CAUTION BEFORE YIELD:

Pesticides and Treeplanters

By

TED M. DAVIS

Photo by Jeannine Caldbeck

Cover photo by Jeannine Caldbeck

FORWARD

Ted first treeplanted in Oregon in 1968. He was earning 2.8¢/tree and just finishing his B.S. in Biology at Portland State. Frequent mountain climbing expeditions in B.C. led to his immigrating that year.

Reforestation in British Columbia began to be let to contract in 1969-70. Beginning in 1970 Ted and I contract planted as partners for seven years, always with a wry laugh that our wilderness lifestyle still risked civilized hazards. This manual clarifies what is known about one of those hazards, nursery pesticides.

During the seventies, the province was dramatically increasing its reforestation program. By 1975 there was contract planting all over B.C. with several thousand planters at the height of the spring season.

In 1977 Ted left for Japan to study iaido and kendo. In the fall of 1978 planters, contractors and co-ops formed the Pacific Reforestation Workers' Association. The unknown hazards of pesticides were a major unifying concern for this diverse group. In the spring of 1979, Ted returned, resumed planting, and got involved in the PRWA Health Committee. In the 1979 general meeting the PRWA membership endorsed the following Health Committee resolution as its basic position statement on pesticides:

RESOLUTION

> Because of the actual and potential mutagenic, carcinogenic, teratogenic and toxic effects of pesticides and the potentially catastrophic results to the biosphere, ourselves, our children
>
> and the human gene pool, we oppose the principle of chemical control of pests and affirm the principle of biological and other means of control that do not carry the distinct risks that chemical pesticides carry.

At this meeting, the Ministry of Forests was asked to notify planters of the pesticides used on the seedlings and to conduct tests to determine how long residues are present. Ministry of Forests officials agreed in principle but did not follow through.

It is fortunate for the planters of B.C. that Ted persevered through the epic two-year volunteer research project which gave birth to this manual.

In the spring of 1981 Bob Farrel, an experienced planter, had an extreme reaction to a Captan-Benlate mix on some seedlings (see Appendix 3). News coverage of this incident, combined with the concurrent exposure of fraudulent I.B.T. Laboratory testing, paved the way for the acceptance of the P.R.W.A. requests for notification and testing. Those agreements were reached in our 1981 spring meeting with the Honorable Tom Waterland, Minister of Forests, the Honorable Stephen Rogers, Minister of Environment and Ron Kobylnyk, director of the Pesticide Control Board, and are detailed in this manual. It is a credit to the Ministry of Forests that they agreed to undertake the notification of this potentially inflammatory information. This manual is an essential complement to that agreement and to the residue testing. Together they provide a thorough information package that will help the planter make an informed choice about planting pesticide-treated trees. May this create a precedent for concerned agricultural workers and food consumers affected by pesticide use.

I encourage everyone who reads this manual to give feedback and support to Ted in this effort to clarify and communicate these issues.

Dirk Brinkman

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ACKNOWLEDGEMENTS

Dirk Brinkman made the notification process happen (See Appendix IV). He had the sense to arrange our meeting with the Ministers and to follow through on the results. His consistent support for this project, both psychologically and financially, was critical.

Jeannine Caldbeck and Rosanne Parker gave eleventh-hour help when my frustration level with the project was running high. Jeannine helped with the project in innumerable ways, especially in being tolerant of my papers, books and typing.

Phillip Ditchburn, Teena Shaw and Verity Rolfe gave the final push to see this report completed.

Dirk Brinkman and Sari Taylor offered valuable criticism.

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TMD

INTRODUCTION

A wide variety of pesticides are used on forest seedlings at BCFS nurseries and unknown amounts of these pesticides are on the trees when we plant them. The pesticides in question are captan, Benlate, Daconil, diazinon, malathion, Orthene and the triazine herbicides propazine, prometryn, prometone, simazine and atrazine. Trees from Alberta may be treated with metiram and/or ferbate. Some trees have been treated with methyl bromide. These pesticides, under certain conditions, can produce a variety of toxic effects. In the mildest acute poisoning, one would experience headache and nausea. With more serious poisoning other symptoms may occur leading to severe illness and death. Some of these pesticides can cause cancer, gene mutations and birth defects in experimental animals and possibly in humans. The particular effect depends on the pesticide and the amount and length of exposure.

In this report, I outline the information a treeplanter needs to assess the hazards and risks that these pesticides pose. I also hope this report will awaken at least a few people to the nature and seriousness of the pesticide problem, and provide a reference source for those who want to know more.

The text has extensive notes to references that support the claims made in this report and to guide the reader to important sources of information on the subject.

Section I provides background information that is essential to understanding the larger problem of pesticide use and abuse. I begin with a brief and general description of pesticides, their history, benefits and potential for agricultural and ecological disaster - the “pesticide treadmill”. Next, to introduce pesticidal health threats, I discuss the concepts of hazard, risk and safety. The health hazards are then described with special emphasis on the most serious long-term diseases: cancer, mutation and birth defects. Finally, I end this section with a discussion of toxicity tests and their limitations.

Section II is a description of pesticide use in the nursery.

In Section III, I outline physical, chemical and toxicological data for each specific pesticide used on forest seedlings. This is followed by a brief conclusion with some suggestions for future work. Finally, there is a glossary, bibliography and appendices.

Appendix I is a discussion of the IBT testing results. Appendix II comments on the on-site use of herbicides, Appendix III describes a case of possible contact dermatitis from pesticide exposure in treeplanters. This has become known as “The Terrace Incident”. In Appendix IV, I describe our efforts with the Provincial government to

get sufficient notification and residue tests. The prolonged correspondence is included. The Ministry of Forests has agreed to provide a warning on the boxes and a history of pesticide application with each invoice. A working plan for new residue tests has been developed.

Photo by Ted Davis

SECTION I: BACKGROUND INFORMATION

PESTICIDES

Pesticides are substances that kill or control pests. A pest is any organism that is “injurious, noxious or troublesome”. (1) Bacteria, protozoa, viruses, pathogenic microorganisms and endoparasites are rather arbitrarily excluded.

Pesticide Classification

Pesticides may be classified according to their use. Fungicides (captan, Benlate, Daconil, etc. J kill fungi. Herbicides (propazine, atra- zine, 2,4-D, etc.) kill plants. Insecticides (diazinon, malathion, Sevin, DDT, etc.) kill insects. Fungicides, herbicides and insecticides are a few of the many kinds of pesticides.

Pesticides may also be classified according to their chemical structure. This is useful because a family of chemically similar pesticides will tend to have similar properties. Thus, we can characterize the organochlorine pesticides (DDT, aldrin, dieldrin, endrin, etc.) as being very persistent in the environment and having a low acute toxicity to mammals. The organophosphate pesticides (diazinon, malathion, parathion, etc.) are not as persistent as the organochlorine compounds, but some of them are very toxic to mammals. There are notable exceptions to these family characteristics, but the general principle is still useful. (l)

There are many ways of classifying pesticides according to their chemistry.[1] In addition to organochlorines and organophosphates, there are the inorganic and simple organic pesticides (arsenic, mercury, cyanide, phosphorus, thallium and fluorine compounds), botanical pesticides (pyrethrin, rotenone, nicotine and strychnine), carbamates (Sevin, aldicarb, etc.), chlorephenoxy herbicides (2,4-D, 2,4,5-T, MCPA, etc.), the triazine herbicides (simazine, atrazine, propazine, etc.), and many others. (l)(2)(3). At the present time, there are over 600 basic pesticide chemicals in more than 5,000 brand-name products in Canada. (1) Group descriptions of the pesticides important to treeplanters can be found at the beginning of Section III.

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[1] Any substance - including a pesticide - can be classified as organic or inorganic. 'Organic' means that the material is based on the chemistry of carbon. The popular meaning of 'organic' - that a plant or animal is grown without pesticides or synthetic fertilizers - has nothing to do with this classification. Most organic pesticides are made by chemical companies and for this reason, are called “synthetic organic pesticides”.

Disease Control and the "Green Revolution"

Pesticides have been used for thousands of years. (4) The Romans burned sulphur to control insects; the Chinese used arsenic and pyrethrin. (1) In 1900 the principal pesticide in the occident was "Paris Green" - lead arsenate. (5) Other common pesticides used before WWII were the highly toxic salts of such metals as chromium, copper, iron, lead, mercury, sodium, thallium and zinc. (1)(4)(6) Since about 1940 these substances have been replaced by the successful and inexpensive synthetic organic pesticides. These pesticides are usually less acutely toxic to people, wildlife and non-target organisms than the older traditional poisons. What is more, they are far more effective. (3)(4)

These organic pesticides are very important in controlling insect-borne diseases, especially in the tropics.The incidence of malaria, yellow fever, sleeping sickness, plague and typhus have been dramatically reduced since the introduction of modern pesticides. Millions of people are alive and free of these devastating diseases because of synthetic organic pesticides, especially DDT. (3)(7)(8)

The reduction in insect-borne disease, along with other public health measures, resulted in a huge population increase and consequent strain on the food supply. To meet this challenge, new plant varieties and production methods were developed that gave higher yields. This so-called "green revolution" has dangerously narrowed the genetic base of crops and made them especially vulnerable to pests and climatic variation. (113)(9) The success of this approach is doubtful. (10) At least one-third of the world's population still goes to sleep hungry. (3) Political, economic, social and distributional inequities compound the problem.

The "green revolution" produced an agriculture heavily dependent on petroleum products, including pesticides. (10) Obviously, since both control of disease and agricultural production now depend on pesticides, the fact that the pests, especially insects, have been developing resistance to pesticides has catastrophic implications (12)(7)(9)(11)

Insects

Pesticides are used primarily for the control of insects. In terms of numbers, diversity of species, adaptability and potential for outliving any natural or artificial disasters, they have no peers. Insects live in soil and freshwater, on plants and animals, including one another, in books, houses, the tropics, the polar regions, almost everywhere. Estimates put the number of species of insects at over one million. About 750,000 species are described, which is more than all

the other animal species combined. (114) The variety of insects and the places in which they can live is markedly increased by the occurrence of two or three different forms of the same species: adult, pupa and larva. The number of individuals and their reproductive potential reaches fantastic proportions. Even in relatively small areas, one talks of billions and trillions of individual insects. (11)

The insects appeared about 300 million years ago and their form has been so successful, that for the last 200 million years they have been much the same as the insects today. (11) Dinosaurs became dominant about 150 million years ago and disappeared about 65 million years ago. (13) Manlike creatures apparently branched off from the other primates about 4 million years ago, but Homo sapiens arose only 100,000 years ago. (14) The insects were around long before humans.

If we cause our own demise through some catastrophe such as pollution, climatic change or nuclear war (all avoidable, but increasingly probable), the insects will still be here. (15)

Of the million or more species of insects, only five to fifteen thousand are considered pests. Fifty thousand to one hundred fifty thousand have pestiferous potential but are controlled by various physical and biological factors in the environment. Two of the most important control factors are predators and parasites. Many of these predators and parasites are insects or organisms that are killed by pesticides. So if we use a pesticide on a crop, we may kill the pest, but we also kill the predators and parasites that naturally keep the pest and potential pests under control. Often the predator that controls the pest is more devastated by the pesticide than is the pest. This is because the predators are usually less robust than the pest species, their numbers are fewer, and the pesticides deplete their food supply (the pest species and other prey). The predators are killed by the pesticide, starve, or leave the area. At this point, we become very dependent on the pesticide for control of the pest. There are always a few members of the pest species that are able to survive the pesticide. With their prodigious powers of reproduction and no predators to hold their numbers in check, these resistant members create an even worse pest problem. Also, new pests begin to appear. These new pests appear because the predators which controlled them are also killed by the pesticide. The “balance of nature” is upset so that the original pests, along with previous non-pest species, which have also lost their predators, have a population explosion. We now have a huge multispecies pest problem far worse than the original one. (11)

The next step has been all too predictable. As the pests become resistant to the pesticide, new and usually more dangerous pesticides become necessary. The farmer adds more pesticide which triggers

another multi pest outbreak. These pesticides often have serious undesirable effects on the soil, water, wildlife and people. (16) The problem gets worse and worse as ever-increasing amounts and kinds of pesticides are needed. This is the "pesticide treadmill". (11)

If the problem were simply a scientific and technological one, we could soon correct this foolishness. Unfortunately, it is also a political and economic problem. The chemical companies have entered the scientific, agricultural and political communities with money, reports containing false data, statistical absurdities and irrelevant conclusions. Politicians and scientists have been bought, threatened and coerced. (17) Fundamental to scientific research is independence, freedom and integrity. These values are being undermined by the producers of synthetic pesticides and chemicals. Not only have they made false claims about the efficiency of their products, but they have lied about the dangers of these chemicals. These dangers include gene damage, birth defects, cancer, destruction of ecosystems, soil and wildlife. (11)(l7)(18)[2]

Integrated Pest Control

There is, however, a way to get off the "treadmill". It is called "integrated pest control". It is based on biological control through insect predators (which may be insects), pest-resistant crops, crop rotation, insect hormones, viruses, sterilizing agents, cultivation and crop handling methods, and so on, which can be used either alone or in combination with minimal application of highly selective, short-lived, "narrow-spectrum" pesticides used with an appreciation of the ecological principles involved. Rather than attempt eradication, the pest is maintained at an economically acceptable level. Pesticides are used rarely. The success of this strategy is proven. (7)(11)(19)

[2] If there are readers who doubt the truth of these statements, or think that I am exaggerating, I refer them to the literature cited.

HAZARD, RISK AND SAFETY

The ecological consequences briefly described above are one of two groups of dangers resulting from the widespread indiscriminate use of pesticides. The other major threat is to human health. To understand this problem, one must clearly understand the nature of hazards, risks and safety.

In ordinary language, these terms are used in a confused and vague manner. Hazard and risk are often used synonymously while safety is usually thought of as freedom from hazard or risk. But since nothing is entirely free of risk, what can the concept of safety mean? Obviously, to think about this issue clearly we need to define these terms in a way that is both precise and heuristic.

The Handbook for Pesticide Applicators and Pesticide Dispensers (1) published by the Pesticide Control Branch, Ministry of Environment, defines the hazard of a chemical as a measure of risk to the user. The Handbook also states that the hazard depends on how a substance is used or abused. Presumably, if you use a toxic substance in the proper manner, the hazard approaches zero, there is essentially no risk and it is safe. Hazard is further defined as a function of toxicity and exposure time.

If all of this seems rather confusing and obtuse, let me reassure you that these terms can be used in a way that is not only precise but illuminating. Clarification of this problem was developed in the 1970s by Chauncey Starr of Electric Power Research Institute, David Okrent at UCLA, William W. Lowrance now at Harvard, and others. (5)(20)(21)(22) I will rely heavily on their work in what follows.

Risk is a measure of the probability and severity of adverse effects. Something is safe if risks are judged to be acceptable. (5) Hazards are the potential adverse effects, irrespective of their probability.

For example, passengers in a rowboat and an ocean liner share a common hazard in crossing the ocean: that of drowning. Those in the rowboat also must contend with the hazards of dehydration and exposure, while those in the ocean liner are subject to fire and falling overboard. Other hazards can be imagined. As far as drowning goes, the individual risk is much greater in the rowboat than in the ocean liner. Since the risk associated with the rowboat is usually considered unacceptable, and the risk associated with the ocean liner is acceptable, we say that the rowboat is “unsafe” while the ocean liner is “safe”. (21)

We can also make a distinction between societal hazards and risks and those faced by an individual. In our example, the maximum societal

hazard in the rowboat is, say, only three deaths. The societal hazard in the ocean liner might be 3,000 deaths. The hazard for each individual in the ocean liner or the rowboat is obviously the same, but the risk in the rowboat is greater. The societal risk is given by the societal hazard multiplied by the probability of the event. (21) Thus, a defective radar on the ocean liner might create a larger societal risk than a leaky rowboat. An important aspect of this issue is the psychological perception of risk. Our intuitive evaluations of risk are often in error. For example, in one study people were asked to estimate the number of fatalities from specific causes annually in the U.S. The perceived risk was often many orders of magnitude in error. Botulism, tornadoes, and pregnancy (among others) were far overrated as causes of death, while cancer, heart disease, and accidents not involving motor vehicles were vastly underrated. (22)

These kinds of studies have shown that low probability events are overrated, while risks of high probability are often underrated. One researcher noted: “One chance of 50,000 of winning a lottery, or having one's house burn down, seems a better chance, or greater risk, than it actually is.” (22) Our perception of risk often has little to do with reality.

Finally, and this is important for us, the risks that society can reasonably take may be totally unacceptable for the individual taking the risk. Treeplanters are taking the risk for society. Pesticides on seedlings might represent a reasonable risk for society, but for the individuals taking the risk, it may be too great to be acceptable.

Photo by Doug Cowell

Estimating the Risk

Since seedlings are grown with pesticides, and pesticides are hazardous, there is some health risk associated with planting. Depending on the particular pesticide and the amount of residue on the trees, the hazards may be relatively minor (e.g. dermatitis) or catastrophic (e.g. birth defects). For most pesticides, the risks are unknown although many of the hazards are well understood. A severe hazard, such as genetic dama9e, even if of low probability, can easily constitute an unacceptable risk.

The risks associated with pesticides are directly related to the amount of exposure. A large exposure for a short period typically results in acute symptoms which usually disappear soon after the exposure. A single such exposure can produce long-term effects such as cancer or gene mutation. Small exposures over a long period can, in terms of chronic effects, add up a large exposure. With these small exposures, you would see no acute symptoms, but you might get cancer or other chronic diseases long after the exposure ceases. However, not all pesticides produce cancer, mutations or birth defects.

Estimating the risk is obviously of immediate practical importance to us and others. However, there is no objective way to dependably quantify the sort of risks we are concerned with.

Biological and economical limitations in testing procedures preclude this possibility. (23) The best we (or anyone) can do is to assess the risks on a qualitative basis. Science can provide us with information about the hazards and, to a very limited degree, an idea of the probability of the event in a large population. The determination of safety is a matter of personal value judgement.

Science can provide information on which to base a decision, but it cannot make the decision. (24)

Health Hazards

Although the problem of risk defies simple and straightforward solution, the nature of the hazards from pesticides are well established. The types of hazards that concern tree planters fall into two broad categories that have some overlap: acute and chronic. Acute hazards are short-term hazards that include things like skin rashes, headaches, nausea, fever, chills, behavioural effects and so on. These symptoms are a reflection of what is called “acute poisoning” and can usually be reversed by simply stopping the exposure. Chronic hazards include cancer, birth defects, gene mutations, and various organ and systematic diseases. These chronic hazards are often subtle in onset, difficult to clearly relate to the cause and usually impossible to reverse. They may be affecting the population at large through exposure via food products and in the environment. Details of these

serious effects are described later. Characteristically, acute poisoning is caused by either a single large dose or frequent small doses, while chronic effects are usually the result of long-term exposure to small doses. However, these are not hard, fast rules. For example, a single dose of any amount of a cancer-causing substance can cause cancer in a person ten to thirty years after the exposure. (25) Another substance might cause nerve damage in a single dose that could result in chronic nervous disease. Another might cause genetic mutation which would only show up in subsequent generations.

There are additional hazards from the breakdown products of pesticides. These are substances that form intermediate steps in the decomposition of pesticides into normal, non-toxic substances. They can be longer-lived and more dangerous than the original compound. (26) (See Section III for specific examples)

Misuse of pesticides is also a problem. Pesticides are dumped along with other toxic chemicals in the face of regulations or common sense. (27) Misuse by uninformed applicators with inadequate training is all too common. (11)(28) Thus, high concentrations of pesticides can occur in local areas. (27)(12)

Some pesticides, especially the organochlorine compounds are magnified in the food chain. If a pesticide is retained in the body of an animal rather than excreted, then it will tend to become more concentrated in each successive predator. This is because a single predator eats many of its prey, each of which makes its contribution of pesticide to the predator. This is a problem of global proportions. (12)(3)

Some pesticides (and pharmaceutical drugs) that are considered too dangerous for use in the industrial world, are exported to the third world where they are applied without proper precautions or understanding of their nature or use. Manufacturers can frequently sell these products without any cautions to the user at all. Ironically, most of the treated crops in the third world carry these banned pesticides back to our supermarkets. (29)

Photo by Jeannine Caldbeck

CANCER, MUTATIONS AND BIRTH DEFECTS

Cancer

Cancer is a disease in which the cells, through damage to the genetic material, are no longer subject to the normal controls of the body which limit growth, but reproduce themselves without regard for the rest of the organism. As they increase in numbers, the cancerous cells invade normal tissue and interfere with its function. Small clumps of cancer cells can break off from the parent growth and be carried by the blood or lymphatic system to other parts of the body and become established as secondary growths. If this happens, death is often the result. (30)

Carcinogens

As much as 70-90% of cancers are caused by environmental agents, called “carcinogens”. (25) These substances may be natural or man-made and can be influenced by environmental, occupational, cultural, dietary and lifestyle factors. (31) Since there are a limited number of carcinogens and specific influences, it is possible, in principle at least, to prevent cancer. (32)(30)(25)(18)

Prevention should be our major concern rather than cure. (25)(30)(33) We have a long way to go before the mechanism of carcinogenesis is understood (34), and this is essential before cures are invented. (It is possible that we will stumble upon a cure, but this would be mere luck.) Whatever kind of therapy you care to consider, “proven” or “unproven”, chemotherapy or laetrile, radiation or chaparral tea, the cure rate for most cancers is not very good. As with infectious diseases, prevention is the name of the game. (33)(25)

Some of the known causes of cancer in humans are aflatoxins (especially aflatoxin B1 which occurs in peanut butter) (35)(25), asbestos, benzene (which is in many consumer products including solvents, carburetor cleaners, paint removers, adhesives and non-leaded gasoline), tobacco, vinyl chloride (polyvinyl chloride (PVC) always contains free vinyl chloride) soot, various pharmaceuticals, sunlight, and ionizing radiation (x-rays, radioactive fallout, etc.). Other substances suspected of causing cancer in humans include various pesticides (especially those that contain dioxins, such as 2,4-D), natural and synthetic estrogens (used as oral contraceptives, for "estrogen replacement therapy" in menopausal women, and as a growth stimulant to poultry, cattle and hogs), certain food additives (36), bracken fern (if you eat it), and chlorinated drinking water. (37) There are many other known and suspected substances, but this should give you an idea of the scope of the problem. (25)(38)

It is important to note, however, that only a very few of the many substances in the world are carcinogens. Unfortunately, some of these are now very common in our environment.[3]

After exposure to a carcinogen, there is a long latency period before cancer will develop. Time scales of 20 to 30 years are not unusual. (25)(33) The cancers we see today are the result of environmental factors in existence decades ago.

Threshold Response

There is a theory boosted by industry that there is a so-called "threshold response" to carcinogens. The theory holds that at certain low doses of a carcinogen, no response, that is no cancer, will occur. The idea is that a carcinogen has to overwhelm the natural defences of the body before a cancer can be produced. Thus, at certain low levels of exposure, the carcinogen is indeed safe, i.e. no risk exists. (40)

There is no convincing evidence of this effect. The best evidence indicates that the smallest amount of any carcinogen can cause cancer in a single exposure. Additional or larger exposures simply increase the chance of the event. (41) There is simply no such thing as a "safe" level of a carcinogen. (25)(42)

Changing Mortality Rates

The mortality from cancer has increased dramatically in the last century. Once a rare disease, it now causes 20% of all deaths in North America. (33) That this is a disease of civilization is unquestioned,

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[3] The fatalistic response of some people that "everything causes cancer, so why bother?" is based on a profound misunderstanding of the problem. Most cancer can be prevented right now. (33)(25)(39)(46)

but to say that our ancestors were relatively free of disease and that civilization is the cause of cancer is a misrepresentation of the facts.

Three or four generations ago scarlet fever, diphtheria, tuberculosis, typhoid fever and dysentery were major causes of death. (13) In 1900, the top four causes of death in the U.S. were: 1) influenza and pneumonia, 2) tuberculosis, 3) gastrointestinal diseases, and 4) heart disease. (Cancer was number eight.) These diseases accounted for nearly 40% of all deaths. (25) Furthermore, infant mortality was 13% in the first year of life. (5) By today's standards, a large proportion of deaths occurred in childhood. (30)

One of the great benefits of science has been the virtual elimination of infectious diseases as a significant cause of death. This was brought about through an understanding of the disease process and the application of public health measures and changes in personal habits. Vaccines and antibiotics merely completed the process. (33)(30)(43)

Today, the four major causes of death are: diseases of the heart 37.9%, cancer 19.8%, stroke 9.9%, and accidents 5.5%. (25) This is a greatly improved situation. One hundred years ago, adult deaths were spread fairly evenly over all age groups, while today most deaths occur in old age, especially those of disease origin. (30) Furthermore, the infant death rate is now down to 2% in North America. (5)

When I say that this is an improved situation, I am expressing a commonly held belief that a twenty-year-old dying of leukemia or accident is a greater loss than a ninety-year-old dying of cancer of the prostate or heart attack. Our lives are improved in that nearly everyone can live to an age which used to be the privilege of a lucky few.

We can carry this one step further. The raw mortality rates can be weighted to reflect the untimeliness of death. We can do this by counting all the deaths before the age of 20 as the loss of 45 years, all the deaths after 65 as no loss, and each death between 20 and 65 as a loss according to the number of years before 65 that it occurred. When looked at in this way, accidents head the list at 26%, followed by heart disease at 19%, infant mortality at 14% and cancer at 13%. (30)[4]

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[4] Of course, everyone dies. The cause of concern is that many people die prematurely of disease to say nothing of the suffering involved. Mortality rates for all causes, in 1900 or now, are exactly 100%.

Cancer is a significant cause of death primarily because people now survive infectious disease long enough to get it. Cancer is a disease of civilization, but it is also a disease of old age (44), and old age is a boon of civilization. However, cancer rates have been increasing over the years independent of the age group, and as we shall see, these increasing rates are reflections of undesirable changes in environmental factors. (25)(45)[5]

The accompanying graph is "age-standardized", meaning that the data have been weighted so that longevity is eliminated as a factor. (The data are based on the calculation of death rates for a standard population with a fixed percentage of people in each age category.) The spectacular drop in stomach cancer mortality is unexplained. (47) The even more spectacular rise in lung cancer mortality is almost entirely due to smoking. (33)(48) The decrease in uterine cancer is a reflection of the success of screening programs, early treatment, and the increasing frequency of elective hysterectomy for non-malignant disease. (47) Although the mortality rate has dropped for this and some other types of cancer, for most types of cancer, improved therapy has not significantly changed the mortality rates. Mortality rates continue to be a very good indication of incidence rates. (49) What is striking about these results is the fact that for most types of cancers the rates are approximately the same now as in 1935, and indeed, the rates have remained the same since at least 1900 when records began to be kept. (46) This means that carcinogens were widespread in the environment in the late 19th century.

From these results, it is obvious that the overall age-adjusted cancer mortality rate is indeed greatly increased over former times and that the main force of the increase is due to the increase in lung cancer. At least 80% of these cancers are caused by cigarette smoking. (50)

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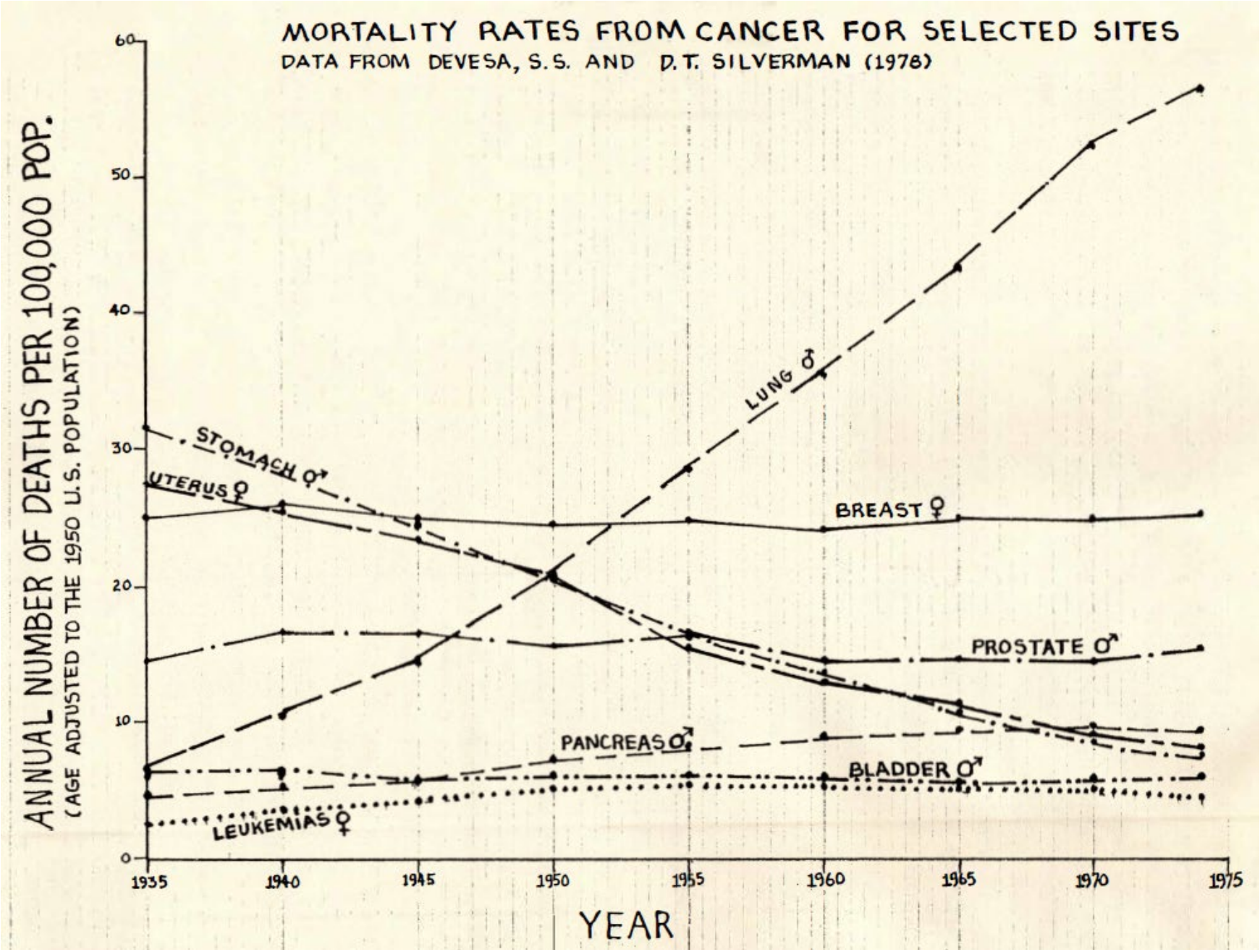
[5] Note that the rate is the proper statistic, not cases. The number of cases of cancer is increased by population and rate increase. (45) Therefore, it is not a meaningful gauge of increasing incidence.


MORTALITY RATES FROM CANCER FOR SELECTED SITES
DATA FROM DEVESA, S.S. AND D.T. SILVERMAN (1978)
ANNUAL NUMBER OF DEATHS PER 100,000 POP.
(AGE ADJUSTED TO THE 1950 U.S. POPULATION)
60
50
40
30
20
10
0
1935
1940
1945
1950
1955
1960
1965
1970
1975
YEAR
LUNG ♂
STOMACH ♂
UTERUS ♀
BREAST ♀
PROSTATE ♂
PANCREAS ♂
BLADDER ♂
LEUKEMIAS ♀

Smoking

One cannot talk seriously about cancer, or even health in the twentieth century without mentioning the effects of smoking. Tobacco smoke is the preeminent carcinogen in this century and is responsible for a third of all cancer deaths. (51) If you smoke cigarettes, you increase your risk of dying of lung cancer ten to fifty times. (33)(52) Cancer of the larynx, mouth, esophagus, bladder, kidney and pancreas are all more common in smokers than in non-smokers. (53)

John Carins, one of the foremost experts in cancer epidemiology and etiology put the contribution of cigarette smoke in perspective in his book "Cancer: Science and Society":

> "Indeed in retrospect, it is almost if Western societies had set out to conduct a vast and fairly well-controlled experiment in carcinogenesis bringing about several million deaths and using their own people as the experimental animals." (54)

Smoking is not only a serious cause of cancer, but also contributes to heart disease. One study found - and this is typical - that men who smoked up to a pack a day were 2.5 times more likely to have a heart attack than non-smokers. For those who smoked more than a pack a day, the rate was 3.2 times higher than for non-smokers. Smoking is also the major cause of chronic bronchitis and emphysema. (52)(25)(55) Women who smoke have about 75% more chronic sinusitis (inflammation of the sinuses) and 50% more peptic ulcers. (55) The risk of smoking is, in general, a 70% increase in the probability of dying at any age. For a two-pack-a-day smoker, the increase is 100%. (56)

Smoking has serious effects on fetal development.There is evidence suggestive of fetal brain damage. (57) Women who smoke during pregnancy greatly increase their chance of miscarriage - 80% more likely than in a non-smoker. (58) They run a significant risk that their babies will be underweight, premature, or die at birth or soon after. (25)(52)(59)

Non-smokers, whether unborn, children or adults, are involuntarily exposed to significant amounts of tobacco smoke, and the effects are extremely serious. (52)(59)(60)(61) Whatever size of group you consider - family to nation - this principle applies: the chances of you dying from lung cancer are directly related to how many members of your group smoke. Even in public buildings smokers overwhelm the ventilation systems in a very short time and expose non-smokers to a significant air pollution burden. (61)

Perhaps the most serious effect - at least in the long run - is the effect of cigarettes on the gene pool. Cigarette smoke is highly mutagenic arid there is every reason to believe that it mutates the reproductive cells of both men and women. (62) Bruce Ames, who developed one of the most important and useful tests for mutagenesis commented:

> "I'd be surprised if there are very many things in the modern chemical world that do as much damage as cigarettes. It's clear that they are causing life-shortening, and I think more and more evidence will come out for genetic effects in the children of smokers." (63)

The financial costs of cigarettes are alarming. Estimates of the total annual costs from cigarettes in the U.S. (fire, health care, health research, etc.) are in the $20 billion range. (64)

Government and industry have distorted and suppressed the facts. (25)(39)(18)(65) For example, in England, steps to reduce cigarette consumption, primarily through education, were not taken partly because cigarettes tend to kill off people, thus saving huge amounts in social security payments. (66) Old people don't generate much revenue for the state.

The tobacco industry is extending its very efficient and effective advertising into the third world. High-tar cigarettes - the most addicting and most harmful to health - are being offered for sale. Millions will die prematurely from smoking these cigarettes. (18)

In a World Health Organization (WHO) report last year, there was the warning that:

> "… smoking disease will appear in developing countries before communicable disease and malnutrition have been controlled, and the gap between rich and poor countries will thus be further expanded." (67)

Photo by Ted Davis

Increasing Incidence and Mortality of Cancer

Although smoking and increased longevity are largely responsible for the increase in cancer incidence and mortality over the last few years, there is an increase in the rates of many types of cancer that cannot be explained by these phenomena. (68) One group of scientists - the so-called "environmentalists" - argue convincingly that industrial chemicals, drugs, pesticides and food additives have made a large and serious contribution to the cancer problem. (48)(60)(69). Samuel S. Epstein, author of The Politics of Cancer (25) and a well-known expert in the field, is the most outspoken of this group. An opposing camp, that of the "lifestyle theorists", led notably by Richard Peto, reader in cancer studies at the University of Oxford, believe that there is no good evidence that these industrial pollutants have produced significant numbers of cancers to date, and that increased rates have more to do with diet, smoking and occupation (i.e. lifestyle) than with environmental pollutants. (18)(70)

Predictably, the chemical industry and agribusiness support Pete's views, in spite of the fact that he is very critical of them. Equally self-serving is the tobacco industry[6] which supports the

[6]In passing, I should point out that all these scientists - Epstein, Peto, Doll, Carlins, etc. - whatever their differences concerning incidence and mortality rates and their causes - use the strongest language I have ever seen in scientific journals on any subject condemning tobacco smoking, government policies concerning tobacco, and especially the tobacco industry. Also see (72).

environmentalist camp in their attempt to divert attention away from themselves. (47)(18) Peto points out that while diet (probably) and smoking (definitely) are the major causes of the age-adjusted cancer increase in this century, lack of concern about industrial pollutants now may result in increased rates in the future. (71)

Regardless of the outcome of this debate, at the very least, prudence demands that we treat these environmental pollutants with a great deal of caution. It is irresponsible to allow exposure to known human or animal carcinogens, especially when alternatives are available. Unfortunately, the large corporate interests have almost always put financial advantage before human health, and governments have bent to this pressure. (25)(18)(11)

Pesticides and Cancer

There are four reasons that pesticides are especially worrisome in terms of carcinogenicity. First, some are known carcinogens or mutagens in experimental animals. Second, many of them are not adequately tested for their toxicity. Third, some of them last a long time and therefore can build up to large concentrations in the food chain, and fourth, they are widespread throughout the environment.

Some of these pesticides are controlled or banned in the industrial world but are "dumped" in third-world markets. These pesticides return to us on imported foods. (29) It is important to note that for many pesticides, some of which are well tested, there is no evidence of carcinogenicity at all.

Treeplanters and Cancer

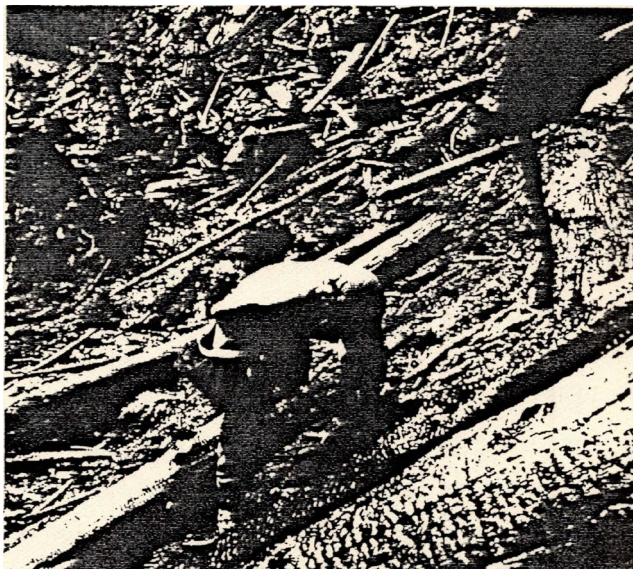


Photo by Doug Cowell

So what does this have to do with treeplanters? First, treeplanters, like everybody else, are involuntarily exposed to carcinogens. Some of these carcinogens are in the natural environment, some are created by modern industry and technology. All of us, whether we like it or not, are exposed to cigarette smoke. Depending on where and how we live, we will be at a greater or lesser risk of getting cancer. Since some of the pesticides used on the trees cause cancer in experimental animals, they may therefore cause cancer in humans. Until there is good evidence to the contrary, treeplanters must assume that

they are at an additional risk of getting cancer. Planters working on burned sites are probably at additional risk: soot and tars are well-known carcinogens. (73) This health hazard deserves serious consideration.

Other occupations are of course, riskier. Pesticide applicators, formulators, and agricultural workers are some obvious examples. It is important to remember that 1) any amount of a carcinogen can cause cancer, 2) the chance of getting cancer is directly related to the amount of exposure, both in quantity and time, and 3) that carcinogens are at least additive and may be synergistic[7] with each other or other substances.

Mutations

Although the cancer problem is serious, the problem of genetic mutation is far more dangerous because it alters the cell at the fundamental level, and this defect can be passed on to subsequent generations. (62)(59)(74)

Within each cell of our body are structures called chromosomes. Chromosomes are made of a molecule called DNA. Genes are sections of the DNA molecule that control virtually every aspect of our structure and physiology. Hormones, stomach acid, hair colour, intelligence and behaviour, etc., are controlled or strongly influenced by the genes.

Each cell of the body contains a complete set of chromosomes containing all the genes possessed by an individual. Each time a cell divides there is a careful and exact replication of the DNA. When there is a mistake in the replication process, damage to the chromosome, or a change in the DNA itself, we have a genetic mutation. If this mutation occurs in the reproductive cells, it can be passed on to offspring.

It is a long-standing notion of genetics that most mutations are lethal or harmful. “Bad” mutations far outnumber “good” mutations. The reason the “good” mutations are not lost in the forest of “bad” mutations is that the “bad” mutations produce individuals that do not survive and reproduce as well as the individuals with the “good” mutations. This is natural selection - the main force of evolution and the foundation of biology in this century.

Many of the mutations that would survive in humans would not be obvious. Mutations such as two heads, three legs, and so on, are

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[7] Synergistic means that the substances can act together to create a stronger effect than a simple addition of their effects would produce.

usually lethal or only last a generation. It is the subtle, recessive mutations that are the serious kind. With this kind of mutation, you or your children appear normal, but your grandchildren may be retarded or susceptible to disease. These mutated genes are essentially impossible to identify, very hard to eliminate from a human population, and persist for generations. If our genes are mutated on a large scale, we would expect to see an increase in the number of spontaneous abortions, along with a general rise in the incidence of disease of all kinds (75), and a lowering of intelligence. (76)

Like cancer, mutations are produced by environmental agents. (77)(74) Nearly everything that is carcinogenic is mutagenic, and most substances which are mutagenic are carcinogenic. A given chemical substance may be carcinogenic, mutagenic, neither, or both. Both cancer and mutations seem to be the result of DNA damage.

For practical purposes, many investigators believe - and this is my opinion as well - that unless there are strong reasons to think otherwise, it is best to assume that if a substance produces cancer in experimental animals, it will also produce cancer in humans. (88)(91)(90) If a substance produces cancer, mutations, or serious disease in experimental animals, even in a single species (91), the logical thing to do is to ban or severely restrict the use of the substance.

A large number of genetic mutations in the human pool would occur from the radioactive fallout of a nuclear war. Less catastrophic, but equally irreversible mutagenic damage may be occurring right now from various human-produced pollutants. Most of the carcinogens in our environment are producing mutations (and may be contributing to the major cause of. death in Canada and other Western countries - atherosclerosis[8]). (95)

The seriousness of this problem cannot be overstated. Damage to the human gene pool would be catastrophic and take hundreds to thousands of generations to repair - if indeed, repair is possible. (78)(77)

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[8] Atherosclerosis is an extremely common form of arteriosclerosis (hardening of the arteries). Atherosclerosis is the major cause of heart attacks and strokes.

Photo by Doug Cowell

Birth Defects

A mutation may show up as a "birth defect". Substances that cause birth defects are said to be teratogenic and are called "teratogens". They can produce their effects through gene mutation or through other physiological processes. Thus, a teratogen is not necessarily a mutagen, but may be. Mutagens are always suspect teratogens.[9]

For pesticides, there is little direct evidence of teratogenesis in humans. (79) However, teratogenetic effects are very difficult to demonstrate. For practical purposes, mutagens in any system must be treated as teratogens in humans. Many pesticides have not been tested for teratogenesis in animals, but some that have been tested have produced birth defects in experimental animals. (79) Until proven otherwise, we should assume that these will also produce birth defects in humans.

[9] For a clear and non-technical discussion of mutagens, teratogens, and genetic toxicity, see Co-Evolution Quarterly, No. 21, Spring 1979.

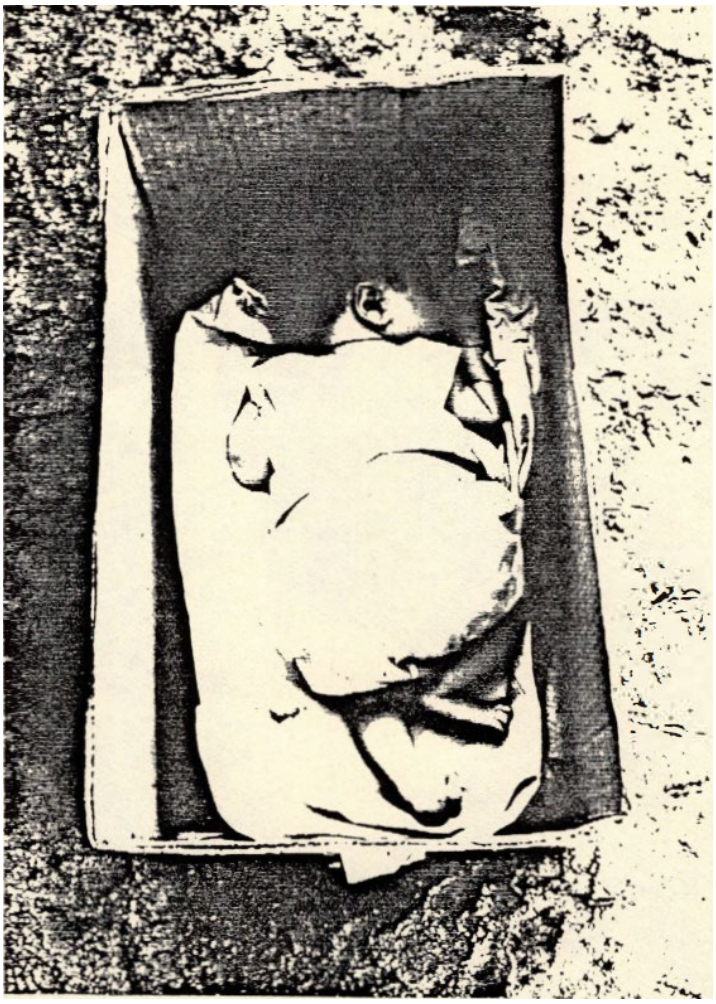

Photo by Doug Cowell

Toxicity Tests and Their Limitations

There is a huge variety of tests that are done with pesticides to determine their potential for harm. (80) The simplest test involves exposing a group of test animals, usually rats or rabbits, to ever-increasing amounts of the test substance. The amount of chemical that kills one-half of the animals is called the "lethal dose to 50%" and is written LD_{50}. The dosage is usually expressed as mg of chemical substance per kg of bodyweight. (Mg/kg is exactly equal to "parts per million" (ppm)).

Thus, a high LD_{50} value indicates that a substance is relatively safe in terms of acute toxicity, while the opposite is true of a low LD_{50}.

Below are some LD_{50} values for some common pesticides and other substances.

Oral LD_{50} values for selected substances (1)

	ppm
antifreeze (ethylene glycol)	3,460
aspirin	1,750
Benlate	10,000
boric acid	3,000
captan	9,000
copper sulfate ($CuSo_4$)	300
DDT	113
diazinon	250-600
hydrogen cyanide	4
nicotine	50-60
Paris Green	22
Roundup (glyphosate)	4,320
Sevin (cabaryl)	560
sodium chloride (table salt)	3,320
thiram	375-865
2, 4-D	375

These LD_{50} values are for oral exposure (feeding). Values for exposure to the skin (dermal) or lungs (inhalation) will produce different values and different relative toxicity between different substances. Substances with LD_{50} values of under 100 are considered very dangerous, while those with values above 1000 are considered relatively safe. (1)

The LD_{50} values are useful as a general guide to the acute toxicity of a substance. Yet these values say nothing about the effect of repeated exposures, accumulation, nor the effect on the body. For instance, eating too much table salt (LD_{50}=3,320) can make you dehydrated, which is dangerous, but the salt is excreted rapidly. DDT (LD_{50}=113) is not excreted and interferes with the nervous system. Captan (LD_{50}= 9,000) would be given in very large doses to show any acute effect but causes cancer in mice. (81)

The obvious way to determine if a pesticide causes cancer, mutations, birth defects or other diseases is to feed or otherwise expose the animals to the pesticide and, after a suitable period of time, examine them for abnormalities. Such an experiment is called a “bioassay”.

Bioassays are frequently done, but the results are often difficult to extrapolate to humans. (82) Some scientists believe that positive results are not predictive, especially if done with mice. (40)(83) Large doses of the pesticide are often necessary to produce cancer, but these doses may be overwhelming the animals’ natural protective systems or simply be creating a stress, which in itself can cause cancer. (84) Also, large doses of pesticide may be upsetting the natural physiology of the animal and thus predispose it to cancer. The

effects of these large doses would never be seen in humans because we would never be exposed to such relatively large quantities of the substance.

However, large doses are necessary for several reasons. Because relatively small numbers of animals were used, to actually prove that a substance is carcinogenic a lot of them have to get cancer. Thus, several dose levels are tried. Another reason for large doses is that they may compensate for the faster metabolic rate and the shorter life span of rats and mice, relative to humans. However, some substances may not produce cancer in the experimental rats and mice, regardless of the dose, simply because they don't live long enough to get cancer. (Remember that cancers in humans can have a 20-to-30-year delay time.) Some argue that the high doses would, in a very sloppy way, compensate for unknown synergistic and cumulative effects. Others argue convincingly that the unknown synergistic and cumulative effects are not compensated for at all, and that despite the apparent sensitivity of these tests, they are not sensitive enough. (76)(48)(85)(86)

Another problem is with the genetics of the experimental animals. To get clear results, strains of rats and mice are used that have very uniform genetics. But the genetics of humans are highly heterogeneous. The test animals might have a gene that would protect them from the effects of a particular substance, or it might be the reverse. In any case, the extrapolation to humans would be incorrect. For example, aniline dyes produce cancer in experimental animals only with extreme difficulty, but among workers with long-term exposure, the rate of bladder cancer approaches 100%.

To get around this problem, different species of animals are tested. Still, we may be unable to predict the effects accurately in humans. (85)(5)(94)

The seriousness of this problem was summed up by the then director of the National Cancer Institute (NCI) Arthur C. Upton:

> "A given exposure to a carcinogen may cause a very low incidence of tumours in one species, whereas the identical exposure may cause a very high incidence in another species. An estimated risk of 4.2 cancers, for example, per 220 million people, as calculated by extrapolation from mouse and rat data, might turn out in reality to be as low as no human cancer, or as high as 420,000 cancers. Although the occurrence of very large errors should be rare, each such error would be a catastrophe. One would not know such errors had occurred until many years after human exposure." (87)

False-negative results have occurred (where the substance produces cancer in humans, but not in rodents), but no evidence has been found of false-positive results, nor is there any evidence on which to estimate the frequency or classes of agents likely to produce such results. However, false-negative and false-positive results are the exception. For most substances, the magnitude of the response in the most sensitive animals tested is reasonably comparable to the response in humans. (88)

A rule of thumb used by the U.S. Environmental Protection Agency (EPA) is that humans may be tenfold more sensitive than the experimental animals used and that there may be in addition a tenfold variation in sensitivity among individuals. (74)(89) This means that some individuals maybe 100 times more sensitive to these tests than are the animals used.

Mutation Testing

Another way to get at the cancer problem is through tests for mutations. (93) Since cancer and mutations often are the result of DNA damage, it is not surprising that a substance that causes mutations will often cause cancer as well. In fact, about 85-90% of mutagens are carcinogens. (96) If a substance is a mutagen, then we should probably treat it as a carcinogen unless we can prove otherwise. (74)(97)

Mutagenicity can be determined in a test called the "dominant lethal mutation system". This involves treating male mice with the suspect substance, then breeding these male mice with female mice.

Since most mutations are lethal, a measure of the mutated male sperm is the number of reabsorbed fetuses in the females. These show up as small black dots in the uterus of the female mice. (75)(98)

Another very useful test done with mice is called the "specific locus method". This test involves mating treated wild-type mice, of either sex, to a strain of mice that breeds true to certain recessive traits that are easily detected, such as coat colour. Since the trait is recessive to the wild type, the trait will only appear in the offspring of the first generation if a mutation to the specific trait occurred in the wild type. This test has the major advantage that the mutant animals can be seen at a glance, thus saving time and labour and virtually eliminating the personal bias that can affect the scoring of mutants in other test systems. However, because only a few genes are studied at a time, large numbers of animals are required. (99)

Mutation testing can also be done with microorganisms. (100)The most common test is called the "Ames Test", developed by Bruce Ames and his associates at Berkeley. The organism used in the Ames test is

a bacterium called Salmonella typhimurium. In the simplest system, a strain is used which is unable to synthesize the essential amino acid histidine. The bacteria are placed in a histidine-free environment, and because they must have histidine to reproduce and grow, they do nothing. If a mutation occurs that reverses the defect, the bacteria start to grow and after a few days, you can see the bacterial population as a circle of opaque material in the culture dish. This elegant system has been refined by adding liver homogenates to the bacterial culture to reproduce some of the metabolic processes going on in mammals. Another method (called the "host-mediated assay", involves putting bacteria into the mouse, treating the mouse with the suspect substance, removing the bacteria, and checking for the histidine mutation. (101)[10]

There are other ways to detect genetic damage. Sperm and chromosomes can be checked for abnormalities. (102) Cells can be checked for the presence of micronuclei, which could indicate chromosome damage. Fruit flies, mice, and other animals can be bred and the young can be checked for genetic changes. (103) Other systems using bacteria and yeast are also being used. (75)(76)(104) Even higher plants, such as corn or barley, may be used to detect mutagens dangerous to humans. (105)

No one test by itself is adequate to determine the mutagenic or carcinogenic potential of a given substance, but together they can be a powerful tool. (112) Unfortunately, these tests are costly[11] and require a great deal of technical expertise. There are about 50,000 untested commercial chemicals in use and a few thousand more in common use. Every year about 1000 new chemicals are introduced. About 7,000 substances have been tested for carcinogenicity in animals and about 1500 of them are carcinogenic. (This should not be taken as an indication that 20% of all substances are carcinogenic. The 7,000 selected for testing were suspected carcinogens in the first place.)

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[10] A negative response in one test does not mean a substance is no longer suspect. For example, the herbicide atrazine induced dominant lethal mutations in mice but was negative in the Ames test. (107) Some researchers have indicated that the Ames test can miss as much as 24% of the chemicals tested and up to 45% of certain classes of substances. (85) Ames and his collaborators argue that these estimates are based on inadequate studies and that the test actually only misses 10% of carcinogens. (106) This seems to me to be the correct estimation.

[11] One estimate is that a complete battery of tests for one chemical would cost more than $1 million. (85)

However, half of these studies are inadequate and we should probably reduce the numbers to 3500 and 750, respectively. (85)(108)

There are simply not enough pathologists to thoroughly test the estimated 63,000 substances that need testing. (85) However, it is possible to do the simple bacterial tests, and many substances have been tested. (109) Any substance that proves mutagenic or carcinogenic in any test system should be treated with extreme caution.

There is one final way to test for carcinogenicity. By studying large numbers of humans who have been exposed to specific environmental hazards, it is often possible to show that there is a relation between the exposure and the disease. This is called epidemiology. The problem with this approach is that by the time we find out that substance is a carcinogen, a large number of people are already exposed to it. Even if the substance was banned, because of the delayed effect, we would still be getting cancers for another 20 or 30 years. (85) Another problem is that to be detectable, the substance has to increase the cancer incidence by at least 100% (that is, a doubling of risk). A weak, but serious carcinogen would never be detected by epidemiology. (76)

Teratogen Testing

Tests for teratogenicity (birth defects) are done with rodents in much the same way that tests for carcinogenicity are done. The problems and limitations are much the same. Thalidomide, which is not a strong teratogen in rodents, is a strong teratogen in humans. Many substances which cause birth defects also cause fetal death and therefore might not be detected. A mutagen is always suspect as a teratogen. Many teratogens, however, have nothing to do with cancer or mutations. They are often very difficult to identify. (75)

One further complication is the shameful involvement of the chemical manufacturers and their associates. This is not the place to get into this complex area in detail, but one must be very careful in reviewing research sponsored by the chemical companies, and in some cases, the universities. (110) The EPA, based on a U.S./Canadian government audit, fortunately, has thrown out from its consideration a large number of experiments done by Industrial Bio-Test Laboratories (IBT), as being poorly designed and executed. (111) These laboratories often found that the substances they tested had ‘no effect’. (See Appendix I)

Photo by Jeannine Caldbeck

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9. Allen, R. (1980), pp. 40-43; CEQ 1980
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11. Van den Bosch, R. (1978)
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13. Valentine, J.W. (1978)
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28. On July 4, 1980, the Vancouver Parks Board sprayed the numerous oak trees on the street in 3900 and 4000 blocks on West 32nd Avenue, in Vancouver, B.C. Several regulations clearly printed in the Handbook for Pesticide Applicators were ignored. They did not stop spraying when the wind came up, the spray drifted into open windows, onto fruit trees, gardens, lawns and pets. The residents were not notified. (Ronald Hyatt, personal comm.) Another close-to-home example: 2,4-D applicators (chemical thinning) have been known to squirt each other as a "joke". (Dirk Brinkman, personal comm.)
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51. Carins, J. (1981)
52. Winter, R. (1980)
53. Bennett, W. (1980); Doll, R. (1978); p. 3580
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57. Norwood, C. (1980) pp. 12,56
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64. Epstein, S.S. (1979)
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69. Albert, R.E. et al. (1977); Ames, B.N. (1979); Fairchild, E.J. (1976); Smith, R.J. (1980f); Epstein, S.S. (1979), p. 17
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91. Fairchild, E.J. (1976)
92. Anon. (1976b)
93. EEMS (1978); Legator, M.S. (1974); Legator, M.S. and S. Zimmering (1975); Mutation Research, Vol. 41 (1976) contains nearly 50 articles on new developments in mutagenicity testing of environmental chemicals.
94. Eckardt, R.E. (1976)
95. Benditt, E.P. (1977)
96. Ames, B.N. (1975); Hooper, N.K. et al. (1979); deSerres, F.J. (1976); Waters, M.D. et al. (1980)
97. Shubik, P. (1977)
98. Bateman, A.J. and S.S. Epstein (1971); Epstein, S.S. et al. (1972)
99. Cattanach, B.M. (1971); Ehling, U.H. (1980)
100. Waters, M.D. et al. (1980); Devoret, R. (1979)

101. Ames, B.N. et al. (1975); Donahue, E.V. et al. (1978)

102. Yoder, J. et al. (1973); Wyrobek, A.J. and W.R. Bruce (1975)

103. Bumb, R.R. et al. (1980)

104. Fishbein, L. et al. (1970); Nagao, M. et al. (1978)

105. Grant, W.F. (1978); Shaw, M.W. (1970)

106. Waters, M.D. et al. (1980); Hooper, N.K. et al. (1979)

107. Ehling, U.H. (1980)

108. Rose, V.E. (1976)

109. Mccann, J. and B.N. Ames (1976); McCann, J. et al. (1975); Ames, B.N. et al. (1975)

110. van den Bosch, R. (1978); Carson, R. (1962); Epstein, S.S. (1979); Peto, R. (1980); Agran, L. (1977); Gilvert, B. (1979); Henig, R.M. (1979); Martin, B. (1980); Graham, F., Jr. (1977); Anon. (1979b); Stadfeld, C.K. (1976)

111. EPA (1980), p. 13

112. Weisburger, J.H. and G.M. Williams (1981)

113. Walsh, J. (1981)

114. Barnes, R.D. (1981)

SECTION II: PESTICIDE USE ON FOREST SEEDLINGS[12]

On January 30, 1980, I discussed pesticide use with Hank Schroeder, superintendent, Surrey Nursery. Most of the following information came from that conversation. Hank definitely has an appreciation of the larger problems of pest control. Commenting on methyl bromide (it kills virtually everything in the soil and is used in the Bellingham nursery), Hank said: "I don't understand why they do it. What makes the soil is the organisms. Without organisms, it's just dead rock."

Pesticides at the Surrey Nursery (which grows 30 to 50 million trees a year) are used as little as possible and at rates often below the recommended doses. They don't engage in large-scale broadcast, indiscriminate use of pesticides. Hank indicated that the chemical companies probably set higher application rates than necessary.

The nursery has more restrictions on what they can use than do farmers. For example, the nursery can't use Furadan (which has a long life) for strawberry root weevils, or the toxic pesticide Cygon. The private farmer can use anything he wants, in amounts he thinks will work, within the law.

Government pesticide use is more tightly controlled than is private use. Farmers can spray pesticides on their own land without doing much more than having a pesticide applicator's license. The B.C.F.S. and forest companies have to have each pesticide approved by the Pesticide Control Branch. That is no great solace, but at least it is on public record. The branch does refuse requests for certain pesticides if there are less toxic alternatives. The case of Furadan (carbofuran) and Cygon are examples.

The Surrey Nursery grows trees in two different environments: fields and containers. The stock grown in the field is generally known as bare-root, but may become mud packs. The container stock is what we usually refer to as plugs. The containers are styrofoam boxes with a hole for each individual tree. The containers are housed in either open sheds or greenhouses. Because the two environments are different, each has its own unique pest problems and solutions, although some are common to both.

Bareroot stock is treated with propazine, Diazinon, Orthene 80 and Sevin (on a restricted basis). Pre-emergent bare-roots are treated with A.W.K. No. l and newly cleared land is treated with Vapam.

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[12] Most of this section appeared in the PRWA Newsletter, Winter/Spring 1980.

Container stock is treated with Diazinon, Sevin, Captan, Benlate and Daconil.

The details are given below:

A.W.K. No. 1 (Agricultural Weed Killer No. 1) is a light petroleum oil that is applied at a rate of 40 gal/acre to kill broadleaf weeds in the fields for pre-emergent fir and spruce. Fir is treated 14-21 days after sowing. It also has the benefit of killing springtails (Order Collembola) which are small primitive wingless insects that eat the leaves of the seedlings.

Diazinon (Speracide, Basudin) is a common organophosphorus insecticide. Aphids attack the leaves of seedlings in May or June. Ladybugs eat the aphids, so if there is a good population of ladybugs, the insecticide is not used. Usually, it is used once or twice a year and sometimes up to four times a year. The aphids show no sign of resistance so far.

Diazinon is also used in a single application in October to control the European Crane Fly (Tipula paludosa) at a rate of 24 oz. a.i./acre. These insects (the larvae are sometimes called leatherjackets) attack the roots of the seedlings.

Acephate (Orthene 80) is a water-soluble contact and systemic insecticide. It is used only on the bare root stock for the Strawberry Room Weevil (Brachyrhinus). This is a common pest throughout North America, which attacks strawberry, grape, raspberry, apple and peach, as well as forest seedlings. Adults girdle the seedlings about 1/4" below the ground level, and the larvae attack the root tips.

Before 1979, the population had been building up for four or five years and was then treated with Orthene. There was an application in 1980, and that is expected to be the end of the problem for several years.

Sevin (carbaryl) is a carbamate insecticide with relatively low toxicity to mammals, but very high toxicity for honeybees. It is applied at a rate of 1/2 lb./greenhouse or 2 lbs/acre. It is used on a very restricted basis for two insects: cutworms (larvae and moth) and June bug larvae. The cutworms occur in the containers anytime between May and October and are treated only where they are found. The June bugs (a beetle) occur in June, cutting off the stem and advancing 2-3 feet/day. Holes are laboriously punched in the soil to get the poison to a proper depth. Sevin is only used in areas where the insects are found. There is usually only one outbreak a year.

Metham (Vapam, SMDC, Metam) is a herbicide, insecticide, or nematocide, depending on the concentration. If needed, it is used for nematodes in a single application after land clearing. Nematodes are

very common roundworms (not earthworms). The roots of fir seedlings are attacked by the dagger nematode (Xiphinema). Approximately 50% of the newly cleared land is treated. The last use of metham was about five years ago. Additional applications are not necessary as long as the soil is cultivated and double cropping is avoided. Further use of metham at Surrey Nursery is unlikely.

Captan and Benlate (both fungicides) are used as a mix (Captan: 1 lb./100 gal., Benlate 1/2 lb./100 gal.) at a rate of about 100 gallons for 500,000 seedlings which cover an area of about 30x200 ft. Only container stock is treated. It is used to control grey mold (Botrvtis cincera). There are two applications. One application controls the mold in the spring during storage. The bareroot seedlings don't have to be treated after lifting because they are frozen and this controls the mold. In the past, container stock was also frozen, but was frequently killed in the field by being thawed too quickly or planted while the trees were frozen. The top became active and begun to transpire while the roots were still frozen. This dehydrated the tree and killed it. If the trees were thawed at the storage site for a week up to ten days at 40° F, the second application would not be necessary. Right now, there are not enough buildings to do this. The Captan-Benlate mix is the only one known to work.

If bareroot stock was suspected of becoming infested, it was dipped in a Captan-Benlate mix just before being put in the boxes. Charlie Johnson, head of silviculture in Victoria, has indicated that this will not be done in the future. This greatly reduces the risk of the treeplanter exposure to these fungicides. (See letter of July 2, 1981, Appendix IV).

Daconil is a broad-spectrum fungicide for use on turf, vegetables, and ornamentals. It is used only on container stock against a seed-borne mold (Sirococcus). The first application is at the first sign of the disease. This occurs about 2-3 weeks after germination. Daconil is needed 1-3 times each spring. During the summer it is applied every two weeks, especially to Hemlock (which can't be grown outside because there is too much light). All greenhouses are treated. In the open sheds (mostly spruce) there is usually only one application. Perhaps 25% of these seedlings are treated.

Malathion is broad-spectrum organophosphorus contact insecticide. It is not used at the Surrey Nursery, but is used at some nurseries as a replacement for diazinon.

Several triazine herbicides may be used to control weeds. Propazine is applied to bareroot stock after the roots are down about 10 cm. Underground, it kills weed seeds on germination before they

break out of the soil. There are two applications a year at a rate of 1/2 lb. a.i./acre. The first application is in late June or early July. The second application is in September or October just before the winter mulch is added. This herbicide is apparently the most selective of the triazine group and is therefore preferred. Prometryn, prometore, simazine and atrazine are similar to propazine and may be used if propazine is unavailable.

Some chemicals that I have not mentioned are no longer used. Sodium fluorosilicate is now outdated. Dalapon was used 7-8 years ago but not now. Chlorine bleach was used as a sterilant against algae that grows on the surface of containers, but seriously interfered with the seedlings. Since it was only effective in large concentrations, it was abandoned. Next coconut oil soap was tried. That controlled the algae, but also stunted the trees. Now the containers are simply washed. This is effective, but laborious. The best control is control of the humidity.

Other chemicals that are used at the nursery are not in contact with the seedlings. Herbicides, such as Round Up, are used selectively in non-growing areas, such as around buildings and along walkways.

The other nurseries follow the same general practices as the Surrey Nursery. Within my limited experience, the approach seems to be along the lines of the integrated pest control management concept. As non-pesticidal control methods are developed, they will probably be-used.

Metiram (Polygram) and Ferbam (Ferbate, Karbam Black) are carbamate fungicides used in Alberta. Bareroot stock is treated with Benlate and metiram while container stock is treated with Benlate and Ferbam.

SECTION III: PROPERTIES OF PESTICIDES

Introduction

The chemical name used here is the Chemical Abstracts name, 9th collective period, 1972-1976. This is the first name in the Merck Index (Windholz, M. et al. 1976); alternate names can be found there.

References are of two types. References I have read are given in the standard author and date form: eq. Windholz, M. et al. (1976). References given in the form 75-1896 are references to Pesticide Abstracts and the number is the number of the abstract. The first two numbers indicate the year of the abstract, and the last four digits are the serial numbers.

I have not had the time or the resources to do a thorough critical review of the literature for any chemical. The pesticides we are most likely to be exposed to I have reviewed in the most detail; others, such as the triazine herbicides, I have reviewed only with the material easily at hand. For some pesticides, the critical toxicological experiments have not been done, and hardly any have been investigated as thoroughly as we would like.

I have been conservative in assessing mutagenicity, carcinogenicity, and teratogenicity. This is only prudent. It is quite backward to assume a substance is "safe" because there is only weak evidence of a hazard. Lack of an effect in a few experiments does not mean that the substance is no longer suspect. Evidence of a hazard in any one test system should make us cautious. See Section I for a more detailed discussion.

How to Use This Section

On the next page is a list of the pesticides reviewed in this report. Find the pesticide you are interested in and turn to the table of contents to find the page number. In describing a particular pesticide, I indicate to which group of pesticides it belongs. Beginning on the next page are group descriptions. For use in the nursery, refer to Section II.

Pesticides Reviewed in Section III

Groups: carbamates; dithiocarbamate fungicides; organophosphorus insecticides; phthalimides and triazine herbicides.

NAME	ALTERNATE NAMES (Miller, A.V. and S.M. Craig, 1979)
acephate	Orthene
atrazine	Aatrex, Gesprim, Marzone, Primatol A
benomyl	Benlate, Tersan 1991
captan	Orthocide
carbaryl	Sevin
chlorothalonil	Bravo, Daconil 2787, Termil
diazinon	Basudin, Spectracide
ferbam	Fermate, Karbam Black
malathion	Cythion
metiram	Polyram
prometone	Gesagard, Primatol Q
propazine	Milogard, Primatol P
simazine	Gestastop, Primatol S, Princep, Simmprim

Group Descriptions

The carbamates were developed after the organophosphorus (q.v.) materials and act in the same manner. However, the inactivation of the nervous system is reversible at a higher rate and recovery is much more rapid. Carbamates are generally metabolized rapidly in both plants and animals. Carbaryl, for example, is degraded rapidly, most animals excreting a high percentage of an ingested dose within 24 hours.

Mammalian toxicity varies from low to high depending on the route of exposure and the specific pesticide. Many carbamates are highly toxic to birds, and all are toxic to honeybees. They are not likely to accumulate in tissues or the environment. (McEwen, F.L. and G.R. Stephenson (1979) pp. 199-206).

The carbamates are suspected mutagens and carcinogens by N-nitrosation. (Seiler, J.P. 1977) See discussion under carbaryl and metiram.

Dithiocarbamate fungicides can be divided into two groups: 1) the dimethyldithiocarbamates (thiram, Ferbate, ziram), and 2) the ethylenebisdithiocarbamates (nabam, maneb, mancozeb, zineb, and Polyram). The second group can decompose into ethylene thiourea (ETU) and is produced under conditions of storage. ETU can produce goiters

and is teratogenic at relatively low doses in rats. (McEwen, F.L. and G.R. Stephenson (1979) pp. 80-84; 450. Also see "metiram".

Organophosphorus insecticides are a large and diverse group.They are often called "organophosphate insecticides", but the technically correct term is "organophosphorus".

These pesticides were developed in Germany during World War II and are related to "nerve gases". This fact is sometimes cited for its emotional appeal but clouds the issue.

These insecticides act on the nervous system by inhibiting acetylcholinesterase at the synapse, i.e., they interfere with nerve impulse transmission. The same mode of poisoning occurs in insects or vertebrates such as ourselves. Symptoms of organophosphorus poisoning are nausea, salivation, giddiness, breathing difficulty, tremors, tearing, contraction of the pupil, coma, convulsions, prostration and death. (McEwen, F.L. and G.R. Stephenson (1979) pp. 179-182).

The organophosphorus compounds are often very toxic to humans and must be used with extreme caution. Unlike the organochlorine compounds, they do not accumulate in the tissues or the environment. (There are exceptions to this general rule). (Miller, A.V. and S.M. Craig, 1979, pp. 13, 35).

The following classification, signs and symptoms of organophosphorus poisoning are modified from Namba, T. et al. (1971).

(1) Latent poisoning. No clinical manifestations. Diagnosis depends on the estimation of serum cholinesterase activity. Prognosis: Good.

(2) Mild poisoning. The patient can walk but complains of fatigue, headache, dizziness, numbness of extremities, nausea and vomiting, excessive sweating and salivation, tightness in the chest, abdominal cramps or diarrhea. Prognosis: Good.

(3) Moderate poisoning. The patient cannot walk and there is generalized weakness, difficulty talking, muscular twitching, contraction of the pupil and severe symptoms described above. Prognosis: Recovery with treatment; without treatment, recovery may not take place.

(4) Severe poisoning. Unconsciousness, extreme pupil contraction, loss of pupillary reflex to light, muscular twitching, paralysis (flaccid), secretions from mouth and nose, moist rales in the lungs, respiratory difficulty. Prognosis: Fatal if not treated.

There are good drugs for the treatment of organophosphorus poisoning, and an objective test (serum cholinesterase activity is inhibited).

Chronic exposure can induce serious nervous disease. This includes memory impairment, slowed thought, minor difficulties in neuromuscular coordination and other "soft neurological signs". (Davis, K.L. et al. 1978)

Phthalimides include three fungicides: captan, folpet, and captafol. Phthalimides may cause skin irritation and rash on the skin of applicators or field workers. The acute oral toxicity is low. (McEwen, F.L. and G.R. Stephenson (1979) pp. 84-85).

Triazine herbicides are moderately persistent in the soil. Residues can persist for years, but the degradation rate depends on the soil and specific herbicide. The triazines are generally low in acute toxicity. (McEwen, F.L. and G.R. Stephenson (1979) pp. 124-133).

Some of the triazine herbicides, especially atrazine and simazine, appear to be mutagenic, especially with plant activation. (Plewa, M.J. 1978) (Galston, A.W. 1979). All the triazine herbicides reviewed here have similar chemical structures and properties. As a group, the triazines are suspect mutagens and carcinogens and should be treated with respect.

The triazine herbicides reviewed in this section are: atrazine, prometone, prometryne, propazine, and simazine.

Photo by Doug Cowell

ACEPHATE

The common name is acephate. It is also known as Orthene, Ortho 12420, and Ortran.

Chemical Name: Acetylphosphoramidothioic acid 0, S-dimethyl ester

Empirical Formula: $C_4H_{10}NO_3PS$

(Windholz, M. et.al. (1976))

Characteristics: Acephate is a white solid which is very soluble in water and reasonably soluble in acetone and alcohol. It is a nonpersistent, organophosphorus insecticide, used against a wide variety of insect pests.

Toxicity

There is good data on the acute toxicity of acephate, but only limited information on teratogenic, mutagenic or carcinogenic effects.

In terms of acute toxicity, acephate is moderately toxic to mammals and birds, slightly toxic to fish and highly toxic to bees. Oral LD_{50} for rats is 866 mg/kg. Dermal LD_{50} for rats is 2,000 mg/kg.

(Miller, A.V. and S.M. Craig (1979)

Like other organophosphorus compounds, this substance interferes with nerve transmission. (See "Organophosphorus Compounds").

The earliest reference I have seen on acephate appeared in 1972 (73-0917). In this review, it is claimed that acephate "does not accumulate in the food chain, has a minimum impact on non-target species, and degrades into non-toxic products." No effects were observed in rats after 90 days at 300 ppm in the diet. In dogs, acephate in the diet for one year produced acute poisoning, but no other effects. The half-life ($T_{½}$) was 3 days in soil.

Waters, M.D. et.al. (1980) reports that acephate is mutagenic in one species of bacteria, increased mitotic recombination in another species and increased abnormal DNA synthesis in a tissue culture. (Human fetal lung). Of thirty-eight pesticides tested only six were positive and one of these was acephate. With Drosophila (fruit flies), no response was shown in a sex-linked recessive lethal test, but was only tested at one dose: 10 ppm (Also see 80-3250).

Clegg, D.J. (1979) cites a three-generation rat study that fed rats at three dose levels. In the second generation, there were decreased mating and fertility indices. The significance of this is not clear.

Douglas fir trees have been treated with acephate in B.C. to control tussock moths. No residues were found after 60 days. "Sunlight

may have been an important factor in the decrease in residues." (78-1543)

The effects on wildlife have been studied (80-2258). "Two Columbian ground squirrels collected on day 3 and one on day 6 had acephate residues in the brain."

Another study (76-1551) studied the effects on humans and concluded that no "allergic, neurotoxic, teratogenic, mutagenic, carcinogenic effects are known" in humans. Acephate apparently produces non-toxic metabolites.

Other reports of half-life ($T_{1/2}$) vary. $T_{1/2}$=15 days (79-2061); $T_{1/2}$=9 days (80-3408).

Recommendations

There is at present no indication that acephate produces carcinogenic, mutagenic or teratogenic effects in mammals. Under certain conditions in the Ames test, acephate caused mutations. This should be cause for more detailed and extensive testing, and at least moderate concern.

At the present time, the most serious threat to human health appears to be acute toxicity, and in this regard, acephate should be avoided. If residues are present, washing your arms and wearing gloves should be adequate protection.

Acephate is one of the IBT pesticides. I would treat it as a mutagen and watch for further testing. (See Appendix I)

ATRAZINE

The common name is atrazine. It is also known as Aatrex, AAtrex, Gesaprim, Marzone, Primatol A, G 30027, Atranex, Atred, Cisazine and Vectal SC. (Miller, A.V. and S.M. Craig (1979); Windholz, M. et.al. (1976); Anon (1980a). Some of these alternate names are mixtures of atrazine and other pesticides.

Chemical Name: 6-Chloro-N-ethyl-N+-(l-methylethyl)-1,3,5-triazine-2,4-diamine.

Empirical Formula: $C_8H_{14}CIN_5$

(Windholz, M. et.al. (1976))

Characteristics: Atrazine is a triazine herbicide slightly soluble in water, but more soluble in organic solvents. In pure form it appears as a colorless crystal. (Anon. 1980a)

Uses: Atrazine is a commonly used herbicide, especially on corn. It is used as a pre-emergence and post-emergence herbicide.

(Miller, A.V. and S.M. Craig (1979))

Persistence: Atrazine is a persistent pesticide.

Acute Toxicity

Atrazine is a relatively safe pesticide having an oral LD_{50} in rats of 3,080 mg/kg and a dermal LD_{50} in rabbits of 7,500 mg/kg. It is relatively safe for wildlife, but has appeared in water and including wells used for drinking water. (McEwen. F.L. and G,R. Stephenson 1979)(76-1629).

A farmer with a history of sensitivity to propachlor suffered contact dermatitis after spraying atrazine. (73-848).

Mutagenicity

There is good evidence that atrazine is a mutagen in several systems. It has been found to induce chromosome breakage, aberrations, and aneuploidy (abnormal number of chromosomes) in plants. One of its metabolites in corn produces mutations in yeast and in corn pollen grains. (Galston, A.W. (1979)) (Plewa, M.J. and J.M. Gentile (1976)) (Plewa, M.J. (1978)).

In the Ames test, atrazine is not mutagenic (Plewa, M.J. and G.M. Gentile) (78-1674), but when done with plant activation it is mutagenic. (Plewa, M.J. 1978).

Atrazine can induce dominant and recessive lethal mutations in Drosophila (fruit flies). (Plewa, M.J. 1978).

Applicators of herbicides, including atrazine, had a maximum of a four-fold increase in chromosome aberrations in their blood lymphocyte cultures as compared to a control population. (Yoder, J. et.al. (1973)). This study is complicated by the fact that applicators were also using 2,4-D which also is mutagenic.

Plewa, M.J. (1978) writes: "Thus, the majority of data reported ... indicate that...atrazine... induce(s) both mitotic and meiotic chromosome aberrations and are biologically activated into agents that induce point mutations." p. 47.

Atrazine produces dominant lethal mutations (Ehling, U.H. 1980) and chromosome damage (80-2592) in mice.

Carcinogenicity

Given the mutagenic properties of atrazine, it is certainly suspect as a carcinogen. Additional concern stems from the fear that atrazine can form N-Nitrosamines in the stomach and/or in soil. N-Nitrosamines are known carcinogens. Since atrazine is widespread in the drinking water of the corn belt, this may have serious implications. (Wolfe, N.L. et.al. 1976)(76-1629)

Teratogenicity

Atrazine produces terata in frogs. (80-2955)

Reviews

For a review of mutagenicity see Plewa, M.J. (1978).

More recently see 80-2600: "Other recent studies developed in Europe by several laboratories confirm the present results, thus indicating that atrazine might represent a genetic hazard for man."

Recommendations

Atrazine should be treated as a mutagen and therefore as a carcinogen. Lack of residues on the plants does not indicate a "no-risk" situation as it is a metabolite of atrazine that appears to be the actual mutagen. Rubber gloves and washing with soap and water is advised. However, significant residues on the seedlings are unlikely.

BENOMYL

The common name is benomyl. It is also known as Benlate, F-1991, and Tersan.

Chemical Name: (1-((Butylamine)carbonyl)-1H benzimidazol-2-yl) carbonic acid methyl ester

Empirical Formula: $C_{14}H_{18}N_4O_3$

(Windholz, M. et.al. (1976))

(McEwen and Stephenson (1979))

Characteristics: Benomyl is a white crystalline solid practically insoluble in water, but soluble in organic solvents. The active component is thought to be the degradation product methyl 2-benzimidazole-carbamate ester (MBC). It is a persistent compound remaining on plant foliage either as the parent compound or as MBC for several months. ($T_{1/2}$=3-6 months on turf, 6-12 months on bare soil) (74-1814)

Classification: Benzimidazole fungicides.

(Windholz, M. et.al. (1976))

(McEwen, F.L. and G.R. Stephenson (1979))

Uses:

Benomyl is a broad-spectrum fungicide used on 43 food crops and 41 ornamentals, (EPA 1979). It is used as a foliar, seed, soil, or turf treatment. It is both a preventative and aneradicant fungicide. Benomyl is used for the control of apple scab, powdery mildew, brown rot, and Botrytis blight. (Miller, A.V. and S.M. Craig (1979)) It is also used to eradicate earthworms (McEwen, F.L. and G.R. Stephenson (1979)) and is toxic to fish (Miller, A.V. and S.M. Craig 1979). Resistance has developed in some species of mold. (78-2459)

Acute Toxicity

Benomyl is of low mammalian toxicity with an oral LD_{50} in the rate of more than 10,000 mg/kg. However, it is toxic to fish and freshwater crustaceans (Daphnia). For this reason, the EPA does not allow it to be applied directly to streams and lakes.

Mutagenesis

There is good evidence that benomyl or its metabolite MBC is a mutagen. (EPA 1977b). In the Ames test (several strains) and tests with E. coli, the results were positive. The mutations were apparently caused by interference with DNA repair. (Seiler, J.B. (1972) (1975) (Kappas, A., et.al. 1976)(78-0838)) However, Fiscor, G. et.al. (1976)

found that benomyl was not mutagenic in the Ames test. The reason for these conflicting results is not clear.

Bignami, M.M. et.al. (1977) found that benomyl was strongly mutagenic in terms of nondisjunction. This is a defect in which the chromosomes are not separated properly during cell division and is a common cause of genetic disease in humans. Although the experiments were done with a mold, the mechanism in humans is basically the same. Other studies confirm this result. (80-3497)

Other studies have shown mutagenic effects in other microorganisms. (Sandhu, S.S. and M.D. Waters 1980) (EPA 1977b) (Seiler, J.P. 1975) (78-2669)

In cultured mammalian cells, benomyl interfered with cell division and causes chromosome damage (not strongly). (Sandhu, S.S. and M.D. Waters 1980) (EPA 1977b)

In higher plants similar mutagenic activity has been detected. (EPA 1977b) (Grant, W.F. (1978)) (75-2480)

In rats, similar effects are observed when benomyl is given by injection, but not when fed. In mice, using the micronucleus test, there were various kinds of interference with chromosome division and structure. (EPA 1977b) In a dominant lethal test in mice, benomyl was not mutagenic. However, in this test, the dosage was low. In addition, it is believed that benomyl is poorly absorbed from the intestine, (Sandhu, S.S. and M.D. Waters 1980) although I have not seen evidence in support.

In 1977, the EPA concluded that "... benomyl and MBC are mutagenic in multitest systems." (EPA 1977b, p. 61792)

There are additional studies that cause concern. Sperm seems to be effected in both rats and dogs. (EPA 1977b) In this case, the exposure route was through the lungs.

Also, rats fed high amounts of MBC showed degeneration of the testis. (EPA 1977b) These results indicate that MBC and benomyl are transported to the gonads.

Investigators in England reported no mutagenic responses in Drosophila (fruit flies) and no increase in chromosome aberrations in cultured human cells. However, only one dosage level was used. (80-0251)

Carcinogenesis

To my knowledge, no adequate experiments have been done to assess carcinogenesis with benomyl. However, there is evidence that MBC and nitrites may form N-nitroso compounds which are carcinogens. There is

no conclusive evidence in this regard, but it is a matter of serious concern. (EPA 1977b) (76-3069) (77-1105)

Teratogenesis

"Benomyl has been shown to induce teratogenic effects in Wistar rats and cause a reduction in spermatogenic activity in both rats and dogs." (EPA 19776, p. 61792) These effects could be of mutagenic origin (e.g., damaged sperm), and/or occur during pregnancy.

One study with Wistar rats showed a clear dose dependent response. (73-0962) These results are a good reason to treat benomyl, for practical purposes, as a teratogen in humans.

Another study (74-0136) confirmed teratogenic effects in Wistar rats.

Additional Comments

The EPA in their RPAR of benomyl (EPA 1979); point out that there is strong evidence that benomyl or its metabolites interfere with chromosome division (which is an important source of genetic disease), that they reach the gonads, and are therefore of serious concern. They also note that teratogenic effects and depressed sperm counts are of concern. There is evidence that gene mutations can occur, but the evidence for this occurring in mammals is not clear. "… the Agency (EPA) is not assured of the safety of benomyl with regard to this mechanism." p. 46 Chromosomal breakage is also of concern.

The EPA requires the following warning on all pesticide products containing benomyl packaged in 5 pound or larger bags:

Warning to Workers

> "The United States Environmental Protection Agency has determined that benomyl causes birth defects and reduced sperm production in laboratory animals. Exposure to benomyl might cause a depressed sperm count. Workers must be sure to wear a cloth mask while mixing benomyl for aerial application. In case of accidental spills or other unusual exposure, cease work immediately and follow directions for contact with benomyl." (EPA 1979)

Contact Dermatitis

Contact dermatitis has been observed in humans after exposure to benomyl. (Savitt, L.E. 1972) In Japan, field workers have suffered skin injuries thought to be the result of an allergic reaction to benomyl. (76-2970) (79-0853)

In rats, benomyl has been shown to be a strong sensitizing agent leading to contact dermatitis. (78-1175)

Bacteria

Fuch and deVries (1978) review the effect of benomyl on bacteria. Benomyl supports the growth of bacteria.

Benomyl stimulated bacterial growth in soil after a two-week incubation period. (72-2380) Microbes can use benomyl as a nutrient.

Reviews

The EPA reviewed benlate under their RPAR system. (EPA 1977b) (EPA 1979a) (EPA 1979b) Sandhu, S.S. and M.D. Waters (1980) reviewed the mutagenic properties of benomyl. An earlier but important review was done by J.P. Seiler (1975). For metabolism and bacterial breakdown of benomyl see Fuchs, A. and F.W. deVries (1978) and Alexander, M. (1981).

Also see 80-3495 for a review of genetic toxicity.

Recommendations

Due to the teratogenic and mutagenic potential of benomyl, I would handle trees with caution. If there are residues on the trees, frequent washing, rubber gloves and clean clothes are in order.

Although the teratogenic potential is not clear, I recommend that pregnant women not plant trees treated with benomyl.

Male exposure is also of concern in teratogenesis.

The evidence indicates that benomyl acts in a manner that could cause serious genetic disease in humans. Be careful.

CAPTAN

The common name is captan. It is also known as Captan, Merpan, Orthocide-406, Vondcaptan, Vancide-89, and SR-406.

Chemical Name: 3a,4,7,7a-Tetrahydro-2-((trichloromethyl)thio)-1H-isoindale-1,3,(2H)-dione.

Empirical Formula: $C_9H_8Cl_3NO_2S$

Windholz, M. et.al. (1976)

Characteristics: The pure form is an odorless, white crystalline substance. The technical grade is a pungent, yellow to buff amorphous powder. (EPA 1980)

It is moderately soluble in many organic solvents including chloroform, benzene and dioxane, but practically insoluble in water at room temperature. (EPA 1980)

Classification: Phthalimide

Uses

Captan is a very widely used fungicide that has been in use since the early fifties. The agricultural usage in the U.S. for 1978-1979 was between 8.5 and 9.7 million pounds. The % of crop or site treated each year for some selected food crops are: apples (32%), peaches (20%), almonds (40%), grapes (30%), strawberries (98%), corn (1OO%), cotton (80%), potatoes (60%). This is only a small sample of the many food products treated with captan. (EPA 1980)

> "Captan is used as a fungicide: 1) on a wide variety of fruit, vegetable and ornamental crops, some of which are grown on home and garden sites; 2) on numerous plant seeds; 3) on food crop packing boxes; 4) in soil preplanting treatment; 5) on surfaces inside and outside the home; 6) in cosmetics and pharmaceuticals, oil based paints, lacquers, paper, wallpaper paste, plasticizers, polyethylene, vinyl, rubber stabilizer and textiles; 7) in combination with insecticides on food crops, seed treatment and household pets. Pesticide products containing captan are most widely used as wettable powders (50-80% captan) and dusts (7.5-15% captan). Other formulations are commercially available including 4-pound per gallon aqueous suspensions and coated granules."
>
> (EPA 1980)

Acute Toxicity

With respect to acute toxicity, captan is considered to be one of the safest pesticides. The acute oral LD_{50} value for rats is 9000 mg/kg. However, pigeons and sheep are at least 10 times more sensitive than rats, and zebrafish larvae are killed by as little as 1 ppm. (Bridges, B.A. 1975) Cattle have been killed by six doses of 250 mg/kg/day. (EPA 1975) No effects on humans are known. Captan may be a mild sensitizer, causing skin irritation. (EPA 1975)

Mutagenesis

There is good evidence in both bacterial and mammalian test systems that captan is a mutagen. Captan can interact with DNA of several species to produce mutations in both reproductive and other cell bodies. There is also good evidence that captan can cause chromosome damage. (EPA 1980)

In the Ames test, several strains of Salmonella typhimurium have undergone mutations due to captan. (Fiscor, G. et.al. 1977, 1978; McCann, J. and B.N. Ames, 1976; Carcre, A. et.al. 1978, EPA (1980); McCann et.al. 1975, Shirasu, et.al. 1977; and Simmon et.al. 1977) In some of these tests, it was found that a S9 microsomal liver fraction decreased the mutagenic action of captan 33-50%. (EPA 1980) This would indicate that captan is less mutagenic in intact higher organisms than in bacteria.

In bacterial tests using E. coli, captan induced mutations. (Shirasu, et.al. 1977; and Simmon et.al. 1977).

Host-mediated assays and fluid-mediated assays have also been used to test the mutagenicity of captan. In the host-mediated assay, bacteria are injected into the peritoneal cavity, circulatory system, or testes of rats or mice. The rodents are exposed to the chemical to be tested and after a few hours, the bacteria are tested for mutation. Four host-mediated assays performed by Fiscor, et.al. (1977) showed no mutagenic activity. Legator, M.S. and H.V. Mal ling (1971) got similar results. Fiscor, et.al. (1977) also did some fluid-mediated assays with human blood, rat blood, plasma, and saline. Blood tends to inactivate captan, but the process is a slow one. (Fiscor, et.al. (1977))

Captan has been shown to be mutagenic in mold (Aspergillus nidulans). (Bignami et.al. 1977)

Captan has shown to be a mutagen in cell cultures of hamster lung fibroblasts. (EPA 1980)

Captan has been shown to cause DNA damage in bacteria, molds, and in hamster and human cell cultures. (EPA 1980)

Chromosomal aberrations have been shown to be caused by captan in two mammalian cell lines (Legator et.al. 1969; EPA (1980). However, another study (Shirasu, Y. et.al. (1977)) found no chromosome aberrations due to captan in rats and human cell line.

Captan is a mutagen in the fruit fly, Drosophila. (Waters, M.D. et.al.,1980)

There have been several dominant lethal tests done with captan. This test involves treating male mice with captan, breeding these male mice with female mice, and checking the females for resorbed fetuses. Since most mutations are lethal, a measure of the mutated male sperm is the number of resorbed fetuses in the females.

Mice and rats thus treated showed the dominant lethal effect in a study by Collins (1972). (See EPA 1980) Other studies which did not show the effect were inconclusive. (EPA 1980)

A heritable translocation test was done for the EPA by Stanford Research Institute (Simmon, et.al. 1977). This test measures a type of chromosome aberration called a translocation. In a translocation, one piece of a chromosome is moved to another chromosome. In man, translocations can result in genetic disease, of which one type is Down's Syndrome. In rats or mice heritable trans locations are measured by treating male rats or mice with the test substance, mating them with females and checking the litter size. Smaller than average litter size is indicative of a trans location in the treated male parent (Malling, H.V. 1978). The test on captan showed that it can produce a heritable translocation. However, there was an unusual trans location in one of the untreated mice as well, and the EPA recommends doing the test again. (EPA 1980)

B.A. Bridges (1975) concluded in his review of captan that “captan is an unambiguous mutagen in cellular systems” and that “captan should be regarded as a base-change mutagen in any system in which it can reach cellular DNA.” He also points out that the dominant lethal assay and other tests indicate that the mutagenic effects of captan can extend to the gonads in laboratory animals. In his conclusions he writes: “A tentative evaluation of the potential genetic hazard to man of captan suggests that the risk is not insignificant and might be appreciable.” He makes the following recommendation: “The use of non-mutagenic substitutes with similar low toxicity should be promoted.” (Bridges, B.A. 1975)

The EPA has done two extensive reviews of captan. (EPA 1975, 1980) Both reviews conclude that captan is a mutagen in several systems. The RPAR Position Document 1 (EPA 1980) quotes regulations

that indicate that a known mutagen should be banned (p.16) and goes on to make the following comments:

> "Human exposure to a mutagen has serious implications. The possible adverse effects to people, especially those of reproductive age, are spontaneous abortions, stillbirths, birth defects in their children and diseases in the adult life of subsequent generations. Any of these effects could result from exposure of the male and/or female parent to a mutagen. In addition, those exposed can be adversely affected by mutations of the somatic cells."

> "…captan is capable of inducing gene mutations, DNA damage and chromosomal aberrations…" (EPA 1980, p. 16)

Carcinogenesis

The only well designed and executed bioassay for carcinogenicity of captan was done by the Gulf South Research Institute and contracted by the National Cancer Institute (NCI 1977). (Revised by Cueto, C. Jr. (1980)) This study found that captan fed mice have an increase in the incidence of duodenal (intestinal) tumors. This tumor is very rare in the strain of mice used, and the compelling conclusion is that captan caused the tumors. (EPA 1980)

Apparently, the manufacturers of captan, Stauffer Chemical Co. and Chevron Chemical Co., did their own study and got similar results. (EPA 1980)

Other studies have been done, but they have been inconclusive. (EPA 1980)

The NCI study (NCI, 1977) required very high doses of captan to induce tumors, but we must not necessarily conclude that captan will only produce cancer under these conditions. The response was dose related, so it is possible that captan could induce cancer at any dose level. There is reason to think that captan might be a potent carcinogen in the lungs, but no work has been done on this question. (B.A. Bridges, 1975)

Teratogenesis

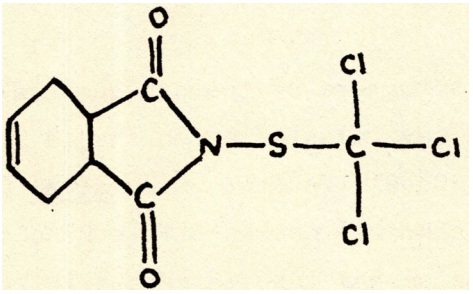
The structure of captan is similar to the structure of the proven teratogen, thalidomide. (See figure 1) For this reason several studies on the teratogenic potential of captan have been done (EPA 1975), (72-0780) Verrett, M.J. et.al. 1969, injected captan into chicken eggs and produced abnormal chicks. This kind of result is very difficult to extrapolate to humans. Captan has induced terata in rabbits (McLaughlin, et.al. 1969), but other studies showed no response. (EPA

1975) Highly significant teratogenic effects were produced in hamsters (Robens, 1970). Other studies on mice, rats, monkeys, dogs and hamsters showed no apparent effects (EPA 1975), but Earl, et.al. (1973) produced effects in dogs.

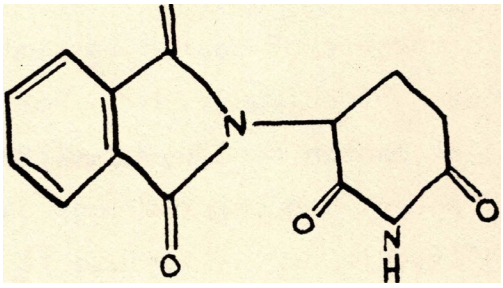
Bridges (1975) concludes that "The evidence that captan and its analogues are teratogens is probably as good as that for thalidomide, taking one species with another and admitting the lack of primate data. It must be emphasized, however, that fairly massive doses are necessary to detect any effect and that repeated application may tend to result in death and resorption of the embryo with consequent loss of teratogenic effect. In man, chronic exposure to these fungicides is generally at a low level and is chronic. The likelihood is probably remote that these agents cause a significant addition to the normal rate of production of embryonic abnormalities, but in situations where these substances are handled in significant quantities it would seem prudent to restrict the workers involved to women past reproductive age and men." Note that this recommendation applies only to the teratogenic potential and not to the mutagenic or carcinogenic potential of captan.

Wilson (1977), in a review of teratogenic chemicals in the environment, comments that captan probably does "not pose appreciable risks under usual conditions of usage." (Wilson, 1977, p. 366)

This is in essential agreement with Bridges (1975). The EPA did not cite teratogenicity as a reason for RPAR review for captan, apparently agreeing with Bridges and Wilson. However, the EPA added that there were other possible health hazards posed by captan: "Currently available data strongly suggest that captan may have teratogenic, fetotoxic and hypersensitivity effect. The Agency is currently seeking more information on these issues." (EPA 1980, p. 36)



Captan



Thalidomide

(After Bridges, B.A., 1975)

Some Russian studies indicate that captan interferes with reproduction and may be teratogenic in both rats and mice. (72-2467)

Additional Comments

The Report of the Secretary's Commission on Pesticides and their Relationship to Environmental Health by the U.S. Department of Health, Education and Welfare, reviewed the significance of adverse effects of a number of pesticides including captan. (U.S. Dept HEW 1969)

The commission recommended unanimously that human exposure to captan be considered a potential health hazard. The following were among the commissions findings relating to captan:

1) it was reported as a cause of dermatitis from apple spraying and allergic dermatitis was demonstrated in agricultural workers,
2) it produced mutagenic effects in bacteria, human embryonic lung cells and cell lines derived from the kidney of the kangaroo rat,
3) it induced teratogenic effects in developing chicken embryos, and
4) it increased tumor incidence in mice.

The commission specifically recommended:

1) reducing exposure of the general population from dietary sources of captan pending the completion and evaluation of additional testing of tumorigenicity;
2) minimizing worker's exposure to captan pending completion of safety studies;
3) giving high priority to captan in a testing program for mutagenesis; and
4) restricting captan immediately should it be found to be teratogenic. (EPA 1980, p. 12)

The U.S. Dept. of Labor, Wage and Hour Division, published final rules pertaining to captan in The Federal Register (1979). The notice indicated that while minors could be employed in hand harvesting of short-term crops, they could not harvest captan treated crops of strawberries or potatoes. (EPA 1980)

Metabolism

For a review see (Lukens, R.J. (1971). It is widely reported that decomposition of captan in human blood is very rapid, the half-life ($T_{1/2}$) as being 0.9 minutes. (Kohn, G.K. (1977)) However, Fiscor, G. et.al. (1977) reported that while blood does inactivate captan's mutagenicity, the process is a slow one.

Captan is broken down rapidly in soil (half-life 3 to 4 days). One of its breakdown products is HCL which may cause damage to plants. (McEwen, F.L. and G. R. Stephenson (1979)).

IBT

Captan was tested by Industrial Bio-test Labratories, Inc. Several studies were shown to be poorly executed or falsified, and these studies are not recognized as valid by a joint U.S./Canadian committee and the EPA. They are not considered in this report. (See Appendix)

Reviews

The best review of captan was done by the EPA (EPA 1980) and was printed in the Federal Register (EPA 1980b). Another excellent review is by Bridges, B.A. (1975) but is somewhat dated. The major development since Bridges (1975) is a positive carcinogenesis result in mice, (NCI 1977). Also see EPA (1975) and Legator, M. and S. Zimmering (1975).

Recommendations

Captan definitely has mutagenic and carcinogenic potential in humans. If there are residues on the trees, I would wear rubber gloves and wash exposed areas frequently. Another measure is to wear clean clothes daily. These precautions are probably adequate to reduce exposure to insignificant levels. For residues above 200 ppm, I would be very cautious.

Although the teratogenic potential is unclear, I recommend that women who are pregnant not plant trees with any residues of captan. Exposure to captan may also affect sperm. (72-2467)

CARBARYL

The common name is carbaryl: It is also known as Sevin, Arylam, and Seffein.

Chemical Name: 1-Naphthalenol methylcarbamate.

Empirical formula: $C_{12}H_{11}NO_2$

(Windholz, M. et.al. (1976))

Characteristics: Carbary] is a carbamate insecticide. The crystals are moderately soluble in organic solvents and slightly soluble in water. It is a broad spectrum insecticide, used on foliage and is highly toxic to honey bees.

It is stable in storage, but short-lived in the field. $T_{1/2}$ on plant foliage is 3 to 4 days; 7 to 9 days in soil; and 1 to 5 days in water.

It is metabolized rapidly by both plants and animals. In animals it is detoxified and excreted. Most animals eliminate a high percentage of an ingested dose within 24 hours.

Carbary] is a very common insecticide in agriculture and for garden pests. (McEwen, F.L. and G.R. Stephenson (1979))

In orchards, carbaryl application results in a rapid buildup of mites as their predators are also killed by the insecticide. (Miller A.V. and S.M. Craig 1979)

Acute Toxicity

Like most carbamates, carbaryl has a low mammalian toxicity. LD_{50} for rats is 560 mg/kg (oral). Dermal LD_{50} for rats is greater than 2,000 mg/kg. (Miller, A.V. and S.M. Craig 1979)

Carbary] is a moderate cholinesterase inhibitor. Symptoms of acute poisoning include headache, nausea, vomiting and blurring of vision. (Label for Sevin)

Mutagenicity

Carbary] was tested by the dominant lethal assay in mice and was found to have no effect. This result is reassuring, but by itself does not indicate non-mutagenicity. (Epstein, S.S. et.al. (1972))

In the Ames test, it was not mutagenic, but in Drosophila (fruit flies) it was a weak mutagen. (75-2508) (Sternberg, S.S. 1979) Various chromosome aberrations were observed. (75-1649) (80-3500)

Carbary] interferes with the proper division of chromosomes in plants. In fact, as a group, the carbamates have been recommended for the artificial induction of polyploidy. (This is the condition of having extra sets of the chromosomes in individual cells. Polyploidy can result in genetic disease.) Other chromosomal effects are known with carbaryl including multinucleate conditions and "stickiness". (Grant, W.F. 1978) Also see (80-2716)

In grasshoppers, chromosome aberrations caused by carbaryl have been observed. (74-2142)

A metabolite of carbaryl, N-Nitrosocarbaryl is produced when added to the common food additive, sodium nitrate. This compound is a potent mutagen in several species of microorganisms. (74-1683) (75-0923) (76-2267) (76-2016) (77-0603) (78-0837) (78-1715) (79-2497) (80-2552)

In a hamster cell line, carbaryl was mutagenic (78-1119)

Carcinogenesis

Under conditions simulating those of the human stomach, carbaryl can be nitrosated to form N-nitrocarbaryl. This compound in several studies, proved to be carcinogenic in rats. (Sternberg, S.S. (1979)) (Wolfe, N.L. et.al. 1976) (76-2239; 75-2461; 76-2240; 77-0844; 80-0505; 80-1102)

In cultured cells, nitrosocarbaryl can induce cancer. (76-0163)

There are several cases of cancer in humans in which carbaryl is a possible cause. However, other pesticides in addition to carbaryl were involved in these cases. (78-2414) (79-2878)

Carbary] fed to mice did not produce tumors by itself, but promoted tumors when fed with other substances (80-1153)

Teratogenesis

"Carbaryl has been reported to be teratogenic at high doses in guinea pigs and at low doses in dogs. In addition, reports of teratogenesis have been cited in tests with mice. Carbary] has been reported to be fetotoxic at high doses in rabbits, and at low doses in cotton rats.... Carbaryl is also teratogenic in chickens. ...At high doses it has reduced reproductive success of bobwhites and pheasants. There is also evidence that 1-naphthol, the most important metabolic breakdown product of carbaryl in mammals, may be teratogenic in mice; further investigation is needed, however, to confirm this finding.... At the very least, those most likely to be affected (pregnant women) should be informed of the risks and be able to avoid exposure." (Nisbet, I.C.I. and D. Miner (1971))

Other researchers have been unable to confirm all the teratogenic effects. (Sternberg, SS. 1979)

Proctor, N.H. et.al. (1976) produced teratogenic effects in the chicken embryo with carbaryl.

"Carbaryl is not only teratogenic in the chicken embryo test, but at high doses is also produces embryonic abnormalities in mice, guinea pigs, and dogs but not in hamsters, rabbits and swine." p. 581 Proctor, N.J. and J.E. Casida (1975)

Weil, C.S. et.al. (1972) argues that the dog studies are not applicable to humans because carbaryl metabolism is different in the two species. Other studies in rabbits, hamsters, and mice are found to be flawed in several ways. In the three-generation rat study performed by Weil, C.S. et.al. (1972), no terata were formed. They cite several other studies with similar results in several species.

However, Murray, F.J. et.al. (1979), in the most recent review of carbaryl I have seen, found that although carbaryl was not teratogenic in mice, it was teratogenic in rabbits. There seems to be a species difference with regard to the teratogenic effects of carbaryl. Murray, F.J. et.al. (1979) speculates that "if any teratogenic hazard to humans exist, it is most likely to be in the case of an overexposure to carbaryl such as in an intentional or accidental poisoning." p.88

A Russian study produced teratogenic effects in rats with carbaryl. (73-0962) Other studies showed fetotoxic effects (74-0136) (74-2948). Also see (78-0612)

Stellman, J.M (1979), in a review of toxic agents which are of occupational importance, lists carbaryl as an agent "toxic to the male reproductive system" and as a substance "observed to induce adverse reproductive outcomes."

Other authors suggest that pregnant women should not be exposed to carbaryl. (80-2229)

Earl, F.L. et.al. (1973) reported that pigs given carbaryl suffered teratogenic effects and a high incidence of resorptions and stillborns.

Additional Comments

Effects on wildlife and ecology are reviewed by Nisbet, I.C.T. and D. Miner (1971). "The real question is whether carbaryl shifts the balance in favor of the pest species or against it. There are dozens of cases in which carbaryl has been reported to upset natural control systems, resulting either in resurgence of the pest population or in

outbreaks of other pests. There is some evidence that the use of carbaryl for gypsy moth control prolongs the outbreak stage and makes the next outbreak worse." p. 14

Carbaryl and its metabolite 1-naphthol is very toxic to fish and shellfish and should not be allowed to get into aquatic ecosystems. (Nisbet, I.C.I. and D. Miner (1971))

Reviews

One of the best reviews will be the IARC monograph on carbaryl. (77-0573) Unfortunately, I have not been able to obtain a copy at this time.

For a review of teratogenic effects see Murray, F.J. et.al. (1979) and Wilson, J.G. (1977).

Recommendations

Acute poisoning is unlikely in treeplanters, but note that if the application is recent, it can produce toxic effects.

Although the evidence is mixed, I think it is prudent to treat carbaryl as a definite teratogen and possible mutagen and carcinogen in humans. The usual precautions of washing and gloves are in order.

CHLOROTHALONIL

The common name is chlorothalonil. It is usually known by one of its trade names: Bravo, Daconil 2787, Termil, DAC-2,787, and Forturf. It is also known as tetrachloroisophthalonitrile.

Chemical Name: 2,4,5,6-Tetrachloro-1, 3-benzenedicarbonitri le

Empirical formula: $C_8Cl_4N_2$

(Windholz, M. et.al. (1976))

(Miller, A.V. and S.M. Craig (1979))

Characteristics: Chlorothalonil is a white crystalline solid, odorless, and soluble in water. It is used primarily as a fungicide, but is also a bactericide and nematicide. It is persistent.

Acute Toxicity

Chlorothalonil causes allergic reactions in some people, but has a very low mammalian toxicity. The LD_{50} (oral) for rats is greater than 10,000.

(Miller, A.V. and S.M. Craig (1979))

Some effects and possible effects have been noted in humans. In a Japanese report, chlorothalonil applicators complained of lumbago, shoulder discomfort, and dimness of vision. Other symptoms including liver and heart disfunction may have been caused by chlorothalonil, but other pesticides were also involved.

Chlorothalonil has caused contact dermatitis due to a hypersensitizing effect in sunlight. (77-1917) In one study, 80% of the people tested ("experienced workers") showed sensitivity to chlorothalonil. (77-2330)

Other studies report sensitivity to chlorothalonil. (80-1644) (80-1645)

Mutagenesis

Attempts to produce chromosome aberrations in plants have failed. (79-2417)

Chlorothalonil was tested for mutagenicity in the Ames test, with and without metabolic activation, and in tissue cultures for chromosomal breakage. It is not mutagenic in these systems. (Shirasu, Y. et.al. (1977)) (73-2365)

Carcinogenesis

In a National Cancer Institute sponsored study, mice showed no increase in tumors over the controls. However, the rats, both male and

female, showed a definite dose related increase in kidney cancer. (80-1118) In view of the lack of mutagenic activity, these results are surprising.

One reviewer uses Chlorothalonil as an example of cancer produced by "metabolic overloading", meaning (as best as I can tell from the abstract) that the dose levels are so high that the test animals' metabolism are stressed far beyond what could ever happen outside the laboratory. Thus, it should not be considered a carcinogen. (80-2503)

Chlorothalonil is on the suspected carcinogens list published by the U.S. Department of Health, Education and Welfare. (Christensen, H.E. et.al. (1976))

Teratogenesis

I have seen no data on the teratogenic potential of chlorothalonil.

Additional Comments

Chlorothalonil is on the IBT list. See appendix.

Recommendations

Without checking the NCI study in detail, I would score chlorothalonil as a carcinogen and therefore avoid exposure. There is not much data on the effects of chlorothalonil, although it does not seem to be a mutagen. If it is a carcinogen, it is one of the rare "false negative" results in the microorganism tests. The fact that it is on the IBT list means that no one is very sure of its safety, mainly because of lack of data.

It definitely has a potential for causing contact dermatitis. Washing and gloves are in order if residues exist.

DIAZINON

The common name is diazinon. It is also known as Basudin, Spectracide, G-24,480, dimpylate, Garden Tox and many others.

Chemical Name: Phosphorothicic acid 0,0-diethyl 0-(6-methyl-2 (1-methylethyl)-4-pryrimidinyl) ester.

Characteristics: Diazinon is an organophosphorus insecticide and nematicide. It is a colorless oil that is practically insoluble in water. The technical grade is a pale to dark brown liquid. It is used as a contact poison and is moderately persistent. It kills a wide variety of insects. It decomposes at high temperatures. In both animal and plant tissues it is converted to diazoxon. It kills insects for 7 to 10 days after application.

(Windholz, M. et.al. 1976)

(Miller, A.V. and S.M. Craig 1979)

(Anon. 1980a)

(McEwen, F.L. and G.R. Stephenson)

Diazinon contains a toxic impurity called sulfotepp (0,0,0,0 tetraethyl dithio-pyrophosphate). Sulfotepp is much more toxic than diazinon and far more stable. It may become concentrated in the environment from repeated applications of diazinon and may be present long after diazinon residues are undetectable. (Meir, E.P. et.al. (1979)) (80-1307) (80-1380)

Diazinon apparently has an adverse effect on testosterone (male hormone) metabolism. (Schein, L.G. et.al. (1976)) and normal function of the prostate gland. (Shain, S.A. et.al. 1977)

Diazinon can persist under certain conditions for longer periods. Several studies reported residues after 28 days. (70-0031) (72-2133) Other studies have reported residues after many months and years, but this only occurs under special circumstances. (71-2121) (72-1433) (72-1651) (72-1849) (76-1627)

(76-1109) reported that residues persist at a low level for a long time although 90% of it disappears within 170-180 days.

Mutagenesis

Waters, M.D. et.al. (1980) reviewed the mutagenesis of several pesticides. Diazinon was not mutagenic in any microorganism test. (Several strains). In tissue cells cultures diazinon had a mutagenic effect, as wel1 as other toxic effects. (Tzoneva-Maneva, M.T. et.al. (1971))

Matsuoka, A. et.al. (1979) tested 29 chemicals in a test for chromosome damage in a hamster cell line (lung) with metabolic activation (S9 mix). Diazinon was strongly positive at relatively low doses.

A study of workers who produce Diazinon found a higher incidence of chromosome aberrations (Chromatid-type) in these workers than in the control group. (Kiraly, J. et.al. (1979))

DNA repair tests with bacteria indicated that diazinon was not mutagenic, but the breakdown products were highly mutagenic. (78-1874)

Carcinogenesis

Diazinon was not carcinogenic in rats and mice of both sexes. (Waters, M.D. et.al. (1980))

It is on the suspected carcinogens list published by the U.S. Department of Health, Education, and Welfare. (Christensen, H.E. et.al. 1976)

Acute Toxicity

Diazinon has a moderate toxicity. LD_{50} (oral) for rats is 250-600.

Like the other organophosphorus compounds, it interferes with nerve transmission. Biskind, M.S. et.al. (1972) reports on cases of poisoning among hospital personnel after application by professional exterminators. Symptoms included nausea, vomiting, diarrhea, headaches, mental confusion, running nose, respiratory distress, visual disturbances, muscle pain, lethargy, and irregular menstruation.

There is one reported case of diazinon induced psychosis. (71-1167)

Chronic nervous disease has resulted from occupational exposure to diazinon (72-1206).

Teratogenesis

Diazinon given to dogs produced a high incidence of stillborns and made the bitches extremely high strung. No clear teratogenic effects were observed, although reproductive success was seriously reduced. In pigs, a clear teratogenic effect occurred. Only a few animals were used so the significance of the result is not clear, but it should be enough to make one extremely cautious. (Earl, F.L. et.al. (1973))

Robens, J.F. (1969) reported no teratogenic effects in hamsters or rabbits given diazinon.

In a study with chicken embryos, diazinon was a potent teratogen. (Proctor, N.H. and J.E. Casida (1975)). This was confirmed by (79-0203) and (80-1419).

Wilson, J.G. (1977) reviewed the teratogenic effects of several pesticides. Earl, F.L. et.al. (1973) is cited along with two studies on rats, both of which produced malformations and resportions of the embryos.

Offspring of mice exposed to diazinon had various degrees of neuropathology. In the high dose group, damage to the brain was observed under the microscope. Mice in the other groups showed subtle behavioral effects. "The behavioral defects observed in offspring of mothers exposed to Diazinon indicate that prenatal exposure to organophosphates may produce subtle dysfunctions not readily detectable until later life." p.997 (Spyker, J.M. and D.L. Avery (1977). Other metabolic teratogenesis has been reported (79-2193). Diazinon appears to be teratogenic to fish. (78-1598)

Additional Comments

Diazinon is on the IBT list. (see appendix). For a review of biodegradation see Alexander, M. (1981).

Recommendations

Although more studies should be done to determine the teratogenic, mutagenic and carcinogenic potential of diazinon, I would certainly treat it as a teratogen, a mutagen, and a suspected carcinogen. Remember that the breakdown product sulfotepp is toxic and persistent. I have not searched for data on the mutagenic, carcinogenic and teratogenic properties of this product.

Most residues will be gone after a few weeks, but more information is needed on the residues on seedlings.

If residues are present, treat with extreme caution.

FERBAM

The common name is ferbam. It is also known as Fermate, Carbamate, Ferbeck, Ferradow, and Karbam Black.

Chemical Name: Tris(dimethylcarbamodithioato-S,S^1)iron

Empirical Formula: $((CH_3)_2NCS_2)_3Fe$

Characteristics: It is a black solid soluble in water. Ferbam is a carbamate fungicide. It leaves a black spray residue and an unpleasant odor.

It is not very persistent, is nonsystemic and degrades to dimethylamine and CS_2. McEwen, F.L. and G.R. Stephenson (1979), pg. 81

Acute Toxicity

The LD_{50} (oral) in rats is 1,000 mg/kg. It may cause irritation of the skin and mucous membranes, and kidney damage.

(Windholz, M. et.al. 1976)

(Miller, A.V. and S.M. Craig 1979)

I have seen very little information on ferbam. One test for mutagenicity gave a positive result in one strain, (bacteria) but not in others. (79-0428) Shirasu, et.al. (1977) list 193 pesticides tested in the Ames test and only 15 proved to be mutagenic. One of these is ferbam.

Until we have more information I would treat ferbam as a mutagen and suspected carcinogen. I have not seen any data on teratogenesis.

MALATHION

The common name is malathion. It is also known as Cythion and several other names.

Chemical Name: ((Dimethoxyphosphinothioyl)thio)butanedioic acid diethyl ester.

Empirical Formula: $C_{10}H_{19}O_6PS_2$

Characteristics: A deep brown to yellow fluid with a characteristic garlic odor, but some formulations are "low-odor products". It is a broad spectrum organophosphorus contact insecticide. It is considered nonpersistent. There are more than 140 different products containing malathion.

Windholz, M. et.al. (1976)

Miller, A.V. and S.M. Craig (1979)

Anon. (1980a)

Malathion is one of the major insecticides for the control of adult mosquitoes. Houseflies in some areas have become persistent. (McEwen, F.L. and G.R. Stephenson (1979))

Malathion contains toxic impurities. One such compound is isomalathion which considerably increases the toxicity of malathion. (Brooks, G.T. (1980))

Acute Toxicity

Malathion has a low mammalian toxicity. The acute oral LD_{50} in rats is in the 2000 mg/kg range, while the dermal toxicity is in the 4000 mg/kg range. (McEwen and Stephenson 1979)

Mutagenesis

Shirasu, Y. et.al. (1977) reported no mutagenic effects in several strains of bacteria found in the Ames test. In a recent report, mutagenic activity was found in one species (80-2917).

Waters, M.D. et.al. (1980) reported no mutagenic effects in several bacterial tests and in Drosophila (Fruit flies). In other reports using Drosophila malathion was weakly mutagenic. (80-0353)

In a chromosomal aberration test (sister chromatid exchange), it was weakly mutagenic. Nevertheless, malathion does not appear to be a mutagen of significance. (Marx, J.L. 1981)

Malathion is not mutagenic in the dominant lethal test in mice, (76-2783)

In plants, malathion produced chromosome damage. (78-0174)

Carcinogenesis

Waters, M.D. et.al. (1980) reported no carcinogenic activity in tests of both rats and mice. Cueto, Jr., C. (1980) cites two rat studies with the same result. I believe that one of these studies is the ne cited by Water, M.D. et.al. (1980). (79-0685)

Malathion has recently been used for the control of the Medfly in California. Because residential areas are being sprayed, there is concern for health hazards. The situation was reviewed by Jean L. Marx in Science 31 July 1981. Although there are some shady areas, malathion does not appear to be a carcinogen.

However, a Canadian study reported cancer in rats treated with malathion. (76-0204)

Teratogenesis

Proctor, N.H. and J.E. Casida (1975) reported malformations in chicken embryos after treatment with malathion.

Sternberg, S.S. (1979) states that "Malathion and other closely related compounds are teratogenic." p. 159. References are given. Other researchers agree. (78-0612)

There is a survey in progress to see if an increase in birth defects can be spotted in areas where malathion has been used. (Marx, J.L. (1981))

Malathion has produced teratogenic effects in rats (72-1971) and chickens. (72-2390) (72-2391) (73-1429) (76-0924) (78-0133) (78-0134)

However, a recent Canadian study proclaimed no effects in rats. (78-2982)

Reviews

The EPA produced an in-depth scientific review of malathion in 1975. This will be an excellent resource to that date. Unfortunately, I have not seen a copy at this time. (77-2262). Also see (78-2430). For biodegradation see Alexander, M. (1981).

Recommendations

Evidence for malathion being a mutagen exists, but it appears to be a rather weak mutagen, if at all. The same can be said for carcinogenicity. Further study is required on this point and probably more experiments. In the meantime, it is prudent to treat malathion as a teratogen. Pregnant women should not plant trees with malathion residues.

METIRAM

Metiram is the common name, but is usually referred to by its trade name of Polyram.

Characteristics: Metiram is a yellowish powder practically insoluble in water and organic solvents. It is a dithiocarbomate of the ethylene-bis-dithiocaromate group which means it has the degradation product ethylenethiourea(ETU) over which there is considerable concern. (Seiler, J.P.·1977) Some experiments indicate that when ETU residues on uncooked food are low, after cooking they become significant. (McEwen, F.L. and G.R. Stephenson 1979) Metiram is a broad spectrum fungicide used on a wide variety of crops.

Acute Toxicity

Metiram is an eye and skin irritant. Mammalian toxicity is low, the oral toxicity for rats LD_{50} = 6,200 to greater than 10,000. (Miller, A.V. and S.M. Craig, 1979) It is apparently goiter producing. (McEwen, F.L. and G.R. Stephenson, 1979)

Mutagenesis: Teratogenesis: Carcinogenesis:

I have very little information on metiram. The degradation product ETU is known to be teratogenic in rats (at 10 and 5 mg/kg) but not in rabbits. (McEwen, F.L. and G.R. Stephenson, 1979)

Several dithiocarbamates have been shown to be mutagenic, but not metiram. Bacterial studies, chromosome studies and a dominant lethal study in mice showed no effect or a very weak effect. However, when ETU is added to sodium nitrate, a common food additive, the resulting nitroso-ETU is very strongly mutagenic in several systems. Apparently, nitrosation takes place in the stomach and the nitrosated compound is circulated in the body. (Shirasu, Y. et.al. (1977))

Metiram is on the IBT list. (See Appendix)

Recommendations

I have not seen enough data on metiram to be confident about recommendations. ETU appears to be a teratogen and pregnant women are advised not to plant trees with residues on them. ETU appears to be a mutagen and suspected carcinogen only on nitrosation. This means that it is a very bad idea to eat when ETU residues may be on your hands. I would treat metiram with considerable caution.

PROMETONE

The common name is prometone. It is also known as Gesafram, Pramitol and Primatol.

Prometone is a pre-emergence and post-emergence triazine herbicide. It has a low mammalian toxicity, and LD_{50} (oral) for rats is 2,245-2,980 mg/kg. (Miller, A.V. and S.M. Craig 1979)

Prometone did not cause mutations in the Ames test. (Plewa, M.J. and J.M. Gentile 1976). However, this is also true of another triazine herbicide, atrazine, which is mutagenic in several systems.

See "triazine herbicides" for more information.

PROMETRYNE

The common name is prometryne. It is also known as G 34161, Gesagard, Caparol, and Primatol Q.

Chemical Name: N, N^1-Bis(l-methylethyl)-6-methylthio-1,3,5-triazine-2,4,diamine.

Empirical Formula: $C_{10}H_{19}N_5S$

Characteristics: A solid crystal at room temperature, soluble in water and organic solvents.

Prometryne is a pre-emergent and post-emergent trizine herbicide. It is fairly persistent in soil, and has a low toxicity to fish, birds and wildlife. Mammalian toxicity is low. The LD_{50} for rats (oral) is 3,150-3,750. The dermal LD_{50} for rabbits is greater than 10,200 mg/kg.

In the Ames test, prometryne was not mutagenic. However, other triazine herbicides that are not mutagenic in the Ames test are mutagenic in other systems. See "triazine herbicides" for more information.

Prometryne forms N-nitroso compounds which are mutagenic after metabolic activation. N-nitroso compounds are known carcinogens. (77-0603) See "atrazine" and (76-2267).

A Russian study concluded that prometryne is "definitely gonadotoxic" to male rats.

There does not seem to be a lot of information on prometryne, but the above information is enough for me to treat it as a mutagen and carcinogen. The N-nitroso compounds will form in the stomach, so be sure to wash before eating. Significant residues on the seedling are unlikely.

PROPAZINE

The common name is propazine. It is also called Milogard, Primatol P, G-30028 and Propazin.

Chemical Name: 6-Chloro-N,N^1bis(l-methylethyl)-1,3,5-triazine-2,4-diamine.

Empirical Formula: $C_9H_{15}ClN_5$

Characteristics: Propazine is a colorless crystalline solid at room temperature. It is slightly soluble in water and difficult to dissolve in organic solvents.

Propazine is a triazine herbicide. It is persistent in the soil and one application is effective for an entire season.

(Miller, A.V. and S.M. Craig, 1979)

(Windholz, M. et.al. 1976)

Acute Toxicity

The LD_{50} (oral) in rats is greater than 5,000 mg/kg. This indicates a low mammalian acute toxicity. (Miller and Craig 1979) Propazine can cause contact dermatitis. (73-0352)

Propazine tested in the Ames test did not show mutagenicity. (Shirasu, Y. et.al. 1977). I have seen no other information on the mutagenic, carcinogenic or teratogenic effects of propazine.

See “triazine herbicides”.

SIMAZINE

The common name is simazine. It is also known as Gestatop, Primatol S. Princep, Simmaprim, and Simanex.

Chemical Name: 6-Chloro-N,N^1-diethyl-1,3,5-triazine-2,4-diamine.

Empirical Formula: $C_7H_{12}ClN_5$

Characteristics: The solid crystal is practically insoluble in water. It is a triazine herbicide similar to atrazine (q.v.). It is held tightly in the soil and does not break down easily.

Acute Toxicity

The acute mammalian toxicity is very low, the oral LD_{50} in rats being 5,000 mg/kg. The dermal LD_{50} for rabbits is 8,160 kg/mg.

(Miller and Craig 1979)

(Windholz, M. et.al. 1976)

Simazine can cause contact dermatitis (73-0352).

Mutagenesis

Like atrazine, simazine is not mutagenic in the Ames test. (Shirasu, Y. et.al. 1977) (Waters, M.D. et.al. 1980) However, both atrazine and simazine are mutagenic after plant activation. Simazine increased the frequency of chromosome aberrations in plants and induced dominant lethal mutations in Drosophila (fruit flies). (Plewa, M.J. 1978) (78-0371) It has also produced sex-linked lethal recessive mutations in the same species.

Plewa, M.J. (1978) writes: "Thus, the majority of data reported in the investigations outlined above indicate that …simazine… induce(s) both mitotic and meiotic chromosome aberrations and are biologically activated into agents that induce point mutations." p. 47.

Carcinogenesis

Simazine was carcinogenic in both rats and mice. (72-1017)

Reviews

For a review of mutagenesis of simazine see Plewa, M.J. (1978).

Recommendations

Simazine should be treated as a mutagen and a suspect carcinogen. Recommendations are the same as for atrazine (q.v.). Also see "triazine herbicides". Simazine is on the IBT list (See Appendix).

WHAT HAPPENS FROM HERE?

Obviously, we have to follow through on the residue tests for all pesticides that could affect planters. We must see that the notification process is carried out. (See Appendix IV)

This report could be revised and updated from time to time, especially Section III: PROPERTIES OF PESTICIDES. For those pesticides that have significant residues on the trees, a more critical and thorough review of the literature is in order. The pesticides can be assessed according to several alternate guidelines that have recently been developed.

Other related information such as the California re-entry times, could be investigated.

Another aspect of the pesticide issue is the contamination of planting sites. 2,4-D and Krenite are briefly reviewed in the appendix, but a more detailed review could be done.

A separate health issue concerns planting in burned areas. Soot and charcoal are well known carcinogens. This could be a more serious health threat than the pesticides and should be investigated.

Specific cases of suspected pesticide poisoning, or contact dermatitis, such as occurred this summer, should be carefully documented, samples taken, and carefully investigated by someone well acquainted with the problems involved.

It will be very difficult to get any of this done on a volunteer basis. Treeplanters are going to have to finance these projects if they want thorough and quality information.

APPENDIX I: INDUSTRIAL BIO-TEST LABORATORIES

The Industrial Bio-Test Laboratories (IBT) conducted many tests on the toxic effects of pesticides. The U.S. Food and Drug Administration inspected the laboratory and found large deficiencies in the testing procedures. In 1977 the EPA started audits on these tests. It took several years for the audits to be completed. For captan, the audits were completed on May 15, 1979. Of the 12 studies on captan done by IBT, none were valid. (EPA 1980a, p. 13) For other pesticides, the results are also invalid. The problem is, many of these studies appeared to be well done on paper, and were often the best evidence available. They often indicated that the substance was not, say, mutagenic or carcinogenic. The tests were used to assess the hazards of pesticides, so many pesticides were put out on the market with safety assessment and regulations based on false data. Apparently, there are over two hundred such substances. A partial list follows:

TABLE 1 - IBT PESTICIDES USED IN BRITISH COLUMBIA

CSA COMMON NAME	TRADE NAME
acephate	ORTHENE
alchlor	LASSO
allidochlor	RANDOX
barban	CARBYNE
bis (tributyltin) oxide	BUTINOX
calcium hypochlorite	HTH
captafol	DIFOLATAN
captan	CAPTAN
carbofuran	FURADAN
chlorbromuron	MALORAN
chlorthalonil	BRAVO
chlorpropham	CIPC
chlorthaldimethyl	DACTHAL
crotoxyphos	CIODRIN
coumaphos	CO-RAL
cyanazine	BLADEX
cyprazine	OUTFOX
daminozide	ALAR
diazinon	BASUDIN
dichlobenil	CASORAN
dichlorvos	VAPONA
dinoseb	SINOX
diquat	REGLONE
disulfoton	SI-SYSTON
endosulfan	THIODAN
ethion	ETHION
fensulfothion	DASANIT
folpet	PHALTAN
formetanate hydrochloride	CARZOL
glyphosate	ROUND-UP

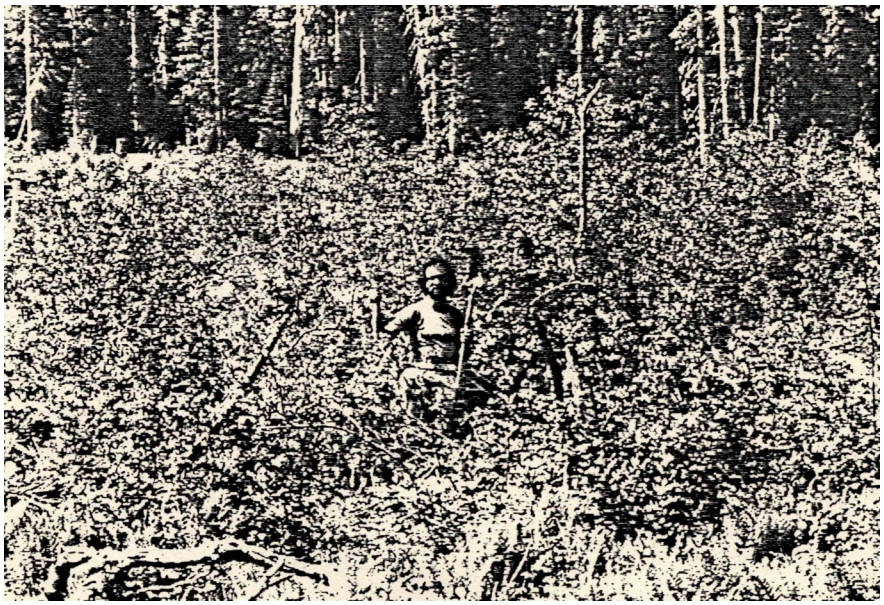
mefluidide	EMBARK
methamidophos	MONITOR
methidathion	SUPRACIDE
methiocarb	MESUROL
metiram	POLYRAM
metobromuron	PATORAN
metolachlor	DUAL
metribuzin	SENCOR
naled	DIBROM
oxydemeton-methyl	METASYTOX-R
paraquat	GRAMOXONE
phenothrin	SUMITHRIN
phosphamidon	DIMECRON
picloram	TORDON
propargite	OMITE
propham	IPC
propoxur	BAYGON
simazine	PRINCEP
sodium chlorate	ATLACIDE
tetrachlorvinphos	GARDONA
tetradifon	TEDION
triallate	AVADEX BW

APPENDIX II: 2,4-D and Krenite

The herbicides 2,4-D and Krenite are used for thinning on or near planting sites. There are studies that chemical thinning doesn't work, but a detailed discussion is beyond the scope of this report.

2,4-D is a phenoxyacetic herbicide which contains the contaminant TCDD. TCDD is a very dangerous mutagen. There is evidence that 2,4-D causes cancer and birth defects. (Galston, A.W. 1979) (Warnock, J.W. and J. Lewis 1978) (Tomatis, L. et.al. 1978) (Seiler, J.P. 1979) (Axelson, 0. and L. Sundell 1974) (NCAP 1978) (CATS (nd.)) I would definitely avoid an area treated with 2,4-D. It is not supposed to be applied near water, but I have talked to foresters who do not take this prohibition seriously.

Krenite is a trade name for fosamine ammonium. It is a carbamate herbicide about which I have very little information. LD50 is 10,200 mg/kg orally for rats. It may irritate eyes, nose, throat, or skin. It is not to be used on any food crops. (DuPont 1976) (Miller, A.V. and S.M. Craig) I have not seen any data on mutagenesis, carcinogenesis, teratogenesis or acute toxicity.



APPENDIX III - THE TERRACE INDICENT

BACKGROUND INFORMATION

The Terrace incident where several planters were exposed to unusually high concentration of Captan and Benolate with disastrous results highlights that planters have had problems with pesticides in treeplanting. We would like to take this opportunity to propose some solutions to our problems.

It is now apparent to all that complete notification is required with shipping invoices and that recent applications must be marked on or noted in the boxes.

It concerns us that treeplanters, as one of the healthiest population groups in Alberta should experience colds, stomach upset, headaches, nausea and diarrhea more frequently during the planting period than during the rest of the year.

Another very healthy population group, mountain climbers/hikers do not experience this during equally stressful and physically demanding activity.

Poor camp and kitchen hygiene was originally blamed for these symptoms but high standards of camp and kitchen cleanliness have failed to eliminate this phenomenon. The drinking water crews are using may be a cause of general malaise in camp.

It is necessary to seriously consider the potential pesticides/fungicides on the trees as a possible contributing factor. The only direct way to begin to determine if this is the cause is through a thorough testing of the trees for pesticide residue. These tests must be characteristic of trees handled by the planters. We would like to participate in the testing to ensure that field conditions are simulated. Such an agreement has been reached with the Minister of Forests in B.C. on July 11, 1981.

During our projects in Alberta this spring, it was commented by the planters that the bareroot trees smelled of some kind of chemical. Especially if the box was out in the hot sun for an hour after which you had to be careful or you would get nauseous from the fumes. One inexperienced planter in our Whitecourt contract, on the second day having pulled trees from his bag and gotten 6 or 8, not knowing where to hold them, put them in his mouth. He reacted immediately by vomiting and remained sick and continued vomiting for 16 hours. This incident resulted in several of our experienced planters; who were aware of the hazard of the pesticides, quitting that project. Notification data must become available.

Both Alberta and BC are experiencing major increases in reforestation and other silvicultural field work. This work is primarily done by crews who live out near the sites.

Recreational camping is increasing more rapidly than the population. Oil exploration is experiencing a boom in the long range. All these people who camp out can be unknowingly exposed to the hazards of the proposed increase in silvicultural pesticides/herbicides, and the current use of pesticides by the railroads, department of highways, Hydro and agriculture, unless filed notices are posted upon spraying. Planters enter an area without local knowledge, in a hurry to set up and begin work. To date planters in B.C. have planted on areas recently treated with 2-4-D, Turdon K and Roundup, tapped into a mine tailings (with mercury) settling pond for water system and drank water draining areas sprayed with defoliants.

In 1980, a thorough check of the BC Pollution Control Board application files for 1979 by Alan Cairns (PhD) a PRWA treeplanter, identified 150 applications for pesticide spraying in areas that could effect planting crews. These applications should require posting of the sprayed areas. Failure to post areas could be made a civil offence.

A program which maps these applications and makes their locations easily available to planting crews might be coordinated through a simple mini-computer system which is programmed with direction of flow in water sheds and records of all applications. A request for information about a particular longitude and latitude would yield the list of applications upstream or on site within the previous year.

This would seem to be an invaluable defense mechanism for the healthy development of this province. The proposal, presently agreed to in BC, that the preparation of a contract include a check of existing or potential hazards, both natural and man made, and that this information be made part of the contract, in our opinion, would satisfy the requirements of the treeplanter.

Dirk Brinkman
Ted Davis
June 30, 1981

Effects of planting 26,000 pine 2+0 bareroot seedlings dipped in Captan and Benolate four days before planting.

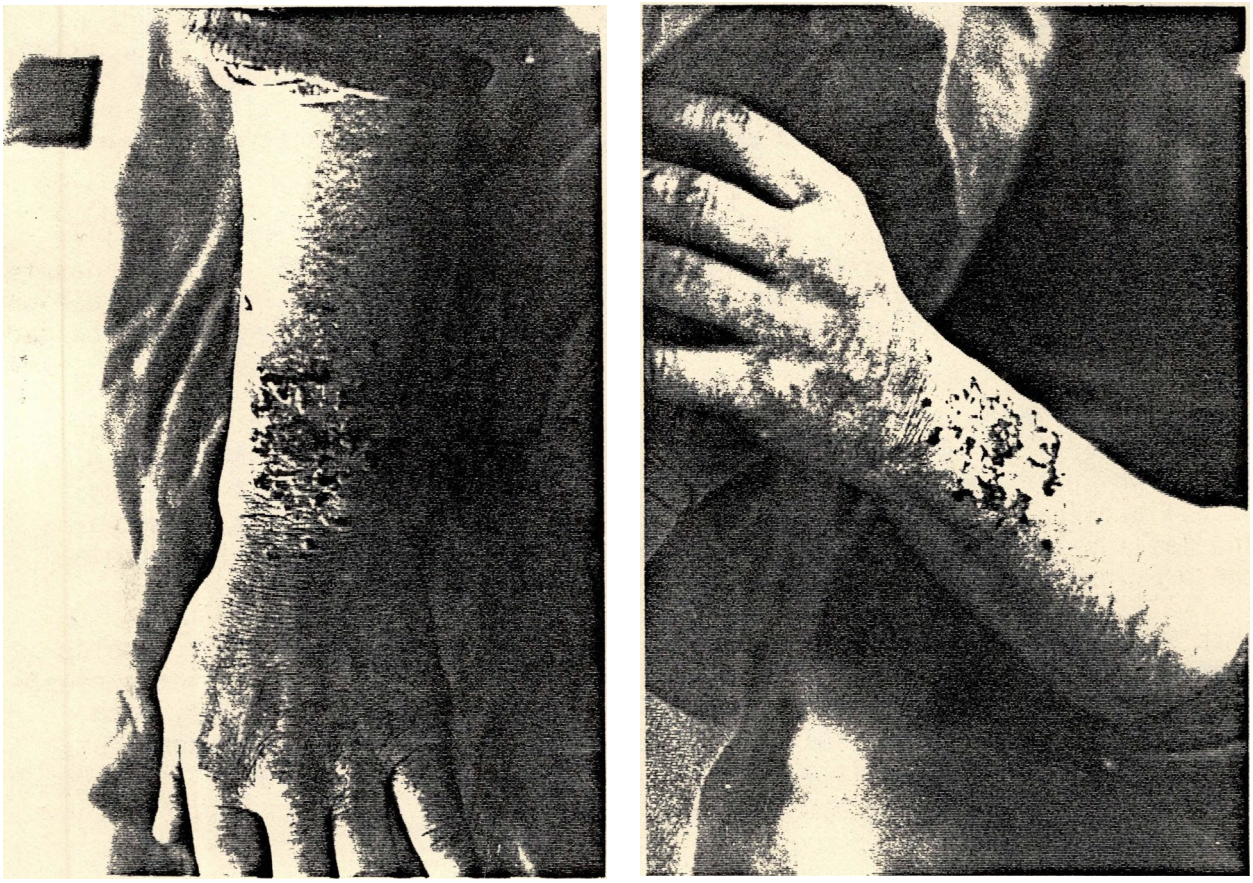


Photo of Bob Farrel's arm taken 1 week after exposure to the unusually high concentrations of Captan and Benolate.

260,000 2+1 pine bareroot seedlings, seedlot #2161, at Surrey nursery, contracted a European moth problem which required fumigation with methyl bromide (64 gram per litre of cubic air) between February 1, 1981 and March 3, 1981. Subsequently a mould developed in the fumigated seedlings. Just before shipping from April 8 to April 21 the seedlings were dipped in a solution of Captan-Benolate and water. (Captan 1 gram/litre, Benolate ½ gram/litre). Immediately after dipping the seedlings were put in water tight paper bag lined boxes and sent to Terrace, Canada. Cellulous received them with many of the boxes soaking wet. The trees were immediately sent up to the planters. Both the company and the planters made some effort to identify the chemicals the trees were obviously soaked with. Unfortunately this was Easter weekend. No one in forestry was in the offices until Tuesday. No data was immediately available. After Bob Farrel and the other planters had planted a day the company came in with a pesticide history of the trees.

Of the two crews of 18 planters and 14 planters, only the following planters were willing to plant these trees on April 19th and 20th with the listed effects.

CREW I Bob Farrel

Re: Rash diagnosed as "contact dermatitis" by

Dr. Chercover of the Terrace Medical Clinic.

Bob began planting the pine the morning of April 16, 1981. He wore gloves. A rash developed by early afternoon on his arms from the edge of the gloves back. Especially on his left tree pulling and delivery arm. Dirt was falling off of the trees perforating his pants. He was wearing loose woven army fatigues with suspenders and the dirt was falling into his pockets and soaking through his pants. Rashes developed on his legs, behind the knee, on the ankle and in his crotch. A hard bacterial infection developed and chewed up his skin like mincemeat. The lymphatic system overloaded and couldn't handle it. His lymph nodes were so swollen he couldn't move. His skin broke out in boils on his groin and underarms. He was on his back for three days. Fifteen boils on each leg.

"That night was hell. The grossest thing you ever want to see. No one would come near me, not even my old lady, it was like leprosy. When I walked into the clinic in Terrace the nurse had a fit. Doctor Chercover brought all his interns over." He said he would not speculate on what caused this. The doctor was amazed at how quickly it developed and how quickly the rash disappeared after Bob stopped planting. The infections were treated with 1500 mg. of Penicillin and other heavy antibiotics, Domero and steroid creams; washing 4 to 6 times a day, Bob stayed in his trailer for 10 days until able to plant again.

WCB claim #XY81026316 - Bob Farrel

Mrs. Marion Schultz - 266-0211 Ext 351

Special claims adjudicator. Claim accepted June 5, 1980.

Ted Davis. "There are extreme allergic reactions to Captan and Benolate in certain individuals. It's possible that the heavy doses of fungicides stimulated the development of the infectious bacteria.

Louis Bernier: developed a rash from wetness soaking through his pants.

Jean Bernier: got a small scrape on his foot which got badly infected. No amount of care, soaking, cleaning, would clear it up. Infection ran up his leg. WCB claim. Occurred one week after planting pine.

Jan Vandendries: infections above the glove line on his wrist.

Graham Albertson: no effects.

Larry Degrauf: no effects.

CREW II Reported by Nora Lilligren, First Aid Attendant.

Ed Bamling: a rash that spread from his hands up to his armpits and down his sides it became so itchy and puffy he had to go to a doctor for antihistamines. The condition persisted for two weeks. Ed has had this so-called "spruce rash" before, this instance was the most severe. At this contract - the Torpy, where the trees grown in the U.S are untreated, he had no reactions and no itchiness.

Ross Beckjord: developed hives over a period of two-three hours after planting a different seed lot. The hives covered from his belly where his pants began over his chest and down his arms. Later his forehead broke out. Towards the end of the contract they began to subside. The condition persisted for over a week.

John Cooper, John Beerbower, Chris Stolley, Kim Smith and Patricia Menton all-expressed that the trees smelled and felt unusual. Out of the 10 planters, 8 developed headaches, irritated eyes, nose and sore throats. Several people had diarrhea for several days. (The same thing occurred on the previous contract at Menzies Bay when we got a shipment of hemlock plugs overdosed with a fungicide.)

Supervisor: John Kragen.

"I, John Kragen, planted several days worth of the pine trees in question. Upon beginning planting these seedlings I experienced a flat, metallic, chemical taste in my mouth and an hour later I had a headache and felt nauseous. Those experiences lasted until I discontinued planting these trees."

****

After these initial effects, all the planters refused to plant any more of these trees. Both the trees which were planted and the trees remaining in the boxes subsequently died within a week.

APPENDIX IV: NOTIFICATION AND RESIDUE TESTING

In August, 1979 at the PRWA "Vallican Jamboree", representatives of the BCFS agrees to provide pesticide notification on the contract description and later updated on the shipping invoice. The notification was to be a complete history of pesticide application for each particular seedlot. We also agreed to develop a series of residue tests. Thus, a strategy for dealing with the pesticide problem was born.

The strategy is simple. First, there was notification on the box to alert the uninformed planter to potential pesticide exposure. Second, the history of pesticide application would be attached to the shipping invoice to the contractor. Third, residue tests would give us an idea of how much pesticide remains on the trees at intervals after the last application. Thus, with the residue tests as a guide to the lifespan of the pesticides on the trees, and the history of pesticide application for a particular seedlot, we should be able to estimate how much pesticide is on any particular seedlot at any particular time. Finally, with the information in this report, an individual treeplanter should be able to make a risk assessment and to take appropriate precautions.

On September 14, 1979, I met with Jim Sweeten and Hans Elias at the Surrey Nursery. We discussed the residue tests and decided the following:

(1) That tests would not be carried out before October 15.

(2) That, if possible, all species grown at the Surrey Nursery be tested to allow for species specific reaction to chemicals.

(3) That both container grown and bareroot stock be tested to allow for possible variations between one year old and older stock.

(4) That, if possible, stock would be tested for residues of as many chemicals as were applied to the stock during the growing season prior to lifting.

(5) That the initial test would be carried out on the day of testing.

(6) That subsequent tests would be carried out in one month intervals to assess the effect of cold storage on chemical degradation (if any).

(7) That these subsequent tests be restricted to species and age-classes which yield residue in the previous test.

HANS ELIAS DEVELOPED AN EXCELLENT WORKING PLAN AS FOLLOWS:

PESTICIDE RESIDUE TESTS 1979

WORKING PLAN

I. OBJECTIVE:

To determine residual amounts of various pesticides on surfaces of forest seedlings with consideration for possible variations for –

(1) Species (foliar differences)

(2) Age-class (1+0 vs 2+)

(3) Length of storage

II. SPECIES AND AGE CLASS:

(a) For 1+0 age class container growth seedlings of three species will be tested.

(1) Sw

(2) Hw

(3) Fc

The use of stock grown for fall-planting is suggested.

(b) For 2+ stock it will be necessary to test some transplant stock as only Sw is available as 2+0.

(1) Sw (2+0)

(2) Fe (1MP+1)

(3) Hw (1P+1)

III. NO. OF TREES:

For II(a)

550 trees will be needed for each of the species (1-3) - 1 carton per species.

For II(b)

Numbers will vary, but again 1 carton per species is recommended.

TREATMENT

1+0 Stock will be treated with Captan-Benlate spray one day prior to lifting, then extracted and packaged in the usual manner.

2+ Stock will be lifted and packaged in the usual manner without fungicide spray.

TESTING

The first samples will be submitted for tests at day of lifting. Subsequent samples will be sent in one-month-intervals from stored cartons.

RESIDUES TO BE TESTED:

If feasible, seedling surfaces will be tested for residues of any pesticide applied to test stock during the previous growing season. A list of dates, amounts and name of pesticide sprayed will be drawn up prior to lifting and will be forwarded to the laboratory with the first samples.

For reasons unknown to me, this plan was never followed. On December 12, 1979, John Bruce, then Director, Silviculture, BCFS sent a letter to Peter Kendall, President, PRWA with some results of the assays. All the assays were done on captan and the results were reported in ppm. The nursery and seedlot numbers were included, but all the important dates of application and testing, and details of the storage procedure were not provided. We were advised that if contractors wanted to know about the pesticides for an individual seedlot, they could contact the nursery that grew the stock.

This letter is reproduced in full:

Province of British Columbia — Ministry of Forests — Forest Service

December 12, 1979

File: 125-39

Mr. Peter Kendall
President, Pacific Reforestation Workers' Association
Box 26
Ymir, B.C. V0G 2K0

Dear Peter:

This is to confirm the various points discussed during our telephone conversation the week before last:

1) Captan/Benlate residue tests being undertaken at the Environmental laboratories in Vancouver.

Seedlot & Stock type			Captan ppm.
✓Koksilah	Hw 2689	1+0P	Nil
✓Surrey	Hw 3006	1+0P	3.86
✓Surrey	Hw 2476	1+0P	2.46
✓Campbell R.	Ba 184	1+0P	21.10
✓Red Rock	S 1827	2+0	not completed'
✓Green Timbers	Pl 2185	1+0 c/Bw	Nil
✓Green Timbers	Se 2871	1+0 c/Bw	Nil

We have received advice that the tests for Benlate have still not been carried out, but are expected in the not too distant future.

2) The Regional Managers have been advised that should a planting contractor apply for a list of pesticides that have been used on an individual seedlot during the growing season prior to planting, such information will be available from the nursery that grew the stock.

3) I have reviewed the Association's request that information on any and all pesticides used in the watershed in which the planting is to take place be made available by the Forest Service. This is not a function or responsibility of the Forest Service and it is my suggestion that if you wish to pursue this aspect you do so through the Ministry of the Environment.

4) I undertook to make a formal reply to the various questions raised by the Association in its submission to the Minister last January. This is being compiled now and I will forward it to you shortly.

Yours truly,

J.B. Bruce
Director
Silviculture Branch

JBB/cm

Pacific Reforestation Workers' Association

Box 26, Ymir, B.C.
V0G 2K0

January 29, 1980

Ministry of Forests
Reforestation

Attention: John Bruce.

RE: Notification of the Planters of the Pesticides Used on the Seedlings In the Nursery.

On August 10, the PRWA Board met with representatives of the Ministry of Forests, including John Bruce (lic Reforestation), Jim Sweeton (Head of Nurseries), and Hans Elias (Surrey Nursery). At this meeting, John Bruce and Jim Sweeton committed the forest service to providing the planters with notification of all the pesticides used on the seedlings in the nursery during the year before planting.

Now, the PRWA understands that the Ministry of Forests plans to make notification the responsibility of the contractor by making a record of some of the pesticides used on the seedlots available only at the regional offices.

At the December, 1979 annual meeting of the PRWA membership, the reaction to this was unanimous: this is in contravention of the agreement arrived at in August.

In August, the PRWA was led to believe that information on all pesticides used on a seedlot was to accompany the shipping invoice with each shipment of contract seedlings.

In August, Hans Elias indicated that at the Surrey Nursery each seedlots pesticide treatments are listed on one page for their records. Pesticide notification with the shipping invoice would require little more than providing the personnel at the nursery who issue the shipping invoice with copies of the record sheet for each seedlot.

The PRWA further understands that the Ministry of Forests is limiting the pesticide information available at the regional offices to the chemicals Captan, Benlate and Daconil.

It is highly disconcerting that now the Ministry of Forests seems to consider this information either too unimportant or too inflammatory to communicate it directly to the crews.

Unfortunately, continuing PRWA Health Committee research into these pesticides is yielding discouraging information. The PRWA finds it increasingly imperative that notification reach the planter without special effort from the contractor. Contractors are notoriously busy during the height of their season. Regional offices are equally busy. Many contractors do not feel very concerned about the possible effects of these pesticides. These contractors will make little or no effort to pass along any information.

The PRWA both at the August meeting and in its publications has made it very clear that it considers the Ministry of Forests responsible for any health effects any pesticide may have on the planter, including long term effects that may only be determined years from now, unless there is good notification. Then it becomes the planters responsibility to choose to plant and to find protection against these chemicals.

Until there is proof that each pesticide used is no longer present on the seedlings, the PRWA regards these pesticides as present. After the results of the UBC labs test of the trees, perhaps this position will be reconsidered. In the present circumstance, the PRWA is obliged to demand that complete notification accompany the shipping invoice with the trees.

Sincerely,

Dirk Brinkman
Legal Committee Chairman

/ams

cc: Tom Waterland
John Bruce
Jim Sweeton
Dave Armit
Tommy Thomas, WCB
Nursery Superintendents
District Offices

Province of British Columbia
Ministry of Forests
Forest Service
Silviculture Branch
1450 Government St.
Victoria, B.C.
V8W 3E7

April 23, 1980

File: 125-39
720-12

Mr. Ted Davis
Pacific Reforestation Workers Association
Suite 102, 3371 Chesterfield
North Vancouver, B.C.
V7N 3N2

Dear Mr. Davis:

Further to the inquiry by you and Kate Riddle, the results of the fungicide residue assays by the Ministry of Environment Laboratory, subsequent to our letter of December 12, 1979 are as follows.

Nursery	Stock Type	Fungicide	Residue P.P.M.
Green Timbers	Pl 1+0P 3587	Captan	17.8
" "	" "	Daconil	1.5
Surrey	Hw 1+0P 3472	Captan	1.0
"	" "	Daconil	9.6
"	Sw 1+0P 3059	Captan	1.0
"	" " "	Daconil	16.8
"	" " "	Benlate	Nil
Koksilah	Hw 1+0P 0211	Captan	49.9
"	" "	Daconil	0.9
"	" "	Benlate	Nil
Skimikin	Pl 1+0P 1620	Captan	1.0
"	" " "	Daconil	58.1
"	" " "	Benlate	Nil
"	Sw 2+0 BR 2498	Benlate	Nil
"	Lo 1+0P 2554	Captan	1.0
"	" " "	Daconil	0.1
"	" " "	Benlate	Nil
Surrey	Sw 2+0 BR 1868	Benlate	Nil
Red Rock	Sw 2+0 BR 1868	Captan	Nil
"	" " "	Benlate	Nil
"	Sw 2+0 BR 1827	Benlate	Nil
"	Pl 2+0 BR 2321	Benlate	Nil

..../2

In addition, testing for residue levels of other pesticides used, provided the following results:

Nursery	Stock Type	Pesticide	Residue P.P.M.
Koksilah	Hw 1+0P 0211	Diazinon	0.2
Skimikin	Pl 1+0P 1620	"	0.1
"	Sw 2+0 2498	"	0.7
"	Lw 1+0P 2554	"	0.1
Surrey	Sw 2+0 1868	Orthene	Nil
"	Sw 1+0P 3059	Diazinon	0.6
"	Hw 1+0P 3472	"	1.1

The Environment laboratory has now processed all stock and residue samples scheduled for assay, so this completes the record on pesticide residue data available at this time.

Sincerely,

David Armit
Manager, Nursery Operations
Silviculture Branch

DA:ka

Next, I wrote each of the nurseries for the history of pesticide application and seedling storage. That information was provided without undue delay, but with some confusing information. As for the testing dates and other details, the Ministry of Environment Laboratory considered that information confidential. Attempting to sort out this material, the following correspondence took place:

#102-3371 Chesterfield
North Vancouver, B. C.
V7N 3N2

13 August 1980

Mr. David Armit
Manager, Nursery Operations
Silviculture Branch
1450 Government St.
Victoria, B. C.
V8W 3E7

Dear Mr. Armit:

I am writing for information concerning the pesticide residue assays completed by the Ministry of Environment Laboratory.

I have received from Jim Sweeten, D. Prideaux, and Helmut Mueller the pesticide application histories of the seedlots listed in your letter of April 23, 1980. There is some confusing information that you may be able to clarify.

1.) Sw 2+0 BR 1868 was not treated with Benlate, yet was tested for the fungicide. Naturally, there was no residue.

2.) Pl 1+0 P 1620 listed as from Skimikin, is, according to Jim Sweeten, "not a Skimikin or Vernon seedlot." The application history of this seedlot would be interesting. Four pesticides were tested, and Daconil had an unusually high residue: 58.1 ppm.

3.) Sw 2+0 BR 1827 was tested for Benlate. D. Prideaux wrote me: "We did not have this seedlot as 2+0. If 1877 was the seedlot, then it had the same treatment as above. (Treatment with Benlate.) We did have this seedlot as 2+1 transplants and they received no Benlate treatment...."

The above information causes me to suspect that there may be errors of transcription in your letter of April 23, 1980. A check on the accuracy of the data in that letter would be appreciated.

I also wrote to the Ministry of Environment Laboratory requesting the following information:

1.) The date they received each seedlot.
2.) How the seedlots were stored until the assay.
3.) The date of assay for each seedlot and pesticide.
4.) The degree of sentisitivity of the assay and the method used.

I also requested comparative data on fruits and vegetables for Benlate, Captan, Daconil, Diazinon, and Orthane.

I. Kalnins, the assistant director of the laboratory, replied that the information I requested had been sent to you, and that the laboratory policy is to only send information to the submitting agency. I would greatly appreciate that information.

Thank you,

Ted Davis
PRWA Health Committee

4 December 1980

Mr. David Armit
Manager, Nursery Operations
Silviculture Branch
1450 Government St.
Victoria, B.C.
V8W 3E7

Dear Mr. Armit:

It has been several months now since I wrote you asking for clarification of the pesticide residue assays. In my letter of August 13, 1980, I requested information clarifying the residue assays as follows:

1.) Sw 2+0 BR 1868 was not treated with Benlate, yet was tested for the fungicide. Naturally, there was no residue.

2.) Pl 1+0 P 1620 listed as from Skimikin, is, according to Jim Sweeten, "not a Skimikin or Vernon seedlot." Four pesticides were tested, and Daconil had an unusually high residue.

3.) Sw 2+0 BR 1827 was tested for Benlate. D. Prideaux wrote me: "We did not have this seelot as 2+0. If 1877 was the seelot, then it had the same treatment as above. We did have this seedlot as 2+1 transplants and they received no Benlate treatment...."

As I mentioned in my lastletter, the above information causes me to suspect errors of transcription in your letter of April 23, 1980. Once again, a check on the accuracy of the data in that letter would be appreciated.

In my letter of August 13, 1980, I also requested the following information:

1.) The date the lab received each seedlot.
2.) How the seedlots were stored until the assay.
3.) The date of assay for each seedlot and pesticide.
4.) The degree of sensitivity of the assay and the method used.

Obviously, the information you sent me on April 23, 1980, is not very meaningful without clarification of the above points.

During the past few months I have been reviewing the scientific literature on Captan and Benlate and have concluded that there is enough evidence of chronic toxic effects of these fungicides to cause reasonable concern. I have written to the U.S. Environmental Protection Agency (EPA) and other organizations. They have provided me with information that agrees with my conclusions.

Captan and Benlate have been identified by the EPA as posing a potential health hazard and have been reviewed under their most rigorous review system, the Rebuttable Presumption Against Registration (RPAR) Program.

The final position document by the EPA on Benlate (PD 2/3, August 22, 1979) makes clear that the toxicity of Benlate is real and serious, but discontinuing its use would have adverse economic and social effects. However, they have recommended that the following warning be placed on all pesticide products containing Benlate that weigh five pounds or more:

"Warning to Workers"

> "The United States Environmental Protection Agency has determined that benomyl causes birth defects and reduced sperm production in laboratory animals. Exposure to benomyl during pregnancy should be avoided. Exposure to benomyl might cause a depressed sperm count. Workers must be sure to wear a cloth mask while mixing benomyl for aerial application. In case of accidental spills or other unusual exposure, cease work immediately and follow directions for contact with benomyl."

The EPA document suggests precautions to reduce exposure such as masks and protective clothing. The document also recommends additional mutagenicity testing as data in this regard is suggestive of adverse effects. Benlate is no longer allowed on rice at a projected loss to growers of 15 million U.S. dollars each year. The EPA obviously considers benomyl dangerous.

I am advised by Marcia Williams, Director, Special Pesticide Review Division, EPA, that a new study has indicated that Benlate is carcinogenic (i.e. oncogenic) and that the EPA may decide on further restrictions on the use of Benlate.

Captan is also under RPAR review, but the progam is not as far along as with Benlate. The EPA has concluded that Captan is a mutagen and oncogen, but have no conclusion about its teratogenic potential. However, there is some very suggestive evidence that Captan may be a teratogen in humans. (PD 1, July 22, 1980). I expect the EPA will have made regulatory restrictions on Captan in about six months.

Other pesticides present potential hazards. For example, Daconil has recently been shown by a National Cancer Institute study to be an oncogen and has been brought under RPAR review. The literature on Diazinon is also of concern.

The EPA regulations are primarily for the protection of applicators and others who are at high risk, and the general population in terms of low level chronic exposure. In the latter case, the effects would be real but undetectable in the general population. The EPA has to consider regulatory action in terms of economic, social and political atmospheres as well as in terms of individual risk. The matter eventually becomes one of subjective determination of risk.

In the past some have felt that the determination of risk should be left to management and "the experts". But because of questions of integrity, competency, and motivations of those in power, and the recognition that the individual taking the risk has a right to know that there is a risk, the system of keeping the workers in ignorance is fast fading.

The PRWA is interested in coming to a practical resolution of the pesticide issue. Our position is that the issues involved can be explained in an understandable way to an intelligent person. The person taking the risk has a right to know that a risk exists and to make his or her own personal risk assessment. To make this assessment one has to have notification, background information, and data. To provide this information should be the function of the PRWA and the BCFS.

Our strategy is simple and inexpensive. First, we need to have notification. The treeboxes should be marked "treated with pesticides" or a warning enclosed to that effect. This warns the planter of the potential hazard, but says nothing about the seriousness of the hazard or degree of risk. To cover this second aspect of the situation, we need to have a copy of the history of pesticide application for the seedlot. This should be attached to each invoice for each contractor. With the appropriate background information (provided by the PRWA and based, in part, on the residue assays) the planters should be able to take appropriate precautions. For example, if the stock was recently treated with Captan, precautions such as protective clothing and washing would be prudent. But if the stock had not been treated recently, and the results of the assays and other information indicate that the pesticide has been metabolized or washed away, then the precautions could be relaxed.

The Health Committee of the PRWA is in the process of providing general background information in a non-technical form, and guidelines to reduce exposure.

Essential to this strategy is cooperation from the BCFS. First, we must have a data base on residues so we can predict the amount of pesticide on untested stock by simply knowing the history of pesticide application. For this we need the information I requested several months ago and outlined in the beginning of this letter. Second, we must have the two types of notification: 1) a general warning on or in each box, and 2) the history of pesticide application with each seedlot and invoice.

If the BCFS continues to take these matters lightly, there my be problems during the coming planting season. We are certainly going to notify treeplanters of the situation, and without sufficient information on which to base their decisions, many planters will assume the worst and not plant. Other planters will treat the trees as contaminated and plant them with time consuming and often unnecessary precautions. This unproductive situation can be avoided with your cooperation.

The PRWA and its members are interested in planting trees. We are not interested in an expensive confrontation with the BCFS. The precautions workers need to take to protect themselves and their children are simple, but must be based on sufficient information.

Sincerely,

Ted M. Davis

Province of British Columbia
Ministry of Forests
Forest Service
Silviculture Branch
1450 Government St.
Victoria, B.C.
V8W 3E7

December 15, 1980

Ref.: 125-39
720-12

Mr. Ted M. Davis
Pacific Reforestation Workers Association
3458 West 20th Avenue
Vancouver, B.C.
V6S 1E4

Dear Mr. Davis:

Your letter of December 4, 1980 requesting additional information on residue assays has been received. The earlier inquiry on the same subject to which you make reference appears to have gone astray.

The information supplied to you last April 23, 1980 by this office is exactly the same as recorded by the Environmental Test Laboratory and forwarded to this office. The "errors of transcription" were discussed with our field agriculturist who was co-ordinating the pesticide residue assay program. He acknowledges such errors might have occured in relay of information between him and the nurseries or between him and the laboratory. As the study was originally initiated for internal Ministry purposes he was principally intent in ensuring he got stock that had been treated with particular pesticides for assay to determine residue levels, and might have occassionally recorded incorrect seedlot or nursery designations. He confirms that the stock tested was treated as reported, and that any discrepancies were in stock type or nursery designation. The data recording and record control procedures have been revised, to reduce the potential for such errors in any future reports circulated on pesticide residue assays.

In respect of your query on Environmental Lab assay

1) Seedlots 3587, 3472, 3059 received Oct. 29, 1979.
" 0211, 1620, 2498, 2554, 1868, received Nov. 21, 1979.
" 2321 received Feb. 8, 1980.
(Seedlot numbers as orginally recorded and reported by the Laboratory)

2) All stock was held in standard refrigerated storage at 1°C in cartons until the assay was carried out.

3) Assays on all seedlots except 2321 were done in later January 1980 and reported on January 29, 1980. Seedlot 2321 was done third week of February and reported on February 22, 1980.

..../2

Defined sensitivity limits are:

Benlate	- less than	1.5	parts	per	million
Captan	- "	"	1.0	"	"	"
Daconil	- "	"	0.04	"	"	"
Diazinon	- "	"	0.10	"	"	"

The Environmental Laboratory reports that these detectable "limits" are larger than the actual detectable limits, but it is necessary to allow some tolerance because detectable levels do vary, depending on the type of sample tested. They advise that any residue level less than the stated detectable limit is considered not detectable for practical purposes.

We will be continuing our pesticide assay tests this coming winter and spring, with attention to maintaining accurate records and reporting procedures.

I note with interest the literature review you have undertaken on pesticide usage and risk factors. The Ministry of Forests share your concern that pesticides are used properly and safely, and only when necessary.

We support and adhere to the regulations and guidelines set down by Environment Canada, B.C. Ministry of Environment, Ministry of Health and Workers Compensation Board on pesticide registration, usage and safety procedures in the application of pesticides in Ministry of Forest nurseries, and the subsequent handling of treated stock.

With respect to notification, the nurseries do provide such notation on stocked treated with pesticides and appropriate safe handling procedures on the invoice of every shipment of stock so treated in Ministry nurseries.

However much of the stock shipped from our nurseries does not go direct to a planting project, but is bulk shipped to Regional storage, for later distribution to district planting projects. Consequently, the originating nursery often is not aware who will be the planting contractor(s) or agent(s) for that stock. Rather, there is the possibility of further treatment after the stock leaves nursery jurisdiction either in regional storage or on-site at the planting project, of which the nursery cannot be aware.

Consequently Mr. J. Bruce the past Director authorized, as indicated in his letter to Mr. Kendall, Dec. 12, 1979 and Mr. C. Johnson the current Director, has reaffirmed, the policy that the Ministry nursery organization will make available to the Regional Manager and his designate District reforestation representatives, a list of pesticide application in the nursery over the previous year for each seedlot shipped to that Region. The Regional representatives will therefore have the necessary information available to deal with planting project inquiries on stock pesticides treatments as they arise. As the Regional and District Silviculture Officers are responsible for administration and supervision of all forestry activities in their areas, it is only proper that matters affecting planting projects in their area should be dealt with by them.

..../3

The P.W.R.A. representatives are aware from previous discussions with me and my nursery staff, and by visits to our facilities, that we do not take the usage of pesticides, or the appropriate safety practises lightly. The effective, safe use of such materials, and the development of alternate non-chemical sotck quality protection procedures is of continuing serious concern to this organization. We also recognize that common interest of the P.W.R.A. in ensuring that reforestation activities in this province are undertaken in as safe an environment as can be developed.

We will provide effective channels of communication that disseminate the necessary information at the field level for the assessment of persons involved in planting projects, as indicated above.

Sincerely,

D. Armit
Manager - Nurseries
Silviculture Branch

DA:lk

At this point I found the time to start correlating the amount of residue with the time since the application of the pesticide. The results are given in the table below:

STOCK TYPE	FUNGICIDE	DATE LAST APPLICATION	DATE TESTED	INTERVAL (DAYS)	RESIDUE PPM
P1 1+0P 3587	Captan	Oct 16	Late Jan(28?)	163	17.8
P1 1+0P 3587	Daconil	July 31	"	181	1.5
Hw 1+0P 3472	Captan	June 20	"	222	1.0
Hw 1+0P 3472	Daconil	Dec 3	"	56	9.6
Sw 1+0P 3059	Captan	June 20	"	222	1.0
Sw 1+0P 3059	Daconil	Sept 21	"	129	16.8
Sw 1+0P 3059	Benlate	Not applied	"	?	NIL
Hw 1+0P 0211	Captan	Oct 4	"	117	49.9
Hw 1+0P 0211	Daconil	June 28:Jan 7	"	21	0.9
Hw 1+0P 0211	Benlate	Jan 27	"	21	NIL
P1 1+0P 1620	Captan	?	"	?	1.0
P1 1+0P 1620	Daconil	?	"	?	58.1
P1 1+0P 1620	Benlate	?	"	?	NIL
Sw 2+0 BR 2498	Benlate	Oct 15	"	105	NIL
Lo 1+0P 2554	Captan	Oct 2	"	119	1.0
Lo 1+0P 2554	Daconil	July 26	"	186	0.1
Lo 1+0P 2554	Benlate	Oct 2	"	119	NIL
Sw 2+0 BR 1868	Benlate	Not applied	"	?	NIL
Sw 2+0 BR 1868	Captan	Not applied	"	?	NIL
Sw 2+0 BR 1868	Benlate	Not applied	"	?	NIL
Sw 2+0 BR 1827	Benlate	Not applied??	"	?	NIL
P1 2+0 BR 2321	Benlate	Oct 4	Late Feb(21?)	72	NIL
Hw 1+0P 0211	Diazinon	Aug 20	Late January	161	0.2
P1 1+0P 1620	Diazinon	??	"	?	0.1
Sw 2+0 2498	Diazinon	June 15	"	227	0.7
Lw 2+0 2554	Diazinon	Not applied	"	?	0.1
Sw 2+0 1868	Orthene	July 25	"	159	NIL
Sw 1+0P 3059	Diazinon	Oct 12	"	108	0.6
Hw 1+0P 3472	Diazinon	Aug 16	"	165	1.1
Ba 184 1+0	Captan	Jan 17:Oct 4?	?	?	21.1
Hw 3006 1+0P	Captan	June 20	?	222	3.68
Hw 2689 1+0P	Captan	??	?	?	2.46
Hw 2476 1+0P	Captan	??	?	?	—
Sw 1827 2+0	Captan	Not applied??	Not completed	?	—
P1 2185 1+0	Captan	??	?	?	NIL
Se 2871 1+0	Captan	??	?	?	NIL

Of thirty six tests, 16 (44%) have no information on the time interval between pesticide application and the testing, 25% of the seed lots were not identifiable by the nurseries. The laboratory did at least 7 tests (19.4%) for pesticides that were never applied to the trees. (According to the nursery data). One seed lot was tested after 3 weeks, another after nearly two months. The average time between application and testing is 139 days, or over four and a half months. I wonder how much money and effort was wasted on this useless project.

Finally, on June 11, 1981 Dirk Brinkman and I met with Minister of Forests, Tom Waterland, Minister of Environment, Stephen Rogers and the director of pesticide control branch, Ron W. Kobylnyk. We presented my letter of December 4, 1980 (to D. Armit), Armit's reply, the working plan developed by Hans Elias, most of the information in Appendix III, and the following requests and background information.

Pacific Reforestation Worker's Association
Legal Committee box 4, riondel, b.c. vob 2bo

Summary of requests made June 11,1981

To the Honorable Steven Rogers,Minister of the Enviornment.

We reqest a combined Ministry of Forests and Ministry of Enviornment study of residue levels of pesticides used on seedlings.

We request notification of the pesticides used on the seedlings be delivered to the planters with the shipping invoice,and notification of all recent pesticide treatment be posted on the boxes.

We request posting of notices on the site of feild applications of pesticides to warn planters against drinking the water in these areas.

Submitted by Dirk Brinkman,Director and Ted Davis,Researcher on behalf of the PRWA.

BACKGROUND INFORMATION

It has come to the attention of the Minister of Environment, the Honorable Mr. Rogers, that several planters were exposed to unusually high concentration of captan and Benolate with disastrous results.

It is now apparent to all that complete notification is required with shipping invoices, and that recent applications must be marked on or noted in the boxes.

This highlights that planters have had problems with pesticides in treeplanting. We would like to take this opportunity to propose some solutions to our problems.

It concerns us that treeplanters, as one of the healthiest population groups in B.C. should experience colds, stomach upset, headaches, nausea and diarreah more frequently during the planting period than during the rest of the year.

Another very healthy population group, mountain climbers/hikers do not experience this during equally stresful and physically demanding climbs.

Poor camp and kitchen hygiene was originally blamed for these symptoms but high standards of camp and kitchen cleanliness have failed to eliminate this phenomenon. The drinking water crews are using may be a cause of general malaise in camp.

It is necessary to seriously consider the potential pesticides/ fungicides on the trees as a major contributing factor. The only direct way to answer this is through a thorough testing of the trees for pesticide residue. These tests must be characteristic of trees handled by the planter. We would like to participate in the testing to ensure that field conditions are simulated. Such an agreement was reached with the Minister of Forests in 1979, but the Ministry did not follow through.

This province is experiencing a major increase in reforestation and other silvicultural field work. This work is primarily done by crews out near the sites. Recreational camping is increasing more rapidly than the population. This year mining exploration is experiencing a boom. All these people who camp out will be unknowingly exposed to the hazards of the proposed increase in silvicultral pesticides/herbicides, and the current use of pesticides by C.P.R., C.N.R., B.C.R., Hydro, Fish & Wildlife, unless field notices are posted upon spraying. Planters enter an area without local knowledge, in a hurry to set up and begin work. To date planters have planted on areas recently treated with 2,4-D, Turdon K, and Roundup, tapped into a mine tailings (with mercury) settling pond for water system, and drinking water draining areas sprayed with defoliants.

In 1980, a thorough check of the PCB application files for 1979 by Alan Cairns (PhD) treeplanter, identified 150 applications for pesticide spraying in areas that could affect planting crews. These applications should require posting of the sprayed areas. Failure to post areas should be a civil offense.

It is a further problem that presently private lands in private or public watersheds, or power lines through crown land, or railroads or department of highways are not required to apply for a permit. In these cases applications are often avoided to minimize public opposition. Applications should be required for all use, including agricultural.

A program which maps these applications and makes their locations easily available to planting crews could be co-ordinated through a simple mini-computer system which is programmed with direction of flow in water sheds and records of all applications. A request for information about a particular longitude and latitude would yield the list of applications upstream or on site within the previous year.

This would seem to be an invaluable defense mechanism for the healthy development of this province.

Dirk Brinkman

June 11, 1981

Ted Davis

Summary of results of the meeting between:

The Hon. Stephen Roger
Minister of Environment
The Hon. Tom Waterland
Minister of Forests
Ron W. Kobylnyk
Director of Pesticide Control Branch
and
Dirk Brinkman
PRWA Director
Ted Davis
PRWA Researcher

11:00 A.M. June 11, 1981

Residue Testing

Minister of Forests Tom Waterland and Ron Kobylnyk, Director of the Pollution Control Board, agreed to initiate a study of the amount of residue on the seedlings. In addition, they will undertake a study to determine what percentage of the residue is undislodgeable and what percentage is dislodgeable. This might simply involve analysis of tree planters' gloves.

Tom Waterland agreed that tests should begin with a seedling right after treatment and continue at appropriate intervals until no residue was found. Test results of application and residue analysis will be published so that tree planters can act according to their own judgement - when or whether to plant the trees.

Seedling Notification

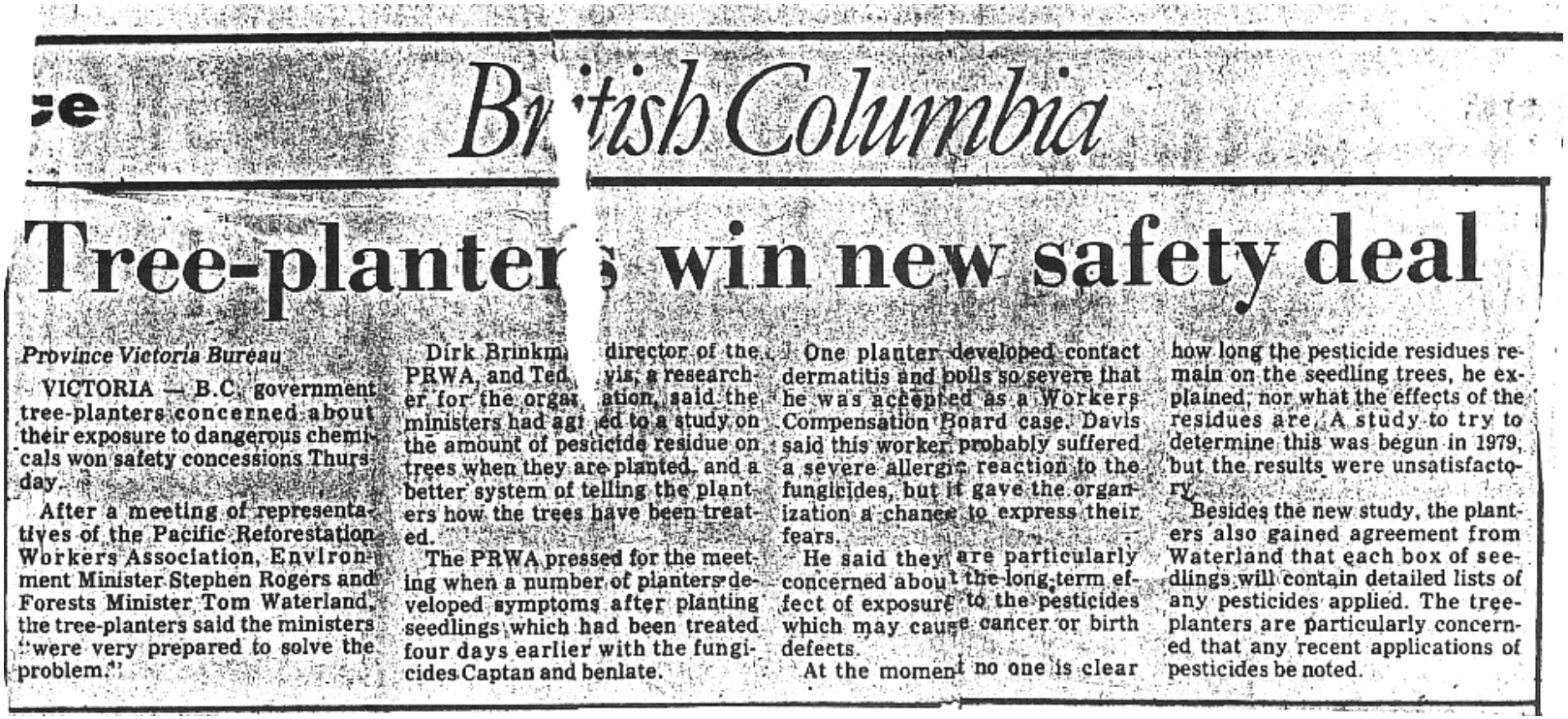
Tom Waterland said that he has already instructed the Forest Service to put shipping invoices with fungicide information in all the boxes, or at least on most of the boxes. He said he did not know what bureaucratic obstacles there might be in the way of complete notification of all the pesticide treatment. He noted that neither date nor amount of the pesticide was presently communicated and said he recognized that that information is necessary to be useful or for it to relate to residue tests. The PRWA said they were only looking for recent (1 month old or younger) applications to be notified in or on the boxes, and a complete pesticide history of the seedlot to be included with the shipping invoice.

Site Notification

Stephen Rogers and Ron Kobylnyk did not consider the posting of treated sites necessary and felt that it would create "an unnecessary additional expense".

Stephen Rogers suggested taht the crew check with the local Ministry of Environment representative. "Take Bella Coola for example: it is isolated but there is a representative there." Dirk Brinkman pointed out that some of the crew that worked in Bella Coola this year flew straight into the area from an isolated inlet in the Queen Charlotte Islands. They could not easily check with Bella Coola.

Tom Waterland pointed out that the preparation of a planting contract takes place over a long enough period to allow the forestry to check with the Ministry of Environment on potential hazards. Waterland said he would try to arrange for this information to be available in the contract. He said he would pursue this with Mike Apsey to see if this land of check could be a part of the preparation of the contract.



:e British Columbia

Tree-planter s win new safety deal

Province Victoria Bureau

VICTORIA — B.C. government tree-planters concerned about their exposure to dangerous chemicals won safety concessions Thursday.

After a meeting of representatives of the Pacific Reforestation Workers Association, Environment Minister Stephen Rogers and Forests Minister Tom Waterland, the tree-planters said the ministers "were very prepared to solve the problem."

Dirk Brinkm director of the PRWA, and Ted vis, a researcher for the orga ation, said the ministers had agr ed to a study on the amount of pesticide residue on trees when they are planted, and a better system of telling the planters how the trees have been treated.

The PRWA pressed for the meeting when a number of planters developed symptoms after planting seedlings which had been treated four days earlier with the fungicides Captan and benlate.

One planter developed contact dermatitis and boils so severe that he was accepted as a Workers Compensation Board case. Davis said this worker probably suffered a severe allergic reaction to the fungicides, but it gave the organization a chance to express their fears.

He said they are particularly concerned about the long-term effect of exposure to the pesticides which may cause cancer or birth defects.

At the moment no one is clear how long the pesticide residues remain on the seedling trees, he explained, nor what the effects of the residues are. A study to try to determine this was begun in 1979, but the results were unsatisfactory.

Besides the new study, the planters also gained agreement from Waterland that each box of seedlings will contain detailed lists of any pesticides applied. The tree-planters are particularly concerned that any recent applications of pesticides be noted.

After meeting with the ministers we met with Charlie Johnson and Bob Jones, Silviculture, B.C.F.S., Victoria.

During the next few months, the following letters were exchanged:

Pacific Reforestation Worker's Association
Legal Committee box 4, riondel, b.c. vob 2bo

June 13, 1981

The Hon. Tom Waterland
Minister of Forests
Parliament Buildings
Vicoria, British Columbia

Dear Mr. Waterland:

We apologize for giving you short notice of our visit to Victoria on June 11, 1981 regarding a question which so involved your Ministry. We appreciate your being available to meet with us.

The initiation of pesticide notification that is complete, and a procedure in preparing the contract that alerts the planters to environmental hazards, demonstrates concern for the planters' interests and will result in greater cooperation between the Ministry of Forests and reforestation workers.

We would appreciate being put in touch with whomever you have assigned to arrange the environmental hazard notification.

After our meeting with you, we met with Charlie Johnson and Bob Jones of the Silviculture Department. We outlined the mornings' discussions and reviewed their procedural or bureaucratic difficulties. Bob Jones asked that we present our requirements for the tests and notifications in writing as explicitly as possible, a copy of which is enclosed.

We hope to see the results soon. Thank you again.

Sincerely yours,

Mirk Brinkman

Ted M. Davis

DB:rp

Pacific Reforestation Worker's Association
Legal Committee box 4, riondel, b.c. vob 2bo

June 13,1981

The Honourable Stephen Rogers
Minister of the Environment
Parliament Buildings
Victoria, British Columbia

Dear Mr. Rogers:

Thank you for meeting with us on June 11 in response to the treeplanters' problems. We found the meeting with you, the Hon. Tom Waterland and Ron Kobylynk to be productive and enjoyable.

We were pleased to find that legislation had recently been passed requiring B.C. Hydro, C.P.R., and the Forest Industry to apply for pesticide application permits. This has created the possibility for Ministry of Forest representatives to alert planting crews of the complete environmental hazards in unfamiliar planting areas. This information is vital and will be highly valued.

Ron Kobylnyk's recommendation that seedlings initially be tested for the dislodgable proportion of pesticide residue is a useful addition to the proposed residue assays. We hope that work can soon begin toward arranging these assays.

We were very satisfied with the general results of the meeting and hope to continue a productive liason with the Ministry of Environment.

Sincerely,

Dirk Brinkman

Ted M. Davis

Pacific Reforestation Worker's Association
Legal Committee box 4, riondel, b.c. vob 2bo

June 13, 1981

Ron W. Kobylnyk
Ministry of Environment
810 Blanshard Street
Victoria, British Columbia
V8W 3E1

Dear Dr. Kobylnyk:

Thank you for meeting with us last Thursday, June 11, 1981. After our meeting we talked with Charlie Johnson and Bob Jones, Silviculture BCFS, about some of the practical details that we discussed with you and the Ministers. We agreed to write them a detailed letter concerning the notification of treatment and residue assays so there would not be any confusion about what we consider necessary. A copy of that leeter is enclosed. Note that guidelines for the residue assays are described there.

If you undertake a study to determine the percentage of dislodgeable residue on seedlings, I strongly recommend that the PRWA be included in the planning and field sampling stages to increase credibility in the eyes of the planters and to take advantage of our considerable knowledge of field conditions.

During our conversation with the Ministers you mentioned that a possible explanation of the events in Terrace might have to do with a reaction to the boxes and/or mould. I am interested in any information you have in this regard and would be appreciative of any alternative explanations to the fungicide hypothesis.

I should point out that our major areas of concern are not in the area of acute toxicity, but rather, chronic effects. Among the papers we gave you at our meeting, there was a letter to Dave Armit (December 4, 1980) in which I outlined our cause of concern. I am sure you are aware of the serious and prolonged controversy within the scientific community concerning the results of carcinogenic, mutagenic, and teratogenic testing, and that application to human situations. Our position is, that since we are taking the risk, and the determination of safety involves a considerable amount of value judging, we should have the best information possible for estimating the risk (exposure being a primary factor) and should be involved in the decision making process. So in terms of our overall concerns, it is a moot point whether the fungicides have a causal connection to the events in Terrace or not. If the fungicides did cause the contact dermititis in Terrace, it must be a very exceptional event. If some other environmental factor is involved it is important for us to determine the nature of that influence. Whatever the outcome (If indeed we can make <u>any</u> causal connection) it has little to do with our strategy of residue testing and notification.

Pacific Reforestation Worker's Association
Legal Committee box 4, riondel, b.c. vob 2bo

I am preparing a handbook for treeplanters concerning these issues. Before publishing we will send you a copy and invite comments to be included in that report from your branch and other appropriate sources.

I am looking forward to hearing from you concerning these matters and again thank you for your contributions at the Thursday meeting.

Sincerely yours,

Ted M. Davis
3656 Ontario Street
Vancouver, B.C.
V5V 3G1

TD:rp

cc: The Hon. Stephen Rogers
Charlie Johnson and Bob Jones, Silvioulture, BCFS
PRWA Directors

Pacific Reforestation Worker's Association
Legal Committee box 4, riondel, b.c. vob 2bo

June 13, 1981

Charlie Johnson
Bob Jones
Silviculture
Ministry of Forests
Bastian Square
Victoria, British Columbia

Dear Charlie and Bob,

Here is our strategy for dealing with the pesticide issue. Briefly, a series of residue assays should be completed which would provide a data base for estimating the amount of residue on treated but untested seedlings. (As suggested by Ron Kobylnyk, Director, Pesticide Control Branch, a study to determine the amount of dislodgeable and undislodgeable pesticide that is on a tree would also be very useful.) Recently treated trees should have marked boxes, and all invoices to the contractor, whether the trees have been treated recently or not, should have the history of application attached. Details of this strategy are described below.

RESIDUE ASSAY

The assay should essentially follow the working plan developed out of the meeting between Jim Sweeten, Hans Elias, and Ted Davis on September 14, 1979. The working plan and guidelines that came out of that meeting were worked out in detail by Hans, but there was no follow-through. A copy of the essential parts of that plan were included in the papers we left with you on June 11, 1981.

That working plan was excellent for the purpose at the time, but some changes are now in order. The following guidelines should be followed in the next series of residue tests:

(1) At least two species of trees should be tested and one of these species should be spruce.
(2) Both container stock and bareroot should be tested.
(3) Stock should be tested for residues for all pesticides which could be applied within six months previous to planting.
(4) Stock selected for testing should be the most recently treated stock. To create a really useful data base we need a series of assays starting very soon after the pesticide application.
(5) The initial assay should be carried out on the day after packaging.
(6) Subsequent assays should be carried out at one week intervals for the first month, two week intervals for the second month, and one month intervals after that or until no residue is detected.

Pacific Reforestation Worker's Association
Legal Committee box 4, riondel, b.c. vob 2bo

(7) Subsequent assays can be restricted to seedlings which yield residues in previous assays.
(8) In the case of captan and Benlate, both dipped and otherwise treated trees should be tested.
(9) Records of the seedlot history of application, lifting and packaging, storage, and date of analysis should be accurately kept and made available to the PRWA as soon as possible.
(10) An initial series of tests for captan and Benlate should be carried out this fall to provide an initial data base. Further testing should, in part, be based on these tests.
(11) The fall tests should be conducted on recently treated seedlings.
(12) A second series of assays should be done in the spring of 1982. This should be the final and complete series necessary.
(13) Storage and handling of the seedlings should be done according to normal procedures.

As suggested by Ron Kobylnyk, assays could be done to determine the exposure levels to nursery workers. The above guidelines could be modified to include that possibility. All studies that are undertaken should be planned and executed with PRWA involvement to lend credibility in the eyes of the planters and to take advantage of our considerable knowledge of practical field conditions.

We have been in contact with Alberta Department of Reclamation and Reforestation and they have been following the pesticide controversy with interest. They have taken measures to minimize the need for fungicides and have done a single assay for residues of captan. We will encourage the Alberta Department of Reclamation and Reforestation to participate in the testing.

NOTIFICATION

We are looking for three kinds of notification. First, we recommend that the warning sheet requested by Bob Ploss in his news release of May21,1981, be conspicuosly printed on the box at the box factory and modified to read as follows:

WARNING

These trees have been treated with pesticides. Residues may be present. You are strongly advised to take the following precautions:
(1) Thoroughly wash your hands before eating or smoking.
(2) Do Not use these boxes for storage of food or clothing.
(3) Change and wash your gloves frequently.

Pacific Reforestation Worker's Association
Legal Committee box 4, riondel, b.c. vob 2bo

The second type of notification is a warning of recent pesticide application. From our conversation the most efficient method of doing this seems to be stamping the boxes. An important part of this notification is the date. Rather than "Treated With Pesticides" or "Treated With Fungicides", we would like to see, for example, "Fungicide March 1981" or "Pesticide April 1981". ("Pesticide..." is probably preferable to "Fungicide..." as pesticide is the more inclusive term.) All boxes should be stamped which have had any kind of pesticide treatment within one month of being packaged for storage or shipment. (This time scale might be revised depending on the results of the assays.) This notification is intended to alert the planters who can then go to their supervisor for more details. These notifications are simply a warning to planters that there is a potential hazard, but say nothing about the seriousness of the hazard, degree of risk, or even the type of pesticide.

The third type of notification is a detail of the pesticide application history of that seedlot. This should be attached to each invoice for each contractor. This should include the name of the pesticide, application date, form of application (if applicable) and concentration used.

The PRWA will recommend guidelines for handling trees based on the scientific literature and the residue tests. We will invite comments from the appropriate Ministries to be appended to this report.

We are more than willing to discuss modifications to the above procedures. However, it is our opinion that the above recommendations are the minimum that must be acted upon to determine the risks to planters, to notify them of the hazards, and to maintain their confidence in the situation.

Sincerely yours,

Dirk Brinkman

Ted M. Davis

DB/TD:rp
cc: The Hon. Stephen Rogers
The Hon. Tom Waterland
Ron Kobylynk
PRWA Directors

July 2, 1981

File: 125-39

Pacific Reforestation Worker's Ass'n.
Box 4
Riondel, B.C. V0B 2B0

Attention: Messrs. Brinkman and Davis

Dear Dirk and Ted:

Re: Nursery Pesticide Use Letter June 13, 1981

Our comments on your letter are as follows:

1. We agree that residue studies are necessary on planting stock. We shall set up a complete working plan for residue studies by October 1, 1981, to which we shall request your agreement before implementing same in November, 1981. Messrs. Matthews and Maxwell, our nursery culturists, will be developing the work plan.

2. The residue work plan will be referred through the Ministry of Environment to ensure they can accommodate the residue testing schedule.

3. We have noted your comments about the Alberta Department of Reclamation and Reforestation, but you will have to deal directly with them on any herbicide use.

4. On reviewing nursery pesticide procedures, we have decided to discontinue any further dip treatment procedures for Benlate and Captan. We shall shortly issue a policy directive to our nurseries to this effect.

5. On your notification request, we shall print on planting boxes the following:

Notice

These trees have been treated with pesticides.
Residues may be present. The following precautions are recommended:

(1) Wash your hands before eating or smoking.

(2) Do not use bags for storage of food or clothing.

(3) Change and wash your gloves frequently.

...2

6. For pesticide treatments made one month before packaging for storage or shipping boxes will be marked:

Fungicide, April, 1981 or
Insecticide, April, 1981

We prefer this terminology, as it is more specific as to the type of pesticide being used.

7. For the third type of notification requested, we shall continue to make the detail of the pesticide application history for each seedlot available to contractors at the Regional and District offices. As planting contracts are tendered in advance of work being carried out, there should be no problem with the contractors sharing some responsibility for obtaining this information if they want it, from either our District or Regional offices, rather than insisting on a greatly increased workload and cost to our Ministry by having nursery staff type this information on each and every shipping invoice.

We would appreciate confirmation from your Association that these procedures are satisfactory.

Yours truly,

Charles M. Johnson
Director
Silviculture Branch

CMJ/cm

Province of
British Columbia

Ministry of
Environment

Pesticide Control Branch
810 Blanshard Street
Victoria
British Columbia
V8W 3E1
Phone: 387-1161

July 20, 1981 File: 18-01-01

Mr. Ted M. Davis
Pacific Reforestation
Worker's Association
3656 Ontario Street
Vancouver, British Columbia
V5V 3G1

Dear Mr. Davis:

Thank you for your letter of June 13, 1981, arising from our informative meeting of June 11, 1981.

1. Fungicide Residues on Seedlings

I have asked Mr. Dan Cronin, Permits Coordinator, to contact appropriate officials in the Ministry of Forests as an initial step in exploring the possibilities of conducting a lab and/or field study of fungicide residues on seedlings. I suspect that a researcher from the Ministry of Forests will be assuming a leadership role in this project.

2. Alternative Hypotheses

I am still awaiting further information concerning a theory that either fungi or viruses could be involved to explain some of the events in Terrace. I shall keep you posted on any progress in this area.

3. Chronic Toxicity

I wish to inform you that there has been a federal consultative committee formed to investigate the merit and safety of all uses for captan. This committee shall be meeting for the

...2

first time in September, and I, as a member, shall be pleased to keep you informed of any new information which may be pertinent to your problem.

4. Handbook for Tree Planters

I would be most pleased to have one of my staff evaluate the pre-publication draft of your handbook from our viewpoint.

5. Residue Assay

I believe that the experimental design proposed in your letter of June 13, 1981, to the Ministry of Forests, is sound, particularly to obtain preliminary decay or disappearance curves.

Thank you in advance for your continued cooperation in these matters.

Yours truly,

Ron W. Kobylnyk
Director
Pesticide Control Branch

RWK/lc

cc: B. Jones
B. DeBoo
S. Rogers
B. Hlatky
D. Cronin
A. Murray
B. Marr
T. Waterland

Pacific Reforestation Workers' Association

Head Office, Box 49, Winlaw, B.C.

V0G 2J0

31 August 1981

Charles H. Johnson
Director
Silviculture Branch
Ministry of Forests
1450 Government Street
Victoria, B.C. V8W 3E7

Dear Mr. Johnson,

Having recently recieved a copy of your 2 July 1981 letter to Dirk Brinkman and Ted Davis, there are several items I will comment on to clarify our position.

The development of the residue assay studies is by far the most important facet in this latest series of negotiations between the Forest Service and the PRWA. I urge all due effort in seeing them through to utilizable conclusions. The results of these tests will have repercussions throughout the industry in such areas as contract procedure and health precautions. As well, the results will assist us in determining the full extent of notification necessary for planters' safety.

As the manner of notification stands at present in point 7 of your letter, I'm in agreement with Gary Ogletree; it is neither a sufficient nor appropriate method of notification.

The purpose of notification is for the benefit of the planters, not the contractors. We are the people who actually handle the trees and plant them, exposing ourselves to the little-known potential dangers of the chemicals on them. Therefore we seek a means of notification that will be most easily accessible to ourselves. Seldom in

traveling to a contract site do the planters pass through the town where the District or Regional office is located. Planters rarely know the exact name and number of the contract or contracts they will be working on to be able to ask for data about the seedlots pertainent to each contract. Finally, it is frequently the case that the actual seedlot that is to be sent to the contract is not known or designated until it is actually sent. Not infrequently, one seedlot is substituted for another for any of a variety of reasons (best known by Fate).

The method you are suggesting will make it extremely difficult for a planter to track down the relevant information prior to the beginning of a contract. Keep in mind also that we function on a rapid-paced schedule, with often only a day or two between contracts, most of which is taken up by travel.

You've raised a concern that the contractor or supervisor on the job may not distribute the information if it is part of the invoice. The simple solution to that problem is to have the information printed on a separate sheet accompanying the invoice. Granted, it is up to the planter to request that information from the contract supervisor, and the PRWA will do its part in informing planters of the form in which the information will be made available by the Forest Service.

What you consider a burdensome increase in the workload of the nursery staff, we see as a vital and duly appropriate measure necessitated by the aforementioned conditions. I am confident that a standardized method can be worked out to our mutual satisfactions. The relevant information that will constitute the notice will be determined when we get the results of the residue assay tests.

One other change that will result in a more exact recognition of the situation. In the second statement of the notice to be printed on the treeboxes the word "boxes" should be substituted for the word "bags". The reason for this is that nobody uses the bags for anything, whereas boxes are constantly utilized for storage and transportation of various materials including personal gear. I suggest also if not currently in the plans for the scheduled residue testing, that both bags and boxes be added.

The decision of the Forest Service to eliminate dip procedures for Captan and Benolate is welcome news to treeplanters. I was working as first aid attendant and treeplanter on the contract outside of Terrace where we recieved the pine seedlings that had been overdosed with fungicides. While unloading the boxes with the crew, we were drenched by a liquid that was sloshing out of the bottom of the boxes. It was an unpleasant experience, and rather frightening, as none of us knew what that liquid was. At that point I decided that personally I would not take the risk in planting the pine. The rest of the crew followed suit after planting pine for a few days and having noticeable reactions to it.

Watching several planters develop rashes and hives worse than they'd ever experienced before during that contract was frustrating. Over the course of the nine years I've worked as both treeplanter and first aid attendant I've seen a number of people get sick, or break out in rashes without any identifiable cause other than some factor consistently present during planting. From the point of view of a first aid attendant, it is essential to have full notification of type and dates of treatment of the trees with chemicals, as the individuals we are responsible for vary in their sensitivities to the different chemicals.

It is my understanding that Messers. Brinkman and Davis will be continuing the official correspondence with you on the development of the residue assay studies. I hope that the results of these important tests will do much to dispell the current uncertainties about the effects of the levels of chemicals we are exposed to.

Looking forward to further exchanges of information,

Yours truly,

Viola Fellegrin

President, Pacific Reforestation Workers' Association

cc; Board of Directors, PRWA
Lorne Nichols cn, MLA

Pacific Reforestation Worker's Association
Legal Committee box 4, riondel, b.c. vob 2bo

Alberta Energy & Natural Resources
Alberta Forest Service
Petroleum Plaza - South Tower
9915 - 108 Street
Edmonton, Alberta
T5K 2C9

Con Dermott:

Enclosed is a presentation to the Ministry of Environment and the Ministry of Forests in British Columbia.

Many of the planters in Alberta are PRWA members. We request a combined Alberta and B.C. study of residue levels of pesticides used on the seedlings.

We request notification of all the pesticides used in the past year on the seedlings be delivered to the planters with the shipping invoice, and notification of any recent pesticide treatment be posted on the boxes, along with a general warning.

We request a check be made to ensure prospective campsites or planting areas do not have anything hazardous in the water or in the area.

We are very hopeful that the Alberta office will contact Charlie Johnson, Silviculture BC, and co-operate with the BC Ministry of Forests seedling assays. If Alberta added the pesticides that they use (eg: ferbate) to the pesticides used in BC, that set of assays would then serve for both provinces. The specifications for the assays and the notification details are outlined in our letter to Silviculture BC, June 13, 1981.

The foundation impetus for asking for this notification will be readily understood by the Reforestation Department. Research indicates Captan and Benelate for example, which are often put on the seedling shortly before shipping, are possibly mutagenic in humans. Gene damage is generally irreversible and detrimental. The human gene pool is our most valuable treasure and hope for the future.

Continued Page 2.....

Pacific Reforestation Worker's Association
Legal Committee box 4, riondel, b.c. vob 2bo

The population group planting trees are characterized by persons very conscious of these values.

We hope you will be responsive to our desire to be informed enough to evaluate the risks we take in planting.

We are looking forward to hearing from you.

Sincerely

Dirk Brinkman
Ted Davis
DB/vb

November 9, 1981

Files: 955
125-54
125-55
720-12
955-20-1

Mr. Dirk Brinkman
Legal Committee
Pacific Reforestation
Workers' Association
Box 4
Riondel, B.C. V0B 2B0

Dear Dirk:

Re: Nursery Pesticide Use

Firstly, I must apologize for the lengthy delay in responding to your letter of August 31, however the issue has now been fully discussed with my staff and we are prepared to agree to the following procedures; this of course supercedes our letter of July 2, 1981.

I. Notification of Pesticide Use (Boxes)

(a) Wording on boxes -

Notice

(i) These trees have been treated with pesticides. Residues may be present. The following precautions are recommended:

(1) Wash your hands before eating or smoking.

(2) Do not use bags or boxes for storage of food or clothing.

(3) Change and wash your gloves frequently.

(ii) For pesticide treatments made one month before packaging for storage or shipping, boxes will be marked:
(e.g.) Fungicide, April 1981 or
Insecticide, April 1981

II. Notification - Pesticide History

A detailed history of pesticide application to the seedlings will be sent with the invoice for each seedlot shipment, indicating for the past year the pesticides used, application dates, method and rate of application and concentration used, in addition to our current practise of supplying such information through the Regional/District offices.

III. Non-Dip Policy

Your Association has been advised of the Branch's instructions to nurseries that dipping in fungicide solution(s) will no longer be used as a treatment method.

IV. Box Testing for Residue

In view of the wide range of conditions to which boxes are subjected, residue testing, as requested to determine extent of residues on boxes, would be meaningless for province-wide evaluation regarding use of boxes for personal storage, etc. The notice in (1) above is explicit - "Don't use bags or boxes for storage of food or clothing."

V. Pesticide Use on Site at or near Silviculture Projects

It is not possible for this Branch's staff to keep a daily update on provincial applications nor be in a position to advise contractors or workers, for as you perceive, isolation can mean delays in contacting. It must be incumbent on contractors or planters to contact the District Manager for up-to date line information on pesticide application in a district, and in particular, contract work areas.

VI. Residue Testing

Our Branch has prepared a residue testing work plan. Mr. Armit will provide you with details.

We trust the foregoing adequately covers the concerns of your Association. In closing, however, we do experience some confusion at times in determining whom we are dealing with in these matters. We understand you represented the PRWA in resolving the use of pesticides on seedlings, but we also received a letter from Gary Ogletree dated August 26, and another from the president of PRWA dated August 31; unless advised otherwise, we will continue to deal with you.

A copy of this letter is also being forwarded to the Western Silviculture Contractors' Association.

Yours truly,

Charles M. Johnson
Director

c.c. Western Silvicultural Contractors

Dea[illegible]:

[illegible]losed is a copy of the plan for testing degradation rates of fungicides used in Forest Nurseries, as referred to in the letter to you from Bob Jones.

Sincerely,

D. Armit
Manager, Nurseries
Silviculture Branch

Degradation Testing of Fungicides Used in Forest Nurseries

The lack of information about degradation rates for fungicides used in Forest Service nurseries results in this proposal for testing which will indicate the degradation rate of those fungicides when applied on forest seedlings under production conditions.

The project is to be carried out at Surrey Nursery. The three fungicides to be tested are captan, benomyl and chlorothalonil, which are applied on seedlings when specific disease control is necessary.

Both bare root and container grown forest tree seedlings will be tested with the three fungicides, regardless of whether they would normally be used. The study will test two species in bare root and two species in containers, and will incorporate an early and late lift for all seedlots tested. Sprays will be applied in accord with standard methods for the two culture systems.

As the test is intended to measure the rate at which these funicides break down while seedlings are insitu, and while they are storage, the study will include

(a) sampling of stock prior to treatment, (b) second day following treatmnet, (c) seven days after, (d) fourteen after (e) twenty-one after, (f) twenty-eight days after, (g) every month on the same date of the month until the stock is shipped and (h) the day stock is shipped.

There will be two parts to the study; (a) one will involve the stock being lifted forty-eight hours after treatment and (b) the second will involve the stock being lifted two weeks later; these represent realistic lift dates for stock when processed on an operational basis. Lifted stock will be wrapped in bundles of twenty-five per sample. Seedlings

will be held in standard cold storage conditions, in standard bags and boxes.

The samples for analysis will be transported to the B.C. Ministry of Environment Laboratory, at B.C. Research Council, for completion of the test work. The number of samples and the size of each sample is constrained by the capacity of the laboratory to handle the test program, which is eight samples per test date. There will be one sample of eight seedlings assayed for each seedlot and each treatment being tested, a total of 8 samples at each test period. To provide a uniform sample, the top five centimeters of each seedling will be used for the analysis.

The assay test will be a analysis of the total pesticide on the seedlings. At the moment there is no operational procedure developed to allow conifer seedlings to be analyzed to distinguish between undislodgeable and dislodgeable.

Storage will be carried on till the end of November at which time the lift for planting program will be finalized. The lift for spring planting is dependent on weather but it is estimated the project will start in November and stock will be stored until the particular seedlots are actually shipped to the planting site.

The Factors To Be Examined Are

1. (a) Captan
   (b) Benomyl
   (c) Chlorothalonil
2. The species and type of stock to be treated:
   (a) 1. Bare root 2+0 S Seedlot 1863
   2. " " 2+0 Fc Seedlot 1330

(b) 1. Container 1+0 PSB S Seedlot 4186
2. Container 1+0 PSB Hw Seedlot 3907

3. Spring and fall lift of bare root and containers.

4. Stock will be sampled from the plots are blocks,

(a) Immediately prior to fungicide application.
(b) Two days after.
(c) One week after.
(d) Two weeks after.
(e) Three weeks after.
(f) Four weeks after.
(g) Second month and every month after until stock is shipped to outplanting.
(h) When the stock is shipped to the planting site.

((g)-(h) will consist of two samples for each seedlot treatment, one each for early lift and later lift stock)

5. The stock will be lifted, sorted, bundled and stored in the Surrey Nursery storage; subsequent samples will be taken from this stored stock.

Spray Applications

Sprays will be applied by the standard production method in both bare root and container on the selected seedlots during the week of October 5th to 9th.

(a) Captan
Captan will be applied as Orthocide 50 w at 1.12 k a.i./ha.

(b) Benomyl
Benomyl will be applied as Benlate 50 w at 565 g a.i./ha.

(c) Chlorothalonil
Chlorothalonil will be applied as Bravo 500 at 800 g a.i./ha.

Spray nozzles will be between 42 - 48 cm above seedling canopy and at 690 k P.a. pressure.

Plots and Blocks

To ensure representative samples are obtained, each seedlot will be divided into plots for bare root and blocks for container culture. There will be sixteen plots or blocks for each seedlot and treatment. Plots in bare root will be one metre length of bed with a 30 cm border at each end. The plots and blocks will be designated by:

(a) Species and seedlot number.
(b) Pesticide treatment.
(c) Plot number.
(d) Date of treatment.
(e) Early or late lift.

Records

A detail record of all pesticides that have been applied to the selected stock during the last twelve months will be maintained. As the trial proceeds the details of each stage will be recorded as well as the weather prior to lifting. Irrigation required for the container stock will also be noted.

Bare root Stock Standards

		Height	Caliper
(a)	Fc	12.5	3.0
(b)	Si	10.0	2.6

Containers Stock Standards

(a) S 7.5 cm with a plug
(b) Hw 7.5 cm with a plug.

Lifted stock will be tied in bundles in the normal manner; each bundle will be tagged, indicating:

(a) Species and seedlot number.
(b) Pesticide treatment and dates.
(c) Plot number.
(d) Period of lift.

Stock for each plot will be placed in individual storage bags which will have the above information written on the outside. Depending on the size of the stock, the same seedlot treatment stock bags will be placed in same standard carton or cartons; information detailing the stock treatment will be written on the box. Cartons will then be placed in storage at Surrey Nursery and held separate from non-trial stock to ensure that there is no error.

Sampling

Each test sample of stock will consist of eight seedlings, from each seedlot treatment plot or block. Each sample will be labelled with:

(a) Treatment.

(b) Species and seedlot.

(c) Lift period.

(d) Sampling date.

The top 5 cm of each seedling will be sampled and placed in plastic bags labelled with the above information. A General Chemistry Requisition form will be completed for each sample, with the relevant information for each sample being collected. For sampling source the following coding abreviations will be used.

(a) Fungicide

captan	CAP
benomyl	BEN
Chlorothalonil	CHL

(b) Species

Fir	F
Spruce	S
Western Hemlock	Hw

(c) Seedlot number

(d) Culture

Bare root	BR
Container	C

(e) Treatment date

Year, month and day.

(f) Lift date

Early	E
Late	L

e.g. CAP F 2810 C 81 10 3 E

The fungicide to be tested will be indicated in the appropriate section.

Requirements at Surrey Nursery

(a) A technican to supervise and co-ordinate the project.

(b) 60 m of 2+0 F and S
48 blocks of 1+0 SB S and Hw

(c) Supplies - Flags or pegs to mark the plots or blocks, bags - plastic and paper, cartons and labels.

(d) The use of calibrated sprayer, tractor and operator, to apply fungicide as scheduled.

(e) Required amounts of captan, Benlate and Bravo 500.

(f) The crew to lift, sort, label, package and store stock on 2nd day after spraying and two weeks after spraying.

(g) Storage space.

Costs

The analysis will cost $104. per sample to be tested. At the end of the first month there will have been 112 samples tested and 24 per month thereafter. Total laboratory test costs are estimated at $25,000, total project costs are estimated at $35,000.

GLOSSARY

Acute Toxicity - Poisoning from a single dose of a chemical.

Carcinogen - A substance capable of producing cancer.

Carcinogenic - Cancer producing.

Carcinogenicity - The power, ability or tendency to produce cancer.

Chromosomal aberrations - An irregularity of the chromosomes which may alter the course of development of the fetus.

Chromosome - A structure in the nucleus of cells containing a linear thread of DNA, containing the genes and the genetic code.

Contact Dermatitis - An acute allergic inflammation of the skin caused by contact of a substance.

Dermal Toxicity - Pertaininq to the poisoning caused by a substance on the skin.

Duodenum - The first part of the small intestine.

EPA - Environmental Protection Agency (U.S. government).

Epidemiology - The study of the relationships of the various factors in the distribution and frequency of disease.

Etiology - The study of the cause or origin of a disease.

F1 generation - The first generation after the parent.

Fetal - Pertaining to the developing young while in the uterus.

Fungicide - A pesticide used to treat or prevent fungus diseases.

Gene - The biological unit of heredity, located at a specific position on a particular chromosome.

Genetics - The study of heredity.

Germ cells - The reproductive cells. The eggs and sperm.

Half-life - The time in which one half of the substance is destroyed. used to describe the time in which the radioactivity of an isotope is reduced by one-half.

Herbicide - A pesticide used to kill weeds.

Heritable - Capable of being passed on to the offspring.

Insecticide - A pesticide used for killing insects.

Intraperitoneally - Within the peritoneal cavity. The peritoneal cavity is the space between the abdominal wall and the internal organs.

LD_{50} - Used to indicate acute toxicity, it is the amount of a substance expressed as mg-kg of body weight of an animal necessary to kill 50 per cent of such animals.

Metabolize - To transform a substance by physical and chemical processes in the body.

Micro-organism - A microscopic organism. Bacteria, yeast, molds, etc.

Mortality - The death rate.

Mutagen - A substance capable of producing mutations.

Mutagenic - Mutation producing.

Mutagenicity - The power, ability or tendency to produce mutations.

Mutation - A genetic change which produce offspring that have different characteristics from their parents.

NCI - National Cancer Institute (U.S.).

Oral Toxicity - Pertaining to the poisoning caused by a substance taken by mouth.

Pesticide - A substance used for killing or controlling plants and animals that are considered pests. Fungicides, herbicides, insecticides, etc. are pesticides.

ppm - Parts per million.

Residue - The amount of a chemical that is left at the time of analysis.

Teratogen - A substance capable of producing deformities in unborn animals.

Teratogenic - Capable of producing deformities in unborn animals.

Teratogenicity - The power, ability or tendency to produce birth defects.

Thalidomide - An infamous teratogen used as a sedative prescribed to mothers because of its apparent absence of side effects. It caused over 7,000 babies to be born with various degrees of limb malformation.

Threshold response - Refers to the theory that there must be a certain amount of a substance present before that substance has any effect. A no-effect level.

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