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INFLUENCES OF INTRODUCED PLAGUE ON NORTH AMERICAN MAMMALS: IMPLICATIONS FROM ECOLOGY OF PLAGUE IN ASIA

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Intercontinental movements of invasive species continue to modify the world's ecosystems. The plague bacterium (*Yersinia pestis*) has colonized and altered animal communities worldwide but has received much more attention as a human pathogen. We reviewed studies on the ecology of *Y. pestis* in ancient foci of central Asia and in western North America, where the bacterium apparently has become established much more recently. Although rodent populations on both continents are affected dramatically by epizootics of plague, the epidemiologically important species of Asia demonstrate resistance in portions of their populations, whereas those of North America are highly susceptible. Individual variation in resistance, which is widespread in Asian rodents and allows a microevolutionary response, has been documented in few North American species of rodents. Plague increases costs of sociality and coloniality in susceptible hosts, increases benefits of disease resistance in general, and increases benefits of adaptability to variable environments for species at higher trophic levels. Prairie dogs (*Cynomys*) epitomize taxa with high risk to plague because prairie dogs have uniformly low resistance to plague and are highly social. Relationships to plague are poorly understood for many North American rodents, but more than one-half of the species of conservation concern occur within the geographic range of plague.

Key words: coevolution, host–parasite relations, invasive species, plague, resistance, rodent, *Yersinia pestis*

Human-assisted invasions of alien species, including microorganisms, are widely recognized threats to global biodiversity and functioning of ecosystems (Daszak et al. 2000; Kaiser 1999; Kinzelbach 1995). For example, avian pathogens introduced by European settlers to Hawaii are implicated in the extinction of nearly one-half of the endemic land bird species (Warner 1968). Expansion of the plague-causing organism (*Yersinia pestis*) into additional continents from its likely origins in Asia has dramatic ecologic and evolutionary ramifications because of its lethality and extraordinary host range. *Y. pestis* is a generalist found in >200 species of mammals (Poland and Barnes 1979; Pollitzer and Meyer 1961), enhancing its ability to colonize new habitat. Plague causes large population reductions in rodents of several species within its native (Chelnokov et al. 1979; Dyatlov 1972) and introduced (Barnes 1993) ranges. Despite the potential of plague to alter ecosystems that it invades and the attention plague has received as a human pathogen. it has received relatively little attention in reviews of effects of nonindigenous organisms (Pimentel et al. 2000).

We initiated this review out of concern for the decline in prairie dogs (*Cynomys*), which is in part due to plague. and the pro-

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posal to list the black-tailed prairie dog (*C. ludovicianus*) as a threatened species (Graber et al. 1999). Our concern extends beyond prairie dogs to other species of North American rodents with shrinking populations and to those that may have been monitored insufficiently to detect reductions in populations. One goal is to raise the level of attention given to plague as an invasive species that may be disrupting North American ecosystems. As a species, *Y. pestis* may be a relative newcomer on an evolutionary time scale (Achtman et al. 1999), but it has occupied central Asia for ≥2,000 years. We discuss North American plague in the context of ecology of plague in central Asian ecosystems that are at least superficially similar to the dry intermountain areas of western North America, where plague has gained a stronghold in the past 100 years. Data are marginally sufficient to allow comparisons for a few Asian and North American rodent species that are affected directly by plague. Asian and North American mustelid predators of those rodents also are compared, enabling consideration of indirect effects of plague in ecosystems. Although plague is found at least sporadically over much of Asia, we emphasize data collected within the natural foci of plague in central Asia, where it is common and persistent. Discussion centers on comparisons of several attributes including mammalian behaviors, life histories, resistance to plague, and population effects.

Biology of Plague

Origin and variability of the plague pathogen.—Plague may have originated on the Central Asiatic Plateau (Wu et al. 1936). The bacterium is relatively homogeneous, characterized by a single serotype, 1 phage type, and 3 biovars. The 3 biovars of *Y. pestis* (Antiqua, Medievalis, and Orientalis), based on whether they produce nitrous acid or ferment glycerol (Devignat 1951), circulate in Asia, but only Orientalis has been found in North America (Pollitzer 1954). The homogeneity of *Y. pestis* is surprising because it had to adapt to various animal hosts and environment conditions (Guiyoule et al. 1994) but supports the hypothesis of recent (1,500–20,000 years ago) clonal origin of plague from *Y. pseudotuberculosis* (Achtman et al. 1999).

Despite description of *Y. pestis* as a single type, variability occurs within and among populations of *Y. pestis* in many Asian plague foci. In some foci, 40.9–55.7% of isolates of *Y. pestis* differed by ≥1 characteristic (Kozlov 1983). Ribotyping of 70 strains of *Y. pestis* isolated from various geographic areas revealed existence of 11 different DNA banding patterns (Guiyoule et al. 1994). However, only 1 ribotype was found among plague strains from North and South America. Bacterial isolates recovered from plague-endemic areas in Asia may have diverse plasmid content. Apart from 3 prototypic plasmids, isolates of *Y. pestis* may be present with alternate plasmid profiles that include additional plasmids, deletion of plasmids, or appearance of novel plasmids (Chu et al. 1998). Of 242 strains of plague bacteria from various natural foci of the former USSR, 20 (8%) were shown to carry additional cryptic plasmids (Filippov et al. 1990). The diversity of plasmid content of plague bacteria in North America is low compared with that of plague bacteria in Asia. Only the cryptic 19-kilobase plasmid associated with isolates from the United States represents a dimer of 1 of the prototypical plasmids (Chu et al. 1998).

Introduction of plague to new areas.—Plague is widespread on all continents of the world except Australia and Antarctica. Two hypotheses have been proposed to explain movement of plague to North America. Plague may have entered via the Siberian–Alaskan route during preceding geologic eras, assuming that plague has been a naturally associated ecologic factor in North American rodents since the Pleistocene, or plague may have come by means of ship-borne rats (*Rattus*) and their fleas (Stark et al. 1966). Kucheruk (1965) and

Rall (1965) explain the origin of natural foci of plague in North America in terms of distribution of marmots (*Marmota*) across the Beringian bridge during the Tertiary, based on similarity of host–parasite diads in Eurasia and North America, and paleontologic data. However, most authors believe that plague was introduced more recently into North America, along with Hawaii, Indonesia, South Africa, and South America in the 3rd pandemic of the late 19th century and early 20th century (Butler 1983; Pollitzer 1954; Wu et al. 1936). Most strains of *Y. pestis* in North America are in the group of glycerine-negative strains typical for *Rattus* in Asia (Meyer 1942; Pollitzer 1954). New molecular-genetic data (Achtman et al. 1999; Guiyoule et al. 1994) also support the opinion that plague probably was introduced to America from Asia quite recently. Nevertheless, plague may have been introduced into the Western Hemisphere by rat-infested ships much earlier than 1900 (Kozlov 1972), in a fashion similar to that of other vector-borne zoonoses (Childs et al. 1999) and to the devastating movement of other human pathogens from Europeans to Native Americans (Diamond 1997). Regardless of the theories concerning the timing of plague introduction into America, the foci of plague in the New World likely are young compared to Asian foci.

Mammals and Plague

Susceptibility to plague in mammals.—More than 200 mammalian species in 73 genera are known to become infected with plague under natural circumstances (Poland and Barnes 1979; Pollitzer and Meyer 1961). Pathogenic effects of plague have been noted in naturally infected mice, voles, rats, gerbils, ground squirrels (susliks in Russian literature), marmots, and prairie dogs (Perry and Fetherston 1997). Many rodents develop a bubonic form of plague with involvement of internal organs and bacteremia.

Susceptibility may differ widely among closely related Asian taxa (Dyatlov 1972). Species of gerbils show high-level resistance (e.g., *Meriones meridianus*), moderate resistance (e.g., *M. libycus*), or low resistance (e.g., *M. tamariscinus*). Most murid rodents in Asia are highly susceptible to plague, but *Rattus norvegicus*, *Apodemus agrarius*, and *A. peninsulae* may be relatively resistant. Similarly, sciurid species of Asia vary from highly susceptible to highly resistant (Kozlov 1979).

Wild rodent species from New Mexico ranged from uniformly susceptible to plague (similar to control laboratory mice) to completely resistant (Holdenried and Quan 1956). Those investigators found the western harvest mouse (*Reithrodontomys megalotis*), silky pocket mouse (*Perognathus flavus*), piñon mouse (*Peromyscus truei*), white-throated woodrat (*Neotoma albigula*), Mexican woodrat (*N. mexicana*), and least chipmunk (*Tamias minimus*) to be highly susceptible to plague bacteria. Although some individuals were moderately resistant, the majority of deer mice (*Peromyscus maniculatus*), brush mice (*P. boylii*), rock mice (*P. difficilis*), southern plains woodrats (*Neotoma micropus*), and Colorado chipmunks (*Tamias quadrivittatus*) died of plague after inoculation with a low dose of *Y. pestis*. In another experimental study of 1,408 rodents of 7 species, only *Microtus californicus* and *P. maniculatus* were highly resistant to infection, whereas *R. megalotis*, *Mus musculus*, *R. norvegicus*, *Peromyscus californicus*, and *P. truei* were susceptible to plague (Quan and Kartman 1962).

California voles (*M. californicus*) taken from an enzootic plague area in California were highly resistant to plague (Goldenberg et al. 1964). Of 173 California voles that were inoculated with virulent strain of *Y. pestis*, only 2 animals died with evidence suggesting plague infection. Two species of kangaroo rats (*Dipodomys spectabilis* and *D. ordii*) were highly resistant (Holdenried and Quan 1956). Die-offs of these species are not observed, despite evidence of

plague activity in their populations; these rodents seroconvert with few animals becoming ill (Poland and Barnes 1979).

Commonly, carnivores are exposed to plague by eating infected rodents or by being bitten by rodent fleas, but many species are resistant to plague (Barnes 1982; Gage et al. 1994). Field and laboratory studies in the United States suggest that domestic dogs, coyotes (*Canis latrans*), and foxes (*Vulpes vulpes* and *Urocyon cinereoargenteus*) typically seroconvert when exposed to *Y. pestis* and rarely die (Gage et al. 1995). However, domestic cats became acutely ill and experienced high mortality in an experimental study (Gasper et al. 1993). The weak susceptibility to plague of some ungulates, canids, birds, and other animals in which plague does not occur may be due to plague specializations rather than evolved resistance in hosts (Dyatlov 1972).

Primary and secondary mammalian hosts.—Mammalian species that are sufficient to maintain plague in natural systems were defined as primary hosts (Fenyuk 1948) or enzootic hosts (Cully and Williams 2001). The ability of these animals to sustain plague is based on their susceptibility and sensitivity to plague, numbers, distribution, and patterns of behavior. Secondary hosts (epizootic hosts) may facilitate spread of plague but are not able to maintain plague for extended periods without primary hosts.

Opinions differ regarding the number of primary host species that are necessary to support continued circulation of plague within a natural focus (a geographic area in which reservoir and vector species coexist and in which the infection is common). Rall (1965) proposed a concept of monohostality for all plague foci based on investigations in central Asia, wherein a single rodent species is required to maintain plague in a particular focus. A unique variant of *Y. pestis* is adapted to that primary host, although other animal species may participate in circulation of the variant (Kozlov 1979; Rall 1965). For example, Petrov and Shmuter (1958) suggested that only the great gerbil (*Rhombomys opimus*) is necessary to sustain plague in the mid-Asian deserts. Others (Kalabukhov 1949; Nekipelov 1959) suggested that maintenance of plague requires a combination of coexisting rodent or lagomorph species. The great gerbil may be joined by other species to maintain plague in the mid-Asian deserts (Lavrovsky and Varshavsky 1970). In the foothills of Kopet-Dag, Mangishlack, in western and northwestern Turkmenia, the Libyan jird (*Meriones libycus*) is involved, whereas coinvolvement comes from the midday gerbil (*M. meridianus*) in the Aral Karakum and northwestern Kizilkum, the little suslik (*Spermophilus pygmaeus*) on lands north of the Aral Sea, and the Aral yellow suslik (*S. fulvus*) on the Krasnovodsky Peninsula. Such combinations in other areas of Asia may be long-tailed susliks (*S. undulatus*) and Pallas' pika (*Ochotona pallasi*) in Mongolia; Daurian susliks (*S. dauricus*) and rats (*Rattus*) in northeastern China; Siberian marmots (*Marmota sibirica*), Daurian susliks, pikas (*Ochotona*), and voles (*Microtus*) in the Daurian enzootic area; and little susliks plus various gerbils and jerboas in the northwestern Caspian enzootic area (Kalabukhov 1949).

The existence of well-defined variants of the plague bacillus that are adapted to particular rodent species supports the concept of coevolution within single primary hosts in areas of central Asia where the disease is enzootic, but the diversity of species participating in plague circulation seems to be an important factor enhancing maintenance of plague in other areas, including those in America where plague foci were established more recently. Generalizations are problematic because "the complex and shifting milieu" created by interacting fleas and mammals through changing seasons make it difficult "to separate the principals from the bit players, especially since their roles may change with time and in space" (Barnes 1982:253).

Variation in resistance to plague in pop-

ulations of mammals: Asia versus North America.—Rodents characterized as primary hosts should have a heterogeneous response to plague with most individuals being moderately resistant (Fenyuk 1948; Rall 1965). Variation in resistance among populations of primary hosts to *Y. pestis* is a commonly reported attribute with evolutionary implications (Pollitzer 1954). Some mammalian species may become primary hosts only after surviving repeated contact with plague (Kucheruk 1965; Pollitzer 1954; Rall 1965). The proportion of individuals resistant or susceptible to plague in a population characterizes the role of that population in transmission and maintenance of plague. In great gerbils, 40–60% of a population was resistant to *Y. pestis* (Rivkus et al. 1973). Symptoms of mild infection by plague were recorded for 47% of some species of susliks, and 15% of infected individuals were resistant (Borisov et al. 1985). The proportion of resistant individuals was about equal for great gerbils (50–80%), little susliks (50–70%), and midday gerbils (44–60%) iu the Ural steppe focus (Atshabar 1999).

In Asian rodents, partial resistance to plague was correlated with genotypic and phenotypic characteristics including blood groups in great gerbils (Ergaliev et al. 1990) and marmots (Akimbaev et al. 1981), hereditary ability to use oxygen in jirds of the genus *Meriones* (Avanian 1984), several biochemical variables in *M. libycus* (Churikova 1988), level of corticosteroids in blood plasma of midday gerbils (Donskaya et al. 1988), and epigenetic cranial features in the great gerbil (Klassovsky et al. 1999). In areas with the most resistant populations of *R. opimus,* resistant homozygotes did not exceed 40%, susceptible homozygotes did not decrease below 15%, and heterozygotes were 40–50% of the populations (Dyatlov 1972).

Resistance to plague may differ dramatically among different populations of the same species of rodent, and the rate may change within populations depending on recency of exposure. Rodents from plague-endemic areas of Asia were more resistant to plague than were rodents from plague-free areas (Kucheruk 1965; Rall 1965). A noteworthy example is the difference in resistance between populations of midday gerbils from different sides of the Volga River (Birukova 1960). Midday gerbils are primary hosts for plague on the east side of the Volga but have a minor role west of the river in the North Caspian Steppe focus. The relatively high resistance of the eastern population of gerbils was confirmed by experiments with flea-bite transmission (Domaradskii 1998). Levi (1994) demonstrated genetic predisposition for resistance to plague between 2 populations of midday gerbils from east and west of the Volga River by comparing dose-rate susceptibility of live-caught individuals from the 2 populations and examining susceptibility of captive-born hybrids. The number of bacteria required to induce mortality in 50% of infected rodents varied, depending on geographic origin of live-caught hosts (2–216 in specimens from west of the Volga River; 1.0–39.4 $\times$ 10^6 in specimens east of the Volga River) and the filial generation of offspring from east–west hybrids (F_1, 1–131,000; F_2, 56,200–170,000).

Plague bacteria persist in some Asian rodents for extended periods. *Y. pestis* survived in individual long-tailed susliks (*S. undulatus*) for 692 days, the period of observation (Maevsky et al. 1988). Long-term persistence of plague in rodents of North America has not been documented.

Relatively resistant species of *Microtus* and *Peromyscus* maintain plague infection in California (Goldenberg et al. 1964; Kartman et al. 1958, 1962). A few individuals (2.1%) in a population of California voles (*M. californicus*) were asymptomatically infected with *Y. pestis* (Goldenberg et al. 1964). Quan and Kartman (1962) showed significant differences in resistance among populations of *M. californicus* separated by as little as 4.8 km. However, other samples of rodents (*N. albigula, P. truei, Peromys-*

cus leucopus, and *P. maniculatus*) trapped in a plague area did not differ in plague susceptibility from corresponding samples from a plague-free area (Holdenried and Quan 1956). Although populations of rock squirrels (*Spermophilus variegatus*) suffer high mortality during plague epizootics, survivors typically remain after epizootic events. Susceptibility of rock squirrels to experimental infection was heterogeneous; some animals survived inoculation with large number of organisms, and others died after inoculation with small numbers (Quan et al. 1985). Captive-born, plague-naive grasshopper mice (*Onychomys leucogaster*) from a plague-free area in Oklahoma were much less resistant to experimental infection with plague (27% survival) than mice from a Colorado population (75% survival) that had been snbjected to a plague epizootic (Thomas et al. 1988). Similarly, 67% of California ground squirrels (*Spermophilus beecheyi*) from a plague enzootic area survived experimental inoculation with plague compared with 39% survival of squirrels from a nonenzootic area (Meyer 1942). Nevertheless, populations of prairie dogs have remained highly susceptible after repeated exposure to plague epizootics (Barnes 1993), although seroconversion occurred in a small proportion of Gunnison's prairie dogs (*Cynomys gunnisoni*) exposed to a plague epizootic (Cully et al. 1997).

Influence of plague infection on density of mammals.—Devastating effects of plagne on some rodent populations in Asia and North America leave little doubt that this infection can cause large-scale mortality in mammals. Short-term fluctuations in local populations of Asian rodents are common (Chelnokov 1979; Gorbunov 1983), but little is known about long-term effects of plague. Epidemics of infectious diseases may be an important population regulator in mammals (Elton 1925, 1942; Formozov 1947; Severtsov 1941). Nevertheless, analysis of influence of plague, tularemia, leptospirosis, and streptococcus infections performed by Kucheruk (1950, 1955) showed that those infections likely were not a main factor responsible for population cycling in Asian rodents, although those common zoonotic diseases were acknowledged to cause die-offs of rodents. A similar conclusion was reached by Nekrasova et al. (1980) based on study of tularemia in lemmings (*Lemmus*). No evidence suggests that plague has caused long-term declines in primary rodent hosts of Asia (Rudenchik et al. 1989). In contrast, plague has resulted in extirpation of prairie dogs from some areas of North America (Fitzgerald 1993; Lechleitner et al. 1962), and the disease often causes local extinctions and population reductions that may be followed by partial or complete recovery (Anderson and Williams 1997; Cully 1993; Cully et al. 1997).

Interactions between sociality and plague resistance in rodents.—Circulation of *Y. pestis* is influenced by spatial distribution of mammals. Important attributes include density, group sizes, sizes of activity areas, migration, and dispersal (Poland and Barnes 1979; Pollitzer and Meyer 1961). Because *Y. pestis* may be transferred by exchange of infected fleas or direct by contact between infected mammals, coloniality enhances the hazard of disease transmission (Hoogland 1995). Selective pressure from plague thus may favor reduced sociality, or increased resistance to tbe disease in social mammals. Within closely related species, are Asian forms less colonial or social than North American counterparts? Although it would be useful to compare *Marmota bobak,* a steppe-dwelling marmot of central Asia, with prairie dogs and other marmots, requisite information is lacking. However, marmots qualitatively are among the most social of Asian sciurids and are among the most plague-resistant species (B. Suleimenov, pers. comm.). Similarly, great gerbils have an advanced social system, and their populations typically have large proportions of plague-resistant individuals (Atshabar 1999).

Indirect effects of plague at higher trophic levels.—The range of the Siberian

polecat (*Mustela eversmannii*) encompasses central Asian plague foci, in contrast to the closely related black-footed ferret (*M. nigripes*) of North America, which apparently evolved in absence of plague. Differences in life histories and behaviors have been noted (Clark 1989; Forrest et al. 1985), including extreme prey specialization on prairie dogs by black-footed ferrets (Campbell et al. 1987). Another notable contrast is litter size, averaging 3.4 young for black-footed ferrets (Hillman 1968) and about 8.5 young for Siberian polecats (Stroganov 1962). Plague leads to population oscillations in susliks and great gerbils of central Asia, secondarily influencing populations and distribution of Siberian polecats, which in turn may affect competitive interactions between Siberian polecats and other carnivores (Chelnokov et al. 1979; Gorbunov et al. 1983).

Discussion

Infectious diseases may affect evolution of animals (Haldane 1949), but mechanisms and precise estimation of effects of diseases on origin, geographic distribution, evolutionary trends, and extinction of species or subspecies remain largely speculative. When populations are subjected to acute epizootics, potential consequences of evolutionary significance include selection by an epidemic for individuals that are genetically most resistant to the parasite; alteration by intense selective pressure of allelic frequencies of other loci that are linked genetically to loci affecting resistance; and possible reduction of a large population to a small number of survivors, causing a bottleneck effect with loss of genetic diversity (O'Brien and Evermann 1988). However, to persist in an ecosystem, parasites should not eradicate populations of their hosts, and various evolutionary models predict coexistence (Alexander 1981; Mode 1958). Some parasitic systems involving Asian plague are spatially and temporally stable and correspond with distributions of particular rodent species, providing evidence for a strong association of *Y. pestis* in mammalian communities. Plague may be an effective, widely distributed, and, in some areas, permanently acting force in evolution of animals in Asia (Dyatlov 1989).

Coevolution of host and parasite species toward a balance between virulence of pathogen and resistance of host is not the only possible scenario. For vector-borne infections, extensive multiplication and spread of pathogen within a vertebrate host should increase the probability that a biting vector will become infected (Ewald 1994). Coadaptation in parasite-host relations may occur at 3 levels: latent infections at the organism level, such as *Bartonella* bacteria or hantaviruses in rodents, with or without clinical manifestation under stressful conditions; interactions between heterogeneous populations of hosts and pathogens; and host-switching species at the community level.

Recent genetic analysis does not support the hypothesis of ancient origin of plague, coincident with evolution of mammalian hosts (Achtman et al. 1999). The 1,500–20,000 years that may have elapsed since *Y. pestis* was derived from *Y. pseudotuberculosis* (Achtman et al. 1999) may be insufficient to develop a harmless symbiosis between bacteria and hosts. Pathogenesis of plague with a high level of bacteremia in mammals also likely facilitates transmission by fleas. Nonetheless, coevolution between those components of a plague parasitic system in Asia resulted in partial resistance in animal populations. This type of coadaptation between *Y. pestis* and mammalian hosts is not typical for vector-borne infections because *Y. pestis* is transmitted by fleas and respiratory secretions (Ewald 1994). Diversity of the plague microbe in Asian foci may be an adaptation to different species of mammalian hosts. Plague likely came to North American from Asia <500 years ago, and probably much later. As evidence and consequence of the recent invasion of western North America by plague, we list low heterogeneity of *Y. pestis*, with no evidence

of bacterial variants specific for any animal host, and absence of resistance or low partial resistance to plague in the North American mammals.

More than one-half of the species of North American rodents of conservation concern (Hafner et al. 1998) occur within the range of plague in western North America (Barnes 1982). Plague relationships for most are understood poorly. Conditions that increase vulnerability of a rodent species to plague include uniformly low resistance, high population densities (including, but not necessarily limited to, coloniality and sociality), potentially abundant flea vectors that are efficient transmitters of plague, and lack of evolved strategies to cope with high demographic or environmental stochasticity. Plague may interact synergistically with other human-induced and natural disturbances that result in fragmentation and isolation of populations of mammals, increasing their risk of local extirpation and range-wide extinction.

Perhaps plagne is responsible for the absence of a highly social ground squirrel in Asia that is ecologically similar to the prairie dog. In North American prairie dog habitats with plague, costs of the prairie dogs' social system may outweigh benefits (Hoogland 1995) in the long term unless prairie dogs become more resistant to plague. If individual variation in prairie dog sociality exists, relatively nonsocial phenotypes may have a substantial advantage during epizootics of plague. A rapid response in resistance due to selective pressure from plague may have occurred in *Onychomys*, suggesting the possibility of evolutionary change in other characteristics of North American rodents, including social behavior. Behaviors may have been altered in some populations because of as much as 100 years of selection from plague.

As suggested by considerations of ferrets and polecats, instability of mammalian populations at lower trophic levels may affect evolved characteristics of animals at higher levels and structure of ecosystems in general (Dobson and Hudson 1986). Plague-induced temporal variation in prey abundance on Asian steppes (Elton 1925) may favor generalist predators by preventing consistent advantage of preying on any single species (sensu Wilson and Yoshimura 1994). In contrast, the historical absence of plague in North America (Barnes 1993) may have allowed greater stability and density in prairie dog populations, promoting specialization on them by ferrets. Specialization on prairie dogs is now disadvantageous to the black-footed ferret because it has no alternatives when prey are depleted by plague. The ferret is in further jeopardy because it is directly susceptible to plague (Williams et al. 1994), has a relatively low reproductive rate, and has specialized on, arguably, the most plague-vulnerable of North American rodents. For at least several species of North American mammals, introduction of plague dramatically has increased environmental stochasticity, an attribute that is fundamentally important in assessments of population viability (Boyce 1992). Estimates for rates of catastrophes (the upper extreme of environmental variation) have a large impact on predicted persistence times for populations (Mangel and Tier 1994).

Given the number of avenues through which plague may exert influences, this disease likely will continue to alter communities of North American mammals and their associates. The pace of change will be influenced by many variables. Epizootic cycles and population declines have been readily apparent, short-term changes, but potential changes due to altered natural selection and indirect effects at the ecosystem level are at least as disconcerting.

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