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## EFFECTS OF PRAIRIE DOG RODENTICIDES ON DEER MICE IN WESTERN SOUTH DAKOTA

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**ABSTRACT.**—Mortality of nontarget small mammals was determined after application of three black-tailed prairie dog (*Cynomys ludovicianus*) rodenticide treatments (prebaited zinc phosphide, prebaited strychnine, and strychnine alone) in western South Dakota. Immediate (September 1983) and long-term (September 1983 through August 1984) impacts on deer mouse (*Peromyscus maniculatus*) relative densities were evaluated, and the three rodenticide treatments were compared for efficacy. The three treatments had no significant ( $\alpha < .10$ ) immediate impacts on deer mouse relative densities, although zinc phosphide did lower them; that impact was not, however, long term. Long-term impacts of the two strychnine treatments were variable, with an increase in deer mouse densities with the strychnine only treatment. Overall, comparisons among the three treatments indicated that zinc phosphide was more effective than either strychnine treatment in reducing deer mouse densities.

Considerable time and money have been spent on control of prairie dogs to reduce the agricultural damage they cause (Collins et al. 1984). However, efforts to evaluate the impact of prairie dog control methods on the total biotic communities of prairie dog towns have been limited. For example, immediate and long-term rodenticidal effects on nontarget wildlife such as deer mice (*Peromyscus maniculatus*) have not been fully evaluated. Applicators, when selecting toxic baits, often overlook information on the margin of safety to nontarget wildlife.

Small mammals are important components of prairie dog towns. Their fossorial activities mix and enrich soils; their food habits may affect vegetation, seed, and invertebrate distribution and abundance; and they provide a food base for predators. When small mammals ingest rodenticides used to control prairie dogs, incidental loss may change the ecological balance on prairie dog towns.

Rodenticides, in addition to causing direct mortality to nontarget wildlife, may impact them indirectly by removing or reducing prairie dog populations. Prairie dogs create niches for small mammals in rangeland ecosystems (Koford 1958, Allen 1967, O'Meilie et al. 1982, MacCracken et al. 1985, Agnew et al. 1986). For example, prairie dogs act as ecosystem regulators by maintaining habitat

suitable for some small mammals, such as deer mice, that are associated with sparse, heterogeneous vegetative cover. Prairie dog burrows provide security cover and nesting habitat for small mammals. When prairie dog activity ceases, burrows are no longer maintained, soil erodes into the holes, and vegetation recaptures the mounds (Klatt 1971, Potter 1980).

Rodenticides used for prairie dog control include zinc phosphide and strychnine. Zinc phosphide is an acute rodenticide that appears to have limited environmental impact (Hilton et al. 1972). Its increased use in recent years (Schenbeck 1982) has resulted in improved formulations and application rates (Tietjen 1976). Secondary poisoning from zinc phosphide poses minimal threat to predators and scavengers that feed on poisoned rodent carcasses (Bell and Dimmick 1975, Schitoskey 1975, Hegdal et al. 1981).

Nontarget wildlife that consume strychnine bait or strychnine-poisoned carcasses are at risk (Rudd and Genelly 1956, Schitoskey 1975, Hegdal and Gatz 1977, Deisch et al. 1989). Apa (1985), in a companion study, found that strychnine used for prairie dog control reduced Horned Lark (*Eremophila alpestris*) densities.

Little information is available on repopulation of small mammals following rodenticide

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treatment (Wood 1965). Such information is needed to formulate guidelines for federal, state, and private landowners for minimizing nontarget wildlife losses caused by prairie dog rodenticides. A program to control black-tailed prairie dogs (*Cynomys ludovicianus*) in western South Dakota provided us the opportunity to assess and compare immediate (direct) and long-term (indirect) impacts on deer mouse densities of three prairie dog control treatments: prebaited zinc phosphide, prebaited strychnine, and strychnine alone.

#### STUDY AREA

This study was conducted on the Buffalo Gap National Grasslands and in the Badlands National Park of western South Dakota at elevations of 820–900 m. Geological formations consisted of sharp pinnacles, towers, steep gorges, and faults. Vegetated tabletop buttes and gently rolling mixed grasslands scattered throughout the area supported prairie dog towns.

The National Grasslands, located in Conata Basin, is grazed by cattle from mid-May to late October each year. Native herbivores include black-tailed (*Lepus californicus*) and white-tailed jackrabbit (*L. townsendii*), eastern cottontail (*Sylvilagus floridanus*), pronghorn (*Antilocapra americana*), mule deer (*Odocoileus hemionus*), and various small mammals. The Badlands National Park excludes cattle, but American bison (*Bison bison*) are present.

Dominant grasses are western wheatgrass (*Agropyron smithii*), blue grama (*Bouteloua gracilis*), buffalograss (*Buchloe dactyloides*), and needleleaf sedge (*Carex eleocharis*). Prairie dogweed (*Dysodia papposa*), Patagonia Indianwheat (*Plantago patagonica*), buckhorn (*Plantago spinulosa*), scarlet globemallow (*Sphaeralcea coccinea*), and prostrate bigbract verbena (*Verbena bracteata*) are dominant forbs.

Climate is semiarid-continental with extremely cold winters and hot, fluctuating summer temperatures. Average annual precipitation is 39.7 cm, most of which falls as high-intensity thundershowers from April through September.

#### METHODS AND MATERIALS

Small mammals were sampled from May through October 1983 (pretreatment) and

May through August 1984 (posttreatment). Eighteen permanent 100 × 100-m (1.0-ha) sampling sites were established on 15 prairie dog towns. Rodenticide treatments were clustered into three separate groups to prevent cross-contamination with respect to wide-ranging nontarget species (6 sites per rodenticide treatment) 13 and 16 km apart. Each rodenticide treatment had 3 control and 3 treated sites. Only zinc phosphide treatments were applied to the park sites because strychnine use is forbidden. Prebaited strychnine and strychnine alone were applied to the grasslands sites.

Relative densities of small mammals (unique mammals/trap session) were determined for each of the 18 sites. A trapping grid included 64 Sherman live traps 10 m apart and a 10-m buffer border. Trapping began in May of each year and continued at four-week intervals. Each trap session consisted of one night of prebaiting followed by four consecutive nights of trapping (256 trap nights/session). Traps were baited with a peanut butter-rolled oats mixture. Captured rodents were identified to species, assigned a unique number by toe amputation (Taber and Cowan 1969), then released. Density was measured as the number of unique captures.

#### Rodenticides and Bait Application

Steam-rolled oats used for prebait and poisoned baits were formulated at the U.S. Fish and Wildlife Service Pocatello Supply Depot. Zinc phosphide was applied to steam-rolled oats at a concentration of 2.0% by weight active ingredients. (Alcolec S, used as an adhesive, was made by American Lecithin Co., Inc.) Strychnine alkaloid was applied to oats at 0.5% by weight. Nontreated steam-rolled oats (4 g) were applied as prebait for zinc phosphide and for one strychnine treatment during 20–21 September 1983. Prebaited areas were visited prior to baiting to assure that most of the prebait had been consumed. Active rodenticides on oats (4 g) were applied three days after prebaiting (22–24 September 1983) in accordance with federal instructions. Both prebait and rodenticides were applied from bait dispensers affixed to Honda 3-wheel ATV's (Schenbeck 1982).

#### Statistical Aspects

Small mammals, including nontarget deer mice, were sampled on each of 18 sites one



TABLE 1. Pretreatment and posttreatment relative densities (unique mammals/trap night) of deer mice (*Peromyscus maniculatus*) on zinc phosphide treated and control sites. Adjusted means were estimated as posttreatment minus pretreatment.

Treatment	Relative density ( $\bar{x} \pm SE$ )			Treatment effect	Significance level (control versus treated) <sup>a</sup>
	Pretreatment (1983)	Posttreatment (1984)	Adjusted means		
IMMEDIATE IMPACTS					
September					
Treated	8.3 $\pm$ 2.6	1.3 $\pm$ 0.7	-7.0 $\pm$ 2.6		
Control	4.3 $\pm$ 1.9	2.7 $\pm$ 0.9	-1.7 $\pm$ 1.2	-5.3 $\pm$ 2.6 <sup>b</sup>	—
POSTTREATMENT IMPACTS					
May					
Treated	8.0 $\pm$ 1.5	8.7 $\pm$ 0.3	0.7 $\pm$ 1.2		
Control	11.0 $\pm$ 3.0	12.3 $\pm$ 3.3	1.3 $\pm$ 3.8	-0.7 $\pm$ 2.1	0.878
June					
Treated	7.0 $\pm$ 2.1	7.6 $\pm$ 0.3	0.7 $\pm$ 2.4		
Control	3.7 $\pm$ 1.9	10.7 $\pm$ 2.6	7.0 $\pm$ 4.2	-6.3 $\pm$ 1.6	0.253
July					
Treated	3.0 $\pm$ 1.2	8.3 $\pm$ 2.3	5.3 $\pm$ 1.9		
Control	2.0 $\pm$ 1.0	10.7 $\pm$ 1.7	8.7 $\pm$ 1.5	-3.3 $\pm$ 1.6	0.223
August					
Treated	8.3 $\pm$ 2.6	4.3 $\pm$ 0.3	-4.0 $\pm$ 2.6		
Control	4.3 $\pm$ 1.9	4.7 $\pm$ 1.9	0.3 $\pm$ 1.9	-4.3 $\pm$ 1.0	0.254

<sup>a</sup>Randomization test used to detect differences between pairs of adjusted means, after significant F-protection at  $\alpha < .10$ .

<sup>b</sup>Treatment effects were not significant ( $P = .295$ ); therefore, statistical significance of contrasts was not determined for September.

week prior to rodenticide application in September 1983 (pretreatment). The fourth day after rodenticides were applied, posttreatment counts were taken on all sites to assess immediate impacts. We evaluated long-term (September 1983 through August 1984) impacts by comparing small mammal data collected during September 1983 with all 1984 trap sessions. Rodenticides were not applied in 1984.

Each rodenticide was evaluated for impacts on nontarget small mammals by comparing the change of mean relative density on each cluster of treated sites with the change observed on respective control sites (Uresk et al. 1988) (Tables 1–3). Five comparisons through time included one for immediate impacts (September 1983), measured between pretreatment and posttreatment (1983) poisoning, and four comparisons that measured differences between pretreatment (1983) and posttreatment (1984) densities. When a significant correlation existed between pretreatment and posttreatment observations, analysis of covariance was used to estimate treatment effect (Deisch 1986, Uresk et al. 1988). Subtraction (Green 1979) was used if the correlation was nonsignificant.

Comparisons between and among rodenticides for impact were produced by forming pairwise contrasts between individual rodenticide treatment effects. Randomization procedures were used to estimate statistical significance of the various contrasts (Edgington 1980, Romesburg 1981, Uresk et al. 1986, Uresk et al. 1988). Rejection of any rodenticide impact (type II error) to nontarget small mammals was considered more serious than potential incorrect acceptance of a significant treatment effect (type I error) (Tacha et al. 1982). After significant ( $P = .10$ ) treatment effects were detected, type II error protection was produced by testing each contrast individually. Type I error protection was afforded by testing for treatment effects with analysis of variance or covariance (Carmer and Swanson 1973).

Individual contrasts were considered biologically significant at  $P = .20$ . Although an alpha of .20 is not a standard level of significance, it is becoming more accepted for ecological field studies (Hayne 1976) and is used here to protect against missing effects on nontarget species. The number of sites available in this study produced a power of .80. This was an acceptable combination of type I



TABLE 2. Pretreatment and posttreatment relative densities (unique mammals/trap night) of deer mice (*Peromyscus maniculatus*) on strychnine only treated and control sites. Adjusted means were estimated as posttreatment minus pretreatment.

Treatment	Relative density ( $\bar{x} \pm \text{SE}$ )			Treatment effect	Significance level (control versus treated) <sup>a</sup>
	Pretreatment (1983)	Posttreatment (1984)	Adjusted means		
IMMEDIATE IMPACTS					
September					
Treated	0.7 $\pm$ 0.7	1.7 $\pm$ 1.7	1.0 $\pm$ 2.1		
Control	9.0 $\pm$ 3.2	6.0 $\pm$ 4.0	-3.0 $\pm$ 2.0	4.0 $\pm$ 2.8 <sup>b</sup>	—
POSTTREATMENT IMPACTS					
May					
Treated	5.7 $\pm$ 3.0	1.7 $\pm$ 1.7	-4.0 $\pm$ 2.1		
Control	11.7 $\pm$ 1.8	3.0 $\pm$ 1.5	-8.7 $\pm$ 3.3	4.7 $\pm$ 2.1	0.314
June					
Treated	2.7 $\pm$ 1.5	0.3 $\pm$ 0.3	-2.3 $\pm$ 1.5		
Control	13.0 $\pm$ 1.2	2.3 $\pm$ 1.9	-10.7 $\pm$ 2.3	8.3 $\pm$ 1.8	0.043
July					
Treated	3.7 $\pm$ 2.7	0.3 $\pm$ 0.3	-3.3 $\pm$ 2.8		
Control	4.3 $\pm$ 2.3	1.0 $\pm$ 1.0	-3.3 $\pm$ 1.7	-0.1 $\pm$ 1.6	0.999
August					
Treated	0.7 $\pm$ 0.7	0.0 $\pm$ 0.0	-0.7 $\pm$ 0.7		
Control	9.0 $\pm$ 3.2	1.3 $\pm$ 1.3	-7.7 $\pm$ 2.0	7.0 $\pm$ 1.1	0.034

<sup>a</sup>Randomization test used to detect differences between pairs of adjusted means, after significant F-protection at  $\alpha < .10$ .  
<sup>b</sup>Treatment effects were not significant ( $P = .295$ ); therefore, statistical significance of contrasts was not determined for September.

and II error protection (Carmer 1976) and allowed for reasonable biological inferences to be drawn from the data.

RESULTS

Effects of Rodenticides

Eleven small mammal species captured on 18 sites included deer mouse (*Peromyscus maniculatus*), northern grasshopper mouse (*Onychomys leucogaster*), Ord's kangaroo rat (*Dipodomys ordii*), thirteen-lined ground squirrel (*Spermophilus tridecemlineatus*), western harvest mouse (*Reithrodontomys megalotis*), hispid pocket mouse (*Perognathus hispidus*), plains pocket gopher (*Geomys bursarius*), prairie vole (*Microtus ochrogaster*), house mouse (*Mus musculus*), olive-backed pocket mouse (*Perognathus fasciatus*), and Norway rat (*Rattus norvegicus*). Deer mouse was the only species captured in sufficient numbers to statistically evaluate for rodenticide effects.

There were no immediate impacts of any of the three rodenticide treatments ( $P = .295$ ) on deer mouse relative densities in September 1983 (Tables 1–3). However, relative densities of deer mice changed 79% from 5.8 to 1.2

unique animals immediately after application of zinc phosphide (Uresk et al. 1988). Long-term impacts of the three rodenticides were detected.

On zinc phosphide sites, deer mouse densities were not significantly different between control and treated sites, but densities on treated sites were consistently lower compared with control sites (Table 1). On strychnine sites, relative densities of deer mice were significantly higher on treated sites in June ( $P = .043$ ) and August ( $P = .034$ ) (Table 2). Sites with prebaited strychnine showed higher densities on treated sites in August 1984 ( $P = .063$ ) (Table 3).

Comparisons of Three Rodenticides for Impacts

Comparisons of the impacts of the three rodenticides immediately after application showed no differences ( $P = .10$ ) for deer mouse densities in September 1983. Zinc phosphide lowered densities of deer mice more than did strychnine alone in June 1984 ( $P = .030$ ) and August ( $P = .018$ ); in May and July no differences in reduction rates were measured. There were no differences among treatment effects of zinc phosphide compared



TABLE 3. Pretreatment and posttreatment relative densities (unique mammals/trap night) of deer mice (*Peromyscus maniculatus*) on prebaited strychnine treated and control sites. Adjusted means were estimated as posttreatment minus pretreatment.

Treatment	Relative density ( $\bar{x} \pm \text{SE}$ )			Treatment effect	Significance level (control versus treated) <sup>a</sup>
	Pretreatment (1983)	Posttreatment (1984)	Adjusted means		
IMMEDIATE IMPACTS					
September					
Treated	9.3 $\pm$ 0.9	4.0 $\pm$ 1.2	-5.3 $\pm$ 1.9		
Control	16.3 $\pm$ 2.7	13.0 $\pm$ 5.5	-3.3 $\pm$ 3.7	-2.0 $\pm$ 2.7 <sup>b</sup>	—
POSTTREATMENT IMPACTS					
May					
Treated	17.0 $\pm$ 3.1	5.3 $\pm$ 0.9	-11.7 $\pm$ 2.3		
Control	20.3 $\pm$ 3.0	7.7 $\pm$ 1.5	-12.7 $\pm$ 4.7	1.0 $\pm$ 2.1	0.864
June					
Treated	20.7 $\pm$ 4.3	0.3 $\pm$ 0.3	-20.3 $\pm$ 4.5		
Control	21.3 $\pm$ 2.2	2.7 $\pm$ 2.2	-18.7 $\pm$ 4.3	-1.7 $\pm$ 1.6	0.795
July					
Treated	10.3 $\pm$ 3.0	0.0 $\pm$ 0.0	-10.3 $\pm$ 3.0		
Control	11.0 $\pm$ 3.8	3.0 $\pm$ 2.1	-8.0 $\pm$ 5.9	-2.3 $\pm$ 1.6	0.726
August					
Treated	9.3 $\pm$ 0.9	0.7 $\pm$ 0.7	-8.7 $\pm$ 0.3		
Control	16.3 $\pm$ 2.7	0.3 $\pm$ 0.3	-16.0 $\pm$ 3.0	7.3 $\pm$ 1.1	0.063

<sup>a</sup>Randomization test used to detect differences between pairs of adjusted means, after significant F-protection at  $\alpha < .10$ .

<sup>b</sup>Treatment effects were not significant ( $P = .295$ ); therefore, statistical significance of contrasts was not determined for September.

with prebaited strychnine on deer mice from May through July. Impact of zinc phosphide in August ( $P = .027$ ) was greater than that of prebaited strychnine. Comparison of treatment effects between the two strychnine rodenticides indicated that strychnine alone was more effective than prebaited strychnine for lowering densities of deer mice in June ( $P = .174$ ).

#### DISCUSSION

Of the three rodenticide applications used for prairie dog control, only zinc phosphide consistently lowered deer mouse densities. On these sites zinc phosphide was also most effective in reducing prairie dog burrow activity (Apa 1985). Deer mice consume seeds (Baker 1968, Flake 1973, Sieg et al. 1986) and are susceptible to granular rodenticides. After initial rodenticide treatments, long-term changes in deer mouse populations are associated with habitat changes such as increased density of vegetation (Uresk 1985) because of lack of clipping by prairie dogs. Deer mice are adapted to live in more open habitat (Baker 1968, Jones et al. 1983, MacCracken et al. 1985, Agnew et al. 1986), and

their numbers decrease with increased vegetation height and canopy cover. Prairie dog burrows were initially devoid of vegetation before rodenticide application; increased plant canopy cover and aboveground biomass occurred with absence of prairie dogs (Klatt 1971, Potter 1980) and contributed to a decrease in deer mouse densities.

Deer mouse densities were variable over the long-term period with the two strychnine treatments, especially when prebaiting was applied. Deer mouse populations generally increased after treatment with the strychnine only. This increase can be attributed to limited control of the black-tailed prairie dogs (Uresk et al. 1986), which provided and maintained suitable habitat for deer mice (Agnew et al. 1986). Changes in densities of deer mice may also be attributed to seasonal movements of these animals from other areas (Terman 1968) and possible lower predation. An influx of rodents usually occurred in the spring when yearling deer mice established home ranges (MacCracken et al. 1985), and lower densities in August were due to dispersal of young-of-the-year (Falls 1968, Metzgar 1980).

Crabtree (1962) and Marsh et al. (1970) found that zinc phosphide produced a



response-stimulating odor that proved attractive to small mammals, but strychnine did not have an attractive effect on rodents. Based on these findings, discontinuation of zinc phosphide for prairie dog control is not recommended or required, but land management plans should include considerations for possible nontarget deer mouse losses. We found that use of strychnine alone or prebaited strychnine generally showed a long-term increase in deer mouse densities. Use of these two strychnine treatments for prairie dog control appears to impose the least threat to nontarget deer mice.

While this study addressed direct effects of rodenticides (zinc phosphide, prebaited strychnine, and strychnine alone) on deer mouse densities, impacts on other nontarget small mammals could not be evaluated because of the small populations observed. We suspect that granivores, such as *Perognathus* spp. and *Dipodomys* spp., found on prairie dog towns in western South Dakota, may also be affected by rodenticides. Further investigations are needed to assess nontarget losses of small mammals other than deer mice.

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