

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 exuviae 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 determine 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 leukopenia 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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“Acute” West Nile Virus Encephalitis (Response to Krishnamoorthy et al.)

To the Editor: In a letter to the editor, Krishnamoorthy et al. question the diagnosis of “acute West Nile encephalitis” in our case report. We did not use the word “acute” in the paper, but the patient did in fact have

an acute illness. We believe that this case report, in which West Nile virus (WNV) was isolated in cell culture, represents the best evidence for a WNV infection in a human in the United States. The diagnosis of West Nile encephalitis was based on clinical analysis (1); not everyone with the diagnosis undergoes an autopsy. In many instances, patients do recover. In our case, the patient had the clinical features of encephalitis consisting of unremitting fever associated with a rapid course of progressive confusion and lethargy followed by coma. In addition, increased depression of respiratory drive existed, pointing to brain stem involvement. We agree that the inflammatory changes in the brain were limited as compared to such changes in other reported cases of WNV; however, this limitation was attributable to the fact that the patient was both immunocompromised and neutropenic at the time of acute infection. Therefore, the usual inflammatory response cannot be expected. Even though the changes were limited, they were consistent with the histologic findings in previously published reports (1,2).

The second point by Krishnamoorthy et al. represents their hypothesis about a human chronic carrier state for WNV. Although a chronic carrier state is possible, the viremic period associated with arboviral infections is typically short (3). While one cannot rule out persistent infection with WNV, until our report attempts to recover the virus by isolation in North America in humans have been uniformly unsuccessful. Also, previous reports of successful WNV isolations by Israeli investigators in immunocompetent hosts (4) have been from blood specimens before seroconversion. These considerations indicate that the virus is not routinely found in the blood in substantial amounts by the time clinical symptoms consistent with WNV infection occur. We do not know, nor have we speculated, about the timing of the infection as the patient had no recollection of a mosquito bite. Tests for both immunoglobulin (Ig) G and IgM antibodies to WNV were negative in our patient. Because the patient was immunocompromised, a humoral response was not expected; therefore, this information cannot be used as

The image shows a screenshot of the CDC Emerging Infectious Diseases Journal homepage. The page features a navigation menu on the left, a main content area with sections for 'Emerging Infectious Diseases', 'Content Highlights', 'Current Issue', 'Perspectives', 'Synopsis', and 'Research'. A large, stylized 'SEARCH EID ONLINE' graphic is overlaid on the right side of the screenshot. Below the screenshot, the URL 'www.cdc.gov/eid' is displayed in a large, bold, black font.