Pesticides and the Reproduction of Birds

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High concentrations of chlorinated hydrocarbon residues accumulate in such flesh-eaters as hawks and pelicans. Among the results are upsets in normal breeding behavior and eggs too fragile to survive.

The birds of prey have had an uneasy coexistence with man. Apart from the training of certain hawks for falconry and the veneration of the eagle as a symbol of fortitude, the predatory birds have been preyed on by the human species. In many parts of the world farmers, hunters and bird-lovers have waged unceasing warfare on the rapacious birds as pests, and egg collectors have further threatened their survival by raiding their nests for the beautifully pigmented eggs. Nevertheless, over the centuries the birds of prey on the whole survived well. The peregrine falcon, for example, is known to have maintained a remarkably stable population; records of aeries that have been occupied more or less continuously by peregrines go back in some cases to the Middle Ages.

About two decades ago, however, the peregrines in Europe and in North America suddenly suffered a crash in population. The peregrine is now rapidly vanishing in settled areas of the world, and in some places, particularly the eastern U.S., it is already extinct [*see illustration, page 256*]. The abrupt population fall of the peregrine (known in the U.S. as the duck hawk) has been paralleled by sharp declines of the bald eagle, the osprey and Cooper's hawk in the U.S. and of the golden eagle and the kestrel, or sparrow hawk, in Europe. The osprey, or fish hawk, has nearly disappeared from its haunts in southern New England and on Long Island; along the Connecticut River, where 150 pairs nested in 1952, only five pairs nested in 1969.

The population declines of all these raptorial birds are traceable not to the killing of adults but to a drastic drop in reproduction. It has been found that the reproduction failures follow much the same pattern among the various species: delayed breeding or failure to lay eggs altogether, a remarkable thinning of the shells and much breakage of the eggs that are laid, eating of broken eggs by the parents, failure to produce more eggs after earlier clutches were lost, and high mortality of the embryos and among fledglings.

Examination of the geographic patterns suggests a cause for the birds' reproductive failure. The regions of population decline coincide with areas where persistent pesticides—the chlorinated hydrocarbons such as DDT and dieldrin—are widely applied. Attrition of the predatory birds has been most severe in the eastern U.S. and in western Europe, where these pesticides first came into heavy use two decades ago. Analysis confirmed the suspicions about the pesticides: the birds were found to contain high levels of the chlorinated hydrocarbons. In areas such as northern Canada, Alaska and Spain, where the use of these chemicals has been comparatively light, the peregrine populations have remained normal or nearly normal. Recent studies show, however, that even in the relatively isolated North American arctic region the peregrines now have fairly high levels of chlorinated hydrocarbons and their populations apparently are beginning to decline.

The birds of prey are particularly vulnerable to the effects of a persistent pesticide such as DDT because they are the top of a food chain. As George M. Woodwell of the Brookhaven National Laboratory has shown, DDT accumulates to an increasingly high concentration in passing up a chain from predator to predator, and at the top of the chain it may be concentrated a thousandfold or more over the content in the original source [see "Toxic Substances and Ecological Cycles," by George M. Woodwell; SCIENTIFIC AMERICAN, March, 1967]. The predatory birds, as carnivores, feed on birds that have fed in turn on insects and plants. Hence the birds of prey accumulate a higher dose of the persistent pesticides and are more likely to suffer the toxic effects than other birds.

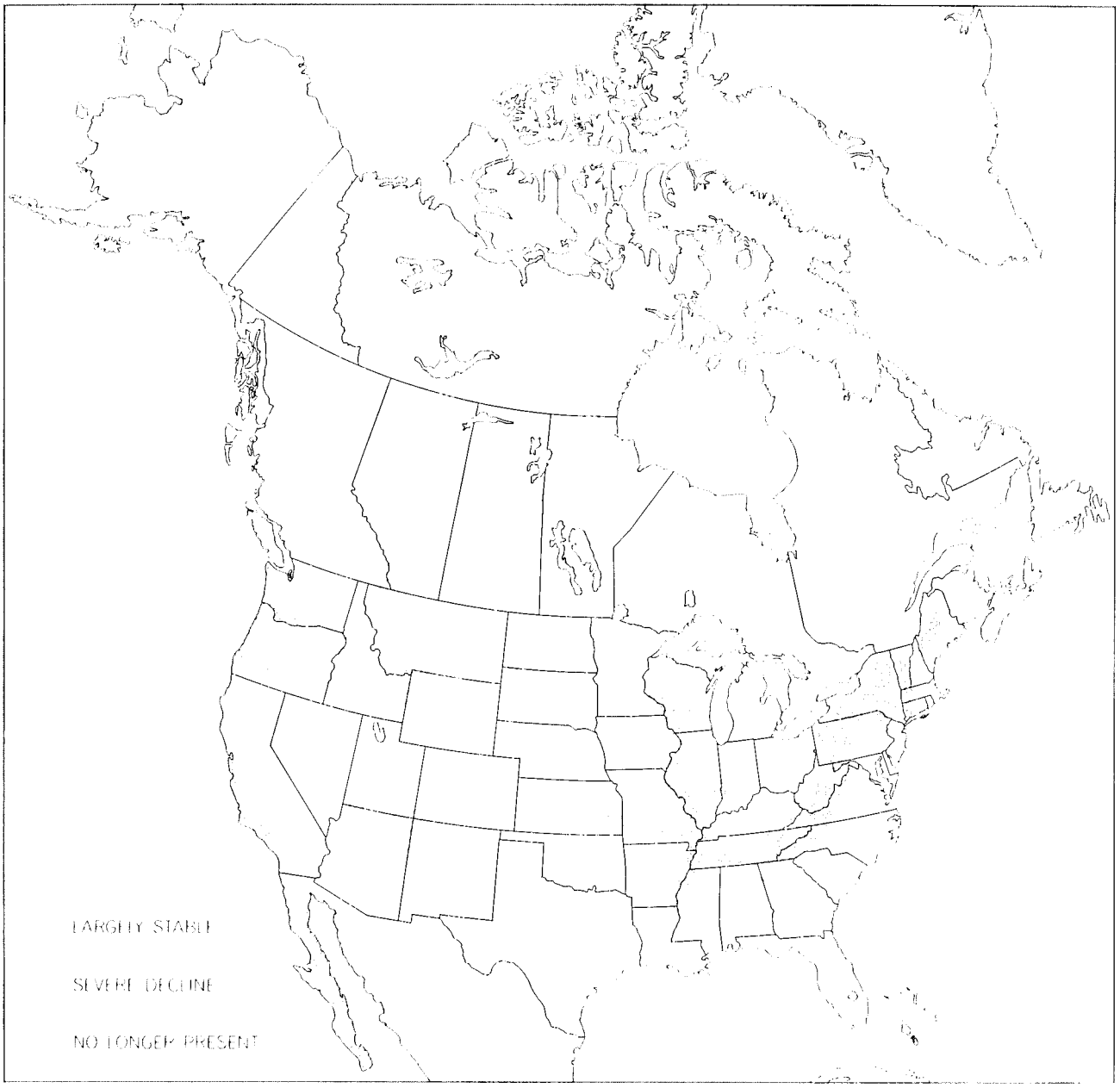
The idea that the predatory birds' decline is due to an internal toxic effect, rather than to a change in their behavior or their habitat, has been verified by many experiments. One of the most interesting was a field test made by Paul Spitzer, now at Cornell University, working in cooperation with the Patuxent Wildlife Research Center in Maryland. He transferred eggs from nests of the failing osprey population in New England to nests of a successful population in the Chesapeake Bay area and placed the Chesapeake eggs in the New England nests. The Chesapeake eggs hatched as successfully in the New England nests as they would have at home with their own parents, whereas the New England eggs transferred to Chesapeake nests produced as few viable young as would have been expected if they had been incubated in their original nests in New England. The experiment thus indicated that the fate of the eggs was determined by an intrinsic factor in the egg itself.

The first clue to what was happening to the predatory birds' reproduction system came in the early 1960's when Derek Ratcliffe of the British Nature Conservancy, puzzled by the extraordinary number of broken eggs he found in peregrine nests, examined the shells of peregrine eggs that had been collected over a period of many years. He found that the eggs collected since the late 1940's show a sharp drop in thickness of the shell, averaging 19 percent. Similar findings were subsequently made

on peregrine eggs in North America and on the eggs of other species of predatory birds whose populations were decreasing. It became apparent that something must be wrong with the birds' calcium metabolism and that the effects of the suspected pesticides would bear looking into.

Experiments were started in several laboratories. At the Patuxent Wildlife Research Center, Richard D. Porter and Stanley N. Wiemeyer, working with kestrels, found that a mixture of DDT and dieldrin in doses measured in a few parts per million brought about a significant decrease in the shell thickness of the birds' eggs. Robert G. Heath of the Patuxent center tested the effects of DDE, the principal metabolic product of DDT, on mallard ducks. DDE is now a ubiquitous feature of the earth's environment; it is estimated that there are a billion pounds of the substance in the world ecosystem, and traces of it have been found in animals everywhere, from polar bears in the Arctic to seals in the Antarctic. Heath found that DDE caused the failure of mallard eggs in two ways: by increasing the fragility of the eggs, leading to increased breakage soon after laying, and by the death of the embryos in intact eggs toward the end of the period of incubation. James H. Enderson of Colorado College and his associate Daniel D. Berger, studying the eggs of prairie falcons in the Southwest desert, established that the amount of thinning of the shells and the mortality rate for the embryos were related to the quantity of DDE in the egg. Enderson and Berger also found that when they fed starlings loaded with dieldrin to falcons, the falcons' eggs showed similar thinning.

The ultimate in thinness of birds' eggshells was discovered recently in colonies of the brown pelican off the California coast. The DDE content in the eggs of this wild population (as measured by Robert Risebrough of the University of California at Berkeley) ranged as high as 2,500 parts per million, and the eggshells were so thin that the eggs could not be picked up without denting the

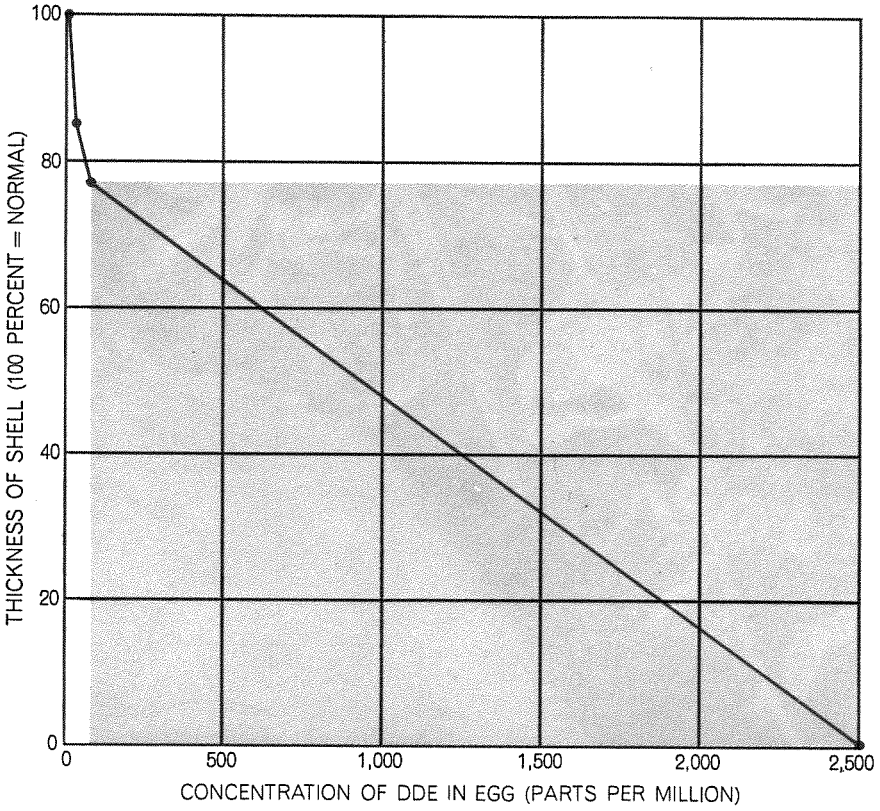


NESTING AREAS of the peregrine falcon, or duck hawk, in the Northern Hemisphere of the New World are shown on this map. Shades of color show the extent of interference with normal r production resulting from ingestion of pesticides by the bird

shells [*see illustration, page 258*]. In a colony on the Anacapa Islands off the coast it was found that the 300 pairs of nesting pelicans had not produced a single viable egg. Their nests, visited shortly after the eggs were laid, contained many broken eggs.

Field studies and laboratory experiments suggest that the thinning of eggshells does not increase in direct proportion to the DDE dose. In fact, small doses can produce dramatic effects. A content of only 75 parts per million in the egg reduces the shell thickness by more than 20 percent; beyond that, as the dose increases the decrease in shell thickness is more gradual [*see illustration at right*]. In the case of the brown pelican very heavy doses may thin the shell to a mere film.

Studies of white pelicans and cormorants have implicated the polychlorinated biphenyls (PCB's), now widely used as plasticizers, as another threat to birds of prey. These compounds cause thinning of the eggshells, although not as effectively as DDT and its metabolites do. Preliminary laboratory studies show that PCB's are particularly effective, however, in delaying the onset of breeding. The PCB's are given off when plastic materials are burned, and they are widely distributed over the earth. They resemble DDT in molecular structure and produce similar physiological actions in animals.



SEVERE EFFECT of the concentration of relatively small amounts of the persistent chlorinated hydrocarbon pesticides is evident in this graph. When the parent's concentration is enough to add as few as 25 parts per million of pesticide to the egg, the shell becomes 15 percent thinner than normal. Soon after the shells become more than 20 percent thinner than normal (*area of light color*) eggs are usually not found in nests because of breakage.

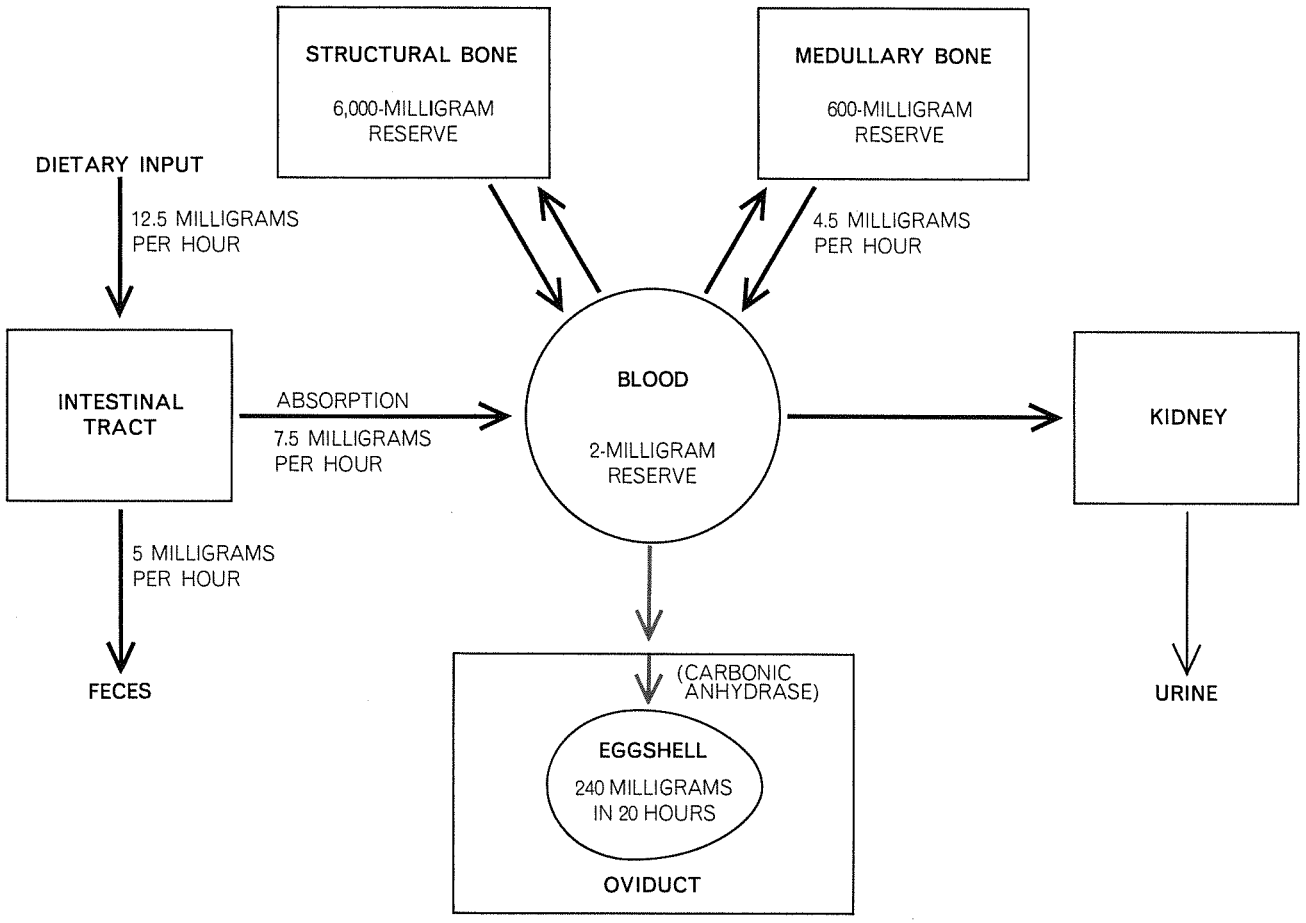
Much interest has focused on the question of how the chlorinated pesticides produce their destructive effects in the predatory birds—a question that is of no small concern to man, who also is the top of a food chain. Oddly enough, the beginning of light on this question came about through an accidental discovery involving an animal totally unrelated to the birds: the laboratory rat. Larry G. Hart and James R. Fouts of the University of Iowa College of Medicine were investigating the effects of food deprivation on the metabolism of drugs in rats. The drug they were using was hexobarbital, and in one experiment they were startled to find that the rats' sleeping time after receiving a standard dose of the barbiturate was much shorter than it had been in previous tests. Reexamining the conditions of the experiment, they found that the only unusual factor was that the cages had been sprayed with chlordane to control bedbugs. Pursuit of this clue led to the finding that chlordane induced rat liver cells to synthesize enzymes that speeded up the metabolism of hexobarbital. The enzymes brought about hydroxylation of the barbiturate, thereby making it more soluble in water and hastening its excretion. Further experiments showed that these enzymes could hydroxylate a wide variety of substances, including the sex hormones: estrogen, testosterone and progesterone.

Because the investigators were interested primarily in drug research and their reports were published mainly in pharmacological journals, these discoveries did not come to the attention of workers studying the effects of pesticides on wildlife until several years later. I myself came on the published findings only incidentally in the course of preparing lectures for medical students. The fact that chlordane could change the balance of sex hormones in animals immediately suggested a possible explanation of the mechanism whereby the chlorinated pesticides inhibit reproduction in birds. It was capable of explaining their reproductive failure in general and the alteration of the calcium balance in the egg in particular.

My colleagues and I at Cornell University launched on a program of experiments designed to explore the interesting questions suggested by this new aspect of the problem. To explain them I must briefly outline the complex chain of physiological events that characterizes breeding by birds. The cycle is initiated by a seasonal or climatic stimulus: the lengthening of daylight in spring in the northern Temperate Zone or rainfall in the arid and tropical regions. These signals cause an increase in the production of hormones in the nerve cells of the medial eminence of the bird's brain. The bloodstream carries these hormones to the anterior pituitary gland, which in turn dispatches to the gonads (the testes or ovaries) hormones that stimulate these organs to produce the sex hormones. The sex hormones not only generate physical changes in the reproductive organs and evoke breeding behavior but also promote the storage of a supply of calcium for the eggs.

Let us look first into the question of how a pesticide may affect the calcium supply. We carried out our experiments on the rather small Asian pigeon known as the ringdove, so that I shall describe the situation in this bird. The female forms the shell of the egg in the uterus within a period of 20 hours, and she needs 240 milligrams of calcium to pro-

CRUSHED EGG in the nest of a brown pelican off the California coast had such a thin shell that the weight of the nesting parent's body destroyed it. The concentration of DDE in the eggs of this 300-pair colony reached 2,500 parts per million; no eggs hatched.

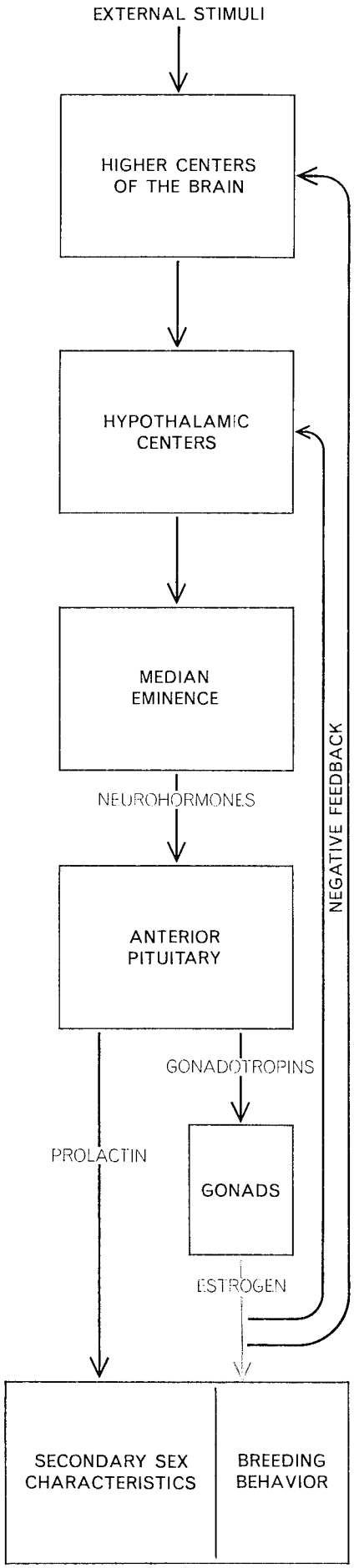


CALCIUM FOR EGGSHELL, which is formed around each egg in the last 20 hours before laying, is drawn in part from the bird's food supply and in part from calcium reserves in the bird's bones. The key to shell formation, however, is the enzyme carbonic anhydrase, which makes the supply of calcium carried in the ringdove's bloodstream available to the bird's oviduct at a rate of 12 milligrams per hour. When laying ringdoves are injected with DDE, the action of the enzyme is severely inhibited, causing thin shells.

duce a shell of normal thickness. Since the calcium content of the circulating blood, even at the time of ovulation, is only two milligrams (barely a 10-minute supply), the bird must draw on other sources to meet the demand. About 60 percent of the demand is supplied by the bird's food intake; the rest is provided by a store of calcium in the marrow of the bones [see "How an Eggshell Is Made," by T. G. Taylor; beginning on page 208]. This calcium reserve is laid down in the bone cavities early in the breeding cycle, and the amount of the deposit is controlled by the levels of estrogen in the blood and tissues. Obviously, therefore, a deficiency of estrogen will reduce the bird's calcium reserve. It seemed unlikely, however, that the reduction of this reserve alone could account for the drastic shell-thinning observed in eggs loaded with pesticides. If the *supply* of calcium were the sole problem, the birds could augment the supply by drawing on the calcium embodied in the skeleton; furthermore, birds on a very low calcium diet have been found to cease egg-laying rather than laying eggs with abnormally thin shells. Was it possible, then, that the thinness of the eggshells was due less to the deficiency in supply than to a failure in delivery of calcium to the shell?

In our experiments we bred pairs of ringdoves in cages and delayed feeding the birds a pesticide until after they had completed at least one successful breeding cycle, thereby demonstrating their natural capability. For the experiment we separated the members of each pair, isolated them in individual cages where they had an eight-hour day instead of their normal 16-hour day and fed them a standard dose of DDT in their food. After three weeks we gave each bird an oral dose of radioactive calcium and returned the birds to cages with their original partners for pairing under long-day conditions. A number of days later we examined the birds, some before they laid eggs, others immediately after they finished laying their clutch. In both cases the birds showed a considerable rise of enzyme activity in the liver. A substantially lower level of estrogen was found in the bloodstream of the birds that had not yet laid eggs. After the eggs had been laid, low estrogen levels were found in both experimental and control birds; this was to be expected because the level of estrogen falls at the time of egg-laying. We found that less labeled calcium was stored in the bone marrow of the experimental birds than in the marrow of control birds that had not been fed the pesticide.

Eggs laid by the pesticide-treated birds were notably thin-shelled, as was to be expected. We proceeded to experiments designed to determine whether this was due simply to the shortage of stored calcium or to something that prevented calcium from reaching the shell. In order to resolve this question we resorted to the tactic of injecting pesticides into females within a period of hours before they laid their eggs. In that short interval there would not be time for any significant change in the supply of calcium by way of an alteration of the



BREEDING SUCCESS in birds involves the five sequential responses to external stimuli shown in the illustration at left. Breeding failures, due to late breeding or an inability to lay more eggs after earlier clutches are destroyed, result from the action of pesticides on the fifth response. They stimulate the activity of enzymes in the breeding bird's liver; the enzymes cut the amount of estrogen in the system below the level that is needed for normal sexual behavior.

estrogen levels through the activity of liver enzymes; consequently if the pesticide produced an effect, it would be not on the stored supply but on the delivery of calcium to the eggshell, which as we have noted is laid down within 20 hours of the laying of the egg. And with regard to delivery it was known that an enzyme, carbonic anhydrase, plays an important role in making calcium available to the eggshell in the oviduct. One could therefore look for a possible effect on the activity of this enzyme.

We tried two chlorinated hydrocarbons: dieldrin and DDE. Dieldrin, when injected into a ringdove shortly before it laid its egg, did not produce any significant thinning of the eggshell or inhibit the activity of carbonic anhydrase in the oviduct. DDE, on the other hand, severely depressed the activity of the enzyme and brought about a marked decrease in the thickness of the eggshell.

Our experiments with ringdoves also showed that the chlorinated hydrocarbons cause a significant delay in breeding by birds. Females that were fed pesticides did not lay eggs until 21.5 days (on the average) and sometimes as long as 25 days after pairing, whereas the normal interval, as indicated by the control birds, is 16.5 days on the average. The delay evidently was caused by the depression of the estrogen level resulting from the induction of liver enzymes by the pesticide. It turned out that dieldrin and the polychlorinated biphenyls were more powerful inducers of these enzymes than DDT was.

Delayed breeding is another factor in the predatory birds' population decline. Most birds do their breeding in the season when food is most plentiful, thus giving their young an optimal chance for survival. An artificial delay in their breeding consequently reduces the chances for reproductive success, and it is most serious for large birds, with their long egg-incubation period and the slower growth of the fledglings to maturity. It was found that the now extinct peregrine colonies along the Hudson River, the declining cormorant rookery at Lake DuBay in Wisconsin and the failing pelican colonies in California were all notably late in breeding.

From this point of view it appears that dieldrin and the PCB's are greater threats to the predatory birds than DDT. Certain field and laboratory studies tend to bear out that deduction. Derek Ratcliffe and J. D. Lockie, in long-term observation of the nests of golden eagles in Scotland, found that although abnormal eggshell breakage began in 1952, about the time that DDT was introduced, marked decline in the breeding success of these birds did not begin until 1960, after the introduction of dieldrin. In laboratory experiments on the bobwhite quail James B. DeWitt and John L. George of the U.S. Fish and Wildlife Service found that one part per million of dieldrin was effective in reducing the success in hatching and survival of chicks, whereas it took 200 parts per million of DDT to produce the same effect. Robert Heath found in his studies of mallard ducks, however, that DDE severely impaired reproductive success at doses as low as 10 parts per million. Thus there appears to be a considerable difference in the effect of DDT and its metabolites on different species of birds.

We come to the following conclusions concerning the physiological mechanisms responsible for the various harmful effects on bird breeding that are brought about by the persistent insecticides. Abnormally late breeding and the failure of birds to lay eggs after their early clutches have been lost can be explained in terms of the induction of liver enzymes that lower the estrogen levels in the birds. The failure, or apparent failure, of birds to lay any eggs at all may be due either to depression of the estrogen level or to the circumstance that the eggs were broken and eaten by the parents shortly after they were laid, so that observers found no eggs in the nest on visiting them. The reduction in clutch size may also be accounted for by early breakage and eating of some of the eggs, as this has been noted mainly in cases where the nests were not checked frequently. The thinning of eggshells and breakage of the eggs evidently is due largely to the inhibition of carbonic anhydrase by DDT and its metabolites. We are left with some phenomena that are still unexplained. Why does a low dose of pesticide produce relatively more thinning of the eggshell than larger doses do? What is the mechanism that kills embryos in the shell? These questions need further investigation.

The effect of the pesticides in disturb-

ing the calcium balance of birds probably is not of direct concern to man, because birds are a special case in their high calcium requirement at breeding time. It seems, however, that we should be concerned about the pesticides' effects on the hormone balance and on other physiological systems. The induction by pesticides of liver enzymes that lower the estrogen levels has been found in a wide variety of vertebrates, including a primate, the squirrel monkey. There is little doubt that this effect applies to man as well. Moreover, the chlorinated hydrocarbons are known to alter the glucose metabolism and inhibit an enzyme (adenosine triphosphatase, or ATPase) that plays a vital role in the energy economy of the human body.

The recent finding by investigators at the National Cancer Institute that a dose of 46 milligrams of DDT per kilogram of body weight can produce a fourfold increase in tumors of the liver, lungs and lymphoid organs of animals indicates that DDT should be banned for that reason alone. Human cancer victims have been found to have two to two and a half times more DDT in their fat than occurs in the normal population. Investigators in the U.S.S.R. recently reported that DDD, another metabolite of DDT, reduces the islets of Langerhans, the site of insulin synthesis.

The peregrine population crash has prompted two international conferences of concerned investigators, in 1965 and again in 1969. It is encouraging to note that in Britain, where severe restrictions were imposed in 1964 on the use of chlorinated hydrocarbon pesticides, the peregrine population has increased in the past two years. The Canadian government recently announced licensing restrictions that are expected to reduce the use of these pesticides by 90 percent, and many states in the U.S. are also instituting or considering such restrictions. Environmental problems do not respect political boundaries, and in the long run it will do little good if restrictions on the use of these hazardous toxins are applied only to certain regions or parts of the globe.

The long-term effects of the chlorinated hydrocarbons in the environment on human beings are admittedly much more difficult to detect or assess than the spectacular effects that have been seen in the predatory birds. Still, the story told by the birds is alarming enough. It seems obvious that agents capable of causing profound metabolic changes in such small doses should not be broadcast through the ecosystem on a billion-pound scale.

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