ISSN:3068-9376

Frontiers in Preventive Medicine

DOI: 10.64904/fpm25011

Review Articles

Chikungunya Virus and Chikungunya Fever: From Molecular Etiology to New Global Prevention and Control Strategies

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Abstract

Chikungunya fever is an acute viral infectious disease caused by the Chikungunya virus (CHIKV) and transmitted by Aedes mosquitoes. Since its first discovery in Tanzania in 1952, CHIKV has caused multiple large-scale outbreaks worldwide, posing a serious threat to public health. CHIKV belongs to the genus Alphavirus in the family Togaviridae. Its genome is a single-stranded positive-sense RNA, encoding 4 non-structural proteins (nsP1-4) and 5 structural proteins (C, E3, E2, 6K, E1). In recent years, adaptive mutations (such as E1-A226V) occurring in the virus during evolution have significantly enhanced its ability to infect and transmit through the vector Aedes mosquitoes, leading to the continuous expansion of its epidemic range. This review systematically summarizes the molecular virological characteristics, genomic evolution rules, pathogenic mechanisms (especially new findings related to joints and the nervous system) of CHIKV, as well as the latest diagnostic technologies, including CRISPR/Cas system-based rapid detection methods and probe-capture metagenomic sequencing. In terms of clinical management, it focuses on interpreting the Diagnosis and Treatment Protocol for Chikungunya Fever (2025 Edition) and a number of expert consensuses, emphasizing the efficacy and safety of integrated traditional Chinese and Western medicine treatment, and points out that non-steroidal anti-inflammatory drugs should be avoided. Although there is currently no specific antiviral drug, progress has been made in the research and development of drugs targeting key links in the viral life cycle and host factors. At the same time, a variety of candidate vaccines (including live attenuated vaccines, virus-like particle vaccines, mRNA vaccines, etc.) have entered preclinical or clinical research stages, showing good application prospects. Finally, combined with the Technical Guidelines for the Prevention and Control of Chikungunya Fever (2025 Edition), this article analyzes the challenges faced by China in preventing local transmission caused by imported epidemics, and proposes a comprehensive prevention and control strategy centered on "eliminating stagnant water, eradicating mosquitoes, and preventing mosquito bites", integrating monitoring and early warning, rapid response, multi-departmental collaboration, and public education. Facing the continuous evolution and global spread of CHIKV, strengthening basic research, drug and vaccine development, and the construction of public health systems are the keys to addressing this threat in the future.

Keywords: Chikungunya virus; Chikungunya fever; Mosquito-borne infectious disease

1. Introduction

Chikungunya fever is an arboviral disease caused by the Chikungunya virus, with main clinical features of fever, rash, and severe joint pain. The name

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"Chikungunya" comes from a dialect in southern Tanzania, Africa, meaning "to bend over", which vividly describes the posture of patients hunching over due to severe joint pain. Since its first isolation and identification in Tanzania in 1952, the transmission range of CHIKV has rapidly expanded from traditional epidemic areas in Africa and Southeast Asia to the Americas, Europe, and the Western Pacific region, becoming one of the most important emerging and re-emerging infectious diseases globally [1-2]. Entering the 21st century, CHIKV has shown unprecedented epidemic vitality. The outbreak in the Indian Ocean islands from 2004 to 2007 infected millions of people; in 2013, the virus achieved local transmission in the Americas (Western Hemisphere) for the first time, causing more than one million infections in a short period. In recent years, with the acceleration of globalization and the increasing frequency of international personnel exchanges, local outbreak epidemics caused by imported cases have become a severe challenge for non-traditional epidemic countries. For example, the local clustered epidemics reported in Foshan City, Guangdong Province, China in 2025 fully reveal the potential risk of CHIKV establishing local transmission chains in China [3]. The successful transmission of CHIKV is closely related to the rapid evolution of the virus. In particular, the A226V mutation in the E1 glycoprotein significantly enhances the replication and transmission efficiency of the virus in Aedes albopictus, making this widely distributed mosquito species an efficient transmission vector and greatly expanding the potential epidemic area of the virus [4-6]. In addition, in addition to causing typical acute symptoms, a considerable number of CHIKV-infected patients will develop chronic Chikungunya arthritis, leading to long-term joint pain, stiffness, and functional impairment, which seriously affects the quality of life and brings a heavy socioeconomic burden. In view of the global epidemic situation of Chikungunya fever and its continuous threat to public health, countries and regions around the world, including China, have continuously strengthened research and prevention and control efforts. In 2025, China successively released a series of authoritative documents such as the Diagnosis and Treatment Protocol for Chikungunya Fever (2025 Edition) and the Technical Guidelines for the Prevention and Control of Chikungunya Fever (2025 Edition), providing the latest basis for the standardized management of the disease. At the same time, a large number of new research results have emerged in the fields of virology, immunology, drug and vaccine research and development [7-9].

This review aims to systematically integrate the latest scientific literature and official guidelines in recent years, especially in 2025, starting from the molecular biological basis of CHIKV, to deeply explore its epidemiological characteristics, pathogenic mechanisms, clinical diagnosis and treatment, and prevention and control strategies, and look forward to future research directions, in order to provide comprehensive references for researchers and public health practitioners in this field.

2. Molecular Biology of Chikungunya Virus

2.1 Virus Classification and Structure

Chikungunya virus belongs to the genus Alphavirus in the family Togaviridae. Under an electron microscope, the virus particles are spherical, with a diameter of approximately 60-70 nanometers, and have a typical icosahedral symmetry structure [4,10]. Mature virus particles consist of a core nucleocapsid and an outer lipid envelope.

Genome structure: The CHIKV genome is a single-stranded positive-sense RNA with a length of approximately 11.8 kb. Its 5' end has a cap structure, and the 3' end has a polyadenylic acid tail, which allows the viral genome to directly serve as messenger RNA in infected cells to translate and produce proteins required for viral replication [11]. The genome mainly contains two open reading frames (ORFs): the non-structural protein ORF at the 5' end and the structural protein ORF at the 3' end, which are separated by a subgenomic promoter sequence downstream of a stop codon [6,11].

Non-structural proteins: Encoded by the 5' end ORF,

they are first translated into a polyprotein precursor, which is then cleaved by a protease encoded by the virus itself into 4 mature non-structural proteins (nsP1, nsP2, nsP3, nsP4). Together, they form the viral replicase/transcriptase complex, which is responsible for the replication of the viral genome and the synthesis of subgenomic RNA. nsP1 has methyltransferase and guanylyltransferase activities and is responsible for capping the 5' end of mRNA; nsP2 has helicase, RNA triphosphatase, and protease activities; nsP3 is involved in the assembly of the replication complex and interacts with a variety of host proteins; nsP4 is the core catalytic subunit of RNA-dependent RNA polymerase [12-13].

Structural proteins: Produced by translation of the 3' end ORF into a polyprotein precursor, which is cleaved by host cell proteases and viral proteases to form mature proteins. ①Capsid protein: Binds to viral RNA and assembles to form the nucleocapsid. ②Envelope glycoproteins E1 and E2: Anchored on the viral envelope in the form of heterodimers, they are crucial for the interaction between the virus and host cells. The E2 protein is mainly responsible for binding to cell receptors, while the E1 protein plays a core role in the fusion process between the viral envelope and the cell membrane [14]. ③E3 protein and 6K protein: The former, as a precursor part of the E2 protein, is involved in the correct folding and transport of the protein; the latter plays an important regulatory role in viral assembly and release [13].

2.2 Viral Life Cycle

The life cycle of CHIKV begins with the binding of virus particles to specific receptors on the surface of host cells through their E2 proteins. Recent studies have identified a variety of possible receptors, such as matrix remodeling-associated protein 8 (MXRA8) and low-density lipoprotein receptor-related protein 3 (LDLRAD3), which are widely expressed in various cell types, including fibroblasts, epithelial cells, and chondrocytes. This explains the broad cell tropism of CHIKV [15]. The virus enters the cell through clathrin-mediated endocytosis. In the acidic environment of the endosome, the E1 protein undergoes a conformational change, mediating the fusion of the viral envelope with the endosomal membrane, thereby releasing the viral nucleocapsid into the cytoplasm [16]. Subsequently, the viral genomic RNA is released and uses the host cell's translation system to directly synthesize the non-structural protein polyprotein (nsP1-nsP4). These non-structural proteins together form the viral RNA-dependent RNA polymerase complex, which is responsible for synthesizing negative-strand RNA intermediates, and then using them as templates to massively replicate full-length positive-strand RNA of the genome and subgenomic RNA required for translating structural proteins [12]. After the newly synthesized structural protein precursor is cleaved and matured by host cell proteases, it assembles with the replicated viral genomic RNA in the cytoplasm to form the nucleocapsid. The nucleocapsid buds through the intracellular membrane system (such as the endoplasmic reticulum and Golgi apparatus) to obtain a lipid envelope containing viral E1/E2 glycoproteins, and finally forms mature virus particles, which are released outside the cell through exocytosis to continue infecting new cells [11, 17].

2.3 Evolution and Genotyping of Viral Genome

Based on differences in the E1 gene sequence, CHIKV can be divided into three major evolutionary lineages: West African lineage, East/Central/South African (ECSA) lineage, and Asian lineage. The ECSA lineage and Asian lineage are the main culprits causing multiple large-scale global epidemics in recent years. It is worth noting that some virus strains in the ECSA lineage have undergone key point mutations (such as A226V) in the E1 protein gene during the process of adapting to new vector Aedes mosquitoes (such as Aedes albopictus). This mutation reduces the difficulty of the virus passing through the midgut barrier of Aedes albopictus by changing the charge characteristics of the viral envelope protein, significantly improving the transmission efficiency of the virus in this mosquito species, and is considered one of the important driving forces for the rapid global spread

of CHIKV [4-6]. A study by Ning Xinhang [5] on the distribution characteristics and risk analysis of CHIKV genotypes detected in China pointed out that the currently imported virus strains in China are mainly of the ECSA lineage and Asian lineage. These virus strains have potential transmission adaptability in Aedes albopictus widely distributed in China, forming the genetic basis for local transmission.

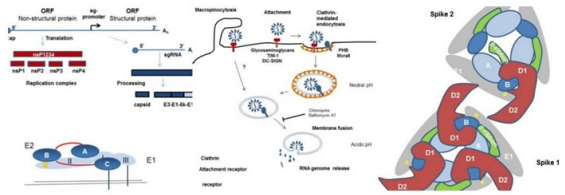

Figure 2: Schematic diagram of CHIKV life cycle

3. Epidemiology and Transmission Dynamics

3.1 Global Epidemic Situation

The epidemic of Chikungunya fever has obvious regional and seasonal characteristics, mainly distributed in tropical and subtropical regions. The epidemic peak coincides with the active season of vector Aedes mosquitoes, i.e., the rainy season or the period of high temperature and humidity [1-2]. However, driven by climate change and global travel, the epidemic map of CHIKV is expanding rapidly. According to the judgment of Han Hui [18] and Teng Daijun [19] on the global infectious disease epidemic situation in 2025, Chikungunya fever remains active in Southeast Asia (such as India and Thailand), the Americas (such as Brazil), and parts of Africa. It is particularly noteworthy that temperate regions such as southern Europe have also experienced local transmission events caused by imported cases, indicating that the threat of CHIKV to non-traditional epidemic areas is increasing. Rong Heng [1] systematically reviewed the global epidemic situation of CHIKV, pointing out that the virus is continuously invading new regions through travelers and trade. Once introduced into areas with efficient vector Aedes mosquitoes, it is very easy to cause community-level outbreaks.

3.2 Epidemic Situation and Challenges in China

Although China is not a traditional epidemic country for CHIKV, it faces continuous imported risks, and local transmission chains caused by imported cases have emerged in some areas. Studies by Chen Yibo [17] and Feng Yun [6] reviewed the epidemiological history of Chikungunya fever in China, pointing out that the cases reported in China have long been mainly imported, mostly from epidemic areas such as Southeast Asia and Africa.

However, 2025 has become a key turning point in the prevention and control of Chikungunya fever in China. The analysis of the clinical characteristics of 213 adult Chikungunya fever patients in Foshan City in 2025 by Zhang Qingsen [3] confirmed that a local outbreak caused by imported cases occurred in Foshan City, Guangdong Province in the summer of 2025. This indicates that CHIKV has successfully established a local transmission cycle in areas of southern China where vector Aedes mosquitoes (mainly Aedes albopictus and Aedes aegypti) exist, and the prevention and control situation is severe.

Most areas in China, especially the Yangtze River Basin and southern regions, have a wide distribution of Aedes albopictus, which provides the necessary ecological conditions for the local transmission of CHIKV [5, 20]. Therefore, strengthening port quarantine, early identification and management of imported cases, and enhancing the monitoring and control of vector mosquitoes are the keys to blocking the spread of the epidemic.

3.3 Transmission Routes and Vectors

The main transmission route of CHIKV is the "human-mosquito-human" cycle. When a female Aedes mosquito bites a patient or host animal in the viremic phase, the virus enters the mosquito body with the blood, replicates in the midgut epithelial cells, and then spreads to the salivary glands. After an extrinsic incubation period, when the mosquito bites a healthy person again, the virus is injected into the human body along with the saliva, causing infection [20].

Main transmission vectors include: ①Aedes aegypti: The most effective vector in the urban transmission cycle, preferring to inhabit indoors and

around human settlements. ②Aedes albopictus: Due to its stronger adaptability to temperate climates and wide geographical distribution, it plays a key role in the spread of CHIKV to new areas. The E1-A226V mutation is evolved by the virus to better adapt to Aedes albopictus [4-5].

In addition to the main mosquito-borne transmission route, rare non-mosquito-borne transmission routes have also been reported, including mother-to-child vertical transmission and transmission through blood products. A study by Luo Keren et al. [21] focused on the mother-to-child transmission of CHIKV and its impact on the neonatal nervous system, pointing out that the risk of vertical transmission increases significantly when the mother has viremia during the perinatal period, which may lead to severe neurological complications in newborns. The expert consensus on neonatal-related viral infections led by Cai Yueju [22] also provides guidance for the diagnosis, treatment, and prevention and control of this special population.

4. Pathogenic Mechanism and Immune Response

4.1 Viral Infection and Replication

The virus enters the subcutaneous tissue of the human body through mosquito bites and first infects local target cells such as fibroblasts and macrophages. After local replication, the virus enters the bloodstream, forming viremia and causing acute phase symptoms. CHIKV has obvious tropism for cells of skeletal muscle, joint synovium, liver, skin, and central nervous system, which is highly consistent with its clinical manifestations. Qi Zhongtian [15] systematically elaborated on the molecular mechanisms of CHIKV infection and pathogenesis in a review, emphasizing the complexity of the interaction between the virus and host factors. The doctoral thesis work of Yan Yao [12] and Zou Meng [13] identified new host factors (such as RACK1) that interact with CHIKV non-structural proteins nsP4 and nsP3, respectively. These findings provide a new perspective for understanding how the virus hijacks the host cell machinery to facilitate its own replication.

4.2 Immunopathological Damage

The host's immune response to CHIKV infection is a double-edged sword. On the one hand, a rapid innate immune response, especially the production of type I interferons, is crucial for controlling early viral replication. On the other hand, excessive inflammatory responses are considered the main cause of tissue damage and chronic symptoms.

①Acute phase: Viremia directly causes high fever. The replication of the virus in muscle and joint tissues, as well as the subsequent "cytokine storm" of pro-inflammatory cytokines (such as IL-6, IL-1β, TNF-α), are the core mechanisms leading to severe joint pain and myalgia [23-24].

②Chronic phase: Some patients experience joint symptoms for months or even years. The mechanism is not yet fully clear, but it is currently believed to be related to the persistent presence of viral antigens in joint tissues, the activation of autoimmune responses, and the persistent chronic inflammatory state [17]. The host's genetic background (such as HLA type) may also affect the susceptibility to chronic arthritis.

4.3 Special Populations and Systemic Effects

①Joint damage: Many scholars have pointed out that joint damage is the most prominent long-term sequela of CHIKV infection. The virus can directly infect joint synovial cells, inducing a strong local inflammatory response, leading to synovitis, tenosynovitis, and even bone erosion.

②Neurological effects: Although relatively rare, CHIKV infection can cause severe neurological diseases such as meningoencephalitis and Guillain-Barré syndrome, with a higher risk in newborns and the elderly [21].

③Mother-to-child transmission: As mentioned earlier, vertical transmission can cause severe encephalopathy and long-term neurodevelopmental disorders in newborns, which is a clinical issue that requires high attention [22].

5. Clinical Manifestations and Diagnosis

5.1 Clinical Manifestations

The incubation period of Chikungunya fever is usually 2-4 days, followed by an acute onset. ①Acute phase: Typical manifestations include sudden high

fever, severe joint pain (often polyarticular and symmetrical, affecting hands, wrists, ankles, knees, etc.), rash (mostly maculopapular), and myalgia. Other common symptoms include headache, nausea, vomiting, and conjunctivitis. ② Chronic phase: Approximately 30%-50% of patients will develop chronic Chikungunya arthritis, manifested as persistent joint pain, stiffness, and swelling. Severe cases can lead to joint deformity and functional loss. The analysis of 213 patients in Foshan by Zhang Qingsen [3] showed that joint symptoms were the most common complaint, and some patients were still troubled by them for a long time after the acute phase. The clinical manifestations of children and the elderly may be atypical. Children are more likely to have severe skin blister-like rashes and neurological symptoms, while the elderly may have aggravated conditions due to underlying diseases.

5.2 Laboratory Diagnosis

Rapid and accurate laboratory diagnosis is crucial for the early identification of cases, epidemic control, and clinical management. Diagnostic methods mainly include viral nucleic acid detection, virus isolation, serological detection, etc.

①Viral nucleic acid detection: In the early stage of the disease (usually the first 5-7 days), when the patient is in the viremic phase, real-time fluorescent RT-PCR and other methods can be used to directly detect viral RNA in blood or other body fluids. This method is sensitive and specific, and is the gold standard for early diagnosis. Tian Rui [25] reported the successful determination of the full-length CHIKV genome sequence from throat swab samples using probe-capture metagenomic sequencing, demonstrating the strong ability of next-generation sequencing technology in virus traceability and mutation monitoring. Xiong Li [26] established a rapid detection method for arboviruses based on RT-RAA and CRISPR/Cas13a, providing a convenient and efficient detection tool for on-site and primary-level use.

②Serological detection: One week after the onset of the disease, the body begins to produce specific antibodies. Detecting IgM antibodies (indicating recent infection) and IgG antibodies (indicating past infection or recovery) are common diagnostic methods. Xu Xiaoli [27] developed a CHIKV IgG antibody ELISA rapid detection kit, improving the efficiency of serological detection. Ma Jiajia [28] reviewed the latest progress in CHIKV serological detection technology.

③Virus isolation: Isolating the virus from patient samples is the "gold standard" for diagnosis, but it is complex, time-consuming, and requires a biosafety level 3 laboratory. It is mainly used for scientific research.

The Diagnosis and Treatment Protocol for Chikungunya Fever (2025 Edition) clearly specify the diagnostic criteria for confirmed cases and suspected cases, emphasizing a comprehensive judgment based on epidemiological history, clinical manifestations, and laboratory tests.

6. Treatment and Clinical Management

Currently, there is no specific antiviral drug for Chikungunya fever. Treatment mainly focuses on relieving symptoms, supportive care, and preventing complications.

6.1 Symptomatic and Supportive Treatment

①Rest and nutrition: Patients in the acute phase should have adequate rest and ensure fluid and nutrient intake.

②Antipyretic and analgesic: Acetaminophen is the recommended first-line drug for antipyretic and analgesic purposes. It is particularly important to emphasize that non-steroidal anti-inflammatory drugs such as ibuprofen and aspirin should be avoided in the acute phase, as they may increase the risk of bleeding, especially when differential diagnosis with hemorrhagic diseases such as dengue fever is required.

③Joint management: For chronic arthritis, non-steroidal anti-inflammatory drugs or short-term, low-dose glucocorticoids can be used as prescribed by a doctor. Physical therapy and rehabilitation exercises are crucial for improving joint function.

6.2 Integrated Traditional Chinese and Western Medicine Treatment

Traditional Chinese medicine has shown unique advantages in relieving the symptoms of Chikungunya fever, especially in the treatment of chronic arthritis. A real-world study of 500 patients conducted by Wu Zelei [29] confirmed that integrated traditional Chinese and Western medicine treatment is superior to pure Western medicine treatment in improving joint pain, shortening the course of the disease, and improving the quality of life, with good safety. The TCM treatment plan in the Diagnosis and Treatment Protocol for Chikungunya Fever (2025 Edition) and the expert consensus of Sichuan Province [30] both provide specific TCM syndrome differentiation and treatment plans and recommended prescriptions. Foshan Hospital of Traditional Chinese Medicine has even set up a special rehabilitation clinic for Chikungunya fever to provide professional TCM rehabilitation services for patients [31].

6.3 Research Progress in Antiviral Drugs and Host-Targeted Therapy

Although there are no marketed specific drugs, researchers around the world are actively developing antiviral strategies against CHIKV. Chen Yibo [17] systematically reviewed the research progress of anti-Chikungunya fever drugs. The current research directions mainly focus on two categories:

①Direct-acting antiviral drugs: Targeting key proteins in the viral replication process, such as nsP1 (methyltransferase), nsP2 (protease), and viral capsid protein. Li Jiaqi [32] established an antiviral drug screening system, providing a platform for drug discovery.

②Host-targeted therapy: Inhibiting viral infection by interfering with host cell factors on which the virus depends for replication. For example, targeting the MXRA8 receptor or host proteins involved in the viral endocytosis process. The advantage of this strategy is that it has a higher genetic barrier and is less likely to cause viral drug resistance [15, 17].

7. Progress in Vaccine Development

A safe and effective vaccine is a fundamental strategy for preventing and controlling Chikungunya fever. Currently, a variety of CHIKV candidate vaccines are in different stages of development worldwide. Chen Xi [7] and Feng Jialu [8] reviewed the progress of clinical research on vaccines in detail.

① Live attenuated vaccines: Attenuated virus strains obtained through in vitro passage or reverse genetics technology can induce strong humoral and cellular immunity. Some candidate vaccines have entered phase III clinical trials.

②Virus-like particle (VLP) vaccines: Self-assembled from viral structural proteins, they do not contain viral genetic material, have high safety, and can effectively induce neutralizing antibodies. A VLP vaccine showed good immunogenicity and safety in clinical trials.

③mRNA/DNA vaccines: Using nucleic acid molecules encoding viral antigens to express antigens in the body and induce an immune response. Such vaccines have the advantages of short development cycle and easy large-scale production. Shu Chang [9] developed a bivalent recombinant DNA vaccine against West Nile virus and CHIKV, which successfully induced a specific immune response in mice, demonstrating the potential of multivalent vaccines.

④Viral vector vaccines: Using modified viruses (such as vaccinia virus, adenovirus) as vectors to deliver CHIKV antigen genes. Zhang Jinxin [33] studied the mouse immune effect of a recombinant vaccinia virus candidate vaccine against CHIKV.

8. Prevention and Control Strategies

The prevention and control of Chikungunya fever is a systematic project that requires a comprehensive strategy, with the core of controlling the source of infection, cutting off the transmission route, and protecting susceptible populations.

8.1 Personal Protection

The most direct and effective way to protect susceptible populations is to prevent mosquito bites. Specific measures include:

①Wearing long-sleeved clothes and long pants, and using mosquito nets.

②Using certified mosquito repellents (containing ingredients such as DEET, picaridin).

③Installing screen doors and windows to reduce

mosquitoes entering the room.

The Popular Science Promotion on Chikungunya Fever (Personal Protection Section) and recommendations from many health experts have repeatedly emphasized these personal protection points.

8.2 Community and Public Health Prevention and Control

①Mosquito vector control: This is the top priority of prevention and control work. The key is to eliminate mosquito breeding grounds, including cleaning up various stagnant water containers (such as vases, tires, idle containers) indoors and outdoors. When an epidemic occurs, emergency mosquito vector control should be initiated, including space spraying of insecticides.

②Monitoring and early warning: Establish and improve a comprehensive monitoring system including infectious disease monitoring, vector mosquito monitoring, and etiological monitoring, promptly detect imported cases and signs of local transmission, and issue early warning information.

③Case management and hospital infection control: Early detection, diagnosis, and isolation of cases (especially during the viremic phase, mosquito-proof isolation should be adopted) are crucial for preventing the spread of the epidemic. The Expert Consensus on Infection Prevention and Control of Chikungunya Fever in Medical Institutions (2025 Edition) formulated by He Ling [34] provides detailed guidance for medical institutions to prevent nosocomial transmission.

④Health education and risk communication: Popularize the prevention and treatment knowledge of Chikungunya fever to the public through various channels, improve disease prevention awareness, and eliminate unnecessary panic. Release epidemic information in a timely and transparent manner and respond to social concerns.

8.3 International Cooperation

In view of the cross-border transmission characteristics of Chikungunya fever, it is crucial to strengthen global cooperation in epidemic information sharing, joint monitoring, and scientific research.

9. Conclusion and Outlook

As a re-emerging mosquito-borne viral disease, Chikungunya fever has an increasingly severe global epidemic situation, and its threat to public health security in China cannot be underestimated. The local epidemic in some areas of China in 2025 has sounded the alarm for us. This review systematically summarizes the latest progress of CHIKV in the fields of virology, epidemiology, pathogenic mechanism, diagnosis, treatment, and vaccine research and development, and closely combines China's latest released diagnosis and treatment and prevention and control plans, aiming to provide comprehensive scientific references for addressing this threat.

Looking forward to the future, the prevention and control and research of Chikungunya fever still face many challenges and opportunities:

①Deepening basic research: It is necessary to further clarify the chronic pathogenic mechanism of CHIKV, the detailed map of virus-host interaction, and the evolutionary dynamics of virus cross-species transmission and adaptation to new vectors, so as to provide new targets for drug and vaccine design.

②Innovation in diagnosis and treatment technology: Continue to develop faster, more sensitive, and portable on-site detection technologies to achieve early diagnosis and genotyping. Promote the standardization and evidence-based medical research of integrated traditional Chinese and Western medicine treatment, and optimize the management plan for patients in the chronic phase.

③Breakthroughs in drugs and vaccines: Accelerate the clinical research of promising antiviral candidate drugs and vaccine platforms, and strive to achieve a "breakthrough" as soon as possible to provide specific treatment for patients and establish an immune barrier for the population.

④Strengthening the public health system: Adhere to the principle of "prevention first", and continuously improve the comprehensive prevention and control mechanism of mosquito-borne infectious diseases featuring "government leadership, multi-

departmental collaboration, and whole-society participation". Strengthen the construction of grassroots prevention and control capabilities, and improve the level of epidemic monitoring, emergency response, and risk communication. Facing the continuous global health challenge of Chikungunya fever, only through continuous scientific exploration, technological innovation, and strong public health actions can we effectively curb its spread and reduce its harm to human health.

Received: October 17, 2025;
Accepted: October 22, 2025

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