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LABORATORY STUDY ON BROMADIOLONE: EFFECTIVENESS ON PRAIRIE DOGS AND SECONDARY HAZARDS TO DOMESTIC FERRETS

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INTRODUCTION

Bromadiolone is a second generation anticoagulant that is currently registered as a toxicant controlling commensal rodents. This laboratory study was conducted to provide us with preliminary information on the effectiveness and secondary hazards of bromadiolone as a prairie dog *(Cynomys* spp.) toxicant. We planned to use this information to decide on the appropriateness of pursuing field research projects using this compound. For an overview on bromadiolone, see Poche (1986).

This was the second laboratory study we conducted on alternative prairie dog toxicants. The first, on chlorophacinone, was reported earlier (Fisher and Timm 1988). Our desire was not to directly compare the effectiveness of bromadiolone to chlorophacinone, but to determine if we could find a safe and effective bait formulation that could be tested later in the field.

METHODS

Twenty-two black-tailed prairie dogs *(C. ludovicianus)* were live-trapped in Morrill County, Nebraska. They were weighed, dusted with the insecticide, Sevin to control ectoparasites, and placed in individual metal cages. We fed them Wayne Rodent Blox (Wayne Pet Food Division, Continental Grain Co., Chicago, 111.) ad lib. and provided sugar beet slices daily as a source of moisture. We also offered the animals untreated crimped oats while they acclimated to the laboratory.

Primary Toxicity

We decided to use a bromadiolone bait containing 0.0025% a.i. to follow the proto col used in our previous trial investigating chlorophacinone. The manufacturer provided us with a hulled, whole oat bait containing approximately 0.0025% a.i. Maki brand of bromadiolone (Chempar Products,

Lipha Chemicals, Inc., New York). We collected random samples of the bait for laboratory analysis for bromadiolone concentration. The concentration was determined to be 22.8 ppm or 0.00228% a.i. (C.T. Male Associates, P.C., Latham, New York).

Fifteen grams of the bait was placed in glass food dishes and offered, individually, to 22 prairie dogs (6 females and 16 males) daily for 3 days. Any bait not consumed was weighed and recorded daily. The laboratory chow was withheld from the prairie dogs those days that bait was available. We continued to provide sugar beet slices throughout the trial. The prairie dogs were observed for 21 days or until death occurred. Prairie dog carcasses were frozen upon death. The identity of each prairie dog was maintained throughout the study.

Secondary Toxicity

Domestic ferrets were chosen as our test model for determining secondary hazards to predators and scavengers. This would, we hoped, closely approximate potential hazards to black-footed ferrets which might consume poisoned prairie dogs in the wild.

Eight domestic ferrets *(Mustela nigripes),* 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 female and 1 male were randomly chosen to serve as controls. All ferrets were given 3 thawed, untreated prairie dog carcasses, 1 every other day, to condition them to eating prairie dogs. To induce feeding behavior more quickly, the skin on the prairie dogs was sliced along the abdomen and peeled off one side, to expose underlying tissue. Care was taken not to cut into the thoracic or abdominal cavities. This procedure was followed on all subsequent prairie dogs offered to all ferrets.

Following this conditioning regime, we gave each treatment ferret 3 thawed prairie dog carcasses poisoned with the bromadiolone bait, 1 every other day. The consumed portions of each treated prairie dog were noted as it was removed from the ferret cage before subsequent carcasses were offered. The control ferrets received untreated prairie dog carcasses. The Cat Chow diet was not available to the ferrets during the period when carcasses were offered.

The ferrets were returned to the Cat Chow diet following removal of the last prairie dog carcass. Ferrets were then observed for 30 days, or until death occurred.

Tissue Residue Analysis

Following the 30 day observation period, all ferrets were sacrificed. Livers were collected for bromadiolone residue analysis. Each ferret liver was placed in a separate plastic bag, labelled for identification, and frozen.

We also collected the livers from 3 bromadiolone-killed prairie dogs not fed to ferrets. The prairie dogs, previously frozen, were thawed and livers removed. These livers were also placed in individual bags, labelled and refrozen. All livers were packed with dry ice and sent for laboratory analysis (C.T. Male Associates, P.C., Latham, New York).

RESULTS

All 22 prairie dogs died of anticoagulant poisoning. The prairie dogs ate an average of 41.2 g of bait with a range of 6

to 45 g. The total dosage of bromadiolone eaten ranged from 0.17 to 1.52 mg/kg with a mean of 1.12 mg/kg. Deaths occurred from 5 to 18 days following removal of bait, with a mean of 10.5 days. All ferrets survived beyond 30 days showing no visible signs of anticoagulant poisoning.

Bromadiolone content in the livers from the 6 treatment ferrets were determined to range from 0.2 to 0.4 ppm, with a mean of 0.3 ppm concentration. Control ferret livers were reported to contain 0.05 and 0.1 ppm bromadiolone. The 3 prairie dog livers contained 1.1, 1.35 and 1.7 ppm bromadiolone.

DISCUSSION

Hulled, whole oats, treated with bromadiolone to a 0.00228% a.i. level were found to be an effective prairie dog toxicant under our laboratory testing regime. All prairie dogs offered 15 g of this bait daily for 3 days died of anticoagulant poisoning.

Testing of secondary toxicity showed no mortality or apparent sublethal effects to domestic ferrets fed 3 bromadiolone-killed prairie dogs over 6 days. While our testing regime cannot demonstrate efficacy or safety in field situations, it would indicate that this compound is worthy of further study.

No direct comparison of efficacy or hazard should be made between bromadiolone and chlorophacinone based only on our 2 studies. Our use of differing testing regimes precluded this comparision.

Our bromadiolone trial does indicate that some anticoagulants may have potential value as prairie dog toxicants. We conclude that some anticoagulants may have potential value as prairie dog toxicants. We conclude that anticoagulants, particularly bromadiolone, deserve further study to define their efficacy and non-target safety when used as prairie dog toxicants.

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