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NEWS & VIEWS

Zika virus: a flavivirus caused pandemics in Latin America

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Since early 2015, an unprecedented outbreak of Zika virus (ZIKV) infection that recognized in northeast Brazil has spread to Latin America (Schuler-Faccini, 2016). As of January 2016, there has been confirmed autochthonous transmission of ZIKV in 19 countries in the Americas outside Brazil (Hennessey, 2016). In September 2015, reports from physicians of an unusual increase in the number of newborn babies with microcephaly in ZIKV-affected regions emerged. In October 2015, the researchers of Brazil confirmed the increase in birth prevalence of microcephaly in northeast Brazil (Schuler-Faccini, 2016) (Figure 1). According to Brazilian health authorities, more than 3,500 microcephaly cases were reported between October 2015 and January 2016 (http://www.cdc.gov/media/releases/2016/s0315-zika-virus-travel. html), which outnumbered the previously reported estimates. However in recent years, the incidence of microcephaly have been around 180 cases per year in Brazil (Triunfol, 2016). It was evidenced that ZIKV infection could be a culprit. ZIKV RNA was found in the amniotic fluid of two women whose fetuses had been diagnosed with microcephaly via prenatal ultrasound, suggesting that it had crossed the placenta and could cause a mother-to-child infection (Triunfol, 2016). It is noteworthy that the number of Guillain–Barré syndrome cases, a rare neurological disorder that can cause temporary paralysis or even death, is markedly increased in other Americas countries (such as Colombia and Venezuela) where ZIKV is currently circulating (World Health Organization, 2016). If this is the case that ZIKV is causing birth defects or neurological conditions, it would be a serious public health concern.

ZIKV is a mosquito-borne flavivirus, a genus that also consists of West Nile virus (WNV), dengue virus (DENV), Japanese encephalitis virus (JEV), and yellow fever virus (YFV) (Kuno et al., 1998). It is a positive-sense, single-stranded RNA virus of approximately 11 kb in genome length. ZIKV is a member of the Spondweni serocomplex and has evolved into three separate genotypes (West African, East African and Asian) based on the phylogenetic relationships (Haddow et al., 2012; Lanciotti et al., 2016). Since the first discovery of ZIKV in Uganda in 1947, only 14 human cases of Zika virus disease have been reported and all were restricted to Africa

Figure 1. An unprecedented outbreak of Zika virus (ZIKV) infection, which may have caused a mother-to-child infection and fetuses with microcephaly, has spread widely in Latin America since early 2015.

and Southeast Asia, such as Uganda, Nigeria, Senegal, and Indonesia (Moore et al., 1975; Olson and Ksiazek, 1981; Duffy et al., 2009; Enserink, 2015). Until 2007, 49 confirmed and 59 probable cases of Zika virus illness in Yap Island, Federated States of Micronesia, represent the transmission of ZIKV outside the regions of Africa and Asia (Duffy et al., 2009). Subsequently, ZIKV caused a major epidemic-close to 30,000 people infected-in the French Polynesia during 2013-2014 (Van-Mai et al., 2014). As of December 2015, approximately 440,000 to 1,300,000 human may have been infected by ZIKV in Brazil (Hennessey, 2016). It seems that ZIKV become more epidemic in human populations. The adaptation of ZIKV to an urban cycle should be of great concern to public health issue.

ZIKV is believed to be transmitted to humans by many *Aedes spp*. mosquitoes, including *Aedes aegypti*, *Aedes africanus*, and *Aedes luteocephalus* (Marchette et al., 1969; Lamb et al., 2001). Two other diseases that are disseminated by the same species of *Aedes* mosquitoes, have caused vast epidemics in Latin America, DENV and chikungunya virus (CHIKV) (Enserink, 2014; Enserink, 2015). Therefore, a new challenge that ZIKV is co-circulating with these two arboviruses in South America has arisen. The majority of ZIKV-infected people are asymptomatic, and only very few develop the mild clinical symptoms, such as headaches, maculopapular rash, fever, conjunctivitis, and arthralgia (Lanciotti et al., 2008), which can be confused with other infectious diseases, especially induced by DENV and CHIKV (Musso et al., 2015). For serological diagnosis, IgM ELISA is a relatively sensitive and specific assay for detecting arboviral infections. However, the ELISA (viral antigens: ZIKV or DENV 1-4 mixture) for IgM antibody against ZIKV could cross-react with that against DENV (Lanciotti et al., 2008; Duffy et al., 2009).

To date (as of 28 February 2016), eight imported cases of ZIKV infection have been reported in mainland China, and infection was also recently identified in Japan (Shinohara et al., 2016). Furthermore, ZIKV have been prevailing in Southeast Asian for a long time and may be experiencing a geographic expansion event within Asian lineage (Haddow et al., 2012; Lanciotti et al., 2016). It is likely that ZIKV infection cases could be greatly underestimated or misdiagnosed in Asia, due to the symptoms clinically similar to dengue fever and many other tropical infectious diseases (Haddow et al., 2012). There are no efficient drugs or vaccines available for ZIKV protection and therapy. Additionally, the links between ZIKV infection and brain damage in fetus are not well determined. Thus, many efforts should be made to answer these unresolved questions. The pandemics of ZIKV in South America underscore the need for investigations of underlying mechanism of viral replication and pathogenesis, as well as the correlation between evolutionary dynamics and epidemiology.

FOOTNOTES

The authors declare that they have no conflict of interest. This article does not contain any studies with human or animal subjects performed by any the authors.

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