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LABORATORY EFFICACY STUDY WITH A WARFARIN BAIT TO CONTROL THE BLACK-TAILED PRAIRIE DOG

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ABSTRACT: Control of the black-tailed prairie dog (*Cynomys ludovicianus*) is important for the reclamation of pasture ground for domestic cattle and limiting the spread of disease to humans and other wildlife. Six different concentrations of warfarin bait were fed to prairie dogs to determine mortality. Without the access to dietary vitamin K, the prairie dogs were susceptible to the warfarin bait. However, some of the prairie dogs recovered and survived the test after bait exposure was terminated. This could be due to physiological differences and the availability of fat-soluble vitamin K. The six different concentrations of warfarin consumed by the prairie dogs were correlated to the increase in treatment group ($r=0.916$). Body weight loss generally increased as the treatment group dosage increased. The control group was the only group which increased in body weight. The whole body tissue analysis of the prairie dogs from treatment groups 44.8, 233.0, and 777.6 ppm was correlated to the increase in treatment group ($r=0.709$).

KEY WORDS: *Cynomys ludovicianus*, black-tailed prairie dog, toxicant, rodenticide, warfarin, bioassay

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INTRODUCTION

Black-tailed prairie dog (*Cynomys ludovicianus*) numbers in the Great Plains were estimated at 5 billion in the early 1900s (Merriam 1902). At that period of history, they were considered to be a pest to the ranchers and farmers because of their feeding on crops and grass planted by the early settlers. Grazing biomass has been decreased by the feeding action of prairie dogs (Taylor and Loftfield 1924; Hansen and Gold 1977; O'Meilia et al. 1982; Knowles 1986). Prairie dogs are also a reservoir of disease that affect humans and other wildlife (Barnes 1982). For these reasons, the prairie dog is still being eradicated with the use of toxicants and firearms resulting in lower densities and widely scattered colonies (Clark et al. 1982; Foster and Hygnstrom 1990).

Even with the current small population of prairie dogs, the farmers and ranchers continue to use control techniques. The author is proposing warfarin as a possible answer because it is a cost effective rodenticide, it is relatively nontoxic to birds (Christopher et al. 1984; Hagan and Radomski 1953), it quickly degrades in the gastro-intestinal tract (half-life of 42 hours) (Ford 1993), it is rapidly excreted from the body (Wong and Solomonraj 1980), animals often do not develop "bait shyness" (Meister 1996), and it is relatively safe with respect to secondary poisonings (Aulerich et al. 1987).

The purpose of this study was to develop a concentration of warfarin bait from five formulations (0.005%, 0.01%, 0.025%, 0.05%, and 0.1%) that would be effective in the management of black-tailed prairie dogs.

OBJECTIVES

The objective of this bioassay test was to determine the efficacy of five concentrations of warfarin for managing black-tailed prairie dogs. Warfarin consumption was monitored to determine possible secondary hazard. Physical, behavioral, and anatomical observations were made to assess anticoagulant poisoning. Whole body tissue analysis was performed to calculate the warfarin accumulation at the time of death. Also, bait

analysis will help determine if the mixing technique is sufficient to produce a homogeneous mixture.

In a companion study, the author tested the secondary hazard potential of warfarin-poisoned prairie dogs on domestic ferrets (*Mustela putorius furo*).

METHODS

Warfarin Formulations

Five concentrations of warfarin bait were tested (0.005%, 0.01%, 0.025%, 0.05%, and 0.1%). The warfarin technical product (99% purity) was supplied by Sigma Chemical. Three warfarin concentrates were made (0.5%, 1.0%, and 2.0% warfarin) for the five different warfarin formulations to aid in obtaining a sufficient homogeneity. Five warfarin formulations were prepared: 0.005%, 0.01%, 0.025%, 0.05%, and 0.1%. The control bait, which contained 0% warfarin, was mixed in the same manner as the other formulations. The exact formulation was kept in the raw data for confidentiality.

After each ingredient was added, the mixer was operated for 20 seconds to mix the ingredients together. When large amounts of the concentrate were to be added (500 and 1,000 ppm groups), small portions of the concentrate were added and then mixed for about 20 seconds. After all of the ingredients were added, the mixer was run for 15 minutes to achieve a homogeneous mixture. Samples were collected from each formulation for freezer storage stability, animal room stability, and homogeneity. All of the formulated bait, including samples, were frozen in plastic bags. The bait was analyzed for freezer storage stability, animal room stability, and homogeneity.

Test System

All methods used in the study were approved by the Genesis Laboratories, Inc. Institutional Animal Care and Use Committee (Project #96018). Black-tailed prairie dogs were live trapped from a colony in Larimer County, Colorado. Traps were set near the burrow openings and worked into the ground to cover the metal bottom with soil. Clean rolled barley was placed on the trigger device

as well as a path leading out of the trap for a distance of about 0.5 meters. Traps were checked at least twice daily.

The prairie dogs were transported to Genesis Laboratories, Inc. in the back of a covered truck in the same trap in which they were captured. All animals were dusted with flea powder containing pyrethrin to control ectoparasites.

The prairie dogs were placed into a cloth bag and plastic container, they were then weighed on a top loading Ohaus dial-o-gram balance. The scale had been calibrated and tared for the cloth bag and the plastic container. Maturity was assessed according to body weight (675 grams and 775 grams for females and males, respectively) (Hoogland 1995). Preliminary body weight was measured on the first day of the acclimation period when the prairie dogs were assigned to treatment groups. A final assessment of maturity by body weight was taken on Day 0 and were replaced with extra animals if they were underweight. Body weights were taken at test termination or death to assess weight loss or gain.

Prairie dogs were observed visually during the acclimation period. They were randomly assigned to cages from group housing one day before the acclimation period. The individual cages had metal screen bottoms with a surface area of at least 4,650 cm² and a minimum height of 46 cm (National Research Council 1992). Prairie dogs received a basal diet of laboratory pellets (Manna Pro Lab Cubes from St. Louis, MO), rolled barley, and water *ad libitum*. The bedding and water bottles were changed and cleaned weekly, and the water and feed levels checked daily. Cages and racks were not cleaned during the study because handling of the animals could cause lesions, bruises, or injury which could bias mortality estimates (Penumarthi and Oehme 1978).

The minimum/maximum temperature and humidity of the animal room was recorded daily during the entire holding period with a calibrated digital hygrometer/thermometer. The temperature and humidity in the study room was maintained at approximately 16 to 26°C and 55 ± 25%, respectively. Ventilation was checked often.

The animals were acclimated to test conditions for 10 days prior to the warfarin bait administration. Five females and three males were placed into acclimation with the initial group after the initial start date. One male was captured one day late, and two males and five females were captured two days late. These extra animals were used in the event some of the other animals did not reach the mandatory weight limits for each sex set by the study protocol.

Animals were randomly assigned to treatment groups, using the computer program "Ran30" (Faisal, Colorado State University, Fort Collins, CO), a program designed to choose random numbers according to the number that is required.

The feeding-test period was conducted for 15 days, during which all formulations were presented until the end of the test period or death. Seventy grams of each diet was presented daily in stainless steel feed cups. The feed cups were attached to a 30 cm X 30 cm sheet of particle board to catch spilled feed and stabilize the feed cup. A flat circular fowler with 10 mm holes, used for rodents,

was placed over the bait to limit spillage. Bait consumption was measured and cups were refilled with fresh bait daily. After the exposure period, prairie dogs were fed the basal laboratory diet and observed for 10 days for signs of warfarin toxicity.

The prairie dogs were observed daily during their entire holding period. Any physical or behavioral signs that could help lead to the identification of sickness or anticoagulant poisoning during the test were recorded. The prairie dogs were observed once each day during the 10-day acclimation period. The prairie dogs were observed twice daily during the feeding-test period and post-test observation periods of the experiment. The Environmental Protection Agency (EPA) recommended the 15 day feeding and a 5 day post-test observation. A 10 day post-test was used to be certain of mortality. Necropsies were conducted on all animals that died during the test. An incision was made through the skin from the anus to the lower jaw. All major organs were observed for hemorrhaging and signs of anticoagulant poisoning in the abdominal and thoracic regions. A few cranial regions were incised to inspect for hemorrhaging.

After the prairie dogs were found dead during the test, the carcasses were labeled, wrapped in foil, and stored in a freezer for later analysis. Nine prairie dogs, three from each of the 50, 250, and 1,000 ppm treatment groups, were randomly chosen from the freezer to be analyzed for whole body residues of warfarin. This gave a representative range of the treatment groups. A laboratory-validated method was used. Also, all baits were analyzed for warfarin levels. A laboratory-validated method was used.

Statistics were performed after the completion of the test by SPSS for Windows, release 7.5. Various tests were performed based upon the capabilities and limitations of the available data. Linear regression was used to compare dependent and independent variables in relation to each other. Levene's test for homogeneity of variances was used to compare the variances within treatment groups. If the variances were significantly different, a Kruskal-Wallis non-parametric test was used to identify differences between variances. If the results of the Levene test showed the variances within the treatment groups were not significantly different, an analysis of variance (ANOVA), a parametric test, comparing treatment group means, was used to identify differences between the means of the variances.

Following analyses of variances, the Tukey's honestly significant difference test (HSD) was performed. This test identifies significant differences of the means of the treatment groups. The test shows which treatment groups are similar and which are different from the others. Significance values classify the significant difference between the treatment groups which are most distant from each other.

The computer program "Probit" (Charles Breidenstein, former section chief of statistics, Denver Wildlife Research Center) was used to calculate the LC₅₀ and LC₉₀ values.

RESULTS

The warfarin baits used in this study were formulated to be a nominal concentration of 50, 100, 250, 500, and

1,000 ppm. The validated analytical extraction procedure resulted in warfarin baits of actual concentrations of 44.8, 89.5, 233.0, 407.0, and 777.6 ppm. For the remainder of the report, the actual concentrations will be used for all calculations and presented results.

The daily observations taken during the exposure period included symptoms of diarrhea, ataxia, immobility, hemorrhage, hyperreactive, hyporeactive, bloody stool, labored breathing, hind limb paralysis, moribund, and found dead. During the first five days of the exposure period, only one female prairie dog showed a sign affiliated with warfarin intoxication—lethargy. On days 6 through 10, the prairie dogs began showing signs of hemorrhaging (nose, mouth, anus) or lethargy, with increasing severity as the study progressed. The first death was on day 8 of the exposure period. On days 11 to 15, the severity of the symptoms continued to progress with more individuals becoming moribund or found dead.

After the feeding test exposure, the animals were placed in a 10-day post-test observation period. Some of the animals continued to deteriorate physically, while some of the animals began to show improvements in their physical condition. In an extreme case, M23, in the 233.0 ppm treatment group, was classified as being "moribund" (M) for 13 days, and then upgraded to lethargic (HY) on day 8 of the post-test observation period.

Variances among the treatment groups differed significantly ($P=0.128$). An ANOVA showed a difference between the treatment groups ($P<0.000$). Tukey's HSD test identified a difference between the control group and the treated groups, and only a small difference among the treated groups (Tukey's HSD=0.430, $P<0.05$) (Figure 1).

Figure 1. Total consumption of bait (grams) by prairie dogs fed six different concentrations of warfarin bait. The prairie dogs were fed for 15 consecutive days in a no-choice regime. Each square represents the consumption of one prairie dog.

Warfarin consumption was highly correlated ($r=0.916$) with the concentration of warfarin baits presented (Figure 2). A Levene test of homogeneity of variances showed that there is a difference between the variances among the treatment groups differed significantly (11.647, $df_1=5$,

$df_2=54$, $P<0.000$). A Kruskal-Wallis test indicated that the treatment means were different ($P>0.000$). Consumption of the two highest concentrations was significantly greater (Tukey's HSD=1.000, 10.00 at $P<0.05$) than the three lower concentrations.

Figure 2. Consumption of warfarin (mg/kg body weight) by prairie dogs fed six different concentrations of warfarin. Prairie dogs were fed warfarin bait in their respective treatment groups for 15 days. Each square represents total consumption of warfarin of one prairie dog.

After the exposure and post-test observation periods were concluded, treatment group mortality was calculated (Table 1). Interestingly, the 777.6 ppm group did not produce 100% mortality like the 407.0 ppm treatment group below it. One male and one female survived. Further investigation revealed that the female prairie dog had eaten only 186.1 mg of warfarin/kg body weight and lost 267 grams of body weight. The male ate only 128.6 mg of warfarin/kg body weight and lost 146 grams of body weight. The female and male ate 43.3 and 121.9 mg of warfarin/kg body weight less than the average of each sex. The female lost 38 grams more than the female mean, and the male lost 24 grams less than the male mean.

According to the efficacy data presented, a computer analysis from "Probit" gives an LC_{50} value of 97 mg/kg body weight. The LC_{50} of the prairie dogs was calculated at 831 mg/kg body weight. Other Probit analyses were performed because of the 80% efficacy achieved in the highest treatment group. One was performed without the 777.6 ppm group and another was performed with 100% efficacy in the 777.6 ppm group. These alternate analyses showed similar LC_{50} values of 94 and 93 mg/kg body weight, respectively. However, the LC_{50} of both of the alternate analyses was much lower, 441 and 376 mg/kg body weight, respectively.

The prairie dog weights in each treatment group were recorded at the beginning of the exposure period. The overall body weight of the prairie dogs was 897 grams. Mean body weights of prairie dogs across treatment were similar ($F=0.657$, $P=0.658$) as were the variances ($L=0.545$, $P=0.741$).

Table 1. Treatment group mortality and mean and range of warfarin consumption per treatment group.

Concentration (ppm)	N	Mortality (%)	Mean Warfarin Consumption mg a.i./kg Body Weight (Range)
Control (0)	10	0	0
44.8	10	30	13.73 (6.30- 21.52)
89.5	10	50	29.85 (16.27- 43.31)
233.0	10	60	55.31 (15.99-107.65)
407.0	10	100	161.42 (65.08-208.16)
777.6	10	80	239.94 (128.63-352.40)

The mean body weight of the animals decreased in all groups except the control group. Table 2 shows the body weight loss or gain according to treatment group, male, and female. Variances among the treatment group showed no significant difference ($L=1.497$, $p=0.208$). As a result, analysis of variance indicated treatment means were different ($P<0.000$). Weight loss from consumption of the treated baits was significantly different from consumption of the untreated control bait (Tukey's $HSD=1.000$, $P<0.05$).

The necropsies showed small to large amounts of hemorrhaging, independent of the treatment dose. During and after the exposure period, hemorrhaging was observed in the nose, eye, mouth, stomach, liver, intestines, cecum, kidneys, anus, heart, lungs, brain, and subcutaneous and neck regions. The hemorrhaging was so extensive in seven animals, the abdomen (5), thorax (1), or subcutaneous (1) became pooled with blood. Twenty-nine of 32 prairie dogs that died during the test

were observed to have extensive fat in the abdominal subcutaneous or within the abdomen. Also, fat was present in the thoracic region in 25 of 32 prairie dogs. Six grams fat was observed from around the heart of one prairie dog.

Using a validated laboratory method, whole body analysis of the prairie dogs resulted in a range from 0.080 to 6.072 ppm in the 50, 250, and 1,000 ppm treatment groups (Table 3).

The warfarin accumulation within the tissue is correlated to the treatment group ($r=0.709$). The mean days to death of the prairie dogs used for the tissue analysis in the 44.8, 233.0, and 777.6 ppm treatment groups is 11, 10, and 18.

Also, using a validated laboratory method, concentration verification, laboratory stability, and freezer storage stability of all treatment group warfarin baits were calculated. The t-test showed there was no significant difference between the same treatment levels with the three different samples taken.

Table 2. Body weight¹ loss or gain from the study initiation to termination or time of death. The control group is the only group which gained weight. All other treatment groups showed a general increase in weight loss as the treatment group dosage increased.

Treatment Group	Mean Treatment Group Loss/Gain (g)	Mean Male Loss/Gain (g)	Mean Female Loss/Gain (g)
Control	31.2	51.4	11.0
44.8	-77.0	-111.2	-42.8
89.5	-80.7	-110.8	-50.6
233.0	-112.7	-118.2	-107.2
407.0	-94.7	-128.2	-61.2
777.6	-142.2	-170.2	-114.2

¹Body weight calculations were taken from Day 0 of the feeding test and at study termination or at time of death.

Table 3. Concentration of warfarin within the whole body tissue of the prairie dogs of treatment groups 44.8, 233.0, and 777.6 ppm, respectively. Animals were chosen at random to be analyzed from animals that had died during the test. The sample identification denotes the sex [male (M) or female (F)], animal number, and sequence of sample used from extraction procedure.

Sample Identification	Concentration (ppm)
M13A	0.091
F17A	0.631
F18A	0.080
M32A	1.495
M34C	1.528
F35A	0.509
M51A	1.139
M52A	6.072
F56A	2.131

DISCUSSION

The results show the physiological variation in the reaction to the different treatments of warfarin baits. Even with the increased amount of warfarin in the higher levels of bait, the 777.6 ppm treatment group failed to produce 100% mortality like the treatment group below it (407.0 ppm). One prairie dog of each sex survived. The male ate much less warfarin bait and lost less weight than the average prairie dog within the same treatment group. The female ate slightly less, but lost much more body weight than the average female within the same treatment group. It is believed that these two individuals survived because of their physiological reactions to the warfarin, possibly involving higher metabolism of the warfarin or a greater ability to synthesize vitamin K from gut bacteria—the antidote for warfarin poisoning (Hadler and Buckle 1992). Mortality is expected to be variable. Physiological difference within an animals species is common. For example, LD₅₀ tests predict the required dosage to kill 50% of the population from a range of responses of hypersusceptibility to levels of appeared resistance to a certain chemical compound. Adjustments in application rates are made for such a variability in susceptibility.

The initial effects of the warfarin appear to be independent of treatment group dosage. The final response, mortality, is the factor which varies most. The two extremes of warfarin consumption, 6.3 and 352.4 mg warfarin/kg body weight, caused death in both cases. Some of the lower treatment groups, even with continuous no-choice feeding, failed to cause sufficient trauma to the prairie dogs to result in death. This is why higher treatment dosages must be used for ample control.

The metabolism of the prairie dogs appears to be variable, requiring different amounts of feed to supply themselves with enough nutrients. It is shown that there is no statistical significant relationship between the size of the prairie dog and the amount of bait eaten. The range

of the body weights of the prairie dogs was between 676 and 1226 g. It was expected that the larger prairie dogs would eat more bait compared to the smaller animals, but this was not the case. There was only a small relationship between these two factors. Varying factors which could be affecting the prairie dogs are stress from laboratory holding and close physical environment to humans. Also, these prairie dogs were captured in late fall. They were probably storing nutrients for the upcoming winter in the form of fat. The larger animals could have had sufficient supplies of nutrients while the other smaller prairie dogs had to continue foraging to supply themselves with sufficient nutrient stores.

Statistical tests show that the control group is significantly different from the other treatment groups because of the larger amount of bait eaten by the control prairie dogs. This difference could be conceived as a palatability problem because of the difference in consumption from the control group, but most likely, this is a negative response from the warfarin. Warfarin poisoning signs appeared at a similar time (days 5 to 11) with no dose response evident. In addition, the warfarin was causing similar decreases in consumption throughout the treatment groups, again, no dose response was apparent. The warfarin is causing illness in all treated groups at a similar time, resulting in a similar decreased consumption.

Statistical analysis of the warfarin consumption displayed a high correlation coefficient. This indicates that as the treatment dosage increases the amount of mg warfarin/kg body weight will increase. Since the ingestion of warfarin did not decrease at the higher treatment levels, no palatability problems existed. Meehan (1984) reports higher warfarin treatments as being unpalatable, but to correct this problem, a high purity of warfarin was used and the taste was disguised with feed additives.

Prairie dogs used in the test were randomized into treatment groups to achieve an unbiased design which the statistical tests showed. Resulting body weights taken at time of death or at test termination revealed significant body weight loss throughout the exposure and post-test observation period.

Nearing the end of the exposure period, the prairie dogs were not eating much bait because of their warfarin-induced sickness. For example, fat and muscle are the only components which could be slowly lost. Fat was a large component of many prairie dogs that were necropsied at the end of the study. The necropsies revealed small to large amounts of hemorrhaging depending upon the individual. Fat was very common in the abdominal and thoracic cavities as well as in other areas. Fat, which is partly composed of vitamin K, could have been used for the metabolism of the warfarin. Vitamin K, a fat soluble vitamin could have been supplied to the liver for the metabolism of warfarin and reversal of the anticoagulant action.

In a similar Genesis Laboratories, Inc. study with prairie dogs, a factor believed to be the cause of the lack of efficacy was presence of alfalfa cubes. Two prairie dogs died and only three observations of warfarin signs were observed. According to the United States-Canadian Tables of Feed Composition (1982), dry alfalfa can have as high as 14.2 mg of fat-soluble vitamin K per kg. According to Donoco and Haft (1976), and Seegars and Walz (1986), vitamin K is needed by the liver to produce prothrombin (factor II), a major component of the blood clotting mechanism.

Other factors directly involved with vitamin K are the factors VII (serum prothrombin conversion accelerator), IX (plasma thromboplastin component), and X (Stuart-Prower factor). In short, these factors, along with factor II, are most important in beginning the clotting system, which is often referred to as a clotting cascade. This procedure has positive feedback which continues to amplify the reaction intensity. When this cascade is inhibited by warfarin, the result is a failure in blood coagulation (Church and Pond 1988).

The calculated prairie dog LC_{50} places this species in close proximity to the other rodents listed above. The calculated LC_{50} (831 mg/kg) seems to be very high because of the survivors in the 777.6 ppm treatment group. If the 777.6 ppm treatment group had 100% mortality, the calculated LC_{50} would have been 376 mg/kg body weight, less than one-half of the actual. The apparent large range of physiological differences of the prairie dogs, therefore, require a higher dosage to achieve high mortality.

With the removal of the alfalfa cubes in the present study, high mortality was achieved in the higher treatment groups demonstrating diet may be an important factor in warfarin efficacy.

In several cases in study 96018, individuals in the lower treatment groups showed a recovery from the warfarin administration. In addition, between days 11 and 15, almost no bait was eaten, exposing the animals to only 10 days of feeding. Prairie dog M23 lost 185 grams (21% of its initial body weight) over the 25 days of test substance exposure and post-test observation period, leaving the animal very thin. The vitamin K in the fat

could have provided enough nourishment throughout this period (Church and Pond 1988). To illustrate, vitamin K is fat soluble and is able to be stored within fat along with vitamins A, D, and E (Church and Pond 1988; Machlin 1984). This allows consumption of vitamin deficient diets over a longer period of time before deficiency signs appear, compared to water soluble vitamins (C, B6, B12, thiamin, and riboflavin).

This could be a reason for the marginal recovery of M23 and the ability of many others to sustain themselves. If vitamin K is stored within the fat reserves of the prairie dogs, which are very extensive, the vitamin K could be metabolized from the fat as an antidote to warfarin.

Vitamin K can also be replaced by gut bacteria (Hadler and Buckle 1992). Bacterial synthesis of vitamin K has also been documented to be absorbed in the lower part of the intestinal tract where the bacterial population is greatest (Machlin 1984). Specifically, vitamin K is absorbed in the large intestine of mammals sufficiently enough to prevent deficiency symptoms when presented a vitamin K deficient diet (Hollander ****).

It would appear with vitamin K being acquired from gut bacteria and the fat in this study, there would be a sufficient supply of vitamin K to produce an antidote. The resulting deaths increased as the concentration level increased, but there was death caused by a large range of warfarin consumption. The two extremes of warfarin consumption, 6.3 and 352.4 mg warfarin/kg body weight, caused death in both cases. The higher warfarin consumption values could be individuals that are metabolizing the warfarin more efficiently than the others. If they were not, more mortality in all treatment groups would be expected.

Certain application techniques can be used to help increase the mortality of the prairie dogs. First, since fat is known to store vitamin K, an antidote to the warfarin, it would be beneficial to eliminate periods of baiting when the prairie dogs are in the process of storing nutrients for winter survival. Second, if bait can be applied when dietary vitamin K is low, an increased mortality could be expected. Using these ideas in conjunction, the significance of vitamin K from the diet and fat reserves must be considered to produce an effective antidote. These ideas signify the pertinence of applying the bait during a period of low vegetative biomass and quality, and when the animals have a low fat content/body weight—early spring.

The tissue analysis resulted in a high correlation between warfarin consumption and tissue accumulation, but the evident variation could be explained by differences in bait consumption and physiology of the prairie dog. The mean days to death of the animals in the tissue analysis is so variable that again, the physiological differences in the prairie dogs is displayed.

CONCLUSIONS

The highest mortality in this study was found to be 100% at the 407.0 ppm level. The highest concentration of 776.7 ppm was not the most efficacious (80%), showing the differences in prairie dog physiology. The fat and dietary vitamin K have the ability to provide an antidote to the warfarin bait which must be considered into a baiting schedule specific to these parameters. At

least a 407.0 ppm bait should be used to control less susceptible prairie dogs.

Prairie dog tissue samples yielded residue levels which increase as the treatment group dosage increases. The technique used for mixing the bait repeatedly produced homogeneous batches with all treatment levels. All concentrations of the warfarin bait are stable in the laboratory environment.

The control of the black-tailed prairie dog is an important issue which influences many other animal population. Where prairie dog control is necessary, a warfarin bait could provide an alternative to other baiting practices. The effects of warfarin are easily reversible which is an important consideration in human and non-target safety.

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