

Chikungunya virus infection-clinical features and epidemiology

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Keywords	Abstract
Chikungunya virus, cultivation, epidemiology, polyarthralgia	The Chikungunya virus is a problem for people all around the world. This virus is spread by mosquitoes, specifically the Aedes. Aedes albopictus. When people get infected with the Chikungunya virus, they usually have a fever, a lot of pain in their joints, a rash and muscle pain. For some people the pain in their joints can last for a time. The Chikungunya virus has changed over time which has helped it spread easily. There are three types of the Chikungunya virus and one type, the East/Central/South African type is responsible for many of the recent outbreaks, including the ones in India since 2005. Recently two vaccines for the Chikungunya virus have been approved, which is a step forward, in preventing the disease. The Chikungunya virus is still a problem and it is hard to keep track of it and control it. This article talks about what we know about the Chikungunya virus how it affects people and how we can try to prevent it. The Chikungunya virus is an issue and we need to keep working to stop it from spreading. The Chikungunya virus is a challenge but with the new vaccines we have some hope to fight the Chikungunya virus.

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1. Introduction

Chikungunya virus (CHIKV) belongs to the Alphavirus genus of the Togaviridae family and is mostly transmitted by *Aedes* species mosquitoes specially *A. aegypti* and *A. albopictus*. Once entered in blood it starts replicating in various cell type, resulting in increase of viremia within 2 to 12 days. Infection in humans is characterized by acute symptoms of Fever, polyarthralgia and rashes. Approximately 50% of patients develop chronic polyarthralgia that may persist for many months. CHIKV infection is reported

with 1 in 1000 death rate. (Ribeiro dos Santos et al. 2025; O’Driscoll et al. 2021)

Chikungunya structure and classification

Chikungunya virus belongs to genus Alpha virus, family Togaviridae. Robinson (1955) reported first outbreak of Chikungunya infection from Africa. Chikungunya Virus was first isolated in 1953 by Ross (1956).

CHIKV is an enveloped virus with 11,805 bp long +ssRNA genome. The Chikungunya genome code for two poly proteins, one contains non-structural proteins (nsP1, nsP2, nsP3 and nsP4) while other

comprised of structural proteins (E1, E2, E3, 6K and C). Mature proteins are formed after posttranslational processing.

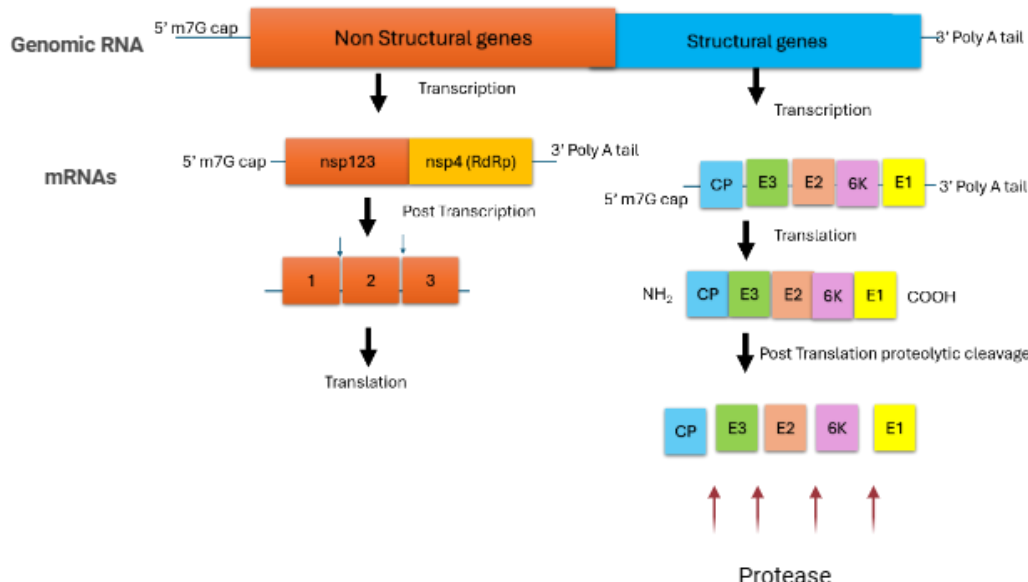


Figure 1: Genome organization of CHIKV. Genome consists of two expression regions i.e. structural and non-structural genes. RNA polymerase read both regions separately transcribing into two polycistronic RNA molecules.

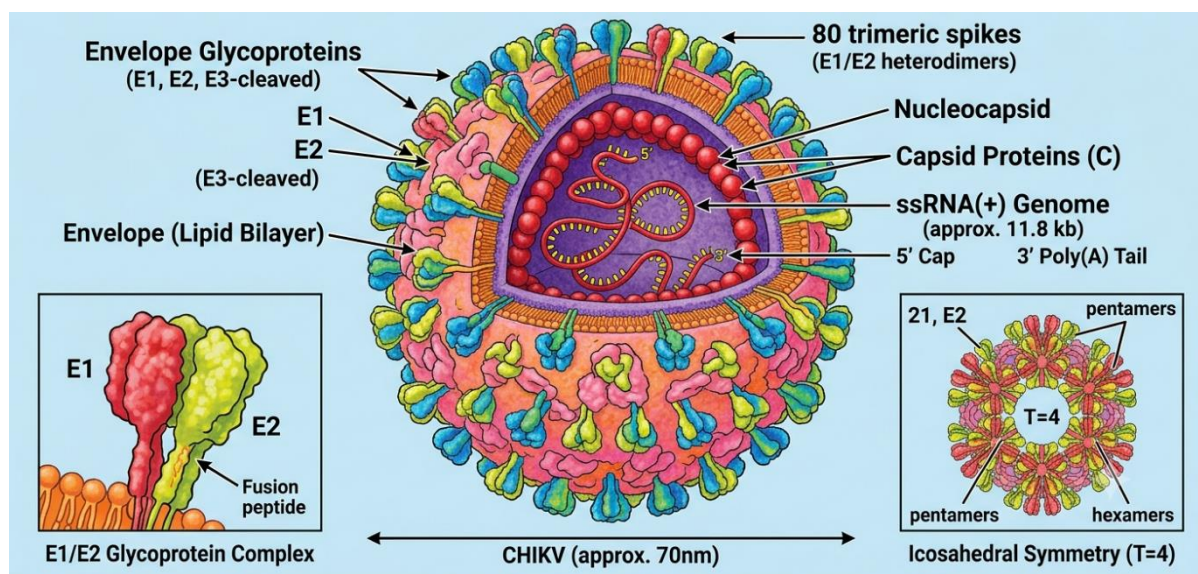


Figure 2: Structure and organization of CHIKV Viron

Genetic variations in these gene products can influence the virulence of virus as well as vector selection. Similar micro evolution of E1 A226V is seen during Indian ocean CHIKV outbreak where Chikungunya virus adopted to *Aedes albopictus* as primary vector than *Aedes aegypti* mosquitoes. E1 gene based phylogenetic analyses revealed the existence of three distinct Chikungunya genotypes: West Africa (African genotype), Asian genotype and Eastern, Central, and Southern African (ECSA

genotype) (Powers et al. 2000).

Clinical features

Incubation period of CHIKV is 2-12 days with generally first symptoms may appear within 48 h of infection. The common clinical symptoms of CHIKV include fever, incapacitating polyarthralgia, muscle pain, and rash. The joint pain is often unbearable and can persist from days to months. Unusual

complications such as hepatitis, pneumonia, mild hemorrhage, pre-renal failure, cardiologic manifestations (heart failure, myocarditis) and neurologic diseases are also seen in Chikungunya patients (Kang et al; Powers et al. 2010; 2024; Ribeiro dos Santos et al. 2025; Singh et al.2020).

Moreover, at least 260 CHIKV-associated fatalities had been reported in La Réunion Island (Josseran et al. 2006). Finally, Cases of severe neonatal CHIKV infection were observed in La Réunion Island, associated with severe disease and encephalopathy, and suggested a possible mother-to-child CHIKV transmission (~3 per 1000 births) (Ramful et al. 2007).

Epidemiology

ECSA genotype is linked to 2005- 2006 massive Chikungunya outbreak at La Reunion Island in Indian Ocean including Mauritius, Madagascar and Seychelles was seen. Later, origin of these strains was linked to Kenya Chikungunya outbreak in 2004 (Parola et al. 2006). Schuffenecker et al. (2006) reported that initially circulating CHIKV strain consist of 226A in E1 protein which during later period of outbreak, changed to A226V. Change E1-A226V was absent in the initial strains but was observed in 90% of subsequent viral sequences from Re'union, denoting evolutionary success possibly due to adaptation to the mosquito vector *A. albopictus* (Simon et al. 2007; Schuffenecker et al. 2006). After initial Chikungunya outbreak in La Reunion Island massive anti mosquito program was launched using pesticides. This results in decline of *A. aegypti* population which was a common Chikungunya vector in this region. Due to environmental pressure favorable replacement of A226 with Valine in CHIKV E1 gene results in adaptation of *A. albopictus* as secondary vector for CHIKV (Thiboutot et al. 2010; Vazeille et al. 2007). E1-A226-V mutation is found to increase the infectivity of CHIKV in *A. albopictus* as compared to *A. aegypti* (Kumar et al. 2008; Lim et al.2025; Simon et al. 2008; Santhosh et al. 2008;

Turell et al. 1992).

During the end of 2005, Chikungunya virus entered in the southern states of India. Initial reports came from the states of Andhra Pradesh and Karnataka in October 2005 (Ravi, 2006). By early 2006, almost all parts of South India including Kerala, Tamil Nadu, Goa, and Maharashtra along with Andaman Nicobar Island were reporting CHIKV infection (Dash et al. 2007, Kumar et al. 2008, Manimunda et al. 2007; Lahariya et al. 2006; Yergolkar et al. 2006). Later, movement of Chikungunya was seen in Central and Western Indian states (Gujarat, Madhya Pradesh and Rajasthan) (Chhabra et al. 2008; Dash et al. 2007). During last quarter of 2006, Chikungunya reaches the northern parts of India, evidenced by reported sporadic cases of CHIKV infection along with massive dengue outbreak in Delhi (Chahar et al. 2009; Kukreti et al. 2008), although only few cases were also reported from Haryana and Uttar Pradesh (NVBDCP). After 2007 nearly all states of India reported Chikungunya infection (NVBDCP). Phylogenetic analysis from southern, central, western and northern Indian Chikungunya virus isolates showed its similarity with Indian Ocean CHIKV strains and all belonged to ECSA genotype (de Souza, et al. 2023; Soni et al. 2013; Singh et al. 2012; Shrinet et al. 2012; Ray et al. 2012; Kumar et al. 2008).

Comparative phylogenetic study from India (Arankalle et al. 2007) using isolates collected from 1963-2006 reveals presence of Thailand strains (Asian genotype) in 1963, while isolates from 1973 belonged to African genotype, however, isolates of 2006 belong to ECSA genotype and were also similar to Indian ocean strains of 2005-2006.

Interestingly, Chikungunya outbreaks do not frequent in many places. Some reports suggests CHIKV transmission outbreaks followed by periods without circulation (Kyungah et al. 2023; Salje et al. 2016; Tran et al. 2023) However, endemic transmission has also been reported (Kumar et al 2021).

Vaccine

Recently, Food and Drug Administration (FDA) and the European Medicines Agency (EMA), European

Union for the first CHIKV vaccines, IXCHIQ (VLA1553), developed by Valneva, epidemiology, licensure was obtained through a correlate of protection rather than traditional phase 3 trials

(Tran, et al. 2023; U.S. Food and Drug Administration

2023; Valneva 2023).

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