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ABSTRACT: Predators play important roles in the ecology, epidemiology, and surveillance of plague in the United States. Most predators are accidental hosts of plague and, with the possible exception of grasshopper mice *(Onychomys* spp.), are not important sources of infection for feeding fleas. However, predators undoubtedly do play an important role in the natural cycle of plague by transporting infected fleas between different populations of plague-susceptible rodents. Predators are known to be at least accidental hosts for 40 of the 50 flea species that have been found to be naturally infected with plague in the U.S. Carnivores, including domestic cats, also play an important epidemiological role and have been sources of infection for 24 human plague cases since 1970. Serosurveillance of rodent-consuming carnivores is currently the most cost-effective method of monitoring plague in the western U.S. During the 1990s, these surveys have allowed CDC and other public health agencies to both identify plague risks for humans living in endemic regions and document the spread of plague into areas where it had not been identified previously.

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INTRODUCTION

Yersinia pestis, the causative agent of plague, is maintained in nature as a flea-borne disease of rodents. Plague probably was first introduced into the United States around 1900 by rat-infested ships entering the port of San Francisco, California (Barnes 1982). Epizootics occurred in local rat populations as the disease spread from the port area to other regions of the city. These epizootics were the source of infection for 121 human cases between 1900-1904 (Link 1955). Within a few years after its introduction, plague passed from San Francisco's urban rat populations into the wild (sylvatic) rodents of the surrounding countryside. Once plague became established in rodents other than *Rattus* spp., it spread rapidly across the western U.S. and by 1940 had been identified as far east as the western edge of the Great Plains (Eskey and Haas 1940).

Plague continues to exist in scattered foci throughout much of the western U.S., although more than 90 percent of the human cases occur in New Mexico, Arizona, Colorado, and California (Figure 1). The ecological relationships of plague in these established foci are extremely complex and each regional focus typically has its own characteristic flea vectors and highly susceptible mammalian rodent hosts (epizootic hosts) (Pollitzer and Meyer 1961, Barnes 1982, and CDC unpublished data). Epizootics usually occur every few years in major foci and often cause mortality in excess of 80 to 99 % among different epizootic hosts (Barnes 1982). Many of these epizootic hosts are associated with important flea vectors and form regional host/flea epizootic complexes. These complexes are important for regional amplification of plague and increase the chances that *Y. pestis* will spread to other rodent species or accidental hosts, such as humans, lagomorphs, or carnivores. The most important epizootic hosts (and their fleas) are discussed in Barnes (1982) and include various ground squirrels *(Spermophilus* spp.), antelope ground squirrels *(Ammospermophilus leucurus),* prairie dogs *(Cynomys* spp.) chipmunks

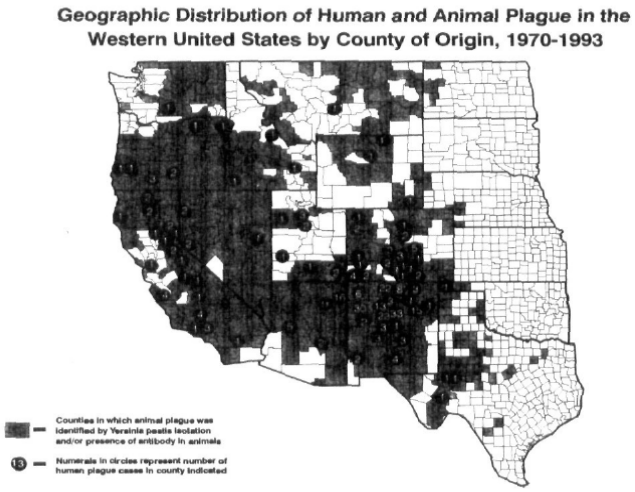

Figure 1. Plague surveillance map showing the distribution of human cases and counties where plague-positive mammals or flea pools have been identified.

(Tamias spp. and *Eutamias* spp.), and woodrats *(Neotoma* spp.). Other rodent species, such as *Peromyscus maniculatus* (deer mice) and *Microtus californicus* (California voles), have populations that differ in their resistance to plague-induced mortality. Rodents from resistant deer mouse or vole populations usually survive infection, but become bacteremic and can serve as sources of infection for feeding fleas. These animals are referred to as maintenance, or enzootic, hosts and are believed, by some, to be critical for maintaining plague during

interepizootic periods (Quan and Kartman 1962, Poland and Barnes 1979).

ROLE OF PREDATORS IN THE ECOLOGY OF PLAGUE

Mammalian predators can become infected with *Y. pestis* after consuming infected prey or being bitten by infected rodent fleas. With the notable exception of grasshopper mice *(Onychomys* spp.), which are discussed separately below, virtually all of these predators are members of the mammalian order Carnivora. Most North American carnivores, including canids, mustelids, procyonids, and ursids, apparently survive plague infections, as indicated by either experimental studies or the large number of seropositive animals found in plague-endemic areas (Rust et al. 1971a, Barnes 1982). Mortality among felids, however, is high and there are numerous records of fatal plague infection in domestic cats and bobcats (Tabor and Thomas 1986, Eidsen et al. 1988, 1991, Gasper et al. 1992, CDC unpublished records).

Although carnivores are susceptible to plague infection, they are only accidental hosts and probably play little or no role as sources of infection for feeding fleas. They do, however, play an important ecological role by transporting plague-infected fleas from one area to another (Poland and Barnes 1979). Carnivores can become infested with plague-infected fleas while consuming prey or exploring rodent nests or burrows. Fleas that have lost their normal rodent hosts during a plague epizootic are especially likely to attack a passing carnivore. Some fleas, such as the prairie dog flea *Oropsylla hirsuta,* actually congregate near burrow entrances after their hosts die and wait for new hosts to appear. When the next host is a carnivore, such as a coyote, the flea can be transported many kilometers before leaving its temporary host. A brief review of records from different treatises on North American Siphonaptera and unpublished CDC records indicates that a wide diversity of fleas at least accidentally infest predators, including a number of rodent fleas that are important plague vectors (Tables 1 and 2) (Hubbard 1947, Hopkins and Rothschild 1953, 1962, 1966, and 1971, Stark 1958, Campos 1971, Wittrockand Wilson 1974, Traub et al. 1983, Holland 1985). Each of the flea species listed in these tables has been reported to be naturally infected with plague (Pollitzer 1961 and CDC unpublished records). It should be noted that 40 of the 50 North American flea species reported to be naturally infected with plague are represented in these tables. Although some of these fleas, such as *Pulex irritans* or *Echidnophaga gallinacea,* are poor plague vectors, other species listed in these tables are extremely important, including many of the *Oropsylla* and *Thrassis* species.

Members of the genus *Onychomys,* especially the northern grasshopper mouse, *O. leucogaster,* play a unique and potentially important role in the interepizootic maintenance and dissemination of plague in North America. With the exception of the Pacific Coast region, the range of the northern grasshopper mouse overlaps much of the known distribution of plague in the United States. Grasshopper mice are unique among North American cricetine rodents in that they are true omnivores (Flake 1973, Landry 1970). In addition to a diet of seeds and insect material, these mice will kill and consume other mice. This behavior exposes them to potentially infected prey and Thomas et al. (1989) demonstrated that 35 *%* of *O. leucogaster* consuming experimentally infected mice became infected. Grasshopper mouse populations have been shown to vary in their susceptibility to plague, with populations from plague endemic areas being highly resistant to plague-induced mortality (Holdenried and Quan 1956, Marchette et al. 1962 and Thomas et al. 1988a). Thomas et al. (1988a) found that first generation mice, reared from *O. leucogaster* parents collected in an area of Colorado where a plague epizootic had occurred recently, were nearly 12,500-fold more resistant to plague-induced mortality than similar offspring from Oklahoma mice collected in a non-endemic area. These data strongly suggest that selection among grasshopper mouse populations during plague epizootics results in a high degree of resistance.

Another important characteristic of grasshopper mice is their ability to serve as temporary or permanent hosts for a great diversity of fleas. Besides becoming infested with fleas while consuming rodent prey, grasshopper mice are likely to be exposed to fleas of other rodents while invading their burrows. It also has been hypothesized that grasshopper mice occupy the burrows of other rodents rather than digging their own (Bailey 1931, Thomas et al. 1988a). In reviewing published flea collection records from *O. leucogaster,* Thomas (1988b) documented collections representing 57 nominate species of fleas from these mice. Of these, 27 are known to be naturally infected with plague (Table 3). Although *Pleochaetis exilis* (formerly Monopsyllus exilis) is rarely found on hosts other than grasshopper mice, most of the fleas collected from *O. leucogaster* are normally associated with other rodents such as *Dipodomys* spp., *Spermophilus* spp., *Peromyscus* spp. and geomyids. The collections cited above indicate that there is a significant degree of flea transfer between grasshopper mice and other sympatric rodents. Given that a male *O. torridus* may utilize a home range as large as 3.71 ha during the breeding season (Frank and Heske 1992), it is likely that grasshopper mice play a role in disseminating plague-infected fleas among rodent populations.

Raptors also have adequate opportunities to become infected with plague, but previous studies indicate that birds are resistant to infection and it is doubtful that predatory birds play any role as sources of infection for feeding fleas (Pollitzer 1954). Raptors, however, have been reported to be infested with potential plague vectors and could transport infected fleas from one susceptible rodent population to another (Hubbard 1947 and Holland 1985). The importance of raptors as transport hosts for plague-infected fleas has received little attention and should be investigated.

ROLE OF PREDATORS IN THE EPIDEMIOLOGY OF PLAGUE

Three hundred and nineteen cases of human plague have occurred in the United States since 1970 (CDC unpublished records). The probably source of infection was determined for 210 of these 319 cases. Most (77.1 *%)* of these 210 cases were associated with various sciurid species and their fleas (Figure 2). Twenty-three

Table 1. Potential plague vectors infesting Felidae and Canidae. Compiled from a list of 50 potential flea vectors from the U.S. that have been found to be naturally infected with plague (Pollitzer 1961 and CDC unpublished data).

FELIDAE	
Felis (domestic cats):	*Ctenocephalides felis, Echidnophaga gallinacaea, Euhoplopsyllus glacialis, Foxella ignota, Oropsylla bruneri, Pulex irritans*
Lynx:	*Cediopsylla inaequalis, Ctenocephalides felis, Echidnophaga gallinacaea, Euhoplopsyllus glacialis, Foxella ignota, Pulex irritans, Thrassis petiolatus*
CANIDAE	
Canis (domestic dogs):	*Cediopsylla inaequalis, Ceratophyllus ciliatus, Ctenocephalides canis, C. felis, Euhoplopsyllus glacialis, Echidnophaga gallinacaea, Hoplopsyllus anomalus, Nosopsyllus fasciatus, Oropsylla bruneri, O. montana, Pulex irritans, P. simulans*
Canis (wild Canis):	*Ctenocephalides canis, Echidnophaga gallinacaea, Euhoplopsyllus glacialis, Eumolpianus eumolpi, Hystrichopsylla dippei, Megabothris abantis, Oropsylla hirsuta, O. idahoensis, O. labis, O. rupestris, Pulex irritans, Pulex simulans, Thrassis acamantis*
Urocyon:	*Ctenocephalides canis, C. felis, Echidnophaga gallinacaea, Pulex irritans*
Vulpes:	*Ctenocephalides canis, C. felis, Euhoplopsyllus glacialis, Megabothris abantis, Oropsylla bruneri, Pulex irritans, P. simulans, Thrassis bacchi*

Table 2. Potential plague vectors infesting Mustelidae and Procyonidae. Compiled from a list of 50 potential flea vectors from the U.S. that have been found to be naturally infected with plague (Pollitzer 1961 and CDC unpublished data).

MUSTELIDAE	
Gulo:	*Euhoplopsyllus glacialis, Oropsylla idahoensis*
Martes:	*Aetheca wagneri, Ceratophyllus ciliatus, Epitedia wenmanni, Euhoplopsyllus glacialis, Foxella ignota, Hystrichopsylla dippei, Nosopsyllus fasciatus, Orchopeas sexdentatus, Thrassis stanfordi*
Mephitis:	*Ceratophyllus ciliatus, Echidnophaga gallinacaea, Eumolpianus eumolpi, Hystrichopsylla dippei, Orchopeas sexdentatus, Oropsylla montana, Pulex irritans, Pleochaetis sibynus, Thrassis acamantis*
Mustela:	*Aetheca wagneri, Catallagia decipiens, C. wymani, Ceratophyllus ciliatus, Ctenocephalides felis, Epitedia wenmanni, Euhoplopsyllus glacialis, Eumolpianus eumolpi, Foxella ignota, Hoplopsyllus anomalus, Hystrichopsylla dippei, Malaraeus telchinum, Megabothris abantis, Megarthroglossus divisus, Nosopsyllus fasciatus, Neopsylla inopina, Orchopeas leucopus, O. sexdentatus, Oropsylla bruneri, O. hirsuta, O. idahoensis, O. labis, O. montana, O. rupestris, O. tuberculata cynomuris, O. t. tuberculata, Peromyscopsylla hesperomys, Pulex irritans, Thrassis acamantis, T. bacchi, T. pandorae, T. stanfordi*
Spilogale:	*Anomiopsyllus nudatus, Atyphloceras echis, Ceratophyllus ciliatus, Ctenocephalides canis, Ctenocephalides felis, Hoplopsyllus anomalus, Orchopeas sexdentatus, Oropsylla montana*
Taxidea:	*Echidnophaga gallinacaea, Hystrichopsylla dippei, Pulex irritans, Pulex simulans, Oropsylla bruneri, O. idahoensis, O. labis, O. rupestris, O. tuberculata cynomuris, O. t. tuberculata, Thrassis acamantis, T. bacchi, T. pandorae*
PROCYONIDAE	
Procyon:	*Ceratophlyllus ciliatus, Ctenocephalides felis, Pulex irritans*
Bassariscus:	*Echidnophaga gallinacaea, Orchopeas leucopus, O. sexdentatus*

Table 3. Potential plague vectors infesting *Onychomys leucogaster*. Compiled from a list of 50 potential flea vectors from the U.S. that have been found to be naturally infected with plague (Pollitzer 1961 and CDC unpublished data).

Aetheca wagneri, Anomiopsyllus spp., Catallagia decipiens, Echidnophaga gallinacaea, Epitedia stanfordi, E. wenmanni, Euhoplopsyllus glacialis, Foxella ignota, Hoplopsyllus anomalus, Malaraeus telchinum, Megarthroglossus divisus, Meringis shannoni, Opisodasys keeni, Orchopeas leucopus, O. sexdentatus, Oropsylla hirsuta, O. idahoensis, O. labis, O. montana, O. tuberculata cynomuris, O. t. tuberculata, Peromyscopsylla hesperomys, Pleochaetis exilis, Pulex irritans, Thrassis bacchi, T. francisi, T. pandorae, T. petiolatus

additional cases occurred in individuals handling infected rabbits *(Sylvilagus* spp.) and another person acquired plague while skinning an infected antelope *(Antilocapra americana).* The remaining 24 cases were associated with handling infected carnivores (Table 4). Fifteen of these cases occurred in pet owners or veterinary personnel exposed to infected domestic cats. Two cases occurred in members of a New Mexico Pueblo tribe who killed and skinned an infected gray fox to obtain its pelt for use in a religious ceremony. The remaining seven carnivore-associated cases occurred in hunters and trappers skinning infected furbearers.

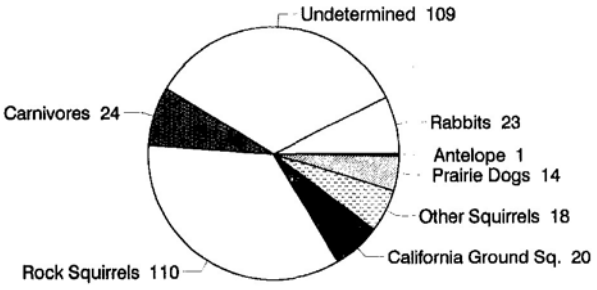

Figure 2. Mammalian sources of infection for human plague cases. Animals were considered to be sources of infection when person became infected after handling an infected member of that species or when a certain species of animal was thought to be the most likely source of infected fleas for a given case.

Table 4. Carnivores as sources of human plague infection in the United States (1970-1993) (CDC unpublished records).

Source	Total number of cases
Felis catus	15
Lynx rufus	4
Canis latrans	2
Urocyon cinereoargenteus	2
Taxidea taxus	1
Total	24

Mortality for the 24 cases associated with carnivores was higher (25.0% of cases fatal) than for the other 295 cases (13.6% of cases fatal) reported from 1970 through 1993. This increased mortality is undoubtedly related to the disproportionately high percentage of carnivore-associated cases that are either primary septicemic plague or primary pneumonic plague (20.8% and 16.7% of 24 cases, respectively). Among the 295 cases where carnivores were not identified as sources of infection, only 12.2% were primary septicemic plague and less than 1 % were primary pneumonic plague. Primary septicemic and primary pneumonic plague are both considered more dangerous than the bubonic form of the disease because they progress more rapidly and are more difficult to diagnose (Poland and Barnes 1979). The higher incidence of primary septicemic plague among carnivore-associated cases is due to persons cutting themselves while skinning animals or exposing existing cuts and abrasions to plague-infected body fluids or tissues. Persons skinning potentially infected carnivores or game animals always should wear thick latex gloves to protect themselves from these exposures. Each of the four carnivore-associated primary pneumonic cases were exposed while handling infected domestic cats. These animals also have been sources of infection for bubonic and primary septicemic cases, and persons should be cautious when handling sick cats from plague-endemic areas.

Pets also can increase human plague risks by bringing infected fleas into homes (Pollitzer 1954, Archibald and Kunitz 1971). Studies in the U.S. have demonstrated that dogs frequently are infested with a variety of fleas, including those potential plague vectors listed in Table 1. Archibald and Kunitz (1971) provided direct evidence that pets can transport infected fleas into human dwellings. These authors collected infected *Pulex irritans* from a plague patient's dog on the Navajo Reservation in Arizona (Archibald and Kunitz 1971). Although *P. irritans* is a relatively poor plague vector, dogs in the Southwest plague focus also have been found infested with important plague vectors, such as *Oropsylla montana* (CDC unpublished data), Persons living in plague-endemic areas should treat pets with insecticidal flea powders and prevent these animals from roaming free in areas where they are likely to be exposed to plague-infected animals or fleas. Trappers and hunters handling infected

furbearers also could be exposed to infected rodent fleas, although this has yet to be demonstrated to be a source of infection.

PREDATORS AS SENTINEL HOSTS

Analyzing serum samples from mammalian predators, especially carnivores, that consume rodent prey is a powerful means of detecting *Y. pestis* in rodent populations (Rust et al. 1971, Cruickshank et al. 1976, Willeberg et al. 1979, Taylor et al. 1981, Barnes 1982, Hopkins and Gesbrink 1982, Smith et al. 1984, Nelson et al. 1985, Isaacson 1986, Clover et al. 1989). Mammalian predators that survive plague infection often develop high antibody titers that persist for as long as four to eight months (Barnes 1982). Sampling even a few rodent-consuming carnivores, such as coyotes, can be roughly equivalent to sampling hundreds of rodents for evidence of plague infection. Carnivore serosurveillance has been used extensively since the 1970's to document the occurrence of plague throughout much of the western U.S., including recent (1992-1993) identifications of epizootic activity in counties of eastern Montana, western North Dakota, and western Nebraska where plague has not been identified previously (CDC unpublished data). The samples collected from western Nebraska represent the first identifications of plague in that state since surveillance activities began in the early 1900s. Carnivore serosurveys are especially recommended when: 1) vast areas must be sampled, 2) investigators want to determine if plague has spread to areas where it has not been detected previously, or 3) plague has not been observed in local rodent populations for many years and it is suspected that the disease may have disappeared from the region.

In North America, the carnivores that have been used most frequently for serosurveillance are domestic dogs, coyotes, and badgers (Rust et al. 1971, Willeberg et al. 1979, Barnes 1982, Hopkins and Gesbrink 1982, Messick et al. 1983, Smith et al. 1984, Nelson et al. 1985). Seropositive foxes, bears, weasels, skunks, martens, fishers, raccoons, ring-tailed cats, domestic cats, bobcats, and mountain lions also have been reported (Barnes 1982, Smith et al. 1984, Zielinski 1984, Clover et al. 1989, and unpublished CDC records).

When carnivores in plague-endemic areas are sampled during interepizootic periods, it is typical to find a small percentage (often < 5%) of animals seropositive at any given time. The percentage of seropositives usually increases dramatically, however, during, or immediately following, an epizootic. Such an increase in seropositives should serve as an early warning of increased plague risks for humans. Studies on the Navajo Reservation in the southwestern U.S. have demonstrated that serosurveillance of domestic dogs is a sensitive indicator of both increased epizootic activity and increased human plague risks. When the percentage of seropositive dogs increased significantly, the level of epizootic activity in local rodent populations also was greater than normal. Increased numbers of canine seropositives and increased epizootic activity also corresponded with increased numbers of human plague cases during these years (Barnes 1982).

Although most serosurveillance programs concentrate on carnivores, grasshopper mice also might be appropriate sentinel hosts in some situations. During a 1984 plague epizootic in Weld County, Colorado, fleas were collected from five species of rodents *(Spermophilus tridecemlineatus, Perognathus hispidus, Dipodomys ordii, Peromyscus matriculates* and *Onychomys leucogaster).* None of the rodents collected were seropositive for plague, but *Y. pestis* was isolated from four species of fleas collected from these same rodents *(Meringis parkeri, Pleochaetis exilis, Thrassis fotus, Meringis hubbardi).* Among these four flea species, only *M. hubbardi* (one flea collected from *S. tridecemlineatus)* was not collected from *O. leucogaster*. More than 450 fleas were collected the following year (1985) from these same rodent species, but none were plague positive. Moreover, among 85 rodent sera tested only seven were positive for plague antibody. Each of these seven positive samples were from *O. leucogaster*. If only grasshopper mice had been used for plague surveillance during this epizootic, all but one infected flea species would have been identified, and serologic evidence of the epizootic would have been observed.

Serosurveillance of predators will continue to be an essential part of plague surveillance programs in the U.S. because the technique is both highly sensitive and cost-effective. The existing serosurveillance program is a collaborative effort involving CDC, state, and local public health officials, as well as animal damage control specialists, wildlife biologists, and others who work with wild or domestic carnivores or other suitable sentinel hosts. Unfortunately cost considerations limit the amount of serological testing that can be done at CDC, but we encourage participation in the program. Samples for these serosurveys can consist of either serum separated from whole blood or drops of blood dried on special paper sampling strips (Nobuto strips) (Wolff and Hudson 1974). Nobuto strips are especially useful for field workers who do not have access to centrifuges or refrigeration. Another advantage is that only about 0.2 ml of blood are required to coat a Nobuto strip with sufficient blood for serological testing. After the strips have been coated with blood, they are allowed to dry before being placed in mailing envelopes that have the appropriate collection data written on the outside. These mailing envelopes are then sent to a central laboratory, such as CDC, for processing. The small amount of blood required for testing can be collected from dead carnivores by opening the body cavity or cutting a large vein. Domestic dogs can be bled from the cephalic vein in the leg without adverse effects, although they should be properly restrained and muzzled to protect both the dog and its handlers. CDC can provide both mailing envelopes and Nobuto strips for appropriate serosurveillance projects.

LITERATURE CITED

ARCHIBALD, W. S., and S. J. KUNITZ. 1971. Detection of plague by testing serum of dogs on the Navajo Reservation. HSHMA Health Reports. 86:377-380.

BAILEY, V. 1931. Mammals of New Mexico. N. Amer. Fauna. No. 53. 412 pp. BARNES, A. M. 1982. Surveillance and control of plague in the United States. Symp. Zoological Society of London 50:237-270. CAMPOS, E. G. 1971. The Siphonaptera of Colorado. M.S. Thesis, Colorado State University. Fort Collins, Colorado. 274 pp. CLOVER, J. R., T. D. HOFSTRA, B. G. KULURIS, M. T. SCHROEDER, B. C. NELSON, A. M. BARNES, and R. G. BOLTZLER. 1989. Serologic evidence of *Yersinia pestis* infection in small mammals and bears from a temperate rainforest in North Coastal California. J. Wildlife Dis. 25:52-60. CRUICKSHANK, J. G., D. H. GORDON, P. TAYLOR, and H. NAIM. 1976. Distribution of plague in Rhodesia as demonstrated by serological methods. Centr. Afr. J. Med. 22:127-130. EIDSON, M., L. A. TIERNEY, O. J. ROLLAG, T. BECKER, T. BROWN, and H. F. HULL. 1988. Feline plague in New Mexico: risk factors and transmission to humans. Amer. J. Publ. Health. 78:1333-1335. EIDSON, M., J. P. THILSTEAD, O. J. ROLLAG. 1991. Clinical, clinicopathologic, and pathologic features of plague in cats: 119 cases (1977-1988). JAVMA. 199:1191-1197. ESKEY, C. R., and V. H. HAAS. 1940. Plague in the western part of the United States. Publ. Hlth. Bull. 254:1-83. FLAKE, L. D. 1973. Food habits of four rodent species in a short-grass prairie in Colorado. J. Mamm. 54:636-647. FRANK, D. H., and E. J. HESKE. 1992. Seasonal changes in space use patterns in the southern grasshopper mouse, *Onychomys torridus torridus*. J. Mamm. 73:292-298. GASPER, P. W., A. M. BARNES, T. J. QUAN, J. P. BENZIGER, L. G. CARTER, M. L. BEARD, and G. O. MAUPIN. 1993. Plague *(Yersinia pestis)* in cats: Description of experimentally induced disease. J. Med. Entomol. 30:20-26. HOLDENRIED, R., and S. F. QUAN. 1956. Susceptibility of New Mexico rodents to experimental plague. Publ. Health Repts. 71:979-984. HOLLAND, G. P. 1985. The fleas of Canada, Alaska, and Greenland (Siphonaptera). Memoirs of the Entomol. Soc. Canada. No. 130. Entomol. Soc. Canada. Ottawa. 631 pp. HOPKINS, D. D., and R. A. GESBRINK. 1982. Surveillance of sylvatic plague in Oregon by serotesting carnivores. Am. J. Public Health 72:1295-1297. HOPKINS, G. H. E., and M. ROTHSCHILD. 1953. An Illustrated Catalogue of the Rothschild Collection of Fleas. Vol. I. Tungidaeand Pulicidae. University Press. Cambridge. 361 pp. HOPKINS, G. H. E., and M. ROTHSCHILD. 1962. An Illustrated Catalogue of the Rothschild Collection of Fleas. Vol. III. Hystrichopsyllidae. University Press. Cambridge. 560 pp. HOPKINS, G. H. E., and M. ROTHSCHILD. 1966. An Illustrated Catalogue of the Rothschild Collection of Fleas. Vol. IV. Hystrichopsyllidae. University Press. Cambridge. 549 pp. HOPKINS, G. H. E., and M. ROTHSCHILD. 1971. An Illustrated Catalogue of the Rothschild Collection of Fleas. Vol. V. Leptosyllidaeand Ancistropsyllidae. University Press. Cambridge. 530 pp. HUBBARD, C. A. 1947. Fleas of Western North America. Iowa State College Press. Ames, Iowa. 533 pp. LANDRY, S. O., Jr. 1970. The rodentia as omnivores. Quart. Rev. Biol. 45:351-372. LINK, V. B. 1955. A History of Plague in the United States. Public Health Monograph No. 26. Public Health Service Publication No. 392. U.S. Gov't. Printing Office, Washington, D.C. 120 pp. MARCHETTE, N. J., D. L. LUNDGREN, P. S. NICHOLES, J. B. BUSHMAN, and D. VEST. 1962. Studies on infectious diseases in wild animals in Utah. II. Susceptibility of wild mammals to experimental plague. Zoonoses Res. 1:225-250. MESSICK, J. P., G. W. SMITH, and A. M. BARNES. 1983. Serologic testing of badgers to monitor plague in southwestern Idaho. J. Wildlife Diseases. 19:1-6. NELSON, J. H., R. H. DECKER, A. M. BARNES, B. C. NELSON, T. J. QUAN, A. R. GILLOGHLY, G. S. PHILLIPS. 1985. Plague surveillance using wild boars and wild carnivore sentinels. J. Environ. Health. 47:306-309. POLAND, J. D., A. M. BARNES, and J. J. HERMAN. 1973. Human bubonic plague from exposure to a naturally infected wild carnivore. Am. J. Epidemiol. 97:332-337. POLAND, J. D., and A. M. BARNES. 1979. Plague. *In* CRC Handbook Series in Zoonoses, Section A: Bacterial, Rickettsial, and Mycotic Diseases. Vol. I. pp. 515-556. (ed.) J.F. Steele. CRC Press. Boca Raton, Florida. POLLITZER, R. 1954. Plague. World Hlth. Organ. Geneva. 698 pp. POLLITZER, R., and K. D. MEYER. 1961. The ecology of plague. *In* Studies in disease ecology: 433-501. May, J.F. (ed.) Hafher. New York, New York. QUAN, S. F., and L. KARTMAN. 1962. Ecological studies of wild rodent plague in the San Francisco Bay area of California. VIII. Susceptiblity of wild rodents to experimental plague infection. Zoonoses Research. 1:99-119. RUST, J. H. JR., D. C. CAVANAUGH, R. O'SHITA, and J. D. MARSHALL, JR. 1971a. The role of domestic animals in the epidemiology of plague. I. Experimental infection of dogs and cats. J. Infect. Dis. 124:522-531. RUST, J. H. JR., B. E. MILLER, M. BAHMANYAR, J. D. MARSHALL, JR., S. PURNVEJA, D. C. CAVANAUGH, and U. S. T. HLA. 1971b. The role of domestic dogs in the epidemiology of plague. II. Antibody to *Yersinia pestis* in sera of dogs and cats. J. Infect. Dis. 124:527-531. SMITH, C. R., B. C. NELSON, and A. M. BARNES. 1984. The use of wild carnivore serology in determining patterns of plague activity in rodents in California. Proc. 11th Vert. Pest Conf. Sacramento,

Calif. D.O. Clark, Ed. Publ. Univ. Calif. Davis, pp. 71-76. STARK, H. E. 1958. The Siphonaptera of Utah. U.S. Department of Health, Education, and Welfare. PHS, Bureau of State Services, CDC. Atlanta, Georgia. 239 pp. TABOR, S. P., and R. E. THOMAS. 1986. The occurrence of plague *(Yersinia pestis)* in a bobcat from the Trans-Pecos area of Texas. Southwestern Naturalist. 31:135-136. TAYLOR, P., D. H. GORDON, and M. ISAACSON. 1981. The status of plague in Zimbabwe. Annals Trop. Med. and Parasitol. 75:165-173. THOMAS, R. E., A. M. BARNES, T. J. QUAN, M. L. BEARD, L. G. CARTER, and C. E. HOPLA. 1988a. Susceptibility to *Yersinia pestis* in the northern grasshopper mouse *(Onychomys leucogaster),* J. Wildlife Dis. 24:327-333. THOMAS, R. E. 1988b. A review of flea collection records from *Onychomys leucogaster* with observations on the role of grasshopper mice in the epizoology of wild rodent plague. Great Basin Nat. 48: 83-95. THOMAS, R. E., M. L. BEARD, T. J. QUAN, L. G. CARTER, A. M. BARNES, and C. E. HOPLA. 1989. Experimentally induced plague infection in the northern grasshopper mouse *{Onychomys leucogaster)* acquired by consumption of infected prey. J. Wildlife Dis. 25:477-480.

TRAUB, R., M. ROTHSCHILD, and J. F. HADDOW. 1983. The Rothschild Collection of Fleas, the Ceratophyllidae: Key to the Genera and Host Relationships. Academic Press, Inc. London. 288 pp.

WILLEBERG, P. W., R. RUPPANNER, D. E. BEHYMER, H. H. HIGA, C. E., FRANTI, R. A. THOMPSON. 1979. Epidemiologic survey of sylvatic plague by serotesting cyote sentinels with enzyme immunoassay. Am. J. Epiydemil. 110:328-334.

WITTROCK, D. D., and N. WILSON. 1974. Ectoparasites of the badger, *Taxidea taxus* (Schreber, 1778), in northwestern Iowa with a list of species recorded from North America. Iowa State J. Res. 49:9-15.

WOLFF, K. L., and B. W. HUDSON. 1974. Paper-strip blood sampling technique for the detection of antibody to the plague organism *Yersinia pestis.* Applied Microbiology 28:323-325.

ZIELINSKI, W. J. 1984. Plague in pine martens and the fleas associated with its occurrence. Great Basin Naturalist 44:170-175.