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# Responses of Quail, Pheasants, and Sparrows to One Oral Dose of Dimethoate and to Consumption of Dimethoate Treated Bran Baits

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The organophosphate insecticide, dimethoate has been used in a liquid formulation to control grasshoppers on cereal crops in the Canadian prairies for two decades (Environment Council of Alberta 1981). A new application technique of broadcasting wheat bran bearing the insecticide into the crop at 165 g ai/3 kg bran/ha is being considered by agriculturalists. Under experimental conditions grasshoppers seek out the bran and are killed within six hours. However using bran bait as a pesticide carrier raises the question of whether some bird species would be attracted to and consume sufficient quantities of bran bait to be poisoned. In this paper we report the brain cholinesterase activities resulting from dimethoate oral dosing and ad libitum bran bait feeding of Coturnix quail, Coturnix coturnix, Ring-necked pheasant, Phasianus colchicus and Savannah sparrow, Passerculus sandwichensis.

The toxicology and metabolism of dimethoate have been studied extensively in insects, mammals and plants. The primary metabolite O, O-dimethyl S-(N-methylcarbamoylmethyl) phosphorothiolate, is thought to be the toxic agent in dimethoate poisoning (Fukuto and Sims 1977). That metabolite is eliminated more slowly in birds than in mammals, leading to greater toxicosis in the former (Sanderson and Edson 1964). $LD_{50}$ measurements of acute toxicity have been reported for several birds and mammals (Sanderson and Edson 1964; Tucker and Crabtree 1970). $LC_{50}$ values for several birds feeding ad libitum for five days were reported by Heath et al. (1972). Schafer and Brunton (1979) suggested that Passeriform birds had greater sensitivity to a wide range of pesticides than Galliform birds.

In our experiments brain cholinesterase activity (ChE) was measured to assess dimethoate exposure because ChE activity has proven to be a sensitive and easily-used method (Hill and Fleming 1982). Depression of ChE activity within the body is an early response to organophosphate poisoning. Interpretation of brain ChE values depends on the biology of the bird species (Grue et al. 1981).

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## MATERIALS AND METHODS

Coturnix quail (5-6 wk old) and Ring-necked pheasants (6 wk old) which had been reared on commercial chicken starter were provided by Alberta Fish and Wildlife. Fifty-eight Savannah sparrows which had been mist-netted in central Alberta were maintained on commercial wild bird seed in an untreated enclosure by the University of Alberta during June, 1983 prior to the experiments.

Cygon 4E, containing 480 mg dimethoate/litre dissolved in Cyclo-Sol 63, was purchased from a commercial outlet; and a small quantity of Cyclo-Sol 63 was provided by Chipman Chemical Co. Agriculture Canada supplied the bran bait.

In the first experiment each bird in groups of eight to ten was given a small oral dose according to its body weight by crop intubation with one mm diameter Tygon tubing attached to a micro-syringe. Dose levels prepared from the Cygon 4E stock of 2.1, 6.3, 11.7, 18.8, 35.2, 41.7, 58.7, 78.2, 104.3 and 105.6 mg dimethoate/kg body weight were used for quail whose mean live weight was 145 $\pm$ 1.2 (SE) g. Dose levels of 2.8, 8.5, 14.1, 18.8, 25.0, 25.4, 31.3 and 41.7 mg/kg were used for pheasants whose mean live weight was 420 $\pm$ 7.7 g. Due to the limited supply of birds, only six Savannah sparrows were dosed at each of 6.0 and 8.1 mg/kg dose levels of dimethoate. This was done with considerable difficulty because of their wilder nature and smaller size compared to the other species. The mean sparrow weight was 18 $\pm$ 0.23 g. To check the effect of the dimethoate solvent, nine quail were given a dose of Cyclo-Sol 63 comparable to that received by quail in the 18.8 mg/kg dose. The birds were released, after dosing by group, into enclosures where feed and water were provided.

In the second experiment birds of each species were fed ad libitum for 24 hours on dimethoate bran bait (5.5%) scattered at four different densities on sand covered (6 mm depth) floors of small enclosures (11.15 sq. m - 10 quail or pheasants, 3.48 sq. m - 10 sparrows). For the primary treatment 3.34 g (quail and pheasants) and 1.04 g (sparrows) of bran bait (165 g ai/3 kg/ha) were scattered by hand onto the floor after the test birds had already been enclosed for 16-18 hours with a surplus (for 48 hrs) of scattered chicken starter (quail and pheasants) or commercial wild bird seed (sparrows). The other floor densities of bran bait were 3, 10 and 100 times the primary floor density for each species.

Twenty-four hours after dosing or ad libitum feeding, all birds were sacrificed. The heads were frozen in liquid nitrogen for ChE analysis of each brain by the third author, using the Ellman et al. (1961) method as modified by Hill and Fleming (1982).

The ChE activities of brains from orally dosed birds were expressed as a percent of the mean ChE activity of non-dosed birds for each species. The arc sin transformed values of these percents were regressed against the logarithms of the dosages. Homogeneity and analysis of variance and differences among the mean brain ChE

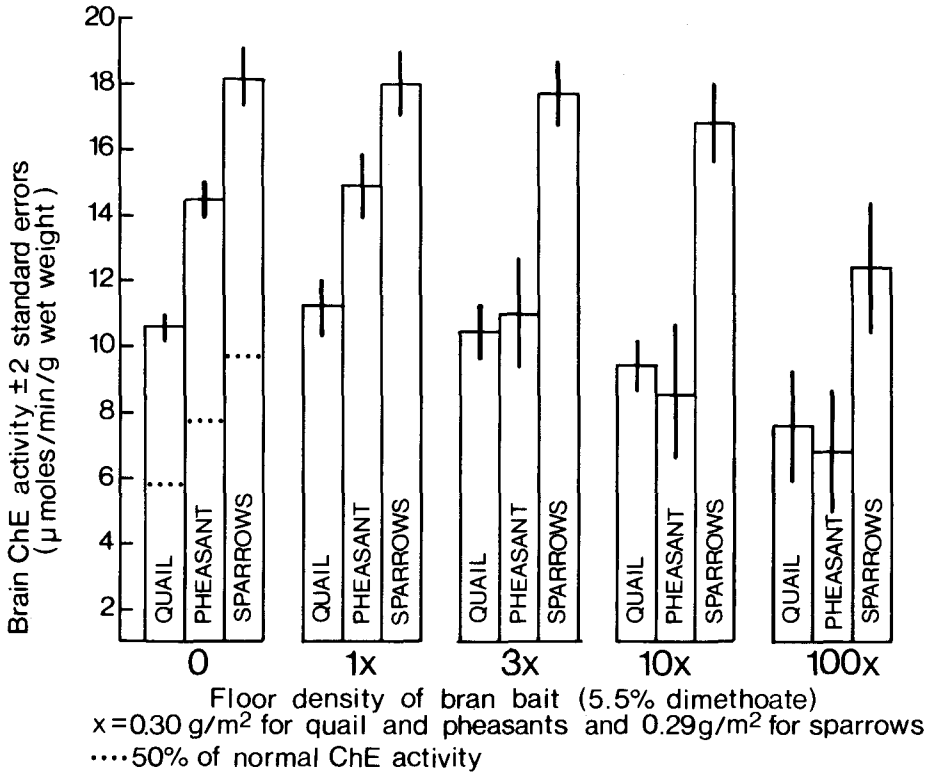


Figure 1. Mean brain cholinesterase activity of three bird species exposed to different densities of dimethoate bran baits.

activities of birds exposed to different floor densities of bran bait were determined (Bartlett's test, 1 way ANOVA, F statistic and Student-Newman-Keul's test).

RESULTS AND DISCUSSION

When exposed to 100X proposed density for application of bran bait (5.5%) for grasshopper control all three bird species had significantly depressed brain ChE activity ($P < 0.05$, Fig. 1).

However, only Ring-necked pheasants suffered significant depression of brain ChE activity at 3X and 10X the proposed density for application of bran bait. At the proposed bran bait density and dimethoate concentration (165 g ai/3 kg/ha) the brain ChE activity did not differ from that of normal birds for each species.

One time, oral exposure to increasing concentrations of dimethoate up to and slightly above the $LD_{50}$ level resulted in significant depression of brain ChE activity for all three species according to regression analysis ($P < 0.01$). The regression equations of arc sin (brain ChE activity as a percent of normal brain activity) against

Ln dosage (mg/kg body weight) were Y = -10.3X+71.6, Y = -18.8X+85.1 and Y = -75.9X+194 for quail, pheasants and sparrows and the respective coefficients of determination were 84%, 86% and 85%. Normal brain ChE activities for untreated (control) birds ranged from 18 for Savannah sparrows, to 14 and 11 moles/min./g wet weight for Ring-necked pheasants and Coturnix quail (Fig. 1).

No birds died during the 24 hours of ad libitum feeding on dimethoate bran bait. However, some test birds of both quail and pheasants did die at the higher dimethoate concentrations in the oral dosing experiment (Table 1). Brain ChE activity was measured on all birds. There were no differences in ChE activity between the sexes or sacrificed and dead birds at the same dose level.

Table 1. Number of birds of three different species dying (number treated) within 24 hours of oral dosing according to dimethoate concentration.

| Dose mg/kg | Coturnix quail | Ring-necked pheasants | Savannah sparrows |
|---|---|---|---|
| 2.1 | 0(10) | - | - |
| 2.8 | - | 0(10) | - |
| 6.0 | - | - | 0(6) |
| 6.3 | 0(10) | - | - |
| 8.1 | - | - | 0(6) |
| 8.5 | - | 0(10) | - |
| 11.7 | 0(10) | - | - |
| 14.1 | - | 0(10) | - |
| 18.8 | 0(10) | 5(10) | - |
| 25.0 | 1(9) | 6(10) | - |
| 25.4 | - | 9(10) | - |
| 31.3 | - | 10(10) | - |
| 35.2 | 1(10) | - | - |
| 41.7 | 0(1) | 11(11) | - |
| 58.7 | 2(10) | - | - |
| 78.2 | 4(9) | - | - |
| 104.3 | 4(8) | - | - |
| 105.6 | 9(10) | - | - |

Brain ChE activity of quail dosed only with Cyclo-Sol 63, the solvent for dimethoate in all other dose levels, was 10.31 $\pm$ 0.18 (1 SD) moles/min/g wet weight. This is almost identical to the brain ChE activity of normal quail which were not given a placebo dose (Fig. 1).

Untreated quail exhibited sand bathing, territoriality and courtship behavior when placed in the experimental enclosures. Immediately after treatment quail were subdued and had considerable gular flutter. After 24 hours, behavioral differences were not

observable between control quail and those exposed to 6.3 mg/kg or less. Quail exposed to 78.2 mg/kg or more did not vocalize or maintain their balance. Subdued behaviors similar to those of quail were noted for pheasants before treatment. At dimethoate exposures of 18.8 mg/kg or more pheasants did not vocalize or maintain their balance.

The brain ChE activity of quail which were given the Cyclo-Sol 63 solvent for dimethoate was the same as that of untreated quail. This suggests that neither the solvent nor the crop intubation process done with all orally dosed birds except the control birds (untreated) had a measureable effect on brain ChE activity.

Hill and Fleming (1982), who reviewed organophosphate poisoning of birds, reported that a 20% reduction in brain ChE activity from normal was sufficient to infer exposure to an organophosphate insecticide and a 50-70% reduction was associated with death. The regression equations from our first experiment related 20% and 50-70% reductions of brain ChE activity to dimethoate doses of 2.2, 13.2-41.3; 3.2, 8.4-15.8 and 5.6, 7.1-8.3 mg/kg live body weight for Coturnix quail, Ring-necked pheasants and Savannah sparrows, respectively. The doses causing 50% reductions are used below in discussing the second experiment. Some deaths of quail and pheasants did occur within or near the dose ranges associated with 50-70% reductions of brain ChE activity (Table 1). No deaths occurred among sparrows in the first experiment although brain ChE reduction was 63% of normal for birds dosed at 8.1 mg/kg. However, because of noticeable but slight regurgitation this reduction probably relates to a somewhat lower dose level.

If the regression equations of the first experiment are applicable in the experiment of ad libitum feeding on dimethoate bran baits and an alternate food, then 50% reductions in brain ChE activity for the average quail (145 g) pheasant (420 g) and sparrow (18 g) would have been associated with consumption of 34.8, 64.2 and 2.3 mg of bran respectively during the 24 hour feeding period. Fifty percent reductions in brain ChE activity occurred only for pheasants at 10X and 100X the recommended field application rate (Fig. 1).

The similarity of brain ChE activities for untreated birds and those exposed 24 hours to the proposed field density of bran bait suggests that the bran bait applied at this density, or lower, should not pose a direct toxic hazard to any of the three test species. However, the significant sharp decline in brain ChE activity for Ring-necked pheasants, even when the proposed bran bait density is only tripled, suggests that overapplication of bran bait may lead to occasional cases of mild toxicosis. Inferences from these results with two constrained galliform and one passeriform species to other birds would be risky for two reasons. A 10-fold range in species sensitivities to a pesticide is to be expected (Tucker and Haegele 1971). A 40% brain ChE reduction in a free-living bird may be more serious than a 60% reduction for

constrained birds because the former must forage, defend territories and avoid predators.

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