Pesticides and Wildlife

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Foreword

The 'sixties and 'seventies will be remembered as a time of widespread public concern about the degradation of the environment. Rachael Carson's *Silent Spring* (1962) and Robert Rudd's *Pesticides and the Living Landscape* (1964) focused international attention on the problems arising from the indiscriminate use of pesticides. Then in 1966, the scientific community was shocked to learn that insecticide residues had been discovered in penguins and seals from the Antarctic – thousands of miles from the nearest site of application. As research progressed, the organochlorine insecticides and some industrial compounds were shown to be universal contaminants of every living biological system. Even more dismaying was the discovery that these chemicals were contributing to the declines of populations of birds, and that they interfered with the reproductive success of some fish and birds.

The Canadian Wildlife Service, through its Toxic Chemicals Section (formerly the Pesticides Section) has contributed much to scientific understanding of the side effects on various wildlife species of the use of agricultural and industrial chemicals. The following four articles are extracts from talks about this research, given by two CWS biologists. J. A. Keith and R. W. Fyfe share CWS concern for the environment as a whole; their personal commitment to the attainment of a healthy global ecosystem is expressed through their work with the Toxic Chemicals Section of CWS.

J. S. Tener, Director
Canadian Wildlife Service

Pesticides and Wildlife*

by Richard Fyfe

Pollution is a very common word these days. It may be defined as any contamination of the natural environment – whether that of a small fish pond, or the total environment of the world – by a substance capable of changing it.

Pesticide contamination is unfortunately an aspect of environmental pollution which is vastly underrated by the public. While many of us have read that pesticide contamination is universal, we may remain unconvinced that this condition actually exists in Canada. In fact, *nearly all* samples of Canadian wildlife analyzed during the past five years contained pesticide residues. These samples, collected throughout southern Canada, along the Atlantic and Pacific coasts and from the Canadian Arctic, have included mammals, fish, fish food, marine invertebrates and both migratory and non-migratory birds. Even the air and rainwater have been shown to contain insecticide residues!

In general, the residue levels in Canadian samples are comparable to those found in the same species, or species at equivalent links of food chains, outside Canada. For example, the average insecticide residue levels in the brains of arctic ground squirrels were similar to those found in ground squirrels from the southern prairies. Also, residue levels in migratory birds from the Arctic tend to be similar to levels in the same species, or species with similar food habits, further south. Many people are surprised that pesticide residues are also found in the tissues of species that remain in the north year-round, even in the arctic lemming, which rarely moves beyond a radius of a few hundred yards during its entire life.

Some scientists argued at one time that the contamination found in our wild birds represented residues picked up during migration, and therefore did not reflect Canadian use patterns or Canadian problems. However, there is evidence to the contrary. An analysis of more than 100 samples of similar tissues from 20 prey species of Saskatchewan and Alberta raptors included material from six species that were year-round resident birds. Yet the average level of pesticides in these six species was actually higher than that found in six of the migratory species.

How have the pesticides spread to such an extent? Obviously, pesticide pollution begins because the effects of these chemicals are not limited to the target species, and is further complicated because the chemicals are persistent and do not remain at the site of application, as revealed by several studies. Instead they are transported in the following ways through the air, water, soil and, of course, in living organisms.

It has been estimated that anywhere from a fraction of one per cent to a maximum of 40 per cent of any pesticide actually hits the target, using conventional application techniques. It has further been demonstrated that usually a much smaller percentage remains in the target area. For example, in spray operations which intentionally produce atomized droplets, a large percentage of the chemical goes directly into the atmosphere. Field studies have shown that, depending on the method of application and the climatic conditions, aerial spraying may result in as much as 60 – 90 per cent of the pesticide never reaching the target. A high percentage commonly goes directly into the atmosphere; and once there, these chemicals can circle the globe in a matter of a few weeks.

In much the same way during spray operations, drift to adjacent fields can cause serious contamination. Anyone driving the prairie roads during spray operations is familiar with the phenomenon of drift, as the

*Presented to the St. Albert Fish and Game Club St. Albert, Alberta, January 1970.

smell of 2, 4-D fills the air. So are the many people who have suffered unintentional damage to fruit trees and shelter belts. In this regard, I was advised by one agricultural field man who travels extensively throughout Alberta that with but one exception all of the Manitoba maple trees examined in 1969 showed the effects of 2, 4-D poisoning.

Following application the chemical combines with organic material or soil particles, and is transported in living organisms or through soil or water erosion. Next to direct dumping, this is undoubtedly the manner in which the greatest percentage of pesticide contamination reaches our lakes, rivers and oceans.

Perhaps the most unusual method of transport, as shown with DDT, is by co-distillation of pesticides with water. DDT moves into the atmosphere from water or wet surfaces to return elsewhere in rainfall or atmospheric fallout.

The last method of transport I will mention is intentional dumping, including the cleaning of equipment and the dumping of excess chemicals and treated seed. These practices are very widespread and have contaminated many watersheds. In particular they have seriously contaminated trout streams in the Maritimes and dumping excess treated seed has been one of the sources of mercury and organochlorine insecticide contamination in Alberta wildlife.

Pesticides are transported by these means in this country and throughout the world. The very properties which lend to their value as insecticides, in particular the residual qualities of some pesticides, result in long-term and widespread contamination.

To understand the significance of pesticide contamination, some of the properties of these chemicals, in particular DDT and the other organochlorines, should be considered. DDT was first invented in 1874; however, its insecticidal properties, which include high toxicity and chemical stability were not recognized until just prior to World War II. Very shortly thereafter this chemical became a panacea for all insect problems. Unfortunately, side effects were of little concern at that time and were even less understood.

DDT was soon followed by the development of the cyclodiene insecticides, a group which includes dieldrin, aldrin, endrin, heptachlor and others which, together with DDT, are classed as chlorinated hydrocarbons or organochlorines. To understand the potential consequences of these materials moving through the natural environment, we must look at some of their chemical, biological and physical properties.

Chemical stability

Chemical stability is a most desirable property for a long-term insecticide.

In general, the chlorinated hydrocarbons are very stable compounds, with estimated half-lives of 10 to 15 years. This residual quality, is further extended by the even greater stability of some of the breakdown products – as in the case of DDT, where certain tissues do bring about a gradual breakdown, and DDT is metabolized into DDD and DDE. DDE, in particular, is apparently far more persistent than the original compound and even now this breakdown product of DDT is the most widely distributed pesticide in our environment. In other words, once we put these chemicals into our environment, they are going to be there for a very long time.

I think this is an especially sobering thought when you stop to think that our environment, in terms of volume or mass, is a constant: there will never be any more water nor any more air on this planet than there is right this minute. Granted, water is constantly changing form, from fluid to gas to solid, etc., but the total amount will always be the same, and the

ratio of one form to another will always remain in relatively constant balance unless we change it. We can do this in a variety of ways, not the least of which is polluting the air and water with toxic chemicals (such as carbon monoxide and pesticides) faster than nature is able to render them harmless.

Absorption

The absorptive property of the organochlorines helps to explain why these chemicals, in particular, are becoming a more and more serious contamination problem.

The chlorinated hydrocarbons rank among the least water soluble substances. DDT is almost insoluble in water. In sharp contrast however, these chemicals are highly soluble in organic solvents such as hexane, benzene, etc., and in lipids, i.e. fat or fat-like materials. Consequently, they are more soluble in biological material than in water; and, as one ecologist has pointed out, we must therefore expect the chlorinated hydrocarbons to flow into organic material. These materials are simply absorbed into biological material through the cell walls in the gills, lungs, integument (e.g., skin) or, when eaten, through the alimentary tract. Once in organic material, a high percentage of the organochlorines and some of the other pesticides are held in the fat and other tissues. Organisms therefore tend to accumulate the residues, becoming contaminated even from an environment with low levels of contamination (as for example the water in a lake or river). This may occur miles away from the source of pollution, as with mercury residues found in Manitoba fish. It is for this reason that we must analyze living organisms rather than water, soil, or air to establish current or potential pesticide pollution problems.

Once the chemicals are in an organic material they are introduced into food chains, and the levels are greatly magnified by the phenomenon of biological concentration.

In simple terms, biological concentration means the way that each organism eats many organisms from the preceding link in the food chain. The food thus eaten is digested and excreted, but much of the insecticide is retained. The actual concentrations vary with the rate of intake and excretion, and the concentration factor may vary from two to three times to as much as 100 or even 1,000 between predator and prey, or as much as a million times between a carnivore and its environment. As a result of this, an organism at the end of a food chain can and does serve as an indicator of the degree of pesticide pollution and in the case of biocides such as the organochlorines or mercury, can give early warning of current or potential pesticide pollution problems. Thus it was prairie falcons and pigeon hawks that warned of mercury contamination in the prairies, and pike and pickerel of mercury contamination in carnivorous fish. For this reason we have been using wildlife, and in particular, birds at the end of the food chain, to monitor pesticides in this country. The animals and birds at the ends of food chains have the highest concentrations of these chemicals and are among the first to show serious population declines and sub-lethal effects. Remember man as a meat-eater is at the end of similar food chains, and he lives in the same environment. In wildlife we have a built-in warning system, and if we cannot learn from what has already happened, just how foolish can we be?

Toxicity

The third property of pesticides, and of equal importance with chemical stability and absorption, is toxicity. Unfortunately, far from being specific to insects alone, almost all pesticides are in fact biocides, in

Richardson's ground squirrels eat mercury treated grain in western Canada. They are in turn eaten by predatory birds such as Swainson's and red-tailed hawks. Photo by Richard Fyfe.

that they are toxic to many different organisms. The organochlorines, in particular, are highly toxic to a broad spectrum of animal life, including arthropods, molluscs and all vertebrates. They are basically nerve poisons – which means that any organism with nerves can be killed by them. The acute toxicity, often referred to as the LD 50, varies with each chemical and with each species. The following examples of acute toxicity are derived from experimental testing to determine the LD 50 in rats and are expressed in parts per million (ppm). For comparison, lead arsenate has an LD 50 of 825 ppm (in other words, 825 ppm of lead arsenate would be a lethal dose for 50 out of 100 rats). Some of the organochlorine toxicities are as follows: DDT 250, lindane 125, aldrin 67, heptachlor 60, and endrin 10.

What do these figures represent? Nothing except the LD 50 for rats and perhaps the relative potential toxicity of the chemicals on other forms of life. They in no way represent the acute toxicity to other organisms, as each organism has specific tolerance levels dependent on size, sensitivities, and different physiological abilities to concentrate or excrete these or other toxic substances, and they are almost meaningless from the point of view of environmental hazard, since we do not know the LD 50's for most organisms. We do know that some organisms are unbelievably sensitive to these poisons. For example, a small crustacean, the brine shrimp, can be killed by a concentration of DDT in water of not more than one part per trillion, and trout fry are readily killed by a few parts per million. However, as frightening as the release and distribution of these biocides in our environment may seem, the acute toxicity is in no way the greatest threat of pesticide contamination. Although there are many recorded instances of wildlife, domestic animals and even humans being poisoned by these chemicals, in general, these are isolated cases of accidental poisoning. Usually these cases result from either the direct intake of the chemicals, or indirectly by one organism eating other organic material which has been contaminated. The chemicals are soon transported beyond the target area, so that a particular hazard of direct acute poisoning is seldom long-lived.

The most important aspect of pesticide pollution is that these chemicals also have properties capable of causing disastrous sub-lethal side effects on organisms and on the environment. Over this worrisome aspect, ecologists seem divided from the pesticides and agricultural industries and their scientific advisers. I do not know why this is the case. Perhaps some of these scientists are so oriented to controlling and killing target species that they have developed an LD 50 complex and simply have not yet accepted the significance of sub-lethal effects. Nevertheless, wildlife ecologists have been required to show over and over again that a given toxic chemical is harmful and, in some cases, to duplicate data already proven by a researcher in another field. This was the case in demonstrating mercury contamination of seed-eaters and their predators as this had been shown for some compounds in Sweden. It was also the case when the effect of DDT on egg shell thinning was demonstrated. Often researchers have to prove the effects of pesticides in different geographical areas. It is difficult to grasp the logic behind this approach when it seems so obvious that the onus should have been on the industries who manufacture, or who may wish to use, any chemical to show conclusively before it is used that it is not harmful. Millions of dollars are spent on testing these chemicals; however, in the past this testing has obviously been inadequate, or we would not be faced with the pollution problems that we have today both with pesticides and other forms of air, water

and soil pollution. Our primary concern, and I sincerely believe everyone's concern at the present time, should be the effect of these chemicals on all living organisms in the environment. Instead, and I speak from personal experience, there are not just a few people who are more upset over the fact that we have proved that a problem does exist than they are over the problem. This is like getting angry at the doctor for discovering that your girlfriend is pregnant.

Sub-lethal effects

Sub-lethal effects, though not serious enough to kill the animal, usually manifest themselves by the particular impairment of a vital function in the organism, in some cases affecting behavior, or in others affecting various physiological functions, including reproduction. The most obvious side effects are those related to reproduction, and usually appear in one of the following ways.

1 A decrease in egg production occurs as has been demonstrated for several different chlorinated hydrocarbons in many species of birds.

2 There is a decrease in hatchability of eggs. This has also been demonstrated in several species of birds and fish, including pheasants with mercury levels as low as .5 ppm. It is ironic that in pheasants, trace levels of mercury can actually increase egg production—the problem is to control the levels.

3 The viability of the young that hatch decreases as demonstrated in several bird species, including chickens, and also in fish, at levels as low as .4 ppm.

4 Genetic change occur. Changes in chromosome structure and cell division because of mercury have been documented with levels as low as .05 ppm in plants. This effect has also been documented in mammals, and can result in abnormal young. In relation to genetic changes I suspect that everyone is aware of the current U.S. measures on the two herbicides, 2, 4, 5-T and 2, 4-D. Research indicates that impurities in 2, 4, 5-T formulation have affected chromosome changes in experimental mammals, and the reportedly "harmless" 2, 4-D is undergoing intensive investigation. Again, it should be noted that the concern is over the potential side effects of the chemicals. We all remember the thalidomide problem – as far as I know, thalidomide didn't kill anybody.

I think it would be appropriate at this time to discuss the mercury problem, since it has been identified as a current problem in western Canada.

Specifically, mercury is one of the heavy metals which is very toxic to both man and animals. Obviously this is always a concern when dealing with any toxin; however, as in the case of the organochlorines, we are equally or perhaps more concerned with the sub-lethal effects that could result from widespread contamination.

In this regard, levels of mercury as low as .5 ppm have been shown to cause a decrease in hatchability, and low levels of mercury can cause a breakage of chromosomes in both plants and mammals. It is not yet known to what extent, if any, the same effect will be manifested in humans. On the other hand, I have been advised by public health officials that small amounts of mercury can damage the kidneys, and apparently some people may be specifically sensitive to this substance.

Too many people are still thinking only in terms of acute toxic levels of chemicals. When mercury contamination was discovered the first and almost only question asked and argued about was whether it constituted a threat to human health, and even more specifically, how many contaminated birds it would take to kill a man. No one seemed aware of, or concerned about, the possible side effects of mercury contamination to humans or even to the pheasants

and partridge, let alone to any other species. I will now go into some details of the mercury investigations in order to clarify the many misconceptions concerning this problem.

Our concern over possible mercury pollution came about largely because of work done in Sweden. In Sweden, mercury has been the chemical pollutant causing the most serious wildlife and environmental consequences, comparable to dieldrin in Britain and DDT in North America. Mercury was used in Sweden as a dressing to reduce fungal attack on seed grain, and was picked up by seed-eating birds, including pheasants, at seeding time. Numbers of seed-eating birds died and there were widespread declines in populations of birds of prey which fed on the seed-eaters, because mercury is another chemical which becomes concentrated in food chains. This led to the eventual prohibition of mercury as a seed dressing. Mercury, used to control fungal growth on wood pulp, contaminated rivers below pulp mills, and this and other industrial sources led to food-chain concentrations in fresh water and coastal areas, so that commercially caught fish, especially those at the ends of long food chains such as northern pike, contained levels dangerous for human consumption. Many commercial and sports fisheries were closed, a serious economic loss in a country that consumes a great deal of fish. These aquatic concentrations of mercury also led to sharp declines in the populations of fish-eating birds.

For several years we have known that a large proportion of the grain sown in Canada is treated with mercurial fungicides, also that mercury is used in some pulp mills and in certain other industries in this country. Because of the similarities between Canadian and Swedish uses, CWS established a research project in 1968 to study possible side effects in Canada.

After research into the current uses of mercury in Canada, CWS made a preliminary survey of wildlife most likely to be contaminated, judging by Swedish experience. That survey, in the early summer of 1968, included seed-eating birds and birds of prey in the western prairies and was dovetailed with a CWS study of the effects of pesticides on prairie falcons and other raptorial birds.

Samples of biological material collected were analyzed by the neutron activation technique developed in Sweden and established internationally as the most sensitive method available. The utmost accuracy and sensitivity was necessary because very small amounts of mercury are known to occur naturally in the environment. Some residues were expected, and indeed found, in all samples. Background levels were determined on the basis of the lowest residue determination from untreated areas. Some measure of these levels was needed in order to identify additional artificially introduced contamination. It was, however, recognized that high levels of mercury contamination would be a serious hazard regardless of the source.

Background levels for mercury in western Canadian animals ranged from 0.008 to 0.035 ppm. Presumably, therefore, higher concentrations in tissue samples resulted from contamination by unnatural sources.

The results of the preliminary survey arrived in late March, 1969, and showed that mercury is being significantly concentrated in prairie seed-eating birds and rodents, and is being then transferred to birds of prey to the extent that it apparently is causing hatching failures. Both DDT and mercury appear to be involved in the present major population declines in certain birds of prey, for the DDT involvement had already been identified by CWS.

Where possible, samples of seed-eating birds and mammals were collected from treated and untreated areas in order to es-

tablish whether there was any correlation between mercury residues and the use of mercurial fungicides in western Canada. Although the number of samples was small, the results established a direct correlation between high mercury residue levels in organisms and areas of mercurial seed treatment.

It should be stated, however, that the actual amount of mercury added to the environment by seed treatment is small when compared with industrial sources. A wildlife problem develops from the seed treatment mainly because birds and seed-eating animals will eat treated grain in farming areas when other food sources are scarce.

Among the seed-eating birds in the 1968 survey were eight hungarian partridges and eight pheasants. Their livers were examined and, by extrapolation, estimates were made of breast muscle concentrations. Many of those birds were judged to be unsafe for human consumption, based on criteria published by Sweden, the Food and Agriculture Organization (FAO) and the World Health Organization (WHO). CWS biologists brought this to the attention of the Director of the Fish and Wildlife Division, Alberta Department of Lands and Forests. Consequently a joint plan was drawn up with the provincial biologists to sample birds from the major pheasant and partridge hunting areas in Alberta as late as possible in the summer of 1969.

The results of these investigations are ancient history now, particularly those results related to upland birds. The presence of high levels of mercury in Alberta pheasants and partridge collected in 1969 resulted in closure of the pheasant season. This decision was made simply because the levels were found to be higher than the actionable level of .1 ppm of the Department of National Health and Welfare, which is itself twice as high as the recommended tolerance of .05 ppm set by FAO and WHO. In one sense, it is perhaps fortunate that two species of game birds were so affected. Since these two species are very important to the sportsman and to the economy of some areas of the province, the problem of environmental contamination was brought home to everyone in Alberta. Equally high levels were found in other seed-eating birds and mammals, particularly in domestic pigeons, horned larks, and in the eggs of prairie falcons (which feed largely on the former species). However, I think that it is extremely doubtful that the same concern would have been shown about the mercury problem had we found these levels only in birds of prey and other non-game species, even though they provide a definite indication of the contamination in the environment.

Apart from the many physiological side effects which have been demonstrated, there are two most important physical side effects – the indirect effects of the supposedly non-toxic DDE on eggshell thickness in birds, and the effect of DDT on photosynthesis in plants, with special reference to marine phytoplankton.

Unexpectedly, changes in eggshell thickness have come into focus as the key indicator of the role of DDE in population declines in several species of birds throughout the world. The thin shells are due to an interference with calcium metabolism during egg laying. This phenomenon apparently results from DDE stimulating the production of hepatic enzymes which in turn break down the steroids that control calcium metabolism. The end result is thin-shelled eggs that crack or are broken or the death of the embryo because of other physiological effects. How many species have been affected? No one knows. We simply haven't been able to check every species. However, population declines, thin-shelled eggs, and a decrease in nest success asso-

ciated with high levels of DDE have been recorded in falcons, eagles, hawks, pelicans, ibis, herons, grebes and recently in loons. All are species at the ends of long food chains and, if the peregrine is any example, their numbers could decline sharply and entire regional populations, or even whole species, become extinct.

What about species in the prairie provinces? Unfortunately high levels of DDE have been found in the eggs of our raptors and fish-eating birds, and changes in eggshell thickness have been documented in three distinct species in our first three years of sampling in western Canada.

In the Alberta and Saskatchewan prairie falcons, our investigations show that the eggs of these birds have decreased in average shell thickness by approximately 11 per cent when compared with samples taken prior to the organochlorine era. By comparing known population data, we know that numbers of breeding pairs have declined by approximately 34 per cent. In one area of Saskatchewan the prairie falcon and pigeon hawk populations have apparently disappeared. In other areas we have documented inverse correlations of eggshell thickness with DDE levels, and of insecticide residue levels and nestling success (in other words we have shown that prairie falcon eggs with the highest residue levels had the lowest nest success). With regard to eggshell thickness, the eggs with the thinnest shells (18 per cent thinner than normal) contained dead embryos. These were found in southern Saskatchewan. Obviously the picture with regard to these birds of prey is dark indeed. However, one bright spot in our results was the discovery of marked variations between regions, indicating that some local populations have very low levels of insecticides and are reproducing satisfactorily. Hopefully, if, as it appears, the main decimating factors are pesticides and the levels of these contaminants can be decreased, then the isolated birds which remain will act as a reservoir to re-establish the species in areas where they once lived.

The second physical side effect is one of the greatest threats to our global environment—the effect that pesticides have on the photosynthetic capabilities of marine phytoplankton. You may wonder how this could be significant, but I think you may draw some terrifying conclusions from three simple facts:

1 Phytoplankton is the essential base of marine food chains.

2 It is responsible for more than half of the world's photosynthesis, and produces an estimated 70 per cent of the total oxygen in the world.

3 It has been clearly shown that a few parts per billion of DDT can result in a decrease in the photosynthetic activity of some species of marine phytoplankton.

It doesn't take much imagination to see what will happen if we interfere with or destroy our single biggest source of oxygen and at the same time pollute the air that is left until it isn't fit for breathing.

We've talked a lot about pollution, its causes and effects, and now we must ask ourselves who is responsible?

The answer to this question is an easy one—although perhaps not easy for us to accept. You and I are responsible for pollution, as is anyone who sprays or treats his lawn or garden for weeds or pests; or who pours phosphate detergents down the drain; or who drives a car with a combustion engine. It is all very easy to pass the buck and blame agriculture, pulp mills, forestry, and a myriad of other industries, forgetting that an industry is nothing more than a composite of individuals with a common interest. I don't say that the industries aren't responsible for pollution; I'm saying that the true blame rests squarely on the shoulders of every one of us, both collectively and as individuals.

In conclusion if it appears that I have been singling out and attacking only one industry it was not intentional. If I have been attacking anything, it is all untested and unjustified use, together with the continued defense of widespread applications, of the broad spectrum persistent biocides which are affecting this environment: our environment and that of our children. If the blame rests with agriculture, forestry, wildlife management, or the homeowner let us admit it and get rid of the fence so that we can work together to eliminate the problem.

For some reason we try to set ourselves apart even though it is often said that man is a product of his environment. Certainly, if you stop to think about it you will realize how dependent we are on our air, our water, our soil and even our soil organisms. It stands to reason that anything that changes this environment will have a profound effect on man.

Pesticides, wildlife and man*

by J. A. Keith

The word "wildlife" carries the delightfully misleading implication that it is fundamentally distinct from the "tamelife" of ourselves and our domestic animals, as if the basic biology of the two groups differed. But don't be misled by our contemptuous language as an ecologically dominant species, we are subject to the same laws of physiology and population dynamics as our wild relatives. And a comforting evolutionary aside is that there is no such thing as a *permanent* ecological dominant.

As far as pesticides are concerned, this point of view is so nicely borne out that it is sure to be a textbook example. We find it extremely difficult to make poisons with a really comfortable separation of toxicity between ourselves and those animal forms we want to dominate, and just that difficulty is a strong proof of physiological unity. Some of us in Canada were embarrassed recently to discover that dieldrin moves in human food chains, from soil to plant to herbivore to carnivore, embarrassed because it had been assumed that that sort of transfer only happened in wildlife ecosystems, somehow the rules would be different for us. And again it seems to shock many people that the milk of cow-eating humans is much higher in organochlorine insecticides than the milk of the cows themselves, as if selective retention of fat-soluble toxicants was some queer thing that only robins do, or that food-chain concentration was the prerogative of peregrines.

Starting with Konrad Lorenz and now with Robert Ardrey's *The Territorial Imperative* it is becoming obvious that many of our basic behaviour patterns have genetic components derived from our evolutionary background, and in the same period of the last forty years, ecology has become the dominant motif of our view of our place in the biosphere. This is the trend of contemporary biology, the belief in special status for humans is a residue of a religious past.

The pesticides with the most serious impact on our biological environment have been mercurial fungicides and organochlorine insecticides. In Sweden, widespread deaths of seed-eating birds, and major population declines of birds of prey feeding on seed-eaters, were caused by mercury-treated seed grain. Water-borne mercury from fungicides used in pulp mills and from other industrial sources has concentrated in fish to the extent that major Swedish inland and coastal fisheries have been closed because of the hazard to human health. This fish contamination has also led to population crashes of fish-eating birds, which does, incidentally, underline the hazard to fish-eating people.

In the Canadian Wildlife Service we are now carrying out preliminary work to assess the hazard of similar uses of mercury in Canada. We are late on the scene, as usual, but, unlike Sweden, no obvious major wildlife disasters have occurred in Canada to galvanize action.

Nevertheless, as if to underline the homily that no data is not the same as a negative answer, our preliminary work is showing that mercury is indeed being concentrated from treated grain into prairie seed-eating birds, and then transferred to birds of prey to the extent that it now looks as though *both* mercury and DDE are responsible for the current population declines of Canadian bird-eating falcons and hawks.

The organochlorine insecticides have caused widespread deaths and major population crashes of birds both in Britain and in North America, dieldrin has been the predominant cause in Britain and in North America the short-term spectacular kills have been caused mainly by dieldrin and heptachlor in large-scale insect "eradication" schemes, although DDT in Dutch elm

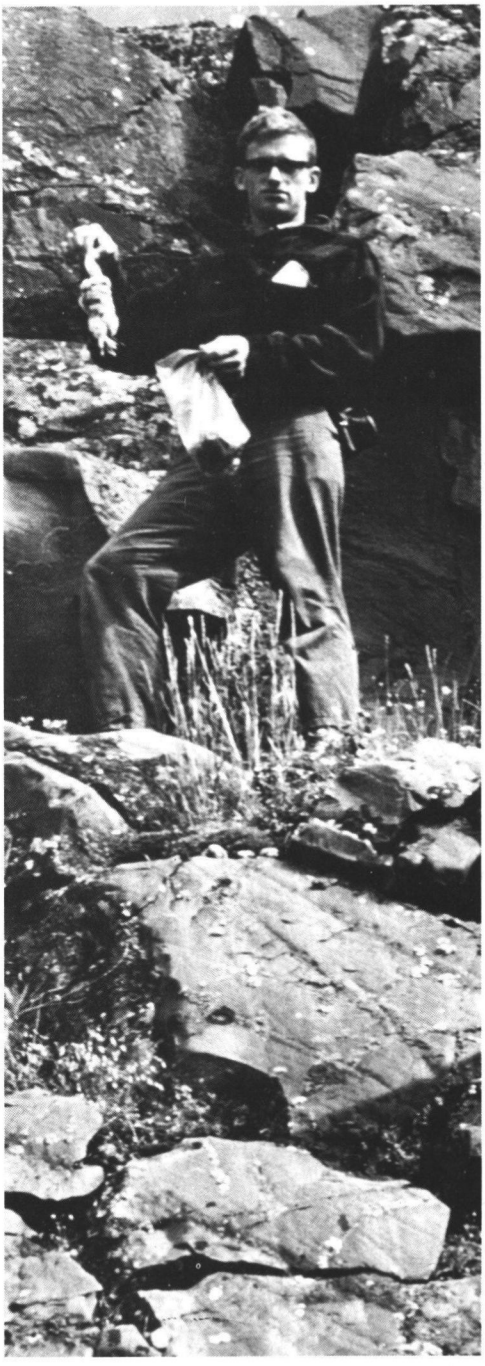
*Presented at the 17th annual meeting and conference of the Canadian Agricultural Chemical Association, Mont Tremblant, 10 September, 1969.

Keith Hodson, a CWS summer assistant, holding up dead young from peregrine nest. Photo by Richard Fyfe.

disease control was responsible for prompt and severe local losses of urban birds. Apart from these short-term spectaculars, whose spray-program origins were obvious, the really intractable problem now is the extraordinarily widespread and uncontrollable contamination of the biosphere with DDE. This contamination is so widespread in biologically significant quantities that one is at a loss to identify its origins in particular programs. Obviously, huge DDT airspray programs, such as the recent New Brunswick episode, can put massive quantities of DDT into atmospheric circulation immediately. Equally obviously, the bulk of the ground-level use of DDT can be found in the soil for years after application, but this may merely mean its dispersion into the biosphere at large is drastically slowed down, not halted. For those DDT residues in agricultural soils *do* decline, and if most of the decline takes ten years, most of the DDT applied to soil in the 'fifties cannot now be found there.

Recent work on air, rainwater, and contamination of the ocean surface, strongly suggests that atmospheric movement of DDT residues is at least as important as the erosion of contaminated soil particles by water. Before this evidence of air transport, with water transport the only hypothesis, it was no wonder that reports of DDT from remote areas in the oceans or Antarctica were greeted with incredulity.

Of course, to a large extent our choice of toxic materials to study for environmental side effects is dependent on the chemists' ability to trace their residues. Where this is possible, as with DDT and mercury, the studies have produced a fistful of surprises. Who would have believed that by going quietly about dusting and spraying insects with DDT that one was also letting loose a biospheric pollutant that now threatens the productivity and safety as human food of the great ocean fisheries? But we obviously



cannot afford many more surprises on the DDT or mercury scale with a human population food crisis arriving within the circulation span of these toxic materials. So surely the first way to avoid these environmental surprises is to be absolutely sure we know the complete chemical fate of a toxic material before we release it. Our society is, with its usual carelessness, already ignoring this obvious point, with the current introduction of stable organophosphate insecticides and stable herbicides. Stability of a toxicant is obviously highly useful in many pest control programs, but it has caused and will cause ecological disaster if it is coupled with any ability to be transported. This is where we really must look at long term costs and benefits. Does the economic benefit gained by a persistent rather than a short-lived toxicant even begin to be worth the cost of environmental degradation on the scale that we have seen come to reality with DDT and mercury? Obviously not.

The procedure now established and functioning for government regulation of the sale or use of pesticides stems from the era when the only likely hazards were thought to be to human users or consumers and when the proliferation of materials with similar functions was to be encouraged because of the increased flexibility afforded to the users. Test procedures with laboratory animals were established, and in general their success as mimics of the human condition is attested to by the astonishing record of human safety achieved in the last twenty years of universal pesticide use.

That era is over. It is clear that the techniques of human toxicology are keeping pace with the introductions of new classes of toxicants, and while the human health question must continually be watched, the situation is under control. The new era began about five years ago when the potential of environmental pollution by pesticides was first widely understood. Since then, more and more weight has been added to that potential by the sort of work on organochlorines and mercury I was describing just now. But in this new era, when environmental problems of pesticides look much more serious than human health problems, we are still approaching regulation with the old concepts and the old techniques.

In fact, of course, environmental biology is in its infancy compared with human medicine, and already it is obviously useless to tackle environmental toxicology with the concepts of human toxicology, LD 50's did not warn us that DDT would eliminate peregrines. On our part, we biologists must come up with conceptually sophisticated techniques for measuring a pesticide's environmental impact, and it may take several more years before much progress is made. On the government's part, the shift in emphasis from human health to biosphere health must be reflected in the regulatory procedure. On your part, as distributors of pesticides, among them persistent materials with the demonstrated potential to make fantastically large changes in the biosphere, you should be aware of the ecological lessons learned so far, particularly the risks of stable toxicants.

To return to my opening theme: The wildlife-pesticide problem is not a question of the trivial hobby of little-old-lady-bird-watchers getting in the way of progress in the production of food and fibre. It is a question of all of us learning how to manage the resources of our unitary biosphere without wrecking the very productivity or usefulness of those resources we will soon need so desperately.

Toxic chemical research by the Canadian Wildlife Service*

by J. A. Keith

Since 1969 the Canadian Wildlife Service mercury project, which was undertaken to look at wildlife hazards of both agricultural and industrial mercury, has established that agricultural mercury used on seed grain is a significant contaminant of prairie seed-eating animals, and that the mercury is then transferred to their avian predators in sufficient concentrations to cause hatching failure. The project has also established that the Canadian chlor-alkali and pulp industries were releasing enough mercury into water systems to cause significant contamination of fish and probably hazardous concentrations of mercury in fish-eating birds.

Our mercury project includes a number of studies, being done both by a university contractor and our own field staff. Our contractor has done penned studies to establish the effects of mercury-treated grain on seed-eating bird reproduction, using pheasants, and the effects of the transfer of mercury through seed-eating birds to avian predators, using red-tailed hawks. Results from these penned studies have been used to interpret the biological significance of our surveys of mercury residue levels in wild populations. From the survey data interpreted this way we hypothesized that certain local populations of predators were likely to have reproduction chronically impaired by mercury. So in the summer of 1970 we tested this hypothesis in New Brunswick with terns and cormorants, and in Saskatchewan with herring gulls.

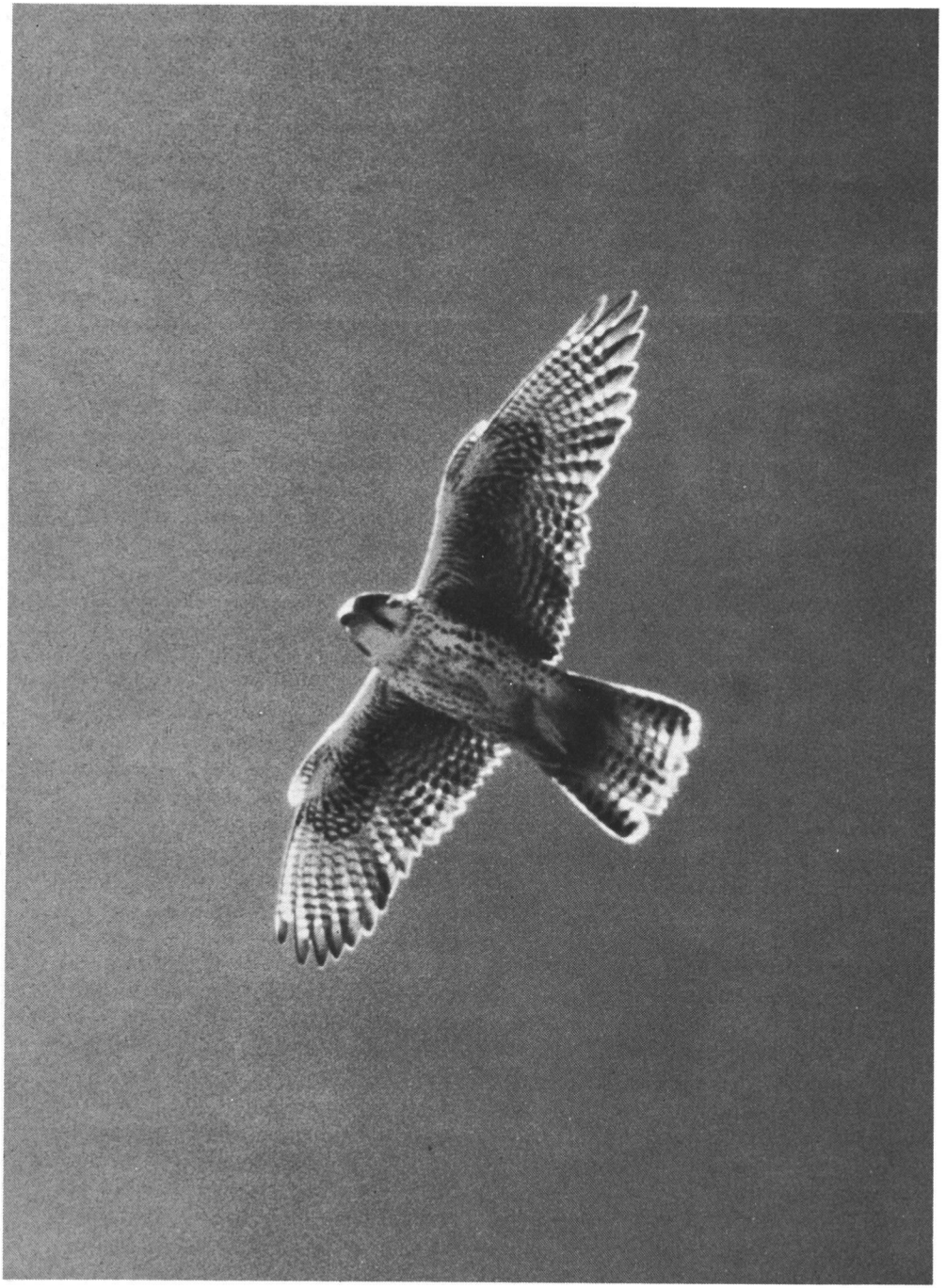
Early results from our mercury project led to a lot of activity by other agencies. The Alberta surveys of mercury in pheasants and partridges precipitated closing the 1969 hunting season and federal and provincial surveys of mercury in commercial fish have resulted in a large number of commercial fishing restrictions from coast to coast. This activity has also spread south into the United States where mercury contamination had not before been seriously considered.

The commercial fishing restrictions have been accompanied by encouragingly rapid restrictions on the release of mercury into industrial waste water, and so we can now expect that mercury levels in the exposed acquatic environments will begin to decline. Judging from the Swedish experience, however, significant reduction in these mercury levels may take a long time, and so we must continue to look for hazardous mercury levels in aquatic wildlife populations for a number of years.

On the other hand, the closing of the pheasant and partridge season in Alberta did not lead to elimination of the seed-treatment source of mercury. Unlike the aquatic situation, the end of mercury seed treatment will be followed by a very rapid reduction in mercury contamination of terrestrial wildlife, probably reaching background levels within a year. This is because the mercury is directly applied to a wildlife food, and is not reaching wildlife by way of general contamination of the environment, as in the aquatic case. Some efforts have been made to reduce the mercury-treated grain spilled around farms, treatment plants or along roads, but we cannot expect the mercury hazards to seed-eaters and their predators to be over until at least the most toxic alkyl mercury seed treatments are eliminated. The alkyl mercurials were not eliminated in 1970 and will not be before 1972 because of the agricultural community's fascinating logic of maximizing grain yields at a time when the surplus on hand is so embarrassing that grain acreage is being reduced. The choice, of course, is not all or nothing, either continuing mercury contamination or no grain crop. Immediate banning of alkyl mercurials would leave other fungicides available, even if they are

*Presented at the 34th Federal-Provincial Wildlife Conference, Yellowknife, July 14, 1970.

Local declines in prairie falcon populations seem definitely related to toxic chemicals. Photo by Richard Fyfe.



less handy or efficient, and anyway the average increase in yield due to fungicide treatment seems to be only about 10 per cent or less. The Scandinavian countries solved their serious mercury contamination from seed treatment by simply banning alkyl mercurials.

Mercury contamination is not the only wildlife problem that results from treating grain seeds. Persistent organochlorine insecticides are routinely used in many areas to combat wireworms, and of these heptachlor and aldrin/dieldrin are of serious concern. High proportions of our prairie raptorial bird samples contain residues of these insecticides, and at least one set of prairie falcon nestlings was killed by heptachlor. It was seed-grain treatment by heptachlor and aldrin/dieldrin that caused such severe mortality among seed-eating birds in Britain between 1956 and 1961. From seed-eaters these insecticide residues were transferred to birds of prey and helped cause widespread population declines. Since restriction on aldrin/dieldrin seed treatments were introduced in the early 'sixties, populations of some of these birds of prey have begun to recover.

Here in Canada the relatively high residues of heptachlor and aldrin/dieldrin in prairie raptors almost certainly originate in seed-grain treatments.

We are now faced, then, with a multiple contamination of western birds of prey. Our model population under study is the prairie falcon, and it is being stressed by residues or metabolites of DDT, aldrin/dieldrin, heptachlor and mercury. The relative contribution of individual compounds is very hard to untangle, but at the least their effects are additive. The local declines within the prairie falcon population seem definitely related to toxic chemicals rather than such other factors as human disturbance, and correlate best with increased levels of DDT metabolites, but the other compounds are contributing to mortality of eggs or nestlings.

The DDT picture in Canada has changed sharply for the better during 1969-70. Wildlife data was very important in the review of all DDT uses during 1969, and the November announcement by the Canadian prime minister of a 90 per cent reduction in the amount of DDT used, starting 1970, was based on both definite harm to wildlife and inconclusive evidence of hazard to people.

Our argument for such broadscale reduction of DDT use was based on the inability to pinpoint individual uses that have caused the all-pervasive DDT contamination. For example, we cannot identify a particular prairie use of DDT that causes the serious contamination of prairie falcons. We are then forced to conclude that the level of this chemical as a general constituent of the environment has reached the point where some avian predators can no longer survive.

Just because use of DDT is now restricted in Canada, our DDT problems are not over. The United States DDT situation is still unclear, and while we expect reductions in use soon, we have no definite idea of their extent or timing. This is, of course, a crucial question for migrant birds and their predators, because it is through eggs that DDT does its population damage, and so DDT levels in spring migrants into Canada are not going to decline as fast as DDT levels in birds resident in Canada. In terms of general airborne contamination of DDT in the northern hemisphere, the Canadian restrictions and the recently announced restrictions in manufacture and use in Russia will hopefully result in declining residues in our general environment.

Whether these DDT reductions will come soon enough to save the collapsing continental peregrine falcon populations is an open question. This bird is being stressed by the same range of chemicals that are found in the prairie falcon with the addition

of a larger amount of PCB residues. These residues are from industrial compounds and show the same flair for widespread distribution and food-chain concentration that DDT does, and their possibly similar role in interfering with egg hatching is now under active research.

For the immediate future, we must pay close attention to wildlife contamination that we think comes from the DDT and mercury uses now being restricted. This may seem a waste of time, but in fact this period is precisely when we can best test our causal hypotheses. Given wild populations subject to a variety of chemical and non-chemical stresses, we have gone out on a limb and said that just a few chemicals are much more important as stresses than anything else. If these chemicals are sharply reduced, and other factors stay roughly the same, a positive response by the wild populations is a convincing test of our original hypothesis.

Next in terms of research priority comes the group labelled PCB, the industrially used polychlorinated biphenyls. We are beginning to learn something of PCB distribution in wildlife populations, and if the rough quantification methods now used are any good, PCBs now occur in many places in concentrations similar to our traditional benefactor DDT. The key research question is biological significance. The evidence so far is conflicting; some research suggests that PCBs are as active enzyme inhibitors as DDT, and some results show a DDT-like effect of PCBs on eggshell thinning. Other research fails to show significant PCB effects on eggshell thinning or hatchability. But the possibility is definitely open that PCBs are as ecologically damaging as the DDT group.

Looking at the problem of trying to screen out dangerous chemicals before they are introduced into the environment, rather than trying to gauge the damage already done, we come up with a very urgent need for research. If you ask, how does a certain toxic chemical behave ecologically, we don't know how to answer. We don't know what would constitute a useful ecological test, and yet we urgently need to work out such tests if we are to regulate intelligently the entry of toxic chemicals into the environment. This is a problem without an instant answer; it will take several years of hard ecological thinking to develop adequate screening tests, and we need the cooperation of every Canadian ecologist who cares to help. We are starting this with a staff position and some university contracts, but we invite any help or suggestions we can get.

Some results and implications of cws pesticide research*

by J. A. Keith

What sort of things has the Canadian Wildlife Service been finding out in the last few years in terms of environmental contamination by persistent pesticides?

The persistent pesticides we know most about are the organochlorine insecticides, especially DDT and dieldrin, and since Canadian uses are usually not radically different from uses in the United States or northern Europe, it is not surprising to learn that there are important similarities between Canada and these countries in the environmental movements and concentration points of this group of insecticides. When we started a few years ago to probe into Canadian wildlife populations we expected, on the basis of European and American work, to find that top carnivores would be concentration points, and that lower trophic levels would have proportionately lower levels of contamination, and this does prove to be the case.

For example, in a series of 15 bird species taken at the end of a winter on the Fraser River delta, or near its shores, the organochlorine contamination of the two hawk species was at least six times higher than that of the others, followed in descending order by owls, herons, shorebirds, passerines, gulls and waterfowl. In Alberta and Saskatchewan, a survey of eggs of 13 species of falcons, hawks, eagles, and owls shows that those that eat birds are more contaminated than those that eat mammals, and collections of their bird and mammal prey show correspondingly lower levels of contamination. In another prairie egg survey, in aquatic systems, waterfowl eggs did not exceed two parts per million (ppm) organochlorine residues, while eggs of gulls and exclusively fish-eating birds ranged between 2 and 26 ppm. In New Brunswick forests where DDT has long been used, the herbivorous showshoe hare and white-tailed deer contain really low DDT residues, but bobcats contain many times higher levels, and mice and voles are less contaminated than shrews. In 1970 we found DDT levels averaging 56 ppm in woodcock from heavily sprayed areas of New Brunswick. Samples of woodcock were taken throughout the province, with the co-operation of the New Brunswick Fish and Wildlife Branch, before the 1970 – 71 hunting season opened. The discovery of such high DDT residues led to cancellation of the hunting season on these birds. This step was taken after consultation with the Department of National Health and Welfare, whose highest tolerance for human food is seven ppm. In a look at resident northern animals, caribou fat averaged around 0.1 ppm organochlorine compounds whereas polar bear fat averaged twenty-five times higher.

Many of the organochlorine residue levels are sufficiently low that it does not seem worth doing more than keeping them under routine observation. This is the case for western waterfowl as a group and for herbivores generally. But, in the cases where residue levels are high, further research is seriously required to assess hazard or damage to species populations.

For this reason, we are actively studying bird-eating and fish-eating birds in the west. Early results show truly astonishing regional within-species variations in residue loads, and these require explanation, presumably in terms of local pesticide-use patterns and local food preferences. For bird-eating birds, where we now have some data on reproduction and on yearly changes in breeding-pair numbers, we can come to some conclusions on effects. As a model for this group, we are concentrating on the prairie falcon in southern Alberta and southwestern Saskatchewan, and here for this bird there has been a substantial decline in occupied territories during the past

*Presented at the 33rd Federal-Provincial Wildlife Conference, Edmonton, July 8, 1969.

decade. In our sample the drop has been one-third. The decline does not include all areas, but it looks as though it is continuing. There are inverse relationships between DDE levels and both eggshell thickness and nestling production.

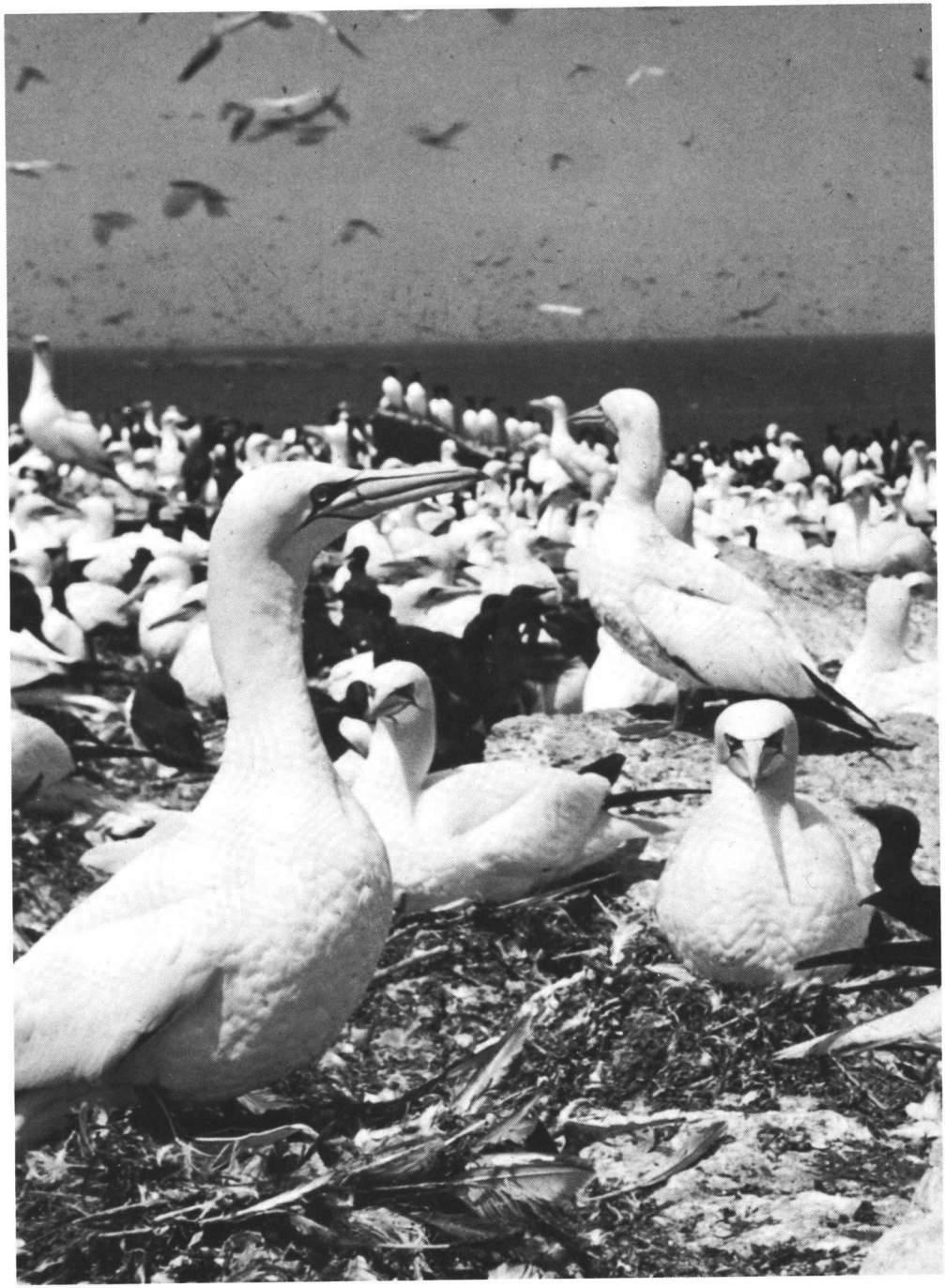
It is widely known that peregrine falcon populations have collapsed in the settled portions of North America in recent decades, but only in the last few years has a connection with pesticides been more than speculation. We are documenting contamination in this bird and its prey in the northern parts of its former breeding range, and we are also following production at a series of eyries in the Thelon and Bathurst areas. In an obviously limited set of samples, egg contamination by organochlorine insecticides in this bird is as high in arctic breeders as it is further south. This may simply reflect a floating situation in which a predator moves north and south with an avian prey in which contamination may be highest during winter and during spring migration. Certainly for effects on eggs, it is the peregrine's body load in early spring that counts, not the latitude of the nest.

Atlantic gannets in Canadian waters, feeding on the mackerel and herring populations that are exploited by human fishermen, are grossly contaminated with organochlorine insecticides, whole-egg levels ranging now between 8 and 100 and averaging about 30 ppm on Bonaventure Island off the Gaspé. This is not as high as the egg levels found in a Lake Michigan herring gull population with abnormally low hatching success, but it is higher than egg levels in the declining populations of peregrines and prairie falcons. Poulin's recent study of the Bonaventure gannets shows breeding success to be only a half that of a Scottish colony which almost certainly has much lower contamination levels, and that hatching success in particular is low on Bonaventure. While insecticides probably are contributing to this situation, the degree of contribution remains to be worked out. The colony on Funk Island, east of Newfoundland, and its summer food are half as contaminated as at Bonaventure, and a very rough census suggests no abnormal drop in productivity.

While organochlorine insecticides are, then, obviously important wildlife contaminants in Canada, there are other toxic chemicals that deserve much more attention than they have had. Since the disastrous Swedish experience with organomercurial fungicides used on grain seeds and on pulp during paper making, we have been trying to start a project to assess the mercury hazard here, where uses are similar. We have finally got such a project underway and results from the prairies suggest that some seed-eating birds taken from areas where mercury-treated seed is used do contain much higher mercury levels than could be expected from fields without mercury-treated seed. Some raptorial birds are, also, concentrating mercury at hazardous levels.

Another group of materials worth immediate attention is the group labelled PBC, the industrially-used polychlorinated biphenyls. These have come to attention because they are chemically similar to DDT and have often been misidentified as DDT. The PCBs have recently been shown to be in the same league as DDT and DDE in breaking down steroid hormones. The chemist who does our analytical work has devised a technique for separating most PCBs from pesticides residues, and so we are beginning to get some idea of the distribution and abundance of PCBs in wildlife samples. The PCBs are most apparent in our marine and Great Lakes samples, in polar bears around Hudson's Bay, in breeding seabirds from both the Atlantic coast (puffin, Leach's petrel, common murre, and gannet) and from the Pacific (ancient murrelet), and in

PCBs are apparent in the gannet from the Atlantic coast. Photo by Leslie M. Tuck.



ring-billed gulls from the Great Lakes. When present, PCBs can account for all of the apparent DDD and much of the p,p'-DDT, but the DDE values are hardly changed at all by PCB separation.

What are the implications of these and similar results of research into toxic chemicals in wildlife? Should we be content with the conventionally safe and proper wildlife management role of doing research and providing information? Or should we be serious about wildlife *management* in this field and consider toxic chemicals as population limiting factors and aim to reduce those uses that limit wildlife numbers?

Pursuit of this innocuous-sounding aim, reducing toxic chemical uses that affect wildlife, involves to a surprising degree an open attack on what is now called progress in agriculture, forestry, outdoor recreation and human population growth, and an open attack on motherhood *is* at first glance a surprising involvement for the wildlife profession. But trying to change agricultural or forestry practice, or to alter trends in human population growth, only differs from controlling wetlands in degree, not in principle, the principle being that environmental biologists do not just study their subject and passively react to changes in environmental stress, but apply their research results actively to moderate those stress factors that prove critical.

This leads us, then, into rather fundamental considerations, for the increasing man-made environmental stresses, such as chemical pollution, are caused by the continuing growth in both human numbers and per-capita environmental demands, and to moderate these stresses, someone obviously must tackle their causes. But is it really the business of biologists to take to the hustings as social critics, would we know what we were talking about? In the 1940's and 1950's the atomic scientists found that their special knowledge had the most catastrophic social implications, and this led them to a vigorous effort to inform society of these implications. We have this sort of special knowledge now. More than any other group in Canada, our knowledge of ecology and population dynamics gives us special insights into the catastrophic environmental consequences of present trends in the expansion of human populations. Because of this, environmental biologists have a deadly serious obligation to be articulate, persistent, and *public* critics of those trends in society which we know to have disastrous implications.