What's Eating You? Cat Flea (*Ctenocephalides felis*), Part 1: Clinical Features and Role as a Disease Vector

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The cat flea, *Ctenocephalides felis*, is a common ectoparasite around the world. It resides on both domestic and feral animals and plays an important role in spreading several infectious agents, including *Rickettsia felis* and *Bartonella henselae*. Although *C felis* is a relatively poor vector for plague, it does have the potential to carry *Yersinia pestis* and play a role in transmission of the disease. This review will discuss the life cycle and distinguishing features of the cat flea as well as the most important pathogens spread by the flea.

Overview

There are 4 subspecies of *C felis*. While *C felis felis* is truly found worldwide, *C felis strongylus* and *C felis damarensis* are more commonly found in Africa, and *C felis orientis* is common in Southeast Asia and the East Indies. *Ctenocephalides felis felis* is the species most commonly found on both dogs and cats in most parts of the United States. The flea also has been reported on calves, goats, sheep, and other domestic animals.[1-4] A survey of several zoos in South Carolina identified *C felis* in both captive and free-roaming animals within the zoo.[5] Wild animals, such as opossum, deer, and raccoon, act as natural reservoirs for fleas. These wild animals often are sources of reinfestation for companion animals.[6-8]

Similar to the dog flea (*Ctenocephalides canis*), the cat flea has combs (ctenidia) that give the genus its name. Both types of *Ctenocephalides* fleas have a pronotal comb that resembles a mane of hair and a genal comb that resembles a mustache (Figure 1). The head of the dog flea is more round and there are 8 hair-bearing notches on the dorsal hind tibia while the cat flea has 6 hair-bearing notches (Figure 2).

Flea bites present as intensely pruritic, excoriated papules or vesicles usually situated on the lower legs. They also may occur on the hands after petting or handling an animal. Large bullae may occur and may mimic pemphigoid with sheets of eosinophils within a subepidermal bulla. Clues to diagnosing flea bites include the distribution as well as the presence of focal overlying spongiosis and an underlying wedge-shaped polymorphous perivascular infiltrate with both lymphocytes and eosinophils. The bite itself causes little reaction; the immune response to the bite produces the clinical lesion and varies substantially from one individual to another. Bizarre exaggerated responses should raise suspicion for an underlying hematopoietic neoplasm. Bite reactions commonly are treated with topical corticosteroids, though severe reactions may require intralesional or systemic administration. Topical antipruritic agents with camphor and menthol or pramoxine hydrochloride may be helpful for symptomatic relief.

Moreover, fleas play a role in the pathogenesis of allergic dermatitis and anemia in dogs and cats and act as vectors for various infectious microorganisms. Most of the pathogens that fleas carry can cause diseases with dermatologic manifestations.[4,9-11] Among these, *R felis* and *B henselae* are most notable.[12,13] In regions where the cat flea is particularly common in areas of human habitation and where the plague remains endemic, the cat flea has the potential to become a substantial vector for *Y pestis*.[14]

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Figure 1. The cat flea *(Ctenocephalides felis)* has combs (ctenidia) that give the genus its name, a pronotal comb that resembles a mane of hair, and a genal comb that resembles a mustache.
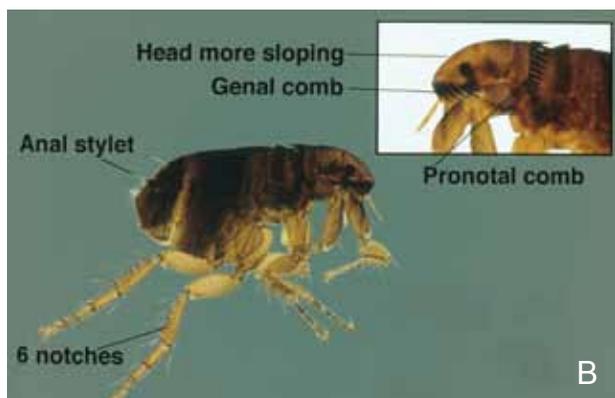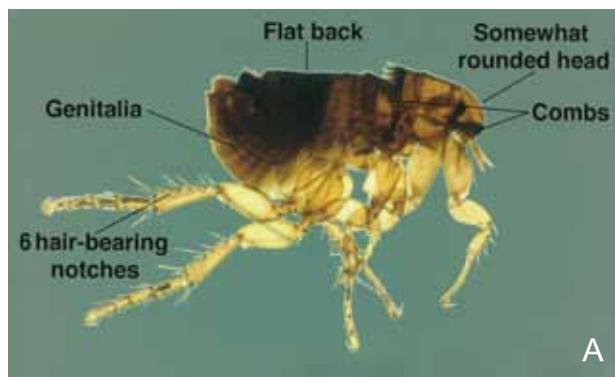
Figure 2. Male (A) and female (B) cat fleas *(Ctenocephalides felis)* have 6 hair-bearing notches on the dorsal hind tibia.

The Flea as a Disease Vector

Rickettsia felis—Rickettsia felis, an agent of endemic typhus, has a worldwide distribution that mirrors the cat flea (Table). The *R felis* genes, gltA and ompA, can be detected through standard polymerase chain reaction (PCR) assays that have been used to screen for the organism in various geographic locations.[15] The presence of *R felis* also can be confirmed through serology studies and more effectively through quantitative real-time PCR assays.[24] *Rickettsia felis* can be found in epithelial cells of the midgut, ovaries, salivary glands, and muscles of fleas. The transmission of *R felis* among cat fleas is maintained both transstadially (passage from one developmental stage to another) and transovarially (passage from adult to ovum).[25,26] *Rickettsia felis* was previously classified in the typhus group because of antigenic similarity to *Rickettsia typhi*. However, there are data to suggest that *R felis* belongs to the spotted fever group, as it encodes the rompA gene, a gene present only in the spotted fever group.[27] The infection rate of *R felis* in cat fleas is varied among the infested host animals and location. In a survey of 5 areas in the state of São Paulo, Brazil, the infection rate of *R felis* in cat fleas was 33.3% to 72.7% among opossum, 13.5% to 52.7% among dogs, and 29.4% to 83.3% among cats.[8] In the United States, PCR assays of 92 fleas from cats in Maryland, Alabama, and Texas showed that 67.4% of the fleas assayed were positive for *R felis*.[17] Human infection with *R felis* has been reported in the United States, Mexico, Brazil, Spain, Thailand, South Korea, Tunisia, and Laos.[28-36] Clinical symptoms generally are mild and similar to *R typhi*, predominantly fever, malaise, and arthralgia. Accompanying these symptoms are dermatologic signs such as a pruritic papular rash on the lower extremities, abdomen, and chest.[35] The patient also can develop central nervous system dysfunction presenting with photophobia and hearing defect. As symptoms often are quite mild, the actual rate of *R felis* infection may be underestimated. Severe illness with multiple organ failure may occur in patients who receive sulfonamides while infected with *R felis*. These patients frequently present with widespread angular hemorrhagic infarcts.

The transmission of this agent often is due to a flea bite or close contact with infested animals. Although PCR can provide a definitive diagnosis, the study can be expensive and the technology is not available in many areas in the developing world. Alternatively, *R felis* also can be detected through serology studies by using *R felis* culture in cell lines.[33]

Rickettsia felis is resistant to erythromycin, gentamycin, amoxicillin, and trimethoprim-sulfamethoxazole. However, doxycycline, rifampin, thiamphenicol, and fluoroquinolones may be effective.[35,37]

Rickettsia typhi—The rat flea, *Xenopsylla cheopis*, is the main vector of *R typhi*, the classic cause of murine typhus. *Ctenocephalides felis* has been shown to act as a vector for *R typhi*. The typical reservoirs for *R typhi* include roof and Norway rats (*Rattus rattus* and *Rattus norvegicus*), but a survey has shown

Prevalence of *Rickettsia felis* Infection in Cat Fleas (*Ctenocephalides felis*) in Several Surveys Around the World

Location	Detection of *R felis* in *C felis,* n (%)
Germany[15]	
Dogs	36/146 (25)
Cats	24/164 (15)
United Kingdom[16]	
Dogs	4/31 (13)
Cats	21/90 (23)
United States[17]	
Cats	62/92 (67)
Uruguay[18]	
Dogs and cats	25/66 (38)
Brazil[8]	
Dogs	278/278 (100)
Cats	59/66 (89)
Spain[19]	
Dogs and cats	22/88 (25)
Taiwan[20]	
Dogs and cats	13/69 (19)
Canada[21]	
Cats	9/50 (18)
France[22]	
Cats	25/309 (8)
Australia[23]	
Dogs	42/116 (36)
Cats	14/43 (33)

that cats and opossum also play an important role as *R typhi* reservoirs in suburban areas, as they do for *R felis*.[38] Clinical cases of human infection have been documented around the world. In the United States, murine typhus has a geographic propensity for Texas and California.[38-41]

Rickettsia typhi is an intracellular, gram-negative, obligate bacterium that requires blood-feeding arthropods and mammals to continue its life cycle. Transmission of bacteria from fleas to humans can occur at the site of a flea bite where feces are deposited while feeding. The cat flea usually feeds on domestic animals, but as an indiscriminate feeder, it will switch to feed on humans when necessary. Patients with murine typhus often present with fever, headache, rash, arthralgia, nonproductive cough, chill, abdominal pain, and malaise.[41,42] A rash is present in 20% to 80% of patients and may be macular or morbilliform. It typically starts at the trunk and then spreads to the extremities. Although sparing of the palms and soles is typical, involvement of these sites has been reported.[41,43] The rash occurs approximately 1 week after the onset of fever and subsides in 1 to 4 days. Patients can develop complications such as pleural effusion, acute renal failure, pulmonary infiltrate, and infarction of the large vessel of the spleen.[42,43] Laboratory abnormalities include anemia, leukopenia, and elevated liver enzymes. The disease can be diagnosed through PCR and indirect immunofluorescence assay, which is considered the gold standard. Doxycycline remains the drug of choice and is superior to either ciprofloxacin hydrochloride or chloramphenicol.[42]

Yersinia pestis—In the past, researchers had concluded that *C felis* was not competent as a vector of *Y pestis*; however, more recent findings have challenged this idea.[14] Epidemiologic evidence in Uganda suggests that the cat flea is capable of transmitting the disease. However, the number of fleas required to maintain *Y pestis* person-to-person transmission was estimated to be 25 fleas per person instead of 4.7 to 7.8 fleas per person for the rat flea, *X cheopis*. Although this study indicates that the transmission potential of plague by *C felis* is low, the high number of fleas concentrated in this region can compensate for the inefficiency of transmission.[14]

Plague is one of the most serious infectious diseases caused by a gram-negative coccobacillus. If not treated appropriately, bubonic plague can lead to septicemia, shock, and multiple organ failure. Typically, transmission of the disease to humans requires close contact with infected animals, eating infected animals, being bitten by infected fleas, or human-to-human transmission (in the case of pneumonic plague).[44,45] The disease is endemic at low levels in the western United States. In recent years, it has declined in incidence in Asia and reemerged in Africa. The reasons for this

shift include the living conditions of rural areas with residents living near rodent-infested locations, the lack of disease awareness, and the inadequacy of the preventive medicine infrastructure in Africa.[46]

In humans, there are 3 forms of plague: bubonic, pneumonic, and septicemic. Bubonic plague is still the most common type reported worldwide.[47] In this form, the disease is transmitted to humans through flea bites. Patients have nonspecific symptoms such as fever, chill, headache, myalgia, arthralgia, prostration, and headache. Following these symptoms, patients develop a very tender bubo, especially at the lymph nodes near the inoculation site, usually in the inguinal area.[47,48] The surrounding areas close to the bubo may exhibit erythema, warmth, and induration, which may progress to sepsis if left untreated.[48] Pneumonic plague is more rapidly progressive, has a high mortality rate compared to the other forms of plague, and is transmitted from person to person through droplets. Typical symptoms include cough, tachypnea, and sputum production. In severe cases, patients experience cardiopulmonary insufficiency and circulatory collapse. Patients with septicemic plague usually have prominent clinical symptoms of gastrointestinal tract infection including nausea, vomiting, diarrhea, and abdominal pain. Disseminated intravascular coagulation and adult respiratory distress syndrome also occur in septicemic plague. The diagnosis can be made with culture from body fluid or tissue, a hemagglutination test to detect F1 antigen, PCR, enzyme-linked immunosorbent assay, or immunochromatographic handheld assays.[48] Streptomycin remains the drug of choice. Tetracyclines and sulfonamides also are active against the organism. When those drugs are not available, gentamycin and fluoroquinolones can be used as alternatives. The reemergence of *Y pestis*, its ability to cause a rapidly fatal disease, and potential use as a biological weapon warrant increased public awareness.

Bartonella henselae—*Bartonella henselae* also appears to be transmitted by *C felis*. This organism has been implicated as the cause of cat-scratch disease (CSD). Documented carriage of *B henselae* by *C felis* had been reported in Spain, the United States, Germany, and France.[12,19,49] *Bartonella henselae* has been detected in *C felis* infesting Virginia opossum. This finding raises a concern that *C felis* from opossum can potentially act as the vector to transmit *B henselae* to pets because opossum are known to approach residential areas in search of food.[12,39] In this situation, humans are at risk for becoming infected with *B henselae* through close contact with their pets.

Bartonella henselae is a gram-negative, facultative, intracellular bacterium that has a propensity to affect red blood cells and endothelial cells. This bacterial agent is transmitted to cats through the inoculation of infected flea feces on an open wound.[13,50] Cats can spread *B henselae* infection to humans through scratches, bites, and licks on open injured skin. Because infected individuals may not show any symptoms, bacteria may be transmitted through blood transfusion if the blood product does not undergo smear or serologic studies for *B henselae*.[51] The presence of *B henselae* also can be detected by PCR and enriched blood agar culture.[12] Histology of an affected lymph node showing a stellate abscess with surrounding histiocytes may suggest the diagnosis.

The clinical presentation of CSD includes the initial development of evanescent papular, pustular, or vesicular lesions after 2 to 3 weeks of infection followed by the appearance of lymphadenopathy. The development of lymphadenopathy may be accompanied by symptoms such as fever, malaise, fatigue, headache, anorexia, weight loss, and vomiting.

Neurologic symptoms, such as signs of encephalopathy and complex partial seizures, also have been seen in patients with CSD.[52] Atypical presentations such as Parinaud oculoglandular syndrome can occur. Patients present with unilateral conjunctivitis with serous discharge and local lymphadenopathy. This clinical sign is caused by direct hand-to-eye contact. Another ocular manifestation includes neuroretinitis, which results in visual change and retinal detachment.[53] In severe cases, patients may develop hepatosplenomegaly, glomerulonephritis, and pleural effusion. Abscesses may occur in the liver, spleen, bone, or mesenteric lymph nodes. In patients with immunodeficiency, the organism has been associated with bacillary angiomatosis, with the development of vascular papules and nodules that can bleed profusely when injured. The disease also may affect the heart. One study found an association between chronic *B henselae* infection and arrhythmogenic right ventricular cardiomyopathy.[54] Patients with prior valvular defects are at high risk for developing endocarditis and eventually require valve replacement surgery.[55-57]

In typical cases, CSD spontaneously resolves within 4 months. Immunosuppressed patients and those with more complicated disease, bacillary angiomatosis, or peliosis hepatis can be treated with oral erythromycin. In patients who develop myocarditis, azithromycin 500 mg daily has demonstrated efficacy.[54]

Dipylidium caninum—Although rarely an infectious agent in children, *D caninum*, a common intestinal cestode of dogs and cats, can be transmitted via *C felis*. *Ctenocephalides felis* acts as an intermediate host for the tapeworm.[58] Humans who accidentally ingest infected adult *C felis* can develop dipylidiasis. *Dipylidium caninum* can mimic infection with

Enterobius vermicularis (pinworm). One case report described a 2-year-old patient who experienced repeated perianal itch that did not remit after treatment with mebendazole. Worms were found in the patient's stool, resembling "small grains of rice."[59] Clinical features of *D caninum* infection may include epigastric pain, decrease in appetite, diarrhea, and weight loss.[59] Many infections remain asymptomatic. The diagnosis can be made by examining proglottids under a light microscope. Treatment involves a single dose of praziquantel. Niclosamide, which is not available in the United States, can be used as an alternative.[59,60]

Conclusion

Cat fleas have the potential to transmit plague and other infectious diseases. Because of their close association with household pets, they are important vectors of human disease.

This article is the first of a 2-part series. The second part providing a discussion of measures for flea control will appear in a future issue of Cutis®.

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