Spotted Fever Group *Rickettsia* sp. Closely Related to *R. japonica*, Thailand

To the Editor: In response to a recent report that suggested human infection with *Rickettsia japonica* in northeastern Thailand (1), we phylogenetically reexamined spotted fever group rickettsiae (SFGR) from Thailand. The organism had been isolated from a male *Haemaphysalis hystricis* tick found on Mt. Doi Suthep, Chiang Mai, northern Thailand, in December 2001. The strain was designated TCM1 and was not distinguishable from *R. japonica* by indirect immunoperoxidase stain using monoclonal antibody (2).

After propagating strain TCM1 in L-929 cell culture, we extracted DNA by using a Wizard Genomic DNA Purification Kit (Promega, Madison, WI, USA). We subjected the DNA to sequencing that targeted a 491-bp fragment of rickettsial outer membrane protein A (*ompA*), a 394-bp fragment of the rickettsial genus–specific 17-kDa antigen gene, and a 1,250-bp fragment of citrate synthase gene (*gltA*). Direct sequencing of amplicons was performed as previously described. (3) Phylogenetic analyses based on *ompA* indicated that strain TCM1 was closely related to and clustered within the same clade as *R. japonica* strain YH (98.4% identity) (Figure, panel A). Also, a 17-kDa antigen gene obtained from strain TCM1 showed 99.5% identity to the corresponding gene of *R. japonica* (Figure, panel B). Our phylogenetic analysis with *ompA* and 17-kDa antigen gene showed that strain TCM1 was closely related to *R. japonica* but distinguished from *Rickettsia* sp. PMK94 (data not shown). Thus, we describe the *R. japonica* group in Thailand. DNA sequences of strain TCM1 were determined and deposited in GenBank/EMBL/DDBJ under the following accession nos.: *ompA*, AB359459; 17-kDa antigen, AB359457; *gltA*, AB359458.

![Figure. Phylogenetic analysis based on *ompA* gene (A) and rickettsial genus–specific 17-kDa antigen gene (B). Sequences were aligned by using the ClustalW software package (http://clustalw.ddbj.nig.ac.jp/top-j.html), and neighbor-joining phylogenetic tree construction and bootstrap analysis were conducted according to the Kimura 2-parameter method (www.ddbj.nig.ac.jp). Pairwise alignments were performed with an open-gap penalty of 10, a gap extension penalty of 0.5, and a gap distance of 8. Multiple alignments were also performed with the same values, and the phylogenetic branches were supported by bootstrap analysis with 1,000 replications (>800 were indicated). *Rickettsia felis* (CP000053) and *R. canadensis* (CP000409) were used as outgroups for *ompA* and 17-kDa antigen gene, respectively. The phylogenetic tree was constructed by using TreeView software version 1.5 (http://taxonomy.zoology.gla.ac.uk/rod/treeview.html). Scale bars indicate nucleotide substitutions (%) per site.](http://cid.oxfordjournals.org/content/suppl/2009/04/15/066.206.F图为)
**Segniliparus rugosus Infection, Australia**

To the Editor: Recently, a female teenager with cystic fibrosis who resided in tropical north Queensland, Australia, was found to be infected with *Segniliparus rugosus*. She was homozygous for the deltaF508 mutation, had well-preserved lung function, and regularly played competitive sports. Unlike many cystic fibrosis patients, she did not have a history of chronic *Pseudomonas aeruginosa* infections, but *Stenotrophomonas maltophilia* and *Achromobacter xylosoxidans* had been previously isolated from her sputum. In May 2007, she described reduced exercise tolerance and increased cough with excess sputum production. Lung function testing showed modest spirometric decline. A computed tomographic scan of the chest showed significant mucus plugging and bronchiectasis, uncommon without previous *P. aeruginosa* infection. Sputum was 3+ smear positive for acid-fast bacilli (AFB), and *S. rugosus* was isolated from liquid culture. Empiric antimicrobial drug therapy was changed to rifabutin and co-trimoxazole because these drugs have been effective in previous cases (1). Clinically, the patient showed response to the treatment. After 12 months of treatment, her sputum was still 3+ positive for AFB, and *S. rugosus* was again found in culture. She was referred to a pediatric teaching hospital in Brisbane with worsening respiratory symptoms precipitated by influenza B infection. Antimicrobial drug therapy with intravenous imipenem, oral moxifloxacin, and co-trimoxazole for 2 weeks resulted in clinical improvement but little reduction in smear positivity.

The initial AFB smear-positive sputum specimen underwent routine decontamination with sodium hydroxide and neutralization and was inoculated into radiometric 12B vials. (Bec-

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