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Effects of Sodium Cyanide and Diphacinone in Coyotes (*Canis latrans*): Applications as Predacides in Livestock Toxic Collars[1]

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Potential environmental contamination and secondary hazards to nontarget animals are controversial issues affecting the use of poisons in predator control. However, application of poisons in toxicant-filled, livestock collars is both "selective" and "specific" for animals causing depredations (see CONNOLLY et al. 1976, CONNOLLY 1976, CONNOLLY et al. 1978, SAVARIE & STERNER, in press). Environmental contamination with predacides is reduced through limited dispensing of toxicants.

The current research sought to compare: (1) the toxic effects of sodium cyanide and the anticoagulant, diphacinone, in coyotes and (2) the effectiveness of NaCN and diphacinone 2-(diphenylacetyl)-1H-indene-1,3(2H)-dione toxic collars for dosing sheep-attacking coyotes.

METHODS

Animals. Test animals were wild-trapped coyotes weighing between 7 and 15 kg. Ten coyotes were used to assess acute oral toxicity and six sheep-attacking coyotes were used in the tests with toxic collars. Throughout the study, coyotes were maintained on ad libitum Purina Dog Chow and water, and were housed in individual pens (3.0 x 1.5 x 1.8 m) with attached shelter boxes (1.5 x 0.8 x 0.7 m). Basic toxicity tests were conducted in the individual pens; toxic-collar tests were conducted in a nearby 31 x 40 m fenced enclosure. To increase the likelihood of attack, the six animals used in toxic-collar tests were food deprived for 72 h prior to pairing with sheep (15 to 30 kg).

Materials. NaCN was formulated as a 33% wt/vol solution in distilled water; the diphacinone solution (Motomco, Inc., Clark, NJ) was a commercial mixture of 5% diphacinone in water.

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[1] This research was supported by an interagency agreement between the U.S. Environmental Protection Agency and U.S. Fish and Wildlife Service (IAG-D6-0910). The current work is derived from the concept of McBRIDE (1974); he patented a leather-sheathed neckband for sheep designed to hold a bladder of poison and reduce environmental contamination from predacides through the selective, specific dosing of attacking coyotes. Reference to trade names does not imply government endorsement of commercial products.

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Toxic collars consisted of four 20-mil polyvinylchloride packets (Elastomer Products, Inc., Denver, CO), each measuring 12.5 x 4.5 cm (see CONNOLLY et al. 1978, SAVARIE & STERNER, in press). Each packet contained 50 mL of the respective solutions. Two packets were laced together with nylon cord and tied lengthwise along each side of the sheep's neck. Collars were camouflaged by gluing a 6-8 mm layer of wool to the outer surface.

Acute-oral-toxicity procedures.--The five coyotes in the NaCN group were randomly assigned to receive 4, 8, 16, 32, or 64 mg/kg of NaCN. Doses were delivered to the back of the coyote's mouth with a 1-mL disposable syringe attached to a 4.5-cm intubation needle. As soon as breathing stopped, the coyote was necropsied; a 5-mL venous blood sample and the entire esophagus-stomach was removed for analysis. One sublethally dosed coyote, still alive 30 min after dosing, was sacrificed by an intracardial injection of pentobarbitol sodium. Samples were frozen and analyzed for NaCN by a commercial laboratory (Poisonlab, Denver, CO) according to the methods outlined by BLANKE (1970).

The five coyotes in the diphacinone group were similarly dosed with either 0.3, 1.2, 2.4, 4.8, or 9.6 mg/kg diphacinone. Venous blood samples (5 mL) were taken from each coyote 1 h before dosing and at 72 and 144 h after dosing; these were frozen and analyzed for residues by a commercial laboratory (The Industrial Laboratories Co., Denver, CO) by the method of BULLARD et al. (1976). Separate 5 mL venous blood samples, taken 1 h before and 48, 96, 144, 216, 288, 360, and 408 h after dosing, were analyzed for prothrombin clotting time by the Quick method (MONKHOUSE 1961).

Toxic-collar procedures.--Six coyotes, known to attack sheep in laboratory pens, were randomly assigned to either a NaCN (n = 3) or diphacinone (n = 3) group. Each was then released for a maximum of 60 min/day into a pen containing a live unrestrained sheep fitted with either a NaCN or diphacinone toxic collar. These pairings were continued daily until each coyote attacked the sheep and punctured a collar.

Toxicity and behavioral measures in these tests were: time spent mouthing the collar, time to immobilization, time to death, and chemical residues (i.e., NaCN in blood and esophagus-stomach; diphacinone in blood). Blood and tissue sampling schedules, collection procedures, and residue analyses were the same as those used in the basic toxicity tests. Because of the slow action of diphacinone, coyotes in this group were allowed to feed on the downed sheep for 15 min, then returned to their holding cages; these coyotes were monitored daily until death or for a period of 30 days (i.e., survival criterion). Behavioral measurements were obtained from videotape recordings of each trial.

RESULTS AND DISCUSSION

Acute oral toxicity of NaCN. Acute toxicity data for NaCN are summarized in Table 1. The coyote given 4 mg/kg NaCN survived initial dosing and was sacrificed; the four given 8 mg/kg or more developed ataxia within 2 min and died within 41 min. Although there is little published information on the toxicity of NaCN in canids, these data agree with two previous reports involving similar formulations: CHEN & ROSE (1952) found an LD_{50} (subcutaneous) of 5.36 mg/kg NaCN for dogs, and SAVARIE (1977) found an LD_{50} (with 95% confidence interval) of 4.1 (2.1-8.3) mg/kg for coyotes.

Acute oral toxicity of diphacinone. Table 2 presents acute toxicity data for diphacinone. Four coyotes survived dosages of 4.8 mg/kg or less of diphacinone; whereas the coyote given 9.6 mg/kg died after 10 days. These data are in agreement with those of BENTLEY & LARTHE (1959) and GATES (1957) that indicated an LD_{50} for dogs of between 5 and 15 mg/kg.

Predose residues of diphacinone were less than 0.001 ppm (the detection threshold of the analytical procedure) for all animals; however, the 72 h residues ranged between <0.001 ppm and 0.005 ppm, and the 144 h residues ranged between <0.001 ppm and 0.003 ppm.

Figure 1 presents the prothrombin response curves for the five coyotes given known dosages of diphacinone. Predose clotting times were highly consistent for all animals, ranging between 8.6 and 9.7 sec. Peak clotting times of between 40-80 sec occurred between 72 and 196 h after dosing for the sublethally-dosed coyotes. The clotting time for the lethally dosed coyote (9.6 mg/kg diphacinone) increased to 111 sec approximately 144 h after dosing.

Toxic collar applications of NaCN and diphacinone. The lower portions of Tables 1 and 2 provide a comparison of toxicity when coyotes were dosed via NaCN or diphacinone collars during attacks on sheep. Only one of the coyotes that punctured an NaCN collar (NaCN-1) ingested a lethal dose of the poison, but all three coyotes in the diphacinone group received lethal amounts of the anticoagulant.

The time to death and time to immobilization data illustrate the drastic difference in speed of action for the two compounds. The lethally dosed NaCN coyote survived only 18 min, versus about 8-15 days for the three diphacinone-dosed animals. Similarly, immobilization (the time at which the coyote was no longer ambulatory) occurred between 1 and 18 min after dosing for the NaCN-dosed coyotes but between 8 and 15 days for the diphacinone group ($t = 5.26$, $df = 2$, $p < 0.05$). Whereas puncture of an NaCN collar interrupted attacks within 15 sec, coyotes in the diphacinone group showed prolonged, sometimes recurrent, mouth contact of up to 2.2 min with punctured collars ($t = 13.05$, $df = 4$, $p < 0.05$).

TABLE 1

Toxicity measurements for coyotes receiving a single forced-oral or toxic-collar dose of NaCN.

Test coyote No.	Sex	Weight (kg)	NaCN dosage (mg/kg)	NaCN residues: Whole blood (ppm)	NaCN residues: Esophagus-stomach (ppm)	Time to death (min)	Time to immobilization (min)	Time mouthing collar (min)
Forced-Oral Dose								
NaCN-1	♂	14.5	4	.032	0.9	Sublethal	13.0	
NaCN-2	♂	9.5	8	.036	0.5	41.0	9.0	
NaCN-3	♀	10.5	16	.009	0.5	8.0	1.0	
NaCN-4	♀	9.5	32	.140	4.0	2.5	1.0	
NaCN-5	♀	7.5	64	.070	13.0	5.0	1.0	
Toxic-Collar Dose								
NaCN-6	♂	12.5		.026	<0.1	18.0	1.0	0.03
NaCN-7	♂	11.0		.014	0.6	Sublethal	18.0	0.05
NaCN-8	♂	9.5		.029	<0.1	Sublethal	2.7	0.25

TABLE 2

Toxicity measurements for coyotes receiving a single forced-oral (known amounts) or toxic-collar dose (unknown amounts) of diphacinone.

Test coyote No.	Sex	Weight (kg)	Diphacinone dosage (mg/kg)	Diphacinone residues in whole blood (ppm) 72 h	144 h	Time to death (days)	Time to immobilization (days)	Time mouthing collar (min)
Forced-Oral Dose								
DIPH-1	♂	8.6	0.3	0.005	<0.001	Sublethal	Not immobilized	
DIPH-2	♀	12.7	1.2	<0.001	0.002	Sublethal	Not immobilized	
DIPH-3	♀	10.9	2.4	0.002	<0.001	Sublethal	Not immobilized	
DIPH-4	♀	7.5	4.8	0.002	0.003	Sublethal	Not immobilized	
DIPH-5	♂	10.9	9.6	0.002	<0.001	≃ 10	≃ 9	
Toxic-Collar Dose								
DIPH-6	♂	11.8		0.028	0.005	≃15	≃14	0.2* 0.3* 2.0*
DIPH-7	♀	9.5		0.004	<0.001	≃12	≃11	2.2
DIPH-8	♂	14.5		0.008	0.003	≃ 8	≃ 8	1.0* 1.7*

* Multiple contacts with the toxic collar occurred throughout the attack; total time spent mouthing the collar is the sum of these measurements for the respective coyotes.

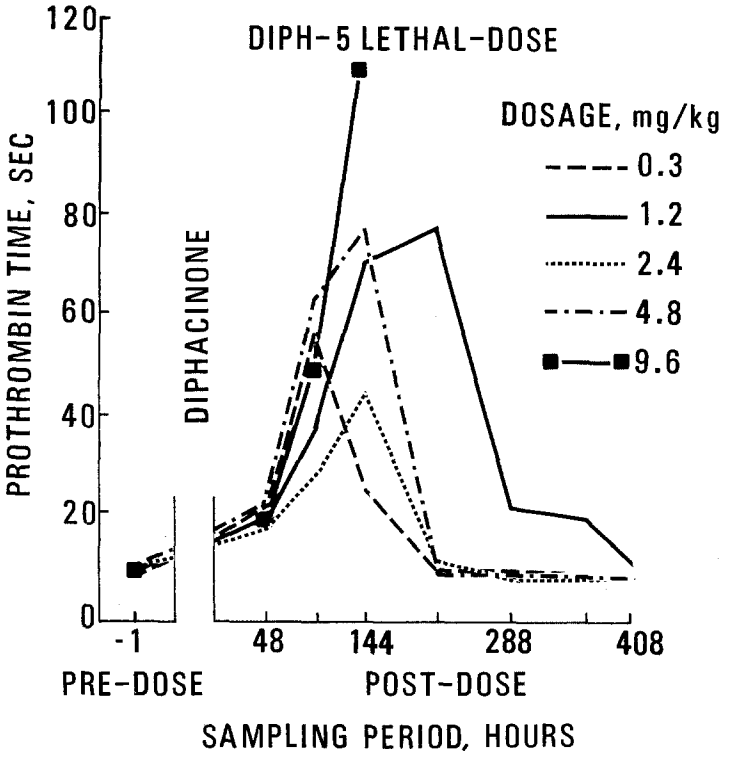

Fig. 1. Prothrombin measurements for each of the five coyotes administered forced-oral dosages of diphacinone. (Note--Dosages of 0.3, 1.2, 2.4, and 4.8 mg/kg proved sublethal; the coyote dosed with 9.6 mg/kg died approximately 10 days after dosing.)

Together, the current results point out the dilemma posed by the use of the fast-acting, noxious NaCN and slow-acting, less noxious diphacinone. Advantages gained in the volume of diphacinone delivered and length of collar contact obtained are compromised by problems associated with field assessments of collar effectiveness (see CONNOLLY et al. 1978). When coyotes die several days after dosing, assessments of effectiveness are difficult; lethally dosed animals may kill additional sheep before they succumb. Thus, despite the advantage of specific dosing of coyotes that attack sheep, the toxic-collar approach to coyote-damage control needs further work. Successful application appears to depend upon development of a fast-acting toxicant.

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