Quanton Biolife Sciences

Tropical Disease Chikungunya

Chikungunya

Chikungunya virus is an alphavirus that is arthropod-borne and transmitted by Aedes mosquitoes. This virus causes a rash, an acute febrile illness, and arthralgia known as chikungunya fever which can develop into potentially chronic and debilitating arthritis that may last for months or years.^{1,2}

Chikungunya virus (CHIKV) is an arthropod-borne alphavirus classified within the *Togaviridae* family, primarily transmitted by *Aedes* mosquitoes. This virus is recognized for inducing an acute febrile illness characterized by fever, rash, and arthralgia, collectively referred to as Chikungunya fever. Following the acute phase, individuals may experience chronic and debilitating arthritic symptoms that can persist for months or even years. Historically, CHIKV was predominantly identified in Africa and Asia, with its initial isolation occurring in the Makonde Plateau of Tanzania in 1952. The term "Chikungunya" is derived from a Makonde expression that translates to "that which bends up." The most significant outbreak recorded took place on Reunion Island between 2005 and 2006, impacting nearly 35% of the local population. Since 2005, the incidence of Chikungunya fever has expanded primarily in tropical and subtropical regions, ultimately reaching the Americas through the Caribbean Island of St. Martin in 2013.^{3,4} Currently, CHIKV is prevalent globally and poses a significant public health challenge. Chikungunya virus (CHIKV) is recognized for its transmission through two distinct cycles: urban and sylvatic. The urban cycle involves the transmission from humans to mosquitoes and back to humans, which is the primary driver of the ongoing epidemic in the Western Hemisphere. In contrast, sylvatic transmission occurs in Africa and involves a cycle of transmission from animals to mosquitoes and then to humans.⁵ Initially, CHIKV was transmitted by the mosquito vector Aedes aegypti; however, the introduction of Aedes albopictus, facilitated by a mutation in the E1 envelope protein, has not only enhanced the virus's fitness in this species but also improved its transmissibility to vertebrates.⁶

The infection process initiated by CHIKV begins with the inoculation and subsequent infection of human epithelial and endothelial cells, as well as primary fibroblasts and monocyte-derived macrophages. Following an initial immune response and subsequent localization in the lymph nodes, CHIKV disseminates through the lymphatic and circulatory systems, leading to significant viremia.

The transport of the virus to target organs, including muscles, joints, liver, and brain, is attributed to infected monocyte-derived macrophages. The inflammatory response, mediated by CD8+ (acute) and CD4+ T lymphocytes, along with pro-inflammatory cytokines, is believed to contribute to the acute symptoms. Additionally, a persistent reservoir of infected monocytes in the joints may play a role in the development of chronic joint disease.^{7,8}

References

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