

73.5% to 81.9% nucleotide sequence identity and from 90.1% to 96.9% amino acid sequence identity (Table). This level of sequence divergence, as well as the geographic specificity of this hantavirus in French Guiana led us to provisionally name it Maripa virus.

Results of a serologic survey to identify cases of respiratory disease with no evident etiology led us to identify an HPS case-patient in French Guiana who had been infected with a new divergent hantavirus strain. Human hantavirus epidemics are associated with fluctuations of rodent populations caused by climatic, ecologic and environmental changes or with changes in human activities associated with nature or agriculture. Therefore, in this region where 90% of the land is tropical rain forest but in which there is increasing economic development, continuous surveillance for the virus in the human population would be beneficial. Surveys of potential reservoirs may help reduce the risk of viral emergence.

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References

1. Nichol ST, Spiropoulou CF, Morzunov S, Rollin PE, Ksiazek TG, Feldmann H, et al. Genetic identification of a hantavirus associated with an outbreak of acute respiratory illness. *Science*. 1993;262:914–7. DOI: 10.1126/science.8235615
2. da Silva MV, Vasconcelos MJ, Hidalgo NT, Veiga AP, Canzian M, Marotto PC, et al. Hantavirus pulmonary syndrome. Report of the first three cases in Sao Paulo, Brazil. *Rev Inst Med Trop Sao Paulo*. 1997;39:231–4.
3. Lopez N, Padula P, Rossi C, Miguel S, Edelstein A, Ramirez E, et al. Genetic characterization and phylogeny of Andes virus and variants from Argentina and Chile. *Virus Res*. 1997;50:77–84. DOI: 10.1016/S0168-1702(97)00053-1
4. Johnson AM, Bowen MD, Ksiazek TG, Williams RJ, Bryan RT, Mills JN, et al. *Laguna Negra* virus associated with HPS in western Paraguay and Bolivia. *Virology*. 1997;238:115–27. DOI: 10.1006/viro.1997.8840
5. Vincent MJ, Quiroz E, Gracia F, Sanchez AJ, Ksiazek TG, Kitsutani PT, et al. Hantavirus pulmonary syndrome in Panama: identification of novel hantaviruses and their likely reservoirs. *Virology*. 2000;277:14–9. DOI: 10.1006/viro.2000.0563
6. Schmaljohn C, Hjelle B. Hantaviruses: a global disease problem. *Emerg Infect Dis*. 1997;3:95–104. DOI: 10.3201/eid0302.970202
7. Klein SL, Calisher CH. Emergence and persistence of hantaviruses. *Curr Top Microbiol Immunol*. 2007;315:217–52. DOI: 10.1007/978-3-540-70962-6_10
8. Bi Z, Formenty PB, Roth CE. Hantavirus infection: a review and global update. *J Infect Dev Ctries*. 2008;2:3–23. DOI: 10.3855/jidc.317
9. Matheus S, Meynard JB, Rollin P, Maubert B, Morvan J. New World hantavirus in humans, French Guiana. *Emerg Infect Dis*. 2006;12:1294–5.
10. Ksiazek TG, Peters CJ, Rollin PE, Zaki S, Nichol S, Spiropoulou C, et al. Identification of a new North American hantavirus that causes acute pulmonary insufficiency. *Am J Trop Med Hyg*. 1995;52:117–23.

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Fatal Human Case of West Nile Virus Disease, Mexico, 2009

To the Editor: West Nile virus (WNV; family *Flaviviridae*, genus *Flavivirus*) was first recognized in the Western Hemisphere in 1999 during an outbreak of human, equine, and avian encephalitis in New York (1). The virus has since spread across the United States and Canada, where it has caused ≈30,000 human infections and ≈1,000 deaths. Serologic evidence has demonstrated that WNV is present throughout Mexico, Central America, South America, and the Caribbean region (2–8). However, WNV illness in humans and vertebrate animals in these regions has been only sparsely reported. For instance, 7 human cases of WNV infection have occurred in Mexico (excluding the case described here), 3 of which were severe. All patients survived. To our knowledge, no fatal human cases of WNV infection have occurred in Central America, South America, or the Caribbean region.

We describe a fatal case of WNV infection in a human in Central America. The patient, a man 40 years of age, lived in Monterrey, Nuevo León State, in northern Mexico. He had not traveled outside of the metropolitan area in the 6 months before illness onset. On June 11, 2009, influenza-like signs and symptoms (i.e., fever, malaise, fatigue, arthralgia, headache, and dizziness) developed in the patient. On June 26, the signs and symptoms had not resolved, and the man was admitted to University Hospital “Dr. José E. Gonzalez” at the Universidad Autónoma de Nuevo León (UANL). At the time of admission, cerebrospinal fluid (CSF) was collected, and laboratory analysis indicated a markedly elevated leukocyte count (182 cells/mm³; reference range 0–5 cells/mm³) and slightly elevated protein and glucose levels.

Several days later, serious neurologic signs that included loss of consciousness developed in the patient. On July 6, he lapsed into a coma and was transferred to the intensive care unit and treated for intracranial hypertension. Another CSF specimen was collected, and laboratory findings demonstrated that the leukocyte count had increased to 495 cells/mm³. CSF cytologic examination showed atypical lymphocytes, some of which resembled plasma cells. Brain magnetic resonance imaging showed hydrocephalus with no brain parenchymal lesions. Because the patient was suspected to have a herpes simplex virus infection, intravenous acyclovir was administered. Several days later, the patient showed signs of improvement; on July 15, he was discharged. Eleven days later, he experienced severe headaches and, on July 29, was readmitted to the UANL Hospital for intracranial hypertension. On July 30, a ventriculoperitoneal shunt was implanted; however, the patient's condition continued to decline, and he died on August 1.

Personnel in the Laboratory of Molecular Infectology at the UANL were informed of the patient and were provided with the remainder of the second CSF specimen several days before his death. Total RNA and DNA were extracted from the CSF by using the QIAamp viral RNA extraction kit (QIAGEN, Valencia, CA, USA) and DNazol (Invitrogen, Carlsbad, CA, USA) and tested for nucleic acid to various pathogens associated with human central nervous system infections, specifically herpes simplex virus types 1 and 2, human enterovirus A–D, dengue virus types 1–4, WNV, and *Mycobacterium tuberculosis*. Complementary DNA samples were generated by using Superscript III reverse transcription (Invitrogen), and PCRs were performed by using *Taq* polymerase (Invitrogen) in accordance with the manufacturer's instructions. PCR amplifications were conducted by us-

ing the following reaction conditions: 94°C for 3 min; 30 cycles of 94°C for 1 min, 50°C for 1 min, and 72°C for 2 min; followed by a final extension at 72°C for 8 min. Reverse transcription–PCRs performed with diethyl pyrocarbonate–treated distilled water in place of nucleic acid were included as negative controls. All test and control reactions were performed in duplicate. PCR products were examined by 2% agarose gel electrophoresis and visualized with ethidium bromide. A PCR product of the expected size was observed when the WNV-specific primers WN-*cap*-F (5'-CAGT GCTGGATCGATGGAGAG-3') and WN-*cap*-R (5'-CCGCCGATTG ATAGCACT GGT-3') were used. These primers amplify a 104-nt region of the capsid gene. All other assay results were negative. Subsequent reactions were performed by using a second set of WNV-specific primers, WN-*env*-F (5'-GATGTGAAG ATGATGAATATGG-3') and WN-*env*-R (5'-AATGCTTCCTTTGCCAA ATAG-3'), which amplify a 216-nt region of the envelope gene. A PCR product of the expected size was again observed. PCR products were purified by using the Purelink Gel Extraction Kit (Invitrogen) and sequenced by using a 3730×1 DNA sequencer (Applied Biosystems, Foster City, CA, USA).

Because of the small volume of CSF obtained, a comprehensive laboratory analysis (virus isolation, plaque reduction neutralization test) could not be performed. Nevertheless, detection of WNV in the CSF of a patient with encephalitis meets the Centers for Disease Control and Prevention established criteria for a case of West Nile neuroinvasive disease (9). Our findings highlight the fact that the low number of WNV cases in Mexico and elsewhere in Latin America should not deter healthcare personnel from performing WNV diagnostic testing and the public from using personal protective measures in these regions.

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References

1. Nash D, Mostashari F, Fine A, Miller J, O'Leary D, Murray K, et al. The outbreak of West Nile virus infection in the New York City area in 1999. *N Engl J Med*. 2001;344:1807–14.
2. Blitvich BJ, Fernandez-Salas I, Contreras-Cordero JF, Marlenee NL, Gonzalez-Rojas JI, Komar N, et al. Serologic evidence of West Nile virus infection in horses, Coahuila State, Mexico. *Emerg Infect Dis*. 2003;9:853–6.
3. Cruz L, Cardenas VM, Abarca M, Rodriguez T, Reyna RF, Serpas MV, et al. Short report: serological evidence of West Nile virus activity in El Salvador. *Am J Trop Med Hyg*. 2005;72:612–5.
4. de Lourdes Garza Rodriguez M, Rodriguez Rodriguez DR, Blitvich BJ, Lopez MA, Fernandez-Salas I, Jimenez JR, et al. Serologic surveillance for West Nile virus and other flaviviruses in febrile patients, encephalitic patients, and asymptomatic blood donors in northern Mexico. *Vector Borne Zoonotic Dis*. 2009 Jun 3; [Epub ahead of print].

5. Dupuis AP II, Marra PP, Kramer LD. Serologic evidence of West Nile virus transmission, Jamaica, West Indies. *Emerg Infect Dis.* 2003;9:860–3.
6. Estrada-Franco JG, Navarro-Lopez R, Beasley DW, Coffey L, Carrara AS, Travassos da Rosa A, et al. West Nile virus in Mexico: evidence of widespread circulation since July 2002. *Emerg Infect Dis.* 2003;9:1604–7.
7. Mattar S, Edwards E, Laguado J, Gonzalez M, Alvarez J, Komar N. West Nile virus antibodies in Colombian horses. *Emerg Infect Dis.* 2005;11:1497–8.
8. Elizondo-Quiroga D, Davis CT, Fernandez-Salas I, Escobar-Lopez R, Velasco Olmos D, Soto Gastalum LC, et al. West Nile virus isolation in human and mosquitoes, Mexico. *Emerg Infect Dis.* 2005;11:1449–52.
9. Centers for Disease Control and Prevention. Neuroinvasive and non-neuroinvasive domestic arboviral diseases [cited 2010 Jan 12]. http://www.cdc.gov/epo/dphsi/casedef/arboviral_current.htm

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Bartonella spp. Infections, Thailand

To the Editor: *Bartonella* are fastidious hemotropic gram-negative bacteria with a worldwide distribution. In Thailand, *Bartonella* species have been demonstrated in mammalian hosts, including rodents, cats and dogs, and in potential vectors, including fleas (1–4). However, data on human infection have been limited to case reports (5,6) and 1 seroprevalence survey, which found a 5.5% prevalence of past *B. henselae* infection (7). No studies have systematically assessed the frequency, clinical characteristics,

or epidemiology of human *Bartonella* infections in Thailand.

We conducted a prospective study to determine causes of acute febrile illness in 4 community hospitals, 2 in Chiang Rai (northern Thailand) and 2 in Khon Kaen (northeastern Thailand). We enrolled patients ≥ 7 years of age with a temperature $> 38^\circ\text{C}$ who were brought to study hospitals for treatment from February 4, 2002, through March 28, 2003. Patients were excluded if they had a history of fever for ≥ 2 weeks or an infection that could be diagnosed clinically. Acute-phase serum samples were collected at the time of enrollment and convalescent-phase serum samples 3–5 weeks later. We enrolled nonfebrile control patients ≥ 14 years of age who had noninfectious conditions; acute-phase serum samples were collected. Clinical information was abstracted from patient charts. Nurses conducted physical examinations and personal interviews to collect information on patients' demographic characteristics, exposures to animals, and outdoor activities.

Serum samples were tested for immunoglobulin (Ig) G antibodies to *Bartonella* spp. by immunofluorescent antibody assay at the Bartonella Laboratory of the Centers for Disease Control and Prevention, Fort Collins, CO, USA. Strains used for antigen production were: *B. elizabethae* (F9251), *B. henselae* (Houston-1), *B. quintana* (Fuller), and *B. vinsonii* subsp. *vinsonii* (Baker). Homologous hyperimmune serum specimens were produced in BALB/c mice as previously described (8). *Bartonella* infection was considered confirmed in febrile patients who had a ≥ 4 -fold rise in IgG antibody titers and a convalescent-phase titer > 64 . Probable infection was defined as 1) a 4-fold antibody titer rise but convalescent-phase titers of 64, or 2) high and stable titers (≥ 512 in acute-phase and convalescent-phase serum samples), or 3) acute-phase titer ≥ 512 with a ≥ 4 -fold titer fall. Paired serum

samples from febrile patients were also tested for serologic evidence of other common causes of febrile illness in Southeast Asia.

Febrile patients with acute-phase and convalescent-phase IgG antibody titers < 128 were considered not to have *Bartonella* infection; we compared demographic and clinical characteristics of these patients to *Bartonella*-infected patients. To evaluate potential risk factors, we compared *Bartonella*-infected case-patients ≥ 14 years of age without serologic evidence of other infections ($n = 20$) to nonfebrile controls with IgG to *Bartonella* < 128 ($n = 70$). Age adjusted odds ratios (AORs) with 95% confidence intervals (CIs) were calculated.

Serologic testing was completed on paired serum samples for 336 (46%) of 732 febrile patients enrolled; 92 (27%) had serologically confirmed (50) or probable (42) *Bartonella* infections. Thirty-five (38%) of these 92 had serologic evidence of infection with another pathogen. The remaining 57 *Bartonella*-infected case-patients (34 confirmed, 23 probable) had a median age of 19 years (range 7–72 years); 65% were males, 47% were students, and 35% were rice farmers. Common clinical characteristics of *Bartonella*-infected patients included myalgias (83%), chills (79%), and headache (77%). Thirty (60%) patients had anemia (hemoglobin level < 13 mg/dL); 18 (32%) had a hemoglobin level < 12 mg/dL, and 4 (7%) had < 11 mg/dL. When compared with 193 febrile patients without *Bartonella* infection, the 57 *Bartonella*-infected patients were similar in age and sex but were more likely to be rice farmers and were more likely to have leukocytosis (Table). Compared with the 70 nonfebrile controls, *Bartonella*-infected case-patients were more likely to report tick exposure (32% vs. 7.9%; AOR = 5.6, 95% CI 1.5–21) and outdoor activities (55% vs. 31%; AOR = 2.7, 95% CI 1.0–7.4) during the 2 weeks before