**Title:** Japanese Encephalitis - A Plague of the Orient (Editorial) (Unclassified)

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**Type of Report:** Editorial

**Date of Report:** Sept. 1986

**Page Count:** PP. 641-643

**Abstract:** N/A

**COSATI Codes:**

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**Subject Terms:** (Continue on reverse if necessary and identify by block number)

**Distribution Statement:** Approved for public release. Distribution Unlimited

**Form:** DD Form 1473, Jun 86
Could Japanese encephalitis be introduced and spread in the United States? Although indigenous mosquitoes, such as *Culex tarsalis*, are efficient vectors, the risk of introduction seems exceedingly remote. Nevertheless, the recent repeated importations of larvae of the Asian tiger mosquito (*Aedes albopictus*) in used tires has raised concern, since Japanese encephalitis virus is known to be vertically transmitted through the eggs of this species.

Americans were first affected by Japanese encephalitis in Okinawa in 1945. Hundreds of clinical cases occurred in American soldiers during the Korean War, and nearly 50 percent of troops in the Pusan perimeter had serologic evidence of infection. In 1969, at least 10,000 Americans were infected in Vietnam, and 57 encephalitic cases were recorded. This history underlies the military interest in Japanese encephalitis, but it also emphasizes the high ratio of subclinical cases to overt infection (200:1 to 300:1). Since 1981, because of a small number of cases among travelers, an American civilian acquired the disease in China, but the epidemic proportions of the disease in Asia have increased attention has been paid to the risks associated with travel. Physicians in the United States should thus be aware of the clinical and epidemiologic features of the disease and of the characteristics of available vaccines.

The most important risk factor for infection of travelers is exposure to *Culex tritaeniorynchus* mosquitoes, which is most likely in the late afternoon to early evening in areas of rice cultivation. Although principally rural, rice paddies are also commonly found on the outskirts of large Asian cities.

After an incubation period of 7 to 14 days, the clinical illness begins with rapid onset of fever, chills, malaise, headache, nausea, and vomiting. This prodromal phase lasts several days and is followed by symptoms and signs of generalized central nervous system infection, including nuchal rigidity, photophobia, confusion, delirium or stupor, generalized convulsions, tremors, muscular rigidity, masklike facies, and localized paresis, generally of the upper-motor-neuron type. A peripheral leukocytosis with relative lymphopenia is typical. The cerebrospinal fluid is under increased pressure and contains mildly elevated concentrations of protein and up to 500 lymphocytes per microliter.

No specific therapy is available. Approximately 10 percent of patients die despite optimal care; the higher fatality rates of 30 to 70 percent reported during epidemics reflect the lack of supportive therapy and recognition of only the most severe cases. Elderly persons are more prone to fatal infection. Neurologic sequelae, including intellectual and emotional changes and motor impairment, occur in up to 80 percent of survivors, especially children. Poor prognostic indicators include prolonged fever, a severely depressed sensorium, and an absence of the lucid interval.
Japanese encephalitis virus has been cloned and sequenced, and expressed in yeast and bacteria. These approaches offer the promise of future vaccines that are inexpensive as well as efficacious and safe.

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REFERENCES