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Rapid spread of emerging Zika virus in the Pacific area

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Zika virus (ZIKV) is an emerging arthropod-borne virus (arbovirus) belonging to the family *Flaviviridae* and genus *Flavivirus*. ZIKV was first isolated from a monkey in the Zika forest of Uganda in 1947 [1].

Subsequently, sporadic human infections were reported in Africa and Asia. In 2007, the first large documented ZIKV outbreak was reported from Yap State, Federated States of Micronesia [2]. No further transmission was identified in the Pacific until October 2013, when French Polynesia (FP) reported the first cases; a subsequent explosive outbreak resulted in an estimated 28 000 cases seeking medical care (approximately 11% of the population) [3,4].

Phylogenetic analyses demonstrated that the FP strain was closely related to Cambodia 2010 and Yap State 2007 strains, corroborating previous findings of the expansion of the ZIKV Asian lineage [3].

During the FP outbreak, most clinical cases presented with mild disease characterized by low-grade fever, maculopapular rash, arthralgia, and conjunctivitis.

In November, a patient presented with Guillain–Barre syndrome (GBS), an autoimmune disease causing acute or subacute flaccid paralysis, 1 week after a confirmed acute ZIKV infection [5]. Subsequent GBS cases were identified, correlating temporally with the ZIKV outbreak. The incidence rate of GBS cases during the ZIKV outbreak was approximately 20-fold higher than expected given the size of the FP population and the established incidence rates of GBS (1–2/100 000 population per year) [6].

No severe disease resulting from ZIKV infection had been reported prior to the FP outbreak, but previous clinical characterization was based on a limited number of confirmed cases. The recent temporal and spatial association between the FP ZIKV outbreak and the highly unusual GBS cluster is very

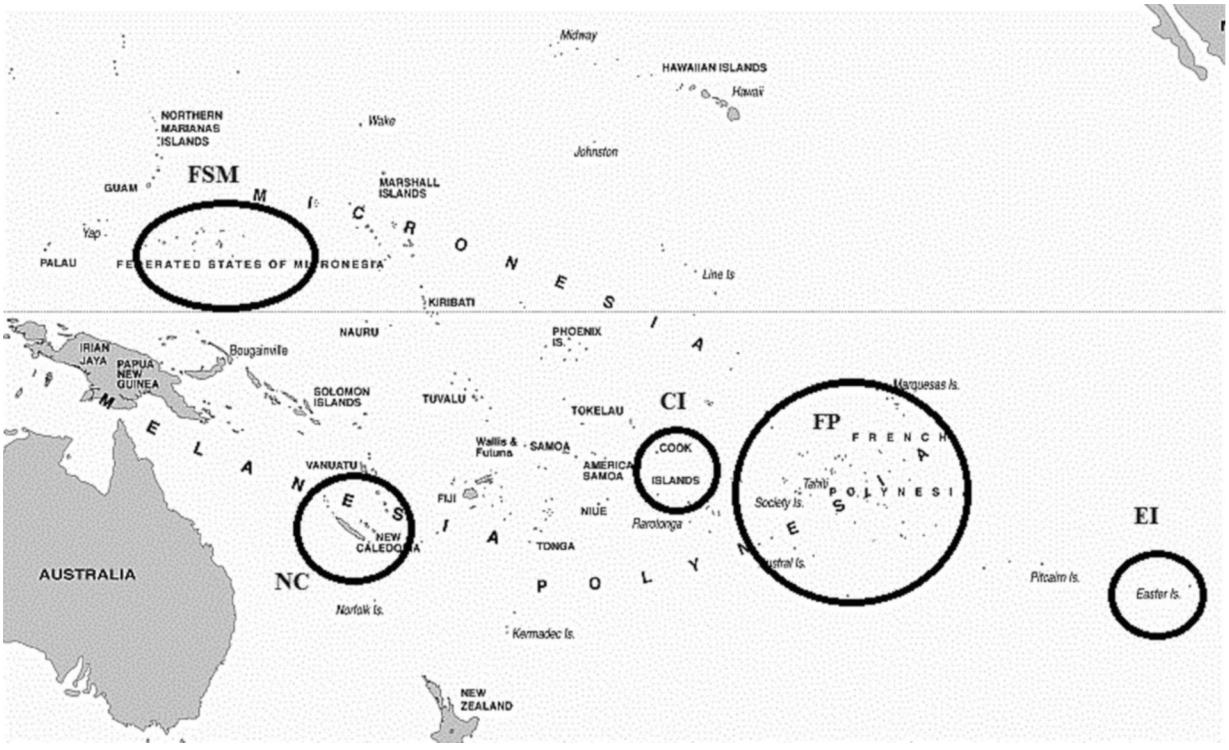

FIG. 1. Circulation of Zika virus in the Pacific: FSM (Federated States of Micronesia, 2007), FP (French Polynesia, 2013/2014), NC (New Caledonia, 2014), CI (Cook Islands, 2014), EI (Ester Island, 2014).

suspicious, but does not confirm ZIKV as the antigenic stimulus predisposing to this autoimmune disease.

ZIKV is transmitted by the bite of infected mosquitoes, and has been isolated from several *Aedes* mosquito species [7], notably *Aedes aegypti*, which is widespread in the tropics and subtropics, and *Aedes albopictus*, which is established in many parts of Europe, especially in Mediterranean countries. In FP, *Aedes polynesiensis* is also suspected to contribute to ZIKV transmission.

Non-vector borne ZIKV transmission through sexual intercourse [8] and perinatal transmission [4] has been reported. Given that transfusion-related ZIKV transmission is a potential risk, molecular screening was implemented in FP for blood donors during the outbreak: 2.8% of blood donors, who were asymptomatic at the time of donation, tested positive for acute ZIKV infection [9].

Following the FP outbreak in late 2013, there were subsequent outbreaks in New Caledonia, the Cook Islands, and Easter Island [10] (Fig. 1). Because of the typically mild clinical symptoms, limited ZIKV diagnostic capacity, and overlapping clinical features of ZIKV, dengue, and chikungunya, which are also circulating in the Pacific, we believe that ongoing and undetected ZIKV transmission in other Pacific island countries, and potentially beyond, is highly probable. The observation that severe clinical complications may occur highlights the need to strengthen surveillance for this emerging virus, and, in the event of a ZIKV outbreak, establish rigorous clinical monitoring to detect GBS or other unusual clinical manifestations.

Transparency Declaration

The authors declare no conflicts of interest.

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