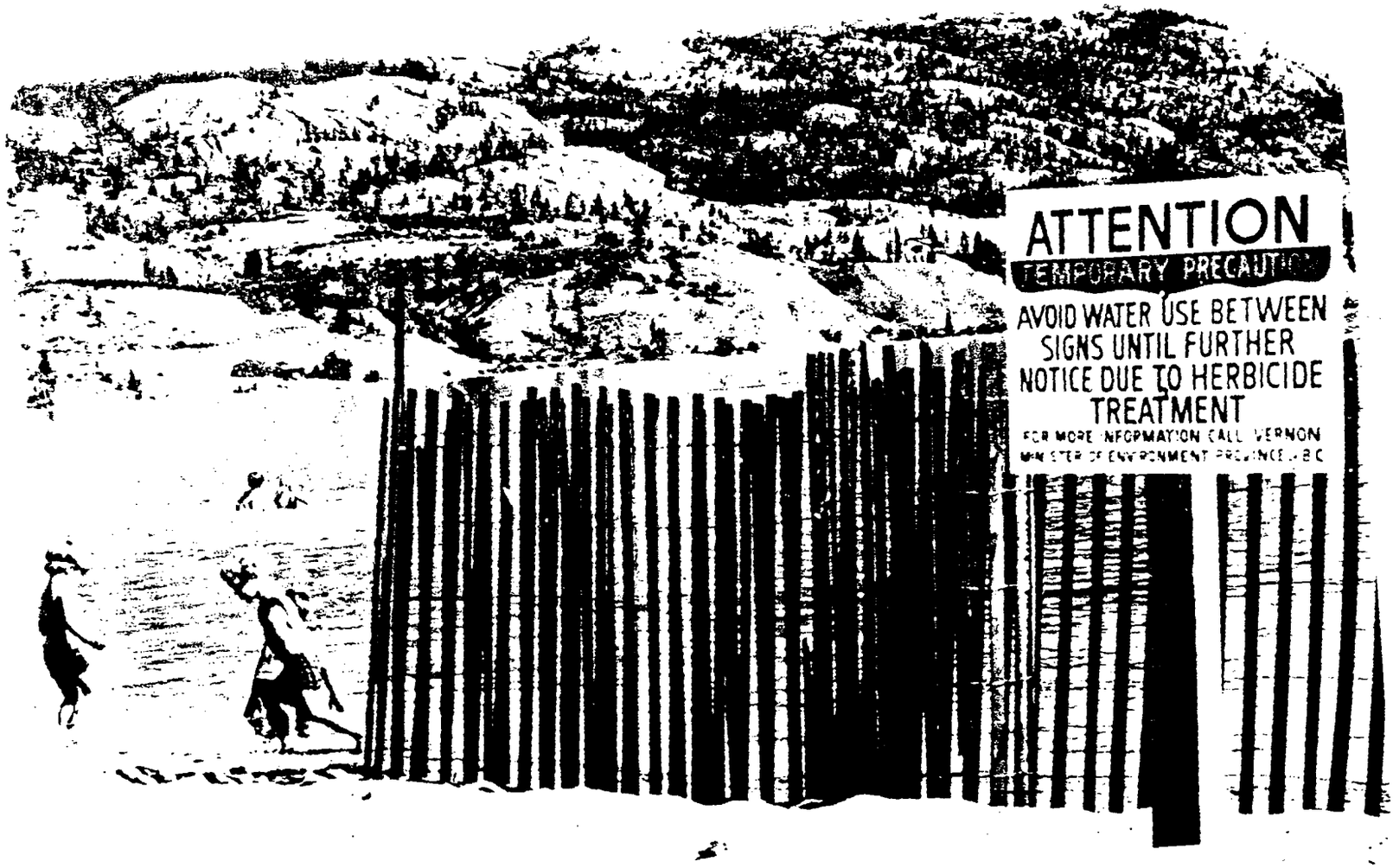


THE OTHER FACE OF 2,4D

A Citizens' Report

SOUTH OKANAGAN
ENVIRONMENTAL COALITION

First Printing - January, 1978

Second Printing - July, 1978

FRONT COVER: Children swimming in the restricted area of Skaha Lake Beach, Penticton, B.C., on June 27th, 1977. Four days earlier this area had been treated with 20 lbs. to 40 lbs. of 2,4-D active ingredient per acre.

TITLE PAGE: Water Investigations Branch team applies herbicide in shallow water on Skaha Lake Beach on June 23rd, 1977. Eight days later, thousands of vacationers used this beach during the July 1st holiday weekend.

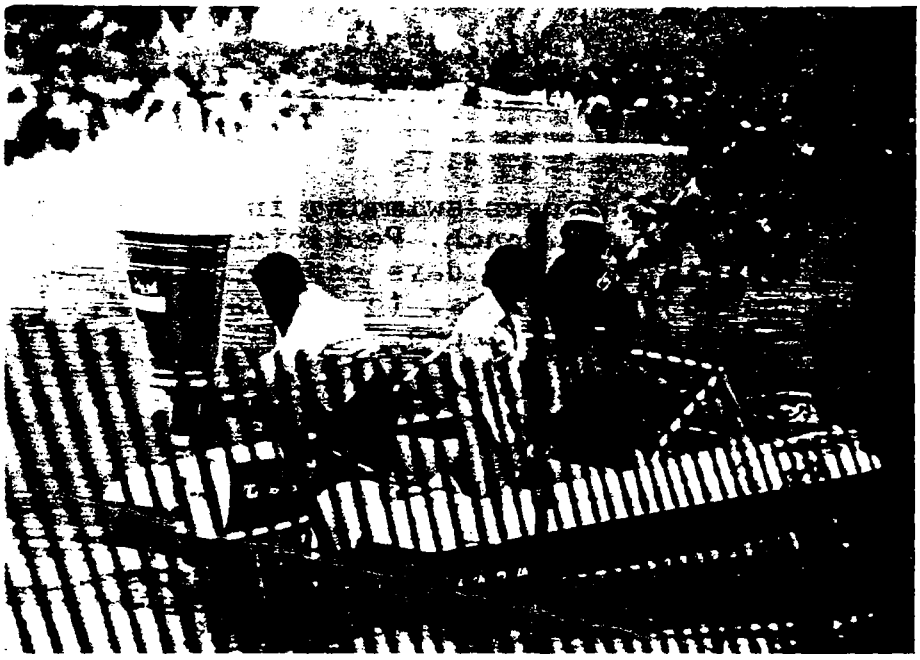
Penticton Commercial Printing Ltd.

THE OTHER FACE OF 2,4D

a citizens' report

JOHN W. WARNOCK

JAY LEWIS



SOUTH OKANAGAN ENVIRONMENTAL COALITION

PENTICTON, BRITISH COLUMBIA

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PREFACE

Genesis

One year ago the South Okanagan Coalition did not exist. Very few people in the Valley knew anything about the phenoxy herbicide 2,4-D. It all began in May of 1976 with the arrival of the B.C. Government's Advisory Committee on the Control of Eurasian Water Milfoil. Public hearings were held, questions asked, and the answers on the safety of 2,4-D were not reassuring enough for many people. The S.O.E.C. was formed and the research began. This report is the result of a seven month literature search and a large amount of primary research. While we believe it to be the first effort of this type on 2,4-D ever published, we realize that we have only scratched the surface. Additional information arrives daily and the picture becomes clearer bit by bit. It is a beginning.

How To Read It

Because of the length and the complexity of this report, some instructions may be helpful. For a quick overview of the situation, read the introduction, the chapter summaries and the conclusion. A more in-depth understanding can be obtained in any particular area by reading that chapter in its entirety. All sources are completely listed in the bibliography and most are available in our files. Please do not attempt to read this report in one sitting - brain damage may result.

Bias, Fairness and Research

We have often been asked whether this report would be "unbiased." We went into this project because we were worried about the conflicting reports which we had read about the health aspects of 2,4-D.

We were suspicious of the phenoxy herbicides because of the widespread reports of the ill effects on humans that emerged during the latter part of the war in Vietnam.

When the Provincial Government appointed the Advisory Committee, they selected three professors from the University of British Columbia who had previously served as the B.C. Royal Commission Inquiry into the Use of Pesticides and Herbicides. In the Final Report of the Royal Commission, and elsewhere, these three individuals clearly established that they believed that there was no damage to public health or the environment from the traditional use of phenoxy herbicides. Could a report by such a group be "unbiased"?

The Ministry of the Environment specifically instructed the three man Advisory Committee to examine "all available data" on the public health problems associated with the aquatic use of 2,4-D. The three Interim Reports total only 44 pages, have only 28 pages devoted to the use of the chemical, and have no footnotes or bibliography. Even at first glance, the end result appears deficient.

As the title suggests, "The Other Face of 2,4-D" is an attempt to look beyond the findings of the Advisory Committee. In fact, we feel that the B.C. Royal Commission did not adequately deal with the evidence it had on the possible dangers to public health from the widespread use of phenoxy herbicides. In addition, much new data has become available since May 1975.

We are publishing only that research which we feel will stand up to rigorous scientific scrutiny. In that sense we feel the report is fair. For the public, it fills in the gap left by the B.C. Royal Commission and the three Interim Reports of the Advisory Committee.

Acknowledgements

So many people helped make this report possible it is difficult to know where to start. Tina Kramer, Clive & Melissa Johnson, Ruel Smith, Don Rees, Peggy and Walt Taylor, Marge Murton, Jim Cluett, Katy Madsen and Margaret Ingram of the Coalition's Research and Information Committees helped with the massive job of collecting and organizing the massive amount of material.

Draft copies of the report were circulated to six outside readers with expertise in various fields, for their comments. Because five of them are employed by the B.C. Government or the B.C. University system, we will not mention them by name. Their suggestions were an immense help to us in producing the final draft. Of course, we take full responsibility for all the material in this report.

A large number of environmental groups from across the U.S. and Canada forwarded material to us for use in this study. First and foremost on our list of contributors are Sid Horton, Bill Boyd and John Hillian of the Kelowna Society for Pollution and Environmental Control (SPEC). They produced the first brief in the Okanagan Valley which pointed to the health hazards associated with 2,4-D. Much of our research data were collected by this group. Other organizations and individuals supplying information were: Linda Pim, Pollution Probe, Toronto; Michael Singleton, Federation of Ontario Naturalists, Toronto; Maureen Hinkle and Norma Watson, U.S. Environmental Defense Fund, Washington, D.C. and New York; Lynn Pederson, Citizens Against Toxic Sprays (CATS), Eugene; Donna Waters, Minnesota Herbicide Coalition, Chaska, Minn.; Michael Mann, Federation of American Scientists, Washington, D.C.; David Fry, Center for Science in the Public Interest, Washington, D.C.; Richard Semiklose,

American Association for the Advancement of Science, Washington, D.C.; Committee For Environmental Information, St. Louis; Mark Luppino, Citizens For a Better Environment, Chicago; Peter Messina, Sierra Club, San Francisco; Robert Nixon, Sierra Club, Victoria; and The National Audubon Society, New York.

Several other organizations were helpful in supplying information: Environmental Canada; Cancer Control Agency of B.C.; Canadian Cancer Society; The U.S. Environmental Protection Agency and most importantly, the South Okanagan Health Unit.

Publication Support

The South Okanagan Environmental Coalition would like to thank the British Columbia Government Employees Union, Area Council #07 - Okanagan, for a substantial grant which helped make reproduction of this report possible.

Production

We would also like to thank Loretta Scott for typing our final draft. Jack Davis designed and produced the Coalition logo and was responsible for the cover artwork. Penticton Commercial Printing got it all through the press. Melody Hessing proof read the final copy and corrected our errors. All the staff at Apex Office Supplies helped with the detail work.

Dedication

It is appropriate that this report should be dedicated to Merriam Doucet of Port Moody, B.C. Without her detailed knowledge of the field, extensive files and delightful sense of humour, this document would not have been possible.

Her speaking tour through the Okanagan in the spring of 1977 provided local residents with the basic information on the hazards associated with 2,4-D and provided the encouragement for the formation of the South Okanagan Environmental Coalition. As long as Merriam is around, people cannot be careless with pesticides and expect to escape unscathed.

Jay Lewis
John W. Warnock

Penticton, B.C.
January 18, 1978

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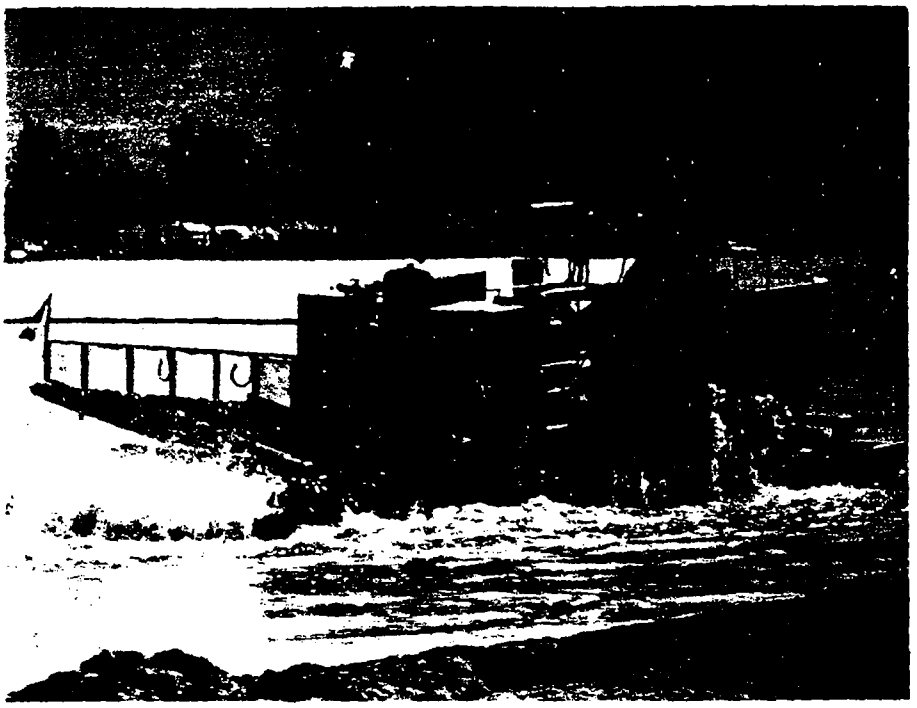
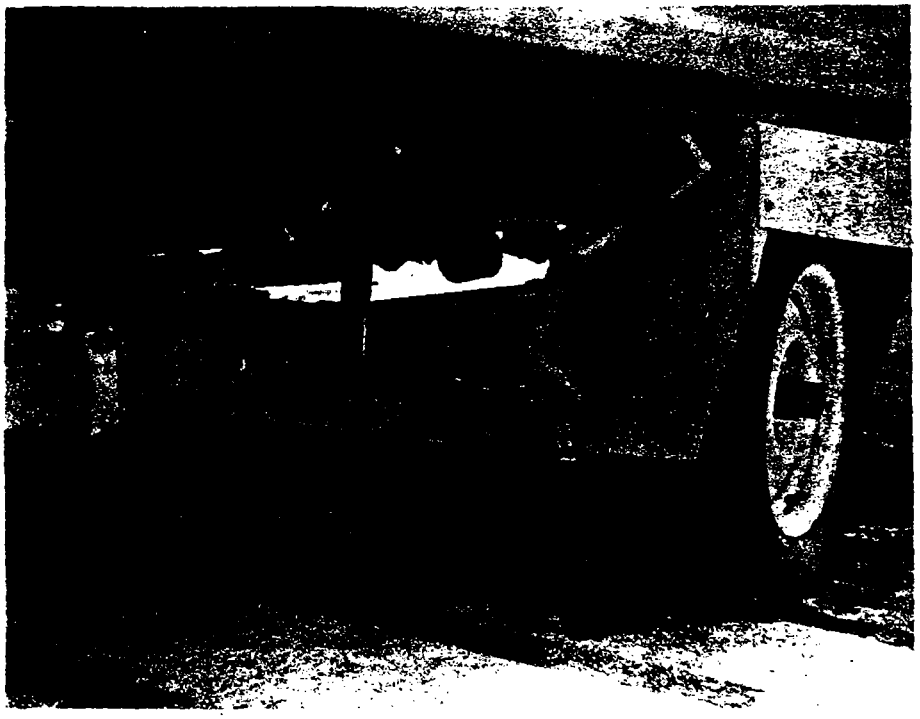


One of the weed eradication machines, designed and built by the Water Investigations Branch, here used in Skaha Lake Beach. While this mechanism was very slow, new designs will soon be available which should speed work.



How Eurasian Water Milfoil spreads. This boat and trailer were on their way to Calgary via Shuswap Lake. The owner was not interested in removing the weed.

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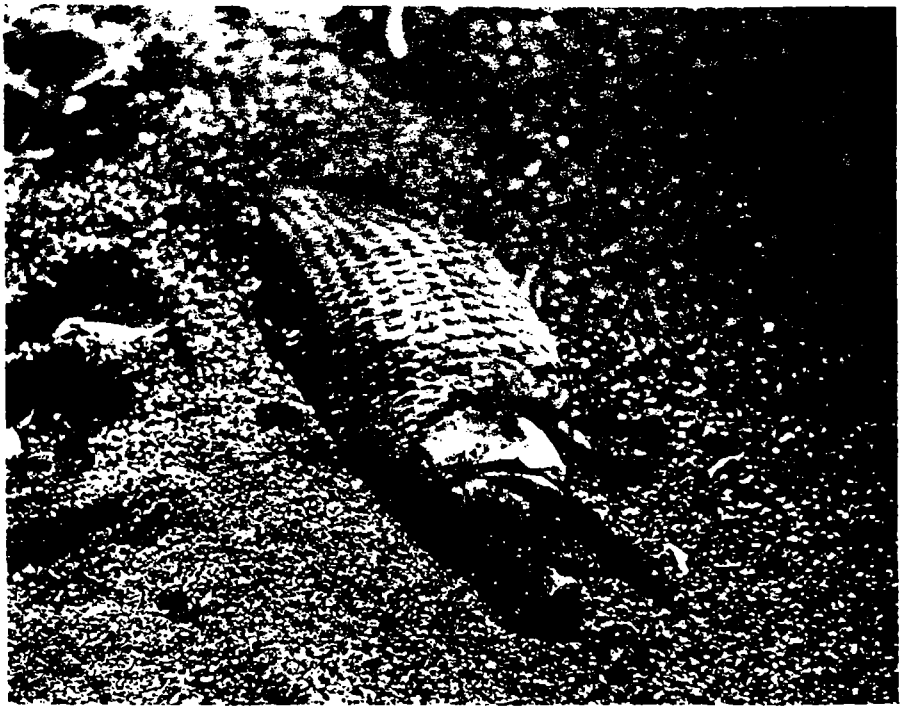
2,4-D monitoring programme by the B.C. Water Investigations Branch. Note the brown tinted sample bottles sitting in the bright Okanagan sunshine. 2,4-D degrades more rapidly in warm water.



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One of the dead carp which showed up on Skaha Lake Beach five days after 2,4-D application. A total of eight were seen. Carp and Suckers taken in the area had high concentrations of the herbicide in the stomach and flesh.

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I

INTRODUCTION

Eurasian water milfoil (Myriophyllum spicatum L.) is spreading through the Okanagan Basin lake system. This is a perennial water plant that has spread throughout Europe and the United States, and is now moving into Western Canada. It has colonized all the lakes in the southern part of Ontario, Quebec as well as upper New York state. (Holm et al, 1969)

During the summer months there is rapid growth from the Milfoil root crown area which reaches up to the surface. It requires sunlight for growth, and in the Okanagan lakes is rarely seen in depths of over 15 feet. If the weed spreads throughout the entire Okanagan lake system, it could conceivably cover 8,000 acres or 10% of the lake surface. (B.C. Department of Environment, 1976)

The weed reproduces from its seeds, which are known to be spread by waterbirds, particularly coots. But the primary method of reproduction is by fragmentation. Parts of the plant break off, float for a period of time, lose their bouyancy, and sink to the bottom. At this point they root and form a new colony. Authorities from the Tennessee Valley Authority report that "a single 2-inch fragment may take root and grow 4 feet or more in length during a single growing season. Calculations indicate that a single fragment theoretically could propagate almost 250 million new fragments in one year." (T.V.A., 1972) It is known to quickly establish where there are high levels of nutrients, as in silt beds and downstream from sewage effluent outfalls. (Holm et al, 1969)

Eradication programmes have been attempted in many areas of the world, but because of the manner in which the milfoil spreads, none have succeeded.

In eastern Canada, government officials looked at the record of past attempts and concluded that it was not worth the cost to try to eradicate the weed.

Alternate Uses of the Milfoil Weed

Eurasian milfoil weed has become a serious problem in the Kawartha lakes in Southern Ontario. Herbicide treatments were tried by some individuals, but the rotting weeds caused equally serious problems, and many felt the game population was threatened. The provincial government turned instead to harvesting the weeds.

The main research project has been carried out by a team of scientists from Ontario Agricultural College at Guelph. Harvesting of the weeds was chosen for it gets the plant out of the water, eliminates the depletion of the oxygen level of the lake areas, and cuts back dangerous algae production. They do not remove all the weeds, leaving beds to protect the local game fish.

At first, the harvested weeds were distributed on farmers fields with manure spreaders. While the weeds contain nutrients and are an excellent soil conditioner, many farmers believed the process took too much time. To easily manage the weeds, it was necessary to remove much of the water.

The second experiment was to press out a "water weed" pellet. This was a success, and the end-product was palatable to both poultry and sheep. However, the costs of production were too high to make it competetive with existing feeds.

The third approach was to utilize the weed as a roughage in cattle feed.

D.H.S.D

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Experiments produced a silage that when top dressed with corn and soy meal was quite successful for finishing cattle. In test animals, the growth record and the beef were very good.

The fourth experiment was conducted by the Horticulture Department at Guelph. The weeds were used as an ingredient in compost, using modern manufacturing equipment to remove the water. The end result was a very good compost; there is a good Ontario market for packaged material for potting and commercial use. ("Nutrition", 1977)

The Herbicide Alternative: The Experience of the TVA

There have been several areas in North America where there has been an attempt to control Eurasian water milfoil weed by chemical means, but the most thorough experiment has been in the Tennessee Valley Authority, a man-made lake system running through six states from Virginia to Kentucky.

Milfoil was first identified in the T.V.A. system in 1960; by the end of 1968, there were more than 25,000 acres of the weed in seven reservoirs. In 1962, the officials of the T.V.A. began large scale applications of 2,4-D in two forms: the butoxyethanol ester (BEE) in granular form and the dimethylamine salt (DMA) in liquid form. Between 1962 and 1969 over 1.4 million pounds of 2,4-D were applied to 35,000 acres of weeds in the reservoir system.

How successful has the programme been? The history of the programme in one lake, as reported in the Tennessee Valley Perspective (Summer 1976), gives us a good indication of what we can expect if a similar programme is introduced into the Okanagan lakes.

In 1969 Guntersville Lake in northeastern Alabama contained around 14,000 acres of milfoil weed. By applying 2,4-D at rates of between 20 and 40 lbs. of active ingredient per acre, the infested acreage was reduced to 3,000 by 1973. Even though the herbicide was continued every year, often over the same beds, by 1976 the milfoil acreage had increased to around 12,000 acres. Control at these levels required herbicide applications every year.

In the overall valley, applications of 2,4-D reduced the infested acreage to around 5,000 by 1973. But in 1976, T.V.A. authorities reported that the total acreage had grown to 16,000 acres in spite of annual applications. (TVA Perspective, Summer 1976)

Officials in the TVA have never believed that eradication was possible. In their Environmental Statement, they state that "the objective of the watermilfoil control program will be to maintain at least the present level of control." (T. V.A., 1972)

There are obvious reasons why eradication is impossible. First, the weed spreads too easily through fragmentation and movement of seeds. Secondly, the herbicides have not been completely successful in killing all of the roots of the plant. Under the best conditions in spring, the T.V.A. reports that their kill rate was only 95%. During the remainder of the year, kills averaged between 55% and 70%. (T.V.A., 1972)

The Drawbacks to the T.V.A. Programme

The experience of the T.V.A. should indicate some of the problems we might experience if a similar programme were to be introduced into the Okanagan.

First, when the weeds are killed by herbicides but not removed from the lake, the rotting weeds not only become a nusiance, but the increase in nutrient release makes further infestation easier. (Moody, 1973; Sculthorpe, 1967)

Secondly, there is the problem of the increase in the slimy dark green weed, filamentous algae. This more dangerous weed is taking over embayments where the herbicide has been applied. This was also a major problem where the herbicide was used in Ontario. In the late 1960's, the outbreak of blue-green algae in the beach area of Skaha Lake at Penticton became a serious health problem, causing illness to residents and tourists. The South Okanagan Health Unit believes that the heavy growth of Milfoil weeds in Skaha Lake near Penticton, virtually eliminated the algae blooms in this area. (B.C. Board of Health, 1976; Brooker and Edward, 1975)

The third problem has been the rising costs of herbicide applications and monitoring. The costs of application in the T.V.A. alone rose from $37 an acre in 1973 to over $100 in 1976. In that year, they abandoned the use of Aqua-Kleen 20, as it costs more than the liquid form. This is spread on the weed beds through the use of a heliocopter. (TVA Perspective, 1976; Bates, 1977)

Finally, there is the concern over the buildup of the herbicide in the lake system. While there is no irrigation in the TVA system, there are many centers which depend on the lakes for their domestic water. During application periods, levels of 2,4-D in the water have exceeded the .1 ppm level (maximum concentration set by the U.S. Government) in a number of the major water purification plants. (See Section VIII) Regular ingestion of low levels of the herbicide may well produce chronic illnesses. These are yet to be recorded, but we do not know of any programme to detect such problems in the T.V.A.

Experiments in the Okanagan Lakes

Research on the control of Eurasian water milfoil in the Okanagan Lake system began in 1972. Tests of the effectiveness of paraquat, diquat and 2,4-D were conducted in experimental plots in 1974, 1975 & 1976. In 1976, the provincial government agreed to a 50-50 cost sharing agreement with the Okanagan Basin Water Board. This was superceded by the agreement of May 5, 1977 whereby the provincial government agreed to finance, design and implement a weed control programme in co-operation with local officials. (Newroth, 1976)

The year 1977 was chosen for major tests of the use of 2,4-D and mechanical harvesting methods in four Okanagan lakes: Kalamalka, Okanagan, Skaha and Osoyoos. The Canadian Wildlife Service excluded Vaseux Lake from the treatments as it is a major waterfowl range. Dr. Peter Newroth, a biologist with the Environmental Studies Division, B.C. Ministry of the Environment, was placed in charge of the programme. In his memorandum on the 1977 programme, Dr. Newroth argued that "it will be impossible to remove the nuisance in one or two years." He supported the five-year programme advanced by the Advisory Committee. Nevertheless, he argued that in 1977 "eradication of Myriophyllum spicatum would be attempted in Kalamalka and Osoyoos lakes." (Newroth, 1977)

In a previous memorandum on application, Dr. Newroth suggested that it would be "inappropriate" to use 2,4-D in three areas: (a) shallow areas frequented by waterfowl; (b) areas immediately adjacent to domestic, industrial or irrigation water intakes; and (c) areas which would be spawning areas for game fish. (Newroth, 1976)

The Reports of the Advisory Committee

On October 18, 1976, the Government of British Columbia appointed a three-man Advisory Committee on the Control of Eurasian Water Milfoil in the Okanagan Lake system. All three members were professors at the University of British Columbia; they were the same three who had served as the members of the Royal Commission of Inquiry into the Use of Pesticides and Herbicides between 1973 and 1975. The Chairman, Dr. C.J.G. MacKenzie of the U.B.C. College of Medicine, also serves as Chairman of the Pollution Control Board, as well as an advisor to the federal government on the question of arsenic poisoning at Yellowknife, N.W.T.

Dr. W.K. Oldham, a civil engineer, was also known as an outside consultant. Between 1971 and 1974 he served on the Joint Federal-Provincial Okanagan Study, and between 1972-1975 on the Kalamalka-Wood Lake Study.

The Third member, Dr. W.D. Powrie, Head of the Department of Food Science, had also been involved as a consultant in the establishment of the government-financed Swan Valley Foods.

The Minister of the Environment instructed the Advisory Committee "to review all available data including information collected by the Royal Commission hearings and to advise him on the programme and any public health problems associated with the use of 2,4-D for the control of aquatic weeds in the Okanagan Lakes."

The first Interim Report of this committee, issued three months later, called for control methods to be utilized in 1977 "with the possible exception of the use of herbicides."

They recommended a "carefully conceived programme" for testing 2,4-D hopefully to fill in for the insufficient data" on the "overall scale of use, application, concentration and exclusion areas." (B.C. Advisory Committee, January 1977)

However, by the time the Second Interim Report was issued in March 1977 the Advisory Committee was arguing that "an all-out attack on the weeds should be launched in the 1977 season." A five-year integrated programme of mechanical, hydraulic, biological and chemical control was outlined. (B.C. Advisory Committee, March 1977)

Around this time widespread concern was being expressed throughout the Okanagan over the use of 2,4-D in the lakes. By the middle of the summer, opposition had been expressed by over 20 groups, ranging from boards of irrigation districts, many trade unions, to the B.C. Medical Association and the Penticton and Kelowna Registered Nurses Association. Only three organizations, all connected with the tourist industry, had passed resolutions supporting its use. This widespread concern was reflected in the public meetings held by the Advisory Committee in the Okanagan in the spring of 1977.

The Advisory Committee made no attempt to alleviate this concern in its Third Interim Report, issued on May 20, 1977. The final report of the Advisory Committee was only four pages long, and contained no new material on 2,4-D. Residents of the Okanagan were simply assured that "water entering intakes in the Okanagan Valley will assuredly contain no detectable 2,4-D three days after the 2,4-D application, if the programme is carried out according to recommendations." They admitted that "the people of the Okanagan are entitled to assurances that there will be no danger to humans or crops from any herbicides used in the lake (sic)." They were sure that this can be given "if zero tolerance is achieved in all cases." (B.C. Advisory Committee, May 1977)

The 1977 Programme

In order to approach the "zero tolerance level", the Advisory Committee recommended that no applications of herbicides should be used in swimming areas, and that no herbicides should be applied within 500 metres of any potable water intake systems or any irrigation intakes. (B.C. Advisory Committee, March 1977) Both of these recommendations were ignored by the provincial government.

The commitment of key officials in the B.C. Department of the Environment to the use of 2,4-D was reflected in the policy set down for the 1977 year of control. All communities which wished provincial government financial support for control of the weed were required to accept a total package which included chemical as well as mechanical control. When the City of Kelowna requested funds for mechanical control, but said that they did not want herbicides used, they were refused provincial assistance. Kelowna officials and others accused the provincial government of engaging in a form of blackmail to force use of 2,4-D where it was not wanted.

During the summer of 1977, test applications of 2,4-D were applied in four areas: one in the North Arm of Okanagan Lake, one at Nasuhito Creek on Okanagan Lake, one at Westside Cays in the Okanagan Lake, and at the Skaha Lake beach at Penticton. Application at the Penticton beach created large public protests. An additional application was scheduled for an area near Kaleden on Skaha Lake, but strong opposition from local residents, plus preparations for civil disobedience, caused the provincial government to abandon this test. While it was at first planned to use the herbicide in Kalamalka Lake and Osoyoos Lake, mechanical methods were used instead. In total, less than 23.5 acres were treated with herbicides.

The cost of mechanical control of herbicides was estimated at $800 an acre by Karl Marsden, public relations director for the Okanagan programme in 1977. He claimed that the application of herbicides cost only $100 an acre. However, monitoring costs were much higher. Close to 5,000 samples were taken in the 1977 monitoring programme. In July, Mr. Marsden estimated that this aspect of the programme would cost between $200,000 and $500,000. (Penticton Herald, July 21, 1977). If the minimum monitoring programme is continued, this would bring the cost of herbicide use at least up to the level of mechanical harvesting. Gordon Jennens, who is building a mechanical harvester, claims he will be able to harvest the weeds for between $200 and$300 an acre, which would be far less than the cost of applying herbicides.

The 1978 "All Out Attack"

The Advisory Committee and the B.C. Ministry of the Environment have both announced that there will be an "all-out attack" on the Eurasian water milfoil weed in 1978, including widespread use of 2,4-D. It has been estimated that there are now between 1,500 and 2,000 acres of the weed in the Okanagan Basin lake system. This means that we could conceiveably expect between 30,000 and 80,000 lbs. of 2,4-D active ingredient in the Okanagan lakes in 1978.

The first problem for the provincial government is how to come up with the finances for such a programme. They will either have to hire more employees or contract out the application. What Okanagan residents fear is that the monitoring programme will be abandoned. That is how the costs were lowered in the Tennessee Valley Authority area. We are certain that no one will want the monitoring programme abandonned.

The second problem concerns the monitoring programme itself. There is only one laboratory in British Columbia capable of doing the laboratory testing. And even here, the gas chromatography, mass spectrometry, computer analysis system used in 1977 is inadequate to test for organic pollutants in water supplies. It can only test down to .001 parts per million. In 1977, by August 10 only 500 of 3000 samples had been processed. In December, not all of the samples had been tested.

If public health officials insist that the monitoring in 1978 be at least as complete as it was in 1977, this means that most of the lake areas that are treated will probably be closed for the entire summer. The laboratory will simply not be able to handle the testing, and public health officials will not dare open recreation areas without first knowing the level of 2,4-D residues in the water and silt. Otherwise, the provincial government will have to abandon the "zero tolerance level" promised by the Advisory Committee.

THE PHENOXY HERBICIDES

The herbicide 2,4-D is one of the chlorinated phenoxy compounds developed at the U.S. Center for Chemical and Biological Warfare at Fort Dietrich, Maryland, during World War II. The three most commonly used members of this herbicide group are 2,4-D, 2,4,5-T and 2,4,5-TP (often called Silvex in the United States and Fenoprop in British Columbia).

All of this family of herbicides are derived from benzene and synthesized from the chlorophenols. All share the benzene ring molecular structure, the main difference being the number and placement of the chlorine atoms. The only difference in the molecular structure between 2,4-D and 2,4,5-T is one additional chlorine atom in the latter. (See figure I)

The only difference between 2,4,5-T and 2,4,5-TP is that the former is manufactured with acetic acid, the latter with picolinic acid. (Ashton and Crafts, 1973)

There are various practical formulations of 2,4-D. Sometimes they are converted to water-soluble amines; at other times, they are in the form of oil-soluble esters. The acid formulation is used to kill weeds like bindweed, Canada thistle and Russian knapweed.

The 2,4-D esters are identified by the name of the alcohol used in the manufacturing process. In 1977, the provincial government used Aqua-Kleen 20, manufactured by the American chemical company, Amchem. This is the butoxyethyl ester formulation of 2,4-D. The esters are generally insoluable in water, but they are considered the most toxic to plants. (Klingman, 1975)

The Mode of Action

All of the phenoxy herbicides initiate the same response when applied to plants: they act like an auxin, a plant hormone. Normal growth ceases within hours. Epinastic bending of the plant occurs within minutes. Over days there is the formation of tumours, secondary roots, and fasciated structure is pronounced.

Meristematic cells cease to divide. Elongated cells stop length growth, but continue radial expansion. In mature plants, parenchyma cells swell and begin to divide. Young leaves stop expanding and develop excessive vascular tissue. The roots lose their ability to absorb water, photosynthesis is inhibited, and the phloem becomes plugged.

It is now recognized that the primary action takes place in the plant cells themselves. 2,4-D is accumulated and retained in the cells. Here, the phenoxy herbicides all disrupt the normal function of the cell nucleus. They increase the RNA, DNA, and protein. RNA and protein synthesis are controlled by DNA, and the phenoxy herbicides disrupt the normal role of DNA. It is also known that the phenoxy herbicides act on the cell membrane, with cell wall loosening. (Ashton and Crafts, 1973)

All of the phenoxy herbicides, including 2,4-D, promote uncontrolled expansion and division of cells. This form of action prompted Professor J. Van Overbeek to state that the phenoxy herbicides "kill like cancer." (Van Overbeek, 1964)

Since the late 1940's, the phenoxy herbicides have been widely used in North America for control of broad leaf plants, brush and trees. 2.4-D is widely used in many agricultural areas. Fenoprop (2,4,5-TP) is used as a growth promoter in some agricultural areas. 2,4,5-T has been banned from general use and use on agriculture since 1971, the result of research by the U.S. government and the experience of its use as a defoliant in Vietnam. But it is widely used in British Columbia in forest management, range and pasture management, spraying along power lines and along rights-of-way for roads and railroads. Since 1971, two formulations of 2,4-D have been registered for use in the United States for control of aquatic weeds; in Canada, Acqua-Kleen 20 is registered for such use under certain conditions and restrictions.

While 2,4-D is quite effective when used on plants, it has a rather low lethal toxicity rating for humans and other animals. The lethal dose of 2,4-D for a human is around 6 grams, or slightly more than a teaspoon of the pure chemical. For this reason, it has been widely used on food crops, as it was believed that the herbicide was relatively harmless to humans.

While there has been considerable research done on 2,4-D, it is only in recent years (since around 1971) that the evidence of its acute and chronic effects on humans has been demonstrated and, more significantly, received widespread publicity.

The Legal Implications of the use of 2,4-D in Okanagan Lakes

It is widely known that herbicides are not supposed to be used in water, or even sprayed near bodies of water or streams. The B.C. Royal Commission of Inquiry into the Use of Pesticides and Herbicides (1975) recommended that 2,4-D not be used any closer than 300 feet to any water source. There are good reasons for these restrictions: fear of possible harmful effects to fish, animals (including humans) and the environment in general.

Therefore, there is an obvious contradiction between the traditional restrictions against the use of phenoxy herbicides in any water environment, and the registration of one or two formulations of 2,4-D for actual use in water. To date, the Control Products Section, Federal Production and Marketing Branch, Department of Agriculture have not responded to our letter requesting them to explain this situation.

In the United States, Aqua-Kleen 20, manufactured by Amchem, has been registered by the U.S. Environmental Protection Agency for use in the control of Eurasian water milfoil, at dosages up to 100 lbs. per acre (40 lbs. active ingredient of 2,4-D). Applicators are warned not to apply the herbicide "within 1/2 mile of potable water intakes." The only other warning is that "fish and other aquatic organisms may be killed at application rates recommended on this label." (EPA Reg. No. 264-109, June 12, 1976)

Aqua-Kleen is also registered for use in Canada, but the restrictions are more significant. The Control Products Section of the federal Department of Agriculture (which registers pesticides in Canada) limits Aqua-Kleen and 15 other aquatic herbicides as follows: "do not apply to waters used for irrigation, agricultural sprays, watering dairy animals, or domestic water supplies." (B.C. South Okanagan Health Unit, 1976)

These restrictions became a public issue in the summer of 1977 when the South Okanagan Environmental Coalition, in co-operation with the West Coast Environmental Law Association of Vancouver, began exploring legal actions to block the use of 2,4-D in the Okanagan lakes system. It was later learned that following consultation with the Control Products Section of Agriculture Canada, the B.C. Ministry of the Environment conducted its chemical program under a clause in the Pest Control Products Act which allows experimental applications. The size of the program was hardly of an experimental nature.

Survey by the B.C. Royal Commission

The legal implications of the use of herbicides was subject to a study done by James M. MacKenzie for the B.C. Royal Commission of Inquiry into the Use of Pesticides and Herbicides (1975). The Final Report mentions some of the federal and provincial laws and regulations which may apply to the use of 2,4-D in the Okanagan lakes.

The most important federal legislation is the Pest Control Products Act. Section 44 prohibits use of any control product (like 2,4-D) in a manner inconsistent with the directions shown on the label. Obviously, the provincial government is violating the restrictions on the use of Aqua-Kleen as imposed by the agency of control, the Control Products Section of the Department of Agriculture. Under Section 5 of the Act, exemptions to normal regulations can be granted by the Governor General in Council.

In reality, this is an action by the federal Cabinet.

Other federal legislation that is involved is the Fisheries Act of 1970. Under section 33 (2) there is a prohibition of depositing any "deleterious substances" in water frequented by fish. This is a "strict liability" section; no criminal intent needs to be established, and there are fines to be layed up to $5000.

Under this act, a "deliterious substance" is defined as "any substance that, if added to any water, would degrade or alter or form part of a process of degradation or alteration of the quality of that water so that it is rendered deleterious to fish or to the use by man or fish that frequent that water." In the past, the courts have held that most pesticides fall into this category.

The third federal law which would seem to apply in this case is the Migratory Birds Act of 1970. Regulations issued in support of this act prohibit anyone from "depositing substances harmful to migratory birds into waters or any area frequented by migratory birds" (sec. 35). As previously stated, the Canadian Wildlife Service has denied permission for the application of 2,4-D in Vaseux Lake, a migratory waterfowl sanctuary.

There is also provincial legislation which directly applies to the use of herbicides in the Okanagan lake system. The Health Act (1960) in section 43 prohibits "the pollution of water courses from which water or ice is taken for domestic purposes." Section 66 of this act prohibits the deposit of waste matter in any stream or lake so as to pollute its waters. It specifically mentions "any poisonous, noxious, or polluting liquid." The Act (Sec. 114) and the implementing regulations (Sec. 71) provide penalties for violations.

It should be noted that there are over 400 domestic water licenses issued for Okanagan Lake alone, plus another 200 unlicensed intakes. Skaha Lake has over 80 licensed intakes. (B.C. South Okanagan Health Unit, 1976)

The report by James M. MacKenzie argues that other provincial legislation is applicable to large scale use of pesticides, including the Pharmacy Act, the Litter Act, the Pollution Act, the Water Act and the Wildlife Act. It would seem that the provincial Ministry of the Environment is violating several federal and provincial acts when it adds herbicides to the Okanagan basin lake system.

The Possibility of Suits for Damages

Across North America there is a growing movement by people to initiate legal suits for damages against companies and governments responsible for pollution and direct damage to individuals and their property. In 1969, the Government of Nova Scotia accidentally sprayed Robin Warren's farm with phenoxy herbicides, resulting in destruction of his crops and deaths to his farm animals. After investigating, the Government of Nova Scotia paid the Warren family $10,000 in damages. (Ogden, 1971)

The people in Globe Arizona who were sprayed by phenoxy herbicides during a range management programme have a $4 million damage suit waiting for court action. Dow Chemical Company and the U.S. Forest Service have tried to postpone this case, fearing it will set a precedent. (Doucet, 1977)

In New Brunswick, Abram Friesen now has a case before the courts claiming damages for illness to his family caused by the provincial government's spraying of fenitrothian as part of the spruce budworm control programme. The spraying of fenitrothian has been linked to Reye Syndrome, which has resulted in the deaths of several children. (Globe and Mail, November 24, 1977)

Another important case involves workers in Ontario who have contracted lung cancer while working in uranium mines and refineries. The United Steelworkers of America, who are pushing the case, have documented over 90 deaths from lung cancer at Elliot Lake and Bancroft. Once the Ontario Workmen's Compensation Board establishes that the industry caused the lung cancer, then the companies involved become subject to legal action for damages. (Globe and Mail, November 24, 1977)

A landmark case is involved in the widely reported case of poisoning by polybrominated biphenyl (PBBs) in Michigan in 1973. This chemical, used as a fire retardant, was accidentally mixed with cattle feed, causing widespread pollution and illnesses among people very similar to those caused by phenoxy herbicides. Like many of the chlorinated phenols, PBBs persist in the environment for a long time. There are 84 damage cases now in court, and if a few of them are successful, they will bankrupt the Michigan Chemical Corporation. (Stadfeld, 1976)

Establishing Safe Levels for Pesticides

Those who are following the 2,4-D/Okanagan Lakes controversy in British Columbia are aware that government officials repeatedly claim that if the water we drink contains less than .1 ppm of 2,4-D, then this is safe and acceptable. How are these levels reached?

First, most of these are at best guestimates, based on established levels for acute toxicity in laboratory animals and sometimes in humans themselves. It is impossible to determine threshold levels for chronic toxicity, as individual resistance levels vary so widely.

For example, prior to 1975 the Plant Products Division of Agriculture Canada required that all commercial formulations of 2,4,5-T contain less than .5 ppm of tetra-dioxin (TCDD). In 1975, they changed this to require a maximum level of .1 ppm TCDD. To a large extent, this could be seen as simply conforming to standards set in the United States. But there was no scientific basis for making the determination. It was just a bureaucratic adjustment, most likely due to the increased publicity of the toxic and chronic effects of TCDD. (Agriculture Canada, 1974)

How such a decision is made is revealed in the case of PBB pollution in Michigan in 1973. At the time, little was known of the toxic levels of PBBs. The first maximum level for PBBs in milk was established at 2.5 ppm; this was the level previously established for polychlorinated biphenols (PCBs). But some tests indicated that PBBs are five times as toxic as PCBs. The U.S. Food and Drug Administration then concluded that they should allow only one-tenth as much as PBBs in milk as PCBs. Thus, they reduced the acceptable level to .3 ppm. The other rationale for setting this level was that .3 ppm could be tested with reasonable accuracy with the average gas chromatograph.

Curtis K. Stadfeld, who investigated the PBB poisoning case, concluded that "the miserable truth about the action levels is that they are purely arbitrary. They are supported only by sophisticated guessing." (Stadfeld, 1976)

The same can be said for the .1 ppm "safe level" set for 2,4-D in drinking water. It is at best a guestimate. It is not based on scientific knowledge of chronic illness, established by testing, experimentation and epidimological surveys. Just because this guestimate has been accepted for the sake of expediency by the World Health Organization/Food and Agriculture Organization Committee on Pesticides is no guarantee of safety from chronic illness.

Summary

Eurasian milfoil weed is spreading throughout the Okanagan lake system and all across North America. Because of the manner by which it spreads, it is impossible to eradicate the weed through the use of herbicides.

Any use of 2,4-D is just "chemical mowing." If the provincial government decides to use the herbicide for this purpose, then it will have to be used far into the future. Otherwise, there is no justification for its use in the first place.

The B.C. Advisory Committee has recommended a five year "all out attack" on milfoil weed including the use of 2,4-D. They recommend that a "zero tolerance level" be maintained with regard to water intakes for irrigation and domestic use. This is simply impossible. There is also the fear that with the rising costs of the herbicide, its application, and monitoring, that the minimum monitoring programme that was used in 1977 will be abandonned.

The phenoxy herbicides, including 2,4-D, kill plants by disrupting the normal pattern of cell growth. There is very little difference between the chemical basis, molecular structure and mode of action of the different phenoxy herbicides.

The use of Aqua-Kleen in the Okanagan basin lakes is in clear violation of the labelling restrictions on use of the herbicide as set by the Control Products Sections, Agriculture Canada. It also violates the federal Fisheries Act and the Migratory Birds Act, as well as the provincial Health Act.

If the provincial government continues to use 2,4-D in the Okanagan basin lake system, it runs the risk of legal actions for damages from injured parties. Such cases are multiplying across North America.

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> "If this is happening, our data collecting system may not be sensitive enough to detect their presence in the population. British Columbia has one of the most advanced registry systems for defects of this type in North America. It is possible, nevertheless, that a very small number of malformed children in the Province have been damaged by herbicides, but their number is so small that they are not distinguishable from the number of damaged children born each year where the cause of their infirmity is not explainable (Royal Commission, 1975).

What we are left with in most cases then, are raw field reports of health damage in certain areas. These are often the best we have as early warning systems after laboratory testing. To wait for proper epidemiology studies may result in delays which would allow toxic chemicals to go unchecked for far too long.

Admittedly, it is very difficult to produce an air tight causal relationship between a chemical and health damage in normal living situations, much less during periods of turmoil such as war or an industrial accident such as in Seveso, Italy. Control groups are difficult to establish and there are an almost unlimited number of possible causal factors. However, to disregard field reports would result in a substantial amount of unnecessary damage to the environment and thus human health.

Thalidomide was identified as a powerful teratogen through field reports rather than lab tests. The drug would probably have been removed from the market sooner had scientists been more willing to heed the warnings being observed by doctors in the field.

The Vietnam Case

Vietnam was the first country to experience the use of herbicides as a weapon of war.

During this period, 2,4-D became widely used as a substitute for 2,4,5-T.

Determining the effects on humans from field use of 2,4-D is often complicated by the fact that "T" and "D" are often used together for defoliation and brush removal. When health problems result, the damage is always blamed on 2,4,5-T and TCDD. The Swedish studies mentioned earlier indicate that both chemicals are teratogenic and may synergize to become even more potent (Bage, et al., 1972).

To date, 2,4,5-T has received a lions share of the attention, while 2,4-D was largely ignored. Laboratory testing shows quite clearly that both chemicals pose serious health problems. 2,4-D has traditionally been the most widely used herbicide and there are consequently tremendous economic forces at work to keep it on the market. This situation may well be responsible for the lack of interest by regulatory agencies under pressure from users and producers in placing restrictions on 2,4-D similar to its sister compound, 2,4,5-T.

Field Reports - Epidemiology

Scientists are loath to give much credence to field reports of damage to health through environmental contamination. Until these reports are accumulated and statistically analyzed into epidemiological data, they do not seem to have much scientific appeal to many. In most countries epidemiological (relating to the incidence, distribution and control of disease) data are simply not collected. Even in the "advanced" nations the science is still a very crude tool.

For instance the B.C. Royal Commission on Pesticides and Herbicides speculates that herbicide induced defects may be occurring now.

Why then is it still being used today with virtually no restrictions outside of a few cursory label warnings?

As with all research that suggests that a product in current use may be a health hazard, many users and producers immediately attacked the testing procedures. One common approach is to say that research on mice and rats has no relationship to human beings. To this, Dr. Theodor Sterling at Simon Fraser University replies that "the mouse is one species that heralds teratogenic effects in man" (Sterling, 1976). Not only will the effects of a chemical often be similar in man, but they may be many times more potent. In the case of thalidomide, Dr. Samuel Epstein reminds us that "humans are 60 times more sensitive to thalidomide than mice, 100 times more sensitive than rats, 200 times more sensitive than dogs and 700 times more sensitive than hamsters." (Epstein, 1970) It is now well established by the authorities in the field that testing with laboratory animals is appropriate to the human experience. Criticism of high dosage levels is treated in the chapter on carcinogenicity.

Perhaps the main reason that 2,4-D is still unrestricted in use is the existance of 2,4,5-T and its contaminant TCDD. When Dow Chemical first produced their study showing the extreme teratogenic potential of TCDD, all attention turned toward 2,4,5-T (Sparschu, et al., 1970). This shift of focus was not warranted as the test results show 2,4-D to be just as damaging as 2,4,5-T. As a result of the studies done on TCDD an effort was made by the U.S. Environmental Protection Agency to have 2,4,5-T removed from use on crops. This further directed attention toward this chemical during the three years of political and administrative manoeuvering which ultimately resulted in restrictions (Epstein, 1972).

Finally, the NCTR division of the U.S. Department of Health, Education and Welfare checked the purified samples of 2,4,5-T and found them to be teratogenic. This latest finding finally puts to rest the notion that the dioxin contaminents are responsible for the teratogenic effects of the phenoxy herbicides. The study also points toward a number of testing deficiencies which should be corrected in the future. These involve: inadequate numbers of test animals; inadequate or nonexistent replications of tests; inadequate testing at doses below 100 mg/kg; and the need for testing in different strains and stocks of mice. (HEW, NCTR, 1975).

The Testing Game

We have found that scientific world is riddled with accusations and back-biting over testing procedures and the interpretation of results. A research programme can be set up which will most likely show a desired result simply through the manipulation of various parameters of the test. There is probably no research effort which is free from bias and fault and cannot be criticized. In fact, criticism by those inside and outside a profession is the chief method of maintaining high quality work. All studies will be picked apart by those on one side or the other of a discussion. That research which stands up the best to such analysis and is continually supported by additional data will probably become generally accepted. Whether any use is made of the material is another matter.

As early as 1968 it was obvious to everyone paying close attention to the research being done that there was reason for concern about the safety of 2,4-D.

Recent Studies

In 1972, the Laboratory of Teratology of the Karolinska Institute in Stockholm, Sweden produced the results of their study on 2,4-D and 2,4,5-T. Unfortunately, in this research, 2,4-D was not tested separately, but in combination with 2,4, 5-T at a ratio of 2:1 (Hormoslyr 64).

However, the Swedish government had concluded that Hormoslyr 64 had caused cancer in applicators working for the national railway. At the high doses (110 mg/kg), fetal mortality was significantly increased and fetal weight considerable reduced. Also noted were: increased rates of cleft palate; rib and vertebral malformations; subcutaneous and renal hemorrhages; and various spontaneous malformations such as exencephaly (brain outside skull); hydrocephaly (abnormal skull enlargement); open eye; small eyes; and ectrodactyly (absence or fusion of toes or fingers).

Of particular interest was the observation that "the teratogenic effects of the two substances (2,4-D & 2,4,5-T) in combination at 110 mg/kg it seems, however, as if the 2,4-D contributes more than would be expected from a purely additive effect." They go on to urge that further studies be undertaken to look into this possible synergistic effect (Bage, et al., 1972).

Most recently, the U.S. Environmental Protection Agency (EPA) has been retesting 2,4-D and 2,4,5-T. These results reaffirmed that the phenoxy herbicides produced cleft palate, depressed fetal weight and increased fetal mortality. Of particular concern for humans who may receive low doses through contamination of water or air, was the finding that "the greatest effects of 2,4-D were produced at the low doses administered over long time periods" (Courtney, 1975).

They then go on to define teratogenesis as "that degree of embryotoxicity which seriously interferes with normal development or survival of the offspring" (Schwetz, et al., 1971). Based on this system, those effects which all other researchers and authorities in the field call teratogenic are now embryotoxic (Epstein, 1970; Royal Commission, 1975).

The study can also be criticized on other grounds. Because the dosages used were low as compared with other studies, particularly the 12.5 mg/kg level, larger test animal populations should have been used for effective statistical analysis. It is also said that rats are more resistant to chemicals than mice and do not present as good a reading as the latter (Sterling, 1974).

Finally, the Dow researchers suggest that the Bionetics studies may have been faulty as the DMSO vehicle used was thought to be a teratogen itself. This criticism doesn't seem to stand up as the DMSO treated control animals in those tests did not show a statistically significant higher rate of abnormalities than the untreated controls. (Royal Commission, 1975). It can also be noted that the doses administered orally in honey also produced defects. The U.S. National Institute of Environmental Health Sciences research looked into this aspect as well and concluded that "the administration of DMSO or honey to mice or rats did not adversely affect the development of the fetuses." (Courtney, et al., 1970)

It is clear from the results of the Dow study, that 2,4-D caused significant defects similar to the other major research efforts. In this case, however, the observed abnormalities have been defined as embryotoxic rather than teratogenic.

Dow Study

The most extensive testing programmes by industry have been conducted by the Chemical Biology Research division of the Dow Chemical Company in Midland, Michigan. In 1971, they produced the results of their study of the effects of various formulations of 2,4-D on Sprague-Dawley rats. 2,4-D acid and its propylene glycol butyl ester (PGBE) and isooctyl (IO) esters were administered orally to the test animals in a corn oil suspension on day 6 to 15 of pregnancy. The dosage levels ranged from 12.5 mg/kg to 87.5 mg/kg. The relatively low upper dose level was selected after it was found that 100 mg/kg was lethal or produced acute toxicity symptoms in their test animals. It is difficult to understand why Dow's test animals are so sensitive to the chemical when other lab's rats apparently tolerate more than twice this dosage without apparent toxic effects.

At the higher dosages, subcutaneous oedema (abnormal accumulation of fluid beneath the skin), delayed ossification of the sternebrae (incomplete breastbone formation), wavy ribs, lumbar ribs (misplaced), delayed ossification of skull bones, and missing sternebrae were statistically significant with some formulations. Hydrocephalus (abnormal swelling of the skull) and dilated urinary bladder were produced extensively in a random litter (Schwetz, et al., 1971)

The report concludes that "no treatment-related teratogenic responses were observed in this study." Now, how could this be, given the list of statistically significant, observed terata (birth defects) mentioned above?

First, they indicate that "embryotoxicity is the toxic effect on an embryo caused by treating pregnant females during that period in which actual tissue differentiation and organogenesis (formation of organs) occur."

The former is used in granular form in the Okanagan weed control programme and the latter is utilized extensively in liquid form for Milfoil control by the Tennessee Valley Authority.

The chemical was administered to the test animals in aqueous oelatin or corn oil on a daily basis from day 6 to 15 of gestation. In the prenatal studies the dams were sacrificed on the 22 day of pregnancy while some post natal studies allowed the females to litter. The research indicated that the dosages ranging from 25 mg/kg to 300 mg/kg of the various formulations of both 2,4-D and 2,4,5-T produced teratogenic effects.

"The 4 prenatal parameters (No. of viable fetuses; No. of dead fetuses; Fetal weight; and % of malformed fetuses) all showed a statistically significant dose-related effect, except for numbers of viable fetuses in the case of 2,4,5-T. The malformative effect was particularly striking: the average incidence of affected fetuses rising from less than 10% in treated controls to usually more than 50% at the 150 mg/kg dose level" (Khera & McKinley, 1972).

The report continues that "at the highest dose (usually 150 mg/kg), all derivities of 2,4-D (including butoxyethynol ester) were associated with a significantly increased teratologic incidence; the butyl and isooctyl esters tended to depress fetal weight." The most common defects observed were: unilateral and bilateral wavy ribs; additional ribs (14); retarded ossification (bone formation) of the frontal and parietal (in skull) bones; sternal (breastbone) defects. In the postnatal studies, a nondose-dependent low incidence of delayed opening of eyelids, hydrocephalus, corneal opacity, and chronic inflamation of the tear glands were seen. It is also interesting to note that "at 25 mg/kg, 2,4,5-T gave negative results while 2,4-D gave significant results regarding litter size and incidence of malformations." (Khera & McKinley, 1972).

The animals were fed three formulations of the chemical at dosages ranging from 20 to 100 mg/kg on the 6th through 10th days of pregnancy. Fetal viability was decreased significantly at the 100 and 60 mg/kg levels in one sample and at the 60 mg/kg dosage in another. Fetal abnormalities rose to 22% at the 100 mg/kg level in one sample while the rate was 3.5% in the control group. Hemorrhages per total live born were consistently increased at the low dose levels. The abnormalities most often seen were fused ribs.

The tests with 2,4,5-T used samples with varying TCDD contents. Abnormalities which occured were clearly dose and dioxin content related. However, samples with no detectable dioxin levels produced a significant increase in abnormalities and a decrease in fetal viability. The most common defects observed were eye abnormalities and delayed skull ossification (bone formation) while exencephaly (brain outside cranium), hind limb deformities, cleft palate, ectopic heart (misplaced) and fused ribs were also seen (Collins & Williams, 1971). During this same period, German experiments also showed "purified" 2,4,5-T to be teratogenic (Roll, 1971)

Canadian Research

Canada's Food and Drug Directorate of the Department of National Health and Welfare produced their research findings in early 1971. The testing, under the direction of K.S. Kera and W.P. McKinley used several formulations of 2,4-D and 2,4,5-T on Wistar rats. Of particular interest in this study was the fact that the butoxyethynol (BEE) ester and dimethylamine (DMA) formulations of 2,4-D were examined.

The problems of extrapolation preclude definition of the hazard on the basis of these studies, but its existence seems clear" (Epstein, 1972).

Further Testing

Shortly after the Bionetics studies were begun, researchers at the U.S. National Institute of Environmental Health Sciences (NIEHS) also began laboratory testing of the phenoxy herbicides. Both 2,4-D and 2,4,5-T were found to produce significant teratogenic effects. (Courtney, et al., 1970) At this time, however, it was learned that the formulations of 2,4,5-T which had previously been used for testing were contaminated with high concentrations (30 ppm) of 2,3,7,8-tetrachlorodibenze-p-dioxin (tetradioxin or TCDD), a substance believed to be extremely teratogenic.

This discovery, set into motion a new testing programme with "purified" phenoxy herbicides. The Dow Chemical Company, NIEHS and the U.S. Food and Drug Administration produced reports of testing 2,4-D and 2,4,5-T (containing less than 1 ppm of TCDD) on mice, rats and hamsters. These all confirmed that the phenoxy herbicides produced teratogenic effects even with very low levels of TCDD. The Dow studies of 2,4,5-T were conducted with very low dosages, the maximum being 24 mg/kg. This research indicated that there was defective sternebral ossification (incomplete bone formation of the breastbone) which is a not uncommon reaction to teratogenic substances (Emerson, et al., 1971).

The NIEHS study tested "purified" 2,4,5-T and TCDD on mice at 100 mg/kg dosage levels. Both compounds produced cleft palate and kidney malformations (Moore & Courtney, 1971).

FDA tests on golden Syrian hamsters with 2,4-D produced generally inconclusive results.

NOTES

1. Trypan Blue - Known Teratogen
2. Significance Levels * (.10) ** (.05) *** (.01)
3. Table modified from Royal Commission, 1975

Some of the abnormalities which were observed in the various strains of test mice were cleft palate (fissure in roof of mouth); anophthalmia (no eyes); incomplete fusion of face; agnathia (no lower jaw); microphthalmia (small eyes); single medial naris (one nasal entry); exencephaly (brain outside cranium); cystic kidney; clubfoot; ectopic intestines (abnormally located); encephalocoele (protrusion of the brain through a cranial fissure) extended legs (produced by breakdown product-2,4-dichlorophenol); and hydrocephaly (abnormal enlargement of the cranium caused by blockage of cerebral fluid) (HEW, 1969). After studying the Bionetics data, the Commission's Panel on Teratogenicity recommended that the butyl, isopropyl and isooctyl esters of 2,4-D be "immediately restricted to prevent risk of human exposure" (Epstein, 1970). Eight years after that statement was made, 2,4-D is the world's most widely used, generally registered herbicide.

2,4-D's sister compound, 2,4,5-T was also among 48 pesticides and industrial chemicals tested by the Bionetics Laboratories. It was in fact tested to a greater extent than any of the others. 2,4,5-T was administered to three strains of mice and one strain of rat both subcutaneously and orally during the 1965 to 1968 period. The dosage levels ranged from 4.6 mg/kg to 113 mg/kg. Both the mice and the rats showed a statistically significant increase in fetal abnormalities as compared with the control groups, even at the low dosage levels. The major abnormalities which were observed where cleft palates and cystic kidneys in the mice and cystic kidneys, gastro-intestinal haemmorrhages and incread fetal mortality in the rats. The Bionetics researchers concluded that "these results imply a hazard of teratogenesis in the use of this compound.

The data from their report, while it has never been made public, was included in Part III of the Mrak Report which was produced after the main work of the Commission had been concluded. The research findings had grave implications for the phenoxy herbicdes. 2,4-D and its sister compound, 2,4,5-T, were both found to be teratogenic in testing on mice.

The acid and a number of the ester formulations of 2,4-D were administered to laboratory mice subcutaneously (beneath the skin) with Dimethyl sulfoxide (DMSO) as a carrier. The dosage levels ranged from 24 milligrams of 2,4-D per kilogram of body weight (mg/kg) to 215 mg/kg with the treatment running from the 6th to the 14th day of pregnancy. On the 18th day the fetuses were sacrificed and examined. The fetal mortality rate ran as high as 74%, while the abnormality rate rose to 100% for the 215 mg/kg dosage of 2,4-D acid. The following table lists the formulations which produced a statistically significant number of birth defects when compared with the control groups. (Similar test animals who were not administered 2,4-D).

Bionetics Test Results-Teratogenic Effects of 2,4-D

Table I

Compound	Dose	% of abnormal litters	% of abn. fet./ all litters	% of abn. fet./ abn. litters
Negative controls				
Untreated controls	-	40	10	25
Controls - DMSO	-	46	13	29
Positive controls				
Trypan Blue (1)	5	60	32	54
Experimental - esters				
2,4-D isooctyl	48 mg/kg	100*(2)	24	24
2,4-D "	130 mg/kg	67	28**	41*
2,4-D butyl	100	75**	25***	34
2,4-D isopropyl	94	70**	26***	37*
2,4-D methyl	106	83	30*	36
2,4-D acid	98	36*	8	23

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For many, the test results may have come too late.

The lead arsenic and chlorinated hydrocarbon (DDT, Aldrin, Dieldrin, etc.) insecticides have been largely removed from use in North America now that their threat to health has been established. It took years of legal maneuvering and political pressure to get the job done, however. When a company spends time and money to get a new pesticide on the market and begins to receive a return on their investment, they are often reluctant to believe that the product might be a health hazard. It is all too easy for them to assert that the testing is faulty or that other environmental factors are to blame. Proving that a chemical is harmful and getting it removed from the market is extremely difficult, as we have seen with cyclamates, saccharin and the chlorinated hydrocarbons. The concept that a pesticide must be proven safe in all respects before it is allowed on the market has not met with very wide acceptance by regulatory agencies.

The Bionetics Study - First Warning

In the early 1960s, the U.S. Department of Health, Education and Welfare (HEW) appointed a Commission on Pesticides and Their Relationship To Environmental Health. The final product of this seven year, 3½ million dollar study has come to be known as the Mrak Report after Commission chairman Emil M. Mrak of the University of California at Davis. In 1969 as the final report was being prepared, the Bionetics research was made available to the Commission Panel on Teratogenicity. The Bionetics Research Laboratories, under contract to the U.S. National Cancer Institute, had been screening pesticides by laboratory testing from 1965 to 1968.

TERATOGENIC EFFECTS OF 2,4-D

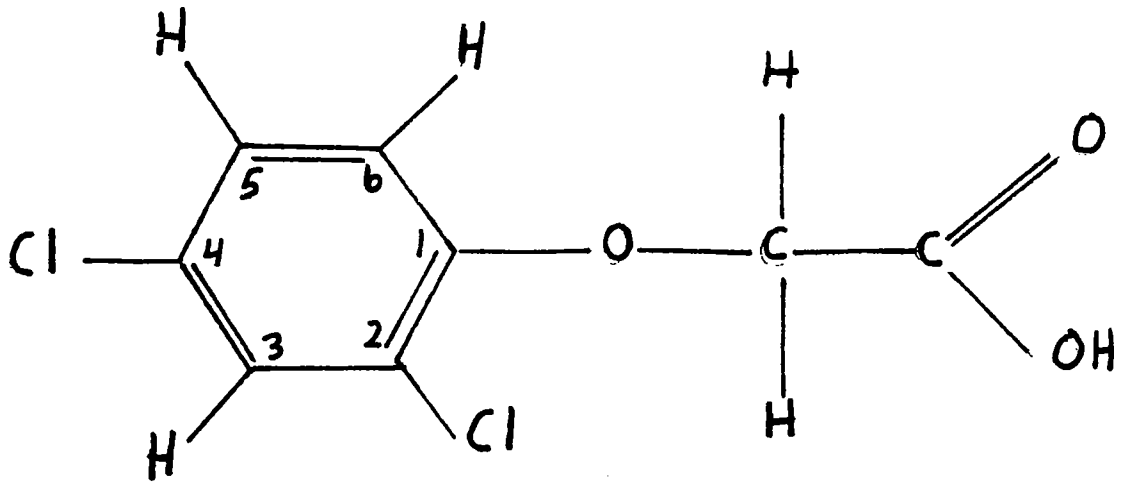
BIRTH DEFECTS

There can be no doubt that the most spectacular example of the teratogenic effects of a chemical on human beings was the Thalidomide tragedy of the early 1960s. Teratogenesis can occur when " a mother mammal is subjected to any condition that can alter her methabolic balance, at a time when the foetus within her is at a stage of developing organs" (Royal Commission Inquiry Into The Use of Pesticides and Herbicides, 1975). The birth defects which resulted from the use of Thalidomide by pregnant women are too easily remembered by everyone. In a macabre sense, the gross foreshortening of the limbs was a blessing in disguise. Had the effects been less noticeable (such as reduced learning capacity or greater susceptibility to disease), the situation could have gone undetected for years. As it turned out, North Americans largely escaped the disaster because, despite intense pressure from the companies involved, a few key health officials here refused to approve the drug until further testing was done (Shea, 1973).

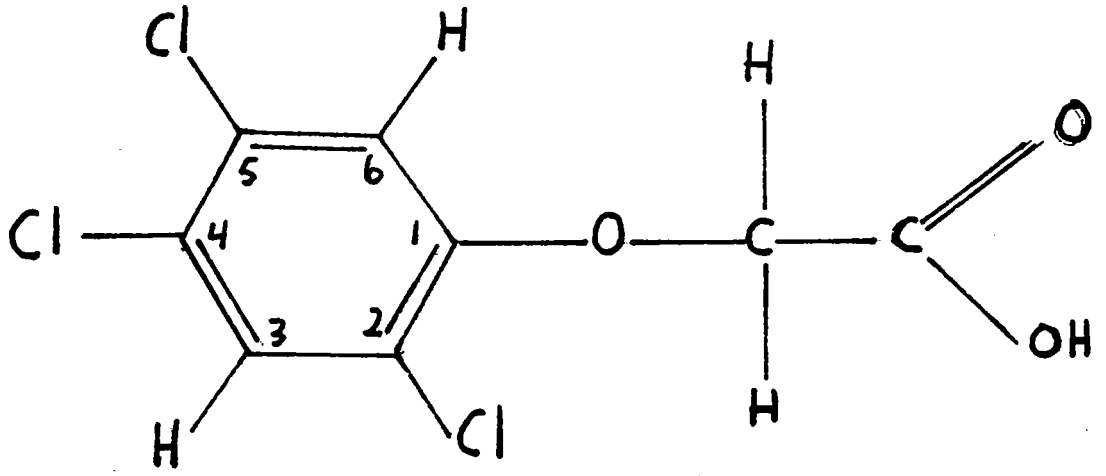
Detecting a teratogen can be a long and expensive process. The standard procedure to date for the registration of a pesticide has involved the submission of a producing company's test results to the regulating agency (Agriculture Canada and the U.S. Environmental Protection Agency). The testing usually centered around acute toxicity factors in laboratory animals. Long term research into the possible teratogenic, carcinogenic, and mutagenic nature of the chemical has not been part of the preregistration procedure. Only after the product has been introduced and health problems identified, have the regulating agencies moved to commission the necessary studies.

FIGURE 1

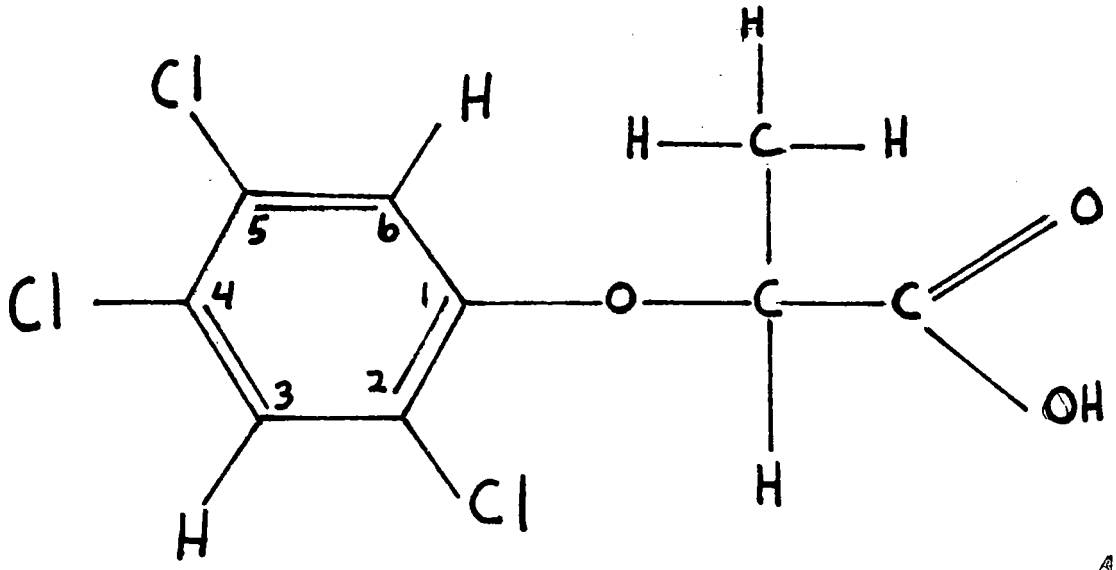
The Most Widely Used Phenoxy Herbicides



2,4-dichlorophenoxyacetic acid (2,4-D)



2,4,5-trichlorophenoxyacetic acid (2,4,5-T)



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2,4,5-trichlorophenoxypropionic acid (2,4,5-TP)

D.4544

The U.S. Defense Department felt that infiltration and ambush activities of the enemy could be reduced if major problem areas were defoliated for better aerial visibility. Between 1961 and 1970, at least 10% of all forested areas or 6.5% of all South Vietnam received at least one herbicide application (Galston, 1974). The main chemicals used in these operations were production grade formulations of 50% 2,4-D and 50% 2,4,5-T (Agent Orange). Also used was a mixture of 80% 2,4-D and 20% Picloram (Agent White) (Orians & Pfeiffer, 1970).

In the late 1960's, reports began to filter out of Vietnam about a dramatic rise in miscarriages and birth defects. (Grant, Moss and Unger, 1971) It was only after the Bionetics test results were released that the medical profession began to question the role of the phenoxy herbicides in the teratogenic effects which were reported. On April 21, 1970, the U.S. Department of Defense, under great pressure from the scientific community, announced that it was discontinuing its spray programme.

Trying to assemble a clear picture of the health impacts following the herbicide applications was extremely difficult. In 1969, the American Association for the Advancement of Science (AAAS) appointed a study team led by Dr. Matthew Meselson of Harvard University to examine the effects. The report which this group produced was instrumental in eliminating the defoliation programme. (AAAS - HAC, 1972)

Another prestigious organization, the U.S. National Academy of Sciences, also launched an investigation which was funded by the U.S. Department of Defense. Both of these efforts were severely hampered by the existing war situation in Vietnam which made on-the-ground inspection of the sprayed areas impossible. The research was carried out largely through the evaluation of those medical records which were available and aerial reconnaisance flights over the defoliated regions.

Because many Vietnamese births do not take place in hospitals, and inspection for birth defects is often not very thorough outside of the major urban centres, both reports recommended additional studies (NAS, 1974).

Some preliminary health problems were noted, which were similar to those produced by 2,4-D in laboratory animals. The Tay Ninh City provincial hospital, in an area where extensive spraying was carried out, reported a stillbirth rate of 58 per 1,000 in 1970. The Saigon hospitals, where a few people came into contact with herbicides, reported a rate of 29 per 1,000 during the same period (Aaronson, 1971). The Saigon Children's Hospital was known to receive patients from the entire country. Spina bifida, a congenital split of the vertebral column which can result in paralysis and other physical problems, increased from one case in 1966 to 13 and 12 cases respectively in 1967 and 1968. The incidence of pure cleft palate went from two cases in 1964 and 1965 to twelve in 1966, twenty-three in 1967 and twelve again in 1968 (Aaronson, 1971; Boffey, 1971).

In 1970, Dr. Ton That Tung and his colleagues at Hospital "A" in Hanoi produced a report on their study of spray effects on refugees from South Vietnam. Of the 73 pregnant women interviewed, 22 had had miscarriages following spray applications. There were also many reports of farm animals aborting and hens failing to lay eggs after defoliation (Ton That Tung, 1970; 1973).

Unfortunately, the Vietnam war presented an opportunity for an examination of the effects of major herbicide applications on human health. All the studies which were completed stressed the need for additional research. For various reasons, it appears that the work may not be done.

We continue to have only a sketchy idea of the damage which resulted.

Field Reports - Arizona

While Agent Orange was being used in Vietnam, the very same formulations were being used regularly in North America. As in Southeast Asia, the applications here have been monitored haphazardly if at all. During the years 1965 to 1969, five herbicide spray applications were made in the Tonto National Forest near Globe, Arizona. The purpose of the programme was to replace the chaparral groundcover with grass for grazing (Shoecraft, 1971). Spray drift resulted in many damaging effects at near-by residences. The Forest Service was quick to deny that there had been any damage. They maintained that only 2,4,5-TP (Silvex, Fenoprop) was used, but it was later learned that 2,4-D and 2,4,5-T had also been applied.

In July, 1970, the Arizona Commissioner of Health wrote in a letter to the Gila County Board of Health (Globe) saying that "... your county is leading the state in deaths caused by diseases of early infancy and fetal deaths....there are excessive deaths from congenital malformations" (Shoecraft, 1971).

The Canadian Experience

In July of 1969, the herbicides 2,4-D, 2,4,5-T and Dicamba were sprayed on the Belleisle Marsh in Nova Scotia near the farm of Robin Warren. During the aerial applications of the herbicides, a wind was blowing which caused spray drift onto the Warren pastures.

One half of the dairy herd was grazing in the treated area and the other half was upwind and did not come into contact with the chemicals. Of those in the sprayed pasture, 22 aborted their calves. The others in the "clean" area produced only one abortion. An analysis of hay samples 15 months after the spraying operations showed 10 parts per billion (ppb) of 2,4-D and a "trace" of 2,4,5-T. Mr. Warren was later compensated by the government for damage done to his farm. Fortunately no children were born in the area at the time (Ogden, 1971).

A powerline right-of-way on Galiano Island, B.C. was sprayed with 2,4-D in 1972. The line ran near the water supply of Dan and Francine Renaud. Their daughters, Sarah and Tamie were born in 1973 and 1975, both with the spina bifida malformations of the spine. There are other reports of a high incidence of spina bifida and cleft palate in the area at the time, as well as one child born with hydrocephalus (abnormal enlargement of the cranium) which died at the age of two (Vancouver Sun, October 3, 1973; The Daily Colonist, May 27, 1977).

Of the malformations reported by delivering physicians in British Columbia, fifty percent are of the type reported in laboratory testing of 2,4-D. These are spina bifida, cleft palate, club foot, hydrocephalus and other skeletal malformations. In the case of cleft palate, British Columbia leads Alberta, Manitoba and New Brunswick by a substantial margin (Elwood and Rogers, 1975)

As Dr. Samuel Epstein states clearly, the "continued use of these herbicides (2,4-D and 2,4,5-T) in the environment constitutes a large-scale human experiment in teratogenicity.. Such an experiment is unwarranted by any conceivable criteria in the face of the unambiguous warning sounded by available scientific data" (Epstein, 1970).

Other Reports

In Guamo, Columbia it was reported that one million inhabitants of the province of Tolima were exposed to 2,4-D and 2,4,5-T spraying. There followed an apparent epidemic of human abortions, stillbirths and malformations. The incident occurred in October of 1974 and the government promised an investigation. As of this writing, we have not been able to track down further research on this incident. (Environment, January, 1975)

From 1971 to 1974, 2,4-D and 2,4,5-T were sprayed from helicopters to control roadside weeds near Pittsville, Wisconsin. Following the applications many abortions, deformities and deaths were noted in wildlife and domestic animals. Six pregnant women in the area aborted (Stevens Point, Wisconsin Daily Journal, March 10, 1975).

During her testimony at the trial of Citizens Against Toxic Sprays versus Earl Butz, Secretary of U.S. Department of Agriculture, Dr. Eloise Kailan described patients which she had examined after exposure to the phenoxy herbicides. All exposure took place in the Siuslaw National Forest in Oregon. Dr. Kailan testified that four of her nine female patients experiencing miscarriages since living in the area, two of them on two occasions and one 16 times (Kailan, 1976).

Summary

The public began to become aware of the potential of 2,4-D to cause birth defects in early 1969. At that time the U.S. Department of Health Education and Welfare released the Mrak Report. This study included the test results of the Bionetics Laboratories' research into the effects of 2,4-D on mice.

The tests indicated clearly that the herbicide was teratogenic in the laboratory animals. The Commission's Panel on Teratogenicity recommended strongly that the butyl, isopropyl and isooctyl esters be "immediately restricted to prevent risk of human exposure." 2,4,5-T was also found to cause malformations during the tests.

In quick succession, the U.S. National Institute of Environmental Health Sciences, the U.S. Food and Drug Administration, the Canadian Food and Drug Directorate and the Dow Chemical Company produced test results on 2,4-D. All published findings which showed that 2,4-D, its various formulations, and one breakdown product produced teratogenic effects in mice, rats and hamsters. Some of the common abnormalities observed were cleft palate; clubfoot; no eyes; no lower jaw; distorted, missing or extra ribs; unformed breastbone; brain mislocation; enlargement of the skull and various hemorrhages.

Testing procedures have been criticized as not being appropriate for human comparison. However, it is asserted that mice are good indicators of teratogenic potential in humans and, as in the case of thalidomide, are often less sensitive. Unfortunately, 2,4,5-T has distracted public concern from 2,4-D because of its highly toxic contaminant, TCDD. There are also powerful economic pressures keeping any restrictions from being placed on this teratogen (2,4-D).

While field reports are a crude tool compared with sophisticated epidemiological investigation, in many cases, they are our only early warning system beyond laboratory testing. With 10% of the forests defoliated, Vietnam stands as the most intensively herbicide contaminated country in history.

Unfortunately, due to the war situation and the lack of adequate birth defect information, we do not have the moutain of data which we should. We do know, however, that the incidence of stillbirths, spina bifida and cleft palate increased significantly among those who had contact with the herbicides.

In North America, birth defects have followed 2,4-D applications in Arizona, Wisconsin, Nova Scotia and British Columbia. There was also a major incident reported in Columbia. Statistics indicate that 50% of the malformations seen by delivering doctors in British Columbia were present in the test animals during herbicide testing. There is clearly reason for concern that 2,4-D may have the potential to produce a significant number of birth defects in humans.

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CANCER AND THE PHENOXY HERBICIDES

Cancer (neoplastic disease) is now a major killing and disabling disease in Canada and the United States. It has clearly reached epidemic proportions. Today, one in every four persons in North America is developing some form of cancer, and 20% of all deaths are attributed directly to the disease. (Epstein, 1976) The death rate since 1900 in North America has risen 240% in terms of deaths per 100,000 people. There is hardly a family today in our country which has escaped this dreaded disease.

Furthermore, there is growing evidence that most carcinogens (agents which produce neoplasms in animals including man) are toxic substances in the environment. It is often said that cancer is "a disease of civilization."

In 1976 the Director of the U.S. National Cancer Institute estimated that 90% of all cancers are caused by environmental carcinogens. (Epstein, 1976) The World Health Organization took a similar stance that same year. (Webster, 1976) This means that not only is man responsible for most cancers, ultimately we can reverse this disastrous course and remove most of the carcinogens from the environment.

What is Cancer?

In the normal body, cell-reproduction is disciplined and orderly. Cell growth occurs during reproduction following fertilization, to repair damaged tissue and to replace "worn out" cells. A tumerous cell has a defect in the genetic apparatus that results in the loss of repression of the DNA synthesis and thus the loss of regulation of cellular reproduction. A cancerous cell reproduces without regard for its original function. It is bizarre in shape, size and in the form of cell groupings.

The nucleus of the cancerous cell is also deformed.

A neoplasm is an abnormal mass of tissue that grows rapidly and continues to grow after the original stimulus ceases. It is referred to as a me tastasizing tumour when it reproduces similar tumours in parts of the body remote from the original site of the disease.

In recent years research has discovered that more and more pesticides are carcinogenic to laboratory animals. There is increasing public pressure to ban these potentially harmful chemicals. In response to this pressure, the manufacturers of pesticides and the primary users have argued that there is a basic difference between malignant tumours (those which exhibit uncontrollable growth) and benign tumours (those that appear to have normal cell structure, and are often characterized by encapsulation). From this they have concluded that pesticides which only cause benign tumours in test animals should not be banned. (C.A.S.T., 1975; Perg, 1977)

However, this position is challenged by medical authorities. Dr. Harold Stewart, former chief of pathology at the U.S. National Cancer Institute, states that "you can't really separate the two terms. We know that if tumours are untreated they will very often metastasize." Dr. Sidney Weinhouse, Director of the Fels Research Institute in Philadelphia, which does extensive cancer research, holds that "there are no strict, quantitative criteria that differentiate benign from malignant tumours. So-called "benign" tumours simply grow more slowly than malignant tumours." (Webster, 1976)

The World Health Organization takes the same stance. The term "chemical carcinogen" is used to indicate a substance that is known to induce or enhance neoplasia. "Induction of tumours and enhancement of tumour induction are not distinguished for present practical purposes", the WHO stated in 1972. They hold that "the terms 'tumorigen', 'oncogen' and 'blastomogen' have all been used synonymously with carcinogen".

(Health Hazards, 1972)

In the United States, the Environmental Protection Agency, which now licenses all pesticides, has been strongly criticized for their decision to ban many of the chlorinated hydrocarbons (the DDT family). In June 1976 the E.P.A. took the position that a carcinogen is an agent which causes the increase of "malignant or benign cancers in humans or animals." (E.P.A., 1976)

The E.P.A.'s stance was supported by Dr. Russell W. Peterson, Chairman of the President's Council on Environmental Quality. At the annual meeting of the Society of Toxicology in Atlanta in 1976, Dr. Peterson argued that "because certain induced tumours transform into malignancies, and because we can seldom predict which tumours will become malignant and which will not, we should regard all tumorigens (tumour causing agents) as possible carcinogens." (Webster, 1976)

Are All Chemicals Carcinogens?

Many public officials, as well as spokesmen for the chemical companies and their major users, claim that all chemicals will cause cancer if given in large dosages. There is absolutely no basis for this statement.

For example, the Bionetics Research Labratories tested around 140 pesticides in the late 1960's for the U.S. National Cancer Institute to determine whether or not they could cause cancer when fed to two strains of labratory mice. Less than 10% of these pesticides were found to be carcinogenic. (Epstein, 1975)

In 1977, the U.S. National Cancer Institute estimated that only around 5% of the chemical compounds tested to that time on labratory animals had proven to cause cancer. The dosage levels of the chemicals when fed to experimental animals made no difference. (Miller, 1977)

Is There a Tolerance Level for Carcinogens?

One often hears public officials claim that there is nothing wrong with consuming toxic substances if it is in very low doses. But this applies only to the acute toxicity of a chemical: its immediate effect as a poison. It does not apply to chronic toxicity.

For example, the B.C. Royal Commission of Inquiry into the Use of Pesticides and Herbicides (1975) points out that "tolerance of pesticides for drinking water have not been established by the Provincial and Federal Governments." The same is true in the United States.

In 1974 the U.S. Congress passed the Safe Drinking Water Act. The U.S. Government asked the National Research Council, an arm of the National Academy of Science, to undertake a study of the major water system contaminants. This was completed in 1977. They were asked to set "acceptable daily intake levels "for 45 major organic chemicals found in water supplies. They refused. The NRC held that "methods do not now exist to establish a threshold for long-term effects of toxic agents." (New York Times, July 21, 1977)

It is widely recognized that there is no tolerance level, or threshold level, for carcinogens. This was the position taken by the International Union Against Cancer in 1954, and it has not been reversed. The Union argues that a sharp distinction must be made between reversible and irreversible adverse effects from chemicals. For chemicals which induced irreversible and possibly cumulative effects, particularly carcinogens, there is no way to define a tolerable limit. (Epstein, 1975)

The World Health Organization has traditionally taken a similar position. They hold that a "no effect" level cannot be determined for carcinogenic substances. As they state in 1972, "this concept is based on experimental animal studies showing that, unlike other forms of intoxication, carcinogenesis is the result of an accumulation of irreversible cellular damage rather than an accumulation of a toxic substance."

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(W.H.O., 1972)

The Delaney Amendment to the U.S. Food and Drug Act, adopted in 1958, reflected this thinking. It holds that "no additive shall be deemed safe if it is found to induce cancer when ingested by man or animal, or if it is found, after tests which are appropriate for the evaluation of the safety of food additives, to induce cancer in man or animal.." The U.S. Department of Health Education and Welfare has supported this law. (Epstein, 1976) There is no comparable law or regulation in Canada.

Paul N. Craig and Gene Miller of the Franklin Institute in Philadelphia have conducted research on this question. After reviewing 151 experiments with different chemicals which produced cancer in labratory animals, they concluded that there is no evidence that there is a threshold dose below which a carcinogen will not cause cancer. (Miller, 1977)

One of the foremost authorities on cancer and teratogens in North America is Samuel Epstein of the Case-Western Reserve College of Medicine. Dr. Epstein argues that the "no threshold level" position "reflects the overwhelming consensus of the qualified scientific community." (Epstein, 1976) In 1976 the U.S. Environmental Protection Agency restated their position in the issue: "The only safe exposure level for a carcinogen is zero." (E.P.A., 1976)

There is a wide variation in susceptibility of humans to cancer depending on many factors, including heredity, race, age, sex, diet and nutrition, and immunologic competence. It is for these practical reasons that it is impossible to set universal tolerance figures. (Meissner and Warren, 1971)

The Danger of Low Doses over Long Periods of Time

It is widely recognized that exposures to carcinogens are irreversible as well as cumulative.

It is true that a single exposure may cause a cancer after a latent period. But most cancers are a result of exposure at low doses over a longer period of time. (B.C. Royal Commission, 1975)

When humans are exposed to a variety of carcinogens, the possibility of contracting the disease increases. For example, it is well known that those who smoke tobacco have a higher rate of cancer than those who do not, and not merely lung cancer. Certain industrial workers (like chemical workers and uranium miners) have very high cancer rates. Those who simply live near to heavy industrialized areas and who are exposed to air pollution have a much higher rate of cancer. (Epstein, 1976)

In some cases carcinogens can be synergistic, increasing the effects through interaction with other carcinogens. There is also the case of "cocarcinogenicity", where a known carcinogen can be enchanced by combination with another agent which itself carries no carcinogenic potency. Croton oil has this effect on the carcinogenic hydrocarbons.

In addition, some known carcinogens are radiomimetic: producing effects similar to that caused by radiation. Chemicals that have an "estrogenic-hormonal-like-activity" fall into this category. For these, low doses over a long period of time have proven to be more dangerous than large doses over the same period of time. The small doses evoke "tissue response", while the large doses do not. The tissue response takes the form of proliferate cellular growth. (Doucet, 1976)

The Time Lag in Exposure to Carcinogens

In many cases, environmental cancer is not detected until many years after the original exposure. This has even been true in the case of industrial workers who have been exposed to high levels of carcinogens on a regular basis.

Very often, cancer caused by environmental factors appears around 15 years after exposure, and often even longer.

For example, in 1975 and 1976 cases of angiosarcoma, a rare form of liver cancer, began to appear in older Wisconsin farmers. Scientists from the U.S. Center for Disease Control in Atlanta and Mt. Sinai Hospital in New York investigated the cases.

Previously, this cancer had been linked to workers in arsenic factories. In this case, the cancer was linked to the use of lead-arsenic pesticides, at one time widely used in Wisconsin, but also all over North America including the Okanagan. The latency for angiosarcoma had been ranging from 20 to 30 years after the original exposure. (Western Producer, April 1, 1976) The B.C. Royal Commission concludes that "the greater frequency of cancers with increasing age suggests a low level of long span exposure to a carcinogen." (B.C. Royal Commission, 1975)

Is Animal Testing Adequate?

In recent years some public figures in North America have criticized the dependence on labratory animals for determining whether a chemical substance is a carcinogen, a teratogen or a mutagen, but those dealing with cancer research remain convinced of the relevance of tests on labratory animals.

For example, in 1977 the U.S. National Research Council released a report on drinking water and health. They were particularly concerned about organic chemicals which could produce chronic toxicity in humans. They argued that effects in animals, properly qualified, are applicable to man:

> This premise underlies all experimental biology and medicine. But because it is continually questioned with regard to human cancer, it is desireable to point out that cancer in men and animals is strikingly similar. Virtually every form of human cancer has an experimental counterpart; and every form of multicellular organism is subject to cancer, including insects, fish and plants. (Miller, 1977)

Dr. John H. Weisburger, Head of the Carcinogen Screening Section of the U.S. National Cancer Institute, adds that "every one of the agents with demonstrated carcinogenic potency in man has also caused cancer in one or more species of animals under specified conditions." We should expect this to be so, for "in the main, biologic systems in animals are not different from those in man." (Weisburger, 1973)

The necessity to use laboratory animals for testing is obvious; it would be out of the question to use human beings. Because it is "unacceptable to wait from 15 to 40 years to find our whether a suspect chemical will produce cancer in man", Dr. Russell Peterson argues that "every chemical that induces cancer in animals must be regarded as a potential carcinogen in humans." (Webster, 1976)

Are High Dosage Tests Relevant to Human Beings?

The other popular attack on the scientific studies deals with the dosage levels of the agent administered to the laboratory animals. Since these are usually higher than the dosages that humans would actually consume, it is argued that the tests are not relevant to the human experience.

The use of high doses is a practical necessity to compensate for the small number of laboratory animals used in a research project.

Dr. Samuel S. Epstein points out that if a particular agent were to cause cancer in 1/10,000 humans exposed, it would be mere good luck for the cancer to show up in a test conducted on only 50 laboratory rats or mice. In theory, such a test would need to use 10,000 rats or mice to yield one cancer. Thus, the higher dosage is used to try to compensate for the size of sample. (Epstein, 1976)

Here it might be well to recall that a carcinogen causes cancer at low levels of exposure as well as at high levels. But exposure to high doses makes the cancer appear more quickly.

In addition, the high doses compensate for the faster metabolism of the test animals relative to humans. Cell turnover is much faster in small animals than it is in humans. Furthermore, we know that laboratory animals, particularly rats and mice, detoxify and excrete chemicals more rapidly than do humans.

The history of the method of testing for carcinogens is outlined by Dr. John H. Weisburger. It was long suspected that 2-naphthylamine was causing bladder cancer in industrial workers. But laboratory tests on animals, using dosage levels similar to human exposure, produced only negative results. Then in 1937 Heuper proved in laboratory tests on dogs that it did cause bladder cancer. He achieved this result by administering high dosages. Dr. Weisburger concludes that "this was a vital finding, and is occasionally forgotten in contemporary discussions. The observations focused major attention on the critical problem of dose and duration of carcinogen administration to reproduce in animals lesions seen in man." (Weisburger, 1973)

The Shortcomings of Laboratory Tests

Research studies on cancer reveal that there are significant differences in laboratories between test species, strain and sex. 4465

And these factors vary with different chemicals. For example, the important human carcinogen, 2-naphthylamine, has proven to be carcinogenic in dogs after a long latent period, in hamsters after high dosage, and in the mouse where it produces liver cancer. But it has not caused cancer in labratory rats.

There are other factors which are related to the environment. Labratory animals are always in apparent good health; those which are ill are discarded, or abandoned half-way through the experiment. Normally, they are fed a well-balanced diet. They may not be under as much stress as many human beings. They live in regulated climates. They are only exposed to the agent suspected of being a carcinogen and used in the test; human beings are constantly exposed to a wide variety of environmental carcinogens. (Epstein, 1970) (Shea, 1973)

Dogs and monkeys appear to be better test animals than rats, mice and hamsters. Their survival levels for acute toxicity are much closer to that of human beings. Furthermore, they have usually been exposed to the human environment and are generally not bred through generations of labratory conditions. (Seabury, 1963) However, experiments with these animals take longer and are more costly.

Finally, most tests are done on only one generation. In the case of diethylstilbestrol (DES), cancer of the reproductive organs appeared in the daughters of women who had taken DES during pregnancy. In the case of saccharin, cancer appeared in the second generation of labratory rats. Most people believe that we need three generations of testing to provide more reliable results. But again, this is a costly and time-consuming approach.

Is 2, 4-D a Carcinogen?

There have always been good reasons for suspecting that all the phenoxy herbicides might be carcinogens. First, they act as an auxin hormone in plants, killing them through a massive reproduction of cells. In plants, then, it has been known that they attack the cell nucleus and upset the normal controls on cell growth. Might they not act the same way in animals?

Secondly, all phenoxy herbicides are manufactured from benzene, a carbonization of coal. The polynuclear aromatic hydrocarbons have been identified as causing cancer in humans. Benzene has for over 60 years been suspected of causing leukemia, although laboratory test results have varied. In 1977, the three year study by New York University's Institute of Environmental Medicine established that benzene fumes caused leukemia in laboratory mice and rats. (Globe & Mail, August 8, 1977)

All the phenoxy herbicides share the common "benzene ring" structure of the molecule onto which other atoms are attached. Many of the known carcinogens have as their core the benzene ring. Some of these are the polynuclear aromatic hydrocarbons, aromatic amines and aze dyes and some of the nitrosamides and hydrazines.

Fourthly, a significant number of chlorine-based chemical compounds have proven to be carcinogens. The most widely known are the chlorinated hydrocarbon insecticides (the DDT family). Another is chloroform. All the phenoxy herbicides have chlorine atoms attached to the benzene ring. (B.C. Royal Commission, 1975)

Finally, there is the fact that the phenoxy herbicides are synthesized from chlorophenols. It has been known at least since the release of the Mrak Commission Report in 1969 that a number of the chlorophenols were carcinogenic to laboratory animals. (Mrak, 1969)

It was not until the release of the Bionetic Research Laboratories' studies in 1969 that interested people became aware of the dangers of the phenoxy herbicides. In laboratory tests on small animals, 2,4-D, 2,4,5-T and 2,4,5-TP (Silvex or Fenoprop) all produced the same types of neoplasia.

The tests using 2,4-D were done by Dr. Berge M. Ulland and were completed on October 18, 1967. Tests performed on two strains of mice produced leukemia (cancer of the bone marrow), reticulum cell sarcoma type A (cancer of the lympth glands and connective tissue), Pulmonary Adenoma (lung cancer), Pulmonary carcinoma (Bronchogenic) and Hepatoma (liver cancer). Benign tumours were also reported in the liver, spleen and stomach of three of the test animals. (Innes, et. al., 1969; Ulland, 1967)

U.S. Food and Drug Administration Tests

In 1963 the U.S. Food and Drug Administration completed a study of the effects of 2,4-D on dogs, and in 1964 they did a study on rats. Summaries of these results did not appear until 1971. (Hansen, et. al., 1971)

In the meantime, 2,4-D was widely being used on food crops and residues were beginning to be noticed. For example, in 1974 there was no tolerance level established for 2,4-D resudues in milk.

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However, one test done at GHT Laboratories in California found .0007 ppm residue of 2, 4-D in milk. It also reported 1.02 ppm residues in apples. (U.S. EPA, November 13, 1974). Pressure was mounting to revise the residue limits.

On April 8, 1976 the EPA mailed reregistration guidance packages to manufacturers and distributors of 670 food products containing 2, 4-D residues. The agency cited the 1963 and 1964 laboratory tests as "sufficient" to satisfy the "chronic" safety testing requirements for reregistration. This action prompted an inquiry by the U.S. Senate Subcommittee on Administrative Practice and Procedure headed by Senator Edward M. Kennedy of Massachusetts.

The Subcommittee sent the data on 2, 4-D that was in the E.P.A. files to Dr. Melvin D. Reuber for his evaluation. Dr. Reuber served as a pathologist at the National Cancer Institute between 1963 and 1971, and is considered a top expert in the field.

Dr. Reuber's report was published in December 1976. The FDA's study revealed that 2, 4-D caused malignant tumours in male and female rats tested: he concluded that "2, 4- Dichlorophenoxyacetic acid is carcinogenic in rats."

The study on beagle dogs was less conclusive. The F.D.A. experiment produced "scattered lesions such as atrophy of the tests and prostrate, interstitial nephritis, hemangioma of the adrenal, atrophic or cystic pituitary, atrophy of the throid, and hypoplasia of the bone marrow. Most of the lesions were seen in the endocrine organs."

However, even though the lesions occured predominately in treated dogs, the FDA researchers concluded that "none of these lesions in the dogs was believed to be due to the ingestion of 2, 4-D."

Dr. Reuber noted that "previous experience at FDA has shown that long-term chronic dog studies should be carried out for six years or longer in order for tumours to develop." (Reuber, December, 1976)

Studies of Organic Carcinogens in Water Supplies

In recent years there has been more research being done on the question of organic and other contaminants in North American drinking water. In 1970 the U.S. Geological Survey found a wide variety of heavy metals in all water supplies. Some of these are toxic at high levels. The main pollutants were arsenic, chromium, cobalt, lead, mercury and zinc. (Durum et al, 1971)

In 1970 the U.S. Environmental Protection Agency conducted a survey of 66 organic chemicals found in drinking water; 43 of these were tested for carcinogenicity, and 15 were found to definitely cause cancer in laboratory animals. Another 87 organic chemicals were identified as "suspected water contaminants," and of these 17 were known to cause cancer in laboratory animals. The phenoxy herbicides were included as suspected carcinogens. (U.S. EPA, 1970)

The most widely cited water contamination study was that done by the EPA on drinking water in the lower Mississippi River. They found 88 organic chemicals in the Louisiana water supply. In the treated water coming out of the Carrollton Water Plant in New Orleans, they found 36 organic contaminants. The EPA concluded by pointing out the strange situation where the U.S. Food and Drug Administration had banned certain chemicals from foods because they were known carcinogens but that they were in most municipal drinking waters.

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There were no standards of water quality in North America covering carcinogens, teratogens and mutagens. (U.S. EPA, 1972)

In a report issued in June 1975, Dr. Robert G. Tardiff, research toxicologist with the EPA's Water Supply Laboratory in Cincinnati, rated 128 organic chemicals commonly found in North American drinking waters. Seven were found to be "extremely toxic", one "super toxic" and 35 were identified as "suspect carcinogens." He cited eight organics as "positive carcinogens", and among them was 2, 4-D. (Haber, 1976; Tardiff, 1976)

These studies on water contaminants reveal the necessity of testing down to the level of parts per trillion. Unfortunately, the testing done in the Okanagan Lakes was only down to .001 parts per million. This is simply inadequate.

For example, one pesticide commonly found in water supplies, Dieldrin, has proven to be carcinogenic to laboratory animals at the lowest tested level, 100 ppb (part per billion). Some carcinogens have produced tumours in test animals at concentrations below 400 parts per trillion, according to the testimony of the Environmental Defense Fund before the U.S. House of Representatives Subcommittee on the Environment and the Atmosphere. (Haber, 1976)

Field Reports

It is very difficult to get field tests on carcinogens. Most chemical carcinogens have been identified through epidemiological surveys of industrial workers. It is difficult, to say the least, to clearly identify a chemical carcinogen in the environment in a direct casual relationship.

We have some evidence of the carcinogenic nature of pesticides through surveys done of agricultural workers. However, most of these workers (and farmers) are exposed to a variety of pesticides, and individual identification is nearly impossible - except where the workers are only performing a specific spraying task.

For example, in 1972 such as case was revealed by The Expresser, the magazine of the Swedish Government Employees Union. It reported that 30 workers had been employed by the Swedish Government to clear brush along the Swedish national railway. They were using a herbicide called Hormoslyr, a mixture of 2, 4-D and 2, 4, 5-T.

By February 1972, five of the thirty workers had died of cancer; a sixth died later in the year. The surviving workers reported that they regularly suffered from headaches, loss of taste, impairment of sight, bladder contraction and impotency, all recognized as symptoms of phenoxy herbicide pois oning. As a result of this case, the Swedish state railway stopped all chemical spraying of weeds. Following the reports from Vietnam in 1971, the Swedish Government had also banned all aerial spraying of phenoxy herbicides. (Province, February 22, 1973; Reuters Dispatch, February 26, 1972)

From this it was not possible to conclude that 2, 4-D alone was the carcinogen. But the Swedish Government concluded that the phenoxy herbicides were the cause.

The other source of epidemiological data comes from Vietnam, from surveys of the people who lived in areas sprayed with phenoxy herbicides during the American defoliation programme.

Normally, there is a relatively long period of latency between exposure to a carcinogen and the development and identification of cancer. However, evidence coming out of Vietnam suggests that Hepatoma (liver cancer) is already appearing in spray victims.

In a paper presented in 1973, Ton That Tung and his associates at the faculty of medicine at Viet Duc Huu Nghi Hospital in Hanoi reported that primary carcinoma of the liver had increased abnormally in Vietnam in the period since the herbicide spraying. (Ton That Tung, 1973)

Similar conclusions were reported in a paper that the group presented in 1976. They found that between 1956 and 1961 hepatic cancers accounted for 2.89% of all cancers in Vietnam. Between 1962 and 1968, the period when the herbicide spraying actually took place, hepatic cancers rose to 9.07% of the total. The Vietnam doctors attribute the rise to the phenoxy herbicides, suggesting that it may possibly be due to the various dioxins in them, particularly TCDD. (Laporte, 1977; Ton That Tung, 1976)

The Position of the Advisory Committee

The Advisory Committee on the Control of Eurasian Water Milfoil covers the question of whether or not 2, 4-D is a carcinogen in one short paragraph in the Second Interim Report. When Senator Kennedy's report on the U.S. Environmental Protection Agency was made public, one member of the provincial Legislature requested the provincial government to suspend the use of 2, 4-D in the Okanagan Lakes until it was absolutely certain that the herbicide did not cause cancer.

The MLA in question stated the medical experts had stated that one molecule of a carcinogen can cause cancer in a receptive cell.

In response, Dr. Courtland J. Mackenzie, Chairman of the Advisory Committee agreed that the herbicide 2, 4-D "may be linked to cancer - at least in theory." However, he then added that there would be no risk if the programme of administering the herbicide was "done properly": the idea is that "nobody will get a molecule." (Penticton Herald, April 20, 1977)

Summary

Not all chemicals are carcinogens, but those that are produce neoplasia at all dosage levels. Because of differences among individual humans, it is impossible to set tolerance levels for carcinogens.

Exposure to carcinogens is irreversible as well as cumulative. Many carcinogens have proven to be more dangerous when exposure is at a low dosage over a long period of time.

Research in both Canada and the United States reveals that our potable water supplies are becoming increasingly contaminated with organic chemicals. A number of these are known carcinogens.

Research by the Bionetic Research Labratory (1967) and the U.S. Food and Drug Administration (1963) demonstrate that 2, 4-D causes malignant tumours in labratory rats and mice. Evidence from Sweden and Vietnam suggests that the phenoxy herbicides cause cancer in humans as well.

IV

IS 2,4-D A MUTAGEN?

A mutation is defined as any inherited alteration in the genetic material. If they occur in germ cells (egg or sperm) of an organism, they may be passed on to future generations.

Mutations can produce a wide diversity of deleterious effects. Some mutant genes are dominant and will show up as foetal deaths, achondroplasiz (dwarfism), polydactyly (having more than normal fingers and toes), retinoblastoma (malignant tumour of the retina) and sterility. There are also recessive mutations which may not be expressed for several generations, among which are albinism, Franconi anemia, amaurotic idiocy, (childhood disease leading to loss of vision, paralysis and death) and phenylketonuria (mental deficiency). The most common genetic abnormalities are enhanced susceptibility to various diseases (such as diabetes and inborn errors of metabolism), increased infertility, premature aging and alterations in sex ratios. (U.S. HEW, 1969; Crow, 1968)

One of the few comprehensive studies of genetic and other diseases was done at Johns Hopkins Hospital, Pediatric Service, between 1965 and 1969. Of the diseases recorded among the patients, 7% were attributed directly to the inheritance of gene or chromosomal abnormalities. When the diseases were added that were possibly genetically influenced, the incidence rose to 39%. (B.C. Royal Commission, 1975)

It should also be noted that most genetic defects result in very early abortions and as a result are not added to these data. It has been estimated that around 25% of spontaneous abortions are due to chromosome abnormalities. (Wellford, 1972)

It has been known for some time that exposure to certain types of radiation can be mutagenic. However, it is less well known that a wide variety of chemicals can also cause chromosome aberrations in plants and animals (including man). The recognition that pesticides can cause mutations dates from around 1931, when it was discovered that nicotine sulphate induced chromosome abnormalities in tobacco, with reduced seed set. In spite of this discovery, there has been relatively little research done in this area. This is perhaps due to the fact that environmentally caused mutations are not different from others, so it has been quite difficult to show a direct causal link. (Kimmins, 1975)

The Phenoxy Herbicides as Hormones

The phenoxy herbicides are "growth regulators" which have hormone-like activity. At relatively low doses, they will bring growth responses in plants that are distant from the point of application.

Hanson and Slife (1969) have pointed out that auxins (plant hormones) can alter nucleic acid metabolism in plants. Holm and Abeles (1968) found that 2,4-D in soybean seedlings increases the RNA and DNA in plant cells. Crispeels and Hanson (1962) conclude that since RNA and protein synthesis are controlled by DNA, the primary side of 2,4-D action is probably the nucleus of the cell. Ashton and Crafts, in their widely used textbook on herbicides, conclude that the evidence available is that 2,4-D attacks the nucleus of the cell. (Ashton and Crafts, 1973)

This is what must be expected. After the application of 2,4-D cell division takes place. If the herbicide acts to unhook the controls over cell division and development, it must attack the nucleus of the cell.

Mutagenesis in Plants

There have been a number of studies of the triazine herbicides for mutagenic properties. Most of these produced mutations. The study by Plewa and Gentile (1976) of the effects of Atrazine on maize plants found that the herbicide was degraded by the plant into environmental mutagenic agents.

It appears that there are not as many tests done on 2,4-D, which is somewhat surprising. However, Sawamura (1961) found that 2,4-D caused chromosomal aberrations such as stickiness, bridges and fragmentation in plants.

K.D. Wuu and W.F. Grant have done considerable research on the mutagenic potential of 2,4-D in plants. In 1966 they reported that barley seeds soaked in 2,4-D produced malformed seedlings. They have concluded that far more rigorous analysis of the health and environmental toxicology of herbicides is required before they can be declared "safe." (Kimmins, 1975)

In 1969 the report on pesticides by the U.S. Department of Health, Education and Welfare (Mrak Commission) listed several studies that showed chromosomal changes in plants treated with a 10 ppm concentration of 2,4-D. They concluded that 2,4-D and 2,4,5-T have "mutagenic potential" as demonstrated with plant systems. (Mrak Commission, 1969)

Reviews of the effects of 2,4-D as a potential mutagen were done by the U.S. Forest Service in 1973 and by J. P. Kimmins for Environment Canada in 1975. They cite studies by Unrau (1953, 1954) and Unrau and Larter (1952) which found "highly significant" abnormalities of chromosome behaviour in rapidly dividing cells of wheat and barley sprayed with 2,4-D. Huhling et al (1960) found chromosomal effects in peas treated with 2,4-D.

Thus, studies show that applications of phenoxy herbicides result in rapid cell division and cell elongation, increased synthesis of DNA and RNA, and chromosomal abnormalities. These aberrations can be inherited. Wuu and Grant (1967) have concluded that the chromosomal aberrations induced by herbicide chemicals are essentially similar to those caused by x-rays.

This was the conclusion of Barbara H. Croker in her study in 1953 on the effects of 2,4-D on onions. Both 2,4-D and 2,4,5-T produced "physiological effects such as stickiness, condensation of chromosomes, and delay in spindle formation." The phenoxy herbicides produced chromatid breaks at all concentrations. "Both 2,4-D and 2,4,5-T affect the nucleic acid cycle, and it is suggested that the effect on the actual chromosome structure is analogous with the action of radiation." In this case study, reversibility or recovery was not demonstrated. (Croker, 1953)

Phenoxy Mutant Effects on Laboratory Test Animals

Plant chromosomes are structurally like mammalian chromosomes, so chromosome aberrations detected in plants are relevant to mammals. (B.C. Royal Commission, 1975) However, tests need to be done on animal species, and it is surprising how little has been done in the field of pesticides in general, and phenoxy herbicides in particular.

In 1971 Darving and Surner fed low doses of 2,4,5-T to fruit flies. The result was a drastic reduction in fertility, very high egg mortality, and total mortality of the first few larvae that were produced. Ovaries of the flies were diminished and rendered abnormally fragile, oogenesis was interferred with, and chromosome aberrations were induced.

Another study involved the feeding of Mongolian gerbils various doses of "pure" 2,4,5-T (i.e., "containing no measurable amount of TCDD") for five days.

Those receiving the higher doses showed substantial chromosome damage, especially chromotid gaps and breaks. (Majundar and Hall, 1972; B. C. Royal Commission, 1975)

In a third study, male rats were fed 2,4,5-T at the very low rate of 1 mg/kg. The results in the treated rats were chromatid aberrations. (Novy and Majundar, 1972) The only reported study of 2,4-D on mice concluded that 2,4-D was not mutagenic at a rate of 125 mg/kg, the same dosage which produced birth defects in hamsters. (B.C. Royal Commission, 1975)

Three professors at the University of Heidelberg studied the effect of 2,4-D on the growth of cultured chicken muscle cells in 1974. Under the influence of 2,4-D, " a marked increase of DNA-synthesizing cells was observed after 22 hours." They concluded that "the morphologic and quantitative results of this study support the assumption that 2,4-D takes effect on the level of chromosomes. Further investigations based on biochemical parameters should be undertaken because of the possible risks of exposure to man." (Haag et al, 1975)

After surveying the research on plants and animals, J.P. Kimmins, Associate Professor of the Faculty of Forestry, University of British Columbia, concludes that it appears that "the mutagenic and chromosomal aberration effects induced by herbicides in plants may also occur in animals." We should be concerned about this because "new and synthetic substances are more likely to be mutagenic than old or natural ones since organisms are less likely to be biochemically prepared to deal with the former than the latter." (Kimmins, 1975)

The Vietnam Experience

The best information of the effect of phenoxy herbicides on human beings comes from the Vietnam experience.

Most areas of Vietnam were sprayed with Agent Orange, a 50-50 mixture of 2,4,5-T and 2,4-D. This same formulation is widely used in British Columbia, although not on argicultural crops. (See Table II)

The first medical report was presented by Dr. Ton That Tung and four colleagues from Viet-Duc University Hospital of Hanoi at the proceedings of the Reunion internationale de scientifiques sur la guerre chimique au Vietnam, Paris, December 12, 1970. As the war was still in progress at this time, this report was the result of tests done on 179 human beings who were refugees from areas sprayed in the Southern part of Vietnam. (Ton That Tung et al, 1970; 1973)

As background, Ton That Tung reports that following spraying there was an abnormal amount of human miscarriages. For example, in the district of Long-Dien and An-trach (Fifth Zone), out of 73 pregnant women, 22 had miscarriages. In the district of An-nghia (Fifth Zone), after the spraying in March 1966 there were many miscarriages in farm animals. Most disturbing was the increase in molar miscarriages. The Vietnamese doctors attribute this to herbicide exposure during the beginning of pregnancy.

In a later report, Dr. Ton That Tung and colleagues report that in the Hue district in the period 1969-1970, following repeated spraying of Agent Orange by U.S. military forces, the rate of stillbirths recorded at hospitals rose to 48.5%, and congenital malformations were observed in 7.4% of children born in the same period. Laporte notes that these figures may be understated, for the Vietnamese word used to substitute for malformation is actually "monstrosity", and therefore tends to exclude lesser malformations. (Ton That Tung et al, 1976; Laporte, 1977)

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News stories also reported a significant increase in Downs Syndrome or Trisomy 21 (formerly called Mongolism) in the areas sprayed by the phenoxy herbicides. In their first study, the Vietnamese doctors report that Downs Syndrome was appearing in very young women from the areas sprayed. Of those cases in Hanoi at the time, the physicans conducted the usual chromosome tests on mothers and children. They concluded that Trisomy 21 was due to chromosomal breaks, "present in a proportion of 3,3/100 of cells from the children." This was roughly "six times as much as was observed in the survivors of Hiroshima as reported by Bloom et al."

Effects on Argricultural Workers

A second important source of information is the study by Yoder et al, which was part of a project sponsored by the U.S. Environmental Protection Agency. Three groups of individuals were tested for chromosome abnormalities over a period of one year: a control group, a group of farm workers who sprayed insecticides, and a group of farm workers who sprayed herbicides. Most of the farm workers in the insecticide group used organophosphates which are widely used in British Columbia, although it did include a few insecticides not used here. There were a number of herbicides applied, but 2,4-D was the most widely used. Tests were made during the spraying season when exposure was high, as well as in the off-season when no spraying was being done.

Blood samples were used. The authors note that an "analysis of chromosomal aberrations. . . is one of the major methods for evaluating chemical mutagenesis."

In the control group the mean number of gaps and breaks in chromatids did not appreciably change at either sampling period. In the groups exposed to insecticides, "the mean number of breaks increased 5-folds" in the samples. Mean breaks per person for those in the herbicide spraying group "increased by a factor of 25 during this period." The authors also found that "the subjects having the most gaps and breaks, respectively, during the high exposure period were members of the herbicide-exposed group." (Yoder et al, 1973)

Exposure to herbicides during the spraying season may pose a danger to applicators. Chromatid abnormalities could cause mutations, although in this case no tests were made on sperm cells. Since mutations are very often carcinogens, there is a likelihood that herbicide applicators will get cancer more often than people not exposed to herbicides. Furthermore, there is also the possibility that they will produce more children with birth defects than those not exposed to herbicides. In reviewing this study, Prof. George Streisinger of the Institute of Molecular Biology at the University of Oregon states that "in view of the proportionality between mutations and exposure, people exposed to any levels of herbicides may be expected to experience some incidence of mutation." (Streisinger, 1976) Dr. R.G. Camfield, in his study on "Mutagenicity of Pesticides" done for the British Columbia Royal Commission, points out that "compounds that produce any type of cytogenetic abnormality have usually been shown to induce point mutations, and it is usually considered that the induction of chromosome abnormalities is an excellent indication of genetic damage." (B.C. Royal Commission, 1975)

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Increase of Congenital Abnormalities in Canada

J. Mark Elwood and Janice R. Rogers have conducted a survey of congenital abnormalities in Canada. In British Columbia, the incidence of Downs Syndrome was .86 per 1,000 births. This was higher than the Canadian average of .78 per 1,000 births. (Elwood and Rogers, 1975)

A more specific study of Downs Syndrome was done by Jane Evans, Alastair Hunter and John Hamerton for the province of Manitoba. Between 1965 and 1973 they found a marked increase in Downs Syndrome among women in the 35-39 age group, rising from 2.5 per 1,000 births in 1965 to 7.6 in 1973. Neither study offers any reason for the present rates or changes. (Evans et al, 1976)

The B. C. Royal Commission notes that around 25% of all disease can be attributed to a genetic origin. This is increasing because of the progressive decline of natural selection in man. (B.C. Royal Commission, 1975)

The Ames Test

The cost and time involved in conducting laboratory tests limits the number that can be done given the present distribution of government budgets. In view of the potential hazard to health, it is somewhat surprising how little testing has been done on the mutagenic potential of chemicals, and in particular pesticides. In recent years, simpler tests have been devised using bacteria, yeast and human tissue (bone marrow and blood cells).

Among these new tests, the Ames Test (named after Dr. Bruce Ames of the University of California, Berkley) is the best known. In a series of tests of 174 chemicals known to produce cancer in laboratory animals, 156 of them proved to be mutagenic as well, using the bacteria test. (Ames et al, 1973; McCann et al, 1975)

Another test was devised by Toronto doctors Robert Bruce and John Heddle. They inject mice with the test substance, and five days later they scan the bone marrow for chromosome breaks. Then in four weeks they count the ratios of misshapen sperm heads to normal sperm to try to determine the extent of genetic damage. This takes longer than the Ames test but has the advantage of using intact animals rather than just cells.

However, they have discovered that some known carcinogens and mutagens do not show up in these tests, indicating that a system of cross-checks is needed. Heddle and Bruce argue that "it is vital to monitor all environmental agents to avoid breeding future populations riddled with genetic disease." (Engel, 1975)

In September 1977 Dr. Bruce Ames described his new improved test in an address to the American Chemical Society. The new test was administered to 300 substances known to produce cancer in animals, and it showed that they were all mutagens. Dr. Ames concluded that "mutagens are also carcinogens, with few exceptions." He also suggested that the "flood of chemicals" to which human beings have been exposed since the 1950's "may be the main cause of death and disability in advanced societies." (Globe & Mail, September 1, 1977)

A similar position is argued by Professor J. P. Kimmins:

> The increased rate of mutation imposed on the human population by mutagenic agents such as radiation, pesticides, and certain other synthetic chemicals poses a great threat to the future of mankind. In a natural population the process of natural selection constantly removes non-adaptive mutants from the population. In our sheltered, well-fed and doctored society, this process has been eliminated, increasing the so-called "genetic load": the percentage of the population carrying deleterious but sublethal genetic mutations. This load will ultimately cost society far more in terms of both human suffering and health-care dollars than the short-term benefits that the mutagenic agents provide. We may indeed be "living on borrowed time." (Kimmins, 1975)

Summary

2,4-D has produced genetic abnormalities in a variety of plants in laboratory tests. Very few tests have been done on the mutagenic potential of the phenoxy herbicides on laboratory animals, but two tests using 2,4,5-T produced chromosome abnormalities in Mongolian gerbials and rats.

The Epidemiologic research done in Vietnam, in areas sprayed with the phenoxy herbicides, reveals widespread and extensive chromosome damage leading to a high rate of miscarriages, still births, congenital abnormalities and Downs Syndrome. Medical authorities in that country compare the effects to those on the survivors of Hiroshima and Nagasaki.

A study sponsored by the U.S. Environmental Protection Agency found that applicators exposed to insecticides and herbicides both experienced a significant increase in chromatid breaks during the season of exposure. The rise in congenital abnormalities in Canada may be attributed to the elimination of much of the process of natural selection in the human species.

There is obviously a need for greatly increased testing in this area. However, there is enough evidence available now to make us very suspicious of 2,4-D and the other phenoxy herbicides.

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In northern Sweden, 250 reindeer died and 40 aborted in a herd of 600 after grazing in an area which had been treated 8 months previously with 2,4-D and 2,4,5-T. An analysis of liver and kidneys of the dead animals showed that they contained .45 - .50 ppm of 2,4-D and .05 - .10 ppm of 2,4,5-T (Erne & Nordkvist, 1971)

In Vietnam, concern focused on the human populations and agricultural crops following the massive spray programme there. With upwards of 10% of the forest area defoliated and mostly killed, the impact on wildlife must have been substantial. Yale University biologist, Arthur Galston, comments that he could obtain "no reliable data on effects (herbicide) on animals, but with a destroyed or at least drastically altered habitat, there is probably a concomitant change of catastrophic proportions in animal populations. The impact would be particularly obvious with respect to insects and birds, but no less important for mammals, reptiles, and other groups. I suspect that the only satisfied animals, at least for the time, would be the termites" (Galston, 1971). Observers did note that they did not see any insectivorous or frugivorous birds in the defoliated mangrove areas except barn swallows which were migrants from the north. They also indicated that while fish eating birds had not been as severely affected, their numbers were also reduced (Orians & Pfeiffer, 1970).

Spray applications of 2,4-D, 2,4,5-T and 2,4,5-TP (Silvex) made near Globe, Arizona during the period from 1966 through 1969 also resulted in reports of damage to wildlife. Charmion McKusik, an ornithologist specializing in osteology (study of skeletons) was living in the spray drift area at the time and made detailed observations. Brown Towhees were unable to fly, had eyes which were discharging or swollen shut, and showed stiffness of the joints. They were unable to eat, drink or walk.

Of the 1066 treated eggs, 702 embryos (66%) were dead before the 19th day of incubation. Of those embryos which reached the latter stages of development, the majority had malformations which prevented hatching. Of those which survived, more than half were sterile and the fertility of the others was greatly reduced. Other defects observed were spinal abnormalities, fused neck vertebrae, muscular atrophy (decrease in size) of the feet, shriveled and clenched toes, and feathers which were missing or lacking pigment (Lutz-Ostertag & Lutz, 1970). The effect of 2,4-D applications were clearly significant in this experiment. This is of particular concern to the Okanagan, as pheasants are just starting to reappear following the ban on DDT and other chlorinated hydrocarbon insecticides.

Wildlife And 2,4-D

Following spray applications (2,4-D, 2,4,5-T and Dicamba) at Belleisle Marsh, Nova Scotia, major effects were observed in wildlife of the area. Sparrows feeding on wheat seed flew poorly and spent a lot of time walking on the ground. A cat which fed on the sparrows died shortly thereafter. A fox found meandering in the area was taken to a veterinarian where it died. During the four years prior to the application of herbicides, the marsh had supported a 300 muskrat pelt per season industry. In 1970, the harvest dropped to 100 pelts and the following year the traps were not set due to the lack of animals. Mr. Warren reported that the muskrats seemed tame and "confused" and could be closely approached. Hay samples from nearby were tested and were found to contain 10 ppb of 2,4-D and a trace of 2,4,5-T, fifteen months after the spray applications (Ogden, 1971).

Ducks and geese feeding on the plants and probing the bottom sediments in these areas could consume a rather large dose of the chemical in a short time. For the herbicide to be most effective, applications are made in the spring. The treatment could result in the exposure of the large migrating waterfowl populations to substantial amounts of 2,4-D.

The Canadian Department of Agriculture reported that 10 Canada Geese were fed a diet of 100 ppm of 2,4-D over a period of time, while another equal size control group was maintained. As the experimental birds died or were killed, they were examined internally and compared with the control group. The geese which had been fed the herbicide showed the following: enlargement of the kidney; jaundiced appearance of other organs; reduced weight gain; organs with general, progressive "disorganization" of cellular structure; hepatic (liver) cell destruction, round cell invasion and fatty degeneration of liver tissue; kidney damage including capillary degeneration and thrombosis, invasion of connective tissue and arteriole wall enlargement (Royal Commission, 1975)

It would seem unlikely that a bird in the wild would receive a daily 100 ppm dose of 2,4-D. It is possible, however, that geese feeding in a treated area could receive a substantial amount of the chemical leading to some of the internal organ deterioration which was observed in the test birds. This information would certainly give wildlife managers cause for concern as large numbers of Canada Geese pause to rest and feed in the Okanagan Valley on their way north to nest.

A major study was conducted in France in an effort to explain a spectacular disappearance of major game birds in Europe. The eggs of pheasant, red partridge and the common gray partridge were sprayed with 2,4-D at a rate of 5.1 lbs per acre (Slightly higher than the normal agricultural application rate). A control group was sprayed with water.

Label restrictions on herbicides generally carry a warning to avoid contamination of water bodies. Forestry and right-of-way applicators are instructed to leave buffer zones near creeks, rivers and lakes. Water can obviously become a means of moving the chemical away from the application site. It is also the source of food and drink for wildlife and human communities. Our understanding of the effects of 2,4-D on the fishery of this province is obviously limited, though some of the data available should give us cause for concern. J.P. Kimmins, a Forestry professor at the University of British Columbia, feels that "our very incomplete knowledge of the sub-lethal effects of herbicide traces on aquatic organisms requires that until we have more information we should consider all herbicide contamination of forest waters undesirable and work to eliminate or minimize it". (Kimmins, 1975)

Effect On Birds

One would expect waterfowl to be the most affected by aquatic applications of 2,4-D. From the limited data available, it would appear that the acute toxicity level for waterfowl to this herbicide is relatively high. One report indicated that the rate for mallard ducks was 1,000 to 2,000 mg/kg (Tucker & Crabtree, 1970). Flocks of "dabbling" ducks have often been observed in or near the Milfoil beds of the Okanagan Lakes system. Apparently they feed on the Milfoil as well as the other aquatic plant species which make up the bulk of their diet.

A study by Lim and Lozoway, following 2,4-D applications in the North Arm of Okanagan Lake in 1976, indicated that Milfoil tissues could contain up to 80 ppm of the herbicide following treatment (Morley, 1977)

It should also be noted that acute toxicity can be increased through a synergistic effect when 2,4-D is combined with other pesticides. This factor is particularly important in the Okanagan Valley where these chemicals are used extensively on crops. In one test, rainbow trout when exposed to a combination of 2,4-D and Carcaryl (Sevin) showed an increased mortality rate of up to 85%.(Stratham & Lech, 1975) It may well be difficult to predict with any degree of accuracy what the toxicity of 2,4-D will be for fish populations given the general contamination of the water basin with other pesticides.

The acute toxicity is not the main concern of wildlife managers. Morley's study indicates that:

> "Low concentrations of 2,4-D maintained over a period of time could cause sublethal damage that is not readily recognizable. Such effects can lead to gradual decreases in fish populations through increased predation, reduced reproduction, increased susceptibility to parasites, disease and stravation. Morphological and histochemical changes in tissues and organs have been demonstrated for sublethal doses of several herbicides."

Loss of habitat, as weeds and their indigenous organisms are removed, is an additional problem. It is well established that "the absence of aquatic plants could reduce the availability of food and increase predation on these fish since shelter would be depleted" (Morley, 1977)

The chemical is also directly toxic to bottom, fish-food organisms. A study indicated that when exposed to a concentration of 1 to 4ppm of 2,4-D for a period of one week, the various nymphs, larvae, midges, beetles and worms suffered a population decline of 43%. At the end of one year of exposure, the rate had climbed to 90% (Walker, 1962) While this test was conducted in aquaria, it does indicate that damage to aquatic food chains can result from relatively low concentrations of this herbicide.

It appears that longer exposure times resulted in generally higher mortality (Pimentel, 1971). The fact that the Salmonid family of fish seems to be particularly susceptible to 2,4-D should be of particular concern in British Columbia now that the Milfoil is known to have spread beyond the Okanagan Water Basin. A substantial concentration of the herbicide in water used as a route to the salmon spawning areas could result in an environmental and financial disaster.

As is emphasized by Provincial Biologist R.L. Morley in his excellent study entitled "2,4-D: A Summary Of Information Relative To Its Possible Effects On Fish & Wildlife When Used In Aquatic Weed Control Programs" - the "toxicity of the ester formulations of 2,4-D to salmonids, and specifically rainbow trout, is very close to the concentration (1-3ppm) required in water to assure control of the target plants (Milfoil). This lack of adequate safety margin, especially when liquid formulations are being used, can result in fish kills" (Morley, 1977) Thus, 2,4-D also poses a threat to Okanagan game fish.

Five days after the application of 2,4-D in the weed control effort on Skaha Lake Beach near Penticton, dead carp and suckers appeared at the shoreline in varying states of decomposition. A total of eight, all weighing over one pound were observed. During and following this same period, a fisheries biologist speared some carp and suckers which were swimming lethargically in the treated area. A pesticide analysis report by the B.C. Department of Agriculture indicated that the carp flesh contained .44 ppm of 2,4-D while the sucker contained .47 ppm. The gullet contents of the carp showed a very high reading of 16.66 ppm. No salmonid species were collected for examination in the application area. (B.C. Fish and Wildlife, 1977)

It is reasonable to conclude then that pesticides in general and the phenoxy herbicides specifically, could pose a threat to any ecosystem. We watched in the past few years as Peregrine Falcons and Ospreys nearly became extinct due to the contamination of their food chains by the chlorinated hydrocarbon insecticides. Species of life are now going extinct at the rate of one per year. This compares with the other period of rapid die-off at the end of the age of dinosaurs when species went extinct at the rate of one per thousand years (Myers, 1977). The long term effect of any pesticide on an ecosystem is still largely unknown at this point.

2,4-D's Impact on Fisheries

Of particular concern to the Fish & Wildlife Branch in British Columbia is the effect of 2,4-D on fish populations of the Okanagan lakes system. In 1977 the granular Butoxyethanol ester formulation of the herbicide (Aqua-Kleen) was placed directly into the weed beds which also serve as fish habitat. The Branch has good reason for concern as previous testing resulted in health authorities advising fishermen not to eat any catch in excess of three pounds. The reason for this warning is that fish which have been sampled were found over recent years to be contaminated with residues of mercury, chlorinated hydrocarbon insecticides and polychlorinated biphenyls (PCB).

The first concern of any researcher when testing a chemical on an organism is the acute toxicity. How much of the substance will it take to kill the test species?

The acute toxicity of 2,4-D on fish depends on the species of fish and the formulation of herbicide used. Pimentel compiled tests results which showed that death occurred in 50% of the species treated (LC 50) at concentrations from .8 ppm to 620 ppm, after 24 to 48 hours exposure.

VII

ENVIRONMENTAL EFFECTS OF 2,4-D

2,4-D is a biocide, which is to say that it has been formulated to kill a certain sector of an ecosystem. The phenoxy herbicides are used to eliminate broad-leaved herbaceous plants. 2,4-D is therefore not very species specific, as the group which it effects is rather large. This chemical also can have an impact or other nontarget species which it may come into contact with, depending on the rate of application and the environmental conditions. Several studies indicate that wildlife can be damaged when their habitats become contaminated with the herbicide. This may happen inadvertently when 2,4-D is transported from the application site by air or water. In some cases, such as the Eurasian Water Milfoil control programme, the chemical is intentionally applied to a wildlife habitat in order to kill the weed. Because the herbicide is used extensively in agriculture (2,4, 5-T has been banned from agricultural use in many countries including Canada and the U.S. due to the health hazards involved with it and the presence of the contaminent Tetradioxin) increased interest in its effects on crops is resulting in an accumulating body of research data.

It is necessary to examine some basic characteristics of ecological systems before going further. While there is some difference of opinion, a healthy ecosystem generally exhibits a diversity of species with many complex interrelationships. Because of this incredible complexity, it is unlikely that the effect of any manipulation of the system can be predicted with long term accuracy. A major alteration in one sector will often set off wide ranging reactions. The impact of an intrusion in one area may be negligible, while the same act in an overburdened, heavily stressed ecosystem may have catastrophic effects. In essence, when we tamper with the natural systems of our planet, we are stumbling in the dark.

For example, in a 12 month survey covering 1972 and 1973 the Illinois Environmental Protection Agency, received notice of or directly participated in 521 pollution accidents involving the evacuation of 8 towns, injury to over 300 persons, and damage to more than 1,100 homes and 350 businesses. Most of these were storage and transportation accidents. (Graham, 1977)

In many of these cases, large damage suits are being filed. The victims of phenoxy herbicide spraying in Globe, Arizona have a large suit against the Dow Chemical Co. and the U.S. Forest Service. In the widely reported case of poisoning of polybrominated biphenyl (PBB) in Michigan, there are now 84 suits against the Michigan Chemical Company. In April 1974, a leak in a storage tank produced a thick fog of hydrogen chloride which soon engulfed 11,000 residents in the Altgeld-Murray housing project in Chicago. Over 300 were hospitalized and one died. The victims have a $100 million law suit now in the courts. (Graham , 1977)

Summary

All the phenoxy herbicides, including 2,4-D cause acute and subacute illnesses and psychological disorders in human beings. Those who report these illnesses include applicators, those engaged in manufacturing the products, and those accidentally sprayed. In relatively high exposure cases, chloracne is produced in the victims.

Many of the chlorophenol-based chemicals have produced serious toxic effects in humans. There are many reported cases. The phenoxy herbicides have also been responsible for a wide variety of illnesses among workers in chemical plants engaged in the production process. Furthermore, environmental accidents are becoming much more frequent in North America as the use of chemicals increases.

(Chlorophenols are known to produce dioxins when heated.) Tree planters have also reported phenoxy herbicide poisioning while working in areas sprayed with the herbicides.

There seems to be a pattern of attributing chloracne and other phenoxy herbicide poisonings to tetra-dioxin in 2,4,5-TP. Some have claimed that this can be solved by removing the TCDD in manufacturing. More will be said about that in the following section.

However, there is evidence that all the phenoxy herbicides produce chloracne, even 2,4-D and "purified" 2,4,5-T. It could be the phenoxy herbicides themselves, or it could be the presence of the other higher isomer dioxins known to be in the phenoxy herbicides. TCDD is a potent stimuli for chloracne.

Poland and his colleagues found chloracne among workers in plants manufacturing 2,4-D and 2,4,5-T, as well as hypomania (mild mental disorder)personality problems. (Poland et al, 1971)

Two doctors have reported treating more than 40 cases of chloracne contracted by workers in plants engaged in the synthesis of 2,4-dichlorophenol and 2,4,5-trichlorophenol. There was some question as to whether TCDD exposure (from the making of 2,4,5-T) was responsible for the chloracne in all cases.

This question was resolved by Dr. Marcus Key of the Indistrial Disease Division of the U.S. Public Health Service. He performed open patch tests on himself with the commercial preparations of 2,4-D and 2,4,5-T. They both induced chloracne on his arm. (Bleiberg and Brodkin, 1964)

Industrial accidents, exposure of workers, and accidents outside the factory are much more widespread than is recognized.

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Fifteen years later the exposed workers still reported the common symptoms of phenoxy herbicide poisoning.

One of the most widely reported cases followed an explosion in an N.V. Philips plant in Amsterdam in 1963. Fifty workers came down with chloracne, and many had damage to internal organs. The plant was closed for ten years, but contamination remained so high that it was completely dismantled, embedded in concrete, and dumped at sea off the Azores.

The Dow Chemical company closed one of its plants at Midland, Michigan when more than seventy workers came down with severe cases of chloracne. This occurred in 1964, when they were manufacturing Agent Orange for use in Vietnam. Yet Dow's representative at Senator Hart's hearings described chloracne as a minor disease. In reality, chloracne is an outward sign that severe poisoning has occurred.

Between 1965 and 1969 a manufacturing plant in Czechslovakia had extensive leaks in the area where 2,4,5-Trichlorophenol was processed. Workers experienced chloracne and other less acute illnesses; eight years later they still reported the illnesses.

An explosion took place in a Coalite and Chemical Product's plant in Bolsover, England in 1968 during the manufacture to 2,4,5-T and hexachlorophene. Seventy-nine workers developed chloracne, as did members of their families having contact with them. Several workers died. The equipment in the factory was removed and buried in an abandonned coal mine. (Whiteside, 1977)

Chloracne has also been reported among people who have been fighting brush and forest fires in areas treated with phenoxy herbicides in Oregon and California.

Polychlorinated biphenals (PCB's) have also produced severe chloracne in humans in proven cases. They are also extremely toxic and build up in the environment and the food chain. The most widely cited case of PCB poisoning occurred in Japan in 1967 when PCBs accidentally contaminated rice oil. Those exposed suffered from chloracne, and still births were widely reported. (B.C. Royal Commission, 1975)

Another product which has been highly toxic is hexachlorobenzene (HCB). In 1960 seed grain which had been treated with HCB was accidently consumed by over 3,000 people in Turkey. Many of these people died from the contamination, and chloracne was widely reported. (B.C. Royal Commission, 1975) (In 1967 six cases of poisoning were reported in Czechoslovakia)

Industrial Accidents Manufacturing Phenoxy Herbicides

The dangers to humans from the phenoxy herbicides has been known for quite some time due to the fact that there have been a number of well-reported industrial accidents. Several of these were cited by the B.C. Royal Commission of Inquiry into the Use of Pesticides and Herbicides, but the best overall account easily available is that of Thomas Whiteside in The New Yorker, July 25, 1977.

In 1949 two hundred and twenty-eight workers at the Monsanto Company herbicide plant at Nitro, West Virginia came down with chloracne as the result of an explosion while manufacturing 2,4,5-T. A similar explosion happened in 1953 in a B.A.S.F. in West Germany. All the male workers and many of their wives, children and pets broke out with chloracne. A number of workers suffered damage to their internal organs.

We can sum up this part by quoting from a letter from a woman who lives in the Siuslaw National Forest Area of Oregon:

> Down here the "flu season" starts when the herbicide use begins. It depresses or destroys the thymus gland which controls resistance to disease. We get colds, flu like symptoms, uterine bleeding, nose bleeds, headaches, double vision, asthma, dizziness, bronchitis and pheumonia. It can cause your hair to fall out, gives you a gut pain, nausea, and chloracne, which is similar to teenage acne but may be anywhere on the body. Brown pigmentation spots appear on the skin exposed to the sun, which once there, stays. It affects memory, the liver, the heart and causes bladder stones. It is vicious. (Pavel, 1976)

Chlorophenol Poisionings

It has been known for some time that many chlorophenol-based chemicals are dangerous to humans. Hexachlorophene, which is synthesized from 2,4,5-Trichlorophenol, is neurotoxic (poisonous to nerve tissue) and causes skin irritations. It was widely used as a disinfectant until 1972, when three dozen French infants died after being treated with a talc powder which had been mixed with hexachlorophene. (B.C. Royal Commission, 1975; Whiteside, 1977)

Pentachlorophenol (PCP) has also proven to be quite toxic. It is used in the processing of oils, leathers, paints, glues, textiles, and as a wood preservative. Commercial preparations have been found to be highly contaminated with tetra-dioxin and dibenzofuran. In 1948, workers in a Germany factory producing PCP broke out in chloracne and experienced nerve pains in the lower extremeities. The B.C. Royal Commission (1975) lists a number of PCP accidents, all of which brought some deaths.

Sodium-PCP has appeared as a pollution problem in effluent from pulp and paper factories which use PCP compounds as slime-controlling agents. It is extremely toxic to fish; human exposure should clearly be avoided. (Crossland and Shea, 1973)

Testimony Before Senator Hart's Committee, 1970

Following the reports of the accidental spraying at Globe, Arizona and the reports out of Vietnam, Senator Philip Hart's Committee on Commerce of the U.S. Senate held hearings on the effects of the phenoxy herbicides on human beings. Those medical specialists who testified reported similar effects for both 2,4-D and 2,4,5-T poisoning.

Medical authorities reported that exposure to phenoxy herbicides produced "pronounced hypochondria" and a decrease in mental capacity. There are often general psychoneuropathic complaints in the region of the extremities. Individuals exposed have a sense of insecurity, inner restlessness, and a feeling of illness. There is a change in normal behaviour, with increased dissatisfaction, sullenness and irritation. Fear and moodiness are common, what some doctors referred to as "psycho-pathological intoxication". (U.S. Senate, 1970; Shoecraft, 1971)

A Case of the Flu

Dr. Granville F. Knight of Santa Monica, California has had considerable experience with pesticides and their effects on humans through his work with California farm workers. He points out that the symptoms produced by exposure to the chlorine-based herbicides (the phenoxy herbicides) and the chlorinated hydrocarbon insecticides (the DDT family) "are similar to, and perhaps identical with, the symptoms of many virus infections which have now become common in the past twenty years." He feels that many people have had pesticide poisoning and have not sought medical help, concluding that they just had a bad case of the flu. (Knight, 1973)

In early 1971 two British professors visited North Vietnam and interviewed a sample of 98 evacuees from sprayed areas. The Vietnamese reported acute illnesses similar to those cited in the previous American studies. However, a greater incidence of skin burning and chloracne was reported. The illnesses had lasted longer among the elderly and children. (Rose and Rose, 1972)

The Committee of the U.S. National Academy of Science reported symptoms of acute illness following spraying of phenoxy herbicides in three separate studies. Children of the Montagnard tribespeople reported coughing, vomiting, skin sores, dizziness, and sometimes death. Similar effects were reported in a survey of two provinces, and in a community in the Rung Sat delta area. (Shapley, 1974)

The most thorough Epidimological work has been done by the group of doctors operating out of Viet-Duc Hospital in Hanoi, where a special monitoring programme was established. They divided the effects of phenoxy herbicide spraying into two categories: (1) clinical symptoms during the first hours, and (2) secondary effects.

The immediate effects on the Vietnamese were similar to those widely reported in North America: tears and watery discharge from the nose, intense weakness, giddiness, vomiting, diarrhea, headaches, sensation of burning on the skin, and fast heart beat. Asthenia (weakness) lasted for several months in those exposed.

The secondary effects reported are prolonged asthenia, chloracne and ocular syndrome (eye problems). The other category of prolonged secondary effects is in the genetic area: chromosomal alterations and congenital malformations, reported before. (Ton That Tung et al, 1970; 1973)

Four of her patients had frequent diarrhea following consumption of water from areas sprayed with the herbicides. Five experience peripheral neuritis (inflammation of the terminal nerves) three had chloracne, and two of the nine women reported unusual uterine bleeding.

Dr. Kailan also stated that she believed that exposure to the phenoxy herbicides reduced resistance to infection, based on the fact that these patients had a very high record of pheumonia, colds, boils and bronchitis. On March 7, 1977, the Judge of the District Court ordered a halt to herbicide spraying in the Siuslaw National Forest. (Kailan, 1976; Skopill, 1977)

In 1976, Harmon Seaver's organic farm in northeastern Minnesota was sprayed with 2,4-D and 2,4,5-TP (Fenoprop) by the U.S. Forest Service. He tried to keep the heliocopters away from his farm by shooting towards them with a deer rifle, but when he was captured by the police, the U.S. Forest Service returned and sprayed his farm. All his family experienced headaches, nausea, dizziness and diarrhea. He was indicted for assault. However, the jury acquitted him, agreeing that he had a right to protect his family from poisoning. (Seaver, 1976; 1977)

The Experience of Vietnam

Early reports out of Vietnam, including those by the U.S. National Academy of Science and the American Association for the Advancement of Science, were of limited use because of the existence of the war. The commissions were unable to conduct direct interviews and to gather on-the-spot data from local medical records. The first firm reports were interviews with refugees from the Central Highlands where there had been considerable herbicide spraying.

Three doctors at the Department of Medicine, University of Kuopio (Finland) report similar symptoms in a farmer exposed to MCPA, one of the phenoxy herbicides. They attribute the illnesses to chemical substances which contain the halogenated benzene ring. (Palva et al, 1975)

Accidental Sprayings

Similar illnesses have been noted in a number of cases of accidental spraying of human beings. We will cite three examples.

In June 1969 the U.S. Forest Service accidentally sprayed a number of people while applying phenoxy herbicides in the Tonto National Forest, near Globe, Arizona. One of these was Mrs. Billie Shoecraft. She experienced swollen arms with open sores, her hair fell out, and she reported the usual "Flu symptoms." Others sprayed in the area reported similar illnesses. (Shoecraft, 1971)

One of the best documented cases involved the spraying of 2,4-D and 2,4,5-T in the Siuslaw National Forest. There are a number of small farms in the National Forest. In 1976, the Citizens Against Toxic Sprays, the Oregon Environment Council and Hoedad Inc. went to court to stop the spraying. Hoedad, Inc. represented people who did contract forest work.

Included in the plaintiff's case was testimony by Dr. Eloise W. Kailan. Dr. Kailan reported on the effects of herbicide spraying on 11 of her patients, nine of whom were female. Seven of the eleven reported severe headaches from breathing the "red smoke" from burning areas which had been treated with herbicides. Eight patients reported irritation of eyes, nose and chest following spraying and exposure to smoke from burn areas treated with phenoxy herbicides.

Electromyographic study showed denervation in several peripheral muscles. Three years later she still had trouble walking.

(3) The third case cited was that of a 65 year old farmer who had wetted his trouser legs while spraying a cornfield with 2,4-D. He suffered from pain in the legs and twitching of muscles for five months. He also reported headaches, nausea, vomiting, and eventual twitching in all skeletal muscles. One year later he reported that he still had constant pain in the upper and lower extremities. (Goldstein, Jones and Brown, 1959)

In 1962 a group of doctors at Budapest, Hungary conducted experiments to test the effect of 2,4-D on the central nervous system. The test were performed on rats, dogs and cats; the treated animals all experienced damage to the higher nervous system. The researchers concluded that "according to conditioned reflex experiments, the higher nervous activity suffered severe damage." Caution and protection in the use of 2,4-D, plus special neruological examinations of all workmen applying the herbicide, was recommended. (Dest et al, 1962)

The Mrak Commission in 1969 reported a similar case involving children playing in a yard which had been sprayed with 2,4-D. During the first few days their eyes, mouths and lips became swollen. Several days later the children experienced incontinence (inability to control discharge of urine) and renal (kidney) damage. They also experienced "fatigue, nausea, vomiting, anorexia (loss of appetite), diarrhea, swelling and aching of the extremities." These symptoms progressed until pain, paresthesias (general feeling of illness) and limb paralysis were severe. Disability was protracted and recovery incomplete even after several years." (Shoecraft, 1971)

D.4600

Billie Shoecraft, who spent 10 years digging into all the studies done on phenoxy herbicides, reported that as early as 1948 "2,4-D was known to produce liver and kidney damage, heart attacks, severe destruction of the central nervous system, genetic changes, reduction in potency, hemmorages, paralysis, personality changes and extreme mental disturbances." (Shoecraft, 1971)

In 1953 Drill and Hiratzka reported that 2,4-D caused leukopenia (decreased production of new blood cells). In 1957 Sellman reported ataxia (an inability to co-ordinate voluntary muscle movements) and coma (a state of profound unconsciousness caused by disease, injury or poison).

The most widely cited study of acute and subacute effects on humans is that done by a group of doctors at the Mayo Clinic. They reported on three cases of 2,4-D poisoning:

(1) A 52 year old farmer spilled 60 cc. of 10% 2,4-D on his forearms. He experienced fatigue, nausea and vomiting. Two months later he accidentally wetted his legs with 2,4-D while spraying, and experienced the same effects along with diarrhea. This was followed by pain and numbness in the extremities, and six weeks later, inability to walk. Two years later he still complained of numbness in feet and fingers and the inability to move his toes and aching feet.

(2) A 50 year old housewife wet her hands and legs with a household 2,4-D preparation while spraying dandelions on her lawn. At the first exposure she experienced swelling and aching of feet and legs which lasted for around three weeks. One year later, while again spraying dandelions, she was similarly exposed. She became agitated and depressed, experienced weakness, rashes, numbness in the extremities, and the inability to walk.

VI

ACUTE AND SUBACUTE ILLNESS CAUSED BY PHENOXY HERBICIDES

So far, we have only looked at severe chronic problems caused by the widespread use of the phenoxy herbicides: the extent to which they are teratogens, carcinogens and mutagens. But there are also dangers in ordinary use of these herbicides which are known to produce illnesses that varys according to exposure and physical health.

Since the phenoxy herbicides are not extremely toxic in the acute sense of immediate poisoning, for a long period of time they were assumed to be safe for general use. The lethal dose for a human is said to be around 80 mg/kg, or for the average-sized person, around six grams. This is slightly more than a teaspoon of pure 2,4-D.

There is no question but that human beings are far more sensitive to 2,4-D than are laboratory mice and rats. Dogs and monkeys have sensitivity rates that are closer to humans. In one study done in 1963, tests of 2,4-D on monkeys revealed the same symptoms of acute illness that have been widely reported in humans. (Seabury, 1963)

Early Evidence of Illnesses from 2,4-D

It has been known for quite some time that 2,4-D produces ill effects in animals and human beings. It 1946 Bucher reported myotonia (muscle spasms), motor disorders, paralysis in the arms and legs, and gastrointestinal symptoms such as vomiting and diarrhea in experimental animals. Hill and Carlisle reported similar findings in 1947.

D-4603

TCDD is one of the most toxic chemicals known to man. It persists in the environment for long period of time and has entered the food chain. There is some evidence that it biocumulates.

When heated (as in burning or cooking) all of the polychlorophenols, including 2,4-D, produce increased amounts of the various dioxins. This has proven to be a hazard to people fighting brush and forest fires in treated areas, and if there are residues could cause a problem when cooking foods containing any residues of 2,4,5-TP.

Hexa-dioxin, which has been identified in 2,4-D by the U.S. Department of Agriculture, is very toxic. It was the cause of the "chick edema factor" in the United States that killed millions of chickens. It is also a teratogen and produces chloracne in tests on laboratory animals.

The highly toxic nature of TCDD and HCDD at minute amounts (the microgram level) reveal the necessity of testing and monitoring for them in the environment at the parts per trillion level.

In rejecting the bid by Dow Chemical Company to re-register 2,4,5-T, the U.S. Environmental Protection Agency cited as one reason the fact that the herbicide was known to contain hexa- and hepta-dioxin isomers "which are known to be teratogenic." (U.S. EPA, 1974)

Chickens were also tested for the "chick edema factor". The pericardial fluid was "markedly increased" when HCDD was administered in the range of .01 to .1 mg/kg per day. The general conclusion of the Dow researchers was that "hexachlorodibenzo-p-dioxin is highly toxic but less toxic than tetra-dioxin." (Schwetz et al, 1973)

These tests for HCCD, and the others cited for TCDD, reveal the major shortcoming of the study of dioxins done by the researchers for the U.S. Department of Agriculture (Woolson et al, 1972). In the USDA test, levels below .5 ppm were considered "non-detectable", and therefore of no significance. We know that both TCDD and HCDD are very toxic at ranges far below that level. For this reason monitoring for pesticides must be done at the parts per trillion level to be of any real significance.

Summary

The various dioxins have been found in all chlorophenols, including the phenoxy herbicides. The most common dioxin found in 2,4-D is 2,7-Dichlorodibenzo-p-dioxin, which is considered to be only slightly toxic. However, the other higher isomer dioxins, hexa-, hepta-, and octa-, have also been isolated in commercial samples of 2,4-D.

The most toxic dioxin, tetra-dioxin (TCDD), is an inevitable contaminant of 2,4,5-T and 2,4,5-TP (Fenoprop). TCDD should not appear in carefully controlled commercial 2,4-D.

During the search for the cause of the "chick edema factor", new information emerged on the dioxins other than TCDD. Firestone found that hexa- and hepta-dioxins are deposited as residues in the tissues and organs of rats and chickens, mainly in the liver. (Firestone, 1970)

Crossland and Shea report that during this period HCDD was tested on a variety of laboratory animals and "adverse effects were found in monkeys, guineau pigs, rats and pigs." Because of the buildup of HCDD in the tissue of chickens, in 1961 the U.S. Food and Drug Administration ruled that commercial fats had to be screened for HCDD before being placed on the market. (Crossland & Shea, 1973)

The highly toxic nature of HCDD was demonstrated in the tests carried out by the research team from Dow Chemical Corporation. The tests were done on Sprague-Dawley rats, the strain which has shown the most resistance to the toxicity of phenoxy herbicides, and chickens. Four rats were used in the test for acute lethality, and one of two died when given an oral dose of 100 mg/kg per day. The two which received a dose of 10 mg/kg per day did not die, but all four tested lost body weight.

HCDD was also tested by applying small amounts to the eye and ear of a rabbit. After 27 days, the eyelid of the rabbit became encrusted. HCDD also caused chloracne in the rabbit ear.

The rats were fed HCDD at a rate of between .01 and .1 mg/kg per day; all dosages were "highly lethal to fetuses during late gestation." At the highest dosage, 79% died. The weight and length of the surviving fetuses were significantly decreased.

At the dosage of .1 mg/kg, the surviving fetuses showed fetal soft tissue and skeletal anomalies. "The incidence of cleft palate, subcutaneous edema, vertebrae with split or unfused centra, and split sternbral was significantly greater than among control litters or the control fetal population." (Schwetz et al, 1973)

D-4606

No traces were found in the liver. (Zitko and Choi, 1972)

The most widely cited study of octa- and hexa-dioxins is that done by a group of researchers employed by Dow Chemical Company. Octa-dioxin was fed to Sprague-Dawley rats and Swiss Webster mice. At the level of 500 mg/kg per day, "the incidence of subcutaneous edema was significantly increased among the fetal population." Among the litters, 50% of the treated experienced the same effect compared to 21% for the control group. OCDD also caused "embryotoxicity" at the level of 500 mg/kg per day. Hepatic lesions were also observed. (Schwetz et al, 1973)

The highly toxic nature of hexa-dioxin (HCDD) was discovered in the research that followed the deaths of millions of commercially raised chickens in the United States in 1957. The chickens affected had droopy and ruffled feathers and had difficulty breathing. Autopsies showed fluid accumulation surrounding the heart, beneath the skin, and in the abdominal cavity. There was also liver and kidney damage. Until the source of the epidemic was found (it lasted into the 1960's), it was referred to as the "chick edema factor." (Crossland and Shea, 1973)

The toxic substance that caused the deaths of the chickens was finally isolated in fats added to commercial poultry feed. Chlorine was found and was at first suspected. Later it was discovered that the cause was hexachlordibenzo-p-dioxin (HCDD). It was believed that the dioxin came from 2,4-D used on corn from which vegetable oil was processed. Others theorized that HCDD could have been formed as condensation from other chlorophenols when the oilseeds were heated. In either cause, the source was the chlorophenols. (Crossland and Shea, 1973)

After two years, the amount of dioxin was significantly increased. (Shoecraft, 1971; Doucet, 1977)

Thomas Whiteside recounts how the U.S. government disposed of the twenty-five thousand steel drums full of Agent Orange left over at the end of the Vietnam war. Because they had been sitting around in the sun for a long time, the level of TCDD in the material was very high. As these drums began to rust and leak, state officials in Mississippi demanded that they be removed because of the hazard to the environment and human health. Both California and Oregon refused to permit the burial of the herbicide in their states at disposal sites.

Eventually, the U.S. Air Force contracted to have the herbicide destroyed at sea. The Vulcanus, a German-built ship owned by a company based in Singapore, took the barrels to Johnston Island. In April 1976, the Vulcanus took the material out to the middle of the Pacific Ocean, where it was burned at temperature over 700 degrees centigrade. TCDD is chemically stable up to that temperature. The drums were to be crushed and smelted at steel mills, where temperatures were said to be high enough to destroy the tetra-dioxin. (Whiteside, 1977)

Hexa- and Octa-Dioxin

Octa-dioxin, which is found in the phenoxy herbicides and other chlorophenal products, appears to be far less toxic than TCDD. In one study done for National Health and Welfare, rats were fed extracts of octa-dioxin. Residues were detected in the faeces, liver and adipose tissue. Between 1% and 3% of the total intake was found in the gastrointestinal tracts of the treated animals.

Independent studies had demonstrated that five different chlorophenols, including 2,4-Dichlorophenol, the precursur and major breakdown product of 2,4-D, formed dioxins, including TCDD, when decomposed by heat. (U.S. EPA, 1974)

Other data by Professors Baughman and O'Keefe of Harvard University was presented in the case of C.A.T.S. v. U.S. Department of Agriculture in Oregon in 1976-7. In an experiment Prof. Baughman produced .1% TCDD by exposing sodium salts of 2,4,5-T to temperatures of 300-400 degrees centigrade at normal atmospheric pressure. The level of dioxin generated in burning was greater than that allowed in commercial 2,4,5-T. The conclusion was that large amounts of dioxins would be formed by fires in 2,4,5-T treated areas, with the distinct possibility that the dioxin would be distributed by smoke particles.

Professor O'Keefe of Harvard University, who along with Professors Meselson and Baughman had done pioneer work in the detection of TCDD, reported that TCDD could be generated from 2,4,5-Trichlorophenol when it took the form of a breakdown product of 2,4,5-T in soil and plants. TCDD could also be generated from pentachlorophenoxyphenol, an intermediate breakdown product of 2,4,5-T, when heated by fire. (C.A.T.S., 1977)

C.A.T.S. also cited the work by Buu-Hoi and his colleagues. These French scientists demonstrated that TCDD was produced by heating the "purified" commercial preparations of 2,4,5-T and 2,4,5-TP (Fenoprop). (Buu-Hoi et al, 1971)

There are other reports that heating increases the amount of TCDD in commercial formulas of the phenoxy herbicides. At Phoenix, Arizona the U.S. Forest Service stored a number of barrels of 2,4,5-TP on a tarmac for two years. The level of tetra-dioxin was measured at the beginning and after two years. Sitting out in the hot summer sun in Arizona greatly increased the temperature of the Fenoprop in the barrels.

Unfortunately, they tested only down to .5 ppm. Most dioxins are present at more minute levels, and several are highly toxic at levels far below .5 ppm.

Nevertheless, the study found that one commercial sample of 2,4-D contained 10 ppm of hexa-dioxin, an extremely high level. Their sampling also found hexa, hepta and octa-dioxins in formulations of tri-, tetra-and penta-chlorophenol. (Woolston et al, 1972)

Heating the Phenoxy Herbicides

A number of studies done in the 1970's reveal that when the phenoxy herbicides are burned, the heat created by the fire intensifies the creation of the various dioxins. There are news reports from California that people fighting brush and forest fires in areas treated by the phenoxy herbicides have come down with chloracne. This was also reported by those who work in the Siuslaw National Forest. (C.A.T.S., 1977)

Much of what we know about the various dioxins was discovered during the search for the cause of the "chick edema" problem of the late 1950's and the 1960's. The Agricultural Research Service of the U.S. Department of Agriculture concluded from all the studies done that "when any chlorophenol is heated to elevated temperatures it can be converted to a dibenzodioxin." (Cowan, 1970)

In May 1974, the U.S. Environmental Protection Agency presented their pretrial brief on the question of re-registration of 2,4,5-T for general use. In rejecting the re-registration, they argued, among other things, that "there is evidence that the polychlorophenols in 2,4,5-T may decompose into dioxin (TCDD) when exposed to high temperatures, such as might occur with incineration or even in the cooking of food."

The buildup of TCDD in humans was also reported in a separate test contracted by the U.S. EPA. In 1976, they reported that breast milk samples were taken from 1400 women in 46 American states. The EPA tests showed continued presence of some of the chlorinated hydrocarbon insecticides. But it also showed TCDD residues that "exceed the acceptable daily intake levels established by the World Health Organization." A separate study done by the New York Department of Health found pesticide levels in women's milk 5 to 10 times higher than average if they had been regularly eating fish from Lake Ontario (Globe & Mail, September 29, 1977)

The Dioxins in 2,4-D

Dr. Samuel S. Epstein, who has served on the Mrak Commission and other advisory committees to the U.S. Department of Health Education and Welfare, pointed out in his testimony before Senator Philip Hart's Subcommittee in 1970 that 2,4-D and its precursor, 2,4-Dichlorophenol, have been shown to contain dioxins. Hexa, hepta and octa-dioxins have been identified in 2,4-Dichlorophenol. (Epstein, 1970)

Professor Gordon W. Gribble of Dartmouth College suggests that the most likely dioxins in 2,4-D would be 3,8-DCDD or 1,3,6,8-TCDD. Both of these would be less toxic than 2,3,7, 8-TCDD. Further chlorination of 2,4-D can produce the higher isomer dioxins. (Gribble, 1977)

The most widely cited study of dioxins is that done by the Plant Science Research Division of the U.S. Department of Agriculture in 1971. They examined 129 samples of 17 different pesticides derived from the chlorophenols.

D-4611

Where Agent Orange was used in Vietnam, measureable amounts of TCDD have persisted in the Environment several years after the final spraying. (Sterling, 1974)

Professors Meselson and Baughman of Harvard University tested fish and crustaceans caught in Vietnam for human consumption. In 1972, three years after the final herbicide spraying, residues of TCDD were found. This study demonstrated that TCDD was entering the food chain. (Baughman and Meselson, 1973; Galston, 1974)

In 1973 the U.S. Environmental Protection Agency reported a test involving beef cattle. They were fed diets containing 100 to 1800 ppm of 2,4,5-T containing .6 ppm of TCDD for 28 days. Their fat and liver retained substantial amounts of the TCDD. (U.S. EPA, 1974)

There is evidence to suggest that at least 25% of the dietary intake of tetra-dioxin may be stored in the body tissue. The Pretrial Brief of the U.S. Environmental Protection Agency on the re-registration of 2,4,5-T reports one study where rats were fed 20 ppb TCDD; 75% of the total retained residue was stored in the liver. In another study cited, 2,4,5-T containing only .04 ppm of TCDD was sprayed on rangeland and then cattle and goats fed on this area. Their fat revealed between 6 and 41 ppt of TCDD. (U.S. EPA, 1974; U.S. EPA, 1975)

Research done by Harvard Professors Meselson, Baughman and O'Keefe in 1976 found TCDD residues in samples of mother's milk taken in San Angelo, Texas and Siuslaw Forest, Oregon where there had been aerial spraying of phenoxy herbicides. They also found TCDD sample in bovine milk and fat samples. (Meselson et al, 1976)

Earlier, researchers with the U.S. Department of Agriculture warned that "the presence of such highly chlorinated compounds in the food chain is of concern because these compounds are generally fat soluble and nonbiodegradable, tending to concentrate in the food chain, and many of them are acutely toxic and even carcinogenic." (Pohland and Yang, 1972)

Early research indicated that TCDD could accumulate on bare surface soil and water. (Crosby et al, 1971) A later study revealed that when 1 ppm of tetra-dioxin was applied to two types of soils, after 350 days 54% of the original TCDD remained. At a higher dosage (100 ppm) 71% was recovered after 350 days. (Kearney et al, 1973) Professor Streisinger comments that "the fate of the 50% that 'disappears' is not known and the possibility of transformation into other toxic substances needs to be investigated." (Streisinger, 1976)

In one laboratory study, rats were administered TCDD five times a week over a period of 13 weeks; the amounts that were measured in the liver and fat appeared to increase steadily up to 7 weeks, when measurements were stopped. (Rose et al, 1976)

The U.S. Airforce carried out some tests of its own on various species of rodents and birds in a testing area which had been sprayed with the phenoxy herbicides. These species contained residues of TCDD which were higher than the levels present in the environment. (Streisinger, 1976)

Dr. Theodore Sterling of Simon Fraser University described the dangers of TCDD in the environment in his brief presented to the B.C. Royal Commission Inquiry into the Use of Pesticides and Herbicides (1975). He reported that studies show that TCDD "clearly is persistent and biocumulative. In general, it resists microbiological deterioration."

At the same trial, Professor George Streisinger argued that the U.S. Forest Service was "negligent in failing to emphasize the possible danger from mutagenic effects of the herbicides, especially in light of the evidence from the tests that have been performed in this area." They also failed "to indicate that herbicides that are mutagenic are likely to be carcinogenic as well: it has been shown that a large majority of the chemicals tested that are mutagenic are also carcinogenic." Similar warning came from Dr. C.D. Dewse in an article in the British medical journal, The Laucet. (Dewse, 1976)

Professor Arthur H. Westing, a member of the AAAS Commission that studied the effects of phenoxy herbicides in Vietnam states: "until new developments alter the current situation the use of TCDD dioxin-contaminated herbicides presents an unacceptable risk to man and his environment." (Skopill, 1977)

Persistence of TCDD in the Environment

Evidence to date indicates that the phenoxy herbicides and their dioxin contaminants may pose a threat to the environment as great as the chlorinated hydrocarbon insecticides. There is evidence that the dioxins persist for a long time and that they biocumulate.

The U.S. Environmental Protection Agency took samples of animal tissues in the Siuslaw National Forest in 1973 and 1974 and they found TCDD residues, presumably from spraying of 2,4, 5-T and/or 2,4,5-TP (Fenoprop). This was confirmed by subsequent tests conducted by Dr. P. O'Keefe of Harvard University.

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Allen and Norback reported in 1973 that TCDD fed to rhesus monkeys resulted in changes in the kinds and growth patterns of cells in the stomach, and changes in the kinds of cells and secretion of the oils in the skin which formed chloracne. (Skopill, 1977)

In more recent tests, eight female monkeys were fed a daily dose of 500 ppt of TCDD. All developed anemia and five of the eight died. They had an almost complete lack of red and white blood cells.

In another experiment, Allen fed rats 5 ppt of TCDD daily for 18 months. Of the 60 treated animals, 23 developed cancerous tumours. Dr. Allen and his colleagues concluded that "the possibility that dioxin (TCDD) is a potent promoter of cancer rather than an inducer cannot be overlooked." (Whiteside, 1977; Cox, 1977)

Virgil C. Boekelheide, Professor of Organic Chemicstry at the University of Oregon, has stated that

> TCDD is fantastically toxic and is commonly quoted as being the most toxic simple organic molecule known to man . . . At sublethal dose levels it still has highly toxic effects on thymus, liver and other organs, as well as being extremely teratogenic. Thus, minute quantities of TCDD well below the acute lethal dose level have adverse toxic effects and a threshold limit, below which no toxic effects occur, has never been demonstrated for TCDD. (Skopill, 1977)

The Chairman of the Laboratory of Pathology at the Oregon Regional Primate Research Center, Dr. Wilbur P. McNulty, Jr., has done research on the effects of TCDD on rhesus monkeys. He suspended his experiments after he discovered how extremely toxic TCDD was to primates. In the C.A.T.S. case against 2,4,5-T in Oregon, he testified that "it is my opinion that the deliberate environmental distribution of TCDD or products known to contain TCDD at any level causes a serious threat to human and animal health."

However, those most often identified in the phenoxy herbicides are tetra-dioxin (four chlorine atoms), hexa-dioxin (six chlorine atoms) hepta-dioxin (7 chlorine atoms) and octa-dioxin (eight chlorine atoms). (Epstein, 1970)

The Extreme Toxicity of Tetra-Dioxin

Arthur W. Galston, Professor of Botany at Yale University, describes TCDD as "one of the most poisonous chemicals ever synthesized by man." The Science Policy Division of the U.S. Library of Congress reports that 6 milligrams (or one drop) of TCDD is enough to kill 1200 human beings. (U.S. Library of Congress, 1974)

The acute oral dose (LD_{50}) for male guinea pigs has been established in a laboratory tests at .005 mg/kg, and in male and female rats at .0225 mg/kg and .045 mg/kg respectively. (Epstein, 1972) Pregnant hamsters were fed .009 mg/kg tetra-dioxin in one laboratory study; 82% of the fetuses were killed, and of those born, 82% were deformed. For rabbits, a dose of .01 mg/kg killed all the animals; a lower dose of .001 mg/kg resulted in chloracne and liver damage. In a study by Dow Chemical Company, an application of .0003 mg on the ear of a rabbit produced chloracne. Dow researcher L.C. Silverstein reports that less than .01 mg on a surface wipe sample creates an acute hazard to man. (Crossland and Shea, 1973)

The most widely publicized studies in recent years are those done by Dr. James R. Allen and his colleagues of the University of Wisconsin Medical School. In 1967 Allen and Carstens reported that repeated feedings of low levels of TCDD to monkeys resulted in their deaths over periods of more than 445 days. The lowest amounts of TCDD tested were lethal.

D.4616

This process yields 2,4,5-Trichlorophenol and tetra-dioxin. The latter is considered an unwanted pollutant but occurrs inevitably in the process. As the temperature rises, more tetra-dioxin is formed. 2,4,5-Trichlorophenol is then treated with sodium chloroacetate and an acid to yield 2,4,5-T. (Gribble, 1974)

In theory, TCDD should not be present in 2,4-D. The chemical phenol is chlorinated at temperature between 80 and 100 degrees contigrade. At these relatively low temperatures, it is argued, TCDD should not be formed. The Plant Products Division of Agriculture Canada wrote in February 1977 that they had analyzed the Aqua-Kleen formulation of 2,4-D for TCDD down to a level of 5 ppb and found no dioxins. However, they also noted that "the laboratories are just now developing the capacility to analyse for the other dioxins ..." (Agriculture Canada, 1977) (Advisory Committee, March 25, 1977)

In a pamphlet released by the B.C. Department of the Environment in July 1977, it was claimed that "TCDD is not produced during the manufacture of 2,4-D." No mention was made of any other dioxins. (B.C. Department of the Environment, 1977) Dow Chemical Corporation , while admitting that commercial preparations of 2,4,5-T and 2,4,5-TP (Fenoprop) contain TCDD, states that "there are no dioxin contaminants in the 2,4-D materials which we sell." (Dow Chemical, 1973)

However, the chlorophenols also produced a number of other dioxins. They differ according to the number of chlorine atoms that are attached to the two benzene rings (see Figure 2). There are eight positions where chlorine atoms can be attached to the basic dibenzo-dioxin structure; thus, depending on the orientation of the molecule, there is a possibility of 60 different dioxins.

D-4617

V

DIOXINS IN PHENOXY HERBICIDES

On July 10, 1977 an industrial accident at the Sevesco, Italy plant of Hoffmann-LaRoche released a deadly cloud of tetra-dioxin (TCDD) over the local valley. The news of the horrible disaster spread around the world, even warranting an article in Maclean's Magazine, July 25, 1977. The regional health officer in Italy called it "our own little Hiroshima", and once again the world began to worry about the phenoxy herbicides.

The Sevesco plant produced trichlorophenol, the chemical substance from which 2,4,5-T is synthesized. Since the Vietnam war, there has been increasing concern over the toxic nature of the various dioxins, and in particular tetra-dioxin (TCDD). While the dangers of the dioxins had disappeared from the headlines following the end of the war in Vietnam, they have come to public attention following recent encironmental accidents involving polychlorinated biphenyl (PCB), pentachlorophenal (PCP) and now, once again, 2,4,5-T. (Whiteside, 1977)

What are the Dioxins?

The Council for Agricultural Science and Technology (CAST), one of the main public relations organizations for the use of pesticides, has pointed out that "a highly poisonous kind of dioxin called TCDD is an unavoidable contaminant in commercial supplies of 2,4,5-T and Silvex (2,4,5-TP)". (C.A.S.T., 1975)

In the manufacture of 2,4,5-T, the chemicals 1,2,4,5-Tetrachlorobenzene and sodium hydroxide in methanol are heated at around 160 degrees centigade.

Populations of Cardinals, Jays, Tanagers and Bewick's Wrens were eliminated entirely. Ducks, geese and chickens being raised in the area failed to hatch any young. The one gosling which did hatch, developed pneumonia, was paralyzed and spastic and finally died. Major infestations of gall insects, brown beetles and other species followed the applications (Shoecraft, 1971). Other residents in the area reported unusual sightings such as quail, a fox, rabbits and dogs all milling around in the same area without paying any attention to each other. Normal instincts it appears were suppressed. Much dead wildlife was reported in and around the spray area. These included several species of birds, skunks, fox and bobcats (Shoecraft, 1971).

2,4-D And Agriculture

2,4-D is being used extensively in agriculture in both Canada and the U.S. With labour and energy costs rising rapidly, it is not unusual to find that farmers are substituting the extensive use of herbicides for these two factors of production. With 2,4,5-T restricted from agricultural use, 2,4-D becomes the least cost acceptable alternative in much of Canada.

We still know very little about the long term effects of the continued use of herbicides on food crops. With substantial amounts of 2,4-D being applied in the lake systems, it is likely that some of the chemical will find its way into orrigation systems. It is well known that grapes are particularly susceptible to herbicide damage. (Pimental, 1971) The B.C. Government's Advisory Committee on weed control was concerned about this problem and recommended that tests be conducted on grape seedlings during the 1977 programme (B.C. Advisory Committee, March 25, 1977).

D-4619

It was reported that the damage to vinyards in eastern Washington became severe during the period from 1970 to 1974 due to the herbicide concentrations in the atmosphere. Experimentation has shown that "a dosage of 1ppb 2,4-D in air during a 6 hour period each week for a total of ten applications can cause a yield reduction in grapes of 50%. Furthermore, Concord grapes can be severely malformed from the application of 0.1 ng of 2,4-D to the leaves" (Farwell, et. al., 1976). In an effort to reduce the damage to the grape crops, even the low volatile 2,4-D esters (such as Butoxyethanol) have been restricted from use for the period from May 16 to October 31 each year in eastern Washington and Oregon.

The effect of various dosages of herbicides on most crops is still not well understood even though some test results may be available. Fruit tree flowering and fruit production abnormalities have been reported following spray drift in both Arizona and Vietnam. In the latter case, trees produced no flowers or fruit for at least three years after contamination. Following an examination of damage done to jack fruit, manioc and guava trees as well as field vegetables, it was reported that the Vietnamese Ministry of Agriculture found the destruction so great "that adequate financial compensation to the owners of damaged trees would probably be impossible" (Shoecraft, 1971; Orians & Pfeiffer, 1970).

On the Robbin Warren farm near Granville Island, Nova Scotia, market garden crops showed highly abnormal growth patterns following herbicide spray drift contact. Corn yields dropped drastically and celery and cauliflower bolted to four and six feet high. The government later compensated Mr. Warren for this damage (Ogden, 1971).

Kimmins discusses the effects of herbicides on flowering crops. He points out that "there can be a reduction or alternation in the number, size and shape of flower parts, and there may be a differential effect on organs of different sex. There can be a multiplication or reduction in flower number and earlier flowering can be stimulated. The resulting fruit may be larger due to increase in cell volume, but the shape of the fruit may be aberrant if the concentration of herbicide within the fruit is uneven" (Kimmins, 1975).

Quite obviously, a substantial amount of damage could be done to fruit and vegetable crops should the chemical get into the irrigation systems of the valley during regular applications or through human error. There appears to be no information available on the effect of small amounts of herbicide on the growth patterns of dward fruit trees. It would be unfortunate if the dwarf orchards which have been planted extensively in the Okanagan Valley grew to standard size.

Research is just beginning into the genetic effects of repeated 2,4-D applications near grain crops. Herbicide use on the prairies is skyrocketing with the institution of the "zero tillage" concept. This method involved the elimination of plowing through an early spring herbicide treatment of new vegetation. The seed is then drilled directly into the sod. This method is thought to conserve soil moisture and topsoil while resulting in reduced costs by the elimination of plowing (McCalla, et. al., 1962).

Genetic alterations have been observed, however, which may have serious implications for crop yields and quality in areas of continuous hebicide use. As early as 1951, it was discovered that barley and wheat plants showed abnormal cell division after being sprayed with 2,4-D at various stages of growth.

This effect persisted for a considerable length of time after the applications (Unrau, 1953, 1954; Unrau & Larter, 1951). Chromosomal defects in herbicide treated peas were reported following testing (Hyhling, et. al., 1960). As genetic defects are carried from one generation to the next, damage done to crops through successive herbicide applications could last indefinitely.

The zero tillage approach to cultivation could also have an effect on seed germination rates as herbicide residues build up in the soil and atmosphere. During the testing of the effects of the vapors of various 2,4-D formulations by Dow Chemical's Biochemical Research Laboratory, it was discovered that the seed germination rate was decreased significantly by the highly volitile esters. The methyl, ethyl and isopropyl esters caused a complete germination failure of turnip, squash, radish and muskmellon seeds. The methyl and ethyl esters produced success rates of 22% and 41% for wheat while the control group rate was 92%. For rye, 40% of the controls sprouted while none germinated after exposure to these same two esters. The following effects of 2,4-D were observed during the experiments:

> "a) the embryonic plants failed to break through the seed coats; b) the seedlings emerged but failed to grow; this was usually accompanied by abnormal swellings on the seedlings, and in this case the roots often did not develop properly; c) leaf modification of the young leaves after emergence; leaves showed typical malformations such as are caused by 2,4-D when applied as a spray; d) stunting of seedlings and a slower germination of the seeds; this might or might not be accompanied by leaf modification" (Mullison & Hummer, 1949).

Swelling of the stem sections (callus growth) was also reported in a study done on 2,4-D treated bean plants (Bach & Fellig, 1961).

There is certainly some evidence to indicate that seed germination rates and early seedling development may be affected when seeds come into contact with this herbicide.

Dr. A.B. Carvers of the University of Ontario reports that some weed species are becoming resistant to herbicides (Doucet, 1973). This is a classic pesticide response. Often, while the target species develops an immunity to a chemical, other nontarget species which may be predators or competitors are sharply reduced in numbers. The result is an explosion of the pest population which cannot be stopped by the use of chemicals.

Poisoning of cattle and other grazing animals has been associated with herbicide applications in British Columbia, Nova Scotia, Arizona, New Mexico and Vietnam. These incidents have resulted from either the acute toxicity of the chemical or from the high nitrate levels in plants after herbicide treatment.

Until recently, it was assumed that the lethal dose of 2,4-D for cattle was between 500 and 2,000 mg/kg of body weight. Following an accident in Australia, this level should be revised downward to 150-188 mg/kg for a fatal dose and 105-132 mg/kg for a toxic dose. Of the two cows poisoned, one died within 12 hours while the other exhibited signs of depression, loss of appetite and muscle weakness which resulted in an inability to stand (McLennan, 1974).

It is difficult to determine if it is possible for grazing animal poisoning to take place as a result of normal applications. Studies have shown that forage in areas just treated would contain about 100 to 150 ppm for each pound of herbicide applied per acre. It would seem, however, that while fatal poisoning is not likely, except in cases of accident, other problems may develop. In tests conducted on sheep and cattle which had been fed 2,4-D in concentrations ranging from 300 to 2,000 ppm residues were found in their internal organs as well as fat and muscle.

The most substantial amounts were located in the kidneys (Clark, et. al., 1975) Beside the possibility of outright poisoning, grazing animals could contain enough herbicide residues so as to complicate marketing.

An increased nitrate level in treated forage can combine with a generally increased palatability to present a problem for livestock and wildlife. Exactly why some plants become preferred by animals after spraying is still somewhat of a mystery although increased protein and mineral content may play a part (Kimmins, 1975; Maxwell & Harwood, 1960). Herbicide applications are known to raise the hydrocyanic acid levels of sudan grass and cherry leaves (Kimmins, 1975). It is not surprising then to find that on Robin Warren's Nova Scotia farm, where herbicide spray drift occurred, the cyanide levels were relatively high. An analysis of damaged brussel sprouts showed 52 ppm of cyanide, while butchered cow meat had 52-56 ppm and an aborted calf's liver showed 5 ppm of the poison (Ogden, 1971). Again, marketing problems would develop for this produce. There are varying theories as to how long livestock should be kept off treated range. They range from one week to two years, depending on the herbicide used and the climate.

As mentioned earlier, herbicide applications have resulted in insect population explosions and consequent damage to trees and plants. It is also thought that the chemicals damage the defense mechanism which have been established by plants against insect attack. In tests conducted on corn which had been sprayed with 2,4-D, the treated group showed 1,679 aphids per unit of plant mass, while the untreated cornstalks had 618 aphids. The sprayed group had a 28% infestation rate of corn borers versus 16% for the unsprayed. Borers in the herbicide treated corn were larger and produced larger eggs (Oka & Pimentel, 1976).

In another experiment, pea aphids were placed in a cage with broad bean plants. Population increases between the treated (with 2,4-D, dimethyl amine) and untreated plants ran as high as 450% and averaged 81.9% (Maxwell & Harwood, 1960).

Other reports indicate that aphid populations in New Brunswick exploded after 2,4-D applications. It was thought there that the predator ladybird larvae were killed and deformed, and pupation was delayed (Rudd, 1964).

Beside insect damage to crops, some plant pathogens are known to increase after 2,4-D applications. Oka and Pimentel noticed an increase in the incidence of southern corn leaf blight during their tests (1976). Another major study on interactions between herbicides and plant pathogens indicated that fungi on tomatoes and wheat, viruses on beans, tobacco, cucumber and cotton and nematodes on oats are all increased by applications of 2,4-D (Katan & Eshel, 1973). It is quite clear that the use of herbicides on or near crops can have a definite effect on overall plant health and therefore, on yield and quality.

Summary

As a biocide, 2,4-D acts to kill members of the broad-leafed plant community. However, because of the complexity of the ecosystem in which it operates, the effects of this chemical can be wide ranging involving many nontarget species. Fish populations can be reduced both directly through poisoning and indirectly by the reduction of habitat and increasing susceptibility to disease and predation. Birds as well risk damage to their health and reduced breeding viability. Other forms of wildlife have been damaged when herbicide applications destroyed homes and contaminated their food supplies.

2,4-D has been used on or near food crops for many years. Grapes are known to be particularly susceptible to the herbicde damage and fruit trees can experience flowering problems. Genetic damage has been noted after exposure to this chemical of several plant species. Germination rates can be seriously suppressed and resistance is developed by some weed species. Both herbicide and associated nitrate poisoning of grazing animals has been documented. Insect pests have been shown to increase in 2,4-D treated crops and some plant diseases may be more prevalent. In general, 2,4-D reduces the overall health of the environment to which it is applied.

VIII

THE PERSISTENCE OF 2,4-D IN THE ENVIRONMENT

Considerable concern has been expressed over what happens to 2,4-D when it is put into a lake system. We know that the provincial government has announced plans for a five year programme for an "all-out attack" on Eurasian water milfoil weed. If we look at the experience of the TVA, then there is a good possibility that once the provincial government becomes dependent on the use of herbicides for control of milfoil, it will continue the programme indefinitely.

The other major concern is the fact that the phenoxy herbicides are based on benzene and chlorine, and other pesticides and chemical products with these two substances as their base persist in the environment for a long time. Many are biocumulative.

The Position of the Provincial Government

In 1977 spokesmen for the government of British Columbia and the City of Penticton repeatedly stated that 2,4-D breaks down into harmless products in a matter of four or five days. Skaha Lake beach was closed on June 23, 1977 following the application of 2,4-D in the swimming area; but it was reopened the July 1st weekend in order to accomodate the influx of tourists. At that time the public was told that tests showed that there were "no detectable levels" of the herbicide in the beach water.

The three Interim Reports of the Advisory Committee on the Control of Eurasian Water Milfoil say very little about what happens to 2,4-D when it is put into the lake system. The Second Interim Report (p. 9) states that 2,4-D will be broken down by micro-organisms in lake bottom mud between 35 and 65 days, the rapidity of the breakdown depending on whether or not the area has been previously treated with 2,4-D."

The report also concludes that the milfoil tissue will absorb and "metabolize" 2,4-D. (Second Interim Report, March 25, 1977)

The Third Interim Report of the Advisory Committee was issued after public meetings were held in the larger population centres of the Okanagan Valley. At these meetings, widespread opposition to the use of 2,4-D was expressed by local residents.

The final report of the Advisory Committee claimed that "the level of 2,4-D in potable water would be around zero, but no higher than 0.0002 ppm when water is drawn from Okanagan Lake for the first few days after herbicide application." (Third Interim Report, May 25, 1977)

At the public meetings, the members of the Advisory Committee reiterated the recommendations of their reports: (1) no herbicides should be used within 500 metres of any water intakes, and (2) mechanical methods of control should be used in public recreation areas. The public was assured that these two precautions would preclude the possibility of human exposure to the herbicide.

Persistence in Water

Under ideal conditions, 2,4-D is broken down by chemical and biological action. Exposure to sunlight increases the degradation process. However, where actual field tests have been monitored, it appears that all the phenoxy herbicides can persist in the environment for quite some time.

In the Tennesse Valley Authority water system, 2,4-D has been used to control Eurasian water milfoil weed since 1961. In their Environmental Statement, they record average levels for Guntersville Reservoir during 1969. Three months after application of the herbicide, residues were still found to average in surface water at .042 ppm. Plankton had levels at 1.1 ppm, and in the sediment it was .35 ppm.

Between April 1 and June 26, 1969 they monitored 2,4-D residues in raw water at eleven water treatment plants on Guntersville Reservoir. Five of the eleven water treatment plants recorded 2,4-D levels in the raw water at above the .1 ppm level set by the U.S. Environment Protection Agency as a maximum permissible concentration. At the North Marshall water treatment plant, 2,4-D residues in water reached the very high level of 5.9 ppm. Even after water treatment by coagulation, sedimentation, filtering and carbon treatment, 2,4-D residues were at a 5.8 ppm level. This report suggests that traditional water treatment systems do not remove 2,4-D.

The TVA concludes that 2,4-D "persists in sediments less than 1 year." However, they note that "significant concentrations of 2,4-D" were found in some sediment samples up to 10 months after treatment. (TVA, 1972)

In the most widely cited study on 2,4-D residues in the TVA system, Smith and Isom (1967) report that 2,4-D was present in sediment samples 10 months after treatment. In one test, the sediment reading was 58.8 ppm. In addition, they report that 2,4-D residues were found in milfoil weed 24 months after application, at a rate of 8 mg/l. (See also Wojatalik, 1971)

J.H. Patterson of the Canadian Wildlife Service found that application of 2,4-D to marshy areas can result in long persistence of the herbicide in outlet streams. The Alberta Conservation Authority has also suggested that wetlands could act as a herbicide sink. (Patterson, 1974) Another researcher has pointed out that repeated applications of a non-persistent pesticide makes that pesticide last in the environment as long as a single dose of a pesticide like DDT. (Holling, 1971)

Higher Risks in Water

There seems to be general agreement that there are greater dangers to using herbicides in water than on terrestrial plants or soil. The herbicides move with the water currents, carrying it far from the target area. (Mitchell, 1974) The U.S. Government, in its manual on disposal of pesticides, warns of the "intimate interconnection between ground and surface water." They report on tests conducted by the U.S. Government which found pesticide residues in 60% of the samples taken from wells. (U.S. Working Group on Pesticides, 1970)

We have found a number of cases which illustrate that the phenoxy herbicides persist far longer in the environment than many people are willing to admit. In the ground water in Colorado, phenoxy herbicides were found seven years after the last spraying. (Walker, 1961; Ogden, 1971) In 1965 the U.S. Department of Agriculture conducted a herbicide residue survey in 6 different areas in the United States, and they found 2,4-D residues in all areas. (Shoecraft, 1971)

In Globe, Arizona tests showed residues in soil as long as six years after spraying. Samples of water taken several weeks after spraying were tested by the Arizona Department of Health; they all revealed phenoxy herbicide residues. One sample, tested over one year later, still showed almost 100 ppm of 2,4,5-T. (Shoecraft, 1971)

In another well-known incident, the U.S. Forest Service sprayed the Cleveland National Forest in California with 2,4-D as part of a tree management programme. The herbicide worked its way through the soil and into the San Gabriel River. It ended up in the reservoir system at Montebello, which supplies water to Los Angeles. Five years later, 2,4-D residues were still detectable. In June 1973 it was still being found in the Morena Reservoir at a level of .27 ppb. (Shoecraft, 1971; U.S. EPA, May-June, 1973)

Recently it was discovered that an industrial plant in the Edmonton area was leaking 2,4-D into the North Saskatchewan River. Tests done by Environment Canada found 2,4-D in the river as far away as Deer Creek, Saskatchewan.

In addition, K.W. Reid, Chief of Fisheries and Environment Canada's Water Quality Branch's Western and Northern Region's office, reported that tests show "low concentrations of 2,4-D in water almost everywhere we have water even if the water is far from any agricultural areas." He concluded that if the source of this 2,4-D is water transport, "it would indicate that the herbicide is relatively resistent to total degradation." (Letter to S.O.E.C., Sept. 22, 1977)

It is well known that 2,4-D in the ester formulation is very volatile and enters the atmosphere as a pollutant.

It has been reported that in Alberta it has caused damage to crops and shelter belts. (B.C. Royal Commission, 1975) In Washington State, grapes have experienced damage from 2,4-D spray drift miles from the application. (Otta, 1974) A recent study in Saskatchewan found significant concentrations of herbicides in the air in spring when grain farmers are spraying to kill weeds. (Sask., 1977)

In 1969, the U.S. Environment Protection Agency tested for 2,4-D residues in wheat and soils in 16 different states. Where herbicides had been used, 20% of all soil samples showed 2,4-D residues. In wheat samples, 8% had residues between .001 and .004 ppm. (Gowen et al, 1976)

2,4-D Residues in Humans

There is growing evidence that 2,4-D and the other phenoxy herbicides are not entirely eliminated from the human body, as claimed by some officials. Dr. E. Stanton Maxey, Diplomat of the American Board of Surgery, reports that tests done on farm workers in Florida who had developed cancer revealed residues of 2,4-D, 2,4,5-T and 2,4,5-TP in tissues. (Maxey, 1974)

Following the spraying of Globe, Arizona by the U.S. Forest Service, Mrs. Billie Shoecraft had tests done on her own tissue. They revealed residues of 2,4-D and 2,4,5-TP. Ten years after the spraying ended Mrs. Shoecraft died of cancer. A biopsy of her tissues revealed residues of both phenoxy herbicides. (Shoecraft, 1971; Doucet , 1977)

Several people living in the Siuslaw National Forest in Oregon had their blood tested in 1972 at GHT Laboratories in Brawley, California, after their area was sprayed with phenoxy herbicides. Their blood samples revealed between .078 ppm and .11 ppm of 2,4-D and between .006 ppm and .03 ppm of 2,4,5-T. (GHT Laboratories, 1972)

The Breakdown of 2,4-D

There have been a fair number of laboratory and field tests done on the behaviour of 2,4-D in a water environment. The consensus is that (1) "water as a carrier seems to increase the volatility of 2,4-D" (Paris and Lewis, 1973) and (2) that "aquatic environments are less favourable for 2,4-D degradation." (Hirst and Bank, 1971; Patterson, 1973)

Degradation of 2,4-D takes place through several processes. Partial metabolism occurs in the plant through hydrolysis and decarboxylation, with the main breakdown product being 2,4-Dichlorophenol. However, as the plant dies the process ceases, leaving some 2,4-D and the breakdown products. The TVA notes that "a small amount of the 2,4-D absorbed or adsorbed by the watermilfoil may be released as it decays, but the levels in the water resulting from such releases would be much lower than the levels when the herbicide was applied." (TVA, 1972)

It is also reported that 2,4-D degrades through photolysis (decomposition by sunlight). The main products here are also the chlorophenols. (Plimmer and Klingebiel, 1971) Two other laboratory studies also reported that 2,4-D broke down when exposed to ultraviolet light; in both cases the primary degradation product was 2,4-Dichlorophenol. (Aly and Faust, 1964; Mitchell, 1961)

The other form of breakdown is by micro-organisms. The spokesmen for the B. C. Department of the Environment repeatedly claim that 2,4-D is quickly degraded through this process. However, existing studies cast some doubt on this claim.

In 1964, Aly and Faust demonstrated that the butyl ester of 2,4-D was biologically degraded to 2,4-D and butyl alcohol, but only the alcohol was oxidized further, not the 2,4-D.

The most widely cited study is that done by Schwartz in 1967. He found that under normal water supply conditions, 2,4-D was strongly resistant to chemical attack, and very little of the herbicide was absorbed onto suspended mineral solids. The microbial breakdown of 2,4-D was slow; over a period of between 3 and 6 months, no more than 37% disappeared. He concluded that in a natural water supply, 2,4-D would not degrade materially. (Schwartz, 1967)

A second widely cited study was done by Demarco and associates in 1967; it seems to be most relevant to the Okanagan situation. They found that the biological breakdown of 2,4-D was greatly reduced by a drop in the oxygen level of the water and the water temperature. (Demarco et al, 1967) This is consistent with the early study by Steenson and Walker (1957) on 2,4-D breakdown in soils. The latter concluded that aerobic conditions were essential for bacterial decomposition.

Hemmett and Faust (1968) also report that 2,4-D lasts for long periods of time in cold lake water in which oxygen has been depleted. They claimed that 2,4-D lasted for more than six months under such circumstances.

Furthermore, there are indications that 2,4-D may not break down at all under certain conditions. In May 1974 Robert Wicklund, a marine biologist from Hydrolab Research Habitat in the Bahamas, and Jim English, a Memorial University diving engineer, found eleven depressions in Resolute Bay in the Canadian Arctic.

These depressions were between 30 and 45 feet deep; the oxygen levels were very low and marine life was non-existent. These holes in the floor of the Artic Ocean had a high concentration of oil and 2,4-D. Wicklund and English described their existence as a "microcatastrophe," which trapped and killed fish and other slow moving organisms. (Dotto, 1974)

The Breakdown Products

One of the major shortcomings of the 2,4-D monitoring programme in the Okanagan lakes in 1977 was the failure of the B.C. Ministry of the Environment to adequately test for breakdown products of 2,4-D. The B.C. Royal Commission of Inquiry into the Use of Pesticides and Herbicides (1975) noted that for most classes of chemicals, the "degradation products may be more toxic or more persistent than the original product." This may be the case with the chlorophenoxy herbicides, including 2,4-D.

It is widely recognized that the major breakdown product of 2,4-D is 2,4-Dichlorophenol. Unfortunately, there appears to be very little research on the toxic nature of 2,4-Dichlorophenol and the other breakdown products of 2,4-D.

As noted before, the chlorophenols which are the breakdown products of the phenoxy herbicides have a greater incidence of the higher isomer dioxins, including HCDD which is known to be quite toxic. (Woolson, et. al., 1972)

The clearest information on the toxicity of 2,4-Di-chlorophenol was provided in the Mrak Commission.

Unfortunately, this material appears in Volume III, which to this date has not been published; therefore, it is available only as pirated, unpublished zerox copies. The report by the Bionetics Laboratory lists the teratogenic nature of 2,4-Dichlorophenol; their tests on mice produced "significant increase in the number of abnormal fetuses; half of the anomolies consisted of extended legs." They also found that "the fetal weights were significantly less than those of the controls." The Bionetics Research Laboratories report concluded that eight compounds studied were "hazardous", enough to be classified as "potentially dangerous, but needing further study." Among these was 2,4-Dichlorophenol. (Shoecraft, 1971 reproduces the unpublished report)

FIGURE III

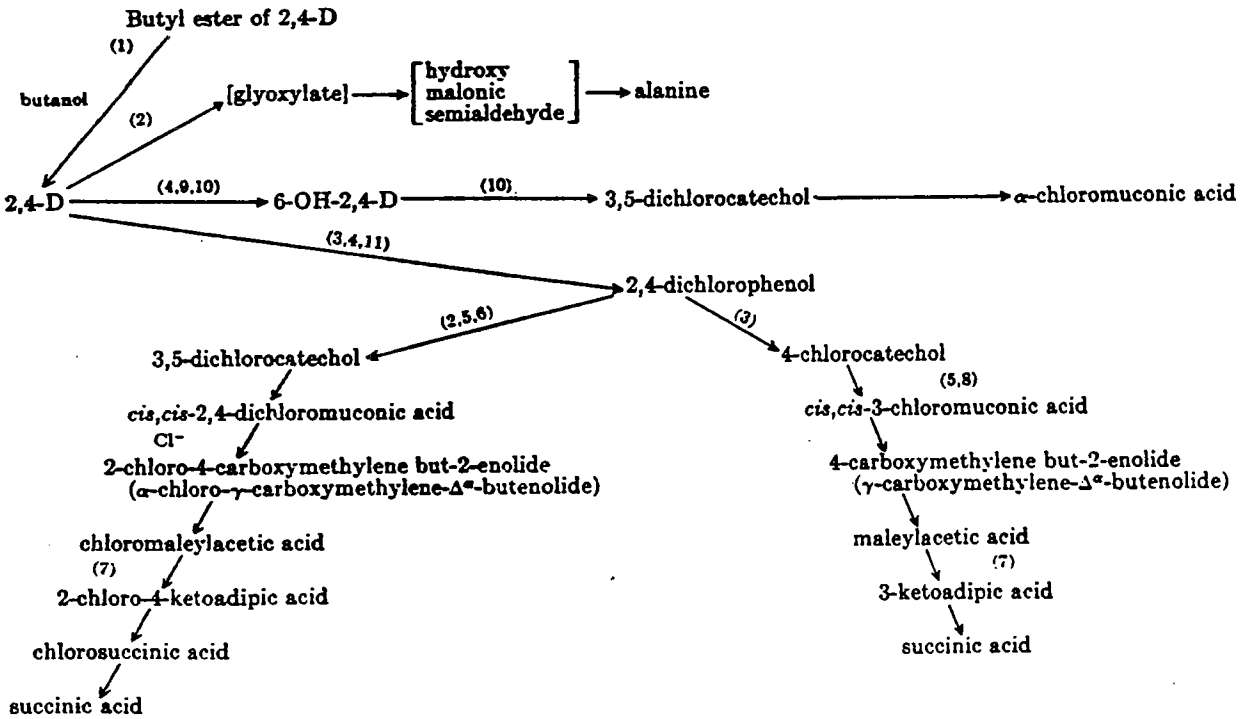

Fig. 1. Pathways of microbial degradation of butyl ester of 2,4-D. Numbers are references: (1) ALY and FAUST 1964, (2) TIEDJE and ALEXANDER 1969, (3) STEENSON and WALKER 1957, (4) BELL 1957, (5) BOLLAG *et al.* 1968, (6) LOOS *et al.* 1967, (7) DUXBURY *et al.* 1970, (8) TIEDJE *et al.* 1969, (9) AUDUS 1960, (10) FERNLEY and EVANS 1959, and (11) EVANS and SMITH 1954.

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SOURCE: Doris F. Paris and David L. Lewis, "Chemical and Microbial Degradation of Ten Selected Pesticides in Aquatic Systems, "Residue Reviews, Volume 45, 1973, p.107

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The Dangers of Succinic Acid

The final breakdown of 2,4-D as identified by Paris and Lewis (1973) is succinic Acid. (See Fig.III). Succinic acid (2,2-dimethylhydrazide) is widely used as a growth regulator on fruits and vegetables in North America, including the Okanagan, where it is known by its brand name, Alar.

The Mrak Commission reported in 1969 that hydrazine derivatives produced hepatomas in tests with laboratory mice. The free enzyme liberation of hydrazine causes tumours. (Mrak, 1969; B.C. Royal Commission, 1975)

Another study revealed that maleic hydrazide was also a mutagen. At the level of 2 mg/ml fed to laboratory mice, there were chromosome aberrations and inhibition of cell division. (Nooden, 1970; B.C. Royal Commission, 1975).

New evidence of the carcinogenic nature of the hydrazines was revealed in March 1977. Dela Toth and Anna Tompa of the Epply Institute for Research in Cancer, University of Nebraska, reported to the American Association of Pathologists on the 10-year research into the "Alar group" of chemicals.

Of the hydrazines tested, thirty-six have been shown to cause tumours of the intestines, blood vessels, lungs, liver, kidneys breast and the central and peripheral nerve tissues of laboratory animals. Some of the hydrazines are natural, found in tobacco and some edible mushrooms, but most of them are man-made.

The Omaha scientists reported that analysis of residue levels in fruit from trees that received 1,500 ppm annually for five years averaged 3.9 ppm. They concluded that "this means the population which consumes these fruits and vegetables is exposed to this hazardous agent." (Hollobon, 1977)

The Effects of Synergism

Most studies on the toxic nature of chemicals deal with the single chemical in an isolated situation. Few are tested to determine how they actually behave in the real environment. Furthermore, toxicology tests are usually conducted in relation to a certain non-target species; rarely are they tested for their effects on a complex ecosystem.

A growing concern in North America is the buildup of organic pollutants in our waters. How do these chemicals interact? In some cases, different chemicals combine to form new, toxic substances. In other instances two or more chemicals co-operate to increase the total effect of the chemical. This is commonly referred to as synergism.

All the recent surveys done in Canada and the United States show a buildup of chemical pesticides in our waters. Professor C.D. Sculthorpe reminds us that few herbicides are chemically inert; most are constantly intereacting with other compounds in the water environment. He warns that "accumulation may occur in mud deposits, decaying organic matter, or living tissues."

As an example, Professor Sculthorpe cites the use of sodium arsenite to control certain water weeds in New Zealand. Where used, mud, and the weeds themselves, contained high levels of arsenic residues. In addition, the treated weed appeared to develop a resistance to the arsenic-based herbicides. He concluded that if accumulation occurs, this "renders any concept of a safe dosage utterly meaningless." (Sculthorpe, 1967)

The Formation of New Chemical Compounds

We now have evidence that in water supplies chemicals can combine to form new and often dangerous compounds. For example, we know that chlorine interacts with other chemicals to form new substances, and in particular chlorinated hydrocarbons. The U.S. Environmental Protection Agency studies of the lower Mississippi River reveal the persistence of biphenyl, chlorine and a number of benzene compounds. Tests have shown that in the presence of ultraviolet light, chlorine and benzene produce Gammoxane and Lindane, both chlorinated hydrocarbon insecticides. (U.S. E.P.A., April 1972)

Subsequent research done by the U.S. Environmental Protection Agency on treated water in 79 American cities conclusively established that chlorine interacts with pollutants to form new organic compounds. At Brownsville, Texas chlorine was found to form dibromochloromethane. Similar results were found in Miami's drinking water, which in this case came from underground sources. The amount of chloroform in all 79 cities increased significantly after chlorine treatment. Chloroform is a known carcinogen. (Haber, 1976)

In July 1977 the U.S. National Research Council completed their 1,000-page study of water pollutants in the United States. Over an 18 month period they identified 161 water-system contaminants; of these, a number were proven or suspected carcinogens and teratogens, including several widely used pesticides, 2,4-D and 2,4,5-T. (New York Times, July 21, 1977)

A similar study was released by the Ontario Ministry of the Environment in November 1977. Chlorine treatment of drinking water and sewage was cited as the cause of the increase in chloroform and several haloforms including bromodichloromethane, chlorodibromethane, carbon tetrachloride and ethylene chloride.

All of these are considered dangerous to the health of humans.

Herbicides and pesticides were found in the water at treatment plants in Amherstburg, Brantford, Cayuga, Dresden, Goderich, Guelph, Niagara-on-the-Lake, Pembroke and Toronto. Aromatic hydrocarbons containing the benzene ring were not found in the raw matter, yet turned up in the water treated by chlorine. (Malarek, 1977)

Another major problem is the buildup of nitrates in North American water supplies. Since World War II there has been a fourteenfold increase in the production of chemical nitrogen fertilizers, and a 70% increase in sewage nitrogen. Fertilizer runoff is widely recognized as one of the main causes of nitrate pollution. (Environment, 1969; Washington University of St. Louis, 1970)

Within the human body, nitrates combine with certain bacteria to form nitrites. One effect of nitrites in the body is the reduction of the ability of the red blood cells to carry oxygen. In children this is known as infant methemoglobinemia, a serious disease which can cause death. (Crossland and Brodine, 1973)

In 1975 Dr. N. Lee Wolfe and Dr. Richard Zepp of the U.S. Environmental Protection Agency research laboratory in Athens, Georgia reported on the results of the interaction of the herbicide Atrazine (a triazine compound) with nitrates which are common in water supplies near agricultural areas. Studies of drinking water in Iowa and Louisiana found that Atrazine interacts with the nitrates from fertilizer runoffs to produce N-nitrosoatrazine, a known carcinogen. This is the same problem caused when sodium nitrite, used as a meat preservative, is heated. (Prevention, November 1975)

Atrazine, as well as other triazine herbicides, are used in British Columbia, particularly along road and railway rights-of-way. (See Table II).

Intensifying Pesticide Toxicity

While there has been a rather general lack of research done on the subject of synergism, that which has been done seems rather conclusive. There are a number of studies which show that polychlorinated biphenyl compounds (PCBs) increase the toxicity of the chlorinated hydrocarbon insecticides (the DDT family) as well as the organophosphorous insecticides now widely used in the Okanagan. (Lichtenstein et al, 1973)

PCBs have been found in many water sources in North America. They are considered a major threat in the Great Lakes and the St. Lawrence River system. In one test, a PCB formulation (Arochlor 1254) was combined with the insecticide carbaryl (Sevin) and administered to DDT-resistant house flies. The toxicity of the pesticide was increased by more than 80 fold. (Frawley, 1965)

In another study, three professors of entomology at the University of Wisconsin tested the effects of four herbicides when combined with 12 common insecticides on fruit flies, houseflies and mosquito larvae. They found that "in most cases all four herbicides increase the toxicity of the insecticides." The phenoxy herbicide 2,4-D increased the toxic nature of parathion and DDT, both of which were extensively used in the Okanagan in the past. The Wisconsin entomologists concluded from their own research, and a survey of that done by others, that "the phenomenon of synergism of insecticides by selected herbicides is rather general." (Lichtenstein, et al, 1973)

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Of perhaps more interest to us is the research done by Dr. Charles N. Statham and Dr. John J. Lech of the Medical College of Wisconsin. They tested the toxic levels of four pesticides when combined with carbaryl (Sevin), the insecticide proposed for use on the Spruce Budworm in the Fraser Valley. It is also widely used in orchards in the Okanagan. The test species in this case was rainbow trout. One of the pesticides included in this study was 2,4-D and it greatly increased the toxicity of Sevin to rainbow trout. (Statham and Lech, 1975)

Effects on Humans

If the toxicity of pesticides is increased by synergism, then it is a matter of considerable concern to us in the Okanagan. Our lakes are deep and cold, with decreasing oxygen levels towards the bottom. These are ideal conditions for the persistence of organic pollutants.

We know that there is a relatively high level of arsenic and lead in our environment due to the widespread use of lead arsenic pesticides. We also know that our environment is polluted with DDT and the other chlorinated hydrocarbons, the result of many years of usage by Okanagan farmers. The B.C. Royal Commission on Pesticides (1975) reports that residues of these insecticides have been found in the tissues of fish and game birds in the Okanagan.

The nitrate level in some water is increasing due to fertilizer runoff and sewage outfalls. Our municipalities are adding chlorine to sewage and the drinking water every day. Heavy metals are also known to be in our water supply.

That is why the Canadian government recommends that we eat no fish larger than three pounds that has been caught in the Okanagan lake system.

We have to be concerned about the effect of all this on our health. The 1977 study by the U.S. National Research Council on water pollution concludes that much of the "intestinal flu" that Americans get each year is probably due to ailments caused by our present drinking water.

In an agricultural area like the Okanagan, where pesticides are widely used and run off into our major source of potable water, there is particular concern. For example, Dr. E. Stanton Maxey argues that "current animal work indicates that herbicides in combination with organic pesticides are up to four times as toxic as either agent alone." He is convinced that the interaction of herbicides and insecticides is a major cause of increased cancer among farm workers in Florida. (Maxey, 1974)

Summary

Under ideal conditions 2,4-D degrades by chemical and biological action in a relatively short period of time. However, it is well known that it persists for a long time in deep, cold water with a low level of oxygen, precisely the kind of conditions we have in the Okanagan lake system. It is also known that 2,4-D persists in bottom sediments for many months. Traces have been found in water sources years after spraying, indicating that it does not all degrade.

2,4-D degrades to 2,4-Dichlorophenol, a toxic chemical. The final breakdown product is reported to be succinic acid, a hydrazine product proven to be a carcinogen in tests on laboratory animals. To date, the B.C. government has not tested for the breakdown products of 2,4-D in areas treated for Eurasian milfoil weed.

Across North America there is growing concern over the increase of organic and other pollutants in our drinking water supplies. New hazardous chemicals are being formed in many waters. There is also good evidence that herbicides, including 2,4-D increase the toxic nature of other pesticides in a water environment.

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CONCLUSION

There is a considerable body of evidence which indicates that the phenoxy herbicides in general and 2,4-D specifically, pose a substantial threat to environmental and thus human health. Laboratory research results show that test animals develop chronic problems after administration of these chemicals. It has been noted that damage results from exposure of people, livestock, wildlife, fish and crops to 2,4-D.

Extensive research on the effects of 2,4-D on test animals indicates that the herbicide is teratogenic (causes birth defects), carcinogenic and very likely mutagenic (causes genetically transmitted defects). These findings have resulted in authorities such as the Mrak Commission (1969) recommending the immediate restriction of three esters of 2,4-D and all formulations of 2,4,5-T. Only the latter has been removed from general use, but it is still applied selectively in British Columbia. 2,4-D remains largely unrestricted.

All the phenoxy herbicides are from the same family with the same chemical base. There is very little difference between them as to molecular structure, mode of action, and the acute and chronic damage which they cause in humans, animals and plants. In certain cases they persist in the environment for far longer than previously thought. It is known that even the so-called "pure" 2,4,5-T and 2,4,5-TP inevitable contain some of the dreaded TCDD, one of the most toxic chemicals known to man. It has also been discovered, however, that 2,4-D can contain the potent HCDD and other higher isomer dioxins (whose effects are largely unknown).

There is evidence to indicate that crop yields and quality can be effected by exposure to 2,4-D. What the effect will be on fruit trees and grape vines of low levels of the herbicide applied through irrigation water over a long period of time, no one really knows.

The fisheries resource is also known to be particularly vulnerable to herbicide use. The concentrations of 2,4-D required to control the weeds is close to the acute toxicity level of the salmonid family. The chronic effects, habitat reduction and disturbance of the food chain which are caused by applications of this herbicide are of some concern to the managers of this resource.

In preparation for the 1977 programme, the B.C. Minister of the Environment instructed the Advisory Committee on the Control of Eurasian Water Milfoil to examine "all available data" on the public health aspects of the aquatic use of 2,4-D. An examination of their three Interim Reports indicates that this was not done. Even the concerns expressed by this same group in the earlier B.C. Royal Commission Inquiry into the Use of Pesticides and Herbicides were not brought forward again with any force.

We have not been able to locate any area in the world where Eurasian Water Milfoil has been eradicated using chemical methods. The application of 2,4-D in the present programme can only be defined as "chemical mowing". Those in charge of the weed control effort have repeatedly pledged that the chemical approach would be discontinued after five years. Yet it is obvious that 2,4-D applications must go on forever if any sort of control is to be maintained by this method. It is also obvious that the cost of this programme will skyrocket along with the price of the petroleum based 2,4-D.

As our research indicates that there are very real problems associated with the use of 2,4-D in the Eurasian Water Milfoil Weed control programme, the South Okanagan Environmental Coalition would like to make the following recommendations.

1) That the use of 2,4-D be discontinued for aquatic weed control. That all phenoxy herbicide use be re-examined in light of the information which is available on their detrimental effects.

2) That mechanical methods be used to control weeds which interfere with recreational activities. This approach would help provide employment as it is labour intensive and could result in the establishment of a weed removal equipment manufacturing industry in this province with considerable export potential. Markets may be found for the harvested weed which will help reduce the overall cost of the operation.

3) That in the long run, a species specific, safe biological control should be sought. These controls exist in other regions where Eurasian Water Milfoil is not a problem. We must discover the controlling agent (pathogen, parasite, etc.) and test it to determine whether it can be safely introduced here. This project will take time and money and would seem to be a logical job for the Federal government as the Eurasian Milfoil problem is national with international implications.

4) That nutrient levels in the lakes and rivers of the province should be reduced to levels which will slow weed proliferation. This involves curtailing man-made sources such as sewage outfalls, storm drainage, septic tank leakage, fertilizer runoff, and induced erosion. The Department of the Environment has taken an important step in this direction in the Okanagan Valley by requiring the city of Penticton to begin land disposal of its effluent.

This is part of a new policy designed to remove all sewage outfalls from the lake system.

5) That there is a pressing need to institute a more elaborate and effective health monitoring programme in British Columbia. A regular sampling analysis of food, drinking water and human blood, hair and milk should be established on a province wide basis in order to detect contaminents in the system. This would lead to identifying problems before they are manifested as disease. Further, epidemiological investigation should be improved particularly in the areas of cancer, teratogenesis, mutagenesis and acute illness so that more causes can be identified. Regional local and occupational data must be made available for purposes of early identification.

6) That a particularly close monitoring system be established for those who regularly come into contact with pesticides, such as government applicators, farmers and farm workers, forestry workers and forest fire fighters. Research in other countries indicates that these groups need to be watched very closely for acute and chronic poisoning.

Many people today feel that there are not likely to be any long term chemical solutions to biological situations. The lake system of the Okanagan Basin is the life blood for all species residing here. Any project which causes the contamination of those waters should be questioned, particularly if the chemical is known to have detrimental effects on the environment.

It has been claimed that health problems will be avoided by the implementation of a "zero tolerance" programme. In other words, the 2,4-D would not be allowed to come into contact with human beings.

One need only look at the cover photograph on this report to see the effectiveness of this approach. The picture was taken on Skaha Lake Beach in Penticton, B.C. four days after the application of 2,4-D at a rate of 20 to 40 pounds of active ingredient per acre.

We should reject emphatically any programme which calls for the contamination of a major water system with a chemical which is a proven hazard to the health of our environment. We cannot accept the claim that the programme will exclude all risk, for that situation is clearly impossible.

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ADDENDUM

The response to the first printing of The Other Face of 2,4-D has been amazing, all the more so because it has been reviewed in only one publication to date, Acres Inc. Yet we have received orders for the book from all over North America, and as far away as Australia, New Zealand and Belgium. The environmental word-of-mouth network is quite impressive.

In January we printed 500 copies, and by May we were sold out. We must apologise to those who have placed orders months ago and have yet to receive the book. We were totally occupied with fighting the provincial government's programme to put 2,4-D in our lakes and simply had no time to update the book and do another printing.

We have also greatly appreciated receiving the many letters from individuals and groups telling of their personal experiences with the phenoxy herbicides. Those that have related personal illness from exposure confirm our position and re-inforce the research data we have collected.

We are also greatly encouraged by the fact that there is such a groundswell around the world against the use of these herbicides. In British Columbia this year, there have been many local citizens' groups fighting spraying in their areas. The United Fishermen and Allied Workers Union, and various native groups, are taking up the struggle, now that they realize the threat that these herbicides pose to the fishing industry.

Just recently the Dene Nation in the North has succeeded in blocking the proposed spraying with Agent Orange of 284 launching sites in the Mackenzie River Delta. The federal government backed this incredible plan, but the government of the Territories was sensitive enough to recognize local concerns. In Saskatchewan, the National Farmers Union has prepared a very good brief to the provincial government questioning the increasing use of these herbicides. Their research and tests results by Environment

Canada revealed the buildup of the phenoxy herbicides, including of course 2,4-D, in the local domestic water supplies.

In the United States, a number of local groups in Arkansas, Idaho, Washington, Oregon and elsewhere have fought the use of phenoxy herbicides in forest management programmes. They have put so much pressure on the federal government that the U.S. Environmental Protection Agency has now put the herbicides manufactured from 2,4,5-Trichlorophenol on the Rebuttable Presumption Against Registration list.

However, the most important development in 1978 is the "discovery" that thousands of American veterans of the Vietnam war have experienced long term chronic illnesses from exposure to phenoxy herbicides while stationed in southeast Asia. Most recently, over 800 have filed a class action suit for damages against the U.S. government (see Uhl and Ensign, 1978).

Herbicide spraying in Vietnam by the U.S. military began in the early 1960's and was abandoned in 1970. Yet it has somehow taken eight years for authorities to recognize (or admit) that damage to veterans had actually happened. It makes us wonder if it would ever be possible to prove damage from the introduction of 2,4-D into the Okanagan Lake system.

Why is there such a growing concern and so many popular movements against phenoxy herbicides springing up all over the world? It is not an accident but stems directly from damage experienced at the local level. It is particularly true of areas where there is herbicide spraying in forest management programmes.

In the 1940's and 1950's insecticides and fungicides were predominant in the sales of pesticides, but this changed in the 1960's. Since then, the sales of herbicides have grown and by 1967 exceeded other pesticide sales. By 1975, sales of herbicides were more than double that of other pesticides (Seiler, 1978, Table I).

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In the 1960's, herbicide residues were being found in foods, but it was not considered to be a serious problem (Duggan, 1966). However, this again had changed by the 1970's, recognizing the growing use of phenoxy herbicides. Residues of 2,4-D now appear regularly in foods. Tests have shown residues as high as .13 ppm in leafy vegetables and some sugar products (Manske and Corneluissen, 1974). Residues of 2,4-D are being widely found in water and air tests. (Manigold and Sehulze, 1969; Gowen, Wiersma and Tai, 1976). One study made at Tallahasee, Florida found 2,4,5-T residues in 24% to 36% of individuals of various groups tested; 60% of people tested had residues of 2,4,5-Trichlorophenol, and absolutely everyone tested had residues of Pentachlorophenol (PCP). Penta residues were also found in local foods sampled: milk, soft drinks, sugar, bread, candy bars, cereals, noodles, rice, and wheat. It was also found in the city water supply. (Dougherty and Piotrowska, 1976). Clearly, the phenoxy compounds persist much longer in the environment than their promoters are willing to admit.

The Okanagan Situation

Readers might be interested in a brief update on the situation in the Okanagan, as this controversy led to the research that produced this book. In the spring of 1978 the Water Investigations Branch of the so-called B.C. Ministry of Environment announced that they had applied for permits to apply 2,4-D at a rate of between 20 and 40 lbs. a.i. to 1,500 acres in five Okanagan lakes. Due to our pressure, and the fact that over 40 organizations in British Columbia had passed resolutions opposing this programme, the civil servants in the provincial government who had to approve the permits were put on the spot. We understand from our inside sources that they did not really want to approve the programme. However,

tremendous pressure was applied by the political forces at the top. They finally approved all the applications, but with major reductions and some time limitations.

In 1977, the Social Credit Government passed new pesticide control legislation, and while it was deemed very inadequate by those outside the industry and users, it did contain appeal procedures. The South Okanagan Environmental Coalition appealed all 28 of the permits, and our action prompted other individuals and groups to also appeal.

We had no illusions on the matter. The W.I.B., of the Ministry of the Environment, was asking for the permits. The administrator approving (or possibly rejecting) the permits was also under the Ministry of the Environment. The Pesticide Control Appeal Board was also nominated by the Ministry of the Environment. If it sounds like Catch-22, it is.

But that wasn't all. Everytime we turned around we faced professors from the University of British Columbia. First, there were the three who made up the Advisory Committee (see Introduction). Then four of the seven members of the Appeal Board were U.B.C. professors. Outside consultation pays well in this province. Furthermore, a majority of the members of the Appeal Board represented institutions or disciplines which are major manufacturers or users of herbicides.

The SOEC decided to go through this seemingly hopeless exercise for several reasons. First, for our own public credibility, we had to exhaust all the proper channels. Secondly, we hoped that we could get some possible court action out of the appeal procedure. Thirdly, this was the very first test of the Appeal Board, and we wanted to demonstrate to the public the realities of these government institutions. Fourthly, we knew that we would have a chance to present our evidence, get some publicity, and strengthen our case with the public.

This certainly happened. Finally, we decided it was cheaper to make a long fight at this level than in the courts.

The hearings were held in Penticton in June. For the first two days, the Appeal Board heard opposition from various groups and individuals in the Okanagan, plus the Consumers Association of Canada (B.C.).

The following week, the SOEC in conjunction with Kelowna SPEC and Vernon SPEC presented our case. We took six and one-half days to present our documentation. We called nine special witnesses: Dr. Melvin Reuber, of the Frederick Cancer Research Center in Maryland; Dr. Charles Huver, of the University of Minnesota; Gil Zemansky, an environmental engineer from Seattle, Washington; Dr. Theodor Sterling, of Simon Fraser University; Merriam Doucet, Chairman of the Pesticide Committee of SPEC and one of the most knowledgeable people on herbicides in North America; Dr. David Clarke, the South Okanagan Health Officer; Drs. Barry Jones and Fred Van Seters of the Kelowna and Penticton Medical Associations; and Gary Taylar of the B.C. Grape Growers Association. We were assisted throughout the hearings by two very competent young legal people from the West Coast Environmental Law Association, Gregg McDade and Kim Roberts.

In contrast, the Water Investigations Branch presented their "case" in just three hours. They made no effort to refute any of our medical testimony. They presented no evidence on the safety of 2,4-D. They called no witnesses, not even the three U.B.C. professors who made up the Advisory Committee. We suspect that the "Three Wise Men" did not want to undergo cross-examination by SOEC. The W.I.B. was extremely confident that the Appeal Board would decide for them. And they did.

While the hearings were going on, the WIB was preparing a major application site at Kelowna. The Board announced it would make its decision on Tuesday, June 20. That morning the

WIB was prepared to apply the herbicide. The pressure was on the SOEC, for we had to get the Board's decision, prepare a court appeal, and try to get an injunction. This would take time. The WIB knew we were going to make a court appeal. They also knew that with their new machines they could cover the 52 acre plot in 2 hours.

In the morning, bad weather held up application, as SOEC lawyers struggled to put together a case. Local Kelowna citizens, under the direction of The Kelowna Chapter of Greenpeace had prepared a canoe demonstration, hoping to hold up application until the courts had had a chance to intervene. The WIB knew this as well. While forecasts were for rain, and a wind or thunderstorm could destroy the effectiveness of the application, the WIB decided to proceed. They wanted to set the precedent of applying 2,4-D.

At noon, despite the forecasts, the WIB decided to proceed. Their application boats were met with a steady stream of canoes manned by Kelowna residents. The RCMP in their launch and one WIB boat began arresting protesters. Others went into the test area. While the WIB first announced to the press that no 2,4-D would be applied while the canoeists were in the test site, by 3 p.m. they changed their minds. They wanted to make the application before the SOEC could get its injunction.

Therefore, the hopper boats began the application. Protesters in the area were sprayed. One of the two WIB hopper boats ran over a canoeist and dumped him in the water. They succeeded in covering around 30% of the test site before the SOEC obtained a temporary court injunction and stopped the application.

However, the court appeal approach has very limited uses in Canada. In this case, the SOEC tried to show in the subsequent appeal hearing in the B. C. Supreme Court in Vancouver

that we were denied natural justice during the Board hearings. This was made difficult by the fact that we had no transcript of the hearings, and only two days to prepare our case. We lost on this question, due to inadequate evidence; but we won a major victory (in the Canadian context) in holding up all major application in recreation areas.

As it now stands (July, 1978), the WIB will be applying 2,4-D to around 130 acres in the Okanagan lakes this summer. We have succeeded in knocking out 90% of the 1978 programme. But we know that we will face another round next year. We plan to take further political action in the elections this fall. We are also going to prepare a paper on alternative control methods for milfoil weed. Venice Lake virus has eliminated the plant in the Chesapeake Bay area, and it is now showing up in other areas of North America. It is the perfect solution. But for the WIB, it is no solution. They want the 2,4-D programme continued. They know it is an ongoing programme, guaranteeing their jobs, as 2,4-D has not been successful against Eurasian milfoil in any area of the world. As the top people in the WIB are biologists, they have similarly exhibited little interest in the mechanical programmes used in other areas.

New General Surveys

Since the first printing of The Other Face of 2,4-D we have received or dug up a number of other papers and bibliographies on 2,4-D and the other phenoxy herbicides. We might mention them here, so others fighting herbicide spraying have other basic sources from which to start.

Gil Zemansky of Friends of the Earth, 4512 University Avenue, N.E., Seattle, Washington, has produced two excellent papers on Eurasian milfoil weed, the use of 2,4-D, and the

politics involved. His paper, "Risks, Benefits, Causal Factors and Cosmetic Solutions," contains a good bibliography on 2,4-D.

The Consumers' Association of Canada (B.C. Branch), at 103 - 163 West Hastings Street, Vancouver, B.C., presented an excellent brief to the Pesticide Control Appeal Board, well documented, and containing a number of references on damages to health and the environment which we did not have.

The Northwest Coalition for Alternatives to Pesticides, P.O. Box 375, Eugene, Oregon, has produced an excellent Herbicide Information Packet which it sells for $3.50. It has good basic material, a good bibliography, and instructions on how to take legal and other action in the United States. There are also instructions on what to do if you get sprayed and how to document damage.

Another good source on 2,4-D is the monograph by the International Agency for Research in Cancer, Volume 15, 1977. It is available in most good university libraries. The monograph is a summary of some of the studies on 2,4-D and its effect on human health, but it is by no means complete, and leaves out some major important studies.

Another basic source is the publication by the World Health Organization in Geneva, "WHO Pesticide Residues Series, No. 1, 1972." This is an interesting source because of the material that is not covered, and their interpretation of some of the basic studies we have reviewed. Their bibliography reveals close contacts with Dow Chemical Co.

At our Appeal Board hearings, the B.C. Water Investigations Branch submitted as supporting evidence Part II, Chapters 6 and 7 from The U.S. National Research Council, Advisory Center on Toxicology, Drinking Water and Health. This was part of the widely publicized study which was released in August, 1977. This American agency produced only a six-page

summary of some studies on 2,4-D, a few of which we had not seen. What is striking is the number of widely publicized major studies which they do not review. They conclude, in classic fashion, that because there are "substantial disagreements in the results of subchronic and chronic toxicity with 2,4-D," we should have more studies done.

But the WIB also produced a paper on "Chlorophenoxy Herbicides," which forms the background of the Canadian Drinking Water Standards. This four-page paper is totally inadequate. Any first year university student could have prepared a much better survey. The paper concludes that "the approval limits for these herbicides are meant to serve in the event that these chemicals inadvertently occur in the water. Deliberate addition of these compounds to drinking water sources is neither implied nor sanctioned." And the WIB actually submitted this as evidence supporting their case!

Cancer and 2,4-D

The above general surveys list other cases of general effects on humans; there are some good studies on exposure of plant workers. But special mention should be made of the issue of 2,4-D and cancer. On February 28, 1978, Richard L. Reising, Special Projects Branch, U.S. Environmental Protection Agency, wrote Gil Zemansky explaining that 2,4-D had been put on the Pre-RPAR list because the Agency's Cancer Assessment Group (CAG), through a review of three studies, "found positive evidence that 2,4-D is carcinogenic." This letter, passed on to the SOEC, was then made public in British Columbia and caused a national news incident.

As a result of "The Canadian Incident," Edwin L. Johnson, Deputy Assistant Administrator for the EPA in Washington, wrote to Jim Nielsen, B.C. Minister of the Environment who is respon-

sible for the 2,4-D programme in the Okanagan. Johnson said that because of "conflicting opinions" the EPA had no intention of banning 2,4-D until there was further information.

However, through sources in the United States the SOEC had received copies of an internal EPA memorandum dated April 27, 1975 from Janet Lambert of the Special Pesticide Review Division to Ed Johnson. In this memo, Lambert explained how, through administrative bungling, 2,4-D had been placed on the Pre-RPAR list. Furthermore, she stated that their office "is not conducting new studies to investigate potential carcinogenicity - nor is there any intent to do so in the future." In addition, the only tests reviewed were the 1963 and 1964 tests by the U.S. Food and Drug Administration; the other published studies (listed below) were not included.

When he was in Penticton, Dr. Melvin Rueber, who had been a member of the EPA Cancer Assessment Group and who had advised Senator Edward Kennedy's Subcommittee on 2,4-D, made several interesting comments on this situation. As a top cancer researcher, who had done a great deal of work with chlorinated hydrocarbons, he found it interesting that there had been so few tests done on 2,4-D considering the results of the Bionetics and FDA studies. Most chlorinated hydrocarbons had been subjected to more than a dozen studies. He then recounted the political and user pressures on the National Cancer Institute and the EPA not to ban 2,4-D, and not to do any further testing. If 2,4-D were found to be carcinogenic, it would have to be banned for use on food crops because of the Delaney Amendment to the U.S. Food and Drug Act. The refusal of the EPA's Cancer Assessment Group to recommend further testing, in the face of what was known, caused him to resign from it.

We now know that the same forces are at work in Canada. Our fight against the phenoxy herbicides, and the publicity resulting from it, has caused a minor panic among government

agencies involved. Dr. W. T. McKinley, now head of the Health Protection Branch, pointed out at a conference in Saskatchewan last winter that no testing was being done in Canada, and none could be expected. No one was doing this testing, as funds were not being allocated.

The Position of the B. C. Government

Our major concern is that the application of 2,4-D in our lakes over a long period of time will cause a chronic health problem. As noted above (Chapter III), we accept the majority position that carcinogens cause irreversible, cumulative damage, even at low dosages. This was the stand taken by the IARC in the Lyon meeting in 1977 (McGinty, 1977). As they noted, because there is enormous individual variation in human reaction to carcinogens, a threshold dose level is a practical impossibility. The SOEC stands behind the position taken by Jens Steensberg, Chief Medical Officer, Danish Environment Protection Agency: "We cannot expect, or wait for, universally accepted proof of carcinogenic risks." We must act now to prevent humans from being test subjects.

In contrast, Dr. Peter Newroth, one of the biologists responsible for the 2,4-D programme in the Okanagan, told the Kelowna Rotary Club on April 26, 1978 that "we have always known that 2,4-D was carcinogenic in tests on small animals but one has to take into account the amounts used. In these tests the lowest dosage level is 600 parts per million. That's the lowest; they go much higher." In contrast, he noted, they had "set the amount to be used in the lake at .6 ppm and that's minute comparatively."

In the public meeting in Penticton on May 2, 1978, Dr. Courtland Mackenzie, a member of the Advisory Committee and the University of British Columbia and the only independent

medical adviser to the B.C. government, stated that "there is a threshold level for many carcinogens." In response to questions from the SOEC, he admitted that on this point he "disagreed with the general medical opinion."

A similar position was taken by Dr. W. D. Prowrie at the public meeting in Kelowna on May 3, 1978. Dr. Prowrie, a member of the Advisory Committee and a professor at the University of British Columbia, stated that "high doses of chemicals are what causes cancer."

New Evidence on Cancer and 2,4-D

A number of other published studies on 2,4-D and cancer should be noted, as we did not have them at the time of the first publication. The U.S. National Academy of Sciences survey reported that "studies on the in vitro and in vivo effect of 2,4-D on the growth of Ehrlich ascites tumor in BALB/c mice showed that the herbicide was inhibitary at 45 mg/kg or more (Walker et al, 1972)."

The IARC reports a Russian study where, of 165 rats fed 2,4-D at a level equal to 1/10 the LD_{50} level in their food, 3 (or 1.8%) developed malignant tumors (Arkhipov & Koslova, 1974). J.P. Seiler reports a Polish study done in 1974 in which an elevated number of tumors were observed in test animals (Jurek, 1974). The U.S. N.A. S. survey also lists another study which did not report any tumors when 2,4-D was administered to mice, but found that it "had a cocarcinogenic effect in mice when it was applied to the skin with 3-methylcholantrene." (Haag et al, 1975).

Several studies we had not heard of are reported in the paper by J. P. Seiler. First he notes the case of the Swedish railroad workers (cited above, Chapter III); the reports cited for this "showed only slightly higher than expected tumor fre-

quencies" (Axelson, Rehn and Sundell, 1974; Axelson and Sundell, 1974). However, this data has been sorted out and reviewed, and Seiler now notes that the recent study indicates that "a significant trend showing a clear relationship with increasing exposure to phenoxy acids was observed" (Axelson and Sundell, 1977).

Seiler also cites a German study of pesticide-exposed agricultural workers, which revealed lung cancer that was twenty times as high as would normally be expected. However, these workers were exposed to pesticides other than just 2,4-D. (Barthol, 1976). Finally, a survey of 87 men in Emea, Sweden who were exposed to phenoxy herbicides in forest management programmes revealed that males had twice as many mesenchymal tumors as women, which was exactly the reverse of the national average (Hardell, 1977).

Birth Defects and 2,4-D

There is very little doubt about the teratogenic nature of 2,4-D, as there are so many studies which have found positive results. No one in the B.C. government disputes this. But Dr. Charles Huber gave us a copy of a paper that Dr. Jacqueline Verrett of the U.S. Food and Drug Administration presented to the Minnesota legislature in 1976. This is a chicken embryo test of 2,4-D, 2,4-5-T and several dioxins. Two tests were made of 2,4-D samples, one with all impurities removed. Both were teratogenic, but interestingly enough, the purified sample proved to be more strongly teratogenic. She also found the same to be true of the purified sample of 2,4,5-T, which supports the research of the National Center for Toxological Research (cited above). Therefore, those groups which are concentrating on the dioxin contaminants in phenoxy herbicides, believing them to be the only danger, are certainly on the wrong track.

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The internal memo of the EPA from Lambert to Johnson reveals that Dr. Diane Courtney has just completed another teratological study with 2,4-D, which again is reported to show positive results.

Dioxins

The history of the edema factor problem (see Chapter V) is also detailed in the paper by Dr. Jacqueline Verrett. The 1969 outbreak in North Carolina was traced to the hexa-, hepta- and Octa-chlorodibenzo-p-dioxins, most likely from 2,4-D residues in fats in chicken feed.

Dr. Verrett carried out her own tests on several dioxins. Tetra-dioxin (TCDD) was highly toxic to chick embryos. TCDD and Hexa-Dioxin (HCDD) also proved to be teratogens. (Verrett, 1976).

Mutagenicity

Our original publication was rather weak on the question of 2,4-D and possible mutagenic effects. This was primarily due to the fact that we had not had time to do adequate research. We have now discovered that in the 1970's there has been considerable research done on the mutagenic potential of 2,4-D and the other phenoxy herbicides.

We have been fortunate to obtain a copy of a review article by J. P. Seiler of the Swiss Federal Research Station, CH-8820, Waedenswil, Switzerland. Our copy of the paper was obtained from Dr. George Streisinger of the Institute of Molecular Biology, University of Oregon, but it is scheduled for publication in the near future in Mutation Research. A similar review article by Dr. W. T. Grant on 2,4,5-T is also scheduled for publication in Mutation Research, and both these

articles will be important contributions to the discussion on the phenoxy herbicides. In addition, the other general surveys cited above also list a number of individual studies. I will not try to duplicate Seiler's review of 133 studies, but I will try to summarize his own conclusions.

The three professors from the University of British Columbia who comprise the Advisory Committee to the B.C. government have repeatedly stated in public that there is no evidence that 2,4-D is a mutagen. While at public meetings they may appear to be propagandists for the government, we have to assume that their position is due to a lack of knowledge on the issue.

J. P. Seiler concludes that there is no question but that "phenoxy acids interact with nucleic acid metabolism." They do "influence nucleic acids and their synthesis." Tests on microrganisms have mostly been negative; however, "a weak mutagenic effect of phenoxy acids cannot be completely excluded on the basis of these bacterial results."

Quite different test results have been obtained using yeasts. Not only 2,4-D but the other phenoxy acids "have mostly given positive results in yeast test systems."

In plants tests (see also Chapter IV), there are many studies which show positive results. At higher dosages, they have produced radiomimetic effects. Seiler concludes that "in general plant systems have shown that phenoxy acids do influence the genetic material or the mitotic cycle or both."

Of more significance to humans are the tests on Drosophila (fruit fly). Seiler concludes that "it can be assumed that in general phenoxy acids possess weak, but significant genetic activity in Drosophila."

The tests on mammals suggest that the primary influence of 2,4-D is on the spindle protein. Several cases cited found

that 2,4-D caused chromosome aberrations in human tissue cultures. "In vivo tests have been performed on mice, and most of the investigations report positive findings with respect to chromosome breakage under the influence of 2,4-D."

Seiler concludes that "until we know more about the penetration of phenoxy acids into different cell types, an evaluation of the mutagenic potency must remain tentative, although they can clearly be considered as potentially mutagenic agents."

Agriculture

We have no new studies on the dangers of 2,4-D to crops to report, simply because we have not had the time to do this research as we have been concentrating on damage to human beings. But during the summer of 1978 the question of damage to grapes became a major news event in the Okanagan Valley.

First, 2,4-D damage to grape vineyards and crops in Washington state had been regularly reported in the Goodfruit Grower, and these news reports were made public at the Pesticide Appeal Board hearings. Both the B.C. Grape Growers Association and the B.C. Fruit Growers Association passed resolutions at their annual meetings opposing the use of 2,4-D in the Okanagan lakes.

Right at this time, a number of vineyards in Osoyoos and Oliver reported 2,4-D damage. The Grape Growers Association made strong public statements and took their appeal to the Board. It is widely known that at the parts per trillion level 2,4-D is damaging to grape vines, and it may be for these reasons that the Board turned down all 2,4-D applications for Vaseaux and Osoyoos lakes, where most of the B.C. vineyards are located. However, the Board never presented any reasons for the decision that they took, and thus they appear to be arbitrary.

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Fish and Wildlife

In British Columbia, there is growing concern about damage to commercial fisheries from phenoxy herbicides. In fact, there is fear that our fishing industry may be destroyed by pollution in general. A very alarming article by Lawrence Wright documents the decline of the commercial fishing industry in the United States due to the pollution of fresh water bodies with chemical pollutants (Wright, 1977).

TABLE I

HERBICIDE USE IN BRITISH COLUMBIA

1973

User		Lbs. of Active Ingredient
1.	The Forest Industry	
	(a) Private use	27,150
	(b) Government use	12,478
	sub total	39,628
2.	B.C. Hydro	
	(a) Transmission Rights of Way	30,070
	(b) Gas Division	377
	(c) Railway Section	675
	(d) Soil Sterilants	13,283
	sub total	44,405
3.	B.C. Department of Highways	37,266
4.	Railways	
	(a) Canadian National Railroad	6,820
	(b) Canadian Pacific Railroad	18,555
	(c) B.C. Railroad	144,013
	sub total	169,388
5.	Agriculture and Home and Garden	
	(a) Phenoxy herbicides	9,318
	(b) Phenols	45,943
	(c) Benzoic Acids	1,435
	(d) Toludines	96
	(e) Chloroal phatic acids	2,094
	(f) Amides	4,428
	(g) Carbamates	8,662
	(h) Ureas	839
	(i) Nitrogen Heterocuclics	44,240
	(j) Inorganic compounds	26,276
	sub total	143,331
6.	TOTAL - all herbicides	434,018

SOURCE: British Columbia. Royal Commission of Inquiry into the Use of Pesticides and Herbicides, Final Report of the Commissioners, May 30, 1975, Vol. II, Part II, Table MM - 1.

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TABLE II

HERBICIDES USED IN BRITISH COLUMBIA

Government and Industrial Use

1973

Herbicides	Trade Name	Lbs. of Active Ingredient
Trichloroacetic Acid	Sodium T.C.A.	99,104
2,4-D		65,836
2,4-D/2,4,5-T	Agent Orange	22,009
Triazine Herbicides	Simmaprim, Primatol	21,266
Picloram	Tordon 10K	19,585
Picloram/2,4-D	Agent White Tordon 101	13,718
2,4,5-T		13,215
2,4-D/2,4-DP	Weedone 170	9,718
Fenitrothion	Sumithion	7,250
M.S.M.A.	Glowon	7,240
Misc.		11,595
T O T A L		290,536

SOURCE: British Columbia. Royal Commission of Inquiry into the Use of Pesticides and Herbicides. Final Report of the Commissioners, May 30, 1975. Volume II, Part II, Table MM - 1.

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TABLE III

2,4-D APPLICATION RATES

Target Plant	Environment	Active Ingredient per Acre
Lambs Quarters, Wild Mustard, Ragweed	Cereal Crop Land	6 oz.
Russian Thistle	Cereal Crop Land	12 oz.
Broad leaf annual weeds	Field Corn	8 oz.
Broad leaf weeds	Pasture and forage grasses	8 oz.
Trees and bushes	Prairie rangeland	32 oz.
Broad leaf weeds	Lawns	1/2 lb.
Woody Plants	Forests	2 - 4 lbs.
Water hyacinth	Aquatic	1 - 2 lbs.
Floating weeds	Aquatic	4 lbs.
Bulrushes, cattails, water lilies	Shoreline areas	4 - 6 lbs.
Forests and rubber plantations	Cambodia - 1969	1/2 - 3 lbs. (Agent Orange)
Jungle vegetation	Vietnam - 1961-1969	5 - 12 lbs. (Agent White)
Eurasian Milfoil weed	Aquatic-Okanagan Lake System - 1977	20 - 40 lbs.

NOTE: It has been claimed that the U.S. military used Agent Orange at as high a rate as 27 lbs. per acre in Vietnam. Normal usage, however, appears to have been far lower, as the Cambodia report and field manuals demonstrate. The UC-123 Aircraft which applied the spray were calibrated to spray at a rate of 3 gallons total fluid per acre.

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According to the B.C. Royal Commission of Inquiry into the use of Pesticides and Herbicides, one gallon of 2,4-D, 2,4,5-T or Agent Orange contains 4 lbs. of active ingredient. Regular commercial preparations used in Vietnam are reported to have the same ratio.

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TABLE IV

2,4-D AND 2,4,5-T USE IN BRITISH COLUMBIA

ESTIMATES 1976

2,4-D	Acres
Home and Garden	5,000
Agriculture	250,000
Forestry and Industry	17,000

2,4,5-T	
Agriculture	200
Forestry and Industry	6,900

SOURCE: Baine Vance
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B.C. Department of Agriculture

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