



Figure: Concentrations of HIV-1 V3 loop specific IgG1 and IgA antibody in mothers who transmitted HIV-1 to their child and those who did not

Note different scales for y-axes.

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.⁹ 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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New arenavirus isolated in Brazil

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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.

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Four arenaviruses (Lassa, Junin, Machupo, and Guanarito) have been associated with haemorrhagic disease in man.^{1,2} 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.^{1,3} 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

	Admission	Day 3	Day 4
Haemoglobin (g/dL)	12.4	11.0	8.3-4.9
Total leucocytes ($\times 10^9/L$)	3.7	5.9	7.7
Neutrophils (%)	86	78	54
Lymphocytes (%)	13	19	38
Platelets ($\times 10^9/L$)	>150	90	36
Prothrombin time (s)	..	14.7	17
Fibrinogen (mg/dL)	..	155	115
Creatinine (mg/dL)	0.9	2.2	3.1
Aspartate aminotransferase (U/L)	96	3230	3180
Alanine aminotransferase (U/L)	35	770	820
Alkaline phosphatase (U/L)	491
Total bilirubin (mg/dL)	0.7	4.1	2.0
Direct bilirubin (mg/dL)	0.3	3.8	1.4

Table: Laboratory findings

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 2 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 leucopenia and slightly elevated aspartate aminotransferase. The differential diagnoses included sepsis, leptospirosis, malaria, hepatitis, and yellow fever. Treatment included intravenous fluids, electrolytes, and cefoxitin (1 g every 6 h) and amikacin (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 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 isolated from brains of the sick and dying mice. Because this agent did not react with immune sera prepared to human pathogenic viruses commonly encountered in Brazil (including yellow fever), it was forwarded to the Evandro Chagas Institute and then to the Yale Arbovirus Research Unit and the US Army Medical Research Institute of Infectious Diseases for further study. Results of complement-fixation, immunofluorescence, and neutralisation indicated that the agent, called Sabiá virus after the name of the community where the patient was staying when she became ill, is a new member of the Tacaribe complex of the genus *Arenavirus*.⁴ 250 nucleotides from the 3' end of the S segment of Sabiá virus RNA were compared with those of five other Tacaribe complex viruses (Junin, Machupo, Guanarito, Tacaribe, and Pichinde) by limited sequence analysis.^{5,6} Sabiá virus was 56% divergent from Junin, Machupo, and Guanarito viruses. The source of infection in the index case is unknown, but it seems likely that Sabiá virus exists in a rodent reservoir.

During characterisation of the virus in Belem, a 39-year-old laboratory technician was infected, probably by

aerosol.⁷ He had a severe illness (temperature 38-40°C, chills, malaise, headache, generalised myalgia, sore throat, conjunctivitis, nausea, vomiting, diarrhoea, epigastric pain, and bleeding gums) for 15 days. Besides leucopenia ($2.5 \times 10^9/L$), laboratory values remained normal. Admission and intravenous fluids were required and the patient recovered. Seroconversion to Sabiá virus was demonstrated in paired acute and convalescent sera.

The signs and symptoms in our two patients were similar to those of the other arenavirus haemorrhagic fevers.^{1,2,8} Liver damage is often observed in patients dying of such fevers.^{8,9} The histopathological appearance of the liver at necropsy is indistinguishable from that of yellow fever.

Our two cases illustrate the difficulty in diagnosing arenavirus infections. These illnesses are insidious and initially indistinguishable from various other common non-specific viral infections.^{1,8} If they become haemorrhagic, they can be misdiagnosed as yellow fever or dengue haemorrhagic fever.^{1,2,8} Virological confirmation is essential to establish a correct diagnosis.

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