Rickettsia felis in Chile

To the Editor: Rickettsiosis due to *Rickettsia felis* is an emerging disease that has been reported worldwide (*1*). Fever, headache, myalgia, and macular rash have been attributed to *R. felis* infection in humans (*1*). In South America, *R. felis* infection in fleas (mostly *Ctenocephalides* spp.) has been reported only in Brazil, Peru, and Uruguay (*2*–*3*). Although a growing number of articles have reported that *R. felis* is transmitted by fleas, the acquisition mechanism of *R. felis* by vertebrates or uninfected fleas in nature remains unknown (*4*).

Cats experimentally exposed to *R. felis*–infected fleas have been shown to become seropositive (*5*). However, neither serologic nor molecular evidence of *R. felis* infection has been reported in cats under natural conditions, despite the fact that most *C. felis* fleas are infected by *R. felis* (*6,7*).

In November 2006, we investigated the presence of rickettsial DNA in 30 *C. felis* fleas randomly collected from 22 domestic cats privately owned and housed indoors in a single household in Santiago, Chile. To detect rickettsial DNA in each individual flea, PCRs were performed that targeted a 398-nt fragment of the rickettsial *gltA* gene and an 856-nt fragment of the rickettsial *ompB* gene (*7,8*).

A total of 21 individual fleas (70%) yielded expected PCR products for both *gltA* and *ompB* genes. PCR *gltA* products from the 21 fleas and *ompB* products from 5 fleas were subjected to DNA sequencing as described (*7*). The *gltA* partial sequences obtained from 21 fleas were identical, as were the *ompB* partial sequences from 5 fleas. These sequences were 100% identical to corresponding sequences in the *R. felis* genome (GenBank accession no. CP000053).

Blood serum samples were collected from the 22 cats and tested by indirect immunofluorescence assay (IFA) with crude antigens derived from 6 *Rickettsia* isolates from Brazil: *R. bellii*, *R. amblyommii*, *R. rhipicephali*, *R. rickettsii*, *R. parkeri*, and *R. felis* (*7,9*). Serum was considered to contain antibodies against rickettsiae if it displayed a reaction at 1:64 dilution. End-point titers against each *Rickettsia* species were determined by testing serial 2-fold serum dilutions. Reactive serum specimens were tested in 2 or 3 replications by 2 readers before the end-point titer was determined. Serum showing a *Rickettsia* species titer at least 4-fold higher than those observed for the other *Ricketttsia* species was considered homologous to the first *Rickettsia* species or to a very closely related genotype (*7,9*). In each slide, a nonreactive cat serum specimen (negative control) and a known reactive cat serum specimen (positive control) were tested at the 1:64 dilution (*7*).

IFA detected antibodies reactive with *R. felis* (titer ≥64) in 16 (72.7%) of 22 cats. Among those, 5 (22.7%) also reacted with *R. rhipicephali*, 4 (18.2%) with *R. bellii*, 3 (13.6%) with *R. parkeri*, 2 (9.1%) with *R. rickettsii*, and 1 (4.5%) with *R. amblyommii*. No serum reacted with any other *Rickettsia* species without reacting with *R. felis* (Table). Four cat serum specimens (cats 1, 3, 8, and 11) showed titers to *R. felis* at least 4-fold higher than those to any of the other 5 antigens. The antibody titers in these 4 animals were considered to have been stimulated by *R. felis* infection. For the remaining 12 seropositive cats, we could not discern whether *R. felis* had been the infection agent because the results displayed a single titer of 64 for *R. felis* or showed similar titers for other *Rickettsia* species.

We report 70% *R. felis*–infected fleas in this study on the basis of the concordant results of 2 PCR amplifications (*gltA* and *ompB*) and DNA

Table. End-point titers of indirect immunofluorescence assay (IFA) for 6 *Rickettsia* species in cats from a household in Santiago, Chile*

Cat no.	IFA titers for *Rickettsia* antigens						PAIHR
	R. felis	*R. rhipicephali*	*R. bellii*	*R. parkeri*	*R. rickettsii*	*R. amblyommii*	
1	128	–	–	–	–	–	*R. felis*
2	256	256	128	–	–	–	
3	512	128	64	–	–	–	*R. felis*
5	64	–	–	–	–	–	
6	64	64	–	–	–	–	
7	64	–	–	–	–	–	
8	128	–	–	–	–	–	*R. felis*
9	64	–	–	–	–	–	
10	64	–	–	–	–	–	
11	128	–	–	–	–	–	*R. felis*
12	64	–	–	–	–	–	
14	128	–	64	128	–	128	
15	64	–	–	–	–	–	
19	64	–	–	–	–	–	
21	128	64	128	128	128	–	
22	64	64	–	64	64	–	

*PAIHR, possible antigen involved in a homologous reaction (serum showing for a *Rickettsia* species titer at least 4-fold higher than that observed for any other *Ricketttsia* species was considered homologous to the first *Rickettsia* species); –, nonreactive at titer ≥64.

sequencing. This infection rate is within the range (13.5%–90%) that has been reported for *R. felis* infecting *Ctenocephalides* fleas in Brazil and Uruguay (*2*,*3*,*7*). Sixteen (72.7%) cats contained *R. felis*–reactive antibodies; 4 of them showed titers to *R. felis* at least 4-fold higher than those to the other 5 rickettsial strains, findings that enabled us to technically conclude that these cats were exposed to *R. felis* or a closely related organism (*1*,*7*,*9*). Our finding of 70% *R. felis* infection in fleas infesting the cats indicates that cats acquired the infection through infected fleas. However, the mechanism of *R. felis* transmission by fleas is yet to be demonstrated under experimental conditions.

To our knowledge, the presence of *R. felis*, or a spotted fever group *Rickettsia* species, has not been reported in Chile. Recent investigations have provided clinical and serologic evidence of canine (*10*) and human (K. Abarca and J. Lopez, unpub. data) infection by spotted fever rickettsia in Chile, confirmed by IFA that used *R. conorii* commercial antigen. Since substantial serologic cross-reaction occurs between *R. conorii* and *R. felis* antigens (*1*), *R. felis* could be causing infection in dogs or humans in Chile.

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Possible Typhoon-related Melioidosis Epidemic, Taiwan, 2005

To the Editor: Melioidosis is a severe infection caused by *Burkholderia pseudomallei*. This organism is present in tropical and subtropical regions where melioidosis is endemic. Before 1995, melioidosis was rare in Taiwan. In 2001, when the annual number of cases of melioidosis in Taiwan was determined to be 1–3 per year from 1996 to 2000, the idea was first proposed that the disease was endemic (*1*).

From July 21 through August 24, 2005, an unusually large number (54) of melioidosis cases occurred in Taiwan. This number exceeded the average case number of 9.4 per year from 2001 to 2004. Since this outbreak appeared to be a common-source epidemic, all persons were suspected of becoming infected from this source at the same time.

To determine this common source, we investigated the role of Typhoon Haitang, which hit Taiwan on July 18 and 19, 2005, and resulted in heavy rainfall. Because the date of this typhoon overlapped the incubation period (1–21 days in most cases) (*2*) and rain is a factor in outbreaks of melioidosis (*3*), Typhoon Haitaing may have been the cause.

All 57 clinical strains of *B. pseudomallei* isolated during this outbreak were typed by pulsed-field gel electrophoresis (PFGE) DNA macrorestriction analysis (*4*). A higher incidence rate (8.86% per million) and clonal diversity (9 PFGE types) of *B. pseudomallei* were observed in the subtropical zone (south of 23.5°N) of Taiwan than in the temperate zone (north of 23.5°N) (0.18% per million and 2 PFGE types) (Table). Because clonal diversity in outbreaks of melioidosis is characteristic of extreme weather (*5*), these data support possible involvement of the typhoon in this outbreak.