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A Genetic Insight and Overview of Zika Virus Infection: An Important Emerging Viral Infection

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Authors' contributions

This work was carried out in collaboration between both authors. Author SAH designed the study, wrote the protocol and wrote the first draft of the manuscript. Author SHS managed the literature searches. Both authors read and approved the final manuscript.

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Review Article

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ABSTRACT

Zika fever is a febrile or sub-febrile illness caused by Zika virus, which mainly spreads through the bite of infected mosquitoes. Zika infection has as of late becoming an emerging infection of medical important. While clinical indications of the infection in adult cases are not serious and ailment isn't related with high death rates, Zika infection can affect foetogenesis and lead to extreme neuro developmental variations from the norm. For better understanding into various parts of Zika infection, this review was performed, with respect to the disease transmission, genetic and geographical distribution of Zika infection. We searched PubMed, Scopus, Embase, HINARI, AJOL, the Cochrane library, Web of Science, and Google Scholar. Zika virus is a member of the family Flaviviridae, which includes dengue viruses, West Nile, and yellow fever viruses. The most common symptoms reported in confirming Zika virus infections are fever, headache, malaise, maculopapular rash, fatigue or myalgia, arthritis and arthralgia. Zika virus was first isolated from the blood of a sentinel rhesus monkey from the Zika Forest in Uganda. The virus has a wide

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geographical distribution, including eastern and western Africa, south and Southeast Asia, and Micronesia, where in 2007, an outbreak of Zika fever was reported on Yap Island. Numerous conventional phylogenetic analyses of Zika virus genomes reveal the presence of two main viral lineages, that is, African and Asian lineages. However, it should be noted that phylogenetic analyses using E and NS5 genes reveal three major lineages of Zika virus with an additional lineage circulating in Africa which is designated African II lineage.

Keywords: Phylogenetic analyses; Zika genome; Aedes spp; microcephaly.

1. INTRODUCTION

Zika virus belongs to the Spondweni sero-complex inside the class Flavivirus, family Flaviviridae [1]. Other mosquito borne flaviviruses of general wellbeing significance include yellow fever, dengue, St. Louis encephalitis, West Nile and Japanese encephalitis infections [2]. In spite of the fact that study have concentrated on a significant number of these infections, other medically significant entities from the mosquito borne flaviviruses, for example, Zika infection, have gotten far less consideration. Zika infection was first isolated from a sentinel rhesus monkey put in the Zika Forest close to Lake Victoria, Uganda in April 1947 and the second disconnection from the mosquito *Aedes africanus* trailed at a similar site in January 1948 [3].

In 2007, the Yap State, a Federated States of Micronesia, reported the first outbreak of Zika virus in areas different from Africa and Asia [4,5]. Subsequent infections of Zika virus in other Pacific islands were not reported until 2013 when this virus reappeared in French Polynesia and then disseminated throughout the Pacific [6,7]. In January-February 2014 one Zika case was confirmed in Eastern Islands (Rapa Nui National Park, Chile) in the Pacific Ocean; [4,8] then in May 2015, 17 cases were confirmed in three states of Brazil [9,10]. Zika virus is probably kept up in a sylvatic cycle including non-human primates and mosquitoes, with cyclic epizootics in monkeys as revealed in Uganda [11]. In the sylvatic transmission cycle, people likely oblige as accidental hosts. Be that as it may, in regions without non-human primates, people most likely serves as essential escalating host and possibly as reservoirs. In spite of the fact that zoonotic Zika infection is kept up basically in a monkey/mosquito transmission cycle, antibodies have been recognized in various other animal species, including water buffalo, elephants, goats, lions, sheep, rodents, wildebeest, and zebras [12]. Luckily, Zika disease to date has been gentle and self-constrained. Clinical indications can be hard to separate from dengue and Chikungunya infections. Furthermore, co-infection with dengue has likewise been as of late proclaimed [13].

2. METHODOLOGY

In this review, we considered these databases: PubMed/MEDLINE, HINARI, Embase, AJOL, Cochrane library, Web of Science, Google Scholar, LILACS, and Scopus which reported studies on the genetic diversities of Zika virus. All searches were limited to, articles published in the last twenty years. All publications were in English, and duplicates, conference abstract, comments and short communications were sorted and removed.

3. RESULTS

A total of 2183 articles were retrieved by literature search, 1098 articles were retained after duplicates were removed; 1048 of them were excluded due to irrelevance based on their title and abstract, and 60 were retained for full-text evaluation.

4. GENOMIC MAKE UP OF ZIKA VIRUS

The Zika virus belongs to Flaviviridae and the genus Flavivirus, and is thus related to the dengue, yellow fever, Japanese encephalitis, and West Nile viruses [14]. Zika viruses like other flaviviruses are enveloped, icosahedral and has a non-portioned, single-stranded, positive-sense RNA genome [15]. It is most firmly identified with the Spondweni virus and is one of the two viruses in the Spondweni virus clade [16]. A positive-sense RNA genome can be legitimately converted into viral proteins. In different flaviviruses, for example, the West Nile virus, the RNA genome encodes seven non-structural proteins and three structural proteins which embody the virus (Fig. 1). The duplicated RNA strand is held inside a nucleocapsid framed from

12-kDa protein; the capsid is contained inside a host-determined layer adjusted with two viral glycoproteins. Replication of the viral genome would first require creation of an anti-sense nucleotide strand.

5. TRANSMISSION OF ZIKA VIRUS

The transmission is mostly by mosquitoes of the Culicidae family and of the *Aedes* genus (sylvatic and urban transmission) including *Aedes aegypti* (urban transmission) as shown in Fig. 2 [17]. Other species have been reported such as *Aedes polynesiensis* and *Aedes albopictus.* The vector *Aedes hensilli* was identified during the Zika epidemic on the island of Yap in 2007, in Micronesia [18]. The virus is usually transmitted by hematophagous arthropods during their blood meal. The virus feeds in the host vector (insect) without affecting it and remains in the insect all life long, and is transmitted to reservoir animals at the next blood meal.

Zika virus is transmitted to individuals basically through the bite of an infected Aedes species mosquito (*A. aegypti and A. albopictus*) [19]. These are similar mosquitoes that spread dengue and chikungunya infections. These mosquitoes regularly lay eggs in or close to standing water in things like cans, bowls, creature dishes, window boxes, and jars. Mosquitoes that spread Zika virus, chikungunya, and dengue, feeds during the day and night and gets infected with the virus when the feed on infected individual during this timeframe. Infected mosquitoes would then be able to spread the infection to other individuals through bite. A pregnant lady can pass Zika infection to her embryo during pregnancy [20].

Mother to Child Transmission: A pregnant lady previously infected with Zika virus can pass the infection to her baby during the pregnancy or around the hour of birth [21]. Conceivable Zika virus diseases have been recognized in

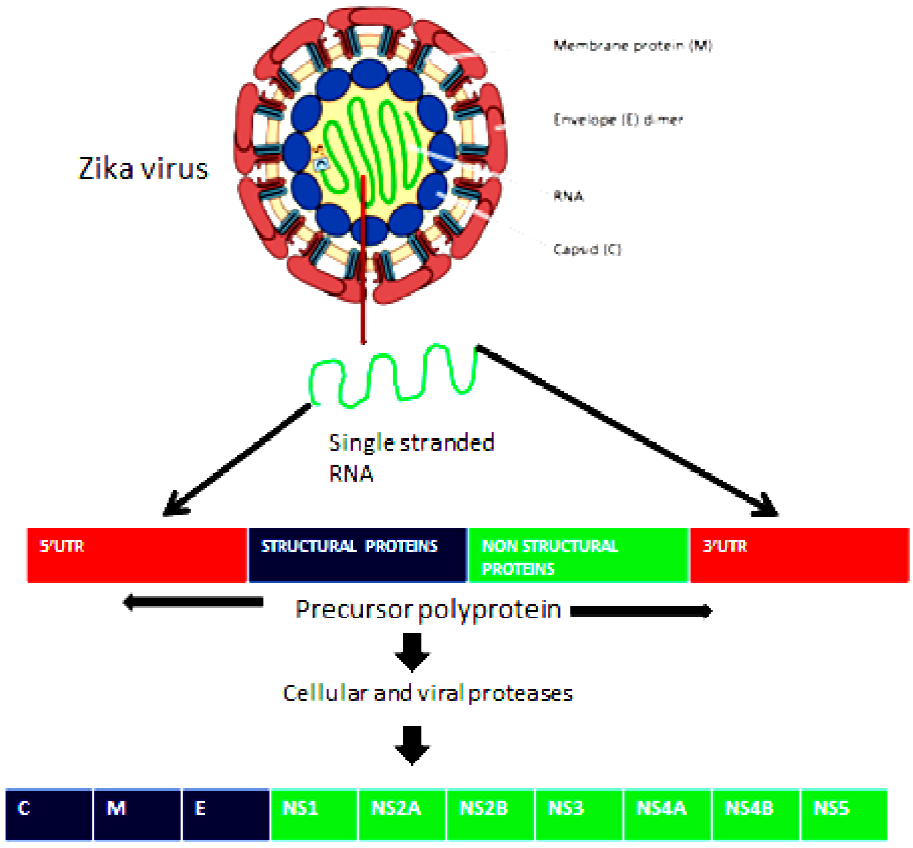

Fig. 1. Illustration of Zika virus proteins

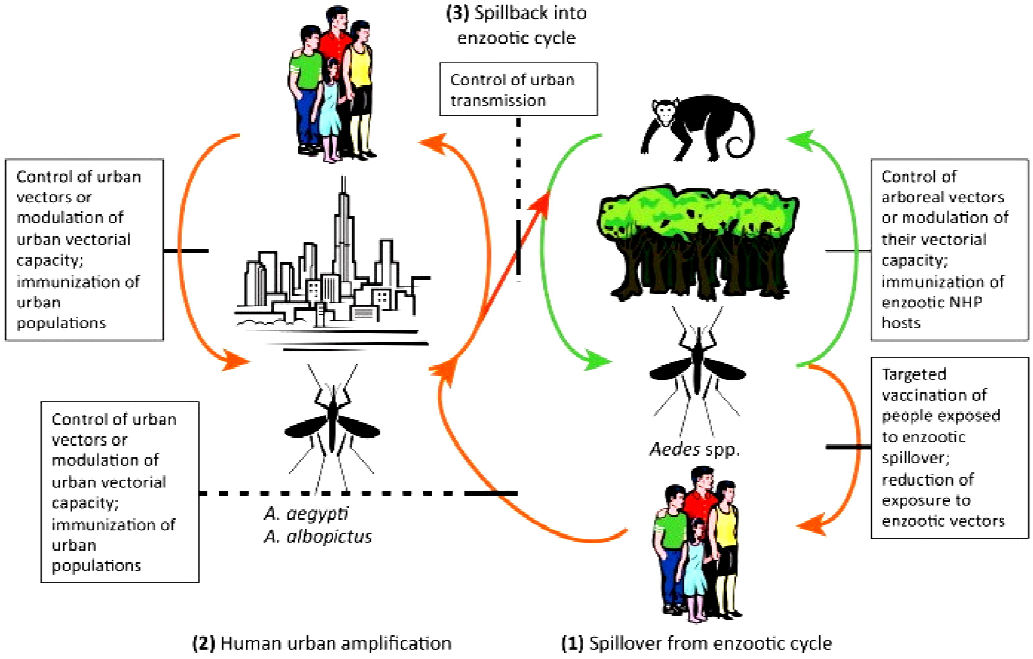

Fig. 2. Sylvatic and Urban transmission of Zika virus

breastfeeding babies; however Zika virus transmission through bosom milk has not been affirmed. Also, we don't yet have a clue about the long haul impacts of Zika virus on youthful new-born children infected after birth. Since current proof proposes that the advantages of breastfeeding exceed the danger of Zika virus spreading through bosom milk, CDC keeps on urging moms to breastfeed, regardless of whether they were infected or lived in or made a trip to a region with danger of Zika and keeps on considering Zika virus and the manners in which it can spread and will refresh suggestions as new data ends up accessible [22].

Sexual Transmission: Zika can be transmitted through sex, regardless of whether the infected individual doesn't have indications at the time [23]. Assort how to secure yourself during sex. It may be transmitted from an individual with Zika before their manifestations start, while they have side effects, and after their side effects termination [24]. Despite the fact that it was not well documented, the infection may likewise be passed by an individual who harbour the infection however never creates side effects. Studies are in progress to discover to what extent Zika remains in the semen and vaginal fluids of individuals who have Zika, and to what extent it tends to be passed to sex accomplices [25].

Transmission through Blood Transfusion: Until this point in time, there has not been any confirmed blood transfusion transmission cases reported. There have been numerous reports of conceivable blood transfusion transmission cases in Brazil [26]. During the French Polynesian flare-up, 2.8% of blood contributors tried positive for Zika and in past episodes, the infection has been found in blood donors [27,28].

Transmission through Laboratory and Healthcare Setting Exposure: There are reports of health care associated Zika virus infection, despite the fact that the course of transmission was not unmistakably settled in all cases. Until this point in time, no instances of Zika virus transmission in health care settings have been recognized. Proposals are accessible for social insurance suppliers to help avoid presentation to Zika virus in health services settings.

6. EPIDEMIOLOGY OF ZIKA VIRUS

Zika virus was described in mosquitoes, primates, and people in 14 nations more than three continents (Africa, Asia, Oceania).The infection had been the object of the few distributed investigations as a result of the recurrence of harmony/asymptomatic presentation and in light of the fact that there was

no reported extreme presentation [29]. The infection was confirmed twice in Nigeria from tests gathered in febrile patients, somewhere in the range of 1964 and 1970 [30]. The authors of a serological investigation of Nigerian symptomatic patients, performed declared that 52% had neutralizing antibodies. Similarly, the authors of another study in Java (Indonesia) reports that out of 219 patients admitted to the Java island medical clinic emergency unit for fever, Zika virus prevalence was 7.1% [31]. Sporadic cases were reported in Thailand, Cambodia, and Indonesia [32]. In a review study, Zika virus epidemic in Gabon, demonstrated the distress of recognizing this pandemic in territories of dengue and chikungunya infection course [33]. A Zika virus first time ever enormous pandemic has been accounted for in French Polynesia. Cases of Zika virus imported from French Polynesia were accounted for in Japan, Norway, Easter Island, and mainland France [34]. Virological investigations, seroprevalence overviews, finding of sporadic cases, and plagues have permitted recognizing the infection in Africa, Asia and Oceania, in the Pacific [35].

7. GENETIC DIVERSITY OF ZIKA VIRUS

Zika viral genome envelope protein E (ENV) sequences that contained the information of collection date and country were retrieved from NCBI data base, the ENV nucleotides sequences were used for subsequent analyses. The phylogenetic analysis of retrieved GenBank sequences revealed two well-separated geographically distinct lineages of Zika virus, namely Asian and African. Western, Middle and Eastern Africa strains were monophyletic. The South-Eastern Asia strains occupied a basal position of the Asian lineage, whereas Oceania and Latin America strains were late-diverging (Fig. 3).

Numerous conventional phylogenetic analyses of Zika virus genomes uncover the nearness of two principle viral genealogies, that is, African and Asian ancestries [9, 37-39]. It ought to be noticed that phylogenetic examinations utilizing E and NS5 qualities uncover three significant genealogies of Zika virus; an extra genealogy has been coursing in Africa assigned African II heredity [40]. Tragically, no genome arrangement has been accessible for strains of African II ancestries to date. In this way, regardless we pursue the conventional two-heredity classification, Asian and African (really African I in the new terminology) genealogies [40]. Be that as it may, Gong, Xu and Han [41] derived an established phylogenetic tree utilizing a technique dependent on shared amino corrosive substitutions however with no specialized detail (Fig. 4B). They recommended that MR766, the strain detached in Uganda in 1947, is basal to the various Zika virus strains and the Asian and African ancestries can't be promptly recognized.

The phylogenetic tree reproduced by setting up the tree topology dependent on the mutual amino corrosive substitutions in their polyproteins and by first assessing the quantity of shared amino corrosive changes for each pair of successions suggested that the tribal Asian strain veered from the normal progenitor of Ada1 and Ada2 in view of one regular amino corrosive substitution at position 3,040 in the polyprotein. One normal substitution may be brought about by united advancement or inversion of the build-up 3,044 in Fig. 4D in the Asian strain Ada1, and Ada2 is probably going to speak to the familial state, as opposed to a common amino corrosive substitution as distinguished in another investigation [42]. The Asian strain imparted a greater number of substitutions to other African strains than with Ada1 and Ada2 Fig. 4D.

The phylogenetic connections between African and Asian Zika virus strains following the rule of shared amino substitutions as appeared in figure 4C had a fundamentally the same topology to the recently distributed regular phylogenetic trees and were accordingly essentially unique in relation to Fig. 4C. In addition, a Bayes Factor (BF) examination by assessing the proportion of the negligible probability of MR766, Ada1, and Ada2 framing a monophyletic bunch as appeared in Fig. 4A to the minimal probability of MR766, Ada1, and Ada2 shaping a paraphyletic bunch in Fig. 4B utilizing the Stepping Stone inspecting strategy. Evidently, the BF is more than 100% recommend that the regular tree is all the more firmly bolstered by the information. In this manner, these discoveries give no proof to the Asian heredity being a continuation of the African genealogy yet bolsters the ordinary idea of an African genealogical strain of Zika virus separating to create unmistakable African and Asian ancestries.

8. CLINICAL PRESENTATION OF ZIKA VIRUS

The indications of Zika infection show up after a brooding time of a couple of days after the bite of

an infected mosquito and normally last three to 12 days [43]. Numerous individuals infected with Zika infection are asymptomatic. Trademark clinical discoveries are intense beginning of fever with maculopapular rash, arthralgia, or conjunctivitis. Other generally detailed manifestations incorporate myalgia and migraine. Clinical ailment is normally mellow with side effects going on for a few days to seven days. Serious ailment requiring hospitalization is phenomenal and case casualty is low. Be that as it may, there have been instances of Guillain-Barre disorder announced in patients following suspected Zika infection disease [44,45]. As of late, considers have uncovered that Zika infection disease during pregnancy is a reason for microcephaly and other extreme foetal cerebrum absconds [46-48]. Because of worries of microcephaly brought about by maternal Zika infection disease, embryos and babies of ladies contaminated with Zika infection during pregnancy ought to be assessed for conceivable intrinsic infection and neurologic variations from the norm.

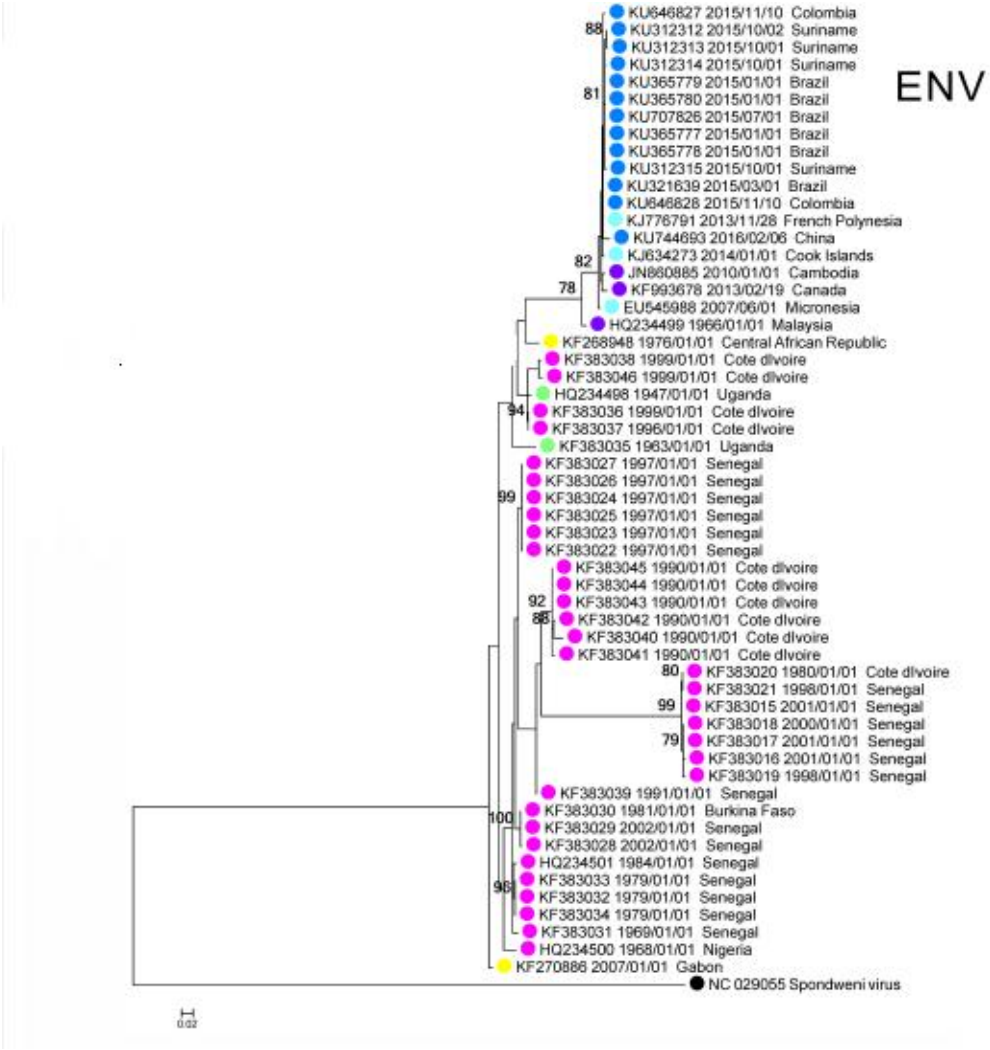

Fig. 3. Maximum likelihood based phylogenetic tree with envelope protein E (ENV) sequences of the Zika virus

These trees are summarized after 1 000 replicates. Bootstrap values smaller than 70 are not shown. Since the evolutionary relationships of flaviviruses have been characterized [36]. We used one of the closest evolutionary relationships species in flaviviruses, namely Spondweni virus (SPOV), to root the trees. The location for imported cases was assigned to the source country

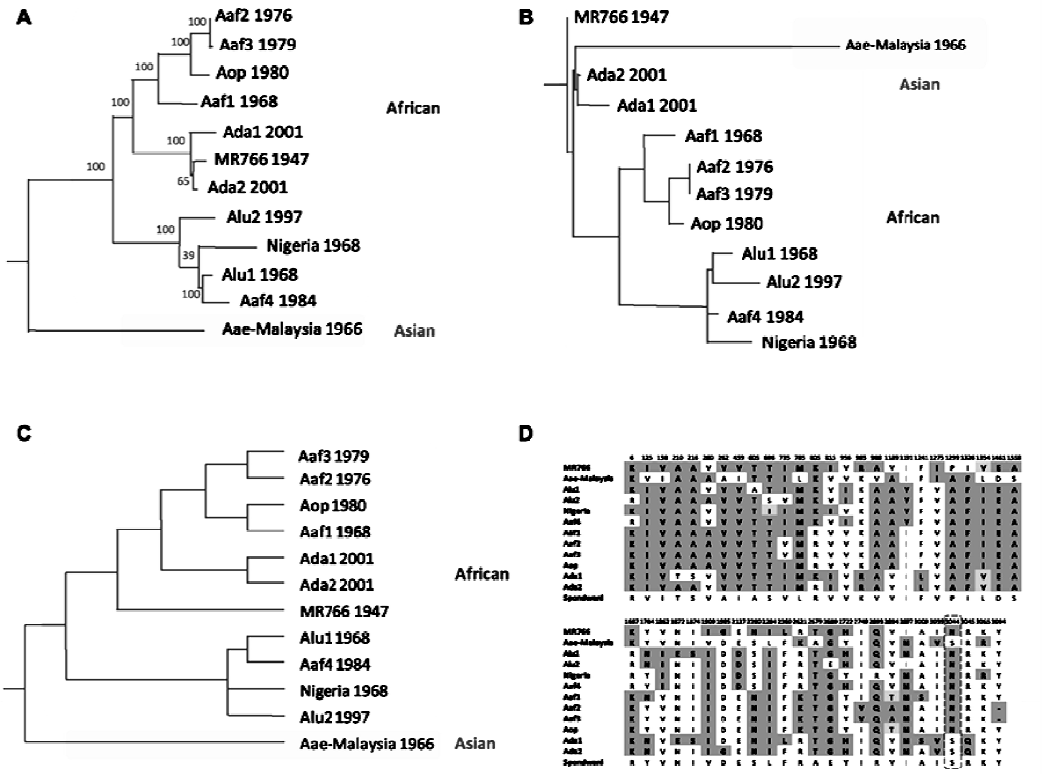

Fig. 4. Different classifications of Zika virus. The phylogenetic tree (A was inferred using maximum likelihood method. The conventional Asian and African lineages are indicated by yellow and blue colours. The numbers on the branches represent the bootstrap values

The tree proposed by Yokoyama and Stammer is shown in the right panel (B. The topology of the tree (C was inferred based on the shared amino acid replacements (D. For the shared amino acid replacements, the mutations were labelled in shaded boxes. The numbers indicate the residue positions in the polyprotein of Zika virus

9. ZIKA VIRUS AND MICROCEPHALY

Microcephaly is characterized as being brought into the world with an unusually little head, built up by estimating the periphery of an infant's head and contrasting it and those of correspondingly matured children of a similar sex a definition that is moderately free. Zika virus has been getting features in view of it's connects to a disturbing birth imperfection called microcephaly. Since the recognizable proof of the Zika virus in Brazil in mid-2015, the infection has spread quickly all through the Americas (www.cdc.gov/zika/geo/dynamic countries.html. opens in new tab. An expansion in the quantity of new-born children with microcephaly in Brazil was first noted in September 2015, after the acknowledgment of Zika virus transmission in the nation previously [49]; this was trailed by the acknowledgment of a comparative increment in French Polynesia after a flare-up there in 2013 and 2014 [50]. In spite of collecting proof that supports the connection between Zika virus disease and microcephaly; most specialists have taken consideration not to express that Zika virus infection is causally identified with these unfavourable results [51]. This careful methodology toward attributing Zika viruses a reason for birth deformities isn't astounding, given that the last time an irresistible pathogen (rubella infection caused a plague of inherent imperfections was over 50 years prior, no Flavivirus has ever been demonstrated conclusively to cause birth deserts in people [52], and no reports of unfriendly pregnancy or birth results were noted during past flare-ups of Zika virus ailment in the Pacific Islands [53-57].

As is commonly the situation in the study of disease transmission and drug, no "conclusive

evidence" or a solitary authoritative bit of proof that affirms Zika viruses a reason for intrinsic imperfections ought to have been envisioned. Rather, the assurance of a causal relationship would be relied upon to rise up out of different lines of proof, every one of which recommends, yet doesn't without anyone else demonstrate, that pre-birth Zika virus infection can cause unfriendly results. Two methodologies have been utilized to distinguish potential teratogens exposures to a mother during pregnancy that harmfully affect her incipient organism or hatchling [58]: first, the recognizable proof of a mix of an uncommon introduction and an uncommon imperfection here and there alluded to as the clever clinician approach [59], and second, the utilization of epidemiologic information to affirm an affiliation. Numerous teratogens were first distinguished by methods for the uncommon introduction uncommon imperfection approach, including rubella infection, which was recognized after an ophthalmologist noticed a trademark type of waterfalls in new-born children whose moms had rubella during pregnancy [60], and overwhelming liquor use, which was distinguished as a teratogen after the acknowledgment of a trademark example of mutations that ended up known as the foetal liquor disorder [61]. Conversely, a few teratogens like valproic corrosive have been recognized based on epidemiologic examinations demonstrated a chances proportion of 20 for the relationship of spina bifida with utilization of this medication during the primary trimester of pregnancy [62].

10. DIAGNOSIS OF ZIKA VIRUS

The diagnosis of Zika infection depends for the most part on the discovery of viral RNA in blood tests: RT-PCR and viral disconnection in blood tests gathered less than five days after the beginning of side effects is the reference strategy [63]. The viremia time frame in people could be short, from the third to the fifth day after beginning of indications. Viremia could last longer than viremia and the RT-PCR discovery of viral RNA in pee could be an elective strategy if the hereditary material is never again present in the serum. Serological tests Elisa or immunofluorescence is likewise generally utilized. The Centres for Disease Prevention and Control CDC in Atlanta had built up an ELISA system to identify explicit enemy of Zika IgM [64]. The recurrence of cross-responses with different flaviviruses dengue, yellow fever may make the finding troublesome. Besides, in the early period of infection, the pace of IgM and IgG might be exceptionally low, making it hard to affirm the finding. The recognition of antibodies ought to be affirmed by an integral sero-balance test permitting deciding the explicitness of the distinguished antibodies like Plaque Reduction Neutralization Test and demonstrating a 4-overlap increment of the immunizer titre at first found [65]. No commercial pack is as of now accessible for the location of antibodies explicitly identified with Zika infection.

11. TREATMENT OF ZIKA VIRUS INFECTION

An estimated 80%of Zika infection is asymptomatic, and most of the remainder are self-limited [66]. There's no vaccine or specific treatment. Instead, the focus is on relieving symptoms and includes rest, rehydration and acetaminophen for fever and pain. Aspirin and non-steroidal anti-inflammatory drugs NSAIDs such as ibuprofen should be avoided until dengue can be ruled out to reduce the risk of bleeding.

12. CONTROL AND PREVENTION OF ZIKA VIRUS INFECTION

Prevention of Zika and other arbovirus is achieved by vector control and insect bite precautions [67]. *Aedes* spp. Insect bite precautions during early morning and late afternoon peak biting times and vector control should be tailored to known epidemiology [68]. To date, there is no vaccine for the prevention of Zika [69]. Thus, it is extremely important to strengthen surveillance, control suspected cases of Zika for early integrated detection of cases. More joint efforts from affected countries are needed in addition to regional efforts of the World Health Organization to provide and establish guidelines and policies.

Mosquito-borne diseases require reducing source populations, including physical like removing water-containing sources and biological like fish that feed on larval controls. Insecticide spraying of mosquito habitats or adult populations can be effective. Although it remains controversial due to ecological concerns; releasing genetically modified sterile male mosquitoes could reduce disease-transmitting mosquito larvae. All these strategies require effective mosquito surveillance to ensure focused interventions.

13. CONCLUSION

The Zika virus has caused two significant scourges in apparent already credulous domains, in less than 10 years. This rising arboviruses transmitted by mosquitoes of the Aedes variety has a high potential for spreading in nations where the vector is available. This circumstance requires the most noteworthy watchfulness, particularly since this ailment isn't outstanding and that a few inquiries stay unanswered, concerning the stores and methods of transmission, the clinical introduction, and potential difficulties. The vector A.albopictus is available in the south of Europe. Various customary phylogenetic examinations of Zika virus genomes uncover the nearness of two primary viral heredities, that is, African and Asian ancestries. This may impact malady counteractive action and the executives systems just as raising moral and sociological issues. Therefore, expanded attention to the therapeutic network together with enhancements in vector control and ailment observation frameworks are of most extreme significance for controlling any potential Zika virus related dangers in various nations. Be that as it may, a few vulnerabilities stay on the result of co-diseases with different arboviruses, for example, the Zika viral infection.

CONSENT

It is not applicable.

ETHICAL APPROVAL

It is not applicable.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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