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Source: Journal of Wildlife Diseases, 42(1) : 74-80

Published By: Wildlife Disease Association

URL: https://doi.org/10.7589/0090-3558-42.1.74

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Journal of Wildlife Diseases, 42(1), 2006, pp. 74–80
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A PLAGUE EPIZOOTIC IN THE BLACK-TAILED PRAIRIE DOG (*CYNOMYS LUDOVICIANUS*)

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ABSTRACT: Plague is the primary cause for the rangewide decline in prairie dog (*Cynomys* spp.) distribution and abundance, yet our knowledge of plague dynamics in prairie dog populations is limited. Our understanding of the effects of plague on the most widespread species, the black-tailed prairie dog (*C. ludovicianus*), is particularly weak. During a study on the population biology of black-tailed prairie dogs in Wyoming, USA, plague was detected in a colony under intensive monitoring, providing a unique opportunity to quantify various consequences of plague. The epizootic reduced juvenile abundance by 96% and adult abundance by 95%. Of the survivors, eight of nine adults and one of eight juveniles developed antibodies to *Yersinia pestis*. Demographic groups appeared equally susceptible to infection, and age structure was unaffected. Survivors occupied three small coteries and exhibited improved body condition, but increased flea infestation compared to a neighboring, uninfected colony. Black-tailed prairie dogs are capable of surviving a plague epizootic and reorganizing into apparently functional coteries. Surviving prairie dogs may be critical in the repopulation of plague-decimated colonies and, ultimately, the evolution of plague resistance.

Key words: Black-tailed prairie dog, *Cynomys ludovicianus*, plague, seroconversion, Wyoming, *Yersinia pestis*.

INTRODUCTION

Plague has been implicated as the primary mechanism behind the rangewide decline in abundance and distribution of prairie dogs (*Cynomys* spp.; Antolin et al., 2002). A flea-transmitted bacterial disease caused by *Yersinia pestis*, plague is exotic to the New World, having arrived at the end of the 19th century (Biggins and Kosoy, 2001). *Yersinia pestis* infects various mammalian taxa but is most prevalent among rodents. In part because of their dense aggregations, colonial rodent species (Evans et al., 1943), particularly prairie dogs, are highly susceptible, exhibiting 85%–100% mortality among colonies (Cully and Williams, 2001).

Prairie dogs are keystone species in North American grassland ecosystems (Miller et al., 1994), supporting predators, including the obligate prairie dog predator, the endangered black-footed ferret (*Mustela nigripes*; Anderson et al., 1986). Prairie dog burrows also provide structural habitat for burrowing owls (*Athene cunicularia*), prairie rattlesnakes (*Crotalus viridis*), and various small mammals (Miller et al., 1994). Through herbivory, prairie dogs alter vegetation and cycle nutrients (Holland and Detling, 1990). Therefore, the population biology of prairie dogs related to plague epizootics has broad implications for North American grassland communities.

Of all North American taxa, population-level effects of plague in prairie dogs is best understood. Yet our knowledge of plague dynamics in prairie dogs is poor. Cully et al. (1997) showed that plague caused 99%–100% mortality among four Gunnison's prairie dog (*Cynomys gunnisoni*) colonies. About half of the survivors exhibited antibodies to *Y. pestis*, and the lack of antibodies in the other survivors suggested that plague avoidance was an important mechanism for survival. However, it was unclear whether animals without *Y. pestis* antibodies avoided infection while in the colony or were recent immigrants. Anderson and Williams (1997) found that plague reduced white-

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tailed prairie dog (*C. leucurus*) abundance on a colony complex by 79%–96%; antibody development was not determined for survivors. Neither study identified demographic effects of the epizootic or reported the spatial distribution of surviving animals.

Colony growth following an epizootic is highly variable. Anderson and Williams (1997) noted that white-tailed prairie dogs were capable of repopulating colonies within 1–2 yr. Cully et al. (1997) found that small groups of surviving Gunnison's prairie dogs experienced variable success in colony repopulation. Several years after the epizootic, however, Cully (1997) determined that recovering Gunnison's prairie dog colonies exhibited growth rates 3.5× higher than noninfected colonies.

Our limited knowledge of plague ecology has hindered our ability to parameterize population-based models for prairie dogs. Our understanding of plague effects on the most widespread species, the black-tailed prairie dog (*C. ludovicianus*), is particularly weak. It has been assumed that plague dynamics for black-tailed prairie dogs are similar to those of Gunnison's prairie dogs (Cully and Williams, 2001). However, this assumption is based on anecdote and the belief that, because the black-tailed and Gunnison's prairie dog occur at similar densities, their susceptibility to plague should be analogous. No study has quantified the population- and individual-level effects of plague on black-tailed prairie dogs. Therefore, we lack reliable estimates of plague mortality rates for the black-tailed prairie dog and have not identified whether black-tailed prairie dogs survive exposure to *Y. pestis* or if these animals avoid infection by occupying uninfected regions of a colony. It also is unknown whether colony structure remains intact following an epizootic and to what extent surviving prairie dogs contribute to the repopulation of plague-decimated colonies.

We report the effects of a plague epizootic on a colony of black-tailed prairie dogs for which a matched, uninfected neighboring colony provided a control. We quantify changes in population size, density, demographic structure, spatial arrangement, body condition, and flea infestation from plague and compare changes to those on the uninfected colony. We compare colony attributes to those previously reported for the Gunnison's prairie dog. Finally, we propose ways in which surviving prairie dogs are important to colony recovery and the evolution of plague resistance.

MATERIALS AND METHODS

Study area

This study was conducted during the summers of 2003–4 while investigating the population biology of black-tailed prairie dogs around Thunder Basin National Grassland (TBNG), Wyoming, USA. This area encompasses >230,000 ha of federal land within a mixture of public and private land in northeastern Wyoming. The region is characterized by mixed grass prairies and sagebrush steppe habitats. Dominant plant species include blue grama (*Bouteloua gracilis*), western wheatgrass (*Agropyron cristatum*), needle-and-thread grass (*Stipa comata*), big sagebrush (*Artemisia tridentata*), and prickly pear cactus (*Opuntia polyacantha*). Plague epizootics in TBNG are relatively common; in 2001 a massive epizootic reduced prairie dog distribution by about 68% (U.S. Forest Service, unpublished data).

Field methods

Two colonies, Antelope Coal Mine (ACM; 105°22′N, 43°30′W) and Jacobs Ranch Mine (JRM; 105°13′N, 43°42′W), were studied. In 2003 ACM and JRM were matched based on habitat characteristics, colony area, prairie dog density, grazing regime, and disturbance history (recent shooting, poisoning, and plague) from a pool of 10 study colonies (Pauli, 2005). In June 2003 we studied the population biology of prairie dogs on both colonies using mark-recapture techniques. In early summer 2004, plague was detected at ACM, allowing us to quantify changes in the colony resulting from an epizootic.

We mapped both colonies after emergence of juveniles in May 2003 and 2004. Colony perimeters were delineated and flagged, and mapped with a Global Positioning System

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(GPS; Trimble Navigation Limited, Sunnyvale, California, USA) following the methodology developed by Plumb et al. (2001). After the epizootic on ACM, we mapped the remaining active area relying principally on outermost active burrows (those with prairie dogs, fresh diggings or scat, or lacking cobwebs) using a GPS unit.

In June 2003 trapping grids were randomly placed on both colonies. Live traps (no. 203, Tomahawk Live Traps, Tomahawk, Wisconsin, USA) were placed 16 m apart in a 9×9 grid. Trapping grids were reestablished in the same locations in June 2004. Trapping was simultaneously conducted on ACM and JRM for six consecutive days. During each trapping session, traps were set, wired open, and prebaited for 24 hr. Because prairie dogs are strictly diurnal, traps were baited and set at sunrise and checked and closed at sunset.

We did not capture any prairie dogs on the trapping grid at ACM after the epizootic. Therefore, prairie dogs were captured opportunistically throughout the colony during June–July 2004. We surveyed ACM by foot and returned to areas exhibiting prairie dog activity for additional observations. With a spotting scope, we monitored presumed active areas for seven consecutive days; if prairie dogs were not detected, we moved to other potential active areas. When prairie dogs were located above ground, we chased them into burrows and surrounded the occupied burrows with traps. Trapping and observations continued until all remaining prairie dogs were captured in mid-July.

We marked captured prairie dogs with fingerling ear tags (National Band and Tag, Newport, Kentucky, USA) and injected adult males with subcutaneous passive integrated transponders (Biomark, Boise, Idaho, USA). Prairie dogs captured on ACM after the epizootic were also marked with nontoxic paint sticks for identification during observations. We recorded sex, weight, and hind foot length from each captured prairie dog. Body condition indices were estimated by dividing each prairie dog's weight (g) by its hind foot length (cm; Wirsing et al., 2002). Using size, pelage, and premolar gap width (Cox and Franklin, 1990), we estimated the age of captured prairie dogs (juvenile, adult). To document flea infestation, we inspected and recorded the number of fleas on each prairie dog with a 5-cm comb. At first capture, we combed the entire pelage of each animal, counting the number of fleas observed in the fur or that were brushed off during combing. Flea intensities were estimated with mean flea indices, the number of fleas on prairie dogs that had ≥1 flea, whereas flea prevalence was calculated as the proportion of prairie dogs with fleas. Blood was collected from surviving prairie dogs on ACM via cardiac puncture to test for antibodies to *Y. pestis*. Animals were anesthetized using halothane, and 1.5 ml of blood was collected and stored at 5 C. After animals had recovered from anesthesia, they were released at the site of capture.

Analyses

Spatial data were transferred to a computer using Pathfinder Office version 2.90 (Trimble Navigation Limited, Sunnyvale, California, USA), and data were differentially corrected with Community Base Station Files (Casper, Wyoming, USA). We estimated the remaining area for ACM after plague with minimum convex polygons in ArcGIS (ESRI Inc., Redlands, California, USA), and used CAPTURE (Otis et al., 1978; White et al., 1982) to estimate the abundance of juvenile and adult prairie dogs. The most appropriate model for juveniles and adults on ACM in 2003 and adults on JRM in 2004 was Jackknife M_h, which assumes variable probability of capture by animal. The most appropriate model for juveniles and adults on JRM in 2003 was M_b, because capture probabilities varied by behavioral response to capture. Model M_0, which assumes constant capture probabilities, was the most appropriate model for juveniles on JRM in 2004.

Densities were estimated by dividing abundance estimates by the effective area trapped (Wilson and Anderson, 1985). Effective area trapped was determined by taking one-half the mean maximum distance moved (MMDM; the average of the maximum distance between recaptures for captured animals) and adding it to the edge of the trapping grid area. Because home range size differed between ages, separate MMDM values were calculated for juveniles. Densities for ACM in 2004 were estimated by dividing the number of survivors by the remaining colony area, and population sizes in 2003 and on JRM in 2004 were estimated by multiplying density estimates by the area of the colony. Variances for abundances and densities were estimated following Otis et al. (1978).

Age ratios and the proportion of flea-infested prairie dogs between colonies and years were compared with log-likelihood ratios, corrected for continuity. Mean flea indices and body conditions were compared between ACM and JRM in 2003 and 2004 with

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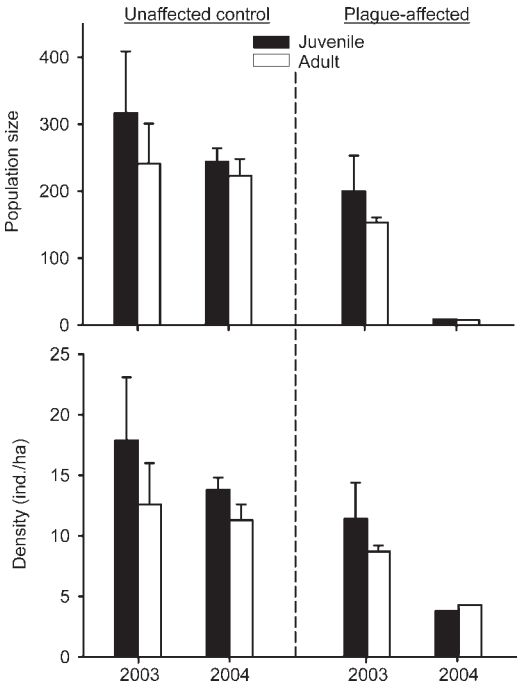


FIGURE 1. Estimates (±1 SE) of population size and density for juvenile and adult black-tailed prairie dogs on a plague-affected colony and an unaffected control colony, Thunder Basin National Grassland, Wyoming, 2003–4. Plague reduced juvenile abundance by 96% and adult abundance by 95% in 2004. Because survivors occupied small coteries, density decreases from plague were moderated; juvenile density decreased by 67%, and adult density decreased by 51%.

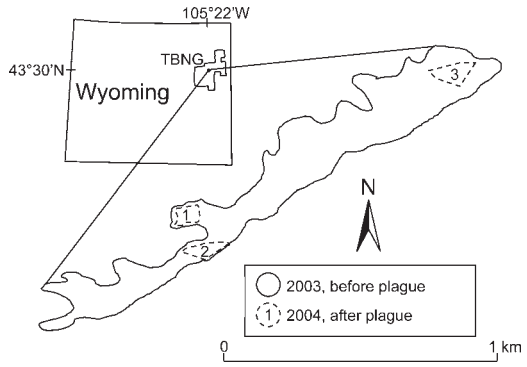


FIGURE 2. Area occupied by black-tailed prairie dogs on Antelope Coal Mine in 2003 (20.5 ha) and 2004 (2.2 ha), Thunder Basin National Grassland, Wyoming. After plague in 2004, surviving prairie dogs occupied three coteries, representing an 89% decrease in colony area.

univariate ANOVAs. Blood samples from ACM were analyzed for plague antibodies with a competitive blocking enzyme-linked immunosorbent assay (cELISA), following standard CDC methods (Chu, 2000). Prevalence of antibodies was compared between adults and juveniles and between males and females using the Fisher exact test (Zar, 1999). All statistical analyses were conducted using SPSS 11.5.

RESULTS

Population size and structure

Antelope Coal Mine and Jacobs Ranch Mine had similar abundances, densities, and colony areas in 2003. Neither colony had experienced poisoning, recreational shooting, grazing, or plague within the previous 10 yr. The plague epizootic was detected on ACM in June 2004 and rapidly spread through the colony; the outbreak had subsided by the first week of July 2004.

Prairie dogs were abundant on both ACM and JRM in 2003 (Fig. 1). In 2004, after the epizootic, juvenile and adult abundances on ACM had plummeted; only eight juveniles (96% decline) and nine adults (95% decline) remained; colony area on ACM decreased by 89% (Fig. 2). Survivors inhabited three small coteries (coterie 1 = 0.5 ha; coterie 2 = 0.8 ha; coterie 3 = 0.9 ha), and, therefore, ACM exhibited a less drastic decrease in prairie dog density; juvenile density decreased by 67%, and adult density decreased by 51% (Figs. 1, 2). On JRM, prairie dog abundance and density decreased slightly from 2003 to 2004. For juveniles, abundance and density decreased by 23%, whereas for adults, abundance decreased by 7%, and density decreased by 10% (Fig. 1). From 2003 to 2004, colony area for JRM increased from 17.7 to 19.7 ha (11.3% increase). Age ratios (1.2:1, juveniles:adults) did not differ between colonies in 2003 ($G_1<0.01$, $P=0.96$) and were constant across years for both ACM ($G_1=0.44$, $P=0.60$) and JRM ($G_1=2.13$, $P=0.14$).

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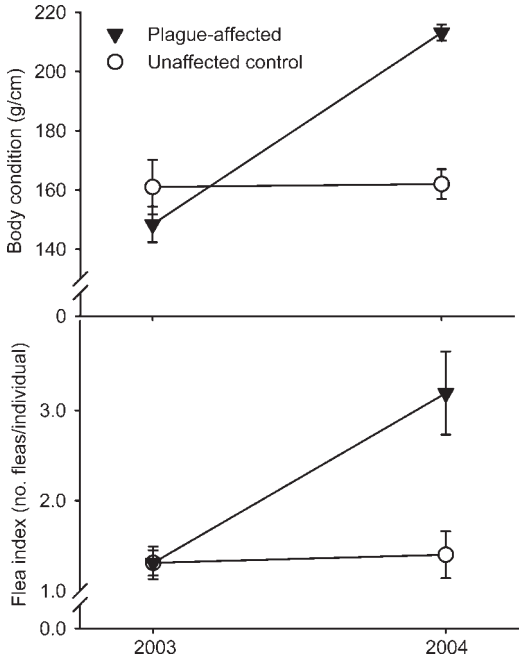


FIGURE 3. Attributes of black-tailed prairie dogs before (2003) and after (2004) a plague epizootic. An unaffected, matched colony is shown for control. (a) Mean body condition (±1 SE) for adults. After plague, adults in the affected colony exhibited a 45% increase in body condition. (b) Mean flea indices (±1 SE). After plague, flea infestation increased on the affected, but not the unaffected, colony.

Characteristics of surviving individuals

Of the 17 remaining prairie dogs on ACM, nine had antibodies to *Y. pestis*. Seroconversion was more common among adults (8/9) than juveniles (1/8; Fisher exact test, $P<0.001$), but did not differ between males (4/8) and females (5/9; Fisher exact test, $P=0.60$). Each remaining coterie contained >1 prairie dog that had developed antibodies to *Y. pestis*.

Body condition was analyzed separately for juveniles and adults because of an interaction among age, colony, and year ($F_{2,135}=7.72$, $P=0.006$). Between 2003 and 2004, adult body conditions diverged between JRM and ACM ($F_{1,78}=24.3$, $P<0.001$; Fig. 3a). On JRM, adult body condition was constant from 2003 to 2004, but on ACM, adult body condition increased by 45% over the same time period ($t_{31}=-6.40$, $P<0.001$). In contrast, juvenile body condition (87±8.2 g/cm, mean ±1 SE) did not show an interaction between colony and year ($F_{1,57}=1.03$, $P=0.31$) and did not differ between colonies ($F_{1,57}=0.74$, $P=0.39$) or years ($F_{1,57}=0.88$, $P=0.35$).

In 2003, flea infestation did not differ between colonies ($G_1=0.15$, $P=0.70$); 39% ($n=90$) of prairie dogs that harbored fleas. Between 2003 and 2004, flea infestation did not change on JRM (39%, $n=41$; $G_1=0.06$, $P=0.81$), but increased to 71% ($n=17$) on ACM ($G_1=5.62$, $P=0.02$). Flea intensity also exhibited an interaction between colony and year ($F_{1,53}=11.9$, $P<0.001$; Fig. 3b). In 2003, the mean flea index for prairie dogs was 1.3 on both ACM (±0.18 no. fleas/individual) and JRM (±0.14 no. fleas/individual). In 2004, the mean flea index did not change on JRM ($t_{28}=0.52$, $P=0.61$) but increased by a factor of 3 on ACM ($t_{25}=4.31$, $P<0.001$).

DISCUSSION

Within <2 mo, plague reduced prairie dog abundance on ACM by 95%. About half of the remaining prairie dogs showed antibodies to *Y. pestis*, indicating that acquired humoral immunity was the primary mechanism for black-tailed prairie dog survival. Although age ratios did not change after plague, antibody development was higher among adults; all but one adult showed antibodies to *Y. pestis*, whereas only one juvenile developed antibodies. Because juveniles do not immigrate (Garrett and Franklin, 1988), most juveniles must have avoided plague by occupying uninfected regions of the colony. Cully et al. (1997) found similar effects of plague in the Gunnison's prairie dog; colonies were extirpated or reduced by 99%, and roughly half of the survivors developed antibodies to *Y. pestis*. They did not, however, determine how plague affected juvenile survival.

Following epizootics in the Gunnison's prairie dog, individual survivors were

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scattered throughout the colony (Rayor, 1985a; Cully et al., 1997). In contrast, we found that surviving black-tailed prairie dogs inhabited distinct coteries. It is unclear whether the coteries persisted through the epizootic or if scattered survivors coalesced into aggregations immediately after the epizootic. Nevertheless, coterie structure was relatively similar to that in a colony that has not experienced an epizootic (Hoogland, 1995); each coterie contained reproductive-age males and females, and juveniles inhabited two of the coteries. Because survivors occurred in small coteries, density declines were less severe than were declines in abundance at the colony level.

Prairie dog colonies in regions with plague exhibit episodic cycles of rapid population growth and plague-caused die-offs (Antolin et al., 2002). It has been suggested that colony growth after an epizootic is attributable to recolonization by intercolonial dispersers (Antolin et al., 2002). Although previously ignored, survivors may be crucial in repopulating plague-decimated colonies. Our results indicate that survivors were not only organized into functional coteries, but also exhibited improved body condition compared to before the epizootic and to the uninfected colony. Two mechanisms could explain the improved body condition of survivors. First, large healthy prairie dogs may have been more resistant to infection, resulting in a disproportionately high mortality rate among smaller-bodied individuals. Second, following the population crash, intraspecific competition for forage should have decreased, providing the few survivors better access to food, resulting in improved body condition. Whatever the underlying mechanism, colonies supporting large healthy individuals should grow more quickly because large prairie dogs reach sexual maturity at a younger age, produce larger litters, and exhibit better over-winter survival (Garrett et al., 1982; Rayor, 1985b). The role of healthy survivors should not be discounted in colony recovery.

Generally in mammal populations, increased host abundance increases ectoparasite infestation in a curvilinear fashion (Krasnov et al., 2002). In contrast, we found that after the population crash both the proportion of prairie dogs with fleas and the number of fleas per prairie dog increased for the survivors. Anderson and Williams (1997) observed a similar trend of increasing flea infestation among white-tailed prairie dogs after an epizootic. Thus, it seems that for colonial mammals, a dramatic reduction in abundance increases ectoparasitism. It appears that as the host population declines, displaced parasites swarm the few survivors, resulting in a temporary increase in infestation. Increasing flea infestations may, consequently, accelerate the transmission rate of plague and, in part, account for the rapid spread of plague through populations of colonial mammals.

This is the first study to quantify changes in both population and individual attributes of black-tailed prairie dogs following a plague epizootic. Our results demonstrate that approximately 5% of black-tailed prairie dogs can survive an epizootic. Over 50% of the surviving prairie dogs developed antibodies to *Y. pestis*, indicating that immune response, not plague avoidance, was the primary mechanism of survival. Survivors exhibited increased body condition and occurred in apparently functional coteries. Therefore, survivors should contribute to the recovery of a colony after an epizootic. If resistance to plague, as demonstrated by antibody development, is a heritable trait among prairie dogs, as it appears to be in other rodent species (Biggins and Kosoy, 2001), surviving individuals may contribute to plague resistance.

ACKNOWLEDGMENTS

We are grateful to Jeffery Schultz, Ruben Mares, Matthew Hamilton, Seth Hansen, Aaron Roe, and Emiliano Donadio for their

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invaluable help with fieldwork. We thank Patrick Bauman (Antelope Coal Company) and Roy Liedtke (Jacobs Ranch Mining Company) for providing unrestricted access to the colonies. This research was funded by the Bureau of Land Management–Wyoming, the Wyoming Game and Fish Department, and the National Wildlife Federation.

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Received for publication 12 April 2004.

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