Importance

Plague is an important zoonotic bacterial disease, and a cause of significant mortality in wild rodents and rabbits. In some animals such as prairie dogs, outbreaks may kill nearly all of the animals in a colony. Sporadic cases also occur in other wild and domesticated mammals, particularly felids. Infections in animals can be transmitted to humans, resulting in life-threatening disease. Pneumonic plague, which is a particularly deadly form of the disease, is usually fatal if antibiotics are not started very soon after the symptoms appear. Bubonic plague is less fulminant, but also has a high mortality rate if left untreated.

At least three major plague pandemics have been seen in human populations. The Justinian plague occurred in the Mediterranean region in the 6th century AD and caused an estimated 100 million deaths, and the Black Death killed a third of the European population beginning in the 14th century. The most recent pandemic, which began in China in the late 1800s, spread worldwide and caused an estimated 12 million fatalities by 1930. The biovars that caused these three pandemics still exist in wild animal reservoirs in parts of the world. The Antiqua biovar, which caused the Justinian plague, occurs in Africa and Central Asia. The Medievalis biovar, associated with the Black Death, is now found only in Central Asia, but the Orientalis biovar, which caused the last pandemic, is widespread. These pathogens occasionally spill over from their reservoirs to affect people or other animals. Approximately 1,000 to 5,000 human cases and 100 to 200 deaths are reported annually to the World Health Organization (WHO), and many additional cases are probably not diagnosed. Most outbreaks occur in Asia and Africa, but sporadic cases and outbreaks can be seen in any endemic region. Plague may reoccur after a long period when the disease seems to disappear; recent outbreaks in India, Indonesia and Zambia followed quiescent periods of 30 to 50 years. This disease is also important because it might be used as a weapon by bioterrorists.

Etiology

Plague results from infection by *Yersinia pestis*, a Gram negative bacillus in the family Enterobacteriaceae. Only one serotype is recognized. *Y. pestis* can be divided into three biovars: Antiqua, Medievalis, and Orientalis. The Antiqua strains are more variable than isolates in the other two biovars. Other classification schemes have also been proposed, including three host-related varieties: ratti (borne by rats), marmotae (borne by marmots), and citelli (borne by susliks [Eurasian ground squirrels]).

Geographic Distribution

*Y. pestis* can be found in parts of Africa, the Middle East, Asia, and North and South America, as well as on Madagascar. The distribution of this organism is patchy. In North America, *Y. pestis* occurs in the western third of the continent, from British Columbia and Alberta, Canada to Mexico, and as far east as Dallas and the western borders of Kansas, Nebraska, Oklahoma and South Dakota. In South America, active foci are found mainly in Brazil and the Andes mountain region of Bolivia, Peru and Ecuador. In Asia, plague has been reported from areas in the former U.S.S.R. east through China, and south to Southwest and Southeast Asia. In Africa, this disease occurs primarily in the eastern and southern regions, but foci are also found in the west and north. Plague is not endemic in Europe or Oceania.

The distribution of each biovar varies. The Antiqua biovar occurs in Africa and Central Asia, and the Medievalis biovar is found in Central Asia. The Orientalis biovar, which caused the last pandemic, is distributed almost worldwide.

Transmission

Plague is usually transmitted by the bites of infected fleas. More than 30 species of fleas are capable of transmitting *Y. pestis*, but they vary in their efficiency as vectors. The oriental rat flea, *Xenopsylla cheopis*, is a particularly effective biological vector. In this flea, *Y. pestis* blocks the gastrointestinal tract, causing the starving flea to bite its host repeatedly and regurgitate the pathogen as it does. Other species of
rodent fleas, including some that are not readily blocked, are also important in transmission. Dog and cat fleas (Ctenocephalides spp) can be infected, but are poor vectors compared to species such as X. cheopis. Human fleas (Pulex irritans) can also carry Y. pestis. Fleas are usually short-lived; however, some may survive for several months, or even a year or more, in rodent burrows after their host have died. During epizootics, there is a high risk that fleas leaving dead animals will bite species they do not usually infest, such as humans.

Other arthropods have also been proposed as potential vectors. Y. pestis has been detected in human lice during outbreaks in people, and lice were able to transmit the infection between rabbits in the laboratory. Ticks have been suggested as possible mechanical vectors in China and the former USSR. Y. pestis has been found in Ornithodoros spp. from Brazil and several ixodid and argasid ticks in Russia.

Direct transmission can also occur between animals. Y. pestis is present in tissues, draining lesions and some body fluids (depending on the form of the disease); these bacteria can be transmitted through mucous membranes and broken skin. People or animals with the pneumonic form of plague may transmit Y. pestis in respiratory droplets. In humans, transmission by inhalation is most common in crowded, poorly ventilated conditions. Animals can transmit bacteria in bites. Carnivores and omnivores, including humans, may also be infected by eating tissues from infected animals. In camels and other herbivores, this may occur when dead rodents or their excretions contaminate the animal’s feed.

Y. pestis can be transmitted on fomites at least for short periods; however, its long-term survival in the environment, particularly in soil, is still poorly understood. This organism is not resistant to desiccation or heat, and on surfaces such as glass and steel, it usually survives for less than 72 hours. However, it is reported to survive for long periods of time in organic material; it may remain viable for up to 100 days in blood and for as long as 9 months in human bodies. Viable Y. pestis was recently found after 24 days in soil that had been contaminated by the blood of a dead mountain lion. In the laboratory, this organism can survive for many months, and possibly years, in autoclaved soil, and for long periods in water. Rodents have been infected experimentally by burrowing in or running over recently contaminated soil, but whether this is an important maintenance mechanism for plague remains to be determined.

**Epidemiology**

In the wild, Y. pestis seems to be maintained in cycles between wild rodents or lagomorphs (e.g., pikas) and fleas. Periodically, these animals experience epizootics, increasing the risk of transmission to other species. What triggers these epizootics, and how Y. pestis persists during interepizootic periods, is poorly understood. Whether this organism circulates in its epizootic hosts between outbreaks, or in a different ‘maintenance’ host, is controversial.

Sporadic cases of plague occur in people who are exposed to tissues from wild animals, or to their fleas. Domesticated animals can act as ‘bridges’ that carry Y. pestis closer to humans. These animals may become infected themselves, or they can simply act as temporary hosts for infected fleas. Infection of rodents in urban areas, particularly rats, can result in epidemic plague in humans. The importance of different transmission routes during human epidemics is still incompletely understood.

**Disinfection**

Y. pestis is susceptible to a number of disinfectants including 1% sodium hypochlorite, 70% ethanol, 2% glutaraldehyde, formaldehyde, and iodine–based and phenolic disinfectants. It can also be inactivated by moist heat (121° C for at least 15 min) or dry heat (160-170° C for at least 1 hour).

**Infections in Humans**

**Incubation Period**

The incubation period for pneumatic plague is 1 to 4 days. The symptoms of bubonic plague appear after 2 to 6 days.

**Clinical Signs**

Three major forms of plague are seen in humans: bubonic plague, septicemic plague and pneumonic plague. All three are acute diseases.

Bubonic plague is the most common form. It begins with the sudden onset of high fever, chills, headache, malaise and myalgia. Dizziness, nausea and vomiting may also be seen. Patients with bubonic plague typically develop an infected, swollen and very painful draining lymph node, called a bubo. Although it can occur anywhere, the bubo is often one of the femoral or inguinal lymph nodes. In some cases, a pustule, vesicle, eschar or papule may be found at the site of the flea bite. People who become infected by ingestion can develop severe pharyngitis and tonsillitis, with swelling of a submandibular lymph node and the neck. Vomiting and abdominal pain may also be seen. If it is not treated, bubonic plague often progresses to septicemia and/or secondary pneumonia.

Approximately 10-25% of plague cases are characterized by primary septicemia. In addition to high fever and other signs in common with bubonic plague, this form has signs of sepsis, but there may be no obvious involvement of the lymph nodes. Epistaxis, hematuria petechiae, disseminated intravascular coagulation (DIC) and neurological signs may also be seen, and the course of the disease can be rapid. Secondary septicemia is similar, but results from disseminated bubonic plague. Meningitis is a relatively rare form of plague; it occurs in approximately 6% of people with the septicemic or pneumonic forms.

Pneumonic plague occurs after the inhalation of bacteria (primary pneumonic plague) or after blood–borne
spread to the lungs. The symptoms of pneumonic plague develop acutely and include high fever, chills, headache, myalgia, malaise and an increased respiratory rate. Within 24 hours, a cough develops; it is initially dry but becomes productive, then bloodstained and/or purulent. The sputum contains only specks of blood at first but eventually becomes foamy and pink or red from blood. Other symptoms may include pleuritic chest pain, nausea, vomiting, diarrhea and abdominal pain. Pneumonic plague is rapidly fatal, with dyspnea, stridor and cyanosis ending in respiratory failure and circulatory collapse.

Pestis minor is a benign form of bubonic plague, usually seen only among people in regions where plague is endemic. Pestis minor is characterized by fever, lymphadenitis, headache and prostration, which resolve spontaneously within a week.

**Communicability**

Pneumonic plague can be transmitted from person to person in respiratory droplets, particularly under crowded, poorly ventilated conditions. This form of plague is most contagious during its final stages, when the number of bacteria in the sputum increases. In the earlier stages, transmission does not seem to occur as readily. Transmission between patients with bacteremia is theoretically possible via ectoparasites.

Person-to-person spread of bubonic plague seems to be rare or nonexistent; however, affected tissues such as draining buboes can contain viable bacteria.

**Diagnostic Tests**

A presumptive diagnosis can be made by identifying the characteristic organisms in sputum, bronchial/tracheal washings, blood, lymph node (bubo) aspirates, cerebrospinal fluid (CSF) or postmortem tissue samples; *Y. pestis* is a Gram negative, facultative intracellular coccobacillus or bacillus with bipolar staining. Bipolar staining is particularly evident when Wright-Giemsa or Wayson stains are used. In some samples such as lymph node aspirates, a relatively homogenous population of bacteria can be found, but samples such as sputum are contaminated by a wide variety of other organisms. *Y. pestis* in clinical samples can be identified by immunofluorescence. Rapid immunoassays can also detect antigens from this organism in clinical samples, and polymerase chain reaction (PCR) assays may be used to detect nucleic acids.

Plague can also be diagnosed by isolating *Y. pestis*. Organisms may be recovered from respiratory secretions, blood and/or aspirates of affected lymph nodes, depending on the form of the disease, as well as from lungs and other tissues postmortem. Organisms are usually present in blood only during septicemia; however, bacteria are sometimes released intermittently from lymph nodes into the blood, and a series of blood samples collected 10-30 minutes apart may be diagnostic. Specimens for culture should be collected before antibiotics are started. *Y. pestis* will grow on ordinary media including blood agar, MacConkey agar, nutrient agar or brain-heart infusion broth. *Yersinia*-specific CIN agar can also be used; this medium is particularly helpful with contaminated samples. *Y. pestis* colonies are small, gray and nonmucoid, and may have a ‘hammered copper’ appearance. Colonies may take up to 48 hours to appear. *Y. pestis* can be identified with routine biochemical tests and other methods. Automated systems may misidentify this bacterium, as it grows slowly and biochemical reactions may be delayed. A specific bacteriophage that lyses only *Y. pestis* and not *Y. pseudotuberculosis* is used as a rapid diagnostic test in reference laboratories. *Y. pestis* may also be recovered in laboratory animals such as mice, particularly when the sample is contaminated with other organisms.

Serology is occasionally helpful. Serological tests include enzyme-linked immunosorbent assays (ELISAs), passive hemagglutination, hemagglutination-inhibition, latex agglutination and complement fixation. A fourfold rise in titer is diagnostic.

**Treatment**

Antibiotics are effective for the treatment of plague; in pneumonic plague, their efficacy is often limited if the symptoms have been present for more than 20 hours. Buboes are occasionally drained but usually resolve with antibiotic treatment. Antibiotic resistant strains seem to be rare, but have been isolated in Madagascar.

**Prevention**

In endemic areas, rodents should be controlled around human homes, workplaces and recreational areas. Buildings should be rodent-proofed, and access to food sources should be prevented. Brush, rock piles, junk and cluttered firewood should not be allowed to accumulate, as they may provide nesting places for rodents. Campers and hikers should not approach rodents or their carcasses, and should avoid sleeping beside rodent burrows. To prevent pets from serving as a link between wild animal hosts and humans, a good flea control program should be established for dogs and cats, and these animals should be kept from hunting or eating tissues from animals that may be infected. Game meat, as well as tissues from domesticated animals that might be infected, should be cooked thoroughly. Die-offs of rodents or lagomorphs should be reported.

Personal protective equipment (PPE) including gloves should be worn when handling animals or tissues if there is any risk that they might be infected. Good hygiene, including frequent hand washing, should be practiced. Insect repellents can also be applied to clothing and skin if exposure to rodent fleas is expected. Veterinarians and their staff should use good infection control procedures and PPE with suspected cases of plague. More stringent precautions are necessary when pneumonic plague is suspected or
higher risk procedures such as necropsies are performed. Specific recommendations for protective measures are available from the U.S. Centers for Disease Control (CDC) and other groups (see Internet Resources, below).

In endemic regions, rodents that host Y. pestis may be monitored and/or controlled. Concurrent insecticidal treatment is often necessary when hosts die or are killed, as fleas leave the carcasses to seek new hosts. People who have been exposed to Y. pestis are treated prophylactically with antibiotics. Good infection control procedures, including the use of disposable surgical masks, are used to prevent transmission from patients with pneumonic plague.

**Morbidity and Mortality**

Y. pestis is endemic in populations of wild rodents and lagomorphs, and occasionally spills over to affect people or other animals. Worldwide, approximately 1,000 to 5,000 human cases of plague and 100 to 200 deaths reported annually to the World Health Organization, and many additional cases are probably not diagnosed. Most outbreaks occur in Asia and Africa, but sporadic cases and outbreaks can be seen in any endemic region. On average, fewer than 20 cases of plague are reported annually in the U.S., but up to 40 cases have been reported in some years. Plague may reoccur after a long period when the disease seems to disappear; recent outbreaks in India, Indonesia and Zambia followed quiescent periods of 30 to 50 years.

Bubonic plague accounts for 80–95% of the cases seen worldwide. Without treatment, the case fatality rate for this form is estimated to be 40-70%; some sources suggest it may be as high as 90%. The availability of treatment lowers the case fatality rate in bubonic or septicemic plague to approximately 5–15%. Untreated pneumonic or septicemic plague is almost always fatal, often within a few days. If appropriate treatment is given very soon after the onset of symptoms, most people survive; however, the narrow window for treatment means that the case fatality rate for the pneumonic form remains greater than 50%.

**Infections in Animals**

### Species Affected

Rodents and lagomorphs are the most important host species for plague. These animals are infested with fleas that can transmit Y. pestis, and develop bacteremia high enough to infect those fleas. Infections have been documented in more than 200 species and subspecies of rodents. In the U.S., significant hosts include prairie dogs (Cynomys spp.), ground squirrels (Spermophilus spp.), antelope ground squirrels (Ammospermophilus spp.), chipmunks (Tamias spp.), wood rats (Neotoma spp.) and mice (Peromyscus spp.) in the southwestern states, and ground squirrels, chipmunks, and wood rats in Pacific coast states. In Asia, important hosts include pikas (Ochotona spp.), which are lagomorphs, and rodents including various species of susliks (Spermophilus spp.), rats (Rattus spp.), Siberian marmots (Marmota sibirica), voles (Microtus spp.), jerboas, and some gerbils (Rhombomys opimus and Meriones spp.). Rats are considered to be the primary hosts for Y. pestis in Madagascar. In some geographic areas, the hosts are not known. Among rodents and lagomorphs, clinical signs are more likely to be seen in some species than others.

Many other species of mammals also become infected, but the majority are incidental hosts. Some species are more likely to develop clinical signs than others. Felids seem to be particularly susceptible to plague; fatal disease has been reported in housecats and wild cats including bobcats and mountain lions. Black-footed ferrets (Mustela nigripes) are also very susceptible. Infrequent cases of plague have been described in ungulates including camels (Camelus bactrianus and Camelus dromedarius), various species of deer, pronghorn antelope, llamas and goats. Y. pestis infections have also been reported in dogs, coyotes, foxes, badgers, skunks and nonhuman primates.

### Incubation Period

Clinical signs develop within 1 to 4 days in cats.

### Clinical Signs

Bubonic plague, septicemic plague and pneumonic plague seem to occur in animals as well as humans; however, plague should be a consideration in any animal with a systemic infection and a history of potential exposure in an endemic area.

Most cats infected with Y. pestis develop the bubonic form of plague. This form is usually characterized by fever, anorexia and lethargy, with an enlarged lymph node (bubo) near the site of inoculation. Many cats are probably infected by ingestion, and the submandibular lymph nodes are most often involved. The affected lymph node may develop abscesses, ulcerate and drain. Some cats also have cellulitis, abscesses at sites other than lymph nodes, mouth lesions including ulcers, or necrotic tonsillitis. Vomiting, diarrhea, ocular discharges, dehydration and weight loss have been reported. Bubonic plague can progress to septicemic plague, with systemic signs including tachycardia, pale or brick red mucous membranes, a prolonged capillary refill time and a weak pulse. DIC and/or respiratory distress may also be seen. Cats with primary septicemic plague have similar clinical signs, but without a bubo. Pneumonic plague can develop in cats with bubonic or septicemic plague, and is characterized by respiratory signs including dyspnea and hemoptysis. Neurological signs such as incoordination have also been reported in infected cats. Studies in experimentally infected cats and serological surveys suggest that some animals might have mild or asymptomatic infections.

Dogs seem less likely to become ill than cats, and subclinical infections may be more common. Only rare descriptions of plague in naturally infected dogs have been...
published: the clinical signs included fever, lethargy, submandibular lymphadenitis, lesions in the mouth and coughing. Experimentally infected dogs inoculated by the subcutaneous or oral routes developed a fever and other signs of illness, but recovered spontaneously during the next week. Two dogs exposed via aerosols died.

In rodents, the outcome varies from subclinical infection or mild illness to severe, rapidly fatal disease. Epizootics with high mortality rates are reported among some rodents and lagomorphs. Infections in other wild animals are poorly understood. Wild felids including mountain lions and bobcats seem to be relatively susceptible to plague, and may be found dead. Other wild carnivores or omnivores might be less susceptible. Fever and lethargy, without bacteremia, were reported in experimentally infected raccoons in one study. In another experiment, neither fever nor deaths were seen in this species, or in coyotes and striped skunks infected by the oral route. However, some individual animals may be more susceptible; *Y. pestis* has been found in the carcasses of dead coyotes, as well as in foxes and other species.

Occasional cases of plague have been reported in domesticated or wild ungulates. Ocular plague, characterized by keratoconjunctivitis, endophthalmitis and panophthalmitis, has been documented in mule deer (*Odocoileus hemionus*) and black-tailed deer (*Odocoileus hemionus columbianus*). Septicemia and pneumonia have also been seen in mule deer, either with or without ocular signs. Overall, plague is not reported to be an important cause of morbidity and mortality in this species. Goats and camels can become ill and die, and a death was reported in a llama in New Mexico. Clinical cases have not been reported in the literature in cattle, horses or pigs.

**Communicability**

Animals with the pneumonic form can transmit *Y. pestis* in respiratory droplets. Bacteria can also be found in draining lesions, in some other secretions and excretions, and in tissues. These organisms can cross mucous membranes or broken skin. They may also be ingested by predators. Some animals, including cats, have transmitted the organism in bites.

Most human cases are associated with wild rodents or lagomorphs, but other species including bobcats, coyotes, mountain lions, foxes and badgers have also been involved. Among domesticated animals, cats seem to be most likely to transmit plague to humans. Small outbreaks have also been reported in people who ate uncooked tissues from infected hosts (e.g., uncooked camel liver or guinea pig flesh). Cases transmitted by direct contact with dogs have not been published; however, a recent study suggests that extended contact with dogs may increase the risk of plague, possibly by bringing infected rodent fleas into the household.

**Post Mortem Lesions**

In cats, necrotic foci may be found in the liver, spleen, lungs and other internal organs. The liver may be pale and the spleen enlarged. Affected lymph nodes can be markedly swollen, with necrosuppurative inflammation, edema and hemorrhagess. Diffuse interstitial pneumonia, focal congestion, abscesses and hemorrhages may be found in the lungs.

In wild animals, the lesions may include hemorrhagic buboes and splenomegaly in some acute cases, or caseous buboes and necrotic lesions in the spleen, liver and lungs when the disease progresses more slowly. Keratoconjunctivitis, endophthalmitis and panophthalmitis, as well as septicemic lesions, pneumonia and lymphadenitis have been reported in deer.

**Diagnostic Tests**

In the U.S., plague diagnosis is usually carried out by state public health laboratories or the Centers for Disease Control and Prevention (CDC). These laboratories should be contacted before collecting samples. Plague is a serious zoonotic disease; samples should be collected, handled and shipped with all appropriate precautions, including appropriate personal protective equipment (PPE) during their collection.

A presumptive diagnosis can be made by identifying the characteristic organisms in clinical samples such as lymph node (bubo) aspirates or swabs of draining lesions. Some types of samples, including lymph nodes, may contain a relatively homogeneous population of bacteria. *Y. pestis* is a Gram negative, facultative intracellular coccobacillus or bacillus with bipolar staining. Bipolar staining is particularly evident when Wright-Giemsa or Wayson stains are used. Bacteria in clinical samples can be identified by immunofluorescence. Rapid immunoassays can also be used to detect *Y. pestis* antigens in clinical samples, and PCR may be used to identify nucleic acids. *Y. pestis* can sometimes be detected by PCR or other techniques in fleas collected from the animal.

Plague can also be diagnosed by isolating *Y. pestis* from blood, nasal/oral swabs, lymph node aspirates, swabs of draining lesions, transtracheal aspirates and/or tissue samples including the liver, spleen, lungs and affected lymph nodes. Specimens for culture should be collected before antibiotics are started. *Y. pestis* will grow on ordinary media including blood agar, MacConkey agar, nutrient agar or brain-heart infusion broth. *Yersinia*-specific CIN agar can also be used; this medium is particularly helpful with contaminated samples. *Y. pestis* colonies are small, gray and nonmucoid, and may have a “hammered copper” appearance. Colonies may take up to 48 hours to appear. *Y. pestis* can be identified with routine biochemical tests and other methods. Automated systems may misidentify this bacterium, as it grows slowly and biochemical reactions may be delayed. A specific bacteriophage that lyases only *Y. pestis* and not *Y. pseudotuberculosis* is used as a rapid diagnostic test in
reference laboratories. Y. pestis may also be recovered in laboratory animals such as mice, particularly when the sample is contaminated with other organisms.

Serology using paired serum samples can be helpful. A single sample, together with consistent clinical signs, may also be supportive. Various serological tests including latex hemaggululation and passive hemaggululation tests may be available.

**Treatment**

Early treatment with antibiotics can be successful.

**Prevention**

A good flea control program should be established for dogs and cats, and they should be kept from hunting or eating tissues from animals that may be infected. Animals that become ill should be examined by a veterinarian. Barrier precautions are necessary during examination and treatment, and suspected cases are isolated. The most stringent measures are needed before antibiotics are begun and during the initial stages of treatment. PPE may include gloves, surgical masks to prevent droplet infection, protective clothing, and eye protection if splashes or sprays are expected. Excellent hygiene should be practiced.

Vaccination has been used to protect endangered black-footed ferrets, which are highly susceptible to plague, during epizootics. Vaccines (in food bait) were also given to prairie dogs, which are the food source for these ferrets, and prairie dog burrows were dusted with an insecticide. Vaccines might also be promising for controlling plague in rodents near human environments. Vaccines are not currently available for domesticated animals.

**Morbidity and Mortality**

In endemic areas, epizootics occur periodically in susceptible rodents and lagomorphs. The mortality rate may approach 100%. Between epizootics, plague persists in wild animals without causing high mortality. Resistance to plague varies between rodent species. Highly susceptible hosts include California ground squirrels (Spermophilus beecheyi), rock squirrels (S. variegatus) and prairie dogs (Cynomys spp.) in North America, and some suslik populations (Spermophilus spp.) in Asia. Other species are more resistant. The percentage of individuals who survive Y. pestis infection is reported to be 40–80% in the great gerbil (Rhombomys opimus), 50%–70% in little susliks (S. pygmaeus) and 44%–60% in midday gerbils (Meriones meridianus). In North America, kangaroo rats (Dipodomys spp.) are reported to be highly resistant, while northern grasshopper mice (Onychomys leucogaster), deer mice (P. maniculatus) and California voles (Microtus californicus) vary in their susceptibility. Populations that live in endemic areas may be more resistant than those that live outside these regions.

Among other mammals, felids seem to be particularly susceptible to plague; fatal disease has been reported in housecats and wild cats including bobcats and mountain lions. One study reported that the mortality rate was 14% in housecats with bubonic plague, 70% in cats with septicemic plague (or cases that were not classified into a form), and 83% in the pneumonic form. In experimentally infected cats with bubonic plague, the case fatality rate can be as high as 60% if the disease is left untreated. Subclinical infections also seem to occur. Surveillance has reported antibodies in healthy cats, and some cats have survived experimental infections. In one study, 20 of 25 cats inoculated by ingestion or subcutaneous inoculation became ill, but three cats seroconverted without clinical signs. Dogs do not seem to be as susceptible to plague as cats. Ten dogs that were infected by subcutaneous or oral inoculation experienced only a brief illness and recovered on their own. Pneumonic infections may be more serious: two dogs infected by aerosols died.

Serological evidence suggests that wild carnivores are frequently exposed to Y. pestis, probably through hunting. Seroprevalence rates are reported to be 13-14% in raccoons and coyotes, and 55% in badgers. Experimentally infected raccoons, coyotes and striped skunks survived the infection. However, fatal infections have occasionally been reported in some species, including coyotes, in the wild. Black-footed ferrets (M. nigripes) are very susceptible to plague, and have a high mortality rate. In contrast, experimentally infected domesticated ferrets (Mustela putorius furo) and Siberian polecats (M. eversmanni) did not become ill.

**Internet Resources**

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Plague

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*Link defunct as of 2009