

OREGON PUBLIC HEALTH DIVISION • OREGON HEALTH AUTHORITY

OF RODENTS, FLEAS, CATS AND MEN: A TALE OF *YERSINIA PESTIS*

For centuries, human plague has terrified humankind by its mysterious contagiousness, virulence and swift fatality. During the Black Death of London of 1348–1350, one-third to one-half of the populace perished. The city's dead overflowed its burial capacity: in the East Smithfield cemetery — especially prepared in advance for the encroaching wave of plague victims from the Continent — bodies were stacked five deep and children's bodies were placed in the small spaces between adults.¹ No one was spared: rich and poor were equally stricken.

In seven months during the 1665 "Great" (and last) Plague of London, 100,000 succumbed.¹ Popular lore maintained that the plague was spread by dogs and cats. In response, the Lord Mayor of London called for their mass extermination. Unbeknownst to him, the plague was harbored in rats and transmitted to humans by their fleas. Once the delicate balance between predators and prey was disrupted, the rat population exploded with unfettered restraint.²

This issue of the *CD Summary* details two recent plague cases and reviews the epidemiology of plague in Oregon.

CAT FEVER OF ANOTHER SORT

In June, a Prineville family's cat went missing; three days later, it returned emaciated, with its neck swollen by enlarged lymph nodes and a rodent carcass hanging from its mouth. While trying to dislodge the dead rodent from the cat's mouth, two adults were bitten on the finger. Out of compassion, the cat was put out of its misery and buried. Two days later, one of the adults (Case 1) developed fever and malaise. He was evaluated and found to have an enlarged axillary lymph node ipsilateral to the cat bite. Azithromycin was prescribed. Four days later, Case 1 returned for evaluation for worsening symptoms despite antibiotics. He was urgently hospitalized for sepsis and respiratory failure, with a primary

diagnosis of suspected plague; treatment with gentamicin was begun. Less than 12 hours later, Gram-negative bacilli were noted in blood culture. *Yersinia pestis* was confirmed 36 hours later. Case 1 suffered multi-organ failure and acral gangrene but survived.

The second adult (Case 2) presented four days after the bite with less severe malaise and chills, and an axillary bubo ipsilateral to the cat bite. Case 2 was also treated with azithromycin for cat bite-associated lymphadenitis. After the diagnosis of Case 1 was entertained, Case 2 was administered doxycycline and thereafter improved rapidly.

INVESTIGATION

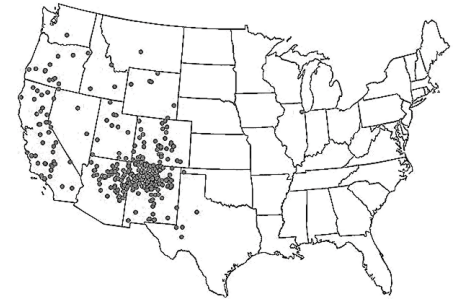
The Crook County Health Department (CCHD), Oregon Public Health Division and Oregon Department of Fish and Wildlife collaborated in the investigation of these cases. The team interviewed neighbors and close contacts of the two cases regarding their exposures, and looked for evidence of epizootic rodent die-off. They conducted a serosurvey of anti-*Y. pestis* antibody among ten cats and five dogs at the local humane society and three neighborhood dogs. The CCHD nurse exhumed and sent the dead cat to CDC's Division of Vector-Borne Diseases, Fort Collins, Colorado, for testing.

The cat that had bitten the affected cases tested positive for *Y. pestis* by culture. Sera from the 18 animals were negative for antibody to *Y. pestis*. Convalescent serum from Case 2 was positive for antibodies to *Y. pestis*.

U.S. AND OREGON: THE NUMBERS

In the U.S., most plague cases occur in the arid regions of the west (Figure 1). Between 1900 and 2010, 999 confirmed or probable human plague cases were reported in the U.S. More than 80% of these cases have been bubonic. In recent decades, an average of seven (range, 1–17) human cases have been reported annually. Plague's case fatality rate in the U.S. decreased from 66% during the pre-antibiotic era (1900–1941) to 11% during 1990–2010.³

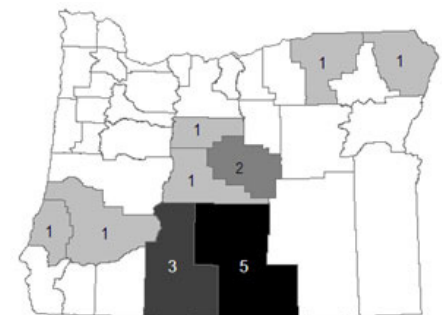
Figure 1. Reported cases of human plague; U.S., 1970-2010*



*1 dot in county of exposure for each plague case

Eighteen cases of plague have been recorded in Oregon since 1934; 17 of those occurred since 1970. Of those with known outcomes, 5 (29%) of 17 died. Most Oregon cases were acquired in the arid central and south-central regions of the state (Figure 2). Thirteen of 17 occurred during June through August (Figure 3, *verso*), as one might expect, when rodents and fleas are more active. Of the 17 cases with known risk factors, 10 had exposures to rodents or fleas. Two cases were rabbit hunters, and four had bites, scratches, or suspected inhalation of infectious exudates from cats.

Figure 2. Plague cases by county of exposure, Oregon, 1970–July 2012 (N=16*)



*Of the 17 Oregon cases, 16 were acquired in Oregon, and 1 was acquired in Wyoming.

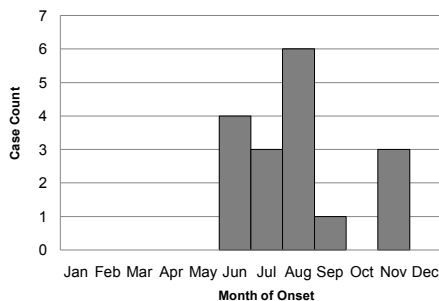
PLAGUE 101

Plague in humans is a life-threatening flea-borne zoonosis caused by the Gram-negative, bipolar-staining bacillus *Yersinia pestis*, discovered by Alexandre Yersin in 1894. Plague is maintained in an enzootic cycle among wild rodents in

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Figure 3. Plague cases by month of onset, Oregon 1970–July 2012 (N=17)



the western United States by fleaborne transmission.⁴ The rock squirrel flea is the primary vector, and the primary hosts are rock squirrels, ground squirrels, prairie dogs, sage rats, wood rats and other rodents that live in burrows or nests. Fleas become infective days or weeks after ingesting blood from a plague-infected rodent.⁴ In 1898, Paul-Louis Simond proved that *Y. pestis* is transmitted by fleas whose midguts (stomach) become obstructed by the replicating bacteria, causing them to regurgitate bacteria into the bite site.

Plague is seasonal because of variations in vector or host activity that affect the likelihood of *Y. pestis* transmission: the risk of flea bites increases markedly during the summer months when epizootic fleaborne transmission among rodents is also at a peak.⁵ During plague epizootics, the disease can spill over to other hosts, including humans. Humans can be infected through a) the bite of an infected flea carried by a rodent (common); b) bites or scratches of infected animals (rare); c) direct contact with contaminated tissues; or d) inhalation of respira-

tory aerosols coughed up by infected persons or animals (very rare).⁶ Bubonic plague, which is characterized by high fever and regional lymphadenitis 1–7 days after the bite of an infected flea or animal, accounts for 80%–90% of cases. Without prompt, effective treatment, infection can quickly spread from the lymph nodes to the blood or lungs, resulting in septicemic plague (10% of cases) or pneumonic plague (<1%).

PNEUMONIC PLAGUE

Pneumonic plague presents the dire potential for person-to-person transmission through respiratory aerosols. There have been but two known instances of such transmission in the United States. In Oakland, California in 1919, a squirrel hunter contracted bubonic plague that progressed to a fatal pneumonia. His case was followed by twelve primary pneumonic plague cases, including two physicians and one nurse, all fatal.⁷ In Los Angeles in 1924, 30 people died following exposure to a secondary case of pneumonic plague. The secondary case presumably acquired the pneumonic form from her father's draining bubo, which had been misdiagnosed as a "venereal bubo."⁸ Rats were the suspected source.

CAT PLAGUE

Cats are highly susceptible to plague and can transmit *Y. pestis* infections to humans. Between 1977 and 1998, 7.7% of human plague cases were associated with cats in eight western states.⁹ Cats who roam freely where *Y. pestis* is enzootic acquire plague by eating bacteremic rodents or through flea bites. Signs and symptoms in cats include fever, lethargy, loss of appetite and subman-

dibular or cervical lymphadenitis or abscess. Infected cats can transmit plague through bites, scratches, or infectious exudates.

CONCLUSION

Regardless of the levels of local epizootic plague activity, a diagnosis of plague should be considered in patients with compatible symptoms and a history of exposure to a sick or dead cat, fleas, or rodents in the western U.S.

FOR MORE INFORMATION

Visit www.cdc.gov/plague

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