Global Journal of Universal Studies

Volume1:Issue1 ***ISSN: 3008-0509***

Article History

Submitted: 21-05-2024

Revised: 11-06-2024

Accepted: 15-06-2024

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Unravelling the Mysteries of Zika Virus: A Comprehensive Overview

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Global Journal of Universal Studies

Volume1:Issue1 ***ISSN: 3008-0509***

Abstract

The Zika virus (ZIKV), which emerged in an epidemic state in 2016, poses a serious risk to the health of society. It could appear as a serious condition with signs such as migraine and a high body temperature or it can appear benign. Aedes species of mosquitoes (family Flaviviridae, genus Flavivirus) are responsible for the transmission of ZIKA virus to humans. Even though over half of civilization lives in areas where Aedes aegypti is common, there is still concern about the possibility of its revival. Global accounts of Zika virus infections have recently been made, especially from America; French Polynesia and other Pacific Island nations; and south-East Asia. ZIKV was first discovered in 1947 in the Ugandan Zika Forest, in a rhesus monkey. Since the beginning of discoveries in Africa and Asia in 2007, the virus expanded around every continent. A serious infection frequently results in a short-term febrile illness, but recent evidence reveals that it may also be linked to additional important adverse effects like microcephaly in infants and Guillain-Barré syndrome. This review provides knowledge about the pathophysiology of ZIKV, reveals essential cellular processes that develop during infections, and recalls the origins of the virus from when it was first discovered to its current worldwide propagation.

Keywords

Zika virus, Epidemic, Aedes species, infection, public health

Introduction

Zika virus is an arbovirus (virus carried by arthropods). It is a microscopic encapsulated positive-stranded RNA virus that is a member of the Flaviviridae family, which is also linked to the viruses that cause dengue, yellow fever, and West Nile [1]. It originally emerged in 1947 in the rhesus monkey inhabiting the Zika forest near Kampala, Uganda [2]. The two main origins of the single-stranded RNA Zika virus are Asian and African [3]. It is believed that the Zika virus has mainly survived in Africa through a cycle of infection between mosquitoes and non-human hosts, including apes, monkeys, and humans serving as rare accidental hosts [4].

However, outside of Africa, humans are most likely the primary host. There are currently just two Zika virus genome sequences from the present pandemic in South America. A Suriname Zika virus's phylogenetic research reveals that it is of the Asian genotype [5]. As they share more than 99.7% and 99.9% of identical nucleotide and amino acid identities, respectively, the strain that was propagating in French Polynesia in 2013 is most closely linked to that strain. Analysis of the envelope genomes from Brazilian patients supports this conclusion [6]. It's possible that a mutation in the Asian lineage caused the virus to adapt to a human host rather than a non-human animal [7].

The Zika virus can spread quickly through skin immune cells and can enter cells through a wide variety of receptors [8]. To learn more about the connection to neurological disorders, research into the virus's capacity for replication in neuronal cells is necessary [9] In February 2016, WHO named Zika as the virus that causes illness, a global public health emergency [10]. Although there has been a decrease in ZIKV transmission since 2017, the virus is still present and has the potential to create major outbreaks in specific areas [11]. Although there has been a decrease in ZIKV transmission since 2017. The virus is still present and has the potential to create major outbreaks in specific areas [12] Therefore, we go over the most recent research on the epidemiology, spread, immunogenicity, host variables, clinical characteristics, and possible therapies for Zika in this review.

Epidemiology

Anxiety emerged as the Zika virus (ZIKV) spread across the world, especially across countries in Latin America and the Caribbean. During the 2016 outbreak, there were between 440,000 and 1300,000 cases in Brazil [13]. In addition, ZIKV spread rapidly, with 60 countries and territories reporting active ZIKV exposure as of July 21, 2016. In light of the aforementioned, on February 1, 2016, the World Health Organization (WHO) declared ZIKV to be “a worldwide health crisis of global frustration” [14] and emphasized the necessity of taking immediate action to reduce its infection, particularly in women who are pregnant and of childbearing age [15].

In 1947, a rhesus monkey in the Zika forest in Kampala, was the first carrier of this virus [16]. After five years, the virus was discovered to be wildly spreading over Africa for the first time [17]. Then, in the 1983s, a distinct strain of the virus—not the same as the one in Africa—migrated to Asia [18].

Furthermore, the Asian strain has led to localized outbreaks outside of Asia, with the result that French Polynesia experienced larger outbreaks in 2007, 2013, and 2014 [19]. In May 2015, the greatest Zika virus outbreak in history happened in northeastern Brazil and reached pandemic proportions. An estimated 1.62 million people worldwide are thought to have contracted ZIKV, with cases peaking along the Pacific, American, and West African coast regions [20].

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Volume1:Issue1 ***ISSN: 3008-0509***

More than 70 countries worldwide have reported instances of the virus. Based on their respective geographic origins, ZIKV has been phylogenetically split into two primary lineages: the African and the Asian. Viral spread in East and West Africa is divided in branch of Africa [21]. **(Figure 1)**

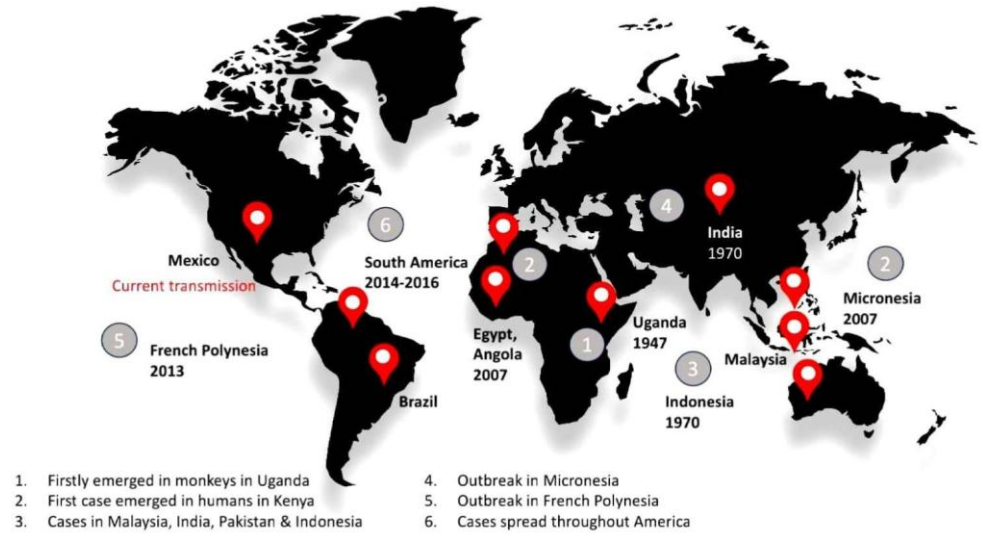

Figure 1: ZIKV infected areas according to time

1. **Genomic Organization of ZIKA VIRUS:**

ZIKV are related in context of size and genetic profiles [22]. The genome of ZIKV is roughly 10.7 kb long. It contains only one polyprotein, three structural (S) proteins, a pre-membrane protein (PrM), a capsule (C) protein, and envelope protein (E). It also contains seven non-structural proteins and Ns1-Ns5 that are important for ZIKV as the make up two main parts of its genome [23]. Untranslated regions 5′ and 3′ around the ZIKV genome. In case of vector borne transmission, the virus is closer to YFV and DENVs [24].

2. **Mode of transmission:**

The primary method of Zika virus transmission to humans is by the bite of an infected Aedes aegypti mosquito, which typically bites during the day time and grow in fresh standing water (both indoors and outdoors) [25]. ZIKV can also be carried by Aedes albopictus and other Aedes mosquito species, such as A. africanus, A. luteocephalus, A. furcifer, and A. taylori [26].

2.1. Vector borne transmission:

Aedes aegypti, Ae. vexans, Ae. notoscriptus, Ae. vittatus, and *Culex quinquefasciatus* are carriers for the transmission of ZIKV Infection [27]. *Anopheles gambiae, Cxpipiens* and *A. stephensi* are Considered to be probable vectors [28]. However, in America Culicidae family compete with Aedes species as a potential vector to transmit ZIKV. When Aedes mosquito bites an infected rhesus monkey, it act as a carrier for ZIKV. When this mosquito bites a healthy person, it transmits zika virus in humans and cause infection as shown in **(Figure 2)**

2.2. Non-Vector borne transmission:

2.2.1. Transmission through urine:

ZIKV is actively found in human urine and cause viremia [29]. When *A. aegypti* and *A. albopictus* were discovered breeding in urine-contaminated or ZIKV-infected environments, they become susceptible to ZIKV infections [29]. These two kinds of mosquitoes can transmit the Zika virus to adult female mosquitoes through transmitting infection to larvae and pupae of mosquitoes in contaminated urine environments [29]. Urine contained infectious ZIKV particles, according to a body of evidence [30]. About five ZIKV infectious strains were found when human urine samples were cultured, indicating that the infectious Zika virus was released by the viruria of ZIKV-infected individuals [30]. Furthermore, two other investigations found that one patient's urine contained Zika virus titer of roughly 10 pfu/ml, while three more patients' urine samples contained 12–20 pfu/ml of the virus, approximately thirty days after the onset of ZIKV symptoms [31].

2.2.2. Sexual transmission

Patients with ZIKV infection have shown evidence of Zika virus RNA in their semen. The virus can spread between the sexes, but male-to-female transmission has the highest transmission frequency [32]. Ratio of viral transmission from male to female: male to male: female to male was 30:1:1 [33].

2.2.3. Vertical transmission

During pregnancy, there is vertical transmission has been observed. ZIKV can be passed from women to their foetuses, as evidenced by the virus's isolation about 4 days later, from the baby brain, amniotic fluid, and baby serum [34].

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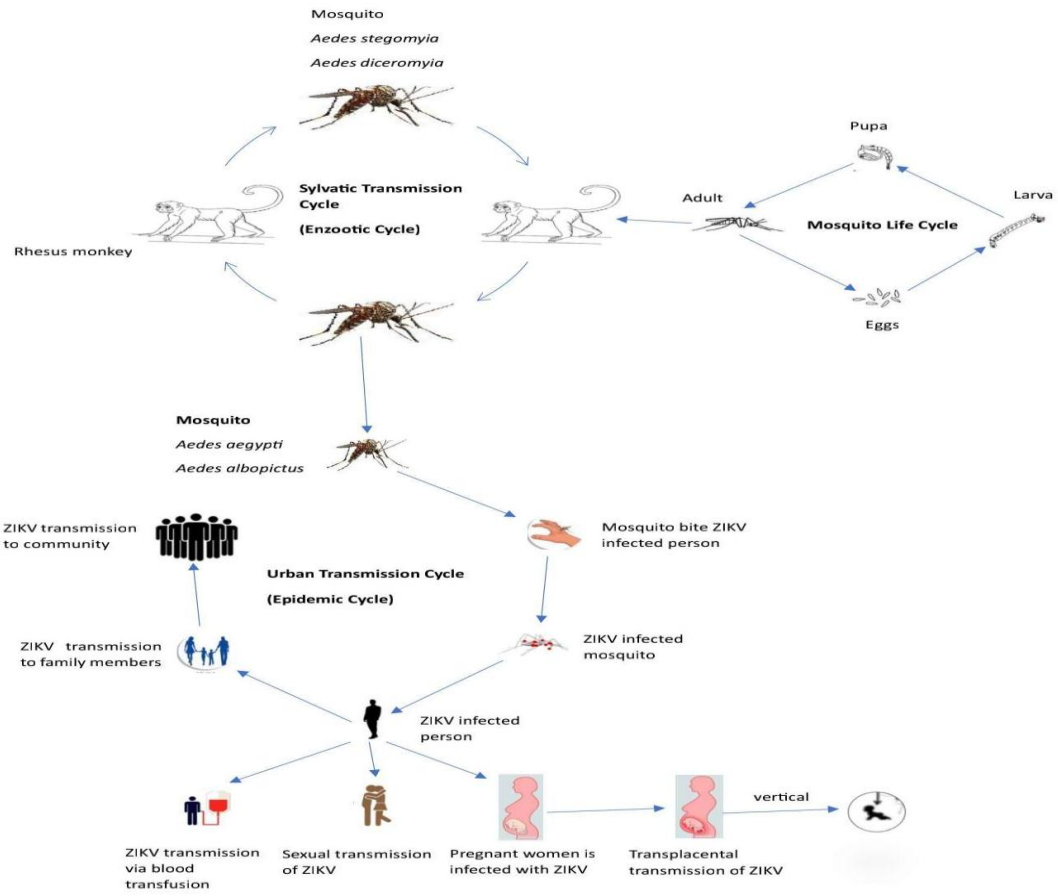
2.2.4. Blood transfusion

Transfusion of blood Brazil has documented incidences of ZIKV transmission [35], which exacerbates the viruses spread because the majority of ZIKV-infected individuals exhibit no symptoms [36]. Blood recipients can easily contract ZIKV from asymptomatic blood donors. Approximately 3% of blood donors tested positive for the virus. The claim that ZIKV can retain in the blood of an infected individual for almost 60 days exacerbates the situation even more [31]. Developing control methods against ZIKV is a challenging task due to its various mechanisms of transmission.



3. Figure 2: Transmission of ZIKV

There is little information about ZIKV's pathology. It was discovered that human keratin, primitive immune cells, and skin cells were favorable routes for the Zika virus infection [37]. The entry of ZIKV is allowed by the DCSIGN, AXL, Tyro, and TIM-1 invasion/adhesion factors [38]. IF-1 is produced by cells that are contaminated upon ZIKV reproduction, which also triggers a protective defense [39]. Dermal cell infection was found to stimulate the development of therapeutic antigen clusters that can detect a range of molecular features related to the virus. The creation of autophagosomes is linked to increased viral reproduction. ZIKV infection generated an autophagous program, which was verified by the fibroblasts infected with the virus exhibiting distinctive autophagosome-like vesicles [40]. Zika fever induces Th1, Th2, Th9, and Th17 T cell activation during its acute phase [41].

5.2. Replicative Cycle:

Zika virus enter through the host receptors by their glycosylated regions which are present on the envelope protein of virus [42]. Clathrin-coated vesicles are responsible to engulf viral partical into the cell. The virus envelope protein exhibits structural alterations in the endosome's low pH environment, which enabling the virus to fuse with the endosome and release its positive strand RNA [43]. In the reticulum, positive strand of ssRNA is converted into functional polyprotein by translation. Polyprotein is further broken down by the proteases enzymes of host or by viral NS3 and NS2B (non-structural proteins). For instance, to produce negative sense RNA strand, helicase (NS3) and RNA dependent RNA polymerase enzymes also use positive RNA strand as a template [44]. A new positive sense RNA strand can be synthesized by using the negative sense RNA strand as a template. Whether translation or additional application for viral genome replication can be done with the freshly created positive sense RNA strand [45]. When structural proteins are assembled around the viral genome, the particles are transported to the Golgi apparatus, where they cleave the precursor membrane protein to become mature virion and leave the host cell [46]. **(Figure 3)**

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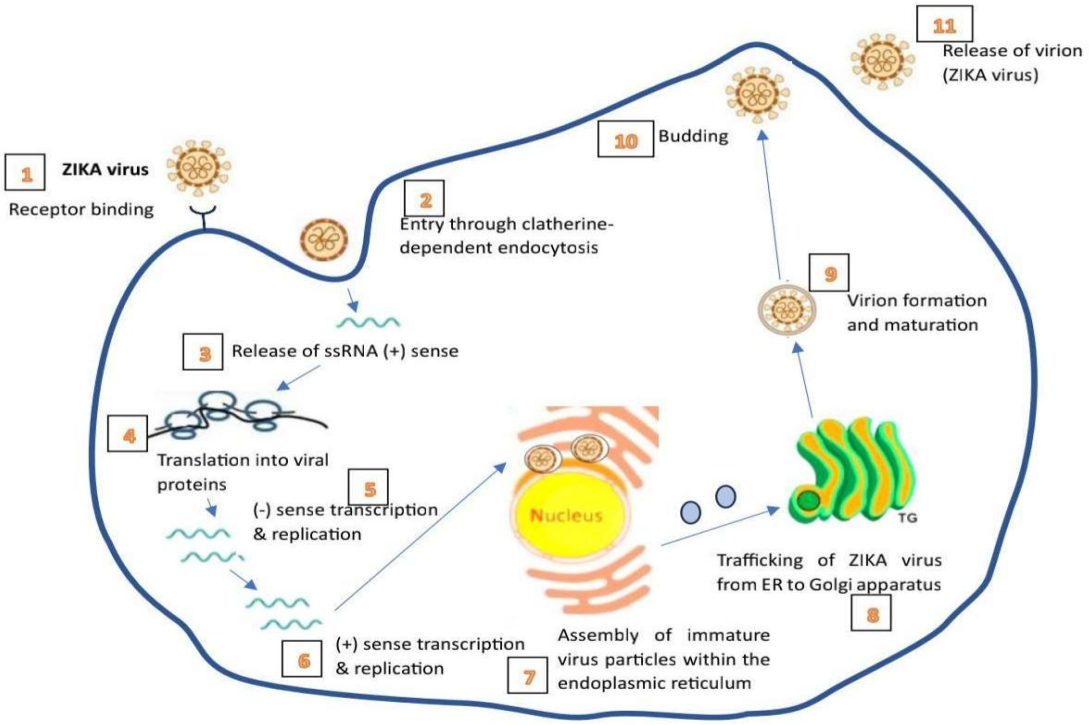

Figure 3: Replication cycle of ZIKV

4. Clinical Manifestations

Only around 20% of infected people have symptoms or indicators of ZIKV infection; mostly patients not show sign and symptoms [47]. Common symptoms include sudden lower level fever with rashes, arthralgia of tiny joints, and non-purulent conjunctivitis [48]. These signs are comparable to the signs of DENV and chikungunya viral disease. Headache, myalgia, asthenia, peripheral edema, and gastrointestinal disruption (abdominal discomfort, nausea, diarrhea) are some other symptoms. After being bitten by an Aedes mosquito, symptoms and indicators usually appear two to twelve days later and go away in two to seven days [49]. Patients who have traveled to regions where ZIKV is endemic in the past and exhibit clinical signs and symptoms should be suspected of having the virus. Infants and children with postnatal ZIKV infection exhibit symptoms and indicators that are comparable to those of adults [50]. Infants (and even young children) with arthritis typically exhibit irritability, limping, movement difficulty, and pain when the affected joint is moved, either actively or passively, or when the joint is palpated [50].

5. Diagnosis:

ZIKV infection can be diagnosed by assays for molecular and serological detection the presence of the virus's vector in the area and clinical signs. By using newborn Swiss albino mice, the zika virus was isolated from mosquito samples using variety of techniques, including subcutaneous, intraperitoneal and intracerebral injections [51]. Zika virus was found in brain material passages and this was further verified by the heamagglutination inhibition test [52]. Zika virus can be grown in cell lines such as vero cells, kidney cells from rhesus monkey such as LLC-192 MK2 and mosquito derived cell lines such as A .albopictus (C6/36) and A. pseudoscutellaris (MOS61 or AP-61) [53].

ZIKV has been determined by compliment fixation test, serum neutralization assay, and heamagglutination inhibition (HI) assay. Antibodies against lgG and lgM are detected as part of the serological diagnosis. IgM antibodies can be examined by using Enzyme Linked Immunosorbant Assay (ELISA) in order to identify antibodies against Zika virus in patient serum. If the titer of the Zika virus neutralizing antibody is greater than four times that of the Dengue neutralizing antibody, the test is regarded as positive otherwise, it is regarded as inconclusive [54]. Due to the irrelevant reaction with other flavivirus members restricts the application of ELISA techniques, the Zika virus induced antibodies can be identified using the Plaque Reduction Neutralization Test (PRNT). PRNT facilitates the differentiation of cross neutralizing antibodies produced by different flaviviruses [55]. Analyzing IgM and IgG titers in paired samples from the acute and convalescent phases of infection confirms seroconversion [56].

Pregnant mothers may undergo ultrasound screening to check for microcephaly [57]. Around five months of pregnancy, a number of fetal abnormalities can be detected; however microcephaly is only detectable around six to seven months gestation or after birth [58]. Molecular and serological tests can be used for additional verification. RT-PCR is used in molecular diagnosis to identify Zika virus RNA [59]. Viral nucleic acids are detectable in the serum during the first seven days of illness. Samples include amniotic fluid, CSF, urine, frozen and fixed placenta, saliva, and nasopharyngeal swab, serum from patients and umbilical cord samples from new-borns [54].

It is necessary to understand the virus shedding in various bodily fluids such as urine which lasts for 15-20 days, in order to develop a more accurate diagnostic tests that uses the right sample [60]. According to a report, viruria may appear more than 15 days after symptoms first appear [61]. Scientist have begun examining antibodies against the Zika virus in amniotic fluid and neonatal blood in order to determine the connection between the virus and microcephaly [62]. DNA sequencing after RT-PCR is considered as confirming [63]. Additional sequencing of the NS5, NS3 and envelope genes verifies the relationship between the different Zika virus strains. It is possible to use real time PCR to find the virus early in the infection process [64]. Additional, it can be verified through the use of immunohistochemistry stainig of antigen that has been fixed on the placenta and umbilical cord of infants who are infected [54].

6. Complications:

Numerous problems, including congenital microcephaly, Guillain-Barre syndrome, and even fetus death in pregnant women infected with ZIKV, have been linked to the virus [65].

6.1. Congenital infection:

Zika virus infection in expectant mothers can happen at any stage of the pregnancy and has been linked to microcephaly in the child. Numerous verified reports from Brazil have demonstrated this link, primarily in cases where the infection occurs in the first trimester. Children born after the start of the ZIKV pandemic in Brazil who had microcephaly have also been shown to have cases of muscle atrophy [66]. Pregnancy-related ZIKV infection is linked to several unfavorable consequences for the fetus, including damage to the central nervous system (CNS), placental damage, in-vitro fetal growth inhibition (with or without microcephaly), and even fetal death [67].

6.2. Neurological complications:

A small number of cases linking ZIKV infection to Guillain-Barre disease has been documented [68]. Six days or less before experiencing neurological symptoms, 90% of the patients reported having ZIKV infection symptoms. There have been cases of meningoencephalitis and acute myelitis linked to ZIKV detection in the CSF, among other neurological symptoms [68]. Additionally, reports of ZIKV infection in Italian travelers have included ankle edema, axillary, lymphadenopathy, leukopenia linked with monocytosis, and thrombocytopenia [69].

Treatment:

People infected with ZIKV infection should drink plenty of water, take enough rest, and use liquid solutions to treat fever and pain. If symptoms become severe then go for counselling and therapeutic strategies. Not treatment or prevention of ZIKA virus is present until now, only the medications related to symptomatic relief are present to treat fever and pain like paracetamol [70]. If patient is already taking drugs, then avoid use of any additional medication like Nonsteroidal anti-inflammatory drugs (NSAIDs) [71]. Homeopathy is an important treatment in ZIKV infection as it shown to be effective in encephalitis virus (same genus as of ZIKA virus) treatment in Japanese [70]. Eupatorium is a natural homeopathic compound that can be used as prophylactic treatment against ZIKV infection as it has shown effective for treating symptoms of ZIKV infection [72]. Atropa belladonna, Eupatorium perfoliatum, and Rhustox are homeopathic medicinal treatment against symptoms of ZIKV infection [73]. To reduce mortality and morbidity rate during epidemics of ZIKV infection, homeopathic pharmaceuticals are more effective than conventional methods [74]. Tinospora cordifolia is a green herbal that is immunomodulator that is use for treating viral infection of any nature. These increase the immune system capacity and increase the macrophages' phagocytic activity [75]. These are effective against intestinal diseases, urinary track infections, dengue and other viral infections so, they might be effective against ZIKV infection [70]. In 2016, WHO listed the public based commercial, governmental, and academic projects for making ZIKV interventions so, its treatment and vaccine development is in progress [76]. Since April, 2019 no vaccine for ZIKV infection approved by FDA, is available in market [77]. Production of vaccines for ZIKV is challenging, due to its ZIKV outbreaks had waned, and vaccines should also be safe for pregnant women and avoid neurological effects in adults [78]. There is also lake of funding for vaccines development. Suramin, chloroquine, nitazoxanide are anti-protozoal drugs; avermectin, niclosamide are anthelmentics; sofosbuvir (effective for hepatitis C virus, has shown promising results in treating ZIKV infection transmission from pregnant mice to fetus (especially sofosbuvir) [79]. **(Table 1)**

Table 1: Antiviral drugs against ZIKV and their mode of action

Drugs	Mode of Action
Isoquercitrin, Curcumin, Gossypol, Conessine, Digitonin, Dictyota menstrualis (F6 fraction), Naringinin, Polydatin, Doratoxylem apetalum.	Inhibit ZIKV internalization.
Delphinidin, Berberine, Dictyota menstrualis (Fac-2 fraction), EGCG, Emodin, Palmatine, Harringtonine.	Virucidal activity.
Schinus terebinthifolia (ethanolic fruits' peel extract or whole fruit extract), Digitonin, Naringinin, Conessine, Cinnamic acid.	Inhibits ZIKV replication.
Docosahexaenoic acid (DHA).	Mitochondrial damage and ROS generation.

1. **Prevention:**

Since there is currently no vaccine against ZIKV on the market, it is essential to develop coordinated, broad, and comprehensive strategies to handle any situation that may arise [80]. Similar to other flavivirus infections. Get lots of rest to manage this infection. Consume liquids to stay hydrated. To lower fever and pain, take medication such as acetaminophen [81]. As Zika-carrying mosquitoes as well as other viruses bite both during the day and at night. So, keep yourself

away from mosquito bites to defeat ZIKV. Everyone should take precautions against mosquito bites, including mothers who are pregnant or breastfeeding. Apply insect repellents with one of the following active chemicals that are registered with the Environmental Protection Agency (EPA). EPA-registered insect repellents are safe and effective when used as prescribed, even for breastfeeding and expectant mothers [82]. On a child's hands, eyes, mouth, cuts, or irritated skin, never use insect repellent. When applying insect repellent to a child's face, adults should first apply it to their hands [83]. Apply sunscreen first and insect repellent second if you're wearing sunscreen. Look both inside and outside for containers that carry water [84]. Restricting the transmission of ZIKAV can be significantly assisted by genetic interference from bacteria such as Wolbachia. A cycling pool of vectors will experience a series of events when Wolbachia bacteria are introduced because the number of mosquitoes carrying the bacteria will grow with each cycle of reproduction [85]. Furthermore, since genetically engineered mosquito strains are successful against DENV, they may also be utilized against ZIKV [86]. The genetically modified (GM) strain A. aegypti OX513A has caused a decrease in the Aegypti population in the area. Because the GM male mosquitoes can mate with wild-type females, they can eradicate them [87]. Pregnant women should refrain from making unnecessary trips to ZIKV-affected areas to prevent the transmission of the virus to the fetus. The government also has to notify anyone who is planning to visit the areas that have been affected [87]. A person who has Zika can transmit the virus to their partners through intercourse. Sharing of sex toys and oral, anal, and vaginal sex are all considered forms of sex. Even in committed relationships, zika can be transmitted through intercourse [88]. The Zika virus can remain in semen longer than in other bodily fluids [89], therefore the periods during which men and women can spread the infection through intercourse vary. It is possible to stop the sexual transmission of ZIKV by avoiding unprotected intercourse, sexual contact with infected individuals, and travel from locations where the virus is common [90]. Travelers who return from Zika-affected locations are prohibited from donating blood for 28 days following their return, or until the danger of infection has diminished [91].

2. Ethical implications and concerns of ZIKV infection:

Developing a thorough strategy to manage this public health emergency may require addressing moral dilemmas and ethical concerns about the Zika virus [92]. It is therefore morally necessary for the health authorities to commit to allocating a reasonable amount of health resources for reacting to this scenario [93]. To stop the Zika virus from spreading, the government should prioritize allocating a sizable sum of money for preventive measures [94]. Setting priorities is typically the most challenging stage in these situations, particularly given the lack of sufficient scientific data to estimate the true cost of disease and the influence of the media and society, which was heightened by the 2016 Brazilian Olympic Games [95].

National and international health authorities should uphold the principle of honesty through the implementation of an unambiguous and transparent information-gathering system aimed at increasing public understanding of the actual circumstances [96]. In light of this widespread pattern of disease transmission, "cosmopolitan solidarity" on a global scale appears to be unavoidable [97]. Similar to the Ebola crisis, governments must establish a global health governance network by being responsive to the demands of accountable international organizations, particularly the World Health Organization [98]. When it comes to controlling mosquito populations, governments should strive to limit the spread and reproduction of these insects while also keeping in mind their moral responsibility to maintain biosafety and safeguard ecosystems [99].

To safeguard fundamental civil and human rights, such as the freedom of mobilization and the right to procreate if birth control or a border are among the alternatives, deliberate ethical evaluation is necessary when developing policies for disease control [100]. Similarly, when rules for reporting likely instances to health authorities are discussed, they must respect the confidentiality of patient's health information and shield individuals from stigma [101].

Abortion is at the forefront of moral issues due to the increased risk of congenital abnormalities, primarily microcephaly, in neonates born by moms infected with the Zika virus during their first trimester of pregnancy [102]. Based on the moral standing that different schools of ethics accord the fetus at each stage of development, their stances on abortion range from outright bans to full

moral justifications. As a result, depending on the social, political, religious, and cultural context, abortion regulations continue to vary between nations [103].

In Iran, the "Therapeutic Abortion Act" of 2005 permits abortions only in the presence of a "definite diagnosis of retardation or malformation of the fetus that is unbearable for the mother." This allows abortions to occur no later than 4 months (19th week) after conception, after which time it is illegal and even criminal to attempt an abortion [104].

Conclusion

Zika virus is a significant international threat that demands urgent global. Achieving success requires ongoing comprehensive research to develop an effective vaccine that can effectively combat the virus. It is essential to investigate the interrelationship, action mechanisms, and transmission routes of DENV, ZIKV infection, and chickengunya. Developing a breakthrough in any of these mentioned viruses would be a notable milestone in the medical field, given their genetic similarities within the Flaviviridae family. Until a breakthrough is achieved, a detailed and multifaceted strategy should be utilized to enhance public awareness and manage ZIKV spread by preventing transmission through sexual contact, travel to affected regions and international commerce.

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