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SECONDARY POISONING OF OWLS BY ANTICOAGULANT RODENTICIDES[1]

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Abstract: Anticoagulants—compounds that prevent clotting of the blood—are extensively used for control of small mammal pests. The potential secondary hazards of 6 anticoagulant rodenticides to birds of prey were examined in this study. Whole rats or mice were killed with each anticoagulant and were fed to 1–3 species of owls. Owls died of hemorrhaging after feeding on rats killed with bromadiolone, brodifacoum, or diphacinone; sublethal hemorrhaging occurred in owls fed rats killed with difenacoum. These results demonstrate potential secondary hazards of 4 anticoagulants to avian predators. No abnormalities were observed in owls fed rats killed with fumarin and chlorophacinone.

Anticoagulant poisons are the most widely used agents for control of small mammal pests throughout the world. Most applications take place against commensal rodents in cities, but their use is increasing in agricultural and forest environments where anticoagulants are replacing acute poisons (such as compound 1080). Rural applications expose a greater diversity of nontarget wildlife to potential hazards of both primary poisoning (through direct ingestion of toxic bait) and secondary poisoning (through ingestion of toxicant from tissues of target species).

Poisoned rodents are likely to be exposed to predators for extended periods during a control program using anticoagulants. In poisoning programs, anticoagulant baits are provided continuously for 10 days or longer. There is also a delay of several days between the ingestion of anticoagulant by a rodent and the appearance of overt symptoms (failure of blood clotting and onset of hemorrhage; Evans and Ward 1967, Howard et al. 1970, O'Reilly 1976). While an anticoagulant is taking effect, poisoned rodents remain active; they may continue to consume bait, and they are available to predators.

Secondary poisoning by anticoagulants has been reported for a few predators including rats (*Rattus norvegicus*) (Bull 1976, Savarie et al. 1979), a cat (*Felis domesticus*) (Mohr 1952), and possibly skunks (*Mephitis mephitis*) (McCulloch 1952). Mink (*Mustela vison*) and dogs (*Canis familiaris*) hemorrhaged when fed poisoned nutria (*Myocastor coypus*) (Evans and Ward 1967), but dogs were not affected by warfarin-poisoned mice (*Mus musculus*) (Prier and Derse 1962). Golden eagles (*Aquila chrysaetos*) displayed sublethal hemorrhaging after feeding for 5 or 10 days on mutton containing diphacinone (Savarie et al. 1979). More evaluation of the hazard for raptors is needed. The potential secondary poisoning hazards of 6 anticoagulants to owls were investigated in this study.

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[1] Chemical names of compounds are given in the Appendix.

Table 1. Secondary toxicity of diphacinone to owls.

Species	Owl wt. (g)	Mice fed to owls: Total wt. (g)	Mice fed to owls: Dose (mg)[a]	Days to death
Great-horned	1,271	175	5.5	—
	1,226	156	4.1	14
	1,135	143	4.6	14
Saw-whet	110	156	6.1	7

[a] Total toxicant consumed by mice.

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METHODS

A preliminary trial was conducted in 1970 at Olympia, Washington. Three great-horned owls (*Bubo virginianus*) and 1 saw-whet owl (*Aegolius acadicus*) were fed diphacinone-killed mice (*Peromyscus maniculatus*). Mice had consumed a lethal dose of toxicant during a 10-day, free-choice bioassay. Each individually caged mouse was fed 1 g daily of an oat-groat bait containing 0.01% diphacinone (Howard et al. 1970). Bait consumption was recorded daily; undosed Purina Lab Chow® and water were available ad libitum throughout the bioassay. Owls were each fed 2 diphacinone-killed mice daily for 5 days. The birds were fed chicken heads before the trial, after each daily treatment with dosed mice, and during a subsequent 20-day observation period. An index of whole-blood coagulation time was measured in all owls on days 0 (pre-treatment) and 8, and in 1 great-horned owl, on days 15 and 22. Blood (0.1 cc or less) was collected from the brachial vein in a nonheparinized microhematocrit tube, and was teased with a hooked needle until the first strand of fibrin appeared. The elapsed time provided an index of coagulation time that was reproducible to ±1 min for normal coagulation. Normal times were approximately 2.0 min.

In the principal experiment, 36 barn owls (*Tyto alba*) were fed rats poisoned with diphacinone, chlorophacinone, fumarin, difenacoum, bromadiolone, or brodifacoum. Rats (*Rattus norvegicus, R. rattus,* and *R. exulans*) were captured in Hilo, Hawaii, and were caged individually. They were fed oat-groat baits containing registered or recommended concentrations of toxicant: 0.025% fumarin, 0.002% brodifacoum, and 0.005% other compounds. Baits were fed free-choice for 5 days (Lab Chow was available as before). Five grams of bait were provided daily for each *R. exulans,* 10 g for *R. rattus,* and 13 g for *R. norvegicus*; bait consumption was recorded daily.

There were 4 feeding regimes, in which anticoagulant-killed rats were fed to owls for periods of 1, 3, 6, or 10 days. These periods represented the range of exposure that seemed likely to occur in the field. Undosed rats killed with CO_2 were then provided in each regime for a total of 20 days of feeding from the start of treatment. One or 2 rats were fed to each owl every afternoon; portions not eaten were weighed and recorded the next morning, including an estimate of the leftover fractions of alimentary tract (containing possible unabsorbed toxicant) and of liver (containing the majority of absorbed residues; Evans and Ward 1967). The experiment was run in 3 sections: (1) feeding of dosed rats for 1 and 6 days, (2) feeding of dosed rats for 3 and 10 days, and (3) replicate of (2). In each section there was 1 owl per toxicant for each feeding regime (6 toxicants × 2 regimes = 12 treated owls) plus 2 controls. All 3 species of rats were fed to each owl in sections 1 and 2; in section 3 all rats were *R. exulans.*

Toxicants present in rats fed to owls were not quantified. Amounts of toxicants originally consumed by rats are listed in Tables 1 and 2, but part of each compound was presumably metabolized and excreted before death.

Owls were obtained from the breeding colony at Patuxent Wildlife Research Center, and were housed during the experiment in individual indoor cages measuring 55 × 75 × 61 cm. Owls were accustomed to the cages and to a diet of rodents before the experiment. Birds that did not adapt well to pre-experimental conditions (that lost weight or were easily agitated and therefore most vulnerable to bruising and possible hemorrhage) were replaced before the start of treatment. Pre-experimental weights ranged from 425 to 605 g.

Coagulation indices were measured 8 days before first treatment with dosed rats, 20 days after first treatment, and (part 2 only) on the 3rd day after the end of treatment. Pre-test coagulation times ranged from 0.25 to 3.35 min ($\bar{x}$ = 0.75 min, SE = 0.10). Birds that died during the experiment were necropsied on the day of death. Owls that survived to day 20 were sacrificed with chloroform (after measurement of coagulation index) and necropsied.

RESULTS

All 4 owls in the preliminary trial displayed anticoagulant poisoning, and 3 died from massive hemorrhaging on days 7–14 (Table 1). Coagulation indices on day 8 (3 days after the end of treatment) were elevated by 22–34+ min; in the survivor, recovery was only partial by day 15 (6 min).

In the principal experiment, 6 barn

Table 2. Secondary toxicity of 6 anticoagulants to barn owls. The full range of doses is shown for the first 3 toxicants; for the last 3 (no effect), only the maximum dose is shown.

Toxicant	Days dosed	Owls		Rats offered		Rats eaten			Intox. signs[b]
		Wt. (g)	Sex	Total wt. (g)	Dose (mg)[a]	Total wt. (g)	Livers	Intestines	
Difenacoum	1	495	M	72	1.74	66	1	0.2	—
	3	430	M	336	6.42	270	3	2.8	—
	3	480	F	189	4.54	125	2.2	3	—
	6	495	M	586	9.81	174	1.2	2.5	H
	10	510	F	1,160	12.54	567	4.8	5.5	H
	10	540	F	595	7.99	477	10	5.8	H
Bromadiolone	1	460	M	118	2.65	52	1	0.8	—
	3	450	M	358	6.60	281	3	3	—
	3	425	M	228	3.96	146	3	2.8	—
	6	490	M	625	11.11	295	5	4	—
	10	540	F	1,106	14.59	576	7.8	4.5	—
	10	635	F	710	9.63	463	8.5	5.2	D(11)
Brodifacoum	1	400	M	71	0.58	67	1	0.5	—
	3	430	M	400	2.50	299	3	2.5	D(8)
	3	475	M	223	1.75	154	3	1.5	D(11)
	6	505	F	580	3.84	370	5.8	3.2	D(9)
	10	470	F	814	3.15	492	6	4.8	D(8)
	10	545	F	558	3.30	368	7	3.8	D(8)
Diphacinone	10	485	F	1,195	11.69	848	10	7.5	—
	10	595	F	575	9.04	490	9.8	7	—
Fumarin	10	520	F	1,137	73.62	751	10	7.5	—
	10	595	F	654	48.89	605	10	8.5	—
Chlorophacinone	10	475	M	1,276	16.07	655	7.2	5.5	—
	10	605	F	712	9.16	576	9	3.5	—

[a] Total toxicant consumed by rat.
[b] Signs of intoxication: — = no signs, H = hemorrhage, survived, D = hemorrhage and death (number indicates day of death from start of dosing).

owls died—5 that were fed brodifacoum rats, and 1 fed bromadiolone rats (Table 2). Birds fed difenacoum rats survived, but those on 6- and 10-day feeding regimes hemorrhaged (Table 2), and 1 of them hemorrhaged severely. Other birds fed bromadiolone-killed rats, and all those fed rats poisoned with diphacinone, fumarin, and chlorophacinone survived without apparent intoxication.

Hemorrhages occurred throughout the owl carcasses, including subcutaneous areas and visceral organs. Three of the 6 had regurgitated blood. Four had extensive bruises on the insides of the limbs, apparently due to normal pressure against the body; 1 had also bled copiously from the site where blood had been sampled 17 days before. Internal hemorrhages occurred in the long bones (3 birds), on the interior of the body wall (3 birds), in intestinal mesenteries and fat (2 birds), adjacent to the liver (2 birds), and intraperitoneally beside the cloaca (1 bird). Cardiac lesions were found in all dead owls; there was variable hemorrhage of the heart wall, and the pericardium was distended by an abnormal amount of clear or bloody fluid. Death in all cases appeared to have followed massive blood loss from at least 1 site. Heart failure may also have contributed to death in some birds.

Sublethal lesions in 3 birds fed difen-

acoum-killed rats were similar to lethal ones, but were less numerous and severe. One bird had regurgitated blood. Hemorrhages were found in 2 birds in subcutanous areas, abdominal fat, and the interior body wall; 1 bird had bled beside the cloaca and 1 at a small spot on the heart. Pericardial fluid appeared normal. The remaining 27 treated birds and all controls had no hemorrhages.

The coagulation index was elevated in the individual sampled shortly before death (10 min). Coagulation times for survivors 3 days after treatment were within normal range ($\bar{x}$ = 0.83 min, SE = 0.17), even in the 3 birds that hemorrhaged. This may indicate either that clotting response in survivors returned to normal within 3 days after treatment in the principal experiment or that a more sensitive measure of clotting time under these conditions is needed.

Birds that died behaved normally until 24 h or less before death, when they became lethargic and stopped eating. Weights at death were not significantly different from those for the same individuals before the experiment (paired t = 0.479, $P > 0.10$). Survivors gained weight slightly but significantly (mean gain = 21 g, paired t = 4.554, $P < 0.001$).

DISCUSSION

We have demonstrated a potential hazard to avian predators of secondary poisoning by 4 anticoagulant rodenticides. Brodifacoum was lethal to 5 of 6 barn owls, and bromadiolone was lethal to 1 of 6. Difenacoum produced sublethal hemorrhaging in the 3 birds that were dosed at least 6 days. Diphacinone—the only compound tested on 3 species of owls—was toxic to great-horned and sawwhet owls during the preliminary trial, but not to barn owls in the principal experiment. Potential weight-specific diphacinone consumption of barn owls was higher than that of great-horned owls. (Potential diphacinone consumption was computed as toxicant consumed by rodents offered to each owl, times the fraction of rodents consumed by the bird, divided by the bird's weight; data in Tables 1 and 2.) This suggests, at first glance, that secondary toxicity of diphacinone to barn owls was lower than to the other 2 species under our conditions. Possible explanations for such a discrepancy include interspecific differences in susceptibility among the owls, or differences in prey species and hence in metabolites presented to them (Townsend and Tarrant 1977). However, since the protocols differed for treatment of the owl species, further tests using a consistent protocol are needed before we can draw any conclusions on interspecific differences in toxicity.

The effects of anticoagulants on raptors in the field remain to be assessed. However, our results suggest that a potential hazard is likely to exist under some conditions. The amount of toxicant ingested from bait stations by rodents in the field is probably similar to that under our regime (free-choice bioassay of each bait at registered or recommended concentrations); an intensive, large-scale control program also exposes predators to poisoned prey for a number of days. If sufficient prey of affected species were taken by a raptor, secondary poisoning would occur. The effect of a given dose on birds in the field may not be the same as under our conditions; impact of the poison may be either greater or less than in the laboratory, but more severe effects can be expected in some cases. Susceptibility to anticoagulants can be exacerbated by stress, changes in diet (Colvin and Wang 1974, Laliberte et al. 1976), or

increased activity (Oliver and Wheeler 1978). Minor injury can also increase susceptibility (Savarie et al. 1979), even if the injury precedes exposure by many days, as in our bird that hemorrhaged at the site where blood had been sampled.

RECOMMENDATIONS

Caution is needed in the use of anticoagulants for rodent control where avian predators may be exposed to poisoned prey. Some combinations of toxicant, predator species, and prey species are highly lethal to the predator. Others may cause little hazard, but a difference in any one of these variables may be critical, and each combination of toxicant and prey to which a predator may be exposed should be tested before safety can be assumed. This is of particular concern where rare or endangered avian predators may be involved.

APPENDIX

Anticoagulants discussed include 5 registered as rodenticides in the U. S.: warfarin (3-[α-acetonylbenzyl]-4-hydroxycoumarin), diphacinone [2-(diphenylacetyl)-1,3-indandione], chlorophacinone (2-[(p-chlorophenyl)phenylacetyl]-1,3-indandione), fumarin [3-(α-acetonylfurfuryl)-4-hydroxycoumarin] and brodifacoum (3-[3-(4′-bromo[1,1′-biphenyl]-4-yl)-1,2,3,4-tetrahydro-1-naphthalenyl]-4-hydroxy-2H-1-benzopyran-2-one); and 2 experimental ones: difenacoum [3-(3-p-diphenyl-1,2,3,4-tetrahydro-1-naphthyl)-4-hydroxycoumarin] and bromadiolone (3-[3-(4′-bromo[1,1′-biphenyl]-4-yl)-3-hydroxy-1-phenylpropyl]-4-hydroxy-2H-1-benzopyran-2-one). Brodifacoum was registered between the time of the principal experiment and completion of the manuscript. Use of trade names does not imply endorsement of commercial products by the Federal government.

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