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A COMPREHENSIVE REVIEW OF ARBOVIRUSES: TRANSMISSION, PATHOGENESIS, AND GLOBAL IMPACT

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ABSTRACT

Through intricate cycles involving the infectious agent, vertebrate hosts, and vectors, arthropod vectors spread a wide variety of viruses collectively referred to as arboviruses. Given the substantial number of infections that affect humans and animals each year, it is essential to comprehend the interaction between biological and environmental factors that underlie arbovirus pathogenesis, disease progression, and transmission. Arthropod vectors and vertebrate hosts are involved in intricate biological transmission cycles that naturally contain arboviruses. These viruses are mainly categorized into families such as Bunyaviridae, Togaviridae (Alphaviruses), and Flaviviridae. Because they can infect humans, mammals, and occasionally plants, these individuals are unique. Due to factors such as increased globalization and environmental changes, arboviruses have reemerged and spread worldwide in recent decades, despite advances in vector management. Millions of infections and significant mortality have been caused by this resurgence, especially in developing nations.

Keywords: Arboviruses, Chikungunya virus, Dengue virus, Ticks, Zika virus, Japanese encephalitis virus, Hemorrhagic fever.

1 INTRODUCTION

Arbovirus infection may trigger a multitude of disease-related syndromes, among which are hemorrhagic fevers, encephalitis, and systemic febrile diseases. Human pathogens encompass the Japanese encephalitis virus, West Nile virus, yellow fever virus, and the four different dengue viruses (dengue viruses 1-4). Bluetongue, Venezuelan equine encephalitis, Nairobi sheep disease, and Rift Valley fever are among those veterinary maladies caused by viruses. With the notable exception of the African Swine Fever virus which is admitted as tick-borne illnesses and is the only member of arboviruses that harbors double stranded DNA genome as an exception, were the most widespread cause of morbidity and illness in humans from the 17th to the 20th century, outpacing all other causes [1]. The World Health Organization (WHO) reports that millions of people die each year from communicable diseases, which continue to be a major cause of death worldwide, especially in developing nations. Among these, vector-borne arboviruses like West Nile, dengue, Zika, and yellow fever are major worldwide risks. Additionally, the cross-border spread and urban amplification of these

pathogens are facilitated by increased international travel and rapid globalization [2]. However, during the recent past few decades, arthropod virus infections have grown more prevalent beyond Africa.

The World Health Organization (WHO) and the Centers for Disease Control and Prevention (CDC) estimate that the yellow fever virus (YFV) causes 200,000 severe clinical cases and up to 30,000 to 60,000 deaths per year, mostly in tropical regions of Africa and South America [3]. Travel and globalization may have contributed to some of these municipal epidemics by making it simple for YFV to spread across national boundaries. The 1990s-related dengue fever outbreak in the Americas was determined to possess a pandemic potential comparable to that of the Southeast Asian region 30 years ago [4]. Over the past 40 years, the dengue virus (DENV) has propagated, so it is currently believed to infect 390 million people annually. Globalization and travel may contribute to some of these urban epidemics by promoting the cross-border propagation of YFV [5]. The 2007 Singapore dengue fever crisis, which showed serotype change and clade substitution, enabling the fusion of numerous genotypes, is another variable that is thought to contribute to serum epidemiological characteristics. After 1986, young adults were primarily affected by the dengue fever outbreak in Singapore. After entering the Western Hemisphere in 1999, the West Nile virus (WNV) swiftly rose to prominence as one of the most prevalent arthropod viruses globally [6]. By 2017, over eight million probable cases of the chikungunya virus (CHIKV), which first showed up in the Americas region in late 2013, had been reported in 44 different countries. Serum epidemiology is found to be influenced by another factor. Up with 1500 WNV cases had been reported in the EU by November 2018, surpassing the total number of infections reported over the preceding five years [7]. The Zika virus (ZIKV) had been quietly circulating in Africa and Asia for decades prior to causing significant epidemics in the Pacific, including Yap Island in 2007. After being brought to the Americas in 2013 or 2014, it caused a significant outbreak in 2015 or 2016 that exposed its link to congenital microcephaly and serious neurological issues.

The vast majority aforementioned arbovirus vectors are hematophagous type insects of the order Diptera, including biting midges, mosquitoes, sandflies, and black flies [8]. The vector-arbovirus relationship is extremely specific because most bug and species of tick do not act as arbovirus vectors, and even within insect or tick species, there is frequently substantial variation in vector proficiency among populations. Vector competence frequently varies significantly between populations, regardless of insect or tick species. The association that exists between the arbovirus and vector thus becomes highly specific.

2 THE LIFECYCLE AND TRANSMISSION OF ARBOVIRUSES

Arboviruses survive in nature through a continuous "vector-host" cycle. This process involves a virus jumping between an animal (the host) and an insect or arachnid (the vector), such as a mosquito or a tick.

2.1 The Core Transmission

2.1.1 Vector Dynamics

Ticks and mosquitoes seem to be the most prevalent arthropod species that arboviruses can infect. About 300 different types of mosquitoes can spread arboviruses in addition to developing long-term survival strategies. The most closely related species, *Aedes* and *Culex* mosquitoes, can carry 115 and 105 different types of arboviruses, respectively. Ticks also usually harbor 116 other species that are known to spread arboviruses, namely sandflies, blackflies, stink bugs, lice, mites, gadflies, bedbugs, and 25 midge species, mostly *Culicoides* (24 types) and *L. iohelea*. In particular, the Japanese encephalitis virus appears to be spread by midges, sandflies, and other mosquito species, such as *Culex*, *Anopheles*, and ticks [9]. Nevertheless, numerous other arthropod species that have been found to carry viruses may contribute to the virus's life cycle without being directly linked to overt disease occurrence in humans or animals. However, if the availability of the terrestrial host—such as birds—becomes scarce toward the end of the summer due to the birds' migration to warmer nations, the vector may switch to a different vertebrate host. This is in line with the finding that *Culex* mosquito vectors are primarily responsible for West Nile virus outbreaks in North America. In contrast, the vector ecology of viruses such as dengue and chikungunya is different: *Aedes albopictus* flourishes in both peri-urban and rural areas, while *Aedes aegypti* is highly adapted to only urban environments and human dwellings. For example, CHIKV efficiently spread in both rural and urban areas on La Réunion island, where *A. aegypti* was absent, mainly through *A. albopictus* [10]. But these are not strict guidelines. on La Reunion, an island in France. Japanese encephalitis is spread by pigs, yellow fever by monkeys, and Russian Spring-Summer encephalitis and VEE by rodents.

2.1.2 The vector Midgut as the Initial Site of Infection

The cardiac (stomodaeal) valve controls the bloodmeal's unidirectional flow and stops the midgut's contents from flowing backward into the foregut. It is situated at the proventriculus junction between the foregut and midgut. The trachea and circular and longitudinal muscle cells surround the midgut. The pharynx, esophagus, crop, and sclerotized proventriculus collectively make up the mosquito's foregut as illustrated in figure 1 [11]. The mosquito midgut is split into a confined anterior region and a wider posterior region. When a bloodmeal is consumed, the midgut's epithelial cell structure becomes significantly altered; rough ER whorls form, electron-opaque material gathers, and the nuclei

condense while the mitochondria enlarge [12]. Bloodmeal acquisition causes significant changes to the midgut's epithelial structure, notably the accumulation of electron-opaque material, the formation of rough ER whorls, and the decomposition of nuclei while the mitochondria grow [13].

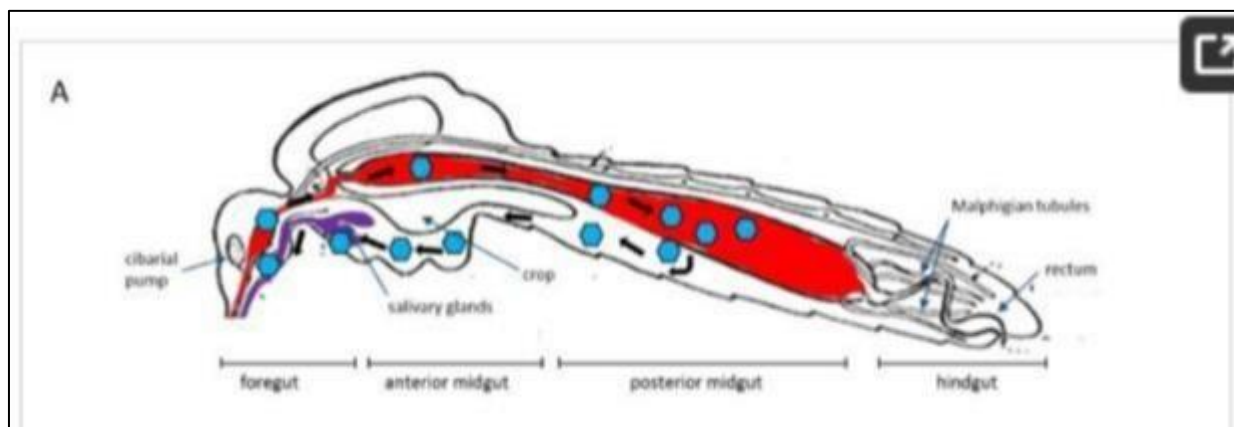


Figure 1: internal anatomy of mosquito's digestive system

In order for an arbovirus to infect a mosquito vector, the virions must successfully pass through tissue barriers. Arboviruses enter the midgut lumen after grazing on a vertebrate host's viremic bloodmeal [14]. In order to infect the midgut epithelial cells through their microvilli, the virions in the bloodmeal must pass through the gut lumen. Early viral entry usually happens quickly before the matrix fully consolidates or through structural pores during its early formation, despite the fact that blood digestion causes the midgut epithelium to secrete a chitinous peritrophic matrix (which in *A. aegypti* begins developing 4–8 hours post-blood meal and matures by 12 hours)[15]. In the beginning, only one to five of the approximately a hundred midgut cells that seemed to be vulnerable to viral infection were repeatedly infected as shown in figure 2.

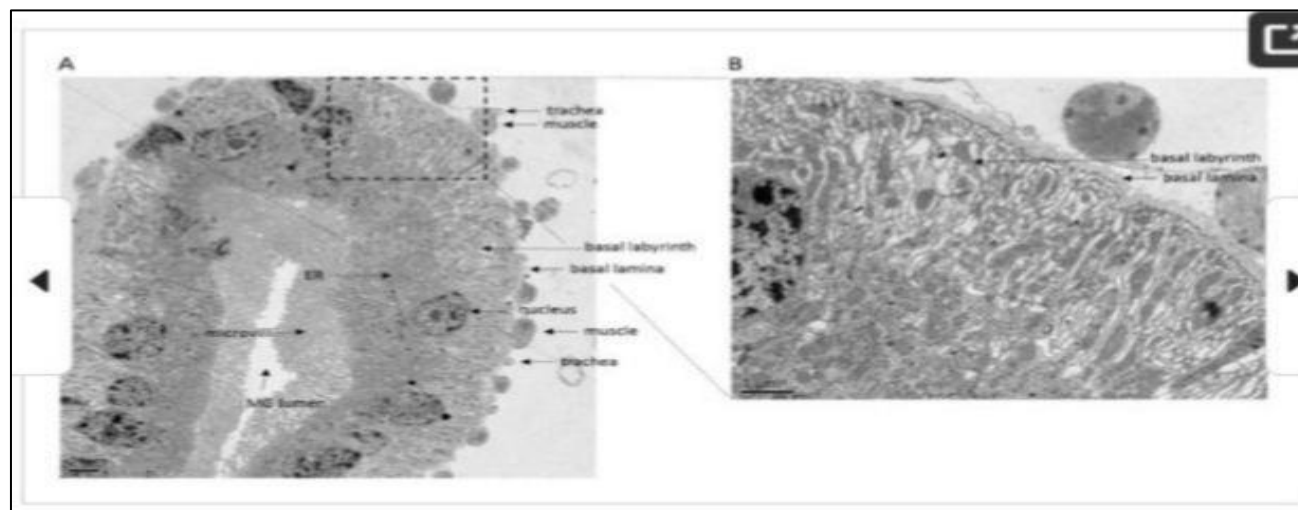


Figure 2: Ultrastructural images of *A. aegypti* posterior midgut tissue.

Viral RNA replication is carried out at the ER membrane one time the virus has penetrated a midgut epithelial cell; though, location of viral RNA maturation could vary based on the virus and type of mosquito. De novo synthesized SLEV virions, for example, first appeared in the cisternae of the ER in *C. pipiens* before building up in the basal labyrinth and the extracellular spaces that separate the BL and the cell plasma membrane. EEEV in *A. triseriatus* and Western equine encephalitis virus (WEEV) in *A. dorsalis* have both demonstrated comparable results [16]. However, while virions are assembled at the plasma membrane lining the basal labyrinth, WEEV in the midguts of *C. tarsalis* and chikungunya virus (CHIKV) in *A. aegypti* do not seem to enter the ER cisternae. Instead, the basal labyrinth's plasma membrane is the location where virions are assembled as explained in figure 3.

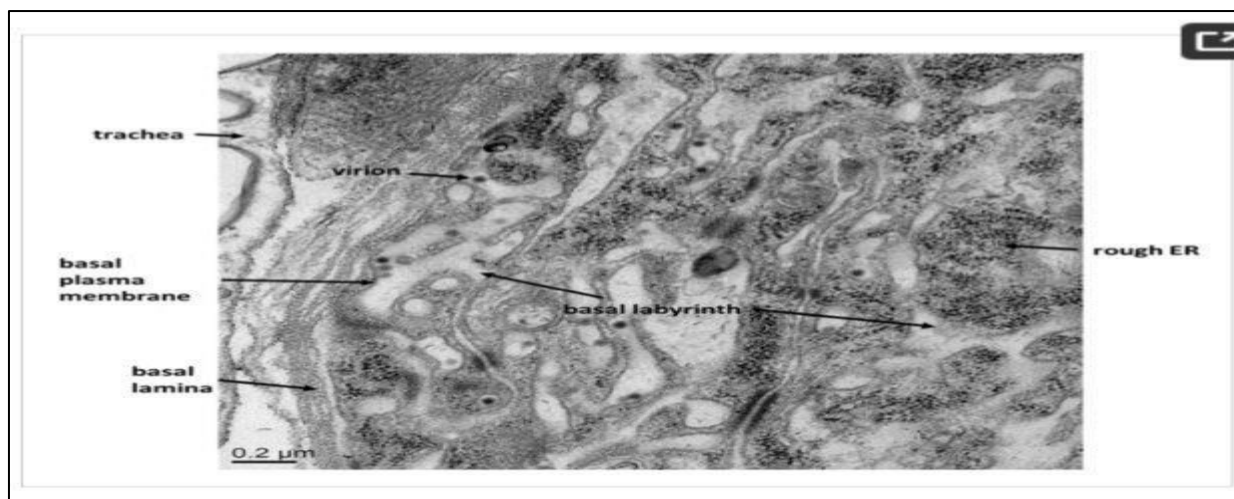


Figure 3: An ultrastructural view of the posterior midgut tissue of a female *A. aegypti* that had contracted CHIKV orally seven days after infection.

2.1.3 Transmission of Viruses from the Midgut to Secondary Tissues

It has been suggested that an arbovirus needs a way to amplify itself after leaving the midgut epithelial cells in order to successfully infect the salivary glands [17]. It has only been demonstrated that a small number of viruses, such as WNV and VEEV, can infect muscle tissue after exiting the midgut. Usually, arboviruses spread to secondary tissues such as nerve tissue, hemocytes, and fat body. Hemocytes may form an essential continuum between the midgut tracheoles and the salivary glands. Prior to the virus infecting the mosquito's salivary glands, hemocyte infection was discovered to be an important amplification step for arboviruses outside the midgut; once the mosquito's salivary glands are infected, the virus spreads through the saliva. SINV infection and viral replication outside the midgut primarily target differentiated phagocytic cells, particularly granulocytes and plasmatocytes, among the different populations of mosquito hemocytes [18].

2.2 Human Infection and "Dead-End" Hosts

While arboviruses such as Zika, Dengue, and West Nile can cause serious illness in humans, humans are often considered dead-end or incidental hosts. This means that although a person may become very sick, they usually do not develop a high enough level of virus in their blood (viremia) to pass it back to a biting mosquito. Consequently, the cycle often ends with the human infection as demonstrated in figure 4.

2.3 Other Ways the Virus Spreads

While insect bites are the primary route, your research highlights that some arboviruses (like Zika) have evolved additional transmission methods:

- Vertical Transmission: An infected female mosquito can sometimes pass the virus directly to her offspring.
- Human-to-Human: In rare cases, viruses can spread through blood transfusions, organ transplants, or from a mother to her baby during pregnancy or delivery. Zika is also unique because it can be transmitted sexually.

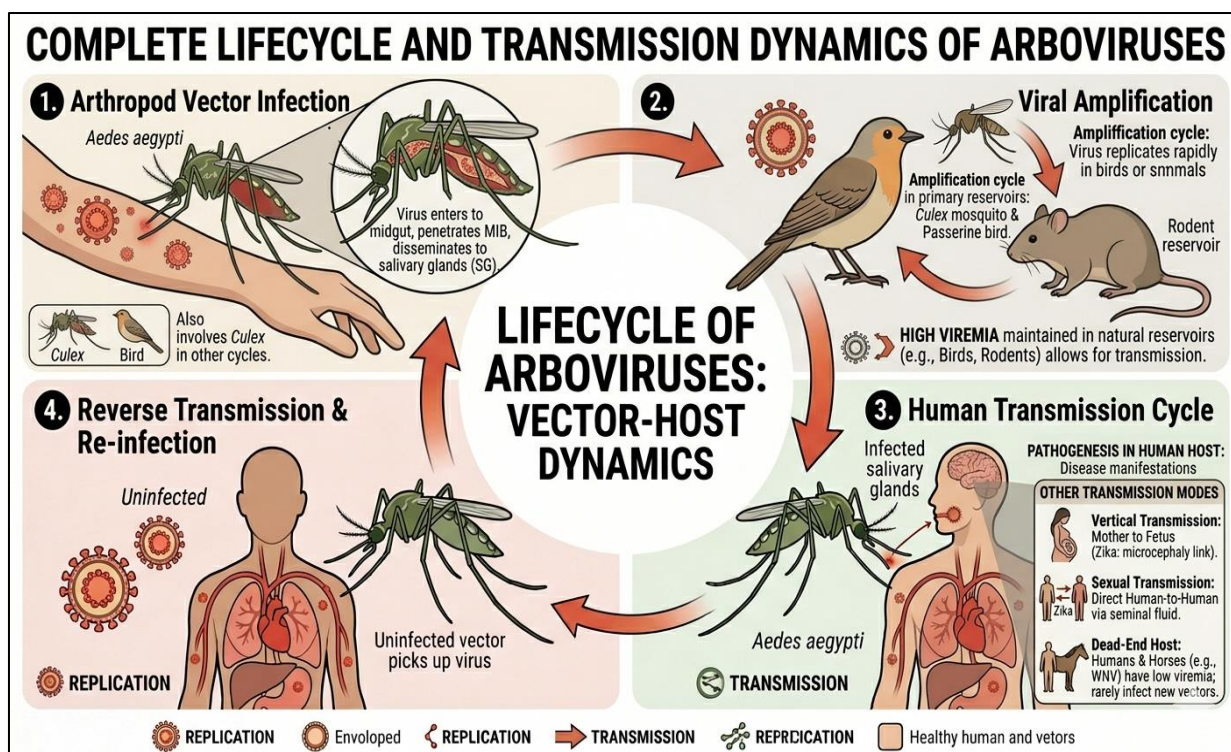


Figure 4: Life cycle of arboviruses

3 CLASSIFICATION OF ARBOVIRUSES:

In times gone by, arboviruses were categorized into four groups: A, B, C, and D. Group C was recognized as the serogroup in the Orthobunya virus genus, Group D was renamed the Guama group in the Orthobunya genus in the mid-1950s, Group A consisted of viruses from the Alphavirus genus, and Group B contained viruses from the Flavivirus genus. Mosquitos transmit many flaviviruses, which are single-stranded, enveloped RNA viruses that include West Nile, Zika, and yellow fever viruses. Additionally, a few examples of tick-borne flaviviruses include Kyasanur Forest disease, Omsk hemorrhagic fever, and tick-borne encephalitis. California encephalitis and Jamestown Canyon virus are two examples of the segmented, enveloped RNA viruses known as bunyaviruses, which are transmitted by a range of arthropods, such as ticks, mosquitoes, and flies. Alphaviruses are single-stranded RNA viruses with a coated virion that are enveloped and a member of the Togavirus family. Notable alphaviruses include western and eastern equine encephalitis, Ross River virus, and chikungunya as mentioned in figure 5.

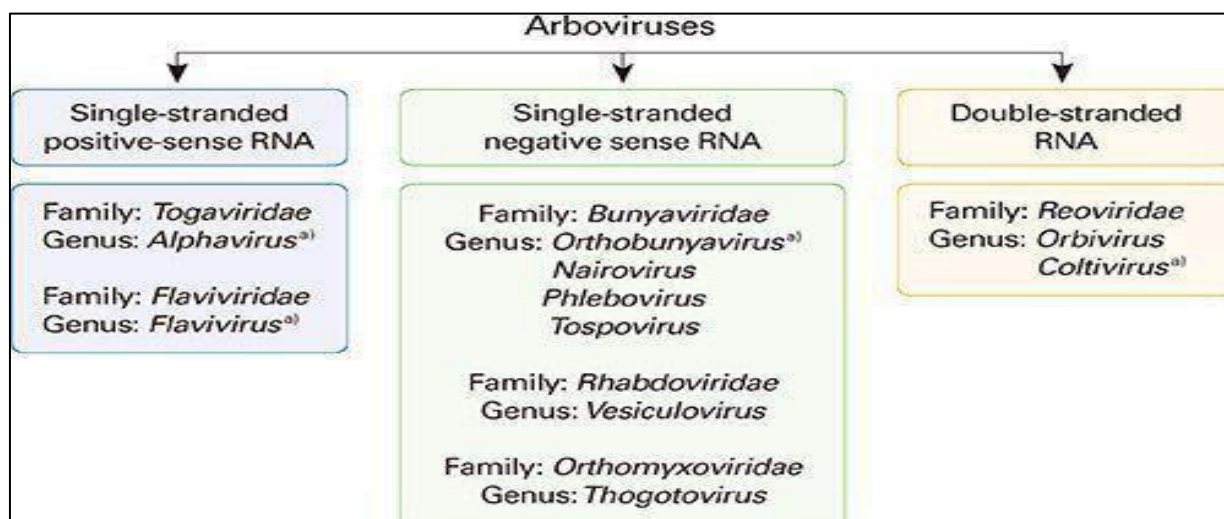


Figure 5: Classification of arbovirus. Within the Rhabdoviridae family, only the Vesiculovirus genus (e.g., Vesicular Stomatitis Virus) includes arboviruses transmitted by arthropod vectors. Other genera in this family, such as Lyssavirus (which includes Rabies virus), are not arboviruses as they are transmitted through bites/saliva of mammals

4 THE MOST COMMON ARBOVIRUSES

4.1 Zika virus (ZIKV)

Aedes mosquitoes that are active during the day, like *A. aegypti* and *A. albopictus*, are responsible for the spread of Zika fever, also referred to as Zika virus disease [19]. ZIKV has been reported in a small equatorial belt that stretches from Asia to Africa. Between 2007 and 2016, the Zika virus spread eastward across the Pacific Ocean to the Americas, causing the epidemic of 2015–2016 [20]. There are currently no approved vaccines for clinical use as of April 2019, but rest and paracetamol may help with symptoms. As of April 2019, none of the several vaccines undergoing clinical trials had been approved for clinical use [21]. Microcephaly, severe brain malformations, and other birth defects may result from Zika transmission from a pregnant woman to her fetus [22]. Rarely, adult Zika infections can cause Guillain-Barré syndrome. According to a long-term study, the cells' mitochondria and vacuoles begin to enlarge six hours after the Zika virus infects them, which sets off paraptosis, a type of programmed cell death that necessitates gene expression. However, viral disruption of neural progenitor cells (NPCs), cell cycle arrest, widespread apoptosis, and other cell death pathways are the main drivers of the multifactorial pathogenesis that results in microcephaly and severe fetal brain malformations. Mosquitoes need blood to lay their eggs. *Aedes africanus*, *Aedes apicoargenteus*, *Aedes furcifer*, *Aedes hensilli*, *Aedes luteocephalus*, and *Aedes vittatus* are some of the arboreal mosquito species from which the virus has also been isolated. Mosquitoes take about ten days to undergo extrinsic incubation [25].

The Zika virus multiplies in the mosquito's midgut epithelial cells and then in its salivary gland cells. After five to ten days, the virus can be found in the saliva of the mosquito. If human skin is injected with mosquito saliva, the virus can infect epidermal keratinocytes, Langerhans cells, and skin fibroblasts. As the virus enters the bloodstream and lymph nodes, it is thought that its pathogenesis continues [26]. Following a Zika outbreak, there have also been cases of birth-related neurological disorders other than microcephaly complication as shown in figure 6 [27].



Figure 6: The effect of the Zika virus on new born

4.2 Dengue virus (DENV)

Dengue fever is caused by DENV, a single positive-stranded RNA virus of the Flaviviridae family that is transmitted by mosquitoes [28]. The sharp increase in infections over the past 20 years indicates that dengue is one of the worst vector-borne human diseases that tropical countries have to contend with. It is estimated that up to 390 million cases of dengue hemorrhagic fever and dengue shock syndrome occur annually, many of which are subclinical or asymptomatic. In tropical and subtropical parts of the world, dengue is primarily found in urban and semi-urban areas [29]. The World Health Organization (WHO) states that clinical warning signs, such as severe abdominal pain, persistent vomiting, mucosal bleeding, fluid accumulation, and liver enlargement, are more predictive of the critical phase and severity of dengue (Severe Dengue/Dengue Shock Syndrome) than allergy symptoms. [30].

4.3 Japanese Encephalitis virus (JEV)

Japanese B Encephalitis (JEV) is a rare yet serious viral infection that carries a mortality rate near 20%, impacting broad areas throughout the Far East and South Asia, where endemic boundaries stretch from Japan and Korea across to Nepal and India [31]. Extended stays in countryside regions carry the greatest exposure risk, so preventing mosquito bites—specifically from *Culex* species that spawn in flooded rice fields—is critical to avoiding infection. The virus circulates when mosquitoes feed on infected wild birds or farm pigs and subsequently transfer JEV to humans and livestock during blood meals. Symptoms generally appear after a 5-to-15-day incubation period; while mild cases might only involve a subtle fever and headache, acute cases hit quickly, causing high fevers, stiff necks, severe headaches, confusion, stupor, coma, muscle tremors, spastic paralysis, and seizures, which are especially prevalent in infants [32].

4.4 Tick-borne Encephalitis (TBE)

The tick-borne encephalitis virus, which is a species of the Flaviviridae family, is the cause of TBE, a type of virus that affects the central nervous system. It is endemic to many European countries, the former Soviet Union, and Asia and is mostly prevalent in temperate regions. The virus mainly infects deer and small rodents, but humans are unintentionally becoming its hosts. Outdoor enthusiasts, hikers, campers, forest workers, and agricultural workers constitute some of the people that a parasitic tick attaches its own to and spreads the virus to. Meningitis, encephalitis, and on rare occasions meningoencephalitis are the sickness's most common symptoms. After a tick bite, the incubation period usually lasts between 7 and 14 days (with a wider range of 4 to 28 days) [33]. Similar to other tick-borne infectious diseases, TBE is more extreme in healthy adults than in children; signs and symptoms consist of an a high temperature, fatigue, bulimia, muscle aches, headache, nausea, and/or vomiting in 23% of patients, as well as meningitis, encephalitis, and drowsiness, disorientation, sensory disturbances, and/or movement abnormalities and paralysis; deaths take place five to seven days after neurological symptoms appear; Since they offer safe and effective protection against TBEV, vaccines are available in disease-endemic areas and are strongly advised by public health authorities (such as WHO and ECDC) for both adults and children over one year of age (using specific pediatric formulations) [34].

4.5 West Nile Virus (WNV)

The West Nile virus (WNV) is a highly prevalent flavivirus in the Near East, Africa, West Asia, and the United States. It can be picked up by humans, animals such as horses, birds, mosquitoes, and some other types of mammals. The most usual method for humans acquire the illness is through bites from mosquitoes [35]. The West Nile virus is known to be carried by at least 36 different mosquito species, with *Culex pipiens* acting as the main vector in the US. After feeding on infected avian hosts, which are the main reservoirs, mosquitoes pick up the virus. They then spread it to humans and other mammals through subsequent blood meals. West Nile fever has become a mild illness with flu-like symptoms that generally lasts only a few days and seems to have no long-term health effects. Typical symptoms consist of a fever, headache, aches in the body, a common skin rash, and swelling lymph glands. West Nile encephalitis, West Nile meningitis, or West Nile infections that usually take three to meningoencephalitis are more severe in fourteen days to manifest [31]. A lot of West Nile virus infections trigger no illness at all. Intensively supportive therapy, which frequently requires inpatient treatment, intravenous fluids, respiratory assistance, management of airways, avoiding the risk of additional infections, and good nursing services, should be considered in more severe cases. When you are outside with infants, wear long sleeves and long pants if at all possible; cover newborn transporters with mosquito netting; and consider staying inside during the hours of dawn, dusk, and early evening when bites from mosquitoes are most prevalent [32].

4.6 Chikungunya virus (CHIKV)

Multiple outbreaks of humans in India as well as Indonesia, Malaysia, Singapore, and Sri Lanka have been attributed to the chikungunya virus since it was initially discovered in Tanzania in 1953. Because *A. albopictus* and *A. aegypti* insects are found everywhere in the world, infected travelers can spread the virus to new regions. The spatial range of the virus is mainly found in Asia and Africa. Travelers with the virus run the risk of carrying it to new areas. The determination is made using laboratory testing, physical indicators (like joint swelling), typical symptoms, and the possibility of mosquito exposure. Severe joint pain is the most prevalent and troublesome chikungunya symptom [33]. Other less frequent symptoms include conjunctivitis, diminished taste, and mouth ulceration. Other symptoms might last longer, but the fever typically subsides in a few days. To avoid contracting the chikungunya virus, wear protective clothing, use insect repellent of any kind, and stay in areas that have already been screened [34].

4.7 Crimean-Congo hemorrhagic fever virus (CCHF)

Hemorrhagic fever viruses (HFVs) are a broad classification of enveloped RNA viruses that fall under families such as Filoviridae, Arenaviridae, Bunyaviridae, and Flaviviridae. They produce severe, possibly terminal illnesses that are distinguished by vascular dysfunction and hemodynamic instability. Ebola, Marburg, and Lassa viruses are typical instances of pathogens that attack host endothelial cell types and normal mononuclear phagocytes, producing an abnormal, hyperactive immune response referred to as a cytokine storm [35]. Widespread microvascular permeability,

disseminated intravascular coagulation (DIC), and multi-organ failure result from this systemic inflammatory cascade's breakdown of the endothelial barrier. HFVs are categorized as high-consequence pathogens requiring biosafety level 4 (BSL-4) containment and continue to be a major focus of global public health surveillance and biodefense strategies due to their high mortality rates, potential for aerosol transmission in laboratory settings, and lack of universally available vaccines or definitive antiviral therapies [36].

4.8 Yellow fever virus (YFV)

A mosquito-borne pathogen responsible for a severe and biphasic hemorrhagic disease that is native to tropical regions of Africa and South America, yellow fever virus (YFV) is a prototypical member of the Flaviviridae family. YFV is primarily transmitted by the bites of infected *Aedes* and *Haemagogus* mosquitoes. It has a high preference for hepatocytes, which results in major liver destruction and distinctive Councilman bodies resulting in the extreme jaundice that gives the illness its name. A subset of patients develop a toxic hemorrhagic phase marked by hematemesis ("black vomit"), epistaxis, and systemic hypovolemic shock caused by widespread endothelial dysfunction and a dysregulated pro-inflammatory cytokine profile, even though many infections remain mild or asymptomatic [37]. Due to suboptimal vaccination coverage, urbanization, and the ongoing danger of sylvatic spillover into densely populated areas, YFV is still a persistent threat to global health even with the availability of a powerful, live-attenuated vaccine (the 17D strain) that offers lifetime immunity [38].

5 DIAGNOSIS

A definitive diagnosis of arbovirus infection is typically determined in a laboratory utilizing a combination of blood tests, especially immunologic, serologic, and/or virologic techniques like ELISA, complement fixation, polymerase chain reaction, neutralization test, and hemagglutination-inhibition test. These blood-drawing tests are mainly utilized in immunology to determine whether a patient's serum sample contains arbovirus antigens or antibodies.

6 HUMAN TO HUMAN TRANSMISSION OF ARBOVIRUSES

Blood transfusions, donor organ transplants, and the use of blood products may pass on arboviruses from person to person if the virus is present in the donor's blood or organs, though this is uncommon [39]. As a result, organs and blood are often checked for viruses prior to donation. Maternal-to-child transmission, or vertical transfer, has been reported in infected pregnant and nursing women on rare occasions. Intravenous drug users and healthcare workers are at risk of infection in areas where the central nervous system (CNS), liver, and capillary Arboviruses can spread through blood transfusions, organ transplantation, and the use of blood products if the virus is present in the donor's blood or organs [40].

7 PREVENTION

Insecticides can be applied in both rural and urban environments, as well as outdoors and inside houses and other constructions. These chemicals are often quite effective at controlling arthropod populations, even though the use of some of them is controversial and some organophosphates and organochlorides (like DDT) have been banned in many countries. In certain regions, infertile male mosquitoes have been introduced to reduce the breeding rate of relevant mosquito species. Swamps and other stagnant water pools (such as old tires, large outdoor potted plants, empty trash cans, etc.) that frequently act as breeding grounds for mosquitoes need to be drained as part of habitat control. The two main ways to prevent arboviral encephalitis are public health initiatives to reduce the number of infected mosquitoes and personal protective measures like sleeping under mosquito nets, wearing protective clothing, applying insect repellents like DEET and permethrin to clothing and exposed skin, and (if possible) avoiding areas known to have high arthropod populations. Temefos is a common mosquito larvicide.

8 VACCINATION

Vaccines are currently being developed for a number of arboviral diseases, including Rift Valley Fever, Chikungunya, West Nil, Dengue fever, and Eastern Equine Encephalitis. Vaccines are currently available for a number of arboviral diseases, including the encephalitis, and Rift Valley Fever (only for veterinary use). Vaccines for Zika virus are not approved to be used clinically till now. As part of public health initiatives, insecticides are frequently sprayed to kill both adult and juvenile mosquitoes (larvae).

9 TREATMENT

Antibiotics cannot treat arboviral encephalitis since it is a viral illness for which there are currently no effective antiviral medications. Brain edema, loss of the brain's natural breathing activity, and other controllable side effects, such as

bacterial pneumonia, are all treated with supportive care. Because aspirin and ibuprofen (NSAIDs) increase the risk of severe bleeding and gastric mucosal bleeding, they should be strictly avoided in patients with dengue fever. The suggested substitute for managing fever and pain is paracetamol.

10 CONCLUSION

The review highlights the serious and expanding threat arboviruses pose to public health worldwide. Although many of these diseases were successfully managed in the middle of the 20th century using insecticides and vector control, there has been a significant resurgence in recent decades. A complex interaction between biological and environmental factors, such as the following, is what is causing this comeback:

- **Global Trends:** Unplanned urbanization, rapid population growth, increased international travel, and globalization all contribute to the quick cross-border spread of viruses like Zika and Yellow Fever.
- **Vector Dynamics:** Viruses can spread from wild animal reservoirs into human populations due to the high adaptability of vectors like *Aedes aegypti* to urban settings and the existence of numerous "bridge vectors."
- **Biological Challenges:** The efficiency of transmission is determined by certain tissue barriers found in mosquitoes, such as salivary gland and midgut barriers, as well as the ongoing co-evolution of viruses and their vectors.
- **Public Health Needs:** Supportive care and vector control strategies like habitat removal and personal protection are still crucial for prevention because the majority of arboviral infections lack specific antiviral treatments and many lack approved vaccines. In order to anticipate and control future arboviral epidemics, it is crucial to comprehend these complex interactions.

STATEMENTS ON COMPLIANCE WITH ETHICAL STANDARDS

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Disclosure of conflict of interest

The authors declare no conflicts of interest regarding this manuscript.

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