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Anticoagulant, avian, brodifacoum, *LC50, mortality, secondary poisoning*
 A study of the toxicity of the anticoagulant brodifacoum to American kestrels (*Falco sparverius*)
 Rodenticide, anticoagulant

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Abstract: The objective of this study was to estimate the secondary median lethal concentration (LC_{50}) to American kestrels (*Falco sparverius*) that were fed meadow voles (*Microtus* sp.) that had consumed a lethal dose of brodifacoum. Individually caged voles were offered 50 g of 100 ppm brodifacoum bait *ad libitum* for 4 consecutive days. Surviving voles were killed on the 5th day, and their tissues were ground and mixed with ground tissues of voles that died prior to the 5th day to form a pooled sample containing 6.7 ppm brodifacoum. The pooled sample was diluted with untreated vole tissues to yield concentrations of 0.3, 0.8, 1.6, 3.2, and 6.0 ppm brodifacoum. Five groups of eight kestrels each were fed one of these five concentrations of brodifacoum-vole tissues for 5 days. After the feeding period, the birds were observed for 90 days. During the observation period, a total of five birds died, one each on Days 2, 3, 6, 10, and 56. Four of the five birds that died had been fed at the 6.0 ppm level and one had been fed at the 0.8 ppm level of brodifacoum-vole tissues. These data suggest that the estimated LC_{50} of brodifacoum-treated vole tissues to American kestrels is near 6.0 ppm.

INTRODUCTION

Brodifacoum is a potent, unique oral anticoagulant rodenticide that kills in a single feeding and is effective against warfarin-resistant rats (Dubock and Kaukenen, 1978). It is Federally registered in the United States by the Environmental Protection Agency (EPA) for the control of commensal rodents in a food bait at 0.005%. Its evaluation and use throughout the world to reduce food losses to field and commensal rodents has generated interest in its primary and secondary toxicity to the other vertebrates. Its potential to cause secondary poisoning to predators or scavengers from eating primary (target) animals is being investigated. Savarie and LaVoie (1979) offered caged American kestrels (*Falco sparverius*) whole brodifacoum-poisoned voles (*Microtus* sp.) for periods of 2 or 6 days. In the 6-day feeding group, four of the ten birds died. Mendenhall and Pank (1980) compared the secondary toxicity of six anticoagulants, including brodifacoum, to barn owls (*Tyto alba*). They fed rats (*Rattus norvegicus*, *R. rattus*, and *R. exulans*) killed by the anticoagulants to caged barn owls for periods of 1, 3, 6, or 10 days. They reported brodifacoum owl mortalities. However, tissue residue levels of brodifacoum in the rats and owls were not determined. Hedgal *et al* (1984) conducted field studies to determine the secondary hazards to barn owls and screech owls (*Otus asio*) from brodifacoum. No barn owl mortalities were observed; however, in the screech owl study, mortalities implicated secondary poisoning with brodifacoum. The study being reported here was conducted in 1981 and was designed to estimate the secondary LC_{50} of brodifacoum in the diet of the American kestrel.

MATERIALS AND METHOD

Meadow voles were live-trapped near Denver, Colorado, and maintained under laboratory conditions for a minimum of 7 days before testing at the Denver Wildlife Research Center (DWRC). During the pretest period, all voles were held in pens approximately 2.5 m x 0.75 m and were provided access to water and rodent laboratory chow *ad libitum*.

American kestrels were live-trapped by Bal-Chatri traps in northern California and eastern Colorado. Mature birds were brought to the DWRC and caged individually under ambient outdoor conditions for a minimum of 30 days before testing. More male than female kestrels were trapped in both Colorado and California. consequently, the test birds consisted of 28 males and 20 females. The outdoor raptor cages used during pretest, test, and observation periods measured 2.24 m long x 0.67 m wide x 0.61 m high. during the first week of captivity, birds were fed whole, untreated voles or mice; they then had daily access to approximately 50 g of food, consisting of 25% commercial bird-of-prey diet (Zu/Preem)^{RI} and 75% horse meat. Test birds were weighed upon arrival at the DWRC, immediately prior to testing and at the end of the test. In addition to weight, sex, place of capture, and estimated food consumption during the test period were noted. Birds were observed daily during the holding, testing, and observation periods. Three days before testing, kestrels were fed untreated ground vole tissues each day.

A total of 125 individually caged voles were offered 50 g each daily of rolled oat groat bait containing 100 ppm brodifacoum *ad libitum* for 4 consecutive days. On the 5th day, all surviving voles were killed by cervical dislocation

and all voles, including those that died and were frozen before the 5th day, were ground whole to form a pooled sample. Five aliquots consisting of 30 g each were drawn from the pooled sample and analyzed for tissue residues of brodifacoum by Imperial Chemical Industries (ICI) Americas, Inc., Biological Research Center, Goldsboro, North Carolina, USA. Treated tissue were then diluted with ground, untreated vole tissues to yield concentrations of 0.3, 0.8, 1.6, 3.2 and 6.0 ppm brodifacoum and frozen until used.

Five groups of eight kestrels each were fed one of the five concentrations of brodifacoum-vole tissues for 5 consecutive days. Birds were assigned to treatment groups as evenly as possible with respect to sex and location (state) of capture. The test protocol was similar to that described by Heath *et al.* (1972) with the exceptions that birds were individually caged, rather than group caged, and each bird was offered 30 g of vole tissues per day (on a metal tray) rather than food supplied *ad libitum*. A sixth group of eight kestrels were offered 30 g per day of untreated ground whole vole tissues for 5 days during the treatment period. All kestrels were observed for 90 days after consuming treated or untreated vole tissues. All dead birds after the start of testing were necropsied by a veterinarian, then were frozen and sent to ICI Americas for analysis; surviving birds were released.

RESULTS

The average total brodifacoum bait consumption by the voles during the 4 test days was 10.4 g ($SE \pm 5.10$). Sixty-four voles died on or before the 5th day. Based on an analysis of the pooled vole tissues, which contained 6.7 ppm of brodifacoum, the weight of the average vole, 48.5 g ($SE \pm 8.9$), and an average consumption of 10.4 g of bait containing 1.06 mg., it appears that about 70% of the ingested brodifacoum was either excreted or metabolized.

Five of the 48 test birds died between Days 2 and 56 of the observation period, and 4 of these 5 were dead at the 6.0 ppm treatment level. These birds could have consumed up to 7.3 mg/kg of brodifacoum. Table 1 shows calculated mean maximum consumption of toxicant in mg and mg/kg for each treatment group based on mean bird weight per group and also shows kestrel mortalities within each group. These values are useful for relative comparisons between groups; however, some spillage of vole tissues during feeding was unavoidable, so maximum consumption was generally not realized. Although some dehydration of vole tissues occurred in the cages prior to consumption, there is no reason to believe that this resulted in a loss of toxicant.

Table 1. Maximum mean possible consumption of brodifacoum* and mortalities of kestrel at each treatment level.

	Treatment level (ppm)					
	0.0	0.3	0.8	1.6	3.2	6.0
Brodifacoum (mg)	0.0	0.05	0.12	0.24	0.48	0.90
Brodifacoum (mg/kg)	0.0	0.4	1.0	2.1	3.9	7.3
Mortalities (died/tested)	0/8	0/8	1/8	0/8	0/8	4/8

* Spillage during feeding by some birds of up to about 10% of ground vole tissues were unavoidable; maximum consumption was never achieved at any treatment level.

Brodifacoum residues were the highest in the three birds dying before the 10th day of observation. It should be noted that residues were nearly the same in these three birds although one was in the 0.8 ppm treatment level (Table 2).

DISCUSSION

Although this study did not estimate the vole-kestrel secondary LC_{50} of brodifacoum, the results in association with those of Savarie and LaVoie (1979) do show that treatment levels of 6.0 ppm are secondarily lethal to kestrels. The LC_{50} probably could be determined by starting at about a 2-ppm-brodifacoum level in vole tissues.

The observed variation in gross pathology of the kestrels relative to body residue of anticoagulants has been noted by others. Jaques (1959) stated: "In fact, it is amazing how completely some of these drugs can interfere with the blood coagulation system without any symptoms of hemorrhage." The variation may also be the result of bird feeding behaviour, such as, food spillage, period of ingestion, and the general health or condition of the birds. Although more males (4) than females died, the 1.4/1 males to females in the test population precludes any conclusions about sex-related specificity.

The persistence of brodifacoum in the body tissues of the one bird that died approximately 2 months after consumption is consistent with elevated prothrombin times observed by Savarie and LaVoie (1979). Brodifacoum may

Table 2. Whole body residues (ppm) and gross pathology of the kestrels that died during the test.

Bird sex	Treatment level (ppm)	Brodifacoum residues (ppm)	Days to death*	Gross pathology
M	0.8	0.23	3	No symptoms
F	6.0	0.24	2	No symptoms
M	6.0	0.20	6	Hemorrhagic
M	6.0	0.12	10	Hemorrhagic
M	6.0	0.10	56	Ruptured cloaca

* Days to death during the 90-day observation period.

warfarin-treated mice in about 9 days after cessation of feeding treated mice. The degree of raptor secondary toxicity that may occur in field evaluations and operations cannot be predicted from this study. It has been shown that anticoagulant hemorrhage can be stress related (Jaques, 1959). Therefore, if low levels (<0.10 ppm) brodifacoum persisted for long periods in birds, they could eventually die when experiencing environmental stresses caused by weather and food shortages. Under field conditions, the degree of hazard to nontarget animals would depend on the bait concentration used, the methods of application, the place of death (above or below ground) by the target species, the amount of bait (kg/ha), local ecological situations, and the behavior of target and nontarget species.

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