Bartonella henselae Antibodies after Cat Bite¹

To the Editor: Bartonella henselae is the causative agent of cat-scratch disease, which is the most common form of human bartonellosis (1). In immunocompromised patients, e.g., HIV-infected patients, B. henselae can give rise to longstanding fever, bacillary angiomatosis, and peliosis hepatitis (2). Domestic cats are the reservoir for B. henselae, and cat fleas transmit the organism between cats (3). The seroprevalence and culture findings of Bartonella spp. in cats have been shown to be low in Sweden (4,5) compared with warmer areas (6). Catscratch disease is most often spread from cats to humans by scratches, but other forms of transmission, including cat bites, have been suggested (7).

To determine seroprevalence of antibodies against *B. henselae* in Sweden, we used data from a recently published prospective study of patients with infected cat bites (8). In addition to the information about bites, information about cat scratches was collected by retrospective review of the patients' medical records. Serum samples were taken during the patient's first visit to a hospital and at a follow-up visit about 2 weeks later. The study was approved by the local ethics committee.

Immunoglobulin G against specific *Bartonella* spp. was detected by the immunofluorescence antibody test (1). Cell-cultivated antigens were prepared from the following strains: *B*.

henselae Houston-1 (ATCC 49882), B. henselae Marseille (CIP 104756), B. henselae Berlin1 (M. Arvand), B. henselae K68 (E. Olsson-Engvall), B. elizabethae (ATCC 49927), and B. grahamii (ATCC 700132). The cutoff values for a positive immunofluorescence antibody test result were chosen as \geq 128 for B. henselae and B. grahamii and \geq 256 for B. elizabethae. The titers are expressed as the reciprocal of the end-point dilution. For controls, we also analyzed serum from 117 blood donors with these antigens. The χ^2 test was used for statistical analysis.

We analyzed antibodies to *Bartonella* spp. in serum from 71 patients (51 women and 20 men), median age 47 years (range 15–85 years). Only 11 patients had fever. Cat scratches were reported for 17 patients. A single serum sample was obtained from 37 of the 71 patients, and an additional convalescent-phase sample was obtained from 34 patients after a median of 16 days (range 6–54 days).

Antibodies against any B. henselae strain were found for 24/71 (34%) patients, against B. elizabethae for 9/71 (13%), and against B. grahamii for 12/71 (17%). A total of 13/71 (18%) patients showed reactivity to B. henselae only. Antibodies to any Bartonella spp. were found for 28/71 (39%) of the patients. As many as 13/24 (54%) serum samples with antibodies against B. henselae reacted to antigens of only that species. More patients (19/71; 27%) reacted to the antigen from the cat in Sweden, K68, than to other strains. The least common reactivity found in this study was against the B. henselae Marseille strain.

Of the 117 controls, 1 (0.8%) had antibodies against *B. henselae* K68 antigens, 3 (2.6%) against Berlin1, 2 (1.7%) against Marseille, 1 (0.8%) against Houston-1, and 4 (3.4%) against any *Bartonella* spp. The difference between patients and controls was significant (p<0.001).

Seroconversion was reported for 6 of the 34 patients (18%) from which 2 serum samples were analyzed (Table). Among those who seroconverted for B. henselae, 1 had fever and only 2 reported having been scratched. Two of the patients who seroconverted were treated with doxycycline, and 1 was treated with ciprofloxacin. In addition, 1 patient with Sjogren disease was initially treated with penicillin, and later a hemangioma-like exanthema developed. Because of severe acne, the patient was treated with doxycycline for 6 months. The other patients who seroconverted were treated with penicillin or amoxicillin.

Seroconversion for *B. henselae* occurred in 4 patients, of which only 2 had reported a scratch. Three of these patients reacted to *B. henselae* Berlin1, and 1 reacted to the Houston-1 strain. Because symptoms did not differ between the patients who did seroconvert and those who did not, these findings could indicate subclinical infection.

The prevalence of immunoglobulin G against *B. henselae* in particular was shown to be much higher than that previously reported in Sweden (9). Earlier studies used only 2 Presented in part at the 4th International Conference on Bartonella as Emerging Pathogens, 2004 Aug 26–28, Uppsala, Sweden.

Table. Titers against Bartonella spp. antigens in 6 patients who seroconverted					
Patient sex/	Reciprocal titer				High titer
age, y	Acute-phase serum	Convalescent-phase serum	Interval, d	Antigen	to other antigen
F/77	32	128	6	B. henselae Berlin1	B. henselae K68
F/50	32	256	17	B. henselae Berlin1	0
M/74	128	512	19	B. grahamii	B. henselae K68
F/62	32	128	44	B. elizabethae	B. henselae K68
M/56	32	128	15	B. henselae Houston-1	B. grahamii
F/23	32	128	10	B. henselae Berlin1	B. grahamii

B. henselae antigens (Houston-1 and Marseille) compared with the 4 different B. henselae antigens used in the present study. Most reactivity to B. henselae in the present study was directed against the Swedish isolate K68 (27%); only 0.8% of controls had antibodies against that antigen.

An increased prevalence of antibodies against *B. henselae* after exposure to cats has been reported from Spain (10). Because seroconversion against *B. henselae* occurred in 2 patients who had not been scratched, cat bites may contribute to transmission of *B. henselae*.

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Letters

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Fatal Case of Israeli Spotted Fever after Mediterranean Cruise

To the Editor: Israeli spotted fever (ISF) is caused by Rickettsia conorii subsp. israelensis. This recently described subspecies is genetically close to R. conorii subsp. conorii, the agent of Mediterranean spotted fever (MSF) (1,2). ISF is likely transmitted by the dog tick Rhipicephalus sanguineus (3). This tick, which is also the vector of R. conorii subsp. conorii, has low affinity for hosts other than dogs. Therefore, like MSF, cases of ISF will likely be sporadic (4,5). ISF was first reported in Israel (1) and was also recently described in Portugal and Italy (6-8). The clinical manifestations of ISF are similar to those of other spotted fever group infections, but an inoculation eschar is rarely observed and a history of tick exposure is not always present (4-6.9). The incubation period is \approx 7–8 days after the tick bite (4).

We describe a 63-year-old man who had fatal ISF despite adequate therapy. The patient, who lived in Switzerland, took a cruise on the Mediterranean Sea, sailing for a week along the coasts of Crete, Libya, and Malta (Figure). With his wife, he visited several archeological sites in Libya (Cyrene, Apollonia, Ptolemais, Leptis Magna, Sabratha). Three days after returning to Switzerland, the patient reported loss of appetite, epigastric pain, and loose stools. Four days later, a fever (40°C) and generalized rash developed. The patient was hospitalized 6 days after symptom onset. At that time, he had fever (38.3°C), hypotension (85/55 mm Hg), tachycardia (100/ min); a maculopapular rash involving the trunk, limbs, palms, and soles; and petechial lesions on the right arm. The patient was confused and exhibited bilateral dysdiadochokinesis. Laboratory investigations yielded the following