Plague In Black–Tailed Prairie Dogs: Implications For Management At Thunder Basin National Grassland

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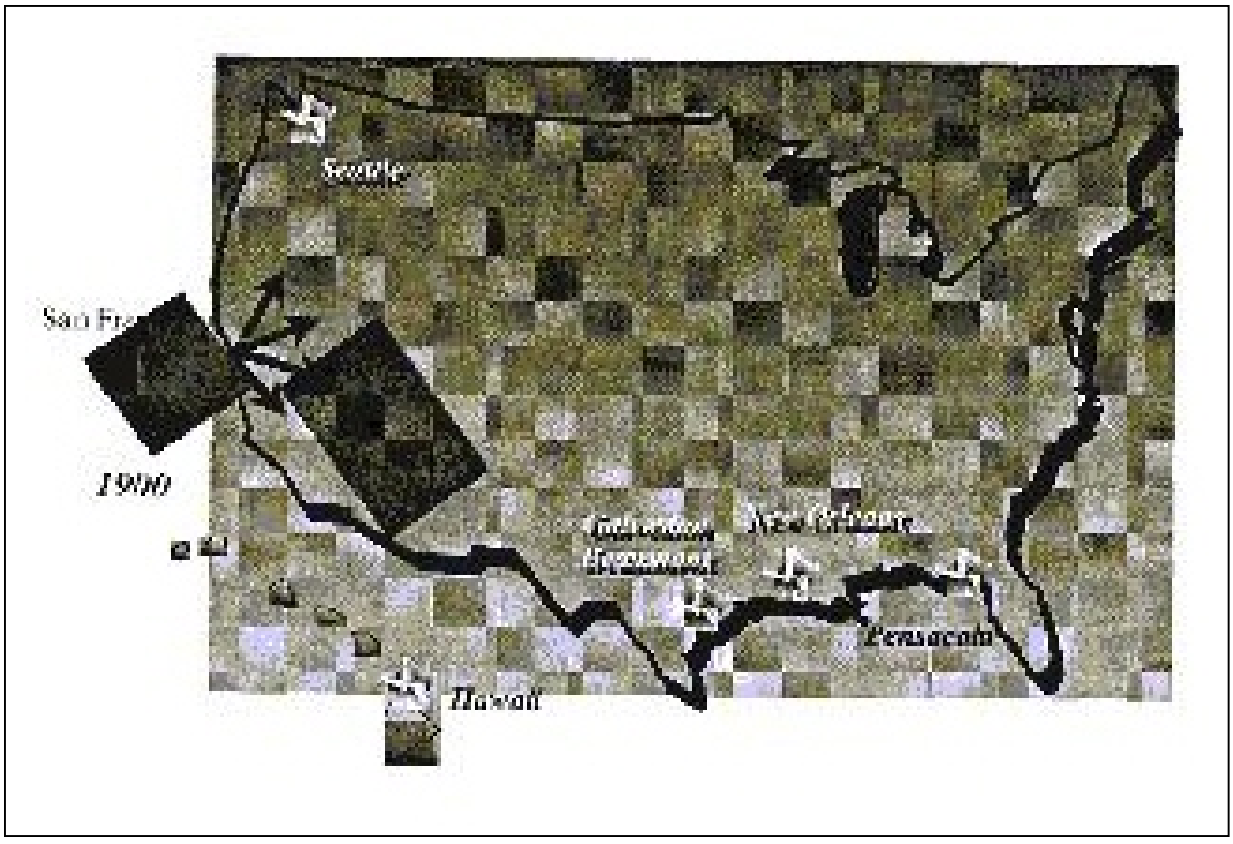
At the beginning of the twentieth century, it is estimated that Black-tailed prairie dogs occupied approximately 40,000,000 ha, mostly in short grass and mixed grass prairie. By 2000, the USFWS estimated that there were less than 800,000 ha remaining, a reduction of more than 98% (USDI 2000). Three factors are responsible for these reductions: habitat conversion, poisoning to reduce perceived competition with cattle for forage, and sylvatic plague. In this presentation I will present information on plague in Gunnison's white-tailed and black-tailed prairie dogs and use these comparisons to try to explain some of the dynamics of the disease in black-tailed prairie dogs (*Cynomys ludovicianus*).

Plague is a bacterial zoonotic disease caused by *Yersinia pestis*. Sylvatic plague refers to plague in wild rodents. A zoonotic disease is a disease of wild animals that occasionally infects people. *Yersinia pestis* is the same organism that caused the famous Black Death in human populations in Europe during the fourteenth century. The Black Death is estimated to nave killed about 30% of the human population of Europe between 1346 and 1349, and it still causes human infections today. Never the less, it is primarily a disease of wild rodents today.

Plague is not native to North America. It came into the United States from Asia in 1899 or 1900, apparently in rats from ships. Plague was introduced into North America at several ports (Figure 1), but disappeared from all except San Francisco where it became

established in the black rat (*Rattus rattus*) populations. Plague was first identified in wild rodents in the United States in California ground squirrels (*Spermophilus californicus*) in the Berkley Hills in 1908 (McCoy 1908, Wherry 1908). By the early 1920s, plague was found in the Los Angeles area to the south and in

Figure 1. Plague entered the United States at several ports at the beginning of the twentieth century, but only became established in the San Francisco Bay area. From there, over the next 50 years, it spread across the western half of the country. (Figure courtesy of the Centers for Disease Control and Prevention)



southern Oregon to the north. By the mid 1930s plague was discovered as far east as Wyoming, Utah, and Arizona, and by 1950 it had spread to approximately its current distribution (Figure 2). In prairie dogs, plague was first identified in Utah prairie dogs in Utah, and white-tailed prairie dogs in Wyoming in 1936. The first records of plague in Gunnison's prairie dogs occurred in New Mexico in 1938 (Eskey and Haas 1940). The first records of plague in black-tailed prairie dogs occurred in Kansas and probably Colorado in 1945, and near Lubbock Texas in 1947 (Cully et al. 2000, Eckey and Johnson 1952, Miles et al. 1952).

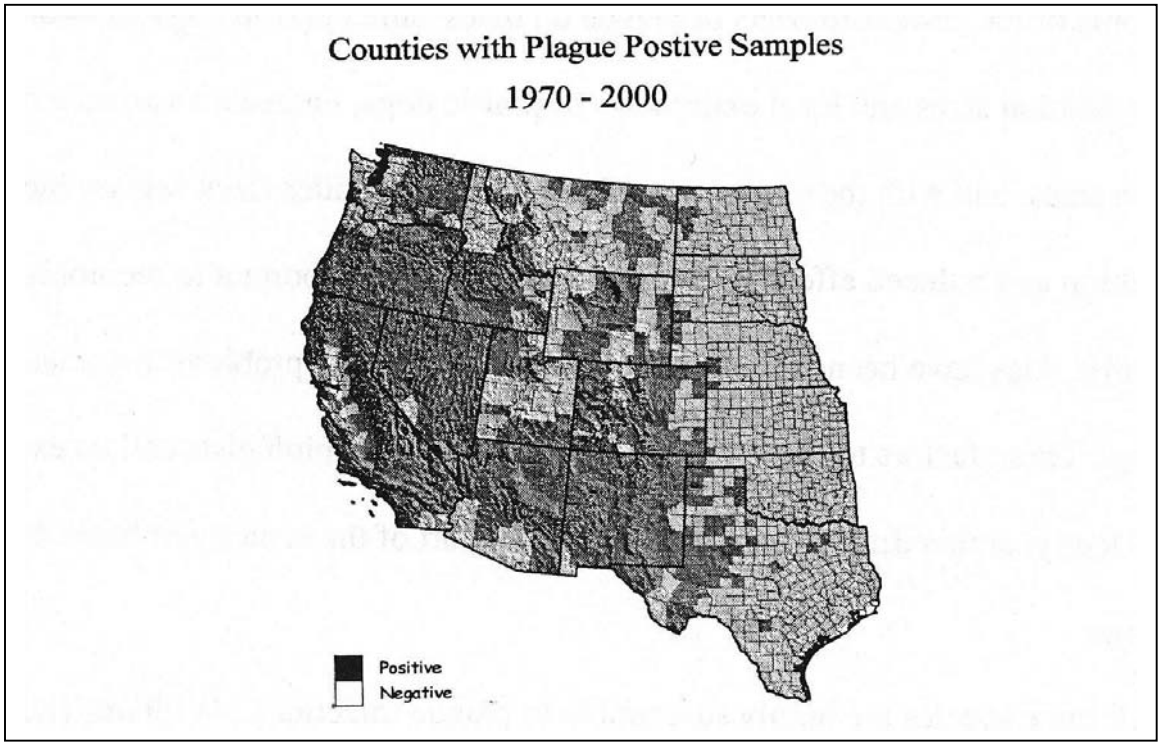


Figure 2. Counties where plague has been found either in humans, wild mammals, or fleas during the period 1970-2000. Plague may occur in counties where it was not documented, but there has not been surveillance during the period. Figure courtesy of the Centers for disease control and prevention.

The overall impact of plague on prairie dogs during the first half of the 20th century is not well documented, but was probably much greater than is generally appreciated. For example, Eckey and Johnson{1952) reported a die-off of a large population of Gunnison's prairie dogs in South Park, Colorado that was later diagnosed as due to a plague epizootic.

This die-off resulted in nearly 100% eradication of GPD over 200,000 acres 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 re-poisoning. Plague was well established in areas with large populations of prairie dogs by 1950. If plague operated on other large colonies of prairie dogs as it did in South Park during 1945-46, its impact may have been as great as that of poisoning in reducing the extent of prairie dogs over extensive areas of their range.

Some of the current impacts of plague on black-tailed prairie dogs are reduced colony population sizes and local extirpation of prairie dogs, increased variance in local population sizes, and with the extirpation of colonies and smaller sizes, we see increased fragmentation and reduced effective dispersal. Dispersal is important to recolonize areas where prairie dogs have been destroyed by plague and to avoid problems associated with inbreeding. These factors together lead to what conservation biologists call an extinction vortex. Clearly, at this time, plague is an important part of the ecology of black-tailed prairie dogs.

All three species are highly susceptible to plague infections. Williams (in litt., 1986) challenged white-tailed prairie dogs with various doses of *Y. pestis* and found the mean lethal dose, the LD50, was 46 organisms. One animal in that study survived a

challenge of 2,300 organisms. On the other hand, 25% of individuals challenged with 2 organisms died. The number of bacteria inoculated by fleas into hosts varies by flea species, but is probably > 10,000 organisms for effective vector species, well above the number necessary to cause infections. Poland and Barnes (1979) do not cite specific laboratory challenge studies but generalize for *Cynomys* species that fewer than 100 organisms serve to cause disease with near 100% mortality. Visible symptoms usually occur 3-4 days following exposure, and death usually occurs within 7 days. Mortality of infected prairie dogs approaches 100 % in all prairie dogs tested.

Fleas are biological vectors of plague. This means that bacteria actually live and reproduce in the flea, at least if the flea is a competent vector. When a flea takes a blood meal from an infected prairie dog or other rodent it ingests *Y. pestis* with its blood meal. The bacteria then multiply in the flea's gut, and together with blood products, form a bolus or clot that blocks the flea's stomach. This process takes from nine days to a month, depending on temperature and perhaps other factors (Eskey and Haas 1940), however fleas can harbor the bacteria for over a year without forming a bolus {Poland and Barnes 1979). When the infected and blocked flea tries to feed again it is unable to hold its blood-meal, which is regurgitated back into the wound, thus inoculating the new host with up to thousands of bacteria (Burroughs 1947). A secondary effect of the block is that because the block prevents the flea from feeding successfully, it begins to starve and becomes voracious. As a result, instead of biting once, feeding to repletion, and retiring to digest its meal, it bites again and again inoculating more *Y. pestis* with each bite. Although many fleas are mostly host specific, the starved condition is thought to cause the flea to become less host-specific and more likely to transmit to other species

when the opportunity presents itself. Thus f1eas are important both as vectors of plague and as long-term host for the bacteria.

We should not underestimate the importance of fleas to the ecology of *Yersinia pestis*. There are more than 200 species of mammals that have been implicated in plague, and the vast majority of those species may harbor more than a single species of flea. Flea species vary greatly in their abilities to transmit plague. Fleas are the arthropod vectors of plague, and although plague can be transmitted directly through blood or other tissue, or through the air (I will discuss this further below), the flea vectors are primarily responsible for maintaining and transmitting plague.

More than 20 species of flea have been found on prairie dogs or in their burrows (Cully and Williams 2001). There are five species of prairie dog specialists that are frequently collected. *Oropsylla hirsuta, O. labis, O. tuberculatis cynomyris,* and *Neopsyll inopina* are frequently implicated in plague. *Pulex* sp. is frequently taken from black-tailed prairie dogs, but is considered a poor vector. Ground squirrel fleas of the genus *Thrassis* are often implicated in prairie dog plague epizootics as are *Aetheca wagneri*, and *Radinopsyla* sp., deermouse fleas. These and the grasshoppermouse flea *Monopsylla exilis* are often implicated in interspecific transmission. All of these fleas are regularly collected infected with *Yersinia pestis* from prairie dog burrows, implicating their normal mammal hosts in plague epizootics (Cully and Williams 2001). Plague positive fleas have been taken from prairie dog burrows as long as three months after the disappearance of the last prairie dog. This long survival of plaque in fleas, in addition to their importance in both intraspecific and interspecific transmission, is a very important component of plague ecology (Cully et at. 1997).

PATTERNS OF PLAGUE IN PRAIRIE DOGS (modified from Cully and Williams 2001)

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 first 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 fleas from either 13-lined ground squirrels (Spennophilus tridecemlineatus) or deer mice at a rock quarry north of the town (Figure 3). In September, 1984, prairie dogs were abundant throughout the grassland of the valley. During winter 1984-1985, most of the prairie dogs in the

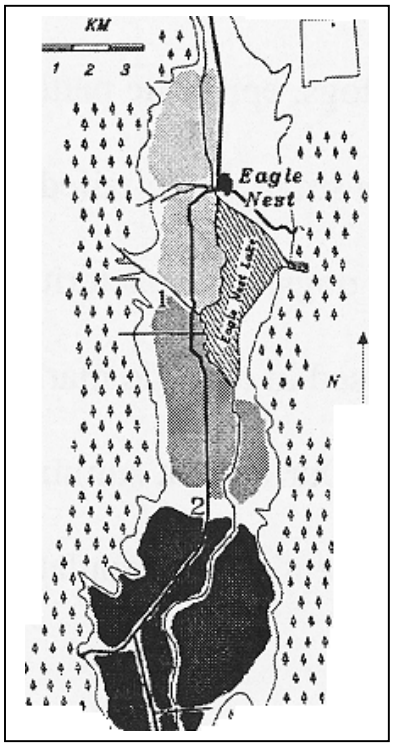


Figure 3. Map of the Moreno Valley showing portions of the Gunnison's prairie dog population that were devastated by plague during 1984-85 (light gray). 1985-86 (medium gray), and 1986-87 (dark gray).

northern one-third of the valley, north of Six -mile Creek disappeared. By late June 1985 only isolated prairie dogs could be found there.

At that time, no indications of plague existed in marked prairie dogs at the study colony, but thirteen-lined ground squirrels, which had been abundant in the previous autumn, were rare and disappeared by early summer. Fleas (*T. bacchi*) of thirteen-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 and no prairie dogs could be found by July 1986. The pattern repeated itself in the southern one-third of the valley between summer 1986 and 1987, except that ground squirrels were not affected. Following 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. Ten years later, in August 1997, prairie dogs were 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. The bacterium, however, 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 also has been present since 1987 at Shirley Basin, Wyoming. Plague was monitored there in association with an attempt to reintroduce black-footed ferrets (*Mustela nigripes*). As at Meeteetse, prairie dog populations at the Shirley Basin have steadily declined with local variation in population size (Menkens and Anderson 1991; Williams et al. 1992; 1997; R. Luce and R. Oakleaf, pers. comm.).

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. The population response of white-tailed prairie dogs to plague is considerably less severe than that of Gunnison's prairie dogs, and affected colonies generally rebounded in 1-2 years. Differences in densities and social interactions probably influence impacts of plague, on these rodent species (Gasper and Watson In press). Plague has been present continuously in the Meeteetse complex since 1985 and at Shirley Basin since at least 1987.

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 first confirmed records of plague in black-tailed prairie dogs were from western Kansas. The current distribution of plague {Fig.2) was established, with minor variations, by the 1950s. It is not known why plague has not spread east beyond its current distribution.

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. 4 and 5). The pattern of rapid simultaneous die-off of multiple colonies was similar to the pattern observed on the Comanche National Grassland, Colorado, in 1995-1996, where all the large towns in the Carizo Unit of the grassland collapsed (Cully 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.), 1945 (Cully et al. 2000) and from 1946-1947 near Lubbock, Texas (Miles et al. 1952).

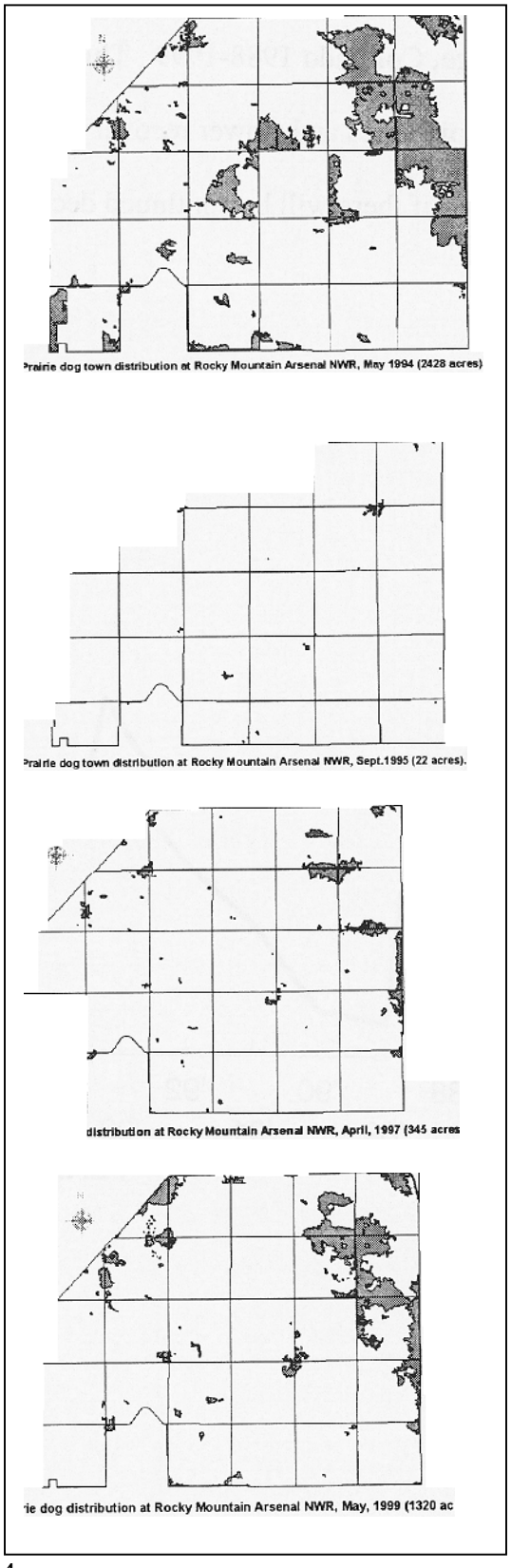
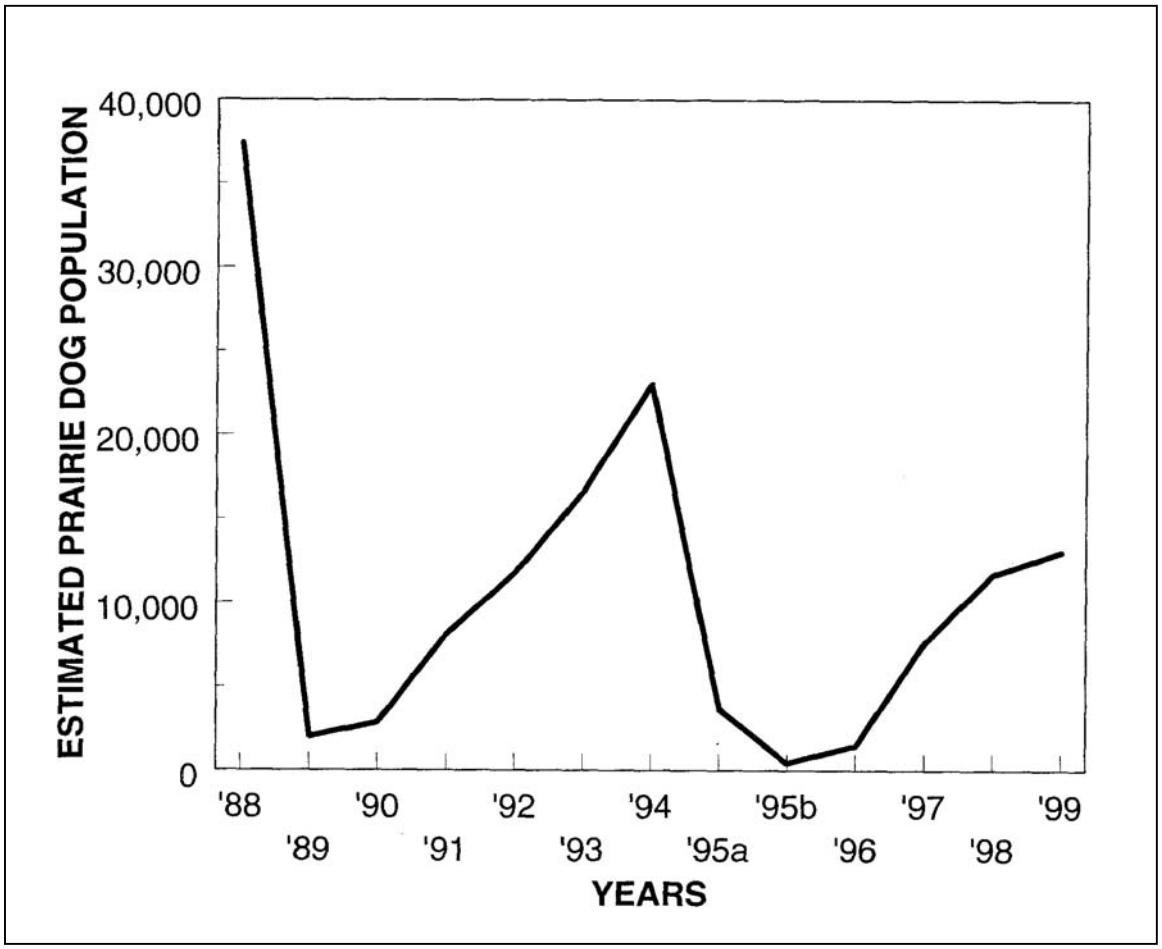


Figure 4.

Figure 5. Estimated populations of black-tailed prairie dogs at Rocky Mountain Arsenal National Wildlife Refuge, Colorado 1988-1999. This figure illustrates well the rapid decline during plague epizootics and slower recovery. Time will tell if the populations will reach 1988 levels, or if there will be continued declines as at Meeteetsie.



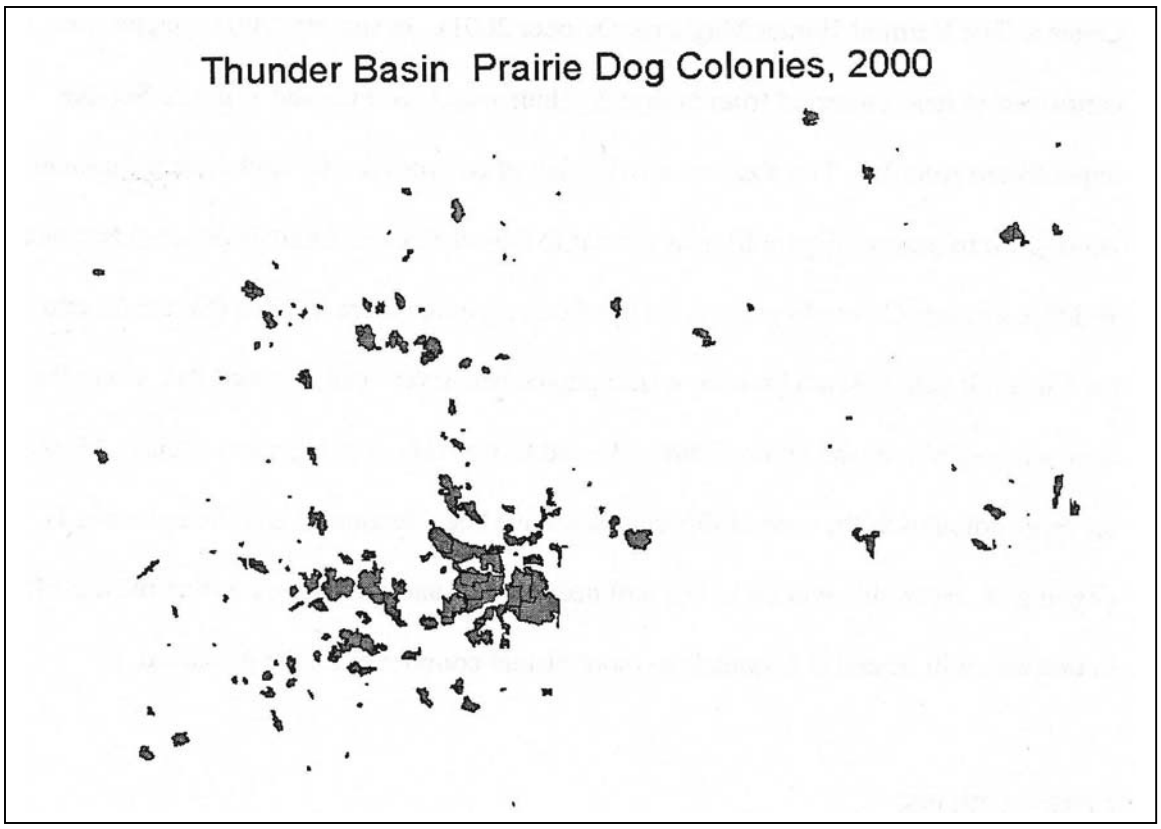


Figure 6. Map of prairie dog colonies at the planned black-footed ferret reintroduction site at Thunder Basin National Grassland, Wyoming. Note the similarity of the large extent of coverage and proximity of colonies to the distribution of colonies at Rocky Mountain Arsenal National Wildlife Refuge prior to the 1994 plague epizootic there.

Thunder Basin National Grassland and Prairie Dogs

Plague was not reported in black-tailed prairie dogs at Thunder Basin National Grassland until colonies began to disappear during summer 2001 (M. Paulick in litt. Letter to The Varmint Hunter Magazine October 2001). In summer 2001, plague was confirmed in fleas collected from prairie dog burrows (U.S. Fish and Wildlife Service unpublished records). The size and distribution of colonies on the landscape in summer 2000, prior to plague (Figure 6), was similar to that of Rocky Mountain Arsenal National Wildlife Refuge, Colorado prior to the epizootic recorded there in 1988 (Figure 5), and at

Conata Basin in South Dakota, where plague has never been reported and where the most successful reintroduction of black-footed ferrets (*Mustela nigripes*) occurs. Most of the large colonies at the core of this complex have been decimated and the epizootic is ongoing (T. Byer, this volume). We will need to wait and see how extensive the die-off in this area will be and if it spreads to more distant complexes on the grassland.

Conclusions

There is clearly a lot that is not known about plague. Some of the questions whose answers would help us manage prairie dogs are: 1) How are plague epizootics started in prairie dogs? Do they result from infected prairie dogs immigrating from other colonies? Do wide-ranging predators such as coyotes (*Canis latrans*) or ferruginous hawks (*Buteo regalis*) bring infected fleas from distant plague infected prairie dog colonies? Do prairie dogs contract plague from other rodent species that may also be involved in plague maintenance? 2) How is plague maintained between prairie dog epizootics? Several suggestions are that the bacteria are maintained in other rodent species, the bacteria survive in soil and reinfect prairie dogs when conditions are right, or that plague is maintained in an avirulent state. At this time there is evidence that many other rodent species and their fleas harbor plague. There is little evidence for the other suggestions. 3) If other species are involved in maintaining plague between prairie dog epizootics, which are they and what are the dynamics of plague in those species? 4) Why has plague not expanded its range beyond the boundaries established around 1950? It appears that host mammal species are available as are competent vector flea species. 5) Are black-tailed prairie dogs viable in the presence of plague?

So what is our current understanding about the ecology of plague in black-tailed prairie dogs? 1) Plague in prairie dog colonies is maintained by metapopulation dynamics of prairie dogs on the grassland. That is, that colonization to repopulate extirpated prairie dog colonies is approximately equal to the plague caused extinction rate of colonies. 2) Between epizootics in prairie dogs, plague is probably maintained in either an epizootic or enzootic state by other susceptible or partially resistant rodent species. We do not know which species are important in different areas and we know nothing about the spatial and temporal dynamics of plague in other rodent species. 3) Colonies that are more than 2 miles from recently infected colonies are probably exposed to plague by fleas of other rodent species, perhaps by fleas left by those species in prairie dog burrows. It remains a possibility that colonies close together are exposed independently to plague in other rodent species. This is an area where a great deal more research is needed.

The scattered distribution of plague positive colonies with unaffected colonies in between, seems to be 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, or that plague bacteria remain in the soil for long time periods and for reasons not understood, become active and reinfect new populations.

My work with black-tailed prairie dogs on the Cimarron National Grassland in New Mexico (Cully et al. 1997), Fitzgerald's with Gunnison's prairie dogs in Colorado (Fitzgerald 1993), and Ubico et al. (1988), Menkens and Anderson (1991) and Anderson and Williams (1997) with white-tailed prairie dogs have all found plague positive fleas of

other rodent species in prairie dog burrows. I believe these flea collections provide strong support to the hypothesis that epizootics in isolated prairie dog colonies in most instances are caused by interspecific contact with other rodent species such as deermice, ground squirrels, voles, or grasshopper mice. This will have important ramifications on the delivery of vaccines to curtail epizootics, and points to another critical research need.

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