



Research Article

Spread of Plague Among Black-Tailed Prairie Dogs Is Associated With Colony Spatial Characteristics

TAMMI L. JOHNSON,¹ *Montana Ecology of Infectious Diseases, Organismal Biology and Ecology, Division of Biological Sciences, University of Montana, Missoula, MT 59812, USA*

JACK F. CULLY, JR., *USGS-BRD Kansas Cooperative Fish and Wildlife Research Unit, Division of Biology, Kansas State University, Manhattan, KS 66506, USA*

SHARON K. COLLINGE, *Department of Ecology and Evolutionary Biology, Environmental Studies Program, University of Colorado-Boulder, Boulder, CO 80309, USA*

CHRIS RAY, *Department of Ecology and Evolutionary Biology, University of Colorado-Boulder, Boulder, CO 80309, USA*

CHRISTOPHER M. FREY, *Arthur Temple College of Forestry and Agriculture, Stephen F. Austin State University, Nacogdoches, TX 75962, USA*

BRETT K. SANDERCOCK, *Division of Biology, Kansas State University, Manhattan, KS 66506, USA*

ABSTRACT Sylvatic plague (*Yersinia pestis*) is an exotic pathogen that is highly virulent in black-tailed prairie dogs (*Cynomys ludovicianus*) and causes widespread colony losses and individual mortality rates >95%. We investigated colony spatial characteristics that may influence inter-colony transmission of plague at 3 prairie dog colony complexes in the Great Plains. The 4 spatial characteristics we considered include: colony size, Euclidean distance to nearest neighboring colony, colony proximity index, and distance to nearest drainage (dispersal) corridor. We used multi-state mark–recapture models to determine the relationship between these colony characteristics and probability of plague transmission among prairie dog colonies. Annual mapping of colonies and mark–recapture analyses of disease dynamics in natural colonies led to 4 main results: 1) plague outbreaks exhibited high spatial and temporal variation, 2) the site of initiation of epizootic plague may have substantially influenced the subsequent inter-colony spread of plague, 3) the long-term effect of plague on individual colonies differed among sites because of how individuals and colonies were distributed, and 4) colony spatial characteristics were related to the probability of infection at all sites although the relative importance and direction of relationships varied among sites. Our findings suggest that conventional prairie dog conservation management strategies, including promoting large, highly connected colonies, may need to be altered in the presence of plague. © 2011 The Wildlife Society.

KEY WORDS *Cynomys ludovicianus*, disease, epizootic, mark–recapture, multi-state, Program MARK, *Yersinia pestis*.

Exotic zoonotic diseases pose a substantial threat to native wildlife species as well as to human health. Management is enhanced by an understanding of the ecological factors associated with the spread of disease. A challenge to studying transmission of mammalian wildlife diseases in natural environments is that infected individuals are rarely observed because common host species such as mice and rats are inconspicuous in their activity and carcasses are removed by scavengers. We overcame these challenges by studying a conspicuous, highly social colonial rodent, the black-tailed prairie dog (*Cynomys ludovicianus*), to characterize the dynamics of wild (sylvatic) plague at large spatial scales. Sylvatic plague is an exotic vector-borne disease caused by the bacterium *Yersinia pestis*. Black-tailed prairie dogs are highly susceptible to plague because they have no known natural immunity (Poland and Barnes 1979, Barnes 1982, Antolin et al. 2002). These colonial mammals are also highly visible and because colonies die very quickly, they serve as an

excellent bioassay for detection of plague and as an early warning system for wildlife managers and public health officials (Cully et al. 2000).

Yersinia pestis is a virulent pathogen in prairie dogs that can be rapidly transmitted among individuals within a colony as well as among colonies across large landscapes. Individual prairie dogs infected with plague suffer mortality of nearly 100% (Barnes 1993). Because plague is highly infectious, it may be more readily transmitted between nearby prairie dog colonies. Understanding plague dynamics within a landscape requires consideration of several scale-dependent processes. At the scale of the colony, with sizes ranging from <1 ha to >1,000 ha, prairie dog mortality rates can be >95% (Cully and Williams 2001). Plague appears to spread within colonies either through contact between prairie dogs and infected fleas or possibly as a result of direct transmission between prairie dogs through contact with infected aerosols, blood, or saliva (Webb et al. 2006).

At coarser scales of resolution (national grasslands), covering anywhere from 40,000 ha to >400,000 ha, mechanisms of plague transmission between colonies are not clear. Possibilities include transport of infected fleas by dispersing

Received: 19 May 2009; Accepted: 27 May 2010

¹E-mail: tammi.johnson@umontana.edu

prairie dogs, transport of infected fleas among colonies by carnivores or raptors that regularly move among colonies, or contact with infected rodents of other species or their fleas (Barnes 1993, Cully 1993, Cully and Williams 2001, Antolin et al. 2002, Salkeld and Stapp 2006). Transmission of plague into prairie dog colonies may be related to spatial characteristics of the colonies or to landscape features that enhance or inhibit movement.

Previous research has considered the importance of colony size and isolation and landscape characteristics such as the proximity and cover of nearby stream corridors, lakes, and roads (Lomolino et al. 2004, Stapp et al. 2004, Collinge et al. 2005, Antolin et al. 2006, Snäll et al. 2008). For example, Lomolino et al. (2004) and Stapp et al. (2004) found increased plague activity in clustered colonies. Similarly, Collinge et al. (2005) and Snäll et al. (2008) found that in Colorado and Montana, die-offs were more likely to occur on larger colonies close to previous die-offs and surrounded by fewer lakes, streams, and roads—geographic features that may act as barriers to movement of plague hosts and vectors. Roach et al. (2001) and Antolin et al. (2006) suggested that drainages may facilitate movement of prairie dogs among colonies. Prairie dogs in colonies connected by (typically dry) drainage corridors were more likely to share genetic similarities providing evidence that higher rates of prairie dog dispersal occur along these corridors.

To expand upon previous research, we sought to further explore the relationship between prairie dog colony spatial characteristics and plague infection. Specifically, we examined the effects of colony size, Euclidean distance to nearest neighboring colony, colony proximity index (Gustafson and Parker 1992), and distances to nearest dry-creek drainages (that may act as dispersal corridors) on probability of plague occurrence.

We used multi-state (MS) mark–recapture models, which are an extension of Cormack–Jolly–Seber (CJS) models that allow for an individual to be encountered in ≥ 2 categorical states (Brownie et al. 1993, Lebreton and Pradel 2002), in this case, affected by plague or not.

Multi-state models have been used previously to examine disease transmission among individuals (Faustino et al. 2004, Senar and Conroy 2004, Lachish et al. 2007). We proposed a novel application of MS modeling to investigate infection dynamics of colony complexes over a 7-year period. Our study is the first attempt to use mark–recapture methods to examine disease dynamics in spatially structured populations. We expected that large colonies close to neighboring colonies and low-lying dry creek drainages would have the highest infection rates.

STUDY AREA

We examined the effect of colony spatial characteristics on plague occurrence at 3 national grasslands with large complexes of black-tailed prairie dog colonies in the western Great Plains (Table 1). We mapped prairie dog colonies to identify mortality events indicative of plague at 3 study sites: Cimarron National Grassland, Kansas (101.9°W, 37.1°N), Comanche National Grassland, Colorado (102.7°W, 37.0°N), and Kiowa and Rita Blanca National Grasslands, at the intersection of New Mexico, Texas, and Oklahoma (102.8°W, 36.4°N), hereafter Cimarron, Comanche, and Kiowa-Rita Blanca, respectively (Fig. 1).

METHODS

We mapped prairie dog colonies only on publicly held portions of each grassland. In 1999, we obtained colony data from J.F. Cully, Jr. (Kansas State University, unpublished

Table 1. Total number and area (ha) of black-tailed prairie dog colonies at 3 national grasslands, Comanche, Cimarron, and Kiowa-Rita Blanca, 1999–2006. GL = total amount of suitable prairie dog habitat at each study area^a.

Study area	Years	GL habitat area (ha)	No. of colonies	Total colony area (ha)	Mean colony area (ha)	SD colony area (ha)
Cimarron	1999	34,174	39	683	17.5	23.3
	2001		41	1,008	24.6	32.5
	2002		45	1,344	29.9	37.9
	2003		49	1,621	33.1	43.7
	2004		53	2,307	43.5	55.1
	2005		52	2,358	45.4	58.5
	2006		42	2,001	51.1	71.7
Comanche	1999	155,016	67	788	11.8	13.2
	2001		87	1,763	20.3	21.3
	2002		101	2,495	24.7	31.7
	2003		106	2,668	25.2	28.8
	2004		122	4,911	40.3	46.7
	2005		134	5,999	44.8	59.9
	2006		133	2,457	18.5	42.5
Kiowa-Rita Blanca	1999	40,749	34	695	20.5	27.5
	2001		42	1,607	38.3	36.2
	2002		61	2,185	35.8	40.8
	2003		65	2,740	42.2	49.3
	2004		66	1,808	27.4	42.8
	2005		61	1,126	18.5	37.6
	2006		58	673	11.6	26.0

^aWe obtained potential habitat area figures from agency biologists for Cimarron National Grassland (A. Chappell, U.S. Forest Service, personal communication) and Comanche National Grassland (D. Augustine, U.S. Forest Service, personal communication) and from the Grassland Management Plan for Kiowa Rita and Blanca National Grasslands (U.S. Forest Service, Cibola National Forest 1999).

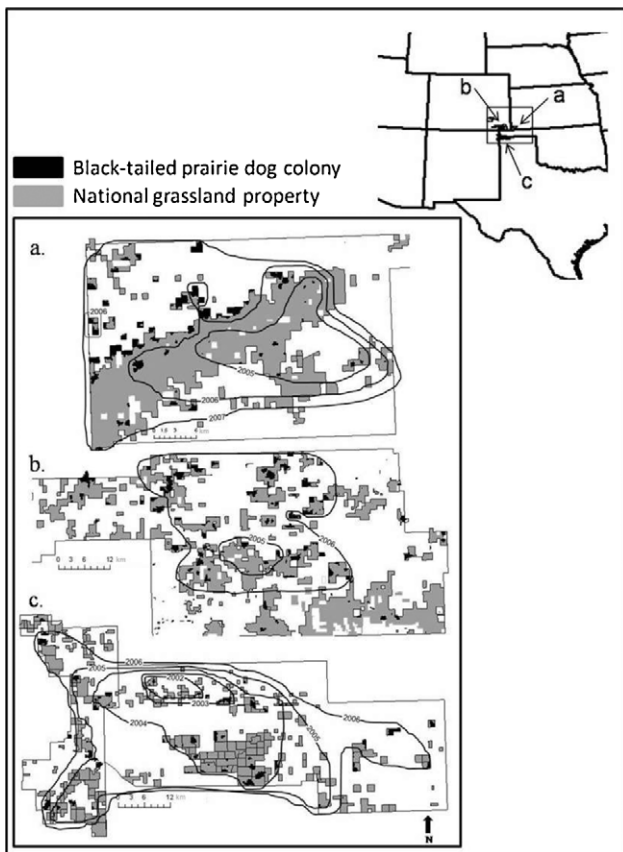


Figure 1. Location of study areas and the annual spread of plague among black-tailed prairie dog colonies (black polygons) at: (a) Cimarron National Grassland, 2005–2007, (b) Carizo Unit of the Comanche National Grassland, 2005–2006, and (c) Kiowa-Rita Blanca National Grasslands, 2002–2006. Contour lines show the spatial and temporal extent of plague across each grassland. We hand-drew contour lines to surround the cumulative area affected by plague each year beginning with the initial outbreak.

data). In 2001–2006, we mapped prairie dog colonies between late May and early October. We mapped all colonies at each grassland during a continuous 1- to 2-week period, depending on the number of colonies. To investigate annual changes in colony size, we mapped only areas actively occupied by prairie dogs. We identified active colonies by visually and audibly locating prairie dogs and determined the boundary of active areas by signs of recent digging on and near burrow mounds, presence of fresh prairie dog scat, and clipped vegetation indicating foraging activity or the characteristic mowing that prairie dogs undertake to enhance visibility on the colony (Hoogland 1995).

Identification of Plague

Yersinia pestis infections in black-tailed prairie dogs cause mortality rates of nearly 100% (Poland and Barnes 1979, Barnes 1993, Cully 1993, Antolin et al. 2002, Gage and Kosoy 2005). When a plague epizootic occurs on a black-tailed prairie dog colony, the population collapses to usually <10% of its former area and often to zero survivors. When a colony collapses, if animals remain, they aggregate into smaller areas and usually occur at densities that appear similar to precollapse densities. We assumed that colony area is a reasonable surrogate for colony population, and we inter-

preted a >90% reduction in active colony area as a plague event.

Where we encountered colonies that had died, we attempted to collect fleas from burrows to be analyzed at the Centers for Disease Control and Prevention (CDC), Bacterial Zoonoses Branch, in Fort Collins, Colorado, for presence of *Y. pestis*. We did not attempt to verify presence of *Y. pestis* at every collapsed colony, but we wanted to verify its presence on each grassland.

Prairie Dog Colony Data

We obtained spatial data by traversing colony boundaries on an all-terrain vehicle and recording a positional reading every second using a hand-held Global Positioning System (GPS). We differentially corrected all spatial data using Trimble Pathfinder software and information from a base station in Elkhart, Kansas, which was located <100 km from all study sites. The dynamic growth and reduction of colonies as a result of plague required us to define closely located colonies as single observational units prior to analysis. Colonies located close to other colonies (usually <1 km) and with no barrier between them often merge together after several years, forming one colony. Likewise, when large colonies are infected with plague, the high level of die-off frequently fragments them into small, unconnected colonies. We treated colonies that coalesced during our study, or large colonies that fragmented as a result of plague, as one colony for analyses; we summed the area of the components to calculate the proximity index, we connected the group of colonies for FRAGSTATS to treat the group as an individual unit, and we selected the shortest measurement of edge to nearest neighbor or to nearest drainage from among the component sub-colonies.

We incorporated yearly GPS data into a Geographic Information System projected to North American Datum 1927, Universal Transverse Mercator zone 13 North, along with drainage data (1995 United States Census Bureau TIGER files, <http://www.census.gov/geo/www/tiger/>, accessed Nov 2004) to quantify 4 spatial characteristics for each colony in each year of the study: colony area (ha), distance to the nearest neighboring colony (m), colony proximity index, and distance to the nearest drainage (m). We quantified nearest neighbor, edge-to-edge, and nearest drainage edge-to-drainage distances using the Nearest Feature ESRI ArcScript (Nearest Features Version 3.8b, http://www.jennessent.com/arcview/arcview_extensions.htm, accessed Nov 2004). We calculated colony proximity, which accounts for both the size and distance to neighboring colonies, using the spatial pattern analysis program FRAGSTATS (FRAGSTATS version 3.3, <http://www.umass.edu/landeco/research/fragstats/fragstats.html>, accessed Dec 2009).

Modeling Approach

We applied MS mark–recapture modeling to colonies, rather than individual animals, which can be encountered in 1 of 3 categorical states: plague, no plague, and not detected–not recovered. Multi-state models allowed us to examine if the effects of plague among colonies were similar among study sites, determine if the effects were constant over time or changed annually, obtain estimates of colony survival rates

for both healthy and infected colonies, and estimate colony infection (collapse) and recovery (growth following collapse) rates. Whereas CJS models allow estimation of apparent survival (ϕ), MS models allow estimation of state-specific apparent survival (S) and transition rates among states (ψ), where $S\psi = \phi$. Categorical states used in previous MS models have included social status (i.e., breeding or non-breeding), age (i.e., ad or juv), and colony location (i.e., occupied or unoccupied; reviewed in Sandercock 2006). More recently, MS models have been applied to the transition of individuals to and from a disease state (Faustino et al. 2004, Senar and Conroy 2004, Lachish et al. 2007). Our approach is novel in that we modeled disease status for whole colonies of black-tailed prairie dogs. Because plague causes mortality of >95% of the individuals within a colony (Cully and Williams 2001), we believe the effects of plague at the colony level is an appropriate measure. Thus, we estimated demographic parameters including survival, infection, and recovery rates, for colonies rather than individuals.

Benefits of our approach included the estimation of demographic parameters that will be useful to managers of prairie dogs, in addition to a model structure that allowed us to take advantage of the full 7-year data set and compensate for occasions when we did not encounter colonies. The MS modeling structure was able to handle missing data, which is a limitation of logistic regression that would have precluded the use of some of our data. After we discovered a colony, it remained in approximately the same place and we visited it each year with a probability of $P \sim 1$. The deviation from 1 was small and resulted from discovery of new colonies, which may have been missed during ≥ 1 year prior to discovery (Table 2). If $P = 1$, then apparent survival is equal to return rate and we can analyze encounter histories using logistic regression (Sandercock 2006). Due to the nature of the data collection discussed above (i.e., revisiting all known colonies each year), we expect $P \sim 1$. Although the modeling structure allowed for estimates of state specific

survival and probability of detection, as well as rates of infection and recovery, our primary goal was to model the transition of colonies from a healthy to a diseased state as a function of colony spatial characteristics.

Multi-State Modeling

At each annual census i , we assigned 1 of 3 states to each colony: Plague (P) if the colony had collapsed since the previous year, not recovered (0) if we did not discover the colony or it was not re-established since an earlier plague event, or no-plague (N) if the colony showed apparently normal prairie dog activity as described above. We thus coded the resulting encounter history for each colony to indicate infection history. For example, 0NNP0NN represents a 7-year period in which we did not detect the colony on the first occasion, detected it as a no-plague colony on occasions 2 and 3, detected it as a plague colony on occasion 4, observed no activity in year 5, and then detected it again as a no-plague colony on occasions 6 and 7. Assuming that the status of a colony at i is a function of its status at $i - 1$ (Arnason 1972, Brownie et al. 1993, Schwarz et al. 1993), and that we were able to correctly assign colonies to each state (Kendall 2004), we used MS encounter histories to estimate 3 types of parameters: S (the state-specific probability of colony survival, i.e., survival of healthy [no-plague] colonies S^N and survival of plague colonies S^P , which means the colony collapsed to <10% of its former size, but was not extirpated), ψ (the probability of a colony transitioning from one state to the other, i.e., the probability of succumbing to plague ψ^{N-P} or the probability of colony recovery ψ^{P-N}), and P (the probability we detected a colony). We analyzed the encounter histories representing 7 encounter periods (1999–2006) with the MS model in Program MARK by selecting the “multi-strata recaptures only” model (White and Burnham 1999). To account for the absence of data in 2000, we specified time intervals of 1 or 2 years in Program MARK.

Table 2. Reduced model selection from Program MARK showing top 5 models fitted to encounter histories of plague infected and uninfected black-tailed prairie dog colonies at Cimarron National Grassland, Comanche National Grassland, and Kiowa-Rita Blanca National Grasslands, 1999–2006. S = colony survival, P = probability of colony detection, ψ^{NP} = probability of infection, and ψ^{PN} = probability of recovery. The best model is that with the lowest Akaike information criterion adjusted for small sample size (AICc) and support for each model is given by Δ AICc, the difference between the AICc value of the minimum AICc model and successive models.

Study area	Model ^a	AICc	Δ AICc	AICc wt	Model likelihood	No. of parameters	Deviance
Cimarron	$S(s) P(c) \psi^{NP}(t) \psi^{PN}(c)$	143.3	0	0.709	1.000	8	126.8
	$S(s) P(s) \psi^{NP}(t) \psi^{PN}(c)$	145.3	2.0	0.266	0.376	9	126.6
	$S(s) P(t) \psi^{NP}(t) \psi^{PN}(c)$	150.3	7.0	0.022	0.031	13	122.9
	$S(s) P(c) \psi^{NP}(t) \psi^{PN}(t)$	154.1	10.9	0.003	0.004	13	126.8
	$S(c) P(c) \psi^{NP}(t) \psi^{PN}(c)$	212.6	69.3	0.000	0.000	7	198.2
Comanche	$S(s) P(c) \psi^{NP}(t) \psi^{PN}(c)$	373.1	0.0	0.866	1.000	9	354.8
	$S(s) P(c) \psi^{NP}(t) \psi^{PN}(t)$	377.7	4.7	0.084	0.097	14	349.0
	$S(s) P(t) \psi^{NP}(t) \psi^{PN}(c)$	378.8	5.7	0.050	0.058	14	350.1
	$S(c) P(c) \psi^{NP}(t) \psi^{PN}(c)$	391.0	17.9	0.000	0.000	8	374.7
	$S(t) P(c) \psi^{NP}(t) \psi^{PN}(c)$	397.4	24.3	0.000	0.000	13	370.8
Kiowa-Rita Blanca	$S(s) P(c) \psi^{NP}(t) \psi^{PN}(c)$	436.7	0.0	0.487	1.000	9	418.2
	$S(s) P(s) \psi^{NP}(t) \psi^{PN}(c)$	437.9	1.2	0.270	0.555	10	417.2
	$S(s) P(t) \psi^{NP}(t) \psi^{PN}(c)$	438.2	1.5	0.229	0.471	14	408.9
	$S(s) P(c) \psi^{NP}(t) \psi^{PN}(t)$	443.8	7.1	0.014	0.030	14	414.5
	$S(t) P(c) \psi^{NP}(t) \psi^{PN}(c)$	472.4	35.7	0.000	0.000	13	445.2

^a Model effects include: s = strata-specific estimate (N = uninfected and P = infected), t = time-based estimate, c = constant estimate.

Multi-state models in Program MARK allow the user to incorporate individual covariates that may influence demographic parameters. We were interested in the effects of colony characteristics on occurrence of plague and included colony area, nearest colony (Euclidean distance), proximity index (which considers the area and distance of all colonies within a search radius of 5 km and ranges beginning at 0 for a colony with no neighbors within the specified distance and increasing as more and larger colonies are within 5 km), and nearest drainage distance as individual covariates for each colony for each of the 7 years. We then modeled relationships between parameters and year-specific spatial data for each colony at each transition interval (i.e., 1999–2001, 2001–2002, etc.). We fit all models using a logit link and defined model structure using the design matrix option of Program MARK.

Model fitting proceeded in 4 steps. First, we performed a priori selection of factors (t = time, c = constant effects, or s = state-specific effects) for each demographic parameter in the model. The reduced model selected for all study sites was: $S_s \psi_t^{N \rightarrow P} \psi_c^{P \rightarrow N} \Pi_c$ (Table 2). Second, we performed goodness-of-fit tests (GOF) to test and control for overdispersion in the reduced model. We performed GOF tests on the Arnason–Schwarz MS model using Test 3G in Program U-CARE version 2.2.5 to detect any overdispersion that may be present in our models (Choquet et al. 2003, Pradel et al. 2003). Test 3G in Program U-CARE tests the assumption of transience in that each colony present at time (t) has the same probability of being present at time ($t + 1$; Pradel et al. 2003, 2005).

Third, to select best-fit reduced models for each site, we systematically substituted time, strata, and constant effects to model variation in parameters. Given the high mortality caused by epizootic plague in prairie dogs, probability of infection and collapse ($\psi^{N \rightarrow P}$) was the parameter of primary interest. We expected that infection rate would vary with time, whereas all other parameters would be constant, but strata-specific (except P , which is constant due to consistent sampling effort). Finally, to evaluate the biological impact of colony spatial characteristics on probability of colony infection, we used the model with the greatest support from our reduced model selection (Table 2) and modeled infection ($\psi^{N \rightarrow P}$) as a function of colony-specific covariate values for colony area, distance to nearest drainage, distance to nearest neighboring colony, and colony proximity.

We determined relative support for each candidate model using Akaike's Information Criterion corrected for small sample size (AICc). We ranked models using Δ AICc values, which represent the difference between the AICc value of the minimum AICc model and successive models. Models with Δ AICc ≤ 2 are considered equally parsimonious and parameter estimates from such models are by definition given equal consideration. To estimate parameters, we used the model averaging techniques described by Burnham and Anderson (1998) and report the weighted average of parameter estimates and unconditional standard errors that correct for model uncertainty. To illustrate changes in real parameter estimates of infection rate ($\psi^{N \rightarrow P}$) as a function of

colony spatial covariates, we transformed the logit function parameters with the user-specified covariate value option in Program MARK. To avoid extrapolating outside the range of the covariate values and to illustrate the trends, we predicted mean $\psi^{N \rightarrow P}$ at 5 evenly spaced intervals within the range of observed covariate values.

RESULTS

We were able to document presence of plague at all 3 grasslands during the course of the study. We confirmed the presence of plague via the detection of *Y. pestis* in fleas collected from burrows in colonies that had collapsed at Kiowa-Rita Blanca, at Cimarron, and in colonies north of the Comanche in Animas County, Colorado, but not on the Comanche. We attributed the die-off of colonies at all 3 grasslands as resulting from plague because the pattern of spread was clearly indicative of contagious spread of disease as described in previous reports of plague. Our criterion for identifying plague was that a colony or suite of colonies declined in active area by $>90\%$, which usually meant that plague had been active since our previous year's census.

Plague Dynamics

The spatial and temporal dynamics of the epizootics differed among the grasslands. We observed colony die-off at all study sites during the study (Fig. 1). At the Cimarron National Grassland in 2005, a group of smaller, isolated colonies on the edge of the complex, south of the Cimarron River, was extirpated. Over the following 3 years, plague expanded its reach to colonies north of the Cimarron River 2 km distant and spread among colonies to the west through 2008 (Fig. 1a).

At Comanche, we first documented colony die-off on the Timpas (north) unit in 2002, and local extirpation of a few neighboring colonies continued through 2004. Colonies on the Timpas unit of the Comanche were small and widely dispersed, and we did not document widespread colony die-off there. In 2005, 6 medium-sized colonies near the center of the colony complex on the Carrizo (south) unit of the grassland were extirpated. In 2006, plague spread among colonies throughout the central Carrizo Unit and decimated or extirpated $>50\%$ of the colonies there (Fig. 1b). The epizootic continued to spread on the Carrizo unit during 2007 (D. Augustine, Comanche National Grassland, personal communication).

At Kiowa-Rita Blanca we first noted colony die-off in 2002. Plague began at the edge of the north-central portion of the grassland, where 3 small colonies were initially affected. Colony die-off continued to occur annually, affecting larger colonies and moving to almost every extent of the grassland over the following 4 years (Figs. 1c and 2a). By 2006, plague had affected 64 of 72 colonies at the Kiowa-Rita Blanca National Grassland. As at Cimarron, where we first recorded the epizootic at the edge of the complex, plague spread over several years in a wave-like pattern across the grassland.

The location and size of the first colonies affected by a plague epizootic influenced the speed and spatial pattern with which the epizootic spreads to neighboring colonies. At the Carrizo unit of the Comanche, where we first

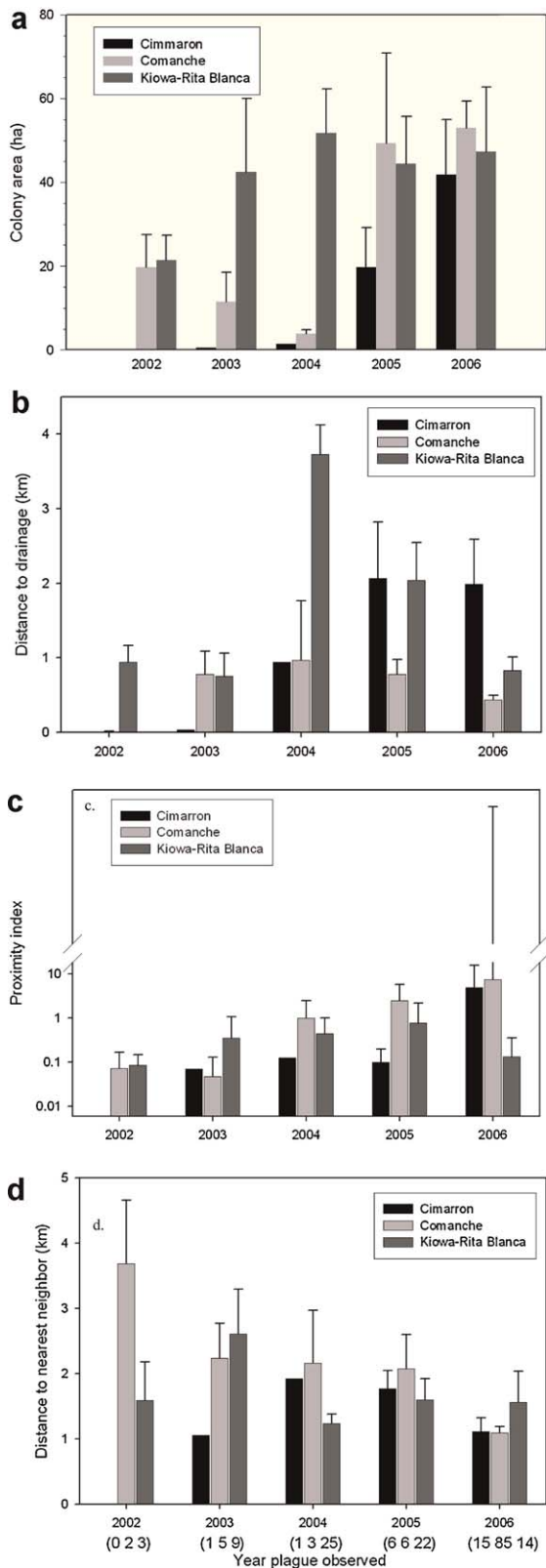


Figure 2. Annual mean covariate values + 1 standard error for black-tailed prairie dog colonies that experienced plague, 2002–2006. Panels display values for: (a) mean colony area (ha), (b) mean distance to drainage (km), (c) colony proximity index, and (d) mean nearest neighbor distance of plague affected colonies calculated from the previous years' data (km). The number of colonies with plague is shown for each grassland below the x-axis labels in panel (d).

observed plague at the center of the colony complex, plague spread rapidly among colonies in all directions and in 2 years, 2005–2007, and reduced colony area by 89%. In contrast, at Cimarron between 2005 and 2007, and at Kiowa-Rita Blanca between 2003 and 2006, where the epizootic began at the edge of each complex, plague spread in a wave-like fashion (Fig. 1) and reduced colony area by 54% and 55%, respectively. The incidence of plague among colonies increased within the 3 grasslands during the study and as time progressed, larger, more clustered colonies spaced further from drainages were infected (Fig. 2).

Due to the different topography among study sites and even within sites, there was substantial annual variation in the distance to nearest drainage of infected colonies. At Comanche, the distance to nearest drainage of infected colonies was lowest relative to the other grasslands (Fig. 2) because plague was initiated in the interior of the colony complex (Fig. 1b) where drainages were numerous and many colonies were located close to drainages. When we first observed plague at Kiowa-Rita Blanca in 2002, the colonies infected were small and located close to drainages relative to the following years, but as the epizootic moved across the grassland, larger colonies spaced further from drainages were infected (Fig. 2a,b).

Mark-Resighting Modeling

The effects of plague on colony survival and recovery, as well as the transition from no-plague to plague and vice versa, differed among sites. At all sites, the data were best represented by MS models that describe state-specific, but constant (not time-varying), estimates of apparent survival (S), a constant (over time) estimate for the probability of encountering a colony (P), a time-dependent estimate of infection rate (ψ^{N-P}), and a constant recovery rate (ψ^{P-N} ; Table 2). We observed no evidence of overdispersion for the global model fit to data from any site (U-CARE GOF P -values: $P = 0.160$, $P = 0.207$, $P = 0.325$, for Cimarron, Comanche, and Kiowa-Rita Blanca, respectively). Results of the analysis of the reduced models indicated that the time-based parameterization of infection rate best-described patterns of the prevalence of plague in the 3 grasslands, and we expected the model estimates of increased infection rates through time based on field observations of epizootic plague. Apparent survival (ϕ) of uninfected colonies was 1.00 in all grasslands (Table 3), whereas survival of infected colonies was variable among the grasslands, ranging from 0.00 at Cimarron to 0.82 at Comanche (Table 3). There was no variation in survival of uninfected colonies in the 3 grasslands and survival of infected colonies in Cimarron, thus it was not possible to estimate a standard error for this parameter (Table 3). Recovery rates varied across study sites, ranging from 0.14 at Cimarron to 0.85 at Kiowa-Rita Blanca.

The colony spatial characteristics represented by individual colony covariates (area, nearest neighbor distance, and colony proximity) contributed to model fit at every site, however, results of model rankings (Table 4) and of parameter estimates (Table 5) varied among sites. For the Cimarron and Comanche there was a reduction of 9.8 and 9.1 AICc

Table 3. Real parameter estimates obtained from the best-fit reduced model in Program MARK fit to encounter histories of plague infected and uninfected black-tailed prairie dog colonies at Cimarron National Grassland, Comanche National Grassland, and Kiowa-Rita Blanca National Grasslands, 1999–2006. Parameter estimates for all parameters except infection rate are constant; infection rate varied with time. S = colony survival, P = probability of colony detection, ψ^{NP} = probability of infection, and ψ^{PN} = probability of recovery.

Study area	S		P	ψ^{NP}						ψ^{PN}
	No plague	Plague	(c)	t_1	t_2	t_3	t_4	t_5	t_6	(c)
Cimarron	1.00	0.00	1.00	0.0	0.0	0.02	0.02	0.13	0.33	0.14
	0.00 ^a	0.00	0.00	0.0	0.0	0.02	0.02	0.05	0.07	0.00
Comanche	1.00	0.82	1.00	0.0	0.02	0.04	0.03	0.05	0.61	0.77
	0.00	0.10	0.00	0.0	0.02	0.02	0.02	0.02	0.04	0.12
Kiowa-Rita Blanca	1.00	0.76	0.98	0.0	0.07	0.14	0.45	0.47	0.28	0.85
	0.00	0.06	0.01	0.0	0.04	0.05	0.07	0.08	0.07	0.06

^a Estimates of the standard errors are listed below parameter estimates.

Table 4. Covariate model selection for no plague to plague transition probability (ψ^{NP}) showing top 5 models fitted to encounter histories of infected and uninfected black-tailed prairie dog colonies at Cimarron National Grassland, Comanche National Grassland, and Kiowa-Rita Blanca National Grasslands, 1999–2006. S = colony survival, P = probability of colony detection, ψ^{NP} = probability of infection, and ψ^{PN} = probability of recovery. The best model is that with the lowest Akaike information criterion adjusted for small sample size (AICc) and support for each model is given by Δ AICc, the difference between the AICc value of the minimum AICc model and successive models.

Study area	Model ^a	AICc	Δ AICc	AICc wt	Model likelihood	No. of parameters	Deviance
Cimarron	$S(s) P(c) \psi^{NP}(t + A + P) \psi^{PN}(c)$	133.5	0.0	0.33	1.00	10	112.6
	$S(s) P(c) \psi^{NP}(t + A + N) \psi^{PN}(c)$	133.6	0.1	0.31	0.92	10	112.8
	$S(s) P(c) \psi^{NP}(t + A) \psi^{PN}(c)$	133.9	0.5	0.26	0.80	8	115.2
	$S(s) P(c) \psi^{NP}(t + A + D) \psi^{PN}(c)$	136.1	2.6	0.09	0.28	10	115.2
	$S(s) P(c) \psi^{NP}(t) \psi^{PN}(c)$	143.3	9.8	0.00	0.01	8	126.8
Comanche	$S(s) P(c) \psi^{NP}(t + A + N) \psi^{PN}(c)$	363.0	0.0	0.41	1.00	11	341.6
	$S(s) P(c) \psi^{NP}(t + N) \psi^{PN}(c)$	365.6	1.6	0.18	0.45	10	345.2
	$S(s) P(c) \psi^{NP}(t + N + P) \psi^{PN}(c)$	365.6	1.6	0.18	0.44	11	343.2
	$S(s) P(c) \psi^{NP}(t + D + N) \psi^{PN}(c)$	366.4	2.4	0.12	0.30	11	344.0
	$S(s) P(c) \psi^{NP}(t + A + D) \psi^{PN}(c)$	369.1	5.1	0.03	0.08	11	346.7
Kiowa-Rita Blanca	$S(s) P(c) \psi^{NP}(t + A) \psi^{PN}(c)$	435.1	0.0	0.346	1.00	10	414.4
	$S(s) P(c) \psi^{NP}(t + A + N) \psi^{PN}(c)$	436.5	1.4	0.180	0.50	11	413.6
	$S(s) P(c) \psi^{NP}(t) \psi^{PN}(c)$	436.7	1.6	0.137	0.44	9	418.2
	$S(s) P(c) \psi^{NP}(t + A + P) \psi^{PN}(c)$	436.9	1.8	0.119	0.41	11	414.0
	$S(s) P(c) \psi^{NP}(t + A + D) \psi^{PN}(c)$	437.2	2.1	0.080	0.34	11	414.4

^a Model effects include: s = strata-specific estimate (N = uninfected and P = infected), t = time-based estimate, c = constant estimate. Covariates include: A = colony area, D = distance to nearest drainage, N = distance to nearest neighboring colony, P = proximity index.

Table 5. Logit function parameter estimates and unconditional standard errors calculated by model averaging 11 covariate models fit for infected and uninfected black-tailed prairie dog colonies at Cimarron National Grassland (CIM), Comanche National Grassland (COM), and Kiowa-Rita Blanca National Grasslands (KRB), 1999–2006. Model parameters include: S = colony survival, P = probability of colony detection, ψ^{NP} = probability of infection, and ψ^{PN} = probability of recovery. Model effects include: s = strata-specific estimate (N = uninfected and P = infected), t = time-based estimate, and c = constant estimate. Covariates include: A = colony area, D = distance to nearest drainage, N = distance to nearest neighboring colony, and P = proximity index.

Study area	S		P	ψ^{NP}						ψ^{PN}				
	No plague	(- ^a) plague	(c)	(-) t_1 ^b	(-) t_2	(-) t_3	(-) t_4	(-) t_5	t_6 ^d	A	D	N	P	(.)
CIM	50.91	-70.51	5.59	0.0	0.0	-3.38	-3.42	-2.02	0.28	-0.026	0.002	-1.40	0.023	-1.13
	3,367.50 ^e	3,364.31	1.00	—	—	1.08	1.08	1.39	0.61	0.011	9.7e ⁻⁰⁴	0.054	0.007	301.75
COM	21.84	-20.36	5.71	0.0	-4.01	-3.50	-3.87	-3.44	0.68	0.003	0.056	-0.232	0.001	1.21
	0.42	0.42	0.71	—	0.75	0.55	0.62	0.47	0.31	8.3e ⁻⁰⁴	0.008	0.079	2.3e ⁻⁰⁴	0.66
KRB	21.49	-20.35	3.72	0.0	-1.67	-0.93	0.66	0.79	-1.02	0.004	0.001	-0.023	-0.002	1.73
	447.60	447.60	0.38	—	0.70	0.52	0.46	0.48	0.40	0.001	0.002	0.007	0.001	0.44

^a Negative symbol indicates the logit parameter is the estimated difference between apparent survival for non-plagued and plagued colonies.
^b We fixed ψ^{NP} at 0 for t_1 within all models fit to the data from the 3 grasslands and also for t_2 within the models for Cimarron.
^c Negative symbol indicates the logit parameter is the estimated difference in transition probability between t_6 and the respective time period.
^d Estimated transition probability for t_6 and the logit equation intercept for all other time intervals.
^e Unconditional SEs are listed below estimates of the logit function parameters.

between the time-based (reduced) model and the most parsimonious model containing area and colony proximity or area and nearest neighbor distance, respectively. The time-based model was an equally parsimonious fit to the colony data at Kiowa-Rita Blanca, with a reduction of 1.6 AICc between the time-based model and the highest ranking model containing area; however, colony area, nearest neighbor, and colony proximity were included in the top models (Table 4). At Comanche and Cimarron, models including colony area, nearest neighbor, and colony proximity provided the best fit to the data (Table 4). Colony area consistently contributed to model fit as indicated by the inclusion of area in the top model ($\Delta\text{AICc} = 0.0$) for each site (Table 4). Slope estimates of 0.004 and 0.003 on the logit scale at Kiowa-Rita Blanca and Comanche, respectively (Table 5), indicate an increasing probability of infection with increasing colony size (Fig. 3a). Whereas at Cimarron, a slope estimate of -0.026 on the logit scale indicates the opposite pattern (Table 5), where larger colonies had a decreased probability of infection (Fig. 3a). Distance to the nearest neighboring colony was important at Comanche. A slope estimate of 0.056 on the logit scale (Table 5) indicates that more isolated

colonies had decreased probabilities of infection (Fig. 3b). Finally, the effects of colony proximity varied among sites. At Comanche and Cimarron, colonies with high proximity values, those nearby other (often large) colonies, had an increased probability of infection as indicated by slope estimates of 0.001 and 0.023 on the logit scale, respectively (Table 5). Whereas at the Kiowa-Rita Blanca a slope estimate of -0.002 on the logit scale indicates the effect of colony proximity on probability of infection had the opposite pattern and was contrary to our predictions. Models containing distance to drainage consistently had a delta AICc of >2.0 indicating distance to drainage did not contribute significantly to the top models (Table 4).

DISCUSSION

Plague is the only disease known to cause high rates of mortality in prairie dogs. Other possible causes of die-offs are poisoning with rodenticides or shooting. Rodenticides have been banned from the national grasslands we studied since the 1980s, so poisoning was not an important issue. Shooting was allowed on the Cimarron and Kiowa-Rita Blanca but has been banned on the Comanche since 2001 (S. Shively, Comanche National Grasslands, personal communication). Shooting has the potential to eliminate prairie dogs from small colonies, perhaps as large as 2 ha, but probably has low-to-moderate impacts on survivorship and persistence on larger colonies. In an experimental shooting treatment, Pauli and Buskirk (2007) showed only small effects on survivorship as a result of shooting, but population growth was reduced relative to control sites with no shooting as a result of reduced reproductive output. The reduced reproduction was attributed to changes in foraging behavior that prevented females from accumulating adequate fat stores to produce and support full litters of pups the year following the experimental shooting. Presence of prairie dogs on colonies where shooting occurs may be less conspicuous than on undisturbed colonies, but indirect evidence in the form of burrow maintenance and digging, fresh scat, and clipped vegetation produce a different appearance than that of a colony where prairie dogs have been eliminated by plague. Within months after population collapse, burrow and crater mounds can be identified as inactive by the appearance of undisturbed dry mud on their surface, spider webs and dead plant material obstructing holes, a general lack of fresh digging (although sometimes signs of digging by other species may be present), emergence of vegetation on burrow mounds, and a profound absence of active, vocalizing prairie dogs. These changes may be seen <1 year after observing the colony as a hive of bustling prairie dog activity.

Multi-state demographic modeling is a new approach for evaluating the effects of disease on wildlife populations. During our 7-year study, we observed outbreaks of plague at 3 grasslands inhabited by black-tailed prairie dogs and found that colony spatial characteristics were correlated with infection. Annual mapping of colonies and mark-recapture analyses of disease dynamics in natural colonies led to 4 major results. First, plague outbreaks exhibited spatial and temporal variation among areas as well as within a given area. Second,

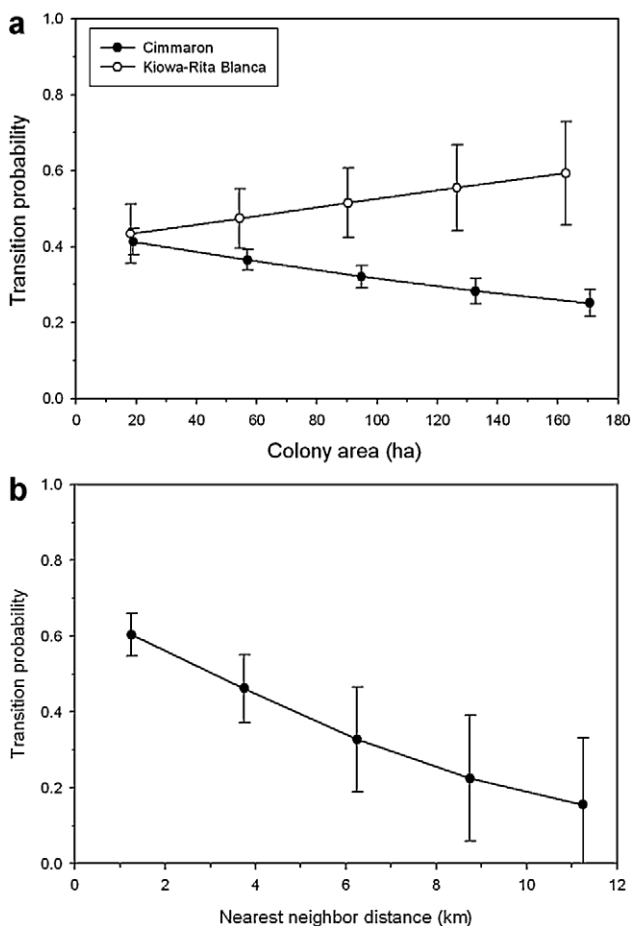


Figure 3. Multi-state model real parameter estimates of the probability of a black-tailed prairie dog colony becoming infected with plague (transition probability) ± 1 unconditional standard error as a function of: (a) colony area for Cimarron and Kiowa-Rita Blanca National Grasslands, and (b) nearest neighbor distances for Comanche grassland. These estimates represent the strongest covariate effects found for each of the three grasslands.

the site of initiation of epizootic plague may have substantially influenced the subsequent inter-colony spread of plague across a prairie dog complex. Third, the effects of plague on individual colonies differed among areas, which may provide insights into the long-term impacts of plague as well as implications for site-specific management during and between plague epizootics. Finally, colony spatial characteristics were related to the probability of infection but the magnitude and direction of the effects of varied among areas.

Annual mapping allowed us to document the multi-year spread of plague among colonies at our study areas. In past studies, when a colony die-off was observed in response to a plague epizootic, it appeared that extensive die-off of neighboring colonies occurred almost simultaneously (Cully et al. 1997, Cully and Williams 2001). For example, there was a major epizootic at Comanche in 1995 that devastated local populations of prairie dogs. Following the major epizootic, active colony area at Comanche decreased from 2,574 ha and had recovered to only 540 ha when colonies were mapped again in 1999 (Toombs 1997, J.F. Cully, Jr., unpublished data). Because these areas were not being actively monitored prior to the 1995 epizootic, it is possible that plague was present and moving through the grassland for several years prior to 1995. Rapid and widespread colony die-off in response to plague is not ubiquitous (Johnson 2005). Colony mapping, flea collection, and the confirmed presence of *Y. pestis* by the CDC at Kiowa-Rita Blanca between 2002 and 2004 revealed that plague was slowly moving across the grassland eliminating a few colonies each year until 2004 when nearly half of the colonies were extirpated, and by 2006, 89% of colonies there had been affected (Fig. 1c). Colony die-offs prior to 2000 at Cimarron appeared to be independent events because die-offs occurred at widely dispersed colonies (Cully et al. 2000, Cully and Williams 2001, Johnson 2005). Isolation and small size of many colonies may have slowed the spread of plague at these grasslands. Additionally at Cimarron, initial die-offs in 2004 and 2005 were primarily confined to small colonies and areas of the grassland south of the Cimarron River, which may usually serve as a barrier to the spread of plague. Once the large colonies on the north side of the river became infected, infection spread more rapidly among the large and less isolated colonies (Fig. 1a).

The location of initial colony die-off may play an important role in epizootic spread. If initial die-off occurs in isolated colonies, infection may be slow to spread to neighboring colonies, whereas if die-off occurs near other colonies, the epizootic may spread rapidly. Inter-colony distances may affect the epizootic spread of plague to neighboring colonies for several reasons. If plague is transferred among colonies by dispersing prairie dogs, inter-colony distance may substantially affect the success of dispersing prairie dogs because they are more vulnerable to predation when they are away from the colony (Hoogland 1995). Short nearest-neighbor distances should decrease time spent off a colony, thus increasing the chance of successful dispersal. Likewise, if plague is spread to neighboring colonies via the transport of infected fleas by pred-

ator species, a predator may be more likely to visit multiple colonies in an area where colonies are clustered on the landscape and close together.

Although our focus was to identify spatial characteristics of colonies that may impact the probability of infection with plague, our results of colony survival may have important implications for the long-term effects of plague. At Cimarron where survival of infected colonies was lowest, the long-term effects of plague may be more severe because fewer colonies infected with plague recovered and continued to support prairie dog populations (Table 3). It is possible that the low rate of colony survival at Cimarron was a result of the size of colonies when they became infected with plague. Smaller colonies were less likely to have surviving prairie dogs and may have been less likely to be recolonized following a plague event.

Colony infection rate was related to colony area at all study areas. Among study areas however, we observed differing trends in infection probability in relation to colony area. At Comanche and Kiowa-Rita Blanca, we observed a trend of increasing infection with increasing colony area. Conversely, at Cimarron, the trend was for smaller colonies to have increased infection probabilities (Fig. 3a). Variation in effects of colony size may be partially explained by examining the location of the initial colony die-off and the subsequent movement of plague among neighboring colonies. If the Cimarron epizootic had started north of the Cimarron River, nearer to the largest colonies, patterns of spread may have led to a different path, and the effect of colony area might have been similar to the other study areas. At Kiowa-Rita Blanca, we also observed the initial colony die-off in small colonies; however, these colonies were nearby larger colonies and, over the following 4 years of monitoring, continued to spread across the grassland in all directions. At Comanche, initial colony die-off occurred in a central portion of the grassland in medium to large colonies and spread more rapidly to large neighboring colonies over the following year.

Colony area has been a consistent indicator of colony susceptibility to plague. Our results at Comanche and Kiowa-Rita Blanca are consistent with previous reports where larger colonies were more likely to be infected with plague (Cully and Williams 2001, Lomolino and Smith 2001, Collinge et al. 2005, Snäll et al. 2008). Larger colonies may have an increased chance of contracting plague for several reasons. Contact with potential reservoir hosts such as deer mice (*Peromyscus maniculatus*) and northern grasshopper mice (*Onychomys leucogaster*; Barnes 1982, Thomas 1988, Gage et al. 1995) and their associated fleas may be more likely to occur on large prairie dog colonies because the colony is spread over a larger portion of the landscape and likely intersects the home ranges of more individuals of reservoir species. Additionally, we expected that a larger number of prairie dogs would be present at larger colonies, which may result in increased numbers of contacts not only between reservoir hosts and prairie dogs, but also among prairie dogs, which could increase the possibility of vector exchange (Cully and Williams 2001). Moreover, prairie dogs

may choose to disperse to larger colonies because of increased habitat suitability (Collinge et al. 2005), which may also increase transmission.

Our results also show that distance and proximity to other colonies may affect transmission. We expected that Euclidean distance to the nearest neighboring colony and colony proximity to other colonies would be important factors contributing to probability of infection. If plague is epizootically transferred among colonies, the proximity to other colonies should impact the inter-colony spread of plague and thus spatially clustered colonies may be more susceptible to epizootic die-off than more isolated colonies. We found that nearest neighbor distance contributed to model fit at all study areas and that probability of infection increased as colonies were located closer together. Models including measures of proximity for each colony emerged in the top models (Table 4). At the Cimarron and Comanche, as proximity increased, that is, as colonies were more clumped on the landscape, probability of infection increased, whereas at Kiowa-Rita Blanca, the effect was opposite (Table 5).

Finally, we did not find the effects of colony proximity to low-lying dry creek drainages to contribute significantly to model fit of the probability of infection at any study area. Drainages have previously been described as routes of dispersal for prairie dogs (Garrett and Franklin 1988). Using microsatellite analyses, Roach et al. (2001) evaluated relatedness among individuals from neighboring colonies and concluded that prairie dogs likely used dry drainages as dispersal corridors. We thus expected that proximity to drainages may also significantly influence the inter-colony spread of plague if prairie dogs are spreading plague via dispersal, if prairie dogs potentially come into contact with infected reservoirs more readily while traveling along drainages, or if predators carrying infected fleas are utilizing drainages to move among colonies.

Collinge et al. (2005) found a negative correlation between plague occurrence in prairie dog colonies and the coverage of lakes and streams on the surrounding landscape for study sites in Phillips County, Montana, and Boulder County, Colorado. However, Collinge et al. (2005) considered percent cover of lakes and total linear length of streams within 5 km of a colony, whereas we considered distance to the nearest (usually dry) drainage. Lakes and perennial streams in Colorado were probably barriers to prairie dog dispersal because of the presence of water, whereas the drainage systems at our study areas at Cimarron, Comanche, and Kiowa-Rita Blanca were almost exclusively dry drainages, which we expected to facilitate rather than impede the movement of animals and spread of disease. Dry drainages may function as corridors for the epizootic spread of plague if they increase successful dispersal of prairie dogs carrying plague infected fleas. Without drainages, plague may be less likely to spread rapidly to neighboring colonies via epizootic transmission. A lack of an extensive drainage network may explain the slow progression and isolated incidences of plague at Kiowa-Rita Blanca and Cimarron.

Overall, our modeling suggests that large colonies close to neighboring colonies are more likely to become infected with plague; however, we were unable to infer proximate mechanisms of transmission from our modeling. We were able to observe how infection rates changed over time and determine the effects of colony spatial characteristics on infection. This information provides a better understanding of the potential long-term effects of plague at individual grasslands. In addition, the importance of colony spatial characteristics as predictors of a colony's susceptibility to plague has important implications for the conservation and management of black-tailed prairie dogs that occur within the current distribution of sylvatic plague.

MANAGEMENT IMPLICATIONS

Our findings suggest that conventional management strategies employed in prairie dog conservation may need to be modified in the presence of plague. Conservation approaches commonly applied to black-tailed prairie dog populations aim to create larger, more densely populated colonies by managing poisoning and recreational shooting and by relocating prairie dogs to previously inhabited areas (U.S. Department of the Interior Fish and Wildlife Service 2000). Prairie dog populations may naturally form metapopulations, sustained by connectivity among colonies (colonies close together and close to dispersal corridors) and high likelihood of successful dispersal (Roach 1999). The introduction of sylvatic plague as an exotic pathogen may have altered natural population dynamics. Decreasing nearest neighbor distances between prairie dog colonies, and increasing colony size and connectivity within a complex, may increase susceptibility of those colonies to plague. Colonies within the range of sylvatic plague may be most effectively managed by limiting connectivity among groups of colonies. Colony connectivity within a complex remains important to the metapopulation structure and persistence of black-tailed prairie dogs, however, limiting the size and connectivity of colony complexes may allow managers to avoid widespread epizootics or to take action before an epizootic becomes widespread. Additionally, our research has shown that plague epizootics in black-tailed prairie dogs may not progress as quickly from colony to colony as was previously thought, which may provide managers time to intervene and potentially lessen the effects of plague within large colony complexes. In the future, creating or enlarging gaps in colony distribution should be explored as a possible method to reduce the transmission of plague among prairie dog colonies.

ACKNOWLEDGMENTS

We thank D. Augustine and T. Peters (Comanche National Grassland), A. Chappell, G. Mason, and J. Hartman (Cimarron National Grassland), D. Garcia, and N. Walls (Kiowa-Rita Blanca National Grassland), and especially the private landowners across our study areas for their cooperation. We thank K. Gage and J. Monteneri, Centers for Disease Control and Prevention, Division of Vector-borne Infectious Diseases (CDC-NVBID),

Ft. Collins, Colorado. We thank R. Marsh, J. Kraft, and J. Kretzer for field assistance and B. Thiagarajan, J. Graham, and 2 anonymous reviewers of an earlier draft for their valuable comments. Financial support for this research was provided by the National Center for Environmental Research (NCER), Science to Achieve Results (STAR) program of the United States-Environmental Protection Agency (EPA; R82909101-0), the National Science Foundation and National Institutes of Health (NSF/NIH) joint program in Ecology and Infectious Diseases (DEB-0224328), United States Geological Survey, United States Forest Service, Kansas Department of Wildlife and Parks, the Division of Biology at Kansas State University, and the Kansas Cooperative Fish and Wildlife Research Unit. Mention of trademarks or commercial products does not imply endorsement by the U.S. government.

LITERATURE CITED

- Antolin, M. F., P. Gober, B. Luce, D. E. Biggins, W. E. Van Pelt, D. B. Seery, M. Lockhart, and M. Ball. 2002. The influences of sylvatic plague on North American wildlife at the landscape level, with special emphasis on black-footed ferret and prairie dog conservation. *Transactions of the 67th North American Wildlife and Natural Resources Conference* 104:127.
- Antolin, M. F., L. T. Savage, and R. J. Eisen. 2006. Landscape features influence genetic structure of black-tailed prairie dogs (*Cynomys ludovicianus*). *Landscape Ecology* 21:867–875.
- Arnason, A. N. 1972. Parameter estimates from mark-recapture experiments on two populations subject to migration and death. *Researches on Population Ecology* 13:97–113.
- Barnes, A. M. 1982. Surveillance and control of bubonic plague in the United States. *Symposium of the Zoological Society of London* 50:237–270.
- Barnes, A. M. 1993. A review of plague and its relevance to prairie dog populations and the black-footed ferret. Pages 28–38 in J. L. Oldemeyer, et al., editors. *Management of Prairie dog complexes for the reintroduction of the black-footed ferret*. US Fish and Wildlife Service Biological Report 13, Washington, D.C., USA.
- Brownie, C., J. E. Hines, J. D. Nichols, K. H. Pollock, and J. B. Hestbeck. 1993. Capture-recapture studies for multiple strata including non-Markovian transitions. *Biometrics* 49:1173–1187.
- Burnham, K. P., and D. R. Anderson. 1998. *Model selection and inference: a practical information-theoretic approach*. p. 488. Springer Verlag, New York, New York, USA.
- Choquet, R., A. M. Rebout, R. Pradel, O. Gimenez, and J. D. Lebreton. 2003. User's manual for U-CARE. Mimeographed document, CEFÉ/CNRS, Montpellier, France. <ftp://ftp.cefe.cnrs-mop.fr/biom/Soft-CR>. Accessed Dec 2004.
- Collinge, S. K., W. C. Johnson, C. Ray, R. Matchett, J. Grensten, J. F. Cully, Jr., K. L. Gage, M. Y. Kosoy, J. E. Loye, and A. P. Martin. 2005. Landscape structure and plague occurrence in black-tailed prairie dogs. *Landscape Ecology* 20:941–955.
- Cully, J. F. 1993. Plague, prairie dogs and black-footed ferrets. Pages 38–48 in J. L. Oldemeyer, D. E. Biggins, B. J. Miller, and R. Crete, editors. *Management of prairie dog complexes for the reintroduction of the black-footed ferret*. US Fish and Wildlife Service Biological Report 1, Washington, D.C., USA.
- Cully, J. F. Jr., and E. S. Williams. 2001. Interspecific comparisons of sylvatic plague in prairie dogs. *Journal of Mammalogy* 82:894–905.
- Cully, J. F. Jr., A. M. Barnes, T. J. Quan, and G. Maupin. 1997. Dynamics of plague in a Gunnison's prairie dog colony complex from New Mexico. *Journal of Wildlife Diseases* 33:706–719.
- Cully, J. F. Jr., L. G. Carter, and K. L. Gage. 2000. New records of sylvatic plague in Kansas. *Journal of Wildlife Diseases* 36:389–392.
- Faustino, C. R., C. S. Jennelle, V. Connolly, A. K. Davis, E. C. Swarthout, A. A. Dhondt, and E. G. Cooch. 2004. *Mycoplasma gallisepticum* infection dynamics in a house finch population: seasonal variation in survival, encounter, and transmission rate. *Journal of Animal Ecology* 73:651–669.
- Gage, K. L., and M. Y. Kosoy. 2005. Natural history of plague: perspectives from more than a century of research. *Annual Review of Entomology* 50:505–528.
- Gage, K. L., R. S. Ostfeld, and J. G. Olson. 1995. Nonviral vector-borne zoonoses associated with mammals in the United States. *Journal of Mammalogy* 76:695–715.
- Garrett, M. G., and W. L. Franklin. 1988. Behavioral ecology of dispersal in the black-tailed prairie dog. *Journal of Mammalogy* 69:236–250.
- Gustafson, E. J., and G. R. Parker. 1992. Relationships between landcover proportion and indices of landscape spatial pattern. *Landscape Ecology* 7(2): 101–110.
- Hoogland, J. L. 1995. *The black-tailed prairie dog: social life of a burrowing mammal*. The University of Chicago Press, Chicago, Illinois, USA.
- Johnson, T. L. 2005. *Spatial dynamics of a bacterial pathogen: sylvatic plague in black-tailed prairie dogs*. M.S. Thesis. Kansas State University, Manhattan, USA.
- Kendall, W. L. 2004. Coping with unobservable and mis-classified states in capture-recapture studies. *Animal Biodiversity and Conservation* 27:97–107.
- Lachish, S., M. Jones, and H. McCallum. 2007. The impact of disease on the survival and population growth rate of the Tasmanian devil. *Journal of Animal Ecology* 76:926–936.
- Lebreton, J. D., and R. Pradel. 2002. Multistate recapture models: modeling incomplete individual histories. *Journal of Applied Statistics* 29:353–369.
- Lomolino, M. V., and G. A. Smith. 2001. Dynamic biogeography of prairie dog (*Cynomys ludovicianus*) towns near the edge of their range. *Journal of Mammalogy* 82:937–945.
- Lomolino, M. V., G. A. Smith, and V. Vidal. 2004. Long-term persistence of prairie dog towns: insights for designing networks of prairie reserves. *Biological Conservation* 115:111–120.
- Pauli, J. N., and S. W. Buskirk. 2007. Recreational shooting of prairie dogs: a portal of lead entering wildlife food chains. *Journal of Wildlife Management* 71(1): 103–108.
- Poland, L. D., and A. M. Barnes. 1979. Plague. Pages 515–556 in J. F. Steele, editor. *CRC handbook series in zoonoses, Section A: Bacterial, rickettsial, and mycotic diseases. Volume 1*. CRC Press, Boca Raton, Florida, USA.
- Pradel, R., C. M. A. Wintrebert, and O. Gimenez. 2003. A proposal for a goodness-of-fit test to the Arnason-Schwarz multisite capture-recapture model. *Biometrics* 59:43–53.
- Pradel, R., O. Gimenez, and J.-D. Lebreton. 2005. Principles and interest of GOF tests for multi-state capture-recapture models. *Animal Biodiversity and Conservation* 28:189–204.
- Roach, J. L. 1999. *Genetic analysis of a black-tailed prairie dog (Cynomys ludovicianus) metapopulation within shortgrass steppe*. M.Sc. Thesis. Colorado State University, Fort Collins, USA.
- Roach, J. L., P. Stapp, B. Van Horne, and M. F. Antolin. 2001. Genetic structure of a metapopulation of black-tailed prairie dogs. *Journal of Mammalogy* 82:946–959.
- Salkeld, D. J., and P. Stapp. 2006. Seroprevalence rates and transmission of plague (*Yersinia pestis*) in mammalian carnivores. *Vector-Borne and Zoonotic Diseases* 6:231–239.
- Sandercock, B. K. 2006. Estimation of demographic parameters from live encounter data: a summary review. *Journal of Wildlife Management* 70:1504–1520.
- Schwarz, C. J., J. F. Schweigert, and A. N. Arnason. 1993. Estimating migration rates using tag-recovery data. *Biometrics* 49:177–193.
- Senar, J. C., and M. J. Conroy. 2004. Multi-state analysis of the impacts of avian pox on a population of Serins (*Serinus serinus*): the importance of estimating recapture rates. *Animal Biodiversity and Conservation* 27:133–146.
- Snäll, T. R. B. O'Hara, C. Ray, and S. K. Collinge. 2008. Climate-driven spatial dynamics of plague among prairie dog colonies. *The American Naturalist* 171(2): 238–248.
- Stapp, P., M. F. Antolin, and M. Ball. 2004. Patterns of extinction in prairie dog metapopulations: plague outbreaks follow El Niño events. *Frontiers in Ecology and the Environment* 2:235–240.

- Thomas, R. E. 1988. A review of flea collection records from *Onychomys leucogaster* with observations on the role of grasshopper mice in the epizootology of wild rodent plague. *Great Basin Naturalist* 48:83–95.
- Toombs, T. P. 1997. Burrowing owl nest-site selection in relation to soil texture and prairie dog colony attributes. Thesis. Colorado State University, Fort Collins, USA.
- U.S. Department of the Interior Fish and Wildlife Service, 2000. Endangered and threatened wildlife and plants: twelve-month finding for a petition to list the black-tailed prairie dog as threatened. *Federal Register* 65:5476–5488. U.S. Department of the Interior Fish and Wildlife Service, Washington, D.C., USA.
- U.S. Forest Service, Cibola National Forest, 1999. Existing conditions report: southern prairie geographic area, Kiowa and Rita Blanca National Grasslands. U.S. Forest Service, Cibola National Forest Service, Albuquerque, New Mexico, USA.
- Webb, C. T., C. P. Brooks, K. L. Gage, and M. F. Antolin. 2006. Classic flea-borne transmission does not drive plague epizootics in prairie dogs. *Proceedings of the National Academy of Sciences of the United States of America* 103(16): 6236–6241.
- White, G. C., and K. P. Burnham, 1999. Program MARK: survival estimation from populations of marked animals. *Bird Study* 46:120–138.

Associate Editor: Jeff Bowman.