stroke located in the left hemisphere. IgG and IgM antibodies against TOSV were detected in a serum sample obtained at that time. The patient was discharged 1 week later with slight aphasia.

The most relevant common signs observed in patients 1 and 2 were the ischemic complications. Few cases of complicated encephalitis with sequelae caused by TOSV have been described (*5*,*6*). Moreover, to our knowledge, persistent neurologic TOSV infection has not been reported. The immune status of these patients probably influenced the clinical outcome in both patients and the delayed serologic response in patient 1.

Patient 3, a 41-year-old woman, sought treatment for exanthema at her health care center in July 2004. Test results for IgM antibodies against rubella, parvovirus B19, and *Rickettsia conorii* were negative. Specific anti-TOSV IgM was detected. The infection was self-limited, and no signs of neurologic involvement were associated with the rash. This was the only case of anti-TOSV IgM detection in 358 serum specimens analyzed from patients with nonneurologic syndromes. Although this finding is not conclusive, it suggests that TOSV infection might be involved occasionally in other mild syndromes. Two other cases of TOSV infection without neurologic involvement have been reported elsewhere: febrile erythema in Italy (*7*) and an influenza-like illness in southern France (*8*).

The unusual manifestations of TOSV infection reported here occurred in persons from rural areas within Granada Province, where seroprevalence rates have been shown to be higher than in urban areas (*3*). These data provide more information about this arboviral infection. Atypical TOSV infection could occur particularly in areas where the virus is endemic.

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References

1. Charrel RN, Gallian P, Navarro-Marí JM, Nicoletti L, Papa A, Sánchez-Seco MP, et al. Emergence of Toscana virus in Europe. Emerg Infect Dis. 2005;11:1657–63.
2. Navarro JM, Fernández-Roldán C, Pérez-Ruiz M, Sanbonmatsu S, de la Rosa M, Sánchez-Seco MP. Meningitis by Toscana virus in Spain: description of 17 cases [in Spanish]. Med Clin (Barc). 2004;122:420–2. DOI: 10.1157/13059543
3. Sanbonmatsu-Gámez S, Pérez-Ruiz M, Collao X, Sánchez-Seco MP, Morillas-Márquez F, de la Rosa Fraile M, et al. Toscana virus in Spain. Emerg Infect Dis. 2005;11:1701–7.
4. Pérez-Ruiz M, Collao X, Navarro-Marí JM, Tenorio A. Reverse transcription, real-time PCR assay for detection of Toscana virus. J Clin Virol. 2007;39:276–81. DOI: 10.1016/j.jcv.2007.05.003
5. Baldelli F, Ciufolini MG, Francisci D, Marchi A, Venturi G, Fiorentini C, et al. Unusual presentation of life-threatening Toscana virus meningoencephalitis. Clin Infect Dis. 2004;38:515–20. DOI: 10.1086/381201
6. Kuhn J, Bewermeyer H, Hartmann-Klosterkoetter U, Emmerich P, Schilling S, Valassina M. Toscana virus causing severe meningoencephalitis in an elderly traveller. J Neurol Neurosurg Psychiatry. 2005;76:1605–6. DOI: 10.1136/jnnp.2004.060863
7. Portolani M, Sabbatini AMT, Beretti F, Gennari W, Matassia MG, Pecorari M. Symptomatic infections by Toscana virus in the Modena province in the triennium 1999–2001. Microbiologica. 2002;25:485–8.
8. Hemmersbach-Miller M, Parola P, Charrel RN, Paul Durand J, Brouqui P. Sandfly fever due to Toscana virus: an emerging infection in southern France. Eur J Intern Med. 2004;15:316–7. DOI: 10.1016/j.ejim.2004.05.006

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Sporadic Oropouche Virus Infection, Acre, Brazil

To the Editor: *Oropouche virus* (OROV), a member of the *Bunyaviridae* family, *Orthobunyavirus* genus, Simbu serogroup, is transmitted to humans in urban areas by the biting midge *Culicoides paraensis* and causes epidemic acute febrile disease (*1*). Since its first isolation in Trinidad in 1955 (*2*), OROV has been associated with large outbreaks in South and Central America; half a million cases have been described during the past 45 years (*1*). The tripartite genome of OROV comprises single-strand, negative-sense large (L), medium (M), and small (S) RNAs that encode RNA polymerase, glycoproteins, and nucleocapsid, respectively. Studies have indicated the existence of 3 genotypes of OROV circulating in Brazil: genotypes I and II in the Amazon Basin and genotype III in the Southeast Region (*3*–*5*).

OROV causes explosive urban epidemics. Serologic evidence of exposure to OROV in populations not affected by known outbreaks suggests that the virus circulates endemically (*1*). However, no sporadic infections have been reported. Here we report a sporadic OROV infection detected by clinical and laboratory surveillance of acute febrile illnesses in Acre, a state in the western Amazon region of Brazil. From March 2004 through October 2006, we prospectively investigat-

ed 69 febrile episodes in persons 6–60 years of age (mean, 28.1 years) living in the town of Acrelândia (10°13′W, 67°00′S) and surrounding rural areas (25.7% and 74.3% of the sample, respectively).

Serum samples for reverse transcription–PCR (RT-PCR) were stored in liquid nitrogen in the field and shipped on dry ice to the laboratory in São José do Rio Preto, 3,500 km southeast of Acre. Because malaria and several arboviruses are locally endemic (*6*), all patients were screened for malarial parasites by thick-smear microscopy and for flaviviruses and alphaviruses by multiplex-nested RT-PCR (*7*). The samples negative for both malaria and other arboviruses were further tested for OROV with primers targeting the S segment of the OROV genome in a seminested RT-PCR strategy (R.V.M. Bronzoni et al., unpub. data; primers and protocol available from the authors by request). The sample also was isolated in Vero cells, and the RT-PCR described by Moreli et al. (*8*) was used for confirmation.

We sequenced amplicons by using the same primers used for RT-heminested amplification and by using BigDye Terminators version 3.1 (ABI, Foster City, CA, USA) in ABI377 automated sequencer. Sequences were edited by DSGene 2.0 (Accelrys, San Diego, CA, USA) and deposited in GenBank (accession no. EU561644). One (1.4%) of 69 samples tested for OROV by heminested PCR was positive. This sample (BR/2004/ACRE27) was collected from a male patient from a rural area in April 2004. Precautions were followed to avoid contamination; positive and negative controls were used in all reactions; and the procedure was reproduced several times. The patient had ill-defined, mild flu-like symptoms; low-grade fever; and nasal discharge but reported no headache or other major symptoms. He recovered without complication.

We built a phylogenetic tree on the basis of the 522 nucleotide sequences (27–200 aa) of nucleocapsid protein gene of OROV sample BR/2004/ACRE27 and other GenBank sequences from different OROV genotypes. We used sequences from Aino, Akabane, and Tinaroo viruses as the outgroup. A phylogenetic analysis was performed by the neighbor-joining method by using the Kimura 2-parameter nucleotide substitution model (*9*).

The tree showed 3 main clades, corresponding to genotypes I, II, and III, and BR/2004/ACRE27 grouped within genotype I strains (Figure). Both genotypes I and II have been described in OROV outbreaks in Acre; genotype I, however, is found mostly in Pará in the eastern part of the Brazilian Amazon region.

A baseline serologic survey in rural Acrelândia during March and April 2004 detected antibodies to OROV in 6 (1.7%) of 357 persons 5–90 years of age who were examined by microplaque hemagglutination inhibition (*10*). Because none of these persons had been exposed to known OROV outbreaks in Acre or elsewhere, these findings further suggest the sporadic circulation of OROV in the area.

We describe a sporadic infection of OROV infection in the Amazon region of Brazil in a mildly symptomatic patient. The nucleocapsid gene of the isolate has been sequenced, placing it in the genotype I group, the most commonly found in the Amazon Basin. These data suggest that OROV circulation may be sporadic and clinically

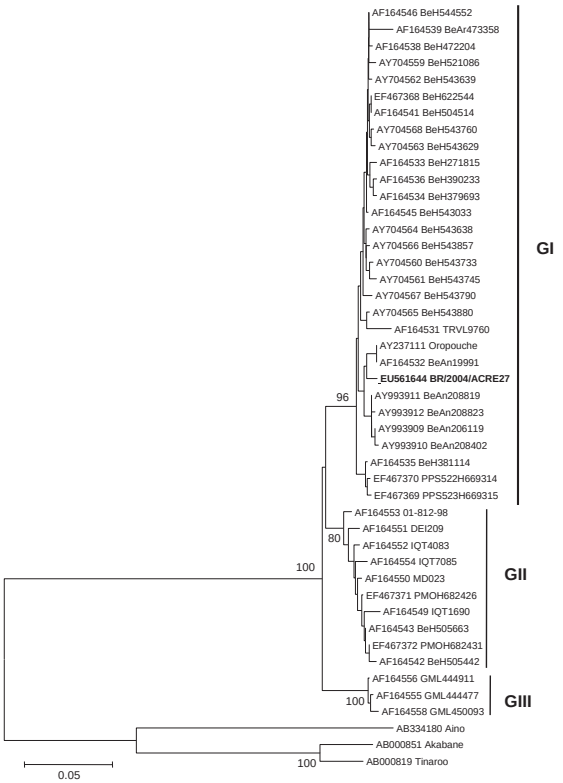

Figure. Phylogenetic tree of Oropouche virus strains; **boldface** shows the sample from the patient in this study. Phylogenetic tree was constructed from partial nucleocapsid gene sequence (522 nt, 27–200 aa) by neighbor-joining method implemented in MEGA 3.0 software (*9*). Kimura 2-parameter nucleotide substitution model was used, and the reliability of the branching patterns was tested by 1,000 bootstrap pseudo replicates. Bootstrap values (%) are shown in main nodes. *Aino*, *Akabane*, and *Tinaroo* viruses were used as the out group. The scale bar represents 5% nucleotide sequence divergence. GenBank accession numbers are provided and are grouped by strain designation. GI, genotype I; GII, genotype II; GIII, genotype III.

silent and, when not associated with outbreaks, most likely neglected by local physicians.

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References

1. Pinheiro FP, Travassos da Rosa APA, Vasconcelos PFC. Oropouche fever. In: Feigin RD, editor. Textbook of pediatric infectious diseases. Philadelphia: WB Saunders Co.; 2004. p. 2418–23.
2. Anderson CR, Spence L, Downs WG, Aitken THG. Oropouche virus: a new human disease agent from Trinidad, West Indies. Am J Trop Med Hyg. 1961;10:574–8.
3. Saeed MF, Wang H, Nunes M, Vasconcelos PF, Weaver SC, Shope RE, et al. Nucleotide sequences and phylogeny of the nucleocapsid gene of Oropouche virus. J Gen Virol. 2000;81:743–8.
4. Nunes MR, Martins LC, Rodrigues SG, Chiang JO, Azevedo RSS, da Rosa AP, et al. Oropouche virus isolation, southeast Brazil. Emerg Infect Dis. 2005;11:1610–3.
5. Azevedo RSS, Nunes MRT, Chiang JO, Bensabath G, Vasconcelos HB, Pinto AYN, et al. Reemergence of Oropouche fever, northern Brazil. Emerg Infect Dis. 2007;13:912–5.
6. Silva-Nunes M, Malafronte RS, Luz BA, Souza EA, Martins LC, Rodrigues SG, et al. The Acre Project: the epidemiology of malaria and arthropod-borne virus infections in a rural Amazonian population. Cad Saude Publica. 2006;22:1325–34. DOI: 10.1590/S0102-311X2006000600021
7. de Morais Bronzoni RV, Baleotti FG, Nogueira RMR, Nunes M, Figueiredo LTM. Duplex reverse transcription-PCR followed by nested PCR assays for detection and identification of Brazilian alphaviruses and flaviviruses. J Clin Microbiol. 2005;43:696–702. DOI: 10.1128/JCM.43.2.696-702.2005
8. Moreli ML, Aquino VH, Cruz AC, Figueiredo LT. Diagnosis of Oropouche virus infection by RT-nested-PCR. J Med Virol. 2002;66:139–42. DOI: 10.1002/jmv.2122
9. Kumar S, Tamura K, Nei M. MEGA3: integrated software for molecular evolutionary genetics analysis and sequence alignment. Brief Bioinform. 2004;5:150–63. DOI: 10.1093/bib/5.2.150
10. Shope RE. The use of a micro-hemagglutination test to follow antibody response after arthropod-borne virus infection in a community of forest animals. [Rio J]. Ann Microbiol. 1963;11:167–71.

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Meningitis Caused by *Streptococcus suis* Serotype 14, North America

To the Editor: *Streptococcus suis* is an opportunistic pathogen that can cause serious systemic infections in pigs and occupation-related infections in humans who work in close contact with pigs or pork by-products. Most *S. suis* organisms isolated from diseased pigs belong to serotypes 1–8 (*1*). The most prevalent strain worldwide is serotype 2, which causes invasive infections in pigs and humans (*2*). We report a case of human meningitis caused by *S. suis* serotype 14.

The patient was a 59-year-old woman from rural Manitoba, Canada; she worked at a hog plant and handled 300–400 piglets/day. In October 2007, when she sought care, she had a 2-day history of fever, vomiting, headache, neck pain, and reduced consciousness. She was febrile and confused and had meningeal signs. Leukocyte count was 19,900/mm^3. Cerebrospinal fluid (CSF) had 284 × 10^6/L leukocytes (59% lymphocytes, 41% polymorphonuclear cells), 2.3 mmol/L glucose, and 1.85 g/L total protein. Gram stain of CSF showed gram-positive cocci in pairs; cefotaxime and vancomycin were prescribed empirically. Results of computed tomography of the head, chest radiograph, and transesophageal echocardiogram were within normal limits. Blood culture was negative after 5 days of incubation. The CSF culture grew small α-hemolytic colonies on blood agar and chocolate agar. The organisms were gram-positive cocci in chains, were catalase negative, and were identified as *S. suis* by Vitek II and API 20 Strep System (both from bioMérieux, St.-Laurent, Quebec City, Canada).

Identification of the organism as *S. suis* was confirmed at the National Microbiology Laboratory, Winnipeg, Manitoba, Canada, by conventional