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## ZIKA VIRUS

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## ABSTRACT

The current Zika outbreak in Southern America, which began in December 2015, has kept the virus in the headlines for a while. The World Health Organization designated it as a public health emergency in February 2016 because of its link to congenital malformations, including microcephaly in children born to infected women. The virus's quick spread across the US and then Asia has caused grave fears for countries around the world. The population of India is seriously threatened by its spread to nearby nations. An overview of the virus, its diagnosis, clinical characteristics, and management is provided in this review article.

## 1. INTRODUCTION

Zika virus (ZIKV) is a member of the Flavivirus genus in the Flaviviridae family, which includes important human infections such as tick-borne encephalitis virus (TBEV), Japanese encephalitis virus (JEV), West Nile virus (WNV), dengue virus (DENV), and yellow fever virus (YFV)(1,2). The positive-sense RNA genome of the enveloped virus ZIKV is approximately 10.7 kilobases in size. Similar to other flaviviruses, the genome of ZIKV encodes a single polyprotein that is cleaved into three structural proteins (capsid [C], premembrane [prM], and envelope [E]) and seven nonstructural proteins (NS1, NS2A, NS2B, NS3, NS4A, NS4B, and NS5) through posttranslational cleavage by both host and viral proteases (3,4). While prM stops premature fusion with host membranes, the C protein binds to viral RNA to create a nucleocapsid, and the E protein is essential for cellular attachment, entry, and fusion (5). The nonstructural proteins play a crucial role in controlling the transcription and replication of viruses. These proteins include NS1, NS2A, NS2B, NS3, NS4A, NS4B, and NS5. Additionally, they have the ability to lessen host antiviral reactions (1,6,7). ZIKV belongs to the mosquito-borne lineage of flaviviruses called Spondweni virus group. It shares a tight relationship with the four serotypes of DENV, sharing about 43% of the amino acid identity in the viral polyprotein and in the E ectodomain.

Comprehending the complex molecular structure and genetic composition of ZIKV is essential for appreciating its pathogenic potential and its interactions with host cells. The similarities and differences with other flaviviruses highlight the necessity for thorough investigation to clarify similarities and differences and eventually aid in the creation of potent antiviral tactics. ZIKV's evolutionary affinities and structural resemblances to other members of the flavivirus family offer a foundation for exploring possible cross-reactivity and synergy in the search for broadspectrum antiviral therapies. The Zika virus (ZIKV) was first discovered in a rhesus monkey in 1947 (8). It first infected humans in Nigeria, Africa, in 1954 (9). Less than 20 human cases were reported during the following 50 years, with the majority of the information coming from serosurveys of the yellow fever virus (YFV). During arbovirus research in Africa and fever studies in Asia, ZIKV was found in a variety of mosquito species (8,10-21). The first Zika fever outbreak occurred in 2007 in the Federated States of Micronesia's island of Yap in the Western Pacific. A larger-scale pandemic that caused an estimated 30,000 symptomatic infections later spread through French Polynesia in the South Pacific in 2013 and 2014. lesser outbreaks followed, with lesser ones in Vanuatu, the Solomon Islands, Samoa, and Fiji in 2015, and in New Caledonia, the Cook Islands, and Easter Island in 2014. Brazil reported the first instance of the virus in March of 2015, marking the year it arrived in the Americas. Autochthonous circulation of ZIKV was observed in over 20 countries or territories in South, Central, and North America, the Caribbean, and at the end of January 2016, an outbreak had been reported in November in West Africa (Cape Verde). Severe neurological consequences, including as microcephaly in newborns in Brazil and Guillain-Barré syndrome (GBS) in adults in French Polynesia, were brought about by the development of ZIKV. ZIKV coexists alongside dengue virus (DENV) and chikungunya virus (CHIKV) in French Polynesia and Brazil; these coexisting viruses are endemic in DENV and CHIKV and are probably found throughout the Americas, Asia, several Pacific islands, and Africa. The virus has spread to every area where Aedes aegypti and Aedes albopictus mosquitoes are present, following the same routes taken by DENV and CHIKV. The goal of this thorough analysis is to compile all of the information currently available on this newly discovered virus, providing insight into its development and effects in various geographical areas.



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2. EPIDEMIOLOGY

The Zika virus (ZIKV) was initially isolated in 1947 after being discovered in a rhesus monkey in Uganda. It was then isolated in 1948 from Aedes africanus mosquitoes from the same area, which began our understanding of this newly emerging infectious agent in its early stages (Dick et al., 1952). Following the discovery of ZIKV cases in humans in 1952, studies using serosurveillance revealed a wider spread of the virus throughout several Asian and African nations (MacNamara, 1954; Weaver et al., 2016). Serosurveillance studies in several African nations—Central Africa, Egypt, Gabon, Nigeria, Sierra Leone, Tanzania, and Uganda—showed that human populations possessed ZIKV antibodies (Smithburn,

1952; Dick, 1953; Smithburn et al., 1954a; Moore et al., 1975; Weaver et al., 2016).

With isolations from Aedes aegypti mosquitoes and human illnesses reported in Indonesia, reports from Malaysia indicated the first cases of ZIKV outside of Africa (Marchette et al., 1969; Olson et al., 1981). Serological evidence of ZIKV infection appeared in a number of Asian nations between the 1950s and the 1980s, including Vietnam, the Philippines, Thailand, India, Indonesia, Malaysia, and Pakistan (Smithburn et al., 1954b; Smithburn, 1954; Hammon et al., 1958; Pond, 1963; Darwish et al., 1983). ZIKV was mainly limited to Africa and Asia for the first sixty years after it was discovered, with occasional outbreaks. However, the first big ZIKV outbreak was recorded on Yap Island in the Federated States of Micronesia in 2007, marking a key turning point in the situation. There was a significant change in the virus's worldwide prevalence, with almost 75% of the population being infected (Duffy et al., 2009). The Asian lineage of ZIKV was identified as the cause of this outbreak (Lanciotti et al., 2008), indicating the virus's potential development as a worldwide concern. After causing outbreaks in French Polynesia in 2013–2014, ZIKV crossed the Pacific Ocean and spread to other Pacific islands, such as the Cook Islands, Easter Island, New Caledonia, and the Solomon Islands (Cao-Lormeau et al., 2014; DupontRouzeyrol et al., 2015; Musso et al., 2014a; Roth et al., 2014; Tognarelli et al., 2016). An estimated 11% of the population was impacted by the French Polynesia outbreak, which alone resulted in over 30,000 recorded cases of sickness (ECDC, 2014; Musso and Gubler, 2016). Between 2013 and 2015, the virus-likely imported from the Pacific-finally made its way to the Americas, causing a major outbreak in Brazil. Early in 2015, reports of the first instances came from Rio Grande do Norte; by May of the same year, autochthonous transmission was documented from Bahia. A startling 440,000 to 1,300,000 probable infection cases were reported by 18 states in Brazil by December 2015 due to autochthonous ZIKV transmission (Hennessey et al., 2016; Musso and Gubler, 2016). The rapid spread of ZIKV infections was made possible by the mosquito vectors' extensive distribution throughout Brazil, where Aedes aegypti was common in the northern, northeastern, and central eastern regions and Aedes albopictus in the southern region. In addition, the virus spread to other American nations, such as Venezuela, El Salvador, Paraguay, Panama, El Salvador, Guatemala, and several Caribbean states (Hennessey et al., 2016; Musso and Gubler, 2016). Concerns regarding a possible connection between ZIKV infection during pregnancy and foetal abnormalities were sparked by the startling increase in the number of instances of microcephaly in Zika-affected areas of Brazil. Almost 4,300 cases of foetal anomalies, such as microcephaly, had been documented by February 2016 (de Araújo et al., 2016; Victora et al., 2016). The impact of ZIKV on public health was given a fresh perspective by this association, which highlighted the need for increased study and surveillance. 2016 saw the introduction of ZIKV into the continental United States; cases were first noted in Florida and Texas, indicating that the virus was now present in North America (McCarthy, 2016). The potential for ZIKV to spread globally and pose a serious threat to international health was highlighted by its recent global dissemination. Asymptomatic illnesses started to appear as the virus carried on with its silent global advance. Three ZIKV-positive cases were reported in Gujarat by the Indian Ministry of Health and Family Welfare in May 2017, and one more case in Tamil Nadu was confirmed (WHO, 2017; Bhardwaj et al., 2017). These cases were noteworthy since they were the first to demonstrate the presence of ZIKV in the Indian population without a history of travel to endemic locations, suggesting that the virus may be endemic in the nation (Sapkal et al., 2018). A significant ZIKV outbreak occurred in the Indian states of Rajasthan and Madhya Pradesh in 2018. The greatest ZIKV outbreak in India happened between September and November 2018, with 130 confirmed cases in Madhya Pradesh and 159 confirmed cases in Rajasthan, including 64 pregnant women (IDSP, 2018). This outbreak brought to light ZIKV's dynamic nature and ability to spread to new areas, presenting continuous difficulties for global public health. In summary, the path of the Zika virus from its original discovery in Africa to its current status as a worldwide health threat highlights the intricate dynamics of infectious diseases in our globalised society. The virus's capacity to propagate covertly, result in serious consequences like microcephaly, and adapt to new habitats highlights the significance of ongoing worldwide surveillance and cooperative research initiatives. It is still essential to be on guard, comprehend the epidemiology of ZIKV, and create practical global prevention and control plans while the virus keeps changing.



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ear	Event	Area or country	No. of infected cases	Reference
947	Discovery of ZIKV in rhesus monkey	Zika forest, Uganda	_	Dick et al. 1952
948	Isolation of ZIKV from Aedes mosquito	Zika forest, Uganda	_	Dick et al. 1952
953	First isolation of ZIKV from human	Nigeria, Tanzania, Uganda	3	MacNamara 1954
950s-1980s	Sporadic mild cases reported from Asian countries	Indonesia, India, Malaysia, Pakistan, Philippines, Thailand	_	Smithburn 1954; Smithburn et al. 1954b; Hammon et al. 1958; Pond 1963; Marchette et al. 1969; Olson et al. 1981; Darwish et al. 1983
007	First large outbreak of ZIKV	Yap Island in the Federated States of Micronesia	49 confirmed and 7391 suspected cases	Duffy et al. 2009
013–2014	French Polynesia ZIKV outbreak, evidence of the Guillain–Barré syndrome; sexual transmission	French Polynesia, Easter Island, Cook Islands, New Caledonia	28 000-30 000 cases in French Polynesia; 1400 confirmed cases in New Caledonia	Cao-Lormeau et al. 2014 Musso et al. 2014b; Roth et al. 2014; Tognarelli et al. 2016; Dupont-Rouzeyrol et al. 2015
015-2016	ZIKV outbreak in the Americas	Brazil, Columbia, El Salvador, Mexico, Paraguay, Venezuela, Caribbean countries	440 000–1 300 000 estimated cases in Brazil; 51 473 suspected cases in Columbia	Zanluca et al. 2015; Hennessey et al. 2016
016	ZIKV in continental USA	Florida and Texas	4 cases in Florida	McCarthy 2016
017	First confirmed ZIKV outbreak in India	Gujarat, Tamil Nadu	3 cases in Gujarat; 1 in Tamil Nadu	Bhardwaj et al. 2017; WHO 2017
018	Biggest ZIKV outbreak of India	Rajasthan, Madhya Pradesh	159 confirmed cases including 64 pregnant women in Rajasthan; 130 cases in Madhya Pradesh	IDSP 2018

## 3. MODES OF TRANSMISSIONS

Vector-borne transmission - The Zika virus (ZIKV) is characterised by vector-borne transmission, which is mostly made possible by mosquitoes. Only a subset of Aedes mosquito species, such as Ae. aegypti, Ae. albopictus, Ae. hensilii, and Ae. polynesiensis, show to be effective vectors for the transmission of ZIKV, despite the virus having been isolated from many of them (22–32). Of them, Aedes aegypti is the most notable vector responsible for the ongoing outbreak of ZIKV in Latin America and the Caribbean. This is explained by the anthropophilic inclinations and urban prevalence of the mosquito. Although it is assumed that monkeys act as reservoir hosts for ZIKV, the primary species is still unknown. We still don't know if ZIKV will become endemic in New World monkeys and initiate a sylvatic transmission cycle in Latin America similar to that of yellow fever virus (YFV) or if it will only spread through urban transmission cycles, as dengue virus (DENV) does. Because humans serve as ZIKV's amplifying hosts, they maintain and spread urban cycles of mosquito-human transmission, which feeds epidemics. Interestingly, a widespread ZIKV outbreak was observed on the Micronesian island of Yap in the absence of nonhuman primates, highlighting the importance of the dynamics of human-mosquito transmission (19). The information available now does not support the involvement of animals other than humans and nonhuman primates as amplifying hosts for ZIKV, which more closely resembles the modes of transmission of DENV, YFV, and CHIKV. While there is no denying that mosquito-borne transmission is the main cause of ZIKV epidemics, there have also been reports of other mechanisms of transmission. The complicated interplay between amplifying hosts, reservoir hosts, and vectors highlights the intricacy of ZIKV transmission dynamics. Developing successful control tactics requires an understanding of the potential for sylvatic cycles and the factors impacting the virus's persistence in urban areas. While ZIKV continues to be a public health concern, research efforts are being made to better understand how it spreads and provide useful information for intervention and prevention tactics.

**Blood-borne transmission** - Similar to other bloodborne illnesses, there is a chance that a viremic ZIKV donor could contaminate the blood supply by blood-borne transmission. Although reports of ZIKV transmission through donated blood transfusions have been made in Brazil, these reports have not yet been officially released. Once a screening test is developed, a similar strategy might be used for ZIKV detection in areas like the United States, Canada, and Europe where the blood supply is regularly checked for West Nile virus (WNV) using nucleic acid amplification testing. In an effort to highlight the value of taking preventative measures, a number of nations have started to test the blood supply for ZIKV or to postpone accepting blood donations from those who have visited nations where the virus is circulating. Although there isn't a recognised diagnostic test for ZIKV, there are methods to render infectious organisms inactive in the bloodstream, which provides an additional layer of protection against possible spread.

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Sexual transmission- There is data that suggests ZIKV can be transferred sexually, which raises further concerns around sexual transmission. Semen has been found to contain ZIKV RNA, and cases of ZIKV infection that have been sexually transmitted have mostly involved infected men infecting their female partners. It is interesting to note that infectious ZIKV was found in semen long after the viremia subsided, casting doubt on theories of bloodborne transmission. It is noteworthy that, unlike other sexually transmitted illnesses, hematospermia-the presence of blood in semen—was not a consistent sign in all cases of sexually transmitted ZIKV. The likelihood of ZIKV replication in urogenital tissues is suggested by recent findings of infectious ZIKV in urine and the survival of ZIKV RNA in urine after viremia has cleared. The distinction between sexual and salivary transmission is made more difficult by the finding of ZIKV RNA in saliva as well as reports of infectious ZIKV in saliva. A historical analogy would be the herpesvirus linked to Kaposi's sarcoma, which was once believed to be spread by intercourse but was subsequently shown to be largely transferred through saliva. Furthermore, pigs have been shown to have significant viral loads in their tonsils and to spread the Japanese encephalitis virus (JEV) via oronasal secretions, suggesting that pigs may be a potential source of flavivirus transmission. Even though ZIKV outbreaks may not be primarily caused by sexual transmission, the virus's presence in semen warrants careful research, particularly in light of recent discoveries showing that ZIKV RNA can be detected in semen for a considerable amount of time after acute symptoms have resolved. Because the testes are immune-privileged, there is a chance that ZIKV will survive in this tissue and act as a reservoir to start fresh cycles of transmission from people who appear to be in good condition. The increase in imported ZIKV cases in parts of the US and Europe where local mosquito transmission is less common offers a chance to examine and understand the importance of other modes of transmission. Comprehensive study and watchful surveillance are essential to identify the various pathways of transmission and adopt effective preventative efforts as the world grapples with the complex issues posed by ZIKV.

Maternity transmission - Zika virus (ZIKV) transmission through breastfeeding adds a complex element; ZIKV RNA has been found in breast milk, which raises questions about possible transmission to nursing infants. Other flaviviruses have been shown to have comparable modes of transmission. It is uncertain, therefore, if infectious ZIKV is found in breast milk and how long it lasts in comparison to acute infection. ZIKV-infected women are currently advised to breastfeed their children in spite of these issues. It is unclear whether ZIKV is transmitted in utero, through breast milk, or through bloodborne transmission at delivery, despite reports of perinatal transmission in French Polynesia. The subject of in utero transmission has become more urgent, especially in Brazil where a sharp increase in cases of microcephaly has corresponded with the introduction of ZIKV. In just four months, the northeastern states recorded almost 4,000 cases—a 20-fold rise over the previous year. The definition of microcephaly, a congenital disorder marked by underdeveloped foetal brains, is not well established, which makes analysis more difficult. Microcephaly during pregnancy can be caused by a variety of reasons, such as genetic mutations, exposure to toxins, and viral infections such as varicella-zoster virus, rubella virus, and human cytomegalovirus. The long-term consequences vary from almost no flaws to significant physical and mental impairments. Most instances of microcephaly reported during the current outbreak are not yet directly linked to ZIKV or have not been confirmed. Current follow-up studies have confirmed about one-third of reported occurrences, pointing to possible other explanations. Further complicating analysis are modifications made to the case criteria for microcephaly throughout the outbreak. Brazil, for example, adopted New born head circumference measurement of 32 cm in December 2015, replacing the previous, less strict 33-cm threshold. In-depth information is necessary to evaluate the possible connection between ZIKV and microcephaly; further epidemiological research, such as prospective cohort and casecontrol studies, should shed light on this issue in the future. Despite the difficulties, mounting data clearly points to ZIKV's causative involvement in the development of microcephaly. The identification of ZIKV RNA in tissues and the placenta of infants with microcephaly, as well as the sequencing of viral genomes in the amniotic fluid of foetuses with microcephaly, are factors that contribute to transplacental infection. The theory of in utero infection is further supported by the finding of anti-ZIKV IgM in the cerebral spinal fluid of infants with microcephaly. Although microcephaly can be caused by other viruses, flaviviruses have not previously been linked to this presentation. In response, international health organisations have issued travel warnings, advised expectant mothers to stay away from ZIKV-affected areas, and advised delaying pregnancy if there is a chance of sexual transmission. Variations in the outcomes of pregnancies exposed to ZIKV highlight the need for ongoing research to completely comprehend the complexity of maternal transmission and its ramifications.

#### PATHOGENISIS

Particularly in light of recent outbreaks in Brazil, research done on mice four to six decades ago has shed light on the possible pathophysiology of the Zika virus (ZIKV) and its connection to microcephaly. Although there have been no recent research on the pathophysiology of ZIKV-related microcephaly in Brazil, previous mouse tests have revealed **@International Journal Of Progressive Research In Engineering Management And Science** Page | 620



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that ZIKV may have a preference for brain cells in certain situations. Understanding the mechanisms that may cause the microcephaly instances that have been seen depends on this observation. It was in 1947 when George Dick and associates discovered the first strain of ZIKV, referred to as MR 766. A 5- to 6week-old Swiss mouse whose brain had been intracerebrally injected with the serum of a feverish sentinel rhesus macaque was the subject of the isolation (34). The same group's later research found that intracerebral inoculation of mice of various ages with passaged ZIKV strains caused symptoms of central nervous system (CNS) illness, such as motor weakness and paralysis (33). It's interesting to note that mature mice showed reduced susceptibility to deadly ZIKV infection when administered intraperitoneally, while mice younger than 7 days of age showed greater susceptibility (36). This age-dependent susceptibility raises the possibility of an early developmental vulnerability.

The pathogenic signs of ZIKV infection were mostly restricted to CNS regions in mice. In areas of the brain and spinal cord, observations included cellular infiltration and neuronal degeneration. Cowdry type A inclusion bodies were also present, a condition linked to herpesvirus-induced neuronal infection (33). The possible neurotropism of ZIKV in mice is highlighted by the specificity of these pathogenic findings in CNS tissues. The results of a pathological assessment of a human foetus infected with ZIKV in gestation corroborated these conclusions. In this instance, the brain stem and spinal cord were also affected, and there was diffuse astrogliosis and microglia activation. It was also observed that the descending corticospinal tracts have Wallerian degeneration (35). The similarity between the pathogenic observations in mice and a human foetus points to a possible ZIKV effect on the growing neurological system.

Interestingly, ZIKV infection did not significantly manifest in mice's kidney, lung, spleen, or liver, among other organs besides the central nervous system. Conversely, even after intracerebral injection, a number of species, including rhesus monkeys, cotton rats, guinea pigs, and rabbits, did not acquire CNS illness (33). These findings highlight the specificity of ZIKV's effect on CNS tissues, highlighting the significance of comprehending the virus's selectivity and possible ramifications. Utilising a ZIKV isolate from French Polynesia, recent research has shed more light on how the virus interacts with human cells. These investigations showed that human keratinocytes, dermal fibroblasts, and skin biopsy tissues were infected with ZIKV. This is consistent with the theory that, like other flaviviruses including West Nile virus (WNV) and Dengue virus (DENV) infections, the skin is the first site of ZIKV replication after insect inoculation (37-40). ZIKV can use DC-SIGN and the TAM receptors Axl and Tyro3 as attachment factors, just like DENV can (37). Furthermore, ZIKV was reported to be inhibited by the antiviral properties of type I and type II interferon when it infected human dendritic cells in culture (37).

This highlights the intricate relationship between the virus and the host's immune system and emphasises the necessity for a thorough understanding of ZIKV immunopathogenesis. One feature of ZIKV that is worth mentioning is its envelope (E) protein, which varies in its N-linked glycosylation. While some ZIKV strains lack anticipated glycosylation sites (41) others have a single N-linked glycosylation site (N154) in their E protein. DENV contains two N-linked glycosylation sites (N67 and N154), but its glycosylation pattern is different. ZIKV's pattern is more akin to that of other flaviviruses that are not closely related, such as WNV and Tick-borne encephalitis virus (TBEV), both of which have N154 glycosylation(42-44). Although N-linked glycosylation on the E protein has been linked to increased virulence in mammals and enhanced mosquito transmission for some flaviviruses, like TBEV and WNV, (45–51), it is still unknown whether or not different glycosylation between ZIKV strains influences or is correlated with pathogenicity.

In conclusion, historical mouse studies reveal important information on ZIKV's possible neurotropism and its connection to CNS illness, drawing comparisons with documented human occurrences of microcephaly. We now know more about the complicated dynamics between the virus and the host's immune response as well as how it interacts with human cells, especially in the skin, thanks to recent research that have used isolates from French Polynesia. The E protein's variable N-linked glycosylation adds another level of intricacy to the possible factors influencing ZIKV pathogenicity. As studies go on, a thorough understanding of ZIKV pathogenesis will be essential to creating methods that effectively prevent, treat, and lessen the virus's negative effects on human health.

#### Signs and symptoms

Most infections caused by the Zika virus have no symptoms at all. If they do occur, the symptoms are typically mild and include rash, fever, conjunctivitis, muscle and joint pain, malaise, headache, and usually manifest 3-14 days after the infection.

They also typically last 2–7 days. Given that these symptoms are present in both arboviral and non-arboviral disorders, the diagnosis of Zika virus infection requires laboratory confirmation. Not all infected individuals will exhibit symptoms. Within two weeks of being bitten, the following symptoms and indicators may appear:



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- High temperature
- Rash
- · Pain in the joints
- Pain in the muscles
- Hadache
- Retinal inflammation (red eyes)

Only 1 in 5 people with the illness show symptoms, which are often moderate.

#### Identification

#### Zika virus infection diagnosis in a lab setting.

Zika virus laboratory diagnosis can be achieved by virus isolation, antigen detection, viral RNA detection with molecular assays, and anti-Zika virus antibody detection with serological assays, depending on the goal of the research (35).

#### **Separating Viruses**

The initial technique to isolate the Zika virus was intracerebral mouse inoculation, which is currently the accepted approach for isolating arboviruses (25, 36). Human clinical materials such as blood (37), urine (38), saliva (39) and semen (40) can be used to cultivate the Zika virus.

#### **Identification of Antigens**

Antigen detection is an effective method for verifying whether the Zika virus is present in postmortem tissues. Using the immunohistochemistry (IHC) method, zika virus antigen has been discovered in the brain and placental tissues of congenitally infected newborns who have suffered microcephaly and miscarriages (41, 42). Recently, a wide range of innovative tests have been created to identify the Zika virus antigen. These include the following: an aptamer-based ELISA assay for targeting the Zika virus NS1 protein (44), NS1 protein-based competitive ELISA (45), and NS1 protein-based fast assays (46). NS3 protein identification in whole blood is achieved by flow cytometry (43).

#### **Molecular Assays**

Zika virus RNA has been detected in a wide range of body fluids, including blood (plasma or serum), urine, saliva, and semen, breast milk, conjunctival fluid, amniotic fluid, and the brain and placental tissues of congenitally infected foetuses. The "gold standard" for diagnosing ZIKV is reverse transcriptase PCR (RT-PCR), which is incredibly sensitive and specific. For Zika virus-specific RT-PCR, a number of traditional and real-time assays that target the prM, E, NS1, NS3, NS4, and NS5 genes have been developed. To yet, nevertheless, the Food and Drug Administration (FDA) has only approved one commercial assay. This is the CobasZika Test (Roche), a qualitative nucleic acid test that can be used to screen blood for Zika virus RNA. contributors Furthermore, several molecular assays have been approved by the FDA under an emergency use authorization (EUA). The AptimaZika virus test, which is based on the transcription-mediated amplification (TMA) technique, is awaited. These include the Triplex Real-Time RT-PCR, Multiplex Assay for Zika Virus, Dengue Virus, and Chikungunya Virus (CDC), Zika Virus RNA Qualitative Real-Time RT-PCR (Quest Diagnostics Infectious Disease, Inc.), and the RealStarZika virus RT-PCR kit (Altona Diagnostics, GmbH). For the molecular diagnosis of Zika virus infection in people, plasma or serum specimens are usually employed within the first week after the onset of clinical symptoms. There have been some intriguing findings demonstrating the ZIKV RNA's shorter persistence in urine as opposed to serum. This is true even though the long period of viral shedding in this readily collected samples provides multiple lines of evidence supporting the urine's advantage for Zika virus RNA detection.

#### Serology Analysis

Even though molecular diagnosis has a high sensitivity and specificity, a brief time of viremia can negatively impact Zika virus RNA detection. Therefore, serological assays for the detection of anti-Zika virus antibodies can be a wise option if you're looking for a longer diagnostic window. Anti-Zika virus antibodies can be found using a variety of serological techniques, such as complement fixation, haemagglutination inhibition, immunofluorescence (IF) testing, ELISA, and neutralisation tests. Anti-Zika virus IgM antibody develops within the first week after the onset of symptoms and is often detected between days five and twelve of illness. IgG antibodies can be monitored for several months to years and rise in response to the Zika virus a few days after IgM. The FDA has not yet approved any serological tests for the Zika virus. However, the FDA has authorised the use of five serological assays with emergency use authorization for the detection of anti-Zika virus IgM antibodies. The CDC's IgM antibody capture



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ELISA (Zika MAC-ELISA), InBios International, Inc.'s ZIKV Detect IgM capture ELISA, DiaSorin Incorporated's Liaison XL Zika capture IgM assay, Siemens Healthcare Diagnostics Inc.'s ADVIA Centaur Zika test, and Chembio Diagnostic Systems, Inc.'s DPP ZikaIgM system are among these assays. Because of the potential for nonspecific reactivity and cross-reactivity with other Flaviviruses, including dengue and yellow fever viruses, results of IgM detection assays should be evaluated with caution. Plaque-reduction neutralisation testing is required to confirm positive or unclear results (PRNT).

#### Laboratory Biosafety

Human sickness caused by the Zika virus is assigned to risk group 2. Therefore, Biosafety Level 2 (BSL-2) facilities should house laboratories that conduct diagnostic tests. The virus can be rendered inactive by ultraviolet (UV) radiation, temperatures above 58 °C, pH-6.2 liquids above <7.8, ether, and 5% potassium permanganate.

## 4. CONCLUSION

Recent ZIKV infection outbreaks in the Americas have raised awareness of this newly discovered arbovirus disease and its potential to cause congenital abnormality (microcephaly) in children born to infected mothers. With ZIKV spreading so quickly and infecting nations throughout Asia, including India's neighbors, there is cause for concern on a global scale. The rising threat of diseases carried by mosquitoes as a result of insufficient control over this vector's breeding is the other epidemic control issue that will need to be addressed in the future. Large-scale research on humans and animals from all over the world is also required in order to create precise management protocols to prevent ZIKV. Moreover, priority should be paid to controlling the new and cryptic virus that is posing a threat.

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