Rickettsia felis in Fleas, Western Australia

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This study is the first confirmation of *Rickettsia felis* in Australia. The organism was identified from 4 species of fleas obtained from dogs and cats in Western Australia, by using polymerase chain reaction amplification and DNA sequencing of the citrate synthase and outer membrane protein A genes.

Several rickettsial diseases have been documented in Australia, namely, Queensland tick typhus (*Rickettsia australis*), scrub typhus (*Orientia tsutsugamushi*), murine typhus (*R. typhi*), and more recently, Flinders Island spotted fever (*R. honei*), as well as the closely related Q fever (*Coxiella burnetii*) and cat scratch fever (*Bartonella henselae*). Cases of murine typhus have been reported in Western Australia (WA) since 1927, and a serologic survey provided evidence that members of the closely related spotted fever group (SFG) rickettsiae are also present in the state (3,4).

*R. felis* is a newly discovered species within the SFG; it is transmitted by fleas, which makes it unique within the biogroup. The species was first detected in the cat flea, *Ctenocephalides felis*, and subsequently has been determined to cause human disease in a number of countries (5–8). A recent study in New Zealand provided the first report of the organism in Oceania (9). Infected domestic and wild animals may not exhibit clinical disease and act as reservoirs of infection for humans. No definitive reports of the organism have been made in Australia, however, a study of cat fleas that used polymerase chain reaction and restriction fragment length polymorphisms of the amplification products (PCR-RFLP), provided strong evidence that *R. felis* exists (10). Our study aimed to confirm the presence of *R. felis* in Australia and to determine the distribution of the organism in WA. This study was approved by the Murdoch University Animal Ethics Committee.

Conclusions

A total of 368 fleas collected from 43 cats and 116 dogs were pooled into 165 flea pools (mixed infections from 6 animals meant 6 more flea pools than the total number of animals). Four different species of flea were identified: *C. felis* (49 from 38 cats and 241 from 99 dogs), *C. canis* (12 from 7 dogs), *Echidnophaga gallinacea* (4 from 3 cats and 57 from 16 dogs), and *Spilopsyllus cuniculi* (5 from 2 cats). Six dogs had a mixed population of fleas; 4 of these had *C. felis* and *E. gallinacea*, and 2 had *C. felis* and *C. canis*.

Forty-two (36%) of the 116 flea pools from dogs were positive for both the gltA and ompA genes. Similarly, 14 (33%) of 43 flea pools from cats were positive for both genes. Notably, positive samples were obtained from all the locations in the study, indicating widespread distribution throughout the state (Table).
Of the 8 samples from *C. felis* positive for both *gltA* and *ompA* genes that were sequenced by using the *gltA* primers, all sequences matched the *gltA* gene from *R. felis* (99% similarity). Of the 2 samples that were also sequenced by using *ompA* primers, the sequences matched the *R. felis ompA* gene (100% similarity).

Our study demonstrates that *R. felis* is present in multiple sites in WA and was conclusively present in 1 of the 4 flea species collected (*C. felis*). The results obtained from 2 rounds of PCR are highly indicative of *R. felis* infection in *E. gallinacea* also. Because *C. felis* has the highest rate of infection and is prevalent, highly mobile, and nonspecific in its choice of hosts (including humans), it is likely to be the most important vector of the organism. *C. canis* has been identified as a vector of *R. felis* (13); however, this finding was not supported by our study. The presence of *R. felis* in *E. gallinacea* has been previously reported (14). To our knowledge, this is the first time a rickettsia has been detected from *S. cuniculi*, which could also be a potential vector for *R. felis*.

The significance of *R. felis* as a cause of human disease in WA has not yet been determined. Because of the often transient and nonspecific symptoms of rickettsioses, infections may not be readily detected. A serologic survey conducted in the Kimberley region of WA (10) showed evidence of scrub typhus and an SFG rickettsia, but no further work has identified the specific organism responsible for the latter. Another serologic study of 866 people throughout southwest WA showed evidence of infection with *R. typhi* (0%–1%) and another undetermined SFG rickettsia (3%–13%). During the same study, fleas were collected from cats and dogs in Perth and screened for rickettsiae by using PCR-RFLP of the *gltA* gene; the results provided evidence for the existence of *R. felis*. However, no sequencing data confirmed its presence (4).

The results from the current study showed that the *gltA* gene from all the sequenced samples most closely matched the *gltA* gene in the species *R. felis*. The identity of the sequenced samples as *R. felis* was confirmed by the *ompA* gene sequences. Therefore, the other samples shown to be positive for SFG rickettsiae in the PCR screening process are probably also *R. felis*.

This study has confirmed the presence of *R. felis* in WA; consequently, this rickettsial disease should be included as a differential diagnosis for influenzalike illnesses in persons who own or work with companion animals.

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Mr Schloderer completed a Bachelor of Science at Murdoch University. This work formed part of his research which concentrated on investigating Rickettsia spp. in companion animals in Western Australia.

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