Chinese scientists reveal mystery of Zika virus

[File Photo: sohu.com]

Chinese researchers said Thursday they might have solved the mystery of why the Zika virus causes microcephaly, a birth defect marked by small head size that can lead to severe developmental problems in babies.

In a study published in the U.S. journal Science, a team led by Cheng-Feng Qin of the Beijing Institute of Microbiology and Epidemiology reported that one single genetic change, likely acquired in 2013, gave the mosquito-borne virus the ability to cause severe fetal microcephaly.

“Our findings offer a reasonable explanation for the unexpected causal link of Zika to microcephaly, and will help understand how Zika evolved from an innocuous mosquito-borne virus into a congenital pathogen with global impact,” Qin said.

Zika was first identified in 1947 in Uganda, and until its recent emergence in the Americas, was a little known one that sporadically causes mild infections.

Then, it rapidly swept through South and Central America in 2015, and due to its link to congenital brain abnormalities, especially microcephaly during pregnancy, the World Health Organization declared in early 2016 the current epidemics a public health emergency of international concern.

However, scientists remain unable to determine why the virus evolved into a pathogen triggering severe neurological syndromes.

By comparing contemporary Zika virus strains from the 2015 and 2016 South American epidemics with an ancestral Cambodian virus that was circulating in

2010, Qin and colleagues found one critical mutation that conferred the ability to cause microcephaly in mouse models of fetal infection.

That one change, S139N, which replaced a serine amino acid with an asparagine at the 139th position of a Zika protein called prM, also made the virus more lethal to human neuron precursor cells in culture compared with the ancestral form.

Zika accumulated numerous changes throughout its genome between 2010 and 2016, of which S139N caused substantially more severe microcephaly and embryonic lethality in mouse models.

Evolutionary analyses revealed that the S139N change likely arose sometime around 2013, which coincided with initial reports of microcephaly.

It was then stably maintained during subsequent spread to the America.

“The discovery should provide guidance for the study of pathogenetic mechanisms of the Zika virus and for the development of vaccines and treatments,” Qin said.