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INTERSPECIFIC COMPARISONS OF SYLVATIC PLAGUE IN PRAIRIE DOGS

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Of the 3 major factors (habitat loss, poisoning, and disease) that limit abundance of prairie dogs today, sylvatic plague caused by *Yersinia pestis* is the 1 factor that is beyond human control. Plague epizootics frequently kill >99% of prairie dogs in infected colonies. Although epizootics of sylvatic plague occur throughout most of the range of prairie dogs in the United States and are well described, long-term maintenance of plague in enzootic rodent species is not well documented or understood. We review dynamics of plague in white-tailed (*Cynomys leucurus*), Gunnison's (*C. gunnisoni*), and black-tailed (*C. ludovicianus*) prairie dogs, and their rodent and flea associates. We use epidemiologic concepts to support an enzootic hypothesis in which the disease is maintained in a dynamic state, which requires transmission of *Y. pestis* to be slower than recruitment of new susceptible mammal hosts. Major effects of plague are to reduce colony size of black-tailed prairie dogs and increase intercolony distances within colony complexes. In the presence of plague, black-tailed prairie dogs will probably survive in complexes of small colonies that are usually >3 km from their nearest neighbor colonies.

Key words: *Cynomys gunnisoni, C. leucurus, C. ludovicianus,* disease, epizootic, landscape, metapopulation, plague, *Yersinia pestis*

Between 1900 and today, the area covered by colonies of black-tailed prairie dogs (*Cynomys ludovicianus*) in the western United States was reduced from about 4×10^7 ha to <600,000 ha, a reduction of >98% (Biggins and Godbey 1995; K. Graber et al., in litt.; Knowles 1998; Nowak 1999). The primary cause of this reduction has been attributed to government and private pest control, habitat loss through conversion of grasslands to crop agriculture, and sylvatic plague (*Yersinia pestis*). After a reduction in control that began in 1973, with an executive order that banned the use of compound 1080, prairie dog species made moderate recoveries. However, continued poisoning since then and sylvatic plague epizootics have resulted in declines of prairie dogs throughout their range during the past 2 decades (United States Department of Interior, Fish and Wildlife Service 2000).

Plague is not endemic to the New World, but entered the United States at several ports around 1900 and became established in commensal rodents in San Francisco in 1900 (Link 1955). The 1st records of plague in wild rodents in the United States occurred in the Berkeley Hills, California, when plague was identified in California ground squirrels (*Spermophilus beecheyi*) in 1908 (McCoy 1908; Wherry 1908). After that, plague spread quickly in a number of species of wild rodents. *Y. pestis* was cultured from fleas of ground squirrels collected in Yellowstone National Park, Wyo-

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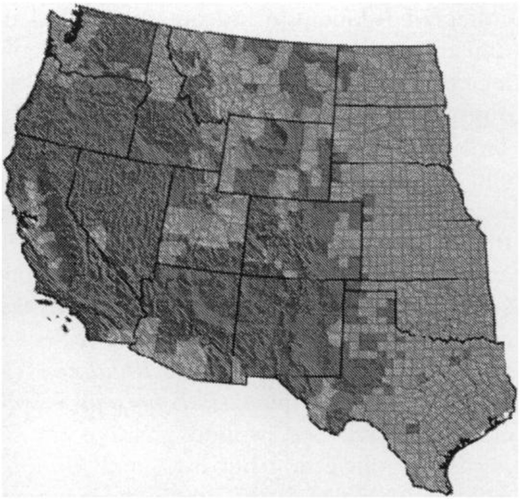

FIG. 1.—Distribution of plague in the United States by county (dark gray) during 1970–2000; unpublished records from Centers for Disease Control, Bacterial Zoonoses Branch. The current distribution was attained by 1950 and has remained relatively stable since that time.

ming, in 1936 (Quan 1982). Plague 1st was identified in Utah prairie dogs (*C. parvidens*) and from fleas on white-tailed prairie dogs (*C. leucurus*) in southwestern Wyoming in 1936 (Eskey and Haas 1940). The 1st records of plague in Gunnison's prairie dogs (*C. gunnisoni*) occurred in New Mexico in 1938 (Eskey and Haas 1940) and in black-tailed prairie dogs in western Kansas in 1945 (Cully et al. 2000) and near Lubbock, Texas, in 1947 (Miles et al. 1952). By around 1950, the current distribution of plague was established near its current limit (Fig. 1). Although minor range extensions have occurred since then, the western boundary of plague has been near its present location since the early 1950s (Barnes 1982, 1993).

The overall impact of plague on prairie dogs during the 1st one-half of the 20th century is not well documented but was probably much greater than is appreciated generally. For example, Ecke and Johnson (1952) reported a die-off of a large population of Gunnison's prairie dogs in South Park, Park County, Colorado, that was later diagnosed as a consequence of a plague epizootic. That die-off resulted in nearly 100% eradication of Gunnison's prairie dogs in 250,000 ha during a 2-year period. At that time, poisoning control was considered effective if 85% of prairie dogs were killed, which resulted in the need for frequent repoisoning. If plague operated on other large colonies of Gunnison's and black-tailed prairie dogs as it did in South Park in 1945–1946, its impact may have been greater than that of poisoning.

Today, plague is an important part of the ecology of the 4 species of prairie dogs in the United States. Some of the important consequences of plague in prairie dogs are local extirpation of colonies, reduced colony size, increased variance in local population sizes, and increased distances between colonies. Black-tailed prairie dog colonies often occur on the grassland landscape in clusters or complexes. The impacts of plague reduce the effectiveness of dispersal in demographic rescue among colonies and increase the probability of extinction of entire complexes.

We review the dynamics of plague in white-tailed, Gunnison's, and black-tailed prairie dogs. We also consider their mammalian associates and fleas. We then identify what differences in plague dynamics among prairie dog species can teach us about the biology of *Y. pestis*. Finally, we use that information to make predictions about the long-term impacts of plague on prairie dogs.

LABORATORY CHALLENGE STUDIES

All 4 species of prairie dogs are highly susceptible to plague infections. The relative susceptibility of rodents to *Y. pestis* typically is determined through laboratory exposure of hosts via subcutaneous inoculation of pure cultures of the bacterium (Holdenried and Quan 1956). Williams (in litt.) challenged white-tailed prairie dogs with titrated doses of *Y. pestis* and found that the mean lethal dose was 46 bacterial cells. In general, laboratory challenges led to signs of illness in 3–4 days, with death following 2–3 days later (E. S. Williams, in litt.). Survival time was related inversely to dose. One animal survived a challenge of 2,300 organisms and developed serum antibodies. On the other hand, 25% of individuals challenged with 2 organisms died.

The number of bacteria inoculated into hosts probably varies by flea species, but may be in the realm of 15,000 organisms for effective vectors (Burroughs 1947), well above the number necessary to cause infections in most rodents. Poland and Barnes (1979) did not cite particular laboratory challenge studies but generalized for species of *Cynomys* that <100 bacteria cause disease with near 100% mortality.

Plague in Fleas

Fleas serve as vectors for *Y. pestis* in a manner somewhat different from other biologically transmitted parasites, in that bacteria grow in the gut of fleas and form a bolus, which obstructs the proventricular valve (stomach valve). When an infected flea takes a blood meal, the blood travels to the stomach, but because the valve is blocked, the blood then is regurgitated with an infective dose of bacteria, which is injected back under the skin of the vertebrate host (Poland and Barnes 1979). This has 2 important effects: it puts a large inoculum into the mammal host, and because the flea is unable to feed successfully, it becomes famished and tries to feed more times than it would if it could successfully retain its meal (Eskey and Haas 1940). This starved condition probably causes host specificity to break down, enhancing multispecific transmission of plague. In laboratory studies, plague infections become established in fleas after about 9–28 days (Eskey and Haas 1940; Poland and Barnes 1979).

More than 20 species of fleas have been collected from prairie dogs or their burrows (Table 1). Five species of fleas (*Opisocrostis labis, Oropsylla hirsutus, O. tuberculatus cynomuris, Neopsylla inopina*, and *Pulex* spp.) that specialize on prairie dog are collected frequently and are implicated in transmission of plague. *Pulex* spp. is collected frequently from black-tailed prairie dogs but is a poor vector (Burroughs 1947). Two ground squirrel fleas, *Thrassis bacchi* and *T. pandori,* are frequently positive for *Y. pestis.* These species most often are found on ground squirrels but frequently are found on prairie dogs and other rodent species (Table 1). Thus, these 2 species may be particularly important as multispecies vectors. *Aetheca wagneri* and *Rhadinopsylla,* fleas of deer mice (*Peromyscus maniculatus*), have been found positive for *Y. pestis* in prairie dog burrows, and *Monopsylla exilis,* a flea of the grasshopper mouse (*Onychomys leucogaster*—Thomas 1988; Thomas et al. 1988) infected with *Y. pestis,* recently was collected from a burrow of a black-tailed prairie dog on Cimarron National Grassland in southwestern Kansas (J. F. Cully, Jr., in litt.). Many of these fleas regularly are positive for *Y. pestis* when collected from prairie dog burrows, implicating their normal mammal hosts in plague epizootics.

Infected prairie dog fleas have been obtained from burrows 3 months (Cully et al. 1997; Fitzgerald 1970) to 1 year (Lechleitner et al. 1968) after disappearance of the last prairie dog. Persistence of fleas infected with *Y. pestis,* in addition to the importance of fleas in both intraspecific and interspecific transmission, contributes to the persistence of plague in the rodent community. Diversity of flea species found in prairie dog burrows provides numerous opportunities for interspecific spread of *Y. pestis* from other species of rodents to prairie dogs, and from prairie dogs to other species of rodents.

Epizootic Patterns of Plague

Plague has been well documented in Gunnison's prairie dogs (Cully et al. 1997; Ecke and Johnson 1952; Fitzgerald 1970; Lechleitner et al. 1968; Rayor 1985). In the Moreno Valley of north-central New Mexico, plague in Gunnison's prairie dogs was

TABLE 1.—Fleas associated with 3 species of prairie dogs and their burrows or with deer mice. P indicates that the flea species collected was infected with *Yersinia pestis*; N indicates that plague has not been documented for the species.

	Mammal species			
Flea species	Gunnison's prairie dog	White-tailed prairie dog	Black-tailed prairie dog	Deer mouse
Aetheca wagneri	N[a–c]	N[d,e]		P[a,d]
Catallagia decipiens	N[a]	N[d]		P[a,d]
Cediopsylla inaequalis		N[d]		
Diamanes montanus	N[f]			
Histrichopsylla dippiei		N[e]		
Hoplopsylla anomalus	N[f]			
Monopsylla eumolpi	P[c]			
Monopsylla exilis			P[g]	
Neopsylla inopina		P[d,e]		N[d]
Opisocrostis labis	P[a–c,f]	P[d–f]		P[a]
Oropsylla hirsutus	P[a–c,f]	P[f]	P[f,g]	N[a]
Oropsylla idahoensis	P[b,c,f]	P[d–f]		P[d]
Oropsylla tuberculatus cynomuris	P[a–c,f]	P[d–f]	P[f]	P[d]
Pulex spp.	N[f]	N[d,f]	P[g]	N[f]
Rhadinopsylla sectilis	P[a]	N[e]		
Rhadinopsylla fraterna	N[c]	P[d,e]		N[d]
Thrassis bacchi	P[a]			N[a]
Thrassis fotus			P[g]	
Thrassis pandori		P[d–f]		

[a] Cully et al. 1997.
[b] Lechleitner et al. 1968.
[c] Fitzgerald 1970.
[d] Anderson and Williams 1997.
[e] Ubico et al. 1988.
[f] Eskey and Haas 1940.
[g] Cully et al. 2000.

1st documented in 1949 (Cully et al. 1997). The next record there involved a human case in the town of Eagle Nest, New Mexico, in 1983, which was attributed to *T. bacchi* or *Rhadinopsylla sectila,* fleas from either 13-lined ground squirrels (*Spermophilus tridecemlineatus*) or deer mice at a rock quarry north of the town. In September 1984, prairie dogs were abundant throughout the grassland of the valley. West of Moreno Creek and south of Eagle Nest (Fig. 2), prairie dogs were abundant at that time. Mark–recapture trapping indicated that the population density in the area was about 30 prairie dogs/ha. During winter 1984–1985, most of the prairie dogs in the northern one-third of the valley, north of Six-mile Creek disappeared. By late June 1985, only isolated prairie dogs could be found.

At that time, no indications of plague existed in marked prairie dogs at the study colony, but 13-lined ground squirrels, which had been abundant in the previous autumn, were rare and disappeared by early summer. Fleas (*T. bacchi*) of 13-lined ground squirrels infected with *Y. pestis* were collected subsequently from nearby prairie dog burrows. In August 1985, plague was documented at the study site in fleas from prairie dog burrows, and the marked population was in decline. Only 25% of those present in June 1985 were estimated to have survived to enter hibernation in October. Seven emerged in spring 1986, and no prairie dogs could be found by July 1995. The pattern repeated itself in the southern one-third of the valley between summer 1996 and 1997, except that ground

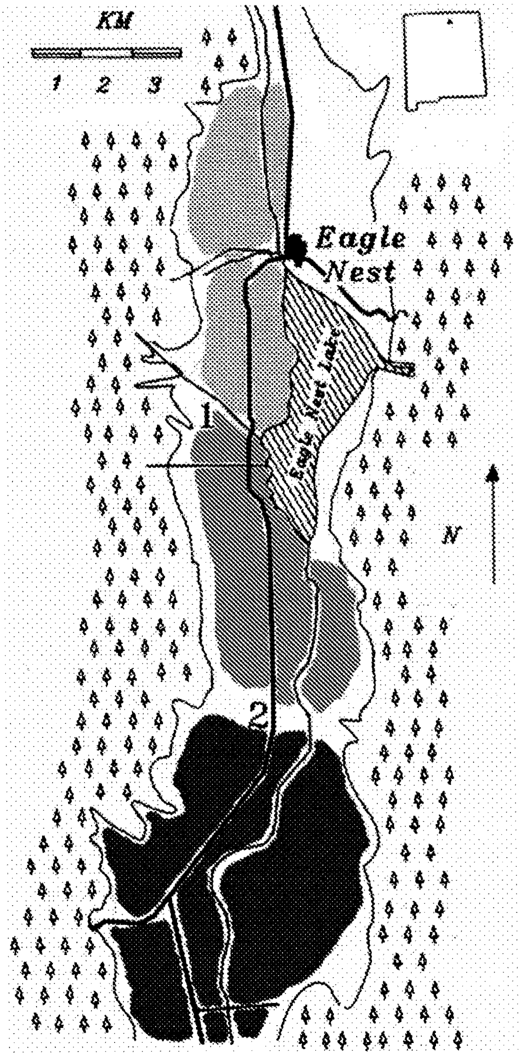

FIG. 2.—Map of the Moreno Valley of New Mexico (modified from Cully et al. 1997) showing the locations of plague epizootics during 1985 (light gray), 1986 (medium gray), and 1987 (dark gray). The 3 regions probably were separated by geographic barriers with 1) deep vegetation at Six-mile Creek and 2) shallow rocky soil at Jackson Hill.

squirrels were not affected. After epizootics, survival of prairie dogs was <1%. In survivors, about 50% had antibody titers, indicating that they had been exposed to plague but had survived. In August 1997, prairie dogs occurred in the Moreno Valley, but colonies were small and scattered, nothing like what had existed there before 1984.

In white-tailed prairie dogs, epizootic patterns are different. Clark (1977) reported a plague epizootic in a small white-tailed prairie dog colony in Wyoming that killed about 85% of the prairie dogs. however, the bacterium quickly disappeared. Years later, plague was diagnosed in a single marked juvenile prairie dog in the same colony (E. S. Williams, in litt.). During that summer no other marked prairie dogs died of plague and no decline was apparent in the population. Conditions likely were not adequate to initiate a plague epizootic in the colony, even though *Y. pestis* was present and prairie dogs were numerous.

Menkens and Anderson (1991) and Anderson and Williams (1997) documented a plague epizootic in white-tailed prairie dogs near Meeteetse, Wyoming, that has continued from 1985 to the present. It was characterized by a slow but continuous decline in the prairie dog population. Plague has been present since 1987 at Shirley Basin, Wyoming. Plague was monitored there in association with black-footed ferret (*Mustela nigripes*). As at Meeteetse, prairie dog populations at Shirley Basin have steadily declined, with local variation in population size (R. Luce and R. Oakleaf, pers. comm.; Menkens and Anderson 1991; Williams et al. 1992, 1997).

The interaction of *Y. pestis* and individual white-tailed prairie dogs is similar to that with Gunnison's prairie dogs. Important vector fleas are also similar (*O. labis* and *O. t. cynomuris*), except that *O. hirsutus* is seldom associated with plague in white-tailed prairie dogs. The population response of white-tailed prairie dogs to plague is considerably less severe than that of Gunnison's prairie dogs. Differences in densities and social interactions probably influence impacts of plague on these rodent species (Gasper and Watson 2001). Colonies of Gunnison's prairie dog that are exposed to plague are very nearly extirpated. Nonetheless, Menkens and Anderson

(1991) reported that variation in populations of uninfected colonies was nearly as great as in infected colonies. Subsequently, Anderson and Williams (1997) revised that opinion; they found that infected colonies declined more precipitously than did uninfected colonies in 1989–1990. However, those declines were less severe than those in Gunnison's prairie dogs, and affected colonies generally rebounded in 1–2 years. Plague has been present continuously in the Meeteetse complex since 1985 and at Shirley Basin since at least 1987. As with Gunnison's prairie dogs, *Y. pestis* has been found in *A. wagneri, T. pandori, O. labis,* and *O. t. cynomuris* collected from burrows of white-tailed prairie dogs (Ubico et al. 1988).

Reports of plague in black-tailed prairie dogs are not as frequent in the literature as they are for Gunnison's prairie dogs, probably because most research on black-tailed prairie dog was done in South Dakota, outside the current range of plague. The 1st published report of plague in black-tailed prairie dogs (Ecke and Johnson 1952) was for Logan and Weld counties, north of Denver, Colorado, but the associated die-off was not confirmed as induced by plague. The 1st confirmed records of plague in black-tailed prairie dogs were from western Kansas in 1945 (Cully et al. 2000) and from 1946–1947 near Lubbock, Texas (Miles et al. 1952). The current distribution of plague (Fig. 1) was established, with minor variations, by the 1950s. Why plague has not spread east beyond its current distribution is not known. Until the mechanistic basis of the limits are understood better, it is unwise to assume plague will not reach previously unaffected colonies east of the current distribution.

When individual black-tailed prairie dogs are infected with plague, the infection follows a pattern similar to that described above for white-tailed and Gunnison's prairie dogs, with nearly 100% mortality. This high individual susceptibility leads to epizootic die-offs similar to those of Gunnison's prairie dogs; colony populations are extirpated or reduced to <1% of preplague levels. The pattern among colonies has been documented for black-tailed prairie dogs at the Rocky Mountain Arsenal National Wildlife Refuge (United States Department of Interior, Fish and Wildlife Service, in litt.). A plague epizootic began there in 1994. By September 1995, the epizootic ran its course, and the prairie dog population was recovering through May 1999 (Figs. 3 and 4). The pattern of rapid die-off for multiple colonies was similar to the pattern observed on Comanche National Grassland, Colorado, in 1995–1996, where all the large prairie dog towns in the Carizo Unit of the grassland collapsed (J. F. Cully, Jr., in litt.). Regrowth of colonies at Rocky Mountain Arsenal was faster than at the Comanche, in part because of transplantation of prairie dogs to aid recovery at the Rocky Mountain Arsenal (D. Seery, pers. comm.).

Epidemiology

Rate of spread of a disease from individual to individual is the transmission rate. If transmission is fast, the disease spreads more quickly through a population than if transmission is slow. All other things being equal, transmission rate will vary with the degree of sociality in a host. Social species have more frequent intraspecific contact than do less social species. As with sociality, high-density populations are expected to yield higher numbers of contacts, with enhanced rates of transmission. Transmission rates also may vary depending on ability of different species of flea to transmit plague. Other factors that are important to transmission rate include numbers of susceptible individuals, infective individuals, and recovered or immune individuals. In the case of plague in prairie dogs, number of recovered individuals is effectively zero. The population can recruit new susceptible individuals via reproduction or migration. If transmission is fast relative to recruitment and infected animals all die, the population will decline. If the recruitment rate is equal

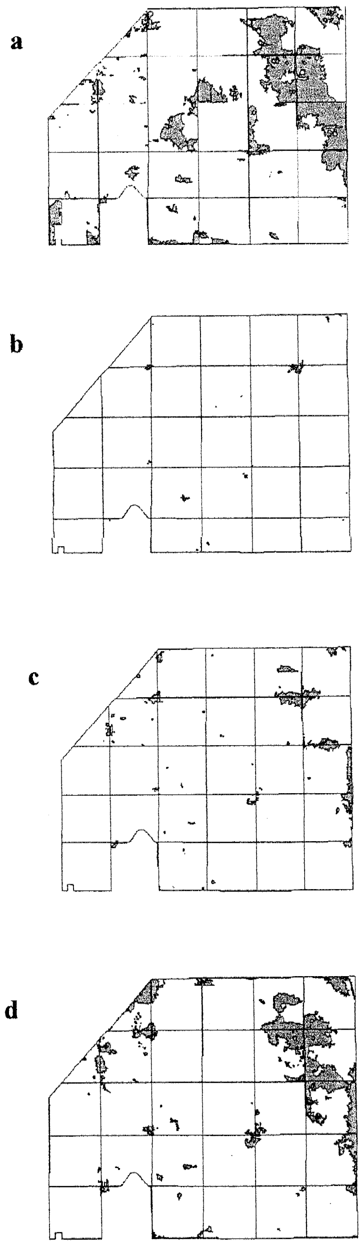

FIG. 3.—Map of black-tailed prairie dog colonies (lines are section lines) at Rocky Mountain Arsenal National Wildlife Refuge near Denver, Colorado (shaded in gray), showing the locations and extent of colonies a) before a plague epizootic in May 1994 (983 ha), b) after a plague epizootic in September 1995 (9 ha), c) after 2 years (140 ha), and d) after 4 years (534 ha) of population growth (United States Department of the Interior, Fish and Wildlife Service, in litt.).

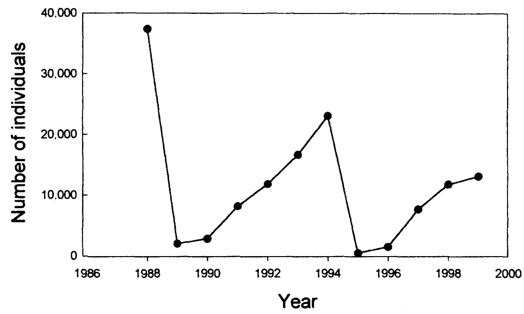

FIG. 4.—Estimated number of black-tailed prairie dogs on Rocky Mountain Arsenal National Wildlife Refuge, Colorado, 1988–1999. Plague epizootics occurred in 1989 and 1995 (modified from United States Department of the Interior, Fish and Wildlife Service, in litt.).

to or faster than the transmission rate, the host population and parasite can persist despite high mortality in infected hosts. Epizootic species are taxa in which disease spreads rapidly and may reduce the size of the susceptible host population either by killing all the hosts or producing a population of immune individuals. In contrast, the disease can persist for a long period with only minor effects on overall population size in enzootic species because loss of individuals from the susceptible population due to death or immunity are compensated by recruitment.

For the past century, scientists have tried to identify enzootic hosts for *Y. pestis* that are characterized by moderate to high resistance to disease, heterogeneity in response to challenge, long and polyestrous breeding season, and short life expectancy (Barnes 1982, 1993; Biggins and Kosoy 2001; Poland and Barnes 1979). Although moderately to highly resistant species have been found with plague from foci around the world (Barnes 1982; Biggins and Kosoy 2001; Poland and Barnes 1979), only rarely has the bacterium been found continuously in a population of rodents (Goldenberg et al. 1964; Hudson et al. 1964).

We believe the expectation for resistant hosts is not necessary for the persistence of *Y. pestis,* a highly virulent disease in which even moderately resistant species may show mortality rates of ≥50% (Holdenried and Quan 1956). White-tailed prairie dogs may provide an example of a new model of an enzootic, or maintenance, host system. White-tailed prairie dogs are as susceptible to plague as are Gunnison's or black-tailed prairie dogs and exhibit epizootics of plague. Individuals infected with moderate doses of *Y. pestis* show essentially 100% mortality. Despite high susceptibility, the low-density colonies of this least social prairie dog species (Nowak 1999) probably contribute to persistence of plague in white-tailed prairie dog colonies because transmission is slow. A relatively rapid rate of recruitment compared with the rate of transmission is an important feature of a species' ability to maintain a highly virulent disease like plague, whether the species is moderately or highly susceptible. Menkens and Anderson (1991) reported that white-tailed prairie dogs were eliminated at a colony with a starting density of 23/ha for 1 year after a plague epizootic. Some animals survived epizootics (Menkens and Anderson 1991) at colonies with lower densities (7–11 prairie dogs/ha). Cully (1989) hypothesized that transmission rates of plague in prairie dogs are density dependent. At low density, transmission of plague is slow enough in white-tailed prairie dogs to allow survivors to reproduce new susceptible individuals at a rate high enough to maintain a host population (i.e., recruitment ≈ mortality).

Adding spatial structure in the form of discrete colonies with intervening unoccupied grassland may enhance persistence by slowing dispersal among colonies where transmission is much slower than it is within colonies. Persistence of virulent organisms in populations of hosts in relatively large geographic areas has been suggested by Yuill (1986) for other arthropod-borne diseases. Migration among colonies can transmit plague from infected to healthy colonies, or provide colonists to restart extirpated colonies. This is the picture that has emerged at Meeteetse and Shirley Basin over the past 15 years.

The Future of Black-Tailed Prairie Dogs

Black-tailed and Gunnison's prairie dogs occur at densities up to 10 times as high as white-tailed prairie dogs and are more social. Thus, they have many more opportunities to exchange fleas or directly transmit the infection. The consequences of the faster spread of plague in these 2 species are profound. Field mortality rates of individuals in infected colonies rise from 85% in white-tailed prairie dogs, which may well be extreme for this species, to nearly 100% in black-tailed and Gunnison's prairie dogs. Infected black-tailed and Gunnison's prairie dog colonies are often extirpated by plague.

Black-tailed prairie dogs on Cimarron National Grassland in Kansas may illustrate a more hopeful pattern, in terms of persistence, at the scale of colony complexes. Despite the fact that plague has been documented from Cimarron National Grassland in 1949, 1997, and 1999 (Cully et al. 2000; J. F. Cully, Jr., in litt.), the area occupied by black-tailed prairie dogs has been fairly stable over the past 10 years. Data for 1989, 1992, 1997, 1998, and 1999 consistently indicate that about 30% of identifiable colony acreage is inactive (United States Forest Service, in litt.). Some of this may be due to shooting, but shooting pressure on Cimarron National Grassland has not been sufficient to eliminate large populations (J. F. Cully, Jr., in litt.). Grassland managers have not allowed any prairie dog poisoning since 1989 (J. Hartman, District Ranger, pers. comm.). Plague is the only disease known to cause extensive die-offs in prairie dogs (Barnes 1993).

Plague may be transmitted intraspecifically into new colonies by prairie dogs dispersing from other, infected colonies, or interspecifically by contact with plague-infected fleas of other rodent species. If transmission among colonies is intraspecific, colonies close together should contract

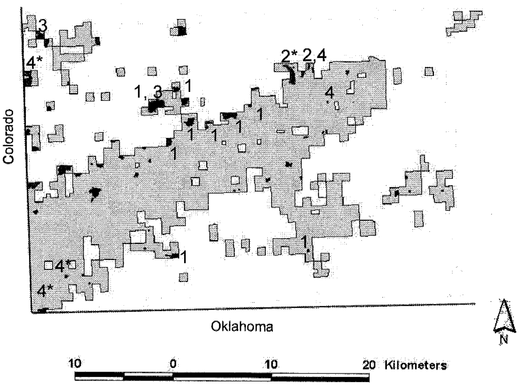

FIG. 5.—Distribution of prairie dog colonies (dark gray) in extreme southwestern Kansas on Cimarron National Grassland (light gray) in 1989–1999. Colonies that were affected by plague are indicated as 1 for 1992, 2 for 1997, 3 for 1998, and 4 for 1999. Asterisks (*) indicate that plague was confirmed at that colony during the given year. Other colonies were assumed to have been reduced by plague because shooting pressure was relatively low, poisoning was not being done, and plague was the only disease known to cause extensive die-offs in prairie dogs.

plague in a wavelike pattern, with close colonies infected before more distant ones. If transmission is interspecific, depending on the dynamics of plague in other host species, plague is expected to occur in prairie dog colonies independent of whether nearby colonies have plague. Staff of the Forest Service at Cimarron National Grassland mapped colonies by hand on 1:126,720 maps in 1989 and 1992, and with global positioning units and geographic information systems in 1997, 1998, and 1999 (Fig. 5). In 1989, 20 colonies were mapped on 303 ha. The 2nd largest town in 1989 covered 92 ha, but <1 ha was active, suggesting a recent die-off. Five additional colonies covering 31 ha also were inactive. In 1992, 30 colonies were mapped. The largest colony from 1989 (102 ha) was 5 ha in 1992. The 1-ha active area in the 92-ha inactive colony mapped in 1989 grew to 63 ha in 1992. A cluster of 7 colonies near the center of the grassland, with intercolony nearest neighbor distances of <3 km, was inactive in 1992 (Fig. 5). Two other colonies (>10 km from the 7 infected colonies) were mostly or totally inactive in 1992. Despite the colonies that became inactive between 1989 and 1992, total active colony area grew 31% to 438 ha during the same period. In 1997, 27 colonies were mapped that covered 504 ha. Two large colonies in the northeastern part of the grassland had plague epizootics, which appeared to extirpate their prairie dog populations in 1997 (Cully et al. 2000). Colony area grew again to 526 ha in 1998 when 30 active colonies were mapped. The large colony in the north-central part of the grassland that was the largest colony in 1989, but 5 ha in 1992, grew to 28 ha in 1997 and again died back in 1998, presumably because of plague, although plague was not confirmed. Another colony in the far northwestern part of the grassland also had a die-off at that time. In 1999, plague was confirmed at 3 colonies, in the northwest, far southwest, and 3 km distant, also in the southwest (Fig. 5).

So what does this say about plague on Cimarron National Grassland? First, the scattered distribution of colonies that were positive for plague in 1997, 1998, and 1999, with intervening unaffected colonies is at odds with the idea that plague is transported by dispersing prairie dogs, although that remains a possibility. Other possibilities are that fleas infected with *Y. pestis* are carried long distances by coyotes or raptors (Barnes 1982, 1993; Cully et al. 2000; Poland and Barnes 1979). Presence of flea species infected with *Y. pestis* and associated with other rodent species in prairie dog burrows supports the hypothesis of interspecific transmission. Second, prairie dog colonies probably are maintained by metapopulation dynamics, in that the rate of colonization of extirpated colonies is about equal to the rate of colony extinction caused by plague. Third, clusters of prairie dog colonies that were extirpated by plague have nearest-neighbor distances <3 km, indicating transmission from recently infected colonies, probably by dispersing prairie dogs.

Work with black-tailed prairie dogs on Cimarron National Grassland (Cully et al. 2000), Gunnison's prairie dog in New Mexico (Cully et al. 1997) and Colorado (Fitzgerald 1970, 1993), and white-tailed prairie dogs in Wyoming (Anderson and Williams 1997; Menkens and Anderson 1991; Ubico et al. 1988) has found fleas of other rodent species, which were positive for *Y. pestis,* in prairie dog burrows. This provides strong support for the hypothesis that epizootics in isolated prairie dog colonies may be caused by contact with fleas of other rodent species such as deer mice, ground squirrels, voles (*Microtus*), or grasshopper mice. This has important ramifications for the delivery of vaccines to curtail epizootics and points to a critical need for research regarding conditions for intraspecific and interspecific plague transmission.

Sylvatic plague is an exotic disease that entered the United States 100 years ago and has become well established in wild rodents in the western one-half of the country. Plague has infected the 4 prairie dog species in the United States for about 60 years with devastating effects. *Y. pestis* is highly virulent to individual prairie dogs of all species, and no evidence suggests that prairie dogs have evolved resistence to plague, although other rodent species have done so in areas of endemic plague (Isaäcson et al. 1983; Quan et al. 1985; Shepherd et al. 1986; Thomas et al. 1988). On Cimarron National Grassland, larger colonies are more likely to become infected with plague compared with smaller colonies. When uninfected colonies occur in close proximity (<3 km) to infected colonies, their likelihood of contracting plague is high. These 2 factors likely result in complexes of small colonies that are mostly >3 km from their nearest neighbors, a situation similar to the distribution on Cimarron National Grassland. Ongoing research must determine if similar patterns of colony distribution within complexes are present at other sites.

The ecology of plague in prairie dogs is highly variable. Prairie dog species differ in density, social behavior, and associated rodents and their ectoparasites. In different parts of the range of black-tailed prairie dogs, rodent associates, their fleas, and climate may be important factors affecting prevalence of plague. It is not possible to predict if plague will cause black-tailed prairie dogs to become dangerously rare, but it is highly likely that they will never attain the dominance on the western plains that was reported at the beginning of the 20th century as long as plague is present.

The current distribution of prairie dogs is modified greatly from the distribution reported 100 years ago. Today, colonies of black-tailed prairie dog are mostly small and widely dispersed, especially in areas where plague is present. Where plague is present, throughout most of the short-grass prairie, it is unlikely that prairie dogs will ever be able to attain their former abundance. To the extent that black-tailed prairie dogs are a keystone species and provide prey or shelter for other wholly or partially dependent species, other components of biodiversity of short-grass prairie also may be threatened by plague. Conservation of black-tailed prairie dogs on mixed-grass prairies, where plague is not present, is essential to maintain the large-scale functional role of prairie dogs on grassland ecosystems so that dependent species can be maintained in viable numbers.

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Literature Cited

Anderson, S. H., and E. S. Williams. 1997. Plague in a complex of white-tailed prairie dogs and associated small mammals in Wyoming. Journal of Wildlife Diseases 33:720–732.

Barnes, A. M. 1982. Surveillance and control of bubonic plague in the United States. Symposia 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. Pp. 28–37 in Proceedings of the symposium on the management of prairie dog complexes for the reintroduction of the black-footed ferret (J. L. Oldemeyer, D. E. Biggins, B. J. Miller, and R. Crete, eds.). United States Fish and Wildlife Service Biological Report 13:1–96.

Biggins, D., and J. Godbey. 1995. Black-footed ferrets. Pp. 105–107 in Our living resources: a report to the nation on the distribution, abundance, and health of U.S. plants, animals, and ecosystems (E. T. LaRoe, G. S. Farris, C. E. Puckett, P. D. Doran, and M. J. Mac, eds.). United States Department of the Interior, National Biological Service, Washington, D.C.

Biggins, D. E., and M. Y. Kosoy. 2001. Influences of introduced plague on North American mammals: implications from ecology of plague in Asia. Journal of Mammalogy 82:906–916.

Burroughs, A. L. 1947. Sylvatic plague studies. The vector efficiency of nine species of fleas compared to *Xenopsylla cheopsis*. Journal of Hygiene, Cambridge 45:371–396.

Clark, T. W. 1977. Ecology and ethology of the white-tailed prairie dog (*Cynomys leucurus*). Milwaukee Public Museum Publications in Biology and Geology 3:1–96.

Cully, J. F., Jr. 1989. Plague in prairie dog ecosystems: importance for black-footed ferret management. Pp. 47–55 in The prairie dog ecosystem: managing for biological diversity (T. W. Clark, D. Hinkley, and T. Rich, eds.). Montana Bureau of Land Management Wildlife Technical Bulletin 2:1–55.

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.

Ecke, D. H., and C. W. Johnson. 1952. Plague in Colorado and Texas. I. Colorado. Public Health Service Monographs 6:1–37.

Eskey, C. R., and V. H. Haas. 1940. Plague in the western part of the United States. Public Health Bulletin 254:1–83.

Fitzgerald, J. P. 1970. The ecology of plague in prairie dogs and associated small mammals in South Park, Colorado. Ph.D. dissertation, Colorado State University, Fort Collins.

Fitzgerald, J. P. 1993. The ecology of plague in Gunnison's prairie dogs and suggestions for the recovery of black-footed ferrets. Pp. 50–59 in Proceedings of the symposium on the management of prairie dog complexes for the reintroduction of the black-footed ferret (J. L. Oldemeyer, D. E. Biggins, B. J. Miller, and R. Crete, eds.). United States Fish and Wildlife Service Biological Report 13:1–96.

Gasper, P. W., and R. W. Watson. 2001. Plague and yersiniosis. Pp. 313–329 in Infectious diseases of wild mammals (E. S. Williams and I. K. Barker, eds.). Iowa State University Press, Ames.

Goldenberg, M. I., S. F. Quan, and B. W. Hudson. 1964. The detection of inapparent infections with *Pasteurella pestis* in a *Microtus californicus* population in the San Francisco Bay area. Zoonoses Research 3:1–13.

Holdenried, R., and S. F. Quan. 1956. Susceptibility of New Mexico rodents to experimental plague. Public Health Reports 71:979–984.

Hudson, B. W., S. F. Quan, and M. I. Goldenberg. 1964. Serum antibody responses in a population of *Microtus californicus* and associated rodent species during and after *Pasteurella pestis* epizootics in the San Francisco Bay area. Zoonoses Research 3:15–29.

Isaäcson, M., P. Taylor, and L. Arntzen. 1983. Ecology of plague in Africa: response of indigenous wild rodents to experimental plague infection. Bulletin of the World Health Organization 61:339–344.

Knowles, C. J. 1998. Status of the black-tailed prairie dog. United States Fish and Wildlife Service, Pierre, South Dakota.

Lechleitner, R. R., L. Kartman, M. I. Goldenberg, and B. W. Hudson. 1968. An epizootic of plague in Gunnison's prairie dogs (*Cynomys gunnisoni*) in south-central Colorado. Ecology 49:734–743.

Link, V. 1955. A history of plague in the United States. Public Health Monographs 70:1–120.

McCoy, G. W. 1908. Plague in ground squirrels. United States Public Health Service, Public Health Report 23:1289–1293.

Menkens, G. E., and S. H. Anderson. 1991. Population dynamics of white-tailed prairie dogs during an epizootic of sylvatic plague. Journal of Mammalogy 72:328–331.

Miles, V. I., M. J. Wilcomb, and J. V. Irons. 1952. Plague in Colorado and Texas. II. Rodent plague in Texas south plains. Public Health Monographs 6:39–53.

Nowak, R. M. 1999. Walker's mammals of the world. 6th ed. The Johns Hopkins University Press, Baltimore, Maryland 2:887–1936.

Poland, J. D., and A. M. Barnes. 1979. Plague. Pp. 515–558 in CRC handbook series in zoonoses. Section A: bacterial, rickettsial, and mycotic diseases (H. Stoenner, W. Kaplan, and M. Torten, eds.). CRC Press, Inc., Boca Raton, Florida.

Quan, T. J. 1982. Plague. Pp. 67–71 in Diseases of wildlife in Wyoming (E. T. Thorne, N. Kingston, W. R. Jolley, and R. C. Bergstrom, eds.). Wyoming Game and Fish Department, Cheyenne.

Quan, T. J., A. M. Barnes, L. G. Carter, and K. R. Tsuchiya. 1985. Experimental plague in rock squirrels, *Spermophilus variegatus* (Erxleben). Journal of Wildlife Diseases 21:205–210.

Rayor, L. S. 1985. Dynamics of a plague outbreak in Gunnison's prairie dog. Journal of Mammalogy 66: 194–196.

Shepherd, A. J., P. A. Leman, and D. E. Hummitszch. 1986. Experimental plague infection in South African wild rodents. Journal of Hygiene, Cambridge 96:171–183.

Thomas, R. E. 1988. A review of flea collection records from *Onychomys leucogaster* with observations on the role of grasshopper mice in the epizootiology of wild rodent plague. The Great Basin Naturalist 48:83–95.

Thomas, R. E., A. M. Barnes, T. J. Quan, M. L. Beard, L. G. Carter, and C. E. Hopla. 1988. Susceptibility to *Yersinia pestis* in the northern grasshopper mouse (*Onychomys leucogaster*). Journal of Wildlife Diseases 24:327–333.

Ubico, S. R., G. O. Maupin, K. A. Fagerstone, and R. G. McLean. 1988. A plague epizootic in the white-tailed prairie dogs (*Cynomys leucurus*) of Meeteetse, Wyoming. Journal of Wildlife Diseases 24:399–406.

United States Department of the Interior, Fish and Wildlife Service. 2000. Endangered and threatened wildlife and plants: 12-month finding for a petition to list the black-tailed prairie dog as threatened. Federal Register 65:5476–5488.

Wherry, W. B. 1908. Plague among the ground squirrels of California. Journal of Infectious Diseases 5: 485–506.

Williams, E. S., K. Mills, A. Boerger-Fields, and C. Lynn. 1992. Survey of prairie dogs and Wyoming ground squirrels for plague in Shirley Basin, Wyoming. Pp. 57–63 in 1991 annual completion report, black-footed ferret reintroduction Shirley Basin, Wyoming (B. Oakleaf, B. Luce, E. T. Thorne, and S. Torbit, eds.). Wyoming Game and Fish Department, Cheyenne.

Williams, E. S., et al. 1997. Survey of coyotes for diseases in Shirley Basin, Wyoming in 1996. Pp. 34–43 in 1996 Annual completion report, black-footed ferret reintroduction Shirley Basin, Wyoming (B. Luce, B. Oakleaf, and E. S. Williams, eds.). Wyoming Game and Fish Department, Cheyenne.

Yuill, T. M. 1986. Diseases as components of mammalian ecosystems: mayhem and subtlety. Canadian Journal of Zoology 65:1061–1066.

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