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MODERN PESTICIDES AND BOBWHITE POPULATIONS

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Abstract: Bobwhite (*Colinus virginianus*) are frequently used as test animals for wildlife tests of pesticides. The organophosphate and carbamate pesticides that have replaced the organochlorines have many desirable properties, but they span a wide range of acute toxicities and some of them affect survival, reproduction, food consumption, behavior, and nervous system enzymes in laboratory tests. Applying these laboratory findings to the field requires assumptions about the severity of exposure in the field. Direct field measurements show that birds may be exposed to significant amounts of these pesticides or even more toxic degradation products under some conditions. Adverse population effects may also result from depression of insect populations during the seasons when bobwhites rely on insects for food.

The possible reduction of wildlife populations by pesticides has been a concern of wildlife biologists since the organic insecticide era began in the 1940's. As early as 1945, tests of DDT's effect on wildlife were being conducted both in the field and laboratory (Coburn and Treichler 1946, Cottam and Higgins 1946, Nelson and Surber 1947). It appears that a 1948 test of parathion was the first wildlife test of an organophosphate (OP) insecticide (Linduska and Springer 1951). Bobwhites were used in these early controlled investigations because they are an important game species and are relatively easily maintained in captivity. The bobwhite is still frequently used as a representative wildlife species for tests of many OP and carbamate (CA) pesticides that are replacing the original organochlorine pesticides like DDT.

Organochlorine insecticides are relatively persistent in the environment. This was initially thought to be a desirable property of these chemicals, but the ecological consequences of the buildup of these chemicals in natural systems have been a major factor in the replacement of the organochlorine insecticides with OP and CA insecticides. These replacement insecticides are degraded relatively rapidly by both chemical and biological processes. They do not appear to be concentrated in higher levels of food chains and their acute effects on birds and mammals seem to be reversible after exposure ceases. Despite these favorable properties, many OP and CA insecticides are extremely toxic and have caused problems. In this paper, I will attempt to provide a practical perspective on the potential effects of these chemicals on bobwhite populations. Many of the questions about effects

on bobwhite populations are not yet answered and I will rely on studies with other species as necessary.

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TOXICITY TESTS

The first logical step in testing a pesticide for wildlife effects is to determine how much is lethal to one or more representative species (Turner 1981). EPA has issued standardized guidelines for such tests and the bobwhite is one of the species that may be used (Anon. 1978), reflecting many years of experience with this species as a test animal and the accumulation of a substantial data base that has been usefully summarized by Hill et al. (1975) and Kenaga (1979). Testing new chemicals during the research and development phase that precedes marketing now allows comparisons with pesticides already in use. The broad range of avian toxicities exhibited by OP and CA insecticides allows crude comparisons of potential acute hazards, but it does not provide one answer to the general question of whether these insecticides affect quail populations. That answer depends on the particular pesticide and the circumstances under which it is used.

Wildlife managers, as applied population biologists, also want to know whether insecticides depress reproductive rates. This is a more difficult question to answer because a bigger commitment of time and facilities is

required for a reproductive study than for an acute toxicity study. Consequently, few bobwhite reproductive studies of OP's or CA's have been published. *Coturnix coturnix* and ring-necked pheasants (*Phasianus colchicus*) have been used more frequently for these studies, and it appears that results with these species may be adequate guides for bobwhites. When I compared the reproductive responses of pheasants and bobwhites to diazinon, the species were qualitatively similar (Stromborg 1977, 1981). In both of those studies, egg laying was inhibited as a function of dietary dose during a three week trial. Although not exactly comparable, the results suggested that bobwhites were somewhat more sensitive to diazinon; the minimum dose that affected egg laying was lower as was the dose that completely blocked egg laying (Stromborg 1981). These reproductive effects were reversible and most birds resumed laying within several weeks after being taken off the pesticide diet. In addition, the hatchability of those eggs that were laid and the survivorship of chicks hatched from them was not affected.

A confounding variable was present in both of those studies that requires some explanation. When diazinon was added to their food, the birds ate less. This behavior is commonly seen in avian tests of highly toxic OP's and CA's, and several studies have demonstrated that caged birds will not eat large quantities of toxic feed if they have access to other foods (Hill 1972, Bennett and Prince 1981). What this means in the field is not clear, but it is known that some species do eat contaminated food under some circumstances (e.g., Mendelssohn and Paz 1977, Stromborg 1979, White et al. 1979). Reduced feeding rate due to pesticide-induced illness may be one of the effects of these insecticides under field conditions. This may lead to nutritional stress, but there is no evidence of any direct effect by these pesticides on energy metabolism (Watkins et al. 1978).

The diazinon tests with quail and pheasants also demonstrated a reduction in egg laying that was not explained by reduced feeding (Stromborg 1977, 1981). These effects may have been related to hormonal imbalances similar to those reported by Rattner et al. (1982 a) for quail fed parathion. EPA guidelines for registration require avian tests for reproductive effects under some circumstances (Anon. 1978), and, as in the toxicity testing requirements, bobwhites are a suggested test species. Therefore, data on reproduction should become available as these relatively new requirements are implemented. However, applying laboratory results to field conditions requires many assumptions about factors such as the availability of the pesticide over time, exposure of birds to it, and the interactions of pesticide effects with natural environmental stress (Rattner et al. 1982 b).

Behavioral alterations caused by OP and CA insecticides could reduce birds' ability to respond to environmental stresses. Kreitzer (unpublished data) found that extremely low

dosages of several OP's lowered the ability of bobwhites to learn visual discrimination of shapes. What this means in a field context is unclear, but an alteration in learning ability might reduce the ability of quail to cope with a complex and varying environment. At higher doses, these chemicals influence behavior in evident ways that are symptomatic of severe poisoning. The toxic action of these chemicals on the nervous system produces uncoordinated muscular activity, prostration, regurgitation, and a variety of other signs all of which would interfere with a poisoned bird's ability to escape from potential predators. Poisoned songbird females temporarily abandoned incubation of clutches (Grue et al. 1982), and this might adversely affect embryonic development if it occurred during sensitive periods. All of these behavioral effects were produced by sub-lethal doses, so detecting their effects on a population under field conditions would require detailed studies and would not be apparent by cursory searches for evidence of poisoning in the form of dead birds.

EXPOSURE OF BIRDS IN THE FIELD

The principal question that must be addressed in attempting to apply laboratory results to field conditions is the extent of exposure, including both dose rates and duration. The usual method of establishing exposure of birds and other wildlife to OP and CA pesticides uses their effect on the enzymes collectively known as cholinesterases (ChE). In fact, the toxic effects of these pesticides are related to these enzyme effects; hence the pesticides are commonly referred to as anticholinesterases, or cholinesterase inhibitors. Applying accepted clinical practice from human medicine, Bunyan and Taylor (1966), in the first of a continuing series of studies, adapted biochemical techniques for quantifying the amount of enzyme inhibition in the brains of birds exposed to cholinesterase inhibitors. Ludke et al. (1975) and Hill and Fleming (1982) have given guidelines for interpreting field results of ChE measurements. At present it is thought that ChE inhibition reflects exposure to significant amounts of a ChE inhibiting pesticide, but some birds with severely depressed ChE recover although others die with less inhibition. As a diagnostic tool, ChE measurements are most useful in determining the nature of a chemical in dead birds. It is unfortunate that no precise predictive relationship between ChE inhibition and overt biological effects has been discovered. If such a relationship did exist and could be measured, interpretation of field results like the demonstration by Smithson and Sanders (1978) that wild quail had depressed ChE in areas sprayed with parathion might be extended to population effects by predictive modelling (Tipton et al. 1980).

In the absence of this relationship, it is necessary to determine the amount of exposure by more direct techniques if laboratory results are to be interpreted and used for predictive

purposes. Quail are directly exposed to ChE inhibitors in a variety of ways. The most obvious is by eating seeds or vegetation treated directly with one of these pesticides. Several of these pesticides are effective seed treatments and have been applied to a variety of crop seeds. The rates of application are usually high enough to kill birds that eat more than a small number of treated seeds (Stromborg 1977). Unfortunately, intentional poisoning of granivorous birds is also easy and apparently not uncommon (Stone 1979). Although this source of exposure actually results from misuse (label statements include appropriate cautions about keeping wildlife and treated seed apart), under field conditions, there are often treated seeds available to birds after normal planting operations, and the rapidity of action of these chemicals localizes mortality so that even intentional poisonings may be undetected.

Vegetation that has been sprayed can also constitute a hazard to birds eating it. A number of instances of mortality, primarily of geese (*Branta canadensis*), have been recorded (Stone 1979). Wild turkeys (*Meleagris gallopavo*) have also been poisoned by feeding on vegetation sprayed with an OP (Nettles 1976). Both of these species are large and carcasses were usually found in the open; these factors combine to increase the probability of detection of poisoning. Similarly, reported die-offs of small birds are usually extensive (e.g., Seabloom et al. 1973) and consequently noticed by the public. In the absence of large conspicuous groups of casualties, detection of mortality may be infrequent (Rosene and Lay 1963).

A less easily studied route of exposure is secondary poisoning. Potentially, this could be extremely important to quail during the times that their diet is high in animal material (Rosene 1969:108, Hurst 1972). It is commonly believed that birds eat poisoned insects (Mills 1973, Stickel 1974) but few data exist on the amounts of ChE inhibitors such poisoned insects might contain. McEwen et al. (1972) found Guthion in grasshoppers during an operational grasshopper control program. Stromborg et al. (In press) found low residues of diazinon in an experimental application designed specifically to determine the feasibility of direct measurements of residues in insects. In another grasshopper control operation, biologically significant residues of acephate and its more toxic metabolite methamidophos were found (Stromborg, McEwen, and Lamont, unpublished data). Although these studies all demonstrate the feasibility of direct residue measurements, the practical difficulties of obtaining adequate samples have precluded widespread use of this technique. Direct demonstrations of secondary poisoning of birds by insects containing ChE inhibitors are equally rare, but White et al. (1979) reported mortality of adult and nestling laughing gulls (*Larus atricilla*) that ate parathion-poisoned insects. This route of exposure should receive much more attention in future studies, particularly the possibility that some ChE inhibitors may be metabolically transformed to

more toxic substances in poisoned insects. This process can lead to erroneous interpretations of laboratory results where only the parent insecticide is tested.

In addition to these oral routes of exposure, bobwhites might be exposed through dermal contact if they occupy a sprayed area either during or after application. Hudson et al. (1979) tested a variety of ChE inhibitors for dermal toxicity in birds and concluded, as did Fowle (1972), that this is a potentially important route of exposure. Labisky (1975) tested this hypothesis with pheasants in a simulated application of a soil insecticide and observed some mortality and evidence of poisoning through dermal contact, but only under presumed worst-case conditions where he simulated a spill of the actual formulated product. During spraying, quail within a field might inhale significant amounts of insecticide. Berteau and Chiles (1978) compared the oral and inhalation routes in laboratory tests and concluded that there was little difference in toxicity between the two routes of exposure. Most probably, this is generally true of field exposures; the observed effect will be a result of total insecticide exposure from all of the potential routes: oral, dermal, and inhaled.

ECOLOGICAL EFFECTS

Although these direct poisoning effects are the usual focus of pesticide research with birds, under field conditions, the reduction of insect populations is probably also quite important to bobwhite populations. Field studies often result in reports of presumed emigration of birds from large spray blocks, but as McEwen et al. (1972:193) point out, emigrants probably rarely find suitable unoccupied habitat, and if they are actively nesting, emigration results in nest abandonment and loss of some reproductive potential. The impact of reduced insect populations is a function of the dependence of the quail on insects at the time of spray and the size of the spray block. If the sprayed areas are small and patchy, suitable foraging areas may be found close enough to the nest site that a simple shift in home range use may enable birds to find adequate insect foods without abandoning an active nest.

These direct and indirect factors acting simultaneously may constitute a serious potential hazard to populations of an agriculturally associated bird like the bobwhite. In fact, studies of organochlorine insecticides and quail clearly demonstrated severe impact on a regional bobwhite population (Rosene 1965). No such comprehensive study of ChE inhibiting insecticides has been undertaken with bobwhites. More field data for pheasants and ChE inhibitors are available than for bobwhites. Because these species are fairly similar in their food habits and agricultural association, it seems reasonable to expect that results from pheasant tests are applicable to bobwhites. Wolfe et al. (1971) exposed young pheasants in 5 acre pens to a simulated parathion spray and found that they ate

large quantities of presumably poisoned insects shortly after the field was sprayed. Although ChE was depressed, they found no evident behavioral effects or mortality. In a similar test of young pheasants, Messick et al. (1974) found behavioral effects that would have increased their vulnerability to predators. Concurrent tests on unconfined wild adults indicated no apparent effects on survival or reproduction. The proportion of insects in the diets of wild juvenile pheasants in sprayed areas was drastically reduced when insect populations were reduced by pesticides. The significance of this reduction of an important protein source for growing birds is hard to assess. Potts (1977) found that when pesticides reduced the vital insect food sources of young partridges (Perdix perdix), the productivity of partridge populations was reduced. Considering the overall similarity of bobwhites and partridge in agricultural areas, it would be surprising if this was not also true for bobwhite populations.

There are many indications from both field and laboratory studies that ChE inhibiting insecticides might be influencing bobwhite populations. Some of these chemicals are extremely toxic to quail, and it is known that wild birds are exposed to biologically significant quantities under some conditions. Whether pesticide-induced mortality or reproductive effects depress bobwhite productivity is not known, and how these hypothetical limits might relate to other factors limiting populations through habitat destruction and degradation is not understood. We need to measure the actual exposure of bobwhites in the field to fully use the results of laboratory studies. We also need to determine whether other influences on mortality and reproduction compensate for pesticide effects. Ultimately, studies of these diverse influences on population dynamics will have to be integrated into a comprehensive study of this species if we want finally to assess pesticide effects on bobwhite populations.

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