

Zika Virus 101: A Mini Review

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Abstract

Zika virus is a mosquito-borne Flavivirus that is the focus of an on going pandemic and public health emergency. The Zika virus outbreak in Brazil in 2015, previously limited to sporadic cases in Africa and Asia, heralded a rapid spread across the Americas. Although most Zika virus infections are characterized by subclinical or mild flu-like illness, severe manifestations have been reported, including Guillain-Barre syndrome in adults and microcephaly in children born to infected mothers. There is no effective treatment or vaccine for the Zika virus; therefore, the public health response is primarily focused on preventing infections, especially in pregnant women. Despite the growing knowledge of this virus, questions remain regarding the vectors and reservoirs of the virus, pathogenesis, genetic diversity, and potential synergistic effects of coinfection with other circulating viruses. These questions highlight the need for research to optimize surveillance, patient management, and public health interventions in the current Zika virus epidemic .Zika virus (ZIKV) is an emerging arthropod-borne Flavivirus that leads to teratogenic effects and neurological disorders after infection. ZIKV infections are a serious global public health problem, leading scientists to increase research into antiviral and vaccines against the virus. These efforts are still on going, as the pathogenesis and immune evasion mechanisms of ZIKV are not yet fully understood. There are currently no specific vaccines or drugs approved for ZIKV; however, some are undergoing clinical trials. Notably, several strategies have been used to develop antiviral, including drugs that target viral and host proteins. In addition, drug reuse is preferred because it is cheaper and less time consuming than other strategies because the drugs used have already been approved for human use. Similarly, various platforms for vaccine design have been evaluated, including DNA, mRNA, peptide, protein, viral vectors, virus-like particles (VLPS), inactivated viruses, and live attenuated virus vaccines. These vaccines have been shown to induce specific humoral and cellular immune responses and reduce viremia and viral RNA in vitro and in vivo. Importantly, most of these vaccines have entered clinical trials. Understanding the mechanism of viral disease will provide better strategies for the development of therapeutic agents against ZIKV. This review provides a comprehensive summary of the viral pathogenesis of ZIKV and current progress in the development of vaccines and drugs against this virus.

Introduction

Zika virus is a flavivirus that was first isolated in 1947 from a febrile rhesus macaque in the Zika forest of Uganda and later identified in *Aedes africanus* mosquitoes from the same forest. In 1954, the first 3 cases of human infection were reported in Nigeria (2). Serologic studies in humans indicate that Zika virus is widespread throughout Africa, Asia, and Oceania (Technical Appendix Table). However, these studies may overestimate the true prevalence of the virus due to the serological overlap between Zika virus and related flaviviruses such as dengue virus (DENV) and West Nile virus (WNV). The virus was first isolated in April 1947 from a rhesus macaque monkey. A scientist placed in a cage in the Zika forest of Uganda near Lake Victoria Yellow Fever Research Institute a second isolate from the mosquito *A. africanus*. Africans were watching art in the same places in January 1948 when the monkey contracted fever, researcher isolated from its serum a "filterable transmission agent" that was named Zika in 1948. Uganda, published in 1952. The first major outbreak of disease caused by Zika infection was reported from Yap Island in 2007. There are several countries that suffer from the Zika virus. A major complication of Zika virus infection is the risk of microcephaly and other congenital brain anomalies in infected pregnant women. The World Health Organization (WHO) has drawn attention to this condition because most cases of microcephaly and defects in newborns caused by the Zika virus have been recorded in economically deprived countries. The organization stressed the need to properly educate the public on preventive measures to prevent a similar phenomenon in the future. In outbreaks over the past decade, Zika virus infection has been found to be associated with an increased incidence of Guillain-Barré syndrome. When the Zika virus emerged in the Americas with a large outbreak in Brazil in 2015, an association between Zika virus infection and microcephaly (smaller than normal head size) was first described; in a retrospective review, there were similar findings in French Polynesia. From February to November 2016, the WHO declared a Public Health Emergency of

International Concern (PHEIC) regarding microcephaly, other neurological disorders, and Zika virus, and a causal link between Zika virus and congenital malformations was soon confirmed (1,2). Outbreaks of Zika virus disease have been identified in most of the Americas and other areas with established *Aedes aegypti* mosquitoes. Infections have been detected in travelers from areas of active transmission, and sexual transmission has been confirmed as an alternative route of Zika virus infection. The greatest concern with Zika virus is its potential to cause severe birth defects, particularly microcephaly, a condition where babies are born with abnormally small heads and underdeveloped brains. Zika virus has also been linked to Guillain-Barré syndrome, a rare neurological disorder that can lead to muscle weakness and paralysis. Zika virus remained a global health concern, but the number of cases had decreased compared to the peak years of the outbreak.

Ongoing research aimed at understanding the virus and developing effective countermeasures continues.

A study of the molecular evolution of Zika virus, based on virus strains collected from 4 West African countries between 1947–2007, identified several sites in the Zika virus genome that were under strong negative selection pressure. This finding suggests the frequent deletion of deleterious polymorphisms in functionally important genes and the possibility of recombination, which occurs rarely in flaviviruses. The implications of this finding require further evaluation with respect to viral spread, zoonotic persistence, and epidemiological potential. A study of the molecular evolution of Zika virus based on virus strains collected from 4 West African countries between 1947–2007 identified several sites in the Zika virus genome that were under strong negative selection pressure. This finding suggests frequent deletion of deleterious polymorphisms in functionally important genes and the possibility of recombination, which occurs rarely in flaviviruses. The implications of this finding require further evaluation with respect to virus spread, zoonotic persistence, and epidemiological potential. Zika virus remained a global health concern, but the number of cases had decreased compared to the peak years of the outbreak. Ongoing research aimed at understanding the virus and developing effective countermeasures continues.

Little is had some significant awareness of the pathogenesis of ZIKV since it includes complex connections among viral and host factors. The atomic systems of infection have communications have been contemplated both in vitro and in vivo to give more knowledge into the pathogenesis of ZIKV. The way that ZIKV causes teratogenic impacts conveys an earnest requirement for the fast advancement of antiviral treatments. WNV and Powassan infection (POWV) have likewise been displayed to bring about critical fetal injury in mice. ZIKV contamination starts with a solitary mosquito nibble of a ZIKV-tainted individual. Viral replication starts in the epithelial cells of the mosquito midgut and continues to the salivary organs. The mosquito can spread the infection following a 10-day brooding period, when its spit becomes tainted. The hatching time frame in people is 3-12 days, and side effects show up following 6-11 days. ZIKV is cleared in the span of 24 days in the vast majority of patients.

Following a mosquito chomp from a ZIKV-tainted mosquito, ZIKV contaminates and duplicates in dendritic cells, spreading through the blood to different pieces of the human body. By and large, the infection is self-restricting; notwithstanding, contaminations in pregnant ladies bring about teratogenic impacts. In pregnant ladies, viral contamination stretches out for a more extended period since the infection duplicates in the fetal mind for a really long time with expanding impacts during the early long stretches of pregnancy

Conclusion

Here we have tried to provide a multi-faceted view of Zika virus infection with more emphasis on the disease status in India (Hyderabad). The Zika virus is not fatal, but the complications associated with it, especially in pregnant women and children, can threaten the quality of life. This can easily be avoided with proper precautions and health considerations. With annual outbreaks around the world, all healthcare groups should come up with a proper plan before the peak season.

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