Women who transmitted HIV-1 to their infants had a significantly higher geometric mean concentration of V3 loop specific IgG1 antibody than non-transmitters. Concentrations of V3 loop-specific IgA antibody, which does not cross the placenta, were not different between transmitting and non-transmitting mothers. The higher concentrations of IgG1 in transmitters could be a direct correlate of transmission or it could simply be a marker for other maternal factors that enhance maternal-infant transmission, such as longer duration of infection or increased viral load. However, in the limited subsets of our subjects for whom data were available, there was no correlation between p24 antigen concentration (a correlate of viral load) or CD4 T-cell numbers (a correlate of disease stage) and envelope-specific IgG1 concentrations. These results are consistent with another report showing that antibody concentration to a specific HIV antigen, p24, declines as disease progresses. The possibility that maternal antibody to the primary neutralising domain of the HIV envelope is positively associated with maternal-infant transmission may have important implications for efforts to interrupt the transmission process.

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### References


### New arenavirus isolated in Brazil

Terezinha Lisieux M Coimbra, Elza S Nassar, Marcelo N Burattini, Luiza Terezinha Madia de Souza, Ivani B Ferreira, Iray M Rocco, Amelia P A Travassos da Rosa, Pedro F C Vasconcelos, Francisco P Pinheiro, James W LeDuc, Rebeca Rico-Hesse, Jean-Paul Gonzalez, Peter B Jährling, Robert B Tesh

A new arenavirus, called Sabiá, was isolated in Brazil from a fatal case of haemorrhagic fever initially thought to be yellow fever. Antigenic and molecular characterisation indicated that Sabiá virus is a new member of the Tacaribe complex. A laboratory technician working with the agent was also infected and developed a prolonged, non-fatal influenza-like illness. Sabiá virus is yet another arenavirus causing human disease in South America.


Four arenaviruses (Lassa, Junin, Machupo, and Guanarito) have been associated with haemorrhagic disease in man. Although each has a distinct geographic distribution, they have common modes of transmission and the diseases associated with them (Lassa, Argentinian, Bolivian, and Venezuelan haemorrhagic fevers, respectively) have similar clinical manifestations and high death rates. We report isolation of a new arenavirus from a fatal case of haemorrhagic fever in São Paulo, Brazil.

The index case was a 25-year-old female agricultural engineer who was admitted on Jan 12, 1990, after 12 days of...
fever, headache, myalgia, nausea, vomiting, and weakness. The patient's history was unremarkable. She worked mainly in an office. She had not travelled out of São Paulo State for months before her illness. The 10 days preceding onset were spent in two different cities with family and friends, all of whom were well.

Examination revealed an acutely ill, somnolent, and mildly dehydrated woman with a very red oropharynx. Laboratory studies (table) indicated leukopenia and slightly elevated aspartate aminotransferase. The differential diagnoses included sepsis, leptospirosis, malaria, hepatitis, and yellow fever. Treatment included intravenous fluids, electrolytes, and cefotaxin (500 mg every 12 h). Over the next 3 days, the patient worsened with haematemesis, vaginal bleeding, and conjunctival petechiae. She developed increasing somnolence, tremors, difficulty in walking, and generalised tonic-clonic convulsions. On the third day, the patient went into coma and an unresponsive shock; laboratory tests were abnormal (table). Death occurred on the fourth day. Principal necropsy findings were: diffuse pulmonary oedema and congestion with intraparenchymal haemorrhages; hepatic congestion with focal haemorrhage and necrosis; renal oedema and acute tubular necrosis; splenic enlargement and congestion; and massive gastrointestinal haemorrhage.

A blood sample taken shortly before death was submitted to the Adolfo Lutz Institute, where it was inoculated intracerebrally into newborn mice. A filterable agent was demonstrated in paired acute and convalescent sera. The source of the agent did not react with immune sera prepared to human yellow fever virus. The histopathological appearance of the liver at necropsy is indistinguishable from that of yellow fever.

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The signs and symptoms in our two patients were similar to those of the other arenavirus haemorrhagic fevers. Liver damage is often observed in patients dying of such fevers. The histopathological appearance of the liver at necropsy is indistinguishable from that of yellow fever.

Table: Laboratory findings

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Admission</th>
<th>Day 3</th>
<th>Day 4</th>
</tr>
</thead>
<tbody>
<tr>
<td>Haemoglobin (g/dL)</td>
<td>12.4</td>
<td>11.0</td>
<td>8.3–4.9</td>
</tr>
<tr>
<td>Total leucocytes (× 10^9/L)</td>
<td>3.7</td>
<td>5.9</td>
<td>7.7</td>
</tr>
<tr>
<td>Neutrophils (%)</td>
<td>86</td>
<td>78</td>
<td>64</td>
</tr>
<tr>
<td>Lymphocytes (%)</td>
<td>13</td>
<td>19</td>
<td>38</td>
</tr>
<tr>
<td>Platelets (× 10^9/L)</td>
<td>&gt;150</td>
<td>90</td>
<td>36</td>
</tr>
<tr>
<td>Prothrombin time (s)</td>
<td>24.7</td>
<td>17</td>
<td></td>
</tr>
<tr>
<td>Fibrinogen (mg/dL)</td>
<td>2.2</td>
<td>1.55</td>
<td>1.15</td>
</tr>
<tr>
<td>Crea (mg/dL)</td>
<td>0.9</td>
<td>2.2</td>
<td>3.1</td>
</tr>
<tr>
<td>Aspartate aminotransferase (U/L)</td>
<td>968</td>
<td>3280</td>
<td>3180</td>
</tr>
<tr>
<td>Alanine aminotransferase (U/L)</td>
<td>35</td>
<td>770</td>
<td>820</td>
</tr>
<tr>
<td>Alkaline phosphatase (U/L)</td>
<td>0.7</td>
<td>4.1</td>
<td>2.0</td>
</tr>
<tr>
<td>Total bilirubin (mg/dL)</td>
<td>0.3</td>
<td>3.8</td>
<td>1.4</td>
</tr>
<tr>
<td>Direct bilirubin (mg/dL)</td>
<td>0.3</td>
<td>3.8</td>
<td>1.4</td>
</tr>
</tbody>
</table>

References


Virus Service, Adolfo Lutz Institute (T L M Coimbra as, E S Nasser as, L T M de Souza vo, I B Ferreira as, I M Rocoo as); São Paulo; and Infectious Diseases Clinics, Paulista Medical School, São Paulo, Brazil (M N Burattini mo); Arbovirus Department, Evandro Chagas Institute, Belem, Brazil (A P A Travassos da Rosa as, Prof P F C Vasconcelos mo); Communicable Diseases Program, Pan American Health Organization, Washington DC, USA (Prof P Pinheiro mo); Microbiology and Immunology Support Section, Health Organization, Geneva, Switzerland (J W LeDuc mo); Yale Arbovirus Research Unit, Department of Epidemiology and Public Health, Yale University School of Medicine, PO Box 208034, New Haven, CT 06620 (R Rico-Hesse mo, J P Gonzalez mo, Prof R B Tesh mo); and Virology Division, US Army Medical Research Institute of Infectious Diseases, Frederick, Maryland, USA (P B Jahrling mo)

Correspondence to: Prof Robert B Tesh