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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)

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[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

AUTHOR:

Pamela W. Smith

Science Policy Research Division

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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.

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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.)

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ARMSTRONG LABORATORY

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

Geoffrey Huse
Jeffrey Driver
Scott Baker
Louis Corio
Sally Kamem
Alfred Pinkney
Gary Whitmyre
Michelle Silkowski
Thomas Piccin
Leslye Wakefield
Nica Mostaghim
Sonja Young

RiskFocus
VERSAR Inc.
6850 Versar Center
Springfield, VA 22151

OCCUPATIONAL AND ENVIRONMENTAL
HEALTH DIRECTORATE
Brooks Air Force Base, TX 78235-5000

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Final Report for Period September 1990 – August 1991

AIR FORCE SYSTEMS COMMAND
BROOKS AIR FORCE BASE, TEXAS 78235-5000

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.

Alan R Holck

ALAN R. HOLCK, Captain, USAF, BSC
Project Scientist

Mark H. Stokes

MARK H. STOKES, Colonel, USAF, BSC
Deputy Chief, Occupational
Medicine Division

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6850 Versar Center
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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).

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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.

Scott R. Baker
Deputy Director, RiskFocus®
Virginia Office

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Contents

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

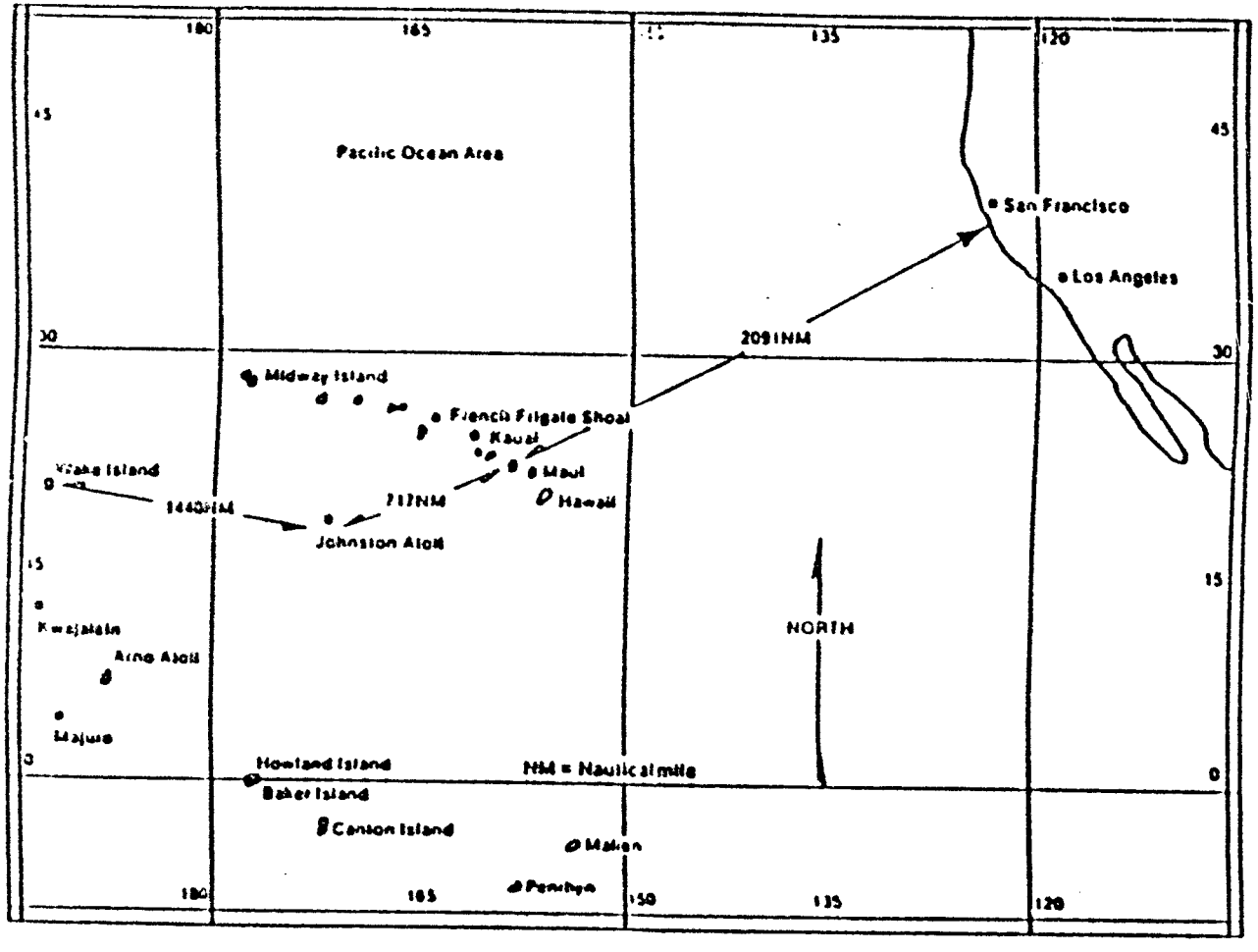
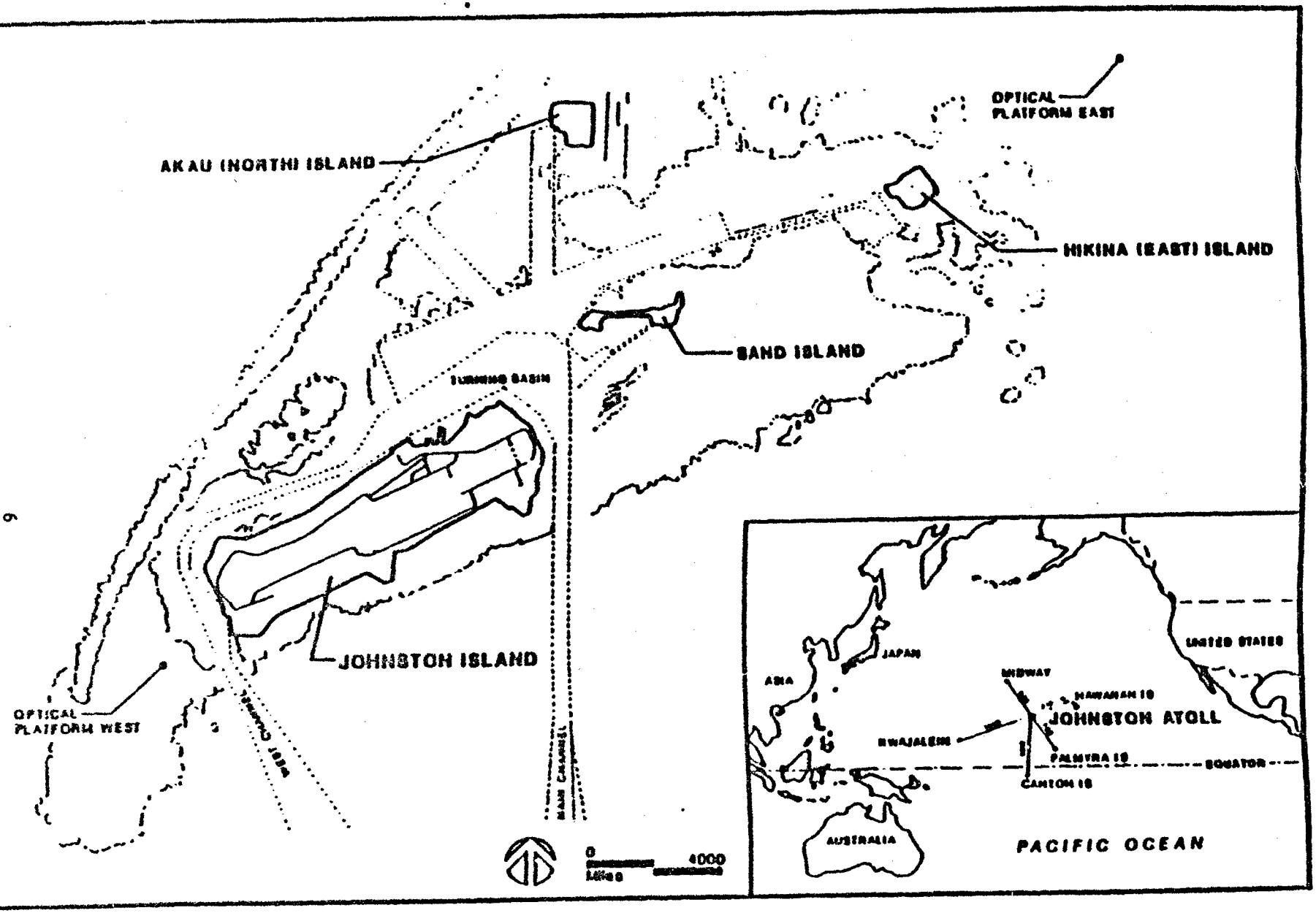


FIGURE 1.1 LOCATION OF JOHNSTON ISLAND
From Crockett AB 1986



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

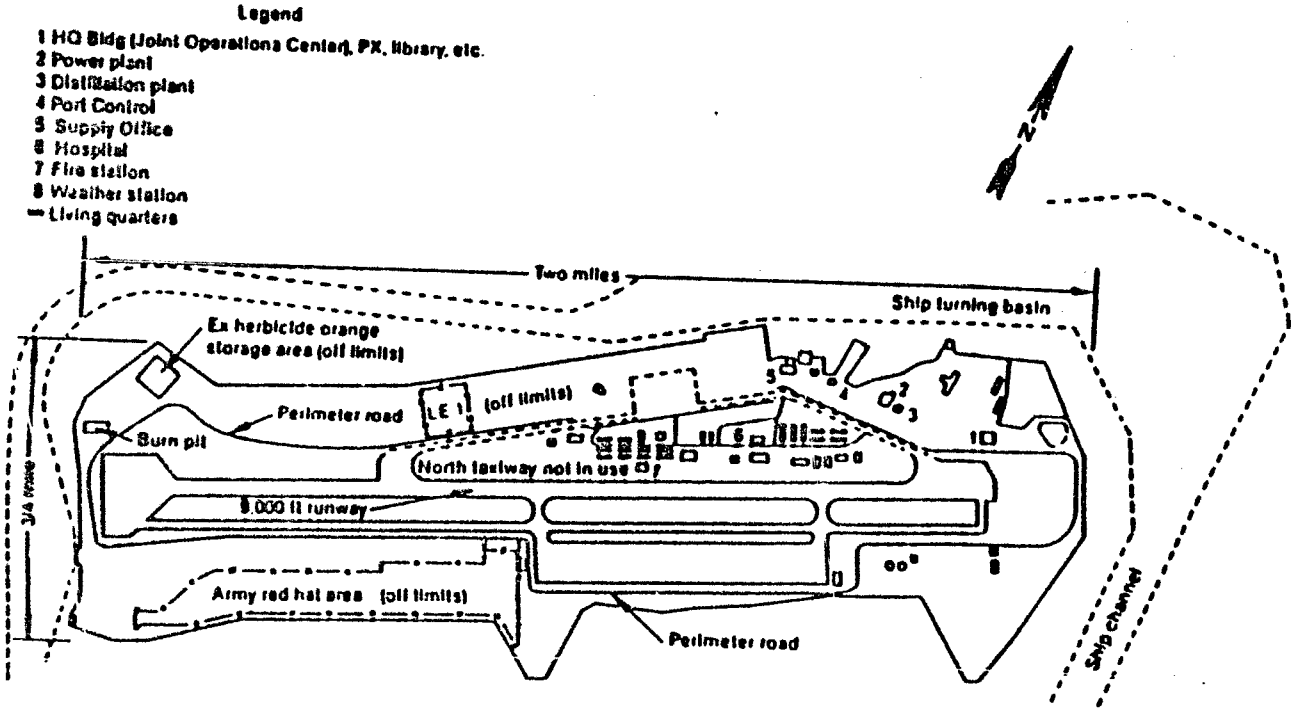


FIGURE 1.3 MAP OF JOHNSTON ISLAND
From Crockett AB 1986

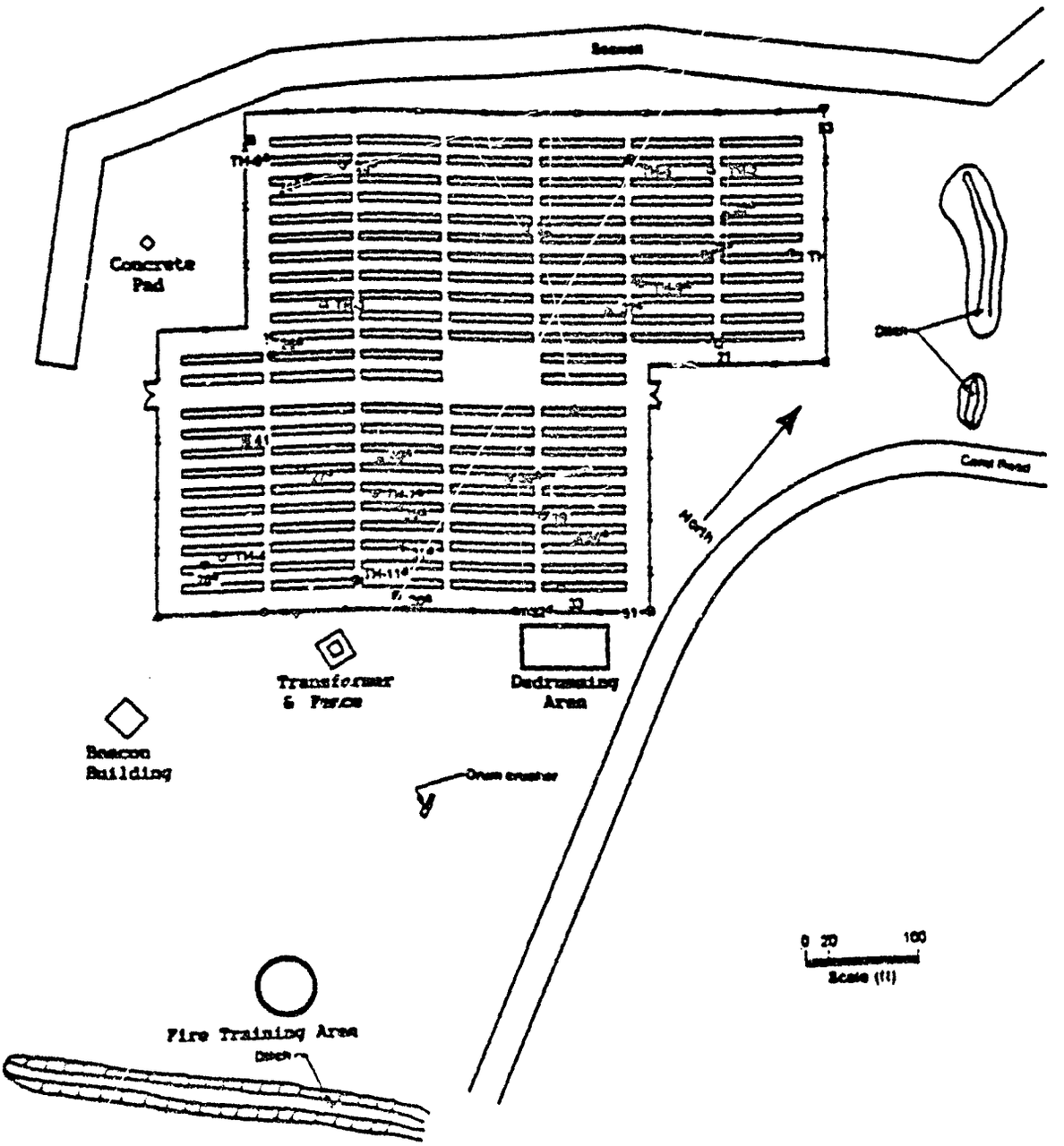


FIGURE 1.4 HERBICIDE ORANGE STORAGE AREA
From Crockett AB 1986

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.

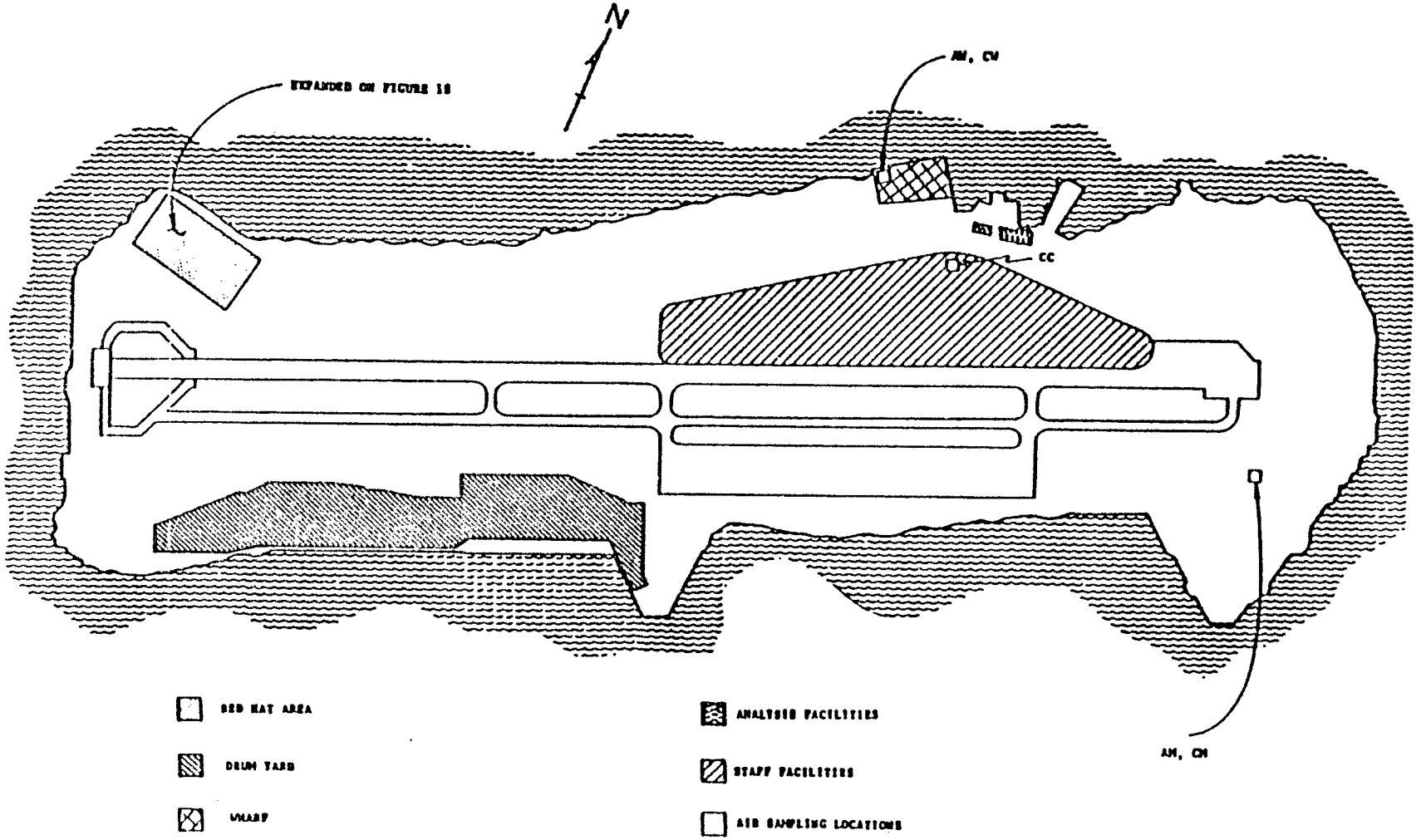


FIGURE 1.5 AIR SAMPLING SITES
From Thomas 1978

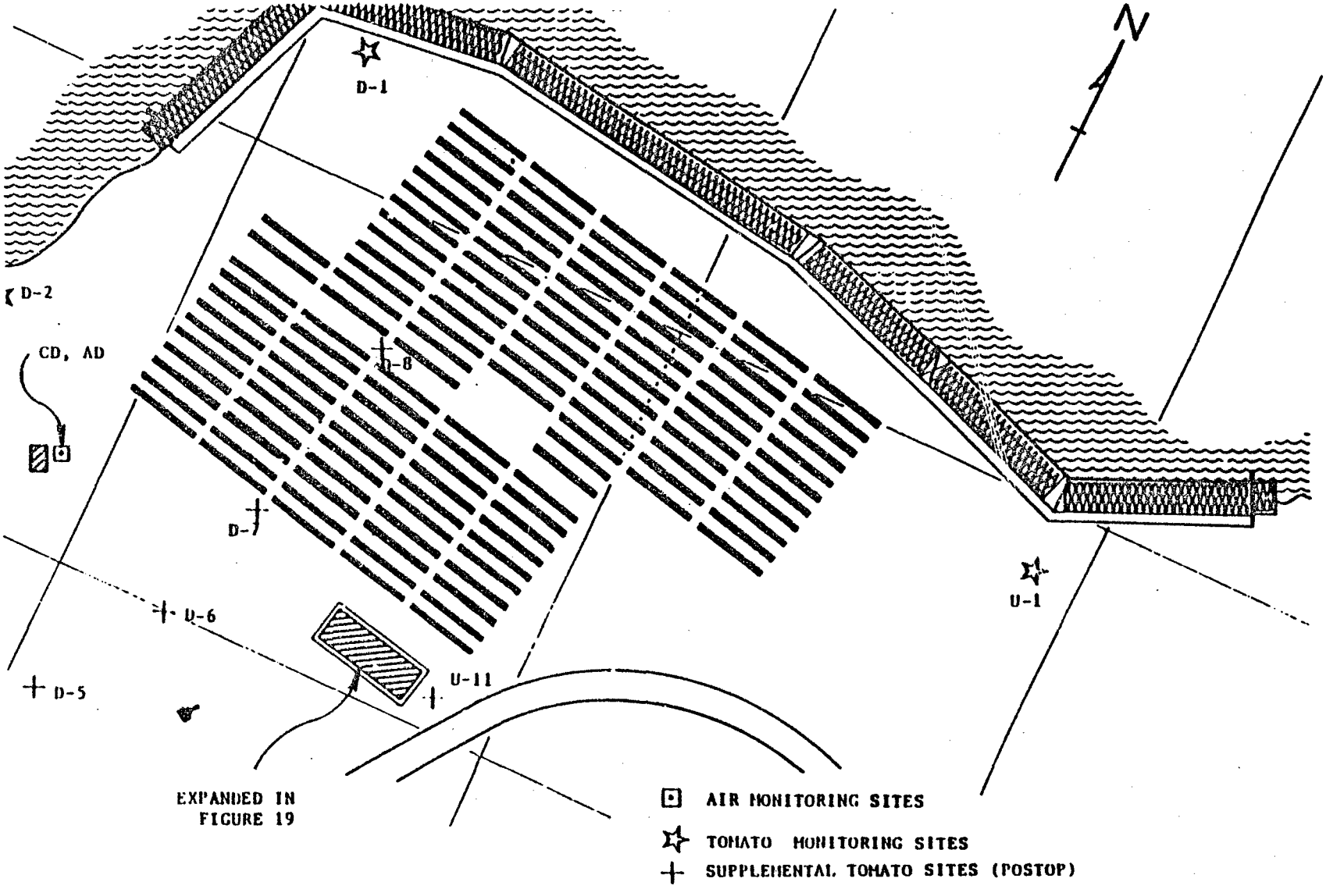


FIGURE 1.6 AIR MONITORING SITES, DRUMYARD
From Thomas 1978

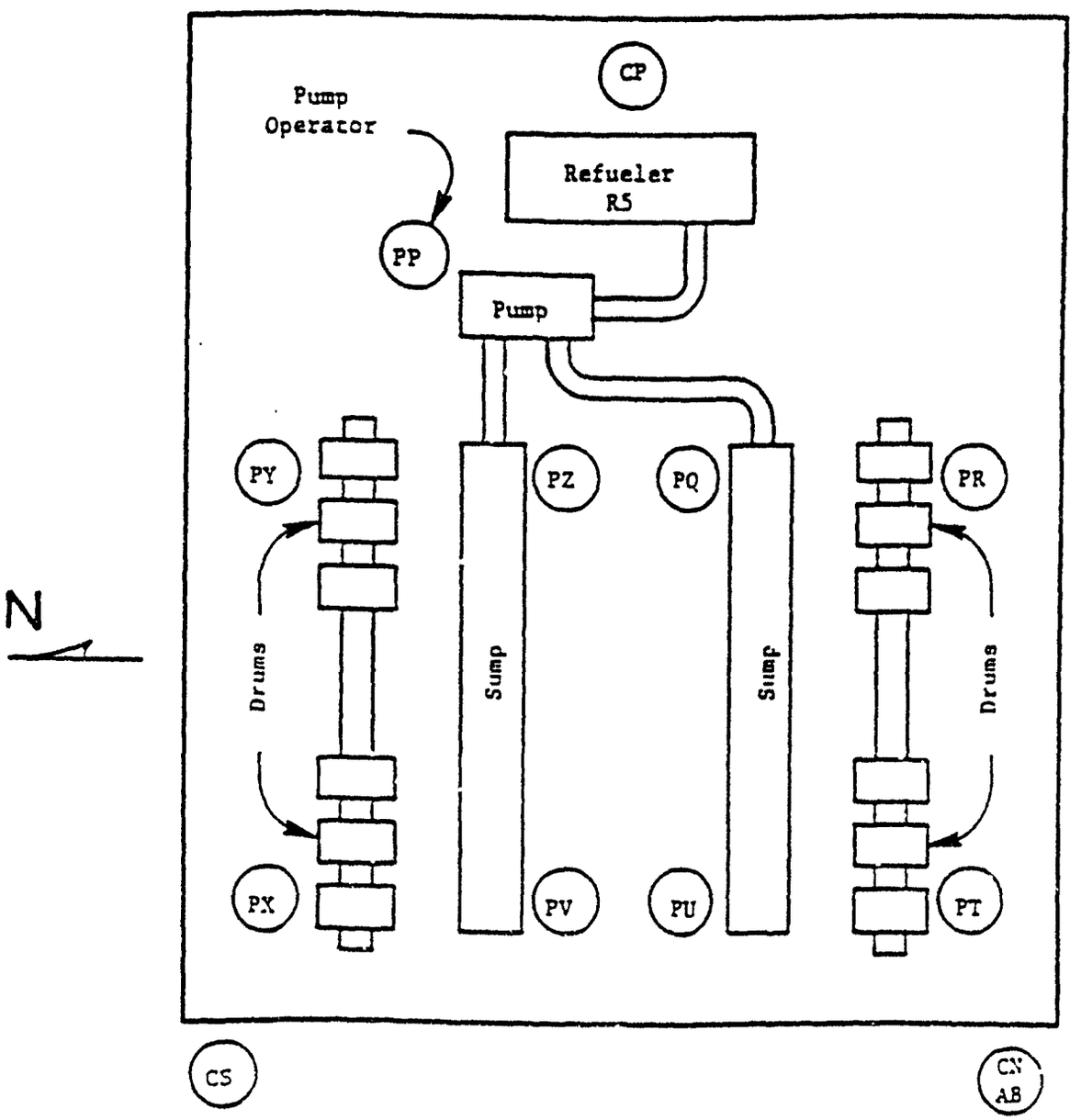


FIGURE 1.7 SAMPLING SITES AT DEDEAM FACILITY
From Thomas 1978

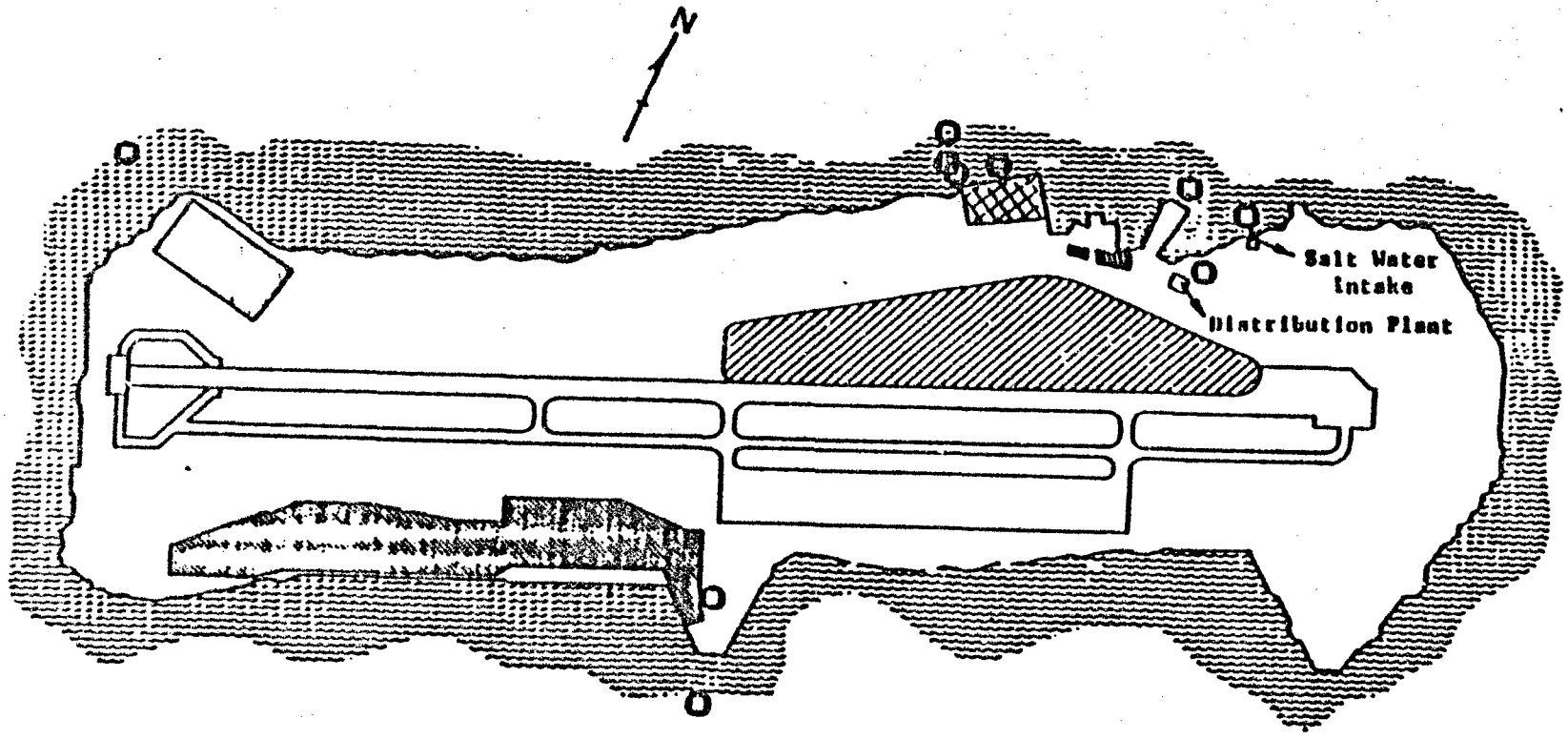


FIGURE 1.8 WATER AND SEDIMENT SITES
From Thomas 1978

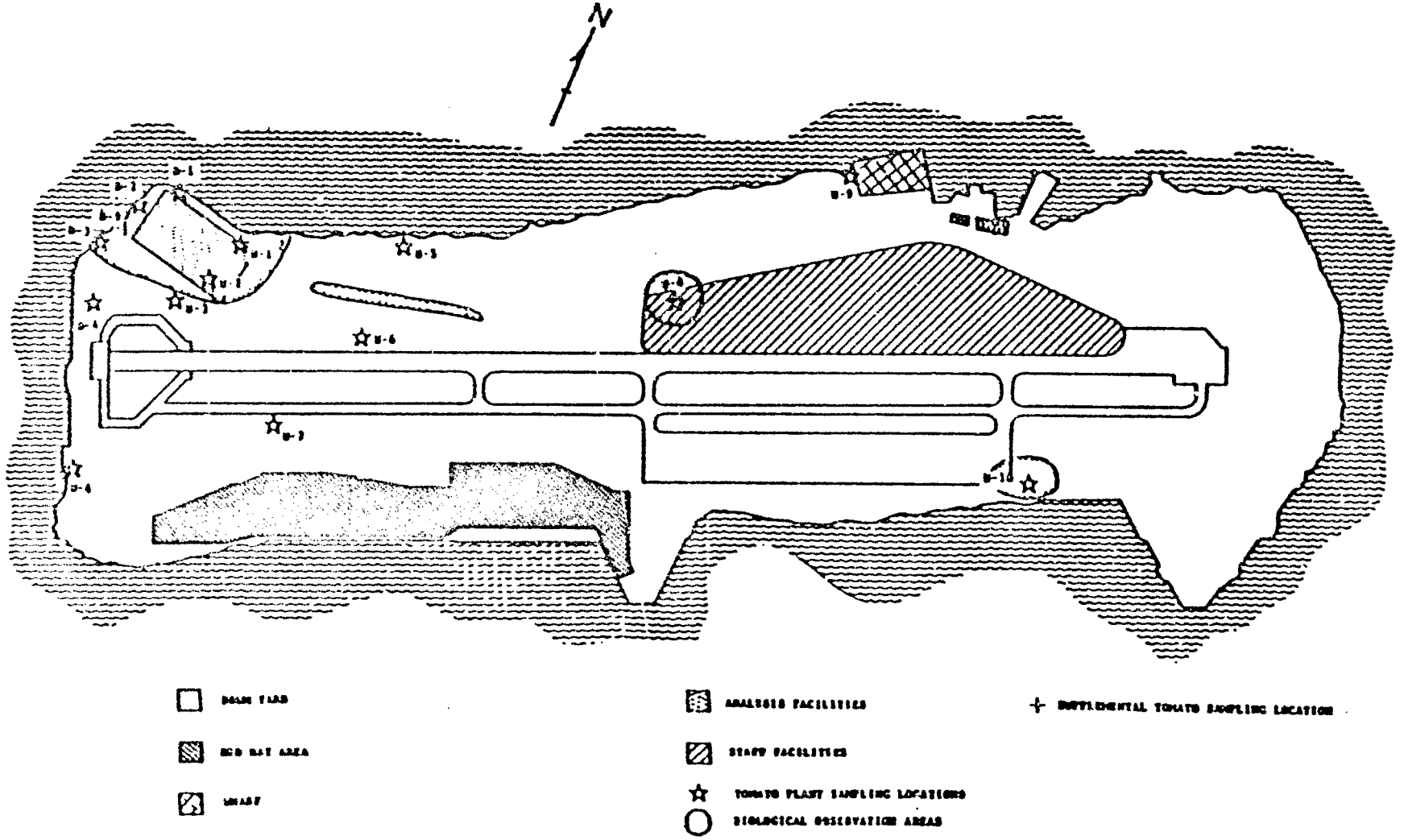


FIGURE 1.9 BIOTA MONITORING SITES
From Thomas 1978

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).

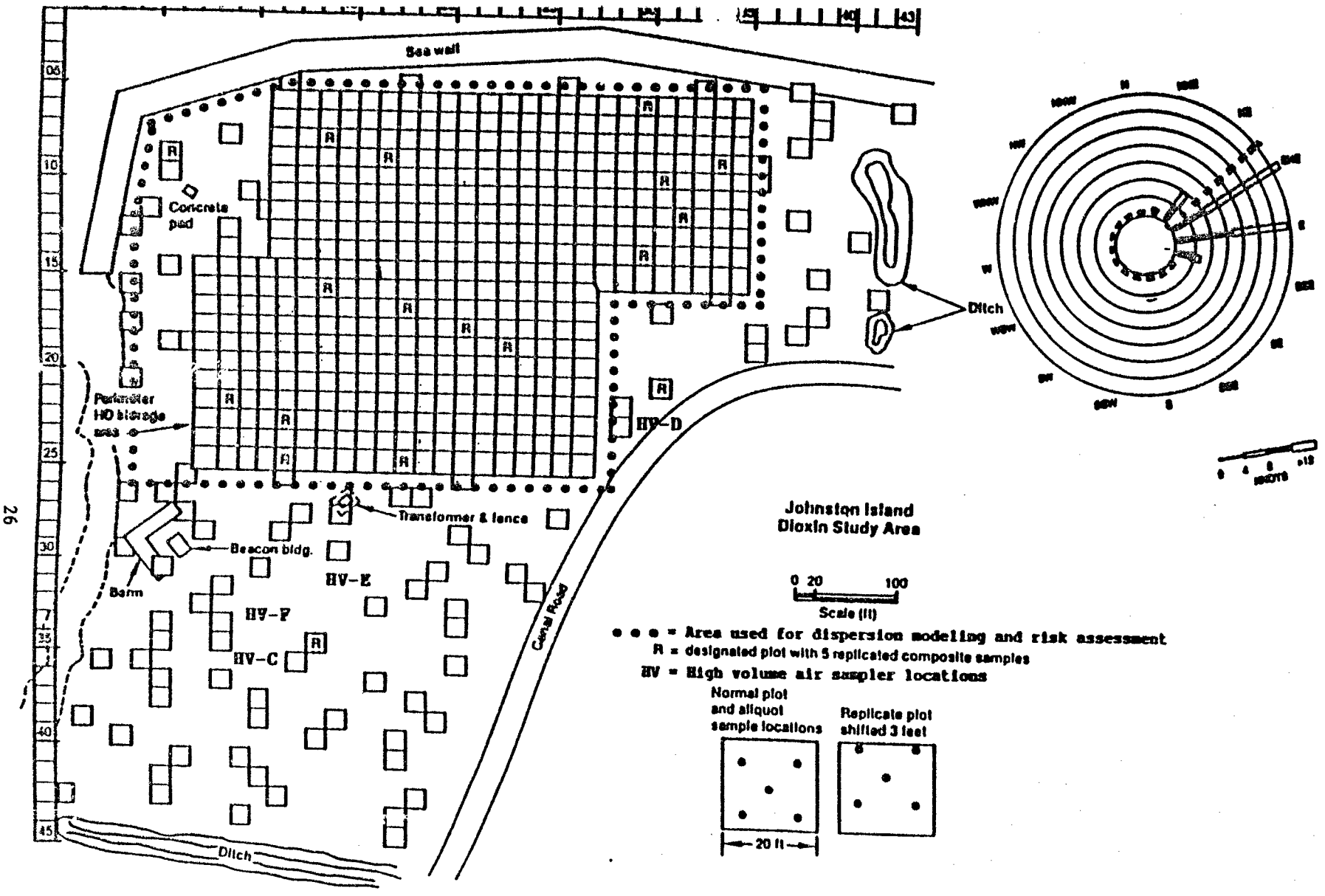


FIGURE 2.1 STUDY AREA GRID WITH REPLICATE SHIFT PATTERN

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)

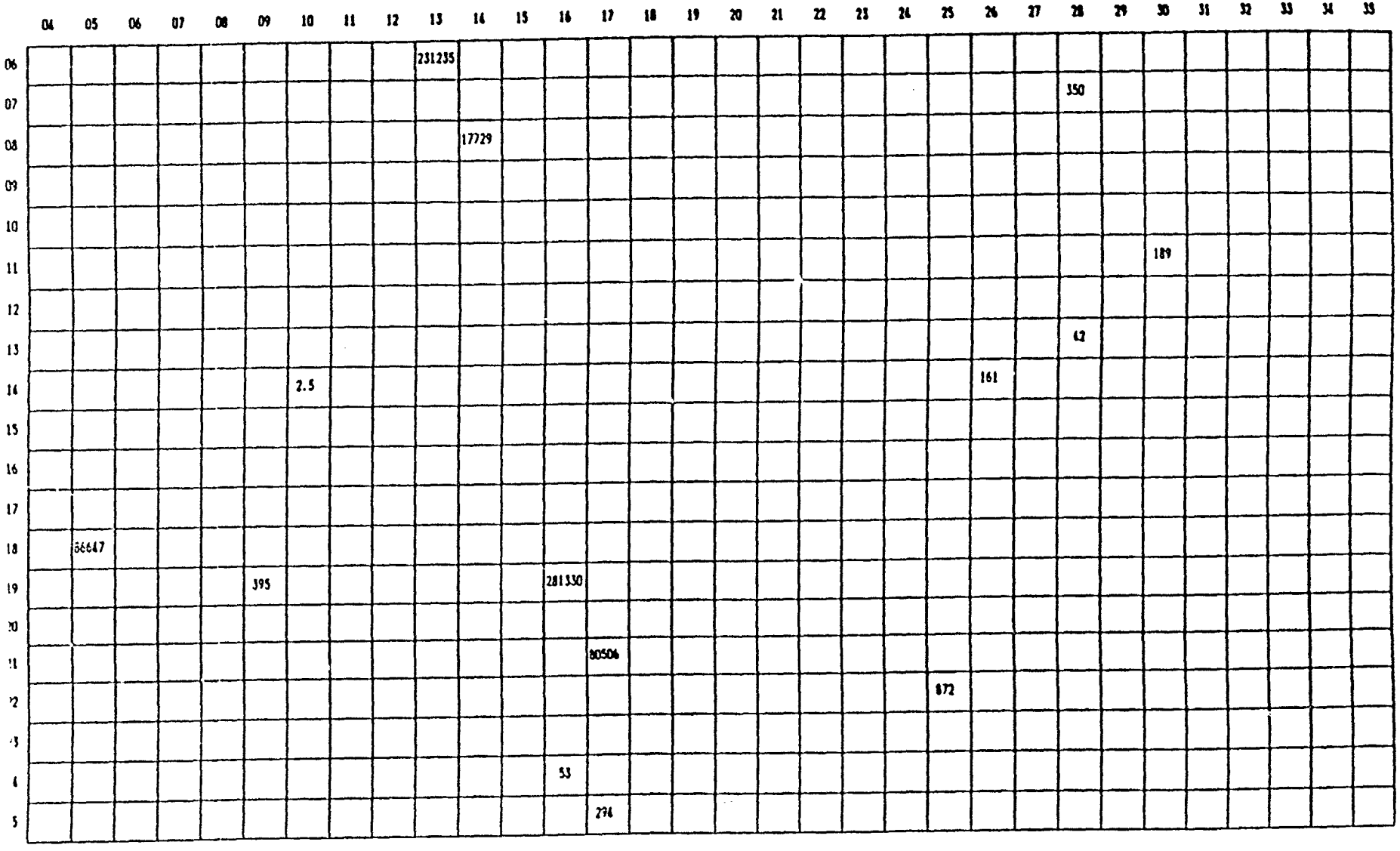


Figure 2.4 2,4,5-Trichlorophenoxy Acetic Acid Concentration (ppb)

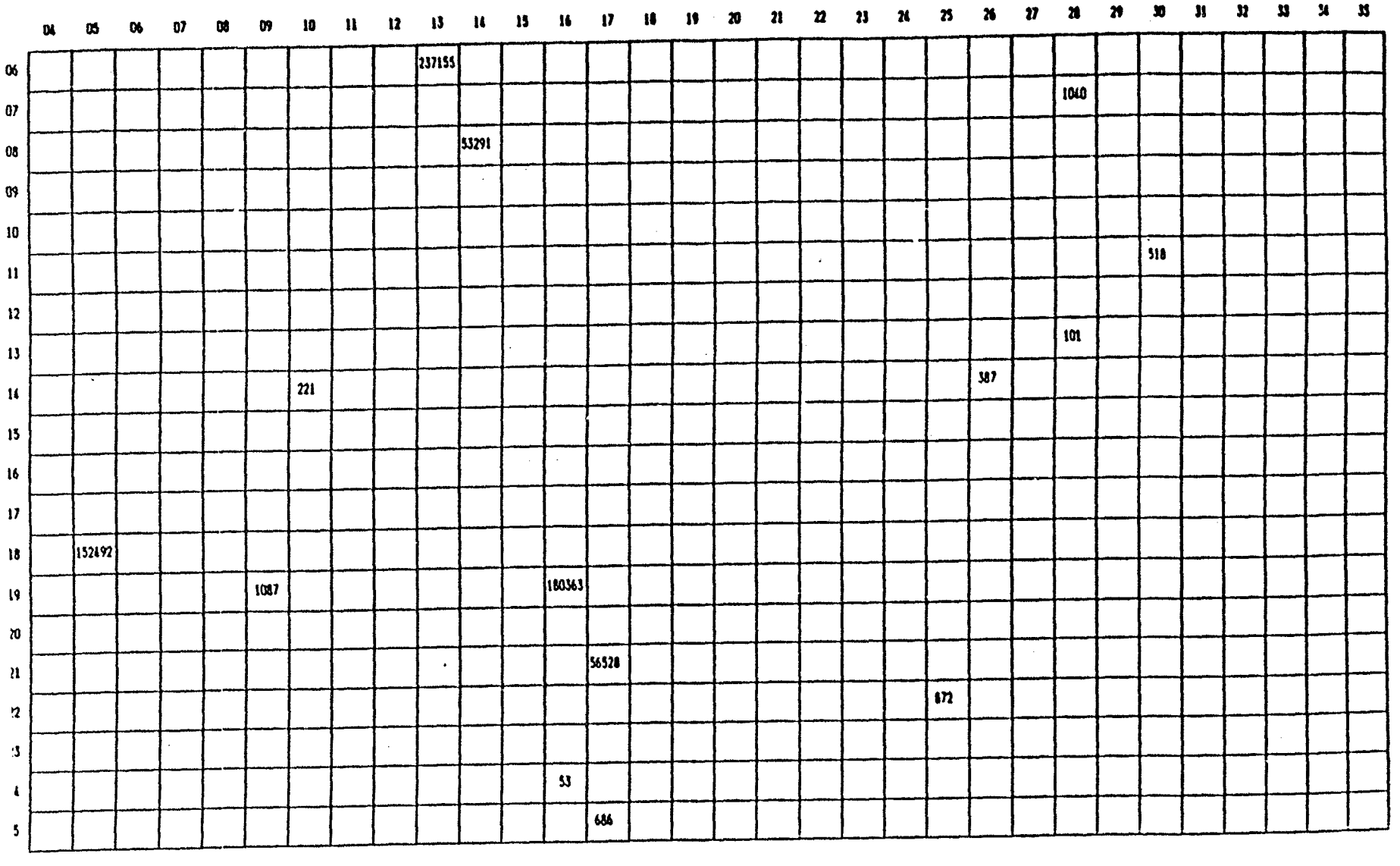


Figure 2.5 2,3,7,8-Tetrachlorodibenzo-p-dioxin Subsurface Soil Concentration (ppb)

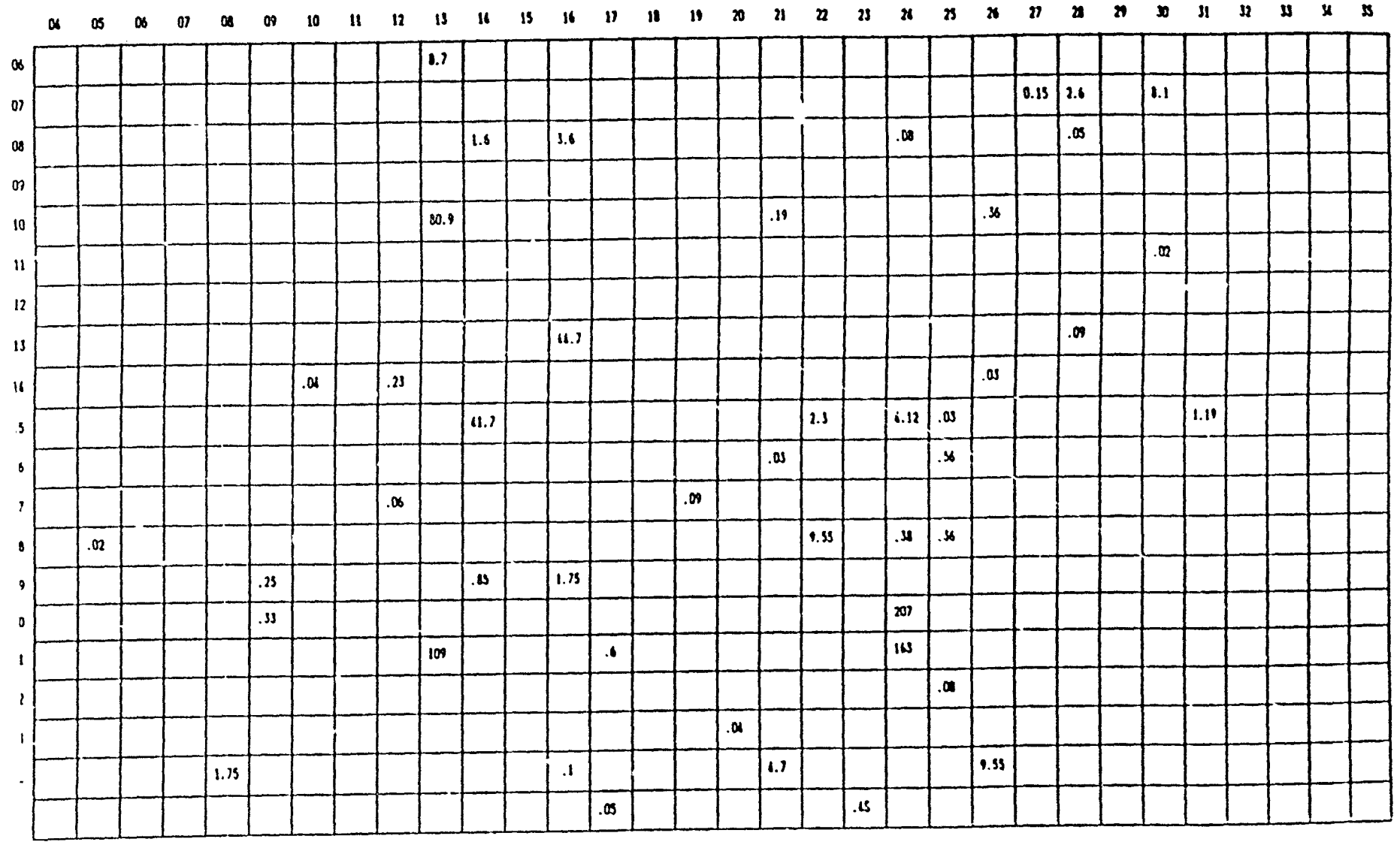


Figure 2.6 3,4-Dichlorophenoxy Acetic Acid Subsurface Soil Concentration (ppb)

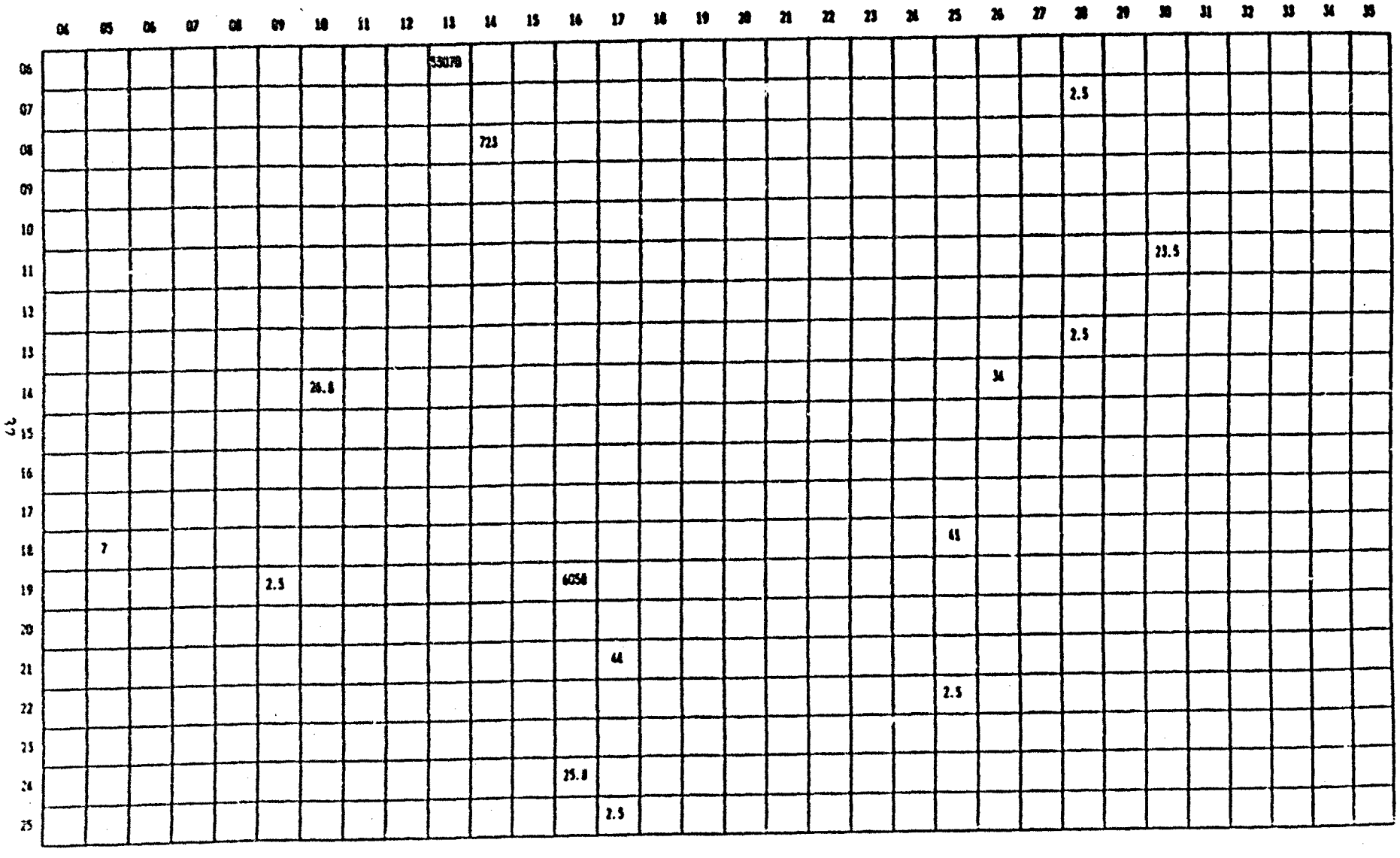
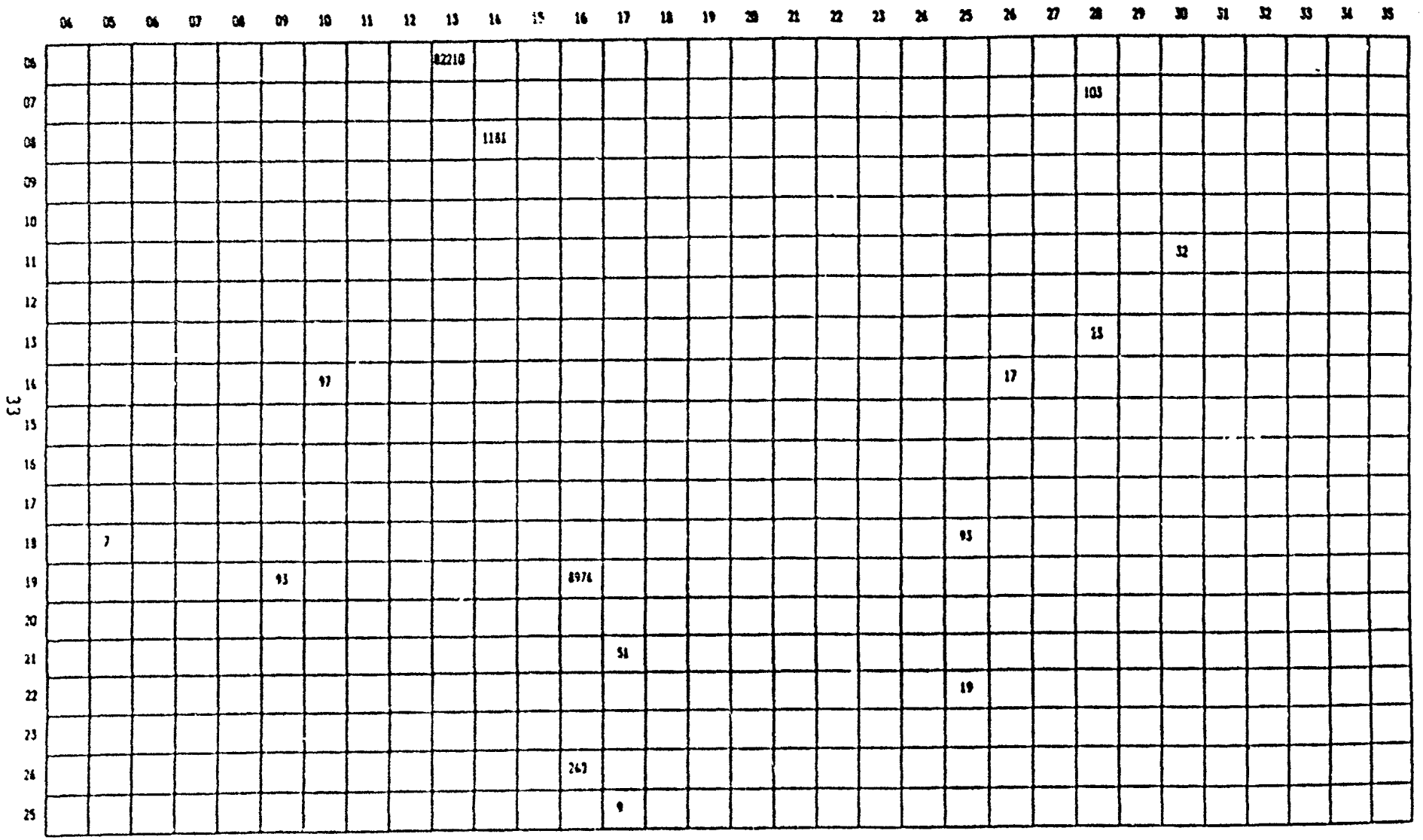


Figure 2.7 2,4,5-Trichlorophenoxy Acetic Acid Subsurface Soil Concentration (ppb)



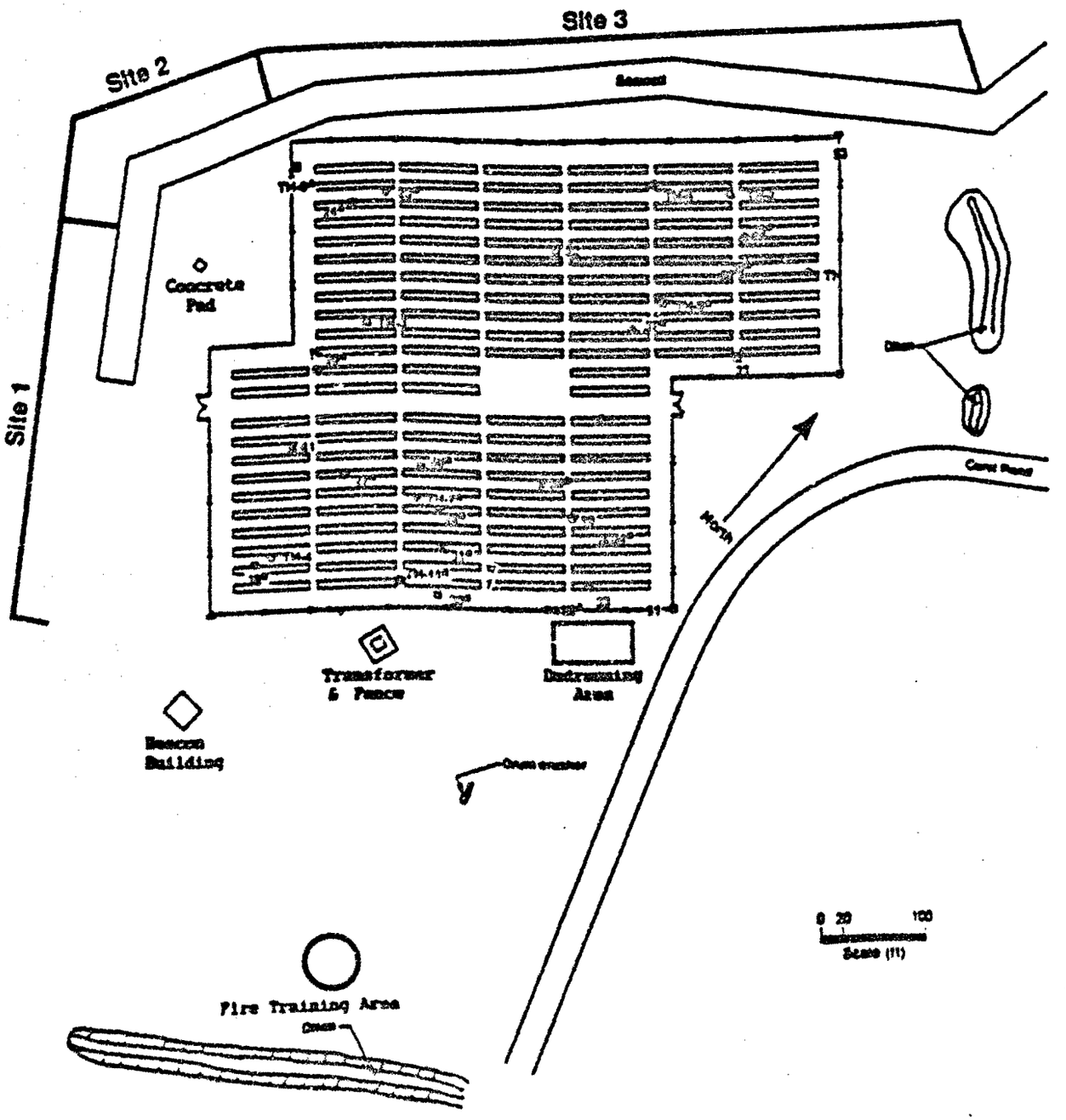


FIGURE 2.8 MARINE BIOTA SAMPLING SITES
Adapted from Crockett AB 1986

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;

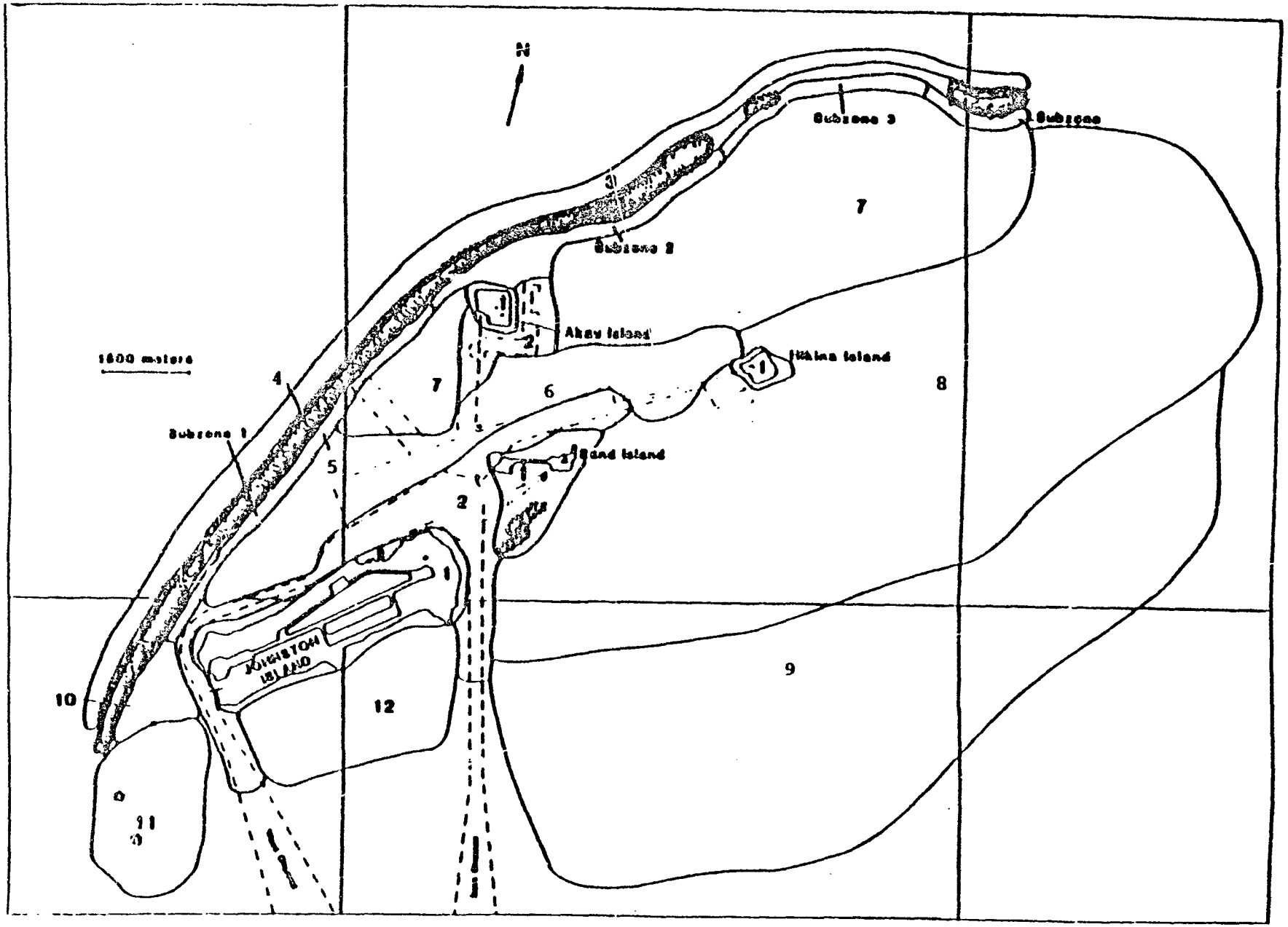


FIGURE 3.1 "ECOTYPE" ZONES (AREAS WITHIN LINE BOUNDARIES) AT JOHNSTON ATOLL From Irons 1990

(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

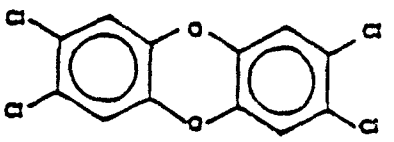
- Migration of contaminated soil particles into the ocean due to water drainage.

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.

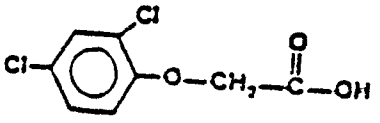
(3) *Sediments.* 2,3,7,8-TCDD is transferred to sediments via leaching from contaminated soil, runoff of contaminated soil particles, and precipitation of resuspended contaminated soil particles and vapor (adsorbed to particles or in rainfall) from the atmosphere into bodies of water. As with soil, microbial 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.

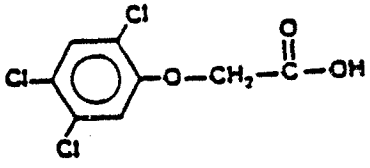


2,4-D

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.