April 1987

Laboratory Trial of Chlorophacinone as a Prairie Dog Toxicant

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Laboratory Trial of Chlorophacinone As a Prairie Dog Toxicant[1]

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Abstract.--A laboratory trial was conducted to investigate the efficacy and secondary toxicity of chlorophacinone oats as a prairie dog toxicant. Bait containing 0.0025% chlorophacinone killed 29 of 31 prairie dogs when offered in 25 gram amounts daily for 6 days. Five of 6 domestic ferrets died of anticoagulant poisoning when fed 4 of these toxicant-killed prairie dogs over 8 days. Chlorophacinone may not be an acceptable prairie dog toxicant due to this potential secondary hazard.

INTRODUCTION

In numerous places throughout their range, black-tailed prairie dog (Cynomys ludovicianus) populations have been increasing in recent years. While these increases may have multiple causes, some authorities point to increased restrictions on the use of toxicants, including the 1972 Presidential Executive Order which limited toxicant use on public lands (Fagerstone 1982). In western Nebraska, prairie dog populations may have increased as much as 60% from 1970 to 1980 (Nebraska Game and Parks, unpubl. data).

Prairie dogs' feeding activities can alter the vegetative composition of rangeland plant communities, resulting in reduced forage productivity (Hansen and Gold 1977). While it is generally believed that prairie dogs and livestock can compete for forage, the amount of competition may vary from site to site (Fagerstone 1982) and from year to year. There are few studies that document the economic impact of these rodents on rangeland.

Despite the absence of such economic assessments, many landowners believe prairie dog control to be desirable. The most cost-effective and practical method of rapidly reducing prairie dog populations is by application of toxic grain bait. Zinc phosphide and strychnine are the only active ingredients presently used in federally registered prairie dog baits (Jacobs 1983). Two fumigants, aluminum phosphide and gas cartridges, are currently available for burrow fumigation. The higher cost and relatively non-selective action of fumigants makes them a viable control option only on small areas or as a follow-up to toxic grain bait treatment.

The efficacy of strychnine and zinc phosphide baits is variable and often control results are not as successful as desired (Holbrook and Timm 1985). "Poor success of toxicant use against prairie dogs often results from such causes as failure to prebait, alternate food resources, weather changes during bait application, and repeated use of toxicants on bait-shy populations. Further, concerns have been raised about the potential hazard of currently-registered toxicants to non-target species, particularly the endangered black-footed ferret (Mustela nigripes). Clearly, alternative prairie dog toxicants are needed.

The purpose of this study was to investigate, in the laboratory, the potential of the anticoagulant chlorophacinone as a prairie dog toxicant. We wanted to find an appropriate bait concentration, determine its effectiveness against prairie dogs, and investigate its secondary toxicity.

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[1]Paper presented at the Eighth Great Plains Wildlife Damage Control Workshop [Rapid City, S.D., April 28-30, 1987].

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BAIT FORMULATION

We live-trapped wild black-tailed prairie dogs from Morrill County, Nebraska. They were weighed, dusted with the insecticide Sevin, and housed in individual metal cages. We fed them Wayne Rodent Blox (Wayne Pet Food Division, Continental Grain Co., Chicago, Ill.) ad lib. and gave them watermelon or sugar beet slices as a source of moisture. We offered the animals untreated crimped oats daily while we acclimated them to the laboratory. Only animals which accepted oats were used in subsequent trials.

To determine the lowest effective bait concentration, we formulated chlorophacinone at three concentrations, 0.01%, 0.005%, and 0.0025% active ingredient (a.i.). Two percent chlorophacinone concentrate (RoZol Dry Concentrate, Chempar Products, New York) was suspended in corn oil and the solution mixed with crimped oats, by hand, until it appeared to be mixed evenly.

Twenty-four prairie dogs which had readily consumed untreated oats were randomly assigned, eight to each of the 3 bait formulations. Twenty-five grams of the respective bait formulation was offered to each prairie dog daily, for 6 consecutive days. The amount of treated oats remaining was recorded daily for each animal. The laboratory chow was not available during the 6 day baiting, while the water source continued to be offered. Following the six days of baiting, the prairie dogs were returned to their laboratory rodent chow and water source diet. They were observed for 21 days or until death occurred. Carcasses of all anticoagulant-killed prairie dogs were frozen upon death. The identity of each prairie dog was maintained throughout the trial.

Each of the bait formulations tested caused total mortality of the test animals. The lowest concentration (0.0025% a.i.) was chosen for further evaluation. Additional dosed prairie dogs were needed to provide sufficient numbers of poisoned prairie dogs for testing of secondary toxicity. Twenty-three additional prairie dogs were offered the 0.0025% bait concentration, following the same procedure as outlined above. Twenty-one prairie dogs died of anticoagulant poisoning while 2 survived beyond the 21-day observation period. The animals which died as a result of the 0.0025% treatment had consumed dosages between 1.3 and 5.5 mg/kg. Of the surviving animals, one consumed relatively little of the treated oats (0.4 mg/kg), while the other consumed a greater quantity than did 17 other test animals which subsequently died.

SECONDARY TOXICITY

Any toxicant that is to be newly registered for prairie dog control will necessarily undergo detailed scrutiny concerning potential non-target hazards. The potential presence of the endangered black-footed ferrets in prairie dog towns underscores this concern. We chose domestic ferrets (Mustela putorius) as surrogate test animals for our secondary toxicity evaluation.

Eight domestic ferrets, 4 of each sex, were housed individually in metal cages. Purina Cat Chow and water were available ad lib. during acclimation to the laboratory.

One male and one female ferret were randomly chosen to serve as controls. All ferrets were given 3 thawed, untreated prairie dog carcasses, one every other day, to condition them to eating prairie dogs. In order to more quickly induce feeding behavior, we had to partially skin the rodent carcasses. The skin on the thawed prairie dogs was sliced along the belly, and peeled off one side, to expose underlying tissue, taking care not to cut into the abdominal cavity. This procedure was followed on all subsequent prairie dog carcasses offered to all ferrets.

Following this conditioning regime, we gave each treatment ferret 4 prairie dog carcasses poisoned with 0.0025% chlorophacinone bait, one every other day, while the control ferrets received 4 unpoisoned carcasses. The consumed portions of each treated prairie dog were noted as it was removed from the ferret cage. The Cat Chow diet was not available to the ferrets during the period when prairie dog carcasses were offered.

The ferrets were returned to the Cat Chow diet following removal of the last treated prairie dog. Ferrets were then observed for 30 days, or until death occurred. Five of the 6 treatment ferrets died of anticoagulant poisoning, as verified by veterinary necropsy. Internal hemorrhaging was found in the neck and thoracic region in each of the poison-killed ferrets. We observed that all ferrets fed on internal organs as well as muscle tissues of the prairie dogs during the treatment phase. Toxicological analyses of ferret and prairie dog tissues are being conducted, and these results will be published elsewhere.

DISCUSSION

Chlorophacinone-treated oats were found to be an effective prairie dog toxicant at 0.0025% a.i., a concentration lower than that in chlorophacinone baits currently registered for use against pocket gophers and commensal rodents. From this standpoint, it would appear that this compound could provide a useful alternative to strychnine and zinc phosphide. Bait shyness should not be a problem when using an anticoagulant, and there should be no need to prebait. However, more than one field application may be necessary to insure that sufficient bait would be present to be eaten over a number of days. Alternatively, the bait could be made available in weather-resistant bait stations, which would be advantageous especially when attempting to prevent prairie dog town expansion at town perimeters or across property lines.

The secondary toxicity of chlorophacinone to domestic ferrets consuming poisoned prairie dogs, at the dosages we tested, indicates that this compound may not be acceptable. On the basis of our study, we believe it would be unwise to use chlorophacinone baits at these dosages against prairie dogs, unless black-footed ferrets are proven absent from the treatment area and it can be demonstrated that potential secondary toxicity poses no significant hazard to other non-target populations.

We do not automatically conclude, however, that all anticoagulants are unsuitable as prairie dog toxicants because of potential secondary hazard. Other compounds may be metabolized differently by prairie dogs and may be of differing toxicity to non-target species. We believe that because of their potential value in cost-effective control, other anticoagulants should be evaluated for prairie dog control.

ACKNOWLEDGEMENTS

We thank R.M. Case and R. Poche for review of an earlier draft of this paper. Cooperation of the staff of the University of Nebraska Panhandle Research and Extension Center is appreciated. We thank S. Kamble for assistance in obtaining funds to conduct this work. This research was funded in part by Pesticide Impact Assessment funds made available to the University of Nebraska by USDA.

LITERATURE CITED

Fagerstone, K.A. 1982. A review of prairie dog diet and its variability among animals and colonies. p. 178-184. In Proc. Fifth Great Plains Wildlife Damage Control Workshop, [Lincoln, Neb., Oct. 13-15, 1981]. R.M. Timm and R.J. Johnson, eds., Univ. of Nebraska.

Hansen, R.M. and I.K. Gold. 1977. Blacktail prairie dogs, desert cottontails and cattle trophic relations on shortgrass range. J. Range Manage. 30(3):210-214.

Holbrook, H.T. and R.M. Timm. 1985. Comparisons of strychnine and zinc phosphide in prairie dog control. p. 73-79. In Proc. Eastern Wildl. Damage Cont. Conf., [Raleigh, No. Carolina, Sept. 22-25, 1985]. P.T. Bromley, ed.

Jacobs, W.W. 1983. Pesticides federally registered for control of terrestrial vertebrate pests. p. G1-G29. In Prevention and Control of Wildlife Damage, Great Plains Agricultural Council and Nebraska Cooperative Extension Service, R.M. Timm, ed.