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Summary Document
Agent Orange at Johnston Island

On November 21, 1971 the New York Times reported in an article entitled "Defoliant Leaving Vietnam" that more than a million gallons of Agent Orange (AO) will be taken back to the United States from Vietnam to be destroyed. The portion of this operation of re-drumming and movement to Johnston Island, aka Johnston Atoll, was named Project **PACER IVY** (see map and photographs) with the remaining herbicide stocks stored at Gulfport, Mississippi.[1]

During the period from 1972 to 1977, Johnston Island was used for storage of Agent Orange, aka Herbicide Orange (HO). A total of 1.37 million gallons of HO in 26,300 fifty-five gallon drums were transferred to Johnston Island from South Vietnam in 1972. The drums were stored on a 4-acre site on the northwest corner of the Island. ***Corrosion of drums while in storage resulted in HO leakage at a rate of approximately 20 to 70 drums per week*** (Emphasis added). Approximately 49,000 pounds of HO are estimated to have escaped into the environment annually during the storage period with the site contaminated with the active ingredients of HO: 2,3,7,8-tetrachloro-dibenzodioxin (TCDD); the n-butyl ester of 2,4-dichlorophenoxy acetic acid (2,4-D); and the n-butyl ester of 2,4,5-trichlorophenoxyacetic acid (2,4,5-T),[2] in addition to approximately 113,400 kilograms that was accidently spilled.[3]

Shamefully, the deception, fraud and political interference that have characterized government sponsored studies on the health effects of exposure to Agent Orange and/or dioxin has not escaped studies ostensibly conducted by independent reviewers, a factor that has only further compounded the erroneous conclusions reached by the government.[4] As documented in the following paragraphs, with excerpts from United States Government agency reports, the United States Government acknowledges the contamination of the potable water supply at Johnston Island from Agent Orange.

Due to the island's small size, remote location in the central Pacific Ocean, and lack of fresh water, Johnston Island, an unincorporated territory of the United States, was uninhabited and never supported an indigenous or permanent human population.[5] Because of the high permeability of the soil and relatively low precipitation, there are no natural bodies of fresh water (DNA 1994). ***The source of potable water on Johnston Island is from groundwater supplied by up-gradient wells and processed through a reverse osmosis system housed in the Water Treatment Plant*** [Emphasis added] [6]

Agent Orange contaminants have the ability to migrate away from actual locations via river channels and the food chain. [Emphasis added] [7] Unfortunately, if a leak occurs during a rain storm or there is unabsorbed herbicide on the ground during a rain storm, the transport of herbicide to drainage ditches can occur.[8] Far more unfortunate and disconcerting is the late acknowledgement that this scenario was possible, because drainage ditches specifically constructed for water collection are not immune from dioxin migration on an isolated, remote island. The report, written in 1977 was four plus years late in determining that Agent Orange could and did drain into the water collection ditches, thereby contaminating the personnel assigned to Johnston Island. A review of Veterans Administration records of claims filed by individuals

assigned to Johnston Island from 1972-1977 that have contracted "qualified" diseases will confirm exposure.

In the 1991 Brooks Air Force Base report (fourteen years after the Agent Orange stockpile was removed from the Island) the government conceded "The site is now contaminated with the active ingredients of HO: 2, 3, 7, 8-tetrachloro-dibenzodioxin (TCDD); the n-butyl ester of 2, 4-dichlorophenoxy acetic acid (2, 4-D); and the n-butyl ester of 2, 4, 5-trichlorophenoxyacetic acid (2, 4, 5-T)."[9] As late as February 2008, The United States Fish and Wildlife Service website stated that, "…dioxin (Agent Orange), which contaminates at least four acres of land and has migrated to the marine environment."[10] The impact of the effect of contamination was not lost on the Environmental Protection Agency as noted in the Brooks Air Force Base Report: "Other release processes (EPA, 1989a) that may be important are apparent from the fish tissue data. These data suggest that one or both of the following release processes may also be important: leaching of TCDD (and possibly 2,4,3 and 2,4,5-T) from the soil via surface and ground water migration into the ocean; and migration of contaminated soil particles into the ocean due to water drainage."[11]

In 1978, when the Department of Defense decided there was no legitimate domestic use for Agent Orange, they decided to burn thousands of barrels left over from the war at sea off Johnston Island, (Project **PACER HO**). The EPA provided major advice for taking care of the personnel on board the incineration ship, *Vulcanus*. Agent Orange was burned there at over 1,000 degrees C. The EPA 1978 manual said: The highly toxic contaminant present in Herbicide Orange is 2, 3, 7, 8-tetrachlorodibenzo-p-dioxin. The US Air Force has analyzed Herbicide Orange stocks and found TCDD concentrations ranging from 0.05 to 47 ppm [parts per million]; Times Beach was evacuated at 2 ppb—parts per billion. Pooled stocks would have an estimated average TCDD concentration of 1.9 ppm. The principal Herbicide Orange constituent of concern, TCDD, has been found to be highly embryo toxic, teratogenic (tending to cause developmental malfunctions and monstrosities,) and acnegenic and is lethal in the microgram-per-kilogram of body weight range and it presents an unacceptable cancer risk when found in water in parts per quadrillion.[12] The contractor responsible for the clean-up, Parsons, founded in 1944, and is one of the largest 100% employee-owned management, engineering, and construction companies in the United States, with revenues exceeding $3.3 billion in 2006, stated "The contract also entailed excavating, transporting, and stockpiling 15,000 tons of soil contaminated with Agent Orange."[13] ***If the authorized protocol for destruction of dioxin required incineration at over 1,000 degrees Celsius, then a reverse osmosis water treatment plant cannot purify water from dioxin contamination.***

It is obvious from the multiple agency referenced government publications and documents that the United States Government has conceded that Agent Orange was stored on Johnston Island, that Agent Orange leaked into the soil and water supply and contaminated the environment and wildlife. The government has also acknowledged debilitating illnesses to veterans that served in Vietnam and from other countries; Australia, Korea, New Zealand, and Canada for example, were subjected to the same

exposure as those personnel that were on Johnston Island yet continues to deny responsibility for its actions at Johnston Island to its own military personnel.

How can it be conceived that if an entire isolated, remote island with no fresh water supply and its surrounding ecosystem was continuously contaminated with dioxin, that its human inhabitants whom ate, swam and drank the food (including indigenous fish caught) and water prepared with the same dioxin contaminated water that polluted that environment can come away unscathed?

Academic periodical documents within the past two years also substantiate new and continuing issues related to Agent Orange:

1. In two new studies, Vietnam veterans with the highest exposure to herbicides exhibited distinct increases in the prevalence of hypertension, says the committee that wrote the report. The analysis is the seventh update since the early 1990s in a congressionally mandated series by IOM that has been examining evidence about the health effects of these herbicides.[14]
2. Exposure to Dioxins Influences Male Reproductive System, Study of Vietnam Veterans Concludes.[15]
3. Agent Orange Causes Genetic Disturbance in New Zealand Vietnam War Veterans, Study Shows.[16]

To this day, the Veterans Administration has yet to address the issues of Johnston Island as requested by former Representative Lane Evans in his letter to then Veterans Administration director Anthony Principi in 2004[17] and continues to deny medical attention to the victims of Agent Orange exposure on Johnston Island, many of whom I know.

With regards to the dioxin contained in Agent Orange, "***No safe exposure levels have been found.*** (Emphasis added) It has been strongly linked to many cancers and is very harmful to all living things. Chemically known as: 2, 3, 7, 8-tetrachlorodibenzopara-dioxin or 2, 3, 7, 8-T."[18] To quote Admiral Zumwalt, "Since science is now able to conclude with as great a likelihood as not that dioxins are carcinogenic directly and indirectly through immunosuppression, and since a large proportion of those exposed to dioxin can be as ascertained; I am of the view that the compensation issue for service-related illnesses with exposure to Agent Orange should be resolved in favor."[19]

As a final thought, two years after the stockpile of Agent Orange had left Johnston Island in 1977 the United States Air Force contracted with the University of Utah to perform soil and water analysis on samples taken from the island. Five of these samples were of the potable water and contained TCDD, [20] corroborating the 1977 USAF Logistics Command report that the drainage ditches were vulnerable to dioxin runoff. How is it possible that the deadliest toxin created by man as a waste by-product from the paper-pulp industry, that is not naturally occurring, can find itself in a "purified, potable water system" on one of the worlds most isolated, remote locations?

Summary Document
Agent Orange at Johnston Island

FOOTNOTES

[1.] THE LIBRARY OF CONGRESS CONGRESSIONAL RESEARCH SERVICE MAJOR ISSUES SYSTEM, June 25, 1982, Agent Orange: Veterans' Complaints Concerning Exposure To Herbicides In South Vietnam Issue Brief Number Ib80040, Author: Pamela W. Smith, Science Policy, Research Division page CRS 5-7. (REF A)

[2.] OCCUPATIONAL AND ENVIRONMENTAL HEALTH DIRECTORATE, Preliminary Public Health, Environmental Risk, And Data Requirements Assessment For The Herbicide Orange Storage Site At Johnston Island, Brooks Air Force Base, Texas 78235-5000, October 1991, page145. (REF B)

[3.] Letter from Lane Evans, Ranking Democratic Member to Honorable Anthony J. Principi, Secretary, Department of Veterans Affairs, Washington, DC 20420, 15 July 2004. (REF C)

[4.] DEPARTMENT OF VETERANS AFFAIRS, (CLASSIFIED) Report To Secretary Of The Department Of Veterans Affairs On The Association Between Adverse Health Effects And Exposure To Agent Orange (C), Admiral E.R. Zumwalt, United States Navy, May 5, 1990, page 13. (REF D)

[5.] DEFENSE THREAT REDUCTION AGENCY (DTRA), Corrective Measures Study/Feasibility Study (CMS/FS), February 2002, Introduction, pages 1-2. (REF E)

[6.] DEFENSE THREAT REDUCTION AGENCY (DTRA), Corrective Measures Study/Feasibility Study (CMS/FS), February 2002, Annex G GROUNDWATER SURVEY, Section G-4 Environmental Setting—Groundwater at Johnston Island, page G3. (REF E)

[7.] DEPARTMENT OF VETERANS AFFAIRS, (CLASSIFIED) Report To Secretary Of The Department Of Veterans Affairs On The Association Between Adverse Health Effects And Exposure To Agent Orange (C), Admiral E.R. Zumwalt, United States Navy, May 5, 1990, page 16. (REF D)

[8.] AIR FORCE LOGISTICS COMMAND, Programming Plan 75-19 for the Disposal of Orange Herbicide, Prepared by San Antonio ALC, April 1977, Annex 8, page 8-2. (REF F)

[9.] OCCUPATIONAL AND ENVIRONMENTAL HEALTH DIRECTORATE, Preliminary Public Health, Environmental Risk, and Data Requirements Assessment For

The Herbicide Orange Storage Site At Johnston Island, Brooks Air Force Base, Texas 78235-5000, October 1991, page145. (REF B)

[10.] http://www.fws.gov/Refuges/profiles/index.cfm?id=12515 (REF G)

[11.] OCCUPATIONAL AND ENVIRONMENTAL HEALTH DIRECTORATE, Preliminary Public Health, Environmental Risk, And Data Requirements Assessment for the Herbicide Orange Storage Site at Johnston Island, Brooks Air Force Base, Texas 78235-5000, October 1991, page 58. (REF B)

[12.] EPA COLLUSION WITH INDUSTRY, "Testimony to the US Environmental Protection Agency (EPA) Presented At Its Hearing of December 14, 1994, Concerning the Reassessment of Dioxin." A Very Brief Overview, Liane C. Casten, Synthesis/Regeneration 7-8, Summer 1995. (REF H)

[13.] http://www.parsons.com/construction/environmental-remediation-and-restoration/projects/johnston-atoll.asp (REF I)

[14.] National Academies Institute of Medicine, July 27, 2007. (REF J)

[15.] http://www.sciencedaily.com/releases/2006/11/061116081851.htm (REF K)

[16.] http://www.sciencedaily.com/releases/2007/04/070419103733.htm (REF L)

[17.] Letter from Lane Evans, Ranking Democratic Member to Honorable Anthony J. Principi, Secretary, Department of Veterans Affairs, Washington, DC 20420, 15 July 2004. (REF C)

[18.] Gary D. Moore, Chairman, Michigan Agent Orange Commission, 5161 Howard Road, Smiths Creek, Michigan 48074-2023. (REF M)

[19.] DEPARTMENT OF VETERANS AFFAIRS, (CLASSIFIED) Report To Secretary Of The Department Of Veterans Affairs On The Association Between Adverse Health Effects And Exposure To Agent Orange (C), Admiral E.R. Zumwalt, United States Navy, May 5, 1990, page 17. (REF D)

[20.] UNIVERSITY OF UTAH, Letter from William H. McClennen to Major Alvin Young, Brooks Air Force Base, Texas, 7 November 1979, page 2. (REF N)

AGENT ORANGE: VETERANS' COMPLAINTS CONCERNING EXPOSURE TO HERBICIDES IN SOUTH VIETNAM

ISSUE BRIEF NUMBER IB80040

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ISSUE DEFINITION

From 1962 to 1971, the United States Air Force (USAF) sprayed various herbicide mixtures (chemicals that kill plants) in South Vietnam. The purpose of the spraying was to defoliate jungle growth to deprive the Communist forces of ground cover, and to destroy enemy crops to restrict food supplies. The most extensively used of these herbicide mixtures was known as Agent Orange, a 50:50 mix of two common herbicides called 2,4,5-T and 2,4-D (2,4,5-trichlorophenoxyacetic acid and 2,4-dichlorophenoxyacetic acid). A third chemical present in the mixture in small amounts was TCDD, an inevitable by-product of the manufacture of 2,4,5-T. This chemical, called tetrachlorodibenzo-para-dioxin or simply "dioxin," is highly toxic to laboratory animals when administered in its pure form. Acute (short-term) toxicity values in humans have not been established, although Gosselin et al., in the 1976 edition of Clinical Toxicology of Commercial Products, puts TCDD in a class of chemicals for which the "probable lethal dose" for humans would be less than 5 mg/kg, or about 7 drops for a 150 lb (70 kg) person.

CRS has been unable to locate any report of a human death from exposure to pure TCDD. The human health effect that has been most consistently documented following exposure to small amounts of TCDD as a contaminant in other compounds is a skin condition known as chloracne. There is other, less consistent, evidence of damage to the liver and the nervous system in humans. Extensive testing on laboratory animals has been done to determine possible long-term effects of exposure to TCDD. It can induce cancer in some strains of rats and mice (carcinogenicity), cause fetal death in several species (fetotoxicity) and birth defects in developing mouse fetuses (teratogenicity), but has been found not to cause genetic changes in mammalian cells (mutagenicity). The American Medical Association's Council on Scientific Affairs concluded that "there is no scientific evidence that 2,4-D, 2,4,5-T or TCDD has caused reproductive difficulties or hazards in the human."

Congressional interest was triggered by receipt of reports from Vietnam veterans who believed they had been harmed by exposure to herbicides, particularly Agent Orange. The 96th Congress held numerous hearings on the use of herbicides in South Vietnam, and various initiatives to deal with the problem were introduced. P.L. 96-151 was enacted to direct the Veterans Administration (VA) to conduct an epidemiological study on Vietnam veterans to determine whether there may be adverse human health effects associated with exposure to phenoxy herbicides and/or dioxin. This study and other studies planned will help elicit answers to the scientific questions posed by the Veterans Administration in determining whether or not the veterans' medical problems, allegedly due to exposure to Agent Orange and associated herbicides used in Vietnam, are compensable. Following recommendations made by the Interagency Work Group on Phenoxy Herbicides (now the Agent Orange Working Group), legislation was introduced in the 97th Congress to expand the scope of the VA's epidemiological study of the health effects of Agent Orange to include other factors related to military service in Vietnam. The legislation also allows veterans with medically certifiable conditions that might possibly have been caused by exposure to Agent Orange to receive medical care in VA facilities. The bill (H.R. 3499) was considered by the House and Senate in June 1981, put into final form in October, and signed by the President Nov. 3, 1981. Its title is the Veterans' Health Care, Training, and Small Business Loan Act of 1981 (P.L. 97-72).

BACKGROUND AND POLICY ANALYSIS

History

During the summer of 1969, the first reports of human birth defects allegedly attributed to Agent Orange appeared in Vietnamese newspapers. Based on these allegations and the results of a study sponsored by the National Cancer Institute that showed that 2,4,5-T contaminated with TCDD caused birth defects in laboratory animals, the USAF stopped spraying 2,4,5-T in South Vietnam by early 1971.

Although the Department of Defense maintains that only a limited number of U.S. military personnel can be positively identified as having been exposed to 2,4,5-T in South Vietnam (i.e., crews of aircraft that were used to spray herbicides), it is theoretically possible that large numbers of both military personnel (from the United States, South Vietnam, North Vietnam, Australia, and New Zealand) and civilians (especially South Vietnamese peasants) were exposed to 2,4,5-T through the USAF spraying program. A growing number of U.S. veterans who served in South Vietnam have begun to attribute the cause of various chronic ailments which they are now experiencing (especially nervous disorders, cancers, and birth defects in their offspring) to exposure to 2,4,5-T in South Vietnam, and many have filed claims with the VA for compensation. The VA has not yet awarded compensation to veterans for any claims related to 2,4,5-T exposure because of the lack of valid human data to prove a cause and effect relationship between exposure to 2,4,5-T and/or TCDD and specific health effects (except for chloracne).

TCDD Contamination

The industrial production of 2,4,5-T always results in some TCDD contamination although TCDD levels can be reduced to about 0.01 parts per million (ppm) with current technology. Because it was not widely recognized until the late 1960s that 2,4,5-T could contain hazardous amounts of TCDD, manufacturers did not start reducing the level of TCDD in 2,4,5-T until the USAF was already winding down its herbicide spraying program. The average TCDD levels in the 2,4,5-T - containing herbicide mixtures used in South Vietnam were approximately 2 ppm in Agent Orange (which accounted for approximately 96% of the 2,4,5-T used in South Vietnam), approximately 32.8 ppm in Agent Purple, and 65.6 ppm in Agents Pink and Green (Agents Purple, Pink, and Green contained the remaining 2,4,5-T used in South Vietnam). [The herbicides procured by the USAF were code named after the colored band that was placed around each 55 gallon drum in order to identify the contents.]

Health Effects -- Animal Data

Although TCDD is well established as one of the most toxic chemicals known for acute (short-term) effects, there is no consensus in the scientific community over the chronic (long-term) effects on humans of exposure to low levels of TCDD (such as those levels found in the herbicides used in South Vietnam).

Statistically significant animal experiments have demonstrated that

2,4,5-T containing low levels of TCDD and/or TCDD alone have caused various tumors in mice and rats. A recently-released National Toxicology Program bioassay of TCDD confirms these earlier reports that TCDD is carcinogenic in some laboratory animals. Thymic atrophy (without a corresponding loss in immune function) and severe weight loss have been observed in many species after TCDD exposure. In some species, acute exposure to TCDD can cause liver damage. Birth defects such as cleft palate and kidney abnormalities have been reported in baby mice when the mothers were exposed during pregnancy. A National Toxicology Program animal study of male reproductive effects of exposure to TCDD, however, has failed to reveal a statistically significant increase in reproductive abnormalities in TCDD-exposed animals or birth defects in the TCDD-exposed male animals' offspring. Although there is some experimental evidence that TCDD may cause mutations (changes in the cell's genetic material that may produce birth defects in as-yet-unconceived offspring), these experiments have been few, they have been done mainly on non-mammalian species or in vitro (in test tubes), and they have basically been inconclusive.

Some investigators feel that humans are less sensitive than animals to the toxic effects of TCDD. There is wide variation of responses to TCDD among different species, and the mechanisms of its toxicity and metabolism are not understood. More work needs to be done to clarify whether human exposure to TCDD can produce the same health effects with the same potency as those observed in animal studies.

Health Effects -- Human Data

If a cause and effect relationship is to be scientifically established between human exposure to a chemical and chronic health effects, a study which meets the following minimum criteria must be conducted to prove that such a relationship exists: a group of people (the "study group") must be identified that has already been exposed to the chemical under study (it would help to know the level of exposure); this study group must be large enough to detect chronic effects with statistical significance (to find an effect that occurred in 1 out of 100 people, one would need to examine at least 100 people); a control group must be found that ideally would differ from the study group only by never having been exposed to the chemical under study (thus, any differences in chronic health effects between the study and control groups could be attributed only to exposure to the chemical under study); and, due to the long latency period for many chronic effects, the study and control groups must be followed for as many years after exposure as it takes for the chronic effects to show up (i.e., in carcinogenicity studies, subjects must be followed for a minimum of 10 to 20 years after exposure to the suspect carcinogen). These exacting criteria are not met by most of the studies that have explored the relationship between human exposure to TCDD and/or 2,4,5,-T and subsequent health effects. Only for chloracne has such a cause and effect relationship been well established.

Workers who have been exposed to TCDD and/or 2,4,5-T in industrial explosions or who have had other occupational exposure are frequently found to have a skin condition known as chloracne -- which resembles normal acne except that it is caused by chemical exposure. Chloracne can appear from weeks to months after initial exposure and while mild cases (blackheads) may clear in a matter of months, severe cases (inflammatory lesions and scars) may last up to 30 years after exposure has ceased. While the severity of chloracne is not thought to correlate precisely with the intensity or duration of exposure to TCDD and/or 2,4,5-T, chloracne is associated so

closely with exposure that some scientists argue that patients who have not exhibited chloracne are unlikely to have suffered other toxic effects of TCDD and/or 2,4,5-T exposure.

Studies of these exposed workers have also indicated a variety of other health problems. For example, the United States Air Force Technical Report on the Toxicology, Environmental Fate, and Human Risk of Herbicide Orange and its Associated Dioxin (1978) listed a number of symptoms, signs, or disorders that had been reported after occupational exposure to TCP (trichlorophenol, 2,4,5-T's precursor), 2,4,5-T, or TCDD (see Appendix). As noted, these studies, which reported symptoms associated with human exposure to dioxin, were not conducted in such a way as to prove a cause-and-effect relationship between exposure to TCDD and/or 2,4,5-T and any of these effects, but they may be indicative of such a relationship.

Several of the above studies have focused on investigating cancer rates among exposed workers. These studies do not show a clear cause/effect relationship between carcinogenicity associated with exposure to TCDD and/or 2,4,5-T because very few exposed workers (with the exception of those in Nitro, West Virginia) have been followed for more than ten years (the latency period for most cancers being 15 to 40 years after exposure) and the results have been equivocal. However, they support a continuing suspicion and indicate a need for further study. When the scientific panel of the Interagency Work Group on Phenoxy Herbicides reviewed five research papers by European scientists, it concluded that despite the studies' limitations, they do "show a correlation between exposure to phenoxy acid herbicides and an increased risk of some forms of cancer." A soft-tissue sarcoma study has been proposed that will be conducted jointly by the Armed Forces Institute of Pathology and the National Cancer Institute.

Studies that have been conducted in non-industrial settings have not been able to prove a cause and effect relationship between exposure to TCDD and/or 2,4,5-T and specific health effects. The National Academy of Sciences (NAS) was directed by Congress [P.L. 91-441, sec. 506(c)] to conduct a study on the effects of herbicides in South Vietnam, including health effects. This NAS study, as well as at least three other similar studies that were conducted in South Vietnam during the early 1970s, were unable to find adequate data upon which to reach any conclusions concerning a causal effect between exposure to herbicides and any health effects, including birth defects.

An explosion in a Hoffman-LaRoche chemical plant in Seveso, Italy in July 1976 caused thousands of people to be exposed to varying doses of TCDD as a toxic cloud drifted across the Italian countryside in a cone-shaped pattern about a mile long and half a mile wide. Some 5400 people lived in the two zones most directly affected, with an additional 40,000 people potentially exposed. Animals began to die 2 to 3 days after the incident with over 1,100 animals killed by direct exposure to TCDD. Over 700 people were evacuated from their homes. Chloracne was reported in 187 people, mostly children, and it tended to heal rapidly. Long-term human health effects of exposure to TCDD at Seveso are still being studied. Preliminary findings reported in 1979 by Hoffman-LaRoche revealed that Seveso residents had suffered liver damage but that there was no permanent breakdown in liver function. They also reported that rates of spontaneous abortions, fetal malformations, congenital defects, chromosome aberrations, reactions to infectious disease, and morbidity and mortality were not affected by TCDD exposure. As reported by the American Medical Association's Council on Scientific Affairs, "The most recent progress report on the long-term epidemiologic survey of the residents of the Seveso area emphasizes the preliminary nature of their

findings and reiterates the conclusions of prior investigators. Except for the skin, no organs or body functions were impaired. No derangement of gestation, no fetal lethality and loss, no gross malformations, no growth retardation at term and no cytogenetic abnormalities have yet occurred."

Health effects of domestic use of 2,4,5-T have been kept under surveillance by various Government agencies for some years. In April 1970, the Departments of Agriculture, Interior, and Health, Education and Welfare jointly announced the suspension of certain uses of 2,4,5-T following studies indicating that it was a teratogen. On Apr. 21, 1978, the Environmental Protection Agency (EPA) issued a Rebuttable Presumption Against Registration (RPAR) on 2,4,5-T, finding that the herbicide had exceeded certain risk criteria and inviting comments from interested parties. The RPAR was based on toxicological data from animal studies showing a correlation between 2,4,5-T exposure and cancer and birth defects. One of the comments received was from Alsea, Oregon, claiming that there was a high incidence of miscarriage among area women following spraying of the local forests with 2,4,5-T. EPA investigated this claim and reported its conclusion that the incidence of spontaneous abortion over a 6-year period in Alsea was higher than the rates in two other regions of Oregon that had lower rates of 2,4,5-T usage. Based on the combination of evidence from the animal studies and the Alsea study, EPA announced the emergency suspension of the domestic use of 2,4,5-T on forests, pastures, and rights-of-way on Feb. 28, 1979. The Alsea study has been criticized on methodological grounds by various groups, and its results are rejected by a number of writers. EPA hearings on cancellation of 2,4,5-T began in June 1979. On Mar. 24, 1981, EPA and Dow Chemical requested a recess in the hearing to discuss the possiblity of negotiating a settlement. The recess has been extended while the negotiations continue.

Herbicide Spraying in Vietnam

Approximately 107 million pounds of herbicides were aerially disseminated on 6 million acres of South Vietnam (an area about the size of Connecticut) from January 1962 to February 1971. Approximately 276,000 gallons of Agents Green, Pink, and Purple were sprayed in South Vietnam prior to 1965 when they were replaced by Agent Orange. Approximately 11 million gallons of Agent Orange were then sprayed in South Vietnam -- making it the most widely used herbicide of the war. Ninety percent of Agent Orange was sprayed on 2.9 million acres of inland forests and mangrove forests for defoliation, 8% was sprayed on enemy crops for crop destruction, and the remaining 2% was sprayed around base perimeters, cache sites, waterways, and communications lines.

The Air Force continued to operate its herbicide spraying program in South Vietnam until the late 1960s when the National Cancer Institute released results of an animal bioassay that showed 2,4,5-T to be teratogenic and/or fetotoxic in rodents, and newspapers in South Vietnam started reporting health problems among the rural populations who had been exposed to such herbicides. The Air Force first restricted the use of Agent Orange to areas remote from populations in October of 1969, then stopped all airplane spraying of Agent Orange in early 1970 and all helicopter spraying of Agent Orange by 1971. All remaining herbicide stocks were gathered and stored at either Gulfport, Mississippi or Johnston Island in the Pacific until they were incinerated at sea in 1977.

The following table outlines major military projects involving the handling of Agents Orange, Purple, Pink, or Green in South Vietnam.

MILITARY PROJECTS INVOLVING AGENTS ORANGE, PURPLE, PINK, OR GREEN

PROJECT	DATES	DESCRIPTION
AGILE	1960-68	Selection of herbicides, and development and evaluation of defoliation techniques.
RANCH HAND	1962-71	Aerial spraying of herbicides in South Vietnam.
Various USAF Projects	1962-70	Development and testing of aerial spray equipment.
PACER IVY	1971	Redrumming and movement of surplus herbicide from South Vietnam to Johnston Island.
Air Force Logistics Command Project	1972-77	Maintenance of herbicide inventory and research on options for disposal.
PACER HO	1977	Dedrumming of herbicide inventory and at-sea incineration of Agent Orange.

Each of these projects involved some human exposure to the herbicide 2,4,5-T and its contaminant, TCDD. The difficulty lies in determining who may have been exposed and at what level.

Personnel Exposed

The early trials that were conducted in South Vietnam to improve aircraft spray systems (1960 to early 1962) were conducted by USAF personnel assigned to the Special Aerial Spray Flight Division, Langley AFB, Va. (USAF personnel engaged in the herbicide program did not receive permanent change of station assignments to South Vietnam until 1964 -- thus making it more difficult to track personnel who may have been exposed to herbicides). During late 1962 and early 1963, the Crops Division at Fort Detrick and the USAF Armament Laboratory at Eglin Air Force Base, Florida were involved in efforts to provide improvements in spray system components in support of Operation RANCH HAND.

Most of the personnel involved in the actual handling of herbicide drums were Vietnamese. However, a USAF flight mechanic or crew chief was responsible for ensuring that the aircraft were properly loaded and that the spray systems were functional. Each herbicide aircrew consisted of a pilot and a copilot (both usually officers) and a flight mechanic/spray unit operator (usually enlisted). The aircrews were frequently joined by South Vietnamese and U.S. observers. As noted in a USAF report, "within the aircraft, it was not uncommon to have herbicide leakage from around the numerous hose connections joining the spray tank and pumps with the wing and aft spray booms. In hot weather, the odor of herbicide within the aircraft was decidedly noticeable."

The USAF has data on 6,542 herbicide spraying missions that took place between August 1965 and February 1971 on its "HERBS" computer tape. These data were compiled on a mission-by-mission basis from reports and files in various commands and offices in South Vietnam and the United States. The HERBS tape contains the following data for each mission: date; mission number; location; province and UTM coordinates; type of herbicide (basically, Agents Orange, White, or Blue); quantity of herbicide; area covered; purpose of mission (defoliation, crop destruction, etc.); and type of aircraft (plane or helicopter). The NAS used the HERBS tape in its evaluation of the effects of herbicides on South Vietnam. After evaluating the HERBS data, the NAS concluded that the HERBS tape accounted for approximately 86% of all herbicide operations in South Vietnam and that "despite certain recognized deficiencies," the HERBS tape is "a reliable source for an assessment of the major part of the herbicide operation in South Vietnam" and "is the best and in fact the only available comprehensive computation of the major part of the herbicide operations conducted in the Vietnam war."

When the DOD suspended all use of 2,4,5-T in South Vietnam, the USAF was left with an inventory of 2.22 million gallons of unused Agent Orange (1.37 million gallons which had been shipped to South Vietnam and 0.85 million gallons which were waiting to be shipped at the Naval Construction Battalion Center at Gulfport, Mississippi). In April 1972, the 1.37 million gallons of Agent Orange were moved from South Vietnam to Johnston Island in the Pacific Ocean for storage. The total amount of TCDD in the remaining Agent Orange stock was approximately 44.1 pounds. Problems began to arise in both locations as drums reportedly began to leak and the USAF expressed concern over further leakage problems that could occur if a tornado hit the Mississippi site or if a typhoon hit the Pacific site. After exploring a number of options, the USAF decided to dispose of the Agent Orange by burning it at high temperatures at sea on the Dutch incinerator ship named the "Vulcanus." The Agent Orange was drained from the drums at each site and

transferred to the Vulcanus. The empty drums were then rinsed with diesel fuel and crushed. The rinse fluid was combined with the Agent Orange for incineration at sea. A total of 15,480 drums of Agent Orange were processed at the Mississippi site between May 24, 1977, and June 10, 1977, by approximately 110 USAF officers/technicians from the five Air Logistics Centers of the Air Force Logistics Command (located at Kelly AFB Texas; Hill AFB, Utah; Warner Robbins AFB, Georgia; Tinker AFB, Oklahoma; and McClellan AFB, California). A total of 24,795 drums of Agent Orange were processed at the Johnston Island site between July 27, 1977, and Aug. 23, 1977. Approximately 100 civilian employees hired by a contractor performed the dedrumming process. At both the Johnston Island and Mississippi sites, workers were provided with daily changes of work clothes and some with protective clothing. The Agent Orange was incinerated at sea in the period from July to September 1977. Results of industrial hygiene studies conducted at the time of the disposal operation by the U.S. Air Force (Gulfport) and the Battelle Memorial Institute (Johnston Island) revealed no immediate adverse health effects among the personnel involved in the operation.

Department of Defense Efforts

The USAF has stated that it can now identify 1,264 servicemen who were directly exposed to Agent Orange as they handled herbicide containers and flew spraying missions in South Vietnam. The Air Force has initiated a health effects study of Air Force personnel involved in operation "Ranch Hand," who sprayed Agent Orange in Vietnam. The Department of Defense (DOD) believes that these individuals had at least 1000 times more exposure to Agent Orange than the average ground troops. The epidemiological study will try to determine whether a causal relationship can be established between exposure to the 2,4-D/2,4,5-T mixture and long-term health effects. Although the study was originally scheduled to begin in October 1979, peer review of its protocols forced delays. The University of Texas School of Public Health, the U.S. Air Force Scientific Advisory Board and the Armed Forces Epidemiological Board reviewed the study protocols and recommended modifications. Then the Air Force asked the National Academy of Sciences (NAS) to review the protocols. On May 6, 1980, the NAS announced recommendations that the scope and duration of the study be expanded to increase the likelihood of obtaining definitive data. NAS also expressed concern about the public perception of credibility and impartiality of a study conducted internally by the Air Force. The Interagency Work Group's Scientific Panel, however, has recommended that the study, as designed by the Air Force, be conducted because, despite its limitations, it provides "a focus as to the type of health effects that may possibly occur in other (ground troop) personnel."

The Ranch Hand study is proceeding in several phases and will continue for 20 years. The first phase consists of a detailed medical history questionnaire, which has been administered to the Ranch Handers in their homes by trained interviewers from Louis Harris and Associates. A carefully matched control group, selected from military records held by the Air Force, has also been interviewed. The first data from the questionnaire should be available by mid-summer 1982. Also underway is the second phase of the study, a 3-day series of physical examinations, including a battery of psychological tests, which will be given to both the study group and the controls. The contractor for this phase is Kelsey-Seabold of Houston. The exams are scheduled to be completed by September 1982, with preliminary findings available 2 to 3 months later. Follow-up exams will be conducted at 1, 3, 5, 10, and 20 years. A mortality analysis on the Ranch Hand group is

in progress at the Air Force School of Aerospace Medicine, with data anticipated around August 1982, and a mortality tracking program will be continued throughout the study. Information on the health status of the veterans, as shown by the questionnaires and the physical examinations, will provide data for a morbidity analysis.

Many of the veterans who have filed claims with the VA for compensation for health effects caused by exposure to TCDD in South Vietnam did not hold jobs that caused direct exposure to 2,4,5-T. They claim that their exposure occurred indirectly either by being sprayed with overhead planes (although substances other than herbicides were also sprayed from planes) or by being exposed to 2,4,5-T in the environment. According to the DOD, military personnel did not usually enter areas sprayed with Agent Orange until 4 to 6 weeks after treatment. However, a recent General Accounting Office investigation concluded that a large number of Marines in the I Corps section of Vietnam from 1966-1969 were in, or close to, areas sprayed with Agent Orange on both the day of spraying and within 4 weeks afterward. Some Army units were also close to Agent Orange spraying.

The Department of Defense has recently made progress in identifying ground troops that may have been exposed to Agent Orange. Two Army and one Marine battalion - 31st Engineer Battalion, 2050 troops; 1st Squadron, 9th Calvary (Air Mobil), 2300 troops and 3rd Battalion, 1st Marines, have been identified as being in areas of Agent Orange operations. Exact numbers, locations, and identities of individuals who may have been sprayed are impossible to determine.

Veterans' Problems and Veterans Administration Efforts

The first reports of veterans' concerns over health effects of exposure to 2,4,5-T began to appear in late 1977 and early 1978, following media coverage of several veterans' claims. Veterans have associated a number of illnesses with exposure to 2,4,5-T, including skin conditions, fatague, nervousness, numbness in extremities, vision and/or hearing impairments, birth defects in offspring, reduced libido, miscarriages, impotency, respiratory problems, gastro-intestinal tract disturbances, and various cancers, as well as a variety of other illnesses.

As of Apr. 1, 1982, the VA had received 13064 claims for damage reportedly related to in-service exposure to herbicides; 2986 claims have been made due only to exposure to the herbicides and not for any specific condition; 10078 claims have been filed for specific conditions related to herbicide exposure, but 3469 of these have not had the diagnosis confirmed by medical authority. Of the 6609 claims with a confirmed diagnosis, 923 (13.7%) have been allowed for reasons other than Agent Orange exposures and 5686 (86.3%) have been denied. Approximately 93% or 858 of the total 923 claims allowed were for service-connected skin conditions, and the remaining 7% or 65 claims were allowed for cancer, psychiatric and neurological conditions, and various other miscellaneous disabilities. The 5686 claims denied fall into the following categories (many claims have more than one claimed diagnosis): 3055 for various skin conditions; 2335 for nervousness, headache, or fatigue; 886 for paralysis or numbness; 751 for gastro-intestinal or genito-urinary conditions; 399 for various malignancies; 356 for impaired sexual activity; 394 for eye, ear, nose, and throat conditions; 274 for lung conditions; 227 for cardiovascular conditions; and 137 for miscellaneous conditions. The VA has not awarded compensation for the claims of chronic illnesses related to Agent Orange exposure because of the lack of valid human data to prove a

cause and effect relationship between exposure to a 2,4,5-T/2,4-D mixture and/or TCDD and specific chronic health effects. Previously, the difficulty of determining which veterans were or were not exposed to Agent Orange was also a factor in denying compensation, but more recently the VA has conceded exposure for all veterans who were in Vietnam.

The VA is maintaining a registry of all Vietnam veterans who have come to VA hospitals and health care facilities expressing concern about possible herbicide-related health problems. Each such veteran, whether experiencing any health problems or not, is given a physical examination; currently, some 2700-2800 exams are being conducted each month. Data from all the exams is being computerized into a central Agent Orange Registry in addition to the individual records being maintained at the local VA facilities. As of Mar. 25, 1982, 81,670 veterans had received the initial exam, and about 61,000 of the records had been coded into the computer. Information from the registry is being analyzed to determine if the veterans have an increased rate of any particular diseases. Thus far, nothing unusual or unexpected has turned up. Treatment of any health problems uncovered by the exams is handled under normal VA procedures regarding service-connection, ability to pay for medical care, etc., with the exception that special guidelines have been issued for the handling of conditions possibly related to Agent Orange. In the Federal Register of Dec. 2, 1981, pursuant to Public Law 97-72, the VA issued guidelines for use by its physicians to "assist them in making determinations in individual cases as to whether a disability may have been caused" by exposure to Agent Orange. Even though treatment may be given for some conditions, the VA specified that "In accordance with congressional intent, a determination to furnish care under this authority does not establish that the condition for which medical care is provided is service-connected" for purposes of compensation or vocational rehabilitation eligibility.

Three additional VA activities on Agent Orange include participation in the tissue registry, the Chloracne Task Force, and investigations into TCDD residues in body fat tissue of veterans. When VA facilities perform surgery or autopsies on Vietnam veterans, tissue samples are taken and sent to the Armed Forces Institute of Pathology where a special tissue registry is being maintained. Examination of approximately 800 specimens has so far shown no significant clustering of tumors or other particular disease features. The Chloracne Task Force was established in response to a congressional request to sift out those cases of skin conditions that either resemble or are truly chloracne. Those veterans whose medical records show a definite possibility of chloracne will be invited to come to non-VA clinics for re-examination by dermatologists who have an expert knowledge of the disease. The VA has conducted a study to determine if TCDD can be detected in the body fat tissues of Vietnam veterans at any higher levels than in veterans who were not in Vietnam. Dioxin in body fat is measured in parts per trillion, levels which are at the technological limits of available detection methods. The test requires surgical removal of tissue from the abdomen and chemical analysis of the sample on gas chromatography/high resolution mass spectrometry instruments. The results of the study were inconclusive, and the VA has decided that the reliability of the procedure is not sufficient to warrant its use in attempting to verify dioxin exposure. An additional problem is that dioxin contamination is so ubiquitous (from domestic herbicide use and from its formation in municipal incinerators) that it may likely be found in everyone's fat tissue.

As mandated in P.L. 96-151, the Veterans' Affairs Amendments, the VA is currently preparing to perform an epidemiological study of Vietnam veterans exposed to Agent Orange. Although the study's protocol has been developed

and validated by an independent group, the VA will perform the testing and collect the data, with oversight by a non-VA scientific committee. Procurement of an independent contractor for the study's protocol was delayed for 14 months by a protest filed by the National Veterans Law Center (NVLC). The NVLC alleged that not only was the VA violating procurement law, but also the study as currently contemplated did not comply with the requirements of P.L. 96-151. On Feb. 2, 1981, the General Accounting Office concluded its investigation and denied the NVLC protest. On May 5, 1981, the VA announced the awarding of a contract to the University of California at Los Angeles (UCLA) School of Public Health for the design of the epidemiological study. UCLA submitted its first draft of the protocol to the VA in August 1981; it was peer-reviewed by the VA Advisory Committee on Health-Related Effects of Herbicides, by the Office of Technology Assessment, and by the Science Panel of the Agent Orange Working Group. All the review groups judged the draft protocol to be inadequate and not in compliance with the contract. UCLA has since modified the protocol, expanding on problem areas and incorporating the suggestions of the review groups; its final submission to the VA is due April 29, 1982. As with the Ranch Hand study, this epidemiological study will have two main parts: a questionnaire on health status and medical and occupational history, and a physical exam with laboratory workup. The study group will be 18,000 veterans, divided into 3 cohorts of 6000 each. Two of the cohorts will have had Vietnam service, and will be distinguished as having a high or a low likelihood of herbicide exposure. The third cohort will be veterans with non-Vietnam military service. Inclusion of the third group will generate data about the health effects of Vietnam service in addition to the information expected about herbicide-related health effects. The study will commence with a pilot project to field test its procedures and the questionnaire.

P.L. 96-151 also mandated the VA to conduct a comprehensive review and scientific analysis of the worldwide literature on Agent Orange and other phenoxy herbicides. JRB Associates prepared the review under contract, and the VA published the 2-volume study in October 1981. The VA is now preparing to contract for an update to the literature review, to reflect new reports and data that have appeared.

The Interagency Work Group on Phenoxy Herbicides and Contaminants, established in December 1979, recommended that the Centers for Disease Control (CDC) perform a case-control study to see if there is an increased incidence of specific malformations in children of Vietnam veterans. The population to be studied is a group of 7500 children who have birth defects and who are registered in CDC's Birth Defects Program (in operation since the late 1960s). Information on the families of these children, gained by extensive interviews and questionnaires, will be compared with that for 300 normal controls. The data will be analyzed to see what risk factors in the parents' lives, including military service in Vietnam, may be related to increased incidence of malformations in their children. CDC has completed a pilot study on a representative sample of the two groups to test the questionnaire and the procedures for finding the families. The main study will be started in late April 1982, and a preliminary report on the issue of Vietnam service is expected in the fall of 1983. Detailed analysis of the data on all risk factors will take several years to complete.

On Sept. 22, 1980, the Work Group held its first public meeting to discuss problems and proposals related to exposure to herbicides. On Jan. 19, 1981, the Secretary of Health and Human Services established the "Advisory Committee on Special Studies Relating to the Possible Long-Term Health Effects of Phenoxy Herbicides and Contaminants" to advise the Secretary and

the Chair of the Interagency Work Group on Herbicides concerning the Advisory Committee's oversight of the conduct of the Ranch Hand Study being conducted by the Air Force. In its seventh report to the White House, the Work Group's Scientific Panel concluded that:

> While it is difficult to accept logically that a single causative factor -- Herbicide Orange -- could be responsible for such a diverse set of health effects [as alleged by Vietnam veteran claims to the VA], there is no definitive evidence that permits selective exclusion of some of these illnesses. Further, it is possible that some of these health effects are occurring as a consequence of Vietnam service but not due to exposure to Herbicide Orange. The Science Panel is not aware of any data that suggest a modification of its previous recommendation that the focus of a study of Vietnam veterans should be broadened to consider Vietnam service as the exposure factor rather than focus solely on Herbicide Orange exposure.... The Science Panel is in receipt of data which indicate that there is at best a remote chance of accurate identification of specific ground troops who were exposed to Herbicide Orange.... The Panel is therefore of the opinion that design of a scientifically valid Herbicide Orange study of ground troops may not be possible. If the focus of a study of Vietnam veterans is broadened to consider Vietnam service as the exposure factor, a study of ground troops is necessary and a scientifically valid study can be designed.

On July 17, 1981, the Interagency Work Group was renamed and its membership expanded. Now called the Agent Orange Working Group, it is part of the Cabinet Council on Human Resources. The Department of Health and Human Services is the lead agency.

Because the VA currently recognizes only chloracne as a human health effect that can be proven to be caused by exposure to 2,4,5-T, veterans may have difficulty being compensated for even those effects for which there is strong animal evidence (i.e., cancer and birth defects caused in utero which are those birth defects that cannot be caused by the father and require the mother and fetus to be exposed during the actual pregnancy). Veterans who claim compensation for health effects which are not supported by strong animal data (i.e., mutations -- which could cause genetic defects in the father's sperm that would affect children conceived after exposure) may have an even tougher case to argue.

The veteran's question then becomes: How much evidence is required to prove the right to compensation? On whom does the burden of proof lie (the veteran or the VA)? If more evidence is needed, who will generate it? And finally, what constitutes fair treatment of veterans while the necessary data are being gathered?

Congressional Action of the 96th Congress

The 96th Congress responded to the problems of establishing a cause and effect relationship between veterans' exposure to herbicides in South Vietnam and the various health problems they are now experiencing by holding hearings and enacting legislation.

The Subcommittee on Oversight and Investigations of the House Committee on Interstate and Foreign Commerce held hearings on June 24 and 25, 1979, to hear testimony from veterans who allegedly have been affected by herbicide exposure and from the Veterans Administration regarding its efforts to unequivocally determine the relationship between herbicide exposure and health effects. The Subcommittee on Medical Benefits and Facilities of the House Committee on Veterans' Affairs held two sets of hearings on the hazards associated with TCDD, veterans' complaints of health effects associated with Agent Orange exposure, and Veterans Administration's efforts to resolve the Agent Orange problem.

The Senate Veterans' Affairs Committee also held hearings to examine the Agent Orange problem.

As a step to gain access to records to locate veterans who may have been exposed to herbicides in-service, Title V of H.R. 2282, the Veterans' Disability Compensation and Survivors' Benefits Amendments of 1979, requires the Director of the National Institute for Occupational Safety and Health, upon request by the VA (or other appropriate agency) to request the current mailing address from the Internal Revenue Service of persons whom the VA certifies may have been exposed to occupational hazards. H.R. 2282 was passed in lieu of its companion bill, S. 689, and became Public Law 96-128 on Nov. 28, 1979.

Title III of H.R. 3892, the Veterans' Affairs amendments, directs the Veterans Administration to conduct an epidemiological study of the long-term health effects on individuals from exposure to dioxins in Vietnam, upon the Office of Technology Assessment's (OTA) approval of its protocol. Its companion bill, S. 1039, was incorporated in H.R. 3892 as an amendment, and the measure was enacted by Congress and signed by the President on Dec. 20, 1979 (P.L. 96-151).

If enacted, S. 2096 would have directed the Secretary of Health, Education, and Welfare (now, Health and Human Services) to undertake an epidemiological study to determine the long-term adverse human health effects associated with exposure to dioxins produced during the manufacture of phenoxy herbicides. This bill proposed to investigate the long-term health effects of exposure to dioxins, in general, not just to Agent Orange. As similarly incorporated in H.R. 3892, S. 2096 would have required that the study's protocol be approved by the Congressional Office of Technology Assessment. This bill was presented to the President on Dec. 21, 1979, and vetoed by him on Jan. 2, 1980. President Carter vetoed the bill because the White House counsel believed that such a procedure violated the separation of power between the legislative branch and the executive branch. He did not feel that the Department of Health and Human Services' study protocol should be subject to approval by a congressional agency.

Title X of H.R. 5288, the Veterans' Rehabilitation Program and Veterans' Educational Assistance Program would have directed the Secretary of Health and Human Services to conduct a study of veterans and other groups exposed to the herbicide known as "Agent Orange" to determine if there may be adverse health effects associated with such exposure. Like H.R. 3892 (P.L. 96-151) and S. 2096, the bill called for OTA approval of the study's protocol. The bill also would have required the Secretary of Health and Human Services to coordinate its efforts with other studies in the Federal Government. During the debate on S. 1188, its companion bill, the Disabled Veterans' Rehabilitation Act, the Senate adopted an amendment offered by Senator

Cranston to expand the study on health effects of exposure to Agent Orange to include other factors related to service in Vietnam. The Senate also adopted an amendment offered by Senator Heinz requiring the VA to promulgate regulations regarding guidelines to resolve veterans' disability claims based on exposure to Agent Orange. The amendments were striken by the House because they were considered to be "non-germane" to the primary focus of the bill.

S. 1872 (the Vietnam Veterans' Act); H.R. 6050 (the Vietnam Veterans' Act); H.R. 6377 (the Vietnam Era Veterans Agent Orange Act); each would have established a presumption of service-connected disability for health effects in Vietnam veterans (and birth defects in their children) exposed to Agent Orange. H.R. 8238 (Independent Agent Orange Study) would have directed the Veterans Administrator to request the National Academy of Sciences to conduct a study on veterans exposed to Agent Orange. H.R. 8300 would have expanded the scope of the Agent Orange study currently being coordinated by the VA and would have established deadlines for promulgating regulations related to Agent Orange exposure claims. These bills received no action.

LEGISLATION

P.L. 97-72, H.R. 3499

Veterans' Health Care, Training and Small Business Loan Act of 1981. Amends title 38, U.S. Code, to extend the Vietnam-era veterans' readjustment counseling program, to provide medical care for Vietnam veterans exposed to herbicide defoliants (including Agent Orange), to recover the cost of certain health care provided by the VA, and authorizes the VA to expand the scope of its epidemiological study on the health effects of Agent Orange, and other purposes. Introduced May 7, 1981; referred to Committee on Veterans' Affairs. Committee consideration and mark-up session held May 12. Reported to House (amended) by Committee on Veterans' Affairs (H.Rept. 97-79) May 19. Passed House (amended) June 2, 1981. Received in the Senate June 3. Senate struck all after the Enacting Clause and substituted the language of S. 921, June 16. Passed Senate in lieu of S. 921 with amendments, June 16, 1981. House concurred in Senate amendments with amendments Oct. 2, 1981. Senate agreed to House amendments Oct. 16, 1981. Signed into law Nov. 3, 1981.

H.R. 523 (Roe)

Amends Title 38, U.S. Code, to waive the 1-year limitation on claims for compensation from the Veterans Administration for disabilities and diseases incurred in or aggravated by military service in the case of claims by veterans who served in Southeast Asia during the Vietnam era for compensation for disabilities resulting from exposure to the phenoxy herbicides known as Agent Orange or other phenoxy herbicides. Introduced Jan. 5, 1981; referred to Committee on Veterans' Affairs.

H.R. 1173 (Montgomery, by request)

Amends section 307 of P.L. 96-151, by assigning the responsibility of designating a protocol for, and conducting an epidemiological study of, veterans who were exposed to Agent Orange, to an independent scientific agency. Introduced Jan. 22, 1981; referred to Committee on Veterans' Affairs.

H.R. 1962 (Gilman)

Amends the Veterans Health Programs Extension and Improvement Act of 1979 to require the Veterans Administration and the National Academy of Sciences to enter into an agreement under which the Academy will conduct an epidemiological study of veterans exposed to Agent Orange. Introduced Feb. 19, 1981; referred to Committee on Veterans' Affairs.

H.R. 2157 (Mottl)

Expands the scope of a study required to be conducted by the Administrator of Veterans' Affairs concerning the effect on humans of exposure to the chemical known as Agent Orange. Introduced Feb. 25, 1981: referred to Committee on Veterans' Affairs. VA requested Executive comment Mar. 2, 1981. Referred to Subcommittee on Hospitals and Health Care Apr. 28. Hearings held Apr. 30. Subcommittee consideration and mark-up session held. Clean bill forwarded to full committee.

H.R. 2297 (Downey)

Amends Title 38, United States Code, to waive the 1-year limitation on claims for compensation from the Veterans Administration for disabilities and disease incurred in or aggravated by military service in the case of claims by veterans who served in Southeast Asia during the Vietnam era for compensation for disabilities resulting from exposure to the phenoxy herbicides known as "Agent Orange" or other phenoxy herbicides. Introduced Mar. 4, 1981; referred to Committee on Veterans' Affairs.

H.R. 2493 (Daschle)

Amends Title 38, United States Code, to provide a presumption of service connection for the occurrence of certain diseases in veterans who were exposed to herbicides in Southeast Asia during the Vietnam era. Introduced Mar. 12, 1981; referred to Committee on Veterans' Affairs.

H.R. 2953 (Daschle)

Entitles veterans exposed to Agent Orange during the Vietnam era to specified medical benefits. Extends the period during which veterans of such era may initially request psychological readjustment counseling. Extends specified educational assistance without delimiting periods for vocational training for specified veterans determined to be in need of such assistance. Introduced Apr. 1, 1981; referred to Committee on Veterans' Affairs. Referred to Subcommittee on Hospitals and Health Care Apr. 28. Hearings held Apr. 28. Subcommittee consideration and mark-up session held Apr. 30, 1981.

H.R. 3163 (Railsback)

Requires the Secretary of Health and Human Services to arrange for an independent epidemiological study of persons exposed to Agent Orange. Introduced Apr. 8, 1981; referred to Committee on Energy and Commerce. Referred to Subcommittee on Health and the Environment Apr. 9, 1981.

S. 636 (Cranston et al.)

Entitles the United States to recover the costs of certain medical care and services furnished to a veteran for a non-service-connected disability when disability is covered by another form of insurance or compensation. Permits the expansion of the scope of the epidemiological and literature

study of the long term adverse health effects of exposure to Agent Orange during the Vietnamese conflict to include the effects of other factors. Introduced Mar. 5, 1981; referred to Committee on Veterans' Affairs.

S. 689 (Heinz)

Amends section 307 of the Veterans Health Programs Extension and Improvement Act of 1979 to require the promulgation of regulations containing guidelines for resolving claims for veterans benefits based on exposure to Agent Orange, and for other purposes. Introduced Mar. 12, 1981; referred to Committee on Veterans' Affairs. Hearings held Apr. 30, 1981.

S. 921 (Simpson)

Extends the authority of the Administrator of Veterans' Affairs to contract for hospital care or medical services in Puerto Rico and the Virgin Islands without reference to patient loads or incidence of provision of medical services for veterans treated by the Veterans' Administration in the contiguous 48 States. Introduced Apr. 8, 1981; referred to Committee on Veterans' Affairs. Reported with amendment May 15, 1981 (S.Rept. 97-89); H.R. 3499 passed in lieu (see P.L. 97-72 above) June 16, 1981.

S. 1345 (Heinz)

Authorizes the Administrator of the Veterans' Administration to provide hospital or nursing home care to a veteran for treatment of a condition associated with exposure to Agent Orange during service in Vietnam. Extends the Vietnam-era veterans' readjustment counseling program. Directs the Administrator to expand the scope of the epidemiological study of long term adverse health effects of other factors involved in such service. Introduced June 8, 1981; referred to Committee on Veterans' Affairs.

S. 1953 (Specter)

Amends title 38, United States Code to provide a presumption of service connection for the occurrence of certain diseases in veterans who were exposed to phenoxy herbicides while serving in Southeast Asia during the Vietnam era. Introduced Dec. 15, 1981; referred to Committee on Veterans Affairs.

HEARINGS

U.S. Congress. House. Committee on Interstate and Foreign Commerce. Subcommittee on Oversight and Investigations. Agent Orange: exposure of Vietnam veterans. Hearing, 96th Congress, 2d session. Sept. 25, 1980. 249 p.

----- Involuntary exposure to Agent Orange and other toxic spraying. Hearings, 96th Congress, 1st session. June 26 and 27, 1979. 256 p.

U.S. Congress. House. Committee on Veterans' Affairs. Ad Hoc Subcommittee. Status of Vietnam veterans in the Bay area. Hearing, 96th Congress, 2d session. Apr. 10, 1980. 64 p.

U.S. Congress. House. Committee on Veterans' Affairs. Subcommittee on Hospitals and Health Care.

Legislation to improve medical programs administered by the Veterans Administration (H.R. 2157, H.R. 2953, and H.R. 2999). Hearings, 97th Congress, 1st session. Apr. 28, 1981. 54 p.

U.S. Congress. House. Committee on Veterans' Affairs. Subcommittee on Medical Facilities and Benefits. Herbicide "Agent Orange". Hearing, 95th Congress, 2d session. Oct. 11, 1978. 62 p.

----- Oversight hearing to receive testimony on Agent Orange. Hearing, 96th Congress, 2d session. Feb. 25, 1980. 121 p.

----- Oversight hearing to receive testimony on Agent Orange. Hearing, 96th Congress, 2d session. July 22, 1980. 459 p.

----- Scientific community report on Agent Orange. Hearing, 96th Congress, 2d session. Sept. 16, 1980. 145 p.

U.S. Congress. House. Committee on Veterans' Affairs. Subcommittee on Oversight and Investigations. Current status of Agent Orange studies. Hearing, 97th Congress, 1st session. May 6, 1981. 385 p.

U.S. Congress. Senate. Committee on Veterans' Affairs. Agent Orange update and appendix: Agent Orange activities (part II). Hearing, 96th Congress, 2d session. Sept. 10, 1980. 1368 p.

----- Oversight on issues related to Agent Orange and other herbicides. Hearing, 97th Congress, 1st session. Nov. 18, 1981. (not yet printed)

----- VA health resources and program extensions and appendix: Agent Orange activities. Hearing, 96th Congress, 1st session, on S. 741 and S. 196. Apr. 10, 1979. 462 p.

----- Veterans' Programs Extension and Improvement Act of 1981. Hearing, 97th Congress, 1st session, on S. 26 (titles II and III, only), S. 380, S. 458, S. 636, S. 689, S. 872, S. 914, S. 921, and related bills. Apr. 30, 1981. 685 p.

----- Vietnam veterans' readjustment. Hearings, 96th Congress, 2d session. Feb. 21, Mar. 4, and May 21, 1980. Part 2. 595-2082 p.

ADDITIONAL REFERENCE SOURCES

American Medical Association. Council on Scientific Affairs. Health effects of "Agent Orange" and dioxin contaminants (Executive Summary). 1981. 6 p.

Behrens, Richard, et al. Comments from CAST (Council on Agricultural Science and Technology): Dioxin. 1978. 9 p.

Buck, Craig. The Death of a nation. New west, v. 3, Aug. 28, 1978: 49-50, 52-55.

Cookson, Clive. "Emergency" ban on 2,4,5-T herbicide in U.S. Nature, v. 278, Mar. 8, 1979: 108-110.

Davis, Donald E. et al. Comments from CAST (Council on Agricultural Science and Technology): Agent orange. 1978. 5 p.

Galston, Arthur W. Herbicides: a mixed blessing. Bioscience, v. 29, February 1979: 85-90.

International Agency for Research on Cancer. IARC monographs on the evaluation of the carcinogenic risk of chemicals to man: 2,5,5-T, vol. 15, August 1977.

JRB Associates. Review of literature on herbicides, including phenoxy herbicides and associated dioxins. Washington, Veterans Administration, 1981. 2v.

Laporte, Joan-Ramon. Multinationals and health: reflections on the Seveso catastrophe. International journal of health services, v. 8, no. 4, 1978: 619-632.

National Research Council. The effects of herbicides in South Vietnam: Part A. Summary and conclusions. Washington, National Academy of Sciences, 1974. AD-774-749. 398 p.

Peterson, Jeannie. Seveso: the event. Ambio, v. 7, no. 5-6, 1978: 232-239.

Reggiani, G. Localized contamination with TCDD - Seveso, Missouri, and other areas. In: Halogenated biphenyls, terphenyls, naphthalenes, dibenzodioxins and related products, ed. Renate D. Kimbrough. Amsterdam, New York, Oxford, Elsevier/North-Holland Biomedical Press, 1980.

Severo, Richard. Seveso: lessons from an escape. Economist, v. 267, June 17, 1978: 101-102, 104-106, 108.

U.S. General Accounting Office. Health effects of exposure to herbicide orange in South Vietnam should be resolved, Apr. 6, 1979. CED-79-22.

----- U.S. ground troops in South Vietnam were in areas sprayed with herbicide orange. Nov. 16, 1979. FPCD-80-23.

Young, Alvin et al. The toxicology, environmental fate and human risk of herbicide orange and its associated dioxin. The Surgeon General, U.S. Air Force, Washington, D.C. October 1978.

Walsh, John. Seveso: the questions persist where dioxin created a wasteland. Science, v. 197, Sept. 9, 1977: 1064-1067.

Whiteside, Thomas. Defoliation. New York, Ballantine, 1970.

----- The pendulum and the toxic cloud. New Haven, Yale

University Press, 1979.

Zimmerman, David R. Agent Orange: Vietnam's lingering poison. American health, v. 1, no. 1, Mar./Apr. 1982: 68-72.

Signs, Symptoms, and Disorders Reported After Occupational Exposure to TCP, 2,4,5-T or TCDD

Source	Headaches	Sensory Nerves and Tracts	Neuralgia or Myalgia	Paresis	Hemorrhage	Porphyria	Hyperpigmentation or Hirsutism	Acne	Fetal Disorders	Cancer	Asthenia	Other Psychiatric	Abdominal Pain or Pressure	Anorexia, Nausea Vomiting, Diarrhea	Death
Baader and Bauer (6)			8	2				17			4				
Bauer, et al. (9)	4[a]	3	6	9			5	8			9	6	5		
Bleiberg et al. (14)						11	18	20							
Poland et al. (62)	8	2		7		1	30	48					+	22	
Dugois et al. (24)	+[b]							17			+		+	+	
Hardell (33)										87					
Kimmig and Schulz (44)				+				31			+				
Kramer (49)	3										4	2			
Jirasek et al. (37)	+		+	+		12	19	78		2	+	+		+	3
Jirasek et al. (38)		+				+						+			
Pazderova et al. (61)			+	+		23	+	53		2	27	8	+	+	3
Miura et al. (54)								+							
Oliver (57)	2	1	1				3	2			3	1	1	1	
Ter Beek et al. (79)	+					+		+			+	+		+	
Zelikov and Danilov (88)								1							
Total number of cases reported[c]	17	6	15	18	0	47	75	275	0	91	47	17	6	23	6

[a]Number entries in table reflect the number of cases in which sign, symptom or disorder was reported. [b]+ = Sign, symptom or disorder reported but number of cases not given.
[c]Numbers do not include cases represented by "+" and totals may represent some double counting due to the overlap to studies by Jirasek et al. and Pazderova et al.

SOURCE: Young, Alvin et al. The Toxicology, Environmental Fate, and Human Risk of Herbicide Orange and Its Associated Dioxin, p. VI-14. (Numbers in parentheses identify sources in Young's bibliography.)

AL-TR-1991-0101

AD-A271 979

ARMSTRONG LABORATORY

PRELIMINARY PUBLIC HEALTH, ENVIRONMENTAL RISK, AND DATA REQUIREMENTS ASSESSMENT FOR THE HERBICIDE ORANGE STORAGE SITE AT JOHNSTON ISLAND

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October 1991

Final Report for Period September 1990 – August 1991

AIR FORCE SYSTEMS COMMAND
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NOTICES

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The Office of Public Affairs has reviewed this report, and it is releasable to the National Technical Information Service, where it will be available to the general public, including foreign nationals.

This report has been reviewed and is approved for publication.

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REPORT DOCUMENTATION PAGE

Form Approved
OMB No. 0704-0188

1. AGENCY USE ONLY (Leave blank)	2. REPORT DATE	3. REPORT TYPE AND DATES COVERED
	October 1991	Final September 1990 - August 1991

4. TITLE AND SUBTITLE Preliminary Public Health, Environmental Risk, and Data Requirements Assessment for the Herbicide Orange Storage Site at Johnston Island

5. FUNDING NUMBERS

C: F33615-87-D-4024/0015

6. AUTHOR(S) Geoffrey Huse, Jeffrey Driver, Scott Baker, Louis Corio, Sally Kamem, Alfred Pinkney, Gary Whitmyre, Michelle Silkowski, Thomas Piccin, Leslye Wakefield, Nica Mostaghim, and Sonja Young

7. PERFORMING ORGANIZATION NAME(S) AND ADDRESS(ES)

RiskFocus
VERSAR Inc.
6850 Versar Center
Springfield, VA 22151

8. PERFORMING ORGANIZATION REPORT NUMBER

9. SPONSORING/MONITORING AGENCY NAME(S) AND ADDRESS(ES)

Armstrong Laboratory
Occupational and Environmental Health Directorate
Brooks Air Force Base, TX 78235-5000

10. SPONSORING/MONITORING AGENCY REPORT NUMBER

AL-TR-1991-0101

11. SUPPLEMENTARY NOTES

12a. DISTRIBUTION/AVAILABILITY STATEMENT

Approved for public release; distribution is unlimited.

12b. DISTRIBUTION CODE

13. ABSTRACT *(Maximum 200 words)*

This report contains the results of a screening-level risk assessment conducted for the Air Force Armstrong Laboratory, Occupational and Environmental Health Directorate, concerning the Herbicide Orange storage site at Johnston Island. The risk assessment is part of a remedial investigation and feasibility study (RI/FS) process established by the U.S. EPA for characterizing the nature and extent of risks posed by hazardous waste sites and for developing and evaluating remedial options. This process is being conducted in the context of the U.S. Department of Defense Installation Restoration Program (IRP).

14. SUBJECT TERMS	15. NUMBER OF PAGES
IRP, Risk Assessment, Herbicide Orange, Agent Orange 2,7,8,8-TCDD, Dioxin, 2,4-D, 2,4,5-T, Johnston Island, Johnston Atoll, RI/FS	308
	16. PRICE CODE

17. SECURITY CLASSIFICATION OF REPORT	18. SECURITY CLASSIFICATION OF THIS PAGE	19. SECURITY CLASSIFICATION OF ABSTRACT	20. LIMITATION OF ABSTRACT
Unclassified	Unclassified	Unclassified	UL

Foreword

This report was prepared under the management and supervision of VERSAR's **RiskFocus** Division located in the Washington, D.C. metropolitan area. **RiskFocus** provides comprehensive *stewardship* for product integrity and registration, worker safety, waste disposal, regulatory interpretation and compliance, and risk communication.

Authorship of this report is credited to Geoffrey Huse, Jeffrey Driver, Scott Baker, Louis Corio, Sally Kamem, Alfred Pinkney, Gary Whitmyre, Michelle Silkowski, Thomas Piccin, Leslye Wakefield, Nica Mostaghim and Sonja Young. Special thanks is given to Sylvia Johnson for word processing and other editorial support. Quality assurance is credited to Gary Whitmyre. Further information about this report may be obtained by writing directly to Robert Tardiff, Director, **RiskFocus** Division or by calling 703/642-6884.

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Ref No 61.5049.15.2

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Preliminary Public Health, Environmental Risk, and Data Requirements Assessment for the Herbicide Orange Storage Site at Johnston Island

Executive Summary

This report contains the results of a screening-level risk assessment conducted for the Air Force Occupational and Environmental Health Laboratory concerning the Herbicide Orange (HO) storage site at Johnston Island (JI). The risk assessment is part of the remedial investigation and feasibility study (RI/FS) process established by the U.S. EPA for characterizing the nature and extent of risks posed by hazardous waste sites and for developing and evaluating remedial options. This process is being conducted in the context of the U.S. Department of Defense (DoD) Installation Restoration Program (IRP).

After the Vietnam war, in April 1972, 1.37 million gallons of unused HO in 24,910 fifty-five gallon drums were transferred to JI and stored on a 4-acre site at the northwest corner of the Island. The HO stored on JI was successfully dedrummed and incinerated at sea in 1977. While stored on the Island, the sea air corroded some of the steel drums, resulting in HO leakage onto the ground and necessitating an active maintenance and redrumming operation at the storage site. It has been estimated that approximately 49,000 pounds of HO

escaped into the environment annually during the period from 1972 to 1977. The HO stock was determined to contain two active ingredients (the n-butyl ester of 2,4-dichlorophenoxy acetic acid (2,4-D) and the n-butyl ester of 2,4,5-trichlorophenoxy acetic acid (2,4,5-T), as well as 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) as a byproduct contaminant of 2,4,5-T. Consequently, through leakage and spillage during maintenance, redrumming, dedrumming, and drum crushing operations, the site was contaminated over a period of six years with 2,4-D, 2,4,5-T, and TCDD. The site has remained essentially untouched since that time.

Objectives of the study. There is some concern that contaminants at the site may be moving offsite into all environmental media: the adjacent air compartment, seawater, sea sediments, and groundwater aquifer that may underlie the site. It follows that if the contaminants are in any or all of these media, humans associated with them and biota contained in them may have a potential for exposure to HO site-derived contaminants and an attendant health risk. Therefore, the site-specific objectives of this investigation are to determine, based on available evidence:

- The potential contaminants at the site;
- The levels of contaminants at the site;
- The potential levels of the contaminants in each offsite environmental compartment;
- The potential levels of exposure to humans and wildlife, and to humans from biomagnification in the food chain; and finally
- The risk of health injury from potential multimedia exposure.

A companion objective is to determine, within the scope of existing environmental regulations, whether the quantified risks fall within acceptable risk limits.

The HO site on JI is a unique environment with exceptionally uneven scientific data (particularly on the monitoring of environmental media) because data collection practices, in accordance with the needs prescribed for a baseline risk assessment, have not been orderly and systematic over the years since HO was stored there and contamination began. As a result, the risk assessment contained in this document includes reasonable conservative assumptions to bridge information gaps where such information is usually present to support the baseline assessment. A more complete baseline risk assessment, suitable for responsible decision-making on remedial alternatives and closure, can be constructed only after additional field data at the HO site are collected.

Chemicals at the site. Thirteen monitoring studies were undertaken during and after disposal of the HO to characterize the site, including sampling of marine biota, ocean sediments, air, and soil. Selected sampling of marine biota have revealed the presence of TCDD. Although sampling has not been systematic and the results are not definitive, 37%, 16%, and 12.5% of the marine biota taken at three sampling sites around the HO site contained measurable quantities of TCDD. Of 38 sediment samples taken between 1985 and 1988, only two have been positive (160 and 190 ppb) above the 50 or 100 ppb detection limit for TCDD. No monitoring has been conducted for 2,4-D and 2,4,5-T in marine sediments and biota.

Air monitoring has occurred in support of the Johnston Atoll Chemical Agent Disposal System (JACADS). Insignificant levels of particle-associated

TCDD were dispersing from the HO site during the sampling period, given that these samplers were downwind of at least the southern portion of the HO site's total surface area, in addition to being downwind of the soil decontamination experiments. However, because of the limited number of samples and the lack of data for the entire downwind area relative to the HO site (i.e., the western fenceline), no conclusions can be made regarding TCDD exposure potential via inhalation of contaminated, airborne particulate at the time the samples where taken in 1986, or particularly prior to 1986, when the site was being used for storage purposes.

The groundwater under the HO site has never been analyzed for HO or dioxin.

Three comprehensive soil characterization activities produced surface and subsurface soil data on 2,4-D, 2,4,5-T, and TCDD throughout the defined waste site and at selected areas around the waste site. These data formed the basis of the risk assessment. The most recent soil study (1984-86) revealed TCDD levels in surface soil ranging from nondetect (0.01 ppb) to 163 ppb, with an average concentration of 0.8 ppb. 2,4-D in surface soil ranges from 2.5 ppb to 281,330 ppb with an average of 49,986 ppb. 2,4,5-T in surface soil ranges from 53 ppb to 237,155 ppb, with an average of 48,914 ppb.

Approximately 25% of the site was sampled for subsurface TCDD in the 3-7 inch layer of subsurface soil. Values ranged from 0.02 ppb to 207 ppb, with an average reading of 15 ppb. Approximately 2% of the site was sampled for subsurface 2,4-D and 2,4,5-T. Values for 2,4-D ranged from 2.5 ppb to 55,070 ppb, with an average reading of 4138 ppb (all but two values were below 44

ppb). Values for 2,4,5-T ranged from 7 ppb to 82,210 ppb, with an average reading of 6210 ppb (two-thirds of the values were below 100 ppb).

Exposure scenarios. Exposure assessment for the HO site included determination of the exposure setting and the exposure pathways that are of particular relevance to the types of human populations present and their respective activity patterns and thus involved characterization of the potentially exposed populations, descriptions of the identified plausible exposure pathways, estimations of human exposure, and identification of uncertainties related to the exposure assessment methods used in this evaluation.

In addition to the current scenario, two future land use scenarios were considered: (1) remediation through excavation and incineration of contaminated soil; and (2) covering of the site with cement.[1] In both of these scenarios, certain activities such as construction vehicles on the site and excavating alter the patterns of particulate suspension and soil volatilization of contaminants from those in the current use scenario. These were incorporated into the calculation of emission factors and exposure estimation. Based on the activities associated with these scenarios and consideration of the currently available soil sampling data, the following potential future exposure pathways were considered for:

- *Future-Use Scenario 1 (Excavation)*: Inhalation of contaminated soil from vehicular traffic, loading and unloading operations during site excavation and treatment, and wind erosion of disturbed soil.

[1]The latter scenario is not intended to be a substitute for prescriptive site capping, which is a more thorough and rigorous form of remediation.

- *Future-Use Scenario 2 (Cement Covering)*: Inhalation of contaminated soil from vehicular traffic and wind erosion of disturbed soil.

Exposure Quantification. Risk to the theoretical maximum exposed individual (MEI) is based on access to any point around the perimeter of the HO site (including the seawall) and selection of the maximum point of exposure around the perimeter. However, in actuality there are certain limitations to where the MEI can be situated because of the restrictions on access to the site. Therefore, risk to an *alternate*, more realistic MEI (a person who has "reasonable maximum exposure"), restricted to the portion of the site boundary that is fenceline and not the inaccessible portion of the site boundary that is seawall, was also calculated for comparison. As a result, risk was calculated for two receptors, the theoretical MEI (TMEI) and the alternate MEI (AMEI).

The Industrial Source Complex (ISC) model was used in a screening mode to conservatively estimate ambient air concentrations of the vapor-phase compounds. A total of 140 ground-level, non-buoyant, point sources were used to represent the area of compound emissions in the modeling. The main HO site was extended westward to the shoreline to include isolated TCDD "hotspots" and this identical area was used for estimating 2,4-D and 2,4,5-T emissions.

Emission rates and exposures were estimated for the current scenario and the two future-use scenarios, taking into account wind erosion, construction, excavation, and vehicular traffic. For both vapor-phase and particulate-bound TCDD, Lifetime Average Daily Dose (LADD) was calculated for the TMEI and AMEI. In similar fashion, Average Daily Dose (ADD) was calculated for 2,4-D, and 2,4,5-T. The results are presented in Table ES-1.

TABLE ES-1

Estimated lifetime average daily absorbed dose (LADD) and average daily absorbed doses (ADD) expressed as mg/kg/day for TCDD, 2,4-D, and 2,4,5-T resulting from inhalation exposure to the TMEI and the AMEI.

CURRENT SCENARIO

Chemical	TMEI		AMEI	
	LADD	ADD	LADD	ADD
TCDD	5.6×10^{-11}	2.3×10^{-10}	5.6×10^{-11}	2.3×10^{-11}
2,4-D		4.1×10^{-6}		1.5×10^{-6}
2,4,5-T		4.5×10^{-6}		2.9×10^{-6}

FUTURE SCENARIO: EXCAVATION

TMEI		AMEI	
LADD	ADD	LADD	ADD
1.5×10^{-12}	1.6×10^{-10}	1.5×10^{-12}	1.6×10^{-10}
----	2.7×10^{-6}	----	1.2×10^{-6}
----	3.0×10^{-6}	----	1.9×10^{-6}

FUTURE SCENARIO: CEMENT COVER CONSTRUCTION

TMEI		AMEI	
LADD	ADD	LADD	ADD
3.5×10^{-13}	7.5×10^{-11}	3.5×10^{-13}	7.5×10^{-11}
----	1.3×10^{-6}	----	5.0×10^{-7}
----	1.5×10^{-6}	----	9.4×10^{-7}

Exposure to contaminated fish. There is TCDD fish contamination in certain areas. The contamination appears to be restricted to the area adjacent to the former HO storage site, which is off-limits to fishing. If contaminated fish migrate into the fishing areas near the former HO storage site, there is a potential for JI inhabitants to consume contaminated fish. For the fish that showed positive TCDD values, the migratory fish species had the lowest values. These values may be low because these fish may not spend all of their time in the contaminated area. It is not possible to quantify this potential exposure because the fishermen's catches have not been sampled. The potential for exposure may be low, but sampling of the fishermen's catches should be performed to confirm this. Sampling at the west wharf has revealed no contaminated fish. This may indicate a low probability of catching a contaminated fish.

Risk assessment. Critical toxicological dose-response data for TCDD, 2,4-D, and 2,4,5-T are presented in Tables ES-2 and ES-3. Application of the slope factors (for carcinogenic effects) and R_fD's (for noncarcinogenic effects) in these tables, representing the toxicity component, to the LADD's and ADD's, respresenting the exposure component, produces estimates of risk. Although all media were considered in the analysis, lack of or inadequate monitoring data on water and marine biota reduced multimedia considerations to air only. For this medium, both vapor phase and chemical-bound particulate were factored into the calculations.

For the *current scenario*, the cancer risk from exposure to TCDD is 3×10^{-5} for the TMEI and 3×10^{-5} for the AMEI. The hazard quotient (for noncarcinogenic risk) from exposure to TCDD is 0.76 for the TMEI and 0.76 for the AMEI. The hazard quotient from exposure to 2,4-D is 0.0014 for the TMEI

TABLE ES-2
Critical Carcinogenic Toxicity Values for Indicator Chemicals

Chemical Name	Slope Factor (SF) $(mg/kg\text{-}day)^{-1}$	Weight of Evidence Classification	Type of Cancer	SF Basis/ SF Source
Oral Route				
2,3,7,8-Tetrachloro-dibenzo-p-Dioxin[a]	1.56×10^5	B1[a]	Lung, liver, hard palate, nasal turbinates	Food/ATSDR
2,4-Dichlorophenoxy acetic acid[b] (n-butyl ester)	No data	No data	No data	No data
2,4,5-Trichlorophenoxy acetic acid[b] (n-butyl ester)	No data	No data	No data	No data
2,4,5-Trichlorophenoxy acetic acid[b] (Iso-octyl ester)	No data	No data	No data	No data
Inhalation Rate	No data	No data	No data	No data

[a] When associated with phenoxy herbicides and/or chlorophenols, B2 when considered alone.

TABLE ES-3
Critical Noncarcinogenic Toxicity Values for Indicator Chemicals

Chemical Name	Chronic R_fD (mg/kg-day)	Confidence Level[a]	Critical Effect	R_fD Basis/ R_fD Source	Uncertainty and Modifying Factors[b]
Oral Route					
2,3,7,8-Tetrachloro-dibenzo-p-Dioxin	1×10^{-9}	No data	Primary: Fetal survival Secondary: Renal	No data/ ATSDR	UF=100 for A, L MF=10
2,4-Dichlorophenoxy acetic acid (n-butyl ester)	1×10^{-2c}	Medium	Primary: Renal Secondary: Hematologic, hepatic	Food/ IRIS	UF=100 for H, A MF=1
2,4,5-Trichlorophenoxy acetic acid (n-butyl ester)	1×10^{-2d}	Medium	Primary: Neonatal survival Secondary: Increased urinary coproporphyrin	Food/ IRIS	IF=300 for H, A, D MF=1
Inhalation Route	No data	No data	No data	No data	No data

[a] Confidence level from IRIS, either high, medium, or low.
[b] Uncertainty adjustments: H=variation in human sensitivity; A=animal to human extrapolation; and D=deficiencies in toxicity data.
[c] R_fD value for acid, n-butyl ester value not available.
[d] R_fD value for acid, n-butyl ester and iso-octyl ester values not available.

and 0.00051 for the AMEI. The hazard quotient from exposure to 2,4,5-T is 0.0015 for the TMEI and 0.00095 for the AMEI.

For the *future-use scenario involving excavation (Scenario 1)*, the cancer risk from exposure to TCDD is 8×10^{-7} for the TMEI and 8×10^{-7} for the AMEI. The hazard quotient from exposure to TCDD is 0.52 for the TMEI and 0.52 for the AMEI. The hazard quotient from exposure to 2,4-D is 0.00090 for the TMEI and 0.00034 for the AMEI. The hazard quotient from exposure to 2,4,5-T is 0.0010 for the TMEI and 0.00063 for the AMEI.

For the *future-use scenario involving paving (Scenario 2)*, the cancer risk from exposure to TCDD is 2×10^{-7} for the TMEI and 2×10^{-7} for the AMEI. The hazard quotient from exposure to TCDD is 0.25 for the TMEI and 0.25 for the AMEI. The hazard quotient from exposure to 2,4-D is 0.00045 for the TMEI and 0.00017 for the AMEI. The hazard quotient from exposure to 2,4,5-T is 0.00049 for the TMEI and 0.00031 for the AMEI.

Ecological effects. Releases of HO have exposed fish and invertebrates and possibly birds to dioxin. Only a rough estimate of risk is possible given the limitations of the data. When possible, risks were assessed by comparing body burdens with levels associated with toxic effects.

The highest concentration of dioxin was reported in the crown squirrelfish. Squirrelfishes tend to remain close to the bottom and do not travel long distances. These behaviors may increase their exposure to localized sources of dioxin in sediments. Out of four samples, TCDD was detected in one sample at 352 ppt and in one sample at 472 ppt. These concentrations exceed the 260 ppt measured in rainbow trout muscle that was associated with decreased growth

and fin lesions. The only other fish species with concentrations exceeding 100 ppt was the yellowfin goatfish. Three samples had concentrations of 11, 85, and 102 ppt. Goatfishes are bottom feeders, which may account for their enhanced body burdens.

Several invertebrate samples were detected at levels between 14 and 28 ppt. The only invertebrate sample detected at greater than 100 ppt was a "snails" sample measured at 120 ppt. No data linking tissue concentrations with effects in snails could be located.

In three samples of birds, there were no detectable concentrations of dioxin.

Data requirements. There has not been a systematic effort in collecting the needed monitoring data at the HO site. To date, the most definitive data-collection activity has been soil characterization. ***In order for a multimedia baseline risk assessment to be considered complete enough to determine whether there is sufficient risk to warrant remediation (including a decision on the best cleanup and closure method from among the range of alternatives), the US Air Force needs to carefully craft a sampling plan and engage in a coordinated sampling and analysis activity[2] to provide the necessary baseline data.*** This is necessary so that:

- The output from the sampling and analysis serves as effective input to the baseline risk assessment;

---

[2] With input from a sampling statistician, marine biologist, and Fish and Wildlife personnel associated with the Island, and in coordination with any other work being done to support JACADS.

- No further analyses will have to be done; and
- The sampling data used to predict exposure and risk are convincing enough to EPA in its decision-making process about clean closure of the site.

The nature of the needed data is described below by medium.

Air - The risk assessment used estimated values for the particulate and vapor phase emissions from the site. Air sampling would characterize the particulates and vapors coming from the site. Particle size distribution will enable determination of the percentage of respirable dust. To determine the wind erosion around the site several Hi-Vol samplers, equipped with particulate traps, could be placed downwind around the fence line. At the southwestern fenceline the odor of 2,4-D was detectable during the site visit, indicating that there may be significant vapor emissions from the site. Organic vapor phase samplers capable of collecting dioxins, 2,4-D, and 2,4,5-T can be placed around the site to characterize ambient air concentrations. There are other potential sources of dioxin on JI, including JACADS, the burn pit, and the fire training area. Sampling would permit source apportionment of dioxin from each of these sites.

Soil - The characteristics of the soil can have an influence on the bioavailability of dioxins and the other chemicals. Soil moisture content, organic content, and particle size distribution are missing elements that are important for lowering the uncertainty in the soil exposure calculations. It was originally planned to vertically sample the TCDD hot spots, but sample results were not available in time to accomplish this, and, therefore, some hot spots were missed in the vertical soil sampling. These hot spots could now be sampled vertically

for all three compounds, TCDD, 2,4-D, and 2,4,5-T. Only 15 plots were sampled for 2,4-D and 2,4,5-T, presenting a spacial distribution for these compounds inadequate for risk assessment. More plots could be sampled for these two compounds. One method that can be used to accomplish this is to revisit the 48 plots that were originally vertically sampled. These 48 plots could be sampled for all three chemicals of concern. This sample design would have two benefits: (1) better knowledge of the spacial distribution for 2,4-D and 2,4,5-T; and (2) knowledge of the fate of these chemicals over time.

Sediment - Positive sediment samples were found near the western shore, prior to construction of the seawall in that area. This area could be revisited to determine if the seawall is performing according to its intended function. More sediment samples are needed to better characterize the spacial pattern of contamination. A grid pattern similar to the soil sampling protocol would help to characterize the spacial contamination pattern. These samples should include areas close to the shoreline.

Water - No seawater sampling has been conducted off the former HO site. TCDD levels of 38 pg/l are toxic to fish. Toxic endpoints include severe adverse effects on survival, growth, and behavioral responses. With this potency, seawater sampling may be important. The groundwater under the former HO site has never been sampled and may be a vital link in any discovery of HO site-related fish contamination.

Biota - More sampling can to be performed at offshore sites adjacent to the HO site to determine if contaminated fish are in this area. No biological samples have been analyzed for 2,4-D or 2,4,5-T. It is not possible to assess the potential impact from fish ingestion for these two chemicals if this analysis is

not performed. Several adult fish species inhabiting the waters surrounding the Island are known to have large migratory movements. A study could be performed to ascertain if these migratory fish species are moving from the waters adjacent to the former HO site into fishing waters. Sampling and analysis of fishermen's catches can be easily used to determine if humans are consuming contaminated fish. This is the only study that would demonstrate if the fish being consumed are contaminated.

Ecological risk - Further field investigations may be needed to adequately characterize the ecological risks at JI. Any additional research should be coordinated with the work underway by Dr. John Labelle of the Woods Hole Oceanographic Institute in support of the JACADS monitoring program. Additional sampling programs could be designed so that statistical comparisons can be made between concentrations in the different areas. In such an investigation sediment sampling would be expanded to allow better characterization of the spatial pattern of contamination. Biota samples would be focussed on species whose behavior may lead to greater levels of contamination (e.g., bottom feeding resident species). Organisms that are important parts of marine food chains (e.g., small invertebrates such as marine worms) would be sampled. Based on the available data, the crown squirrelfish, yellowfin goatfish, snails, and crabs are good candidates for further sampling. Increased sampling of birds may be required to determine whether populations are at risk due to consumption of contaminated prey (e.g., fish and snails). Sampling could focus on one or two bird species that tend to be localized on the Island.

Although the contaminant studies should remain focussed on dioxin, it would be useful to examine several fish samples for 2,4-D. This compound has

been measured at levels as high as 281 ppm in soil samples on the Island. Although it is not bioaccumulated to the same extent as dioxin, measurable residues have been reported in fish from lakes treated with the compound and toxicity data are available.

Preliminary Public Health, Environmental Risk, and Data Requirements Assessment for the Herbicide Orange Storage Site at Johnston Island

1.0 Introduction

This report contains the results of a screening-level risk assessment conducted for the Air Force Occupational and Environmental Health Laboratory concerning the Herbicide Orange (HO) storage site at Johnston Island (JI). This risk assessment is part of the remedial investigation and feasibility study (RI/FS) process established by the U.S. EPA for characterizing the nature and extent of risks posed by hazardous waste sites and for developing and evaluating remedial options. This process is being conducted in the context of the U.S. Department of Defense (DoD) Installation Restoration Program (IRP). The following section provides a conceptual overview of the risk assessment for the HO storage site, site specific objectives of this investigation, a description of background information concerning the site, and defines the risk assessment's scope and study design.

1.1 Overview

During the Vietnam war, HO was widely used as a broad-scale defoliant. Large quantities of technical grade material were shipped to Vietnam. After the war, in April 1972, 1.37 million gallons of unused HO were transferred to JI from the stockpile in Vietnam for temporary storage. This was the result of the suspension of certain uses of 2,4,5-trichlorophenoxy acetic acid, a component of HO, by the Secretary of Health, Education and Welfare, and the Secretary of the Interior on April 15, 1970, following reports that HO may be teratogenic. The 24,910 fifty-five gallon drums of HO were stored on a 4-acre site at the northwest corner of JI (Figure 1.3). Further toxicity studies were conducted, and in September 1971 the Secretary of Defense directed the Joint Chiefs of Staff to dispose of all stocks of Herbicide Orange (HO). The HO stored on JI was successfully dedrummed and incinerated at sea in 1977. While stored on the Island, the sea air corroded some of the steel drums, resulting in HO leakage onto the ground and necessitating an active maintenance and redrumming operation at the storage site. Patrols of the storage area revealed approximately 20 to 70 leaking drums per week. It has been estimated that approximately 49,000 pounds of HO escaped into the environment annually during the period from 1972 to 1977 (Thomas et al., 1978). The HO stock was determined to contain two active ingredients (the n-butyl ester of 2,4-dichlorophenoxy acetic acid (2,4-D) and the n-butyl ester of 2,4,5-trichlorophenoxy acetic acid (2,4,5-T)), as well as 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) as a byproduct contaminant of 2,4,5-T (Holmes and Narver, 1989). Consequently, through leakage and spillage during maintenance, redrumming, dedrumming, and drum crushing operations, the site was contaminated over a period of six years with 2,4-D, 2,4,5-T, and TCDD. The site has remained essentially untouched since that time. Significant activities that

have occurred include a trial burn of contaminated soil (Helsel et al., 1987), construction of a seawall for those portions of the site adjacent to the ocean (as referenced in Channell and Stoddart, 1984), and extensive soil sampling in 1984.

There is some concern that contaminants at the site may be moving offsite into all environmental media: the adjacent air compartment, seawater, sea sediments, and groundwater aquifer that may underlie the site. It follows that if the contaminants are in any or all of these media, humans associated with them and biota contained in them may have a potential for exposure to HO site-derived contaminants and an attendant health risk. Therefore, the site-specific objectives of this investigation are to determine, based on available evidence:

- The potential contaminants at the site;
- The levels of contaminants at the site;
- The potential levels of the contaminants in each offsite environmental compartment;
- The potential levels of exposure to humans and wildlife, and to humans from biomagnification in the food chain; and finally
- The risk of health injury from potential multimedia exposure.

A companion objective is to determine, within the scope of existing environmental regulations, whether the quantified risks fall within acceptable risk limits. As such, this is *not* an Applicable or Relevant and Appropriate Requirement (ARAR) analysis, which is based on remediation alternatives, associated cleanup levels, and their compliance with relevant and applicable regulations. An ARARs analysis follows later in the RI/FS process.

1.2 Site Background

Johnston Atoll (JA) is a group of isolated coral islands located in the central Pacific Ocean lying approximately 717 nautical miles southwest of Honolulu Hawaii (Figure 1.1). Four small islands, Johnston Island, Sand Island, North (Akau), and East (Hikina) Island, comprise the egg-shaped atoll (Figure 1.2). JI the largest of the islands, 625 acres, has been enlarged over the years with dredged calcareous sand and coral rubble. The Island is approximately two miles long and one-half mile wide. JI is very flat with its highest elevation at seven feet. The Island has a 9000 foot runway down its middle. Details of the construction of JI can be found in Holmes and Narver (1989).

JI is an unincorporated territory of the United States. It was originally created as a bird refuge by Executive Order 4467 on June 29, 1926, and on July 25, 1940 was designated a National Wildlife Refuge. Historically, the Island has been under the control of various federal agencies. The Island is currently under the control of the Defense Nuclear Agency (DNA). A detailed outline of the agencies that have controlled the Atoll can be found in Table 1.1.

Figure 1.2 illustrates the location of JI to the other islands on the Atoll. Sand Island is the major brooding grounds for the birds. A detailed history and description of the atoll can be found in the following references: U.S. Air Force (1974), Thomas et al. (1978), Crockett et al. (1986), and Holmes and Narver (1989).

The Island is currently used for two major purposes. First, in the late 50's and early 60's it was used to launch missiles for atmospheric testing of nuclear

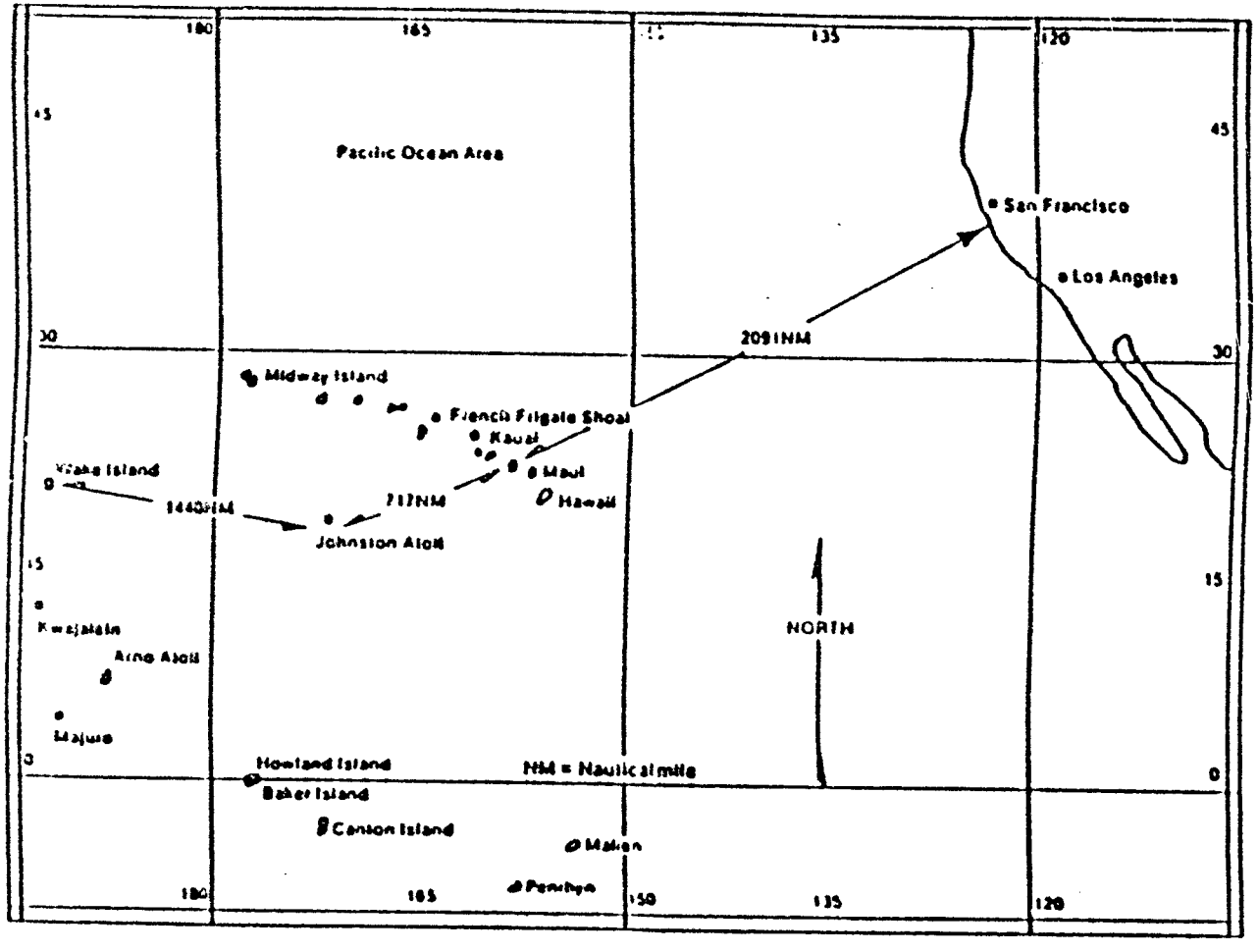
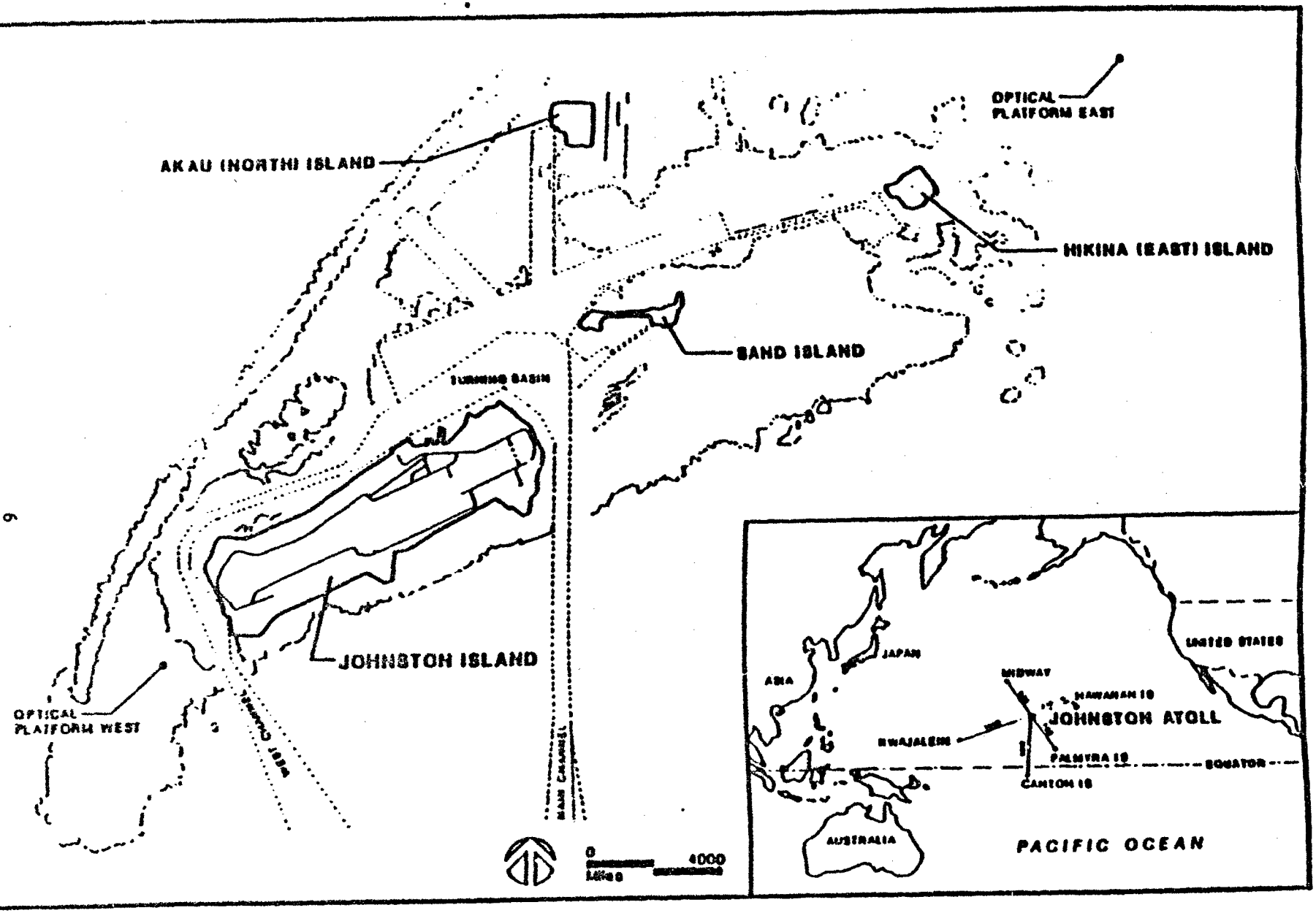


FIGURE 1.1 LOCATION OF JOHNSTON ISLAND
From Crockett AB 1986



JOHNSTON ATOLL LOCATION MAP
FIGURE 1.2
From Holmes & Marver 1989

TABLE 1.1 Ownership and Control of Johnston Atoll

Period	Jurisdiction	Operational Control	Purpose of Document	Authority	Event/Use
1926	Dept. of Agriculture	Agriculture	Plant and sea life surveys	*Executive Order 4467	Bird refuge (Executive Order June 29, 1926)
1934	Dept. of the Navy (USN)	USN	Pacific Defense	*Executive Order 6935	
1940	Dept. of the Navy (USN)	USN	Redesignation	Presidential Proclamation No. 2416	Johnston Island National Wildlife Refuge (July 25, 1940)
1941	Dept. of the Navy (USN)	USN	Established Naval Defense Sea Area for military sea and air operations	*Executive Order 8682	Became Johnston Island Naval Air Station
1947	Dept. of the Navy (USN)	USN	---	Secretary of the Navy	Became a Naval Air Facility
1948 July 1	Dept. of the Navy (USN)	USAF	Transfer of operational control to USAF	Agreement	Pacific Air Command (MATS, ARS, AACS, AWS Dots) (SecNAV ordered transfer to USAF)
1949 June 1	Dept. of the Navy (USN)	USAF	---	---	Pacific Air Command inactivated; Pacific Division MATS took over
1951-52	Dept. of the Navy (USN)	USAF	---	---	Korean airlift support
1957 Jan. 25	Dept. of the Navy (USN)	USAF	---	---	USAF granted Treasury Department five-year use for USCG LORAN
1957 Sept. 13	Dept. of the Navy (USN)	USAF	---	---	USAF granted Department of Commerce five-year use for U.S. Weather Bureau
1958 April 22	Dept. of the Navy (USN)	CJTF-7	---	Agreement	Atomic tests in Pacific area under Commander Joint Task Force-7 (CJTF-7) until August 19, 1958; then roll-up

TABLE 1.1 Ownership and Control of Johnston Atoll (continued)

Period	Jurisdiction	Operational Control	Purpose of Document	Authority	Event/Use
1959 June 30	Dept. of the Navy (USN)	USAF	—	—	Secretary of the Treasury asked Secretary of Defense for Sand Island as LORAN Station, to be under operational control of Commander-in-Chief, Pacific
1962 Jan. 17	Dept. of the Navy (USN)	CTJF-8/AEC	—	Agreement	USAF signed Operations Agreement for 1962 nuclear tests
1962 Jan. 18	Dept. of the Navy (USN)	CTJF-8/AEC	—	Agreement	Commander-in-Chief, Pacific, signed agreement with Commander, Joint Task Force Eight (CJTF-8)
1963 June 11	Dept. of the Navy (USN)	CTJF-8/AEC	—	—	Joint Chiefs of Staff reaffirmed operational control of Joint Task Force Eight
1970 July 1	Dept. of the Navy (USN)	USAF	Transfer of operational control to USAF	—	JTF-8 inactivated. Deputy Secretary of Defense Memorandum to Secretary of Air Force
1973 July 1	Dept. of the Navy (USN)	DNA (FCDNA)	Transfer of operational control to DNA	Agreement	Department of the Air Force signed agreement with DNA
1976	Dept. of the Navy (USN)	DNA	—	Agreement	Safeguard "C" revised, JA placed in caretaker status
1976	Dept. of the Navy (USN)	DNA	Responsibilities and jurisdiction guideline	Memorandum of Understanding DOD/DOI	Allow the Departments to perform their functions in a manner that is mutually compatible and agreeable

*Executive Orders are still in effect and have not been amended or rescinded as to affect "ownership."

Source: Johnston Atoll Chemical Agent Disposal System (JACADS) Final Environmental Impact Statement, November, 1983.

From: Holmes and Narver, 1989.

weapons. In 1963 the Limited Test Ban Treaty banned atmospheric nuclear testing. The facilities at JI are still maintained for this purpose in case this type of testing is deemed necessary for national defense. These facilities are currently held in a caretaker status. During 1962, three missile aborts caused transuranic contamination on parts of the Island, the section labelled LE-1 on Figure 1.3. The second purpose of operations at the Island has been to destroy chemical weapons at the Johnston Atoll Chemical Agent Disposal System (JACADS) facilities, which is a state-of-the-art incineration operation. The JACADS facilities are located in the "Red Hat" area of the Island.

Figure 1.3 illustrates the location of the HO site relative to the other facilities on the Island. A detailed map of the HO site is provided in Figure 1.4. The dedrumming area was used to redrum HO that was leaking from the corroded drums during their storage, and later during the HO removal process to transfer the HO from the drums to the trucks for transport to the wharf area and loading onto the incineration ship. A drum crusher was used in 1977 during the removal operation. The dedrumming and drum crushing areas are of particular interest in this investigation because they are potential sources of contamination. The purpose of a concrete pad in the northwest corner of the HO site has not been determined. A transformer, Hi-Vol air sampling station, beacon building, and a berm are adjacent to the site immediately downwind. The Hi-Vol sampler is associated with the JACADS operation. A fire training area and burn pit are located further downwind.

Thirteen separate media sampling and analysis studies have been conducted on JI. These are summarized in Table 1.2. The first study was conducted during the disposal of HO in 1977. The sites of sampling in various environmental media are presented in Figures 1.5 through 1.9. This study was

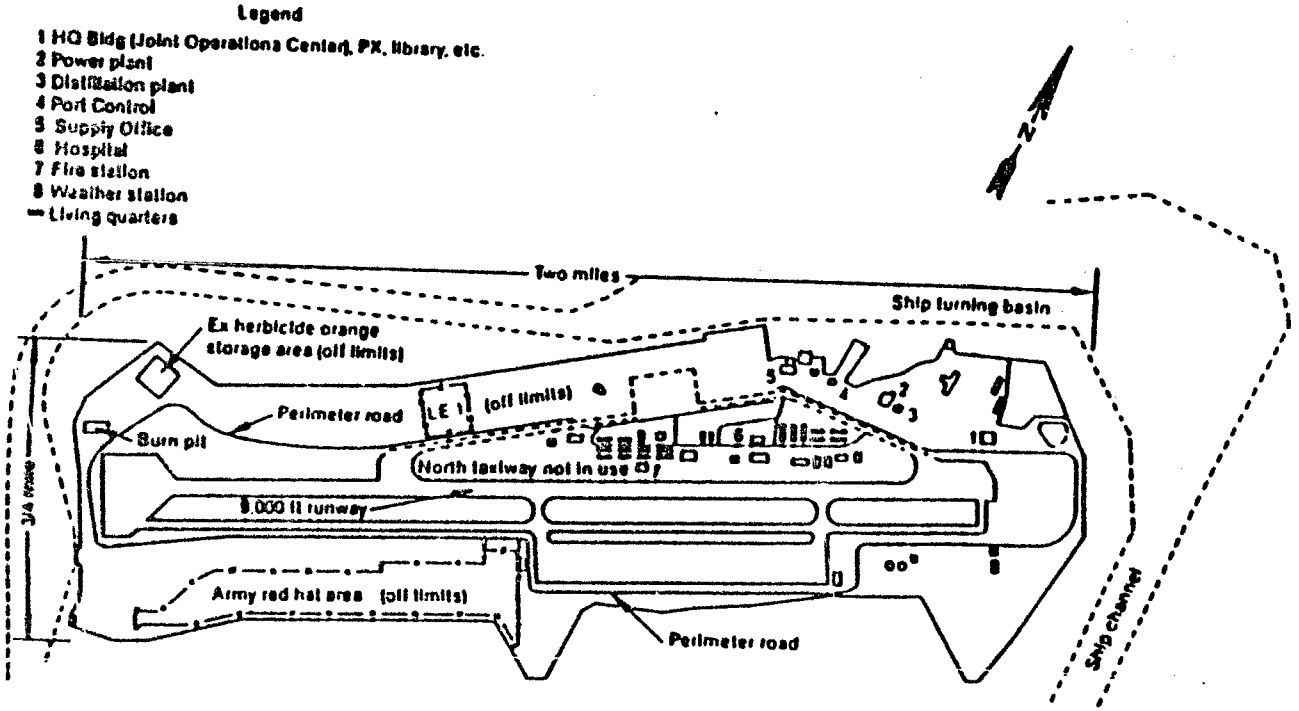


FIGURE 1.3 MAP OF JOHNSTON ISLAND
From Crockett AB 1986

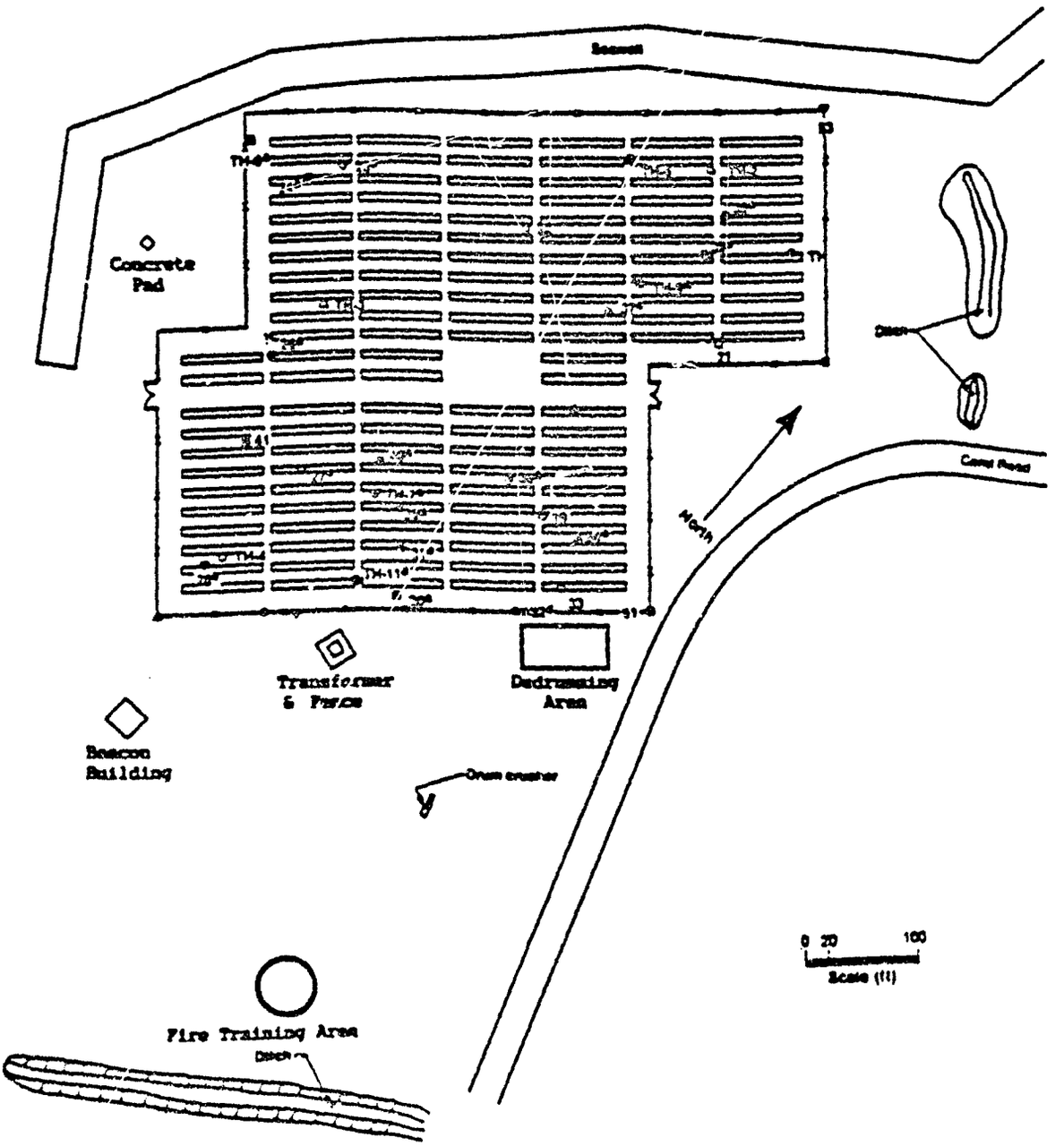


FIGURE 1.4 HERBICIDE ORANGE STORAGE AREA
From Crockett AB 1986

TABLE 1.2 Sampling Studies of Johnston Island

Study	Period of Performance	Soils	Ocean Sediments	Water	Air	Biota	Reference Document(s)
Associated with the Dedrumming Operation							
1. HO Monitoring during disposal of HO by OEHL	May 1977 - September 1978		X	X	X	X	Thomas, T.J., D.P. Brown, J. Harrington, T. Stanford, L. Taft, B.W. Vigon, September 1978, *Land-Based Environmental Monitoring at Johnston Island: Disposal of Herbicide Orange, May 1977 - September 1978*, OEHL TR-78-87, OEHL, AFOEHL, Brooks Air Force Base (AFB), Texas.
Associated with the Period Subsequent to the Disposal Operation							
2. Initial HO Monitoring Program by OEHL and ESL	August 1977 - September 1984	X	X				Channell, R.E., and T.L. Stoddart, April 1984, *Herbicide Orange Monitoring Program: Interim Report, January 1980-December 1982*, ESL-TR-83-56, ESL, AFESC, Tyndall AFB, Florida.
3. Supplementary Dioxin Biomonitoring Program	1984					X	Rhodes, 2 Lt., Albert N., January 2, 1985, *Johnston Island Fish Samples*, Letter to USAF OEHL/EC.
4. Supplementary Dioxin Biomonitoring Program	1985		X			X	Markland, Col. Darryl T., January 3, 1986, *Dioxin Monitoring at Johnston Island*, Consultative Letter, 85-192 EQ 805 MBC, to HQ USAF/SGES (Lt.Col. Capell).

TABLE 1.2 Sampling Studies of Johnston Island (continued)

Study	Period of Performance	Soils	Ocean Sediments	Water	Air	Biota	Reference Document(s)
5. Comprehensive Soil Characterization Study	April 1984-April 1986	X					Crockett, A.B., A. Propp, and T. Kimes, EG&G/Idaho, Inc., Idaho Falls, Idaho, October 1986, *Soil Characterization Study of Former Herbicide Storage Site at Johnston Island: April 1984-April 1986*, Final Report, ESL-TR-86-18, ESL, AFESC, Tyndall AFB, Florida.
6. JI Survey Sampling and Analysis Project in Support of the Johnston Atoll Chemical Agents Disposal System (JACADS)	September 1985	X			X[1]		Casanova, J.N., January 1986, *JI Survey Sampling and Analysis Project*, EG&G/Idaho, Inc., Idaho Falls, Idaho.
7. Supplementary Dioxin Biomonitoring Program	1986		X			X	Markland, Col. Darryl T., March 18, 1987, *Dioxin Monitoring Analytical Results, Johnston Island*, Consultative Letter, 87-031-EQ-805-CEF, to HQ USAF/SGPA.
8. Supplementary Dioxin Biomonitoring Program	May 1987		X			X	Forsell, Doug, May 11, 1987, *Second Quarter Samples Collected from Johnston Island for Dioxin Testing*, Letter to Chief Ecology Functions (Maj. Thomas Doane).

[1] Dust and sweepings sampling.

TABLE 1.2 Sampling Studies of Johnston Island (continued)

Study	Period of Performance	Soils	Ocean Sediments	Water	Air	Biota	Reference Document(s)
9. Supplementary Dioxin Biomonitoring Program	October 1987		X			X	Forsell, Doug, October 4, 1987, *October Samples Collected from Johnston Island for Dioxin Testing*, Letter to Ecology Function (Maj. Elliott Ng), USAF OEHL.
10. Supplementary Dioxin Biomonitoring Program	January 1988		X			X	Forsell, Doug, January 16, 1988, *January Samples Collected from Johnston Island for Dioxin Testing*, Letter to Chief Hazardous Waste Function (Maj. Elliott Ng), USAF OEHL.
11. Supplementary Dioxin Biomonitoring Program	August 1988		X				Forsell, Doug, August 26, 198, *August samples from Johnston Island for Dioxin Testing*, Letter to Chief Hazardous Waste Function (Maj. Elliott Ng), USAF OEHL.
12. Supplementary Dioxin Biomonitoring Program	December 1988		X			X	Forsell, Doug, December 17, 1988, *December Samples Collected from Johnston Island for Dioxin Testing*, Letter to Chief Hazardous Waste Function (Maj. Elliott Ng), USAF OEHL.
13. Supplementary Dioxin Biomonitoring Program	December 1989					X	Mertens, Sharon K., December 7, 1989, *Analytical Results for December 1989 Regarding Contract F 33615-84-D-4402/0012/Analytical Work*, Letter to HSD/YAQI (Mr. Rodriquez).

Adapted from Holmes & Narver, 1989.

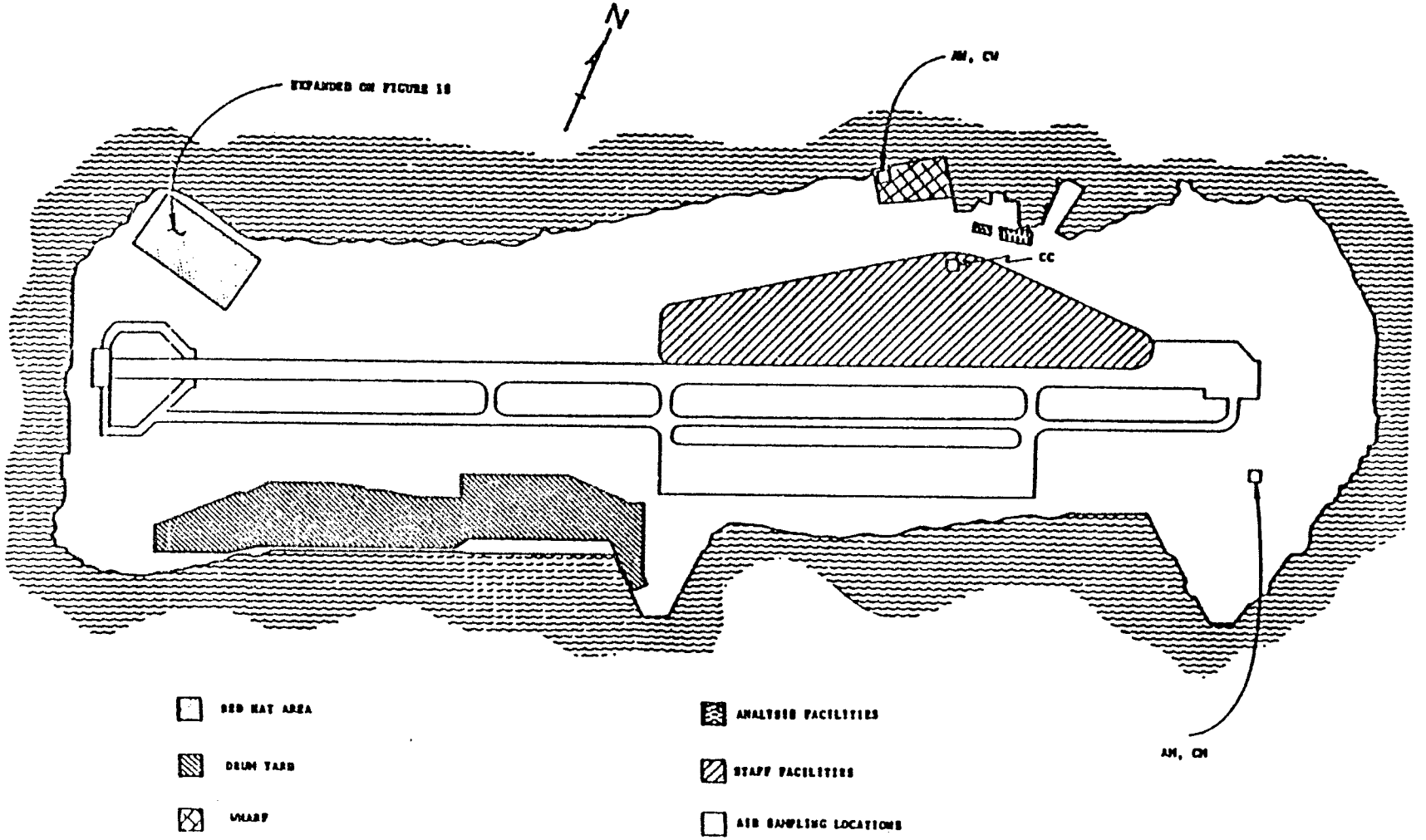


FIGURE 1.5 AIR SAMPLING SITES
From Thomas 1978

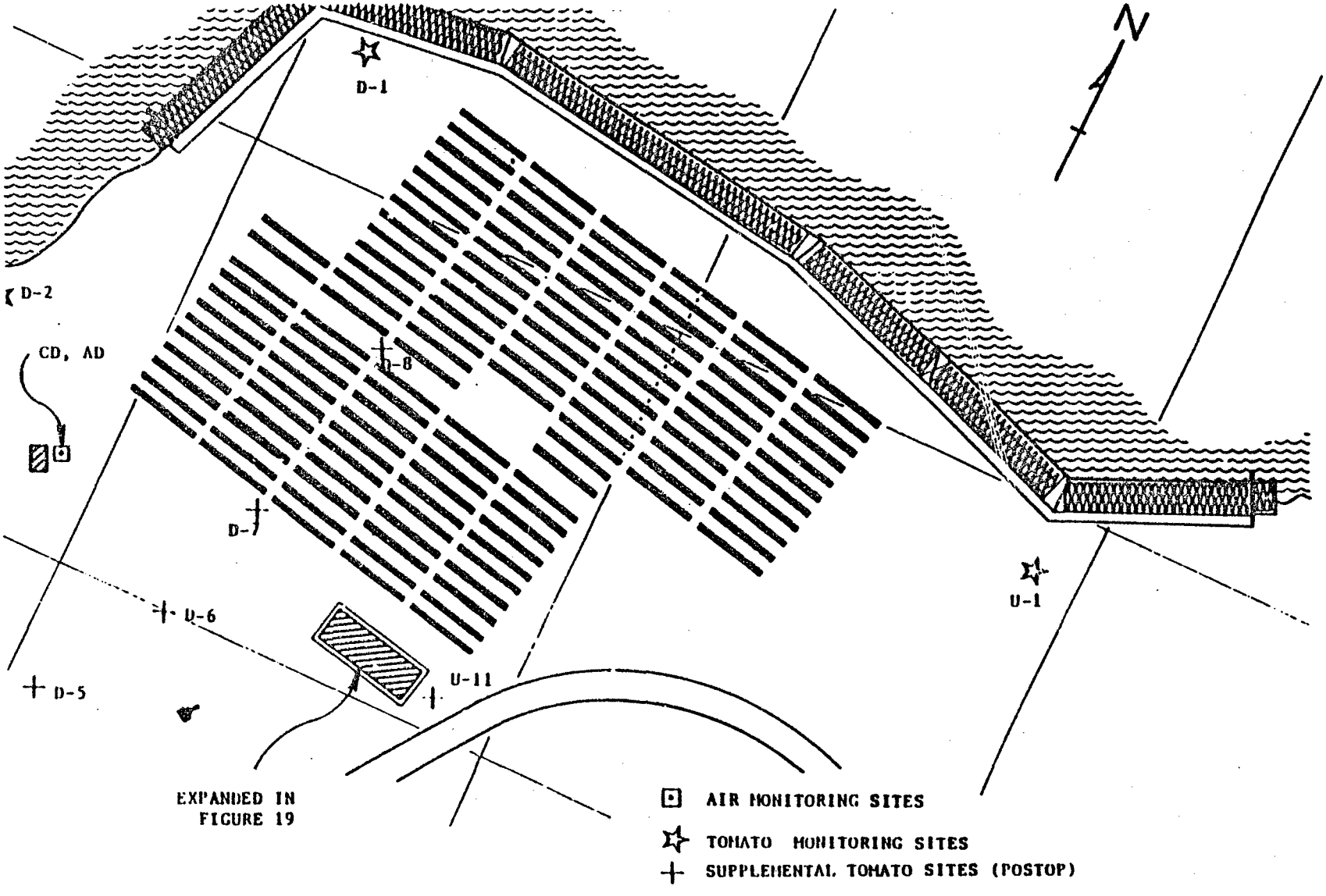


FIGURE 1.6 AIR MONITORING SITES, DRUMYARD
From Thomas 1978

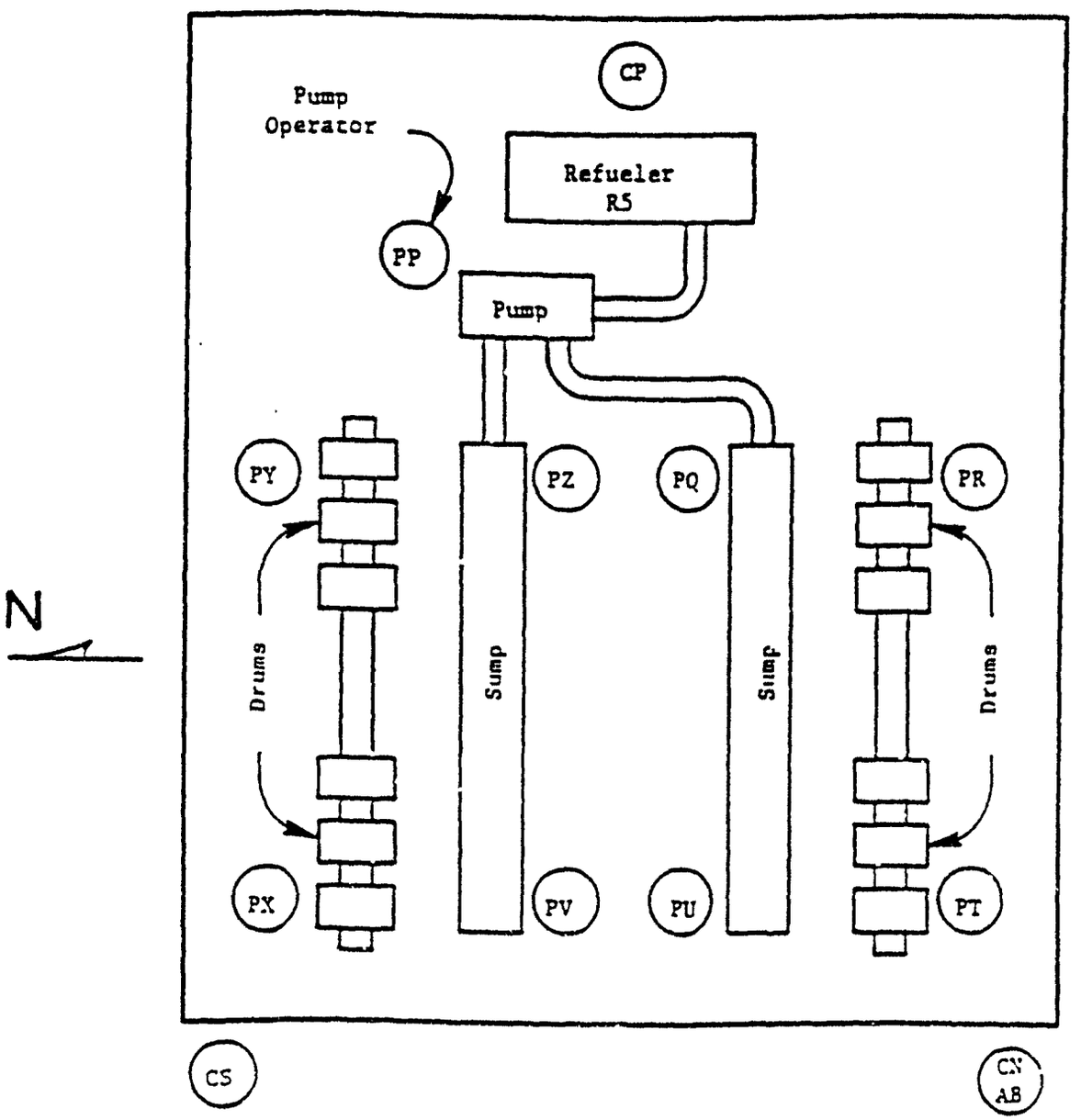


FIGURE 1.7 SAMPLING SITES AT DEDEAM FACILITY
From Thomas 1978

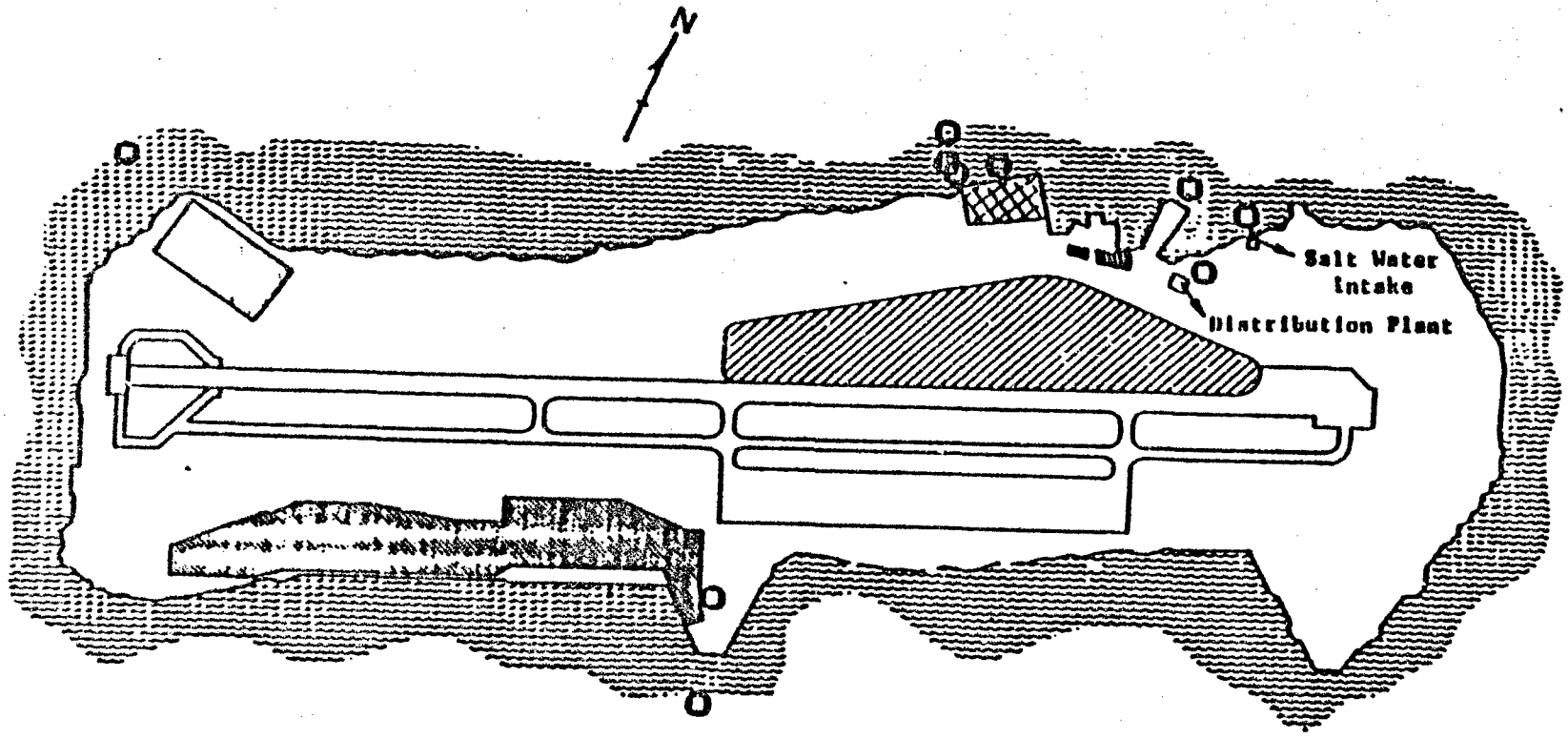


FIGURE 1.8 WATER AND SEDIMENT SITES
From Thomas 1978

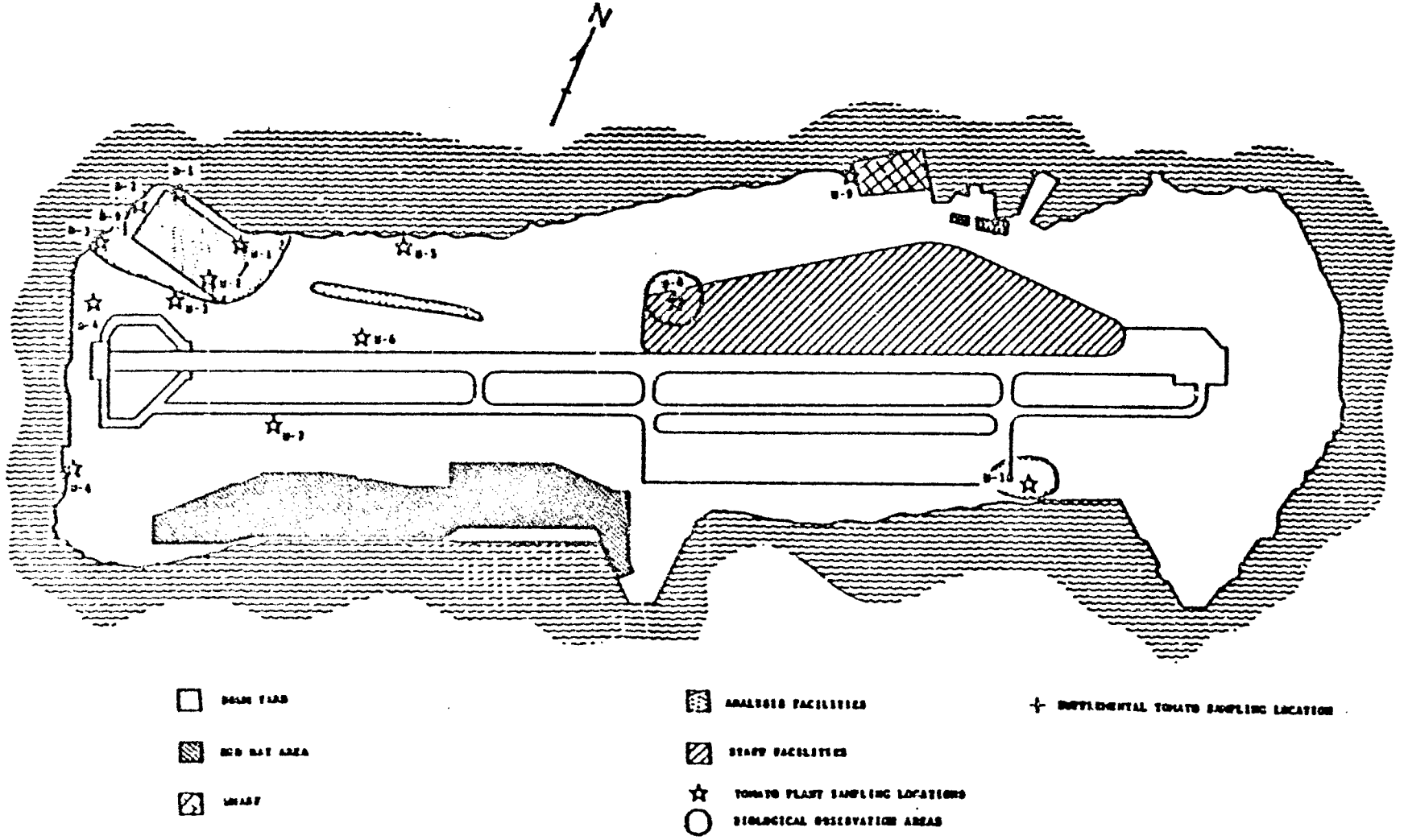


FIGURE 1.9 BIOTA MONITORING SITES
From Thomas 1978

used to assess the possible environmental impacts resulting from the disposal of HO. The ground water under the HO site has never been analyzed for HO or dioxin. The second through thirteenth studies continued to measure the impacts to the environment from the HO storage site after disposal was completed. Studies 3, 4, and 7 through 13 are part of a continuing effort to monitor biological effects from the former HO storage site. These studies include invertebrates, fish, and sediments around the former HO site and the west wharf, where sport fishing is conducted by Island inhabitants. The fifth study was conducted to obtain a comprehensive soil profile of the former HO storage site and the immediate surrounding area. The sixth study was initiated in support of the JACADS operation. It included TCDD soil measurements.

1.3 Scope of the Risk Assessment

This analysis follows the conventional structure of a risk assessment as laid out in documents of the EPA (1988c, 1989c). Its basic features include a health hazard assessment, exposure assessment, dose-response determination, and a risk characterization. The results of the risk characterization are then used to determine if existing concentrations on the site present a level of risk to human health and the environment that is acceptable or unacceptable and, if deemed to be unacceptable, the degree to which remediation is necessary to lower risks to an acceptable level.

This is a multimedia assessment that includes air, soil, water, and the food chain. The HO site has some unique features that make some of the multimedia components of the risk assessment straightforward and others complex. Among the straightforward components, the meteorological features of the Island and the surrounding area are the strongest, being well

characterized, predictable, and relatively nonvariable. There is a finite human population that has a potential for exposure from all media and whose exposure is controllable should it be necessary. Access to the site can be limited or expanded to any degree desired, and there are a limited number of optional future uses for the site which limit the need for more elaborate analyses. On the complex side, possible offsite contamination means that the HO site is uncontained and extended into the surrounding environment. The site may be contiguous with the sea and marine environment via ground water and provides some element of runoff into the open water. The dynamics of the ocean as an environmental compartment are too difficult to characterize for predicting potential zones of contamination; nevertheless dynamic transfer from one environmental compartment to another (e.g., emission factors from soil into air, partitioning of TCDD into sediments and seawater) must be quantified. The soil composition (variable coral) is unusual and its characteristics poorly defined. Fate and transport phenomena must be accounted for to predict contaminant form and concentration in secondary media. As a mixture, chemical-chemical interactions, particularly associated with possible additive, potentiative, or synergistic effects of the mixture's toxicity must be considered. TCDD is a potent carcinogen and even though there is considerable evidence of carcinogenic and noncarcinogenic toxicity on 2,4-D and 2,4,5-T, there are no published benchmark toxicity values (UCR, RfD) that quantitatively represent their dose-response characteristics. There is a potential confounding effect posed by other sources and their contaminants on the Island (i.e., JACADS and the launch area). Lastly, as will be described in detail later, data on the site and surrounding area are quite limited.

This analysis should be considered as a *preliminary* baseline risk assessment. In a *full* baseline risk assessment that forms an integral part of

the RI/FS process, prescribed procedures are followed as specified in key documents of the EPA, such as the *Human Health Evaluation Manual* (EPA, 1989c) and the *Superfund Exposure Assessment Manual* (EPA, 1988c). To the extent possible, these prescribed procedures were utilized. However, the HO site on JI is a unique environment with exceptionally uneven scientific data (particularly on the monitoring of environmental media) because data collection practices, in accordance with the needs prescribed for a baseline risk assessment, have not been orderly and systematic over the years since HO was stored there and contamination began. As a result, the risk assessment contained in this document includes reasonable conservative assumptions to bridge information gaps where such information is usually present to support the baseline assessment. Accordingly, *this risk assessment should be viewed only as a screening-level evaluation*, to:

- Provide a plausible preliminary estimate of risk;
- Identify the areas where information is needed to provide more quantitative estimates of risk with less associated uncertainty for decision-making by risk managers; and
- Provide a basis for determining what future data development ought to be undertaken to:
    - Decide if remediation is necessary and, if so, to what level of cleanup;
    - Enable adequate analyses of remedial options (including an assessment of residual risk associated with implementation of each viable remedial option and future use scenario); and
    - Aide in the sensible selection of the most appropriate option.

A more complete baseline risk assessment, suitable for responsible decision-making on remedial alternatives and closure, can be constructed only after additional field data at the HO site are collected. The default assumptions used in this screening-level risk assessment and the data needed to develop a more definitive risk assessment for the site are clearly laid out in discrete sections of this report.

1.4 Organization of the Report

This report generally follows the organizational structure recommended by the EPA (1989c) and is progressive in laying out the sequential components along the path to determination of human health risk. The site features relevant to this analysis, scope, and rationale are presented in Section 1.0. Data collection and evaluation practices, and identification of chemicals of concern are addressed in Section 2.0. A complete exposure assessment, including pathway analysis and exposure quantification for different scenarios is presented in Section 3.0. A toxicity assessment is presented in Section 4.0. Characterization of risks for current and future land-use conditions are presented in Section 5.0. An ecological assessment is presented in Section 6.0. Data needs for the various preceding components of the analysis are presented in Section 7.0. A summary of the report is presented in Section 8.0.

2.0 Identification of Chemicals of Potential Concern

Identification of chemicals of potential concern is based on consideration of the types of chemicals known or expected to be present at the site, the toxicity and physicochemical properties of these chemicals, and potential human exposure pathways. Evaluation of the potential human exposure pathways which are relevant to a given site includes consideration of the types of environmental media of concern, geographical/physical areas of concern, potential routes of contaminant transport through the environment (e.g., inter-media transfer, food chain), and the human populations present and their activity patterns. This section provides information regarding site-specific data collection and evaluation considerations and identifies chemicals of concern based on human exposure pathways of potential relevance to the HO storage site.

2.1 Site-Specific Data Collection

Monitoring data that have been collected since 1977 are presented in Table 1.2. Study number 1 was conducted during ocean incineration of HO. Study number 2 was the first investigation conducted after the disposal operation. Data from Study

numbers 3 through 13 (except number 6) were utilized for this risk assessment because they comprise the most recent data available. The water samples taken in Study number 1 were from drinking water supplies on the east side of JI. These samples showed no detectable levels of TCDD. No water samples have been taken since that study. Particulates and vapor phase organics were not sampled. Air sampling for Study number 6 was taken for two criteria pollutants: SOx and NOx. For this risk assessment, limited data are available for residues in soil, fish, birds, and sediment.

Crockett et al. (1986) performed an extensive soil study of the HO site from 1984 to 1986. Approximately 900 soil samples were analyzed for TCDD, 2,4-D, and 2,4,5-T. The sample grid (Figure 2.1) contained 445 plots, each 400 ft^2. Each plot was sampled five times to produce one composite sample for analysis. Replicate samples were taken from 18 plots. Vertical chemical profiles were taken for TCDD to a depth of 1 ft in 33 plots, and for TCDD, 2,4-D, and 2,4,5-T to a depth of 5.5 ft in 15 plots. For 1-foot profiles, samples were taken at depths of 0, 0.1, 0.4, and 0.8 ft. for 5.5-ft profiles, samples were taken at depths of 0, 0.1, 0.4, 0.8, 2.0, 3.0, 4.0, and 5.0 ft.

Surface samples for 2,4-D and 2,4,5-T were taken in 15 vertical sampling plots. The authors originally intended to perform vertical sampling in the plots where high levels of TCDD were detected. However, sample processing time was insufficient to permit this. The vertical sampling plots were chosen by three criteria: brown staining of the soil surface, random selection, and results from previous soil studies. Some of the plots with the highest TCDD surface concentrations were not identified before completion of vertical sampling; therefore vertical sampling of these plots were not performed. Greater detail of the sampling protocol can be found in Crockett et al. (1986).

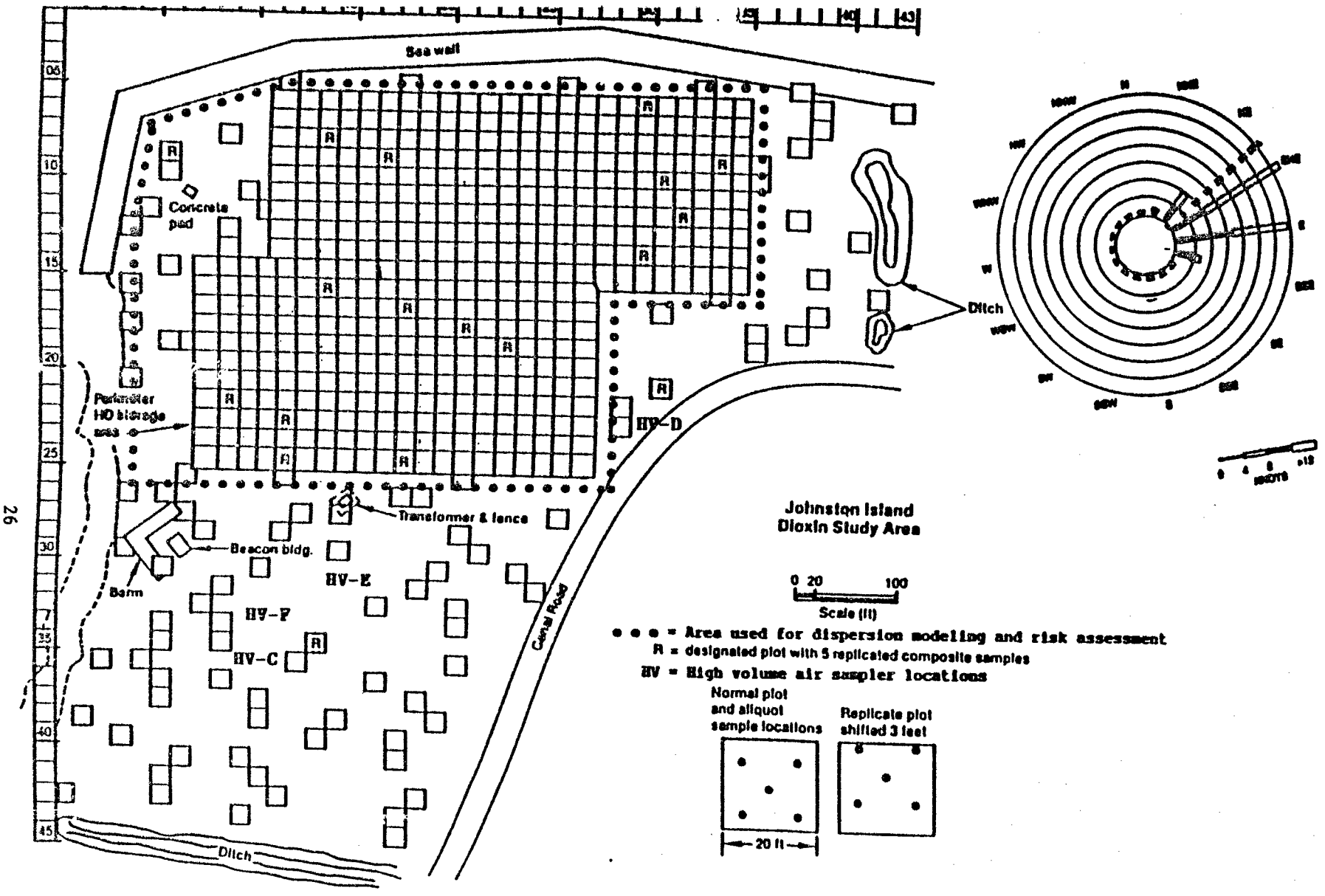


FIGURE 2.1 STUDY AREA GRID WITH REPLICATE SHIFT PATTERN

Results of the *surface* soil analysis are presented in Figures 2.2 to 2.4. The X,Y coordinates in all figures correspond the to X,Y coordinates in Figure 2.1. The 2,4-D and 2,4,5-T values were taken from the 0-3 inch vertical depth sample.

Results of the *subsurface* soil analysis are presented in Figures 2.5 to 2.7. The value for each plot is the median concentration from all vertical samples taken within that plot. Results reported to be invalid by the authors of the study were not considered in the calculation of the median value. The highest concentration of all three chemicals analyzed were found in the 3 to 7 inch layer of soil: 510 ppb for TCDD, 365,202 ppb for 2,4-D, and 682,247 ppb for 2,4,5-T. The authors suggested that remediation to a vertical depth of 30 inches would result in TCDD levels below 1 ppb in all plots but one (at 1.3 ppb). The highest concentration of 2,4-D below 30 inches was 140 ppb and of 2,4,5-T was 450 ppb. The plots south and east of the fenceline were considered to be outside the HO site for purposes of this risk assessment. This is because the plots are small and isolated, there are no data available on concentrations for adjacent areas, and the concentrations are relatively low and therefore not expected to contribute significantly to offsite risk were access to them limited. In a few of these isolated plots, the concentrations are likely to be representative of what is expected to have been leaky drums on similar plots of the HO site.

In this risk assessment, marine biota, sediment, and avian samples were used from data that have been collected since 1984. These samples were analyzed only for TCDD. Samples of marine biota were obtained from six sites (Figure 2.8), according to the protocol described in Forsell (1987). Sites 1 through 3 are located in the water adjacent to the former HO site. Site 4 is located on the east side of JI and serves as a control. Site 5 is located at the west wharf, and Site 6 is located at the coral reef off the northwest corner of JI. Site seven is located on the former HO area. Some of the samples were not identified by site number. The marine biota samples were collected as grab samples by divers using a spear. Prior to September 1987,

Figure 2.2 2,3,7,8-Tetrachlorodibenzo-p-dioxin Surface Soil Concentration (ppb)

	04	05	06	07	08	09	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26	27	28	29	30	31	32	33	34	35
06									ND	8.7	ND	ND	0.1	0.1	0.1	0.1	31	ND	ND	0.1	1.3	ND	7.8	ND	ND	1	0.6	ND	ND	ND	ND	ND
07									0.3	ND	1.5	2.0	1.6	17	13	24	27	5.0	6.8	0.1	2.5	45	0.8	20	19	18	1.6	13	6.6	57	0.8	ND
08						0.6			136	163	91	20	6.7	0.8	1.1	0.3	0.1	9.5	ND	0.1	ND	0.2	3.1	0.2	ND	0.2	1.9	41	0.2	0.2	32	39
09			9.6						26	50	46	21	7.7	0.5	9.6	ND	0.2	ND	ND	ND	0.1	19	0.5	ND	ND	9.8	0.5	0.8	2.6	25	1.7	0.1
10			77						0.9	26	59	25	7.2	1	1	0.3	0.1	0.6	1.3	0.2	0.3	ND	1.6	ND	ND	18	1.6	1.1	20	8.5	0.6	0.2
11							0.2		0.2	5.0	18	1.8	ND	0.1	12	41	0.5	12	M	M	1.9	1.2	31	0.6	34	0.3	5.2	1.7	0.9	18	6.6	0.3
12		36						0.2	0.7	0.3	2.2	ND	ND	ND	ND	ND	ND	5.6	2.7	1.3	0.6	18	53	0.6	7.1	29	1.5	0.1	6.2	22	63	ND
13	23					0.6			0.1	ND	ND	ND	0.5	ND	0.1	ND	0.1	29	ND	0.7	1.0	ND	ND	0.2	63	3.9	32	ND	0.1	0.3	0.1	0.3
14						1.0			0.2	0.8	0.6	1.6	21	1.8	0.2	ND	ND	ND	0.2	2.2	0.6	18	0.2	ND	0.5	1.7	1.0	3.1	5.1	1.6	0.2	1.8
15			2.8		1.1	12	13	59	0.5	0.6	6.0	15	27	ND	0.6	15	10	0.2	0.3	63	9.7	24	ND	1.6	55	27	3.5	3.1	24	9.0	0.2	ND
16	1.1				1.1	1.2	0.5	0.3	35	19	6.7	0.3	0.1	ND	0.6	0.1	0.9	ND	2.7	0.9	1.1	ND	0.1	0.6								
17					27	26	10	9.7	0.1	0.3	0.6	0.5	11	14	16	0.2	6.9	0.6	17	0.8	2.9	1.7	0.9	ND								
18	9.0				0.5	21	ND	0.5	0.3	0.6	ND	0.6	M	0.2	61	ND	ND	17	1.8	18	ND	17	28	ND								
19			0.7	0.2	0.9	148	6.0	ND	59	2.8	1.2	0.8	0.7	ND	3.5	5.2	0.3	0.1	2.1	0.1	0.1	0.1	ND	ND								
20					0.2	1.5	13	0.6	0.1	0.2	0.6	0.6	0.9	0.1	7.1	0.6	ND	ND	ND	0.1	0.1	1.5	0.2	ND								
21	1.9				0.6	6.7	46	53	ND	41	23	27	ND	ND	1.3	22	0.6	61	0.6	0.2	0.6	ND	ND	ND								
22					ND	0.2	0.5	2.7	0.9	21	1.6	12	ND	0.1	16	0.6	ND	22	24	62	11	132	11	0.9								
23					0.1	0.1	0.3	0.1	5.5	3.7	0.6	0.7	0.1	0.1	0.2	ND	ND	1.3	1.0	0.7	0.1	0.3	ND	ND								
24					2.6	1	9.0	10	0.3	0.1	0.1	5.6	0.2	0.1	25	10	51	1.5	1.8	0.1	0.1	0.1	0.1	1								
25					37	ND	ND	ND	0.1	2.3	38	13	63	18	20	0.3	6.8	9.0	2.1	0.8	62	62	7.2	0.5								

I = Invalid Result
M = Missing Sample
ND = Not Detected

Figure 2.3 2,4-Dichlorophenoxy Acetic Acid Surface Soil Concentration (ppb)

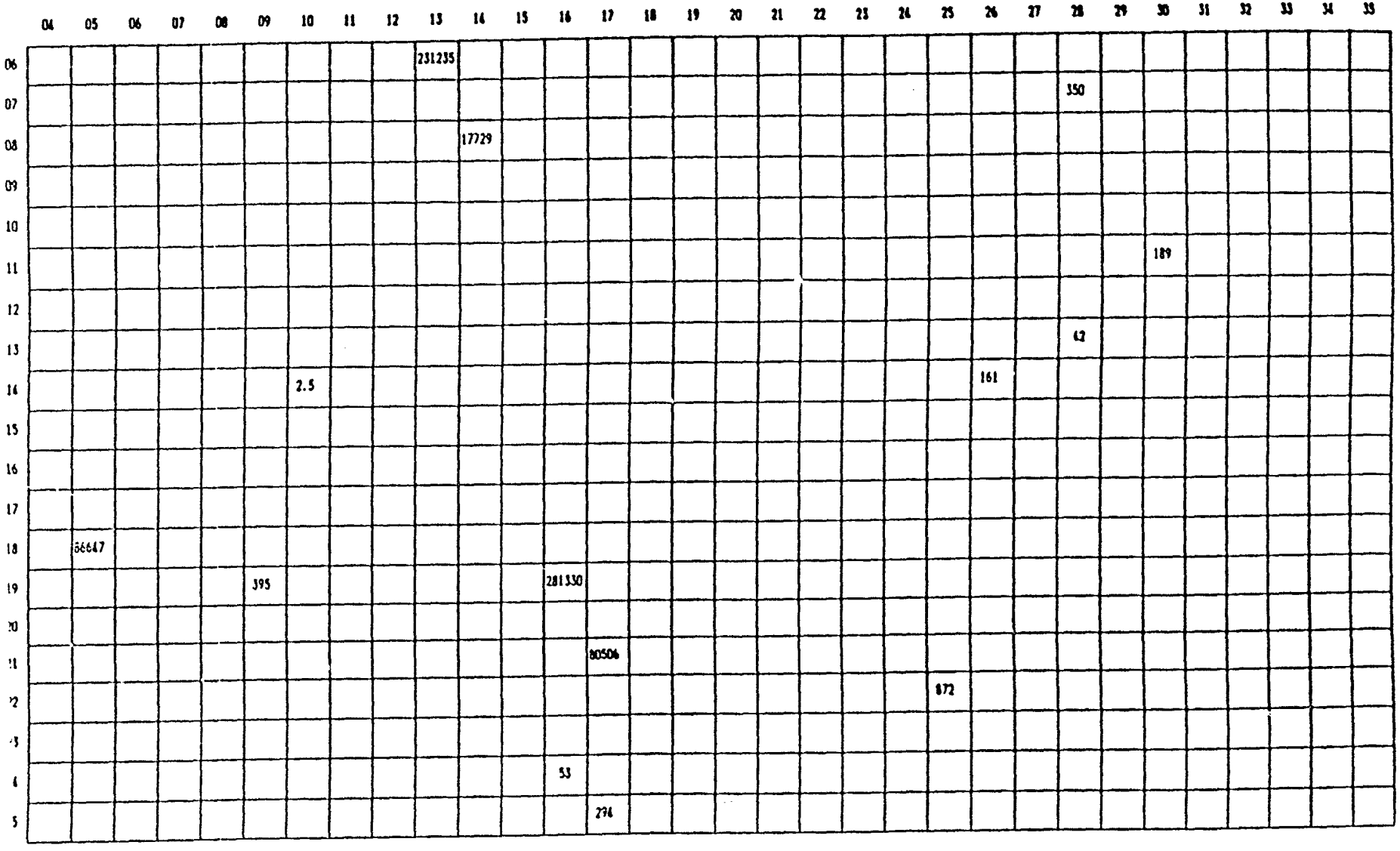


Figure 2.4 2,4,5-Trichlorophenoxy Acetic Acid Concentration (ppb)

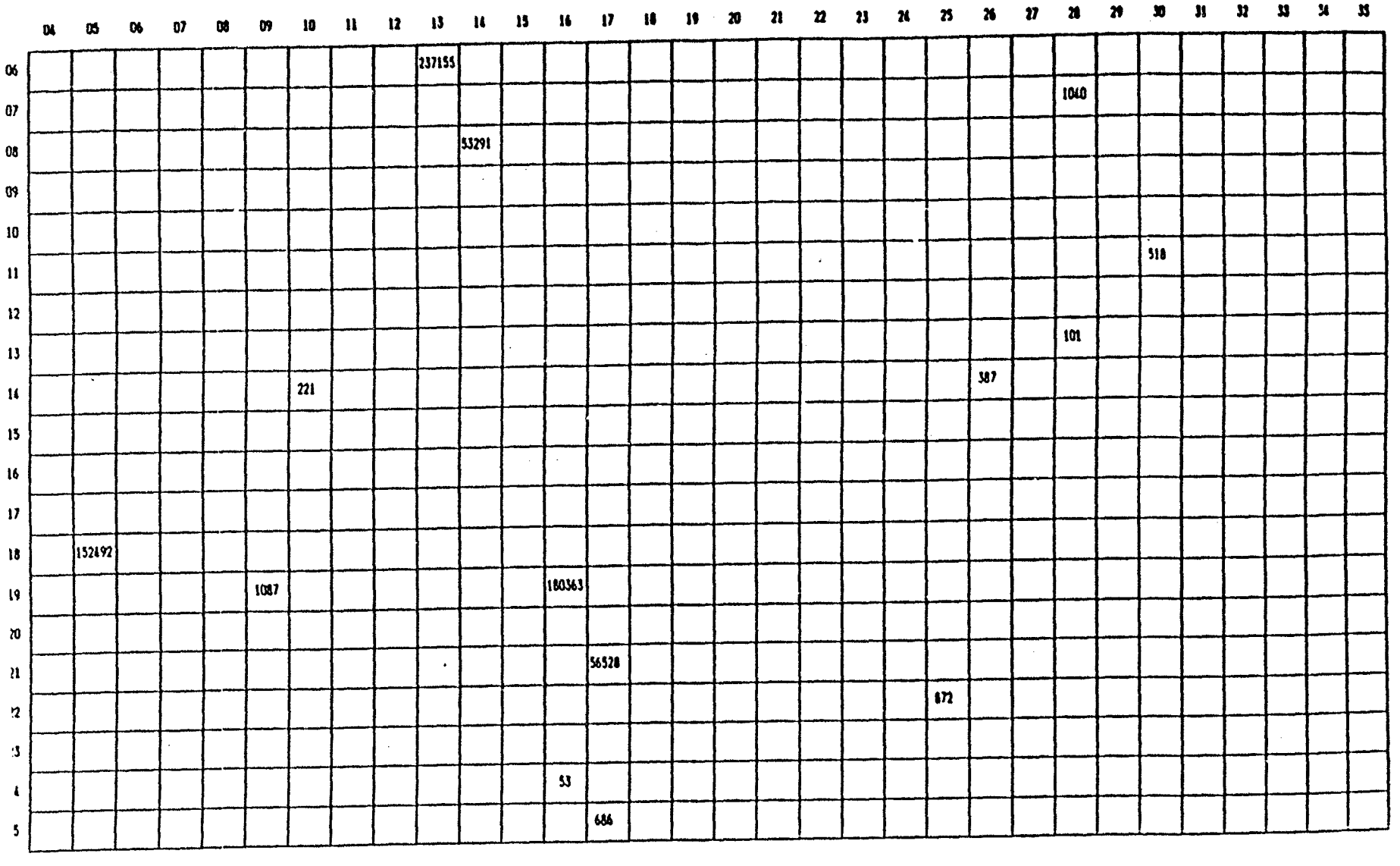


Figure 2.5 2,3,7,8-Tetrachlorodibenzo-p-dioxin Subsurface Soil Concentration (ppb)

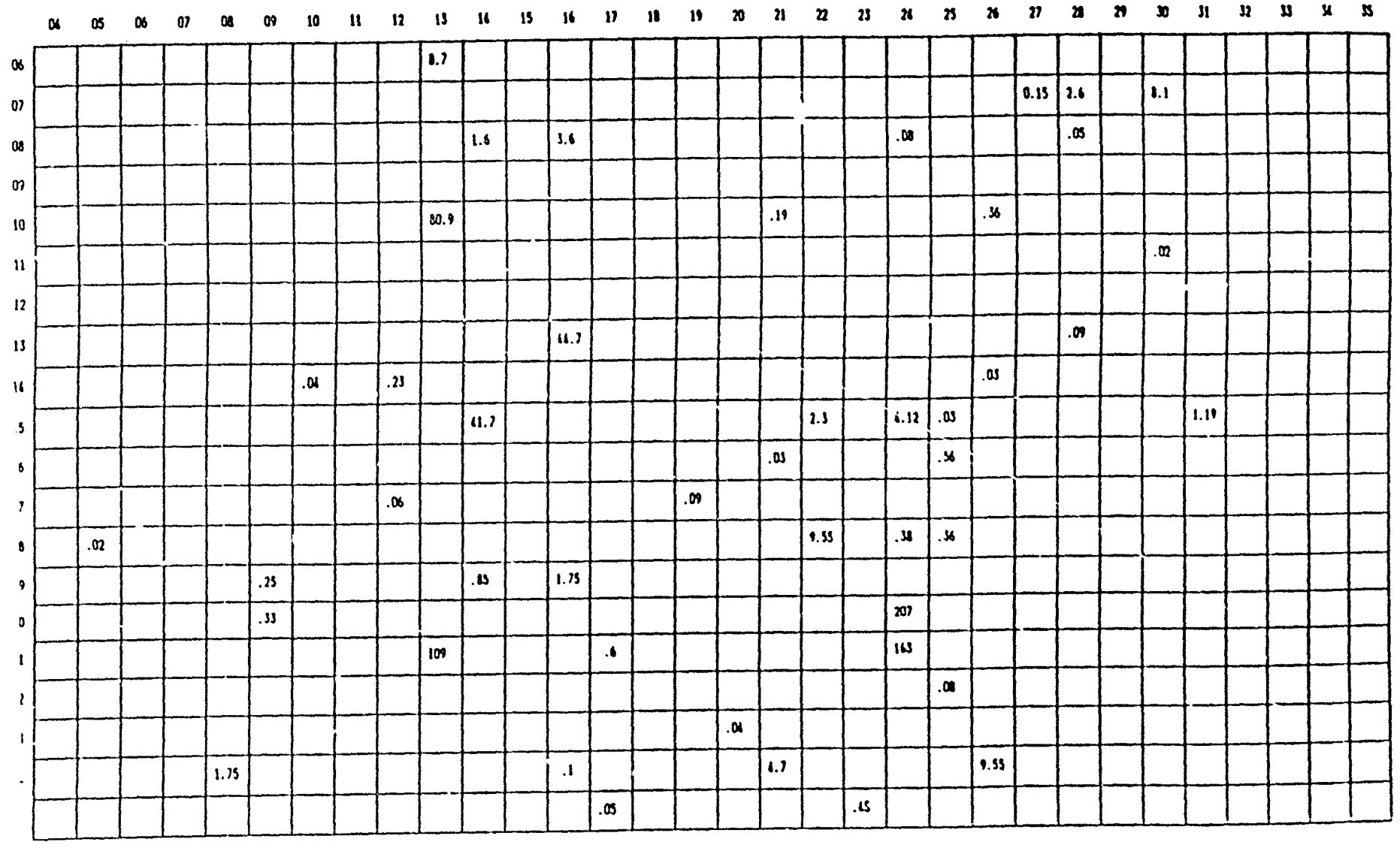


Figure 2.6 3,4-Dichlorophenoxy Acetic Acid Subsurface Soil Concentration (ppb)

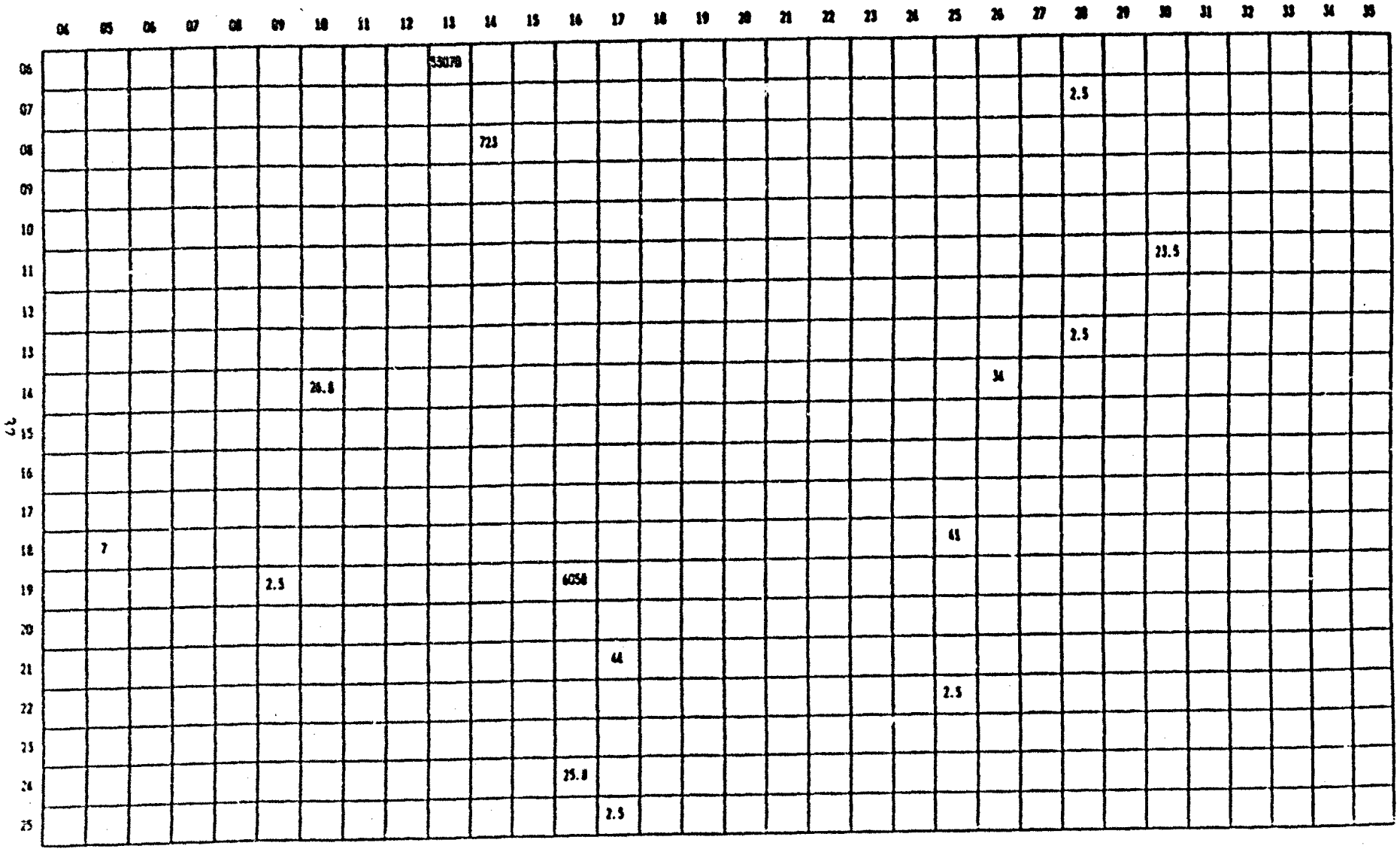
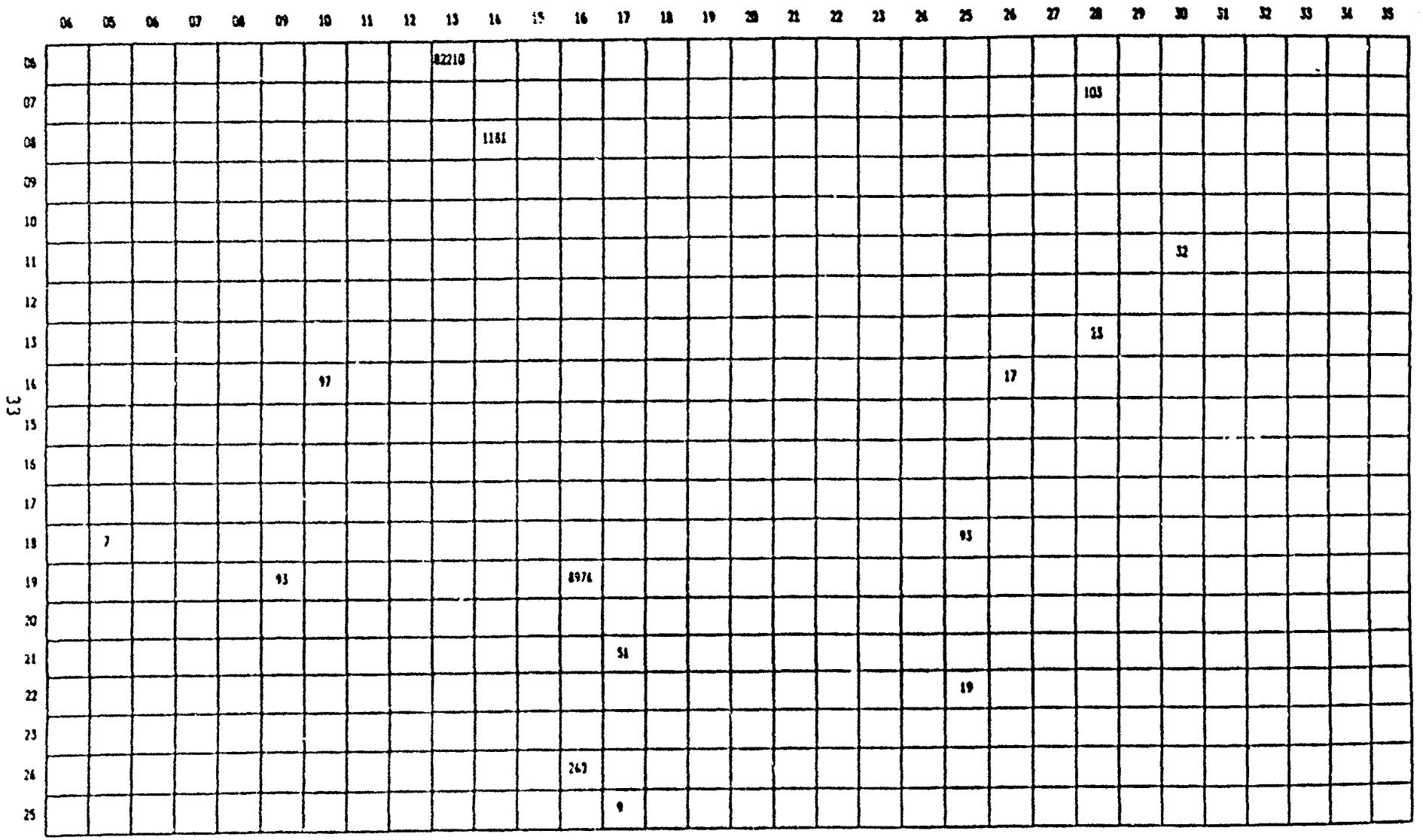


Figure 2.7 2,4,5-Trichlorophenoxy Acetic Acid Subsurface Soil Concentration (ppb)



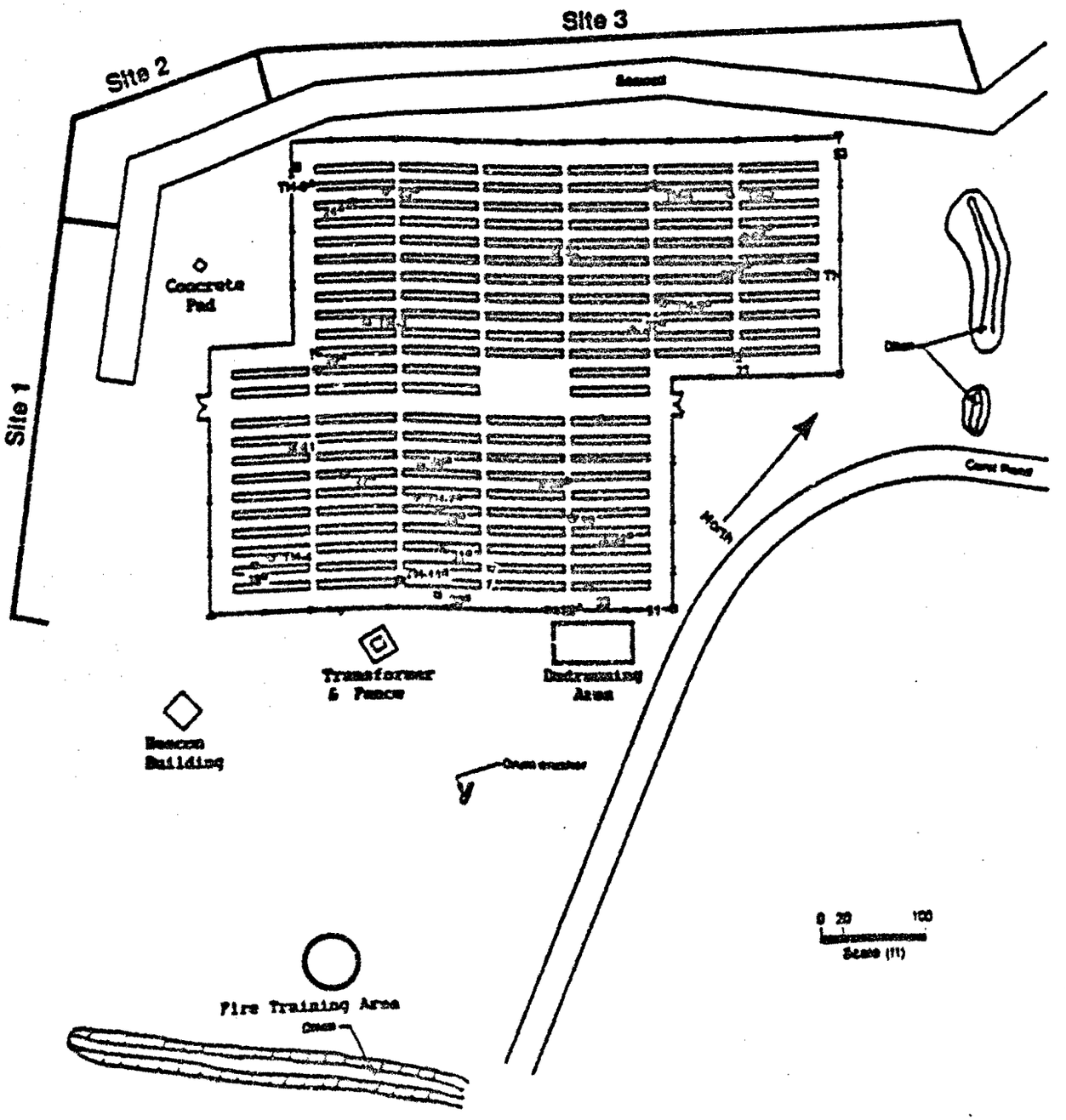


FIGURE 2.8 MARINE BIOTA SAMPLING SITES
Adapted from Crockett AB 1986

monitoring consisted of collecting one fish, one invertebrate, and one sediment sample from Sites 1 through 4. After September 1987, the monitoring program progressed to a more systematic collection procedure. Site 4, the control site, was deemed to be unnecessary because of the low frequency of positive values from Sites 1 to 3. From Sites 1 to 3, two fish from each of the following species or species groups were collected and combined:

- Bullethead parrotfish (*Scarus sordidus*) or spectacled parrotfish (*Scarus perspicillatus*);

- Convict tang (*Acanthurus triostegus*) or goldring surgeon fish (*Ctenochaetus strigosus*); and

- Goatfish (*Pseudupenus* sp. or *Mulloides* sp.).

An additional three to four fish samples from Sites 1 to 3 were collected. These fish had different feeding habits than the algal or bottom feeders listed above. The additional samples included:

- Coral feeders such as chevron butterfly (*Megaprotodon trifascialis*); predators such as eels, octopus, or jacks (*Caranx* sp.); and

- Nocturnal feeders such as shoulderfish (*Myripristis* sp.), squirrelfish (*Sargocentron* sp. or *Neoniphon* sp.), or trigger fish (*Rhinecanthus* sp. or *Melichthys* sp.).

Two to three samples of invertebrates were collected and combined. These included crabs, snails, cucumbers, gastropods, or worms. Two to four fish were collected from the west wharf. These species were to be representative of the species caught by sport fishermen on JI. One or two sediment samples from Sites 1 to 3

were also taken. It should be noted that no fish caught in wharf fishing have been analyzed.

Results of the marine biota and avian analyses are presented in Table 2.1. All avian samples were taken from Site 7. The number of marine biota and avian samples from each site are presented below and the percentages with positive residue values:

Site	Number	Positive values (%)
1	62	37
2	32	16
3	8	12.5
4	6	0
5	47	0
6	23	0
7	3	0

Eighteen samples had no site numbers. Sites 1 to 3, the areas adjacent to the HO site, generated 28.4% positive samples. From all sites combined, 16% of the samples were positive. Fourteen samples, or 7% overall, had values above 25 ppt, FDA's limit for levels in edible fish.

Results of the sediment analysis are presented in Table 2.2. Thirty-eight samples were taken; two were positive. Many samples are missing site numbers. Previously, Channell and Stoddard (1984) took three sediment samples prior to construction of the seawall on the west side of the Island. These samples averaged 57 ppt of TCDD. The authors felt that sediment contamination was due to soil runoff from the site.

Table 2.1
Johnston Island Fish Data

Sample Species	Sample Tissue	Sample Date	Site Taken	Dioxin Level PPT	Detection Limit PPT
Achilles Tang	Muscle	Sep-89	1	ND	10
Achillies Tang	Muscle	Dec-88	1	ND	10
Blackspot Sergeant	Muscle	Dec-88	1	41	10
Blackspot Sergeant	Muscle	Sep-89	1	26	10
Bluelined Surgeonfish	Muscle	Jan-88	1	ND	10
Bluelined Surgeonfish	Muscle	Dec-88	1	14	10
Bluelined Surgeonfish	Muscle	Sep-89	1	ND	10
Brick Soldierfish		Jan-88	1	ND	10
Bullethead Parrotfish	Muscle	May-87	1	ND	10
Bullethead Parrotfish	Muscle	Oct-87	1	ND	10
Coelenterate		Oct-87	1	ND	10
Cone	Muscle	May-87	1	ND	10
Cone	Muscle	Oct-87	1	18	10
Cone	Muscle	Dec-88	1	14	10
Cone Shells	Muscle	Sep-89	1	15	10
Convict Tang		May-87	1	12	10
Convict Tang	Muscle	Oct-87	1	ND	10
Convict Tang	Muscle	Dec-88	1	19	10
Convict Tang	Muscle	Sep-89	1	ND	15
Crab		Sep-84	1	ND	9
Crabs		Feb-84	1	20	
Crown Squirrelfish	Muscle	Dec-88	1	352	10
Crown Squirrelfish	Muscle	Sep-89	1	ND	10
Crown Squirrelfish	Muscle	Sep-89	1	ND	10
Dolabella	Muscle	Sep-89	1	ND	21
Doublebar Goatfish		Oct-87	1	ND	10
Eel		Sep-84	1	ND	21
Eel	Muscle	Sep-89	1	ND	10
Fish		Nov-85	1	8.9	10
Fish		Nov-85	1	13	10
Fish		Sep-86	1	ND	10
Goldring Surgeonfish	Muscle	Oct-87	1	15	10
Goldring Surgeonfish	Muscle	Sep-89	1	ND	14
Hermit Crab	Muscle	Dec-88	1	ND	10
Hermit Crabs	Muscle	Oct-87	1	ND	10
Hermit Crabs	Muscle	Sep-89	1	ND	10
Live Coral		Sep-84	1	ND	13
Manybar Goatfish	Muscle	Sep-89	1	ND	10
Moana Kali	Muscle	Sep-84	1	ND	73
Moana Kali	Liver	Sep-84	1	ND	10
Moray eel		Feb-84	1	64	
Moray eel		Feb-84	1	30	
Octopus	Muscle	Dec-88	1	28	10

Table 2.1 (cont.)
Johnston Island Fish Data

Sample Species	Sample Tissue	Sample Date	Site Taken	Dioxin Level PPT	Detection Limit PPT
Octopus	Muscle	Sep-89	1	ND	10
Orange Spine Unicornfish	Muscle	Sep-89	1	ND	10
Orangemouth Lizardfish	Muscle	Dec-88	1	21	10
Sea Cucumber		Nov-85	1	ND	10
Sea Cucumber		Sep-86	1	ND	10
Sea Cucumber	Muscle	Dec-88	1	ND	10
Sea Cucumber	Muscle	Sep-89	1	ND	10
Slipper Lobster	Muscle	Sep-89	1	ND	10
Snail		Sep-84	1	ND	24
Snails	Muscle	Oct-87	1	ND	10
Snails	Muscle	Dec-88	1	ND	10
Stocky Hawkfish	Muscle	Sep-89	1	ND	10
Tahitian & Spotfin Squirrelfish	Muscle	Jan-88	1	ND	10
Tahitian Squirrelfish	Liver	Oct-87	1	27	10
Threadfin Butterflyfish		Oct-87	1	12	10
Yellowfin Goatfish	Muscle	Dec-88	1	102	10
Yellowfin Goatfish	Muscle	Sep-89	1	11	10
Yellowfin Goatfish	Muscle	Sep-89	1	85	10
Yellowstripe & Yellowfin Goatfish	Muscle	Jan-88	1	49	10
Achilles Tang	Muscle	Sep-89	2	ND	10
Bluelined Surgeonfish	Muscle	Sep-89	2	ND	10
Bullethead Parrotfish	Muscle	May-87	2	ND	10
Cheveron Butterflyfish	Muscle	Dec-88	2	ND	10
Cone		May-87	2	ND	10
Cone		Jan-88	2	ND	10
Convict Tang	Muscle	Jan-88	2	ND	10
Convict Tang	Muscle	Dec-88	2	ND	10
Convict Tang	Muscle	Sep-89	2	ND	10
Crown Squirrelfish	Muscle	Dec-88	2	472	10
Dolabella	Muscle	Dec-88	2	ND	10
Fish		Nov-85	2	ND	10
Fish		Nov-85	2	ND	10
Fish		Sep-86	2	40	10
Goldring Surgeonfish	Muscle	Jan-88	2	ND	10
Goldring Surgeonfish	Muscle	Sep-89	2	ND	10
Hermit Crab		Jan-88	2	ND	10
Manybar Goatfish	Muscle	Sep-89	2	23	10
Moana	Whole Fish	Sep-84	2	ND	10
Octopus		Sep-84	2	ND	19
Orange Mouth Lizardfish	Muscle	Sep-89	2	ND	10
Red Snapper	Muscle	Sep-84	2	ND	10
Red Snapper	Liver	Sep-84	2	ND	14
Red Snapper	Fat	Sep-84	2	ND	25

Table 2.1 (cont.)
Johnston Island Fish Data

Sample Species	Sample Tissue	Sample Date	Site Taken	Dioxin Level PPT	Detection Limit PPT
Sea Cucumber		Jan-88	2	ND	10
Sea Cucumber	Muscle	Sep-89	2	ND	11
Snails		Feb-84	2	120	
Spectacled Parrotfish		May-87	2	ND	10
Threadfin Butterflyfish	Muscle	Dec-88	2	ND	10
Trigger Fish	Muscle	Sep-84	2	ND	10
Trigger Fish	Liver	Sep-84	2	18	
Yellowfin Goatfish	Muscle	Dec-88	2	ND	10
Fish		Nov-85	3	4.6	10
Fish		Sep-86	3	ND	10
Menpachi	Whole Fish	Sep-84	3	ND	5
Moana	Whole Fish	Sep-84	3	ND	4
Moana Papa	Muscle	Sep-84	3	ND	10
Moana Papa	Liver	Sep-84	3	ND	35
Sea Cucumber		May-87	3	ND	10
Snapper		May-87	3	ND	10
Cone		May-87	4	ND	10
Crab		Sep-84	4	ND	5
Fish		Nov-85	4	ND	10
Fish		Sep-86	4	ND	10
Fish	Liver	Sep-86	4	ND	18
Snail		Sep-84	4	ND	3
Achilles Tang	Muscle	Sep-89	5	ND	10
Ahole Hole	Whole Fish	Sep-84	5	ND	2
Ahole Hole	Whole Fish	Sep-84	5	ND	1
Ahole Hole	Whole Fish	Sep-84	5	ND	31
Ahole Hole	Whole Fish	Sep-84	5	ND	18
Ahole Hole	Whole Fish	Sep-84	5	ND	27
Blackspot Sergeant		Jan-88	5	ND	10
Blackspot Sergeant	Muscle	Dec-88	5	ND	10
Bluelined Surgeonfish	Muscle	Sep-89	5	ND	10
Convict Tang		Oct-87	5	ND	10
Convict Tang	Muscle	Dec-88	5	ND	10
Convict Tang	Muscle	Sep-89	5	ND	10
Dracula	Whole Fish	Sep-84	5	ND	3
Dracula	Whole Fish	Sep-84	5	ND	7
Dracula	Muscle	Sep-84	5	ND	7
Eel	Muscle	Dec-88	5	ND	10
Goldring Tang	Muscle	Dec-88	5	ND	10
Halalu	Whole Fish	Sep-84	5	ND	2
Lowfin Chub		May-87	5	ND	10
Lowfin Chub	Muscle	Dec-88	5	ND	10
Mackerel Scad		Oct-87	5	ND	10

Table 2.1 (cont.)
Johnston Island Fish Data

Sample Species	Sample Tissue	Sample Date	Site Taken	Dioxin Level PPT	Detection Limit PPT
Manybar Goatfish	Muscle	Sep-89	5	ND	10
Manyray Flatfish	Muscle	Dec-88	5	ND	10
Moana	Whole Fish	Sep-84	5	ND	4
Moana	Whole Fish	Sep-84	5	ND	2
Moana Kali	Muscle	Sep-84	5	ND	10
Moana Papa	Muscle	Sep-84	5	ND	300
Moana Papa	Liver	Sep-84	5	ND	10
Octopus		Sep-84	5	ND	7
Palani	Muscle	Sep-84	5	ND	10
Palani	Liver	Sep-84	5	ND	15
Palani	Whole Fish	Sep-84	5	ND	1
Papio	Muscle	Sep-84	5	ND	1
Papio	Liver	Sep-84	5	ND	1
Papio	Fat	Sep-84	5	ND	8
Papio	Muscle	Sep-84	5	ND	3
Papio	Liver	Sep-84	5	ND	6
Papio	Fat	Sep-84	5	ND	48
Parrot Fish	Muscle	Sep-84	5	ND	1
Parrot Fish	Liver	Sep-84	5	ND	22
Parrot Fish	Fat	Sep-84	5	ND	604
Parrot Fish	Muscle	Sep-84	5	ND	3
Parrot Fish	Liver	Sep-84	5	ND	3
Red Weke	Whole Fish	Sep-84	5	ND	53
Sheephead	Whole Fish	Sep-84	5	ND	1
Stocky Hawkfish	Muscle	Sep-89	5	ND	10
Yellowfin Goatfish		Oct-87	5	ND	10
Ahole Hole	Whole Fish	Sep-84	6	ND	8
Blue Ulua	Muscle	Sep-84	6	ND	1
Blue Ulua	Liver	Sep-84	6	ND	3
Blue Ulua	Fat	Sep-84	6	ND	18
Hinalaya	Whole Fish	Sep-84	6	ND	15
Hinalaya	Muscle	Sep-84	6	ND	12
Hinalaya	Liver	Sep-84	6	ND	46
Moana	Whole Fish	Sep-84	6	ND	1
Moana Papa	Muscle	Sep-84	6	ND	22
Moana Papa	Liver	Sep-84	6	ND	343
O'Paka Paka	Muscle	Sep-84	6	ND	1
O'Paka Paka	Liver	Sep-84	6	ND	7
O'Paka Paka	Muscle	Sep-84	6	ND	1
O'Paka Paka	Liver	Sep-84	6	ND	1
Palani	Muscle	Sep-84	6	ND	1
Palani	Liver	Sep-84	6	ND	3
Papio	Muscle	Sep-84	6	ND	1

Table 2.1 (cont.) Johnston Island Fish Data					
Sample Species	Sample Tissue	Sample Date	Site Taken	Dioxin Level PPT	Detection Limit PPT
Papio	Liver	Sep-84	6	ND	7
Papio	Fat	Sep-84	6	ND	6
Trigger Fish	Whole Fish	Sep-84	6	ND	1
Trigger Fish	Whole Fish	Sep-84	6	ND	3
Trigger Fish	Muscle	Sep-84	6	ND	1
Trigger Fish	Liver	Sep-84	6	ND	6
Pacific Golden Plover	Immature Male	May-87	7	ND	10
Ruddy Turnstone	Adult Male	May-87	7	ND	10
Turnstone & Plover	Liver	May-87	7	ND	10
Biota		Jun-86		ND	10
Biota		Jun-86		ND	10
Biota		Jun-86		ND	10
Fish		Nov-85		11	10
Fish		Nov-85		ND	10
Fish		Nov-85		ND	10
Fish		Nov-85		ND	10
Fish		Dec-86		ND	10
Fish		Dec-86		14	10
Fish	Liver	Dec-86		150	10
Fish		Dec-86		ND	10
Fish		Dec-86		ND	10
Liver		Nov-85		ND	10
Liver		Jun-86		ND	10
Liver		Jun-86		ND	10
Sea Cucumber		Nov-85		ND	10
Sea Cucumber		Nov-85		ND	10
Shell Fish		Dec-86		ND	10

Table 2.2 Johnston Island Sediment Data			
Sample Date	Site Taken	Dioxin Level PPT	Detection Limit PPT
Nov-85	1	ND	50
Sep-86	1	ND	100
May-87	1	ND	100
Oct-87	1	160	100
Jan-88	1	ND	100
Jan-88	1	ND	100
Jan-88	1	ND	100
Aug-88	1	ND	100
Aug-88	1	ND	100
Aug-88	1	ND	100
Dec-88	1	ND	100
Dec-88	1	ND	100
Dec-88	1	ND	100
Nov-85	2	ND	50
Sep-86	2	ND	100
May-87	2	ND	100
Oct-87	2	ND	100
Jan-88	2	ND	100
Aug-88	2	190	100
Dec-88	2	ND	100
Nov-85	3	ND	50
Sep-86	3	ND	100
May-87	3	ND	100
Jan-88	3	ND	100
Nov-85	4	ND	50
Sep-86	4	ND	100
Nov-85		ND	50
Nov-85		ND	50
Nov-85		ND	50
Nov-85		ND	50
Jun-86		ND	100
Jun-86		ND	100
Jun-86		ND	100
Jun-86		ND	100
Dec-86		ND	100
Dec-86		ND	100
Dec-86		ND	100
Dec-86		ND	100

Helsel et al. (1987) collected a variety of liquid, solid, and gas samples as part of a series of monitoring tests for evaluating thermal desorption and ultraviolet photolysis of contaminated soil. To determine if any downwind exposure occurred as a function of distance, four high-volume air particulate samplers were positioned based on the prevailing easterly trade wind direction.

The specific locations for the downwind samplers were determined by using a simple Gaussian plume dispersion model. The model estimated the distance downwind from the test area where the ground level particulate impact could be anticipated. The dispersion model used the exhaust stack of the test process as the emission point. The stack was situated approximately 15 feet above the ground surface. An average wind velocity of 11 miles per hour blowing parallel to the island's runway (i.e., 60 degrees) was used. Pasquill-Gifford Stability Class A (unstable) conditions were assumed for measuring contaminant migration during the daylight testing activities, and Stability Class D (neutral) conditions were assumed for measuring nighttime testing activities. The layout of the high-volume air particulate samplers, in relation to the Agent Orange site are shown in Figure 2.1. The sampler located nearest the east side of the site, referred to as HV-D, served as an upwind control; whereas, the remaining three samplers, HV-E, HV-F, and HV-C, were placed 80, 160, and 240 feet downwind, respectively. Sampler HV-E was used to monitor offsite migration at the predicted maximum impact location, HV-F acted as a monitor of offsite migration of contaminated particulate due to natural processes, and HV-C was used to monitor contaminated particulate migrating off the island.

The ambient air filter samples (11 samples total) were analyzed for the amount of particle-associated TCDD collected on each filter. TCDD was not detected on any of the samples analyzed. A summary of the TCDD concentrations in the ambient air filter samples is presented in Table 2.3. The detection limits presented as ng of TCDD and as air concentrations (pg/m^3). The results of this study suggest that

TABLE 2.3

Summary of 2,3,7,8-TCDD Concentrations in Ambient Air Filter Samples

Run	Migration Path Monitored[a]	Sampler	Sample Number	Quantity (ng)	Average Concentration (pg/m^3)
1	**Equipment Setup and Testing**				
	Upwind control	HV-D	R1-12A	<1.4[b]	<0.52[b]
	Offsite	HV-E	R1-12B	<2.4	<0.88
	Offsite control	HV-F	R1-12C	<1.4	<0.55
	Off island	HV-C	R1-12D	<1.1	<0.44
2	**Operation of TD/UV Photolysis System**				
	Upwind control	HV-D	R2-12A	<0.96	<0.24
	Offsite	HV-F	R2-12C	<1.1	<0.27
	Offsite control	HV-E	R2-12B	<1.5	<0.36
	Off island	JV=C	R2-12D	<0.67	<0.17
3	**Decontamination and Demobilization**				
	Upwind control	HV-D	R3-12A	<0.75	<0.25
	Offsite	HV-F	R3-12C	<0.94	<0.33
	Offsite control	no sample	---	---	---
	Off island	HV-C	R3-12D	<1.3	<0.30

[a] See Figure 2.1 for layout of air samples.
[b] Not detected. Detection limit value shown.

Source: Helsel et al., 1986.

virtually no exposure to TCDD occurred as a result of the soil decontamination experiments conducted by Helsel et al. (1987). Further, these data suggest that insignificant levels of particle-associated TCDD were dispersing from the site during the sampling period, given that these samplers were downwind of at least the southern portion of the site's total surface area, in addition to being downwind of the soil decontamination experiments. However, because of the limited number of samples and the lack of data for the entire downwind area relative to the site (i.e., the western fenceline), no conclusions can be made regarding TCDD exposure potential via inhalation of contaminated, airborne particulate at the time the samples where taken in 1986, or particularly prior to 1986, when the site was being used for storage purposes.

2.2 Data Quality Assurance

The study design and sample collection procedure for the soil study (Crockett et al., 1986) appear to be adequate. The study design was approved by EPA. However, the apparent problems that occurred during sample analysis may have been corrected, but their resolution not reported. On this basis, the quality of the soil data in this report cannot be accurately judged. Quality assurance concerns are discussed below.

The analytical procedure used in this study was adapted from an existing EPA method for dioxin analysis where the detection limit was 0.1 ppb for surface samples. The sample digestion procedure was modified and the detection limit was lowered to 0.01 ppb. There is no indication that a method validation study was performed to verify that this modified procedure worked adequately with this coral matrix and lower detection limit. [However matrix spikes at 1.0 ppb analyzed concurrently with the soil samples indicated good recoveries; accordingly, the analytical method appears to have been adequate for the coral matrix.] According to the EPA method for TCDD

analysis, sample extraction must be completed within 7 days after sample collection, and the resulting sample extract must be analyzed within 40 days thereafter. Only one laboratory, U.S. Testing Laboratories, analyzed all samples collected in this study, approximately 900 samples. With such a large influx of samples to one laboratory along with shipping problems, it is possible that the holding times may not have been met. This report did not indicate if a storage stability study was conducted to ensure the stability of samples until analysis could be performed.

Matrix spike standards and surrogate spikes were used at the 1.0 ppb level to test the accuracy of the analytical procedure. More than one spike concentration should have been used to test the accuracy of the procedure over a range of the expected soil concentrations. Spikes of 0.1 and 10 0 ppb should also have been used because these concentrations reflect the range found in many of the soil samples. A spike of 1.0 ppb is 100 times the reported detection limit, therefore the method was not rigorously tested near the detection limit. The report indicated that the average percent recoveries and the standard deviations from the matrix spike analyses were well within the guidelines of the protocol. The analytical guidelines describing data acceptability, (e.g., recovery and standard deviation ranges), were not provided with this report such that criteria used to evaluate the data is unclear. The report also indicated that five recoveries were considered outliers. Reasons for the outliers were explained only for two of the recoveries. The method used to determine why the other three values were outliers was not explained.

An independent QA/QC laboratory was utilized to perform various QA functions. The QA/QC laboratory submitted summaries of its findings in various reports, but these reports were not appended to the soil study report. The report indicated that there were several discrepancies between the performing and QA/QC laboratories. The average relative percent difference (RPD) for split sample analysis between the two labs was reported as 51% with a standard deviation of 76%. This is a large difference between the two labs. The report stated that most of the outliers

had RPD's of 200%, and they represented sample pairs where one sample value was not detected and the other value was low. An RPD of greater than 200% was also reported for split sample analysis within the performing laboratory for the same stated reasons. This indicates that the analytical method used may not have been as rugged near the detection limit as originally intended. Other discrepancies between the two labs included differences in results from field performance audit samples and performance evaluation standards. As stated above, these discrepancies may have been resolved, but this report did not discuss if they were or how.

The report stated that two field blanks, considered as outliers, were not rerun because the level of contamination at 0.2 ppb was not considered significant. A review of Figure 7 in the report shows that approximately 46% of the samples had values at 0.5 ppb or lower. The report did not indicate how many samples were collected with these positive blank samples, nor did it indicate if the positive sample blank values were subtracted from the positive soil samples. If the positive sample blanks were not subtracted from the positive soil values, then some of the reported positive soil samples could be false positive values.

The sample collection protocol for fish, sediments, and birds was made more systematic in October of 1987, but it still appears to be lacking in some aspects. The protocol does not specify that different stages in the fish life cycle be sampled. This information would be helpful to determine to what degree the adult fish are bioaccumulating the contaminants. Not all trophic levels of the marine biota have been sampled, (e.g., filter feeders). No systematic protocol has been established for sediment sampling. Many of the reports did not specify the exact location where the sediment samples were taken. Channell and Stoddart (1984) noted three positive sediment sample near the shore on the west side of the site. This area should be resampled to determine if the seawall is preventing further contamination of the lagoon. Only three birds have been sampled; more birds should be sampled to assess the possible impact of the site on the nesting birds. There are no data for 2,4-D or

2,4,5-T in fish, sediment, or birds, and there are no data for TCDD, 2,4-D, or 2,4,5-T in sea water and in groundwater under the site.

Data validation for the fish, sediment, and avian analyses can not be performed for several reasons. First, the exact EPA method used to analyze these samples was never mentioned in the reports. Second, there are no data from the performing laboratory on their QA/QC procedures, or results of their QA/QC analyses. Percent recovery data were given, but comprehensive data validation cannot be made on this one piece of QA/QC data. Third, since the samples must have been shipped a great distance, there is no information on whether a storage stability study had been performed.

2.3 Summary of Chemicals of Potential Concern

Herbicide Orange (HO) was used in two different formulations (U.S. Air Force, 1974). Orange was composed of a 50:50 mixture of n-butyl 2,4-dichlorophenoxyacetic acid and n-butyl 2,4,5-trichlorophenoxyacetic acid. Orange II was composed of a 50:50 mixture of n-butyl 2,4-dichlorophenoxyacetic acid and isooctyl 2,4,5-trichlorophenoxyacetic acid. The ratio of these two lots on JI was not known. The arithmetic mean TCDD concentration on JI was determined to be 1.909 mg/kg (U.S. Air Force, 1974). The sample analysis did not differentiate between the two 2,4,5,-T compounds. The only dioxin isomer tested in all of the samples was 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD). Other isomers of dioxin could have been present in the HO, and therefore could also be contaminants at the HO site. Both phenoxy herbicides and TCDD have been detected at the site, and TCDD has been detected in biological samples. Therefore, these three chemicals are of potential concern, along with any other possible isomer of dioxin as of yet unanalyzed.

3.0 Exposure Assessment

The following section describes the procedures used for conducting the exposure assessment for the HO site. The objective of the exposure assessment is to estimate the type and magnitude of current exposure and, to the extent possible, future exposures to the chemicals of potential concern at JI. The exposure assessment methods used in this evaluation are those described in various documents developed by the U.S. Environmental Protection Agency (EPA) and include Cowherd *et al.* 1985, EPA 1988b, EPA 1988c, EPA 1989a, EPA 1989b, and EPA 1989c. The methods used in the exposure assessment for the HO site at JI include consideration of the exposure setting and the exposure pathways which are of particular relevance to the types of human populations present and their respective activity patterns. This section presents the following:

(1) Characterization of the physical setting of the HO site and the resulting potentially exposed populations;
(2) Descriptions of the identified plausible exposure pathways;
(3) Estimations of human exposure; and

(4) Identification and discussion of uncertainties related to the exposure assessment methods used in this evaluation.

3.1 Characterization of the Exposure Setting

The potential for exposure is dependent on the physical setting of the HO site, including the climate, vegetation, soil type, and hydrology, as well as the features of the potentially exposed population, dependent on population characteristics and land use.

3.1.1 Physical Setting

The physical setting of JI has been extensively characterized and reported (U.S. Air Force, 1974; Thomas et. al., 1978). The features are briefly synopsized below.

The climate is marine and tropical with little variation in temperature, wind speed, and wind direction over its entire surface due, in part, to the small land area, uniform terrain, and low elevation. The mean temperature is 79°F ranging from 62°F to 89°F. The mean annual rainfall is 26 inches; the lowest annual rainfall recorded was 13 inches and highest 42 inches. The annual mean relative humidity is 75%.

Wind characteristics are important for the dispersion modeling component of exposure via the air medium. The mean annual windspeed is 15 mph with little variation throughout the year due to dominating surface trade winds. Monthly means are 14 mph to 16 mph. Winds are from the northeast and east 85% of the time, at least 62% of the time in every month. Occasionally from December through March, the winds are light and variable or westerly.

Mean monthly sky cover, sunrise to sunset, averages 6 on a scale of 0 to 10 with little variation.

To a large extent, the type and density of vegetation is determined by the amount of rainfall. To a lesser extent at the HO site, it is influenced by residual levels of 2,4-D and 2,4,5-T. Vegetation consists of a few grasses, herbs, and dwarf shrubs. Most are not indigenous and have been introduced to JI by humans. Terrestrial animal life is equally limited in variety. These are described in Section 6.0.

Soil is the most critical physical component of the Island with respect to risks posed by the HO site because it is the medium within which the chemical contaminants of concern are contained. Environmental fate and transport, which characterizes the movement of the contaminants from the soil medium, is largely dependent on the soil type and its ability to release or retain them. The surface of JI is mainly coral sand with a mixture of fine coral fragments. The area of the HO site is not part of the original Island but, through dredging and reconstruction, was built up artificially with alternating layers of coral and sand of various consistency and porosity. Beach rock on the Island is formed by sand and coral gravel loosely cemented together by calcium carbonate. The HO site has been left relatively undisturbed since the dedrumming operation (a trial soil burn and comprehensive soil sampling program are the only major activities to have occurred for relatively brief time periods). As a consequence, most of the loose fines on the surface have been blown away, leaving the surface covered with a combination of cobble-sized or compacted coral fragments. The soil has not been well characterized for its physical features (composition, density, porosity, pH, organic content). During the most recent chemical characterization study (Crockett et al., 1986), moisture content was determined to be approximately 9.57% and 9.0% by air and oven drying, respectively.

There is no surface water on the HO site due to the coarse texture and extreme permeability of the coral sand and rubble within the first few feet of the regolith. Groundwater on the Island lies in general at a depth of 1.2 to 2.4 meters (4 to 8 feet). The aquifer under the HO site, if it exists, has neither been characterized nor its chemical composition determined. A thin lens of brackish water (dissolved solids greater than 1,000 mg/L) that is rust colored and has an odor of hydrogen sulfide underlies the original Island. Characteristics of the groundwater are important for determining the fate and transport of contaminants at the site.

3.1.2 Current and Future Land Use Conditions

The site is currently not in use, is dormant, and has access limited by a surrounding fence. Potential avenues of human exposure include volatilization of the contaminants into the air, suspension of particle-laden contaminants into the air, and consumption of edible marine life that have become contaminated in the waters adjacent to the site.

Two future scenarios that would alter exposure potential from that presented by current land conditions and which form the basis of the quantitative estimations of risk in this analysis are: (1) remediation through excavation and incineration[2] of contaminated soil; and (2) covering of the site with cement. The latter scenario is not intended to be a substitute for prescriptive site capping, which is a more thorough and rigorous form of remediation. In both of these scenarios, certain activities such as construction vehicles on the site and excavating alter the patterns of particulate suspension and soil volatilization of contaminants from those in the current use scenario. These are explained in Section 3.3 as they are incorporated into the calculation of emission factors and exposure estimation.

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[2] Although incineration is a plausible remediation alternative, potential exposures resulting from incinerator emissions during thermal desorption and combustion of TCDD, 2,4-D, and 2,4,5-T in soil were not included in this evaluation.

3.1.3 Potentially Exposed Populations

The permanent and semi-permanent Island population is a mixture of military personnel whose stay on JI generally ranges from one to three years and civilians employed by a DoD service contractor who remain on JI for longer periods. Some individuals have been on JI for over 15 years and at least two who are still on JI were involved in the HO dedrumming operation. Any occupational and recreational activities of these individuals at certain distances downwind of the HO site create a potential for exposure to contaminants at the site. These activities are a matter of specific job functions and responsibilities of individuals as well as lifestyle on the Island.

The circumstances that create a potential for human exposure are related *not* to activities at the site itself (it is assumed that individuals working on the actual site would be wearing appropriately protective gear and clothing), but rather to activities beyond the boundary of the HO site (Figure 2.1).

For exposure through the air medium, these activities include but are not necessarily limited to any occupational operations associated with the seawall, the electrical transformer, the Hi-Vol sampler, the beacon building in the immediate area, the fire training area, the rip-rap area used as a boat-launch site, and the burn pit at an intermediate distance. The time that an individual is located in these areas conducting operations related to facilities for any one episode and the frequency with which these areas are visited is variable. As important components in the calculation of potential human exposure, it was necessary to assume reasonable values for time and frequency within the range of 0 to 24 hours per day, 0 to 7 days per week. Typical values used for atmospheric dispersion estimates are one hour, eight hours, and annual averages concentrations (e.g., mg/m^3), which are usually based on continuous exposure. Without the benefit of actual time-activity data and considering the structures around the site, their functions, and the need to choose exposure

parameters that are conservative but nevertheless reasonable, a value of 1 hour per day, five days per week was assumed to be appropriate for the time and frequency that an individual would be located in proximity to the site. This represents a reasonable approximation, although actual values may be greater or lesser.

Sport fishing presents a potential for exposure through the food chain, since fish sampling data indicate a potential for TCDD exposure though consumption of contaminated fish. Sport fishing is an important recreational activity on Johnston Atoll (JA). Approximately 350 boxes of frozen fish are exported each year for home leave (Irons et al., 1990). Many fishermen give some of their catch to nonfishermen for consumption on the island, and for export during home leave. Fishing is conducted from the shorelines around the islands and from boats. Both line fishing and spear fishing are allowed on JA. Line fishing is conducted both at night and during the daytime. The only area that is off limits to fishing is the area adjacent to the former HO site out to the shipping channel. Residents are aware of this restriction and it is not violated. Fishing is allowed on the other side of the channel out to the reef (Zone 5 in Figure 3.1). Irons et al. (1990) has conducted an extensive fish catch survey to characterize the fish population on JA, a portion of which is attached in Appendix A of this report.

3.2 Identification of Exposure Pathways

The identification of exposure pathways involves consideration of the environmental fate and transport of a chemical in media where its presence has been detected and if possible, quantified, as well as human activities which may present opportunities for exposure to occur. An exposure pathway generally consists of four elements:

(1) A source and mechanism of chemical release;

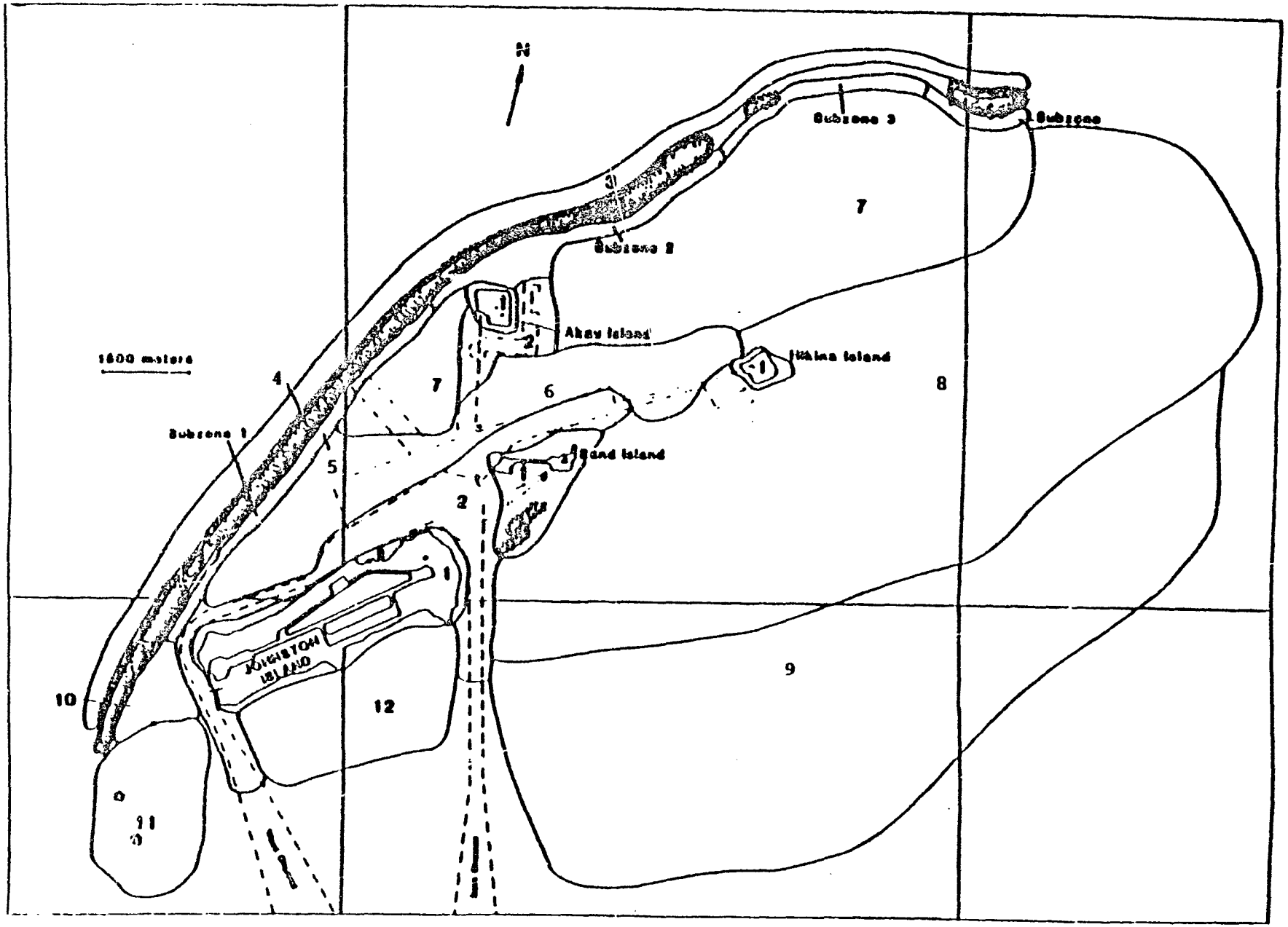


FIGURE 3.1 "ECOTYPE" ZONES (AREAS WITHIN LINE BOUNDARIES) AT JOHNSTON ATOLL From Irons 1990

(2) A retention or transport medium;

(3) A "point" of potential human contact with the chemical or contaminated medium; and

(4) An exposure route (e.g., inhalation, ingestion, or dermal contact) by which the chemical may be absorbed into the body.

The following sections (3.2.1 through 3.2.3) present the plausible exposure pathways for persons at JI which form the basis for quantification of exposure in Section 3.3.

3.2.1 Identification of Sources and Receiving Environmental Media

As described in Section 1.2, the primary source of environmental release of HO at JI (i.e., corroded steel drums containing HO) was removed in 1977. However, contaminated soil has subsequently served as a source for environmental release of the active ingredients of HO (i.e., 2,4-D, 2,4,5-T) and the contaminant TCDD. As described in Section 2.0, the environmental media which has been sampled and analyzed is the soil directly beneath the HO storage site. In addition, ocean sediment and limited fish species, which are native to the reef surrounding the island, were caught and subjected to tissue analyses. The soil samples were analyzed for TCDD, 2,4-D, and 2,4,5-T, whereas the fish tissue and sediment samples were analyzed for TCDD only. Based on an evaluation of the sampling data provided to RiskFocus (see Section 2.0), the receiving media for the contamination is the soil at the site and apparently, through an unknown mechanism, the aquatic biota near the site. Air and groundwater sampling has not yet been performed and thus, cannot be evaluated as to their potential significance as receiving media (see Section 7.0).

Potential significant mechanisms of release for TCDD, 2,4,-D and 2,4,5-T from the soil at the HO site include volatilization and emission as soil-associated airborne particles (EPA, 1988b). Emission of the compounds adsorbed to airborne particulate matter is particularly important to consider if the surface of the soil at the HO storage site is disturbed (e.g., during excavation) which creates dust emissions from activities such as vehicular traffic and of vehicular loading and unloading of contaminated soil and which allows wind erosion to occur unless dust control measures are taken (EPA, 1988b). Wind erosion of the undisturbed soil at the HO site is assumed not to be significant for several reasons:

- JI experiences continuous air movement (see Section 3.1) across the island's surface. Thus, any fine particles available for erosion would have eroded soon after activity ceased on the site in 1977, leaving it relatively undisturbed with the exception of the most recent soil sampling effort (Channell and Stoddart, 1984);

- Based on direct observation during a site visit in 1990, the particle size distribution of the surface soil at the site was found to include large coral rocks which would tend to prevent wind erosion; and

- Vegetation covers approximately 20% of the surface area of the HO site, further preventing significant wind erosion.

- Helsel et al. (1987) conducted a study in 1986 which included sampling airborne particles and subsequent analysis of TCDD levels; this study suggested that particle-associated TCDD was not dispersing from the undisturbed site.

Other release processes (EPA, 1989a) that may be important are apparent from the fish tissue data. These data suggest that one or both of the following release processes may also be important:

- Leaching of TCDD (and possibly 2,4,D and 2,4,5-T) from the soil via surface and ground water migration into the ocean; and

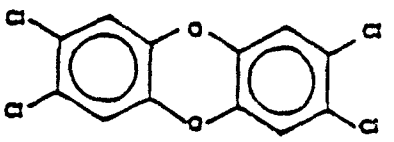
- Migration of contaminated soil particles into the ocean due to water drainage.

The rate and extent of bioconcentration of these compounds in the local reef ecosystem cannot be assessed with the available data. Similarly, without air sampling data (e.g., vapor phase and particulate matter) the extent to which the compounds may be directly volatilizing or emitted as contaminated dust from the site is unknown. The next section (3.2.2) presents further rationale for the exposure pathways of potential concern based on physicochemical characteristics, and the environmental fate and transport of these compounds.

3.2.2 Evaluation of Environmental Fate and Transport

3.2.2.1 Environmental Fate and Transport of Dioxin

Polychlorinated dibenzo-p-dioxins are tricyclic aromatic compounds consisting of two benzene rings connected through oxygen atoms and containing a varying number of chlorine atoms at different positions on the benzene rings. There are 75 possible isomers of polychlorinated dibenzo-p-dioxins (EPA, 1979). Most of the environmental fate and transport data on this class of compounds are on the 2,3,7,8 isomer. Its structure is shown below.



2,3,7,8-Tetrachlorodibenzo-p-dioxin

TABLE 3.1
Physical/Chemical Properties of Constituents of Herbicide Orange Found at Johnston Island
Herbicide Orange Storage Area
Johnston Island, Johnston Atoll

Chemical Name	Molecular Weight	Specific gravity	Water solubility (mg/L)	Vapor pressure (mm Hg)	Henry's Law Constant (atm-m^3/mol)	Log (K_{oc})	Log (K_{ow})
2,3,7,8-Tetrachloro-dibenzo-p-Dioxin[a]	321.97	1.827	1.93×10^{-5}	1.52×10^{-9}	8.1×10^{-5}	6.0-7.39	6.15-7.28
2,4-Dichlorophenoxy acetic acid[b] (n-butyl ester)	277.15	No data	2.47	4.62×10^{-6}	6.8×10^{-7}	4.0	4.60
2,4,5-Trichlorophenoxy acetic acid[b] (n-butyl ester)	311.59	1.316-1.340[d]	0.268	5.08×10^{-7}	7.77×10^{-7}	5.0	5.34
2,4,5-Trichlorophenoxy acetic acid[b] (Iso-octyl ester)	367.7	1.2-1.22[d]	NA[c]	6.12×10^{-9}	NA[c]	NA[c]	7.33

[a] Values from ATSDR, June 1989.
[b] All values except specific gravity estimated by GEMS.
[c] Not available (no estimation method available).
[d] From Department of the Air Force, 1974.

TCDD is formed as a byproduct under the conditions of synthesis of polychlorinated phenols and products formed from them, including the herbicide 2,4,5-T. The amount of TCDD occurring in 2,4,5-T appears to vary with each batch and with each manufacturer (EPA, 1979). Table 3.1 lists the key physical properties of 2,3,7,8-TCDD. The ultimate environmental fate of 2,3,7,8-TCDD appears to be strong adsorption to soils and sediments and bioaccumulation in biota.

(1) *Soil*. Once 2,3,7,8-TCDD moves into soils, it is strongly sorbed and only limited migration through the soil is expected to occur [(as suggested by its low water solubility (200 ppt)] and high log K_{OC}) unless organic solvents are present that are able to elute the compound from the soil particles (EPA, 1990). Transport of 2,3,7,8-TCDD through or from contaminated soil occurs to a limited extent through:

- Slow movement of the compound through the soil column as a result of leaching;
- Overland transport of contaminated soil particles as runoff;
- Wind erosion; and
- Diffusion of 2,3,7,8-TCDD vapor through the soil pore spaces and ultimately to the atmosphere (EPA, 1988b).

The latter process, however, is expected to be slow due to the high affinity of the compound for soil particles and the low vapor pressure of 2,3,7,8-TCDD (on the order of 10^{-9} to 10^{-11} mm Hg at 25°C) (EPA, 1990). As a result, the half-life of volatilization from soil is measured in weeks for surface soil and in years for 2,3,7,8-TCDD occurring below 5 cm of soil (EPA, 1990).

Chemical degradation of 2,3,7,8-TCDD via hydrolysis or oxidation in soil is unlikely to be an important fate process in light of the very low rate constants for these reactions in aqueous media (EPA, 1988b). Laboratory studies indicate that after deposition of 2,3,7,8-TCDD onto surfaces, there is initially a high loss due to photodegradation in the presence of hydrogen donors, and possibly volatilization (EPA, 1990). However, there is little evidence to support the suggestion that photolysis plays a significant role in the fate of 2,3,7,8-TCDD in soils, especially when the compound occurs in horizons below the soil surface (EPA, 1988b). Some loss due to the biodegradation by microorganisms in the soil may occur, but the extent of loss through this mechanism is highly dependent on the type and concentration of organisms present in the soil; under most circumstances; biodegradation is not expected to make a significant contribution to the fate of 2,3,7,8-TCDD (EPA, 1988b).

(2) *Water.* The major fate of 2,3,7,8-TCDD in aquatic ecosystems is related to adsorption and loss to sediments and suspended particulate matter, due to the low water solubility and high K_{OC} of this compound. Half-lives in water due to photolysis, as estimated from quantum yield data, are from roughly 1 to 4.6 days; however measured half-lives of 2,3,7,8-TCDD in water due to photolysis exceed 28 days (EPA, 1990). 2,3,7,8-TCDD is probably stable to oxidation in aquatic environments, based on limited data (EPA, 1990). There is no available evidence that 2,3,7,8-TCDD would be degraded to any extent by hydrolysis in water (EPA, 1990). The estimated Henry's Law constant of 1.6×10^{-5} atm-m^3/mol suggests that 2,3,7,8-TCDD may volatilize from water and enter the atmosphere.

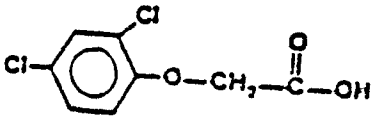
(3) *Sediments.* 2,3,7,8-TCDD is transferred to sediments via leaching from contaminated soil, runoff of contaminated soil particles, and precipitation of resuspended contaminated soil particles and vapor (adsorbed to particles or in rainfall) from the atmosphere into bodies of water. As with soil, microbial degradation is expected to be slow and, hence, not an important fate mechanism for this compound.

(4) ***Air.*** The air over a contaminated site will contain limited amounts of 2,3,7,8-TCDD as a result of slow volatilization from the soil and resuspension of contaminated soil particles from the site. Laboratory studies indicate that indirect photolysis occurs through reaction of atmospheric hydroxyl radicals with 2,3,7,8-TCDD, indicating a half life of airborne gaseous 2,3,7,8-TCDD in sunlight of 5 to 24 days (EPA, 1990). Methods for estimating photolysis half life are inconsistent with measurements in the laboratory, producing values of 1 to 200 hours as the half-life (EPA, 1990).

(5) ***Biota.*** 2,3,7,8-TCDD has been shown to be bioavailable to fish and other aquatic organisms primarily from sediments (EPA, 1988b). In fact, of the possible substituted dioxin isomers in the tetra- through octachlorinated homologous series, the 2,3,7,8 isomer has the highest bioaccumulation in fish (EPA, 1988b). The extent of actual bioaccumulation will depend on the species, lipid content, ratio of surface area to weight, food intake rate, density of suspended particulate matter, the time each species spends in given contaminated areas, and the concentrations of the compound in the contaminated sediments (EPA, 1988b). Marine biota may bioaccumulate 2,3,7,8-TCDD from intake of sediments, from intake of contaminated food, and via absorption from external surfaces (although the latter is probably a minor route). While no data exist to determine whether a correlation exists between the bioconcentration factor (BCF) and concentration in the water for marine species, studies with warm- and coldwater freshwater species indicate that the lower the water concentration, the higher is the BCF observed (EPA, 1990). Estimated BCFs for 2,3,7,8-TCDD based on measured versus estimated Log K_{OW} values range from 3,000 to 68,000 and from 7,000 to 900,000, respectively (EPA, 1984). Adequate measured data to characterize the actual range of BCFs for marine species for 2,3,7,8-TCDD are not available. Measured data for freshwater fish include a whole-body BCF of 2,000 for channel catfish (after 28 days) and a steady-state BCF of 5,450 to 9,270 in rainbow trout (EPA, 1984). Section 6.0 of this report contains additional information on the uptake of TCDD in biota.

3.2.2.2 Environmental Fate and Transport of 2,4-D

The chemical structure of 2,4-D is shown below.

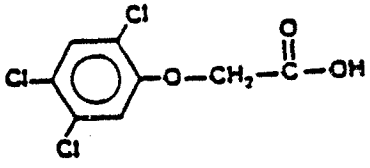


2,4-D

There is only limited fate information available on 2,4-D; however, its environmental fate and transport properties can at least be inferred in part from the physicochemical properties listed in Table 3.1. The log K_{OC} value of 4 (K_{OC} = 10,000) indicates that 2,4-D will absorb strongly to soil, but 100 or more times less tenaciously than 2,3,7,8-TCDD. Due primarily to the higher water solubility of 2,4-D relative to that of 2,3,7,8-TCDD, 2,4-D will volatilize even less than 2,3,7,8-TCDD from contaminated waters, as suggested by the difference in Henry's law constant. Because of its lower log K_{OW}, 2,4-D is expected to bioaccumulate in fish to a much lesser extent than 2,3,7,8-TCDD. Because the magnitude of its vapor pressure is 3 orders greater than that of TCDD, 2,4-D is expected to volatilize to a greater extent from contaminated soil. 2,4-D is biodegraded by soil microorganisms, and there is reportedly no accumulation of 2,4-D in soil as a result of normal agricultural use (IARC, 1977). Based on experience in Southeast Asia, less than or equal to 0.02 percent of the amount originally applied remained in the soil after 6 to 7 years (IARC, 1977). 2,4-D is reported to have a half-life of considerably less than 28 days in sediments from freshwater ponds (IARC, 1977).

3.2.2.3 Environmental Fate and Transport of 2,4,5-T

The chemical structure of 2,4,5-T is shown below.



2,4,5-T

There is only limited fate information available on 2,4,5-T; however, its environmental fate and transport properties can at least be inferred in part from the physicochemical properties listed in Table 3.1. The fate properties of 2,4,5-T closely resemble those of 2,4-D. Thus:

- Strong adsorption to soil is expected, but not as high a binding strength as with 2,3,7,8-TCDD;
- Less volatilization from water and greater volatilization from soil are expected relative to 2,3,7,8-TCDD; and
- Less bioaccumulation is fish and other marine life is expected relative to TCDD.

2,4,5-T is reported to be biodegraded more slowly than 2,4-D by soil microorganisms; however, it is also reported that no accumulation of 2,4,5-T in soil occurs as a result of annual agricultural applications (IARC, 1977). Based on experience in Southeast Asia, less than or equal to 0.3 percent of the original applied amount remained in the soil 3 to 5 years after application (IARC, 1977).

3.2.3 Identification of Exposure Points and Routes

Based on the current exposure setting at the HO site, the physicochemical properties of TCDD, 2,4-D, and 2,4,5-T, their fate and transport, and the currently available environmental sampling data for soil and fish tissue, the following exposure pathways were considered in evaluating potential current exposures:

Current Scenario:

(1) Inhalation of vapor-phase TCDD, 2,4-D, and 2,4,5-T by persons working near the existing site (see Section 3.1.4); and

(2) Ingestion of contaminated fish.

Similarly, two proposed future-use scenarios for the HO site were considered based on options for future use known to have been considered by the U.S. Air Force (Jeffers, 1984):

(1) Excavation of the contaminated soil and concurrent treatment by incineration; or

(2) Construction of a cement layer on top of the entire HO site for use as a storage depot.

Thus, based on the activities associated with these scenarios and consideration of the currently available soil sampling data, the following potential future exposure pathways were considered for:

Future-Use Scenario:

- *Scenario 1 (Excavation)*: Inhalation of contaminated soil from vehicular traffic, loading and unloading operations during site excavation and treatment, and wind erosion of disturbed soil.

- *Scenario 2 (Cement Covering)*: Inhalation of contaminated soil from vehicular traffic and wind erosion of disturbed soil.

For both of the future-use scenarios, direct exposure to workers engaged in the remediation activities was not considered likely. It was assumed that these individuals would be adequately protected by personal protective equipment (e.g., clothing, gloves, respirators) used site remediation/modification involved in the two future-use scenarios. Thus, the exposure points (receptor sites) being evaluated include inadvertent exposure to individuals working near the site (see Section 3.1.4).

3.3 Quantification of Exposure

3.3.1. Estimation of Reasonable Maximum Exposure

The theoretical most exposed individual (MEI) is assumed to represent the risk receptor. This is consistent with procedures recommended by the EPA (1989c). In this assessment, risk to the MEI is based on access to any point around the perimeter of the HO site (including the seawall) and selection of the maximum point of exposure around the perimeter. However, in actuality there are certain limitations to where the MEI can be situated because of the real limitations on access to the site. Therefore, risk to an alternate, more realistic MEI (a person who has "reasonable maximum exposure"), restricted to the fenceline and not the seawall, is also calculated for comparison. As a result, risk is calculated for two receptors, the theoretical MEI (TMEI) and the alternate MEI (AMEI).

3.3.2 Inhalation of Vapors

As discussed in Section 3.2.2, volatilization is an important mechanism by which TCDD is depleted from the soil (EPA, 1988b). Further, based on EPA's analyses, the fate of TCDD in soil is so slow by water leaching that other transport mechanisms, such as volatilization and erosion, are much more important. However, in view of the very low vapor pressure of TCDD, volatilization itself may be an extremely slow process depending upon variables such as diurnal temperature changes on the surface of the soil, as well as concurrent processes such as photolysis of the compound at the surface, and microbial degradation (EPA, 1988b). Given the similar physicochemical properties of 2,4-D and 2,4,5-T, vapor-phase emission is also considered to be an important release mechanism for these compounds.

To assess potential inhalation exposure from vapor-phase TCDD, 2,4-D, and 2,4,5-T originating from contaminated soil at the HO site, a screening-level air modeling analysis was conducted to estimate one-hour, eight-hour, and annual average concentrations of these compounds at the fenceline of HO site beginning after removal of the drums containing HO. These predicted air concentrations were then used to estimate inhalation exposure to individuals working near the site (proximate to the fenceline).

The EPA-approved Industrial Source Complex (ISC) model (EPA, 1987) was used in a screening mode to conservatively estimate ambient air concentrations of the vapor-phase compounds. Model runs were made for wind directions every 10 degrees around the compass (36 runs total), starting from north (0 degrees). A wind speed of 1.0 m/s and an extremely stable atmosphere (Pasquill stability category 6) were assumed in the modeling.

A total of 140 ground-level, non-buoyant, point sources were used to represent the area of compound emissions in the modeling. The main HO site was extended

westward to the shoreline to include isolated TCDD "hotspots" and this identical area was used for estimating 2,4-D and 2,4,5-T emissions (Figure 2.1).

Individual sample blocks with nondetectable measurements of the compounds (labelled "ND") were each assigned a value of one-half the detection level (EPA, 1989), whereas missing values within the fenceline were assigned the median value for all plots sampled and analyzed at the site (Figure 3.2, 3.3, and 3.4). Finally, for purposes of modeling point emission sources across the surface of the soil sampling grid, a point source was located at the center of each four-plot sampling area. The soil concentration of TCDD, 2,4-D, and 2,4,5-T for each point source was calculated by averaging the four measured concentrations (ppb) associated with the set of four adjacent sample plots (see Figures 3.5, 3.6, and 3.7).

Methods developed by EPA for estimating exposures to TCDD (EPA, 1986a; Hwang and Falco, 1986) were used to calculate time-averaged compound vapor-phase emission rates for TCDD as well as 2,4-D and 2,4,5-T. It is important to note that environmental fate processing (e.g., photolysis, microbial degradation) which reduce the concentration of these compounds in soil over time are not accounted for using this estimation procedure; thus, the emission rate estimates represent overestimates for long exposure durations (e.g., greater than approximately 10 years). These emission rates (N_D), expressed as grams per cm^2 per second, were estimated for each four-plot average soil concentration as follows:

$$N_D = (2D_i)\,(e^{\frac{4}{3}})\,(K_{ai})\,\frac{(C_{ai})}{\sqrt{3.14\alpha T}} \qquad \text{(3-1)}$$

Figure 3-2. TCDD (Dioxin) Surface Soil Concentrations (ppb): Measured and Estimated Values

	04	05	06	07	08	09	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26	27	28	29	30	31	32	33	34	35
06					0.8	0.8	0.8	0.8	0.005	8.7	0.005	0.005	0.1	0.1	0.1	0.1	31	0.005	0.005	0.1	1.3	0.005	7.0	0.005	0.005	0.8	0.4	0.005	0.005	0.005	0.005	0.005
07		0.8	0.8	0.8	0.8	0.8	0.8	0.8	0.3	0.005	1.5	3.0	1.4	17	13	24	27	5.0	6.8	0.1	2.5	45	0.8	20	19	18	1.4	13	6.4	57	0.8	0.005
08		0.8	0.8	0.8	0.8	0.6	0.8	0.8	136	163	91	20	6.7	0.8	1.1	0.3	0.1	9.5	0.005	0.1	0.005	0.2	3.1	0.2	0.005	0.2	1.9	61	0.2	0.2	32	39
09	0.8	0.8	9.6	0.8	0.8	0.8	0.8	0.8	26	50	46	21	7.7	0.5	9.4	0.005	0.2	0.005	0.005	0.005	0.1	19	0.5	0.005	0.005	9.8	0.5	0.6	2.6	25	1.7	0.1
10	0.8	0.8	77	0.8	0.8	0.8	0.8	0.8	0.9	26	59	25	7.2	0.8	0.8	0.3	0.1	0.4	1.3	0.2	0.3	0.005	1.4	0.005	0.005	18	1.6	1.1	20	8.5	0.4	0.2
11	0.8	0.8	0.8	0.8	0.8	0.8	0.2	0.8	0.2	5.0	18	1.8	0.005	0.1	12	61	0.5	12	0.8	0.8	1.9	1.2	31	0.4	34	0.3	5.2	1.7	0.9	10	6.6	0.3
12	0.8	36	0.8	0.8	0.8	0.8	0.8	0.2	0.7	0.3	2.2	0.005	0.005	0.005	0.005	0.005	0.005	5.6	2.7	1.3	0.4	18	53	0.6	7.1	29	1.5	0.1	4.2	22	43	0.005
13	23	0.8	0.8	0.8	0.8	0.6	0.8	0.8	0.1	0.005	0.005	0.005	0.3	0.005	0.1	0.005	0.1	29	0.005	0.7	1.0	0.005	0.005	0.2	43	3.9	32	0.005	0.1	0.3	0.4	0.3
14	0.8	0.8	0.8	0.8	0.8	1.0	0.8	0.8	0.2	0.8	0.4	1.4	21	1.8	0.2	0.005	0.005	0.005	0.2	2.2	0.6	18	0.2	0.005	0.5	1.7	1.0	3.1	5.1	1.6	0.2	1.8
15	0.8	0.8	2.8	0.8	1.1	12	13	59	0.5	0.4	6.0	35	27	0.005	0.6	15	10	0.2	0.3	43	9.7	24	0.005	1.4	55	27	3.5	3.1	24	9.0	0.2	0.005
16	1.1	0.8	0.8	0.8	1.1	1.2	0.5	0.3	35	19	4.7	0.3	0.1	0.005	0.4	0.1	0.9	0.005	2.7	0.9	1.1	0.005	0.1	0.6								
17	0.8	0.8	0.8	0.8	27	26	10	9.7	0.1	0.3	0.4	0.5	11	14	16	0.2	4.9	0.4	17	0.8	2.9	1.7	0.9	0.005								
18	9.0	0.8	0.8	0.8	0.5	21	0.005	0.5	0.3	0.4	0.005	0.4	0.8	0.2	61	0.005	0.005	17	1.8	18	0.005	17	28	0.005								
19	0.8	0.8	0.7	0.2	0.9	148	6.0	0.005	59	2.8	1.2	0.8	0.7	0.005	3.5	5.2	0.3	0.1	2.1	0.1	0.1	0.1	0.005	0.005								
20	0.8	0.8	0.8	0.8	0.2	1.5	13	0.4	0.1	0.2	0.4	0.4	0.9	0.1	7.1	0.6	0.005	0.005	0.005	0.1	0.1	1.5	0.2	0.005								
21	1.9	0.8	0.8	0.8	0.4	6.7	46	53	0.005	41	23	27	0.005	0.005	1.3	22	0.4	61	0.6	0.2	0.6	0.005	0.005	0.005								
22	0.8	0.8	0.8	0.8	0.005	0.2	0.5	2.7	0.9	21	1.6	12	0.005	0.1	14	0.4	0.005	22	24	42	11	132	11	0.9								
23	0.8	0.8	0.8	0.8	0.1	0.1	0.3	0.1	5.5	3.7	0.4	0.7	0.1	0.1	0.2	0.005	0.005	1.3	1.0	0.7	0.1	0.3	0.005	0.005								
24	0.8	0.8	0.8	0.8	2.4	0.8	9.0	10	0.3	0.1	0.1	5.4	0.2	0.1	25	10	51	1.5	1.8	0.1	0.1	0.1	0.1	0.8								
25	0.8	0.8	0.8	0.8	37	0.005	0.005	0.005	0.1	2.3	38	13	43	18	20	0.3	4.8	9.0	2.1	0.3	42	42	7.2	0.5								

Figure 3-3. 2,4-D Surface Soil Concentrations (ppb): Measured and Estimated Values

	04	05	06	07	08	09	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26	27	28	29	30	31	32	33	34	35
06					372.5	372.5	372.5	372.5	372.5	231235	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5
07		372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	350	372.5	372.5	372.5	372.5	372.5	372.5
08		372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	17729	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5
09	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5
10	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5
11	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	189	372.5	372.5	372.5	372.5	372.5
12	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5
13	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	42	372.5	372.5	372.5	372.5	372.5	372.5	372.5
14	372.5	372.5	372.5	372.5	372.5	372.5	2.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	161	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5
15	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5
16	372.5	372.5	372.5	.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5								
17	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5								
18	372.5	86647	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5								
19	372.5	372.5	372.5	372.5	372.5	395	372.5	372.5	372.5	372.5	372.5	372.5	281330	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5								
20	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5								
21	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	80506	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5								
22	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	672	372.5	372.5								
23	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5								
24	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	53	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5								
25	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	294	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5	372.5								

Figure 3-4. 3,4,5-T Surface Soil Concentrations (ppb): Measured and Estimated Values

	04	05	06	07	08	09	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26	27	28	29	30	31	32	33	34	35
04					956	956	956	956	956	237155	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956
05		956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	1040	956	956	956	956	956	956	956
06		956	956	956	956	956	956	956	956	956	53291	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956
07	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956
08	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956
09	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956
10	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956
11	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	518	956	956	956	956	956
12	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956
13	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	101	956	956	956	956	956	956	956
14	956	956	956	956	956	956	221	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	387	956	956	956	956	956	956	956	956	956
15	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956
16	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956								
17	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956								
18	956	152492	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956								
19	956	956	956	956	956	1087	956	956	956	956	956	956	180363	956	956	956	956	956	956	956	956	956	956	956								
20	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956								
21	956	956	956	956	956	956	956	956	956	956	956	956	956	56528	956	956	956	956	956	956	956	956	956	956								
22	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	872	956	956								
23	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956	956								
24	956	956	956	956	956	956	956	956	956	956	956	956	53	956	956	956	956	956	956	956	956	956	956	956								
25	956	956	956	956	956	956	956	956	956	956	956	956	956	606	956	956	956	956	956	956	956	956	956	956								

Figure 3-5. TCDD (Dioxin) Surface Soil Concentrations (ppb): Aggregated Cells

	04	06	08	10	12	14	16	18	20	22	24	26	28	30	32	34
06	0.80	0.80	0.80	0.80	2.25	0.88	4.65	9.50	15.75	1.75	12.20	6.95	9.45	3.70	15.85	0.20
08	0.80	3.00	0.75	0.80	93.75	44.50	3.93	2.70	2.45	0.03	4.83	0.95	2.50	11.05	7.00	18.20
10	0.80	19.85	0.80	0.65	8.03	25.95	2.03	18.53	3.25	0.78	0.85	8.20	13.08	2.40	9.85	1.88
12	15.15	0.80	0.75	0.65	0.28	0.55	0.08	0.03	8.68	1.18	4.85	13.45	20.75	8.40	6.65	10.85
14	0.80	1.30	3.73	18.40	0.48	5.70	12.45	3.95	2.55	11.43	13.08	0.40	21.05	2.68	9.93	0.55
16	0.88	0.80	13.83	5.13	13.60	1.48	6.28	4.18	1.55	5.35	1.43	0.40				
18	2.85	0.63	42.60	1.13	15.62	0.60	0.43	17.43	4.35	5.50	4.30	7.00				
20	1.08	0.80	2.25	28.10	10.33	12.70	0.25	7.75	15.35	0.23	0.55	0.05				
22	0.80	0.80	0.10	0.90	7.78	3.68	0.08	3.65	5.83	16.93	35.85	2.98				
24	0.80	0.80	10.05	4.75	0.70	14.13	15.33	13.83	16.58	1.20	21.05	2.15				

Figure 3-6. 2,4-D Surface Soil Concentrations (ppb): Aggregated Cells

	04	06	08	10	12	14	16	18	20	22	24	26	28	30	32	34
06	372.50	372.50	372.50	372.50	58088.13	372.50	372.50	372.50	372.50	372.50	372.50	372.50	366.88	372.50	372.50	372.50
08	372.50	372.50	372.50	372.50	372.50	4711.63	372.50	372.50	372.50	372.50	372.50	372.50	372.50	372.50	372.50	372.50
10	372.50	372.50	372.50	372.50	372.50	372.50	372.50	372.50	372.50	372.50	372.50	372.50	372.50	326.63	372.50	372.50
12	372.50	372.50	372.50	372.50	372.50	372.50	372.50	372.50	372.50	372.50	372.50	372.50	289.88	372.50	372.50	372.50
14	372.50	372.50	372.50	280.00	372.50	372.50	372.50	372.50	372.50	372.50	372.50	319.63	372.50	372.50	372.50	372.50
16	372.50	372.50	372.50	372.50	372.50	372.50	372.50	372.50	372.50	372.50	372.50	372.50				
18	21941.13	372.50	378.13	372.50	372.50	372.50	70611.88	372.50	372.50	372.50	372.50	372.50				
20	372.50	372.50	372.50	372.50	372.50	372.50	20405.88	372.50	372.50	372.50	372.50	372.50				
22	372.50	372.50	372.50	372.50	372.50	372.50	372.50	372.50	372.50	372.50	497.38	372.50				
24	372.50	372.50	372.50	372.50	372.50	372.50	273.00	372.50	372.50	372.50	372.50	372.50				

Figure 3-7. 2,4,5-T Surface Soil Cencentrations (ppb): Aggregated Cells

	04	06	08	10	12	14	16	18	20	22	24	26	28	30	32	34
06	956.00	956.00	956.00	956.00	60005.75	956.00	956.00	956.00	956.00	956.00	956.00	956.00	977.00	956.00	956.00	956.00
08	956.00	956.00	956.00	956.00	956.00	14039.75	956.00	956.00	956.00	956.00	956.00	956.00	956.00	956.00	956.00	956.00
10	956.00	956.00	956.00	956.00	956.00	956.00	956.00	956.00	956.00	956.00	956.00	956.00	956.00	846.50	956.00	956.00
12	956.00	956.00	956.00	956.00	956.00	956.00	956.00	956.00	956.00	956.00	956.00	956.00	742.25	956.00	956.00	956.00
14	956.00	956.00	956.00	772.25	956.00	956.00	956.00	956.00	956.00	956.00	956.00	813.75	956.00	956.00	956.00	956.00
16	956.00	956.00	956.00	956.00	956.00	956.00	956.00	956.00	956.00	956.00	956.00	956.00				
18	38840.00	956.00	989.75	956.00	956.00	956.00	45807.75	956.00	956.00	956.00	956.00	956.00				
20	956.00	956.00	956.00	956.00	956.00	956.00	14849.00	956.00	956.00	956.00	956.00	956.00				
22	956.00	956.00	956.00	956.00	956.00	956.00	956.00	956.00	956.00	956.00	935.00	956.00				
24	956.00	956.00	956.00	956.00	956.00	956.00	662.75	956.00	956.00	956.00	956.00	956.00				

where, D_i = molecular diffusivity of the vapor-phase compound in air (i.e., for TCDD, $D_i = 4.7 \times 10^{-2}$ cm^2/s; for 2,4-D, $D_i = 6.2 \times 10^{-2}$ cm^2/s; for 2,4,5-T, $D_i = 5.91 \times 10^{-2}$ cm^2/s)[3];

ε = porosity of soil (i.e., approximately 0.35 for the calcium carbonate soil at JI);

K_{as} = air/soil partition coefficient (mg/cm^3 air)/(mg/g soil)[4];

C_{so} = initial compound concentration in soil (g/g); and

T = exposure duration (i.e., 25 years in units of seconds[5]).

Using the parameters defined above, alpha (α) is expressed as follows:

$$\alpha = \frac{(D_i)\ (\varepsilon^{\frac{4}{3}})}{[\varepsilon + \frac{\rho_s(1 - \varepsilon)}{K_{as}}]} \qquad (3\text{-}2)$$

where, ρ_s = soil density (i.e., approximately 1.76 g/cm^3 for the calcium carbonate soil at JI).

To convert the area emission rate to a point source emission rate for this modeling analysis, each compound emission rate was divided by the area of the four plots equal to 1,600 ft^2 (1.5×10^6cm^2). Receptors were placed along the border, or fenceline, of the storage area at intervals of 20 feet (104 receptors total) which

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[3] D_i values for 2,4-D and 2,4,5-T were obtained from R. Coutant, Batelle Memorial Institute Columbus, based on formulas cited in Fuller, Schettler, and Giddings. 1966. Title. Ind. Eng. Chem. 58:19, and A. Bondi. 1968. Physical properties of molecular crystals, liquids, and glasses. Wiley and Sons. New York.

[4] $K_{as} = 41\ H_c / K_d$. For TCDD $H_c = 5.00 \times 10^{-5}$, $K_d = 3.65 \times 10^6$. For 2,4-D, $H_c = 1.02 \times 10^{-8}$, $K_d = 1.66 \times 10^1$. For 2,4,5-T, $H_c = 8.68 \times 10^{-9}$, $K_d = 1.22 \times 10^1$.

[5] It was assumed that the HO site would exist for no longer than twenty-five years before remediation is conducted; thus, the longest potential exposure duration would be twenty-five years.

correspond to the original study area sampling grid. These receptors enclosed the entire perimeter of the storage area.

The ISC model was used to calculate a 1- and 8-hour average ambient air concentration (g/m^3) at each receptor for each wind direction. In order to convert this value to an annual average concentration, each model-predicted concentration was multiplied by a conversion factor of 9.925 (EPA, 1990). It should be noted that there is an unknown measure of uncertainty associated with this factor, as applied in this analysis, because it was developed using data for elevated point source releases.

Tables B-1 through B-9 (see Appendix B) present results of the atmospheric dispersion modeling, i.e., g of vapor-phase compound (TCDD, 2,4-D, and 2,4,5,-T) per m^3 of ambient air at the fenceline receptor sites. The receptor sites are presented as x,y coordinates which have their origin (i.e., x = 0 and y = 0) at the lower, southwest corner of the HO site (Figure 2.1) and proceed clockwise around the fenceline of the entire site. Air concentrations were estimated as 1-hr and 8-hr averages, as well as annual averages.

Given the fenceline receptor concentrations, the next step involved determination of the plausible "zone of impact" or zone where potential human inhalation exposure might occur. As discussed in Section 3.1.4, human activities near the HO site are assumed to be almost entirely confined to short durations (approximately 1 hour) at locations south and west of the HO site. Cross-referencing these locations with a wind rose for JI (Figure 2.1), reveals that, on an annual basis, the prevailing frequency of winds (i.e., greater than 95 percent) are from the 40 to 110 degree wind direction sector; therefore, it is plausible that inhalation exposure may occur for individuals working at downwind locations (e.g., burn pit, fire training area). Thus, to estimate reasonable maximum exposure (EPA 1989b), the maximum 1-hr average concentration occurring along the prevailing, downwind side of the HO site's fenceline (i.e., the north, south, and west sides) was selected. This ambient air

concentration was considered to represent the reasonable maximum ambient air concentration which an individual may breath while in the zone of impact.

TABLE 3.2

Maximum 1-hour average vapor-phase concentrations (mg/m^3) of TCDD, 2,4-D, and 2,4,5-T estimated to occur for the TMEI and AMEI at the perimeter of the HO site.

Chemical	TMEI	AMEI
TCDD	1.01×10^{-8}	1.01×10^{-8}
2,4-D	1.81×10^{-4}	6.79×10^{-5}
2,4,5-T	2.00×10^{-4}	1.27×10^{-4}

Table 3.2 presents the selected maximum 1-hr average ambient air concentrations (mg/m^3) of vapor-phase TCDD, 2,4-D, and 2,4,5-T estimated to occur for TMEI and the AMEI at the fenceline of the site and in the zone of impact. These ambient air concentrations were then used in the following equation to estimate the daily absorbed dose (EPA 1988b, 1989b, 1989c):

$$AbsorbedDose\ (mg/kg\text{-}day) = \frac{CA \times IR \times ET \times EF \times ED \times ABS}{BW \times AT} \qquad (3\text{-}3)$$

where,

CA = contaminant ambient air concentration (mg/m^3);

IR = inhalation rate (i.e., 2.1 m^3/hour for an average adult engaged in a moderate activity level);

ET = exposure time (i.e., 1 hour/day for persons engaged in activities in the zone of impact);

EF = exposure frequency (i.e., 250 days/year);

ED = exposure duration [i.e., 0.68 years (250 days/365 days)];

ABS = absorption fraction (0.75, EPA, 1988b);

BW = body weight (i.e., 70 kg for an average adult); and

AT = averaging time [i.e., 250 days for noncarcinogenic effects; 25,550 days (365 days/year x 70 years) for carcinogenic effects].

Table 3.3 presents the estimated lifetime average daily absorbed dose for TCDD, and average daily dose for TCDD, 2,4-D, and 2,4,5-T resulting from vapor-phase inhalation exposure.

TABLE 3.3

Estimated lifetime average daily absorbed dose (LADD) and average daily absorbed doses (ADD) expressed as mg/kg/day for TCDD, 2,4-D, and 2,4,5-T resulting from vapor-phase inhalation exposure to the TMEI and the AMEI.

Chemical	TMEI		AMEI	
	LADD	ADD	LADD	ADD
TCDD	5.6×10^{-11}	2.3×10^{-10}	5.6×10^{-11}	2.3×10^{-11}
2,4-D		4.1×10^{-6}		1.5×10^{-6}
2,4,5-T		4.5×10^{-6}		2.9×10^{-6}

3.3.3 Inhalation of Contaminated Soil

Inhalation of contaminated airborne particles emitted from the HO site represents a plausible exposure pathway resulting from potent[illegible] future uses as discussed in Section 3.2.3. Although data collected by Helsel et al. (1987) suggested that virtually no particle-associated TCDD exposure (via inhalation) was occurring

as the result of airborne particulate originating from the undisturbed site, disturbances to the site may result in dispersion of contaminated soil particles and thus, present the potential for inhalation exposure to downwind receptors. The following Sections (3.3.3.1 through 3.3.3.3) present the methods for estimating potential particle-associated inhalation exposures resulting from persons being engaged in activities in the zone of impact during two distinct future-use activities at the HO site: (1) excavation of contaminated soil; and (2) construction of a cement cover over the existing site. To estimate the compound concentration in soil which is disturbed during site activities associated with these figure-use scenarios, first, the median value of the subsurface concentrations for each verticle profile (see Section 2.0) was calculated, and then the grand median of these median values was calculated. Thus, the grand median values for TCDD, 2,4-D, and 2,4,5-T were 0.42, 25.8, and 93 ppb, respectively.

3.3.3.1 Wind Erosion

Wind erosion was evaluated with respect to its contribution to airborne particulates emitted from the site as the result of disturbances to contaminated soil during either excavation or construction of a cement cover. The flux of dust particles less than 10 gm in diameter from surfaces with an "unlimited reservoir"[6] of erodible particles can be estimated as follows (Cowherd *et al.* 1985; EPA, 1988b):

$$E = 0.036\ (1-V)\ \frac{(U_m)}{(U_t)}\ F(x) \qquad \textbf{(3-4)}$$

where,

E = total dust flux of <10 gm diameter particles ($g/m^2/hr$);

[6] Soil surfaces that are exposed to the wind, uncrusted, and which consist of finely divided particles (EPA, 1988b).

V = fraction of vegetation (i.e., assumed to be 0.20 on the HO site at JI);

U_m = mean annual wind speed (i.e., 6.75 m/s at JI);

U_t = threshold wind speed (i.e., assumed to be 8.2 m/s, see EPA 1988c); and

F(x) = model function (i.e., 1.5, based on a comparison of $(U_t/U_m)0.886$ versus F(x) as presented in Cowherd *et al.*, 1985).

Then, the total dust flux (E), is converted to an emission rate using the following relationship (Cowherd *et al.* 1985):

$$Q = (C_s)\ (E)\ (A)\ \frac{(1\ hr)}{(3{,}600\ seconds)} \qquad \textbf{(3-5)}$$

where,

Q = compound emission rate (ng/second);

C_s = compound concentration in soil (ng/g); and

A = surface area of the site disturbed per day (i.e., 86 m^2/day during excavation and 173 m^2/day during cement cover construction).

Thus, the particle-associated compound emission rate estimates (g/hr) for wind erosion from either excavation or construction of cement cover were calculated as follows:

Chemical	Emission Rate (g/hr)	
	Excavation	Cement Cover
TCDD	1.4×10^{-11}	2.9×10^{-11}
2,4-D	8.9×10^{-10}	1.8×10^{-9}
2,4,5-T	3.2×10^{-9}	6.5×10^{-9}

3.3.3.2 Vehicular Traffic

The emissions of soil-associated TCDD, 2,4-D, and 2,4,5-T which may result from vehicular traffic on the HO site for either future use scenario (i.e., excavation or cement cover construction) can be estimated from an emission factor. The derivation of this factor is contained in EPA (1985, 1988b), and takes the form of:

$$E_v = k[1.7(\frac{s}{12})] \ (\frac{S}{48}) \ (\frac{W}{2.7})^{0.7} \ (\frac{w}{4})^{0.5} \ (365-\frac{p}{365})$$

where,

	E_v	=	Emission factor (kg/vehicle kilometer traveled);
	k	=	Particle size multiplier (i.e., 0.36 to 0.45, EPA, 1983);
	s	=	Silt content of road surface material (i.e., 0.2, EPA, 1988b);
	S	=	Mean vehicle speed (i.e., 8 km/hr);
	W	=	Mean vehicle weight (i.e., approximately 45 Mg for front-end loader and dump truck used during excavation and 35 Mg for loaded cement truck used during construction of cement cover);
	w	=	Mean number of wheels (i.e., 20 during excavation using at least two vehicles, and 10 during cement covering using one vehicle); and
	p	=	Number of days with at least 0.254 mm (0.01 in) of precipitation per year (i.e., 162 at JI).

This emission factor is provided in units of kg particulate emitted per vehicle kilometer traveled (kg/VKT). The particle size multiplier (k) varies with aerodynamic particle size range. Of particular interest is the respirable particle size range, because particles in this range may be inhaled and retained in the respiratory tract allowing for possible desorption from the surface of the particles and subsequent absorption through the capillaries (Paustenbach *et al.*, 1986). For unpaved surfaces,

U.S. EPA (1983) has estimated k to be 0.45 for aerodynamic particle diameters less than 10 μm; whereas, for soil loading and unloading operations and maintenance of outdoor storage piles, k is estimated to be 0.36 for aerodynamic particle diameters less than 10 μm.

Thus, the compound emission rate estimates (g/hr) associated with particle emissions from vehicular traffic involved in excavation or construction of cement cover were calculated as follows:

Chemical	Emission Rate (g/hr)	
	Excavation	Cement Cover
TCDD	8.0×10^{-9}	6.0×10^{-9}
2,4-D	4.9×10^{-7}	3.6×10^{-7}
2,4,5-T	1.8×10^{-6}	1.3×10^{-6}

3.3.3.3 Loading and Unloading Operations

The emission of particle-associated TCDD, 2,4-D, and 2,4,5-T during excavation activities (e.g., loading and unloading of contaminated soil) can be estimated from an emission factor described in Cowherd *et al.* (1985) and EPA (1988b):

$$E = k\,(0.0018)\,\left[\frac{(\frac{S}{5})\,(\frac{U}{5})\,(\frac{H}{5})}{(\frac{M}{2})^2\,(\frac{Y}{6})^{0.33}}\right] \qquad (3\text{-}7)$$

where,

E = Emission factor (lb emission per ton of soil moved);

k = Particle size multiplier (i.e., 0.36, EPA 1988b);

s = Silt content (i.e., 0.2, EPA 1988b);

U = Mean wind speed (i.e., 15.1 mph at JI);

H = Drop height (i.e., 12 ft);

M = Soil moisture content (i.e., 0.09, Crockett et al., 1986); and

Y = Dumping device capacity (i.e., 4 yd^3).

The particle-associated emission rate values were estimated as follows:

Chemical	Emission Rate (g/hr)
	Excavation
TCDD	5.6×10^{-8}
2,4-D	3.4×10^{-6}
2,4,5-T	1.2×10^{-5}

3.3.3.4 Estimated Emission Rates of Compounds Associated with Soil During Excavation or Construction of a Cement Cover and Estimated Inhalation Exposure and Absorbed Doses for Exposed Individuals

The estimated emission rates of particle-associated TCDD, 2,4-D, and 2,4,5-T for wind erosion and vehicular traffic associated with excavation and cement cover construction, and loading and unloading operations associated with excavation, were summed to provide an estimate of the total emission expected per hour, which results from these activities. Thus, during construction of the cement cover, it was assumed that both wind erosion and vehicular traffic would contribute to particle-associated compound emissions; therefore, their respective compound-specific emission rates were summed. Loading and unloading operations were not considered to be necessary for construction of the cement cover. However, for the excavation scenario,

compound-specific emission rates associated with particle emissions due to wind erosion, vehicular traffic and loading and unloading operations were summed.

The total emission rates for both excavation and construction of a cement cover were then used as input rates for the atmospheric dispersion model described in Section 3.3.2. The emissions of the particle-associated compounds were assumed to originate from the center of the soil sampling grid for purposes of dispersion modeling. The modeling provided estimates of 1-hr and 8-hr concentrations (g/m^3) of the particle-associated compounds across the same receptor perimeter as described above (Section 3.3.2) for the vapor-phase ambient air concentrations estimates.

The duration of exposure was assumed to be 243 days (0.67 years) for excavation and 120 days (0.33 years) for construction of a cement cover. Tables B-10 through B-15 and B-16 through B-20 (see Appendix B) present the estimated particle-associated ambient air concentrations (g/m^3) of TCDD, 2,4-D, and 2,4,5-T resulting from excavation and cement cover construction, respectively.

Absorbed inhalation doses were then calculated for both the TMEI and AMEI using equation 3 described above. The pulmonary absorption of the particle-associated compounds was assumed to be 3.0 percent for all three compounds; whereas, vapor-phase pulmonary absorption was assumed to be 75 percent for all three compounds (EPA, 1988b). In addition to particle-associated compound inhalation, it was assumed that vapor-phase inhalation could also occur simultaneously; thus, the vapor-phase absorbed doses estimated in Section 3.3.2 (see Table 3.2) were summed with the particle-associated absorbed doses to yield a total absorbed dose for both the excavation and cement cover construction scenarios. These total absorbed dose estimates are provided in Table 3.4. It is important to note that the TMEI and AMEI were selected based on the highest possible concentration resulting from the sum of both the vapor-phase concentration and the particle-associated concentration for each receptor location.

TABLE 3.4

Estimated Lifetime Average Daily Dose (LADD) and Average Daily Dose (ADD) expressed as mg/kg/day for TCDD, 2,4-D, and 2,4.5-T resulting from vapor-phase and particle-associated inhalation exposure to the TMEI and the AMEI during excavation and construction of a cement cover.

EXCAVATION

Chemical	TMEI		AMEI	
	LADD	ADD	LADD	ADD
TCDD	1.5×10^{-12}	1.6×10^{-10}	1.5×10^{-12}	1.6×10^{-10}
2,4-D	----	2.7×10^{-6}	----	1.2×10^{-6}
2,4,5-T	----	3.0×10^{-6}	----	1.9×10^{-6}

CEMENT COVER CONSTRUCTION

Chemical	TMEI		AMEI	
	LADD	ADD	LADD	ADD
TCDD	3.5×10^{-13}	7.5×10^{-11}	3.5×10^{-13}	7.5×10^{-11}
2,4-D	----	1.3×10^{-6}	----	5.0×10^{-7}
2,4,5-T	----	1.5×10^{-6}	----	9.4×10^{-7}

3.3.4 Ingestion of Contaminated Fish

A review of Table 2.1 shows that there is TCDD fish contamination in certain areas. The contamination appears to be restricted to the area adjacent to the former HO storage site, which is off-limits to fishing. Walsh III (1984) states that many coral reef fishes are strongly site-attached, and therefore move about only in relatively small areas. However, he points out that other coral reef fish can undergo

extensive daily movements. These large movements are usually restricted to adults. Randall (1961) studied the Convict Tang and noted that adults could move up to 300 yards in several hours. Walsh studied these movements in several Hawaiian fish species that are also present on JA. Table 2.1 indicates that these authors have identified the following species of fish as potentially having large daily movements:

Achilles Tang
Bluelined Surgeonfish
Bullethead Parrotfish
Convict Tang
Goldring Surgeonfish
Parrotfish
Spectacled Parrotfish
Threadfin Butterflyfish

Some of these fish species have been found to have TCDD contamination. If they migrate into the fishing areas near the former HO storage site, (Zones 5 and 10, Figure 3.1), then there is a potential for JI inhabitants to consume contaminated fish. For the fish that showed positive TCDD values, the migratory fish species had the lowest values. These values may be low because these fish may not spend all of their time in the contaminated area. It is not possible to quantify this potential exposure because the fishermen's catches have not been sampled. The potential for exposure may be low, but sampling of the fishermen's catches should be performed to confirm this. Sampling at the west wharf has revealed no contaminated fish, and this may be an indication of the low probability of catching a contaminated fish.

3.4 Uncertainties Associated with the Assessment of Exposure

There are many input values that must be selected along the path to developing a quantitative estimate of potential exposure. They involve making assumptions about the chemicals, the environment in which they are located, and the potential for human contact with them. In addition, input values, whether selected

by assumption or by existing empirical evidence, are all associated with some individual variability to a lesser or greater degree. In the aggregate, the use of assumptions and the variability underlying input values both create an element of uncertainty that is important to keep in mind when considering quantitative estimates of exposure and risk. Where the uncertainties are large, bounding them with statistical measures and sensitivity analyses can place quantitative limits on their range. This procedure was considered to be beyond the scope of this investigation because the risk assessment is screening-level and missing a lot of needed information. Instead, a qualitative description of the uncertainties is presented below.

Future use scenarios for HO site. The two future use scenarios were chosen to represent situations where site disruption was either minimal (concrete cover without remediation) or maximal (excavation of contaminated soil). As such, these are hypothetical scenarios that may not necessarily reflect the actual future use. This in itself creates an elements of uncertainty about the true risks at the site. Further, it is expected that paving this site would not occur without some form of prior treatment to stabilize the contaminated soil.

Assumptions in calculating exposure to chemicals at the HO site. There are two classes of assumptions that were necessary to have made in the estimation of exposure: those associated with human receptors and those associated with the calculation of emission factors. The *human receptor assumptions* include use of the TMEI or AMEI (the AMEI is more realistic), body weight, inhalation rate, and pulmonary deposition rate. It is important to recognize that under typical conditions, EPA recommends calculation of risk for the TMEI. However, at the HO site, locations that would normally produce a TMEI are inaccessible, making the AMEI a more viable alternative for prediction of exposure and risk. The *emission factor assumptions* associated with the excavation and paving scenarios include construction vehicle weight, number of wheels, duration of excavation scenario, duration of cement

covering scenario, physical parameters of soil (moisture content, density, pH, carbon content), threshold wind velocity, diffusion coefficients (computer estimates) and air-soil partition coefficients, concentrations of chemicals in soil (missing values, invalid values, unknown spatial distribution of 2,4-D and 2,4,5-T on surface and in vertical profiles), and QA issues. The first three are assumed to be of low variability; the rest are assumed to be of higher variability. In addition, the levels of particle-association inhalation exposure prior to the soil sampling study conducted by Crockett et al. (1986) are unknown. During this period, i.e., 1972 to 1986 (the period when Agent Orange storage began until the first soil sampling study was conducted) it was assumed that the average inhalation exposure levels estimated to occur over the lifetime exposure period (i.e., 25 years), which were based on the 1986 soil sampling study (Crockett et al. 1986), were representative of inhalation exposures levels occurring prior to 1986.

In addition, there are several variables *unaccounted for in this analysis*. These include:

- Transience of the potentially exposed population (transience implies that duration is variable);
- Differences in exposure between males and females;
- Other chemicals of concern at the site (e.g., other isomers of dioxin);
- Other chemicals on the Island (e.g., solvents, radiation, combustion products);

- Prior or concurrent occupational or environmental exposures to TCDD, 2,4-D, or 2,4,5-T, or other substances affecting the same target organs from the HO site or other sources:

Dedrumming operation	TCDD, 2,4-D, 2,4,5-T
Smoking	PIC (especially PAHs)[7]
Fire training area	TCDD and other PIC
JACADS stack plumes	TCDD, TCDFs[8], and other PIC
Fish consumption	Potential TCDD contamination
Launch area	Plutonium and progeny

  and other occupational hazards on JI involving in particular solvents or metals;

- Atmospheric transformation and soil photodegradation of TCDD, 2,4-D, and 2,4,5-T;

- Confounding exposure presented by accidental release of CW from JACADS; and

- Groundwater contamination and its relation to exposure of marine biota.

Uncertainty in dispersion modeling. The uncertainty in model predictions is a function of (1) "inherent" uncertainty; (2) uncertainties in model input variables; and (3) model physics errors. The inherent uncertainty arises from the random nature of the turbulent flow in which the plume is embedded (i.e., its variation from

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[7] PIC = Products of incomplete combustion; for example, polynuclear aromatic hydrocarbons (PAHs).

[8] TCDFs = Tetrachlorinated dibenzo furans.

one realization (i.e., observation) to the next) and the finite averaging time of the concentrations. Almost without exception, existing air quality models predict the ensemble-averaged concentration field (i.e., the mean concentration at any location over a large number of realizations of the same experiment). Overall, based on comparisons of model predictions to observations, the deviation between the predicted ensemble-average and an individual realization is large (i.e., of the order of the prediction).

For the horizontal scale of distance for this application, the principal cause of inherent uncertainty is three-dimensional boundary layer turbulence. This category of turbulence arises in ideal, homogeneous terrain and is caused by the stochastic nature of turbulence in the boundary layer; it is dominant over distances of less than approximately 20 km.

Model input variables that introduce uncertainty to the concentration estimate include (but may not be limited to) wind speed, wind direction, temperature, and emission rate. For this analysis, conservative meteorological parameters (in terms of plume dispersion) were used in the modeling; therefore, in terms of a peak model-predicted impact, the uncertainty introduced by the prescribed meteorological data should be small compared to the uncertainty introduced by the estimate of emissions for the emission area. The uncertainty in the emission estimates may be on the order of several magnitudes. Because the model-predicted impact is directly proportional to the emission rate, the uncertainty in the impacts may also be on the order of several magnitudes. Uncertainty contributed by errors in the representation of atmospheric physical processes in the model may also be large; however, quantification of this uncertainty for a particular model is a complicated process.

4.0 Toxicity Assessment

This section provides a review of the toxicological properties of TCDD, 2,4-D, and 2,4,5-T. These chemicals, which are present at the HO site, have been identified in Section 2.0 as having the potential for exposure in humans. The toxicity assessment of these chemicals examines the weight-of-evidence available regarding their ability to cause adverse health effects in exposed individuals. This evaluation also includes an estimation of the relationship between the extent of exposure to these compounds and the likelihood and severity of adverse effects.

4.1 Toxicological Profile for 2,3,7,8-Tetrachlorodibenzo-*p*-dioxin (TCDD)

4.1.1 Chemical Characteristics

2,3,7,8-Tetrachlorodibenzo-p-dioxin (TCDD) is one of 75 compounds that are referred to as dioxins. TCDD is a man-made chemical with no known natural sources. It is not intentionally manufactured except for research purposes. This chemical is produced as a byproduct in the manufacture and/or use of herbicides containing 2,4,5-trichlorophenoxy acids; 2,4,5-trichlorophenol in wood preservatives;

hexachlorophene in germicides; and pulp and paper plants. TCDD can also be produced during incineration of municipal or certain industrial wastes; transformer/capacitor fires involving chlorinated benzenes and biphenyls; and the burning of wood in the presence of chlorine. A summary of the physical-chemical properties of TCDD can be found in Table 3.1. Much of the toxicological information in this review was extracted from three key documents, definitive reviews in their own rights: ATSDR (1989), IARC (1977), and IARC (1986). Primary citations acknowledged in these documents were also used as citations in this review.

4.1.2 Pharmacokinetics

4.1.2.1 Absorption

There are no data on the absorption of TCDD via inhalation. For oral and dermal absorption, the vehicle used to administer the compound has a great influence on its absorption. Lipophilic vehicles enhance the absorption of this chemical, while soil, fly ash, and activated carbon greatly reduce its bioavailability. One human study (Poiger and Schlatter, 1986), showed that >87% of the dose was absorbed after ingestion of the compound in a corn oil vehicle. Animal studies have shown a 50 to 80% absorption in a lipophilic vehicle when given by gavage (Nolan et al., 1979; Olson et al., 1980; Piper et al., 1973), and a 50 to 60% absorption when administered in the diet (Fries and Marrow, 1975). McConnell et al. (1984) and Lucier et al. (1986), investigated the difference in TCDD gastric absorption when two different vehicles were used, corn oil and soil. The soil vehicle was discovered to reduce the bioavailability of TCDD by 50%. Paustenbach et al. (1986) reviewed several papers on the oral bioavailability of TCDD from soil. The reviewed papers reported bioavailabilities ranging from 0.5% to 85%. The authors stated that several factors could influence the oral bioavailability of TCDD from soil, these include: bolus size of dose; method for calculating bioavailability; and organic content of the soil. These authors concluded that the upper estimate for the oral bioavailability of TCDD in soil

would be 30%. Dermal absorption of TCDD is also greatly influenced by the dosing vehicle. When applied on rat skin with methanol (Poiger and Schlatter, 1980), TCDD was 40% absorbed, whereas with an acetone-carbon disulfide mixture it was 77% absorbed (Driver et al., 1990). When bound to soil, Driver et al. (1990) showed that TCDD after 24 hours was less than 1% absorbed.

4.1.2.2 Distribution

There are no data on the distribution of TCDD following inhalation. In a human study Poiger and Schlatter (1986) discovered that approximately 90% of the absorbed dose was sequestered in the fat after an oral dose of TCDD in corn oil. Rats and mice preferentially sequestered TCDD in the liver and then adipose, whereas in guinea pigs this trend was reversed this (EPA, 1985). In studies with mice, Gasiewicz et al. (1983a,b) and Birnbaum et al. (1986), demonstrated that inducible mouse strains sequestered more TCDD in their livers than non-inducible strains. Weber and Birnbaum (1985) and Krowke (1986), demonstrated that TCDD crosses the mouse placenta and 75% of the total fetal body burden is located in the liver. Nau et al. (1986), further revealed that the mouse pup was also exposed via the mother's milk.

4.1.2.3 Metabolism

The only metabolic data available are either from in vitro studies or oral animal studies. Poiger et al. (1982) analyzed the bile of dogs to determine the possible metabolites of TCDD. They found five phenolic compounds: 1,3,7,8-tetrachloro-2-methoxydibenzo-p-dioxin; 2,7,8-trichloro-3-methoxydibenzo-p-dioxin; trichloro-dimethoxydibenzo-p-dioxins; tetrachloro-dimethoxy diphenylether; and 1,2-dichloro-4,5-dimethoxybenzene. Isolated rat hepatocytes were studied by Sawahata et al. (1982), and they identified 1-hydroxy-2,3,7,8-tetra-chlorodibenzo-p-dioxin and 8-hydroxy-2,3,7-trichlorodibenzo-p-dioxin as the metabolites in this study. Mason and

Safe (1986a,b) demonstrated that these metabolites had less biological activity than TCDD. Several authors have studied the differences in TCDD metabolism between species to attempt to explain the wide difference in species sensitivity to TCDD (Olson and Wroblewski, 1985; Poiger and Schlatter, 1985; and Wroblewski and Olson 1985). Pretreatment with TCDD in dogs (*in vivo*) and rats (*in vitro*) resulted in a greatly increased rate of metabolism of a subsequent dose, 100 and 320% respectively, but no increase was noted with the same experiment in guinea pigs. These results may partly explain why guinea pigs are 25 times more sensitive than rats to the effects of TCDD.

4.1.2.4 Excretion

Excretion data following inhalation or dermal exposure to TCDD are not available. Poiger and Schlatter (1986), investigated the elimination of TCDD in a human volunteer. They discovered that 11% of the dose was eliminated in the feces in the first three days, but during days 7 through 125 only 3.5% of the dose was eliminated. This led to a half-life calculation for this study of 2,120 days. In contrast, laboratory animals have a much shorter half-life: guinea pigs, 22 to 30 days; rats, 17 to 31 days; and mice, 11 to 24 days. Rats and guinea pigs eliminated 91 to 99% in the feces, mice, 54 to 72%; and 59% was eliminated in the hamster feces (EPA 1985).

4.1.2 Noncancer Toxicity

The noncancer toxicity of TCDD following inhalation exposure is not available. The summary of the oral R_fD values can be found in Table 4.6. This compound has shown to be lethal at very low concentrations in all laboratory animals tested, but there is a wide range of LD50 values between species. Oral administration of TCDD in lipophilic solvents has resulted in the following LD_{50} values: 0.6 to 2.1 ug/kg in guinea pigs (Schwetz et al., 1973), 20 to 60 ug/kg in rats, 100 to 600 ug/kg in mice,

and 1,000 to 5,000 ug/kg in hamsters (EPA, 1985; McConnell, 1985). One dermal study by Schwetz et al. (1973), with TCDD in acetone on New Zealand white rabbits produced an LD_{50} of 142 to 531 ug/kg. Death in all of the above experiments was delayed, and was not observed until 5 to 40 days after TCDD administration.

Toxicity data for humans are difficult to interpret because no one has been exposed to pure TCDD. Humans have been exposed to TCDD only as a minor contaminant in mixtures of other chlorinated aromatics or phenolics, and in the case of pesticide formulations various solvents are also present. It is not always known if the effects seen are from TCDD or from the other chemicals present, or a combination of the chemicals in the mixture. Many of the toxic effects described below have been reported in humans, but no confirmation linking these effects solely to TCDD can be made because of the confounding factors, including adequate exposure data, involved in the epidemiological studies. Therefore, the only data available on pure TCDD exposure are in laboratory animals.

TCDD is a potent inducer of chloracne in both humans and animals. Greig (1984) and Puhvel et al. (1982), produced chloracne lesions in hairless mice by both oral administration and dermal application respectively of TCDD. A threshold dose is not available since both investigations used only one dose level. Both children and adults developed chloracne lesions after the Seveso accident, with a greater prevalence showing in children. The higher frequency in children may have due to their greater activity patterns with soil (Suskind, 1985; Taylor, 1979).

In laboratory animals, a characteristic effect seen with both acute and long term studies, and usually seen with lethal doses, is the wasting syndrome. Weight loss and/or severely limited weight gain can begin to appear within 24 hours after TCDD administration, and continues until death 15 to 30 days after exposure (EPA, 1985; Peterson et al., 1984). Lu et al. (1986) showed that this syndrome is not entirely caused by a loss of appetite. Guinea pigs' weights when fed were stable until

a few days before death, but at that time weight loss began and was observed until death. This study did show that most of the observed weight loss can be attributed to appetite loss, but not all of it. This syndrome has not been reported in humans (ATSDR 1989).

Rats and mice are sensitive to the hepatic effects of TCDD, but guinea pigs and monkeys do not appear to be quite as sensitive (EPA, 1985). Types of lesions include necrosis, proliferative changes, cellular membrane alterations, bile duct proliferation, altered lipid metabolism, and excess amounts of porphyrin. Turner and Collins (1983), noted mild changes in guinea pig livers following a single gavage dose ranging from 0.1 to 20 ug/kg. Changes included hypertrophy, steatosis, focal necrosis, and hyalin-like bodies. A LOAEL of 0.001 ug/kg/day for liver effects in rats and mice was determined by EPA (1985) after a review of the literature (Kociba et al., 1979; NTP, 1982b).

Rats, mice, and guinea pigs are all very sensitive to the immunotoxic effects of TCDD. Reviews by EPA (1985, 1988a) and Knutsen (1984) revealed minimum effective oral doses of 1 ug/kg/week for mice, 5 ug/kg/week for rats, and 0.04 ug/kg/week for guinea pigs. Strain differences in mice have been observed to segregate with the Ah locus response (Dencker et al., 1985). C57B1/6 mouse thymus cultures, which are Ah-responsive, proved to be very sensitive to the immunotoxic effects of TCDD, whereas DBA/2J mouse thymus cultures, which are not Ah responsive, showed no effects. Luster et al. (1982) demonstrated that Fischer rat pups and B6C3F1 mice pups were sensitive to the immunotoxic effects of TCDD following in utero and postnatal lactation exposure.

The teratogenic effects of TCDD have been extensively studied, and rats and mice have been shown to be sensitive to these effects. Cleft palate and hydronephrotic kidney were the effects seen in mice after an oral dose of only 1 µg/kg (Courtney, 1976; Moore et al., 1973; Neubert and Dillmann, 1972; Smith et al., 1976).

Gavage administration of 0.125 to 0.25 µg/kg to rats during organogenesis produced hemorrhage of internal organs and subcutaneous edema (Sparschu et al., 1971a,b; Khera and Ruddick, 1973). As with hepatic effects, the teratogenic effects were only seen in Ah-responsive C57B1/6J mice (Poland and Glover, 1980; Dencker and Pratt, 1981).

The fetotoxicity of TCDD has been seen in rats, mice, and monkeys, with the monkey being the most sensitive species. In studies reviewed by EPA (1985, 1988a), fetal death and vaginal bleeding was seen at oral doses between 2 and 9 ug/kg/day. Murray et al. (1979), conducted a three-generation dietary study with Sprague-Dawley rats. Doses of 0.01 and 0.1 ug/kg/day resulted in decreased litter size, decreased fetal survival, and decreased neonatal survival. A decrease in fertility was observed at the 0.1 ug/kg/day dose. McNulty (1984, 1985) reported a high incidence of spontaneous abortions in Rhesus monkeys at total oral doses of 0.2 and 1.0 µg/kg on days 20 to 40 of gestation. Khera and Ruddick (1973) reported a decrease in male Wistar rat reproductive performance after oral administration of TCDD.

Several epidemiological studies have been conducted to determine if there is a correlation between TCDD exposure and birth defects (Aldred, 1978; Bisanti et al., 1980; Bonaccorsi et al., 1978; Department of Health, New Zealand, 1980; McQueen et al., 1977; Nelson et al., 1979; Reggiani, 1980; Smith et al., 1982; and Thomas, 1980). All of theses studies failed to demonstrate a correlation between birth defects and possible exposure to TCDD. Erickson et al. (1984) conducted a case control study of Vietnam veterans to determine if the offspring of these men had an increased risk of birth defects. This study showed that when all types of defects were combined there was not an increase in risk to birth defects among Vietnam veterans. They did find an increase in certain types of defects which include spina bifida, cleft palate, and certain congenital tumors. The authors noted that these increased risks may have been due to several factors including, unmeasured confounding factors, chance, or some other experience in Vietnam. The increased risks were low.

4.1.3 Carcinogenicity

The genotoxicity data for this compound have yielded conflicting results. Many of the studies have given negative results, while the positive tests showed weak response. The results of these studies can be found in Tables 4.1 and 4.2. The insolubility and high toxicity of TCDD has caused problems in some of these test systems. More testing must be done to resolve the conflicting data obtained so far (ATSDR, 1989).

As with noncancer effects, there are no inhalation carcinogenic data available. Several studies have shown that TCDD is carcinogenic by oral administration, the key studies being NTP (1982b) and Kociba et al. (1978a,b). A summary of the results of these studies can be found in Tables 4.3 and 4.4. In contrast to the oral studies, dermal studies have demonstrated limited or conflicting results. In the NTP (1982a) study, female Swiss mice had an increase incidence of fibrosarcomas in the integumentary system (but not the males). Berry et al. (1978) and Slaga and Nesnow (1985), reported no promotion or weak promoting activity in CD-1 mice and Sencar mice, respectively, when TCDD was applied to the skin. On the other hand, Poland et al. (1982) showed promotion in CD-1 mice, and that promotion was affected by genetic differences in the mice. These inconsistencies have not been resolved yet.

Human data on the genotoxicity and carcinogenicity of TCDD are inconclusive because of the previously described confounding factors involved in the epidemiological studies. There appears to be limited evidence that there may be an increased risk of soft-tissue sarcomas and lymphomas from exposure to phenoxyacetic acid herbicides and/or chlorophenols contaminated with TCDD (EPA, 1985). A recent retrospective cohort study (Fingerhut et al., 1991) found an increased risk of soft-tissue sarcomas in workers exposed for over one year to chemicals contaminated with TCDD, with a latency period of over 20 years. Limitations of this study were the

TABLE 4.1 Genotoxicity of 2,3,7,8-TCDD *in vitro*

End point	Species (test system)	Results	References
Gene mutation	*Salmonella typhimurrum* (reverse mutation)	—/—	McCann, 1978 Gilbert et al., 1980 Geiger and Neal, 1981 Mortelmans et al., 1984
	S. typhimurium (reverse mutation)	Not tested/+	Hussain et al., 1972 Seiler, 1973
	Escherichia coli (reverse mutation)	Not tested/+	Hussain et al., 1972
	Saccharomyces cerevisiae (reversion)	+/—	Bronzetti et al., 1983
	L5178Y mouse lymphoma cells (forward mutation)	Not tested/+, and not tested/—	Rogers et al., 1982
Cytogenetic	*S. cerevisiae* (gene conversion)	+/—	Bronzetti et al., 1983
	S. cerevisiae (host mediated)	+/NA[a]	Bronzetti et al., 1983
	Chinese hamster cells (sister chromatid exchange)	Not tested/—	Toth et al., 1984
Cell transformation	Baby hamster kidney cells - BHK	Not tested/+	Hay, 1982
	C3H/10T1/2 cells	Not tested/—	Abernathy et al., 1985

[a] Not available.

Source: ATSDR, 1989.

TABLE 4.2 Genotoxicity of 2,3,7,8-TCDD *in vivo*

End point	Species (test system)	Results	References
Gene mutation	*Drosophila* (sex-linked recessive lethal)	—	Zimmering et al., 1985
Cytogenetic	*Drosophila* (sister chromatid exchange)	—	Zeiger, 1983
	Drosophila (structural aberration)	—	Zeiger, 1983
	Rat (sister chromatid exchange)	—	Lundgren et al., 1986
	Rat - marrow cells (structural aberration)	—	Green and Moreland, 1975
	Rats - marrow cells (structural aberration)	+	Green et al., 1977
	Mouse - marrow cells (structural aberration)	+	Loprieno et al., 1982
	Mouse - marrow cells (sister chromatid exchange)	—	Meyne et al., 1985
	Mouse - marrow cells (structural aberration)	—	Meyne et al., 1985
	Mouse - marrow cells (micronucleus)	—	Meyne et al., 1985

Source: ATSDR, 1989.

TABLE 4.3 Summary of the oral carcinogenicity bioassay of Kociba et al. (1978 a,b)

Animal	Sex	Drug tested	Tumor type	Incidence
Sprague-Dawley rats	M	Control	Squamous cell carcinoma of the tongue, adenoma of the adrenal cortex, and squamous cell carcinoma of tyhe hard palate	0/85
		0.001	Squamous cell carcinoma of the tongue	1/50
		0.01	Squamous cell carcinoma of the tongue	1/50
			Squamous cell carcinoma of the adrenal cortex	2/50
		0.1	Squamous cell carcinoma of the tongue	3/50
			Adenoma of the adrenal cortex	5/50
			Squamous cell carcinoma of the hard palate	4/50
	F	Control	Hepatocellular carcinoma	1/86
		0.001	Hepatocellular carcinoma	0/50
		0.01	Hepatocellular carcinoma	2/50
			Squamous cell carcinoma of the hard palate	1/50
		0.1	Hepatocellular carcinoma	11/49
			Squamous cell carcinoma of the hard palate	4/49
			Squamous cell carcinoma of the lung	7/49

Source: ATSDR, 1989.

TABLE 4.4 Other Oral Studies Supporting the Conclusion that 2,3,7,8-TCDD is an Animal Carcinogen

Method of Exposure	Animal	Sex/ number	Doses tested	Tumor type	References
Diet	Sprague-Dawley rats	M/10	0.01, 0.005, 0.05, 0.5, 1.0, or 5 ppb	Increase in total tumor incidence	Van Miller et al., 1977a,b
Gavage	Osborne-Mendel rats	M/50	0.01, 0.05, or 0.5 µg/kg/ week	Follicular-cell adenomas and carcinomas of the liver	NTP, 1982b
	Osborne-Mendel rats	F/50	0.01, 0.05, or 0.5 µg/kg/ week	Neoplastic nodules and hepatocellular carcinomas of the liver	NTP, 1982b
	B6C3Fl mice	M/50	0.01, 0.05, or 0.5 µg/kg/ week	Hepatocellular carcinomas	NTP, 1982b
	B6C3Fl mice	F/50	0.01, 0.05, or 0.5 µg/kg/ week	Hepatocellular carcinoma and follicular-cell adenomas of the thyroid	NTP, 1982b
	Swiss mice	M/44	0.007, 0.7, or 7.0 µg/kg/ week	Hepatomas and hepatocellular carcinomas	Toth et al., 1979

Source: ATSDR, 1989.

limited number of cases, and the misclassification of soft-tissue sarcomas. A summary of the unit cancer risk values can be found in Table 4.9.

4.2 Toxicological Profile for 2,4-Dichlorophenoxyacetic Acid (2,4-D)

The purpose of this toxicological profile is to describe the known behavior of 2,4-D by using the most current and related information available. It is important to note that the n-butyl esters of 2,4-dichlorophenoxyacetic acid can hydrolyzed in biological and aquatic systems. Therefore, the behavior of the pure acid and their salts are pertinent and will be discussed in the following paragraphs along with studies on the esters when they are available (USAF, 1974).

4.2.1 Chemical Characteristics

2,4-Dichlorophenoxyacetic acid (2,4-D[9]) is a man-made chemical with no known natural sources. The chemical is produced by the interaction of 2,4-dichlorophenol, with the sodium salt of monochloroacetic acid, typically followed by an acid treatment to convert the 2,4-D salt to an acid (Sittig, 1980, 1986).

2,4-D is a systemic herbicide used for the control of broad leaf weeds in cereal crops, sugar cane, turf, pastures and other non-cropland (Weed Science Society of America, 1974). It is also used to control the ripening of bananas and citrus fruits (WHO, 1975). An estimated 27 million kg of 2,4-D acid equivalent, in the form of esters and salts, were used in the US in 1975 (IARC, 1977). 2,4-D was used as a jungle defoliant during the Vietnam War in the mid-1960's, where it was a component of "Agent Orange" (a 50:50 mixture of the n-butyl esters of 2,4-D and 2,4,5-trichlorophenoxyacetic acid). About 40 million liters of "Agent Orange" were sprayed

[9] 2,4-D refers to the acid derivative unless otherwise stated.

in South Vietnam between 1965-1971 (Committee on the Effects of Herbicides in Vietnam, 1974).

Various physical and chemical properties of 2,4-D are discussed in Section 4.5.

4.2.2 Pharmacokinetics

The differences in toxic effects caused by the various salts, amines and esters of 2,4-D can be explained on a pharmacometric basis. The concentrations of chemicals at the receptor sites in an organism depends on the absorption and distribution rates in relation to rates of metabolism and excretion. The rate of absorption in animals or plants is based on the route of entry and rate of membrane transport. Specific membrane transport rates depend upon the characteristics of the membrane in relation to the size, shape, polarity and lipid solubility of the particular molecule considered (USAF, 1974).

4.2.2.1 Absorption

The most common route of exposure to herbicides in mammals is via ingestion, although exposure via inhalation and cutaneous routes is possible. The literature indicates that gastric absorption of 2,4-D, its amines and alkali salts occur readily as would be predicted from the Henderson-Hasselbalch relationships (USAF, 1974). The gastro-intestinal absorption of 2,4-D esters may be incomplete (Erne, 1966 as cited in USAF, 1974).

Frank et al. (1985) calculated that a maximum of 4.5% of the amount of 2,4-D deposited on the bare skin of a person directly sprayed with 2,4-D was absorbed. Among those occupationally exposed, dermal exposure appears to be the most important route of absorption.

4.2.2.2 Distribution

After oral administration of 2,4-D to sheep and cattle, analyses of muscle, fat, liver and kidney showed the presence of 2,4-dichlorophenol (Clark et al., 1975 as cited in USDIFWS, 1978). There are no data concerning distribution after other relevant routes of administration.

4.2.2.3 Metabolism

Most studies indicate that 2,4-D is rapidly eliminated via the kidneys by active tubular secretion into the urine. Cattle and rabbits excrete 2,4-D in their urine mostly unchanged (USAF, 1974). Erne (1966) as cited in USAF (1974), found that 2,4-D had a half-life from three to twelve hours and that urinary excretion was the primary route of elimination in the rat, rabbit, calf and chicken. Berndt and Koschier (1973), as cited in USAF (1974), concluded that renal tubular transport by the organic anion mechanism may account for the relatively rapid disappearance of 2,4-D and that might account for 2,4-D's low toxicity.

4.2.2.4 Excretion

In a study on the kinetics of 2,4-D, five male volunteers were administered a dose of 5 mg/kg bw. Absorption was nearly complete, as indicated by the recovery of 88-100% of the dose in the urine within 144 h. Approximately 80% of the 2,4-D was excreted unchanged in the urine. The additional 20% was excreted as an acid-labile conjugate (Sauerhoff et al., 1977a). Extensive and rapid gastrointestinal absorption of 2,4-D was also observed by Kohli et al. (1974b).

Maximum concentrations of 2,4-D were detected in urine three days after dermal exposure (Feldman and Maibach, 1974).

4.2.3 Toxicity

Toxicity data for humans are difficult to obtain because people are rarely exposed to pure 2,4-D. Most occupational exposure studies are difficult to evaluate because of the combined exposures of many workers to more than one herbicide or greater than one derivative of a single herbicide.

4.2.3.1 Noncancer Toxicity

Most of the data derived from acute toxicity studies indicate that 2,4-D has low toxicity. In the rat, the single dose LD_{50} is 620 mg/kg for the butyl ester derivative of 2,4-D and 100 mg/kg for the dog in the 2,4-D acid derivative (Rowe et al., 1954; Edson et al., 1964 as cited in USAF, 1974).

Groups of 3 male and 3 female beagle dogs were fed 10, 50, 100, or 500 mg/kg of diet 2,4-D for 2 years, beginning at 6-8 months of age. Twenty-eight dogs survived the 2 year period and were clinically normal. No adverse effects related to 2,4-D were observed (Hansen et al., 1971).

Results of teratological studies are variable; teratogenic effects are observed with doses close to maternal toxicity. In a study by Bjorklund and Erne (1966), Sprague-Dawley rats were given 1000 mg/l 2,4-D (50 mg/kg) in the drinking water during pregnancy and for an additional 10 months after that, and 2,4-D was administered to the second generation for up to 2-years. Pregnancy and parturition were normal, the litter size was not significantly reduced, and no malformations were noted in the young. Except for retarded growth and increased mortality in the second generation, no clinical or morphological changes were seen.

In a three-generation study, Osborne-Mendel rats were orally administered 100 or 500 µg/kg (4 µg/kg or 20 µg/kg) of diet 2,4-D. No adverse effects were observed.

Diets containing 1500 µg/kg (60 µg/kg) 2,4-D significantly reduced the percentage of pups surviving to weaning and their weights (Hansen et al., 1971).

No significant increases in embryonic effects were noted when 2,4-D was orally administered to hamsters at doses up to 100 mg/kg on days 6-10 of gestation (Collins and Williams, 1971).

An Oral Reference Dose (Oral R_fD), of 0.01 mg/kg/day has been set by EPA (IRIS, 1991). This is based on data from Dow Chemical Co. (1983). Hematologic, hepatic and renal toxicity were demonstrated in Fisher 344 rats during a subchronic feeding. 2,4-D was fed to the rats for 91 days at doses calculated to be 0, 1, 5, 15, or 45 mg/kg/day. There were a total of 200 animals in the study. Criteria examined to determine toxicity were survival, daily examination for clinical symptomology, weekly change in body weights and clinical, gross and histopathologic alterations. The results demonstrated statistically significant reductions in mean hemoglobin (both sexes), mean hematocrit and red blood cell levels (both sexes), and mean reticulocyte levels (males only) at the 5 mg/kg/day dose or higher after 7 weeks. There were also significant reductions in liver enzymes LDH, SGOT, SGPT, and alkaline phosphatase at week 14 in animals treated at the 15 mg/kg/day or higher doses. Kidney weights (absolute and relative) showed significant increases in all animals at the 15 mg/kg/day dose or higher at the end of the experimental protocol. Histopathologic examinations correlated well with kidney organ weight changes showing cortical and subcortical pathology. The dose used to derive the R_fD_o was 1 mg/kg/day (IRIS, 1991). The R_fD_o was set at 0.01 mg/kg bw/day by using a total uncertainty factor of 100 to account for uncertainty in the interspecies and interhuman variability in the toxicity of 2,4-D in regard to these specific data (IRIS, 1991). Because the analysis of the 90-day and a follow up 1-year interim study, results suggest that the NOAEL would also be relevant for the full 2-year duration. Inclusion of the subchronic-to-chronic uncertainty factor is not warranted (IRIS, 1991). The EPA has medium confidence (tending towards high) in this oral R_fD (IRIS, 1991). Confidence

in the study is medium because of a reasonable number of animals were used of both sex, the four doses were given, and a generous number of parameters were examined (IRIS, 1991). Confidence in the data base is medium because several studies support both the observation of critical toxic effects and the levels at which they occur (IRIS, 1991).

Critical noncarcinogenic toxicity values for 2,4-D are discussed in Section 4.5.

4.2.3.2 Carcinogenicity

Osborne-Mendel rats were orally administered 5, 25, 125, 625, or 1250 mg/kg (0.2, 1.0, 5.0, 25.0, or 50 mg/kg) 2,4-D for 2 years. A significant increase in tumors was seen only in the highest dose group, but tumors were randomly distributed and were typical of those found in aging rats of this strain (Hansen et al., 1971). Because of the limitations of this study (including the small number of animals used) no evaluation of carcinogenicity could be made based on the available studies (IARC, 1987).

IARC (1987 and 1977) state that the evidence for carcinogenicity in animals is inadequate for 2,4-D.

4.2.3.3 Additional Data

The genotoxicity data for 2,4-D have yielded fairly inconsistent results overall. Many *in vitro* studies have given positive results in absence of metabolic activation, but a few negative results have been noted. The results of these studies can be found in Tables 4.5 (*in vitro* data) and 4.6 (*in vivo* data).

TABLE 4.5 Genotoxicity of 2,4-D *in vitro*

End point	Species (test system)	Results	References
Gene Mutation	*Salmonella typhimurium* (reverse mutation)	-/-[a]	Nishimura et al., 1982 Mortelmans et al., 1984
	S. typhimurium (reverse mutation)	0[b]/-	Anderson and Styles, 1978
	S. typhimurium (reverse mutation)	-/0	Zetterberg et al., 1977 Anderson et al., 1972
	Saccharomyces cerevisiae (reverse mutation)	+/0	Zetterberg, 1978
Cytogenetic	*S. cerevisiae* (gene conversion)	+/0	Zetterberg et al., 1977
	S. cerevisiae (gene conversion)	(+)[c]/0	Siebert and Lemperle, 1974
	Chinese hamster cells (sister chromatid exchange)	-/-	Linnainmaa, 1984
	Human lymphocytes (sister chromatic exchange)	+/0	Korte and Jalal, 1982
	Human lymphocytes (chromosomal aberration)	+/0	Pilinskaya, 1974 Mustonen et al., 1986

[a] In presence of metabolic activation/absence of metabolic activation
[b] Not tested
[c] Weakly positive

Source: IARC, 1987.

TABLE 4.6 Genotoxicity of 2,4-D *in vivo*

End point	Species (test system)	Results	References
Gene mutation	*Drosophila melanogaster* (sex-linked recessive lethal)	-	Vogel and Chandley, 1974 Zimmering et al., 1985
	Drosophila melanogaster (sex-linked recessive lethal)	+	Magnusson et al., 1977
Cytogenetic	*Drosophila melanogaster* (somatic mutation/ recombination)	+	Rasmuson and Svahlin, 1978
	Drosophila melanogaster (aneuploidy)	-	Ramel and Magnusson, 1979 Magnusson et al., 1977 Woodruff et al., 1983
	Mouse (micronucleus test)	-	Seiler, 1978 Jenssen and Renberg, 1976
	Mouse (dominant lethal test)	-	Epstein et al., 1972
	Human lymphocytes (sister chromatid exchange)	-	Linnainmaa, 1983
	Human lymphocytes (sister chromatid exchange)	(+)[a]	Crossen et al., 1978
	Human lymphocytes (chromosome aberration)	-	Mustonen et al., 1986
	Human lymphocytes (chromosome aberration)	(-)[b]	Hoegstedt et al., 1980

[a] Weakly positive
[b] Weakly negative

Source: IARC, 1987.

4.3 Toxicological Profile for 2,4,5-Trichlorophenoxyacetic Acid (2,4,5-T)

The purpose of this toxicological profile is to describe the known behavior of 2,4,5-T by using the most current and related information available. It is important to note that the n-butyl esters of 2,4,5-trichlorophenoxyacetic acid can be hydrolyzed in biological and aquatic systems. Therefore, the behavior of the pure acid and their salts are pertinent and will be discussed along with studies on the esters when they are available (USDAF, 1974).

4.3.1 Chemical Characteristics

2,4,5-Trichlorophenoxyacetic acid (2,4,5-T[10]) is a man-made chemical with no known natural sources. The chemical is currently produced by the reaction of 2,4,5-trichlorophenol with the sodium salt of monochloroacetic acid, typically followed by an acid treatment to convert the 2,4,5-T salt to an acid (Sittig, 1980).

2,4,5-T was used as a jungle defoliant during the Vietnam War in the mid-1960s, where it was a component of "Agent Orange" (a 50:50 mixture of the n-butyl esters of 2,4,5-T and 2,4-dichlorophenoxyacetic acid). About 40 million liters of "Agent Orange" were sprayed in South Vietnam between 1965-1971 (Committee on the Effects of Herbicides in Vietnam, 1974).

Various physical and chemical properties of 2,4,5-T are discussed in Section 4.5.

[10] 2,4,5-T refers to the acid derivative unless otherwise stated.

4.3.2 Pharmacokinetics

The differences in toxic effects caused by the various salts, amines and esters of 2,4,5-T can be explained on a pharmacometric level. The concentrations of chemicals at the receptor sites in an organism depends upon the absorption and distribution rates in relation to rates of metabolism and excretion. The rate of absorption in animals or plants is dependent on the route of entry and the rate of membrane transport. Specific membrane transport rates depend upon the characteristics of the membrane in relation to the size, shape, polarity and lipid solubility of the particular molecule considered (USDAF, 1974).

4.3.2.1 Absorption

The most common route of exposure to herbicides in mammals is via ingestion, although exposure via inhalation and cutaneous routes is possible. The literature indicates that gastric absorption of 2,4,5-T and its amines and alkali salts occur readily as would be predicted from the Henderson-Hasselbalch relationships (USDAF, 1974). There is no information in the available literature about the absorption of 2,4,5-T via the skin or inhalation.

4.3.2.2 Distribution

There was no available information on the distribution of 2,4,5-T.

4.3.2.3 Metabolism and Excretion

Most studies indicate that animals rapidly eliminate 2,4,5-T via the kidney by active tubular secretion into the urine. Cattle and rabbits excrete 2,4,5-T in their urine mostly unchanged (USDAF, 1974). Erne (1966), as cited in USDAF (1974), found that 2,4,5-T had a half-life from three to twelve hours and that urinary

excretion was the primary route of elimination in the rat, rabbit, calf and chicken. Berndt and Koschier (1973), as cited in USDAF (1974), concluded that renal tubular transport by the organic anion mechanism may account for the relatively rapid disappearance of 2,4,5-T, which may account for 2,4,5-T's low toxicity.

[1-^{14}C]2,4,5-T was administered to pregnant and non-pregnant rats by stomach tube in a study by Fang et al. (1973), as cited in USDIFWS (1978). The rate of elimination for both groups was the same. Ninety to 95% of the label was eliminated in the form of unchanged 2,4,5-T in the urine. In addition, two non-polar and one water soluble metabolite were observed. Acid hydrolysis of the water soluble metabolite produced 2,4,5-T suggests potential ester formation.

Studies in humans confirm the results observed in animals. Gerring et al. (1973) orally administered 2,4,5-T directly or in milk in 5 human male volunteers. An average of 88% of the dose was excreted in the urine within 96 hours of administration, and renal clearance was 180 to 260 ml/min. The ingested 2,4,5-T was eliminated unchanged into the urine (USDAF, 1974). There was no free trichlorophenol detected in the urine. Clearance from the plasma and excretion both followed first-order kinetics with a half-life of 23 hours. Fecal excretion was <1% of the dose (Gerring et al., 1973).

In a similar study, 2,4,5-T was administered orally at 2, 3, or 5 mg/kg bw. Maximum plasma concentrations were detected 7 to 24 hours after administration. Following the 5 mg/kg bw dose, the half-life averaged 19 hours. For all of the doses examined, an average of 63 to 79% of the dose was recovered in the urine within 96 h of administration (Kohli et al., 1974a).

4.3.3 Toxicity

Toxicity data for humans are difficult to obtain because people are rarely exposed to pure 2,4,5-T. In the majority of cases, the available data do not distinguish between the possible effects of exposure to 2,4,5-T and those of exposure to associated chemicals or more toxic contaminants such as TCDD.

4.3.3.1 Noncancer Toxicity

Most of the data derived form acute toxicity studies indicate that 2,4,5-T has low toxicity. In the mice, the single dose LD_{50} was 940 mg/kg for the butyl ester derivative for 2,4,5-T and 500 mg/kg in the rat for the 2,4,5-T acid derivative (Rowe and Hymas, 1954 as cited in USDAF, 1974).

Dogs fed 2,4,5-T 5 times a week for 90 days at a dosage level of 2, 5, or 10 mg/kg bw exhibited no adverse effects. Daily doses of 20 mg/kg bw resulted in deaths 11-75 days after the first dosing (Drill and Hiratzka, 1953).

Results of teratology studies in animals are variable. 2,4,5-T (containing less than 0.02 mg/kg TCDD) orally administered on days 6-15 of gestation was embryotoxic to NMRI mice. The frequency of cleft palate was significantly increased when doses of greater than 20 mg/kg bw were administered. Reductions in fetal weight were found with doses of 10-15 mg/kg bw, but there was no increase in embryolethality over controls. Cleft palates were produced following a single oral dose of 150-300 mg/kg bw. 2,4,5-T butyl ester was found to have similar embryopathic effects as 2,4,5-T following administration on days 6-15 of gestation (Neubert and Dillmann, 1972).

To the contrary, 2,4,5-T (containing 0.5 mg/kg TCDD) was neither teratogenic or fetotoxic when orally administered to CD rats at doses ranging from 1-80 mg/kg

bw (Courtney and Moore, 1971), or in Sprague-Dawley rats at doses ranging from 1-24 mg/kg bw (Emerson et al., 1971) on days 6-15 of gestation. The butyl ester of 2,4,5-T had no effect when orally dosed at 50 or 150 mg/kg bw in Wistar rats, but 2,4,5-T (containing less than 0.5 mg/kg) did induce skeletal anomalies following single daily doses of 100-150 mg/kg bw on days 6-15 of gestation (Khera and McKinley, 1972).

Sjoden and Soderberg (1977), reported that prenatal exposure to 2,4,5-T may lead to behavioral abnormalities and changes in thyroid activity as well as brain serotonin levels in the progeny. Crampton and Rogers (1983) reported that prenatal exposure to 2,4,5-T has long-term effects on behavior in rats. After exposure to a single dose of 2,4,5-T (6 mg/kg) on day 8 of gestation, abnormalities were observed in tests for novelty responses.

An oral Reference Dose (oral R_fD), of 0.01 mg/kg/day has been set by EPA (IRIS, 1991). This is based on data from two well conducted studies (Kociba et al., 1979; Smith et al., 1981). Kociba et al. (1979) maintained Sprague-Dawley rats (50/sex) on diets supplying 0, 3, 10, or 30 mg 2,4,5-T/kg bw/day for 2 years. Toxicological endpoints measured were body weight, food consumption, tumorigenicity, hematology, urinalysis, serum chemistry, and histopathology. No effects were seen at 3 mg/kg/day. An increase in urinary excretion of coproporphyrin (at 4 months only) was reported for males at 10 and 30 mg/kg/day and for females at the 30 mg/kg bw dose level. A mild dose-related increase in the incidence of mineralized deposits in the renal pelvis was reported for females after 2 years. Smith et al. (1981) conducted a three generation reproduction study. Rats were fed levels of 2,4,5-T corresponding to 0, 3, 10, or 30 mg 2,4,5-T/kg bw/day. No effects were observed at the lower doses. Reduced neonatal survival was observed at both higher doses. The dose used to derive the R_fD_o was 3 mg/kg/day (IRIS, 1991). The R_fD_o was set at 0.01 mg/kg bw/day by using a total uncertainty factor of 300 to account for uncertainty in the extrapolation of dose levels from laboratory animals to humans

(10), uncertainty in the threshold for sensitive humans (10), and uncertainty because of deficiencies in the chronic toxicity data base (3) (IRIS, 1991). The EPA has medium confidence (tending towards high) in this oral R_fD (IRIS, 1991). There is high confidence in the studies used to determine the R_fD_o because of the completeness of the studies and the data base is supportive of the magnitude of the reproductive effect. The relative weakness of the chronic toxicity data base precludes a higher overall confidence level (IRIS, 1991).

Critical noncarcinogenic toxicity values for 2,4,5-T are discussed in Section 4.5.

4.3.3.2 Carcinogenicity

2,4,5-T has been tested in mice by oral administration. In a study by Mutanyi-Kjovacs et al. (1976), 20 male and 19 female 6-week old inbred XVBII/G mice were given 100 mg/l (5 mg/kg) 2,4,5-T (containing less than 0.05 mg/kg chlorinated dibenzodioxins) in the drinking water for 2 months. Subsequently, 2,4,5-T was fed orally at a concentration of 80 mg/kg (3.2 mg/kg) of diet for lifespan. No significant increase was noted in the incidence of tumors. In a similar study by the same authors, C3HF mice were treated in the same manner. The treated female mice showed a significant increase in the total number of tumors. Although an increased incidence of tumors at various sites were observed in this study, no evaluation of carcinogenicity of 2,4,5-T could be made because of the limitations of this study (small number of animals used) (IARC, 1987).

IARC (1987, 1977) state that the evidence for carcinogenicity in animals is inadequate for 2,4,5-T.

4.3.3.3 Additional Data

The genotoxicity data suggest that 2,4,5-T is not likely to effect genetic material. Most studies have given negative results, while the positive studies had only weak responses. The results of these studies can be found in Tables 4.7 (*in vitro* data) and 4.8 (*in vivo* data).

TABLE 4.7 Genotoxicity of 2,4,5-T *in vitro*

End point	Species (test system)	Results	References
Gene mutation	*Salmonella typhimurium* (reverse mutation)	-/-[a]	Herbold et al., 1982 Nishimura et al., 1982 Mortelmans et al., 1984
	Salmonella typhimurium (reverse mutation)	0[b]/-	Anderson and Styles, 1978
	Salmonella typhimurium (reverse mutation)	-/0	Andersen et al., 1972
	Saccharomyces cerevisiae (reverse mutation)	+/0	Zetterberg, 1978

[a] In presence of metabolic activation/absence of metabolic activation
[b] Not tested

Source: IARC, 1987.

TABLE 4.8 Genotoxicity of 2,4,5-T *in vivo*

End point	Species (test system)	Results	References
Gene mutation	*Drosophila melanogaster* (sex-linked recessive lethal)	+	Majumdar and Golia, 1974
	Drosophila melanogaster (sex-linked recessive lethal)	(+)[a]	Magnusson et al., 1977
	Drosophila melanogaster (sex-linked recessive lethal)	-	Zimmering et al., 1985
Cytogenetic	*Drosophila melanogaster* (somatic mutation/recombination)	-	Rasmuson and Svahlin, 1978
	Drosophila melanogaster (aneuploidy)	-	Ramel and Magnusson, 1979 Magnusson et al., 1977
	Mouse (micronucleus test)	-	Jenssen and Renberg, 1976
	Mouse (dominant lethal test)	-	Buselmaier et al., 1972
	Rat (dominant lethal test)	-	Herbold et al., 1982
	Human lymphocytes (sister chromatid exchange)	(+)	Crossen et al., 1978

[a] Weakly positive

Source: IARC, 1987.

4.4 Toxicity Profile for the Mixtures of 2,4,5-Trichlorophenoxyacetic Acid (2,4,5-T), 2,4-Dichlorophenoxyacetic Acid (2,4-D), and 2,3,7,8-Tetrachlorodibenzo-p-Dioxin (TCDD) as Chlorophenoxy Herbicides

4.4.1 Toxicity

Toxicity data for humans are difficult to obtain because people are rarely exposed to pure 2,4,5-T, 2,4-D or TCDD. Most occupational exposure studies are difficult to evaluate because of the combined exposures of many workers to more than one herbicide or greater than one derivative of a single herbicide. In the majority of cases, the available data do not distinguish between the possible effects of exposure to 2,4,5-T or 2,4-D and the exposure to associated chemicals such as TCDD. Many studies involve the occupational exposure to the general category of chlorophenoxy herbicides.

4.4.2 Noncancer Toxicity

4.4.2.1 Chloracne

In a reaction incident with exposure to 2,4,5-T and its contaminant TCDD in 1949, workers were who were exposed were followed for 4 years. Directly after exposure, workers had complaints including chloracne and respiratory tract, liver and nervous system disorders. By 1953, liver and nervous system problems subsided, but chloracne still persisted in some cases (Suskind, 1985).

4.4.2.2 Reproduction and Prenatal Toxicity

Effects on reproduction and prenatal toxicity have been addressed in several studies in humans. A study in Arkansas, USA, divided the state into low, medium and high 2,4,5-T use areas on the basis of rice acreage. No significant differences in

rates of facial cleft were found among the different areas between 1943 and 1974 (Nelson et al., 1979). The USEPA investigated spontaneous abortion rates in areas of Oregon, USA, in relation to 2,4,5-T spray rates between 1972 and 1977. Significantly higher spontaneous abortion rates were noted in areas in which 2,4,5-T was used. IARC (1986) noted that some of the methods in the study were inadequate.

A study of the pregnancy outcomes of wives of professional herbicide (2,4,5-T) sprayers was conducted in New Zealand (Smith et al., 1981). There were a total of 1172 births among families in the exposed group (1969-1979 for spraying of 2,4,5-T; 1960-1979 for spraying of any pesticide) and 1122 births in a control group. Major congenital defects were reported in 2% (24) of births to applicator families and 1.6% (18) of births to the control group; the difference was not significant. Similar rates were observed for the two groups for stillbirths and miscarriages. In further analysis, the pregnancy outcomes associated with spraying of 2,4,5-T by the father in the same year or in the previous year of the birth were selected and compared to the control group. The relative risk for congenital defects in children of exposed fathers was 1.19 and for miscarriages 0.89 (Smith et al., 1982b). These results were not statistically significant.

4.4.3 Cancer

4.4.3.1 Case-Control Studies

4.4.3.1.1 Soft-Tissue Sarcomas

Hardell and Sandstrom (1979), conducted a case-control study of 52 male patients with soft-tissue sarcoma and 220 matched controls. A person was classified as being exposed if he had at least one full day of exposure more than 5 years before a tumor was diagnosed. Of the 52 cases, 13 cases were exposed to chlorophenoxy herbicides (12 had been exposed to 2,4,5-T or 2,4-D, and one to 4-chloro-2-methyl-

phenoxy acetic acid (MCPA) alone; combined exposure to 2,4,5-T and 2,4-D was reported in 9 cases). A significant association was observed (odds ratio = 5.3; 95% CI, 2.4 to 11.5) with prior exclusion of exposure cases to chlorophenol. Latency from first exposure was 10 to 20 years. The average duration of exposure was three to four months (range, 2 days to 49 months).

Eriksson et al. (1981) undertook a case-control study with 110 cases with soft-tissue sarcomas and 220 matched controls in an area of Sweden where MCPA and 2,4-D had been widely used in agriculture. A significant association was observed (odds ration = 8.5) for exposure to chlorophenoxy herbicides alone for more than 30 days (7 cases), and 5.7 for exposures of less than or equal to 30 days (7 cases). The odds ratio for exposure to chlorophenoxy herbicides other than 2,4,5-T was 4.2 (95% CI, 1.3 to 15.8).

An initial analysis of occupations recorded with the National New Zealand Center Registry between 1976 and 1980 did not find and excess of soft-tissue sarcoma cases in agricultural and forestry workers (Smith et al., 1982). After this preliminary analysis, nearly 90% of the cases (or next of kin) were interviewed regarding past occupations and actual exposure to chlorophenoxy herbicides. A significant association was observed (odds ratio = 1.6; 90% CI, 0.7 to 3.3) was calculated for those who had probably or definitely been exposed for more than one day greater than 5 years prior to the diagnosis of the tumor. None of the cases was of a professional applicator. The possibility of recall bias based on the previous study was noted.

In a study by Smith et al. (1984) 82 persons with soft-tissue sarcomas and 92 controls (with other types of cancers) were interviewed for a case-control study. For those potentially exposed to phenoxyherbicides for more than one day not in the 5 years prior to cancer diagnosis, no significant association was observed (odds ratio = 1.3; 90% CI, 0.6 to 2.5). In addition, no significant association was observed for

chlorophenol exposure (odds ratio = 1.5; 90% CI. 0.5 to 4.5). The authors concluded that further studies were needed to clarify whether human exposure to these chemicals increase the risk of soft-tissue sarcoma.

4.4.3.1.2 Malignant Lymphomas

A case-control study of 169 cases of malignant lymphoma was undertaken with 338 matched controls (Hardell et al., 1981). The study design, including determination of exposure, was similar to the Swedish soft-tissue sarcoma studies (see Hardell and Sandstorm, 1979). A significant association (odds ratio = 4.8; 95% CI, 2.9 to 8.1) was obtained for exposure to chlorophenoxy herbicides, excluding cases and controls exposed to chlorophenols. Stratifying by duration of exposure, the relative risk estimate was 4.3 for less than 90 days and 7.0 for 90 days or more exposure to chlorophenoxy herbicides. The majority of chlorophenoxy herbicide-exposed cases reported exposure to both 2,4,5-T and 2,4-D (25 cases), two reported exposure to 2,4,5-T, 2,4-D and MCPA, seven to 2,4-D alone and 5 to MCPA alone (Hardell, 1981a).

An analysis of reported occupations appearing on the New Zealand Cancer Registry indicated an excess of malignant lymphoma and multiple myeloma among men in agricultural occupations during 1977-1981. The main findings of a subsequent case-control study concerned 88 cases of malignant lymphoma (covering non-Hodgkin's lymphoma other than lymphosarcoma and reticulosarcoma), classified as ICD 202, and 352 matched controls. A subsequent study with 83 cases of ICD 202 suggested that exposure to chlorophenoxy herbicides was not associated, since the odds ratio of 1.3 (90% CI. 0.7 to 2.5) was obtained when controls were people with other cancers were used, and an odds ratio of 1.0 (90% CI. 0.5 to 2.1) when the controls were the general population (Pearce et al., 1986).

4.4.3.1.3 Nasal and Nasopharyngeal Cancer

Hardell et al. (1982), described an odds ratio of 2.1 (95% CI, 0.9 to 4.7) for exposure to chlorophenoxy herbicides.

4.5 Conclusion and Summary

There is limited evidence that occupational exposures to chlorophenoxy herbicides are carcinogenic to humans (IARC, 1986). Benchmark values for all relevant toxicological indicators, carcinogenic and noncarcinogenic, are presented in Tables 4.9 and 4.10, respectively.

TABLE 4.9
Critical Carcinogenic Toxicity Values for Indicator Chemicals
Herbicide Orange Storage Area
Johnston Island, Johnston Atoll

Chemical Name	Slope Factor (SF) $(mg/kg\text{-}day)^{-1}$	Weight of Evidence Classification	Type of Cancer	SF Basis/ SF Source
Oral Route				
2,3,7,8-Tetrachlorodibenzo-p-Dioxin[a]	1.56×10^5	B1[a]	Lung, liver, hard palate, nasal turbinates	Food/ATSDR (June 1989)
2,4-Dichlorophenoxy acetic acid[b] (n-butyl ester)	No data	No data	No data	No data
2,4,5-Trichlorophenoxy acetic acid[b] (n-butyl ester)	No data	No data	No data	No data
2,4,5-Trichlorophenoxy acetic acid[b] (Iso-octyl ester)	No data	No data	No data	No data
Inhalation Rate	No data	No data	No data	No data

[a] When associated with phenoxy herbicides and/or chlorophenols, B2 when considered alone.

TABLE 4.10
Critical Noncarcinogenic Toxicity Values for Indicator Chemicals
Herbicide Orange Storage Area
Johnston Island, Johnston Atoll

Chemical Name	Chronic R_fD (mg/kg-day)	Confidence Level[a]	Critical Effect	R_fD Basis/ R_fD Source	Uncertainty and Modifying Factors[b]
Oral Route					
2,3,7,8-Tetrachlorodibenzo-p-Dioxin	1×10^{-9}	No data	Primary: Fetal survival Secondary: Renal	No data/ ATSDR	UF=100 for A, L MF=10
2,4-Dichlorophenoxy acetic acid (n-butyl ester)	1×10^{-2}[c]	Medium	Primary: Renal Secondary: Hematologic, hepatic	Food/ IRIS	UF=100 for H, A MF=1
2,4,5-Trichlorophenoxy acetic acid (n-butyl ester)	1×10^{-2}[d]	Medium	Primary: Neonatal survival Secondary: Increased urinary coproporphyrin	Food/ IRIS	IF=300 for H, A, D MF=1
Inhalation Route	No data	No data	No data	No data	No data

[a] Confidence level from IRIS, either high, medium, or low.
[b] Uncertainty adjustments: H=variation in human sensitivity; A=animal to human extrapolation; and D=deficiencies in toxicity data.
[c] R_fD value for acid, n-butyl ester value not available.
[d] R_fD value for acid, n-butyl ester and iso-octyl ester values not available.

5.0 *Risk Characterization*

Characterization of risk is based on the results of the exposure assessment (as summarized in Table 3.12) and the benchmark toxicity values (presented in Table 4.10). The basic algorithm for calculation of risk for carcinogenicity is:

$$Risk = Lifetime\ Average\ Daily\ Dose\ (mg/kg/day) \times unit\ cancer\ risk\ (mg/kg/day) \quad (5\text{-}8)$$

and for systemic toxicity (as the hazard quotient) is:

$$Noncancer\ hazard\ quotient = \frac{Average\ Daily\ Dose\ (ADD)}{Reference\ Dose\ (R_fD)} \quad (5\text{-}9)$$

Among the chemicals of concern, TCDD is the only known carcinogen. The Unit Cancer Risk (UCR) on which risk was calculated is 1.56×10^5. TCDD, 2,4-D, and 2,4,5-T are all systemic toxicants. It is important to note that, in the case of systemic

toxicity, hazard quotients are *not* additive for different chemicals where their respective R_fD's are based on different target organs. R_fD's and their bases are listed in Table 4.10 as the primary effect on which each chemical's R_fD is based. For TCDD the primary effect is fetotoxicity; for 2,4-D it is renal toxicity; and for 2,4,5-T it is reduced neonatal survival. As a result, hazard quotients are presented separately for all three chemicals and are not added into a single hazard index.

The noncancer hazard quotient assumes that there is a level of exposure (i.e., R_fD) below which it is unlikely for even sensitive populations to experience adverse health effects. If the exposure level (i.e., average daily intake) exceeds this threshold (i.e., if the hazard quotient exceeds unity), there may be concern for potential noncancer effects. It is important to note that the level of concern does not increase linearly as the R_fD is approached or exceeded because R_fDs do not have equal accuracy or precision and are not based on the same severity or toxic effects. Thus, the slopes of the dose response curve in excess of the R_fD can range widely depending on the substance (EPA, 1989c).

For all three compounds (i.e., TCDD, 2,4-D, and 2,4,5-T) inhalation, only oral R_fDs were available, and only an oral cancer potency factor (or UCR) was available for TCDD. Therefore, it was necessary to adjust these toxicity benchmark values, which were based on exposure (administered) dose to account for absorption. This route-to-route extrapolation method as been described by EPA (1989c) and is used to express the toxicity expected from an absorbed dose. Additionally, these adjusted toxicity benchmark values must then be used with inhalation exposure values which have also been adjusted to estimate absorbed dose. The uncertainties associated with this method include the fact that "point-of-entry" toxicity (i.e., in the lungs) cannot be estimated from oral toxicity data. Furthermore, unlike orally administered compounds, inhaled chemicals would not be subjected to first-pass hepatic metabolism before reaching the systemic circulation. Therefore, a toxic effect attributable to an active metabolite might be more pronounced if the compound was administered

orally. Conversely, the pulmonary absorption of a toxic parent compound that undergoes little or no first-pass metabolism may result in a greater dose of the toxic moiety entering the systemic circulation than if the compound was absorbed orally.

5.1 Quantitative Assessment of Risk

All parameters used in calculations leading to the expression of carcinogenic and systemic toxicity risks are presented in Table 5.1 for the current scenario and Table 5.2 for the two future use scenarios. Although all media were considered in the analysis, lack of or inadequate monitoring data on water and marine biota reduced multimedia considerations to air only. For this medium, both vapor phase and chemical-bound particulate were factored into the calculations.

For the *current scenario*, the cancer risk from exposure to TCDD is 3×10^{-5} for the TMEI and 3×10^{-5} for the AMEI. The hazard quotient from exposure to TCDD is 0.76 for the TMEI and 0.76 for the AMEI. The hazard quotient from exposure to 2,4-D is 0.0014 for the TMEI and 0.00051 for the AMEI. The hazard quotient from exposure to 2,4,5-T is 0.0015 for the TMEI and 0.00095 for the AMEI.

For the *future-use scenario involving excavation (Scenario 1)*, the cancer risk from exposure to TCDD is 8×10^{-7} for the TMEI and 8×10^{-7} for the AMEI. The hazard quotient from exposure to TCDD is 0.52 for the TMEI and 0.52 for the AMEI. The hazard quotient from exposure to 2,4-D is 0.00090 for the TMEI and 0.00034 for the AMEI. The hazard quotient from exposure to 2,4,5-T is 0.0010 for the TMEI and 0.00063 for the AMEI.

For the *future-use scenario involving paving (Scenario 2)*, the cancer risk from exposure to TCDD is 2×10^{-7} for the TMEI and 2×10^{-7} for the AMEI. The hazard quotient from exposure to TCDD is 0.25 for the TMEI and 0.25 for the AMEI. The hazard quotient from exposure to 2,4-D is 0.00045 for the TMEI and 0.00017 for the

Table. 5.1 Estimated Lifetime Average Daily Absorbed Dose a, i
Average Daily Absorbed Dose and Subsequent Risk from Inhalation of Vapor-Phase TCDD, 2,4-D, and 2,4,5-T within the Impact Zone of the Existing Herbicide Orange Site.

Compound	Ambient Air Conc (mg/m^3) (1hr avg)	Inhalation Rate (m^3/hr)	Exposure time (hr/d)	Exposure Freq (d/yr)	Exposure Duration (yr)	Absorption Fraction (vapor)	Body Weight (kg)	Avg. Time (d)	ABS. DOSE (mg/kg/day)	CANCER RISK	CHRONIC RfD (Adjusted) (mg/kg/day)	HAZARD RATIO (DOSE/RfD)
TMEI												
TCDD-c	1.01E-08	2.1	1	250	25	0.75	70	25550	5.55E-11	2.89E-05		
TCDD-nc	1.01E-08	2.1	1	250	1	0.75	70	250	2.27E-10		3.00E-10	7.56E-01
2,4-D	1.81E-04	2.1	1	250	1	0.75	70	250	4.06E-06		3.00E-03	1.35E-03
2,4,5-T	2.00E-04	2.1	1	250	1	0.75	70	250	4.51E-06		3.00E-03	1.50E-03
AMEI												
TCDD-c	1.01E-08	2.1	1	250	25	0.75	70	25550	5.55E-11	2.89E-05		
TCDD-nc	1.01E-08	2.1	1	250	1	0.75	70	250	2.27E-10		3.00E-10	7.56E-01
2,4-D	6.79E-05	2.1	1	250	1	0.75	70	250	1.53E-06		3.00E-03	5.09E-04
2,4,5-T	1.27E-04	2.1	1	250	1	0.75	70	250	2.85E-06		3.00E-03	9.50E-04

Table. 5.2 Estimated Lifetime Average Daily Absorbed Dose and Average Daily Absorbed Dose and Subsequent Risk from Inhalation of Vapor-Phase and Particle-Associated TCDD, 2,4-D, and 2,4,5-T within the Impact Zone During either Excavation or Construction of a Cement Cover.

SCENARIO 1: Excavation

Compound	Ambient Air Conc (mg/m^3) (1hr avg; adjusted for absorption)	Inhalation Rate (m^3/hr)	Exposure time (hr/d)	Exposure Freq (d/yr)	Exposure Duration (yr)	Absorption Fraction	Body Weight (kg)	Avg. Time (d)	ABS DOSE (mg/kg/day)	CANCER RISK	CHRONIC RfD (Adjusted) (mg/kg/day)	HAZARD RATIO (DOSE/RfD)
TMEI												
TCDD-c	7.80E-09	2.1	1	243	0.67	1	70	25550	1.48E-12	7.70E-07		
TCDD-nc	7.80E-09	2.1	1	243	0.67	1	70	243	1.56E-10		3.00E-10	5.19E-01
2,4-D	1.35E-04	2.1	1	243	0.67	1	70	243	2.70E-06		3.00E-03	9.01E-04
2,4,5-T	1.50E-04	2.1	1	243	0.67	1	70	243	3.00E-06		3.00E-03	1.00E-03
AMEI												
TCDD-c	7.80E-09	2.1	1	243	0.67	1	70	25550	1.48E-12	7.70E-07		
TCDD-nc	7.80E-09	2.1	1	243	0.67	1	70	243	1.56E-10		3.00E-10	5.19E-01
2,4-D	5.09E-05	2.1	1	243	0.67	1	70	243	1.02E-06		3.00E-03	3.39E-04
2,4,5-T	9.5E-05	2.1	1	243	0.6658	1	70	243	1.9E-06		3.00E-03	6.33E-04

SCENARIO 2: Construction of Cement Cover

Compoun	Ambient Air Conc (mg/m^3) (1hr avg; adjusted for absorption)	Inhalation Rate (m^3/hr)	Exposure time (hr/d)	Exposure Freq (d/yr)	Exposure Duration (yr)	Absorption Fraction	Body Weight (kg)	Avg. Time (d)	ABS DOSE (mg/kg/day)	CANCER RISK	CHRONIC RfD (Adjusted) (mg/kg/day)	HAZARD RATIO (DOSE/RfD)
TMEI												
TCDD-c	7.58E-09	2.1	1	120	0.33	1	70	25550	3.51E-13	1.83E-07		
TCDD-nc	7.58E-09	2.1	1	120	0.33	1	70	120	7.48E-11		3.00E-10	2.49E-01
2,4-D	1.35E-04	2.1	1	120	0.33	1	70	120	1.34E-06		3.00E-03	4.45E-04
2,4,5-T	1.50E-04	2.1	1	120	0.33	1	70	120	1.48E-06		3.00E-03	4.94E-04
AMEI												
TCDD-c	7.58E-09	2.1	1	120	0.33	1	70	25550	3.51E-13	1.83E-07		
TCDD-nc	7.58E-09	2.1	1	120	0.33	1	70	120	7.48E-11		3.00E-10	2.49E-01
2,4-D	5.09E-05	2.1	1	120	0.33	1	70	120	5.02E-07		3.00E-03	1.67E-04
2,4,5-T	9.50E-05	2.1	1	120	0.33	1	70	120	9.37E-07		3.00E-03	3.12E-04

AMEI. The hazard quotient from exposure to 2,4,5-T is 0.00049 for the TMEI and 0.00031 for the AMEI.

5.2 Uncertainties

As in exposure assessment (see Section 3.4), there are uncertainties associated with the dose-response component of risk assessment. The EPA is now considering new evidence to suggest that TCDD may be a threshold carcinogen dependent on receptor-mediated (aryl hydroxylase) binding into a ligand-receptor complex for all dioxin-induced effects, and that this binding is rate-limiting. Furthermore, the complex must undergo activation and translocation into the nucleus as a prerequisite for effect. The Agency is now considering lowering the slope factor by two-fold, which would have an impact on the ultimate expression of risk. At this time of report preparation, the IRIS file on TCDD has been pulled while deliberations are underway on this issue.

As recorded in Table 4.10, the level of confidence in the studies used to develop RfD's for all three chemicals can be highly variable for a great variety of reasons having to do with the quality of available science. No level of confidence is presented for TCDD; levels of confidence for 2,4-D and 2,4,5-T are described as medium, creating a margin of uncertainty.

Susceptibility to chemical toxicity among potential human receptors can also be highly variable due to preexisting general morbidity of residents on the Island, particular sensitivities among individuals (e.g., pregnant women), and such other factor as genetic predisposition to cancer.

Determination of carcinogenic risk from exposure to TCDD is typically amortized over a lifetime of 70 years. While exposure for the current scenario was assumed to have a maximum duration of 25 years (based on first exposure in 1972

and paving, excavation, or some other modification to the site in 1997), for some individuals, lifetime may be fewer or greater than 70 years, creating an element of uncertainty in the risk calculation.

Section 4.0 included a discussion on the toxicity of HO as a mixture. However there is insufficient evidence to formulate either a composite R_fD or additive hazard quotients. As a result, any synergistic, potentiative, or antagonistic effects posed by exposure to the three chemicals in combination could alter the benchmark values used to calculate risk. These toxicological phenomena could not be accounted for in this analysis.

Finally, the uncertainties posed by dose-response data and the toxicity benchmark values derived from them for the determination of risk are compounded on top of the uncertainties associated with exposure assessment, as expressed in Section 3.4. Together they may result in a risk determination that can be off by as many as two orders of magnitude.

6.0 Ecological Effects

Johnston Island is a coral atoll occupying 626 acres in the Pacific Ocean, 717 nautical miles southwest of Honolulu. The island was expanded from an area of 60 acres by the deposition of local dredged material in 1942. The marine ecosystem in the waters surrounding the Johnston Atoll is typical of a diverse tropical Indo-Pacific reef community. One hundred ninety-three fish species and 164 invertebrate species have been identified (Amerson and Shelton, 1976). The terrestrial fauna at the Johnston Atoll comprises about 40 species of birds, many of which brood on the nearby Sand Island. Relative to the marine community, the terrestrial ecosystem is less diverse since the island is arid, only seven feet above sea level, and has no tropical forest. No information was available on other terrestrial fauna and flora. Most of the land on the island is taken up by a 9,000 foot runway and military buildings associated with the chemical agent disposal system and, therefore, would provide poor habitat for most species.

As part of the investigation of contaminant effects at JI, this section describes the sampling and analysis of TCDD in sediments and biota, analyzes possible exposure of ecological receptors (fish, invertebrates, and birds) to dioxin, and assesses

risks. Risks to the ecological community resulting from exposure to 2,4-D and 2,4,5-T have not been assessed because these substances were not monitored in the present study.

6.1 Sampling Data

From 1985 through 1988, sediments were sampled from four areas of JI. Areas 1 through 3 are near the inner reef in the vicinity of the HO site, while Area 4 is on the opposite side of the Island (Figure 1). While a total of 38 samples were collected (Table 1), only 26 were identified by sampling area. In Area 1, dioxin was detected in one of 11 samples at a concentration of 160 parts per trillion (ppt). In Area 2, dioxin was detected in one of seven samples at a concentration of 190 ppt. Dioxin was not detected in the four Area 3 samples or the two Area 4 samples.

Samples were collected from a variety of fish, invertebrate, and bird species from 1984 through 1989 (Table 2). A total of 199 tissue samples (44 fish species, 13 invertebrate species, 2 bird species) were analyzed for dioxin. Samples of aquatic species were collected from Areas 1 through 4, Area 5 (inner reef), and Area 6 (outer reef) (see Figure 1). Samples of birds were collected on land near the Formal HO Storage Area.

A total of 32/199 tissue samples contained detectable concentrations of dioxin. Frequency of detection for the fish, invertebrate, and bird samples from each area is listed in Table 2.1. Analysis of the fish and invertebrate tissue data is complicated by the use of different organs (liver, muscle, and unspecified organs) for various samples. In addition, differences in habitat and feeding strategies are likely to result in variable uptake. Nevertheless, for the purpose of summarizing the data, all fish (whole body, muscle, or unspecified), crab, snail, octopus, and sea cucumber data have been summarized for each area.

A total of three bird samples were analyzed. TCDD was not detected in any of the samples which included one liver sample and two unspecified organ samples.

6.2 Toxicological Profile for TCDD

The toxicity of dioxin to fish and wildlife was reviewed by Eisler (1986). Dioxin is toxic to fish at low and sub-ng/L levels which makes it one of the most toxic compounds tested in aquatic organisms. Mehrle et al. (1988) reported significant increases in mortality and decreases in growth in rainbow trout (*Oncorhynchus mykiss*) exposed for 28 days to 0.038 ng/L followed by a 28-day observation period. Recently, Wisk and Cooper (1990) exposed Japanese medaka (*Oryzias latipes*) embryos to dioxin beginning on the day of fertilization and continuing until hatch (11 to 14 days). A statistically significant increase in the incidence of lesions occurred at 0.4 ng/L. Eisler's (1986) review stated that the highest tested concentration that did not produce adverse effects was 0.01 ng/L.

Due to its low water solubility, estimated at less than 20 ng/L (Marple et al., 1986), releases of dioxin to the aquatic environment tend to result in accumulations in sediments and biota (Eisler, 1986). Eisler (1986) cited studies in which higher levels of dioxin were found in bottom-feeding versus top-feeding fish, indicating the likely importance of sediments as a source. Dietary uptake may also contribute to body burdens as substantial levels of dioxin were measured in fish gut contents (Young and Cockerham, 1985; as cited in Eisler, 1986). Mehrle et al. (1988) estimated a bioconcentration factor (steady state fish muscle concentration divided by water concentration) of 39,000. Monitoring studies have identified measurable levels of dioxin in field samples of fish and crab tissues (e.g., Belton et al., 1985; Ryan et al., 1984). Studies in New Jersey have resulted in closure of the Passaic River to the harvesting of fish and shellfish because dioxin was frequently found in fish and crabs at concentrations exceeding the FDA levels of concern (Belton et al., 1985).

Several studies were found linking tissue residues with toxic effects. The Mehrle et al. (1988) study, which reported increased mortality and decreased growth, measured mean whole body dioxin concentrations of 0.74 ng/g (=740 ppt). Branson et. al. (1985) exposed rainbow trout to 0.107 ng/L dioxin for 6 hours and monitored elimination over 139 days. Dioxin body burdens at the end of the study were 650 ppt in whole fish, 260 ppt in muscle, and 2710 in liver. In these fish, there was reduced growth relative to controls and evidence of fin rot. The embryo exposure study of Wisk and Cooper (1990) reported that lesions were reported in embryos containing 240 ppt dioxin.

Dioxin is known to bioaccumulate in fish-eating birds (reviewed by Walker, 1990). Braune and Norstrom (1989) measured dioxin concentrations in herring gulls (*Larus argentatus*) and alewife, which comprise a major portion of their diet, from Lake Ontario. Mean whole body dioxin concentrations were 127 ppt in gulls and 4 ppt in fish. A biomagnification factor (whole body bird/whole body alewife concentration) of 32 was calculated. Egg levels may be similar to whole body levels; mean dioxin levels in herring gull eggs and whole body tissues were 83 and 127 ppt, respectively.

Elliott et al. (1989) reported that population declines in great blue herons (*Ardea herodias*) in British Columbia coincided with a tripling of dioxin levels in eggs from 66 to 210 ppb. These researchers cited studies in which colonial waterbird population declines occurred when dioxin levels exceeded 2000 ppt and began to recover when levels decreased to below 500 ppt. These field studies have not established causal relationships; controlled laboratory studies are required. Eisler (1986) cited a laboratory study in which chick edema disease (pericardial, subcutaneous, and peritoneal edema accompanied by liver enlargement and necrosis) occurred in domestic chickens fed dioxin at 1 or 10 ppb for 21 days. This disease was frequently lethal.

6.3 Risk Assessment

Releases of HO have exposed fish and invertebrates and possibly birds to dioxin. Only a rough estimate of risk is possible given the limitations of the data. When possible, risks were assessed by comparing body burdens with levels associated with toxic effects.

6.3.1 Aquatic life

The highest concentration of dioxin was reported in the crown squirrelfish. Squirrelfishes tend to remain close to the bottom and do not travel long distances (Migdalski and Fichter, 1976). These behaviors may increase their exposure to localized sources of dioxin in sediments. Out of four samples (three Area 1; one Area 2), TCDD was detected in one sample from Area 1 at 352 ppt and in one sample from Area 2 at 472 ppt. These concentrations exceed the 260 ppt measured in rainbow trout muscle that was associated with decreased growth and fin lesions (Branson et al., 1985).

The only other fish species with concentrations exceeding 100 ppt was the yellowfin goatfish. Three samples were collected in Area 1, where concentrations were 11, 85, and 102 ppt. TCDD was not detected in single samples of this species from Areas 2 and 5. Goatfishes are bottom feeders (Migdalski and Fichter, 1976), which may account for their enhanced body burdens. The maximum reported concentration is nearly one-half the 260 ppt reported as toxic by Branson et al. (1985).

Several invertebrate samples were detected at levels between 14 and 28 ppt. The only invertebrate sample detected at greater than 100 ppt was a "snails" sample from Area 2 measured at 120 ppt. No data linking tissue concentrations with effects in snails could be located.

Uncertainties in the analysis result from the collection of a small number (usually less than five) samples of each species in each area. In addition, in some samples either the species or organ that was analyzed or the collection site was not reported.

6.3.2 Birds

In three samples of birds, there were no detectable concentrations of dioxin. Further sampling is recommended to more adequately characterize risks.

6.4 Regulatory Concentrations

EPA has not issued ambient water quality criteria for the protection of aquatic life from exposure to dioxin (F. Gostomski, EPA, personal communication, January 22, 1991). FDA advisory levels are for the protection of human health rather than aquatic species. No sediment quality criteria have been published or proposed for dioxin.

7.0 *Data Requirements Assessment*

The EPA (1989) recommends that the data needs for the RI/FS be addressed at the site scoping meetings. Developing a comprehensive sampling and analysis plan (SAP) during the scoping meeting allows all of the data needs for the RI/FS, including the risk assessment, to be met. The data needs are identified by determining the type and duration of possible exposures (e.g., acute, chronic), potential exposure routes (e.g., fish ingestion, dust inhalation), and key exposure points (e.g., work areas) for each medium. These same types of considerations are also important for the ecological risk assessment. Data needs may have to be addressed before a more comprehensive risk assessment can be performed.

While there is always a need for better empirical data on toxicity, dispersion modeling, and general methodologies for expressing risk, monitoring data is usually site-specific and can be tailored to specific features of the site. There has not been a systematic effort in collecting the needed monitoring data at the HO site. To date, the most definitive data-collection activity has been the soil characterization study by Crockett et al. (1986). Data that can be obtained to convert this risk assessment into a more realistic multimedia approach are presented below. Many of these needs

were presented in the trip report for the site visit (Appendix C). Although the indicated supplemental data collection would provide the complete range of information needed for a full baseline risk assessment, there are some pieces of information that are more important than others, so that the individual needs may need to be ranked in priority order. This may preclude the necessity of having to perform all recommended procedures.

7.1 Air Sampling

The risk assessment used estimated values for the particulate and vapor phase emissions from the site. Air sampling would characterize the particulates and vapors coming from the site. Particle size distribution will enable determination of the percentage of respirable dust. To determine the wind erosion around the site several Hi-Vol samplers, equipped with particulate traps, could be placed downwind around the fence line. At the southwestern fenceline the odor of 2,4-D was detectable during the site visit, indicating that there may be significant vapor emissions from the site. Organic vapor phase samplers capable of collecting dioxins, 2,4-D, and 2,4,5-T can be placed around the site to characterize ambient air concentrations. There are other potential sources of dioxin on JI, including JACADS, the burn pit, and the fire training area. Sampling would permit source apportionment of dioxin from each of these sites.

7.2 Soil Sampling

The characteristics of the soil can have an influence on the bioavailability of dioxins and the other chemicals. Soil moisture content, organic content, and particle size distribution are missing elements that are important for lowering the uncertainty in the soil exposure calculations. It was originally planned to vertically sample the TCDD hot spots, but sample results were not available in time to accomplish this, and, therefore, some hot spots were missed in the vertical soil sampling. These hot

spots could now be sampled vertically for all three compounds, TCDD, 2,4-D, and 2,4,5-T. Only 15 plots were sampled for 2,4-D and 2,4,5-T, presenting a spacial distribution for these compounds inadequate for risk assessment. More plots could be sampled for these two compounds. One method that can be used to accomplish this is to revisit the 48 plots that were originally vertically sampled. These 48 plots could be sampled for all three chemicals of concern. This sample design would have two benefits: (1) better knowledge of the spacial distribution for 2,4-D and 2,4,5-T; and (2) knowledge of the fate of these chemicals over time.

7.3 Sediment Sampling

Channell and Stoddart (1984) found positive sediment samples near the western shore, prior to construction of the seawall in that area. This area could be revisited to determine if the seawall is performing according to its intended function. More sediment samples are needed to better characterize the spacial pattern of contamination. A grid pattern similar to the soil sampling protocol would help to characterize the spacial contamination pattern. These samples should include areas close to the shoreline.

7.4 Water Sampling

7.4.1 Seawater Sampling

No seawater sampling has been conducted off the former HO site. The U.S. Fish and Wildlife (1987) report that TCDD levels of 38 pg/l are toxic to fish. Toxic endpoints include severe adverse effects on survival, growth, and behavioral responses. With this potency, seawater sampling may be important.

7.4.2 Groundwater Sampling

The groundwater under the former HO site has never been sampled and may be a vital link in any discovery of HO site-related fish contamination. Groundwater sampling could proceed as described in Appendix C.

7.5 Biological Sampling

More sampling can to be performed within Site 3 to determine if contaminated fish are in this area. No biological samples have been analyzed for 2,4-D or 2,4,5-T. It is not possible to assess the potential impact from fish ingestion for these two chemicals if this analysis is not performed. Walsh III (1984) and Randall (1961) demonstrated that several adult fish species can have large movements. A study could be performed to ascertain if these migratory fish species are moving from the waters adjacent to the former HO site into fishing waters (e.g., Zones 5 and 10 in Figure 3.1). Sampling and analysis of fishermen's catches can be easily used to determine if humans are consuming contaminated fish. This is the only study that would demonstrate if the fish being consumed are contaminated.

7.6 Ecological Risk Sampling Recommendations

Further field investigations may be needed to adequately characterize the ecological risks at JI. Any additional research should be coordinated with the work underway by Dr. John Labelle of the Woods Hole Oceanographic Institute in support of the JACADS monitoring program. Additional sampling programs could be designed so that statistical comparisons can be made between concentrations in the different areas. In such an investigation sediment sampling would be expanded to allow better characterization of the spatial pattern of contamination. Biota samples would be focussed on species whose behavior may lead to greater levels of contamination (e.g., bottom feeding resident species). Organisms that are important

parts of marine food chains (e.g., small invertebrates such as marine worms) would be sampled. Based on the available data, the crown squirrelfish, yellowfin goatfish, snails, and crabs are good candidates for further sampling. Increased sampling of birds may be required to determine whether populations are at risk due to consumption of contaminated prey (e.g., fish and snails). Sampling could focus on one or two bird species that tend to be localized on the Island.

Although the contaminant studies should remain focussed on dioxin, it would be useful to examine several fish samples for 2,4-D. This compound has been measured at levels as high as 281 ppm in soil samples on the Island (Crockett et al. 1986). Although it is not bioaccumulated to the same extent as dioxin, measurable residues have been reported in fish from lakes treated with the compound (Frank et al. 1987) and toxicity data are available (e.g., Cope et al., 1970).

8.0 *Summary*

Scope of the study and physical setting. This report contains the results of a screening-level risk assessment conducted for the Air Force Occupational and Environmental Health Laboratory concerning the Herbicide Orange (HO) storage site at Johnston Island (JI). The risk assessment is part of the remedial investigation and feasibility study (RI/FS) process established by the U.S. EPA for characterizing the nature and extent of risks posed by hazardous waste sites and for developing and evaluating remedial options. This process is being conducted in the context of the U.S. Department of Defense (DoD) Installation Restoration Program (IRP).

JI is currently used for three purposes:

1. In the late 1950's and early 1960's, the island was used to launch missiles for atmospheric testing of nuclear weapons. During 1962, three missile aborts caused transuranic contamination on parts of the island. Launch and support facilities at JI are maintained in a caretaker status in case testing is deemed necessary for national defense.

2. JI has been designated as a chemical warfare destruction site and the Department of the Army maintains the Johnston Atoll Chemical Agent Disposal System (JACADS) on the Island. JACADS is involved in active thermal destruction of CW agents.

3. Johnston Atoll, including JI, is a National Bird Refuge, largely because of bird populations on nearby Sand Island. Among the few species of animal life swimming in waters off JI is the green sea turtle, currently classified as an endangered species. The Island is also used as a chemical munitions storage site.

The Island is inhabited with military personnel and civilian employees of DoD support contractors. The tour of duty for military personnel has generally run 1 to 2 years. Civilian personnel have generally been on the Island for longer periods of time (5 years but as many as 15 years or more). No children reside on the Island, although there is a potential for fetal exposures.

Site characterization. During the period from 1972 to 1977, JI was also used for temporary storage of Herbicide Orange (HO). A total of 1.37 million gallons of HO in 26,300 fifty-five gallon drums were transferred to JI from South Vietnam in 1972. The drums were stored on a 4-acre site on the northwest corner of the Island. The HO was successfully incinerated at sea in 1977. Corrosion of drums while in storage resulted in HO leakage at a rate of approximately 20 to 70 drums per week. Approximately 49,000 pounds of HO are estimated to have escaped into the environment annually during the storage period. The site is now contaminated with the active ingredients of HO: 2,3,7,8-tetrachloro-dibenzodioxin (TCDD); the n-butyl ester of 2,4-dichlorophenoxy acetic acid (2,4-D); and the n-butyl ester of 2,4,5-trichlorophenoxyacetic acid (2,4,5-T).

For this risk assessment, the chemicals of primary concern are TCDD, 2,4-D, and 2,4,5-T. The site is bounded by a seawall to the west-northwest, an open area and storage area to the east-southeast, a roadway to the south, and several limited-use operations to the west: a transformer, beacon building, Hi-Vol sampler associated with JACADS, fire training area, and burn pit. Access to the site itself is restricted by a fence on all landlocked sides. Soil on the site is contaminated with the three chemicals of concern. Soil samples taken in 1986 contained surface residues of TCDD (nondetect at 0.1 ppb to 163 ppb), 2,4-D (2.5 to 281,330 ppb), and 2,4,5-T (53 to 237,155 ppb). Soil samples also contained subsurface residues of TCDD (nondetect to 510 ppb), 2,4-D (nondetect to 365,202 ppb), and 2,4,5-T (nondetect to 682,247 ppb). Measurement of these substances in air, groundwater, seawater, and sediments have not been conducted. Analysis of marine biota for TCDD has revealed residues ranging from nondetect to 472 ppb. Subsurface soil and marine biota samples were limited to the point of greatly confining the scope of the exposure and risk assessments.

Exposure assessment. The potential for exposure to TCDD, 2,4-D, and 2,4,5-T for persons engaged in activities proximal to the HO site is dependent on numerous factors including the physical setting of the site (i.e., climate, vegetation, soil type, and hydrology), as well as features of the potentially exposed populations. The frequency and duration of potential exposure depends on population demographics and human activities patterns associated with land-use around the site.

The site is currently not in use, is dormant, and has limited access by a surrounding fence. However, potential avenues of human exposure include volatilization of the contaminants into the air, suspension of particle-associated compounds into the air due to wind erosion, and consumption of edible marine life that have become contaminated in the waters adjacent to the site. For purposes of assessing current or "baseline" risk from exposures related to the HO site, only the air pathway was evaluated. Wind erosion was judged to be non-significant for the

undisturbed site, whereas, ingestion of contaminated marine biota, while considered plausible, could not be performed to due the lack of sufficient data.

For exposure through the air medium, important human activities include, but are not necessarily limited to, occupational operations associated with the seawall, the electrical transformer, the Hi-Vol sampler, the beacon building in the immediate area, the fire training area, the rip-rap area used as a boat-launch site, and the burn pit at an intermediate distance.

Two future scenarios that would alter exposure potential from that presented by current land conditions which were considered in this report are: (1) remediation through excavation; and (2) covering of the site with cement. For purposes of assessing potential inhalation exposures due to the release of particle-associated compounds resulting from future-use activities, emission rates were estimated for each activity (i.e., unloading and loading of contaminated soil, vehicular traffic, wind erosion) within each scenario (i.e., excavation or cement cover construction).

For both vapor-phase inhalation potentially occurring during the current scenario, as well as vapor-phase and particle-associated inhalation potentially occurring during the two future-use scenarios, exposure was estimated for the Theoretical Most Exposed Individual (TMEI), as well as an Alternate Most Exposed Individual (AMEI). The TMEI was assumed to have access to the entire perimeter of the HO site; whereas, the AMEI has access to only the fenceline (southern side of the site).

To estimate the air concentrations (g/m^3) of both vapor-phase and particle-associated TCDD, 2,4-D, and 2,4,5-T, a screening-level atmospheric dispersion modeling analysis was conducted to estimate one-hour, eight-hour, and annual average concentrations of these compounds around the perimeter of the HO site. These predicted air concentrations were then used to estimate inhalation exposures

and lifetime average and average daily absorbed doses to the TMEI and the AMEI. The estimated absorbed doses where then used to assess cancer and noncancer risks, respectively.

Toxicity assessment. For noncarcinogenic toxic endpoints, TCDD appears to be approximately seven orders of magnitude more potent than either 2,4-D or 2,4,5-T, with oral R_fD's of 1×10^{-9}, 1×10^{-2}, and 1×10^{-2}, respectively. The primary critical effect seen for TCDD was fetal survival and the secondary critical effect seen was renal damage. The primary critical effect seen for 2,4-D was renal damage and the secondary critical seen was hematologic and hepatic effects. The R_fD for 2,4-D was based on studies producing a medium level of confidence. For 2,4,5-T the primary critical effect was neonatal survival, and the secondary critical effect was increased urinary coproporphyrin excretion. The R_fD for this chemical was based on studies producing a medium level of confidence.

For both 2,4-D and 2,4,5-T an evaluation of their carcinogenicity cannot be made on the limited animal data available. TCDD is classified as a B1 carcinogen when associated with phenoxy herbicides and/or chlorophenols. In animal studies TCDD has been shown to be a potent carcinogen with an oral slope factor of 1.56×10^5 $(mg/kg/day)^{-1}$. Increased incidences of cancer have been observed in lungs, liver, hard palate, and nasal turbinates. Epidemiological studies have produced only a potential correlation of an increased risk of soft-tissue sarcomas for chemicals contaminated with TCDD.

Human health risk assessment. Characterization of risk based on the results of the exposure assessment for inhalation of vapor-phase TCDD revealed that current or baseline lifetime excess cancer risk associated with the undisturbed HO site was approximately 3×10^{-5} for both the TMEI and the AMEI. This is equivalent to 3 excess cancer cases occurring among 10,000 individuals exposed for a period of 25 years during their lifetime. TCDD-associated estimated cancer risks resulting from

excavation and cement cover construction activities were 8×10^{-7} and 2×10^{-7}, respectively, for both the TMEI and the AMEI. The magnitude of these cancer risk estimates are within the Superfund site remediation goals (i.e., cancer risk range of 10^{-4} to 10^{-7}); however, it is plausible that additional lifetime excess cancer risk may be present due to ingestion of contaminated marine biota. This exposure pathway has not been adequately characterized and was not included in the risk characterization.

For the current scenario, noncancer risks, as measured by hazard quotients from exposure of the TMEI to TCDD, 2,4-D, and 2,4,5-T, were 0.76, 0.0014, and 0.0015, respectively; whereas, the hazard quotients from exposure of the AMEI to TCDD, 2,4-D, and 2,4,5-T were 0.76, 0.00051, and 0.00095, respectively.

For the future excavation scenario, noncancer risks, as measured by the hazard quotients from exposure of the TMEI to TCDD, 2,4-D, and 2,4,5-T, were 0.52, 0.00090, and 0.0010, respectively; whereas, the hazard quotients from exposure of the AMEI to TCDD, 2,4-D, and 2,4,5-T were 0.52, 0.00034, and 0.00063, respectively.

For the future cement cover construction scenario, noncancer risks, as measured by the hazard quotients from exposure of the TMEI to TCDD, 2,4-D, and 2,4,5-T, were 0.25, 0.00045, and 0.00049, respectively; whereas the hazard quotients from exposure of the AMEI to TCDD, 2,4-D, and 2,4,5-T were 0.25, 0.00017, and 0.00031, respectively.

Similar to the cancer risk estimates for TCDD, these noncancer hazard quotients are within the Superfund site remediation goals (i.e., less than 1.0). However, noncancer risk resulting from ingestion of contaminated marine biota has not been evaluated.

Uncertainties associated with this analysis. There are several significant uncertainties associated with soil characterization, exposure assessment, and risk characterization. The two future-use scenarios, remedial excavation or surfacing with unknown pretreatment, are hypothetical and not necessarily reflective of actual future use. Many empirical and site-specific assumptions were made in the exposure assessment, including body weight, inhalation rate, pulmonary deposition rate, construction vehicle weight, number of wheels rolling over the site, duration of excavation, duration of the soil covering activity, physicochemical features of the soil, threshold wind velocity, diffusion and air-soil partition coefficients, and spatial distribution of 2,4-D and 2,4,5-T on the surface and in vertical profiles. In addition, other variables were unaccounted for in the analysis. They include population transience, male/female differences in exposure, presence of other isomers of dioxin and other chemicals on the Island and prior or concurrent exposures to them, atmospheric transformation and soil photodegradation of the chemicals of concern, groundwater contamination, and potential concurrent exposures from JACADS.

With regard to toxicity and dose-response parameters associated with the risk calculation, uncertainties include what is currently a rethinking of the mechanism of toxicity of TCDD in the scientific community (which would affect the benchmark toxicity value used in the risk calculation), medium levels of confidence in the R_fD values used, and the potential for sensitive individuals and those with preexisting morbidity to be exposed to chemicals at the HO site. In addition, the assumed periods of maximum exposure (25 years) and lifetime risk (70 years) may be incorrect. Lastly, synergistic or other toxicological phenomena caused by chemical interaction are unknown.

Ecological risk. A limited data base permitted only a preliminary ecological risk assessment. Sediment sampling indicates several locations of dioxin contamination. Among resident fish species sampled at the site, the crown squirrelfish had the highest dioxin levels in several samples (352 and 472 ppb).

These concentrations exceed levels reported to be associated with toxic effects in the rainbow trout. Further sampling of fish, invertebrates, birds, and sediments is needed to characterize the spatial pattern of contamination and to assess ecological risks.

Needs assessment. There is a fairly large uncertainty associated with the calculation of human health and ecological risks for the HO site because of a consistent lack of appropriate scientific information. It is recommended that uncertainty reduction be given a high priority in any future activities concerning HO site closure. With specific regard to the air component of the risk assessment, it is recommended that particulate and vapor-phase concentrations of TCDD, 2,4-D, and 2,4,5-T be conducted. Since ambient air concentrations of these chemicals is dependent on soil characteristics, it is recommended that additional soil sampling be performed to characterize soil moisture and organic content, particle size distribution, and spacial distributions of the chemical contaminants. Sediment and water sampling is recommended to determine which medium or media contain the potential source of the fish contamination. Further biological sampling is recommended to better characterize the potential for human exposure to contaminated fish, and (as a National Bird Sanctuary) the risks to the avian populations on the Atoll.

9.0 References

Abernathy, D.J., W.F. Greenlee, J.C. Hubard, and C.J. Boreiko. 1985. 2,3,7,8-Tetrachlorodibenzo-*p*-dioxin (TCDD) promotes the transformation of C3H/10T1/2 cells. *Carcinogenesis* 6:651-653.

Amerson, A.B., and P.C. Shelton. 1976. *The Natural History of Johnston Atoll, Central Pacific Ocean.* Atoll Research Bulletin No. 192. University of Kansas, Department of Systematics and Ecology, Lawrence, Kansas.

Anderson, D., and J.A. Styles. 1978. The bacterial mutation test. *Br. J. Cancer* 37:924-930.

Anderson, E., N. Browne, S. Duletsky, J. Ramig, and T. Warn. 1985. *Development of Statistical Distributions or Ranges of Standard Factors Used in Exposure Assessments. Final Report.* Report No. EPA/600/8-85/010, prepared by Technology Division, GCA Corporation, Chapel Hill, N.C. Washington, D.C.: U.S. Environmental Protection Agency, Office of Research and Development, Exposure Assessment Group. Available from National Technical Information Service, Springfield, Virginia, as PB85-242667.

ATSDR (Agency for Toxic Substances and Disease Registry). 1989. *Toxicological Profile for 2,3,7,8-Tetrachlorodibenzo-p-Dioxin.* Report No. ATSDR/TP-88/23, prepared by Syracuse Research Corporation. Atlanta: Agency for Toxic Substances and Disease Registry. 138 pp. (available from National Technical Information Service, Springfield, Virginia, as PB89-214522).

Belton, T.J., R. Hazen, B.E. Ruppel, K. Lockwood, R. Mueller, E. Stevenson, and J.J. Post. 1985. *A Study of Dioxin (2,3,7,8-Tetrachlorodibenzo-p-Dioxin) Contamination in Select Finfish, Crustaceans and Sediments of New Jersey Waterways.* Office of Science and Research, New Jersey Department of Environmental Protection, Trenton, N.J.

Berry, D.L., J. DiGiovanni, M.R. Juchau, W.M. Bracken, G.L. Gleason, and T. J. Slaga. 1978. Lack of tumor-promoting ability of certain environmental chemicals in a two-stage system of mouse skin tumorigenesis: Potent anticarcinogenic effects. *Ann. N.Y. Acad. Sci.* 320:405-414.

Birnbaum, L.S., M.W. Harris, C.P. Miller, R.M. Pratt, and J.C. Lamb, IV. 1986. Synergistic interaction of 2,3,7,8-tetrachlorodibenzo-*p*-dioxin and hydrocortisone in the induction of cleft palate in mice. *Teratology* 33:29-35.

Branson, D.R., I.T. Takahashi, W.M. Parker, and G.E. Blau. 1985. Bioconcentration kinetics of 2,3,7,8-tetrachlorodibenzo-*p*-dioxin in rainbow trout. *Environ. Toxicol. Chem.* 4:779-788.

Braune, B.M., and R.J. Norstrom. 1989. Dynamics of organochlorine compounds in herring gulls: III. Tissue distribution and bioaccumulation in Lake Ontario gulls. *Environ. Toxicol. Chem.* 8:957-968.

Bronzetti, G., E. Zeiger, I. Lee, . . Suzuki, and H.V. Malling. 1983. Mutagenicity study of TCDD and ashes from urban incinerator "*in vitro*" and "*in vivo*" using yeast D7 strain. *Chemosphere* 12:549-553.

Channell, R.E., and T.L. Stoddart. 1984. *Herbicide Orange Monitoring Program: Interim Report, January 1980-December 1982.* Report No. ESL-TR-83-56. Engineering & Services Laboratory, Air Force Engineering & Services Center, Tyndall AFB, Florida. 58 pp.

Cope, O.B., E.M. Wood, and G.H. Wallen. 1970. Some chronic effects of 2,4-D on the bluegill (*Lepomis macrochirus*). *Trans. Am. Fish. Soc.* 99:1-12.

Courtney, K.D. 1976. Mouse teratology studies with chlorodibenzo-*p*-dioxins. *Bull. Environ. Contam. Toxicol.* 16(6):674-681.

Cowherd, C., Jr., G.E. Muleski, P.J. Englehart, and D.A. Gillette. 1985. *Rapid Assessment of Exposure to Particulate Emissions from Surface Contamination Sites.* Report No. EPA/600/8-85/002, prepared by Midwest Research Institute, Kansas City, Missouri. Washington, D.C.: U.S. Environmental Protection Agency, Office of Health and Environmental Assessment. [159 pp.]

Crockett, A.B., A. Propp, and T. Kimes. 1986. *Soil Characterization of Former Herbicide Storage Site at Johnston Island.* Report No. ESL-TR-86-18 to U.S. Air Force Engineering and Services Center, Tyndall AFB, Florida. Idaho Falls: EG&G Idaho, Inc. 178 pp.

Dencker, L., and R.M. Pratt. 1981. Association between the presence of the Ah receptor in embryonic murine tissues and sensitivity to TCDD-induced cleft palate. *Terat. Carcinog. Mutag.* 1:399-406.

Dencker, L., E. Hassoun, R. D'Argy, and G. Alm. 1985. Fetal thymus organ culture as an *in vitro* model for the toxicity of 2,3,7,8-TCDD and its congeners. *Mol. Pharmacol.* 27:133-140.

Driver, J.H., G. Whitmyre, J.J. Konz, T.A. Roy, J.J. Yang, and A.J. Kreuger. 1990. Dermal exposure assessments for toxicants in soil: Soil adherence to skin and dermal bioavailability. In: *Total Exposure Assessment Methodology. A New Horizon.* Proceedings of the EPA/AandWMA Specialty Conference, November 1989, Las Vegas, Nevada.

Eisler, R. 1986. Dioxin hazards to fish, wildlife, and invertebrates: A synoptic review. *U.S. Fish Wildl. Serv. Biol. Rep.* 85(1.8).

Elliott, J.E., R.W. Butler, R.J. Norstrom, and P.E. Whitehead. 1989. Environmental contaminants and reproductive success of great blue herons *Ardea herodias* in British Columbia, 1986-87. *Environ. Pollut.* 59:91-114.

EPA (U.S. Environmental Protection Agency). 1977. *Compilation of Air Pollutant Emission Factors, 3rd ed. with Supplements 1-7. Parts A and B.* Report No. AP-42. Research Triangle Park, N.C.: U.S. Environmental Protection Agency, Office of Air and Waste Management and Office of Air Quality Planning and Standards. [477 pp.]

EPA (U.S. Environmental Protection Agency). 1979. *Water-Related Environmental Fate of 129 Priority Pollutants. Volume I.* Washington, D.C.: U.S. Environmental Protection Agency, Office of Water Planning and Standards. Report No. EPA-440/4-79-029a.

EPA (U.S. Environmental Protection Agency). 1983. *Methods for Assessing Exposure to Windblown Particulates.* Report No. EPA-600/4-83-007. Prepared by Enviro Control Division, Dynamac Corporation, for EPA Office of Solid Waste and Emergency Response. Washington, D.C.: U.S. Environmental Protection Agency, Office of Health and Environmental Assessment. 65 pp.

EPA (U.S. Environmental Protection Agency). 1984. *Ambient Water Quality Criteria Document for 2,3,7,8-Tetrachlorodibenzo-p-Dioxin.* Report No. EPA-440/5-84-007.

Washington, D.C.: U.S. Environmental Protection Agency, Office of Water Regulations and Standards.

EPA (U.S. Environmental Protection Agency). 1985. *Drinking Water Criteria Document for 2,3,7,8-Tetrachlorodibenzo-p-Dioxin.* Report No. EPA 600/X-84-194-I. Cincinnati, Ohio: U.S. Environmental Protection Agency, Environmental Criteria and Assessment Office.

EPA (U.S. Environmental Protection Agency). 1987. *Industrial Source Complex (ISC) Dispersion Model User's Guide, Volumes 1 and 2,* 2nd ed. (revised). Report Nos. EPA-450/4-88-002a and 002b. Research Triangle Park, N.C.: U.S. Environmental Protection Agency.

EPA (U.S. Environmental Protection Agency). 1988a. *A Cancer Risk-Specific Dose Estimate for 2,3,7,8-TCDD.* Appendices A through F. Report No. EPA/600/6-88/007Ab (Draft). Washington, D.C.: U.S. Environmental Protection Agency, Office of Research and Development, Office of Health Effects Assessment.

EPA (U.S. Environmental Protection Agency). 1988b. *Estimating Exposures to 2,3,7,8-TCDD.* External review draft. Report No. EPA/600/6-88/005A. Washington, D.C.: Office of Health and Environmental Assessment. 368 pp.

EPA (U.S. Environmental Protection Agency). 1988c. *Superfund Exposure Assessment Manual.* Report No. EPA/540/1-88/001. Washington, D.C.: U.S. Environmental Protection Agency, Office of Remedial Response. 169 pp.

EPA (U.S. Environmental Protection Agency). 1989a. *Assessing Human Health Risks from Chemically Contaminated Fish and Shellfish: A Guidance Manual.* Report No. EPA-503/8-89-002. Washington, D.C.: U.S. Environmental Protection Agency, Office of Marine and Estuarine Protection and Office of Water Regulations and Standards. 136 pp.

EPA (U.S. Environmental Protection Agency). 1989b. *Exposure Factors Handbook.* Report No. EPA/600/8-89/043, prepared by Versar Inc., Springfield, Virginia. Washington, D.C.: U.S. Environmental Protection Agency, Office of Health and Environmental Assessment, Exposure Assessment Group. [282 pp.]

EPA (U.S. Environmental Protection Agency). 1989c. *Risk Assessment Guidance for Superfund. Volume I. Human Health Evaluation Manual (Part A). Interim Final.* Report No. EPA/540/1-89-002. Washington, D.C.: U.S. Environmental Protection Agency, Office of Emergency and Remedial Response. [294 pp.]

EPA (U.S. Environmental Protection Agency). 1990. Background document to the integrated risk assessment for dioxins and furans from chlorine bleaching in pulp and

paper mills. Washington, D.C.: Office of Pesticides and Toxic Substances, Exposure Evaluation Division, Exposure Assessment Branch.

Fingerhut, M.A., W.E. Halperin, D.A. Marlow, L.A. Piagitelli, P.A. Honchar, M.H. Sweeney, A.L. Griefe, P.A. Dill, K. Steenland, and A.J. Suruda. 1991. Cancer mortality in workers exposed to 2,3,7,8-tetrachlorodibenzo-*p*-dioxin. *N. Eng. J. Med.* 324:212-218.

Forsell, D. 1987. Proposal for a Dioxin Sampling Program for Johnston Island, with cover letter to Elliott Eng, Chief, Ecology Function, U.S. Air Force Occupational & Environmental Health Laboratory, Brooks AFB, Texas. September 15, 1987. U.S. Department of the Interior, Fish and Wildlife Service. 5 pages.

Frank, R., A.G. Carpentier, and D.L. Mackenzie. 1987. Monitoring for 2,4-residues in fish species resident in treated lakes in east-central Ontario 1977-80. *Environ. Monit. Assess.* 9:71-82.

Fries, G.F., and G.S. Marrow. 1975. Retention and excretion of 2,3,7,8-tetrachlorodibenzo-*p*-dioxin by rats. *J. Agric. Food Chem.* 23(2):265-269.

Gasiewicz, T.A., J.R. Olson, L.E. Geiger, and R.A. Neal. 1983a. Absorption, distribution and metabolism of 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD) in experimental animals. Pp. 495-525 in R.E. Tucker, A.L. Young, and A.P. Gray, Eds. *Human and Environmental Risk of Chlorinated Dioxins and Related Compounds.* New York: Plenum Press.

Gasiewicz, T.A., L. E. Geiger, G. Rucci, and R.A. Neal. 1983b. Distribution, excretion, and metabolism of 2,3,7,8-tetrachlorodibenzo-*p*-dioxin in C57BL/6J, DBA/2J, and B6D2F1/J mice. *Drug Metab. Dispos.* 11(5):397-403.

Geiger, L.E., and R.A. Neal. 1981. Mutagenicity testing of 2,3,7,8-tetrachlorodibenzo-*p*-dioxin in histidine auxotrophs of *Salmonella typhimurium*. *Toxicol. Appl. Pharmacol.* 59(1):125-129.

Gilbert, P., G. Saint-Ruf, F. Poncelet, and M. Mercier. 1980. Genetic effects of chlorinated anilines and azobenzenes on *Salmonella typhimurium*. *Arch. Environ. Contam. Toxicol.* 9(5):533-541.

Greig, J. 1984. Differences between skin and liver toxicity of 2,3,7,8-tetrachlorodibenzo-*p*-dioxin in mice. Pp. 391-397 in A. Poland and R.D. Kimbrough, Eds. *Biological Mechanisms of Dioxin Action.* Banbury Report 18. Cold Spring Harbor, New York: Cold Spring Harbor Laboratory.

Green, S., and F.S. Moreland. 1975. Cytogenetic evaluation of several dioxins in the rat. *Toxicol. Appl. Pharmacol.* 33:161.

Green, S., F.S. Morland, and C. Sheu. 1977. Cytogenic effect of 2,3,7,8-tetrachlorodibenzo-*p*-dioxin on rat bone marrow cells. *FDA By-Lines* 6:292.

Hay, A. 1982. Toxicology of dioxins. Pp. 41-47 in *The Chemical Scythe: Lessons of 2,4,5-T and Dioxin.* New York, London: Plenum Press.

Helsel, R.W., J.N. Casanova, and R.W. Thomas. 1987. *Thermal Desorption / Ultraviolet Photolysis Process Research, Test and Evaluation Performed at Johnston Island for the USAF Installation Restoration Program, Vol. 1: Technical Report.* Final Report (July 1986 — August 1986) to Engineering & Services Laboratory, U.S. Air Force Engineering & Services Center, Tyndall Air Force Base, Florida, by EG&G Idaho, Idaho Falls. ESL Report No. TR-87-37, Vol. 1. Available from National Technical Information Service (NTIS), Springfield, Virginia, as AD-A195-595.

Holmes and Narver, Inc. 1990. RCRA B Permit Outline. Draft document. Honolulu: Holmes and Narver, Inc., Energy Support Division.

Holmes and Narver, Inc. 1989. Preliminary Assessment of Johnston Atoll. Report to U.S. Defense Nuclear Agency. Honolulu: Holmes and Narver, Inc. 61 pp.

Hussain, S., L. Ehrenberg, G. Lofroth, and T. Gejvail. 1972. Mutagenic effects of TCDD on bacterial systems. *Ambio* 1:32-33.

IARC (International Agency for Research on Cancer). 1977. *IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Man, Vol. 15.* Lyon, France: World Health Organization, International Agency for Research on Cancer.

IARC (International Agency for Research on Cancer. 1987. 2,4-D. Pp233-236 in *Genetic and Related Effects: An Updating of Selected IARC Monographs from Volumes 1 to 42. IARC Monographs of the Evaluation of the Carcinogenic Risk of Chemicals to Humans. Supplement 6.* Lyon, France: World Health Organization, International Agency for Research on Cancer.

Irons, D.K., R.K. Kosaki, and J.D. Parrish. 1990. *Johnston Atoll Resource Survey: Final Report - Phase Six (21 Jul 89 - 20 Jul 90).* Report submitted by Hawaii Cooperative Fishery Research Unit, Honolulu, Hawaii, to U.S. Army Engineer District, Hononulu, Department of the Army, Fort Shafter, Hawaii. 59 pp.

Irwin, J. 1990. Internal memorandum to J. Tikvart, 19 June 1990. Research Triangle Park, N.C.: U.S. Environmental Protection Agency.

Jeffers, T.P. 1984. Letter to R.A. Capell, Bolling Air Force Base, on future use scenarios of the HO site. March 28, 1984. Defense Nuclear Agency. 4 pages.

Khera, K.S., and J.A. Ruddick. 1973. Polychlorodibenzo-*p*-dioxins: Perinatal effects and the dominant lethal test in Wistar rats. *Adv. Chem. Ser.* 12C:70-84.

Knutsen, A.P. 1984. Immunologic effects of TCDD exposure in humans. *Bull. Environ. Contam. Toxicol.* 33:673-681.

Kociba, R.J., D.G. Keyes, J.E. Beyer, and R.M. Carreon. 1978b. Toxicologic studies of 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD) in rats. *Toxicol. Occup. Med.* 4:281-287.

Kociba, R.J., D.G. Keyes, J.E. Beyer, R.M. Carreon, and P.J. Gehring. 1979. Long-term toxicologic studies of 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD) in laboratory animals. *Ann. N.Y. Acad. Sci.* 320:397-404.

Kociba, R.J., D.G. Keyes, J.E. Beyer, et al. 1978a. Results of a two-year chronic toxicity and oncogenicity study of 2,3,7,8-tetrachlorodibenzo-*p*-dioxin in rats. *Toxicol. Appl. Pharmacol.* 46(2):279-303.

Korte, C., and S.M. Jalal. 1982. 2,4-D induced clastogenicity and elevated rates of sister chromatid exchanges in cultured human lymphocytes. *J. Hered.* 73:224-226.

Krowke, R. 1986. Studies on distribution and embryotoxicity of different PCDD and PCDF in mice and marmosets. *Chemosphere* 15:2011-2022.

Linnainmaa, K. 1984. Induction of sister chromatid exchanges by the peroxisome proliferators, 2,4-D, MCPA, and clofibrate *in vivo* and *in vitro*. *Carcinogenesis* 5:703-707.

Loprieno, M., I. Sbrana, D. Rusciano, D. Lascialfari, and T. Lari. 1982. *In vivo* cytogenic studies on mice and rats exposed to 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD). Pp. 419-428 in O. Hutzinger et al., Eds. *Chlorinated Dioxins and Related Compounds: Impact on the Environment.* New York: Pergamon Press.

Lu, C.H., R.B. Baggs, D. Redmond, E.C. Henary, A. Schecter, and T.A. Gasiewicz. 1986. Toxicity and evidence for metabolic alterations in 2,3,7,8-tetrachlorodibenzo-*p*-dioxin-treated guinea pigs fed by total parenteral nutrition. *Toxicol. Appl. Pharmacol.* 84:439-453.

Lucier, G.W., R.C. Rumbaugh, Z. McCoy, R. Hass, D. Harvan, and P. Albro. 1986. Ingestion of soil contaminated with 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD) alters hepatic enzyme activities in rats. *Fundam. Appl. Toxicol.* 6:364-371.

Lundgren, K., M. Andries, C. Thompson, and G.W. Lucier. 1986. Dioxin treatment of rats results in increased *in vitro* induction of sister cchromatid exchanges by α-naphthoflavone: An animal model for human exposure to halogenated aromatics. *Toxicol. Appl. Pharmacol.* 85:189-195.

Luster, M.I., J.H. Dean, and G. A. Boorman. 1982. Altered immune functions in rodents treated with 2,3,7,8-tetrachlorodibenzo-*p*-dioxin, phorbol-12-myristate-13-acetate, and benzo[a]pyrene. Pp. 199-213 in *Environmental Factors in Human Growth and Development.* Banbury Report 11. Cold Spring Harbor, New York: Cold Spring Harbor Laboratory.

Magnusson, J., C. Ramel, and A. Eriksson. 1977. Mutagenic effects of chlorinated phenoxyacetic acids in *Drosophila melanogaster. Hereditas* 87:121-123.

Marple, L., R. Brunck, and L. Throop. 1986. Water solubility of 2,3,7,8-tetrachlorodibenzo-*p*-dioxin. *Environ. Sci. Technol.* 20:180-182.

Mason, G., and S. Safe. 1986a. Synthesis, biologic and toxic effects of the major 2,3,7,8-dichlorobenzo-*p*-dioxin metabolites in the rat. *Toxicology* 41:153-159.

Mason, G., and S. Safe. 1986b. Synthesis, biologic and toxic properties of 2,3,7,8-tetrachlorodibenzo-*p*-dioxin. *Chemosphere* 15:2081-2083.

McCann, J. 1978. Unpublished study cited in EPA, 1985.

McConnell, E.E., G.W. Lucier, R.C. Rumbaugh, et al. 1984. Dioxin in soil: Bioavailability after ingestion by rats and guinea pigs. *Science* 223:1077-1079.

McConnell, E.E. 1985. Comparitive toxicity of PCBs and related compounds in various species of animals. *Environ. Health Perspect.* 60:29-33.

McNulty, W. 1985. Toxicity and fetotoxicity of TCDD, TCDF, and PCB isomers in Rhesus macaques (*Macaca mulatta*). *Environ. Health Perspect.* 60:77-88.

McNulty, W. 1984. Fetotoxicity of 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD) for Rhesus macaques (*Macaca mulatta*). *Am. J. Primatol.* 6:41-47.

Mehrle, P.M., D.R. Buckler, E.E. Little, L.M. Smith, J.D. Petty, P.H. Peterman, D.L. Stalling, G.M. DeGraeve, J.J. Coyle, and W.J. Adams. 1988. Toxicity and bioconcentration of 2,3,7,8-tetrachlorodibenzo-*p*-dioxin and 2,3,7,8-tetrachlorodibenzofuran in rainbow trout. *Environ. Toxicol. Chem.* 7:47-62.

Meyne, J., D.C. Allison, K. Bose, S.W. Jordan, P.F. Ridolpho, and J. Smith. 1985. Hepatotoxic doses of dioxin do not damage mouse bone marrow chromosomes. *Mutat. Res.* 157:63-69.

Migdalski, E.C., and G.S. Fichter. 1976. *The Fresh and Salt Water Fishes of the World.* New York: Alfred A. Knopf.

Moore, J.A., B.N. Gupta, J.G. Zinkl, and J.G. Yos. 1973. Postnatal effects of maternal exposure to 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD). *Environ. Health Perspect.* 5:81-85.

Mortelmans, K., S. Haworth, W. Speck, and E. Zeiger. 1984. Mutagenicity testing of Agent Orange and related chemicals. *Toxicol. Appl. Pharmacol.* 75:137-146.

Murray, F.J., F.A. Smith, K.D. Nitschke, C.G. Humiston, R.J. Kociba, and B.A. Schwetz. 1979. Three-generation reproduction study of rats given 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD) in the diet. *Toxicol. Appl. Pharmacol.* 50:241-251.

Mustonen, R., J. Kangas, P. Vuojalahti, and K. Linnainmaa. 1986. Effects of phenoxyacetic acids on the induction of chromosome aberrations *in vitro* and *in vivo*. *Mutagenesis* 1:241-245.

Neubert, D., and I. Dillmann. 1972. Embryotoxic effects in mice treated with 2,4,5-trichlorophenoxyacetic acid and 2,3,7,8-tetrachlorodibenzo-*p*-dioxin. *Arch. Pharmacol.* 272(3):243-264.

Nishimura, N., H. Nishimura, and H. Oshima. 1982. Survey on mutagenicity of pesticides by the salmonella-microsome test. *J. Aichi Med. Univ. Assoc.* 10:305-312.

Nolan, R.J., F.A. Smith, and J.G. Hefner. 1979. Elimination and tissue distribution of 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD) in female guinea pigs following an single oral dose. *Toxicol. Appl. Pharmacol.* 48(1):A162.

NTP (National Toxicology Program). 1982a. *Bioassay of 2,3,7,8-Tetrachlorodibenzo-p-Dioxin for Possible Carcinogenicity (Dermal Study).* DHHS Publ. No. (NIH) 82-1757. Research Triangle Park, N.C.: National Toxicology Program.

NTP (National Toxicology Program). 1982b. *Bioassay of 2,3,7,8-Tetrachlorodibenzo-p-Dioxin for Possible Carcinogenicity (Gavage Study).* DHHS Publ. No. (NIH) 82-1765. Research Triangle Park, N.C.: National Toxicology Program.

Olson, J.R., and V.J. Wroblewski. 1985. Metabolism of 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD) in isolated hepatocytes from guinea pigs and rats. *Chemosphere* 14:979-982.

Olson, J.R., T.A. Gasiewicz, and R.A. Neal. 1980. Tissue distribution, excretion, and metabolism of 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD) in the Golden Syrian hamster. *Toxicol. Appl. Pharmacol.* 56(1):78-85.

Paustenbach, D.J., H.P. Shu, and F.J. Murray. 1986. A critical examination of assumptions used in risk assessments of dioxin contaminated soil. *Reg. Toxicol. Pharmacol.* 6:284-307.

Peterson, R.E., M.D. Seefeld, B.J. Christian, C.L. Potter, C.K. Kelling, and R.E. Keesey. 1984. The wasting syndrome in 2,3,7,8-tetrachlorodibenzo-*p*-dioxin toxicity: Basic features and their interpretation. Pp. 291-308 in A. Poland and R.D. Kimbrough, Eds. *Biological Mechanisms of Dioxin Action.* Banbury Report 18. Cold Spring Harbor, New York: Cold Spring Harbor Laboratory.

Pilinskaya, M.A. 1974. Cytogenic effect of the herbicide 2,4-D on human and animal chromosomes. *Cytol. Genet.* 8:6-10.

Piper, W.N., R.Q. Rose, and P.J. Gehring. 1973. Excretion and tissue distribution of 2,3,7,8-tetrachlorodibenzo-*p*-dioxin in the rat. *Environ. Health Perspect.* 5:241-244.

Poiger, H., and C. Schlatter. 1986. Pharmacokinetics of 2,3,7,8-TCDD in man. *Chemosphere* 15:9-12.

Poiger, H., and C. Schlatter. 1985. Influence of phenobarbitol and TCDD on the hepatic metabolism of TCDD in the dog. *Experientia* 41:376-378.

Poiger, H., and C. Schlatter. 1980. Influence of solvents and adsorbents on dermal and intestinal absorption of TCDD. *Food Cosmet. Toxicol.* 18(5):477-481.

Poiger, H., H.-R. Buser, H. Weber, U. Zweifel, and C. Schlatter. 1982. Structure elucidation of mammalian 2,3,7,8-tetrachlorodibenzo-p-dioxin metabolites. a*Experientia* (Basel) 38(4):484-486.

Poland, A., and E. Glover. 1980. 2,3,7,8-Tetrachlorodibenzo-*p*-dioxin segregation of toxicity with the Ah locus. *Mol. Pharmacol.* 17(1):86-94.

Poland, A., D. Palen, and E. Glover. 1982. Tumor promotion by TCDD in skin of HRS/J mice. *Nature* 300(5389):271-273.

Puhvel, S.M., M. Sakamoto, D.C. Ertl, and R.M. Reisner. 1982. Hairless mice as models for chloracne: A study of cutaneous changes induced by topical application of established chloracnegens. *Toxicol. Appl. Pharmacol.* 64:492-503.

Ramel, C., and J. Magnusson. 1979. Chemical induction of nondisjunction in drosophila. *Environ. Health Perspect.* 31:59-66.

Randall, J.E. 1961. A contribution to the biology of the convict surgeonfish of the Hawaiian Islands, *Acanthurus triostegus sandvicensis*. *Pacif. Sci.* 15:215-272.

Rasmuson, B., and H. Svahlin. 1978. Mutagenicity tests of 2,4-dichlorophenoxyacetic acid and 2,4,5-trichlorophenoxyacetic acid in genetically stable and unstable strains of *Drosophila melanogaster*. *Ecol. Bull.* (Stockholm) 27:190-192.

Rogers, A.M., M.E. Anderson, and K.C. Back. 1982. Mutagenicity of 2,3,7,8-tetrachlorodibenzo-*p*-dioxin and perfluoro-*n*-decanoic acid in L5178y mouse lymphoma cells. *Mutat. Res.* 105:445-449.

Ryan, J.J., P.Y. Lau, J.C. Pillon, D. Lewis, H.A. McLeod, and A. Gervais. 1984. Incidence and levels of 2,3,7,8-tetrachlorodibenzo-*p*-dioxin in Lake Ontario commercial fish. *Environ. Sci. Technol.* 18:719-721.

Sawahata, T., J.R. Olson, and R.A. Neal. 1982. Identification of metabolites of 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD) formed on incubation with isolated rat hepatocytes. *Biochem. Biophys. Res. Commun.* 105(1):341-346.

Schwetz, B.A., J.M. Norris, G.L. Sparschu, V.K. Rowe, P.J. Gehring, J.L. Emerson, and C.G. Gerbig. 1973. Toxicology of chlorinated dibenzo-*p*-dioxins. *Environ. Health Perspect.* 5:87-99.

Seiler, J.P. 1973. A survey on the mutagenicity of various pesticides. *Experientia* 29:622-623.

Siebert, D., and E. Lemperle. 1974. Genetic effects of herbicides: Induction of mitotic gene conversion in *Saccharomyces cerevisiae*. *Mutat. Res.* 22:111-120.

Skidmore, E.L., and N.P. Woodruff. 1968. *Wind Erosion Forces in the United States and Their Use in Predicting Soil Loss.* Agriculture Handbook No. 346. Washington, D.C.: U.S. Department of Agriculture, Agricultural Research Service, in cooperation with Kansas Agricultural Experiment Station. 42 pp.

Slaga, T.J., and S. Nesnow. 1985. Sencar mouse skin tumorigenesis. Pp. 230-250 in H.A. Millman and E.K. Weisburger, Eds. *Handbook of Carcinogen Testing.* Park Ridge, New Jersey: Noyes.

Smith, F.A., B.A. Schwetz, and K.D. Nitschke. 1976. Teratogenicity of 2,3,7,8-tetrachlorodibenzo-*p*-dioxin in CF-1 mice. *Toxicol. Appl. Pharmacol.* 38(3):517-523.

Sparschu, G.L., Jr., F.L. Dunn, Jr., and V.K. Rowe, Jr. 1971a. Study of the teratogenicity of 2,3,7,8-tetrachlorodibenzo-*p*-dioxin in the rat. *Food Cosmet. Toxicol.* 9:405-412.

Sparschu, G.L., F.L. Dunn, R.W. Lisowe, and V.K. Rowe. 1971b. Effects of high levels of 2,4,5-trichlorophenoxyacetic acid on fetal development in the rat. *Food Cosmet. Toxicol.* 9:527-530.

Suskind, R.R. 1985. Chloracne, "the hallmark of dioxin intoxication." *Scand. J. Work Environ. Health* 11:165-171.

Taylor, J.S. 1979. Environmental chloracne: Update and overview. *Ann. N.Y. Acad. Sci.* 320:295-307.

Thomas, T.J., D.P. Brown, J. Harrington, T. Stanford, L. Taft, and B.W. Vigon. 1978. *Land Based Environmental Monitoring at Johnston Island — Disposal of Herbicide Orange.* Report No. USAF OEHL TR-78-87 to U.S. Air Force Occupational and Environmental Health Laboratory, Brooks AFB, Texas. Columbus, Ohio: Battelle Columbus Laboratories. 323 pp.

Toth, K., E. Olah, S. Somfai-Relle, and J. Sugar. 1984. Effect of herbicide 2,4,5-trichlorophenoxyethanol (TCPE) containing dioxin on mutation and induction of sister chromatid exchanges. *Carcinogenesis* 5:1725-1728.

Turner, J.N., and D.N. Collins. 1983. Liver morphology in guinea pigs administered either pyrolysis products of a polychlorinated biphenyl transformer fluid or 2,3,7,8-tetrachlorodibenzo-*p*-dioxin. *Toxicol. Appl. Pharmacol.* 67:417-429.

U.S. Air Force. 1974. *Final Environmental Statement on Disposition of Orange Herbicide by Incineration.* Washington, D.C.: U.S. Air Force, Office of the Special Assistant for Environmental Quality. 150 pp.+ appendices.

U.S. Fish & Wildlife Service. 1987. *Dioxin (TCDD) and Furan (TCDF) Toxicity to Rainbow Trout.* Res. Info. Bull. No. 87-55. Washington, D.C.: U.S. Department of Agriculture, Fish & Wildlife Service. 3 pp.

Van Miller, J.P., J.J. Lalich, and J.R. Allen, Jr. 1977a. Increased incidence of neoplasms in rats exposed to low levels of 2,3,7,8-tetrachlorodibenzo-*p*-dioxin. *Chemosphere* 6(9):537-544.

Van Miller, J.P., J.J. Lalich, and J.R. Allen, Jr. 1977b. Increased incidence of neoplasms in rats exposed to low levels of 2,3,7,8-tetrachlorodibenzo-*p*-dioxin. *Chemosphere* 6(10):625-632.

Vogel, E., and J.L.R. Chandler. 1974. Mutagenicity testing of cyclamate and some pesticides in *Drosophila melanogaster*. *Experientia* 30:621-623.

Walker, C.H. 1990. Persistent pollutants in fish-eating sea birds — Bioaccumulation, metabolism and effects. *Aquat. Toxicol.* 17:293-324.

Walsh, W.J., III. 1984. Aspects of Nocturnal Shelter, Habitat Space, and Juvenile Recruitment in Hawaiian Coral Reef Fishes. Ph.D. Dissertation. Honolulu: University of Hawaii, Graduate Division of Zoology. 475 pp.

Weber, H., and L.S. Birnbaum. 1985. 2,3,7,8-Tetrachlorodibenzo-*p*-dioxin (TCDD) and 2,3,7,8-tetrachlorodibenzofuran (TCDF) in pregnant C57BL/6N mice: Distribution to the embryo and excretion. *Arch. Toxicol.* 57:159-162.

Wisk, J.D., and K.R. Cooper. 1990. The stage-specific toxicity of 2,3,7,8-tetrachlorodibenzo-*p*-dioxin in embryos of the Japanese medaka (*Oryzias latipes*). *Environ. Toxicol. Chem.* 9:1159-1169.

Wroblewski, V.J., and J.R. Olson. 1985. Hepatic metabolism of 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD) in the rat and guinea pig. *Toxicol. Appl. Pharmacol.* 81:231-240.

Young, A.L. and L.G. Cockerham. 1985. Fate of TCDD in field ecosystems. Pp. 153-171 in M.A. Kamrin and P.W. Rodgers, Eds. *Dioxin in the Environment.* New York: Hemisphere Publishing Corp. (as cited in Eisler, 1985).

Zeiger, E. 1983. Memorandum to Dr. E.E. McConnell on the results of test performed for the Environmental Mutagenesis Development Program. Research Triangle Park, N.C.: National Toxicology Program/National Institute for Environmental Health Sciences.

Zetterberg, G. 1978. Genetic effects of phenoxy acids on microorganisms. *Ecol. Bull.* (Stockholm) 27:193-204.

Zetterberg, G., L. Busk, R. Elovson, I. Starec-Nordenhammar, and H. Ryttman. 1977. The influence of pH on the effects of 2,4-D (2,4-dichlorophenoxyacetic acid Na salt) on *Saccharomyces cerevisiae* and *Salmonella typhimurium*. *Mutat. Res.* 42:3-18.

Zimmering, S., J.M. Mason, R. Valencia, and R.C. Woodruff. 1985. Chemical mutagenesis testing in drosophila. II. Results of 20 coded compounds tested for the National Toxicology Program. *Environ. Mutag.* 7:87-100.

Appendix A

JOHNSTON ATOLL RESOURCE SURVEY
FINAL REPORT - PHASE SIX
(21 JUL 89 - 20 JUL 90)

SUBMITTED BY:

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DATE SUBMITTED: 20 JUL 90

JOHNSTON ATOLL RESOURCE SURVEY
FINAL REPORT - PHASE SIX
(21 JUL 89 - 20 JUL 90)

INTRODUCTION

Construction of the Johnston Atoll Chemical Agent Disposal System (JACADS) project has been completed, and operations began in June 1990. The potential for adverse environmental effects is a concern, which has been addressed in environmental impact statements (U.S. Army Corps of Engineers 1983, 1985). This concern has led to a number of studies of the atoll's surrounding environment and biota (Applied Eco-Tech Services, Inc. 1983; Balazs 1984; Irons et al. 1984; Lobel 1984, 1985; Agegian and Abbott 1985; Dee et al. 1985; Keating 1985; Randall et al. 1985; Irons et al. 1986; Irons et al. 1987, 1988, 1989). There have been several previous studies of elements of the Johnston Atoll lagoon flora and fauna (Smith and Swain 1882; Edmondson et al. 1925; Fowler and Ball 1925; Clark 1949; Schultz et al. 1953; Halstead and Bunker 1954; Gosline 1955; Banner and Helfrich 1964; Moul 1964; Brock et al. 1965, 1966; Buggeln and Tsuda 1966; Jones 1968; Brock 1972, 1982; Bailey-Brock 1975; Amerson and Shelton 1976; Jokiel 1976; Maragos and Jokiel 1986). A systematic survey of the nature and distribution of the living aquatic resources is of particular concern because of the status of Johnston Atoll as a National Wildlife Refuge.

The first portion of the initial study (Irons et al. 1984) was designed to characterize, describe and evaluate the shallow-water ecosystem of the atoll as a whole, in an attempt to better assess its environment and resources. This included identifying the zones or "ecotypes" (Fig. 10), based on physical and biological similarities, that appeared distinctive within the atoll ecosystem (Irons et al. 1984).

The second portion of the initial study (Dee et al. 1985) had two distinct but related objectives: 1) detailed resource measurement and status monitoring, and 2) assessment of the nature and level of harvest. Subsequent work during Phase Two (Irons et al. 1986), Phase Three (Irons et al. 1987), Phase Four (Irons et al. 1988), Phase Five (Irons et al. 1989), and the present phase (Phase Six) have continued with the same objectives. The detailed resource measurement and status monitoring is intended to obtain more complete and quantitative abundance, distribution, and population characteristic data for the non-cryptic macrofauna within a representative set of long-term monitoring stations. Using standardized methods, the resources at the long-term stations have been monitored periodically to detect differences in the resource populations as JACADS progresses.

To the extent that spatial patterns of fishing/collecting activity permit, it is desirable to maintain a pair of physically and ecologically similar stations, one with a fairly high present level of harvest and one with a low level. Differences over time in the unharvested monitoring station will reflect changes unrelated to harvest - either natural variability or changes

1 (pages 2-4 not included)

abundance and distribution of cryptic species, such as soldierfish and bigeyes. These were conducted by searching all possible hiding places where cryptic species may be found throughout two areas of 900 m^2 each, within a station.

The overall area characterization consisted of a quantitative estimate of percent algal and coral cover (corals by species), invertebrate abundances, and physical characteristics of the station area. Overall characterization methods were basically as in Irons et al. (1984) except that a numerical value was assigned for bottom coverage of most sessile forms (Appendix A).

To assess the fishery at Johnston Atoll, two methods were used: 1) fishermen's catch reporting, and 2) creel census. The catch reporting program was started in February 1984, and has been ongoing throughout the project whenever fishing was permitted. Boxes containing catch report forms (Appendix B, Fig. 1) were placed at the six most frequently fished locations on Johnston Island: port control, Hama point, Hashi's shack, the east and west ends of the main pier, and the boathouse (between port control and the main pier) (Fig. 1). Catch reports provided information on species and numbers of animals caught and/or collected: date, time, and location caught/collected; amount and types of gear used; hours spent fishing; and identity of fishermen. A catch report was requested each time anyone did any kind of fishing and/or collecting, even if there was no catch. The catch report format was designed and the report boxes were located and maintained so as to make the reporting process as simple and painless as possible for all fishermen. Consistent and accurate catch reporting was constantly stressed by Unit project staff. Serious declines in voluntary catch reporting during the report year ending 1987 resulted in the implementation of a new form (Appendix B, Fig. 2) combining recreational boat sign-out procedures with a mandatory catch report to be filled out upon the fisherman's return. A serious decline in JI shoreline catch reporting during the report year ending 1989 made this shoreline information unusable. Subsequently, Unit personnel and Island management personnel have been unable to determine a satisfactory method of enforcing mandatory reporting of JI shore catch. As a result, no data for JI shore catch will be reported. However, Unit personnel continue to encourage JI shore catch reporting and continue to collect the completed JI shore catch forms.

Creel census was performed by the Unit project staff on catches made by fishermen. It consisted of recording pertinent data, such as numbers of each species caught, weights, lengths, and sex (if discernible) of specimens, date, gear used, and the names of fishermen. Catches involving the use of boats were censused at the boathouse. Due to the work schedule of Johnston Atoll people, approximately 70% of all fishing occurs on Sundays. For this reason creel census was routinely conducted only on Sundays. This allowed a significant portion of the harvest to be examined with minimum time and effort.

5 (pages 6-24 not included)

considerably reduced the negative trend in "mean total number per census" (Table 3). By extension, variability of recruitment occurring for a good many species might contribute heavily to the overall population pattern observed.

All the community analyses combined showed no clear seasonal variations in the fish communities at the monitoring stations. However, there were differences in the fish communities between stations. Stations P3 and P7, which are both located in different habitat types from Stations P1, P5, and P6, have very different fish communities. Station P3 has a significantly lower mean number (as determined by paired t-tests) of total individuals observed on the fish transect censuses when compared to Stations P1, P5, and P6. In some previous phases of this study and in the present phase, Station P7 has had a significantly higher number (as determined by paired t-tests) of Ctenochaetus strigosus and Acanthurus nigroris juveniles than any other station. Station P5 showed no significant differences from Stations P1 and P6 in the t-tests and dendrograms, but it is the only place where the whitecheek surgeonfish (Acanthurus glaucopareius) is seen.

In addition, paired t-tests were performed on some species that are often important in the catch (i.e., Myripristis amaenus, the doublebar goatfish (Pseudupeneus bifasciatus), the manybar goatfish (P. multifasciatus), the blue goatfish (P. cyclostomus), the Samoan goatfish (Mulloides flavolineatus), the rudderfish (Kyphosus vaigiensis), the blue jack (Caranx melampygus), the spectacled parrotfish (Scarus perspicillatus), and Acanthurus triostegus) seen at Stations P5 and P6. These results also showed no significant differences between these two stations. The lack of significant differences between these stations, with similar habitats and substantially different fishing effort, is consistent with the harvest assessment results in suggesting that there is no significant impact on the fish communities at Johnston Atoll from the present level of fishing.

THE FISHERY

General Characteristics

All fishing at Johnston Atoll (JA) is supposedly for recreational purposes. The majority of the fishing activity and a very large fraction of the finfish catch is due to long-term "residents" - almost all employees of Holmes and Narver, the prime contractor for JA operations. These fishermen fish mostly for enjoyment, to add fresh fish to their diet, and to accumulate fish to freeze and carry home when they take home leave from JA at infrequent intervals. The remainder of the catch is due to "transients" - personnel stationed for one to two years at JA, such as military personnel, and the employees of various JACADS contractors. As a rough estimate, 350 boxes of frozen fish are "exported" annually for home leave. During years of good deep-sea fishing conditions, a majority of these boxes may contain deep sea fish, primarily wahoo (Acanthocybium solandri). Most of the "exported" fish terminates in Honolulu. There is no definite

information as to how it is disposed of. While there are no subsistence implications to the consumption of fish locally at JA, eating fresh caught fish is clearly an important recreational and social activity for a number of residents. There is apparently little waste of the total fish catch. Many fishermen give fish to nonfishermen to take home on leave. There is no monitoring or control of "export". Coral and gastropods are taken by both residents and transients. Disposition of these and most other invertebrate species appears to be for personal collections, or they are used as gifts for family and friends. The following is a brief description of the nature of the fishery for some of the species (fish and invertebrates) that were major items in the catch when the study began.

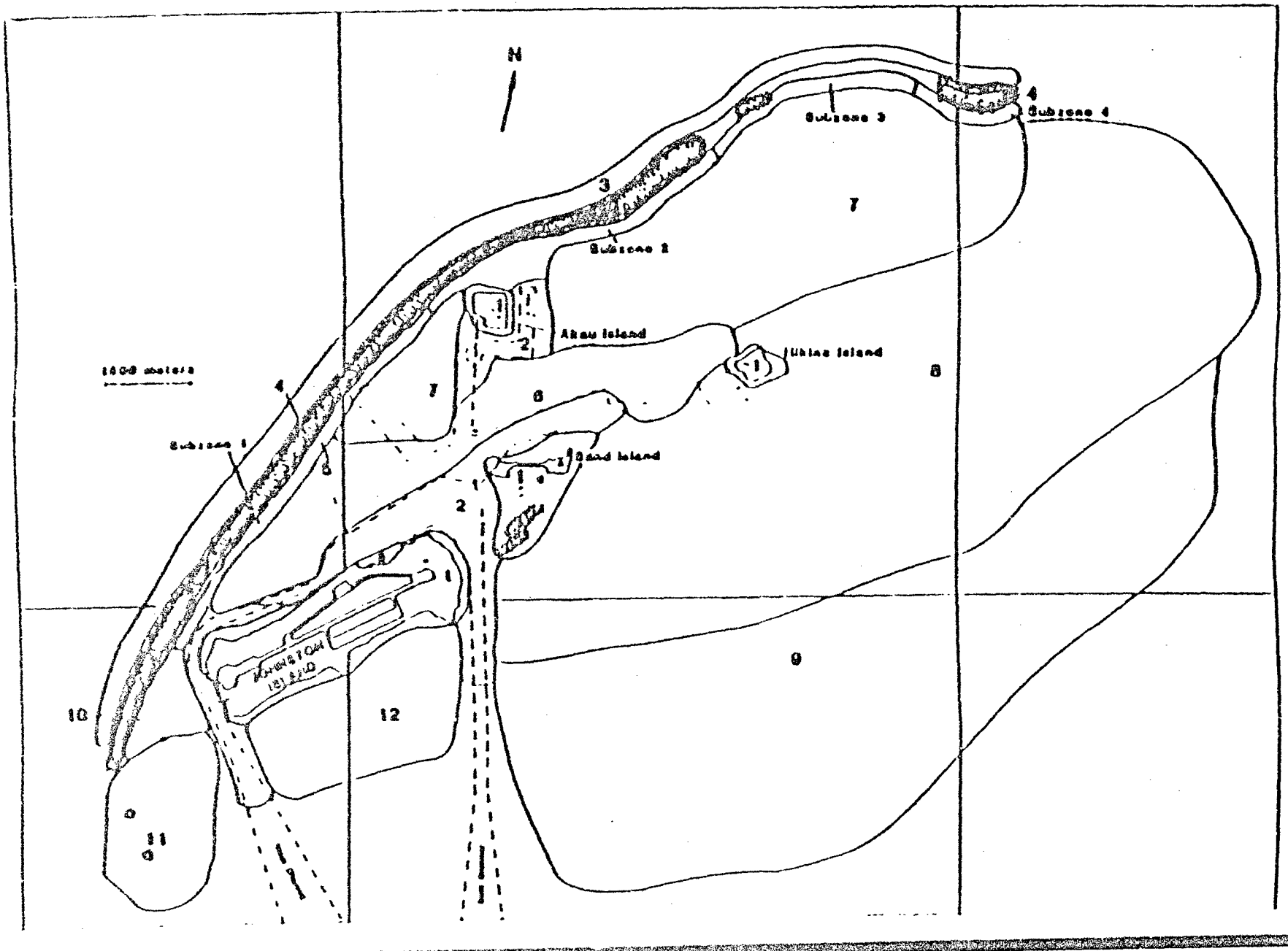
Myripristis amaenus, the most common of the "menpachi", constitutes the largest catch in numbers of all fish species at JA. Large numbers of this soldierfish are taken by fishermen throughout the year. Prime areas for nighttime line fishing for menpachi include Hama point and Red Hat seawall on Johnston Island (JI), as well as at the Sand Island pier (Fig. 1). During the day, large numbers of menpachi are taken by spear throughout Zones 5 and 10 (Fig. 10), with most taken in the vicinity of Station P5. No menpachi are taken by net. Menpachi fishing, like most fishing at JA, is done almost exclusively by residents. Most menpachi taken is used for local get-togethers, or is frozen by fishermen for home leave export.

Priacanthus cruentatus or "aweoweo" is one of the most prized fish species at JA. Bigeyes are taken at night by line from several locations on Johnston Island - main pier, Hama point, Red Hat seawall - as well as from the Sand Island pier. During the day, they are occasionally taken by spear throughout Zone 5, with most of these taken in the vicinity of Station P5. No aweoweo are taken by net. Aweoweo fishing is done almost exclusively by residents. They are taken in small numbers most of the year. Occasionally (only a few times a year, usually in January and February), they are taken in large numbers. When this occurs, many fishermen go to the main pier at night to fish exclusively for aweoweo, which usually bite heavily for one or two days. Aweoweo are usually frozen for home leave export.

Kuhlia marginata or "aholehole" and Chaenomugil leuciscus or "uouoa" are taken almost exclusively by throw net. Schools of these flagtail and mullet frequent the shallow rubble flats around the shorelines of Akau, Hikina, and Sand Islands, and occasionally Johnston Island. There are a few regular throw net fishermen (all residents) who take these species in large numbers. Thus small changes in the fishing activity of these fishermen can produce wide fluctuations in the annual catch figures for these species. They are either eaten locally, given to others, or frozen for home leave export.

Kyphosus vaigiensis or "nenue" are taken by line and spear mostly from JI. Rudderfish taken by residents are usually consumed; those taken by transients are considered incidental catch and are either used as bait or are returned alive.

Mulloidichthys flavolineatus or "weke" are taken using all three gear types - line, spear, and net - from shallows around all islands and occasionally from Zone 5. During the summer months,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juvenile weke or "oama" are taken in large numbers by throw net from the shallows around the islands. Approximately 50% of all weke taken are oama. Residents, mostly throw netters, take the majority of weke, with transients taking small numbers by line fishing. This goatfish is eaten locally or given away for home leave export. Juveniles are often collected for use as bait.

Pseudupeneus bifasciatus or "moano papa" is a prized fish species at JA and is taken almost exclusively by residents by line fishing or spearing. Line fishing for moano papa is done by boat along the channel edges, primarily the north edge of the main channel. This goatfish is taken by spear throughout Zones 5 and 10, mostly from the vicinity of Station P5. Moano papa are usually frozen by fishermen for their own home leave export.

Pseudupeneus cyclostomus or "moano kea" are highly prized at JA. A large part of the catch is taken by residents using lines or spears. Most moano kea are taken along the edges of the main channel; many are also taken from rubble shoreline areas around Johnston Island. This goatfish is speared throughout Zones 5 and 10, with most taken in the vicinity of Station P5. Moano kea are usually frozen for home leave export.

Pseudupeneus multifasciatus or "moano" are taken almost exclusively by residents, by line fishing along the channel edges, with some also taken from Johnston and Sand Island shorelines. This goatfish is speared throughout Zones 5 and 10, with most taken in the vicinity of Station P5. Most moano are frozen for home leave export.

Caranx melampygus and Forskal's jack (Carangoides orthogrammus), known locally as "papio" (those under 10 lbs.) or "ulua" (those over 10 lbs.), are taken mostly by residents and some transients by line fishing along channel edges, or from several locations on Johnston Island, as well as from Sand and East Island piers. These jacks are only occasionally taken by spear, usually in the vicinity of Station P5. Most papio are frozen for home leave export.

Scarus perspicillatus or "uhu" are taken predominantly by residents using spears. This parrotfish is speared throughout Zones 5 and 10, with some also taken around Sand and Johnston Island shorelines. Uhu are prized by fishermen and are usually frozen for home leave export.

Acanthurus triostegus or "manini" are taken exclusively by residents using throw nets, or spears. About 40% of the total catch is taken by throw nets around the shallows of all islands. Spearing, which accounts for the remaining 60% of the total catch, is done throughout Zones 5 and 10, with most fish taken in the vicinity of Station P5. This surgeonfish is usually eaten at local get-togethers or given to others for home leave export.

Ctenochaetus strigosus or "kole" are taken almost exclusively by residents. Practically all are taken by spear from Zones 5 and 10, primarily in the vicinity of Station P5. This surgeonfish is also eaten locally or is given to others to freeze for home leave export.

Acropora cytherea or "tabletop coral" is frequently collected by hand by both residents and transients. Most A. cytherea colonies collected are ~15-30 cm in diameter. This coral is commonly used for making coral trophy boxes. Most A.

cytherea is taken in the vicinity of Station P5, but it is also taken from other locations throughout the lagoon. Other species of coral, including Pocillopora sp. and Millepora are taken in much smaller numbers for similar purposes.

The red coral (Distichopora sp.) is prized by collectors and is primarily used for decorative purposes such as coral boxes. It is taken by hand throughout Zone 4 by both residents and transients. It is somewhat scarce in various sections of Zone 4, especially from Station P5 northward toward Station P6 (Irons et al. 1984), but is abundant in areas inaccessible to collectors (outside the barrier reef).

The mushroom coral (Fungia [P.] scutaria), the sea urchin (Echinothrix calamaris/diadema), and various gastropods such as augers, cones and small cowries occur in Zone 5 and other locations throughout the lagoon. These are collected by hand by both residents and transients, and are used for decorative purposes.

The tiger cowrie (Cypraea tigris) is prized by residents and transients and is used for decorative purposes. C. tigris is taken by hand throughout Zone 4, mostly from the reef-top around and between Stations P5 and P6. It is somewhat scarce and scattered throughout Zone 4.

Octopus sp. or "tako" are prized by residents and are occasionally found in the rubble of shallows along the shorelines of all four islands. Tako are speared or hand collected and are usually eaten locally.

The spiny lobster (Panulirus penicillatus) is taken by hand exclusively from Zone 4 and is highly prized by both residents and transients. Any P. penicillatus taken are usually eaten locally.

The crab (Grapsus sp.) is collected by hand and eaten exclusively by residents. It is found along stretches of all the island shorelines. Only a few people occasionally collect this crab.

Many other fish and some invertebrate species produce small catches of some minor recreational value.

Correction for Underreporting of Catch

The basic quantitative data used to estimate catch came from fishermen's catch reports. There was substantial underreporting, and adjustments were made in an attempt to obtain a reasonable approximation of the annual catch. Fishing involving use of boats includes all fishing done on and around Akau, Hikina, and Sand Island, as well as all fishing done directly from boats. Underreporting of fishing done by boat was estimated by counting the catch report forms that were turned in not completed by fishermen who used boats. (Catch reports are now located on the back of the boathouse "boat check-out" records (Appendix B, Fig. 2) that are filled out for the recreation department each time a boat is used). Since it is mandatory for everyone who checks out a boat to fill out the catch form on the back, a single estimate of underreporting was calculated for all species caught using boats. During the current report year, 77% of all boats that

were checked out for fishing reported on catch. Thus, we estimated that 77% of the catch of each species was reported. Catch data recorded from JI shore fishing were neither analyzed nor reported because there is no means for estimating underreporting, which is known to be substantial.

Annual Catch and Effort

The total boat catch of each species, for the period Jun 89 to May 90 (year ending 1990), corrected for underreporting, is shown in Table 5, including major gear types used and primary location(s) of catch. The first 13 species listed were those that initially provided the largest catches. For historical reasons, this group continues to be referred to as the "major catch species", and most of these species have provided important landings in most years of the study. In the last few years, catches of Kyphosus vaigiensis have been very low (zero by boat in the current year), and catches of Carangoides orthogrammus, Selar crumenophthalmus, and Decapterus macarellus have been as high as many of the "major catch species".

Table 5. Estimated total annual boat catch of all species reported in the JA fishery, including major types of fishing gear and locations of catch, for Jun 89 - May 90.

FISH SPECIES[1]	TOTAL NUMBER CAUGHT	MAJOR GEAR TYPE[2]	PRIMARY LOCATION(S)[3]								
			AI	HI	SI	PI	PS	Z6	Z10	CH	LA
Myripristis [illegible]	3362	LI SP		HI		227	2047		690		375
Ctenochaetus strigosus	1201	SP	596	HI		PI			161		385
Acanthurus triostegus	828	SP HT	112	521					172		LA
Chaetodon [illegible]gii leucisecus	509	HT	112	392							
Kuhlia marginata	225	LI HT	65	160							
Caranx [illegible]	186	LI SP	24	110	SI					CH	LA
Pseudocaranx cyclostomus	129	LI SP	AI	73	SI					CH	
Mullioides flavolineatus	123	LI SP HT	56	69	SI						
Scarus perspicillatus	83	SP	56	18					210		18
Pristipomoides [illegible]	79	SP	20						52		LA
Pseudocaranx bifasciatus	66	LI SP	56						210	CH	LA
Pseudocaranx multifasciatus	58	LI SP	AI	HI	SI					CH	LA
Kyphosus vaigiensis	0	.									
Selar crumenophthalmus	593	LI HT	31	566							
Carangoides orthogrammus	157	LI SP	30	102	SI					CH	LA
Acanthurus [illegible]	26	LI		HI							
Acanthurus nigricans	16	SP	AI								
Balistes sp.	13	LI									LA
Aphareus furca	5	LI		HI							
Aulostomus chinensis	2	HT		HI							
Carcharhinus [illegible]	2	LI		HI							
Scomberoides lysan	2	LI		HI							

Table 5 (continued).

BENTHIC SPECIES[1]	TOTAL NUMBER CAUGHT	MAJOR GEAR TYPE[2]	PRIMARY LOCATION(S)[3]								
			AI	MI	SI	P1	P5	Z6	Z10	CH	LA
Corals											
Acropora cytherea	456	HC					P5				
Diatichopora sp.	402	HC						Z6			
Fungia scutaria	135	HC	AI								LA
Acropora valida	108	HC					P5				
Millepora tenera	4	HC							Z10		
Non-sessile invertebrates											
Octopus sp.	121	SP HC	AI	MI					Z10		
Penutirus penicillatus	74	HC						Z6			
Linckia sp.	14	HC									LA
Gracosus sp.	8	HC	AI								
Cypraea tigris	57	HC						Z6			
Terebra sp.	36	HC	AI		SI						
Conus sp.	8	HC						Z6			
Charonia tritonis	7	HC						Z6	Z10		
Cypraea sp.	5	HC									LA

[1] See Appendix A for common names.

[2] Gear abbreviations:

LI : Line
SP : Pole spear
HC : Hand collected
NT : Throw net

[3] Location abbreviations:

AI : Shoreline and/or shallow waters around Akau Island
MI : Shoreline and/or shallow waters around Nikina Island
SI : Shoreline and/or shallow waters around Sand Island
P1 : Long-term Station P1 and adjacent similar areas
P5 : Long-term Station P5 and adjacent similar areas
Z6 : Zone 6
Z10: Zone 10
CH : All channels
LA : Elsewhere in JA lagoon within the shallow platform atoll area.

Note : For species with a substantial total number caught in more than one location, the number caught in each major location is shown.

Some fishing and collecting have occurred throughout all areas of the lagoon where boat use is permitted and at all the islands of JA. However, there are a number of locations that are fished much more than others.

Trolling and bottom fishing are done in all the channels. About 95% occurs along the north edge of the main channel and turning basin from Hama point around JI to the garbage chute. Catch from the channels consists primarily of Caranx melampygus, Carangoides orthogrammus, Pseudupeneus multifasciatus, P. cyclostomus, and P. bifasciatus. There are only a few fishermen who fish this area once and occasionally twice a week.

Another location that receives considerable fishing pressure from spearfishermen and coral collectors is the area between the north edge of Akau Island and the barrier reef, extending from Station P5 west to the NW corner of Akau Island. Very little line fishing occurs in this area. Major catch species are Myripristis amaenus, Ctenochaetus strigosus, Pseudupeneus multifasciatus, P. bifasciatus, Acanthurus triostegus, and Priacanthus cruentatus. Acropora cytherea, Cypraea tigris, and Panulirus penicillatus are the primary hand collected species from this area.

The area in Zone 10 between the west edge of the main channel and the barrier reef, extending past the west camera stand to the SW end of the barrier reef, receives a moderate amount of fishing pressure. Major catch species taken are Ctenochaetus strigosus, Pseudupeneus bifasciatus, P. multifasciatus, P. cyclostomus, Acanthurus triostegus, and Scarus perspicillatus. Most are speared, but some are taken with lines from the channel edge near Station P3. The reef flat immediately adjacent to the west camera stand is regularly visited by fishermen looking for octopus.

The area around and containing Station P1 is occasionally visited by spearfishermen and collectors. Major catch species from this area are Myripristis amaenus, Priacanthus cruentatus, Ctenochaetus strigosus, and Scarus perspicillatus. Less fishing occurs here during winter months due to strong surge and currents resulting from large surf breaking just outside the reef. The region of Zone 5 extending from Station P5 to P6 and Donovan's Reef is occasionally visited by spearfishermen and collectors. Major catch species from this area are Ctenochaetus strigosus and Myripristis amaenus. Hand collected species are Cypraea tigris, Panulirus penicillatus, and Diatichemora sp.

Various locations around Johnston Island receive a considerable amount of fishing pressure. The main pier is line fished for Caranx melampygus, Carangoides orthogrammus, Pseudupeneus cyclostomus, and Priacanthus cruentatus when barge traffic allows. The port control pier, which formerly was line fished for Myripristis amaenus, is now off limits to fishing. During the day, Pseudupeneus cyclostomus, P. multifasciatus, and occasionally Octopus sp. are taken primarily by line along the shoreline from the Point house to the southeast corner of JI. Myripristis amaenus and Priacanthus cruentatus are taken by line and are the major catch species from Hama point. Throw nets are occasionally used along the shoreline from Hama point to the West point to take Acanthurus triostegus and Chaenomugil leucosagus.

At night the Red Hat seawall is line fished for Myripristis amaenus, the big-scale soldierfish (Myripristis berndti), and Priacanthus cruentatus. Hashi's shack is line fished for the needlefish (Platybelone argalus) and Scarus perspicillatus. The grey reef shark (Carcharhinus amblyrhynchos) is also occasionally taken by military personnel using handlines from Hashi's shack and Hama point. The white-tipped reef shark (Triaenodon obesus), which was formerly caught at these sites, is now protected by an FCJ regulation. The garbage chute, formerly a popular fishing site, has been condemned due to structural damage by a storm. Fishing previously done at the garbage chute is now done at nearby Hashi's shack on the west wharf. However, some shark fishermen have been frequenting the garbage chute again.

Sand Island also receives some line and net fishing pressure. At night the pier is line fished for Myripristis amaenus and Priacanthus cruentatus. Caranx melampygus and Carangoides orthogrammus are occasionally taken there also. During the day, throw netters take Acanthurus triostegus, Kuhlia marginata, and Chaenomugil leuciscus from the shorelines around the east part of Sand Island.

Akau and Hikina Islands are frequented by throw netters taking Acanthurus triostegus, Chaenomugil leuciscus, Kuhlia marginata, and Mulloides flavolineatus. Pseudupeneus cyclostomus, Caranx melampygus, and Carangoides orthogrammus were also taken by line from the Hikina Island pier. These islands are off limits for all human visitation most of the year due to the large numbers of nesting seabirds there.

Weather permitting, all the locations above are easily accessible to fishermen. Locations in Zone 5 are somewhat less accessible due to occasional strong currents and surge. The areas around Stations P1, P3, P5 and P6 are visited primarily by divers spearing and/or hand collecting. Very little, if any, line fishing occurs at or near these areas. The channel areas are fished almost exclusively using lines, with some spearing occurring along the channel edge near Station P3. Line fishing from shore on JI is done at all the locations mentioned above. There is a low level of throw netting on JI done by a handful of regular fishermen.

A more detailed breakdown for annual catch of the 13 "major catch species" is presented in Table 6. Catch was separated by gear types. Catch, effort, and catch per unit effort (CPUE) were calculated for each situation.

Table 6. Estimated annual boat [1] catch, effort, and catch per unit effort (CPUE) of the 13 "major catch species" in the JA fishery for the period Jun 89 - May 90, broken down by gear type.

SPECIES	GEAR TYPE: LINE	SPEAR	THROW NET	TOTAL
Myripristis amoenus (Brick soldierfish)				
CATCH[2]	65	3297		3362
EFFORT[3]	29	737		
CPUE[4]	2.24	4.47		
Priacanthus cruentatus (Bigeye)				
CATCH		79		79
EFFORT		194		
CPUE		0.41		
Kuhlia marginata (Hawaiian flagtail)				
CATCH	38		187	225
EFFORT	28.5		30	
CPUE	1.33		6.23	
Kyphosus vaigiensis (Rudderfish)				
CATCH				0
EFFORT				
CPUE				
Mulloides flavolineatus (Yellowstripe goatfish)				
CATCH	35	29	64	128
EFFORT	98	82.5	21	
CPUE	0.36	0.35	3.05	
Parupeneus bifasciatus (Doublebar goatfish)				
CATCH	6	60		66
EFFORT	26	131		
CPUE	0.15	0.46		
Parupeneus cyclostomus (Blue goatfish)				
CATCH	124	5		129
EFFORT	429	18		
CPUE	0.29	0.28		
Parupeneus multifasciatus (Manybar goatfish)				
CATCH	31	7		38
EFFORT	226	10.5		
CPUE	0.14	0.67		

Table 6 (continued).

SPECIES	GEAR TYPE: LINE	SPEAR	THROW NET	TOTAL
Caranx melampygus (Blue jack)				
CATCH[2]	177	9		186
EFFORT[3]	506	41		
CPUE[4]	0.35	0.22		
Chaenomugil leuciscus (Chaotall's mullet)				
CATCH			509	509
EFFORT			81	
CPUE			6.28	
Scarus perspicillatus (Spectacled parrotfish)				
CATCH		74	9	83
EFFORT		145	15	
CPUE		0.51	0.60	
Acanthurus triostegus (Convict surgeonfish)				
CATCH		480	348	828
EFFORT		222.5	45.5	
CPUE		2.16	7.65	
Ctenochaetus striatus (Yellow eyed surgeonfish)				
CATCH		1201		1201
EFFORT		359		
CPUE		3.34		
GRAND TOTAL FOR MAJOR SPECIES IN CATCH				
CATCH	474	5241	1117	6832
EFFORT	1342.5	1940.5	192.5	
CPUE	0.35	2.70	5.80	

[1] Any fishing from shores of islands other than it involved the use of boats and is reported here.
[2] Catch in number of individuals.
[3] Effort units:
Line : line-hours
Spearing : spear-hours
Throw netting : throw net-hours
[4] Catch per unit effort:
Line : number of fish per line-hour
Spearing : number of fish per spear-hour
Throw netting : number of fish per throw net-hour

Catch and effort were highly variable among species, and for most species, they were highly variable over time. Most of the CPUE values for individual species from the year ending 1990 were generally within the range of the corresponding values from the previous years of the study (Table 7). However, all the CPUE values were highly variable with no clear trends between the years.

Total catch has varied considerably over the 6 years of the study (Table 7) as well as the subtotals by each type of gear (Fig. 11-13). No particular temporal pattern is recognizable. However, for most of the total time series for each gear type, the pattern of fishing effort corresponds rather closely with that of catch. Therefore, CPUE, which is sometimes used as an indicator of fish abundance, is much less variable than catch. CPUE for each gear type is considerably more stable for all species combined than for most single species. It shows no meaningful temporal trend for any of the gear types. CPUE's for spearing and netting (Fig. 11-12) seem to vary randomly above and below their initial values. The CPUE for line fishing (Fig. 13) decreases irregularly. These temporal patterns and the limited range of CPUE values for each gear type suggest that the year-to-year fluctuations in catch primarily reflect fluctuations in effort.

Effort and CPUE may have been noticeably affected by some observable shifts in the fishermen's fishing patterns in recent years. Several of the "resident" fishermen have retired and left JA in the past two years. Other "resident" fishermen have stated that they have been "taking a break" from fishing and have only gone fishing a few times in the past two years. Competition by increasing numbers of "transient" SCUBA divers (who seem to catch little) for the use of the limited supply of boats at JA appears to have reduced the amount of productive effort by experienced, skilled fishermen. Other fishermen new to JA have been replacing the older "resident" fishermen in the fishery, but these new fishermen do not seem to catch as much as the "resident" fishermen did. A decrease in CPUE may have resulted, especially where consistent line fishermen have left JA for good. The "resident" fishery has been shifting to mostly a few groups of spear fishermen. Consequently, some of the species previously caught mostly by line fishing were collected in low numbers this report year, while some of the spear catches were high. Overall, there are now fewer fishermen who catch a high volume of fish. Inconsistent reporting of catch and effort, months of bad weather (especially in the years ending 1985 and 1986), as well as the home leaves, travel and work schedules of "resident" fishermen all can have significant effects on this small fishery.

Clearly there are some unresolved anomalies in the catch and effort data. However, all the catch and effort data together do not produce any consistent trends that would indicate any major change in abundance of the resident fished populations.

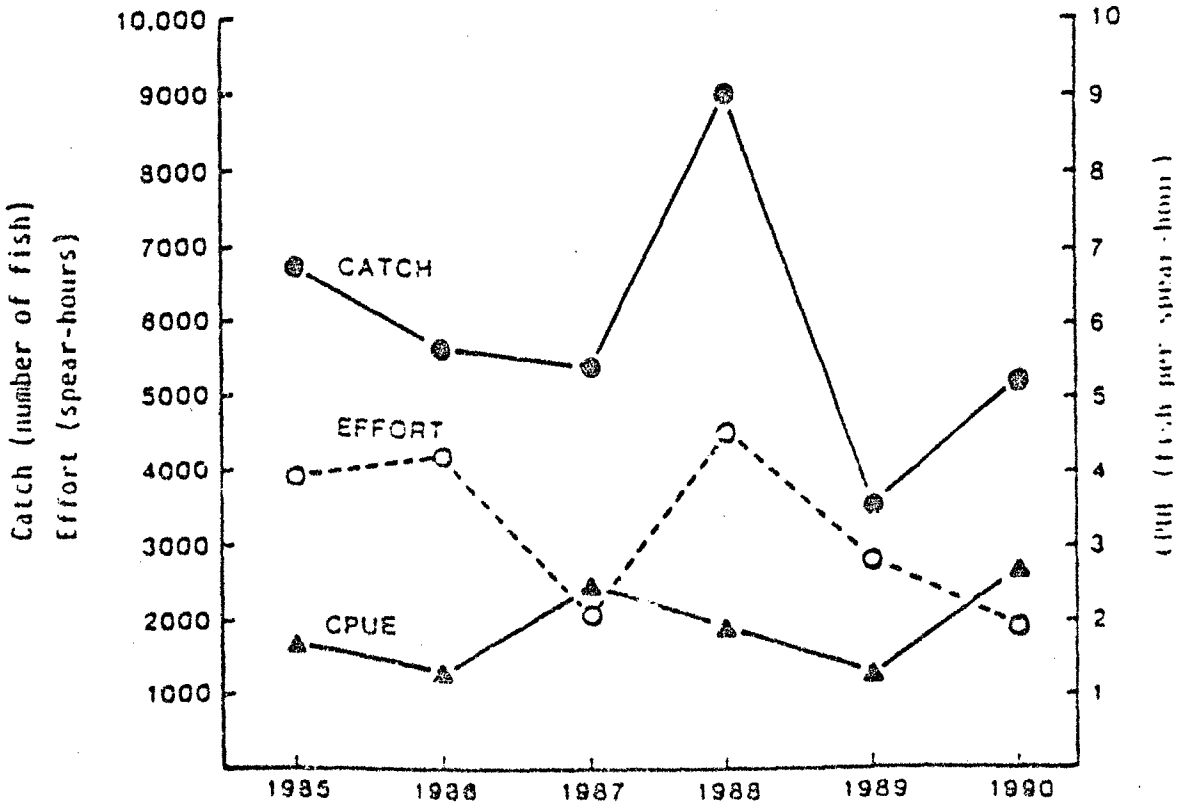


Figure 11. History of catch, effort and catch per unit effort (CPUE) of all species caught by spear fishing (using boats) over the full course of the study.

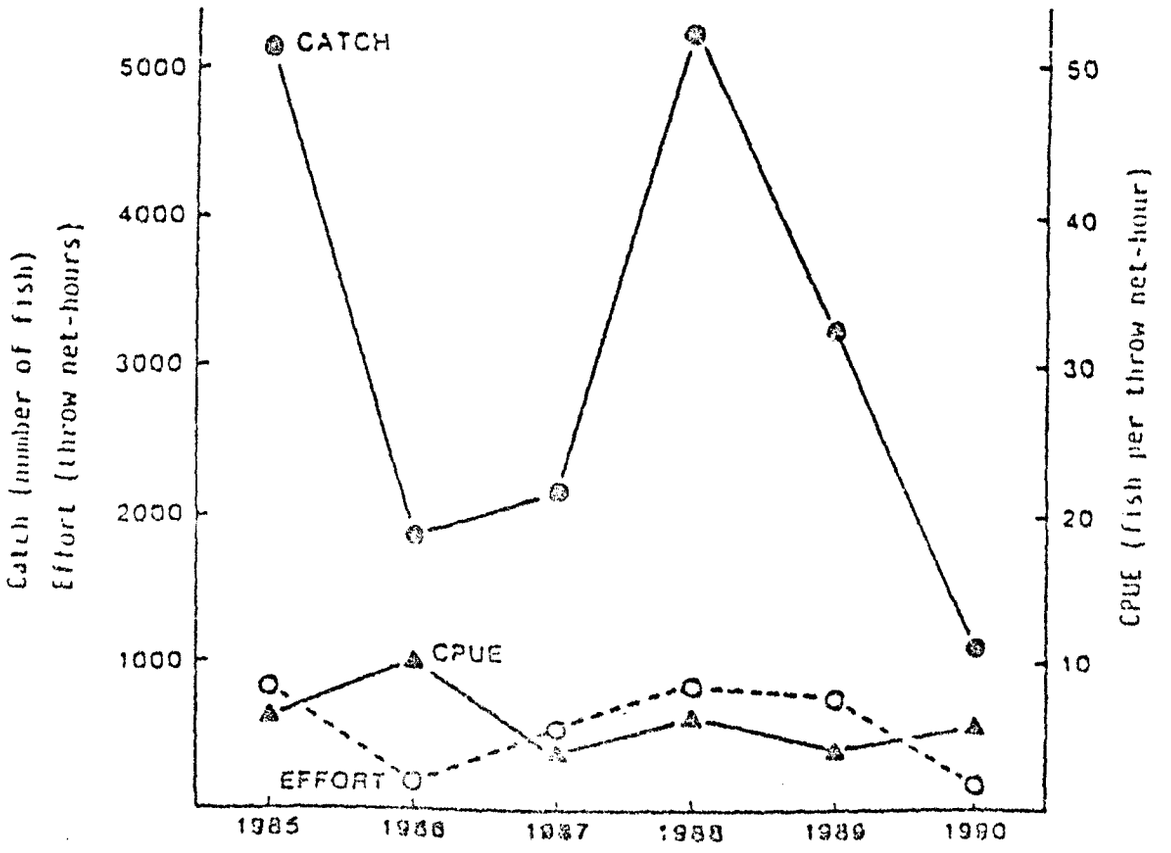


Figure 12. History of catch, effort and catch per unit effort (CPUE) of all species caught by throw net fishing (using boats) over the full course of the study.

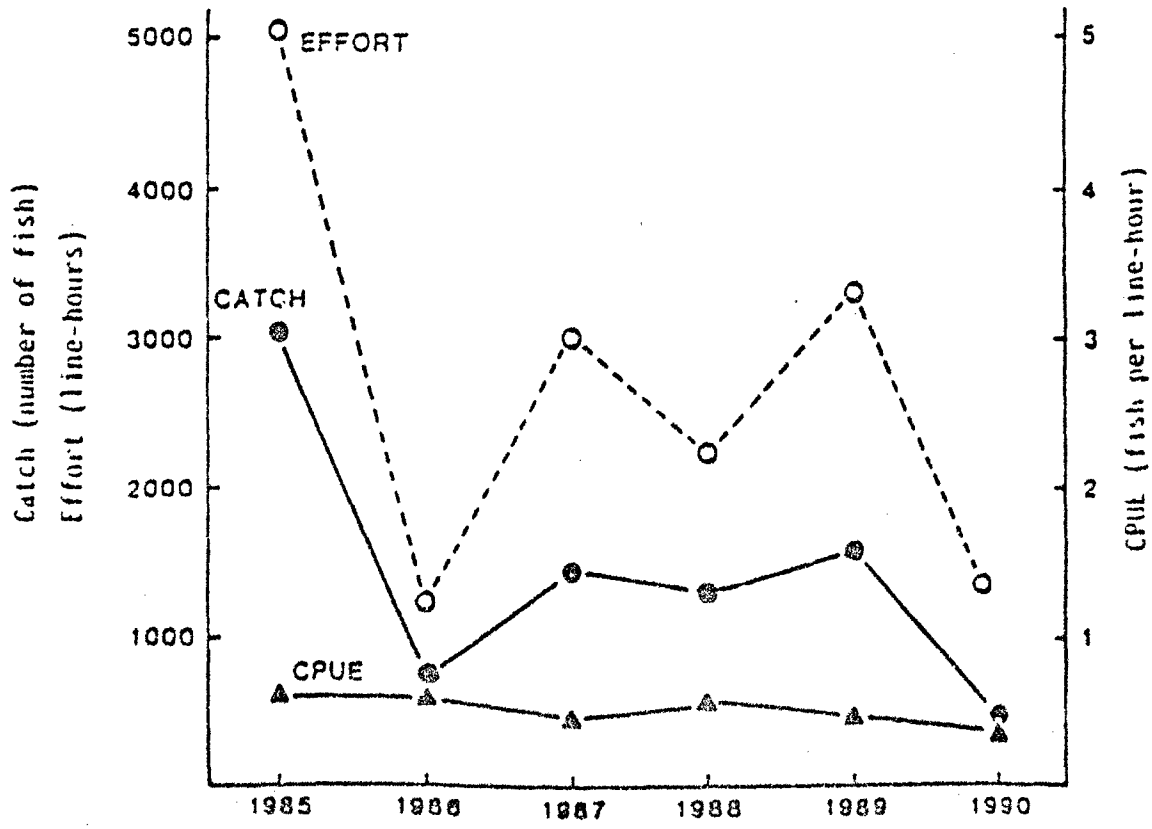


Figure 13. History of catch, effort and catch per unit effort (CPUE) of all species caught by line fishing (using boats) over the full course of the study.

Table 7. Boat catch and effort data for six successive phases of the project for the "major catch species". Resu
include the total estimated annual catch, and for the major gear type, the total effort and the catch per unit ef

Species	Estimated Annual Boat Catch (all gear combined), for year ending: 1990	1989	1988	1987	1986	'9
Myripristis amaenus	3362	1799	4474	4206	2029	30
Priacanthus cruentatus	79	49	63	94	95	1
Kuhlia marginata	225	240	555	75	293	14
Kyphosus vaigiensis	0	19	78	28	48	
Mulloides flavolineatus	128	903	396	269	266	3
Pseudocheilinus bifasciatus	64	144	370	207	358	3
Pseudocheilinus cyclostomus	129	435	322	282	239	5
Pseudocheilinus multifasciatus	38	338	289	288	198	-
Caranx melampygus	186	310	405	362	552	5
Chaerodon fasciatus leucisca	509	1201	3772	769	557	8
Scarus perspicillatus	83	315	353	185	289	1
Acanthurus triostegus	828	1657	2940	1222	1162	24
Ctenochaetus strigosus	1201	936	1609	1066	2188	31
Total	6,832	8,396	15,652	9,051	8,274	14,9

Effort and Catch per Unit Effort by major gear type for year ending:

Species	Major Gear Type	1990 Effort	1990 CPUE	1989 Effort	1989 CPUE	1988 Effort	1988 CPUE	1987 Effort	1987 CPUE	1986 Effort	1986 CPUE	'9 Effort
M. amaenus	spear	737	4.47	631	2.12	999	4.35	569	6.78	576	3.40	546
P. cruentatus	spear	194	0.41	74	0.60	404	0.13	198	0.38	276	0.27	526
K. marginata	net	30	6.23	46	4.97	33	16.03	63	1.28	30	9.93	134
K. vaigiensis	line	0	0	0	0	39	1.03	78	0.23	0	0	324
M. flavolineatus	net	21	3.05	166	4.44	60	3.92	59	4.14	27	6.23	63
P. bifasciatus	spear	131	0.46	269	0.28	505	0.63	117	0.62	556	0.57	448
P. cyclostomus	line	429	0.29	1227	0.28	766	0.39	492	0.55	362	0.63	1072
P. multifasciatus	line	226	0.16	737	0.38	743	0.38	348	0.63	207	0.62	890
C. melampygus	line	506	0.35	526	0.56	201	1.78	1063	0.32	472	0.61	1334
C. leucisca	net	81	6.28	251	4.28	353	10.69	268	2.87	51	10.47	245
S. perspicillatus	spear	143	0.51	624	0.50	591	0.58	343	0.54	618	0.47	370
A. triostegus	net	46	7.43	264	6.40	366	1.99	167	7.49	53	11.40	327
C. strigosus	spear	357	3.34	491	2.01	826	1.95	391	2.72	566	3.30	609

Fish Population Characteristics Based on Creel Census

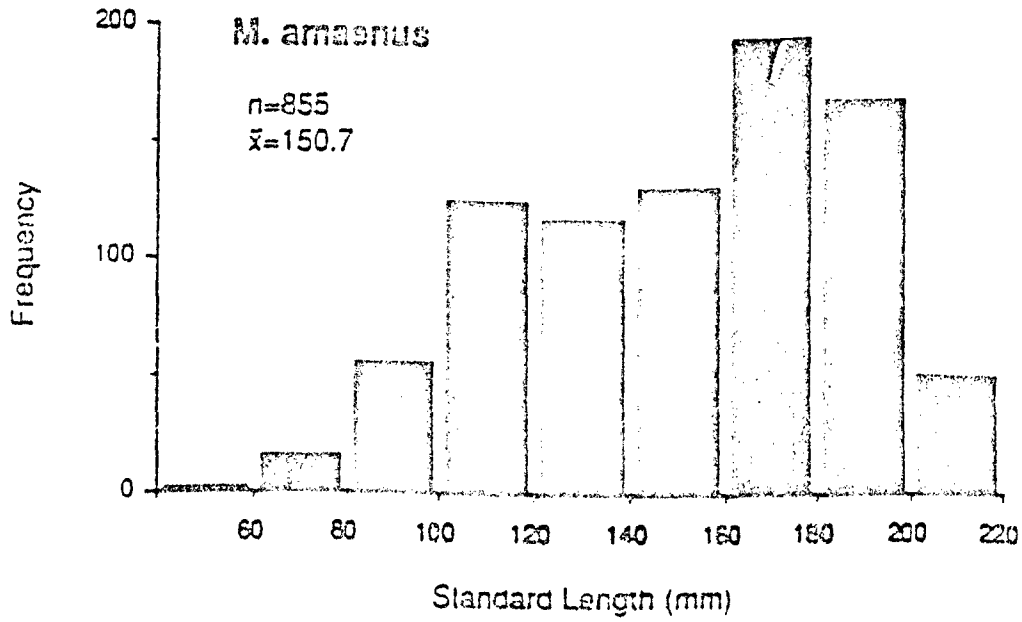
Some basic descriptive statistics for 11 of the "major catch species" were calculated from the creel census size data using SAS (version 5.16) on the University of Hawaii's mainframe IBM 3081 computer (Table 8). Only species with 70 or more specimens examined in creel census (from Feb 84 to May 90) were analyzed. Table 8 shows a summary of the data, as well as length-weight regression equations generated for each species, and the size at first reproduction for some of the species. Figures 14-24 are histograms of the standard lengths (SL) and weights of the individuals examined from Feb 84 to May 90. Appendix G contains frequency tables of SL and weights for the species shown in these histograms.

Most of the catch was of a fairly large size. The absence of very small individuals and the presence of several ascending size classes below the mode probably reflect selection for larger individuals by the gear and fishing techniques. However, very small individuals of any species were rarely seen in censuses or surveys. At body sizes above the mode, strong selection by fishermen for larger individuals of M. amaenus appears to produce a distribution that may be much different from the natural population at large (Fig. 14). For some species, the descending limb of the distribution curve (to the right of the mode) is rough (perhaps because of limited sample size). However, there seems to be no reason to believe that this portion of the distributions is far from representative of the natural populations in most cases. A cluster of large outliers of C. melampygus (Fig. 19) is produced by the efforts of a few fishermen specifically targetting large size classes.

Few cases of multiple modes appear clearly in any of the histograms. None of the data sets in their present condition appear promising for detecting cohorts for age or mortality estimation. No adequate data for size frequency are available from areas with low fishing effort for comparison with these data (which came primarily from the more heavily fished areas).

The sizes at first reproduction (SFR) for six of the 11 species shown in Table 8 were taken from the results of other investigators working in the Hawaiian Islands. No estimates were available for the SFR of Priacanthus cruentatus, Carangoides orthogrammus, Scarus perspicillatus, Ctenochaetus strigosus, and Chaenomugil leucosoma. No data were available from JA for the SFR of any species except Myripristis amaenus (Dee 1986), but it seems unlikely that any are greatly different from Hawaiian populations.

The number of fish caught and examined in creel census was inadequate to do many types of fishery analyses. The results presented here are thus somewhat limited, but they are adequate in light of the low level of catch. Since there has been no sustained and significant increase in fishing effort since the beginning of the project, all the basic descriptive data taken to date will serve as a useful baseline for comparison with samples taken after any future major changes in fishing effort. The frequency distributions of the catch species will be especially useful if fishing pressure significantly increases at JA.



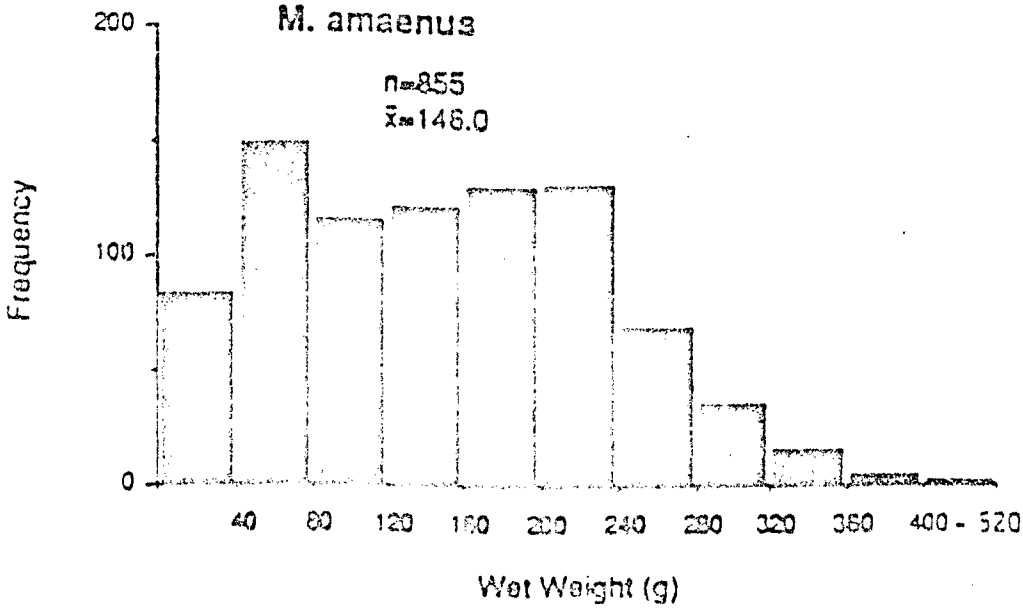
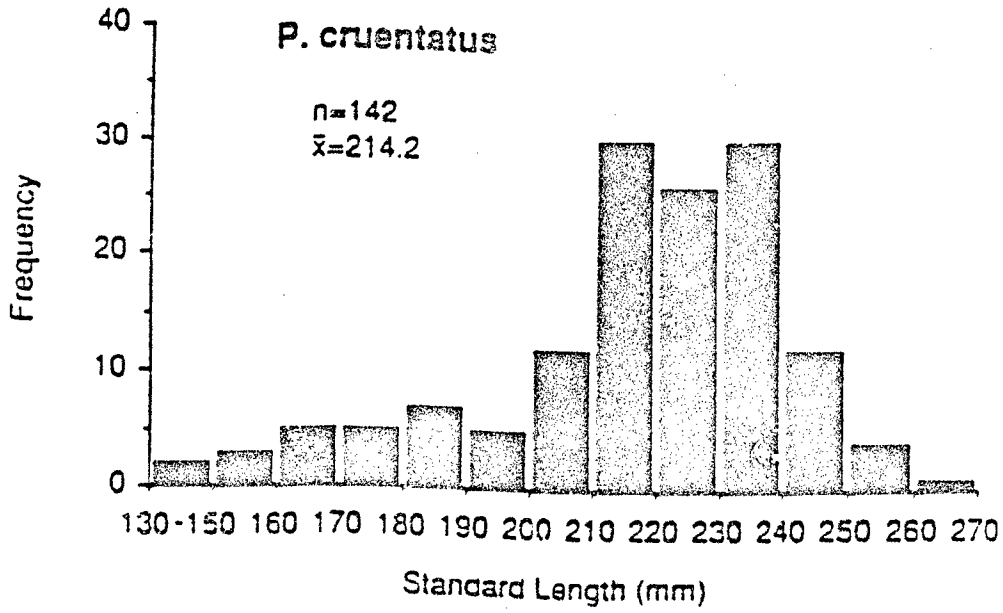


Fig. 14. Frequency histograms of standard lengths (mm) and wet weights (g) of M. amaenus creel censused between Feb 84 and May 90. The means ($\bar{x}$) represent the arithmetic average of all data taken during this period.



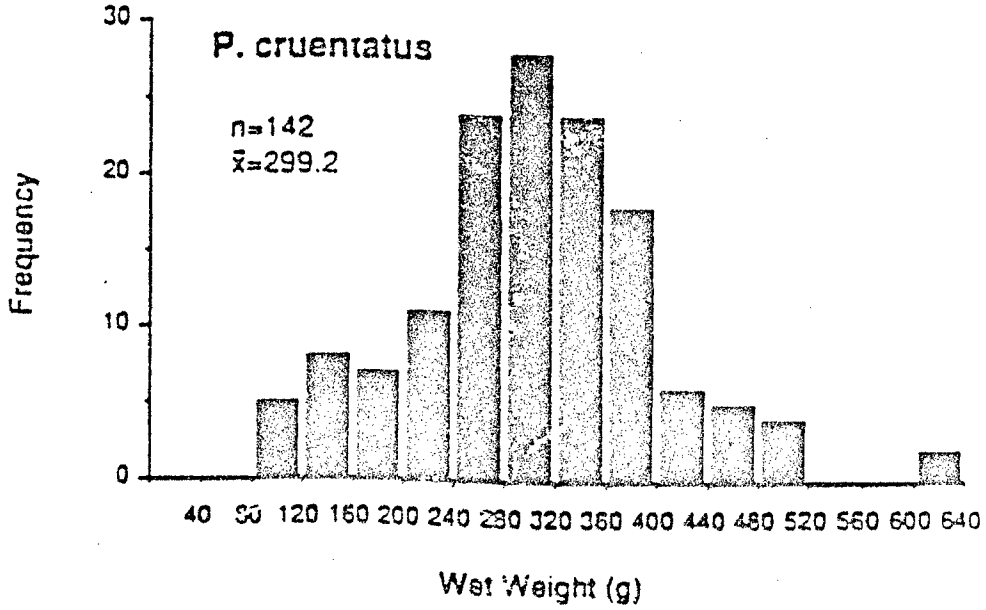
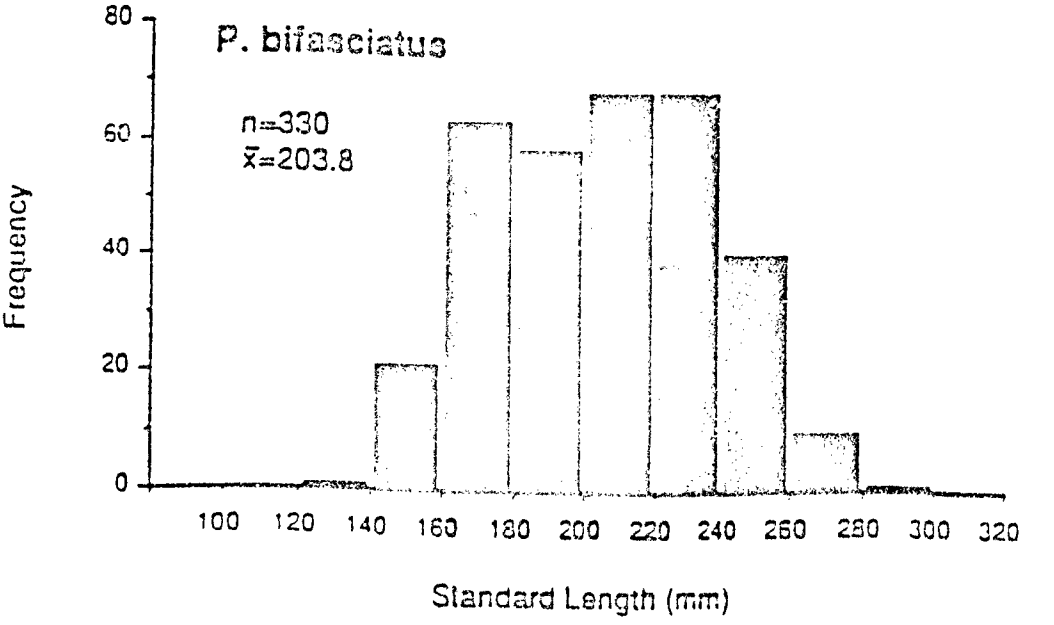


Fig. 15. Frequency histograms of standard lengths (mm) and wet weights (g) of P. cruentatus creel censused between Feb 84 and May 90. The means (x̄) represent the arithmetic average of all data taken during this period.



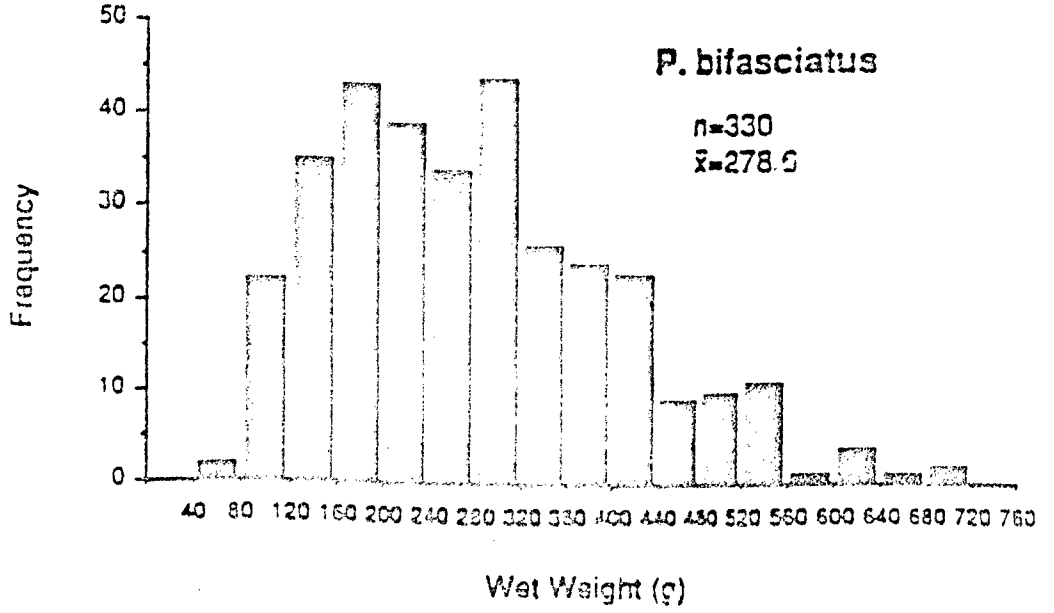
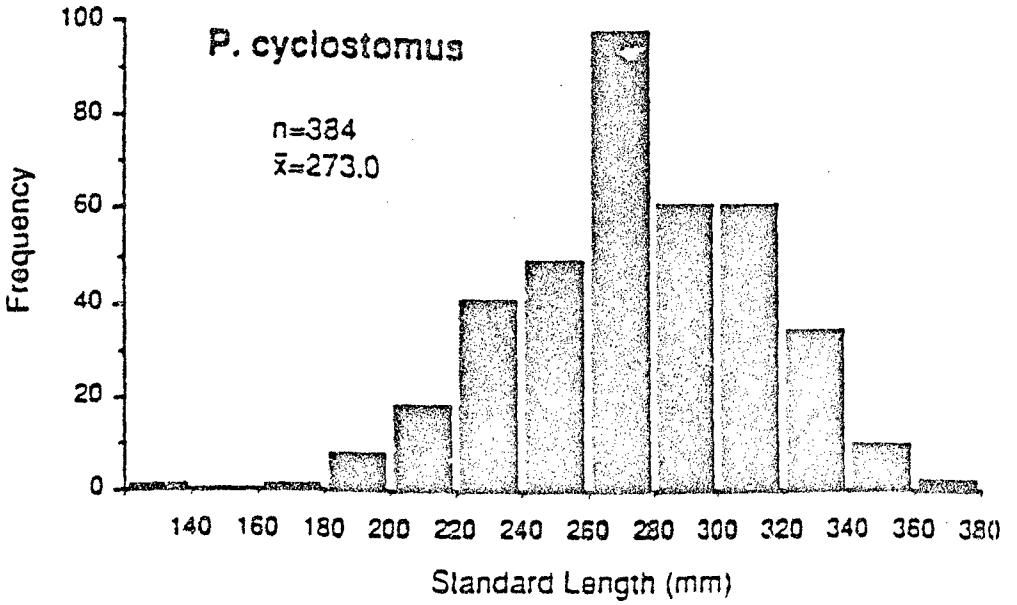


Fig. 16. Frequency histograms of standard lengths (mm) and wet weights (g) of P. bifasciatus creel censused between Feb 84 and May 90. The means ($\bar{x}$) represent the arithmetic average of all data taken during this period.



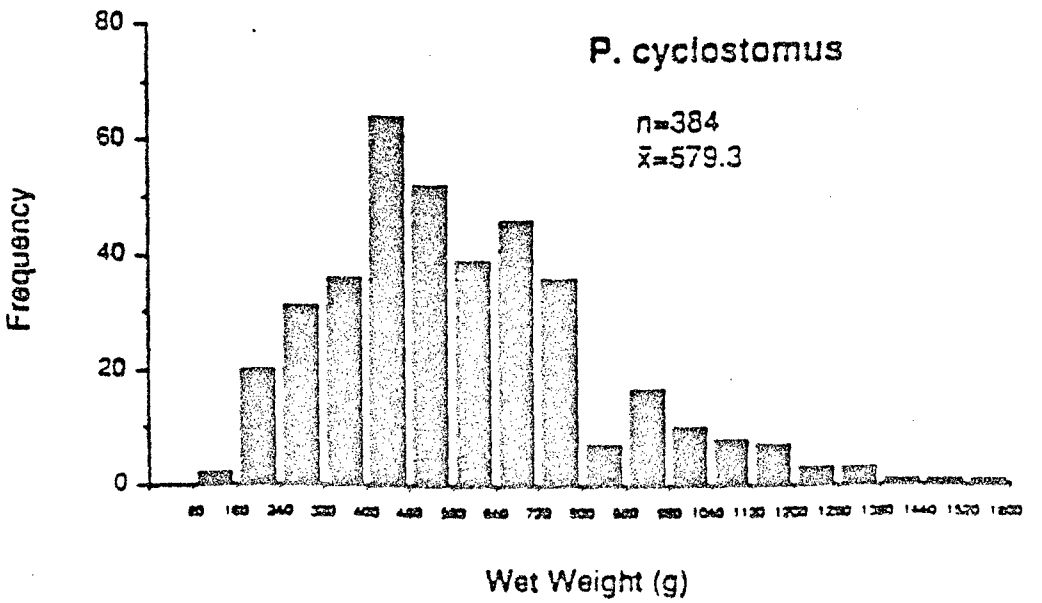
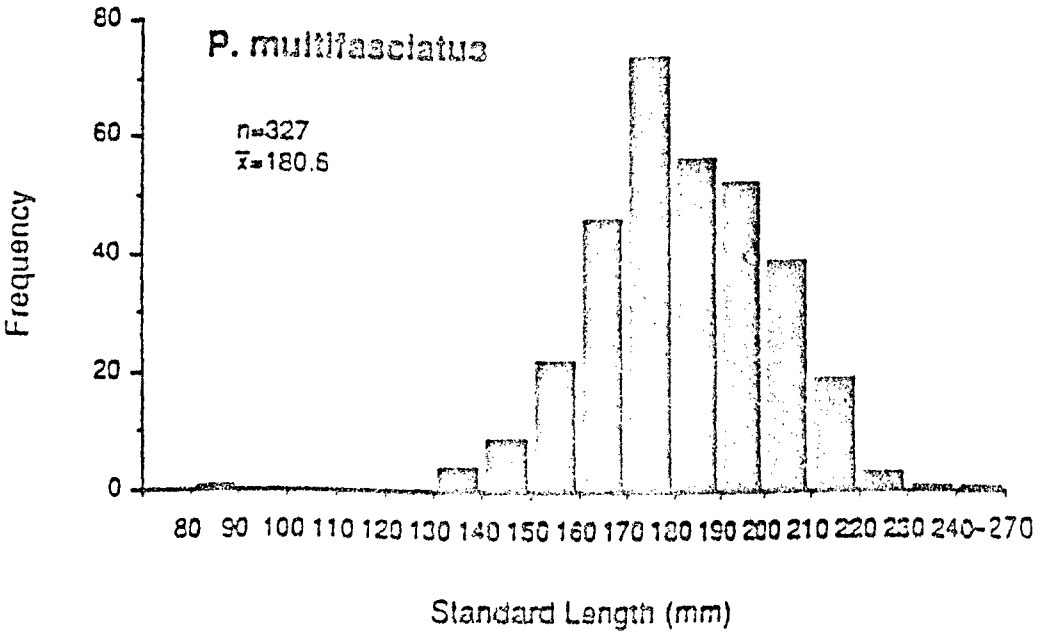


Fig. 17. Frequency histograms of standard lengths (mm) and wet weights (g) of P. cyclostomus creel censused between Feb 84 and May 90. The means (x̄) represent the arithmetic average of all data taken during this period.



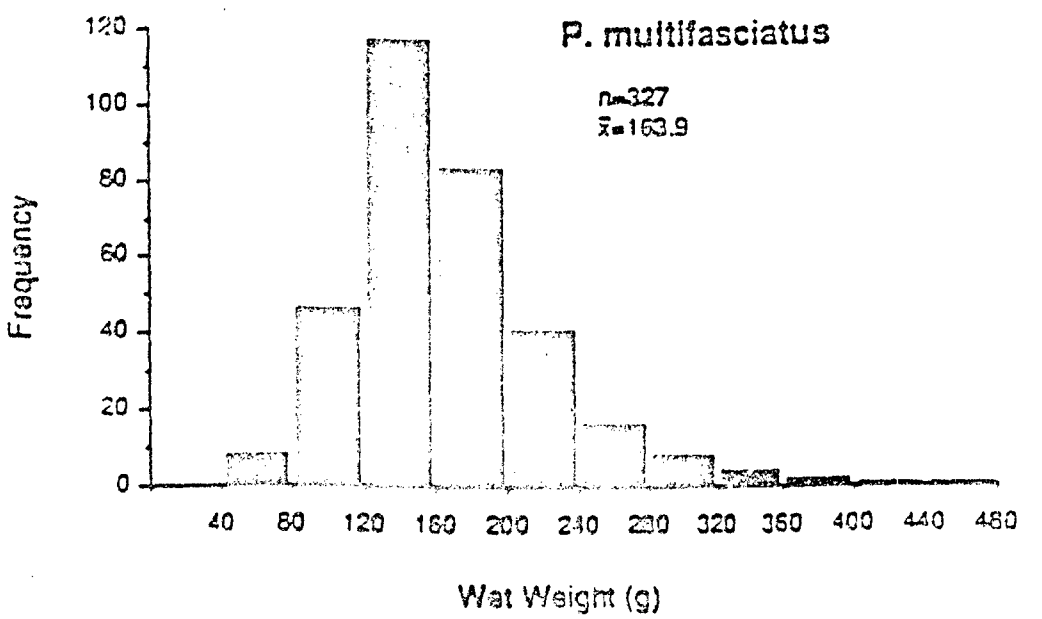
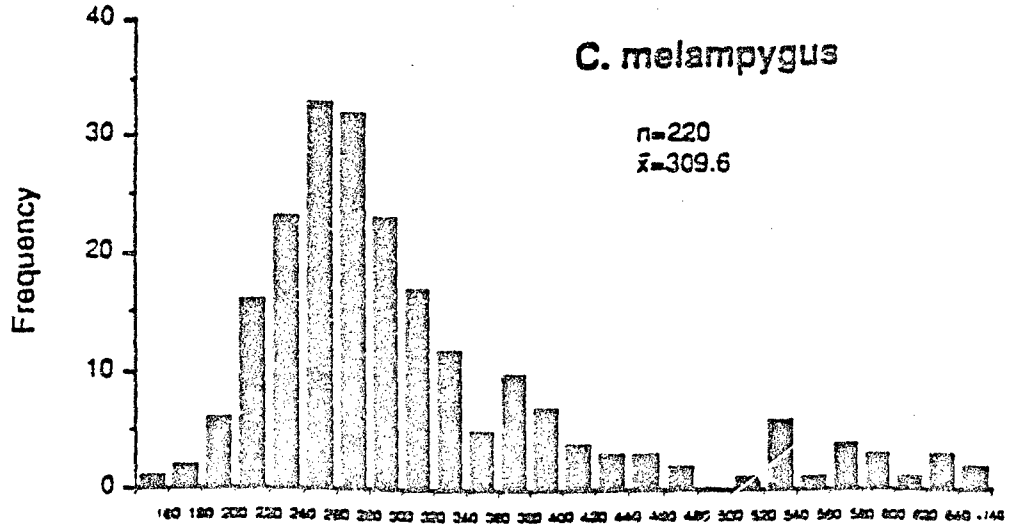


Fig. 18. Frequency histograms of standard lengths (mm) and wet weights (g) of P. multifasciatus creel censused between Feb 84 and May 90. The means ($\bar{x}$) represent the arithmetic average of all data taken during this period.



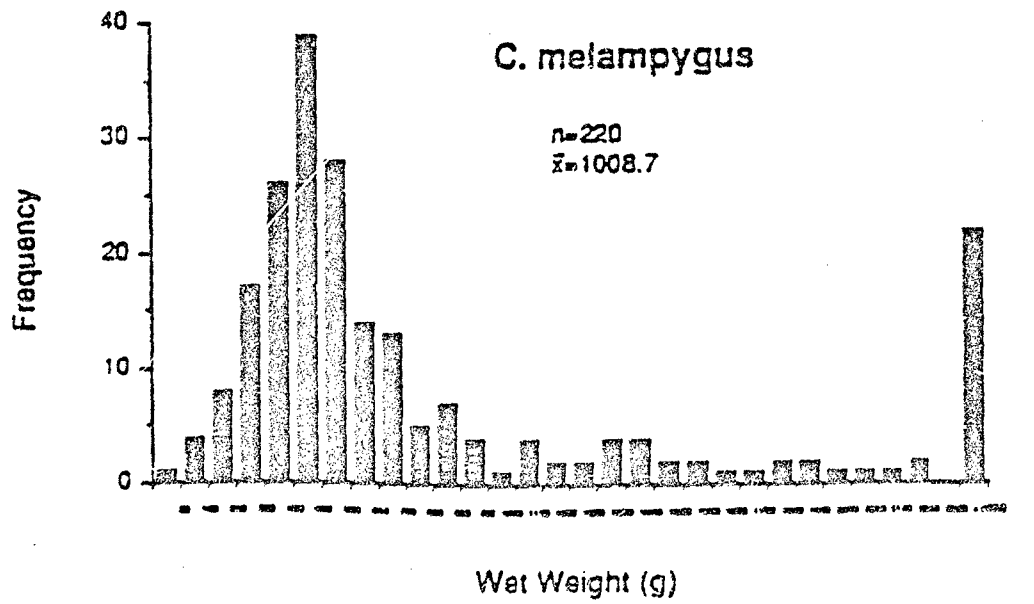
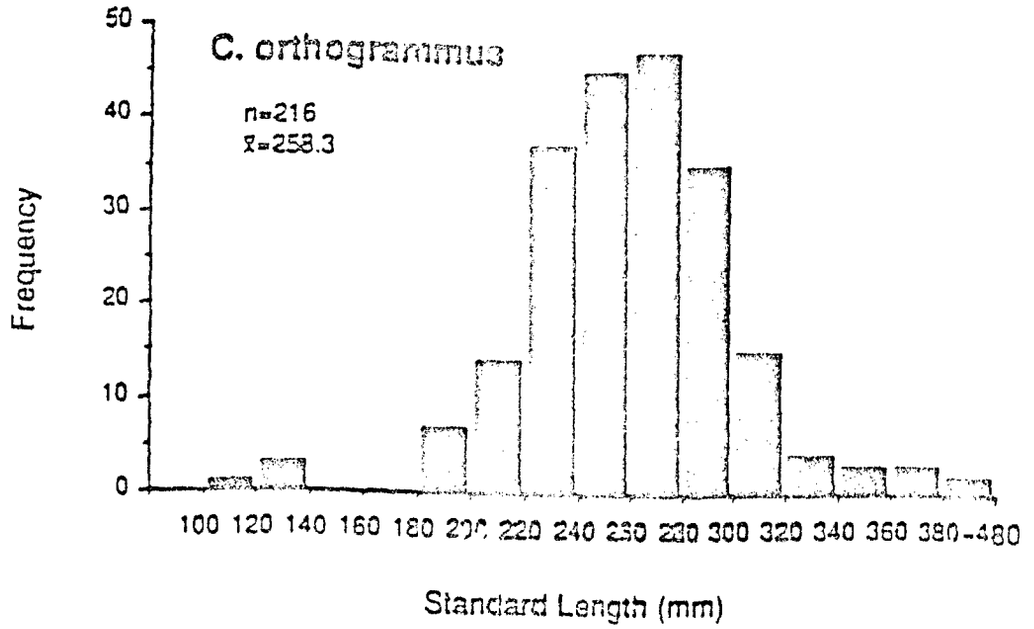


Fig. 19. Frequency histograms of standard lengths (mm) and wet weights (g) of C. melampygus creel censused between Feb 84 and May 90. The means ($\bar{x}$) represent the arithmetic average of all data taken during this period.



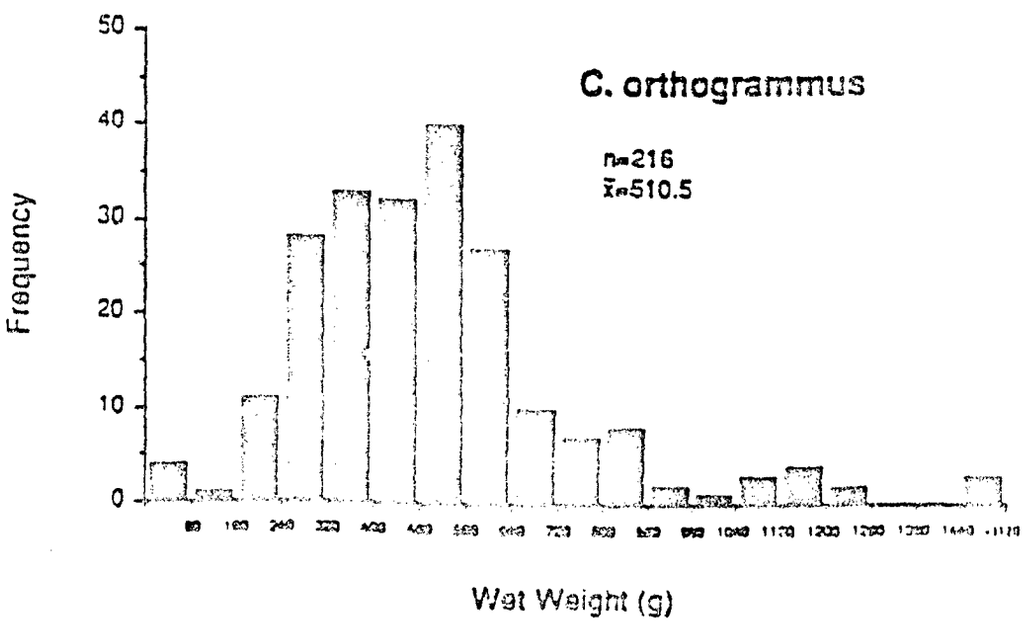
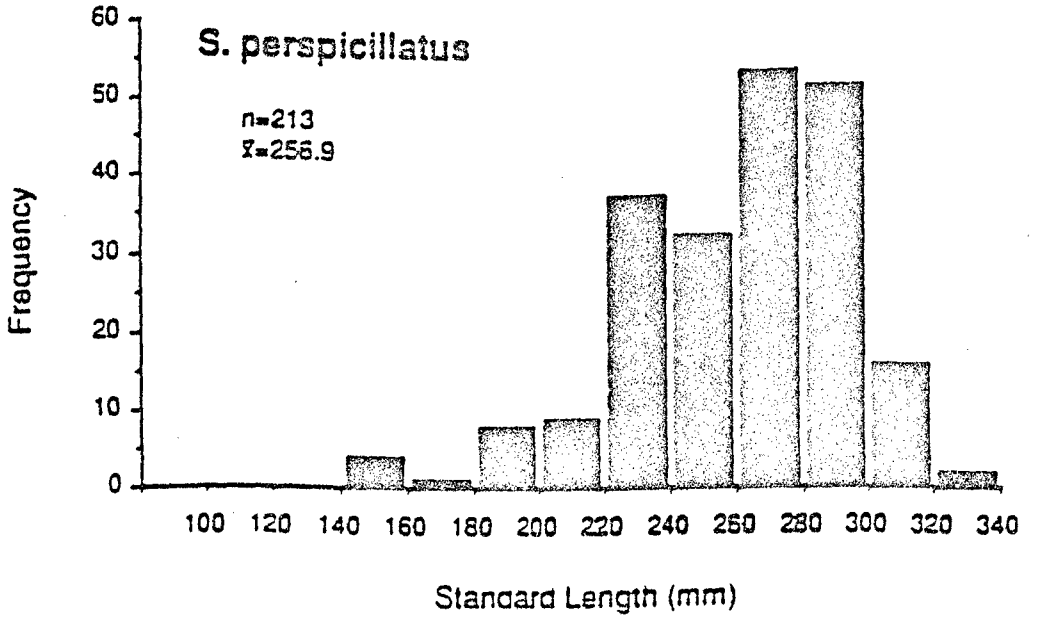


Fig. 20. Frequency histograms of standard lengths (mm) and wet weights (g) of C. orthogrammus creel censused between Feb 84 and May 90. The means ($\bar{x}$) represent the anthmetic average of all data taken during this period.



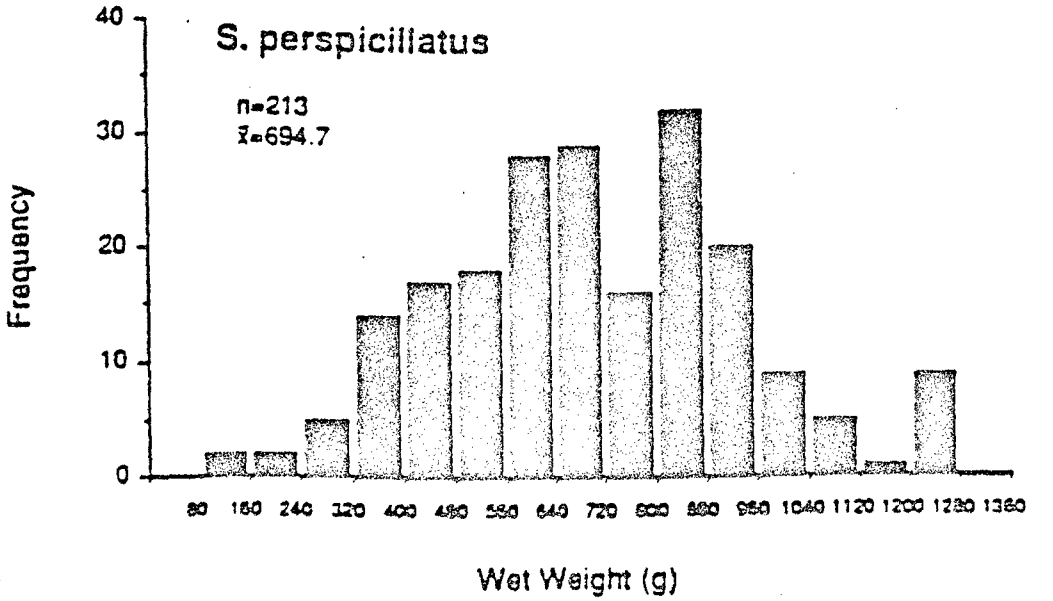


Fig. 21. Frequency histograms of standard lengths (mm) and wet weights (g) of S. perspicillatus creel censused between Feb 84 and May 90. The means ($\bar{x}$) represent th anthmetic average of all data taken dunng this period.

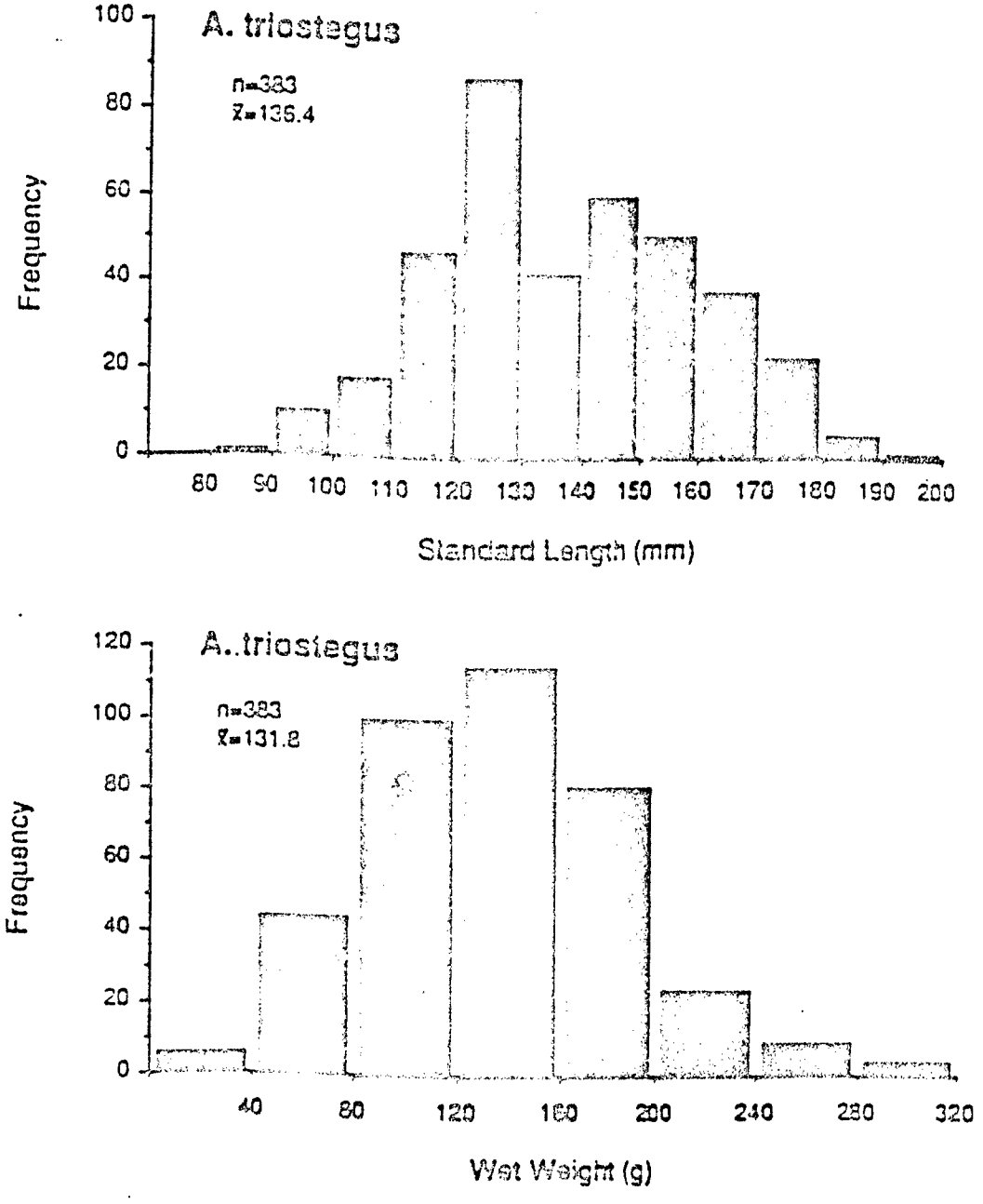
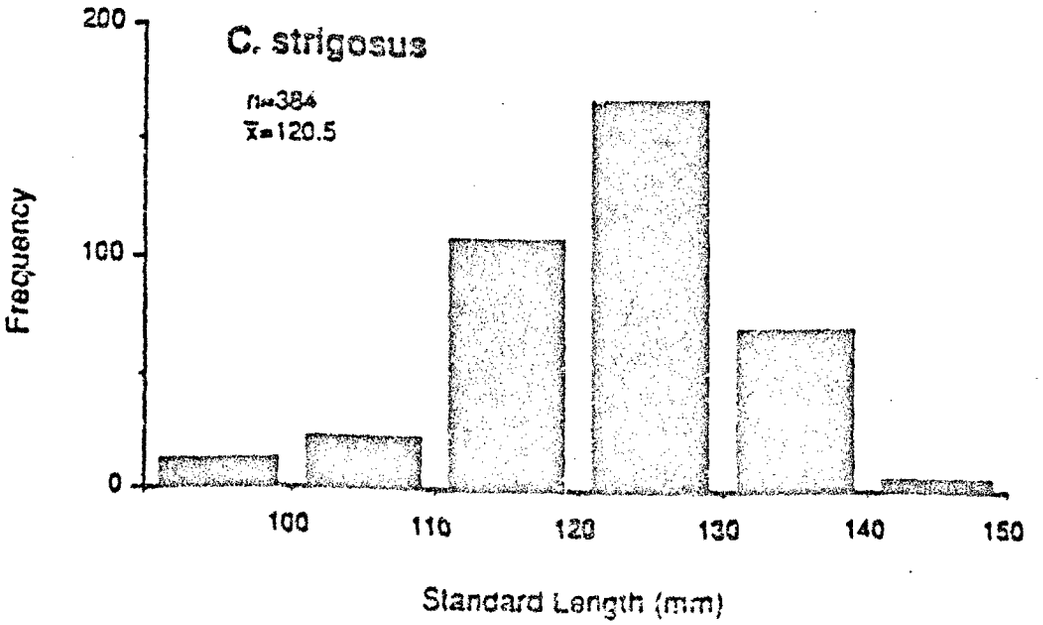


Fig. 22. Frequency histograms of standard lengths (mm) and wet weights (g) of A. triostegus creel censused between Feb 84 and May 90. The means ($\bar{x}$) represent the arithmetic average of all data taken during this period.



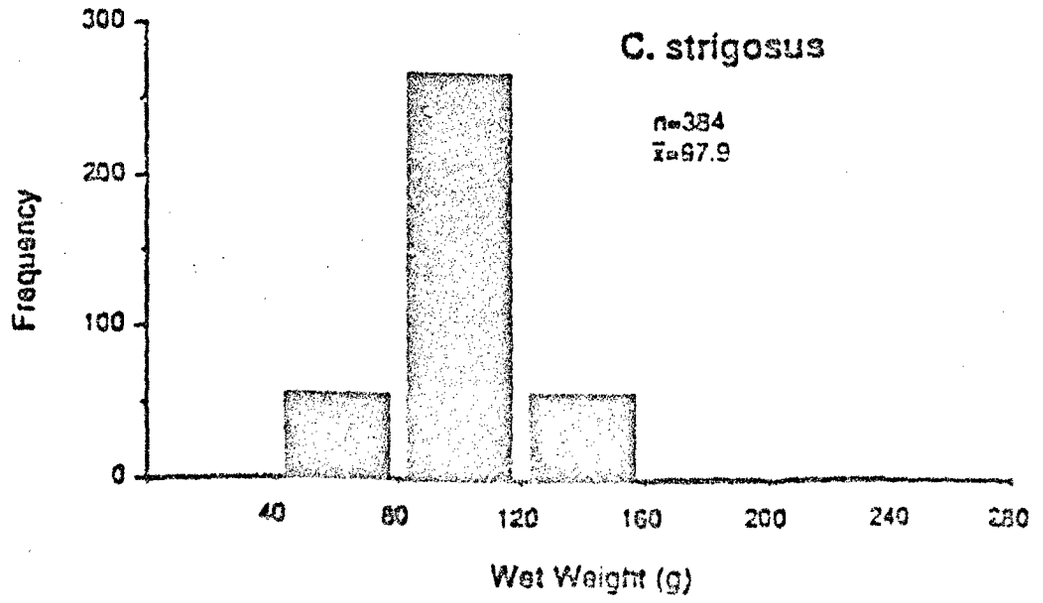
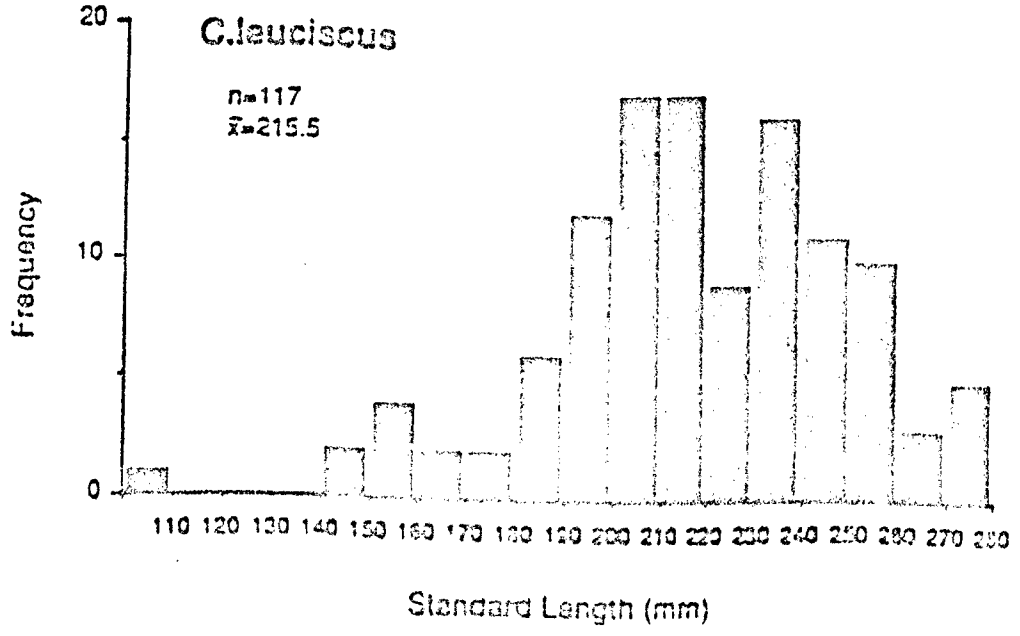


Fig. 23. Frequency histograms of standard lengths (mm) and wet weights (g) of C. strigosus creel censused between Feb 84 and May 90. The means ($\bar{x}$) represent the arithmetic average of all data taken during this period.



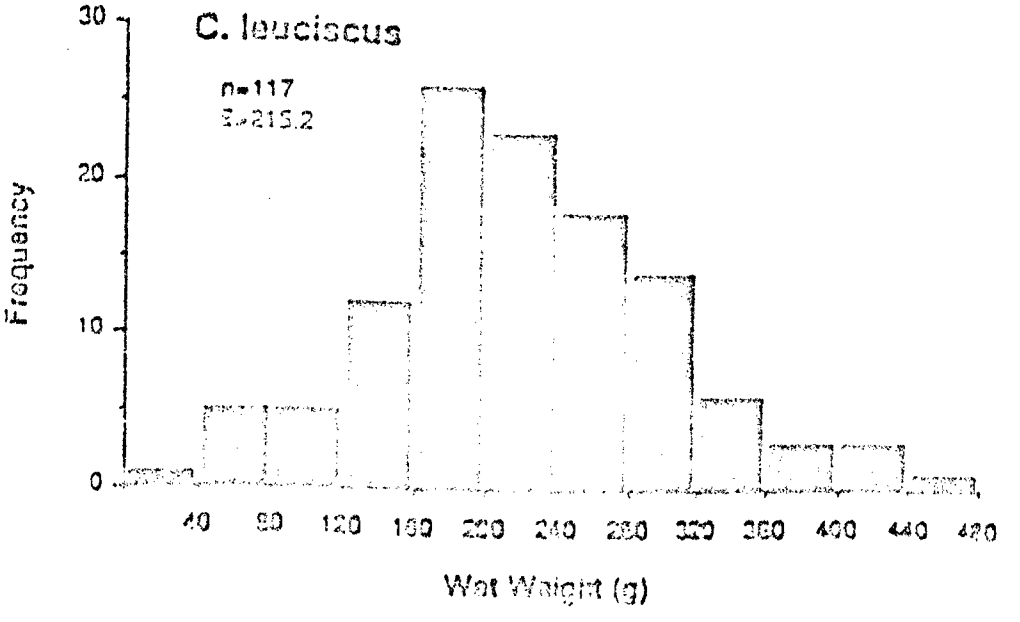


Fig. 24. Frequency histograms of standard lengths (mm) and wet weights (g) of C.leuciscus creel censused between Feb 84 and May 90. The means (x̄) represent the arithmetic average of all data taken during this period.

Table 8. Summary size data for 11 important catch species, based on creel census.

Catch species	Creel Census Data[1] Mean Standard Length, SL (mm)	Mean Weight, W (g)	Range of Weight (g)	Regression Equation $W = a(SL)^b$ a	b	r^2	n	Size at Fir Reproductio
Myripristis amoenus	150.7	146.0	2.8-480.0	7.00×10^{-5}	2.87	0.97	855	F 153-156 mm
								M 149-156 mm
Priacanthus cruentatus	214.2	299.2	80.0-600.0	5.41×10^{-5}	2.89	0.88	142	-
Pseudocheilinus bifasciatus	203.8	278.6	72.0-700.0	5.96×10^{-5}	2.88	0.86	330	~181 mm
Pseudocheilinus cyclostomus	273.1	579.2	130.0-1560.0	5.33×10^{-5}	2.88	0.82	384	~181 mm
Pseudocheilinus multifasciatus	180.6	163.9	40.0-440.0	1.35×10^{-4}	2.69	0.76	327	F < 115 mm
								M 164-200 mm
Caranx melampygus	309.6[2]	1008.7[2]	60.0-9000.0	7.35×10^{-5}	2.81	0.96	220	F 325-375 mm
Carangoides orthogrammus	258.3	510.5	30.0-3100.0	1.92×10^{-5}	3.06	0.91	216	-
Scarus perspicillatus	256.9	696.7	140.0-1265.0	1.07×10^{-3}	2.61	0.85	213	-
Acanthurus triostegus	136.4	131.8	20.0-310.0	5.51×10^{-4}	2.51	0.83	383	F 101 mm
								M 97 mm
Ctenochaetus strigosus	120.5	97.9	40.0-200.0	1.79×10^{-3}	2.27	0.63	384	-
Chaenomugil leucosus	215.5	215.3	20.0-470.0	2.93×10^{-5}	2.87	0.83	117	-

[1] Data only for species with 70 or more specimens examined from Feb 84 - May 90.

[2] There was one large outlier of SL = 736.6 mm and W = 9000.0 g that was excluded from the means.

[3] From Hayes et al. (1982) unless otherwise specified. (F = female, M = male).

[4] From Moffitt (1979) for Pseudocheilinus octotaenia.

[5] From Sudekum (1984).

[6] From Dee (1983).

ATOLL-WIDE ESTIMATES OF FISH POPULATIONS AND CATCHES

Rough atoll-wide population estimates for 10 of the 13 "major catch species" are presented in Table 9, column 1 (Dee et al. 1985). (For the remaining three "major catch species", data were insufficient to arrive at reasonable atoll-wide estimates.) Using these population estimates, the percent of the species population caught annually for the year ending 1990 was calculated and compared to that for the years ending 1989, 1988, 1987, 1986 and 1985 (Table 9, column 3).

Table 9. Estimated percents of species populations caught annually from boats for the years ending 1990 (Jun 89 - May 90), 1989 (Jun 88 - May 89), 1988 (Jun 87 - May 88), 1987 (Jun 86 - May 87), 1986 (Jun 85 - May 86), and 1985 (Feb 84 - May 85).

SPECIES	1 ESTIMATED ATOLL POPULATION	2 ESTIMATED TOTAL 1990 BOAT CATCH	3 ANNUAL CATCH/POPULATION (%) 1990	1989	1988	1987	1986	1985
Ctenochaetus striosus	1,650,300	1201	<0.1	<0.1	<0.1	<0.1	0.1	0.2
Acanthurus triostegus	599,600	823	0.1	0.3	0.5	0.2	0.2	0.4
Myripristis amaenus	383,400*	3362	0.9*	0.5*	1.2*	1.0*	0.5*	0.8*
Kuhlioides flavolineatus	128,900	123	<0.1	0.5	0.2	0.1	0.1	0.2
Pseudocheneus multifasciatus	61,650	35	<0.1	0.5	0.5	0.5	0.3	1.3
Pseudocheneus bifasciatus	43,000	66	0.1	0.3	0.8	0.4	0.7	0.8
Scarus perspicillatus	29,450	83	0.3	1.1	1.2	0.6	1.0	0.6
Pseudocheneus cyclostomus	27,600	129	0.5	1.6	1.2	1.0	0.9	2.0
Carenx melampygus	26,500	186	0.7	1.1	1.5	1.4	2.1	1.9
Kyphosus vaigiensis	22,350	0	0	0.1	0.3	0.1	0.2	0.2

* The atoll population estimate is probably a considerable underestimate because of its cryptic habits.

STATUS OF STOCKS

Harvested Species

The harvest assessment shows that few species were taken in sizable numbers and that the annual catches this past year, as in previous years, were insignificant compared to the estimated standing stocks of the respective species (Table 9).

More Myripristis amaenus are caught than any other species at JA. However, this catch estimate is quite small compared to the total population figure (Table 9, which is undoubtedly an underestimate for this cryptic species). In the year ending 1985, of the 193 measured specimens caught from shore by lines, approximately 93% were below the maximum SPR (Dee et al. 1985). No individuals caught by line fishing from shore were examined in the years ending 1986, 1988, 1989 and 1990. In the year ending 1987, of the 30 measured individuals caught from shore by lines, 90% were below the maximum SPR. Among measured specimens in the speared catch, about 22% of the individuals were below the maximum SPR in the years ending 1985 (n=233), 1986 (n=54), and 1987 (n=100); about 14% were below in 1988; about 25% in 1989, and none were below in 1990. This result is consistent with visual observations of individual size ranges at the long-term stations. Since the taking of individuals from the lagoon below the maximum SPR has apparently not increased much over the period of the study, the total atoll population should not be reduced by the present level of harvest.

There are no population size estimates for Kuhlia marginata or Chaenomugil leucosoma because of the nature of their habitat. These two species frequent the inland shorelines to feed. These areas are the only places where they are seen and caught. Under completely natural conditions, these species would probably make

similar use of shoreline habitat. No quantitative surveys or censuses were done in these habitats to provide population estimates. Net fishing for these species occurred less frequently this year than during the previous three years. In the absence of other data, little can be said about the status of these stocks except that the absolute catch values do not seem extremely high for an area of the general size of JA.

No information on SFR is available for Kyphosus vaigiensis, Mulloides flavolineatus, Scarus perspicillatus, or Ctenochaetus strigosus. All their catches are insignificant compared to their respective populations.

Based on the available Hawaiian values for SFR, our data suggest that approximately 30% of Pseudupeneus bifasciatus*, 1% of P. cyclostomus*, and 3% of Acanthurus triostegus are caught at sizes below their respective maximum SFR (based on data for all six years combined).

The total number of Pseudupeneus multifasciatus caught annually is not significant compared to the estimated standing stock (Table 9). Only one of the P. multifasciatus caught was below the SFR for females, but the male SFR falls in the range of sizes caught most frequently. Approximately 87% of the P. multifasciatus catch is below the maximum male SFR value.

About 82% of the Caranx melampygus catch is below the maximum SFR value. However, most of the individuals seen at the monitoring stations were much larger than the SFR. This seems to be due to the occasional presence of small schools of small individuals feeding near the piers of the islands where they are especially vulnerable to catch. The annual catch is very small compared to the standing stock.

When the 13 "major catch species" are considered as a group, the small size at capture of some species seems to offer some potential for concern if the catch levels were to increase greatly. In agreement with the results of the five previous phases, at present levels of effort, there appears to be very little impact on atoll fish populations as a result of fishing pressure.

The mandatory catch reporting system incorporated during the 1988 report year has resulted in higher reporting rates (compared with those of previous years) of invertebrates that previously went largely unreported. The catches of most species of coral and of total coral declined from last year, but comparisons with years prior to that would be misleading due to the substantial reduction in underreporting of boat catches that has resulted from the mandatory reporting system. However, the relatively small portion of the atoll accessible to coral collectors as well as the abundance of Acropora corals make it unlikely that the populations of these species will be threatened. A large majority of the coral populations (especially Distichopora sp., which is found primarily in the restricted area outside the barrier reef) lie outside the areas where recreational diving is permitted. In addition, the diurnally cryptic habits of most molluska popular with shell collectors are sufficient to prevent overcollection at the present low levels of fishing pressure. In

*Estimated from SFR for Pseudupeneus porphyreus.

spite of higher levels of reported catches compared with report years 1985-87, the major invertebrate catch species (coral, cephalopods, gastropods, crustaceans, and echinoderms) continue to be collected in insignificant numbers compared to their respective abundances.

Protected Species

Protected species occurring at JA are the threatened green sea turtle (Chelonia mydas) and the endangered Hawaiian monk seal (Monachus schauinslandi). Turtles are most often found in the vicinity of Zones 11 and 12. This is the area where their major food source, the algae (Caulerpa spp.), occurs in abundance. Turtles are also seen occasionally throughout the lagoon and channel areas. One turtle was censused in April 1986 at Station P5. Hawaiian monk seals have been seen occasionally by residents at various locations throughout JA over the past several years. In November 1984, nine male monk seals were brought to JA from Laysan Island. At last report, none of these monk seals appears to have remained at JA; the last reported sighting was in the summer of 1986. Most of the other monk seals have not been seen since shortly after their arrival.

DEEP SEA FISHING

Although the scope of this project and report focuses on the lagoon and shallow platform waters, a brief discussion of the fishery for pelagic species of the deep waters surrounding the atoll as a whole will complete the picture of atoll fisheries. Deep sea fishing at JA is done from several landing craft ~13 m long (known locally as "Mike boats"), operated by port control personnel. All deep sea fishing is for recreational purposes and is done on weekends only. One or two "Mike boats" with five to seven residents and/or transient personnel each, go out Saturday and Sunday (weather permitting) for three to four hours. Table 10 presents rough annual catch estimates for the fish species occurring in the deep sea catch during Jun 89 - May 90 (1990), Jun 88 - May 89 (1989), Jun 87 - May 88 (1988), Jun 86 - May 87 (1987), Jun 83 - May 86 (1986), and Feb 84 - May 85 (1985), based on catch reports and creel census. Little time and effort was spent collecting catch data for these trips. The data set is small, and no underreporting estimate was made for these deep sea catches. Although there is a broad decreasing trend in the estimated deep-sea catch over the period of Table 10, in the absence of effort data, little can be said about changes in the local abundance of these species. The deep sea catch at JA is essentially independent of the lagoon and its fishing activity. There is probably little or nothing that JA resource management can do that will affect these species significantly.

Table 10. Estimate of annual catch of deep sea species (uncorrected for underreporting).

SPECIES	ESTIMATED NO. CAUGHT					
	1990	1989	1988	1987	1986	1985
Acanthocybium solandri (wahoo)	136	149	180	175	201	201
Thunnus albacares (yellowfin tuna)	70	65	110	120	135	111
Sphyraena barracuda (great barracuda)	28	8	15	10	12	-
Katsuwonus pelamis (skipjack tuna)	23	29	60	50	90	134
Elagatis bipinnulatus (rainbow runner)	13	15	20	15	15	6
Coryphaena hippurus (dolphin)	5	6	10	6	8	5

SUMMARY

Environmental studies in the lagoon at Johnston Atoll continued through the project year in an attempt to detect any effects of JACADS activities (including any increase in recreational fishing) on the marine ecosystem. Established, long-term stations were monitored by visual, underwater censuses of fish and invertebrates. Catch and effort of the recreational fishery were monitored by use of catch reports completed by fishermen and by direct observation of fishing activity. Samples of the catch were examined to determine species and size composition.

Of the five stations censused, the three that appeared visually to provide similar habitat (Stations P1, P5, and P6) had similar fish communities, even though Station P5 was much more heavily fished than the physically very similar Station P6. Stations P3 and P7, which appeared visually different in habitat from each other and from the preceding stations, had distinctly different fish communities. Results of analyses by both similarity index and paired t-tests indicated these results. Similarity index analysis indicated relatively high levels of similarity within each station over the six years of the study, suggesting that activities related to JACADS development had not made a detectable change in these fish communities. The time series of population size as estimated by census was analyzed for temporal trends by two methods of correlation/regression. It seems likely that there has been a decreasing trend in the total number of fish and in the numbers of a good many species over the six years of the study. The changes do not seem associated with fishing, and there is no evidence to link them with any other human activity. It seems likely that this is a natural phenomenon, perhaps related to variability in recruitment. The available data on this apparently natural variability provide a valuable baseline for comparison with changes in fish populations that may occur in the future.

Fourteen fish species, octopus, and a few species of decorative coral made up the bulk of the recreational fishery. A

few decorative shelled mollusc species, lobsters, and occasional other invertebrates were also collected, as well as a few individuals of many other fish species. Comparing years was difficult because of variable underreporting of catch and effort. However, there seemed to be no evidence of significant or consistent increase in either total catch or effort over the six years of the study (despite a more than three-fold increase in JA human population at maximum). Most transient changes in catch seem to be explained by corresponding changes in effort. For all the major fish species caught, the total annual catch was small compared to the estimated size of the species population. Continued fishing at levels observed during the study is unlikely to affect the fish populations seriously. Increases reported in the 1989 catch of several invertebrates (e.g., corals, shelled molluscs, octopus) may reflect an artifact of reporting by fishermen. Catches of most of these species declined somewhat in the present year, but the trend will bear watching in future years.

The serious problem with compliance by boat fishermen with the catch reporting system during the year ending 1987 has largely been remedied. Mandatory catch reporting was incorporated into the sign-out/return procedure for recreational boat use in the year ending 1988, and the requirement for reporting all types of animals caught was stressed. Catch estimates for the past two years based on boat catch reports are believed to be reasonably accurate; the loss of data from previous years is irreparable and will continue to hamper analysis and interpretation of temporal trends. It is essential that compliance with reporting requirements for all catch be maintained high in order that the studies on the fishery can produce meaningful results. This issue must receive the necessary attention and continuing effective supervision by JA management if the project is to succeed.

During the project year, it became clear that compliance with reporting of shoreline catch and effort had deteriorated to the point that the data were not reliable for making the main quantitative estimates useful for management decisions. Compliance by fisherman cannot be enforced by project staff, and it is not feasible for project staff to collect the data directly. In response to our report of this status, JA administration indicated that they would not enforce compliance nor apply other means to secure shoreline catch and/or effort data. The attempt to use such data for quantitative analysis in the project has therefore been abandoned, and the effects of shoreline fishing on the fish stocks will remain unknown.

As of the end of the project year, the JACADS facility was just beginning operation, so monitoring of any environmental effects due to operation is still to come. A good baseline has been acquired, and no effects of construction have been detected. Lack of effects on the fishery may be due to a lack of increased fishing effort; it is not clear what the trend of human population and fishing effort will be in the future. However, if the effects of any future changes due to plant operation or fishing are to be detected, the study program presented here must be continued using much the same sampling methods and analyses.

Appendix B

TABLE B-1. Estimated 1-Hour Average Concentrations of Vapor-Phase TCDD at Receptor Locations (x, y Coordinates) Around the Perimeter of the Herbicide Orange Site.

X Coordinate (m)	Y Coordinate (m)	1-Hour Average Concentration (g/m^3)
0.000	0.000	0.10869E-05
0.000	6.096	0.71606E-06
0.000	12.192	0.15821E-05
0.000	18.288	0.92511E-06
0.000	24.384	0.13211E-05
0.000	30.480	0.11776E-05
0.000	36.576	0.17014E-05
0.000	42.672	0.89331E-06
0.000	48.768	0.12986E-05
0.000	54.864	0.10935E-05
0.000	60.960	0.10394E-05
0.000	67.056	0.23507E-05
0.000	73.152	0.72389E-05
0.000	79.248	0.25512E-05
0.000	85.344	0.66881E-05
0.000	91.440	0.23620E-05
0.000	97.536	0.19368E-05
0.000	103.632	0.17130E-05
0.000	109.728	0.19697E-05
0.000	115.824	0.12683E-05
0.000	121.920	0.12411E-05
6.096	121.920	0.82771E-06
12.192	121.920	0.17928E-05
18.288	121.920	0.25317E-05
24.384	121.920	0.13754E-05
30.480	121.920	0.33187E-05
36.576	121.920	0.65311E-05
42.672	121.920	0.70387E-05
48.768	121.920	0.41036E-05
54.864	121.920	0.33110E-05
60.960	121.920	0.42264E-05
67.056	121.920	0.64511E-05
73.152	121.920	0.58688E-05
79.248	121.920	0.34911E-05
85.344	121.920	0.46393E-05
91.440	121.920	0.28861E-05
97.536	121.920	0.66784E-05

TABLE B-1. Estimated 1-Hour Average Concentrations of Vapor-Phase TCDD at Receptor Locations (x, y Coordinates) Around the Perimeter of the Herbicide Orange Site. (Continued)

X Coordinate (m)	Y Coordinate (m)	1-Hour Average Concentration (g/m^3)
103.632	121.920	0.27536E-05
109.728	121.920	0.67676E-05
115.824	121.920	0.27149E-05
121.920	121.920	0.54310E-05
128.016	121.920	0.22306E-05
134.112	121.920	0.52685E-05
140.208	121.920	0.20922E-05
146.304	121.920	0.47859E-05
152.400	121.920	0.16793E-05
158.496	121.920	0.40241E-05
164.592	121.920	0.23911E-05
170.688	121.920	0.73955E-05
176.784	121.920	0.26016E-05
182.880	121.920	0.77590E-05
188.976	121.920	0.27115E-05
195.072	121.920	0.10147E-05
195.072	115.824	0.29191E-05
195.072	109.728	0.84478E-05
195.072	103.632	0.32479E-05
195.072	97.536	0.81633E-05
195.072	91.440	0.27307E-05
195.072	85.344	0.51753E-05
195.072	79.248	0.21901E-05
195.072	73.152	0.52978E-05
195.072	67.056	0.18375E-05
195.072	60.960	0.10187E-05
188.976	60.960	0.20641E-05
182.880	60.960	0.48878E-05
176.784	60.960	0.17248E-05
170.688	60.960	0.45996E-05
164.592	60.960	0.37120E-05
158.496	60.960	0.93241E-05
152.400	60.960	0.36129E-05
146.304	60.960	0.93482E-05
146.304	54.864	0.33913E-05
146.304	48.768	0.34357E-05

TABLE B-1. Estimated 1-Hour Average Concentrations of Vapor-Phase TCDD at Receptor Locations (x, y Coordinates) Around the Perimeter of the Herbicide Orange Site. (Continued)

X Coordinate (m)	Y Coordinate (m)	1-Hour Average Concentration (g/m^3)
146.304	42.672	0.14158E-05
146.304	36.576	0.34726E-05
146.304	30.480	0.24876E-05
146.304	24.384	0.24098E-05
146.304	18.288	0.14316E-05
146.304	12.192	0.20872E-05
146.304	6.096	0.27877E-05
146.304	0.000	0.32758E-05
140.208	0.000	0.35537E-05
134.112	0.000	0.10083E-04
128.016	0.000	0.39054E-05
121.920	0.000	0.85703E-05
115.824	0.000	0.31626E-05
109.728	0.000	0.71679E-05
103.632	0.000	0.28354E-05
97.536	0.000	0.78186E-05
91.440	0.000	0.32782E-05
85.344	0.000	0.67743E-05
79.248	0.000	0.26083E-05
73.152	0.000	0.73547E-05
67.056	0.000	0.27275E-05
60.096	0.000	0.62408E-05
54.864	0.000	0.22823E-05
48.768	0.000	0.19447E-05
42.672	0.000	0.15307E-05
36.576	0.000	0.40823E-05
30.480	0.000	0.17803E-05
24.384	0.000	0.45009E-05
18.288	0.000	0.19206E-05
12.192	0.000	0.13845E-05
6.096	0.000	0.80730E-06

TABLE B-2. Estimated 1-Hour Average Concentrations of Vapor-Phase 2,4-D at Receptor Locations (x, y Coordinates) Around the Perimeter of the Herbicide Orange Site.

X Coordinate (m)	Y Coordinate (m)	1-Hour Average Concentration (g/m^3)
0.000	0.000	0.19477E-02
0.000	6.096	0.39631E-02
0.000	12.192	0.26655E-02
0.000	18.288	0.97173E-02
0.000	24.384	0.70420E-02
0.000	30.480	0.25542E-01
0.000	36.576	0.67556E-01
0.000	42.672	0.26382E-01
0.000	48.768	0.67592E-01
0.000	54.864	0.25488E-01
0.000	60.960	0.69852E-02
0.000	67.056	0.96678E-02
0.000	73.152	0.26252E-02
0.000	79.248	0.46039E-02
0.000	85.344	0.19071E-02
0.000	91.440	0.55104E-02
0.000	97.536	0.33685E-02
0.000	103.632	0.40676E-02
0.000	109.728	0.60926E-02
0.000	115.824	0.21389E-02
0.000	121.920	0.61288E-02
6.096	121.920	0.60058E-02
12.192	121.920	0.49756E-02
18.288	121.920	0.30086E-02
24.384	121.920	0.67717E-02
30.480	121.920	0.25632E-01
36.576	121.920	0.18519E-01
42.672	121.920	0.67457E-01
48.768	121.920	0.18052E+00
54.864	121.920	0.67357E-01
60.960	121.920	0.17853E+00
67.056	121.920	0.67358E-01
73.152	121.920	0.18427E-01
79.248	121.920	0.25561E-01
85.344	121.920	0.66975E-02
91.440	121.920	0.29347E-02
97.536	121.920	0.49055E-02

TABLE B-2. Estimated 1-Hour Average Concentrations of Vapor-Phase 2,4-D at Receptor Locations (x, y Coordinates) Around the Perimeter of the Herbicide Orange Site. (continued)

X Coordinate (m)	Y Coordinate (m)	1-Hour Average Concentration (g/m^3)
103.632	121.920	0.59388E-02
109.728	121.920	0.60642E-02
115.824	121.920	0.56506E-02
121.920	121.920	0.50112E-02
128.016	121.920	0.43326E-02
134.112	121.920	0.37016E-02
140.208	121.920	0.31521E-02
146.304	121.920	0.26677E-02
152.400	121.920	0.23038E-02
158.496	121.920	0.40571E-02
164.592	121.920	0.20113E-02
170.688	121.920	0.24965E-02
176.784	121.920	0.13524E-02
182.880	121.920	0.16639E-02
188.976	121.920	0.19454E-02
195.072	121.920	0.19422E-02
195.072	115.824	0.15801E-02
195.072	109.728	0.15036E-02
195.072	103.632	0.19604E-02
195.072	97.536	0.21416E-02
195.072	91.440	0.17412E-02
195.072	85.344	0.13755E-02
195.072	79.248	0.14653E-02
195.072	73.152	0.25189E-02
195.072	67.056	0.13646E-02
195.072	60.960	0.14306E-02
188.976	60.960	0.12030E-02
182.880	60.960	0.14390E-02
176.784	60.960	0.20232E-02
170.688	60.960	0.18035E-02
164.592	60.960	0.22019E-02
158.496	60.960	0.33033E-02
152.400	60.960	0.47735E-02
146.304	60.960	0.58437E-02
146.304	54.864	0.21440E-02
146.304	48.768	0.63571E-02

TABLE B-2. Estimated 1-Hour Average Concentrations of Vapor-Phase 2,4-D at Receptor Locations (x, y Coordinates) Around the Perimeter of the Herbicide Orange Site. (continued)

X Coordinate (m)	Y Coordinate (m)	1-Hour Average Concentration (g/m^3)
146.304	42.672	0.14449E-02
146.304	36.576	0.63571E-02
146.304	30.480	0.17778E-02
146.304	24.384	0.58435E-02
146.304	18.288	0.12571E-02
146.304	12.192	0.50959E-02
146.304	6.096	0.18491E-02
146.304	0.000	0.26943E-02
140.208	0.000	0.52016E-02
134.112	0.000	0.29861E-02
128.016	0.000	0.19225E-02
121.920	0.000	0.86733E-02
115.824	0.000	0.20161E-02
109.728	0.000	0.90958E-02
103.632	0.000	0.28467E-02
97.536	0.000	0.93074E-02
91.440	0.000	0.12760E-01
85.344	0.000	0.62689E-02
79.248	0.000	0.32600E-02
73.152	0.000	0.62469E-02
67.056	0.000	0.11686E-01
60.096	0.000	0.91177E-02
54.864	0.000	0.28482E-02
48.768	0.000	0.90952E-02
42.672	0.000	0.22583E-02
36.576	0.000	0.86766E-02
30.480	0.000	0.19236E-02
24.384	0.000	0.30140E-02
18.288	0.000	0.52274E-02
12.192	0.000	0.27159E-02
6.096	0.000	0.17363E-02

TABLE E-3. Estimated 1-Hour Average Concentrations of Vapor-Phase 2,4,5-T at Receptor Locations (x, y Coordinates) Around the Perimeter of the Herbicide Orange Site.

X Coordinate (m)	Y Coordinate (m)	1-Hour Average Concentration (g/m^3)
0.000	0.000	0.40998E-02
0.000	6.096	0.30514E-02
0.000	12.192	0.51275E-02
0.000	18.288	0.18139E-01
0.000	24.384	0.13190E-01
0.000	30.480	0.47583E-01
0.000	36.576	0.12670E+00
0.000	42.672	0.48208E-01
0.000	48.768	0.12590E+00
0.000	54.864	0.47446E-01
0.000	60.960	0.13036E-01
0.000	67.056	0.18007E-01
0.000	73.152	0.50186E-02
0.000	79.248	0.53689E-02
0.000	85.344	0.39782E-02
0.000	91.440	0.60774E-02
0.000	97.536	0.44624E-02
0.000	103.632	0.45234E-02
0.000	109.728	0.68376E-02
0.000	115.824	0.32081E-02
0.000	121.920	0.69038E-02
6.096	121.920	0.67646E-02
12.192	121.920	0.56497E-02
18.288	121.920	0.35287E-02
24.384	121.920	0.77137E-02
30.480	121.920	0.27982E-01
36.576	121.920	0.20313E-01
42.672	121.920	0.73438E-01
48.768	121.920	0.20046E+00
54.864	121.920	0.73290E-01
60.960	121.920	0.19416E+00
67.056	121.920	0.73231E-01
73.152	121.920	0.20133E-01
79.248	121.920	0.27832E-01
85.344	121.920	0.11522E-01
91.440	121.920	0.40237E-02
97.536	121.920	0.55060E-02

TABLE B-3. Estimated 1-Hour Average Concentrations of Vapor-Phase 2,4,5-T at Receptor Locations (x, y Coordinates) Around the Perimeter of the Herbicide Orange Site. (continued)

X Coordinate (m)	Y Coordinate (m)	1-Hour Average Concentration (g/m^3)
103.632	121.920	0.66277E-02
109.728	121.920	0.67720E-02
115.824	121.920	0.63372E-02
121.920	121.920	0.56586E-02
128.016	121.920	0.49383E-02
134.112	121.920	0.42681E-02
140.208	121.920	0.36863E-02
146.304	121.920	0.43281E-02
152.400	121.920	0.27947E-02
158.496	121.920	0.58916E-02
164.592	121.920	0.22107E-02
170.688	121.920	0.47877E-02
176.784	121.920	0.22674E-02
182.880	121.920	0.17181E-02
188.976	121.920	0.18273E-02
195.072	121.920	0.17304E-02
195.072	115.824	0.13019E-02
195.072	109.728	0.12953E-02
195.072	103.632	0.18293E-02
195.072	97.536	0.18026E-02
195.072	91.440	0.16046E-02
195.072	85.344	0.10864E-02
195.072	79.248	0.19185E-02
195.072	73.152	0.20209E-02
195.072	67.056	0.12299E-02
195.072	60.960	0.13806E-02
188.976	60.960	0.13524E-02
182.880	60.960	0.15511E-02
176.784	60.960	0.32245E-02
170.688	60.960	0.37882E-02
164.592	60.960	0.35080E-02
158.496	60.960	0.38885E-02
152.400	60.960	0.37995E-02
146.304	60.960	0.44871E-02
146.304	54.864	0.29576E-02
146.304	48.768	0.49692E-02

TABLE B-3. Estimated 1-Hour Average Concentrations of Vapor-Phase 2,4,5-T at Receptor Locations (x, y Coordinates) Around the Perimeter of the Herbicide Orange Site. (continued)

X Coordinate (m)	Y Coordinate (m)	1-Hour Average Concentration (g/m^3)
146.304	42.672	0.22117E-02
146.304	36.576	0.49690E-02
146.304	30.480	0.29834E-02
146.304	24.384	0.61789E-02
146.304	18.288	0.17737E-02
146.304	12.192	0.45863E-02
146.304	6.096	0.16218E-02
146.304	0.000	0.41072E-02
140.208	0.000	0.39609E-02
134.112	0.000	0.41424E-02
128.016	0.000	0.17067E-02
121.920	0.000	0.88917E-02
115.824	0.000	0.19144E-02
109.728	0.000	0.67356E-02
103.632	0.000	0.28113E-02
97.536	0.000	0.66454E-02
91.440	0.000	0.95679E-02
85.344	0.000	0.46037E-02
79.248	0.000	0.34387E-02
73.152	0.000	0.45487E-02
67.056	0.000	0.81558E-02
60.096	0.000	0.64398E-02
54.864	0.000	0.26765E-02
48.768	0.000	0.78736E-02
42.672	0.000	0.33379E-02
36.576	0.000	0.89007E-02
30.480	0.000	0.16248E-02
24.384	0.000	0.50808E-02
18.288	0.000	0.66992E-02
12.192	0.000	0.40998E-02
6.096	0.000	0.24769E-02

TABLE B-4. Estimated 8-Hour Average Concentrations of Vapor-Phase TCDD at Receptor Locations (x, y Coordinates) Around the Perimeter of the Herbicide Orange Site

X Coordinate (m)	Y Coordinate (m)	8-Hour Average Concentration (g/m^3)
0.000	0.000	0.76103E-06
0.000	6.096	0.50137E-06
0.000	12.192	0.11078E-05
0.000	18.288	0.64774E-06
0.000	24.384	0.92498E-06
0.000	30.480	0.82454E-06
0.000	36.576	0.11913E-05
0.000	42.672	0.62548E-06
0.000	48.768	0.90928E-06
0.000	54.864	0.76562E-06
0.000	60.960	0.72775E-06
0.000	67.056	0.16459E-05
0.000	73.152	0.50685E-05
0.000	79.248	0.17863E-05
0.000	85.344	0.46829E-05
0.000	91.440	0.16538E-05
0.000	97.536	0.13561E-05
0.000	103.632	0.11994E-05
0.000	109.728	0.13791E-05
0.000	115.824	0.88805E-06
0.000	121.920	0.86897E-06
6.096	121.920	0.57955E-06
12.192	121.920	0.12553E-05
18.288	121.920	0.17726E-05
24.384	121.920	0.96304E-06
30.480	121.920	0.23237E-05
36.576	121.920	0.45730E-05
42.672	121.920	0.49284E-05
48.768	121.920	0.28733E-05
54.864	121.920	0.23183E-05
60.960	121.920	0.29592E-05
67.056	121.920	0.45170E-05
73.152	121.920	0.41092E-05
79.248	121.920	0.24444E-05
85.344	121.920	0.32833E-05
91.440	121.920	0.20208E-05
97.536	121.920	0.46761E-05

TABLE B-4. Estimated 8-Hour Average Concentrations of Vapor-Phase TCDD at Receptor Locations (x, y Coordinates) Around the Perimeter of the Herbicide Orange Site (continued)

X Coordinate (m)	Y Coordinate (m)	8-Hour Average Concentration (g/m^3)
103.632	121.920	0.19280E-05
109.728	121.920	0.47386E-05
115.824	121.920	0.19009E-05
121.920	121.920	0.38027E-05
128.016	121.920	0.15618E-05
134.112	121.920	0.36889E-05
140.208	121.920	0.14649E-05
146.304	121.920	0.33510E-05
152.400	121.920	0.11758E-05
158.496	121.920	0.28176E-05
164.592	121.920	0.16742E-05
170.688	121.920	0.51782E-05
176.784	121.920	0.18216E-05
182.880	121.920	0.54327E-05
188.976	121.920	0.18986E-05
195.072	121.920	0.71046E-06
195.072	115.824	0.20439E-05
195.072	109.728	0.59150E-05
195.072	103.632	0.22741E-05
195.072	97.536	0.57158E-05
195.072	91.440	0.19120E-05
195.072	85.344	0.36236E-05
195.072	79.248	0.15334E-05
195.072	73.152	0.37094E-05
195.072	67.056	0.12366E-05
195.072	60.960	0.71327E-06
188.976	60.960	0.14452E-05
182.880	60.960	0.34224E-05
176.784	60.960	0.12077E-05
170.688	60.960	0.32206E-05
164.592	60.960	0.25990E-05
158.496	60.960	0.65285E-05
152.400	60.960	0.25297E-05
146.304	60.960	0.65454E-05
146.304	54.864	0.23745E-05
146.304	48.768	0.24056E-05

TABLE B-4. Estimated 8-Hour Average Concentrations of Vapor-Phase TCDD at Receptor Locations (x, y Coordinates) Around the Perimeter of the Herbicide Orange Site (continued)

X Coordinate (m)	Y Coordinate (m)	8-Hour Average Concentration (g/m^3)
146.304	42.672	0.99130E-06
146.304	36.576	0.24315E-05
146.304	30.480	0.17418E-05
146.304	24.384	0.16873E-05
146.304	18.288	0.10024E-05
146.304	12.192	0.14614E-05
146.304	6.096	0.19519E-05
146.304	0.000	0.22937E-05
140.208	0.000	0.24882E-05
134.112	0.000	0.70600E-05
128.016	0.000	0.27345E-05
121.920	0.000	0.60008E-05
115.824	0.000	0.22144E-05
109.728	0.000	0.50188E-05
103.632	0.000	0.19853E-05
97.536	0.000	0.54744E-05
91.440	0.000	0.22953E-05
85.344	0.000	0.47432E-05
79.248	0.000	0.18263E-05
73.152	0.000	0.51496E-05
67.056	0.000	0.19097E-05
60.096	0.000	0.43697E-05
54.864	0.000	0.15980E-05
48.768	0.000	0.13617E-05
42.672	0.000	0.10718E-05
36.576	0.000	0.28584E-05
30.480	0.000	0.12465E-05
24.384	0.000	0.31514E-05
18.288	0.000	0.13448E-05
12.192	0.000	0.96941E-06
6.096	0.000	0.56525E-06

TABLE B-5. Estimated 8-Hour Average Concentrations of Vapor-Phase 2,4-D at Receptor Locations (x, y Coordinates) Around the Perimeter of the Herbicide Orange Site.

X Coordinate (m)	Y Coordinate (m)	8-Hour Average Concentration (g/m^3)
0.000	0.000	0.13637E-02
0.000	6.096	0.27748E-02
0.000	12.192	0.18663E-02
0.000	18.288	0.68037E-02
0.000	24.384	0.49306E-02
0.000	30.480	0.17884E-01
0.000	36.576	0.47511E-01
0.000	42.672	0.18472E-01
0.000	48.768	0.47326E-01
0.000	54.864	0.17846E-01
0.000	60.960	0.48908E-02
0.000	67.056	0.67691E-02
0.000	73.152	0.18381E-02
0.000	79.248	0.32235E-02
0.000	85.344	0.13353E-02
0.000	91.440	0.38582E-02
0.000	97.536	0.23585E-02
0.000	103.632	0.28480E-02
0.000	109.728	0.42659E-02
0.000	115.824	0.14976E-02
0.000	121.920	0.42912E-02
6.096	121.920	0.42051E-02
12.192	121.920	0.34838E-02
18.288	121.920	0.21065E-02
24.384	121.920	0.47413E-02
30.480	121.920	0.17947E-01
36.576	121.920	0.12966E-01
42.672	121.920	0.47231E-01
48.768	121.920	0.12640E+00
54.864	121.920	0.47161E-01
60.960	121.920	0.12500E+00
67.056	121.920	0.47162E-01
73.152	121.920	0.12902E-01
79.248	121.920	0.17897E-01
85.344	121.920	0.46894E-02
91.440	121.920	0.20548E-02
97.536	121.920	0.34347E-02

TABLE B-5. Estimated 8-Hour Average Concentrations of Vapor-Phase 2,4-D at Receptor Locations (x, y Coordinates) Around the Perimeter of the Herbicide Orange Site. (continued)

X Coordinate (m)	Y Coordinate (m)	8-Hour Average Concentration (g/m^3)
103.632	121.920	0.41582E-02
109.728	121.920	0.42460E-02
115.824	121.920	0.39563E-02
121.920	121.920	0.35087E-02
128.016	121.920	0.30336E-02
134.112	121.920	0.25918E-02
140.208	121.920	0.22070E-02
146.304	121.920	0.18819E-02
152.400	121.920	0.16131E-02
158.496	121.920	0.28406E-02
164.592	121.920	0.14082E-02
170.688	121.920	0.17480E-02
176.784	121.920	0.94689E-03
182.880	121.920	0.11650E-02
188.976	121.920	0.13621E-02
195.072	121.920	0.13599E-02
195.072	115.824	0.11063E-02
195.072	109.728	0.10528E-02
195.072	103.632	0.13726E-02
195.072	97.536	0.14995E-02
195.072	91.440	0.12191E-02
195.072	85.344	0.96305E-03
195.072	79.248	0.10260E-02
195.072	73.152	0.17636E-02
195.072	67.056	0.95543E-03
195.072	60.960	0.10017E-02
188.976	60.960	0.84227E-03
182.880	60.960	0.10075E-02
176.784	60.960	0.14166E-02
170.688	60.960	0.12627E-02
164.592	60.960	0.15417E-02
158.496	60.960	0.23129E-02
152.400	60.960	0.33423E-02
146.304	60.960	0.40916E-02
146.304	54.864	0.15012E-02
146.304	48.768	0.44511E-02

TABLE B-5. Estimated 8-Hour Average Concentrations of Vapor-Phase 2,4-D at Receptor Locations (x, y Coordinates) Around the Perimeter of the Herbicide Orange Site. (continued)

X Coordinate (m)	Y Coordinate (m)	8-Hour Average Concentration (g/m^3)
146.304	42.672	0.10117E-02
146.304	36.576	0.44510E-02
146.304	30.480	0.12448E-02
146.304	24.384	0.40915E-02
146.304	18.288	0.88019E-03
146.304	12.192	0.35680E-02
146.304	6.096	0.12946E-02
146.304	0.000	0.18865E-02
140.208	0.000	0.36420E-02
134.112	0.000	0.20908E-02
128.016	0.000	0.13461E-02
121.920	0.000	0.60728E-02
115.824	0.000	0.14116E-02
109.728	0.000	0.63686E-02
103.632	0.000	0.19931E-02
97.536	0.000	0.65167E-02
91.440	0.000	0.89339E-02
85.344	0.000	0.43893E-02
79.248	0.000	0.22826E-02
73.152	0.000	0.43739E-02
67.056	0.000	0.81820E-02
60.096	0.000	0.63839E-02
54.864	0.000	0.19942E-02
48.768	0.000	0.63682E-02
42.672	0.000	0.15812E-02
36.576	0.000	0.60751E-02
30.480	0.000	0.13468E-02
24.384	0.000	0.21103E-02
18.288	0.000	0.36601E-02
12.192	0.000	0.19016E-02
6.096	0.000	0.12157E-02

TABLE B-6. Estimated 8-Hour Average Concentrations of Vapor-Phase 2,4,5-T at Receptor Locations (x, y Coordinates) Around the Perimeter of the Herbicide Orange Site

X Coordinate (m)	Y Coordinate (m)	8-Hour Average Concentration (g/m^3)
0.000	0.000	0.28706E-02
0.000	6.096	0.21365E-02
0.000	12.192	0.35901E-02
0.000	18.288	0.12700E-01
0.000	24.384	0.92349E-02
0.000	30.480	0.33319E-01
0.000	36.576	0.88714E-01
0.000	42.672	0.33753E-01
0.000	48.768	0.88149E-01
0.000	54.864	0.33220E-01
0.000	60.960	0.91275E-02
0.000	67.056	0.12608E-01
0.000	73.152	0.35139E-02
0.000	79.248	0.37591E-02
0.000	85.344	0.27854E-02
0.000	91.440	0.42552E-02
0.000	97.536	0.31244E-02
0.000	103.632	0.31671E-02
0.000	109.728	0.47875E-02
0.000	115.824	0.23162E-02
0.000	121.920	0.48338E-02
6.096	121.920	0.47363E-02
12.192	121.920	0.39557E-02
18.288	121.920	0.24707E-02
24.384	121.920	0.54009E-02
30.480	121.920	0.19592E-01
36.576	121.920	0.14222E-01
42.672	121.920	0.51419E-01
48.768	121.920	0.14036E+00
54.864	121.920	0.51315E-01
60.960	121.920	0.13595E+00
67.056	121.920	0.51274E-01
73.152	121.920	0.14097E-01
79.248	121.920	0.19487E-01
85.344	121.920	0.80671E-02
91.440	121.920	0.28173E-02
97.536	121.920	0.38551E-02

TABLE B-6. Estimated 8-Hour Average Concentrations of Vapor-Phase 2,4,5-T at Receptor Locations (x, y Coordinates) Around the Perimeter of the Herbicide Orange Site (continued)

X Coordinate (m)	Y Coordinate (m)	8-Hour Average Concentration (g/m^3)
103.632	121.920	0.46405E-02
109.728	121.920	0.47415E-02
115.824	121.920	0.44371E-02
121.920	121.920	0.39620E-02
128.016	121.920	0.34576E-02
134.112	121.920	0.29884E-02
140.208	121.920	0.25810E-02
146.304	121.920	0.30304E-02
152.400	121.920	0.19568E-02
158.496	121.920	0.41251E-02
164.592	121.920	0.15478E-02
170.688	121.920	0.33522E-02
176.784	121.920	0.15876E-02
182.880	121.920	0.12029E-02
188.976	121.920	0.12794E-02
195.072	121.920	0.12116E-02
195.072	115.824	0.91155E-03
195.072	109.728	0.90693E-03
195.072	103.632	0.12808E-02
195.072	97.536	0.12621E-02
195.072	91.440	0.11235E-02
195.072	85.344	0.76067E-03
195.072	79.248	0.13433E-02
195.072	73.152	0.14150E-02
195.072	67.056	0.86112E-03
195.072	60.960	0.96662E-03
188.976	60.960	0.94691E-03
182.880	60.960	0.10860E-02
176.784	60.960	0.22577E-02
170.688	60.960	0.26523E-02
164.592	60.960	0.24562E-02
158.496	60.960	0.27226E-02
152.400	60.960	0.26603E-02
146.304	60.960	0.31417E-02
146.304	54.864	0.20708E-02
146.304	48.768	0.34792E-02

TABLE B-6. Estimated 8-Hour Average Concentrations of Vapor-Phase 2,4,5-T at Receptor Locations (x, y Coordinates) Around the Perimeter of the Herbicide Orange Site (continued)

X Coordinate (m)	Y Coordinate (m)	8-Hour Average Concentration (g/m^3)
146.304	42.672	0.15485E-02
146.304	36.576	0.34791E-02
146.304	30.480	0.20889E-02
146.304	24.384	0.43263E-02
146.304	18.288	0.12419E-02
146.304	12.192	0.32112E-02
146.304	6.096	0.11355E-02
146.304	0.000	0.28757E-02
140.208	0.000	0.27733E-02
134.112	0.000	0.29004E-02
128.016	0.000	0.11950E-02
121.920	0.000	0.62257E-02
115.824	0.000	0.13404E-02
109.728	0.000	0.47161E-02
103.632	0.000	0.19684E-02
97.536	0.000	0.46529E-02
91.440	0.000	0.66992E-02
85.344	0.000	0.32234E-02
79.248	0.000	0.24077E-02
73.152	0.000	0.31848E-02
67.056	0.000	0.57105E-02
60.096	0.000	0.45089E-02
54.864	0.000	0.18740E-02
48.768	0.000	0.55128E-02
42.672	0.000	0.23371E-02
36.576	0.000	0.62320E-02
30.480	0.000	0.11376E-02
24.384	0.000	0.35574E-02
18.288	0.000	0.46905E-02
12.192	0.000	0.28706E-02
6.096	0.000	0.17343E-02

TABLE B-7. Estimated Annual Average Concentrations of Vapor-Phase TCDD at Receptor Locations (x, y Coordinates) Around the Perimeter of the Herbicide Orange Site

X Coordinate (m)	Y Coordinate (m)	Annual Average Concentration (g/m^3)
0.000	0.000	0.27181E-07
0.000	6.096	0.17907E-07
0.000	12.192	0.39565E-07
0.000	18.288	0.23135E-07
0.000	24.384	0.33036E-07
0.000	30.480	0.29449E-07
0.000	36.576	0.42548E-07
0.000	42.672	0.22340E-07
0.000	48.768	0.32476E-07
0.000	54.864	0.27345E-07
0.000	60.960	0.25992E-07
0.000	67.056	0.58786E-07
0.000	73.152	0.18103E-06
0.000	79.248	0.63799E-07
0.000	85.344	0.16725E-06
0.000	91.440	0.59067E-07
0.000	97.536	0.48434E-07
0.000	103.632	0.42838E-07
0.000	109.728	0.49257E-07
0.000	115.824	0.31717E-07
0.000	121.920	0.31036E-07
6.096	121.920	0.20699E-07
12.192	121.920	0.44833E-07
18.288	121.920	0.63311E-07
24.384	121.920	0.34396E-07
30.480	121.920	0.82992E-07
36.576	121.920	0.16333E-06
42.672	121.920	0.17602E-06
48.768	121.920	0.10262E-06
54.864	121.920	0.82799E-07
60.960	121.920	0.10569E-06
67.056	121.920	0.16133E-06
73.152	121.920	0.14676E-06
79.248	121.920	0.87302E-07
85.344	121.920	0.11727E-06
91.440	121.920	0.72175E-07
97.536	121.920	0.16701E-06

TABLE B-7. Estimated Annual Average Concentrations of Vapor-Phase TCDD at Receptor Locations (x, y Coordinates) Around the Perimeter of the Herbicide Orange Site (continued)

X Coordinate (m)	Y Coordinate (m)	Annual Average Concentration (g/m^3)
103.632	121.920	0.68860E-07
109.728	121.920	0.16924E-06
115.824	121.920	0.67892E-07
121.920	121.920	0.13581E-06
128.016	121.920	0.55781E-07
134.112	121.920	0.13175E-06
140.208	121.920	0.52321E-07
146.304	121.920	0.11968E-06
152.400	121.920	0.41996E-07
158.496	121.920	0.10063E-06
164.592	121.920	0.59795E-07
170.688	121.920	0.18494E-06
176.784	121.920	0.65060E-07
182.880	121.920	0.19403E-06
188.976	121.920	0.67809E-07
195.072	121.920	0.25374E-07
195.072	115.824	0.72998E-07
195.072	109.728	0.21126E-06
195.072	103.632	0.81222E-07
195.072	97.536	0.20414E-06
195.072	91.440	0.68287E-07
195.072	85.344	0.12942E-06
195.072	79.248	0.54768E-07
195.072	73.152	0.13249E-06
195.072	67.056	0.45951E-07
195.072	60.960	0.25475E-07
188.976	60.960	0.51618E-07
182.880	60.960	0.12223E-06
176.784	60.960	0.43134E-07
170.688	60.960	0.11503E-06
164.592	60.960	0.92827E-07
158.496	60.960	0.23317E-06
152.400	60.960	0.90349E-07
146.304	60.960	0.23377E-06
146.304	54.864	0.84808E-07
146.304	48.768	0.85918E-07

TABLE B-7. Estimated Annual Average Concentrations of Vapor-Phase TCDD at Receptor Locations (x, y Coordinates) Around the Perimeter of the Herbicide Orange Site (continued)

X Coordinate (m)	Y Coordinate (m)	Annual Average Concentration (g/m^3)
146.304	42.672	0.35405E-07
146.304	36.576	0.86842E-07
146.304	30.480	0.62209E-07
146.304	24.384	0.60264E-07
146.304	18.288	0.35802E-07
146.304	12.192	0.52195E-07
146.304	6.096	0.69714E-07
146.304	0.000	0.81921E-07
140.208	0.000	0.88869E-07
134.112	0.000	0.25215E-06
128.016	0.000	0.97665E-07
121.920	0.000	0.21432E-06
115.824	0.000	0.79088E-07
109.728	0.000	0.17925E-06
103.632	0.000	0.70906E-07
97.536	0.000	0.19552E-06
91.440	0.000	0.81979E-07
85.344	0.000	0.16941E-06
79.248	0.000	0.65227E-07
73.152	0.000	0.18392E-06
67.056	0.000	0.68207E-07
60.096	0.000	0.15607E-06
54.864	0.000	0.57075E-07
48.768	0.000	0.48633E-07
42.672	0.000	0.38279E-07
36.576	0.000	0.10209E-06
30.480	0.000	0.44520E-07
24.384	0.000	0.11256E-06
18.288	0.000	0.48030E-07
12.192	0.000	0.34623E-07
6.096	0.000	0.20188E-07

TABLE B-8. Estimated Annual Average Concentrations of Vapor-Phase 2,4-D at Receptor Locations (x, y Coordinates) Around the Perimeter of the Herbicide Orange Site

X Coordinate (m)	Y Coordinate (m)	Annual Average Concentration (g/m^3)
0.000	0.000	0.48697E-04
0.000	6.096	0.99089E-04
0.000	12.192	0.66645E-04
0.000	18.288	0.24296E-03
0.000	24.384	0.17607E-03
0.000	30.480	0.63862E-03
0.000	36.576	0.16966E-02
0.000	42.672	0.65962E-03
0.000	48.768	0.16900E-02
0.000	54.864	0.63726E-03
0.000	60.960	0.17465E-03
0.000	67.056	0.24172E-03
0.000	73.152	0.65633E-04
0.000	79.248	0.11511E-03
0.000	85.344	0.47684E-04
0.000	91.440	0.13777E-03
0.000	97.536	0.84221E-04
0.000	103.632	0.10170E-03
0.000	109.728	0.15233E-03
0.000	115.824	0.53477E-04
0.000	121.920	0.15324E-03
6.096	121.920	0.15016E-03
12.192	121.920	0.12440E-03
18.288	121.920	0.75222E-04
24.384	121.920	0.16931E-03
30.480	121.920	0.64088E-03
36.576	121.920	0.46302E-03
42.672	121.920	0.16866E-02
48.768	121.920	0.45136E-02
54.864	121.920	0.16841E-02
60.960	121.920	0.44637E-02
67.056	121.920	0.16842E-02
73.152	121.920	0.46073E-03
79.248	121.920	0.63910E-03
85.344	121.920	0.16746E-03
91.440	121.920	0.73376E-04
97.536	121.920	0.12265E-03

TABLE B-8. Estimated Annual Average Concentrations of Vapor-Phase 2,4-D at Receptor Locations (x, y Coordinates) Around the Perimeter of the Herbicide Orange Site (continued)

X Coordinate (m)	Y Coordinate (m)	Annual Average Concentration (g/m^3)
103.632	121.920	0.14849E-03
109.728	121.920	0.15162E-03
115.824	121.920	0.14128E-03
121.920	121.920	0.12530E-03
128.016	121.920	0.10833E-03
134.112	121.920	0.92551E-04
140.208	121.920	0.78812E-04
146.304	121.920	0.67201E-04
152.400	121.920	0.57602E-04
158.496	121.920	0.10144E-03
164.592	121.920	0.50288E-04
170.688	121.920	0.62420E-04
176.784	121.920	0.33813E-04
182.880	121.920	0.41601E-04
188.976	121.920	0.48641E-04
195.072	121.920	0.48562E-04
195.072	115.824	0.39507E-04
195.072	109.728	0.37595E-04
195.072	103.632	0.49014E-04
195.072	97.536	0.53545E-04
195.072	91.440	0.43535E-04
195.072	85.344	0.34390E-04
195.072	79.248	0.36637E-04
195.072	73.152	0.62979E-04
195.072	67.056	0.34118E-04
195.072	60.960	0.35770E-04
188.976	60.960	0.30077E-04
182.880	60.960	0.35979E-04
176.784	60.960	0.50585E-04
170.688	60.960	0.45091E-04
164.592	60.960	0.55053E-04
158.496	60.960	0.82593E-04
152.400	60.960	0.11935E-03
146.304	60.960	0.14611E-03
146.304	54.864	0.53606E-04
146.304	48.768	0.15895E-03

TABLE B-8. Estimated Annual Average Concentrations of Vapor-Phase 2,4-D at Receptor Locations (x, y Coordinates) Around the Perimeter of the Herbicide Orange Site (continued)

X Coordinate (m)	Y Coordinate (m)	Annual Average Concentration (g/m^3)
146.304	42.672	0.36126E-04
146.304	36.576	0.15895E-03
146.304	30.480	0.44450E-04
146.304	24.384	0.14610E-03
146.304	18.288	0.31431E-04
146.304	12.192	0.12741E-03
146.304	6.096	0.46232E-04
146.304	0.000	0.67366E-04
140.208	0.000	0.13006E-03
134.112	0.000	0.74662E-04
128.016	0.000	0.48068E-04
121.920	0.000	0.21686E-03
115.824	0.000	0.50407E-04
109.728	0.000	0.22742E-03
103.632	0.000	0.71174E-04
97.536	0.000	0.23271E-03
91.440	0.000	0.31903E-03
85.344	0.000	0.15674E-03
79.248	0.000	0.81509E-04
73.152	0.000	0.15619E-03
67.056	0.000	0.29218E-03
60.096	0.000	0.22797E-03
54.864	0.000	0.71213E-04
48.768	0.000	0.22741E-03
42.672	0.000	0.56405E-04
36.576	0.000	0.21694E-03
30.480	0.000	0.48095E-04
24.384	0.000	0.75358E-04
18.288	0.000	0.13070E-03
12.192	0.000	0.67904E-04
6.096	0.000	0.43413E-04

TABLE B-9. Estimated Annual Average Concentrations of Vapor-Phase 2,4,5-T at Receptor Locations (x, y Coordinates) Around the Perimeter of the Herbicide Orange Site

X Coordinate (m)	Y Coordinate (m)	Annual Average Concentration (g/m^3)
0.000	0.000	0.10251E-03
0.000	6.096	0.76294E-04
0.000	12.192	0.12820E-03
0.000	18.288	0.45352E-03
0.000	24.384	0.32978E-03
0.000	30.480	0.11898E-02
0.000	36.576	0.31679E-02
0.000	42.672	0.12053E-02
0.000	48.768	0.31478E-02
0.000	54.864	0.11863E-02
0.000	60.960	0.32594E-03
0.000	67.056	0.45022E-03
0.000	73.152	0.12548E-03
0.000	79.248	0.13424E-03
0.000	85.344	0.99465E-04
0.000	91.440	0.15195E-03
0.000	97.536	0.11157E-03
0.000	103.632	0.11310E-03
0.000	109.728	0.17096E-03
0.000	115.824	0.82712E-04
0.000	121.920	0.17261E-03
6.096	121.920	0.16913E-03
12.192	121.920	0.14126E-03
18.288	121.920	0.88228E-04
24.384	121.920	0.19286E-03
30.480	121.920	0.69962E-03
36.576	121.920	0.50783E-03
42.672	121.920	0.18362E-02
48.768	121.920	0.50121E-02
54.864	121.920	0.18325E-02
60.960	121.920	0.48547E-02
67.056	121.920	0.18310E-02
73.152	121.920	0.50338E-03
79.248	121.920	0.69537E-03
85.344	121.920	0.28307E-03
91.440	121.920	0.10060E-03
97.536	121.920	0.13767E-03

TABLE B-9. Estimated Annual Average Concentrations of Vapor-Phase 2,4,5-T at Receptor Locations (x, y Coordinates) Around the Perimeter of the Herbicide Orange Site (continued)

X Coordinate (m)	Y Coordinate (m)	Annual Average Concentration (g/m^3)
103.632	121.920	0.16571E-03
109.728	121.920	0.16932E-03
115.824	121.920	0.15845E-03
121.920	121.920	0.14148E-03
128.016	121.920	0.12347E-03
134.112	121.920	0.10672E-03
140.208	121.920	0.92168E-04
146.304	121.920	0.10821E-03
152.400	121.920	0.69877E-04
158.496	121.920	0.14731E-03
164.592	121.920	0.55273E-04
170.688	121.920	0.11970E-03
176.784	121.920	0.56691E-04
182.880	121.920	0.42956E-04
188.976	121.920	0.45687E-04
195.072	121.920	0.43265E-04
195.072	115.824	0.32551E-04
195.072	109.728	0.32386E-04
195.072	103.632	0.45736E-04
195.072	97.536	0.45069E-04
195.072	91.440	0.40119E-04
195.072	85.344	0.27163E-04
195.072	79.248	0.47968E-04
195.072	73.152	0.50529E-04
195.072	67.056	0.30750E-04
195.072	60.960	0.34518E-04
188.976	60.960	0.33814E-04
182.880	60.960	0.38783E-04
176.784	60.960	0.80621E-04
170.688	60.960	0.94715E-04
164.592	60.960	0.87710E-04
158.496	60.960	0.97224E-04
152.400	60.960	0.94999E-04
146.304	60.960	0.11219E-03
146.304	54.864	0.73949E-04
146.304	48.768	0.12424E-03

TABLE B-9. Estimated Annual Average Concentrations of Vapor-Phase 2,4,5-T at Receptor Locations (x, y Coordinates) Around the Perimeter of the Herbicide Orange Site (continued)

X Coordinate (m)	Y Coordinate (m)	Annual Average Concentration (g/m^3)
146.304	42.672	0.55298E-04
146.304	36.576	0.12424E-03
146.304	30.480	0.74594E-04
146.304	24.384	0.15449E-03
146.304	18.288	0.44348E-04
146.304	12.192	0.11467E-03
146.304	6.096	0.40550E-04
146.304	0.000	0.10269E-03
140.208	0.000	0.99033E-04
134.112	0.000	0.10357E-03
128.016	0.000	0.42672E-04
121.920	0.000	0.22232E-03
115.824	0.000	0.47864E-04
109.728	0.000	0.16841E-03
103.632	0.000	0.70291E-04
97.536	0.000	0.16615E-03
91.440	0.000	0.23923E-03
85.344	0.000	0.11511E-03
79.248	0.000	0.85978E-04
73.152	0.000	0.11373E-03
67.056	0.000	0.20392E-03
60.096	0.000	0.16101E-03
54.864	0.000	0.66920E-04
48.768	0.000	0.19686E-03
42.672	0.000	0.83456E-04
36.576	0.000	0.22254E-03
30.480	0.000	0.40624E-04
24.384	0.000	0.12703E-03
18.288	0.000	0.16750E-03
12.192	0.000	0.10251E-03
6.096	0.000	0.61930E-04

TABLE B-10. Estimated 1-Hour Average Concentrations of Particle-Associated TCDD at Receptor Locations (x, y Coordinates) Around the Perimeter of the Herbicide Orange Site During Excavation

X Coordinate (m)	Y Coordinate (m)	1-Hour Average Concentration (g/m^3)
0.000	0.000	0.79800E-07
0.000	6.096	0.51560E-06
0.000	12.192	0.64930E-06
0.000	18.288	0.10700E-06
0.000	24.384	0.69100E-06
0.000	30.480	0.54920E-06
0.000	36.576	0.33510E-06
0.000	42.672	0.91270E-06
0.000	48.768	0.17620E-06
0.000	54.864	0.10329E-05
0.000	60.960	0.11560E-06
0.000	67.056	0.10329E-05
0.000	73.152	0.17630E-06
0.000	79.248	0.91270E-06
0.000	85.344	0.33530E-06
0.000	91.440	0.54920E-06
0.000	97.536	0.69120E-06
0.000	103.632	0.10700E-06
0.000	109.728	0.64920E-06
0.000	115.824	0.51560E-06
0.000	121.920	0.79800E-07
6.096	121.920	0.36670E-06
12.192	121.920	0.78420E-06
18.288	121.920	0.38060E-06
24.384	121.920	0.28400E-06
30.480	121.920	0.10137E-05
36.576	121.920	0.26380E-06
42.672	121.920	0.95500E-06
48.768	121.920	0.52050E-06
54.864	121.920	0.10385E-05
60.960	121.920	0.43080E-06
67.056	121.920	0.13628E-05
73.152	121.920	0.16800E-06
79.248	121.920	0.13630E-05
85.344	121.920	0.43080E-06
91.440	121.920	0.10389E-05
97.536	121.920	0.52050E-06

TABLE B-10. Estimated 1-Hour Average Concentrations of Particle-Associated TCDD at Receptor Locations (x, y Coordinates) Around the Perimeter of the Herbicide Orange Site During Excavation (continued)

X Coordinate (m)	Y Coordinate (m)	1-Hour Average Concentration (g/m^3)
103.632	121.920	0.95530E-06
109.728	121.920	0.26360E-06
115.824	121.920	0.10137E-05
121.920	121.920	0.28420E-06
128.016	121.920	0.38040E-06
134.112	121.920	0.78420E-06
140.208	121.920	0.36680E-06
146.304	121.920	0.79800E-07
152.400	121.920	0.33040E-06
158.496	121.920	0.54250E-06
164.592	121.920	0.43790E-06
170.688	121.920	0.20460E-06
176.784	121.920	0.62800E-07
182.880	121.920	0.85300E-07
188.976	121.920	0.18310E-06
195.072	121.920	0.27900E-06
195.072	115.824	0.34480E-06
195.072	109.728	0.14160E-06
195.072	103.632	0.66800E-07
195.072	97.536	0.30220E-06
195.072	91.440	0.37230E-06
195.072	85.344	0.11370E-06
195.072	79.248	0.12900E-06
195.072	73.152	0.40520E-06
195.072	67.056	0.27770E-06
195.072	60.960	0.40000E-07
188.976	60.960	0.44500E-07
182.880	60.960	0.49900E-07
176.784	60.960	0.56200E-07
170.688	60.960	0.63700E-07
164.592	60.960	0.72900E-07
158.496	60.960	0.84000E-07
152.400	60.960	0.98000E-07
146.304	60.960	0.11550E-06
146.304	54.864	0.10330E-05
146.304	48.768	0.17620E-06

TABLE B-10. Estimated 1-Hour Average Concentrations of Particle-Associated TCDD at Receptor Locations (x, y Coordinates) Around the Perimeter of the Herbicide Orange Site During Excavation (continued)

X Coordinate (m)	Y Coordinate (m)	1-Hour Average Concentration (g/m^3)
146.304	42.672	0.91280E-06
146.304	36.576	0.33520E-06
146.304	30.480	0.54940E-06
146.304	24.384	0.69110E-06
146.304	18.288	0.10700E-06
146.304	12.192	0.64930E-06
146.304	6.096	0.51550E-06
146.304	0.000	0.79800E-07
140.208	0.000	0.36680E-06
134.112	0.000	0.78420E-06
128.016	0.000	0.38040E-06
121.920	0.000	0.28420E-06
115.824	0.000	0.10137E-05
109.728	0.000	0.26360E-06
103.632	0.000	0.95520E-06
97.536	0.000	0.52030E-06
91.440	0.000	0.10387E-05
85.344	0.000	0.43060E-06
79.248	0.000	0.13629E-05
73.152	0.000	0.16810E-06
67.056	0.000	0.13629E-05
60.096	0.000	0.43090E-06
54.864	0.000	0.10387E-05
48.768	0.000	0.52060E-06
42.672	0.000	0.95520E-06
36.576	0.000	0.26360E-06
30.480	0.000	0.10137E-05
24.384	0.000	0.28410E-06
18.288	0.000	0.38040E-06
12.192	0.000	0.78420E-06
6.096	0.000	0.36680E-06

TABLE B-11. Estimated 1-Hour Average Concentrations of Particle-Associated 2,4-D at Receptor Locations (x, y Coordinates) Around the Perimeter of the Herbicide Orange Site During Excavation

X Coordinate (m)	Y Coordinate (m)	1-Hour Average Concentration (g/m^3)
0.000	0.000	0.48800E-05
0.000	6.096	0.31510E-04
0.000	12.192	0.39680E-04
0.000	18.288	0.65400E-05
0.000	24.384	0.42230E-04
0.000	30.480	0.33560E-04
0.000	36.576	0.20480E-04
0.000	42.672	0.55780E-04
0.000	48.768	0.10770E-04
0.000	54.864	0.63120E-04
0.000	60.960	0.70600E-05
0.000	67.056	0.63120E-04
0.000	73.152	0.10770E-04
0.000	79.248	0.55780E-04
0.000	85.344	0.20490E-04
0.000	91.440	0.33560E-04
0.000	97.536	0.42240E-04
0.000	103.632	0.65400E-05
0.000	109.728	0.39680E-04
0.000	115.824	0.31510E-04
0.000	121.920	0.48800E-05
6.096	121.920	0.22410E-04
12.192	121.920	0.47920E-04
18.288	121.920	0.23260E-04
24.384	121.920	0.17360E-04
30.480	121.920	0.61950E-04
36.576	121.920	0.16120E-04
42.672	121.920	0.58360E-04
48.768	121.920	0.31810E-04
54.864	121.920	0.63460E-04
60.960	121.920	0.26330E-04
67.056	121.920	0.83280E-04
73.152	121.920	0.10260E-04
79.248	121.920	0.83300E-04
85.344	121.920	0.26330E-04
91.440	121.920	0.63490E-04
97.536	121.920	0.31810E-04

TABLE B-11. Estimated 1-Hour Average Concentrations of Particle-Associated 2,4-D at Receptor Locations (x, y Coordinates) Around the Perimeter of the Herbicide Orange Site During Excavation (continued)

X Coordinate (m)	Y Coordinate (m)	1-Hour Average Concentration (g/m^3)
103.632	121.920	0.58380E-04
109.728	121.920	0.16110E-04
115.824	121.920	0.61950E-04
121.920	121.920	0.17370E-04
128.016	121.920	0.23250E-04
134.112	121.920	0.47920E-04
140.208	121.920	0.22420E-04
146.304	121.920	0.48800E-05
152.400	121.920	0.20190E-04
158.496	121.920	0.33160E-04
164.592	121.920	0.26760E-04
170.688	121.920	0.12510E-04
176.784	121.920	0.38400E-05
182.880	121.920	0.52100E-05
188.976	121.920	0.11190E-04
195.072	121.920	0.17050E-04
195.072	115.824	0.21070E-04
195.072	109.728	0.86500E-05
195.072	103.632	0.40800E-05
195.072	97.536	0.18470E-04
195.072	91.440	0.22750E-04
195.072	85.344	0.69500E-05
195.072	79.248	0.78800E-05
195.072	73.152	0.24760E-04
195.072	67.056	0.16970E-04
195.072	60.960	0.24500E-05
188.976	60.960	0.27200E-05
182.880	60.960	0.30500E-05
176.784	60.960	0.34300E-05
170.688	60.960	0.38900E-05
164.592	60.960	0.44500E-05
158.496	60.960	0.51400E-05
152.400	60.960	0.59900E-05
146.304	60.960	0.70600E-05
146.304	54.864	0.63130E-04
146.304	48.768	0.10770E-04

TABLE B-11. Estimated 1-Hour Average Concentrations of Particle-Associated 2,4-D at Receptor Locations (x, y Coordinates) Around the Perimeter of the Herbicide Orange Site During Excavation (continued)

X Coordinate (m)	Y Coordinate (m)	1-Hour Average Concentration (g/m^3)
146.304	42.672	0.55780E-04
146.304	36.576	0.20490E-04
146.304	30.480	0.33570E-04
146.304	24.384	0.42230E-04
146.304	18.288	0.65400E-05
146.304	12.192	0.39680E-04
146.304	6.096	0.31500E-04
146.304	0.000	0.48800E-05
140.208	0.000	0.22420E-04
134.112	0.000	0.47920E-04
128.016	0.000	0.23250E-04
121.920	0.000	0.17370E-04
115.824	0.000	0.61950E-04
109.728	0.000	0.16110E-04
103.632	0.000	0.58370E-04
97.536	0.000	0.31800E-04
91.440	0.000	0.63480E-04
85.344	0.000	0.26310E-04
79.248	0.000	0.83290E-04
73.152	0.000	0.10270E-04
67.056	0.000	0.83290E-04
60.096	0.000	0.26330E-04
54.864	0.000	0.63480E-04
48.768	0.000	0.31820E-04
42.672	0.000	0.58370E-04
36.576	0.000	0.16110E-04
30.480	0.000	0.61950E-04
24.384	0.000	0.17360E-04
18.288	0.000	0.23250E-04
12.192	0.000	0.47920E-04
6.096	0.000	0.22420E-04

TABLE B-12. Estimated 1-Hour Average Concentrations of Particle-Associated 2,4,5-T at Receptor Locations (x, y Coordinates) Around the Perimeter of the Herbicide Orange Site During Excavation

X Coordinate (m)	Y Coordinate (m)	1 Hour Average Concentration (g/m^3)
0.000	0.000	0.17290E-04
0.000	6.096	0.11171E-03
0.000	12.192	0.14069E-03
0.000	18.288	0.23180E-04
0.000	24.384	0.14972E-03
0.000	30.480	0.11900E-03
0.000	36.576	0.72610E-04
0.000	42.672	0.19775E-03
0.000	48.768	0.38170E-04
0.000	54.864	0.22380E-03
0.000	60.960	0.25040E-04
0.000	67.056	0.22380E-03
0.000	73.152	0.38200E-04
0.000	79.248	0.19775E-03
0.000	85.344	0.72660E-04
0.000	91.440	0.11900E-03
0.000	97.536	0.14976E-03
0.000	103.632	0.23180E-04
0.000	109.728	0.14067E-03
0.000	115.824	0.11171E-03
0.000	121.920	0.17300E-04
6.096	121.920	0.79450E-04
12.192	121.920	0.16991E-03
18.288	121.920	0.82450E-04
24.384	121.920	0.61540E-04
30.480	121.920	0.21963E-03
36.576	121.920	0.57150E-04
42.672	121.920	0.20692E-03
48.768	121.920	0.11278E-03
54.864	121.920	0.22501E-03
60.960	121.920	0.93340E-04
67.056	121.920	0.29528E-03
73.152	121.920	0.36390E-04
79.248	121.920	0.29532E-03
85.344	121.920	0.93340E-04
91.440	121.920	0.22510E-03
97.536	121.920	0.11278E-03

TABLE B-12. Estimated 1-Hour Average Concentrations of Particle-Associated 2,4,5-T at Receptor Locations (x, y Coordinates) Around the Perimeter of the Herbicide Orange Site During Excavation (continued)

X Coordinate (m)	Y Coordinate (m)	Annual Average Concentration (g/m^3)
103.632	121.920	0.20698E-03
109.728	121.920	0.57110E-04
115.824	121.920	0.21963E-03
121.920	121.920	0.61570E-04
128.016	121.920	0.82420E-04
134.112	121.920	0.16991E-03
140.208	121.920	0.79480E-04
146.304	121.920	0.17290E-04
152.400	121.920	0.71590E-04
158.496	121.920	0.11755E-03
164.592	121.920	0.94880E-04
170.688	121.920	0.44340E-04
176.784	121.920	0.13610E-04
182.880	121.920	0.18480E-04
188.976	121.920	0.39660E-04
195.072	121.920	0.60440E-04
195.072	115.824	0.74710E-04
195.072	109.728	0.30680E-04
195.072	103.632	0.14470E-04
195.072	97.536	0.65480E-04
195.072	91.440	0.80670E-04
195.072	85.344	0.24640E-04
195.072	79.248	0.27940E-04
195.072	73.152	0.87790E-04
195.072	67.056	0.60170E-04
195.072	60.960	0.86700E-05
188.976	60.960	0.96500E-05
182.880	60.960	0.10810E-04
176.784	60.960	0.12170E-04
170.688	60.960	0.13810E-04
164.592	60.960	0.15730E-04
158.496	60.960	0.18210E-04
152.400	60.960	0.21230E-04
146.304	60.960	0.25030E-04
146.304	54.864	0.22381E-03
146.304	48.768	0.38180E-04

TABLE B-12. Estimated 1-Hour Average Concentrations of Particle-Associated 2,4,5-T at Receptor Locations (x, y Coordinates) Around the Perimeter of the Herbicide Orange Site During Excavation (continued)

X Coordinate (m)	Y Coordinate (m)	Annual Average Concentration (g/m^3)
146.304	42.672	0.19777E-03
146.304	36.576	0.72630E-04
146.304	30.480	0.11903E-03
146.304	24.384	0.14973E-03
146.304	18.288	0.23190E-04
146.304	12.192	0.14069E-03
146.304	6.096	0.11169E-03
146.304	0.000	0.17290E-04
140.208	0.000	0.79480E-04
134.112	0.000	0.16991E-03
128.016	0.000	0.82420E-04
121.920	0.000	0.61570E-04
115.824	0.000	0.21963E-03
109.728	0.000	0.57110E-04
103.632	0.000	0.20696E-03
97.536	0.000	0.11273E-03
91.440	0.000	0.22506E-03
85.344	0.000	0.93290E-04
79.248	0.000	0.29530E-03
73.152	0.000	0.36410E-04
67.056	0.000	0.29530E-03
60.096	0.000	0.93370E-04
54.864	0.000	0.22506E-03
48.768	0.000	0.11280E-03
42.672	0.000	0.20696E-03
36.576	0.000	0.57110E-04
30.480	0.000	0.21963E-03
24.384	0.000	0.61570E-04
18.288	0.000	0.82420E-04
12.192	0.000	0.16991E-03
6.096	0.000	0.79480E-04

TABLE B-13. Estimated 8-Hour Average Concentrations of Particle-Associated TCDD at Receptor Locations (x, y Coordinates) Around the Perimeter of the Herbicide Orange Site During Excavation

X Coordinate (m)	Y Coordinate (m)	8-Hour Average Concentration (g/m^3)
0.000	0.000	0.55860E-07
0.000	6.096	0.36092E-06
0.000	12.192	0.45451E-06
0.000	18.288	0.74900E-07
0.000	24.384	0.48370E-06
0.000	30.480	0.38444E-06
0.000	36.576	0.23457E-06
0.000	42.672	0.63889E-06
0.000	48.768	0.12334E-06
0.000	54.864	0.72303E-06
0.000	60.960	0.80920E-07
0.000	67.056	0.72303E-06
0.000	73.152	0.12341E-06
0.000	79.248	0.63889E-06
0.000	85.344	0.23471E-06
0.000	91.440	0.38444E-06
0.000	97.536	0.48384E-06
0.000	103.632	0.74900E-07
0.000	109.728	0.45444E-06
0.000	115.824	0.36092E-06
0.000	121.920	0.55860E-07
6.096	121.920	0.25669E-06
12.192	121.920	0.54894E-06
18.288	121.920	0.26642E-06
24.384	121.920	0.19880E-06
30.480	121.920	0.70959E-06
36.576	121.920	0.18466E-06
42.672	121.920	0.66850E-06
48.768	121.920	0.36435E-06
54.864	121.920	0.72695E-06
60.960	121.920	0.30156E-06
67.056	121.920	0.95396E-06
73.152	121.920	0.11760E-06
79.248	121.920	0.95410E-06
85.344	121.920	0.30156E-06
91.440	121.920	0.72723E-06
97.536	121.920	0.36435E-06

TABLE B-13. Estimated 8-Hour Average Concentrations of Particle-Associated TCDD at Receptor Locations (x, y Coordinates) Around the Perimeter of the Herbicide Orange Site During Excavation (continued)

X Coordinate (m)	Y Coordinate (m)	8-Hour Average Concentration (g/m^3)
103.632	121.920	0.66871E-06
109.728	121.920	0.18452E-06
115.824	121.920	0.70959E-06
121.920	121.920	0.19894E-06
128.016	121.920	0.26628E-06
134.112	121.920	0.54894E-06
140.208	121.920	0.25676E-06
146.304	121.920	0.55860E-07
152.400	121.920	0.23128E-06
158.496	121.920	0.37975E-06
164.592	121.920	0.30653E-06
170.688	121.920	0.14322E-06
176.784	121.920	0.43960E-07
182.880	121.920	0.59710E-07
188.976	121.920	0.12817E-06
195.072	121.920	0.19530E-06
195.072	115.824	0.24136E-06
195.072	109.728	0.99120E-07
195.072	103.632	0.46760E-07
195.072	97.536	0.21154E-06
195.072	91.440	0.26061E-06
195.072	85.344	0.79590E-07
195.072	79.248	0.90300E-07
195.072	73.152	0.28364E-06
195.072	67.056	0.19439E-06
195.072	60.960	0.28000E-07
188.976	60.960	0.31150E-07
182.880	60.960	0.34930E-07
176.784	60.960	0.39340E-07
170.688	60.960	0.44590E-07
164.592	60.960	0.51030E-07
158.496	60.960	0.58800E-07
152.400	60.960	0.68600E-07
146.304	60.960	0.80850E-07
146.304	54.864	0.72310E-06
146.304	48.768	0.12334E-06

TABLE B-13. Estimated 8-Hour Average Concentrations of Particle-Associated TCDD at Receptor Locations (x, y Coordinates) Around the Perimeter of the Herbicide Orange Site During Excavation (continued)

X Coordinate (m)	Y Coordinate (m)	8-Hour Average Concentration (g/m^3)
146.304	42.672	0.63896E-06
146.304	36.576	0.23464E-06
146.304	30.480	0.38458E-06
146.304	24.384	0.48377E-06
146.304	18.288	0.74900E-07
146.304	12.192	0.45451E-06
146.304	6.096	0.36085E-06
146.304	0.000	0.55860E-07
140.208	0.000	0.25676E-06
134.112	0.000	0.54894E-06
128.016	0.000	0.26628E-06
121.920	0.000	0.19894E-06
115.824	0.000	0.70959E-06
109.728	0.000	0.18452E-06
103.632	0.000	0.66864E-06
97.536	0.000	0.36421E-06
91.440	0.000	0.72709E-06
85.344	0.000	0.30142E-06
79.248	0.000	0.95403E-06
73.152	0.000	0.11767E-06
67.056	0.000	0.95403E-06
60.096	0.000	0.30163E-06
54.864	0.000	0.72709E-06
48.768	0.000	0.36442E-06
42.672	0.000	0.66864E-06
36.576	0.000	0.18452E-06
30.480	0.000	0.70959E-06
24.384	0.000	0.19887E-06
18.288	0.000	0.26628E-06
12.192	0.000	0.54894E-06
6.096	0.000	0.25676E-06

TABLE B-14. Estimated 8-Hour Average Concentrations of Particle-Associated 2,4-D at Receptor Locations (x, y Coordinates) Around the Perimeter of the Herbicide Orange Site During Excavation

X Coordinate (m)	Y Coordinate (m)	8-Hour Average Concentration (g/m^3)
0.000	0.000	0.34160E-05
0.000	6.096	0.22057E-04
0.000	12.192	0.27776E-04
0.000	18.288	0.45780E-05
0.000	24.384	0.29561E-04
0.000	30.480	0.23492E-04
0.000	36.576	0.14336E-04
0.000	42.672	0.39046E-04
0.000	48.768	0.75390E-05
0.000	54.864	0.44184E-04
0.000	60.960	0.49420E-05
0.000	67.056	0.44184E-04
0.000	73.152	0.75390E-05
0.000	79.248	0.39046E-04
0.000	85.344	0.14343E-04
0.000	91.440	0.23492E-04
0.000	97.536	0.29568E-04
0.000	103.632	0.45780E-05
0.000	109.728	0.27776E-04
0.000	115.824	0.22057E-04
0.000	121.920	0.34160E-05
6.096	121.920	0.15687E-04
12.192	121.920	0.33544E-04
18.288	121.920	0.16282E-04
24.384	121.920	0.12152E-04
30.480	121.920	0.43365E-04
36.576	121.920	0.11284E-04
42.672	121.920	0.40852E-04
48.768	121.920	0.22267E-04
54.864	121.920	0.44422E-04
60.960	121.920	0.18431E-04
67.056	121.920	0.58296E-04
73.152	121.920	0.71820E-05
79.248	121.920	0.58310E-04
85.344	121.920	0.18431E-04
91.440	121.920	0.44443E-04

TABLE B-14. Estimated 8-Hour Average Concentrations of Particle-Associated 2,4-D at Receptor Locations (x, y Coordinates) Around the Perimeter of the Herbicide Orange Site During Excavation (continued)

X Coordinate (m)	Y Coordinate (m)	8-Hour Average Concentration (g/m^3)
97.536	121.920	0.22267E-04
103.632	121.920	0.40866E-04
109.728	121.920	0.11277E-04
115.824	121.920	0.43365E-04
121.920	121.920	0.12159E-04
128.016	121.920	0.16275E-04
134.112	121.920	0.33544E-04
140.208	121.920	0.15694E-04
146.304	121.920	0.34160E-05
152.400	121.920	0.14133E-04
158.496	121.920	0.23212E-04
164.592	121.920	0.18732E-04
170.688	121.920	0.87570E-05
176.784	121.920	0.26880E-05
182.880	121.920	0.36470E-05
188.976	121.920	0.78330E-05
195.072	121.920	0.11935E-04
195.072	115.824	0.14749E-04
195.072	109.728	0.60550E-05
195.072	103.632	0.28560E-05
195.072	97.536	0.12929E-04
195.072	91.440	0.15925E-04
195.072	85.344	0.48650E-05
195.072	79.248	0.55160E-05
195.072	73.152	0.17332E-04
195.072	67.056	0.11879E-04
195.072	60.960	0.17150E-05
188.976	60.960	0.19040E-05
182.880	60.960	0.21350E-05
176.784	60.960	0.24010E-05
170.688	60.960	0.27230E-05
164.592	60.960	0.31150E-05
158.496	60.960	0.35980E-05
152.400	60.960	0.41930E-05
146.304	60.960	0.49420E-05
146.304	54.864	0.44191E-04

TABLE B-14. Estimated 8-Hour Average Concentrations of Particle-Associated 2,4-D at Receptor Locations (x, y Coordinates) Around the Perimeter of the Herbicide Orange Site During Excavation (continued)

X Coordinate (m)	Y Coordinate (m)	8-Hour Average Concentration (g/m^3)
146.304	48.768	0.75390E-05
146.304	42.672	0.39046E-04
146.304	36.576	0.14343E-04
146.304	30.480	0.23499E-04
146.304	24.384	0.29561E-04
146.304	18.288	0.45780E-05
146.304	12.192	0.27774E-04
146.304	6.096	0.22050E-04
146.304	0.000	0.34160E-05
140.208	0.000	0.15694E-04
134.112	0.000	0.33544E-04
128.016	0.000	0.16275E-04
121.920	0.000	0.12159E-04
115.824	0.000	0.43365E-04
109.728	0.000	0.11277E-04
103.632	0.000	0.40859E-04
97.536	0.000	0.22260E-04
91.440	0.000	0.44436E-04
85.344	0.000	0.18417E-04
79.248	0.000	0.58303E-04
73.152	0.000	0.71890E-05
67.056	0.000	0.58303E-04
60.096	0.000	0.18431E-04
54.864	0.000	0.44436E-04
48.768	0.000	0.22274E-04
42.672	0.000	0.40859E-04
36.576	0.000	0.11277E-04
30.480	0.000	0.43365E-04
24.384	0.000	0.12152E-04
18.288	0.000	0.16275E-04
12.192	0.000	0.33544E-04
6.096	0.000	0.15694E-04

TABLE B-15. Estimated 8-Hour Average Concentrations of Particle-Associated 2,4,5-T at Receptor Locations (x, y Coordinates) Around the Perimeter of the Herbicide Orange Site During Excavation

X Coordinate (m)	Y Coordinate (m)	8-Hour Average Concentration (g/m^3)
0.000	0.000	0.12103E-04
0.000	6.096	0.78197E-04
0.000	12.192	0.98483E-04
0.000	18.288	0.16226E-04
0.000	24.384	0.10480E-03
0.000	30.480	0.83300E-04
0.000	36.576	0.50827E-04
0.000	42.672	0.13842E-03
0.000	48.768	0.26719E-04
0.000	54.864	0.15666E-03
0.000	60.960	0.17528E-04
0.000	67.056	0.15666E-03
0.000	73.152	0.26740E-04
0.000	79.248	0.13842E-03
0.000	85.344	0.50862E-04
0.000	91.440	0.83300E-04
0.000	97.536	0.10483E-03
0.000	103.632	0.16226E-04
0.000	109.728	0.98469E-04
0.000	115.824	0.78197E-04
0.000	121.920	0.12110E-04
6.096	121.920	0.55615E-04
12.192	121.920	0.11894E-03
18.288	121.920	0.57715E-04
24.384	121.920	0.43078E-04
30.480	121.920	0.15374E-03
36.576	121.920	0.40005E-04
42.672	121.920	0.14484E-03
48.768	121.920	0.78946E-04
54.864	121.920	0.15751E-03
60.960	121.920	0.65333E-04
67.056	121.920	0.20670E-03
73.152	121.920	0.25473E-04
79.248	121.920	0.20672E-03
85.344	121.920	0.65333E-04
91.440	121.920	0.15757E-03
97.536	121.920	0.78946E-04

TABLE B-15. Estimated 8-Hour Average Concentrations of Particle-Associated 2,4,5-T at Receptor Locations (x, y Coordinates) Around the Perimeter of the Herbicide Orange Site During Excavation (continued)

X Coordinate (m)	Y Coordinate (m)	8-Hour Average Concentration (g/m^3)
103.632	121.920	0.14489E-03
109.728	121.920	0.39977E-04
115.824	121.920	0.15374E-03
121.920	121.920	0.43099E-04
128.016	121.920	0.57694E-04
134.112	121.920	0.11894E-03
140.208	121.920	0.55636E-04
146.304	121.920	0.12103E-04
152.400	121.920	0.50113E-04
158.496	121.920	0.82285E-04
164.592	121.920	0.66416E-04
170.688	121.920	0.31038E-04
176.784	121.920	0.95270E-05
182.880	121.920	0.12936E-04
188.976	121.920	0.27762E-04
195.072	121.920	0.42308E-04
195.072	115.824	0.52297E-04
195.072	109.728	0.21476E-04
195.072	103.632	0.10129E-04
195.072	97.536	0.45836E-04
195.072	91.440	0.56469E-04
195.072	85.344	0.17248E-04
195.072	79.248	0.19558E-04
195.072	73.152	0.61453E-04
195.072	67.056	0.42119E-04
195.072	60.960	0.60690E-05
188.976	60.960	0.67550E-05
182.880	60.960	0.75670E-05
176.784	60.960	0.85190E-05
170.688	60.960	0.96670E-05
164.592	60.960	0.11053E-04
158.496	60.960	0.12747E-04
152.400	60.960	0.14861E-04
146.304	60.960	0.17521E-04
146.304	54.864	0.15667E-03
146.304	48.768	0.26726E-04

TABLE E-15. Estimated 8-Hour Average Concentrations of Particle-Associated 2,4,5-T at Receptor Locations (x, y Coordinates) Around the Perimeter of the Herbicide Orange Site During Excavation (continued)

X Coordinate (m)	Y Coordinate (m)	8-Hour Average Concentration (g/m^3)
146.304	42.672	0.13844E-03
146.304	36.576	0.50841E-04
146.304	30.480	0.83321E-04
146.304	24.384	0.10481E-03
146.304	18.288	0.16233E-04
146.304	12.192	0.98483E-04
146.304	6.096	0.78183E-04
146.304	0.000	0.12103E-04
140.208	0.000	0.55636E-04
134.112	0.000	0.11894E-03
128.016	0.000	0.57694E-04
121.920	0.000	0.43099E-04
115.824	0.000	0.15374E-03
109.728	0.000	0.39977E-04
103.632	0.000	0.14487E-03
97.536	0.000	0.78911E-04
91.440	0.000	0.15754E-03
85.344	0.000	0.65303E-04
79.248	0.000	0.20671E-03
73.152	0.000	0.25487E-04
67.056	0.000	0.20671E-03
60.096	0.000	0.65359E-04
54.864	0.000	0.15754E-03
48.768	0.000	0.78960E-04
42.672	0.000	0.14487E-03
36.576	0.000	0.39977E-04
30.480	0.000	0.15374E-03
24.384	0.000	0.43099E-04
18.288	0.000	0.57694E-04
12.192	0.000	0.11894E-03
6.096	0.000	0.55636E-04

TABLE B-16. Estimated 1-Hour Average Concentrations of Particle-Associated TCDD at Receptor Locations (x, y Coordinates) Around the Perimeter of the Herbicide Orange Site During Cement Cover Construction

X Coordinate (m)	Y Coordinate (m)	1-Hour Average Concentration (g/m^3)
0.000	0.000	0.70900E-08
0.000	6.096	0.45830E-07
0.000	12.192	0.57720E-07
0.000	18.288	0.95100E-08
0.000	24.384	0.61420E-07
0.000	30.480	0.48820E-07
0.000	36.576	0.29790E-07
0.000	42.672	0.81130E-07
0.000	48.768	0.15660E-07
0.000	54.864	0.91820E-07
0.000	60.960	0.10270E-07
0.000	67.056	0.91820E-07
0.000	73.152	0.15670E-07
0.000	79.248	0.81130E-07
0.000	85.344	0.29810E-07
0.000	91.440	0.48820E-07
0.000	97.536	0.61440E-07
0.000	103.632	0.95100E-08
0.000	109.728	0.57710E-07
0.000	115.824	0.45830E-07
0.000	121.920	0.71000E-08
6.096	121.920	0.32600E-07
12.192	121.920	0.69700E-07
18.288	121.920	0.33830E-07
24.384	121.920	0.25250E-07
30.480	121.920	0.90100E-07
36.576	121.920	0.23440E-07
42.672	121.920	0.84890E-07
48.768	121.920	0.46270E-07
54.864	121.920	0.92310E-07
60.960	121.920	0.38290E-07
67.056	121.920	0.12114E-06
73.152	121.920	0.14930E-07
79.248	121.920	0.12116E-06
85.344	121.920	0.38290E-07
91.440	121.920	0.92350E-07
97.536	121.920	0.46270E-07

TABLE B-16. Estimated 1-Hour Average Concentrations of Particle-Associated TCDD at Receptor Locations (x, y Coordinates) Around the Perimeter of the Herbicide Orange Site During Cement Cover Construction (continued)

X Coordinate (m)	Y Coordinate (m)	1-Hour Average Concentration (g/m^3)
103.632	121.920	0.84920E-07
109.728	121.920	0.23430E-07
115.824	121.920	0.90110E-07
121.920	121.920	0.25260E-07
128.016	121.920	0.33810E-07
134.112	121.920	0.69710E-07
140.208	121.920	0.32610E-07
146.304	121.920	0.70900E-08
152.400	121.920	0.29370E-07
158.496	121.920	0.48230E-07
164.592	121.920	0.38920E-07
170.688	121.920	0.18190E-07
176.784	121.920	0.55900E-08
182.880	121.920	0.75800E-08
188.976	121.920	0.16270E-07
195.072	121.920	0.24800E-07
195.072	115.824	0.30650E-07
195.072	109.728	0.12580E-07
195.072	103.632	0.59300E-08
195.072	97.536	0.26860E-07
195.072	91.440	0.33090E-07
195.072	85.344	0.10110E-07
195.072	79.248	0.11460E-07
195.072	73.152	0.36010E-07
195.072	67.056	0.24690E-07
195.072	60.960	0.35600E-08
188.976	60.960	0.39600E-08
182.880	60.960	0.44300E-08
176.784	60.960	0.49900E-08
170.688	60.960	0.56600E-08
164.592	60.960	0.64800E-08
158.496	60.960	0.74700E-08
152.400	60.960	0.87100E-08
146.304	60.960	0.10270E-07
146.304	54.864	0.91820E-07
146.304	48.768	0.15660E-07

TABLE B-18. Estimated 1-Hour Average Concentrations of Particle-Associated TCDD at Receptor Locations (x, y Coordinates) Around the Perimeter of the Herbicide Orange Site During Cement Cover Construction (continued)

X Coordinate (m)	Y Coordinate (m)	1-Hour Average Concentration (g/m^3)
146.304	42.672	0.81140E-07
146.304	36.576	0.29800E-07
146.304	30.480	0.48830E-07
146.304	24.384	0.61430E-07
146.304	18.288	0.95200E-08
146.304	12.192	0.57720E-07
146.304	6.096	0.45820E-07
146.304	0.000	0.70900E-08
140.208	0.000	0.32610E-07
134.112	0.000	0.69710E-07
128.016	0.000	0.33810E-07
121.920	0.000	0.25260E-07
115.824	0.000	0.90100E-07
109.728	0.000	0.23430E-07
103.632	0.000	0.84910E-07
97.536	0.000	0.46250E-07
91.440	0.000	0.92330E-07
85.344	0.000	0.38270E-07
79.248	0.000	0.12115E-06
73.152	0.000	0.14940E-07
67.056	0.000	0.12115E-06
60.096	0.000	0.38300E-07
54.864	0.000	0.92330E-07
48.768	0.000	0.46280E-07
42.672	0.000	0.84910E-07
36.576	0.000	0.23430E-07
30.480	0.000	0.90100E-07
24.384	0.000	0.25260E-07
18.288	0.000	0.33810E-07
12.192	0.000	0.69700E-07
6.096	0.000	0.32610E-07

TABLE B-17. Estimated 1-Hour Average Concentrations of Particle-Associated 2,4-D at Receptor Locations (x, y Coordinates) Around the Perimeter of the Herbicide Orange Site During Cement Cover Construction

X Coordinate (m)	Y Coordinate (m)	1-Hour Average Concentration (g/m^3)
0.000	0.000	0.44300E-06
0.000	6.096	0.28640E-05
0.000	12.192	0.36070E-05
0.000	18.288	0.59400E-06
0.000	24.384	0.38390E-05
0.000	30.480	0.30510E-05
0.000	36.576	0.18620E-05
0.000	42.672	0.50710E-05
0.000	48.768	0.97900E-06
0.000	54.864	0.57390E-05
0.000	60.960	0.64200E-06
0.000	67.056	0.57390E-05
0.000	73.152	0.98000E-06
0.000	79.248	0.50710E-05
0.000	85.344	0.18630E-05
0.000	91.440	0.30510E-05
0.000	97.536	0.38400E-05
0.000	103.632	0.59400E-06
0.000	109.728	0.36070E-05
0.000	115.824	0.28640E-05
0.000	121.920	0.44400E-06
6.096	121.920	0.20370E-05
12.192	121.920	0.43570E-05
18.288	121.920	0.21140E-05
24.384	121.920	0.15780E-05
30.480	121.920	0.56310E-05
36.576	121.920	0.14650E-05
42.672	121.920	0.53060E-05
48.768	121.920	0.28920E-05
54.864	121.920	0.57700E-05
60.960	121.920	0.23930E-05
67.056	121.920	0.75710E-05
73.152	121.920	0.90300E-06
79.248	121.920	0.75720E-05
85.344	121.920	0.23930E-05
91.440	121.920	0.57720E-05
97.536	121.920	0.28920E-05

TABLE B-17. Estimated 1-Hour Average Concentrations of Particle-Associated 2,4-D at Receptor Locations (x, y Coordinates) Around the Perimeter of the Herbicide Orange Site During Cement Cover Construction (continued)

X Coordinate (m)	Y Coordinate (m)	1-Hour Average Concentration (g/m^3)
103.632	121.920	0.53070E-05
109.728	121.920	0.14640E-05
115.824	121.920	0.56320E-05
121.920	121.920	0.15790E-05
128.016	121.920	0.21130E-05
134.112	121.920	0.43570E-05
140.208	121.920	0.20380E-05
146.304	121.920	0.44300E-06
152.400	121.920	0.18360E-05
158.496	121.920	0.30140E-05
164.592	121.920	0.24330E-05
170.688	121.920	0.11370E-05
176.784	121.920	0.34900E-06
182.880	121.920	0.47400E-06
188.976	121.920	0.10170E-05
195.072	121.920	0.15500E-05
195.072	115.824	0.19160E-05
195.072	109.728	0.78700E-06
195.072	103.632	0.37100E-06
195.072	97.536	0.16790E-05
195.072	91.440	0.20680E-05
195.072	85.344	0.63200E-06
195.072	79.248	0.71700E-06
195.072	73.152	0.22510E-05
195.072	67.056	0.15430E-05
195.072	60.960	0.22200E-06
188.976	60.960	0.24700E-06
182.880	60.960	0.27700E-06
176.784	60.960	0.31200E-06
170.688	60.960	0.35400E-06
164.592	60.960	0.40500E-06
158.496	60.960	0.46700E-06
152.400	60.960	0.54400E-06
146.304	60.960	0.64200E-06
146.304	54.864	0.57390E-05
146.304	48.768	0.97900E-06

TABLE B-17. Estimated 1-Hour Average Concentrations of Particle-Associated 2,4-D at Receptor Locations (x, y Coordinates) Around the Perimeter of the Herbicide Orange Site During Cement Cover Construction (continued)

X Coordinate (m)	Y Coordinate (m)	1-Hour Average Concentration (g/m^3)
146.304	42.672	0.50710E-05
146.304	36.576	0.18620E-05
146.304	30.480	0.30520E-05
146.304	24.384	0.38390E-05
146.304	18.288	0.59500E-06
146.304	12.192	0.36070E-05
146.304	6.096	0.28640E-05
146.304	0.000	0.44300E-06
140.208	0.000	0.20380E-05
134.112	0.000	0.43570E-05
128.016	0.000	0.21130E-05
121.920	0.000	0.15790E-05
115.824	0.000	0.56310E-05
109.728	0.000	0.14640E-05
103.632	0.000	0.53070E-05
97.536	0.000	0.28900E-05
91.440	0.000	0.57710E-05
85.344	0.000	0.23920E-05
79.248	0.000	0.75720E-05
73.152	0.000	0.93400E-06
67.056	0.000	0.75720E-05
60.096	0.000	0.23940E-05
54.264	0.000	0.57710E-05
48.768	0.000	0.28920E-05
42.672	0.000	0.53070E-05
36.576	0.000	0.14640E-05
30.480	0.000	0.56310E-05
24.384	0.000	0.15790E-05
18.288	0.000	0.21130E-05
12.192	0.000	0.43570E-05
6.096	0.000	0.20380E-05

TABLE B-18. Estimated 1-Hour Average Concentrations of Particle-Associated 2,4,5-T at Receptor Locations (x, y Coordinates) Around the Perimeter of the Herbicide Orange Site During Cement Cover Construction

X Coordinate (m)	Y Coordinate (m)	1 Hour Average Concentration (g/m^3)
0.000	0.000	0.15960E-05
0.000	6.096	0.10312E-04
0.000	12.192	0.12987E-04
0.000	18.288	0.21400E-05
0.000	24.384	0.13820E-04
0.000	30.480	0.10985E-04
0.000	36.576	0.67030E-05
0.000	42.672	0.18254E-04
0.000	48.768	0.35230E-05
0.000	54.864	0.20659E-04
0.000	60.960	0.23110E-05
0.000	67.056	0.20659E-04
0.000	73.152	0.35260E-05
0.000	79.248	0.18254E-04
0.000	85.344	0.67070E-05
0.000	91.440	0.10985E-04
0.000	97.536	0.13824E-04
0.000	103.632	0.21400E-05
0.000	109.728	0.12985E-04
0.000	115.824	0.10312E-04
0.000	121.920	0.15970E-05
6.096	121.920	0.73340E-05
12.192	121.920	0.15684E-04
18.288	121.920	0.76110E-05
24.384	121.920	0.56800E-05
30.480	121.920	0.20273E-04
36.576	121.920	0.52750E-05
42.672	121.920	0.19100E-04
48.768	121.920	0.10411E-04
54.864	121.920	0.20770E-04
60.960	121.920	0.86160E-05
67.056	121.920	0.27256E-04
73.152	121.920	0.33590E-05
79.248	121.920	0.27261E-04
85.344	121.920	0.86160E-05
91.440	121.920	0.20778E-04
97.536	121.920	0.10411E-04

TABLE B-18. Estimated 1-Hour Average Concentrations of Particle-Associated 2,4,5-T at Receptor Locations (x, y Coordinates) Around the Perimeter of the Herbicide Orange Site During Cement Cover Construction (continued)

X Coordinate (m)	Y Coordinate (m)	1 Hour Average Concentration (g/m^3)
103.632	121.920	0.19106E-04
109.728	121.920	0.52710E-05
115.824	121.920	0.20274E-04
121.920	121.920	0.56830E-05
128.016	121.920	0.76080E-05
134.112	121.920	0.15684E-04
140.208	121.920	0.73360E-05
146.304	121.920	0.15960E-05
152.400	121.920	0.66080E-05
158.496	121.920	0.10851E-04
164.592	121.920	0.87580E-05
170.688	121.920	0.40930E-05
176.784	121.920	0.12570E-05
182.880	121.920	0.17060E-05
188.976	121.920	0.36610E-05
195.072	121.920	0.55800E-05
195.072	115.824	0.68970E-05
195.072	109.728	0.28320E-05
195.072	103.632	0.13350E-05
195.072	97.536	0.60440E-05
195.072	91.440	0.74460E-05
195.072	85.344	0.22750E-05
195.072	79.248	0.25790E-05
195.072	73.152	0.81030E-05
195.072	67.056	0.55540E-05
195.072	60.960	0.80000E-06
188.976	60.960	0.89100E-06
182.880	60.960	0.99700E-06
176.784	60.960	0.11240E-05
170.688	60.960	0.12740E-05
164.592	60.960	0.14580E-05
158.496	60.960	0.16810E-05
152.400	60.960	0.19600E-05
146.304	60.960	0.23100E-05
146.304	54.864	0.20659E-04
146.304	48.763	0.35250E-05

TABLE B-18. Estimated 1-Hour Average Concentrations of Particle-Associated 2,4,5-T at Receptor Locations (x, y Coordinates) Around the Perimeter of the Herbicide Orange Site During Cement Cover Construction (continued)

X Coordinate (m)	Y Coordinate (m)	1 Hour Average Concentration (g/m^3)
146.304	42.672	0.18256E-04
146.304	36.576	0.67040E-05
146.304	30.480	0.10988E-04
146.304	24.384	0.13821E-04
146.304	18.288	0.21410E-05
146.304	12.192	0.12987E-04
146.304	6.096	0.10310E-04
146.304	0.000	0.15960E-05
140.208	0.000	0.73360E-05
134.112	0.000	0.15684E-04
128.016	0.000	0.76080E-05
121.920	0.000	0.56830E-05
115.824	0.000	0.20273E-04
109.728	0.000	0.52710E-05
103.632	0.000	0.19104E-04
97.536	0.000	0.10406E-04
91.440	0.000	0.20775E-04
85.344	0.000	0.86120E-05
79.248	0.000	0.27258E-04
73.152	0.000	0.33610E-05
67.056	0.000	0.27258E-04
60.096	0.000	0.86190E-05
54.864	0.000	0.20775E-04
48.768	0.000	0.10412E-04
42.672	0.000	0.19104E-04
36.576	0.000	0.52720E-05
30.480	0.000	0.20273E-04
24.384	0.000	0.56830E-05
18.288	0.000	0.76080E-05
12.192	0.000	0.15684E-04
6.096	0.000	0.73360E-05

TABLE B-19. Estimated 8-Hour Average Concentrations of Particle-Associated TCDD at Receptor Locations (x, y Coordinates) Around the Perimeter of the Herbicide Orange Site During Cement Cover Construction

X Coordinate (m)	Y Coordinate (m)	8-Hour Average Concentration (g/m^3)
0.000	0.000	0.49630E-08
0.000	6.096	0.32081E-07
0.000	12.192	0.40404E-07
0.000	18.288	0.66570E-08
0.000	24.384	0.42994E-07
0.000	30.480	0.34174E-07
0.000	36.576	0.20853E-07
0.000	42.672	0.56791E-07
0.000	48.768	0.10962E-07
0.000	54.864	0.64274E-07
0.000	60.960	0.71890E-08
0.000	67.056	0.64274E-07
0.000	73.152	0.10969E-07
0.000	79.248	0.56791E-07
0.000	85.344	0.20867E-07
0.000	91.440	0.34174E-07
0.000	97.536	0.43008E-07
0.000	103.632	0.66570E-08
0.000	109.728	0.40397E-07
0.000	115.824	0.32081E-07
0.000	121.920	0.49700E-08
6.096	121.920	0.22820E-07
12.192	121.920	0.48790E-07
18.288	121.920	0.23681E-07
24.384	121.920	0.17675E-07
30.480	121.920	0.63070E-07
36.576	121.920	0.16408E-07
42.672	121.920	0.59423E-07
48.768	121.920	0.32389E-07
54.864	121.920	0.64617E-07
60.960	121.920	0.26803E-07
67.056	121.920	0.84798E-07
73.152	121.920	0.10451E-07
79.248	121.920	0.84812E-07
85.344	121.920	0.26803E-07
91.440	121.920	0.64645E-07
97.536	121.920	0.32389E-07

TABLE B-19. Estimated 8-Hour Average Concentrations of Particle-Associated TCDD at Receptor Locations (x, y Coordinates) Around the Perimeter of the Herbicide Orange Site During Cement Cover Construction (continued)

X Coordinate (m)	Y Coordinate (m)	8-Hour Average Concentration (g/m^3)
103.632	121.920	0.59444E-07
109.728	121.920	0.16401E-07
115.824	121.920	0.63077E-07
121.920	121.920	0.17682E-07
128.016	121.920	0.23667E-07
134.112	121.920	0.48797E-07
140.208	121.920	0.22827E-07
146.304	121.920	0.49630E-08
152.400	121.920	0.20559E-07
158.496	121.920	0.33761E-07
164.592	121.920	0.27244E-07
170.688	121.920	0.12733E-07
176.784	121.920	0.39130E-08
182.880	121.920	0.53060E-08
188.976	121.920	0.11389E-07
195.072	121.920	0.17360E-07
195.072	115.824	0.21455E-07
195.072	109.728	0.88060E-08
195.072	103.632	0.41510E-08
195.072	97.536	0.18802E-07
195.072	91.440	0.23163E-07
195.072	85.344	0.70770E-08
195.072	79.248	0.80220E-08
195.072	73.152	0.25207E-07
195.072	67.056	0.17283E-07
195.072	60.960	0.24920E-08
188.976	60.960	0.27720E-08
182.880	60.960	0.31010E-08
176.784	60.960	0.34930E-08
170.688	60.960	0.39620E-08
164.592	60.960	0.45360E-08
158.496	60.960	0.52290E-08
152.400	60.960	0.60970E-08
146.304	60.960	0.71890E-08
146.304	54.864	0.64274E-07
146.304	48.768	0.10962E-07

TABLE B-19. Estimated 8-Hour Average Concentrations of Particle-Associated TCDD at Receptor Locations (x, y Coordinates) Around the Perimeter of the Herbicide Orange Site During Cement Cover Construction (continued)

X Coordinate (m)	Y Coordinate (m)	8-Hour Average Concentration (g/m^3)
146.304	42.672	0.56798E-07
146.304	36.576	0.20860E-07
146.304	30.480	0.34181E-07
146.304	24.384	0.43001E-07
146.304	18.288	0.66640E-08
146.304	12.192	0.40404E-07
146.304	6.096	0.32074E-07
146.304	0.000	0.49630E-08
140.208	0.000	0.22827E-07
134.112	0.000	0.48797E-07
128.016	0.000	0.23667E-07
121.920	0.000	0.17682E-07
115.824	0.000	0.63070E-07
109.728	0.000	0.16401E-07
103.632	0.000	0.59437E-07
97.536	0.000	0.32375E-07
91.440	0.000	0.64631E-07
85.344	0.000	0.26789E-07
79.248	0.000	0.84805E-07
73.152	0.000	0.10458E-07
67.056	0.000	0.84805E-07
60.096	0.000	0.26810E-07
54.864	0.000	0.64631E-07
48.768	0.000	0.32396E-07
42.672	0.000	0.59437E-07
36.576	0.000	0.16401E-07
30.480	0.000	0.63070E-07
24.384	0.000	0.17682E-07
18.288	0.000	0.23667E-07
12.192	0.000	0.48790E-07
6.096	0.000	0.22827E-07

TABLE B-20. Estimated 8-Hour Average Concentrations of Particle-Associated 2,4-D at Receptor Locations (x, y Coordinates) Around the Perimeter of the Herbicide Orange Site During Cement Cover Construction

X Coordinate (m)	Y Coordinate (m)	8-Hour Average Concentration (g/m^3)
0.000	0.000	0.31010E-06
0.000	6.096	0.20048E-05
0.000	12.192	0.25249E-05
0.000	18.288	0.41580E-06
0.000	24.384	0.26873E-05
0.000	30.480	0.21357E-05
0.000	36.576	0.13034E-05
0.000	42.672	0.35497E-05
0.000	48.768	0.68530E-06
0.000	54.864	0.40173E-05
0.000	60.960	0.44940E-06
0.000	67.056	0.40173E-05
0.000	73.152	0.68600E-06
0.000	79.248	0.35497E-05
0.000	85.344	0.13041E-05
0.000	91.440	0.21357E-05
0.000	97.536	0.26880E-05
0.000	103.632	0.41580E-06
0.000	109.728	0.25249E-05
0.000	115.824	0.20048E-05
0.000	121.920	0.31080E-06
6.096	121.920	0.14259E-05
12.192	121.920	0.30499E-05
18.288	121.920	0.14798E-05
24.384	121.920	0.11046E-05
30.480	121.920	0.39417E-05
36.576	121.920	0.10255E-05
42.672	121.920	0.37142E-05
48.768	121.920	0.20244E-05
54.864	121.920	0.40390E-05
60.960	121.920	0.16751E-05
67.056	121.920	0.52997E-05
73.152	121.920	0.65310E-06
79.248	121.920	0.53004E-05
85.344	121.920	0.16751E-05
91.440	121.920	0.40404E-05
97.536	121.920	0.20244E-05

TABLE B-20. Estimated 8-Hour Average Concentrations of Particle-Associated 2,4-D at Receptor Locations (x, y Coordinates) Around the Perimeter of the Herbicide Orange Site During Cement Cover Construction (continued)

X Coordinate (m)	Y Coordinate (m)	8-Hour Average Concentration (g/m^3)
103.632	121.920	0.37149E-05
109.728	121.920	0.10248E-05
115.824	121.920	0.39424E-05
121.920	121.920	0.11053E-05
128.016	121.920	0.14791E-05
134.112	121.920	0.30499E-05
140.208	121.920	0.14266E-05
146.304	121.920	0.31010E-06
152.400	121.920	0.12852E-05
158.496	121.920	0.21098E-05
164.592	121.920	0.17031E-05
170.688	121.920	0.79590E-06
176.784	121.920	0.24430E-06
182.880	121.920	0.33180E-06
188.976	121.920	0.71190E-06
195.072	121.920	0.10850E-05
195.072	115.824	0.13412E-05
195.072	109.728	0.55090E-06
195.072	103.632	0.25970E-06
195.072	97.536	0.11753E-05
195.072	91.440	0.14476E-05
195.072	85.344	0.44240E-06
195.072	79.248	0.50190E-06
195.072	73.152	0.15757E-05
195.072	67.056	0.10801E-05
195.072	60.960	0.15540E-06
188.976	60.960	0.17290E-06
182.880	60.960	0.19390E-06
176.784	60.960	0.21840E-06
170.688	60.960	0.24780E-06
164.592	60.960	0.28350E-06
158.496	60.960	0.32690E-06
152.400	60.960	0.38060E-06
146.304	60.960	0.44940E-06
146.304	54.864	0.53173E-06
146.304	48.768	0.63530E-06

TABLE B-20. Estimated 8-Hour Average Concentrations of Particle-Associated 2,4-D at Receptor Locations (x, y Coordinates) Around the Perimeter of the Herbicide Orange Site During Cement Cover Construction (continued)

X Coordinate (m)	Y Coordinate (m)	8-Hour Average Concentration (g/m^3)
146.304	42.672	0.35497E-05
146.304	36.576	0.13034E-05
146.304	30.480	0.21364E-05
146.304	24.384	0.26873E-05
146.304	18.288	0.41650E-06
146.304	12.192	0.25249E-05
146.304	6.096	0.20048E-05
146.304	0.000	0.31010E-06
140.208	0.000	0.14266E-05
134.112	0.000	0.30499E-05
128.016	0.000	0.14791E-05
121.920	0.000	0.11053E-05
115.824	0.000	0.39417E-05
109.728	0.000	0.10248E-05
103.632	0.000	0.37149E-05
97.536	0.000	0.20230E-05
91.440	0.000	0.40397E-05
85.344	0.000	0.16744E-05
79.248	0.000	0.53004E-05
73.152	0.000	0.65380E-06
67.056	0.000	0.53004E-05
60.096	0.000	0.16758E-05
54.864	0.000	0.40397E-05
48.768	0.000	0.20244E-05
42.672	0.000	0.37149E-05
36.576	0.000	0.10248E-05
30.480	0.000	0.39417E-05
24.384	0.000	0.11053E-05
18.288	0.000	0.14791E-05
12.192	0.000	0.30499E-05
6.096	0.000	0.14266E-05

TABLE B-21. Estimated 8-Hour Average Concentrations of Particle-Associated 2,4,5-T at Receptor Locations (x, y Coordinates) Around the Perimeter of the Herbicide Orange Site During Cement Cover Construction

X Coordinate (m)	Y Coordinate (m)	8-Hour Average Concentration (g/m^3)
0.000	0.000	0.11172E-05
0.000	6.096	0.72184E-05
0.000	12.192	0.90909E-05
0.000	18.288	0.14980E-05
0.000	24.384	0.96740E-05
0.000	30.480	0.76895E-05
0.000	36.576	0.46921E-05
0.000	42.672	0.12779E-04
0.000	48.768	0.24661E-05
0.000	54.864	0.14461E-04
0.000	60.960	0.16177E-05
0.000	67.056	0.14461E-04
0.000	73.152	0.24682E-05
0.000	79.248	0.12778E-04
0.000	85.344	0.46949E-05
0.000	91.440	0.76895E-05
0.000	97.536	0.96768E-05
0.000	103.632	0.14980E-05
0.000	109.728	0.90895E-05
0.000	115.824	0.72184E-05
0.000	121.920	0.11179E-05
6.096	121.920	0.51338E-05
12.192	121.920	0.10979E-04
18.288	121.920	0.53277E-05
24.384	121.920	0.39760E-05
30.480	121.920	0.14191E-04
36.576	121.920	0.36925E-05
42.672	121.920	0.13370E-04
48.768	121.920	0.72377E-05
54.864	121.920	0.14539E-04
60.960	121.920	0.60312E-05
67.056	121.920	0.19079E-04
73.152	121.920	0.23513E-05
79.248	121.920	0.19083E-04
85.344	121.920	0.60312E-05
91.440	121.920	0.14545E-04
97.536	121.920	0.72377E-05

TABLE B-21. Estimated 8-Hour Average Concentrations of Particle-Associated 2,4,5-T at Receptor Locations (x, y Coordinates) Around the Perimeter of the Herbicide Orange Site During Cement Cover Construction (continued)

X Coordinate (m)	Y Coordinate (m)	8-Hour Average Concentration (g/m^3)
103.632	121.920	0.13374E-04
109.728	121.920	0.36897E-05
115.824	121.920	0.14192E-04
121.920	121.920	0.39781E-05
128.016	121.920	0.53256E-05
134.112	121.920	0.10979E-04
140.208	121.920	0.51352E-05
146.304	121.920	0.11172E-05
152.400	121.920	0.46256E-05
158.496	121.920	0.75957E-05
164.592	121.920	0.61306E-05
170.688	121.920	0.28651E-05
176.784	121.920	0.87990E-06
182.880	121.920	0.11942E-05
188.976	121.920	0.25627E-05
195.072	121.920	0.39060E-05
195.072	115.824	0.48279E-05
195.072	109.728	0.19824E-05
195.072	103.632	0.93450E-06
195.072	97.536	0.42308E-05
195.072	91.440	0.52122E-05
195.072	85.344	0.15925E-05
195.072	79.248	0.18053E-05
195.072	73.152	0.56721E-05
195.072	67.056	0.38878E-05
195.072	60.960	0.56000E-06
188.976	60.960	0.62370E-06
182.880	60.960	0.69790E-06
176.784	60.960	0.78680E-06
170.688	60.960	0.89180E-06
164.592	60.960	0.10206E-05
158.496	60.960	0.11767E-05
152.400	60.960	0.13720E-05
146.304	60.960	0.16170E-05
146.304	54.864	0.14461E-04
146.304	48.768	0.24675E-05

TABLE B-21. Estimated 8-Hour Average Concentrations of Particle-Associated 2,4,5-T at Receptor Locations (x, y Coordinates) Around the Perimeter of the Herbicide Orange Site During Cement Cover Construction (continued)

X Coordinate (m)	Y Coordinate (m)	8-Hour Average Concentration (g/m^3)
146.304	42.672	0.12779E-04
146.304	36.576	0.46928E-05
146.304	30.480	0.76916E-05
146.304	24.384	0.96747E-05
146.304	18.288	0.14987E-05
146.304	12.192	0.90909E-05
146.304	6.096	0.72170E-05
146.304	0.000	0.11172E-05
140.208	0.000	0.51352E-05
134.112	0.000	0.10979E-04
128.016	0.000	0.53256E-05
121.920	0.000	0.39781E-05
115.824	0.000	0.14191E-04
109.728	0.000	0.36897E-05
103.632	0.000	0.13373E-04
97.536	0.000	0.72842E-05
91.440	0.000	0.14543E-04
85.344	0.000	0.60284E-05
79.248	0.000	0.19081E-04
73.152	0.000	0.23527E-05
67.056	0.000	0.19081E-04
60.096	0.000	0.60333E-05
54.864	0.000	0.14543E-04
48.768	0.000	0.72884E-05
42.672	0.000	0.13373E-04
36.576	0.000	0.36904E-05
30.480	0.000	0.14191E-04
24.384	0.000	0.39781E-05
18.288	0.000	0.53256E-05
12.192	0.000	0.10979E-04
6.096	0.000	0.51352E-05

Appendix C

RISK FOCUS

November 13, 1990

Captain Alan Holck
AFOEHL/EHT
Brooks Air Force Base, TX 78235-5501

Dear Captain Holck:

Enclosed please find a trip report for the Johnston Island site visit conducted on October 10-11, 1990. Please note the questions and needs expressed at the end of the report. This information is important to the successful completion of the project. Some of the information (e.g., location of fish sampling stations 4 and 6) will be easily obtained by us in a phone conversation with Roger DiRosa of FWS.

Yours truly,

Scott R. Baker, Ph.D.
Deputy Director

Attachment

Trip Report for Visit to Johnston Island

October 10-11, 1990

Background for the Trip and its Objectives

The RiskFocus Division of Versar is conducting a baseline risk assessment for the Occupational and Environmental Hygiene Laboratory for the Herbicide Orange (HO) storage site at Johnston Island. This risk assessment is part of the site investigation/remediation process related to EPA's regulations on the cleanup of hazardous waste and is being performed in the context of DoD's Installation Restoration Program. A major objective of the risk assessment is to determine the potential for human exposure to contaminants at the HO storage site (using the existing information on site characterization) and the potential human health risk that is the consequence of exposure. In this regard, the site was visited as part of the "investigation" phase of the study, during which several points of information to support the objectives of the study were identified and obtained (to the extent possible). The information to be obtained during the site visit included the following:

- The nature of morbidity (related to the known health effects of HO) among long-term residents of the island, particularly those who participated in the HO leak containment, dedrumming, and drum crushing operations;

- The sampling strategy used by personnel of the Fish and Wildlife Service to determine the levels of dioxin, 2,4,-D and 2,4,5,-T in water, sediments, and biota;

- The need for and possible arrangement for additional sampling and monitoring;

- The relation of site to other activities on the island that might present confounding factors on the risk from exposure to the HO site (e.g., potential for exposure to dioxin from the JACADS operation as it impacts the dioxin risk potential from exposure to the HO site);

- Background information on the potential for contamination of seawater with dioxin at the HO site (e.g., design and construction of the seawall surrounding the site), and

- Based on the physical layout of the island, activities of its residents, and prevailing meteorology, preliminary impressions about the potential for exposure to contaminants at the HO site.

The knowledge gained from the site visit in relation to these points of information is presented in the following descriptions. Recommendations for additional data collection activities, based on site-visit observations and the objectives of the baseline risk assessment, are presented in text in context with specific observations that are being made.

The Nature of Morbidity Among Long-term Residents of the Island

In accordance with the objectives of the study, it is important to determine if current long-term residents on the island are at risk from exposure to contaminants at the HO site. This includes, in particular, residents who participated in the HO removal activities in 1977 and who are still on the island (estimated to be 16 individuals). It does not include residents who are on the island for short durations

(one year or less) because short-term exposure to low levels of potential contaminants at the HO site are not presumed to result in a health risk from a toxicological perspective. It also does not include residents who have resided on the island in the past and who are not currently residing there. Current and future exposure for these latter individuals is presumed to be zero; therefore, their attendant current and future risk is presumed to be zero.

The staff of the medical unit indicated that limb injuries (sprains, bruises) constitute most of the health complaints on the island. Dr. Patrick, a physician currently assigned to JI, estimated that fewer than 50% of the residents smoke, although he did not have enumerative statistics on smoking incidence. He also observed that, to his knowledge, few residents have clinically diagnosed allergies (respiratory, dermal, and other immunologic responses from plants, food, dust, pollen, and in particular chemical exposure). In part, this may be the result of the relatively pollution-free atmosphere over the island, the lack of extensive pollen-bearing plant life on the island, and the relatively constant winds that promote high air exchange around the atoll. Three or four cases of breast cancer have occurred over the years, in addition to one melanoma (which was present prior to residence on the island but which metastasized while on the island), and one case of lung cancer in a smoker. Any hematological workups that were needed were done at the Straub Clinic on Oahu.

As a matter of due course, a more aggressive occupational medicine program should be instituted on the island, including medical monitoring, to determine if the island's hazards, including the HO site, are impacting the health of its long-term civilian residents.

Sixteen (16) individuals who are still on the island worked at the HO site. A list of these individuals was provided. Their medical histories should be examined for HO-related illnesses.

Sampling Strategy Used to Determine the Levels of HO Constituents in Water, Sediments, and Biota

Because the island is a National Wildlife Refuge, personnel of the U.S. Fish and Wildlife Service were present to manage the animal life on land and in the surrounding waters. Their activities center around identification, enumeration, and further characterization of biota in the island environment, and in assisting Federal departments in the sampling and analysis of biological and environmental samples for evidence of chemical contamination. In that context, the FWS staff were drawing fish and sediment samples to support the JACADS monitoring program for dioxin. Samples of fish and sediment are being drawn on a semiannual basis from the area surrounding the HO site. Although a degree of order and record keeping are maintained by FWS staff in their sampling regimen, there is no scientifically-based, systematic collection scheme (i.e., sampling method, frequency, location, and fish-type) in place with an objective of monitoring the potential migration and bioaccumulation of contaminants in the aquatic environment. Sampling parameters are left to the discretion of FWS staff. Reports of tissue and sediment analyses being conducted by Radian Corporation have been made available. The most recent analytical results were provided by FWS staff during the site visit. FWS staff are embarking on a sample collection and monitoring program to support the JACADS activity. This will be centered on the coral reef downrange of the HO site and presents a potential for collaboration with sampling needs for the HO site investigation (see below).

Need for and Possible Arrangement for Additional Sampling and Monitoring

A potential protocol for future aquatic sampling was discussed at length with FWS staff on the island. The stated objective is to determine the possible link between HO site contamination, sediment/water/fish contamination, and human consumption of contaminated fish (by catching them off the west wharf near the HO

site). The sampling plan should be responsive to this objective and was conceived as presented below for further consideration:

The physical layout of the area consists of, on land, the HO site and west wharf, and, in water, a seawall, reef, and intermediate area between the seawall and reef. To draw links between the HO site and the potential human consumption of contaminated fish caught at the fishing wharf, samples should be taken at the following locations:

- *Snails (a representative of filter feeders) and sediment (to determine if HO site contaminants are leaching from site to sediment or seawater) immediately off the HO site;*
- *Goat fish (representative of an intermediate aquatic trophic level) and sediment in the intermediate area off the HO site;*
- *Herbivores and predatory fish (representative of a higher trophic level) and sediment at the reef off the HO site;*
- *Sediment at the reef off the fishing wharf;*
- *Sediment at the intermediate area off the fishing wharf;*
- *Sediment at the seawall off the fishing wharf; and*
- *Fish that are caught by individuals fishing off the wharf.*

There is some question as to whether or not fish migrate between waters off the wharf area and waters off the HO site, and whether fish at the reef come inland as potential catch. The fish tagging and tracking effort that would be required to

address this issue is a costly and labor-intensive undertaking. The above plan circumvents the need for such an elaborate activity by drawing links between HO site contamination and actual catch.

Dr. Phillip LaBelle of the Woods Hole Oceanographic Institute will be embarking on a sampling regimen related to the JACADS operation to monitor the existence of furans, dioxins, and PCB's in sediments and fish at the reef and west camera stand. This presents an opportunity for the Air Force to collaborate on any need for further sampling with that being conducted by Dr. LaBelle for the Aberdeen Proving Ground. The JACADS monitoring program will begin shortly so that timely decisions on the need for additional sampling related to the HO site are needed. It is anticipated that, as long as stack monitors at the JACADS incinerators do not detect these chemicals at the stack, no JACADS-related chemicals will appear in biota off the west end of the island.

Well-placed locations for drawing *a few* water samples should be ascertained. As a substitute for taking extensive water samples, it may be sufficient to place current meters in the water to gain additional knowledge of present-day current patterns. This, in combination with existing empirical information on currents in the Atoll in general, may provide information on the potential role of currents in the distribution of HO site contaminants and further information on the land/water/fish/sediment interfaces.

There is a need to get as accurate information as possible on consumption (frequency and quantity) of fish caught off the west end of the island, as well as the dioxin levels in those fish.

With regard to air monitoring, there is a distinct aroma of formulation constituents in the area of the transformer west of the HO site. Based on dioxin levels at selected locations within the site as determined in the 1986 soil characterization

study, it is plausible that dioxin and other HO formulation ingredients (2,4-D, 2,4,5-T, emulsifiers, pH buffers, detergents, stabilizers, etc.) as cocontaminants may be volatilizing from the site. Since fire-training, burn-pit, and possibly other activities occur in this downwind area, the air as a potential source of personnel exposure to HO-site derived chemicals should be monitored for 2,4,-D and 2,4,5-T and in particular 2,3,7,8-dioxin that may be volatilizing from the HO site. Tomato plant bioassays provide only crude estimates of the presence of dioxin according to the severity of epinastic growth. This bioassay is not sufficient for human exposure estimation.

Activities on the Island as Potential Confounders to Risks from the HO Site

There is a potential for a confounding effect presented by two possible carcinogen-generating sources on the island other than the HO site:

- The JACADS facility is located upwind of the HO site and activities west of the site. The potential for dioxin release from JACADS is unknown. For purposes of the baseline risk assessment related to the HO site, it will be assumed that the potential for JACADS to pose a confounding influence in air or water media is negligible. Nevertheless, should there be airborne dioxin, furan, or other carcinogenic releases from the JACADS incinerators and dioxin releases from the HO site, any concentrations at locations west of the HO site would have to be apportioned between the two sources by air dispersion modeling (requiring knowledge of the source term). The reliability of results presented by modeling may be questionable enough to warrant additional monitoring. Currently, monitoring for dioxin related to the JACADS operation is being conducted only at the stack; downrange (Hi-Vol) samplers are monitoring for criteria pollutants and not for organics.

- The current fire training area is located immediately downrange of the HO site. Since this is a combustion operation (probably fueled by a petroleum-based product), there is a possibility that the area is contaminated with PAH's (i.e., carcinogens) including benzpyrenes and dioxin. Soil analyses of this area as presented in the 1986 soil characterization study reveal levels of 15 and 24 ppb in the fire training area. This may impact health risks associated with the HO site through both air and water media in ways that are difficult to predict with existing data.

Potential for Contamination of Seawater with Chemicals at the HO Site

Some aquatic and sediment samples have contained dioxin to varying degrees. If continuing monitoring of sediments and fish reveals contamination, particularly if the levels that are not diminishing with time, the possibility that the HO site as a source of dioxin in water must be explored. The seawall risers surrounding the HO site are lined with an impervious tough material near to the top of the seawall as it adjoins the ground of the HO site. There are two potential sources of migration of contaminants at the site to the surrounding aquatic environment:

- Backwash of contaminated soil over the seawall on those rare meteorological occasions when seawater is able to climb over the wall;

- Possible confluence between the groundwater aquifer under the site with the sea. The groundwater aquifer under the HO site has not been characterized. To ascertain if groundwater is a potential source of fugitive escape, the following prudent protocol should be conducted:

  - At hot cells on the HO site, bore holes into the water table;

- If groundwater is contaminated, characterize both the aquifer and the contaminant plume;
- Determine if the plume is (or is predicted to) reach the seawater;
- Determine the frequency of topsoil being washed out to sea;
- Estimate wind erosion and sea deposition of topsoil from the site; and
- Determine levels of dioxin in sediments and biota (see above: Need for and Possible Arrangement for Additional Sampling and Monitoring).

Preliminary impressions about the potential for exposure to contaminants at the HO site based on the physical layout of the island, activities of its residents, and prevailing meteorological features

Because the HO site is at the western edge of the island in the presence of prevailing easterly winds, there is not much potential for exposure via the air. There is also not much potential for confounding effects from the JACADS facility due to design and safety features of that facility; any JACADS releases will be acute episodic with health consequences (if any) that are different from those posed by HO-site contaminants. The fire training area poses a more plausible source of confounding synergistic or potentiative exposure because of its proximity to the HO site (i.e., the possibility that personnel working around the fire training area might receive exposures from the HO site) and the probable similarity in mode of action of contaminants from the HO site and the fire training area. The health status of islanders is a complete unknown (smoking histories, morbidity). As a result it will be difficult to select likely sensitive individuals. In accordance with HHEM procedures, risk will be determined for the MEI (most exposed individual) and MEAP (most exposed actual person). Considering the air and water as transport media for HO-derived dioxin and other HO-site contaminants (i.e., the only potential sources of

exposure), water poses a greater risk because of fish contamination and human consumption.

Followup Information Needed

In order to conduct a thorough analysis for the baseline risk assessment, we would like to obtain answers to the following questions:

- *What is the formulation composition of HO (chemicals and % wt)?* This will help us determine the range of contaminants present at the site. Presumably the maker (Dow Chemical) of HO would have this information. It may be more readily available in Air Force files than by starting with a cold call to Dow.

- *How much time (frequency and time interval per occurrence) do people spend downwind of the HO site (at the burn pit and the fire training area)?* Someone (who?) on JI would have to provide estimates.

- *Where would we be able to obtain automated meteorological data (data tape or disk) for the island?*

- *Who designed the seawall?* We would like to find out the principle of seawall operation, water dynamics through the seawall, and the likelihood of leakage of water through it.

- *Can you help us locate Colonel Nay (?) at Tyndall AFB?* He was the base engineer during the time of the HO removal operation. He may be able to provide information on the location of specific operations (e.g., burning of dunnage, use of ash for fill).

- *Can you help us obtain a copy of JACADS EIS Second Supplemental for Storage and Ultimate Disposal of the European Chemicals (first and/or second versions)?*

- *What are stations 4 and 6 identifying locations from which fish are being sampled?*

- *Can you please furnish the following documents cited in the Holmes and Narver Preliminary Assessment of Johnston Atoll (October 1989):*

  - Channell, R.E., and T.L. Stoddart, April 1984, *Herbicide Orange Monitoring Program: Interim Report, January 1980-December 1982*, ESL-TR-83-56, ESL, AFESC, Tyndall AFB, Florida.

  - Rhodes, 2 Lt., Albert N., January 2, 1985, *Johnston Island Fish Samples*, Letter to USAF OEHL/EC.

  - Casanova, J.N., January 1986, *JI Survey Sampling and Analysis Project*, EG&G/Idaho, Inc., Idaho Falls, Idaho.

  - Casanova, J.N., March 1986, *Johnston Island Survey Sampling and Analysis Project Addendum I*, EG&G/Idaho, Inc., Idaho Falls, Idaho.

July 15, 2004

Honorable Anthony J. Principi
Secretary
Department of Veterans Affairs
Washington, DC 20420

Dear Mr. Secretary:

Thank you for your reply to my letter concerning the exposure of veterans who served on Johnston Atoll between 1971 and 1977. I am puzzled at your conclusion that there is not enough evidence to concede exposure of these veterans to Agent Orange.

Johnston Island, the largest of the islands comprising Johnston Atoll, is less than 2 miles long and less than a half mile wide. Approximately 113,400 kg of Agent Orange accidentally spilled in 1972 during redrumming after the Air Force brought approximately 5.18 million liters of unused Agent Orange from Vietnam to Johnston Island. In addition, 49,000 gallons per year of Agent Orange are estimated to have leaked from drums at the Johnston Island storage site. Dioxin contamination was attributed to soil transport (wind transport or surface water runoff).

Given the very small size of Johnston Island, and the wind transport and water runoff of contaminated soil, I am at a loss as to how it would be possible for a servicemember assigned to Johnston Island to avoid exposure to Agent Orange. I am enclosing copies of selected pages from "An Ecological Assessment of Johnston Atoll" which provided some of the information referenced in this letter.

I am requesting that you reconsider your decision concerning the likelihood that all veterans who served on Johnston Island during 1971 – 1977 were exposed to Agent Orange and dioxin. I also note that as late as 1994, the most toxic dioxin isomer (TCDD) with concentrations as high as 901.00 was still present at 28% of the soil samples tested at the Agent Orange storage site on Johnston Island. It is also possible that servicemembers who were stationed at the Agent Orange site as late as 1994 (such as those assigned to guard the area) were exposed to TCDD.

Kindly provide me with a response to this request by September 1, 2004. If you have any questions about this request or need further information, please contact Mary Ellen Mc Carthy, Democratic Staff Director, Subcommittee on Benefits at 202-225-9756. Thank you for your cooperation in this matter.

Sincerely,

LANE EVANS
Ranking Democratic Member

Department of Veterans Affairs

Report

REPORT TO TO SECRETARY OF THE DEPARTMENT OF VETERANS AFFAIRS

ON THE ASSOCIATION BETWEEN ADVERSE HEALTH EFFECTS

AND EXPOSURE TO AGENT ORANGE

CLASSIFIED

CONFIDENTIAL STATUS (1)

As Reported by Special Assistant

Admiral E.R. Zumwalt, Jr.

May 5, 1990

WARNING

NOT FOR PUBLICATION AND

RELEASE TO THE GENERAL PUBLIC

I. INTRODUCTION

On October 6, 1989 I was appointed as special assistant to Secretary Derwinski of the Department of Veterans Affairs to assist the Secretary in determining whether it is at least as likely as not that there is a statistical association between exposure to Agent Orange and a specific adverse health effect.

As special assistant, I was entrusted with evaluating the numerous data relevant to the statistical association between exposure to Agent Orange and the specific adverse health effects manifested by veterans who saw active duty in Vietnam. Such evaluations were made in accordance with the standards set forth in Public Law 98-542, the Veterans' Dioxin and Radiation Exposure Compensation Standards Act and 38 C.F.R. 1.17, regulations of the Department of Veterans Affairs concerning the evaluation of studies relating to health effects of dioxin and radiation exposure.

Consistent with my responsibilities as special assistant, I reviewed and evaluated the work of the Scientific Council of the Veterans' Advisory Committee on Environmental Hazards and commissioned independent scientific experts to assist me in evaluating the validity of numerous human and animal studies on the effects of exposure to Agent Orange and/or exposure to herbicides containing 2,3,7,8 tetrachlorodibenzo-para-dioxin (TCDD or dioxin). In addition, I reviewed and evaluated the protocol and standards employed by government sponsored studies to *assess* such studies' credibility, fairness and consistency with generally accepted scientific practices.

After reviewing the scientific literature related to the health effects of Vietnam Veterans exposed to Agent Orange as well as other studies concerning the health hazards of civilian exposure to dioxin contaminants, I conclude that there is adequate evidence for the Secretary to reasonably conclude that it is at least as likely as not that there is a relationship between exposure to Agent Orange and the following health problems: non—Hodgkin's lymphoma, chloracne and other skin disorders, lip cancer, bone cancer, soft tissue sarcoma, birth defects, skin cancer, porphyria cutanea tarda and other liver disorders, Hodgkin's disease, hematopoietic diseases, multiple myeloma, neurological defects, auto—immune diseases and disorders, leukemia, lung cancer, kidney cancer, malignant melanoma, pancreatic cancer, stomach cancer, colon cancer, nasal/pharyngeal/esophageal cancers, prostate cancer, testicular cancer, liver cancer, brain cancer, psychosocial effects and gastrointestinal diseases.

I further conclude that the Veterans' Advisory Committee on Environmental Hazards has not acted with impartiality in its review and assessment of the scientific evidence related to the association of adverse health effects and exposure to Agent Orange.

In addition to providing evidence in support of the conclusions stated above, this report provides the Secretary with a review of the scientific, political and legal efforts that have occurred over the last decade to establish that Vietnam Veterans who have been exposed to Agent Orange are in fact entitled to compensation for various illnesses as service-related injuries.

II. AGENT ORANGE USAGE IN VIETNAM

Agent Orange was a 50:50 mixture of 2,4-D and 2,4,5-T. The latter component, 2,4,5-T, was found to contain the contaminant TCDD or 2,3,7, 8-tetrachlorodibenzo-para-dioxin (i.e. dioxin), which is regarded as one of the most toxic chemicals known to man.[1]

From 1962 to 1971 the United States military sprayed the herbicide Agent Orange to accomplish the following objectives: 1) defoliate jungle terrain to improve observation and prevent enemy ambush; 2) destroy food crops; and 3) clear Vegetation around military installations, landing zones, fire *base* camps, and trails [2]

Unlike civilian applications of the components contained in Agent Orange which are diluted in oil and water, Agent Orange was sprayed undiluted in Vietnam. Military applications were sprayed at the rate of approximately 3 gallons per acre and contained approximately 12 pounds of 2,4-D and 13.8 pounds of 2,4,5-T.[3]

Although the military dispensed Agent Orange in concentrations 6 to 25 times the manufacturer's suggested rate, "at that time the Department of Defense (DOD) did not consider herbicide orange toxic or dangerous to humans and took few precautions to prevent exposure to it."' Yet, evidence readily suggests that at the time of its use experts knew that Agent Orange was harmful to military personnel.[5]

The bulk of Agent Orange herbicides used in Vietnam were reportedly sprayed from "Operation Ranch Hand" fixed wing aircraft. Smaller quantities were applied from helicopters, trucks, riverboats, and by hand. Although voluminous records of Ranch Hand missions are contained in computer records, otherwise known as the HERBS and Service HERBs tapes, a significant, if not major source of exposure for ground forces was from non— recorded, non Ranch Hand operations.[6]

Widespread use of Agent Orange coincided with the massive buildup of U.S. military personnel in Vietnam, reaching a peak in 1969 and eventually stopping in 1971. [7] Thus, according to an official of the then Veterans Administration, it was "theoretically possible that about 4.2 million American soldiers could have made transient or significant contact with the herbicides because of [the Ranch Hand Operation]." [8]

A. REASONS FOR PHASE OUT

Beginning as early as 1968, scientists, health officials, politicians and the military itself began to express concerns about the potential toxicity of Agent Orange and its contaminant dioxin to humans. For instance, in February 1969 The Bionetics Research Council Committee ("BRC') in a report commissioned by the United States Department of Agriculture found that 2,4,5-T showed a "significant potential to increase birth defects." [9] Within four months after the BRC report, Vietnamese newspapers began reporting significant increases in human birth defects ostensibly due to exposure to Agent Orange.[10]

By October, 1969, the National Institute of Health confirmed that 2,4,5—T could cause malformations and stillbirths in mice, thereby prompting the Department of Defense to announce a partial curtailment of its Agent Orange spraying.[11]

By April 15, 1970, the public outcry and mounting scientific evidence caused the Surgeon General of the United States to issue a warning that the use of 2,4,5-T might be hazardous to "our health". [12]

On the same day, the Secretaries of Agriculture, Health Education and Welfare, and the Interior, stirred by the publication of studies that indicated 2,4,5-T was a teratogen (i.e. caused birth defects), jointly announced the suspension of its use around lakes, ponds, ditch banks, recreation areas and homes and *crops* intended for human consumption.[13] The Department of Defense simultaneously announced its suspension of all uses of Agent Orange.[14]

B. HEALTH STUDIES

As Agent Orange concerns grew, numerous independent studies were conducted between 1974 and 1983 to determine if a link exists between certain cancerous diseases, such as non-Hodgkin's lymphoma and soft-tissue sarcomas, and exposure to the chemical components found in Agent Orange. These studies suggested just such a link.

In 1974, for example, Dr. Lennart Hardell began a study which eventually demonstrated a statistically significant correlation between exposure to pesticides containing dioxin and the development of soft tissue sarcomas.[15]

In 1974, Axelson and Sundell reported a two—fold increase of cancer in a cohort study of Swedish railway workers exposed to a variety of herbicides containing dioxin contaminants.[16]

By 1976, the Occupational Safety and Health Administration, established rigorous exposure criteria for workers working with 2,4,5-T. [17]

In 1977 the International Agency for Research on Cancer (IARC), while cautioning that the overall data was inconclusive, reported numerous anomalies and increased mortality rates in animals and humans exposed to 2,4-D or 2,4,5-T.[18]

In 1978, the Environmental Protection Agency issued an emergency suspension of the spraying of 2,4,5-T in national forests after finding "a statistically significant increase in the frequency of miscarriages" among women living near forests sprayed with 2,4,5-T.[19]

In 1980, another provocative mortality study of workers involved in an accident at an industrial plant which manufactured dioxin compounds suggested that exposure to these compounds resulted in excessive deaths from neoplasms of the lymphatic and hematopoietic tissues. [20]

On September 22, 1980, the U.S. Interagency Work Group to Study the Long-term Health Effects of Phenoxy Herbicides and Contaminants concluded "that despite the studies' limitations, they do show a correlation between exposure to phenoxy acid herbicides and an increased risk of developing soft-tissue tumors or malignant lymphomas."[21]

To be sure, there remain skeptics who insist that the studies failed in one respect or another to establish a scientifically acceptable correlation.[22] Yet, it can fairly be said that the general attitude both within and outside the scientific community was, and continues to be increasing concern over the mounting evidence of a connection between certain cancer illnesses and exposure to dioxins.

III. VETERANS' DIOXIN AND RADIATION EXPOSURE COMPENSATION STANDARDS ACT OF 1984

With the increasing volume of scientific literature giving credence to the belief of many Vietnam Veterans that exposure to Agent Orange during their military service was related to their contraction of several debilitating diseases -- particularly non-Hodgkin's lymphoma, soft tissue sarcoma ("STS") (malignant tumors that form in muscle fat, or fibrous connective tissue) and porphyria cutanea tarda ("PCT") (deficiencies in liver enzymes) --Vietnam Veterans rightfully sought disability compensation from the Veterans Administration ("VA").

The VA determined, however, that the vast majority of claimants were not entitled to compensation since they did not have service connected illnesses. [23] As a consequence, Congress attempted to alter dramatically the process governing Agent Orange disability claims through passage of the Veterans' Dioxin and Radiation Exposure Compensation Standards Act of 1984 (hereinafter the "Dioxin Standards Act") [24] To ensure that the VA provided disability compensation to veterans exposed to herbicides containing dioxin while serving in Vietnam,[25] Congress authorized the VA to conduct rulemaking to determine those diseases that were entitled to compensation as a result of a service-related exposure to Agent Orange.[26]

In promulgating such rules, the Dioxin Standards Act required the VA to appoint a Veterans' Advisory Committee on Environmental Hazards (the "Advisory Committee") -- composed of experts in dioxin, experts in epidemiology, and interested members of the public -- to review the scientific literature on dioxin and submit periodic recommendations and evaluations to the Administrator of the [27] Such experts were directed to evaluate the scientific evidence pursuant to regulations promulgated by the VA, and thereafter to submit recommendations and evaluations to the Administrator of the VA on whether "sound scientific or medical evidence" indicated a connection to exposure to Agent Orange and the manifestation of various diseases.[28]

In recognition of the uncertain state of scientific evidence and the inability to make an absolute causal connection between exposures to herbicides containing dioxin and affliction with various rare cancer diseases.[29] Congress mandated that the VA Administrator resolve any doubt in favor of the veteran seeking compensation. As stated in the Dioxin Standards Act:

> It has always been the policy of the Veterans Administration and is the policy of the United States, with respect to individual claims for service connection of diseases and disabilities, that when, after consideration of all the evidence and material of record, there is an approximate balance of positive and negative evidence regarding the merits of an issue material to the determination of a claim,

> **the** benefit of the doubt in resolving each such issue shall be given to the claimant. [30]

A. NEHMER V. U.S. VETERANS ADMINISTRATION

Despite Congressional intent to give the veteran the benefit of the doubt, and in direct opposition to the stated purpose of the Dioxin Standards Act to provide disability compensation to Vietnam Veterans suffering with cancer who were exposed to Agent Orange, the VA continued to deny compensation improperly to over 31,000 veterans with just such claims. In fact, in promulgating the rules specified by Dioxin Standards Act, the VA not only confounded the intent of the Congress, but directly contradicted its- own established practice of granting compensable service-connection status for diseases on the lesser showing of a statistical association, promulgating instead the more stringent requirement that compensation depends on establishing a cause and effect relationship.[31]

Mounting a challenge to the regulations, Veterans groups prosecuted a successful legal action which found that the VA had "both imposed an impermissibly demanding test for granting service connection for various diseases and refused to give the veterans the benefit of the doubt in meeting the demanding standard." Nehmer v. U.S. Veterans Administration, 712 F. supplement 1404, 1423 (1989) (Emphasis in original) As a result, the court invalidated the VA's Dioxin regulation which denied service connection for all diseases other than chloracne; ordered the VA to amend its rules; and further ordered that the Advisory Committee reassess its recommendations in light of the court's order.[32]

Thus, on October 2, 1989, the VA amended 38 C.F.R. Part 1, which among other things set forth various factors for the Secretary and the Advisory Committee to consider in determining whether it is "at least as likely as not" that a scientific study shows a "significant statistical association" between a particular exposure to herbicides containing dioxin and a specific adverse health effect.[33] Equally important, the regulation permits the Secretary to disregard the findings of the Advisory Committee, as well as the standards set forth at 38C.F.R. § 1.17 (d) and determine in his own judgment that the scientific and medical evidence supports the existence of a "significant statistical association" between a particular exposure and a specific disease 38 C.F.R. § 1.17 (f).

The Secretary recently exercised his discretionary authority under this rule when he found a significant statistical association between exposure to Agent Orange and non-Hodgkin's lymphoma, notwithstanding the failure of his own Advisory Committee to recommend such action in the face of overwhelming scientific data.[34]

B. THE WORK OF THE VETERANS' ADVISORY COMMITTEE ON ENVIRONMENTAL HAZARDS

To assess the validity and competency of the work of the Advisory Committee, I asked several impartial scientists to review the Advisory Committee transcripts. Without exception, the experts who reviewed the work of the Advisory Committee disagreed with its findings and

further questioned the validity of the Advisory Committee's review of studies on non-Hodgkin's lymphomas.

For instance, a distinguished group at the Fred Hutchinson Cancer Research Institute in Seattle, Washington, upon reviewing the Advisory Committee transcripts, concluded "that it is at least, as likely as not that there is a significant association *(as* defined by the Secretary of Veterans Affairs) between (exposure to phenoxy acid herbicides and non-Hodgkin's lymphoma.)" 35 This same group further asserts that the Committee's work was "not sensible" and "rather unsatisfactory" in its review and classification of the various studies it reviewed. Additionally, these scientists regarded Dr. Lathrop's views as "less than objective" and felt that the possibility exists that "his extreme views (e.g., in respect to the role of dose--response testing) may have unduly affected the Committee's work." Finally, the Hutchinson scientists argue that the issue of chemical-specific effects, in which animal studies have been sufficient to demonstrate the carcinogenicity of dioxin, is an important factor "not well considered by the Committee." (Emphasis in original)

A second reviewer of the Committee's work, Dr. Robert Hartzman (considered one of the U.S. Navy's top medical researchers), effectively confirms the views of the Hutchinson group. Dr. Hartzman states that "the preponderance of evidence from the papers reviewed [by the Advisory Committee) weighs heavily in favor of an effect of Agent Orange on increased risk for non-Hodgkin's lymphoma."[36] Dr. Hartzman also attests that: An inadequate process is being used to evaluate scientific publications for use in public policy. The process uses scientific words like 'significant at the 5% level' and a committee of scientists to produce a decision about a series of publications. But in reality, the Committee was so tied by the process, that a decision which should have been based on scientific data was reduced to vague impressions... Actually, if the reading of the rules of valid negative found in the transcript is correct ('a valid negative must be significant at the p=.05 level' that is statistically significant on the negative side) none of the papers reviewed are valid negatives. [37]

A third reviewing team, Dr. Jeanne Hager Stellman, PhD (Physical Chemistry) and Steven D. Stellman, PhD (Physical Chemistry), also echo the sentiments expressed by the Hutchinson Group and Dr. Hartzman on the validity of the Committee's proceedings and conclusions. In fact, the Stellmans' detailed annotated bibliography and assessment of numerous cancer studies relevant to herbicide exposure presents a stunning indictment of the Advisory Committee's scientific interpretation and policy judgments regarding the link between Agent Orange and Vietnam Veterans. [38]

A fourth reviewer, a distinguished scientist intimately associated with government sponsored studies on the effects of exposure to Agent Orange, states the same conclusions reached by the other reviewers:

> The work of the Veterans' Advisory Committee on Environmental Hazards, as documented in their November 2, 1989 transcript, has little or no scientific merit, and should not serve as a basis for compensation or regulatory decisions of any sort...

> My analysis of the NHL articles reviewed by the committee reveals striking patterns which indicate to me that it is much more likely than not that a statistical association exists between NHL and herbicide exposure.

As these various reviewers suggest, the Advisory Committee's conclusions on the relationship between exposure to Agent Orange and non-Hodgkin's lymphoma were woefully understated in light of the clear evidence demonstrating a significant statistical association between NHL and exposure to phenoxy acid herbicides such as Agent Orange.

Perhaps more significant than the Committee's failure to sake such obvious findings is the distressing conclusion of the independent reviewers that the Committee's process is so flawed as to be useless to the Secretary in making any determination on the effects of Agent Orange. From a mere reading of Committee transcripts, these reviewers detected overt bias in the Committee's evaluation of certain studies. In fact, some members of the Advisory Committee and other VA officials have, even before reviewing the evidence, publicly denied the existence of a correlation between exposure to dioxins and adverse health effects.[40] This blatant lack of impartiality lends credence to the suspicion that certain individuals may have been unduly influenced in their evaluation of various studies. Furthermore, such bias among Advisory committee members suggests that the Secretary should, in accordance with the Dioxin Standards Act, appoint new personnel to the Advisory Committee.

III. THE CDC STUDIES

Were the faulty conclusions, flawed methodology and noticeable bias of the Advisory Committee an isolated problem, correcting the misdirection would be more manageable. But, experience with other governmental agencies responsible for specifically analyzing and studying the effects of exposure to Agent Orange strongly hints at a discernible pattern, if not outright governmental collaboration, to deny compensation to Vietnam Veterans for disabilities associated with exposure to dioxin .

A case in point is the Centers for Disease control ("CDC"). As concerns grew following the first studies of human exposure to Agent Orange, Congress commissioned a large scale epidemiological study to determine the potential health effects for Vietnam Veterans exposed to Agent Orange. Initially, this study was to be conducted by the VA itself. When evidence surfaced, however, of the VA's foot-dragging in commencing the study (and initial disavowal of any potential harm from exposure to Agent Orange), Congress transferred the responsibility for the study to the CDC in 1983. [41]

Unfortunately, as hearings before the Human Resources and Intergovernmental Relations Subcommittee on July 11, 1989 revealed, the design, implementation and conclusions of the CDC study were so ill conceived as to suggest that political pressures once again interfered with the kind of professional, unbiased review Congress had sought to obtain.[42]

The Agent Orange validation study, for example, a study of the long-term health effects of exposures to herbicides in Vietnam, was supposedly conducted to determine if exposure could, in fact, be estimated.[43] After four years and approximately $63 million in federal funds,

the CDC concluded that an Agent Orange exposure study could not be done based on military records. [44] This conclusion was based on the results of blood tests of 646 Vietnam Veterans which ostensibly demonstrated that no association existed between serum dioxin levels and military-based estimates of the likelihood of exposure to Agent Orange.[45] Inexplicably, the CDC then used these "negative" findings to conclude that not only could an exposure study not even be done, but that the "study" which was never even conducted proves that Vietnam Veterans were never exposed to harmful doses of Agent Orange.

Even more disturbing, when the protocol for this "study" and the blood test procedures were examined further, there appeared to be a purposeful effort to sabotage any chance of a meaningful Agent Orange exposure analysis. For, the original protocol for the Agent Orange exposure study understandably called for subject veterans to be tracked by company level location.[46] By tracking company level units of 200 men, rather than battalions of 1,000 men, the location of men in relation to herbicide applications would be known with greater precision, thereby decreasing the probability that study-subjects would be misclassified as having been or not been exposed to Agent Orange.

However, in 1985 the CDC abruptly changed the protocol to have battalions, rather than companies, serve as the basis for cohort selection and unit location. [47] By the CDC's own admission, changing the protocol to track veterans on the broader batta1ion basis effectively diluted the study for the simple reason that many of the 1,000 men in a battalion were probably not exposed to Agent Orange. Why then did the CDC change the protocol in 1985?

According to Dr. Vernon Houk, Director of the Center for Environmental Health and Injury control, the department within the CDC responsible for conducting the Agent Orange study, the protocol was changed because the CDC concluded that company-specific records were unreliable and contained too many gaps of information. As a result, military records could simply not be used to assess exposure.[48]

Richard Christian, the former director of the Environmental Study Group of the Department of Defense ("ESG") testified that not only was this conclusion false, but that he had personally informed the CDC that adequate military records existed to identify company-specific movements as well as spray locations.[49] Furthermore, in a February 1985 report to the Congressional Office of Technology Assessment, the CDC reported that in analyzing 21 of 50 detailed computer HERBs tapes developed by the ESG on company movements that it was possible to correlate the exposure data to areas sprayed with Agent Orange with consistent results.[50] Indeed, a peer reviewed study sponsored by the American Legion conclusively demonstrated that such computerized data could be used to establish a reliable exposure classification system essential to any valid epidemiologic study of Vietnam Veterans.[51]

In addition to altering the protocol from company units to battalions, the CDC further diluted the study by changing the protocol on the length of time study subjects were to have served in Vietnam. Whereas the original protocol required subjects to have served a minimum of 9 months in combat companies, the CDC reduced the minimum to 6 months. Furthermore, the CDC eliminated from consideration all veterans who served more than one tour in Vietnam. Finally, while the original protocol called only for subjects who served in Vietnam from 1967 to

1968, the years that Agent Orange spraying was at its height, the CDC added an additional 6 months to this time period. The net effect of these various changes was seriously to dilute the possibility that study subjects would have been exposed to Agent Orange, which in turn would impair any epidemiological study's ability to detect increases in disease rate.[52]

Although the above referenced problems cast serious suspicion on the work of the CDC, perhaps its most controversial action was to determine unilaterally that blood tests taken more than 20 years after a veteran's service in Vietnam were the only valid means of determining a veteran's exposure to Agent Orange. In addition, Dr. Houk further "assumed" that the half-life for dioxin in the blood was seven years. [53] When the underlying data for Houk's assumptions were recently reviewed, however, 11 percent of the blood tests were invalid (i.e. study subjects had higher values of dioxin in their blood in 1987 than in 1982 even though the subjects had no known subsequent exposure to dioxin) and the half lives of dioxin in the remaining study subjects ranged from a low of 2 to a high of 740 years! [54] Yet despite this tremendous variance in the data and the high incidence of false results, Houk and the CDC concluded, rather remarkably, that a large scale exposure study was simply not possible since "negative" blood tests appeared to "confirm" that study subjects were not even exposed to Agent Orange.

Such conclusions are especially suspect given the fact that scientists have consistently cautioned against the use of blood tests as the sole basis for exposure classification. Although blood and adipose tissue tests can be used to confirm that Vietnam veterans were heavily exposed to Agent Orange and the contaminant dioxin[55], even the CDC's own researchers have unequivocally stated that "much more has to be learned about the kinetics of dioxin metabolism and half-life before current levels can be used to fully explain historic levels of exposure."[56]

While the CDC's changes in protocol have been "justified", however unreasonably, on the basis of "scientific" explanations[57], what cannot be justified is the evidence of political interference in the design, implementation and drafting of results of the CDC study by Administration officials rather than CDC scientists. As early as 1986, the Subcommittee on Oversight and Investigations of the Committee on Energy and Commerce documented how untutored officials of the Office of Management and Budget (0MB) interfered with and second-guessed the professional judgments of agency scientists and multidisciplinary panels of outside peer review experts effectively to alter or forestall CDC research on the effects of Agent Orange, primarily on the grounds that "enough" dioxin research had already been done.[58] These Agent Orange Hearings revealed additional examples of political interference in the CDC~s Agent Orange projects by members of the White House Agent Orange Working Group.[59]

Dr. Philip Landrigan, the former Director of the Environmental Hazards branch at the CDC, upon discovering the various irregularities in CDC procedures concluded that the errors were so egregious as to warrant an independent investigation not only of the methodology employed by the CDC in its validation study, but also a specific inquiry into what actually transpired at the Center for Environmental Health of the CDC.[60]

With these suspicions in mind, it should come as no surprise that those familiar with the CDC~s work found little credence in the conclusions reached by the CDC in its recently released Selected Cancers Study. Even though the CDC has previously stated that it believes exposure to

Agent Orange is impossible to assess, it found no difficultly in reporting to the *press* upon the release of the Selected Cancers Study that exposure to Agent Orange does not cause cancer. This conclusion was reached despite the fact that the CDC made no effort to determine, through military records or blood/adipose tissue tests, if study subjects were, indeed, exposed to dioxins; nor did the CDC attempt to verify exposure to Agent Orange of those study subjects who actually contracted cancerous diseases. In fact, according to scientists who have made preliminary reviews of the CDC's findings, the statistical power of any one cancer grouping, with the exception of non-Hodgkin's lymphoma, was so low as to make any conclusion virtually impossible.

IV. RANCH HAND STUDY

Unfortunately, political interference in government sponsored studies associated with Agent Orange has been the norm, not the exception. In fact, there appears to have been a systematic effort to suppress critical data or alter results to meet preconceived notions of what alleged scientific studies were meant to find.[61] As recently as March 9, 1990 Senator Daschle disclosed compelling evidence of additional political interference in the Air Force Ranch Hand study, a separate government sponsored study meant to examine the correlation between exposure to Agent Orange and harmful health effects among Air Force veterans who participated in Agent Orange spraying missions under Operation Ranch Hand. As Senator Daschle explained:

> In January 1984, the scientists in charge of the Ranch Hand Study issued a draft baseline morbidity report that described some very serious health problems in the Ranch Hand veterans and stated that the Ranch Handers, by a ratio of five to one, were generally less well than the veterans in the control group. The opening sentence of the draft report's conclusion was clearly stated: "It is incorrect to interpret this baseline study as 'negative.'
>
> After the Ranch Hand Advisory Committee, which operates under the White House Agent Orange Working Group of the Domestic Policy Council, got its hands on the document, the final report was changed in some very important ways. Most notably, the table and exposition explaining that the Ranch Handers were generally less well than the controls was omitted, and the final conclusion was altered substantially. The statement that the baseline study was not negative was completely omitted and the study was described as "reassuring." [62]

By altering the study's conclusion, opponents of Agent Orange compensation were able to point to "irrefutable proof" that Agent Orange is not a health problem: if those veterans most heavily exposed to Agent Orange did not manifest any serious health problems, they argued, then it could safely be deduced that no veteran allegedly exposed to Agent Orange in smaller doses could have health problems. Yet, when Senator Daschle questioned Air Force scientists on why discrepancies existed between an Air Force draft of the Ranch Hand Study and the final report actually released to the press, the answers suggested not merely disagreements in data evaluation, but the perpetration of fraudulent conclusions. In a word, the major premise was badly flawed.

For example, in 1987 Ranch Hand scientists confirmed to Senator Daschle that an unpublished birth defects report shows that birth defects among Ranch Hand children are double those of children in the control group and not "minor" as originally reported in l984.[63]

This increase in birth defects takes on added significance when one considers that the original CDC birth defects study, which found no increase in birth defects, merely examined birth defects as reported on birth certificates, rather than as reported by the child's parent or physician. The CDC never recorded hidden birth defects, such as internal organ malformations and other disabilities that only became apparent as the child developed. Consequently, it is very likely that the CDC's negative findings on birth defects were also vastly understated.[64]

In addition to elevated birth defects, Ranch Handers also showed a significant increase in skin cancers unrelated to overexposure to the sun as originally suggested in the 1984 report. Air Force scientists also admitted that Air Force and White House Management representatives were involved in scientific decisions in spite of the study's protocol which prohibited such involvement.[65]

On February 23, 1990, the Air Force released a follow-up morbidity report on the Ranch Handers. That report, "1987 Follow-up Examination Results," described statistically significant increases in health problems among Ranch Handers including: all cancers--skin and systemic combined, both verified and suspected; skin cancers alone; hereditary and degenerative neurological diseases and other problems. The Air Force-concluded, however, that these and other problems cannot necessarily be related to Agent Orange/dioxin exposure, as they do not always show a "dose-response" relationship--particularly since the exposure index used in the data analysis "is not a good measure of actual dioxin exposure." [66]

With this conclusion, the Air Force for the first time officially acknowledged that the conclusions reached in its original 1984 Ranch Hand study are not simply moot, but that the Ranch Hand study is not, at this date, an Agent Orange study at all since dioxin exposure could not be determined reliably in the first place. In other words, the Air Force could just as easily have concluded that the health problems associated with the Ranch Handers were not necessarily related to eating beer nuts.

For the Air Force to have made the statement in 1990 of no evidence of a link between exposure to Agent Orange and the cancer problems experienced by Ranch Handers is, as Senator Daschle notes, "patently false."[67]. Although not yet conclusive, what the Ranch Hand and CDC studies demonstrate is that there is evidence of a link between health problems and dioxin exposures which may become definitive when a new and reliable exposure index is used to evaluate the data.

As stated by Dr. James Clary, one of the scientists who prepared the final Ranch Hand report:

> The current literature on dioxin and non-Hodgkin's lymphoma and soft tissue sarcoma can be characterized by the following:

1. It underestimates (reduced risk estimates) the effect of dioxins on human tissue systems. As additional studies are completed we can expect to see even stronger correlations of dioxin exposure and NHL/STS.

2. Previous studies were not sensitive enough to detect small, but statistically significant increases in NHL/STS. As time progresses, and additional evidence is forthcoming, it will be increasingly difficult for anyone to deny the relationship between dioxin exposure and NHL/STS

V. INDEPENDENT STUDIES

Shamefully, the deception, fraud and political interference that has characterized government sponsored studies on the health effects of exposure to Agent Orange and/or dioxin has not escaped studies ostensibly conducted by independent reviewers, a factor that has only further compounded the erroneous conclusions reached by the government.

For instance, recent litigation against the Monsanto Corporation revealed conclusive evidence that studies conducted by Monsanto employees to examine the health effects of exposure to dioxin were fraudulent. These same fraudulent studies have been repeatedly cited by government officials to deny the existence of a relationship between health problems and exposure to Agent Orange. According to court papers:

Zack and Gaffey, two Monsanto employees, published a mortality study purporting to compare the cancer death rate amongst the Nitro workers who were exposed to Dioxin in the 1949 explosion with the cancer death rate of unexposed workers. The published study concluded that the death rate of the exposed worker was exactly the same as the death rate as the unexposed worker. However, Zack and Gaffey deliberately and knowingly omitted 5 deaths from the exposed group and took 4 workers who had been exposed and put these workers in the unexposed group, serving, of course, to decrease the death rate in the exposed group and increase the death rate in the unexposed group. The exposed group, in fact, had 18 cancer deaths instead of the reported 9 deaths (P1 Ex 1464), with the result that the death rate in the exposed group was 65% higher than expected (emphasis in original) [69]. Similarly, recent evidence also suggests that another study heavily relied upon by those opposed to Agent Orange compensation to deny the existence of a link between dioxin and health effects was falsified. Three epidemiologic studies and several case report studies about an 1953 industrial accident in which workers at a BASF plant were exposed to dioxins concluded that exposure to TCDD did not cause human malignancies.[70] A reanalysis of the data that comprised the studies, all of which was supplied by the BASF company itself, revealed that some workers suffering from chloracne (an acknowledged evidence of exposure to dioxin) had actually been placed in the low--exposed or non--exposed cohort groups. Additionally, 20 plant supervisory personnel, not believed to have been exposed, were placed in the exposed group.

When the 20 supervisory personnel were removed from the exposed group, thereby negating any dilution effect, the reanalysis revealed statistically significant increases in cancers of the respiratory organs (lungs, trachea, etc.) and

When the 20 supervisory personnel were removed from the exposed group, thereby negating any dilution effect, the reanalysis revealed statistically significant increases in cancers of the respiratory organs (lungs, trachea, etc.) and cancers of the digestive tract.[71] According to the scientist who conducted this study, "(t)his analysis adds further evidence to an association between dioxin exposure and human malignancy."[72]

Recent evidence also reveals that Dow Chemical, a manufacturer of Agent Orange was aware as early as 1964 that TCDD was a byproduct of the manufacturing process. According to Dow' s then medical director, Dr. Benjamin Holder, extreme exposure to dioxins could result in "general organ toxicity" as well as "psychopathological" and "other systemic" problems. [73] In fact, a recent expert witness who reviewed Dow Chemical corporate documents on behalf of a plaintiff injured by exposure to dioxin who successfully sued Dow[74] states unequivocally that "the manufacturers of the chlorphenoxy herbicides have known for many years about the adverse effects of these materials on humans who were exposed to them."[75]

VI. CURRENT SCIENCE ON HEALTH EFFECTS OF HERBICIDES AND DIOXIN

Despite its poor record in carrying out its responsibility to ascertain the health effects of exposure to Agent Orange, the CDC has been candid in some of its findings. As early as 1983, for instance, the CDC stated in the protocol of its proposed Agent Orange Studies "(t) hat the herbicide contaminant TCDD is considered to be one of the most toxic components known. Thus any interpretation of abnormal findings related to 2,4,5-T must take into consideration the presence of varying or undetermined amounts of TCCD." [76]

In 1987, after first being leaked by the New York Times, a VA mortality study was released indicating a 110 percent higher rate of non-Hodgkin's lymphoma in Marines who served in heavily sprayed areas as compared with those who served in areas that were not sprayed. [77] The study also found a 58 percent higher rate of lung cancer among the same comparative groups. [78]

Also in 1987, a second VA study found a suggestive eight-fold increase in soft tissue sarcoma among veterans most likely to have been exposed to Agent Orange.[79]

A proportionate mortality study of deaths in pulp and paper mill workers in New Hampshire from 1975 to 1985 showed that one or more of the exposures experienced by such workers (dioxin is a byproduct of pulp and paper production) posed a "significant risk" for cancers of the digestive tract and lymphopoietic tissues. [80]

Another case control study of farmers in Hancock County, Ohio, showed a "statistically significant" rise in Hodgkin's disease and non-Hodgkin's lymphoma. Although the study speculates that exposure to phenoxy herbicides may be the cause of such elevated cancers, the study recognizes that, given the size of its cohort, the only credible conclusion that can be drawn

is that it "adds to the growing body of reports linking farming and malignant lymphoma, particularly NHL." [81]

A study of disease and non-battle injuries among U.S. Marines in Vietnam from 1965 to 1972 showed a significantly higher rate of first hospitalizations for Marines stationed in Vietnam as opposed to Marines stationed elsewhere, particularly for neoplasms, diseases of the blood and blood forming organs and diseases of the circulatory and respiratory systems.[82] Of particular significance is the fact that the rate of first hospitalization for disease and non-battle injuries among Vietnam personnel rose steadily, reaching a peak in 1969, while the rate of non-Vietnam personnel remained relatively constant.[83] This rise in hospitalization for non-combat injuries coincides exactly with the increased use of Agent Orange, reaching a peak in 1969, and declining thereafter until its elimination in 1971.

In a recently published article entitled "2,4-D, 2,4,5-T, and 2,3,7,8-TCDD: An Overview", the authors acknowledge that at least three weaknesses in research related to dioxins are sufficient to cast doubt on the validity of any study. [84] The authors report that while the data on soft tissue sarcoma and phenoxy acids are too inconsistent to allow for any comment at this time, there is evidence of a strong association between STS and the suspect chemicals in 2 of the 8 studies analyzed in their article. Furthermore, the birth defect studies analyzed "suggest that adverse reproductive effects can be caused by (dioxin). [85]

Recent studies in Vietnam continue to show statistically significant reproductive anomalies and birth defects among women, and children of women presumably exposed to Agent Orange spraying.[86]

In the December 1, 1989, issue of *Cancer*, a study of the cancer risks among Missouri farmers found elevated levels of lip and bone cancer as well as nasal cavity and sinuses, prostrate, non-Hodgkin’s lymphoma and multiple myeloma. Smaller elevations, but elevations nonetheless, were found for cancers of the rectum, liver, malignant melanoma, kidney and leukemia. According to the authors, evidence of the cause for the elevated risks for these illnesses "may be strongest for a role of agricultural chemicals, including herbicides, insecticides and fertilizers." [87]

Both the U.S. Environmental Protection Agency (EPA) and the International Agency for Research on Cancer (IARC) have concluded that dioxin is a "probable human carcinogen." [88]

In a work entitled "Carcinogenic Effects of Pesticides" to be issued by the National Cancer Institute Division of Cancer Etiology, researchers conclude that while confirmatory data is lacking there is ample evidence to suggest that NHL, STS, colon, nasal and nasopharyngeal cancer can result from exposure to phenoxy herbicides .

A just released case control study of the health risks of exposure to dioxins confirmed previous findings that exposure to phenoxyacetic acids or chlorophenols entails a statistically significant increased risk (i.e. 1.80) for soft tissue sarcoma.[89]

As recently as February 28, 1990 an additional study found that farmers exposed to various herbicides containing 2,4-D may experience elevated risks for certain cancers, particularly cancers of the stomach, connective tissue, skin, brain, prostate, and lymphatic and hematopoietic systems."[90]

This week a scientific task force, after reviewing the scientific literature related to the potential human health effects associated with exposure to phenoxyacetic acid herbicides and/or their associated contaminants (chlorinated dioxins) concluded that it is at least as likely as not that exposure to Agent Orange is linked to the following diseases: non-Hodgkin's lymphoma, soft tissue sarcoma, skin disorders/chloracne, subclinical hepatotoxic effects (including secondary coproporphyrinuria and chronic hepatic porphyria), porphyria cutanea tarda, reproductive and developmental effects, neurologic effects and Hodgkin's disease.[91]

On the same day that this scientific task force reported a statistically significant linkage between exposure to the dioxins in Agent Orange and various cancers and other illnesses, the Environmental Protection Agency reported that the cancer risk posed by the release of such a "potent carcinogen" as dioxin in the production of white paper products is "high enough to require tighter controls on paper mills."[92]

CONCLUSIONS

As many of the studies associated with Agent Orange and dioxins attest, science is only at the threshold of understanding the full dimension of harmful toxic effects from environmental agents on various components of the human immune system. [93] In fact, a whole new discipline-immunotoxicology - has developed to explore further the effects of environmental chemicals on human health and to relate animal test results to humans.[94]

Immunotoxicology has established, however, at a minimum that at least three classes of undesirable effects are likely occur when the immune system is disturbed by environmental exposure to chemicals such as dioxin, including: 1) immunodeficiency or suppression; 2) alteration of the host defense mechanism against mutagens and carcinogens (one theory is that the immune system detects cells altered by mutagens or other carcinogenic trigger and destroys these cells. Thus, an impaired immune system may not detect and destroy a newly forming cancer); and 3) hypersensitivity or allergy to the chemical antagonist. Because of dioxin's ability to be both an immunosuppressant and a carcinogen, as early as 1978 immunologists were suggesting that "(a) gents such as TCDD may be far more dangerous than those possessing only one of these properties."[95]

While scientists are not in agreement, some immunotoxicologists argue that one molecule of a carcinogenic agent, like dioxin in the right place and at the right time can cause the human immune system to turn on itself, manifesting such breakdowns in the form of cancer. Indeed, even some courts have accepted this theory of causation in matters specifically related to exposure to dioxin.[96]

With additional evidence from Vietnam suggesting that Agent Orange contaminants have the ability to migrate away from actual spray locations via river channels and the food chain, the

opportunity for a Vietnam Veteran to have been exposed to dioxin contaminant molecules increases significantly.[97]

It cannot be seriously disputed that any large population exposed to chemical agents, such as Vietnam Veterans exposed to Agent Orange, is likely to find among its members a number who will develop malignancies and other mutagenic effects as a result of being exposed to harmful agents.

To be sure, decisions today with regard to the seriousness of Agent Orange health effects must be made while the science of immunotoxicology is in its infancy. After having evaluated and considered all of the known evidence on Agent Orange and dioxin contaminants, it is evident to me that enough is known about the current trends in the study of dioxins, and their linkage with certain cancers upon exposure, to give the exposed Vietnam Veteran the benefit of the doubt.

This benefit of the doubt takes on added credence given two separate means for determining exposure to Agent Orange - 1) HERBs and Service HERBs tapes establishing troop location for comparison with recorded Ranch Hand spraying missions; and 2) blood testing from living Veterans, to ascertain elevated dioxin levels. The inexplicable unwillingness of the CDC to utilize this data has had the effect of masking the real increase in the rate of cancers among the truly exposed. There is, in my opinion, no doubt that had either of these methods been used, statistically significant increased rates of cancer would have been detected among the Veterans for whom exposure can still be verified.

Since science is now able to conclude with as great a likelihood as not that dioxins are carcinogenic directly and indirectly through immunosuppression, and since a large proportion of those exposed to dioxin can be so ascertained, I am of the view that the compensation issue for service-related illnesses associated with exposure to Agent Orange should be resolved in favor of Vietnam Veterans in one of the two following ways:

COMPENSATION FOR SERVICE RELATED ILLNESSES

Alternative 1:

Any Vietnam Veteran, or Vietnam Veteran's child who has a birth defect, should be presumed to have a service-connected health effect if that person suffers from the type of health effects consistent with dioxin exposure and the Veteran' s health or service record establishes 1) abnormally high TCDD in blood tests; or 2) the veteran's presence within 20 kilometers and 30 days of a known sprayed area (as shown by HERBs tapes and corresponding company records); or 3) the Veteran' s presence at fire base perimeters or brown water operations where there is reason believe Agent Orange have occurred.

Under this alternative compensation would not be provided for those veterans whose exposure came from TCDD by way of the food chain; silt runoff from sprayed areas into unsprayed waterways; some unrecorded U.S. or allied Agent Orange sprayings; inaccurately recorded sprayings; or sprayings whose wind drift was greater than 20 kilometers. Predictably,