THE HEALTH EFFECTS OF HERBICIDE 2,4,5-T

A Report by the American Council on Science and Health

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Introduction

Consumers today are expressing a continued concern about the state of their environment and its relationship to human health. There is a consensus that human life and personal well being should always be the first consideration in any environmental debate. Yet there is also a growing feeling that decisions on the regulation of environmental chemicals such as food additives, pesticides, herbicides, and other tools of agriculture and food technology should be based on scientific facts. Many believe that unnecessary and expensive product bans should be avoided.

During the 1970s, the media throughout the world gave considerable attention to a particular herbicide, 2,4,5-trichlorophenoxyacetic acid, or 2,4,5-T. Public interest in this chemical was based on two health concerns. First, the safety of its use in forest management was questioned following reports of miscarriages among women living near sprayed areas. Second, 2,4,5-T was a component of Agent Orange, a defoliant used in Vietnam during the period 1962-1970 and alleged to cause serious illnesses among veterans and their children.

A number of public interest groups and veteran's organizations have urged that use of 2,4,5-T be banned in the United States and that veterans exposed to Agent Orange in Vietnam be compensated for their illnesses. The American Council on Science and Health, a consumer education association directed by a panel of scientists, has reviewed the available scientific information on the relationship between 2,4,5-T and human health. This report is a summary of a more detailed literature review.

Because of an absence of scientific data on the health effects of Agent Orange as used in Vietnam, this report addresses only the domestic use of 2,4,5-T in agriculture and forestry. ACSH has approached this issue from the viewpoint of the consumer: if 2,4,5-T is indeed hazardous to health, consumers would benefit if it were banned. On the other hand, if there is no credible scientific evidence of hazard associated with traditional 2,4,5-T use, a ban would be unjustified. Banning 2,4,5-T in the absence of convincing scientific data would increase consumer costs for such products as food, paper, building materials, and other forest products which are dependent on the herbicide for efficient production. At the same time, consumers would receive no additional health benefits in terms of disease prevention despite the increased economic costs.

Position Statement

Based on its review of the scientific evidence, the American Council on Science and Health (ACSH) concludes that there is insufficient evidence to support a ban on 2,4,5-T. No scientific reports presented to date have shown any convincing relationship between the traditional use of 2,4,5-T and adverse health effects in humans. Laboratory evidence of 2,4,5-T's toxicity in animals cannot be reliably used to predict human health risk. However, the laboratory data strongly suggest that 2,4,5-T, like other potentially hazardous chemicals, should continue to be regulated.

The toxicity of 2,4,5-T and its contaminant, 2,3,7,8-TCDD (2,3,7,8-tetrachlorodibenzo-p-dioxin), has been demonstrated under laboratory conditions at doses far higher than those to which humans are exposed. Estimates of human health risk derived from animal experiments are unreliable. Furthermore, those risks which have been calculated for 2,4,5-T and 2,3,7,8-TCDD are extremely small.

ACSH recommends that the current use of 2,4,5-T in rice fields and rangeland be continued and the suspended use in forests, railways and highways, and landscaping be reinstated.

ACSH recognizes the problems posed by 2,4,5-T's unavoidable dioxin contaminant (2,3,7,8-tetrachlorodibenzo-p-dioxin) and urges that every effort be made to further reduce dioxin contamination during the manufacturing process. ACSH recommends that applications of 2,4,5-T, particularly aerial spraying, be strictly monitored to minimize unnecessary environmental and human exposure. ACSH believes that stringent safeguards for manufacture and application of 2,4,5-T will effectively reduce any potential adverse effects of dioxin while allowing for the continued use of this herbicide.

2,4,5-T Chronology of Events

1945 2,4,5-T developed as an herbicide.

1948 2,4,5-T registered for use with the U.S. Department of Agriculture as a pesticide under the provisions of the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).

1949 Industrial accident involving 2,4,5-T at Nitro, West Virginia, manufacturing plant.

1957 2,4,5-T's unavoidable contaminant dioxin (2,3,7,8-tetrachlorodibenzo-p-dioxin) identified.

1962 2,4,5-T first used as a defoliant in Vietnam.

1966 U.S. Department of Agriculture and Food and Drug Administration order 2,4,5-T residue tolerances be established for food products.

1967 Regulatory deadline for obtaining 2,4,5-T tolerances on food and feed products and byproducts.

1969 Reports of birth defects in experimental animals exposed to the herbicides 2,4,5-T and 2,4-D.

1970 U.S. Department of Agriculture requests further studies of 2,4,5-T's potential to cause birth defects.

1970 U.S. Department of Defense cancels use of 2,4,5-T in Vietnam.

1970 U.S. Department of Agriculture cancels approved use of granular 2,4,5-T around residential and recreational sites and use on food crops intended for human consumption.

1970 Congress orders a Department of Defense contract with the National Academy of Sciences to study ecological and physiological effects of herbicide use in Vietnam.

1970 Italy and the Netherlands ban all uses of 2,4,5-T.

1971 Animal death and human illness attributed to dioxin contamination at a St. Louis, Missouri, racecourse; 2,4,5-T not involved.

1971 FIFRA advisory committee recommends continued use of 2,4,5-T on forest, rangeland, rice fields, and rights of way; also recommends that dioxin contamination of 2,4,5-T be reduced to 0.1 parts per million (ppm) and that 2,4,5-T be applied no more than once a year in a manner that will not contaminate humans.

1971 Environmental Protection Agency (EPA) cancels use of 2,4,5-T on most food crops.

1972 Amendments to the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA) mandate registration of all pesticides and their uses as well as EPA certification of pesticide applicators.

1972 Dow Chemical Company, a manufacturer of 2,4,5-T, obtains a court injunction against EPA preventing further regulatory action against 2,4,5-T.

1973 Vietnamese Dr. Ton That Tung publishes a report in the medical journal **Chirurgie** linking U.S. military use of herbicides with liver cancer.

1973 EPA appeal of the court injunotion preventing further regulation of 2,4,5-T is upheld in federal court.

1973 EPA publishes a notice of intent to hold hearings on all food uses of 2,4,5-T following completion of monitoring program for dioxin residues in foods in the parts per trillion (ppt) range.

1974 National Academy of Sciences report on the effects of herbicides in South Vietnam issued concluding there is insufficient evidence of a relationship between military herbicide use and adverse health effects in humans.

1974 EPA withdraws from information hearings because of inability to monitor dioxin residues in food; hearings are suspended.

1974 EPA establishes a Dioxin Implementation Plan to identify analytical methods to detect dioxin residues in the ppt range.

1976 Industrial accident at a chemical manufacturing plant in Seveso, Italy, releases an estimated 1.7 kg (3.7 lb) of dioxin in a densely populated area.

1977 New Zealand government decides to continue use of 2,4,5-T.

1978 United Kingdom Ministries of Agriculture, Fisheries and Food (MAFF) advisory committee concludes that 2,4,5-T-containing herbicides are safe "if used in the recommended way for the recommended purposes."

1978 A U.S. Vietnam veteran forms Agent Orange Victims, International.

1978 EPA publishes notice of Rebuttable Presumption Against Registration (RPAR) of remaining uses of 2,4,5-T based on evidence that 2,4,5-T and dioxin cause cancer, birth defects, and fetal deaths; 2,4,5-T can still be used pending a final agency decision.

1978 ABC-TV 20/20 news magazine program reports on 2,4,5-T and Agent Orange.

1979 Formal hearings on 2,4,5-T suspension begin on April 19, 1979.

1979 2,4,5-T hearings halted on May 15, 1979, after all herbicide registrants withdraw from regulatory proceeding.

1979 NBC San Francisco affiliate KRON-TV airs program on 2,4,5-T, "Politics of Poison."

1979 American Farm Bureau Federation sponsors scientific dispute resolution conference on 2,4,5-T.

1979 House of Representatives subcommittee on Oversight and Investigations holds hearings on involuntary exposure to toxic herbicides and pesticide products.

1979 Publication of the **Pendulum and the Toxic Cloud** by Thomas Whiteside describing Seveso, Italy, dioxin accident.

1979 EPA takes regulatory action to cancel all suspended uses of 2,4,5-T.

1979 Epidemiologic report on miscarriages among women in Alsea, Oregon, (Alsea II) submitted to EPA.

1979 FIFRA scientific advisory panel reviews nonsuspended uses of 2,4,5-T.

1979 EPA issues emergency suspension order to ban 2,4,5-T use on rights of way, pastures, forests, home gardens, aquatic weeds, ditch banks, and ornamental turf; rangeland and rice field uses of 2,4,5-T remain approved.

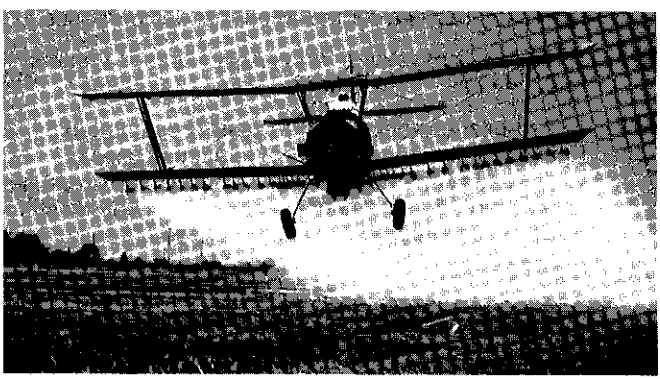
1979 PBS Nova program, "A Plague on Our Children," deals in part with 2,4,5-T.

1979 EPA issues a notice of its intent to hold public hearings on its action to ban all uses of 2,4,5-T.

1980 Dow Chemical Company files suit against the United States claiming government negligence in use of Agent Orange and failure to inform servicemen of potential health risks resulting from dioxin exposure.

1980 Public hearings on 2,4,5-T begin on March 14, 1980.

1980 Epidemiological study of Vietnam veterans, Operation Ranch Hand, under supervision of Department of Defense begins.



The 2,4,5-T Controversy

Since it was first registered for use with the U.S. Department of Agriculture in 1948, the herbicide 2,4,5-T has proven successful in controlling the growth of undesirable plants, especially broadleaf weeds and brush. However, within the past ten years some studies have suggested that 2,4,5-T may cause birth defects and cancer in both humans and laboratory animals.

As a result of these reports, the Environmental Protection Agency (EPA) questioned whether it should allow continued use of this herbicide. In 1978, the EPA began regulatory proceedings which could lead to an eventual ban on all uses of 2,4,5-T. In February 1979, following the release of a controversial epidemiological study in Alsea, Oregon, the EPA

immediately suspended all but two uses of 2,4,5-T. The Agency took this emergency action and bypassed the regulatory proceedings it began in 1978 because the Alsea study found an apparent increase in miscarriages allegedly caused by 2,4,5-T spraying. Many scientists, herbicide users, and trade groups objected to this sudden regulatory action. 2,4,5-T manufacturers also objected to the sudden ban on their product—a ban they believed to rely unjustifiably on an unconfirmed and widely criticized report.

Individuals and organizations supporting the EPA proposal argue that 2,4,5-T causes illness and disease in man and animals in addition to its plant-killing effects. Proponents of the ban cite the results of various animal studies which show that 2,4,5-T is toxic at high dose levels.

Those groups opposed to the ban believe that the EPA's action is not supported by an objective analysis of the entire scientific record.

The central issue in the 2,4,5-T controversy is the chemical popularly known as dioxin, or 2,3,7,8-tetrachlorodibenzo-p-dioxin. Dioxin is an unavoidable contaminant of 2,4,5-T created during the manufacturing process. Dioxin is also a potent toxin in man and animals.

2,4,5-T Chemistry

2,4,5-T (2,4,5-trichlorophenoxyacetic acid) is a white, crystalline solid which dissolves very slowly in water. 2,4,5-T is biodegradable and almost completely decomposes in warm, organic soil in about three months.

2,4,5-T's Dioxin Contaminant

2,3,7,8-tetrachlorodibenzo-p-dioxin is one of a large group of related compounds generally known as dioxins. It was first discovered as an impurity of 2,4,5-T in 1957. Unlike other members of the dioxin group, this specific contaminant is a potent toxin; small amounts of the chemical can cause extensive damage to animal cells. Although in the 1960s some samples of commercially supplied 2,4,5-T are reported to have contained 70 parts per million (ppm) of this dioxin, 2,4,5-T currently sold in the U.S. contains an average of only 20 parts per billion (ppb), or 3000 times less. As used in agricultural and forestry applications, 2,4,5-T is usually mixed with oil, which dilutes its dioxin concentration even further. Thus, health risk estimates based on the earlier dioxin concentrations are likely overstated.

Dioxin is a white crystalline solid which is slightly soluble in most organic solvents and water but which dissolves readily in fat. It can accumulate in the environment under some conditions but is rapidly decomposed by sunlight.

An Historical Perspective

Forty years ago, while studying the properties of naturally occurring plant growth regulators (so-called plant hormones) scientists discovered that phenoxyacetic acid compounds could artificially alter plant growth. Researchers in the United States and Great Britain further determined that some of these phenoxy compounds, especially 2,4-D (2,4-dichlorophenoxyacetic acid), could selectively kill many broadleaf weeds in grain fields, grasslands, and coniferous forests, while leaving these cash crops unharmed. In 1945, the U.S. Department of Defense

also began to study the herbicidal properties of the phenoxy compounds as jungle defoliants.

By 1946, 2,4-D was used extensively to control weeds in grass, grain, and corn fields, but it was ineffective in killing undesirable brush. Researchers then began looking for an herbicide that could solve this problem and found an answer in 2,4,5-T. Since its registration for use in 1948, 2,4,5-T has been used to control broadleaf weeds and brush along highways and railways, in rangeland and forests, and in wheat, rice, corn, and sugarcane fields. It allows for efficient production of foods as well as paper and other forest products.

2,4-D is the most popular phenoxy herbicide used in the United States because of its low cost and effectiveness. 2,4-D is also manufactured by a different process which does not create dioxin as a contaminant. For those shrubs and plants which are resistant to 2,4-D, other herbicides such as 2,4,5-T are substituted.

How 2,4,5-T Works

2,4,5-T and other phenoxy herbicides are sprayed on plants and trees and are absorbed through the leaves, roots, and soft stem tissue. Once inside the plant, phenoxy compounds concentrate in the growing parts of the roots and stems and cause malformations. Plants that are most susceptible to phenoxy herbicides may die within a few days; less sensitive plants may survive for weeks or months while some plants may not be affected at all.

It is not exactly known how natural plant growth regulators or synthetic herbicides like 2,4,5-T actually work. It is known that phenoxy herbicides affect the plant's own growth mechanisms and interfere with cell division, enlargement, and food utilization—processes vital to normal plant growth.

The phenoxy herbicides are far more toxic to plants than to animals and humans. These so-called plant hormones act on plant cell compounds such as cellulose and lignocellulose which are not present in animals.

How 2,4,5-T Is Used

2,4,5-T has a variety of specialized agricultural and forestry uses:

Land Preparation before Planting: 2,4,5-T is used in forests to keep cleared land from sprouting undesired plants before conifer seedlings (pinecone bearing evergreen trees) are planted.

Control of Competing Plants: Because conifers are especially resistant to 2,4,5-T, it is often applied to the ground around seedlings to selectively destroy competing plants which rob seedlings of sunlight, nutrients, and water.

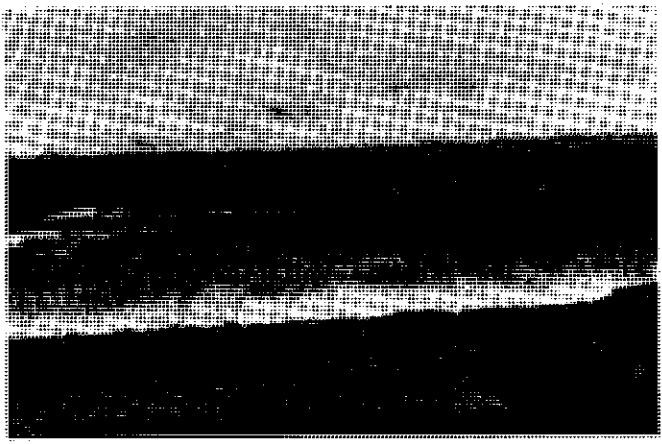
Rangeland Rehabilitation: Annual use of 2,4,5-T on open land will kill sagebrush, chaparral oak, and other hardwoods. In time these plants will be replaced by grasses thus making previously unusable land suitable for grazing livestock.

Fire Prevention: 2,4,5-T is used to create and maintain fuelbreaks in wooded areas to prevent fires from spreading uncontrollably.

Railroads and Highways: 2,4,5-T is used on these rights of way to control weeds and brush which represent potential fire hazards, obscure road signs, and interfere with access to utility lines and equipment.

Landscaping: 2,4,5-T kills dandelions, plantain, chickweed, and other broadleaf weeds in and around lawns, ornamental shrubbery, shade trees, and other recreational areas such as golf courses.

Rice Fields: Apart from rangeland use, the only remaining non-suspended use of 2,4,5-T is controlling weeds in rice fields. In southern states where weeds such as the curly indigo are particularly resistant to other phenoxy herbicides, 2,4,5-T is the only effective method for controlling these weeds.



In some cases, alternatives to 2,4,5-T can be adequately substituted. Mechanical tillage, manual labor, or the use of other phenoxy compounds and other herbicides can be used to control weeds. However, in many applications 2,4,5-T is the most effective weed control method. It is a highly selective herbicide when used properly in agricultural and forestry management and is both inexpensive and efficient.

Animal Studies of 2,4,5-T and Dioxin

Several animal species have been studied to determine the possible health effects of 2,4,5-T. Experiments have been conducted with pure 2,4,5-T and dioxin as well as with the commercial herbicide which contains the dioxin contaminant. Monkeys, sheep, dogs, rabbits, guinea pigs, hamsters, rats, and mice have all been tested to study the effects of these chemicals on cells, tissues, organs, and whole animals.

The results of these studies have been as varied as the species of animals tested. Many experimental and physiological factors affect the way an animal responds to a test chemical. Animals react differently to 2,4,5-T when it is fed rather than applied directly to the skin. Administering 2,4,5-T directly through a stomach tube will also produce different responses than when it is mixed with food.

Physiological and biochemical differences among animal species also produce differing test results. Dogs, for example, are far more sensitive to the effects of 2,4,5-T and dioxin than mice. However, even within a single species, different strains or breeds vary in their response to 2,4,5-T and dioxin.

Experimental conditions, particularly the length of time and the amount of 2,4,5-T and dioxin to which an animal is exposed will profoundly affect its response to both chemicals. At sufficiently high dose levels, both 2,4,5-T and dioxin are lethal; however, the exact levels vary among species. Intermediate dose levels will cause observable physiological changes and illness. Yet there are also low dose levels for 2,4,5-T and dioxin which produce no observable effects in any animals tested.

Although animal testing is widely used for many scientific studies, there are difficult problems in applying animal test results to human beings. Many animal cells, organs, and body systems are similar to those of the human body, yet there are also important differences in anatomy and biochemistry between animals and humans. While animal testing is an invaluable method for determining potential health risks for humans, it cannot provide an exact model of our human environment and experience. The differences in response to 2,4,5-T and dioxin among animal species further complicate the accurate prediction of human health risks from these chemicals. Animal test results may be used in addition to valid human evidence but cannot act as a perfect substitute for it.

The many animal studies of pure 2,4,5-T without dioxin have shown that at high doses it will cause muscle weakness, weight loss, and tissue changes. In contrast, animals exposed to large doses of pure dioxin will develop damage to the thymus gland, lymphoid tissue, and certain blood forming tissues such as the bone marrow.

Both dioxin and 2,4,5-T can cause reproductive problems if administered in large doses to animals at critical times during gestation. Spontaneous abortions will occur, and there is an increase in risk of cleft palate and kidney defects in liveborn offspring. However, animal studies have also demonstrated that there are presumably "safe" levels of exposure to 2,4,5-T and dioxin which produce no observable changes in newborn animals or increase the rate of spontaneous abortion.

Another type of test using animal cells cultured in the laboratory is used to study the probability that 2,4,5-T and dioxin will produce harmful changes in genetic material. The results of these so-called short term mutagenicity studies can then be used to evaluate possible cancer causing activity.

Results of all 2,4,5-T mutagenicity studies have been either negative or inconclusive for primary DNA (deoxyribonucleic acid) damage, gene mutations, and chromosome abnormalities. While the inconclusive test results cannot be interpreted as showing that there is no harmful effect, there is a general agreement that 2,4,5-T does not cause observable mutagenic changes in cultured cells. On the other hand, test results from several studies have shown that dioxin exposure does increase the frequency of gene mutations.

Perhaps the biggest health question is "Do 2,4,5-T and dioxin cause cancer?" A number of animal studies using mice and

rats have addressed this question but have failed to produce any clear evidence that 2,4,5-T causes cancer.

Studies of pure 2,4,5-T and 2,4,5-T contaminated with dioxin have not conclusively shown that 2,4,5-T causes cancer. A 1980 report by an EPA advisory group found suggestive evidence of cancer at high dose levels. However, this preliminary conclusion must be confirmed by additional tests and interpreted in light of actual human exposure levels.

Rat studies have been used to determine if dioxin causes cancer in animals. In these tests, a special strain of rat that is particularly sensitive to cancer causing chemicals was used. Results showed that dioxin, when given in relatively large doses, did increase the frequency of lung and liver tumors in these animals. However, there is a difference of opinion among scientists about how these results should be interpreted.

Some argue that dioxin may not be a true cancer causing chemical, or cancer initiator. Instead, they interpret the test evidence to suggest that dioxin accelerates, or promotes, the carcinogenic action of other chemicals. Other scientists argue that dioxin is a cancer initiator rather than a promoter.

This controversy is further complicated by the lack of observable tumors in studies where dioxin was fed to animals at low dose levels. This may mean that the tumors observed at higher doses are actually due to physiological changes caused by dioxin poisoning or that the tests themselves could not detect a small increase in tumors at low dioxin levels. Additional research is needed to clearly resolve the question of dioxin's cancer causing potential.

Human Exposure to 2,4,5-T and Dioxin

There is very little evidence on the long-term adverse effects of 2,4,5-T and dioxin in man because most human exposure to these chemicals has been accidental rather than experimental. However, from the evidence that has been gathered, no conclusive relationship has been established between 2,4,5-T or dioxin and human cancer, spontaneous abortion, or birth defects. The only human illness so far proven to occur from

exposure to these chemicals is chloracne, a severe acne-like skin disorder. This condition is caused solely by dioxin and not by 2,4,5-T.

Agent Orange

From 1962 to 1970, U.S. military forces sprayed herbicides in Vietnam to defoliate jungle areas. One of the defoliants widely used in Vietnam was Agent Orange (named for the color of its container) which contained an equal mixture of 2,4-D and 2,4,5-T. Use of Agent Orange was discontinued in 1970 after the potency of 2,4,5-T's dioxin contaminant was discovered.

Several years after the final spraying of Agent Orange, a number of Vietnam veterans exposed to it during the war filed medical claims with the Veterans Administration (VA). They argued that Agent Orange caused serious illnesses such as cancer in themselves and birth defects in their children. Some veterans have alleged that Agent Orange also caused their psychiatric and neurological disorders.

At present, these veterans are seeking compensation for their illnesses from the VA. However, because of the controversy over the scientific evidence concerning 2,4,5-T and dioxin toxicity, the VA is providing compensation only for the skin disorder chloracne.

No adequate studies of the relationship between Agent Orange and human health have yet been attempted. During the next few years, the U.S. Department of Defense will conduct a large scale epidemiological study of Vietnam veterans exposed to Agent Orange. When completed, this study will likely present a clearer picture of any adverse health effects caused by Agent Orange.

Seveso, Italy

One of the most publicized chemical accidents in recent years occurred on July 10, 1976, in Seveso, Italy. Following the explosion of a chemical reaction chamber at the ICMESA chemical company, a section of the densely populated Seveso community was contaminated by an estimated 1 to 4 pounds of dioxin. In this case, the dioxin was produced during the manufacture of hexachlorophene, an antibacterial agent.

Plants, birds, rabbits, and chickens died soon after the accident. In addition, children and adults exposed to the chemical dust complained of nausea, nervous symptoms, and chloracne-like skin disorders accompanied by redness and swelling. Following these reports, the local authorities evacuated some 5000 persons from the contaminated area.

An extensive health surveillance system was put into effect to record the middle- and long-term effects of dioxin exposure on the population. Medical examinations and laboratory tests were performed, pregnant women were closely monitored to record miscarriages and birth defects, and a cancer registry was created to track any new cases of cancer among exposed individuals.

A thorough analysis of the health data gathered from the Seveso population has been completed and published. The results of the study showed that Seveso residents developed chloracne and minor, temporary nerve damage. No other

organs or body functions were found to be affected. There was no increase in the number of miscarriages, birth defects, or infant deaths that could be linked to dioxin. However, the study period was not long enough to uncover any cases of cancer related to the accident.

Alsea, Oregon

The epidemiological study referred to as "Alsea I" is based on the experiences of a small number of women in the Alsea region of Oregon. In 1977, these women, who lived near a forest area sprayed with 2,4,5-T, became concerned about the number of miscarriages they had experienced. They believed that their miscarriages were related to the annual spraying of 2,4,5-T near their homes. At their request, the Environmental Protection Agency (EPA) began a preliminary epidemiological study.

Initially 10 miscarriages were reported by eight women. The final total, however, included 13 miscarriages experienced by nine women. Twelve of these miscarriages occurred within the first 20 weeks of pregnancy.

The EPA investigation concluded that there was a seasonal variation in the miscarriage rate among the Alsea women compared with a control group from the nearby city of Corvallis but that there was insufficient evidence to prove a relationship between the miscarriages and 2,4,5-T spraying.

The so-called "Alsea II" study was a more detailed continuation of the earlier epidemiological investigation. Using hospital records, EPA scientists collected information on miscarriages from three study areas: the Alsea region along the Oregon coast; the city of Corvallis immediately inland from Alsea; and a sparsely populated rural area near the Idaho border. Both the Corvallis and rural areas were to serve as controls for the Alsea region since 2,4,5-T was not used in these areas during the study period from 1972 through 1977.

Unlike the Alsea I report, this second study suggested that there was a relationship between the use of 2,4,5-T and an increase in miscarriages. It concluded, "The Agency's systematic survey of the occurrence of spontaneous abortions in an area of 2,4,5-T use indicates that there was an unusually high number of spontaneous abortions in the area, and that the incidence of spontaneous abortions may be related to 2,4,5-T in that area."

Based on the findings of this report, the EPA immediately issued an emergency suspension of all remaining uses of 2,4,5-T except for rangeland clearing and rice field weed control.

After the Alsea II report was made public, however, a number of scientists examined the study and challenged the EPA's conclusions. More than 18 reviews of this study have uncovered numerous flaws in the study design, statistical analysis, and interpretation of its results.

Based on this widespread criticism, the Alsea II study is not regarded by the scientific community or the EPA as a valid indicator of a relationship between 2,4,5-T and miscarriages.

2,4,5-T Regulation

The course of 2,4,5-T regulation over the years has been a

stormy one. Acting under the provisions of the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA), the EPA has gradually restricted 2,4,5-T use.

In 1971, the EPA banned the use of 2,4,5-T on most food crops. In 1974, the Agency first held public hearings on a proposal to ban all food uses of the herbicide but discontinued the hearings because of its inability to accurately detect dioxin residues in food.

In 1978, the EPA instituted a Rebuttable Presumption Against Registration (RPAR). In this regulatory proceeding, the Agency began formal action to ban all uses of 2,4,5-T. However, during the time the hearings are in process, 2,4,5-T may still be used in forestry and agriculture.

After the release of the Alsea II report, however, the EPA immediately banned most uses of 2,4,5-T. Under the FIFRA provisions, the EPA may bypass the normal regulatory channels in emergency situations such as the Agency presumed to exist in Oregon.

On March 14, 1980, hearings began in Washington to decide whether to permanently ban all uses of 2,4,5-T. Because of the complexity of the issues and the involvement of manufacturers, herbicide users, consumer groups, the EPA, and the U.S. Department of Agriculture, a long and expensive legal fight is expected. Until this proceeding is settled, all uses of 2,4,5-T with the exception of rangeland and rice field applications will continue to be banned.

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