Rickettsia parkeri in Uruguay

To the Editor: During 1990 in Uruguay, a rickettsiosis in the spotted fever group was presumptively diagnosed for 3 patients who had fever, an initial small maculopapulous lesion at the site of a tick bite on the scalp, and subsequent regional lymphadenopathy. Microimmunofluorescent serologic assay, with Rickettsia conorii as the sole antigen source, gave positive results for all patients, and these infections were presumptively identified as spotted fever caused by R. conorii (1). During 1993–1994, a total of 23 patients who had a history of tick bite, some of whom had exanthema and inoculation eschars, were identified from Canelones County, Uruguay. These patients had antibodies against R. conorii, according to microimmunofluorescence testing; however, R. conorii was again the sole antigen source used in the assay (2). Because 1 of the major limitations of serologic testing for diagnosis of rickettsioses is the cross-reactivity between different Rickettsia species, the association of R. conorii with the spotted fever group cases in Uruguay was considered inappropriate (3). In addition, R. conorii has never been found in the Western Hemisphere (3).

Amblyomma triste, a neotropical tick species with a variety of hosts, is the main tick species that feeds on humans in Uruguay and the primary candidate vector for tickborne rickettsioses in that country (4). A recent investigation demonstrated DNA of R. parkeri in A. triste ticks collected from humans and animals, indicating that this rickettsia could be the pathogenic agent of spotted fever group rickettsioses in Uruguay (5). In the United States, where A. maculatum ticks infected with R. parkeri have been reported since the 1930s, the role of this rickettsial agent as a human pathogen was confirmed only recently (3). Our study is the first to isolate R. parkeri from A. triste collected in Uruguay and confirms the presence of this emerging pathogen in South America.

During September 2004, 78 adult flat ticks (25 males, 53 females) identified as A. triste were collected from vegetation in the suburban area of Toledo Chico (34°44′53″S, 56°06′19″W) in Canelones County, southern Uruguay. At the laboratory, the legs of live ticks were extirpated for DNA extraction, and the tick bodies were immediately frozen at −80°C. Each group of legs from 1 tick was subjected to DNA extraction by boiling at 100°C for 20 min as described (6). DNA extracted from each tick was tested by PCR by using primers CS-78 and CS-323 (Table), which targeted fragments of 3 rickettsial genes: gltA, ompB, and ompA. PCR products of expected size were obtained in all reactions and subjected to DNA sequencing as described (6). Fragments of 1,084, 775, and 491 nt of the gltA, ompB, and ompA genes, respectively, were obtained and showed 100% identity to the corresponding sequences available in GenBank (accession nos. U59732, AF123717, and U43802, respectively) for the Maculatum strain of R. parkeri from United States. Although isolation of Rickettsia from the male tick was unsuccessful, DNA extracted from remnants of the male and female ticks was tested by PCR (ompA, Table) and yielded product that after

Table. Primer pairs used for amplification of rickettsial genes

<table>
<thead>
<tr>
<th>Primer pairs</th>
<th>Genes and primers</th>
<th>Primer sequences (5′→3′)</th>
<th>Fragment size (nucleotides)</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>gltA</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>CS-78</td>
<td>GCAAGTATCGTGGAGGTGGTAA</td>
<td>401</td>
<td>7</td>
</tr>
<tr>
<td></td>
<td>CS-323</td>
<td>GCTTCTTAAATTTCAATACGGAG</td>
<td>7</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>CS-239</td>
<td>GCTCCTCTCATCCTATGGCTATT</td>
<td>834</td>
<td>7</td>
</tr>
<tr>
<td></td>
<td>CS-1069</td>
<td>CAGGCTTCTCTGATTCCTTCTT</td>
<td>7</td>
<td></td>
</tr>
<tr>
<td>ompB</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>120-M59</td>
<td>CCAGCAGGTGTGTTAATCTCG</td>
<td>862</td>
<td>8</td>
</tr>
<tr>
<td></td>
<td>120-807</td>
<td>CCTTTTAGATTTACCGCTAAA</td>
<td>8</td>
<td></td>
</tr>
<tr>
<td>ompA</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>R190.70p</td>
<td>ATGGGCGAATTTTCTCCAAA</td>
<td>530</td>
<td>9</td>
</tr>
<tr>
<td></td>
<td>R190.602n</td>
<td>AAGTGACCGACAGTTGCCCTCC</td>
<td>9</td>
<td></td>
</tr>
</tbody>
</table>
sequencing (491 nt) showed 100% identity to the R. parkeri sequence from GenBank (U43802).

These procedures enabled the identification of R. parkeri in 2.56% of the A. triste ticks from Uruguay. Previous findings of R. parkeri DNA in A. triste ticks from Uruguay (5) are corroborated by our isolation of a Uruguayan strain of R. parkeri in cell culture. The only other country where R. parkeri has been previously reported is the United States, where it is associated with A. maculatum ticks and is the causative agent of an emerging rickettsiosis (3). As A. maculatum and A. triste are established in at least 12 other Latin American countries (10), the distribution of R. parkeri in the Americas is likely continental. Finally, our results corroborate recent reports (3,5) that suggest R. parkeri is the causative agent of previously reported cases of rickettsiosis in Uruguay.

This study was financially supported by Foundation of Support to the Research of the State of São Paulo (FAPESP).

Richard C. Pacheco,*
José M. Venzal,†
Leonardo J. Richtzenhain,*
and Marcelo B. Labruna*  
*University of São Paulo, São Paulo, SP, Brazil; and †University of La República, Montevideo, Uruguay

References

Address for correspondence: Marcelo B. Labruna, Laboratório de Doenças Parasitárias, Departamento de Medicina Veterinária Preventiva e Saúde Animal, Faculdade de Medicina Veterinária e Zootecnia, Universidade de São Paulo, Av. Prof. Dr. Orlando Marques de Paiva 87, São Paulo, SP, Brazil 05508-270; email: labruna@usp.br

Influenza-related Death Rates for Pregnant Women

To the Editor: Articles about influenza in the January 2006 issue of Emerging Infectious Diseases discussed a pandemic possibly as profound in its effect as the 1918–19 pandemic, when attack rates were >20% worldwide and death rates were 1%–2%. Then, as when subsequent virus antigenic shifts have occurred, all age groups were affected. Governments are now preparing contingency plans against the effects of an expected further antigenic shift.

However, insufficient consideration may have been given to how, in the absence of effective prophylaxis against a novel strain of influenza virus, to avoid deaths on the scale seen in the fall and winter of 1918–19. In particular, the vulnerability of pregnant women and their offspring appears to have been forgotten. Bland reported on pregnant influenza patients in Philadelphia and elsewhere in the fall of 1918; of 337, 155 died (1). Harris obtained by questionnaire from obstetricians medical histories of 1,350 pregnant patients in Maryland and in 4 large US cities (2). Pneumonia developed in half (678) of these patients and 365 died. Death rates from pneumonia were >40% for every month of pregnancy; fetal loss

Correction: Vol. 12, No. 10

In Human Prion Disease and Relative Risk Associated with Chronic Wasting Disease by Samantha MaWhinney et al., an error occurred in the list of references. Missing from the list is reference no. 36: Belay ED, Maddox RA, Gambetti P, Schonberger LB. Monitoring the occurrence of emerging forms of Creutzfeldt-Jakob disease in the United States. Neurology. 2003;60:176-81. The corrected list of references appears in the online article at http://www.cdc.gov/ncidod/EID/vol12no10/06-0019.htm

We regret any confusion this error may have caused.