

What's Eating You? Oriental Rat Flea (*Xenopsylla cheopis*)

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The oriental rat flea (*Xenopsylla cheopis*) is best known for its ability to spread 2 potentially lethal diseases to humans: plague and endemic (murine) typhus. Plague has caused 3 great pandemics that killed almost a third of the population in Europe.¹ Similar to other fleas, *X cheopis* has a laterally compressed body and large hind legs (Figure). Female fleas have a more rounded body; males have a flatter back, rounded ventral surface, and a prominent retroverted genital apparatus approximately half the length of the entire flea.

Unlike cat and dog fleas, *Xenopsylla* fleas are combless, that is they lack the prominent combs resembling a mustache and mane of hair on cat and dog fleas. Additional identifying features for *X cheopis* include a rounded frons (forehead). The flea that is closest in appearance is *Pulex irritans* (the human flea). Unlike *P irritans*, *X cheopis* has several setae (hairs) posterior to the antennae as well as a pleural rod visible within the mesopleuron above the second pair of legs. In contrast, *Pulex* fleas lack the pleural rod and have only 1 hair on the postantennal head.

Disease Vector

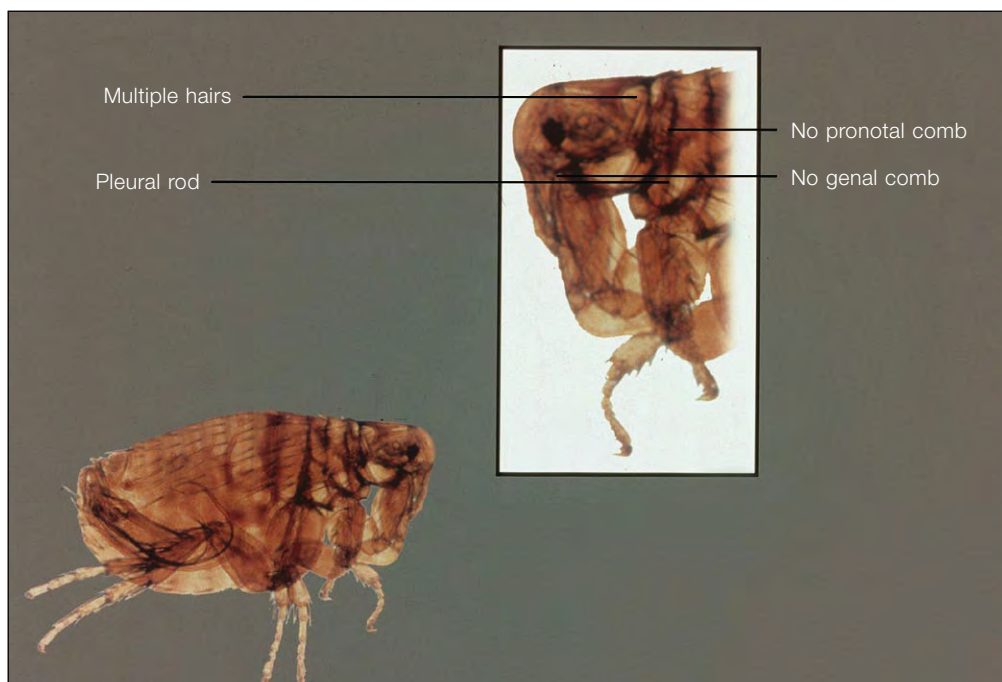
Xenopsylla cheopis serves as an important arthropod vector for *Yersinia pestis* and *Rickettsia typhi*, the organisms that cause the plague and endemic (murine) typhus, respectively. Additionally, the flea has been shown to harbor *Rickettsia felis* (cat flea rickettsiosis)² and 2 species of *Bartonella*: *Bartonella tribocorum* and *Bartonella vinsonii* subsp *vinsonii*.³ It should be noted that the cat flea is now the most

important vector for endemic (murine) typhus in South Texas. The cat flea has been shown to be a competent vector but has low efficiency and a decreased bacterial load when compared with the rat flea.⁴ Although *X cheopis* remains the most important plague vector in endemic zoonotic disease, *P irritans* and pulmonary transmission of pneumonic plague were more important means of spread during the great pandemics.

The method of acquiring the 2 bacteria (*R typhi* and *Y pestis*) is the same: the flea takes a blood meal from an infected mammal such as a rat, mouse, or opossum, and then transmits the organism through a bite or defecation. The consumed bacteria must survive long enough in the flea's gut to be transmitted. *Yersinia pestis* survives by forming cohesive aggregates that protect the bacteria from being lysed, allowing for further propagation. The aggregate of bacteria can become so large that it can block the flea's midgut, resulting in starvation.^{5,6} The source of the blood (eg, rat, mouse, opossum) impacts the flea's ability to maintain *Y pestis* in an adequate bacterial load. The rat is the most infectious source of blood, possibly due to nutrient content, passage time, and decreased complement content in the blood.⁷ *Rickettsia typhi*, an obligate intracellular organism, survives by entering the flea's midgut epithelial cells. Within 3 to 5 days, the entire midgut epithelium is infected.⁸ Despite the potential for starvation with massive propagation of *Y pestis*, the flea's overall fitness usually is not affected by the presence of either of the bacteria, which is a marked difference from the ancestor of *Y pestis*, *Yersinia pseudotuberculosis*, whose toxins detrimentally affect the flea, inflicting diarrhea and immobility.⁹ Temperature-regulated gene expression plays a role in the host-vector relationship. Certain key genes in each species of bacteria are turned on or off due to the relatively low body temperature of the flea. If the bacteria are introduced to a warmer environment,

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*Xenopsylla cheopis*.

such as a human, the gene expression changes and infection ensues.^{10,11}

Humans become a target when the infected flea is in search of a blood meal, often due to the death of its prior host. When rat infestations are treated through extermination, there may be a transient increase in bites, as the fleas look for alternative hosts. The bacteria can be transmitted in several ways. Small pieces of the *Y pestis* bacterial aggregate, which have blocked the flea's midgut, can be regurgitated during feeding. Alternatively, *Y pestis* can enter the skin through infected mouthparts or flea feces.¹² Unblocked fleas have been shown to be as efficient as blocked fleas at transmitting *Y pestis* and are able to infect another mammal only 1 day after acquiring the bacteria themselves.¹³ Without a blocked gut, fleas can survive longer, allowing for more opportunity to spread the bacteria.¹⁴ *Rickettsia typhi* is mainly transmitted to humans when the flea feeds and its infected feces come in contact with the bite site. The feces are inoculated with bacteria that have been released from the gut's epithelial cells into the lumen via binary fission.¹⁵

Plague—After *X cheopis* transmits *Y pestis* to the human, signs and symptoms of bubonic plague typically begin after a 2- to 6-day incubation period, including fever, painful lymphadenitis, and buboes (fluctuant necrotic lymph nodes). If left untreated, the disease can enter the bloodstream, leading to shock, disseminated intravascular coagulation, and death. Patients with septicemic plague may present with large ecchymoses similar to meningococcemia

as well as gangrene of acral areas including the nose and digits, which prompted the historical nickname "black death."¹⁶ The disease persists at a low-level enzootic state in the western United States. From 1947-1996, 390 cases of endemic plague were reported in the United States with the majority of cases occurring in New Mexico, Arizona, Colorado, and California.¹⁷ Because of the severity of the disease and its ability to be weaponized, *Y pestis* is considered a potential agent of bioterrorism. The bacteria could be spread by the mass release of fleas but is more likely to be released as aerosolized droplets leading to pneumonic plague. In the latter case, lymphadenitis and buboes would not be present.

Endemic (Murine) Typhus—*Rickettsia typhi* is manifested as endemic (murine) typhus, with the majority of US cases reported in Texas and southern California. After an incubation period of 7 to 14 days, the most common symptoms include fever, headache, rash, arthralgia, and gastrointestinal tract and respiratory symptoms. Thus the presentation can mimic and often be confused with more common viral illnesses. The rash has been reported in only 54% of patients, varying greatly in presentation but most commonly presenting on the trunk; it is nonpruritic and is usually macular or morbilliform in appearance.^{18,19} Severe manifestations may occur in patients who have received a sulfa drug during the course of their illness. These patients may present with multiorgan failure, coma, and stellate purpuric infarcts.¹⁸

Prevention and Treatment

Keeping rats and other mammals that potentially harbor infected fleas away from humans can decrease the incidence of disease transmission. It also has been suggested to use pesticides to exterminate fleas. However, fleas may develop resistance and tolerance to commonly used pesticides, such as DDT, malathion, and permethrin, which may limit the efficacy of these products.²⁰

If either of these infections is suspected, treatment should be immediately started, even before diagnosis is confirmed, to avoid further complications. Bubonic plague can be treated with streptomycin. Alternatively, gentamicin has been suggested as a treatment option. Tetracycline and doxycycline can be used as prophylaxis.¹⁸ Endemic (murine) typhus can be treated with either tetracycline or doxycycline, which have been shown to decrease the length of the febrile illness.¹⁹

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