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Rickettsia felis, *Bartonella henselae,* and *B. clarridgeiae,* New Zealand

To the Editor: The cat flea (*Ctenocephalides felis felis* Bouché, 1935) is a ubiquitous parasite of domestic and wild animals that also feeds readily on people. Recent studies have implicated the cat flea as a vector of new and emerging infectious diseases (1). To determine the pathogens in *C. felis* in New Zealand, we collected 3 cat fleas from each of 11 dogs and 21 cats at the Massey University Veterinary Teaching Hospital from May to June 2003. The fleas were stored in 95% alcohol until they were identified by using morphologic criteria and washed in sterile phosphate-buffered saline. The DNA from each flea was extracted individually by using the QiaAmp Tissue Kit (QIAGEN Ltd., Hilden, Germany), according to the manufacturer's instructions. When polymerase chain reaction (PCR) was performed with primers for *glt*A and *rOmp*A as described (2), products were obtained with DNA from 15 (15%) of the fleas. The sequences of the products were identical to those of *Rickettsia felis* (GenBank AF191026) with infected fleas taken from both dogs (3/11; 27%) and cats (7/21; 33%). When PCR was performed with primers for the 16S-23S rDNA interspacer region as described (3), products were obtained with DNA of four fleas. The sequences of the products from three fleas (from two cats) were identical to that of *Bartonella henselae* (GenBank AF312495), and the sequence of the product of one flea (from a cat) was identical to that of *B. clarridgeiae* (GenBank AF167989).

Our study is the first to identify *R. felis* in Oceania. The organism is a recently described human pathogen, and infections with this spotted fever group rickettsia have already been reported in 11 persons: 4 persons in the United States, 2 persons in Brazil, 4 persons in Europe, and 1 person in Thailand. The symptoms of the patients were nonspecific and included fever, headache, and rash. Diagnoses were made by sequencing products obtained by PCR with primers for the 17-kDa protein (4), citrate synthase (4), and PS 120 protein (5) genes. *R. felis* has been established in tissue culture (XTC-2 and Vero cells) (6), and serologic testing has been used to diagnose infections (5). Reports indicate that patients respond rapidly to doxycycline therapy (5), and in vitro studies have shown the organism is susceptible to rifampin, thiamphenicol, and fluoroquinolones.

B. henselae is an agent of cat-scratch disease, bacillary angiomatosis, bacillary peliosis, endocarditis, bacteremia, and various neurologic and ocular conditions. Cats are the reservoir hosts, and contact with cats and their fleas is an established risk factor for most infections. Although *B. henselae* has been isolated from 17% of domestic cats in New Zealand (7), only two human infections have been reported in the country; neuroretinitis was diagnosed in both patients (8). In neighboring Australia, however, cat-scratch disease, bacillary angiomatosis, and endocarditis have been diagnosed in numerous patients. Cats are also the reservoir hosts of *B. clarridgeiae* which has been implicated as an agent of cat-scratch disease in humans and aortic valve endocarditis and hepatic disease in dogs (9). The organism has been found in cat fleas (as great as 17%) in Europe (1), and although we found only one flea infected with *B. clarridgeiae* in New Zealand, this description is the first of the organism in Oceania. However, *B. clarridgeiae* has been found in domestic cats in nearby Indonesia and the Philippines (10).

Our findings add to the accumulating data on *R. felis, B. henselae,* and *B. clarridgeiae* and should alert medical workers in New Zealand, a common tourist destination, to the possibility that their patients may be infected with these organisms.

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Enterohemorrhagic *Escherichia coli* O157, Kinshasa

To the Editor: During the rainy season, from April to September 2003, 463 children ≤15 years of age (median 10 months) with severe diarrhea were admitted to the Pediatric Hospital of Kalembelembe in Kinshasa, the capital of the Democratic Republic of Congo. The population of the outbreak area was approximately one million.

Several children with bloody diarrhea without fever were treated. They came from six districts of Kinshasa (Bumbu, Selembao, Makala, Kimbanseke, Masina, and Ndjili). Abdominal cramps, nausea, vomiting, and dehydration were uncommon. The duration of illness ranged from 5 days to 2 weeks. Available antiparasitic drugs, trimethoprim-sulfamethoxazole, and ampicillin showed no effect against the illness. Fifty-six infants died between June and July. Symptoms of hemolytic uremic syndrome developed in most of them.

Stool samples from 32 patients were screened for parasites, enteropathogenic bacteria, rotavirus, and adenovirus. Three samples were positive for rotavirus. In contrast, all stool cultures were positive for *Escherichia coli* which always grew as pure cultures on purple bromocresol agar, a nonselective medium containing lactose. The *E. coli* isolates appeared sorbitol negative when tested on MacConkey sorbitol; they were agglutinated by O157 and H7 antisera (Difco Laboratories, Detroit, MI) and lacked expression of β-glucuronidase. All *E. coli* isolates were sent to the Pasteur Institute in Bangui, Central African Republic, for further characterization. Polymerase chain reaction allowed detection of Shiga-like toxin *slt-1* and *slt-2* genes (1,2) in isolates from all patients. The Vero cell assay phenotypically confirmed cytotoxicity of these isolates, with most of them being seroneutralized by rabbit antisera against Shiga toxin (3). Thus, all *E. coli* isolates responded to the definition of enterohemorrhagic *E. coli*.

Before 2003, sporadic infections or outbreaks caused by enterohemorrhagic *E. coli* were not reported as a cause of bloody diarrhea in the Democratic Republic of Congo. A case-control study could not be performed because of political unrest in Kinshasa. Although reported outbreaks of *E. coli* O157 in sub-Saharan Africa have been few to date, available information indicates that the pathogen has wide geographic distribution. *E. coli* O157–related diarrhea outbreaks that occurred before 2003 have been reported in South Africa, Swaziland (4), and Malawi (5) in 1992; Central African Republic (6) and Kenya (7) in 1996; Cameroon in 1998 (8); and Nigeria (9) and Ivory Coast (10) in 2000. In the Central African Republic and in Zémio, a small village located on the Democratic Republic of Congo border, outbreaks of bloody diarrhea in 1996 were attributed to *E. coli* O157 from molecular test results (6).

Since 2001, an increasing number of cases of acute bloody diarrhea have been reported in Kinshasa between June and August. During this 2003 outbreak, an investigation could not be conducted; possible routes of transmission would include person-to-person contact related to lack of hygiene, and contaminated food and water.

In 1996 in the Central African Republic and in 1998 in Cameroon, the major contributing factors of the *E. coli* O157 outbreak were consumption of smoked zebu meat and contaminated drinking water. Studies of *E. coli* O157 carriage rates among livestock, food, and environment in this central African area might be useful in assessing the potential for future outbreaks.