the present study, PARV4 and PARV5 have been identified in blood samples obtained from persons from the United Kingdom. For parvovirus B19, there is evidence of persistent virus infection, at low levels, in bone marrow of previously exposed persons (7) and in plasma of immunocompromised and immunocompetent persons (8,9). There is also evidence for the lifelong persistence of parvovirus B19 (genotypes 1 and 2) in tissues such as skin and synovia (10). PARV4 and PARV5 virus genomes share only limited homology with parvovirus B19 (<30% amino acid similarity). Although they have been detected in blood and plasma, nothing is known about the role of these viruses in human disease or their ability to persist in infected persons, healthy or otherwise. Further studies will be required to determine the prevalence of PARV4 and PARV5 in healthy persons compared with its prevalence in those with chronic infections and at high risk, such as IVDUs, and to investigate the nature of persistence of these novel viruses.

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References


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**Saint Louis Encephalitis Virus, Brazil**

To the Editor: Saint Louis encephalitis virus (SLEV), a member of the Flaviviridae family, is widely dispersed in the Americas (1,2). In Brazil, SLEV was first isolated in the 1960s from a pool of mosquitoes at the Amazon Basin. Subsequently, the virus was repeatedly isolated from animals and arthropods in the Amazon region and Sáo Paulo state (3). Nonetheless, isolation of SLEV from humans is rare; only 2 isolates from humans were described before 2005. Each isolate was from a patient who had jaundice and febrile illness without any neurologic symptoms (1,3). Recently in Sáo Paulo, SLEV was isolated from a patient who had an incorrect diagnosis of dengue fever (2,4).

Despite the rare isolation of SLEV from humans, antibodies to this virus have been found in ≈5% of studied populations in the north and southeast regions of Brazil. However, because of antibody cross-reactivity among different flaviviruses and the fact that this population is vaccinated against yellow fever and exposed to dengue virus (DENV), such results should be interpreted carefully. Nevertheless, in these areas, SLEV may circulate and infect humans, although most infections are undiagnosed (1,3,5).

In contrast to previous instances in which the disease was detected in only 1 patient, we describe the first community outbreak of SLEV in Brazil. The outbreak was detected in Sáo José do Rio Preto (population 400,000), in northwest Sáo Paulo state. This outbreak was concurrent with a large outbreak of DENV serotype 3 (DENV-3), which occurred during the first half of 2006, with >15,000 possible cases reported to public health authorities. During this time, we were involved in an epidemiologic study to monitor the disease. We tested ≈250 samples for DENV, and 65% were positive. We tested for SLEV only those patients who were in our hospital or those who were referred to us for SLEV testing after an initial diagnosis of SLEV or DENV. The protocol approved by our ethical committee allowed us to test only samples from these patients (process no. 300/2004).
We used a multiplex nested reverse transcription–PCR (RT-PCR) assay to identify the most common flaviviruses in Brazil (DENV-1, DENV-2, DENV-3, yellow fever virus) as well as DENV-4, Ilheus virus, Iguaque virus, Rocio virus, and SLEV. Of 54 samples (49 serum and 5 cerebrospinal fluid [CSF]) that were negative for DENV and yellow fever virus, SLEV RNA was detected in 6 (4 serum and 2 CSF) (6). RT-PCR results were negative for all other tested flaviviruses. Sequences of the amplified SLEV cDNAs from the 2 CSF samples were determined by using an ABI377 automated sequencer (Applied Biosystems, Foster City, CA, USA). The resulting sequences (GenBank accession nos. DQ836336 and DQ836337) were identical and showed 96% homology to an Argentinean SLEV isolate (AY6-32544). All 6 SLEV-infected patients had an initial diagnosis of dengue fever or viral encephalitis; 3 had a diagnosis of viral meningoencephalitis, and the other 3 had signs of hemorrhagic disease (Table).

Dengue is widely disseminated in Brazil and causes large outbreaks almost every year. The high prevalence of antibodies in the Brazilian population (1,3,6) suggests that SLEV infections are being misdiagnosed; its importance is underestimated. Brazil has no SLEV surveillance programs, and health professionals do not usually consider SLEV among their differential diagnoses. This SLEV outbreak was detected in a large urban center and was not specifically linked to patients who dwell in pockets of tropical forests, as previously reported (1-4).

This outbreak may represent the first time that hemorrhagic signs have been linked to SLEV infections. SLEV-associated hemorrhagic manifestations have not been reported in the literature. However, of our 6 SLEV-infected patients, 3 had hemorrhagic signs. Substantiating a causal link between SLEV infection and such clinical manifestations is difficult because DENV is endemic in the studied region (7). Possibly, SLEV-infected patients with hemorrhagic signs may have been previously infected by DENV. No reports have linked hemorrhagic manifestations to sequential DENV and SLEV infections; this possible link needs to be carefully evaluated.

In Argentina, SLEV has been isolated several times from animals (8). In some regions, SLEV seroprevalence in humans is ≈13% (9), but the number of documented human infections is small (10). These findings indicate either that SLEV is more prevalent than reported or that SLEV is reemerging. The Brazilian cases may parallel the situation in Argentina.

Our results clearly indicate an SLEV outbreak among this local population in Brazil. This outbreak differs from isolated infections previously described and indicates that this disease may be more prevalent in Brazil. In fact, the number of samples tested for SLEV during this DENV outbreak was relatively small. Had more samples been investigated, more cases of SLEV infection might have been found. A more comprehensive epidemiologic study is required to fully assess the magnitude of SLEV infection in Brazil.

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<table>
<thead>
<tr>
<th>Patient no. (age)</th>
<th>Sample tested by RT-PCR</th>
<th>Date of hospital admission</th>
<th>Initial diagnosis at admission</th>
<th>Signs, symptoms, selected laboratory results</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 (27 y)</td>
<td>Serum</td>
<td>Feb 25</td>
<td>Dengue fever</td>
<td>Clinical: fever, abdominal pain, diarrhea, Serum: AST 58 IU/mL, ALT 69 IU/mL</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Clinical: fever, abdominal pain, melena, petechiae, positive tourniquet test</td>
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<tr>
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<td></td>
<td></td>
<td>Serum: platelets 311,000/mm³, hematocrit 29%</td>
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<tr>
<td></td>
<td></td>
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<td></td>
<td>CSF: 13 cells/mm³, lymphocytes 86%, monocytes 14%</td>
</tr>
<tr>
<td>2 (7 mo)</td>
<td>Serum</td>
<td>Mar 06</td>
<td>Dengue hemorrhagic fever, viral encephalitis</td>
<td>Clinical: fever, headache, chills, myalgia, maculopapular rash, positive tourniquet test</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Serum: hematocrit 43%, platelets 280,000/mm³</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>History: previous DENV infection (2002)</td>
</tr>
<tr>
<td>3 (37 y)</td>
<td>Serum</td>
<td>Apr 22</td>
<td>Dengue hemorrhagic fever</td>
<td>Clinical: fever, headache, chills, myalgia, maculopapular rash, positive tourniquet test</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Serum: platelets 141,000/mm³, hematocrit 36%, AST 81 IU/mL, ALT 56 IU/mL</td>
</tr>
<tr>
<td>4 (34 y)</td>
<td>Serum</td>
<td>Apr 23</td>
<td>Dengue hemorrhagic fever</td>
<td>Clinical: fever, headache, chills, myalgia, maculopapular rash, positive tourniquet test</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Serum: platelets 286 cells/mm³, lymphocytes 60%, polymorphonuclear cells 37%, eosinophils 3%</td>
</tr>
<tr>
<td>5 (5 y)</td>
<td>CSF</td>
<td>Jun 05</td>
<td>Viral meningoencephalitis</td>
<td>Clinical: fever, facial palsy</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>CSF: 12 cells/mm³, lymphocytes 100%</td>
</tr>
<tr>
<td>6 (11 y)</td>
<td>CSF</td>
<td>Jun 07</td>
<td>Viral meningoencephalitis</td>
<td>Clinical: fever, facial palsy</td>
</tr>
</tbody>
</table>

*RT-PCR, reverse transcription–PCR; AST, aspartate aminotransferase; ALT, alanine aminotransferase; CSF, cerebrospinal fluid; DENV, dengue virus.
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Cryptococcus gattii
Risk for Tourists Visiting Vancouver Island, Canada

To the Editor: An unprecedented outbreak of Cryptococcus gattii genotype amplified fragment length polymorphism (AFLP) 6/VGII on Vancouver Island, British Columbia, Canada, is affecting both human and animal hosts with normal immunity (1/3). So far, >100 human cases, including at least 6 fatalities, have been reported by the British Columbia Centre for Disease Control (4), (www.bccdc.org, www.cbc.ca). Vancouver Island is a major tourist destination, with ≈7.5 million visits each year (www.bcstats.gov.bc.ca). We report the first known intercontinental transmission of C. gattii from this outbreak in a tourist from Denmark who visited Vancouver Island. This case indicates a potential risk for tourism-related acquisition.

A 51-year-old, HIV-negative, apparently immunocompetent man from Denmark, with known psoriatic gout and under treatment with a non-steroidal antiinflammatory drug, was admitted to a hospital in Herning, Denmark, with chest pain radiating to the left shoulder and arm, lasting for 1 day. Six weeks before his admission, he returned to Denmark from a 3-week trip to Canada, during which he visited Victoria and surrounding areas on the eastern coast of Vancouver Island for 7 days. During their stay, the patient and his 3 fellow travelers visited gardens and studied the local natural vegetation.

During his stay in Canada, the patient had no symptoms, and symptoms had not developed in any of his family members as of October 2006. On admission to the hospital, his temperature was 38.2°C, and a chest radiograph showed 3 large nodular infiltrates suspect for malignancy or abscesses. Neither bacterial nor