Five (15.5%) of the 33 pregnant women had *P. jiroveci* DNA in their nasal swab samples versus none (0%) of the 28 nonpregnant controls (p=0.04 by 1-sided Fisher exact test). Immunologic parameters were not tested. The *P. jiroveci*–positive women were all multiparous with 1 (n=2), 2 (n=2), or 3 (n=1) previous pregnancies.

These results suggest that pregnancy is a host factor that favors asymptomatic nasal carriage of *P. jirovec*. However, PCR detection of *P. jiroveci* DNA in the nares of pregnant women does not necessarily indicate either a mild active pulmonary infection or viable or transmissible organisms. In animal models, detection of *P. carinii* DNA in nasal and oral samples is a good indicator that *Pneumocystis* is in the lungs (8).

These results also support the hypothesis that pregnant women who nasally carry *P. jiroveci* may play a role as contagious sources for susceptible persons, especially their immunologically naive newborn infants. This hypothesis warrants further study. Mother-to-infant transmission may explain the accumulating evidence that the primary infection is widely acquired very early in life (9). Recent animal model studies have documented the early acquisition of *P. carinii* (within 1 to 2 h after birth) in neonatal rats, likely transmitted by the dams (10). Evidence of mother-offspring transmission would be clinically relevant for infants born to HIV-infected mothers, who currently rely on empiric anti-*Pneumocystis* chemotherapy started at 1 month of age as their only prophylactic option.

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First Evidence of *Aedes albopictus* (Skuse) in Southern Chiapas, Mexico

To the Editor: The mosquito *Aedes albopictus* (Skuse, 1894) was first identified in the Americas in Texas in 1985 (1,2). That year, this newly introduced species had dispersed widely in Texas and was implicated in the transmission of dengue virus (3). Later, the first states in Mexico that were infested by *Ae. albopictus* were along the northern Mexican border: Coahuila, Nuevo Leon, and Tamaulipas (4,5; J.P. Martínez-Muñoz, thesis). In 1997, this species was reported farther south in Veracruz (6). Although *Ae. albopictus* was expected to spread to southernmost Mexico, this mosquito has never been reported there until now. We have confirmed *Ae. albopictus* in the city limits of Tapachula, southern Chiapas, Mexico.

On September 13, 2002, one of the authors, who resides in Tapachula, was bitten by a mosquito. He collected the specimen, which was later identified as *Ae. albopictus* by the Centro de Investigación de Paludismo (CIP). Nearby larval habitats were then comprehensively searched to collect the immature stages of the species; the sampling area was located at 14° 55' 22.5" north and 92°15' 05.7" west at an altitude of 220 m along the periphery of Tapachula. We found the following containers with larval stages of mosquitos: five water containers, two discarded tires (con-

taining 300–3,000 mL of water), one thermal bottle (250 mL), one plastic bottle (50 mL), and one bucket (2,500 mL). Larvae were placed in plastic bags and transported to CIP laboratories, where they were allowed to emerge to adults during 17 days. The fourth instar larval and pupal exuvias were fixed and identified to species according to Darsie (7) and Superintendência de Campanhas de Saúde Pública (8). Twenty-five female and male *Ae. albopictus* from these collections are available from CIP laboratory upon request.

Additional field collections are being conducted to establish the distribution range of this species along the Chiapas coastal plain, to determinate the entomologic levels of infestation, and to determine its susceptibility to insecticides. Considering the epidemiologic relevance of this discovery, we have notified the proper health authorities to take necessary control measures to reduce the possibility of increased dengue transmission and to prevent other arboviruses, such as West Nile virus (9), from being spread by this new species in southern Mexico.

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Virus Isolation and "Acute" West Nile Virus Encephalitis (Response to Huang et al.)

To the Editor: We read with interest a recent article in your journal, First Isolation of West Nile virus from a Patient with Encephalitis in the United States (1); in the report, we were unable to ascertain indisputable evidence that this patient had indeed acquired acute West Nile virus (WNV) encephalitis. In animals (2,3) and humans (4), West Nile virus can persist in the host even after the host has recovered from an acute WNV infection, presumably more so in the immunocompromised persons. Therefore, in the case described by Huang et al. (1), proving that the patient did not have a history of WNV infection is important, particularly because this patient is from a geographic area where WNV is known to exist. The findings at autopsy of perivascular lymphocyte cuffing in mammillary bodies of the brain are not the classic findings reported during the West Nile encephalitis outbreak in New York City (5). The immunoglobulin (Ig) G antibody against WNV, if it had been present, would have been useful in that IgG antibody in the absence of IgM antibody is indicative of past rather than acute infection.

The WNV copy numbers in clinical samples and clinical indices (leukocyte count) suggest that the virus multiplies in the setting of leukopoenia or immune suppression and cannot be definitive proof that it was an acute infection, unless a negative preillness sample was available. The cause of the transient viremia, whether acutely acquired or from increased proliferation in a chronic infection, needs to be clarified further. In the future, antigen detection will guide patient management decisions; therefore, the possibility of a human chronic carrier state warrants study.

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