

DDT, HUMAN HEALTH AND THE ENVIRONMENT

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For the past two years there has been an intensive campaign to ban the insecticide DDT (dichlorodiphenyltrichloroethane) from use in, manufacture in, and export from the United States. This article evaluates the evidence for some of the charges in this campaign. These include charges that DDT is a "biocide" (a name implying that it is poisonous to all forms of life), permeates the environment, is virtually indestructible, kills many desirable forms of wildlife, and is dangerous to human beings. The last of these claims is particularly astonishing in view of the fact that DDT has saved millions of human lives from malaria, typhus fever, plague and other deadly diseases without harming a single person, except in a few cases of accidental or suicidal overdosage.

The propaganda against DDT has been so extensive and successful that DDT is now widely regarded by the public as a dangerous poison. There have been a series of newspaper cartoons, skillfully staged television shows, and a display of bumper stickers with skull and cross-bones, all aimed at exposing the evils of DDT. The National Audubon Society printed 700,000 copies of a leaflet urging that the export of DDT be stopped. This leaflet was distributed at about the same time a resolution requesting the continuation of the use of DDT was passed at a meeting of the WHO Regional Committee for Southeast Asia held in 1969 in Nepal, attended by representatives of eleven tropical countries totaling over 700 million in population—about 1000 people for each Audubon leaflet.

Many people have become self-constituted authorities on DDT as a result of exposure to mass media. For example, the chief judge of the Circuit Court of Appeals in Washington, D.C., an-

nounced in January, 1971, that DDT kills honeybees and is dangerous to people. Yet, while other insecticides, such as parathion and Sevin, do kill bees, DDT's effect on these insects is only minimal. As regards DDT's effects on people, the principal consequence has been to increase population, not endanger lives.

What are the facts of this strange paradox? What motivates those who crusade against the most useful chemical in history? Is the attack on DDT partly directed against its role in accelerating the population explosion?

The organizations that are most active in the movement to ban DDT include some of the large conservation groups. Despite the size of some of these groups, they do not speak for all segments of the population. Some environmental groups, in fact, have recently been challenged by organizations which represent racial minorities. As the following statement suggests, the needs of the urban poor are not likely to be assuaged by the Thoreau-like pre-occupations of many of the conservation associations:

In general, black people probably do not know much about the science of ecology or the study of human conservation as now offered in most universities. Probably they know very little about bay fill, polluted streams, soil erosion or redwood trees. And probably they couldn't care less. But precisely because they are black and poor, they do know a great deal about 125th Street in New York, about South Street in Philadelphia and the Fillmore in San Francisco. About those environmental disasters they are very knowledgeable and they can also tell you quite a bit about human conservation as practiced on any of those nearby street corners, in the filthy two-, three-room walk-ups, in the fetid housing projects or in their urine-, vomit-, whiskey-, blood-stained hallways which exist all over, everywhere, in the uninhabitable cities of America's enraged and inconsolable slum communities.¹

The National Audubon Society, which appears to have a predominantly white and middle-class membership, is one of the most active anti-DDT organizations. For a member to condone the use of pesticides would be tantamount to the deepest heresy in a religious sect. For an official of the Society to approve such use would be fiscal lunacy, in view of the tremendous amount of free publicity that the Society has received as a result of *Silent Spring*² and other publications which have established a new mythology—the extermination of wild birds by agricultural pesticides. The Society shows underlying resentment of human beings

and all their works, including cities, farms, highways, and especially private industry. Membership in the Society is a form of expiation of the sin of being one of the human race, the species that consumes "the environment". The Society stated recently that one of its two main purposes is "the education of man regarding his relationship with and his place within the natural environment as an ecological system."³ This pious pronouncement is actually intended to exclude man as an inhabitant of the Earth, except in small numbers and in a primitive, mythical, aboriginal state. The Audubon Society has no program for the relief of suffering among millions of human beings in the tropics.

Two other organizations that have attacked DDT are the National Geographic Society⁴ and the Sierra Club. The National Geographic Magazine advertises plush overseas tours for the wealthy people. The magazine has beautiful photographs of wild animals, birds, and under-dressed natives in picturesque attitudes. These pictures do not show the ravages of tropical diseases that can be controlled by DDT. The Sierra Club, which is seeking legal action to obtain a ban on DDT,⁵ also features expensive outings to remote lands, again largely for the healthy and economically secure. The motivation of conservation organizations is primarily to protect the landscape and its wildlife. This may be in conflict with combatting hunger and disease in human beings.

Why is it necessary to defend DDT? Why can't other insecticides be used instead? The answer is that DDT is specifically needed to protect millions of people in tropical countries from death by malaria. This has repeatedly been made plain by the World Health Organization in statements such as the following:

The withdrawal of DDT would mean the interruption of most malaria programs throughout the world. . . . DDT used as a residual spray of the interior surface of houses. . . led to the idea of nation-wide malaria control campaigns including the whole of the rural areas of a country. The success of these campaigns resulted in the concept of malaria eradication which was adopted. . . for the world by the Eighth World Health Assembly in May, 1955.

Since then DDT has been the main weapon in the world-wide malaria eradication program. Research has continued for the development of other methods of attack against malaria and for the development of alternative insecticides. To date, there is no insecticide that could effectively replace DDT which would permit the continu-

ation of the eradication program or maintain the conquests made so far.

The withdrawal of DDT will therefore represent a regression to a malaria situation similar to that in 1945. The reestablishment of malaria endemicity would be probably attained following a period of large-scale outbreaks and epidemics which would be accompanied by high morbidity and mortality due to loss of immunity by population previously protected by eradication programs.

Toxicological observation of spraymen working for a number of years in malaria eradication, and even in formulation plants, has not revealed toxic manifestations in them or in people residing in houses that have been repeatedly sprayed at six month intervals.

We therefore believe that a great harm will result from the unqualified withdrawal of DDT. We feel that selective use of DDT is justified and warranted.⁶

This is what the argument is all about. If the manufacture and export of DDT are banned in the United States, the world-wide antimalarial program will collapse. Most of the DDT manufactured in the United States is for this program. Furthermore, a ban in the United States would lead to prejudice against the use of DDT elsewhere.

Those who are fighting the ban are struggling to save lives. The objective is not to "protect the chemical industry," since the substitute insecticides are more expensive and more profitable than DDT. These substitutes can be used, with varying degrees of lower efficiency, against the agricultural pests that are controlled by DDT. But there is no effective substitute for DDT in the world-wide campaign against malaria. The other compounds either decompose rapidly, produce resistance too fast, or they are too poisonous to people.

TOXICOLOGY

There is a saying among toxicologists that this subject can be easily learned in two lessons—each five years long.

One of the oldest principles in toxicology was stated by Paracelsus almost 500 years ago: "Everything is poisonous, yet nothing is poisonous." This is quite familiar to biochemists, who recognize that several chemical elements commonly regarded as poisonous are essential in small amounts to life. Examples of these are copper, chromium, manganese and selenium. The last named of these is also carcinogenic (*i.e.*, tends to produce cancer). Traces of practically all the elements can be detected by spectroscopic

tests in most biological materials, and all living creatures contain radioactive carbon and radioactive potassium. The crucial matter is the quantity of such substances that we consume in proportion to the amount that is toxic. It is comparatively easy to poison animals with table salt in high dosage.

The development of modern analytical methods of sensitivity has enabled many substances to be detected in concentrations of less than one part per billion (ppb). If the substance thus detected is commonly regarded as a poison, then the detection of such traces in unexpected places may cause public alarm, especially when the news is presented in a sensational manner. The claimed detection of DDT in Antarctic penguins at levels in the range of one part per billion has been used to imply that the whole world has been poisoned. The finding actually shows that molecules can be dispersed widely, and that the analytical device known as vapor phase chromatography, or electron capture, is extraordinarily sensitive. Such procedures can easily give erroneous results if substances are present which simulate the compound whose measurement is sought. The analysis must be carried out by an expert, or better, by two or three experts working independently, if reliable results are to be obtained.

Another basic concept of toxicology was stated by Lucretius two thousand years ago: "Quod cibus est aliis; aliis est acre venenum"—"One man's meat is another man's poison." The scientific application of this proverb is known as comparative toxicity; it is a keystone of therapeutic medicine. A significant goal of researchers in the area is easily understood in terms of the metaphor of the "magic bullet."⁷ Drug therapists have continually searched for chemical "magic bullets" which hit disease carriers while missing patients. DDT is such a "magic bullet" because it kills mosquitoes and other insects that carry disease, but does not injure human beings. The idea of the "magic bullet" is more precisely expressed by the therapeutic ratio, which is the fraction of the minimum lethal dose of a drug that is therapeutically effective. The smaller the fraction, the safer the compound is for general use.

Research in comparative toxicity has led to the discovery and development of chemicals, such as antibiotics and pesticides, which enable the human species to compete successfully with other forms of life, especially disease organisms. Although it is

possible that advances in comparative toxicity will contribute to a serious overpopulation in man, it is *certain* that inattention to this field of knowledge will permit a critical fall in global food supplies and global public health.

Malaria

From time immemorial human beings have been hosts to a genus of protozoa known as *Plasmodia*. There are many species in this genus; three of the principal ones that attack human beings are *Plasmodium vivax*, *P. malariae*, and *P. falciparum*. They produce respectively three different types of malaria. Of these, the most malignant is that produced by the *falciparum* species, which often attacks the brain. These three *Plasmodia* spend part of their life cycle in mosquitoes, but the cycle is not complete without going through several stages in man, where they reach maturity in the red blood cells and reproduce, in enormous numbers, into a form called *merozoites*. These change into the sexual stage, which enters the body of a blood-sucking mosquito. Other stages of the life cycle then take place, and the parasite reaches the salivary gland of the mosquito, from which it is inoculated into the next victim bitten by this insect. The cycle then continues from man to mosquito, and mosquito to man. The principal method for breaking this pernicious chain is to kill mosquitoes with DDT by spraying the interior walls of human dwellings.

Public health authorities in the tropics apparently use a figure of 1 per cent per year to estimate the mortality from malaria; thus 75 million cases in India in one year were calculated to be responsible for 750,000 deaths.⁸ The survivors in many cases are severely debilitated and unable to work.

The mosquitoes rest on the walls by day and attack sleeping people at night. If DDT is sprayed on the walls, it kills the mosquitoes. An insecticide for this purpose must be *persistent* because it is not possible for spray teams to go into the same house frequently. Malathion, for example, is non-persistent and soon decomposes; it also has a pronounced and offensive odor. The procedure must be *safe* to those dwelling in the house, and to the sprayers, who are intensively exposed to the insecticide. The development of resistance by mosquitoes to the insecticide must be slow enough to enable the life cycle of the mosquito to

be broken. DDT as opposed to lindane, for example, fulfills this requirement. Note that the WHO program favors spraying walls to kill adult mosquitoes rather than spraying ponds and swamps to kill larvae.

I have not mentioned cost, because this should not be a prime consideration when many human lives are at stake. However, Russell estimates that the appropriations of the United States for overseas malaria control and eradication amounted to a half-billion dollars in the past 25 years.⁹ As a result of international cooperation, WHO has maintained an effective world-wide malaria eradication program:

Today more than 960 million people who a few years ago were subject to malaria endemicity are now free of malaria; another 288 million live in areas where the disease is being vigorously attacked and transmission is coming to an end. Because much of Africa remains highly malarious and because about 288 million people live in malarious areas not yet subject to eradication measures, it is logical that the United States should maintain an active interest in this disease.¹⁰

These estimates by Dr. Russell indicate that the United States contribution to saving lives from malaria has paid off quite well in terms of human welfare. As I said recently however, "some Americans, by demanding a ban on DDT, are reversing the traditional role of their country in relieving the sufferings of others."¹¹

DDT in Agriculture

Insects compete with mankind for food. They devour all parts of plants—leaves, stems, fruits and seeds. A plant attacked by insects will often die without producing seeds or fruit. The vulnerability of plants to attack by insects is greatly increased by agriculture, which inevitably leads to what is called "monoculture"—the growing of a single crop in a large area of land, such as a field. Obviously a group of crop plants, such as potatoes, corn, tomatoes and alfalfa, cannot be grown as a mixture. Food must be grown on farms using monoculture, unless and until some other method of providing nourishment is developed.

Prior to World War II methods of controlling agricultural pest insects included the use of "stomach poisons", the most effective of which was lead arsenate; the use of contact poisons, *e.g.*, nicotine sulfate; and the use of hand labor, *e.g.*, for burning corn-stalks and plowing them underground in the fall to control the European corn borer, or for picking bugs off potato plants. The

discovery of DDT revolutionized the control of agricultural insects. It also replaced lead arsenate, which is virtually indestructible, and highly toxic.

DDT is intensely poisonous for many insects, and is less toxic for plants and warm-blooded animals. Like all chemicals, however, DDT has a level of toxicity for any species of animal or plant. The extensive use of DDT, without adequate controls, has resulted in the killing of non-target species. In some instances this was an accepted risk. For example, when forests were sprayed to control the spruce budworm, many fish were killed in the streams. These were replaced by planting other fish.

DDT is poisonous to crustacea, such as crabs. Careless use of DDT and other pesticides that results in their drainage into rivers, swamps, lakes, estuaries and coastal waters must not be allowed. This precaution would remove a major source of friction between agriculture and those who are interested in wildlife. Actually, a rapid reduction is currently taking place in the agricultural use of DDT, and this will undoubtedly lead to a lessening of its movement through "food chains," which concentrate fat-soluble substances. However, if DDT suddenly disappeared this would not end the problem. Most of the fish that are killed by pollutants in inland waters are the victims of petroleum wastes, industrial wastes and sewage, just as most eagles found dead are killed by gunfire. The role of other pollutants is just beginning to emerge.

Solubility in fats is not an exclusive property of DDT or of the other pesticides that are similar to it. Recently it has been found that mercury can be converted into a fat-soluble form, methyl mercury, by bacteria. This form can enter food chains and, by stepwise concentration, produce effects that are toxic to animals and birds that eat fish. Mercury has been present in sea water ever since the oceans were formed, and occurs in the earth's crust. It does not become a problem until its concentration is greater than the toxic level. This level can result from industrial contamination, but the possibility exists that such levels also occur under natural conditions.

Space does not permit an adequate discussion of the vast topic of the effect of chemicals on wildlife. Many of the conclusions are based on inferences, rather than on controlled experiments. There arise therefore ambiguities, which lead in turn to disputes. The final answer may differ from the first guess. It is thus best to

react cautiously to preliminary judgments regarding the death or disappearance of wildlife. For example, a few winters ago robins were unusually scarce in the coastal cities of California and DDT was blamed widely. The robins were actually back in the mountains, feeding on an unusually fine crop of berries, and the following winter they were in town as usual.

HOW TO ALARM THE PUBLIC—A STUDY IN “ECO-TACTICS”

A syndicated newspaper article has described the history and activities of the Environmental Defense Fund (EDF), asserting that the EDF “has swiftly become the public defender of the environmental movement.” The article stated that

The turning point came when Cameron decided to spend about \$5,000 of the organization’s total remaining assets of \$23,000 on an advertisement in the New York Times on Sunday, March 29, headlined ‘Is Mother’s Milk Fit for Human Consumption?’ It referred to the amounts of DDT in the human body.

The ad appealed for members, starting at \$10 for a basic membership. It produced \$7,000, a profit, and the EDF turned to a direct mail campaign and now has 10,000 members, a stable financial base and a chance at major foundation support.¹²

This is a most interesting revelation. The EDF appealed to the public on the basis of the DDT content of human milk. As a means of arousing alarm concerning DDT, the EDF and the National Audubon Society have both stated that DDT causes cancer. The implication that DDT in breast milk may cause cancer in babies is superlatively sensational copy. The following lurid passage is from an article by Ed Chaney, Information Director, National Wildlife Federation:

A five-day-old human being lies asleep in the other room. His name is Eric. His tiny, wiggly, red body contains DDT passed on to him from his mother’s placenta. And every time he sucks the swollen breasts, he gets more DDT than is allowed in cow’s milk at the supermarket. Be objective? Forget it. Objective is for fence posts. How can you be objective in the face of a global insanity that is DDT? In the face of abdicated responsibility by the men the public pays to protect its interests. Are the anarchists right? Are ashes the only fertile seed bed for growing new responsiveness to the public interest? Picture a swarm of angry citizens bathed in the light of flames engulfing the Agriculture Department.¹³

It is distressing that an official of a large organization should discard objectivity and propose anarchy in its stead.

Let us examine the factual and scientific background for the propaganda campaign regarding DDT in human milk. The background starts with the improvements in technology that made it possible to detect fantastically small quantities of DDT. Note that such extremely delicate tests can easily give "false positive" readings because of accidental contamination of the equipment or lack of expertise by the tester. We must next note that cows' milk has occupied an unusual position among foods with respect to regulations. "Zero tolerance" has been the policy with respect to additives to milk, except for vitamin D. The improvements in testing procedures made it necessary to re-examine the definition of zero, since every chemist knows that zero content, in molecular terms, does not exist. For example, all fish and all human beings have contained mercury for millions of years (*i.e.*, before the chemical industry existed). To get back to milk, more than ten years ago it was evident that the entire canned milk stocks of the United States contained DDT. It was therefore necessary to face facts and choose one of two alternatives: ban cows' milk from interstate commerce, or set a tolerance.

The second alternative was chosen, and the tolerance set at 0.05 parts per million (ppm). This was a far lower level than the 7 ppm permitted for most agricultural products. A rule of thumb for tolerance levels is 1% of the toxic dose which is lethal to 50% of a group of experimental animals. Obviously, if 7 ppm had been estimated to be non-injurious, a tolerance of 0.05 ppm provided an unusually large margin of safety. The low tolerance was possible primarily because cows metabolize and break down DDT very effectively, and also because great attention was paid to avoiding the use of DDT on crops, such as alfalfa, which are consumed by dairy cattle. In contrast, human beings are less efficient than cows in metabolizing DDT, and they do not eat hay. There is a straight-line relationship between DDT intake and DDT level in body fat.¹⁴ If the dosage decreases, the content of DDT in the fat becomes less. This is the result of an equilibrium level between intake, breakdown and excretion in the urine. A level of 10 ppm in the body fat is apparently harmless; far higher levels occur in spray operators and workers in DDT manufacturing and formulating factories who remain in good

health despite prolonged exposure for periods up to 20 years.¹⁵ Dr. J. M. Barnes (Director, Toxicology Research Unit, British Medical Research Council) summed up the matter as follows:

Unfortunately, DDT is relatively slowly metabolised and excreted by the mammal and by virtue of its solubility characteristics tends to get laid down in tissue fat. Here it would have remained as an innocent and unrecognised passenger but for the fact that the chemists invented a sensitive chemical method, since further enhanced by the gas-liquid-chromatographic technique capable of detecting the chlorine and indicating its source even in minute quantities. Thus it has become possible to establish an anxiety neurosis in respect to a few parts per million of a compound in a tissue such as fat where a few parts per thousand in the whole animal are of no toxicological significance.¹⁶

A good example of one of the many studies on the prolonged effects of DDT on human subjects is the recent publication by Hayes and co-workers who reported that:

Twenty-four volunteers ingested technical or p,p'-DDT at rates up to 35 mg. per man per day for 21.5 months. They were then observed for an additional 25.5 months, and 16 were followed up for five years. Storage of DDT and DDE and excretion of DDA were proportional to dosage. The fat of those receiving technical insecticide at the highest rate contained 105 to 619 ppm of DDT when feeding stopped. The average dosage of p,p'-DDT administered in this study was 555 times the average intake of all DDT-related compounds by 19-year-old men in the general population and 1,250 times their intake of p,p'-DDT. Since no definite clinical or laboratory evidence of injury by DDT was found in this study, these factors indicate a high degree of safety of DDT for the general population.¹⁷

DDE and DDA are two breakdown products of DDT. DDE is not insecticidal, but it has an effect similar to that of DDT in inducing the production of microsomal enzymes. DDA is an acetic acid derivative. It is inert, is soluble in water and is excreted in the urine.

The DDT in human beings enters the fat of breast milk. This was noted and published in 1950 by Laug and co-workers who found an average concentration of DDT of 0.13 ppm in 32 samples taken in Washington, D.C., with a range from undetectable ("zero") to 0.77 ppm.¹⁸ Several similar reports have since appeared, and the results of an extensive survey were described by Quinby and co-workers.¹⁹

It may be concluded from the preceding discussion that the DDT level in human milk is about twice as high as the tolerance allowed for cows' milk by the FDA. That bald conclusion, however, requires explanation: it must be explained in terms of its underlying premises and toxicological implications. The use of the unqualified conclusion to create public alarm is a scientifically irresponsible act.

The DDT content of human milk has also been scrutinized by the World Health Organization and the Food and Agricultural Organization of the U.N. They set a permissible rate of intake of 0.01 mg. of DDT per kilo of body weight for breast-fed infants. The DDT intake of breast-fed babies in the United States may be higher than this; estimates range from 0.014 and 0.02 mg/kilo/day at birth, if the infant consumes 600 ml. (about 1½ pints) of breast milk daily. As the infant grows the intake of milk on a per-kilo basis decreases because food intake per unit of body weight lessens when the size of an animal increases. Furthermore, breast-fed infants usually receive supplementary feeding with other foods.

The "permissible rate" set by the WHO-FAO, according to the chairman of the meeting that established the value, is highly conservative, and he points out that

it offers a safety factor of about 25 compared with what workers in a DDT manufacturing plant have tolerated for 19 years without any detectable clinical effect (see Laws *et al.*, *Arch. Environ. Health*, 15: 766-775, 1967). The safety factor of the WHO-FAO permissible rate is 150 compared to the dosage of DDT given daily for 6 months to a patient with congenital unconjugated jaundice without producing any side effects (Thompson *et al.*, *Lancet* 11, (7610): 2-6, July 5, 1969).

Infants are more susceptible than adults to some compounds, but the difference is seldom great—usually about 2 to 3 times. In a study of 49 different compounds, newborn rats were found to vary from 5 times less susceptible to 10 times more susceptible than adults. Although there is no information on the relative susceptibility of human infants and adults to DDT, it is shown by Lu *et al.* (*Food and Cosmetic Toxic.*, 3: 591-596, 1965) that weanling rats are slightly more resistant than adult rats to this compound, and that preweanling rats are more than twice as resistant and newborn rats are over 20 times more resistant than adults.²⁰

Evidently it is possible for breast-fed infants to obtain DDT from the milk at a level up to twice the WHO-FAO "permissible

rate." Again, background information indicates that no toxic effects have been detected or could be anticipated at this level. Nevertheless, the EDF and its collaborators have conspicuously proclaimed a warning that DDT may cause cancer. This adds to public apprehension, especially among nursing mothers. The question of carcinogenicity therefore should be discussed.

The "Delaney Clause" of the Food Additives Amendment²¹ prohibits the use of any food additive that has been found to cause cancer in experimental animals. It is difficult to think of a more meritorious or public-spirited objective than is implied by this clause. It is even more difficult to *comply* with it, because all foods contain substances which can be shown to cause cancer in experimental animals, given the appropriate dose and the susceptible animal. All foods contain traces of radioactive elements which are present naturally. All meat products contain sterols and steroid hormones, which produce breast cancer in mice.

Pyrolysis—scorching—such as occurs in barbecuing of meat or the roasting of coffee, produces carcinogens and is only one of many examples of processes or substances in foods that can produce cancer in experimental animals when at high levels. The Delaney clause is usually regarded among scientists as being impossible either to administer or repeal. The Secretary's Commission on Pesticides, Department of Health, Education and Welfare made the following recommendation and comments:

Recommendation 8: Seek modification of the Delaney clause to permit the Secretary of the Department of Health, Education, and Welfare to determine when evidence of carcinogenesis justifies restrictive action concerning food containing analytically detectable traces of chemicals.

The effect of the Delaney clause is to require the removal from interstate commerce of any food which contains analytically detectable amounts of a food additive shown to be capable of inducing cancer in experimental animals. This requirement would be excessively conservative if applied to foods containing unavoidable trace amounts of pesticides shown to be capable of inducing cancer in experimental animals when given in very high doses. If this clause were to be enforced for pesticide residues, it would outlaw most food of animal origin including all meat, all dairy products (milk, butter, ice cream, cheese, etc.), eggs, fowl, and fish. These foods presently contain and will continue to contain for years, traces of DDT despite any restrictions imposed on pesticides. Removal of these foods would present a far worse hazard to health than uncertain carcinogenic risk of these trace amounts.

Commonly consumed foodstuffs, contain detectable amounts of unavoidable naturally occurring constituents which under certain experimental conditions are capable of inducing cancer in experimental animals. Yet, at the usual low level of intake of these constituents, they are regarded as presenting an acceptable risk to human health.

Exquisitely sensitive modern analytical techniques which became available since enactment of the Delaney clause permit detection of extremely small traces of chemicals at levels which may be biologically insignificant. Positive response in carcinogenic testing has often been shown to be dose-related, in that the carcinogenic response increases with increasing dose levels of the carcinogen; when the dosage of a carcinogen is minimized, the risk for cancer is also minimized or eliminated. . . .

The recommendation for revision of the Delaney clause is made in order to permit determinations essential to the protection of human health, not to justify irresponsible increases in the exposure of the population to carcinogenic hazards.²²

However, any attempt at such a revision would meet with great political opposition in the light of current fears and superstitions regarding "chemicals."

The above quotation speaks of the "uncertain carcinogenic risk" of trace amounts of pesticides. I shall review this statement with regard to DDT. The question of carcinogenicity of DDT was examined extensively starting in 1947. The above statement by the Commission was based on a recent report by Innes *et al.*²³ which is essentially a repetition of observations made about 20 years ago. The extensive earlier work on DDT and tumors in experimental animals includes about 20 articles in the scientific literature. In 1944, Lillie and Smith described hepatic alterations in rats kept on a diet containing 1000 ppm of DDT for 14 weeks.²⁴ Similar changes were observed repeatedly by subsequent investigators.

These findings aroused much interest and a number of toxicologists studied the effects of DDT in various experimental animals. Cameron and Burgess fed very high levels of DDT to rats, and produced liver damage that was severe enough to account for death in a number of the animals.²⁵ However, when the DDT was discontinued "the dead cells were removed by autolysis and phagocytic action and repair was complete without any fibrosis, although calcification was occasionally seen."²⁶ In plain language, if the rats were fed enough DDT to produce acute liver damage, and then the DDT was stopped, the animals got better. No cancer was found.

A more prolonged ordeal for rats, which were given a level of 100 ppm of DDT in the diet, was described by Fitzhugh and Nelson.²⁷ After two years, which is roughly equivalent to 70 years for a human being, a "minimal hepatocarcinogenic tendency" was noted by the authors. The authors could not decide whether the tumors were benign (adenomas) or low grade hepatic cell carcinomas.

These findings draw attention to an interesting matter known as the *dose-response curve*. It is possible to calculate the time of onset of symptoms from the daily dose of a toxic substance. If the dose is low enough, a calculation may show that the average animal will die of old age before it develops tumors. Since biological responses are subject to individual variability, the "average" animal does not represent all the animals in a group. There will be a few animals, perhaps only one in a million, that will develop tumors at a considerably lower dose than the average. This is the basis for suggesting so-called "mega-mouse" experiments (a million mice per experiment!) to detect borderline effects of mutagens and carcinogens. But in a million animals there are enough spontaneous tumors and mutations to make the results undecipherable at low levels of the chemical.

Fitzhugh and Nelson also described recovery experiments with rats on 1000 ppm of DDT for 12 weeks.²⁸ Extensive necrotic changes in the liver were produced during this period. The livers returned to normal after 8–10 weeks, indicating an absence of malignancy in the 12-week lesions. In these and various other studies with DDT, the minimum concentration in the diet was 100 ppm. At lower levels, the smallest detectable morphologic effect was at 5 ppm, reported by Laug *et al.*²⁹ These authors also reported hepatic cell changes in rabbits, mice and guinea pigs fed DDT, but the changes were not as marked as in rats. They were absent from chickens, dogs, cats, monkeys and large domestic animals. As a result, the liver changes are regarded by many pathologists as being characteristic of rodents. Various investigators have been unable to produce any pathology in rats fed DDT; indeed, the British investigators Cameron and Cheng concluded that the histological changes reported by others were "fixation artefacts."³⁰ These differences of opinion stimulated further research. This extensive series of publications and investigations was reviewed in 1965 by Arnold Lehman, chief of toxicology for the United States Food and Drug Administration, and his conclusion was that "DDT is not a carcinogen."³¹

The liver changes produced by DDT in rats and other rodents were described by Hayes as involving the cellular tissue that produces the "microsomal enzymes", as being reversible, and as being peculiar to rodents.³² He also pointed out that the changes could also be produced by phenobarbital, by pyrethrum (a "natural" insecticide), by ethyl alcohol and by oxidized fats. The microsomal enzymes have various effects including the breakdown of some toxic substances and certain hormones.

The allegations by the EDF that DDT is a carcinogen are based primarily on three reports. The first is an article by Innes and co-workers with mice, which the authors call a "preliminary note."³³ Just why it was necessary to publish a "preliminary note" on a subject (the effect of DDT on rodents) that had been covered exhaustively over a period of ten years by a large number of experiments is not clear. The amount of DDT fed was the maximum tolerated dose, 140 ppm in the diet, and the experiment lasted 18 months, which is most of the lifetime of a mouse. The results were as follows:³⁴

Supplement (and no.)	Level used		% Mice with tumors		
	mg/kg*	ppm	Hepatomas	Lung	Lymphoma
None (controls) (338)	—	—	4.1	6.2	4.1
<i>p,p'</i> -DDT (72)	46.4	140	31.9	5.5	10.9

* Dosage was oral for 7-28 days, then added to diet.

In confirmation of experiments reported earlier by other scientists, there was an eight-fold increase in hepatomas over the controls, and a borderline effect on lymphoma, to which mice are highly susceptible. Hepatomas are defined as tumors that are on the borderline between benign and malignant. Dr. Hayes commented as follows:

Innes, *et al.* (1969) reported that the tumorigenicity of selected pesticides and industrial compounds was tested by continuous oral administration to both sexes of two hybrid strains of mice, starting at the age of 7 days. The chemicals were given by stomach tube until weaning and thereafter as a mixture in the diet. Maximal tolerated doses were given for the entire period of observation, about 18 months. The authors stressed that the dose received by the mice was far in excess of that likely to be consumed by humans. One of the compounds that gave a statistically significant positive result was DDT. The incidence of tumors was comparable to the mean tumor incidence

produced by a group of positive control compounds, most of which are weak or even questionable carcinogens of no demonstrated importance to human health. The authors made no distinction between hepatomas and carcinomas. It is difficult to understand why, in denying the practicality of making this important distinction, they entirely neglected the matter of reversibility. A full account of the study is promised later. In the meantime there is no assurance that the small number of tumors observed in mice exposed to DDT were different from the "nodules" described by Fitzhugh and Nelson in 1947.³⁵

In this experiment, the mice received about 3000 times as much DDT in their diet as is consumed by people in the United States. It is primarily on this experiment that the allegations rest that DDT causes cancer. The other two reports are trivial; in one of them the diet of mice was suspected of being contaminated³⁶ and the second was based on a far-fetched inference which one of the authors (Deichmann) later "soft-pedaled."³⁷ The inference was that DDT was found in higher concentrations in fatty tissues of persons with terminal degenerative diseases than in fatty tissues from "normal" autopsy samples. However, such terminal diseases are usually accompanied by emaciation, which would concentrate the DDT in the remaining fat, and Deichmann, *et al.* have commented that the "investigators did not demonstrate a causal relationship between those diseases and pesticide retention in body tissues."³⁸ Yet a causal relationship is repeatedly emphasized by the lawyers for the EDF, the National Audubon Society and the Sierra Club.

The argument over DDT and cancer is important because millions of lives hang on it. If DDT were officially tabbed as a carcinogen in the United States, its use in the world-wide malaria control program would be severely inhibited or even stopped. What would this do? Ceylon has provided an excellent object-lesson:

Following a country-wide malaria eradication campaign in the 1950's and early 1960's, the number of confirmed malaria cases reached lows of 31 in 1962 and 17 in 1963, when full-scale house spraying was partially withdrawn, and subsequently terminated in 1964. The cases increased annually thereafter, numbering 150 in 1964, 308 in 1965, 499 in 1966, and 3466 in 1967, most of them occurring in the last few months of that year. In 1968, the epidemic flared rapidly—16,493 confirmed cases being reported in January and 42,161 in February. No DDT supplies were on hand with which to reinstate the house

spraying program on the wide scale needed, and months were required for the procurement and delivery of them. As a result, more than a million cases of malaria occurred throughout the country in 1968.³⁹

What is the *real* effect of DDT on babies? Let us reverse the coin—What is the effect of malaria? Burnet had something to say on this. He wrote in 1953 that malaria

is the great devitalizer of the tropics. . . and it is the main agent of infantile mortality. If malaria could be suddenly eliminated from the globe, the racial, economic and political consequences within a very few years would probably be appalling. India and parts of Africa are populated up to and beyond the capacity of the land to provide adequate food by present methods, and even with the tremendous infantile and prenatal mortality caused by malaria, the populations are increasing steadily. The sudden conversion to a more vigorous and rapidly increasing population would undoubtedly produce famine (emigration) and intense internal and external social repercussions.⁴⁰

Nine years after Burnet's article, the effects of the anti-malaria program in India were described by Pal:

The control programme [with DDT] was launched in April, 1953 and it was designed to give protection to 200 million people living in the malarious areas of the country. . . . Improved knowledge on malaria control led to the revision of the original strategy and the aim became the eradication of the disease for the entire sub-continent. In April, 1958, the National Malaria Eradication Programme was launched. It consists of three phases: attack, consolidation and maintenance. The attack phase is aimed at total interruption of transmission by spraying with residual insecticides all roofed structures throughout the country. . . . Since 1953, about 147,593,270 lb. of DDT have been used, with small amounts of BHC and dieldrin. As a result, malaria morbidity has been significantly reduced in the country. The proportional case rate of malaria (per cent of malaria cases to total diseases as clinically diagnosed) in each year of this programme has shown a decline. . . . Estimates of actual morbidity and mortality are difficult but it would appear, from the available data, that malaria in India has been reduced from 75 million cases to less than 5 million. A new era in economic development and social progress has been initiated with its beneficial transformation of the life of the people. The average span of life in India is now 47 years, whereas before the eradication campaign it was 32 years. This improvement has resulted in better agriculture and industrial production. In the Terai region (Uttar Pradesh), land under cultivation and food grain production

has increased and this region, once abandoned by its inhabitants because of the high incidence of malaria, has become a beautiful and prosperous area.⁴¹

WILDLIFE, NATURE STUDY AND INFERENCES

It is often difficult to obtain controlled and reliable results on the question of toxicity of chemicals to wildlife under field conditions. The temptation to blame pesticides indiscriminately for the death of wild creatures seems irresistible to organizations committed to the protection of wild animals. A trace of DDT reported in a dead bird or fish often triggers a chain reaction of publicity and incrimination. On the other side of the ideological fence, the farmers and entomologists give considerable weight to the fact that, in order to be able to eat and to protect themselves against major diseases, human beings must vigorously wage war on noxious insects. Chemical pesticides are essential in this fight. The release of pesticides into the environment, and the presence of traces of pesticides in our food, are inevitable if the human race is to maintain its present numbers and control of disease. It is obvious, of course, that the use of pesticides must be kept down to the minimum level commensurate with adequate crop production and disease control.

Modern agricultural technology as practiced in the United States is a development that has taken place largely in the past half-century, with major advances in the last 25 years. Geneticists, chemists and engineers have made great contributions to increasing the food supply and simultaneously lifting the burden of toil from farming. The easy availability of food has accelerated the movement of people to the cities.

Food is never pure. In the 1930's, contamination of food by pests was a major problem; for example, canned vegetables were spot-checked for pieces of insects. Today much testing is done for pesticide residues. With rare exceptions, the amounts found are well below the tolerances, which in themselves are far below the toxic levels. Pesticide residues in foods are not a public health problem. The absence of such residues could be brought about by stopping the use of pesticides. This would create a real problem—food shortages.

It would not be possible to reverse this movement and replace chemical technology by hand labor on farms without a great social dislocation. If chemical herbicides are not to be used, "the

man with the hoe" must return to the farm and work long hours. In China, until recently, grasshoppers were killed by hand, but most of the major pest insects are too elusive for even this archaic procedure, and "biological control" can play only a minor part in keeping such insects from destroying crops.

Many of the charges that DDT destroys wildlife are based on inference. Often the charges have been based on tests that appeared to detect traces of DDT or other pesticides, and no consideration was given to the quantitative aspects of the results. Sometimes the effect of non-pesticide factors is disregarded or ignored. An example is the occurrence of large numbers of dead fish in the lower Mississippi River. This was blamed, with great fanfare, on pesticide contamination of the water. Subsequently the deaths were attributed to bacteria, *Aeromonas liquefaciens*, and to a lack of oxygen resulting from run-off of flooded fields.

The decline of the crab catch in the vicinity of San Francisco in 1969 was blamed on DDT. A scare article and banner headline appeared on the front page of the *San Francisco Chronicle* stating that the decline resulted from the toxic effects of DDT.⁴² The story warned the public that crab meat might be contaminated. Strangely enough, the crab fishery further north on the California coast reported record catches for three seasons, and the most recent one was 14 million pounds (making one wonder whether the species will be "fished out"). In November, 1970, another San Francisco newspaper headlined: "What Happened to Our Crabs? Pollution!"⁴³ The article stated:

The dumping of millions of gallons of highly poisonous wastes off the Farallones is probably responsible for the drastic slump in the San Francisco crab fishery.

State Fish and Game Biologists Don Lollock and John Ladd in a report to the Regional Water Quality Control Board said that although absolute proof is lacking, evidence of the decline points to the oceanic pollution.

The catch has dropped from a high of nearly nine million pounds in the 1956-57 season to 1.4 million last season.

The biologists recommended the board take prompt action to stop such industrial waste discharging.

On receiving the State Fish and Game Department document, the board yesterday took emergency procedures to place the subject on the agenda for action at its Dec. 22 San Francisco meeting.

Three firms use the ocean for major dumping. U. S. Steel, given a Dec. 15 deadline yesterday for improving the quality of discharges

from its Pittsburg works, dumps in the neighborhood of 15 million gallons of acid steel waste containing sulphuric and hydrochloric acid annually.

Sulfates and large concentrations of heavy metals also are barged and unloaded in the Farallones Gulf, nine miles from the City.⁴⁴

What happened to our crabs? What happened to the DDT that was blamed in 1969?

DDT was found in the livers of dead sea lions on the coast of California in the late summer of 1970, and, as usual, the newspapers swung into action to condemn the insecticide. In November, a memorandum was issued by Dr. Richard Hubbard of the Marine Mammal Study Center, Fremont, California, describing the findings of a team that had diagnosed the deaths as being due to leptospirosis. The diagnosis was based on symptomatology, post-mortem findings, identification of *Leptospira pomona*, blood antigen tests and epidemiology. He commented that "there is no correlation between mercury and DDT levels, and sick animals." But, by November, who was interested? Certainly not the EDF. *Leptospira* is "part of the environment."

Robinson Jeffers has written, "Give your heart to the hawk." The bird protection societies would seemingly have us follow this admonition and, indeed, the speed and audacity of the bird of prey appeal to the Walter Mitty who lurks in most of us. Eagles have many admirers, while chickens (at least, while alive) have but few. The dire warnings that bald eagles were threatened by DDT were enough to arouse horror against this insidious chemical. For example, a photograph on the cover of *Science*⁴⁵ showed a bald eagle's nest with one unhatched egg and one apparently healthy eaglet. The failure of one of the eggs to hatch was attributed to the presence of DDT in fish in the Great Lakes area. But is the story true?

An examination of scientific literature which antedates the extensive use of DDT is instructive. It reveals that even in such earlier years the survival of the eagle was deemed a critical issue. For example, in 1921 an article entitled "Threatened Extinction of the Bald Eagle" appeared in *Ecology*. In 1943, F. Thone stated in *Science News Letter*: "When the timber was cleared, it was inevitable that the eagles had to go. Moreover, the cities grew and befouled the rivers with sewage and industrial wastes. The once teeming fish population vanished."

The Territory of Alaska paid a bounty of 25 cents per claw for 115,000 bald eagles assassinated between 1917 and 1952. There

are now an estimated 7000 bald eagles in Alaska, about 6% of the number that was slaughtered. Gunfire continues to be the main cause of death of bald eagles, according to United States Department of Interior figures: of 76 dead specimens examined between 1960 and 1965, 44 were shot, 7 died of impact injuries, 3 of other violent forms of death, 4 of disease or old age, and the remaining 18 of undetermined causes. More recently, the same Department has reported "poisoning from dieldrin in growing numbers of bald eagles found dead in the United States"⁴⁶ and also that mercury poisoning has been detected in bald eagles.⁴⁷ The last finding draws attention to the presence of potentially toxic levels of mercury in fish in the Great Lakes, and suggests that mercury may be the cause of reproductive failure in the bald eagles in this region.

Why was DDT blamed? As George Mallory said of a certain mountain, "Because it was there."

How are the eagles doing? According to the National Park Service, a record number of 373 bald eagles, 120 of which were immature, were counted on November 20, 1969, below Lake Macdonald in Glacier National Park and 268 (with the birds still flying in) on November 25, 1970. The percent of immatures has held "a steady 31" during the past five years. This meant, according to the Park Service, that the birds came from "areas not yet seriously affected by pesticides," an explanation which is on a logical par with stating that the birds are alive because they are not dead. Evidently pesticide poisoning plays no more than a minor role in the demise of bald eagles. The alleged effects of DDT are now being second-guessed in favor of other chemicals.

In Scotland, the use of dieldrin in sheep dips was concluded to be the cause of a decline in the breeding ability of the golden eagle, which eats sheep carrion. This use of dieldrin was discontinued in 1966, following which there was an improvement in breeding success in golden eagles.⁴⁸

The story of the peregrine falcon is similar in many respects to that of the eagle, except that the peregrine is even rarer. It is not only the object of man's hostility because it eats racing pigeons, but it is also harassed by the robbing of its eyries to provide young birds for falconry, and eggs for collectors. The peregrine falcon is considered as no longer breeding in the eastern United States. However, the counts of migrant peregrines at Hawk Mountain, Pennsylvania, were as follows:

1935-1942	ave.	32
1967		22
1968		21
1969		26
1970		27

It is obvious that the peregrine was rare even prior to 1940; the total annual count of hawks at Hawk Mountain ranges between 10,000 and 20,000.

The rapid decline of the peregrine in the eastern United States took place prior to the introduction of organic pesticides,⁴⁹ and seems to have been caused by harassment.⁵⁰ The peregrine population in the eastern United States is estimated as having been something less than 275 breeding pairs in 1940. In contrast, the peregrine population in Northern Canada and Alaska is reported to be thriving and was estimated by Fyfe (1969) as about 7,500 breeding pairs.⁵¹

Whether or not counts like those above can be read as linking DDT with animal deaths, the counts must be kept in proper perspective. All such statistics should be reviewed in the context of other "human" statistics. One cannot ignore, for example, that before the advent of DDT, 2000 *people per day* were dying in India from malaria.

Other analyses have been made of the effects of DDT on the peregrine. High levels of DDT and its metabolites were reported in the fat of peregrine falcons in the Yukon⁵² but a "seemingly normal average" of viable eggs and young was found in 15 nests in this region. Fat biopsy samples from nine females had an average content of 37 ppm DDT, 284 ppm DDE and 40 ppm TDE (DDD). The samples also contained an average of 3.3 ppm diel-drin and 4.4 ppm heptachlor epoxide. During this survey, the nests were robbed to obtain eggs for analysis, and female falcons were trapped and slit open to take samples of body fat. Following this, the unfortunate birds were sutured and released, after which they "showed normal behavior." One wonders how long they survived. The authors noted that the eggs were taken from eyries where reproduction was normal and averaged about 27 ppm of total organochlorine residues. Ratcliffe stated that the residues in eggs from unsuccessful eyries were 17.4 ppm; these were to be compared with the residues of 12.7 ppm in eggs from successful eyries in Great Britain.⁵³ The disparity between the two sets of findings suggests that the reason for reproductive failure in the peregrine has not been identified.

The British Advisory Committee on Pesticides and other Toxic Chemicals, in a comprehensive review on organochlorine pesticides published in 1969, stated:

There is no close correlation between the declines in populations in predatory birds, particularly in the peregrine falcon and the sparrowhawk, . . . and the use of DDT. Therefore DDT does not appear to have been the principal cause.⁵⁴

In summary, the peregrine as a breeding species in the eastern United States appears to have been extirpated primarily by harassment, and no role for DDT has been shown. The peregrines in northern Canada are breeding successfully despite high tissue levels of organochlorine pesticides. The current reduction in the use of these pesticides should tend to a lowering of these levels, and the future of peregrines in this region does not appear to be in jeopardy.

The osprey, or fish hawk, was a conspicuous species along the eastern seaboard of the United States, even in populated areas such as Gibson Island on Chesapeake Bay. A decline in the population of ospreys in such areas has been attributed to DDT. Harassment seems more likely to be the major cause, because the osprey is evidently increasing in the eastern United States and Canada. An obvious explanation is that the birds have moved away from the region of suburban developers and outboard motors in search of peace and quiet. The osprey count at Hawk Mountain averaged 172 per year from 1935 to 1942, pre-DDT years. Recent counts are:

1965	444
1966	405
1967	467
1968	403
1969	530
1970	600 (a record high)

The counts at Hawk Mountain must be interpreted with care, because up until about 10 years ago there was only one look-out, and now there are three, at least two of which "are manned every day, all day, from mid-August through November," according to Dr. J. W. Taylor, who estimates that 20 years ago, under today's conditions, the "number of hawks seen would have been at least three times, and probably five or six times, what our figures in 1969 show." Dr. Taylor nevertheless states that "the

Osprey population on the interior lakes has greatly increased, and these are the birds we are now seeing at Hawk Mountain."

Naturally all of us hope that hawks and eagles will survive the onslaughts of human interference. However, blaming DDT seems a convenient excuse. If a species is still being counted at Hawk Mountain, it is obviously *not* extinct, and if the numbers have not decreased greatly in recent years an interpretation that the species is drastically declining is questionable in the absence of further study.

DDT AND PHOTOSYNTHESIS IN THE OCEAN

DDT is said to be steadily accumulating in the seas by distillation from the surface of the land, by drainage into rivers and by the blowing of dust. It is also alleged that DDT is, in effect, indestructible, because its principal metabolite, DDE, resembles DDT in inducing the production of microsomal liver enzymes. One of the most sensational anecdotes about DDT is the prediction that it will stop photosynthesis in the oceans, as a result of which life on Earth will become extinct. This story was repeated by diverse authorities ranging from U Thant to Ehrlich, who stated⁵⁵ that it originated in Wurster's report.⁵⁶ This report described the effects of adding graded amounts of an alcoholic solution of DDT to algal cultures in sea water and measuring photosynthesis by carbon-14 uptake. The DDT was added to produce concentrations up to 500 ppb. Not surprisingly, photosynthetic uptake of carbon-14 was depressed at the higher levels, since DDT is phytotoxic above certain levels. Its maximum solubility in sea water is 1.2 ppb, and DDT would be precipitated and adsorb to the algae above this level. The results as presented by Wurster show that at the points corresponding to 1 and 2 ppb, there was no depression of carbon-14 uptake.

The findings at these levels were as follows:⁵⁷

Species	Effect of DDT on C ¹⁴ Uptake*	
	1 ppb	2 ppb
<i>Skeletonema costatum</i>	—	—
<i>Coccolithus huxleyi</i>	+	+
<i>Pyramimonas</i> sp.	—	+
<i>Peridinium trochoideum</i>	+	+
Mixed culture**	+	+

* Increases over controls, +; decreases —.

** Typical neritic phytoplankton community.

According to these fragmentary findings, a saturated solution of DDT in sea water would not depress photosynthesis. Wurster states:

The fact that these data apparently follow sigmoid curves is typical of dose-response relations and suggests the absence of a threshold concentration of DDT below which no effects occur. Experimental scatter produced some uptake of C^{14} above 100 percent at low concentrations of DDT, however. This should not be interpreted as a low-level stimulatory effect, a possibility that cannot be evaluated from these data.⁵⁸

Obviously if the data show an indication, even slight, of a low-level stimulatory effect, it follows that the same data cannot be used to postulate a "no threshold" situation. Wurster pointed out that "water near a direct application of DDT to the environment, however, commonly contains concentrations comparable to those applied by me."⁵⁹ This is a far cry from stopping photosynthesis in the oceans as suggested by Ehrlich. Wurster goes on to state that "selective toxic stress by DDT on certain algae" may "favor species normally suppressed by others, producing population explosions. . . . Such effects are insidious."⁶⁰ (The word "insidious" is a favorite word in the lexicography of DDT.) Increases and decreases in aquatic photosynthesis can both be blamed on DDT. It is a well-documented observation that over-application of DDT to green crop plants may cause not only depression of photosynthesis, but death. Over-application of various chemicals kills plants; common salt, for example, used to be used as a weed-killer.

To produce a concentration of 1 ppb of DDT in the 300 million cubic miles of sea water in the oceans would take 9,000 years if the total annual production of DDT, 300 million pounds, were dispersed in the oceans each year and there was no breakdown. If the half-life of DDT in sea water is one thousand years or less, this concentration would never be reached.

The absurdity of these figures illustrates the need for quantitative examination of allegations, but is not intended as a suggestion that it is safe or desirable to use the ocean as a sink for pollutants.

CONCLUSION

This article has treated only a few of the points that demonstrate DDT's ultimate safety and significant contributions to

man. I have chosen to discuss these few points at length, rather than to mention a large number of topics briefly. A final response, however, must be made to the charge that DDT has injurious effects on human beings. The particular charge to which I respond is based on the observation that DDT inhibits certain enzymes in laboratory experiments. Contrary to what DDT opponents would have us believe, a similar observation can be made of any of a number of substances. For example, many of the substances that we eat—salt, phosphate, magnesium, citric acid—drastically affect enzymes in test tube experiments. These experiments, moreover, do not involve intact animals. By contrast, the everyday use of DDT does involve intact animals, and the effects are remarkably minimal. In point of fact, *several hundred million people* have been exposed to DDT for prolonged periods of time without any sign of ill effect. Some of them have received heavy doses, over periods at up to 19 years⁶¹—the 130,000 spraymen listed by the WHO, for example, and numerous people in DDT factories and formulating plants—all without any reports of illnesses attributable to DDT. In the 1940's, enthusiastic volunteers allowed themselves to be used as experimental animals and swallowed large amounts of DDT, in some cases daily for prolonged periods.⁶² For some reason, nothing seemed to happen except transient tingling of the extremities. Some day the true story of DDT, buried in the scientific literature, will be brought out into the open.

We may certainly expect an increase in dialogue between scientists and lawyers as the number of legal questions involving the environment grows apace. Such dialogue, however, when it occurs in the tightly structured setting of the courtroom, may have unfortunate consequences. If a scientist is asked, for example, whether a pesticide is poisonous, he will say "Yes;" at the same time, however, he may recommend its use at an appropriate level as a proper response to human needs. A skillful attorney can effectively exploit such an apparent contradiction, following which the scientist will probably withdraw into his shell. In the 1969 Wisconsin hearings on DDT, such incidents repeatedly took place during examinations by Environmental Defense Fund (EDF) attorney, Victor J. Yannacone, Jr. An EDF spokesman, C. F. Wurster, expressed much satisfaction with Mr. Yannacone's efforts:

Victor J. Yannacone, Jr., the Environmental Defense Fund's at-

torney, has an impressive grasp of scientific material, especially the environmental sciences. His cross-examination is usually aggressive and may be devastating where a witness is scientifically weak.⁶³

Wurster, however, later altered his opinion. On March 9, 1971, he stated:

It has come to my attention that certain remarks, attributed to me by Mr. Victor J. Yannacone, Jr. in May 1970, have been inserted into the record of your hearings on pesticides during the testimony of Edward Lee Rogers of the Environmental Defense Fund.

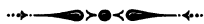
I wish to deny all of the statements of Mr. Yannacone. His remarks about me, attributable to me, and about other trustees of EDF are purely fantasy and bear no resemblance to the truth. It was in part because Mr. Yannacone lost touch with reality that he was dismissed by EDF, and his remarks of May 1970 indicate that his inability to separate fact from fiction has accelerated.⁶⁴

Yannacone's fiery and uncompromising onslaught on DDT in Wisconsin was evidently undertaken for the purposes of advocacy, for on September 27, 1970, he said, in comparatively moderate tones:

Any law simplistically banning the use, sale, manufacture or distribution of DDT in your state, county, city or even the United States, without at the same time establishing an ecologically sophisticated pesticide regulation program, is a bad law. It won't satisfy anyone very long and will permanently polarize agriculture and conservation to such an extent that common problems can no longer be considered in rational discourse.⁶⁵

These remarks by Mr. Yannacone reflect a scientific sophistication that he did not reveal during the Wisconsin hearings. Regrettably, however, the earlier remarks seem to have gained greater attention.

This anecdote serves to illustrate an essential difference between the advocate and the scientist. The advocate seeks a prompt and unequivocal decision on a particular issue. The scientist, however, simply cannot expect such results; he thus strives to obtain facts which enable him to ascend the spiral staircase of knowledge. With each step of his ascent, he obtains a wider and superior view of his subject matter, and, although he never reaches the top, he should on his way up help his fellow men by telling them what he sees.



FOOTNOTES

❖ Professor of Medical Physics, University of California, Berkeley.

¹ J. Slater, "Third World Ecology Course to Be Offered," *The Journal of Educational Change* 2:1 (Oct. 1970).

² Rachel L. Carson, *Silent Spring* (1962).

³ Advertisements by National Audubon Society, 1970, in various magazines.

⁴ "Our Ecological Crisis," *National Geographic Magazine* (Dec. 1970).

⁵ U. S. Court of Appeals, District of Columbia, Circuit Petition for Review of Order of U. S. Dept. of Agriculture, No. 23, 813 (Apr. 6, 1971).

⁶ Martin G. Garcia (WHO), Personal Communication to S. Rotrosen (June 19, 1969).

⁷ See P. DeKruif, "The Magic Bullet," *Microbe Hunters*, p. 308 (1926).

⁸ R. Pal, "Contributions of Insecticides to Public Health in India," *World Review of Pest Control* 1:6 (1962).

⁹ P. F. Russell, The United States and Malaria: Debits and Credits *Bulletin of the New York Academy of Medicine* 44:623 (1968).

¹⁰ *Id.*

¹¹ T. H. Jukes, "DDT: The Chemical of Social Change," *Clinical Toxicology* 2:359 (Dec. 1969).

¹² "Environmental Scorecard," *Richmond Independent* (Calif.) 10 (Dec. 17, 1970).

¹³ E. Chaney, *Conservation News* (June 15, 1970).

¹⁴ W. J. Hayes, Jr., "Toxicity of Pesticides to Man: Risks from Present Levels" *Proc. Roy. Soc. B.* 167:101 (1967).

¹⁵ W. J. Hayes, Jr., W. E. Dale, and C. I. Prikle, "Evidence of Safety of Long Term, High, Oral Doses of DDT for Man," *Arch. Environ. Health* 22:119 (1971).

¹⁶ J. M. Barnes, "Food and Health—The Safe Use of Pesticide," *British Food Journal* (May–June 1967).

¹⁷ *Id.*

¹⁸ E. P. Laug, F. M. Kunze and C. S. Prickett, "Occurrence of DDT in Human Fat and Milk," *Arch. Indus. Hyg.* 3:245 (1951).

¹⁹ G. E. Quinby, J. F. Armstrong, and W. F. Durham, "DDT in Human Milk," *Nature* 207:726 (Aug. 14, 1965).

²⁰ Personal communication from W. J. Hayes, Jr. to P. Gyorgy, (June 4, 1970).

²¹ 21 U.S.C. §348 (c) (3) (A).

²² U. S. Department of Health, Education and Welfare, Report of the Secretary's Commission on Pesticides and Their Relation to Environmental Health, pts. I and II, at i-xvii and 1-677 (Dec. 1969).

²³ J. R. M. Innes, B. M. Uland, M. G. Valerio, L. Petrucelli, L.

Fishbein, E. R. Hart, and A. J. Pallotta; R. R. Bates, H. L. Falk, J. J. Gart, M. Klein, I. Mitchell, and J. Peters, "Bioassay of Pesticides and Industrial Chemicals for Tumorigenicity in Mice: A Preliminary Note," *J. Natl. Cancer Inst.* 44:1101 (1969).

²⁴ R. D. Lillie and M. I. Smith, Pathology of Experimental Poisoning in Cats, Rabbits and Rats with DDT" *U. S. Pub. Health Reports* 49:979 (1944).

²⁵ A. R. Cameron and F. Burgess, *British Medical Journal* 1:865 (1945).

²⁶ *Id.*

²⁷ O. G. Fitzhugh and A. A. Nelson, The Chronic Oral Toxicity of DDT, *J. Pharmacol.* 89: 18 (1947).

²⁸ *Id.*

²⁹ E. P. Laug, A. A. Nelson, O. G. Fitzhugh, and F. M. Kunzer "Liver Cell Alteration and DDT Storage in the Fat of the Rat Induced by Dietary Levels of 1 to 50 ppm DDT," *J. Pharmacol.* 98:268 (1950).

³⁰ G. R. Cameron and M. B. Cheng, "Failure of Oral DDT to Induce Toxic Changes in Rats," *British Medical Journal* 2:819 (1951).

³¹ A. J. Lehman, Summaries of Pesticide Toxicity (Assoc. Food and Drug Officials of the U. S., 1965).

³² W. J. Hayes, Jr. testimony at State of Washington Hearings on DDT (Seattle Oct. 15, 1969).

³³ J. R. M. Innes *et al.*, *supra* note 23.

³⁴ *Id.*

³⁵ W. J. Hayes, Jr., *supra* note 32.

³⁶ The Place of DDT in Operations Against Malaria and Other Vector Borne Diseases, WHO Statement EB 47/WP/14 (Jan. 22, 1971).

³⁷ Statement by Committee on Experimental Toxicology, *J. Am. Med. Assoc.* 212:1055 (1970).

³⁸ *Id.*

³⁹ National Communicable Disease Center, Department of Health, Education and Welfare, DDT in Malaria Control and Eradication (July 12, 1969).

⁴⁰ Sir Macfarlane Burnet, *Natural History of Infectious Disease*, (2d ed. 1953).

⁴¹ R. Pal, *supra* note 8.

⁴² "DDT Killing Crabs," *San Francisco Chronicle* (May 3, 1969).

⁴³ "What Happened to Our Crabs? Pollution!" *San Francisco Examiner*, p. 11. (Nov. 14, 1970).

⁴⁴ *Id.*

⁴⁵ *Science*, Vol. 162 (Feb. 7, 1969).

⁴⁶ *San Francisco Chronicle* (June 13, 1970).

⁴⁷ "Science for the Citizen," *Scientific American* p. 86 (Sep. 1970).

⁴⁸ J. D. Lockie, D. A. Ratcliffe, & R. Balharry, *Journal of Applied Ecology* 6:381 (1969).

⁴⁹ J. J. Hickey, "Eastern Population of the Duck Hawk," *Auk* 59:176 (1942).

⁵⁰ R. A. Herbert and K. G. S. Herbert, "The Extirpation of the Hudson River Peregrine Falcon Population," *Peregrine Falcon Populations—Their Biology and Decline* pp. 133–154 (1959).

⁵¹ R. Fyfe, "The Peregrine Falcon in Northern Canada," *Peregrine Falcon Populations—Their Biology and Decline*, pp. 101–114 (1959).

⁵² J. H. Enderson & D. D. Berger, *Condor* 70:149 (1968).

⁵³ D. A. Ratcliffe, "The Peregrine Situation in Great Britain," 1965–66 *Bird Study* 14, 238 (1968).

⁵⁴ Advisory Committee on Pesticides and Other Toxic Chemicals, Department of Education and Science, London, *Further Review of Certain Persistent Organochlorine Pesticides Used in Great Britain* (1969).

⁵⁵ P. Ehrlich, "Eco-catastrophe," *Ramparts* 8:24 (Sep. 1969).

⁵⁶ C. F. Wurster, "DDT Reduces Photosynthesis by Marine Phytoplankton," *Science*, 159:1474 (1968).

⁵⁷ *Id.*

⁵⁸ *Id.*

⁵⁹ *Id.*

⁶⁰ *Id.*

⁶¹ W. J. Hayes, Jr., W. E. Dale, and C. I. Prinkle, "Evidence of Safety of Long-Term High, Oral Doses of DDT for Man," *Arch. Environ. Health*, 22:119 (1971).

⁶² W. J. Hayes, Jr., in "DDT The Insecticide Dichlorodiphenyltrichloroethane and its Significance," *Human and Veterinary Medicine* 11:252 (P. Muller, ed., 1959).

⁶³ C. F. Wurster, "DDT Goes to Trial in Madison," *BioScience* 19:809 (1969).

⁶⁴ C. F. Wurster, in transcript of Congressional Hearings on Agriculture, Representative Poage, Chairman, (Mar. 9, 1971).

⁶⁵ V. J. Yannacone, Jr., in *Highlights* 70, Congress for Recreation and Parks, Philadelphia, Pa., p. 24, (Sep. 27–30, 1970).