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# A Model of the Impact of Methyl Parathion Spraying on a Quail Population

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The use of computer simulation models has gained wide acceptance in both research and management of ecological systems. One of the primary reasons for this acceptance is the realization that computers and computer models can be useful in integrating large amounts of information and providing insight into problem areas of research and management.

Numerous books and papers have been written on the justification of models (PATTEN 1971, 1972, 1975, 1976; JEFFERS 1972; MAKI & THOMPSON 1973; deWIT & GOUDRIAAN 1974; THORNLEY 1976; GOLD 1977; HALL & DAY 1977; SOLOMON & WALTERS 1977) and more specifically the use of models in the natural resource field (DAVIS 1967; WATT 1968; GILES & SCOTT 1969; HAYNE 1969; LOBDELL et al. 1972; GROSS et al. 1973; NICHOLS et al. 1976; TIPTON 1977; COWARDIN & JOHNSON 1979). We will illustrate herein the potential use of models in evaluating the impacts of pesticides and environmental contaminants on wildlife populations.

Organophosphate pesticides have desirable characteristics from the standpoint of control of many crop-damaging insects as their half-lives in the environment are much shorter than other pesticides (DIETER & LUDKE 1975). However, their acute toxicity to vertebrate species is often quite high (TUCKER & CRABTREE 1970). Methyl parathion, for example, is used as an insecticide on cotton and soybeans, and laboratory studies have indicated a high acute toxicity in avian species and an acute oral $LD_{50}$ of 8.2 mg/kg in 2-month old pheasants (Phasianus colchicus) (TUCKER & CRABTREE 1970). Since field applications (0.57-1.13 kg/ha) of methyl parathion occur frequently during summer and early fall months in the habitats of local bobwhite quail populations (SMITHSON & SANDERS 1978), there is need to evaluate the potential increased mortality of bobwhite quail exposed to different methyl parathion application rates. The present state of knowledge on the

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effects of pesticides would necessitate the use of both laboratory and field data in the initial modeling effort. As additional information becomes available more sophisticated and biologically realistic models can be developed.

Since organophosphates generally do not accumulate in the bodies of exposed organisms, identification of exposure in collected samples is usually by enzymatic assays rather than by chemical analytical techniques. Acetylcholinesterase (AChE) inhibition in the brain is often used to indicate exposure to organophosphates such as methyl parathion.

Field applications of 1.13 kg/ha ethyl parathion have been found to cause severe mortality in a laughing gull (Larus atricilla) colony (WHITE et al. 1979). Insects poisoned by parathion were ingested resulting in death of adults as well as in young that received food from adults that ingested poisoned insects. Intoxicated gulls found sick or dead had significant reductions in brain AChE activity (>50% inhibition). Substantial laboratory information is available on AChE inhibition in avian species and inhibition greater than 50% is generally considered indicative of death from organophosphate exposure (LUDKE et al. 1975) should dead or dying birds be found in a treated area.

The model developed in this paper utilizes a projection matrix model developed by TIPTON (1975). LESLIE (1945) and LEWIS (1942) first used this type of matrix structure to model the dynamics of populations with discrete age classes. The important attributes of this group of models is their ability to include age specific mortality and reproduction. Since the initial development of LESLIE'S (1945) model numerous modifications have been made to the model in order to include more realistic biological information. For a review of the modifications and uses of the Leslie matrix see USHER (1972).

Data collected by SMITHSON & SANDERS (1978) from a field study of a local bobwhite quail population exposed to methyl parathion applications (1.13 kg/ha) were used for this modeling effort. Quail were collected before application and 24 h after each spray (generally weekly) of the area. Brain AChE tended to be lower after each successive spray and following the later applications many individual AChE activities were in the toxicologically significant range (>50% inhibition). Therefore, using brain AChE inhibition as an index of mortality, the model is used to evaluate the impact of 2 levels of methyl parathion spraying on mortality in a local bobwhite quail population.

## METHODS

Two 6 x 6 matrices (Tables 1 and 2) were constructed and indicate states of brain AChE inhibition in bobwhite quail for 2 levels of spraying, 1.13 kg/ha and 0.57 kg/ha. Rows 1 through 5 in the matrices represent stages of AChE depression. An individual animal can pass through 5 stages of depression from 0 inhibition (100% AChE activity) to death (< 25% normal activity or > 75% brain AChE inhibition). Progressive states of inhibition were associated with higher levels of toxicosis from pesticide poisoning. Row 6 represents mortality from natural causes. ROSENE (1969) found that from March 1 to November 1 about 50% of a southeastern bobwhite quail population died from natural causes. Using this value, weekly mortality rates were calculated to be 2% of the adult birds in the population.

Table 1. Probabilities of survival and mortality under 1.13 kg/ha weekly spraying of methyl parathion.

| | | | | | | |
|---|---|---|---|---|---|---|
| 100* | 0.23 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 |
| 75* | 0.70 | 0.04 | 0.0 | 0.0 | 0.0 | 0.0 |
| 50* | 0.0 | 0.79 | 0.1 | 0.0 | 0.0 | 0.0 |
| 25* | 0.0 | 0.0 | 0.48 | 0.01 | 0.0 | 0.0 |
| Pesticide Mortality | 0.05 | 0.15 | 0.49 | 0.97 | 1.0 | 0.0 |
| Natural Mortality | 0.02 | 0.02 | 0.02 | 0.02 | 0.0 | 1.0 |

*Percent brain AChE activity (100 = normal brain AChE activity; 25 = 25% of normal or 75% inhibition).

The individual elements in each column of the matrices (Tables 1 and 2), therefore, contain the probabilities of an animal either: (1) staying in the same AChE depression stage; (2) moving to the next stage; (3) dying of poisoning; or (4) dying of natural causes. For example, if spraying occurred at 1.13 kg/ha (Table 1) then 23% of the healthy animals (100% AChE activity) would remain in that stage, 70% would move into the 75% activity stage, (25% inhibition) 5% would die from poisoning and 2% from natural causes. At the same time, 4% of the birds in the 75% activity stage (25% inhibition) would remain in that stage, 79% would move into the 50% activity stage (50% inhibition), 15% would die from poisoning and 2% from natural causes. Hypothetical selection of these probabilities was based on integration of reported field (SMITHSON & SANDERS 1978) and laboratory (LUDKE et al. 1975, WHITE et al. 1979) data.

Table 2. Probabilities of survival and mortality under 0.57 kg/ha weekly spraying of methyl parathion.

| | | | | | | |
|---|---|---|---|---|---|---|
| 100* | 0.62 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 |
| 75* | 0.33 | 0.51 | 0.0 | 0.0 | 0.0 | 0.0 |
| 50* | 0.0 | 0.40 | 0.49 | 0.0 | 0.0 | 0.0 |
| 25* | 0.0 | 0.0 | 0.24 | 0.49 | 0.0 | 0.0 |
| Pesticide Mortality | 0.03 | 0.07 | 0.25 | 0.49 | 1.0 | 0.0 |
| Natural Mortality | 0.02 | 0.02 | 0.02 | 0.02 | 0.0 | 1.0 |

*Percent brain AChE activity.

At methyl parathion application rates of 0.57 kg/ha the percentage of animals staying in the same stage or moving to a lower stage would be determined by the reduced probabilities in Table 2. For this exercise, it was assumed that spraying occurred weekly for the 8-week period, animals could not improve AChE activity due to the short period between spraying and could only move down 1 stage of AChE activity with each spraying. In addition, only adult birds were considered, and it was assumed that males and females were equally affected by the pesticide and no reproduction, emigration or immigration occurred in the treated areas.

Using the 2 probability matrices (Tables 1 and 2) the mortality of a local quail population was investigated. One thousand healthy birds (0% inhibition; 100% AChE activity) were assigned to a hypothetical area. Eight weekly applications of methyl parathion (1.13 kg/ha or 0.57) were simulated.

## RESULTS AND DISCUSSION

The computer simulations indicated that different levels of methyl parathion spraying had a major impact on the number of bobwhite quail that remained healthy (100% AChE activity) in the treated area. Table 3 contains the number of healthy birds, those dying from pesticide poisoning and natural causes over the 8-week period. Spraying at 1.13 kg/ha resulted in totally removing all healthy quail (99% mortality) from the population after 6 weekly methyl parathion applications. Although this mortality is probably higher than might be expected a reduction of the weekly application to 0.57 kg/ha substantially reduced (55% mortality) the number of individuals removed by methyl parathion poisoning . WHITE et al. (1979) reported mortality trends in a laughing gull colony that was exposed to parathion applied at 1.13 kg/ha, which was double the recommended rate of application. Birds found dead or dying were determined to have 57-89% depression in AChE activity, and therefore, were diagnosed as dying from parathion exposure. Laboratory studies reported by LUDKE et al. (1975) substantiate that brain AChE inhibition exceeding 20% indicated organophosphate exposure, and inhibition greater than 50% was sufficient for diagnosing cause of death. The ability to predict the impact of pesticide spraying (e.g. parathion induced mortality in laughing gulls) on wildlife populations would be extremely useful for their management.

In comparing the mortality values in Table 3, the bobwhite quail population could probably withstand application rates at 0.57 kg/ha for 8 weeks without any major die-off possibilities. ROSENE (1969) has reported that up to 72% of a quail population is normally replaced each year. Although natural mortality is generally high in this species, reproductive potential is sufficiently high for population replenishment. However, a substantial reduction in population size can result in population recovery being delayed by one to several years ROSENE (1969). ROSENE (1965) reported that heptachlor applications of 4.56 kg/ha reduced the number of coveys of quail in a treated area from an average 19.4 coveys in a 405 ha area to 4.4 coveys the following year. Two years after pesticide application, the population was still depressed with only 11 coveys in the area. Although residual effects of heptachlor could have been interacting with natural factors causing population depression, the population still required several years to recover after a major pesticide induced die-off.

Although in the presently discussed model only mortality is considered, the potential use of models to evaluate impacts of environmental contaminants on wildlife populations appears to be quite valuable. As more information is collected the model could be made more sophisticated to include effects on the reproductive characteristics or differential mortality of sexes or age groups. Further integration of field and laboratory information used in conjunction with models such as this projection matrix could be useful in improving decision-making processes particularly as related to the use of toxic chemicals in the environment. There presently exists substantial information concerning contaminant effects on wildlife which could be synthesized in the form of computer models, producing significant gains in the management strategies for many wildlife species.

Table 3. Simulated effects of spraying methyl parathion weekly on the survival of 1000 healthy bobwhite quail.

| Application Rate kg/ha | | Weeks | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | 0 | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 |
| 1.13 | Healthy (100% AChE) | 1000 | 232 | 54 | 13 | 3 | 1 | - | - | - |
| | Pesticide Mortality | 0 | 49 | 163 | 464 | 807 | 902 | 923 | 930 | 931 |
| | Natural Mortality | 0 | 20 | 39 | 55 | 65 | 67 | 68 | 68 | 68 |
| | Total Mortality | 0 | 69 | 202 | 519 | 872 | 969 | 991 | 998 | 999 |
| 0.57 | Healthy (100% AChE) | 1000 | 620 | 386 | 239 | 148 | 52 | 57 | 36 | 22 |
| | Pesticide Mortality | 0 | 24 | 64 | 134 | 232 | 344 | 455 | 554 | 637 |
| | Natural Mortality | 0 | 20 | 39 | 58 | 80 | 88 | 99 | 108 | 115 |
| | Total Mortality | 0 | 44 | 103 | 192 | 312 | 432 | 554 | 662 | 752 |

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