PLAGUE IN NEW MEXICO

I. INTRODUCTION

Plague is an acute bacterial zoonotic disease that is transmitted primarily by the fleas of certain species of rodents. In most parts of the world, commensal rodents (*Rattus rattus* and *R. norvegicus*) are the most important hosts. In the western U.S., however, it is native rodents, primarily ground squirrels, which serve as amplifying hosts of the disease and the source of most human plague cases.

II. HISTORY

A. Plague has occurred in three great (known) pandemics (widespread epidemics over time) during the last 15 centuries. The first recorded pandemic, known as Justinian’s Plague, began in East Africa around 540 AD and spread north to the entire Mediterranean region. It is estimated this pandemic lasted 50 to 60 years and claimed 40 million lives (out of an estimated world population of slightly more than 260 million people).

The second pandemic, which occurred during the 14th century, became known as the infamous "Black Death". It arose in central Asia and spread through China, India, and eventually by trading ships to the Middle East, North Africa and Europe. Unlike the first pandemic, which was probably almost entirely flea-borne, the Black Death also caused explosive outbreaks of primary pneumonic plague, an air-borne form of the disease that can be passed directly from person to person by coughing and is virtually always fatal unless promptly treated with appropriate antibiotics (non-existent in the 14th century). It is estimated that one-fourth to one-third of Europe's population, or about 25 million people, died in this pandemic.

The modern pandemic began in southwest China in the latter part of the 19th century, and spread to Canton and Hong Kong by 1894. From Hong Kong the disease was carried by rats on ships to Calcutta and Bombay by 1896, and to San Francisco in 1899. It eventually reached every continent except Antarctica. The modern pandemic did not produce the tremendous mortality and social chaos associated with the previous two pandemics, but it did result in an expansion of the natural distribution of plague to include North America. It was also during this time that the etiologic agent of plague, the bacteria *Yersinia pestis*, was identified, and transmission by fleas was proven.

B. History of urban plague in the U.S. — Plague was introduced into urban rat populations of several port cities around the turn of the century, including San Francisco, Seattle, New Orleans, Galveston and Pensacola, resulting in hundreds of human plague cases. In most of these cities only domestic rat populations became infected; rat and flea control contained the disease and stopped its spread.

C. Establishment of natural plague foci in the U.S. — Plague spread from domestic rats in the San Francisco Bay area to California ground squirrels and their fleas by 1908 and continued to spread through wild rodents, rabbits and carnivores throughout the West. Plague was first
detected in New Mexico in prairie dogs from Catron County in 1938; plague has been detected in wild rodents and their fleas as far east as Central Kansas and Dallas, Texas.

D. Human plague in New Mexico — The first three cases were reported in 1949; the total through 2007 is 255, of which 33 (13%) were fatal. Since 1970, slightly more than half of U.S. cases have been reported from New Mexico. Most of the remaining cases come from Arizona, Colorado and California. Although plague in wild animals or their fleas has been found in every New Mexico county except one (Hidalgo), 207 of 255 human cases (81%) have occurred in seven northern N.M. counties (Bernalillo, McKinley, Rio Arriba, San Miguel, Sandoval, Santa Fe and Taos).

III. THE DISEASE

A. Agent — Plague is caused by a bacterium called *Yersinia pestis*, a gram-negative coccobacillus shaped somewhat like a safety pin with bipolar-staining nuclei. *Y. pestis* is killed by heat and desiccation, but can survive for up to 7 years in frozen tissues.

B. Host — several species of rodents can serve as hosts of the plague bacterium. Rodents vary widely in their susceptibility. Highly susceptible species are called epizootic hosts and include *Rattus* and members of the squirrel family. The maintenance of plague during interepizootic periods is not well understood, and may involve more resistant species (enzootic hosts) such as voles (*Microtus*) and mice (*Peromyscus*).

C. Vector — the Oriental rat flea, *Xenopsylla cheopis*, is the most important plague vector historically. Plague-infected rodents become bacteremic (bacteria are present in the blood) and serve as sources of infection for feeding fleas. When a flea ingests a blood meal containing plague bacteria, the bacteria begin to grow in the flea's gut and eventually block the proventriculus (throat valve). A blocked flea cannot swallow any more blood and will become very aggressive, attempting to feed repeatedly from any available host. In so doing, it regurgitates plague bacteria into the animal (or person) it is feeding upon, thus transmitting the disease.

In New Mexico, of approximately 105 species of fleas, 33 have been found plague-infected. This does not mean they are all equally capable of transmitting the disease. The rock squirrel flea, *Oropsylla montana*, is the most important plague vector in New Mexico in terms of transmission to humans. The common dog and cat fleas do not transmit plague; however, dogs and cats can transport rodent fleas into the home environment.

D. Symptoms in humans — Plague is an acute febrile illness that causes high mortality unless promptly diagnosed and treated. Common symptoms of plague occurring within 2 to 6 days after exposure include high fever, chills, headache, myalgia, weakness, malaise, gastrointestinal symptoms, and often acute lymphadenopathy (buboes). The three predominant clinical forms of plague are: *bubonic*, *septicemic*, and *pneumonic*. In bubonic plague, typically the lymph node nearest the site of infection becomes tender and swollen and is called a bubo. For example, a flea bite on the right leg may result in an inguinal bubo on the right side of the groin. The three most common bubo locations, in descending order, are inguinal, axillary and cervical. Septicemic plague is characterized by the presence and proliferation of *Y. pestis* in the bloodstream.
Septicemic plague is likely to develop in inadequately treated bubonic cases (called secondary septicemic plague), but can also occur without prior lymphadenopathy, making diagnosis more difficult. The most dangerous, and least common, form of the disease is pneumonic plague. This can develop from inadequately treated septicemic plague (secondary pneumonic plague), or occur due to the inhalation of infectious materials (primary pneumonic plague). The latter form is usually acquired as a result of close contact with a coughing person or cat with plague pneumonia. There has not been human-to-human transmission of pneumonic plague in the U.S. since an outbreak in Los Angeles in 1924-1925. However since 1980 there have been five cases of primary pneumonic plague acquired from cats. Most human cases in the U.S. are bubonic (82% of cases from 1970 through 2005).

Untreated bubonic plague is fatal about 50% of the time, whereas untreated septicemic and pneumonic plague is almost always fatal. However, all forms of the disease can be treated with antibiotics if medical help is promptly sought. Effective antibiotics include streptomycin, gentamicin and the tetracyclines. Most fatalities in the U.S. today are a result of a delay in treatment.

E. Transmission to humans — the majority of cases are acquired via flea bite (63% of U.S. cases from 1970 through 2005). Other modes of transmission are by direct contact with infected tissues or blood of rodents, rabbits, carnivores or, very rarely, ungulates; by inhalation of infectious respiratory droplets expelled from a person or cat with pneumonic plague; or by bite or scratch of infected domestic cats.

IV. NATURAL HISTORY OF PLAGUE IN THE SOUTHWEST UNITED STATES

Plague occurs in a cyclical nature for reasons not completely understood, but probably related to favorable weather (above average rainfall and moderate temperatures) which leads to increased food abundance for rodents, thus supporting higher rodent populations and an increased risk for disease. Moderate temperatures and adequate humidity are also necessary for survival of fleas. Certain highly to moderately susceptible rodents and their fleas maintain *Y. pestis* in ongoing rodent-flea-rodent transmission cycles. A high population density among these susceptible rodents increases the rodent-flea transmission cycle and may result in amplification of plague over a wide geographic area. This is called the epizootic cycle of plague. Epizootic hosts in New Mexico include ground squirrels (especially the rock squirrel, *Spermophilus variegatus*), prairie dogs and woodrats.

How plague is maintained between epizootics (enzootic cycle) is not well understood, but may involve moderately resistant enzootic or maintenance hosts. Mice of the genera *Peromyscus* and *Microtus* are considered enzootic plague hosts.

Epizootics among susceptible rodent species, and subsequent human cases, are most common during summer when rodent and flea activity is at its highest. Most human cases also occur during the summertime for the same reason. Winter plague cases do occur; these are more likely to be as a result of direct contact with infected animals, such as skinning a rabbit or carnivore.
Wild and domestic carnivores may become infected by preying on infected rodents or rabbits, or by being bitten by plague-infected fleas. Most carnivores are resistant to plague; they may seroconvert (meaning antibodies to plague have been detected in a blood sample) but they do not exhibit symptoms. However, wild and domestic cats are susceptible and often die of the disease if not treated (see following section on feline plague). Human cases have resulted from skinning or otherwise handling infected bobcats, coyotes, gray fox, badgers, and, rarely, ungulates.

Two areas of the U.S. are known plague foci for human cases. The first is the Southern Rocky Mountains-Four Corners area of Colorado, New Mexico, Arizona, and Utah. Most of the U.S. cases since 1949 have occurred there and most of these were the result of infected flea bites. The second area is the Pacific Coast and Western Great Basin of California, Oregon, and Nevada, where infected fleas cause most of the human cases. Outside of these two areas, most of the cases involve handling of infected animal tissues, whether rodent, rabbit, or carnivore. The exact reason for this is not known, but it may be related to the absence of certain rodent flea vectors in peripheral areas of these foci. In New Mexico, there is a strong association between human plague cases and pinyon-juniper woodland habitat.

Most persons are exposed to plague when rodent epizootics occur near their homes. Risks of peridomestic exposure are especially high in New Mexico, Arizona and Colorado where ongoing suburbanization has resulted in large numbers of people living within active plague foci. Rock squirrels are peridomestic and readily find suitable food and habitat in these new developments, such as woodpiles, gardens and pet food. Rock squirrels are extremely susceptible to plague, and their fleas are efficient vectors of plague to humans and other animals. Rock squirrels have been the sources of infection for almost one-third of U.S. cases since 1970.

V. FELINE PLAGUE AND IMPLICATION FOR HUMAN DISEASE

In 1977, a young boy from Cibola County acquired plague from the bite of his infected cat. The child recovered; during the investigation the cat was found dead and tissue samples tested plague-positive. This was the first confirmed case of feline plague in the state, and the first human case in New Mexico acquired from a cat. Just a few months earlier, the first cat-associated human plague case ever recorded in the U.S. occurred in Arizona. A total of 24 human plague cases in the U.S. (five fatal) were acquired directly from domestic cats between 1977 and 1998. Seven of these were from New Mexico; none was fatal.

In New Mexico, 345 domestic cats have been diagnosed with plague since 1977. This is probably less than the true incidence of the disease, as only pets that are taken to a veterinarian would be diagnosed. Unlike humans, who usually acquire plague from a flea bite, cats usually acquire the disease through eating an infected rodent or rabbit. Epidemiological evidence has also shown that cats which are hunters are much more likely to contract plague than cats which are non-hunters.

Symptoms in cats include fever, lethargy, loss of appetite, and they may develop a discharge from the nose and mouth. The submandibular lymph node is most often affected, although more than one lymph node may become infected. Cats can also develop septicemic infection without bubo formation. Bubonic or septicemic plague in cats may progress to the lungs to produce pneumonic
The cat may then expel plague bacteria in respiratory droplets when it coughs or sneezes. A person thus exposed to the coughing cat could acquire primary pneumonic plague. Veterinarians, veterinary assistants and cat owners are most at risk. Of the 23 cat-associated human plague cases in the U.S., five developed primary pneumonic plague. This is particularly significant in light of the fact that there has been no human-to-human transmission of pneumonic plague in the U.S. since 1925.

Besides pneumonic plague, cats can be a source of plague infection to their owners or veterinarians through biting, scratching, or through direct contact with lesions, abscesses, oozing buboes, saliva, or other infectious fluids or tissues. Cats can also carry plague-infected rodent fleas into the home. Cats can be treated with antibiotics if the disease is detected in time.

Domestic dogs are generally more resistant to plague infection than cats, but may develop symptoms similar to those seen in cats if they have had contact with an infected rodent.

VI. PLAGUE SURVEILLANCE

Suspect and confirmed plague cases are required to be immediately reported to the New Mexico Department of Health, Epidemiology and Response Division, so that appropriate diagnostic tests can be ordered and effective treatment begun. Human or feline plague cases are immediately investigated to assess the risk to other people, either from human-to-human or cat-to-human transmission, and the environmental risk. Decisions on prophylactic treatment of contacts of sick humans or pets are made on a case-by-case basis. Case patients are interviewed to determine their whereabouts during the probable incubation period (typically two to six days). Surveillance personnel can then begin environmental investigations at likely exposure sites to determine probable sources of infection and assess the potential risks for other persons living in the area.

A. Rodent Surveys — Rodents are live-trapped, identified, and anesthetized. Their fleas are removed and collected for lab analysis, rodent blood samples and tissue samples are taken, and sex and reproductive status are determined. Dead rodents that are found by the public, or during the course of an investigation or routine survey, can also provide valuable information. Liver and spleen samples are taken from dead rodents, or, if these are too decomposed, the femur can be collected. These can be analyzed by either the Plague Section of the Centers for Disease Control and Prevention in Fort Collins, Colorado (CDC) or the state lab (Scientific Laboratory Division – SLD) in Albuquerque.

B. Flea Surveys — Rodent fleas can also be collected from burrows by using a flannel flag on a 9-ft. sewer snake. The flag-bound fleas are anaesthetized and placed in saline solution in vials. Rodent and flea samples are sent to the CDC in Fort Collins for identification and analysis. Results of rodent and flea surveys indicate status of plague and potential risk to humans.
VII. PREVENTION AND CONTROL

Human plague risks can be reduced by:

- Rodent-proofing homes
- Removing structures that provide shelter for rodents
- Limiting rodent access to water and food sources near homes
- Avoiding sick or dead animals and rodents nests or burrows
- Using insect repellents
- Preventing pets from hunting
- Using caution when handling sick cats (which should be examined and treated by a veterinarian)
- Treating pet dogs and cats with effective flea repellents to prevent transport of infected fleas into the home.

Control of wild rodent populations on a large scale is not feasible or ecologically desirable, but some control or reduction in local populations can be achieved in time through elimination of rodent food sources and shelter around human habitation. In the event that rodenticides are used, they should not be applied until an aggressive flea control campaign has been implemented. Killing rodent hosts without first eliminating fleas is likely to increase the risk of a human plague case.

In the event that large numbers of fleas are obtained from rodent burrows or epizootic hosts, control of the possibly plague-infected vectors is achieved by dusting the burrows with either 5% Sevin (carbaryl) or a synthetic pyrethroid called Pyraperm 455 dust, which is a mixture of pyrethrin and permethrin. A hand-powered duster is used to blow Sevin into burrows, but a newly-developed compressed-air duster is best for applying permethrin to burrows. Sevin provides 2-4 weeks of flea control in the best of circumstances, but the chemical deteriorates within 1-2 days if exposed to sun and rain. Permethrin has a much longer residual action of 10-12 weeks and is applied in much smaller amounts (about 1 - 2 ounces per burrow). Pyraperm 455 is no longer being manufactured, but deltamethrin (Deltadust) has been used effectively in rodent burrows in the state of California.
Human Plague by Year
New Mexico, 1949-2007

Number of Cases
N = 255

Human Plague by Onset Month
New Mexico, 1949-2007

N = 255
Feline Plague by Year
New Mexico, 1977-2007

Number of Cases
N = 345

Feline Plague by Onset Month
New Mexico, 1977-2007

Number of Cases
N = 342 *

* 3 cases onset date unknown
Human Plague in New Mexico
Cases by County 1949 - 2007

Feline Plague in New Mexico
Cases by County 1977 - 2007