

Effect of Flea Control
on Survival of Utah Prairie Dogs (*Cynomys parvidens*)

Final Report

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Abstract

Plague causes periodic epizootics that decimate populations of prairie dogs, but the means by which the causative bacterium (*Yersinia pestis*) persists between epizootics is poorly understood. We examined the possibility that the bacterium persists in and around Utah prairie dog colonies, affecting prairie dogs during inter-epizootic periods. We attempted to reduce the numbers of fleas on portions of 6 prairie dog colonies during 2001-2004 using deltamethrin powder delivered into burrows as a pulicide. We compared survival rates of prairie dogs on treated plots with survival rates of prairie dogs on paired plots where burrows were not dusted. Flea numbers were reduced by 96-98 % by the dusting. Survival rates for prairie dogs were about 98 % higher in dusted plots than in non-dusted plots, even though epizootic plague was not detected during the study. *Y. pestis* was not found in any of the 4,578 fleas taken from 1,258 prairie dogs, and only 35 of those prairie dogs carried serum antibodies for plague (34 of the seropositive prairie dogs were from a single colony). Our data suggest that plague may be resident on prairie dog colonies rather than periodically invading from reservoirs elsewhere. The effects of enzootic plague raise serious concerns for conservation of prairie dogs and other dependent species.

Introduction

Human-assisted invasions of alien species, including microorganisms, are widely recognized threats to global biodiversity and functioning of ecosystems (Andrewes 1967, Dobson and Hudson 1986, Hobbs and Humphries 1995, Kinzelbach 1995, Kaiser 1999, Daszak et al. 2000). Expansion of the plague-causing organism (*Yersinia pestis*) into additional continents from its likely origins in Asia or Africa (Achtman et al. 1999) has dramatic ecological and evolutionary ramifications due to its lethality and extraordinary host range. *Y. pestis* is a generalist found in > 200 species of mammals (Pollitzer and Meyer 1961, Poland and Barnes 1979), enhancing its ability to colonize new habitat. Plague causes large population reductions in rodents of several species within both its native range (Dyatlov 1972, Chelnokov et al. 1979) and introduced range (Barnes 1993). Despite the potential of plague to alter ecosystems it invades, and the attention it has received as a human pathogen, it has received relatively little attention in reviews of effects of non-indigenous organisms (e. g. , Pimentel et al. 2000).

This study was motivated by our concern for the general decline in prairie dogs (*Cynomys* spp.) and the particularly precarious status of Utah prairie dogs (*Cynomys parvidens*). The total number of Utah prairie dogs, estimated at >95,000 prior to 1920, declined to about 3,300 by 1973, when the species was listed as endangered. Declines were attributed to plague, poisoning campaigns, and habitat alteration (U.S. Fish and Wildlife Service 1991). The species was down-listed to threatened in 1984, due to an increase in the estimated population (to 7,988) by 1983. Most population expansions occurred on private lands, creating numerous conflicts (McDonald 1993). Populations on public lands have been less successful, and their long-term viability has been questioned (Roberts et al. 2000). Some populations of Utah prairie dogs were known to have been decimated by epizootics of plague (U. S. Fish and Wildlife Service 1991, Roberts et al. 2000). Recent proposals to list the black-tailed prairie dog (*C. ludovicianus*) as threatened (Graber et al. 1999) and re-list the Utah prairie dog as endangered similarly exemplify the plight of prairie dogs in general. Our concern about plague, however, extends beyond prairie dogs to other species of mammals with shrinking populations in western North America (Biggins and

Kosoy 2001a,b), and to those species that have not been monitored or have been monitored so poorly that even large-scale population reductions may have escaped notice.

All species of prairie dogs are highly susceptible to plague (Ecke and Johnson 1950, Williams 1986, Ubico et al. 1988, Barnes 1993). Although epizootics of plague that cause high mortality of prairie dogs over wide areas are a commonly noted phenomenon, plague may be enzootically maintained between the epizootics (as opposed to the hypothesis of maintenance of the disease by continued movement of the organism from community to community). Postulated mechanisms for maintenance of *Y. pestis* include (1) survival of the bacterium in the soil, (2) persistence of the bacterium in fleas, and (3) maintenance of the bacterium in “reservoir” species of mammals in which *Y. pestis* is relatively non-lethal. Although *Y. pestis* has been identified as the probable cause of several population crashes of Utah prairie dogs, little is known about the ecology of plague in Utah prairie dog communities, particularly the inter-epizootic maintenance of *Y. pestis* populations in the rodent community and its potential effect on prairie dogs as an enzootic disease. If enzootic plague is affecting populations of prairie dogs, an ambitious effort to remove the disease should result in increased survival rates of prairie dogs even in the absence of catastrophic epizootics of plague. We evaluated this possibility by controlling the fleas that serve as vectors of plague using deltamethrin powder applied to prairie dog burrows on 6 sites during 2001-2004.

Methods

Treatments for plague control commenced in 2001, except at Horse Hollow where work began in 2002 (Table 1). Experimental areas selected were up to 50 ha each, encompassing entire prairie dog colonies or portions thereof. The treatments were repeated in pairs (dusted and non-dusted) with as little physiographic and biotic variation as possible between members of each pair (e.g., slope, aspect, burrow density, quantity and type of vegetative cover, initial prairie dog density). Paired plots were in close proximity to each other, either by splitting colonies into two parts or by using colonies that were separated by < 1 km. We attempted to reduce the incidence of plague (or eliminate it) from test areas by reducing the populations of fleas which transmit the disease. We controlled fleas by infusing a powdered deltamethrin formulation (DeltaDust[®]) into burrows during summer months with pressurized applicators (Beard et al. 1992, Seery et al. 2003), at a rate of about 4 g per burrow.

Prairie dogs were captured and ear-tagged for individual identification, using a #1 monel fingerling fish tag in each ear. The East Creek colony, in Bryce Canyon National Park and adjacent Dixie National Forest, presented a situation somewhat different from other study sites because prairie dogs had declined to just a few animals (purportedly due to plague) before our study began. In 2001, we released 46 prairie dogs on the dusted half of the colony and 48 animals on the non-dusted half. All were ear-tagged and treated in analyses as the marked population for that year. We conducted visual counts on 4-9 ha plots once each year on each study area during 3 consecutive days (Fagerstone and Biggins 1986, Menkens et al. 1990). We collected small quantities of blood from prairie dogs via toe nail clipping and spent about 20 seconds combing fleas from each animal captured (Karhu 1999). Blood and fleas were tested for *Y. pestis* by the U.S. Centers for Disease Control and Prevention, Plague Branch, Fort Collins, Colorado. Blood samples were tested via passive hemagglutination assay (PHA). Fleas from individual hosts were pooled by species and sex, and emulsions were tested by mouse

inoculation and subsequent isolation. Direct fluorescent antibody tests were done on tissues from a few prairie dogs found dead.

We used several methods to compare reencounter rates (the product of probability of capture and probability of survival) and true survival rates of ear-tagged prairie dogs on dusted and non-dusted plots at the 6 study sites. Reencounter rates can be a reasonable index to survival rate under some conditions, and are often called minimum survival rates, similar to the minimum number alive of Krebs (1966). The degree that reencounter rates underestimate survival rates is proportional to the probability of capture (because $[\text{survival probability}] = [\text{reencounter probability}] / [\text{capture probability}]$). If the assumption of equal probability of capture for all treatment groups can be reasonably met, use of reencounter rates to compare groups may be valid.

For the simplest assessment of reencounter rates, we used the non-parametric Wilcoxon test of signed ranks, which took full advantage of the paired nature of study sites; observations were differences between members of each pair. We assumed that the 17 pairs of year-site observations (Table 1) were independent and that probability of recapture was the same for the dusted and non-dusted plot at each site. It is important to note that, for this analysis, the assumption of equal probability of capture among sites and years was not necessary. Potential effects of other variables were ignored. Sample size for this analysis was 859 marked prairie dogs.

Our second approach involved logistic regression analysis, again using overall reencounter rates but allowing assessment of simultaneous effects of multiple variables. For meaningful interpretation, it is necessary to assume equal probability of capture for all animals, or at least to control for factors that may cause probabilities of capture to vary. Among the most important of those factors is the amount of trapping effort at each site during each year. This variable was recorded for most (but not all) of the years and sites. Because trapping effort was theoretically influential, only the subset of years and sites with data on effort were used in the logistic regression analyses ($n = 506$ marked prairie dogs). Animals recaptured at the end of each annual interval were re-used at the beginning of the next time interval and were treated the same as newly-marked animals. The variable of primary interest was dusting, but covariates included in the general model were sex, elevation, capture history, effort and density (all but density and effort were treated as categorical variables).

In our logistic regression model, the interaction between the primary treatment (dust) and density during the year prairie dogs were marked was considered more important to examine than the main effect of density, because we were primarily interested in detection of disproportionately different effects of density on reencounter rates for dusted and non-dusted plots. If plague is density dependent, we predicted that its effect would be greater on the unprotected prairie dogs of the non-dusted plots than on their dusted counterparts.

In the final approach, we used complete capture histories for each animal marked at any time during the study (which included multi-year consecutive recaptures and alternate year recaptures) to simultaneously estimate probabilities of capture and survival (program RELEASE, Burnham et al. 1987). The methodology for estimation in this approach and for the logistic

regression approached described above relies on maximum likelihood theory. Choices for components of general models were made *a priori* on ecological and biological grounds outlined in original study plans. We attempted to simplify general models to the most parsimonious sub-models that explained significant variation (Lebreton et al. 1992). Stepwise model reduction was used in logistic regression (using *t*-values), and Akaike's Information Criterion (AIC, program RELEASE) was used for model selection in the final analysis of true survival rates.

The Tom Best Spring site was the only Utah prairie dog study area where we found a large proportion of adult prairie dogs to be carrying serum antibodies for plague-specific antigens (Biggins 2002b), and those antibodies may provide protection during subsequent exposure to plague. We therefore conducted a separate logistic regression analysis on data for adult prairie dogs at Tom Best Spring to examine the effect of former plague exposure on survival. We predicted that the greatest benefit of immunity should be seen where the prairie dogs were least protected by our flea control; thus, the interaction between dust and titer was the most important feature to be statistically assessed.

Results

The effect of flea control on reencounter rates and survival rates was substantial, regardless of analysis approach and attendant assumptions. The simple signed rank comparison of reencounter data for 17 paired plots (Fig. 1) yielded a highly significant pattern of differences ($Z = -2.689$, $P = 0.007$). In the logistic regression modeling, dust was again influential ($t = -2.880$, $P = 0.004$) in the reduced model, which had 4 additional covariates. Overall reencounter rates were 77 % higher in the dusted plots than in the non-dusted plots, using unadjusted means (Fig. 2). Finally, modeling within program RELEASE resulted in several closely-competing models, all of which maintained separate estimates of true survival rates for dusted and non-dusted plots. Differences between models mostly resulted from varied pooling over time. The model with the lowest AIC value (with 8 parameters) retained four separate estimates of survival (2 each for intervals 2001-2002 and 2002-2003); the average rate for the two intervals was 98% higher for the dusted than for the non-dusted plots (Fig. 3). The results from "Test 1" in RELEASE, considered an omnibus test of treatment effects, reinforced the conclusion that dusting had a significant effect ($X^2 = 14.852$, $df = 5$, $P = 0.011$).

We failed to generate any support for density dependence of plague, as implied by lack of a meaningful statistical interaction between dusting and prairie dog density during the year animals were marked ($t = -0.485$, $P = 0.628$). Fig. 4 illustrates the simple relationship unadjusted by covariates. The main effect of density was similarly equivocal ($t = -0.363$, $P = 0.716$); so both the interaction and main effect terms were removed from the logistic regression model.

The final logistic regression model provided evidence of a positive correlation between reencounter rate and trap hours of effort ($t = 1.933$, $P = 0.053$), reinforcing our *a priori* decision (based on theoretical considerations) to retain effort as a control variable in all logistic regression models, regardless of *P*-values. The interaction between elevation and dust was not influential in the logistic regression model ($t = -0.349$, $P = 0.727$), but the reencounter rates seemed different at different elevations ($t = 1.980$, $P = 0.048$) (Fig. 5). The final model also suggested that

reencounter rates were lower for males than for females ($t = 1.803$, $P = 0.071$) (Fig. 6) and that reencounter rates were positively correlated with the lag between first capture and recapture ($t = 4.859$, $P < 0.001$) (Fig. 7).

The separate logistic regression analysis of data from the Tom Best Spring colony, to examine the effect of plague titers on reencounter rates, failed to detect an interaction between dusting and plague titers ($t = 0.648$, $P = 0.517$), and an overall main effect of titers on reencounter rates likewise was not supported ($t = 0.795$, $P = 0.426$).

Plague detection rates were extremely low, except at Tom Best Spring. *Y. pestis* was not cultured from any of the 4,578 fleas taken from 1,258 Utah prairie dogs during 2001-2003. Fluorescent antibody tests failed to detect plague in tissues from the few dead prairie dogs encountered aboveground. Relatively few of the 1,258 prairie dogs had serum antibodies for plague. In 2001, however, 77 % of the adult prairie dogs (17 of 22) carried antibodies for plague at Tom Best Spring. Only 9 % (2 of 23) of juveniles were seropositive in 2001; their low titers (1:32) could have been from passive immunity, as some adult females at the site had high antibody levels. In 2002, 17 of 20 adults were seropositive, but by 2003, only 2 of 28 were positive. At Tom Best Spring, a juvenile prairie dog residing on the non-dusted plot was seropositive for plague (1:32) on 4 June 2003. Thus, it was likely exposed to the disease during the previous month, suggesting that plague was active at the site in 2003. Because few adults were seropositive for plague in 2003, it seems unlikely that the juvenile's titer was maternally derived. PHA titers declined for 5 of the 6 prairie dogs recaptured in 2002 that were seropositive in 2001 (Table 2). Serum antibodies were detected in only 1 additional Utah prairie dog (at Wild Pea Hollow) during all years of the study (Table 2).

Discussion

Fleas were effectively controlled by the annual treatments of deltamethrin, with reductions of 96-98 percent 1 month after treatment and reductions of 52-86 percent 10 months after treatment (Biggins 2002). There is little doubt that treating prairie dog burrows with deltamethrin improved the survival rates of prairie dogs. The lack of epizootics of plague on our study sites was both fortunate and problematic. It would have been more difficult to study enzootic plague if epizootics had intervened. Most detections of plague, however, occur during or after epizootics, so plague was difficult to detect during our study. Our failure to detect plague at most sites raises the question of whether or not the disease was present. One might wonder whether the effect we observed was due to some other vector born disease or due to ectoparasites themselves. Our proposed flea control methods may reduce or eliminate other ectoparasites that may play a role in other vector-borne diseases (e.g., ticks and tularemia). Also, the collective reduction in populations of various parasites could have direct effects on host mammals. Nevertheless, we suggest that plague is the most likely explanation for the changes in survival we observed because (1) plague is known to be lethal to prairie dogs, (2) fleas that serve as vectors for plague are by far the most common ectoparasites on Utah prairie dogs (our data and those of Hoogland et al. 2004), (3) our study showed that deltamethrin was highly effective in controlling fleas, and (4) plague has been detected in these study areas historically, in other recent studies (Hoogland et al. 2004), and in our monitoring. Additionally, there is evidence that pyrethroid applications to control fleas in prairie dog burrows also halted plague epizootics

(Seery et al. 2003, Hoogland et al. 2004). Alternative explanations for the effects we observed in this study seem less likely because (1) ticks that are vectors for tularemia are relatively uncommon on prairie dogs, (2) flesh flies and bot flies are only rarely found in prairie dogs (our observations and those of D. Long, pers. comm.), and we have no evidence that deltamethrin powder applied to burrows will control them, (3) fleas seem to produce rather benign direct effects on other species of rodents (Khokhlova et al. 2002), (4) other effects (e.g., other diseases, insect prey eaten by prairie dogs) are not supported by data and remain highly speculative.

Nine marked prairie dogs were known to have changed plots between years. We cannot be certain where they spent the most time during the intervals assessed. Because there were few such animals and they moved nearly equally from and to dusted plots, we assumed that they had little effect on results of our analyses. This sparse immigration and emigration suggests that populations were not maintained in equilibrium by source-sink dynamics (e.g., dispersal from dusted to non-dusted plots). We cannot, however, discount the effect of proximity of dusted and non-dusted plots. Flea movements could have occurred even without major prairie dog dispersal, and other species of mammals may have moved frequently between the plots.

A close examination of effect of prairie dog density on survival was important because of prevalent theory regarding density dependence in disease transmission in general and plague cyclicity in particular (Barnes 1993). If plague reduced survival of prairie dogs on the colonies we studied and its effect was density dependent, we should have detected an interaction between dusting and density in the logistic regression model of reencounter rates. Furthermore, the wide range of densities present (0.3 to 51.0 prairie dogs/ha) (Fig. 4) should have provided ample opportunity for a density dependent effect. Our failure to detect an interaction could have been due to low power of the experiment, but our experiment was sufficiently powerful to detect numerous other effects. It seems more likely that either density dependence is not a factor of great importance in the dynamics of enzootic plague, or that some other factor is counteracting the density dependence of plague. At least two factors, competition for forage and rate of disease transmission, would be expected to cause a negative correlation between prairie dog density and survival rate. In prairie dogs and other colonial species, however, there may be a positive correlation between prairie dog density and survival due to increased efficiency of predator avoidance as density increases. We presume that this is a relatively powerful selective force to have favored the degree of sociality present in prairie dogs (Hoogland 1995). Thus, our failure to detect a relationship between density and survival may be due to the balancing of these two tradeoffs. This line of reasoning spawns speculation about whether the introduction of this exotic disease may have tipped the balance toward a lower level of sociality for optimum survival in prairie dogs.

Considering the evidence that human cases of plague are positively correlated with precipitation (Parmenter et al. 1999, Enscoe et al. 2004) and evidence positively correlating plague detections with elevation (Hoar et al. 2002, Arjo et al. 2003), we anticipated that effects of dusting might vary with elevation. Conditions tend to become more mesic with increasing elevation. Our prediction that the disparity between reencounter rates for dusted and non-dusted plots would increase with elevation was not strongly supported, however. Our data gave equivocal results, showing a tendency for decreasing reencounter rates with increasing elevation, but failing to demonstrate the expected interaction. The trend is intriguing, but more data are needed on the

subject. The shortage of data at high elevation and low elevation (one site each) weakened this aspect of the experiment.

We predicted that reencounter rates for prairie dogs with serum antibodies for plague (at Tom Best Spring) might be higher than for seronegative animals, but no relationship was detected. Many animals (18/34) had low titers (1:32, 1:64) which may not be protective. Also, power to detect this effect was low due to small sample sizes (8 of 34 "vaccinated" prairie dogs were recaptured, a rate of 24% compared to 16% for non-vaccinated animals).

Additional methods of plague control are under development and testing. Most promising are vaccines for plague and insect growth regulators (IGRs) that interrupt growth or reproductive cycles of fleas. The vaccine will directly target plague in rodents, with little chance of altering the community of arthropods in the prairie dog ecosystem. The IGRs are somewhat more attractive than pyrethroids for large scale applications because they can be administered directly to prairie dogs and other rodents via baits (Karhu 1999, Karhu and Anderson 2000); thus, they are more target-specific. Future studies incorporating both the vaccine and flea control methods would make it possible to distinguish between direct effects of parasites and effects of plague itself.

Similar objectives are being addressed in our studies of plague ecology in white-tailed prairie dogs (*C. leucurus*) and black-tailed prairie dogs. On white-tailed prairie dog study areas, *Y. pestis* was detected on sites which had not undergone noticeable die-offs of prairie dogs (Biggins 2000), although population densities had remained low for an extended period. *Y. pestis* was detected in *Pulex simulans* flea pools from 25 of 48 prairie dogs captured and marked in 2 experimental areas. On the two experimental areas with plague, the recapture rate after 2 months for prairie dogs harboring plague positive fleas (22 %) was similar to the rate for prairie dogs with plague negative fleas (26 %). Given the lethality of plague in prairie dogs, the lack of detectable effect of plague-positive fleas on recapture rates of prairie dogs was unexpected. Perhaps *Pulex simulans*, a relatively inefficient transmitter of *Y. pestis* (K. Gage, pers. comm.), helps maintain plague in prairie dog communities by harboring the organism without exposing large numbers of prairie dogs to it. These findings support the contention that our understanding of transmission routes for sylvatic plague in North America remains rather rudimentary.

Most discussion of the effects of plague in prairie dog populations focuses on the catastrophic effects of epizootics. Modeling of prairie dog dynamics often invokes periodic catastrophies due to plague (Roberts et al. 2000). A prevalent perceptual model of plague in prairie dog communities involved periodic invasion of colonies by *Y. pestis* organisms that come from other sources when the densities of prairie dogs reached appropriate levels to sustain rapid transmission, resulting in nearly complete die-offs and subsequent local disappearance of the bacterium due to lack of hosts. Attributes of epizootics, including synchrony of outbreaks, rates of mortality, and rates of spread influence estimates of persistence for prairie dog colonies. The ecology of plague during inter-epizootic periods is less well understood.

What are the ramifications if plague persists at low levels as a chronic disease of prairie dogs? Effects of chronic persistence of low levels of plague may be sufficiently severe that populations of Utah prairie dogs may not persist over moderate time spans, even in the absence of

catastrophic losses due to epizootic plague. The mortality rate changes we observed may result in struggling populations under all but the most favorable conditions. The failure to demonstrate that this form of plague is density dependent is particularly disturbing. The disease may have an additive effect that is superimposed over background stochasticity. Population persistence should be modeled with VORTEX or other suitable modeling software for assessing extinction rates, to examine long-term effects of enzootic plague suggested by our mortality data. Second, if *Y. pestis* is resident on prairie dog colonies instead of a periodic invader, previous conceptual views of diseases dynamics may be invalid. This change in outlook has several ramifications. The organism may not need a "reservoir" away from prairie dog colonies in order to persist, and looking for reservoir species of mammals may prove fruitless. Similarly, discussions regarding how plague seems to move from prairie dog colony to prairie dog colony during epizootics, and how the disease may skip colonies within a complex, need to be revisited. The organism may benefit from being transported by mammalian hosts and their fleas in terms of broad scale spread, but may be less reliant on those mechanisms once it has become a resident. Our observations of "spread" in epizootic plague may simply be an artifact of relatively independent population expansions of *Y. pestis* on separate prairie dog colonies that have undergone synchronous changes in conditions favoring bacterial growth rates (e.g., temperature, moisture, flea populations). The concepts of source-sink metapopulation dynamics and population amplification of *Y. pestis* in prairie dog communities also should be reviewed. Our data were insufficient to document interactions between plague effects and elevation of prairie dog colonies, but these relationships need further investigation. We hypothesize that source-sink dynamics are operative in relatively large scales of space and time. For example, plague may be persistent where the climate is typically optimal, but may migrate periodically into peripheral habitat when conditions are most favorable. It seems likely that good and poor conditions for plague are correlated to weather phenomena at several scales (Parmenter et al. 1999, Ensore et al. 2002).

Our data suggest that *Y. pestis* is not periodically invading prairie dog colonies from distant reservoirs, but remains in the vicinity of the colonies or within the colonies. The elevated mortality rates for prairie dogs on our non-dusted plots infer that prairie dogs themselves may serve as the reservoir hosts for *Y. pestis*. Perhaps the disease is dynamic simply because, during good years for prairie dogs (e.g., due to abundant forage or low predation), population growth rates for the rodents temporarily exceed the capability of *Y. pestis* to colonize new hosts, causing a lag effect. Similarly, during poor years for *Y. pestis* (e.g., due to low flea numbers or low densities of combined species of hosts), the bacteria may become too rare to be detected with the monitoring methods we used.

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Table 1. Utah prairie dog colonies where plots dusted with deltamethrin were paired with non-dusted plots.

Site	Elevation (meters)	Land Management Agency	Years
West Desert			
Horse Hollow	1622	Bureau of Land Management	2002-2004
Wild Pea Hollow	1951	Bureau of Land Management; State	2001-2004
Paunsaugunt Plateau			
Tom Best Spring	2347	Forest Service	2001-2004
East Creek	2384	Forest Service; Park Service	2001-2004
Johnson Bench	2408	Forest Service	2001-2004
Awapa Plateau			
The Tanks	2697	State	2001-2004

Table 2. Results from passive hemagglutination assays on Utah prairie dogs that were seropositive for plague.

Site	Sex	Age	Animal	Maximum dilution (1:X) at which agglutination was detected (for year indicated)		
				2001	2002	2003
Tom Best Spring	M	A	3325	32	0	0
Tom Best Spring	M	J	3323	32		
Tom Best Spring	M	A	3383	32		
Tom Best Spring	F	A	3331	64	128	
Tom Best Spring	F	A	3329	64		
Tom Best Spring	F	A	3335	64	0	
Tom Best Spring	M	A	3391	64		
Tom Best Spring	M	A	3814	64		
Tom Best Spring	F	A	3350	128	32	
Tom Best Spring	M	A	3816	128		
Tom Best Spring	F	A	3852	128		
Tom Best Spring	M	A	3802	256	64	
Tom Best Spring	F	A	3396	256		
Tom Best Spring	F	A	3812	1024	128	
Tom Best Spring	F	A	3347	1024		
Tom Best Spring	F	J	3385	1024		
Tom Best Spring	F	A	3387	1024		
Tom Best Spring	F	A	3862	1024		
Tom Best Spring	F	A	3339	4096		
Tom Best Spring	F	A	3350		32	
Tom Best Spring	F	A	3381		32	
Tom Best Spring	F	A	3381		32	
Tom Best Spring	F	A	3824		32	
Tom Best Spring	F	A	3824		32	
Tom Best Spring	F	A	2498		64	
Tom Best Spring	F	A	3686		64	
Tom Best Spring	M	A	3802		64	
Tom Best Spring	F	A	3331		128	
Tom Best Spring	M	A	3680		128	
Tom Best Spring	F	A	3812		128	
Tom Best Spring	F	A	3876		128	
Tom Best Spring	F	A	3676		256	
Tom Best Spring	M	J	1874			32
Tom Best Spring	F	A	1880			64
Wild Pea Hollow	F	A	1614			32

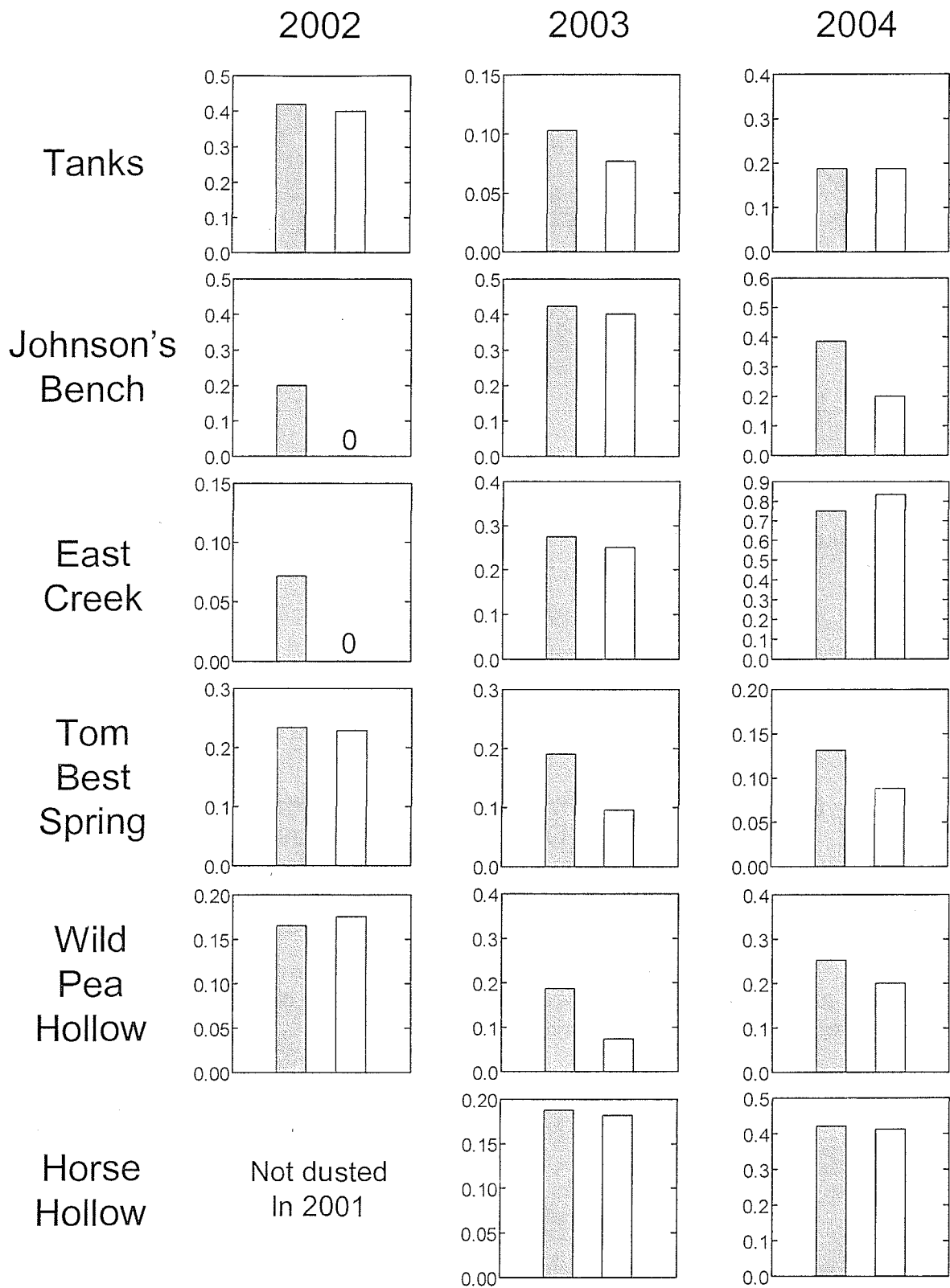


Figure 1. Reencounter rates for 859 marked Utah prairie dogs on plots dusted with deltamethrin (shaded bars) and non-dusted plots (non-shaded bars) at 6 study sites during 2002-2004. Scales were allowed to vary to emphasize comparisons between treatments within study sites and years.

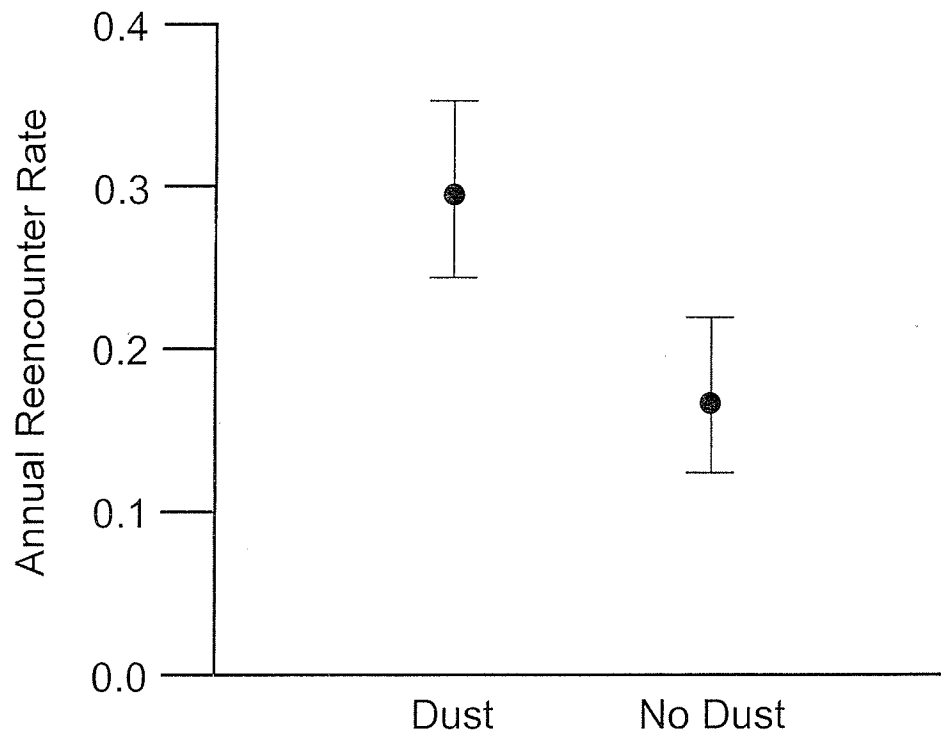


Figure 2. Reencounter rates (means, 95% CIs) for Utah prairie dogs on dusted and non-dusted plots during 2001-2003, without considering effects of other variables.

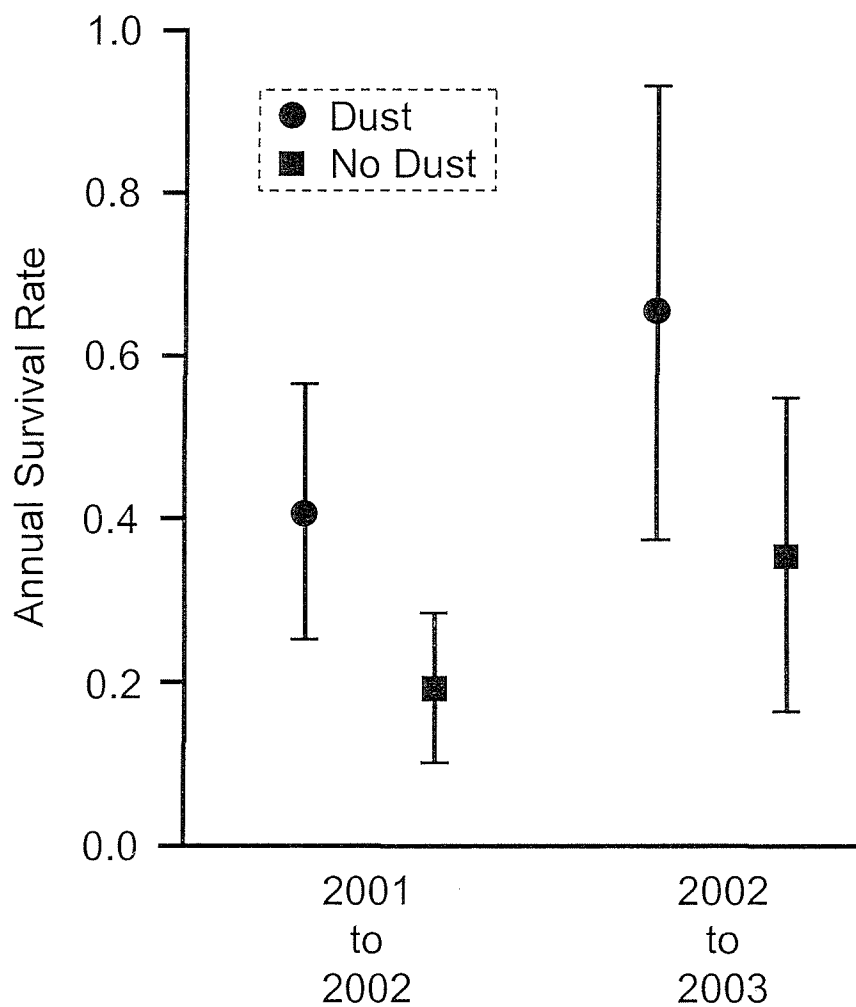


Figure 3. Survival rates (means, 95% CIs) for Utah prairie dogs on dusted and non-dusted plots during 2001-2003, as estimated by the preferred model of program RELEASE (model selection via AIC).

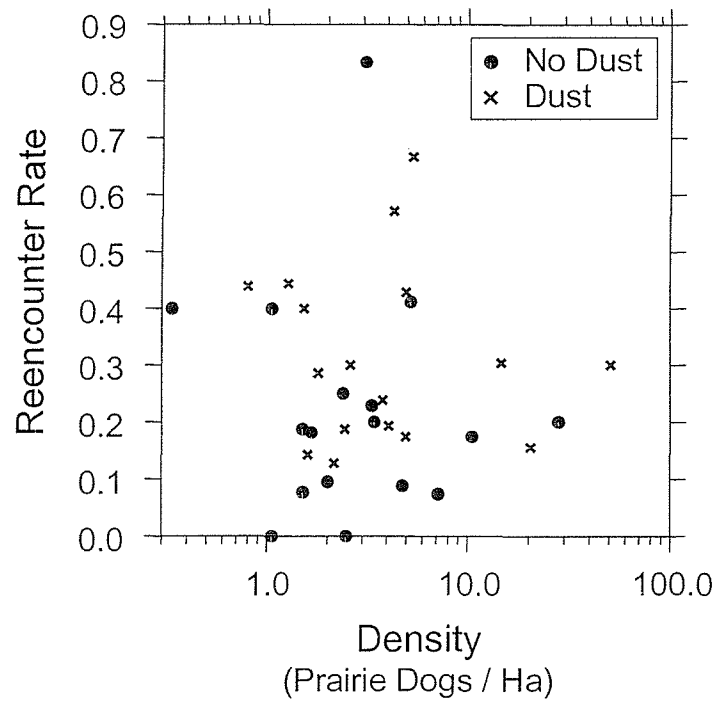


Figure 4. Relationship between densities of Utah prairie dogs estimated by visual counts and reencounter rates of marked prairie dogs trapped the following year.

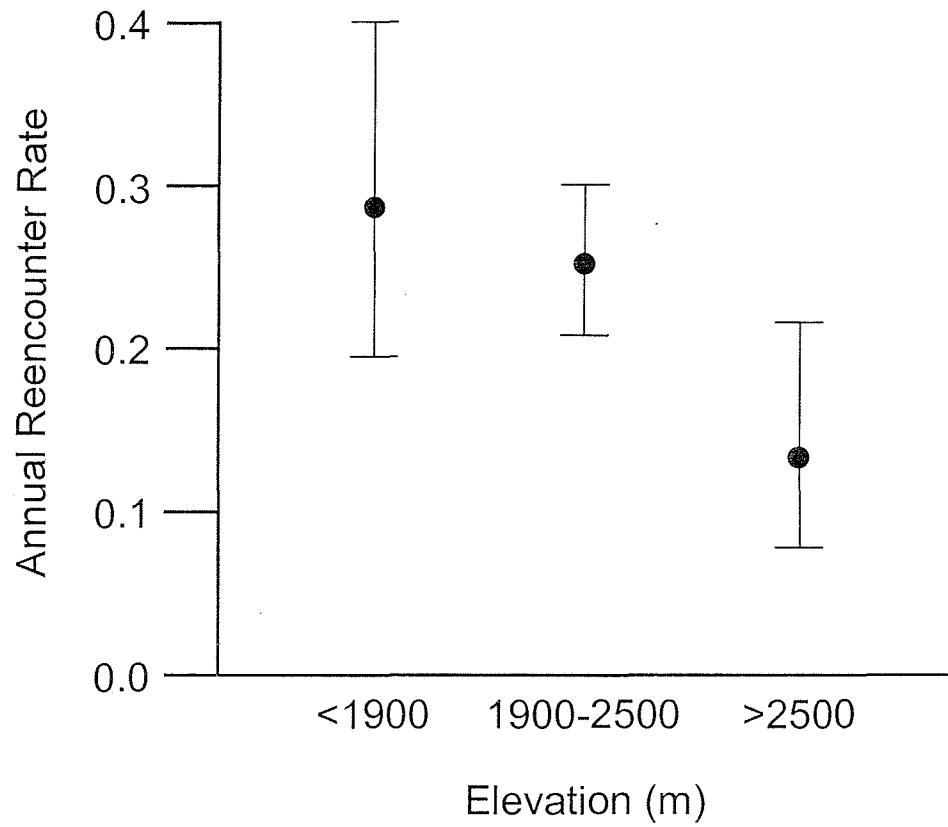


Figure 5. Reencounter rates (means, 95% CIs) for Utah prairie dogs at low, medium, and high elevations during 2001-2003, without considering effects of other variables.

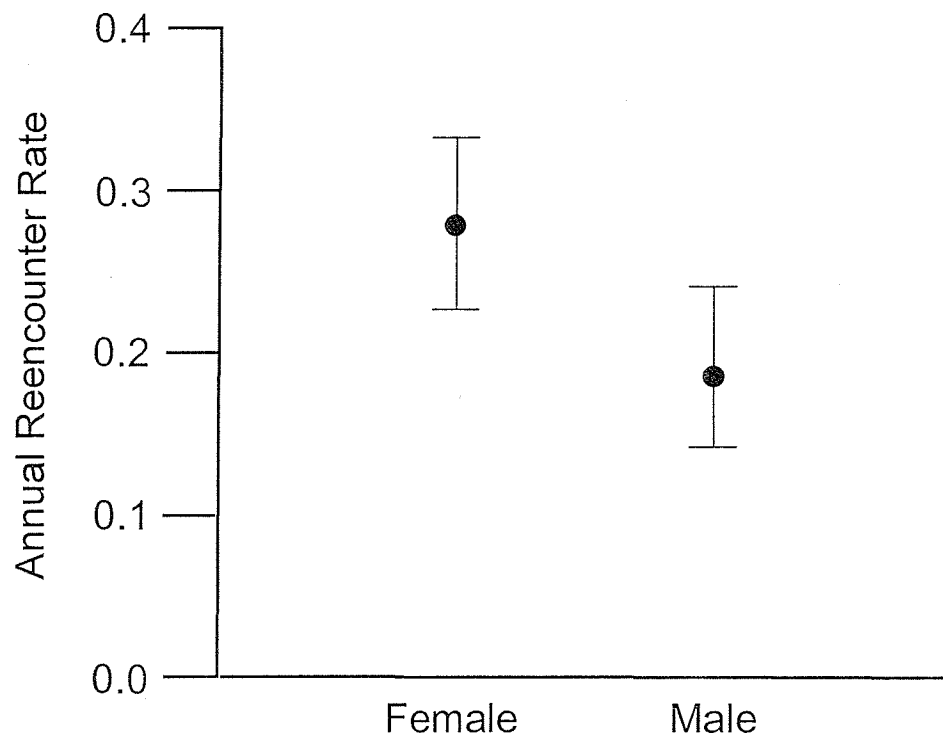


Figure 6. Reencounter rates (means, 95% CIs) for male and female Utah prairie dogs during 2001-2003, without considering effects of other variables.

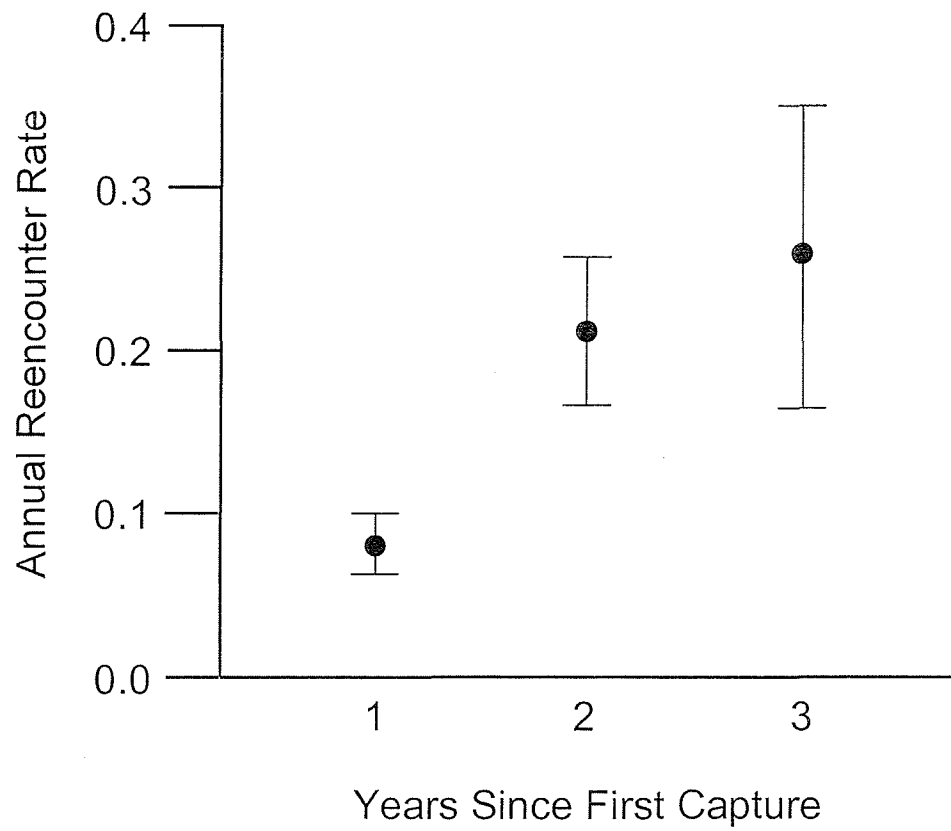


Figure 7. Reencounter rates (means, 95% CIs) for Utah prairie dogs with various capture histories during 2001-2003, without considering effects of other variables.

