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10/29/81

DATA EVALUATION RECORD

1. CHEMICAL: Brodifacoum, Diphacinone, Chlorophacione, Fumarin, Difenacoum, Bromadiolone

2. FORMULATION: 0.025% fumarin, 0.002% brodifacoum, the rest 0.005% A.I formulations.

3. CITATION: (Note: This Article was submitted in a draft form by ICI Americas Inc. However, we had access to the published article and utilized it for this DER.) Mendenhall, V.M. and Pank, L.F. (1980). Secondary Poisoning of Owls by Anticoagulent Rodentcides. Wildlife Society Bulletin 8(4) pp 311-315. Acc# 245704.

4. REVIEWED BY: Russel Farringer
   Wildlife Biologist
   EBB/HED

5. DATE REVIEWED: 10/29/81

6. TEST TYPE: Secondary Poisoning

   A. Test Species: Great-horned, saw-whet and barnowls

7. REPORTED RESULTS:

   See attached Tables from above reference.
   Reference attached.

8. REVIEWER'S CONCLUSIONS: This study demonstrated that Bromadiolone, Brodifacoum and diphacinone (0.01%) caused secondary mortality to owls. Difenacoum cause hemoragging in owls. Diphacione (0.005%), Fumarin and Chlorophacinone did not appear toxic to barn owls.

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Material & Methods

Test Procedure (from above reference)

Apreliminary trial was conducted in 1970 at Olympia, Washington. Three great-horned owls (Bubo virginianus) and I 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 $\pm$ 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. 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% broadifacoum, 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; batit 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 x 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 x 75 x 61 cm. Owls were accustomed to the cages and to a diet of rodents before the experiment. Birds that did not adapt well to preexperimental 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.

Table 1. Secondary toxicity of diphacinone to owls.

Species	Owl wt. (R)	Mice fed to owls		
		Total wt. (g)	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.

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 Wt. (g)	Owls Sex	Rats offered Total wt. (R)	Rats offered Dose (mg)[a]	Rats eaten Total wt. (R)	Rats eaten Livers	Rats eaten Intestines	Intox signs
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	541	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, survided, D = hemmorrhage and death (number indicates day of death from start of dosing).

Reviewer's Conclusions

Anticoagulent rodenticide could pose a secondary hazard to avian predators in the field. Even those rodenticides which didnot cause death in this laboratory study could cause the death of avian predators where the following conditions (one or all) exist.

1) Stress on the avian predator due to climitac conditions.

2) Increased activity due to lowprey base or breeding season activities.

3) Prey species readily available in treatment area allowing avian predators the increase exposure potential.

4) Smaller prey allow for more potential doses over time and the greater potential of a mega dosed prey being taken.

5) Prey species would be available to avian predators due to the nature of anticoagulents which can take as long as 14 days (some cases more) to cause death.