RJNMSR
Review Journal of Neurological & Medical Sciences Review

Volume 4, Issue 3, 2026

IMMUNE IMPRINTING BY PRIOR INFECTIONS: MECHANISMS LINKING ZIKA EXPOSURE TO SEVERE DENGUE DISEASE

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DOI: https://doi.org/10.5281/zenodo.20537239

Received	Accepted	Published
30 January 2026	14 March 2026	31 March 2026

ABSTRACT

Zika virus and dengue virus are an important and a significant global health burden transmitted by mosquitoes. These viruses are often co-circulating in tropical and subtropical areas which makes it a high chance of sequential infection among populations that have been exposed to such viruses. There is growing evidence to the effect that being infected by one flavivirus may be instrumental in influencing immune responses to that of other related viruses. This is referred to as immune imprinting or original antigenic sin and can influence antibody and cellular responses in such a manner that it changes the severity of the disease. Specifically, the cross-reactive antibodies produced during the initial infection with the flaviviruses might not be able to neutralize second infection and instead promote viral entry into immune cells with Fc receptor-bearing via antibody-dependent enhancement (ADE). This process has also been closely linked to extreme cases of dengue including dengue hemorrhagic fever and dengue shock syndrome. The experimental and epidemiological literature also indicates that the immunological interaction between these two viruses is complicated because under some circumstances anti-body formation during these infections helps infectious cells multiply. Besides ADE, novel processes like viral entry by B-cell receptor and cross-reactive immune memory could have a part in the amplification of viral replication and immunopathology in sequential infections. Such immune responses pose a profound problem when attempting vaccines development because vaccines should induce body defenses but not cross-reactive immunoglobulins that can promote an infection. The following review provides an overview of existing information on the pathogenic processes of immune imprinting between Zika and dengue virus infection, with a focus on antibody-dependent enhancement, cross-reactive immunity, and some new avenues of immunology that could mediate disease severity. The elucidation of these mechanisms is important in making better it is essential in designing vaccines against flaviviruses, anticipating the risk of the disease in the endemic areas, and providing safer immunization protocols.

Keywords: *Dengue, Zika, Antibody-dependent enhancement, Immune imprinting, Cross-reactive immunity, Flavivirus infection, Sequential infection, Dengue*

INTRODUCTION

Mosquito transmitted flaviviruses, particularly dengue virus (DENV) and Zika virus (ZIKV), cause a significant and increasing global health threat. Dengue infects hundreds of millions of people annually, while Zika has become a significant

public health concern because of its link to birth defects and neurological problems. These viruses circulate together in the same environments, increasing the chances that people will be exposed to multiple flaviviruses over their lifetimes, particularly in tropical and subtropical regions (Tomalin et al., 2025; Parker et al., 2025. One of the important characteristics of flavivirus infections is the antigenic similarity, particularly in the structural proteins of flavivirus infections, such as the envelope protein of the virus. This antigenic similarity allows the immune system to react to several flavivirus infections, as the immune response elicited in response to a flavivirus infection tends to cross-react with other flavivirus infections. Although this cross-reaction provides a degree of immunity, this immune response might in subsequent infections, which might affect the intensity of the disease process. This occurs when the memory for the immune system from the first infection affects the memory from the second infection with the same type of virus, which is known as immune imprinting (Salem et al., 2025).

The antibodies that the individual has from the first flavivirus infection may not neutralize the next infection effectively but rather facilitate the entry of the virus into the immune system through the phenomenon known as antibody-dependent enhancement (ADE), which is associated to severe cases of dengue (Chelluboina et al., 2025; Kawiecki & Christofferson, 2016). Such complex immune interactions make vaccine development more complicated because a vaccine has to be effective enough to induce immunity without any cross-interactions, which might result in adverse effects. Therefore, it is vital to comprehend how past flavivirus infections affect immune interactions and disease severity in order to improve disease control measures. This review explores the pathogenic mechanisms by which prior infections shape the immune system and contribute to severe disease upon subsequent exposure, focusing particularly on the interactions between Zika and Dengue viruses.

Overview of Zika Virus Infection

The Zika virus (ZIKV) is a virus that is spread by arthropods and is a member of genus Flavivirus in the family Flaviviridae. The dengue virus, yellow fever virus, West Nile virus and other medically significant viruses are closely related to the virus. ZIKV was identified in people in Asia and Africa after being initially isolated in 1947 from a rhesus monkey in the Zika forest of Uganda (Tajik et al., 2024; Li et al., 2017). The virus primarily circulated in a sylvatic cycle involving non-human primates and mosquito vectors with very occasional human infections, for a number of decades. But in the 21st century, most famously in the Americans in 2015-2016 and in Micronesia in 2007, significant outbreaks started to appear (Camacho-Moll et al., 2025; Fassola et al., 2025). Humans contract ZIKV mostly by the bite of an infected *Aedes* mosquitoes, specifically *Aedes albopictus* and *Aedes aegypti.* Because of their wide spread distribution in tropical and subtropical areas, these vectors help virus spread around the world (de Jong & Grobusch, 2025; Fassola et al., 2025). There are a number of known non-vector transmission pathways in addition to vector borne transmission. These illustrate the complexity of ZIKV epidemiology and include sexual transmission, organ donation, blood transfusion and vertical transmission from mother to fetus (Tajik et al., 2024; Ferdous et al., 2026).

In terms of structure, ZIKV is a 50nm diameter encapsulated virus with a 10.8-11 kilobase single stranded positive sense RNA genome. The single polyprotein that encodes by viral genome is broken down into seven non-structural proteins (NS1-NS5) that are involved in viral replication and assembly, as well as three structural proteins (capsid (C), premembrane/membrane (prM/M) and envelope (E) by host and viral proteases (Wong et al., 2024; Fassola et al., 2025). While non-structural proteins remodel host cell membranes, especially the endoplasmic reticulum, to form replication complexes that promote viral RNA production, the envelope protein is essential for host cell attachment and membrane fusion during viral entry (Wong et al., 2024). Clinically, ZIKV infections frequently show no symptoms or very minor ones. Common symptoms include

fever, headache, conjunctivitis, maculopapular rash and joint pain. These symptoms rarely lead to hospitalization and often go away in a few days (Fassola et al., 2025; Ferdous et al., 2026). Because ZIKV infection is linked to serious neurological results, it has gained international interest despite its relatively mild presentation. Congenital Zika syndrome, which can happen when infection is contracted during pregnancy, is one of the most serious consequences. Microcephaly, eye sight impairment, brain abnormalities and developmental issues in neonates are the hallmarks of this illness (Fassola et al., 2025; Tajik et al., 2024). Guillain-Barre syndrome, an inflammatory neurological condition that can result in paralysis and muscle weakness, has also been connected to ZIKV infection in adults (Tajik et al., 2024).

Due to increased urbanization, globalization and mosquito vector proliferation, ZIKV has epidemiologically spread to many parts of the world. There have been reports of outbreaks in different regions of Asia, Latin America and Pacific Islands, and there is currently proof of transmission in many tropical and subtropical regions (de Jong & Grobusch, 2025; Fassola et al., 2025). The distribution of mosquito vectors and consequently the risk of ZIKV transmission are influenced by environmental factors as temperature, population mobility and rainfall (Camacho-Moll et al., 2025). Currently, there is neither a licensed vaccine nor a commonly accepted antiviral treatment for ZIKV infection. In order to minimize exposure to infected vectors, management mainly concentrates on symptomatic treatment and preventive techniques, such as mosquito control measures and personal protective behaviors (de Jong & Grobusch, 2025; Tajik et al., 2024). In order to better prepare for and control outbreaks, research is being done to create vaccines and therapeutic interventions as well as to enhance diagnostic techniques and surveillance systems.

One of the most notable vector born hazard to global public health is dengue virus. In 2024, according to World Health Organization, dengue burden reached at groundbreaking level with 7.6 million cases reported out of which 3.4 million were confirmed positive with infection and 3000 were died in endemic region. In more than 100 countries, it is endemic. Each year dengue effects 390 million people chiefly in tropical and subtropical regions, out of which 96 million showed clinical symptoms. Rebirth and spread of dengue in geographical region is due to following factors which includes; inadequate vector control, high rate of urbanization and change in climate. (Ghosh et al., 2025) There are four numbers of serotypes of dengue virus. Which includes; DENV-1, DENV-2, DENV-3, DENV-4. These all are genetically different and show complex immunological cross reactivity. (Yamanaka et al., 2021)

With host antibodies, they make different types of interactions and show their specific antigenic variations. In dengue patients, infecting serotype should be identified. Because they show different clinical results, leading to infection. In case of co infection, identification of serotype is very important for early and good management of disease. (Sabrina et al., 2025)

Clinical symptoms and tendency of dengue is important to understand. No or little symptoms are showed during primary dengue infection. In case of secondary dengue infection, dysfunction of hemostatic and failure of many organs occur. Different stages of dengue infections can be identified with the help of symptoms (febrile, critical, convalescent, recovery). Symptoms during febrile phase includes; high grade fever and dehydration. On the other hand, rash, itching and hunger are symptoms of recovery phase. (Pulock et al., 2025)

Immune Memory and Immune Imprinting

Those immunogenes that develop in case of the primary viral infection have a significant influence upon immune responses in case of exposure to secondary viruses. The first occurrence of exposure to a pathogen stimulates naive B and T lymphocytes, which differentiate into effector as well as long-term memory cells. These memory cells provide quick protection against reinfection of the same pathogen. However, when the immune system faces an antigenically different, although related, virus, then the system might be

occupied by the already existing immune memory and this may hamper the secretion of the neutralizing antibodies against new viral epitopes. This is commonly referred to as immune imprinting or original antigenic sin (Salem et al., 2025).

Dengue virus and Zika virus are flaviviruses and have similar structures, particularly when it comes to envelope proteins, and whose major antigenic determinants are shared. As a result of such similarity, the antibodies formed in response to an infection with one flavivirus may cross-link with the related viruses. Previously developed memory B cells may be chosen in the case of the second infection with a flavivirus as it results in antibody response that is biased on the antigen that was previously encountered and not the new pathogen (Salem et al., 2025).

The evidence of the inclusion of the immune imprinting was propounded to be supported by the dengue infection studies. In case of infection by one serotype, serotype protects the infected against this serotype but not the other serotypes only temporarily. The immune responses of secondary infection with another serotype are generally largely controlled by antibodies of the initial infection. In the majority of the cases, the highest antibody titres of infection are associated with the serotype, associated with the prior exposure than the infectious virus, showing the impacts of the antigenic seniority in the determination of the immune responses (Cracknell Daniels et al., 2024)).

Exposure to different flaviviruses may have therefore been confusing the immune responses in a sequence. Though, cross-reactive immune memory may provide partial protection against the related viruses, it may also acquire sub-optimal immune responses resulting in severity of the disease. The similarity of the antigens of viruses, the affinity of the antibodies, and the quantity of antibodies in circulation will determine the final result (Salem et al., 2025).

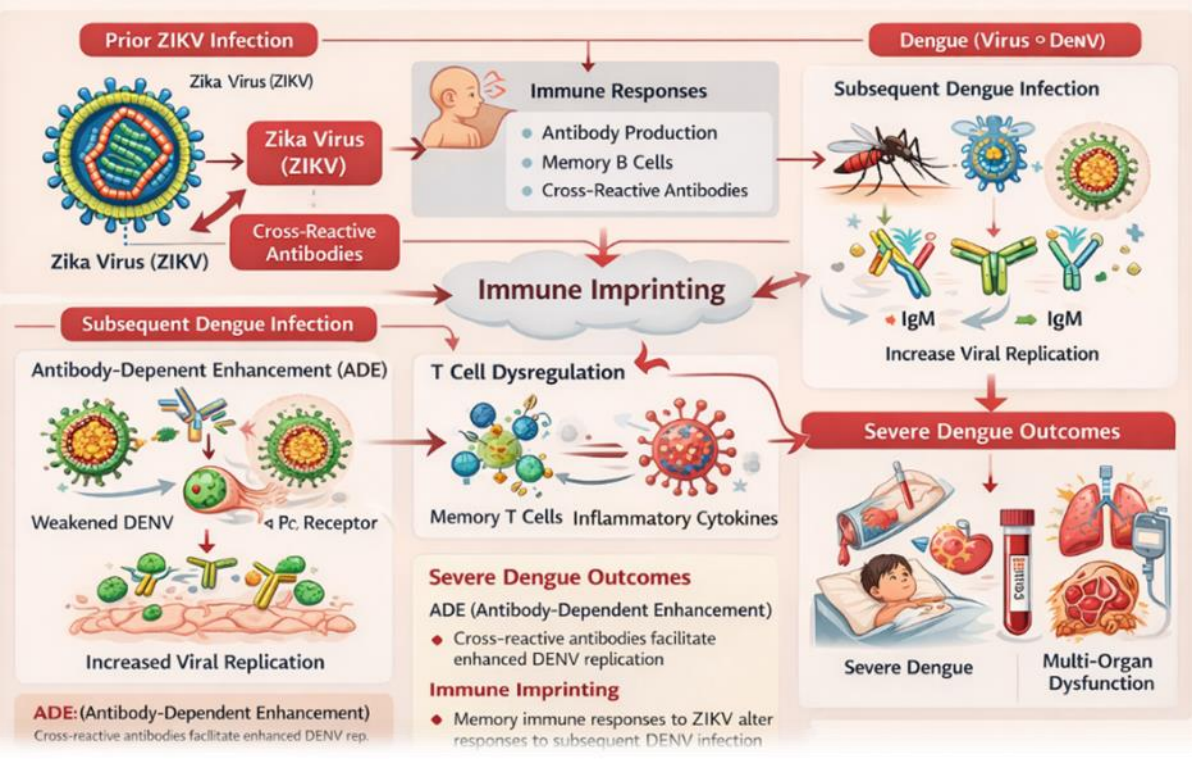

Figure 1: Immune imprinting between Zika virus and Dengue virus and its role in severe dengue disease

Antibody-Dependent Enhancement

Antibody-Dependent Enhancement One of the most studied possible mechanisms of association of past exposure to flaviviruses with severe illness in case of secondary infection is the association with antibody-dependent enhancement (ADE). ADE occurs when the antibodies which were developed due to an earlier infection are bound to a virus, but they are unable to neutralize the virus. The reason as to why these antibodies do not prevent infection is because they do not form immune complexes with viral particles, which are then internalized by immune cells using Fc-gamma receptors. This aids in the penetration and replication of the virus by the monocytes, macrophages, and dendritic cells (Chelluboina et al., 2025). Dengue virus infection is strongly connected with ADE which is closely related to severe clinical outcomes. Cross-reactive antibodies may likewise be induced to bind heterologous serotype during secondary infection following the original infection of differing serotype and cannot be used to neutralize. These sub-neutralizing antibodies enhance cellular processes that stimulates virus uptake to Fc receptor-expressing cells that result in increased viral growth and enhanced viremia (Salem et al., 2025).

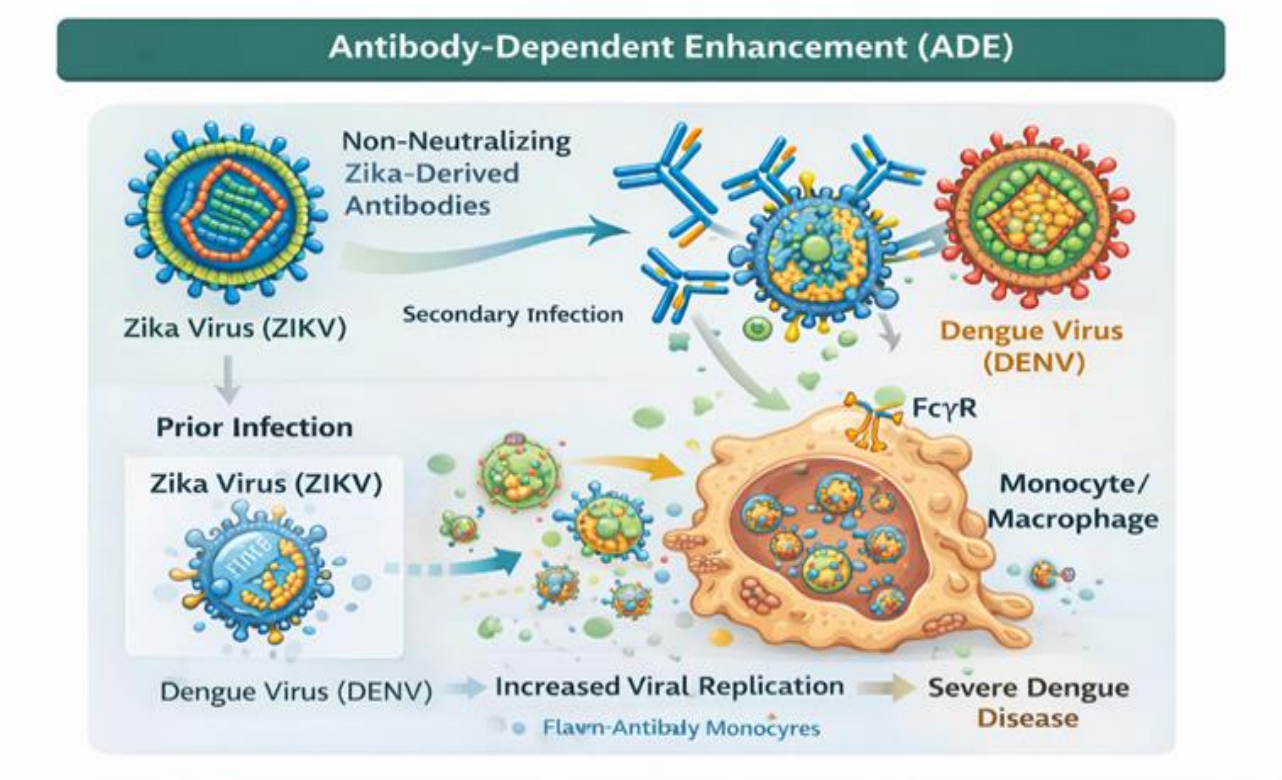

Figure 2: Antibody-Dependent Enhancement in Zika and Dengue virus infections.

This mechanism has experimental evidence to support it. Experiments in laboratory with the use of serum samples containing dengue specific IgG antibody revealed that the antibodies enhanced infection of dengue virus in cell lines expressing Fcg receptors in sub-neutralizing concentrations. Individuals who had been previously infected or had antibodies showed a high rate of infection to the anti-dengue antibody negative samples (Chelluboina et al., 2025). The rising of infection of the dengue virus can also be accomplished as a result of the production of the cross-reactive antibodies that are generated during the infection of the Zika virus. The polyclonal Zika-immune serum experimental evidence has shown that the antibodies with the potential to neutralize Zika virus may enhance the replication of dengue virus serotype 2 in vitro, which suggests that cross-

reactive flavivirus antibodies might play a role in disease enhancement (Kawicki and Christofferson, 2016).

Recent research indicates that there are other mechanisms which may lead to an increased infection of dengue virus after exposure to it in the previous instance. Infection is one of the mechanisms which is facilitated by B-cell receptor (BCRs). It has been shown in experimental research that the dengue-specific B-cell receptors are capable of serving as viral entry receptors thus enabling dengue virus to infect antigen-specific B cells. Dengue-specific BCR-expressing cells were identified to be very vulnerable to dengue viruses as well as the percentage of infectable B cells rose after initial infection. These results indicate that the viral entry via B-cell can be another target of the immune memory in the spread of viruses during secondary infections (Gebo et al., 2024) The discovery of BCR-dependent viral entry also explains the intricacy of immune-mediated dengue pathological processes. A combination of these mechanisms, together with ADE, depict the role of immune memory that is formed in the course of past infection in supposedly boosting a viral infection during subsequent exposures.

Experimental evidence

In United State, ELISA was developed by FAMERP group used to examine history of Dengue and Zika in suspected dengue patients. A patient has 2.34 times more risk of suffering from severe dengue with zika infection history as compared to no zika history. Aged person (59) has more risk of suffering from severe dengue. (Estofolete et al., 2023) During 2015-2016, children were at risk of getting DHF/DSS after ZIKV infection in western Hemisphere. Animal studies have revealed that mother mice who has developed ZIKV immunity gave birth to mice with increased mortality when infected with DENV. Macaques resistant to ZIKV, when treated with DENV2 showed rise in viral burden and promoting inflammatory reactions. Epidemiology studies were not clear about increase in DENV complexity with ZIKV immunity. Later in 2019-2020, examination of Nicaraguan children cohort had showed that DENV2 severity surged with ZIKV immunity. Further, it was hypothesized that ADE in other DENV serotypes could be promoted by preceding ZIKV (Bonheur et al., 2021).

Cross-Reactive Immunity among Flavivirus

The coexistence of numerous flaviviruses in endemic areas enhances the likelihood of consecutive infections. Because flaviviruses have conserved antigenic structures, antibodies produced after infection with one virus typically cross-react with other viruses. Such cross-reactivity can aggravate immune responses and increase illness severity. Cross-Reactive Immunity to Flavivirus. Population-based serological studies have found significant overlap in antibody responses to dengue and Zika viruses. A cross-sectional serosurvey done in Ethiopia found significant levels of dengue virus IgG antibodies as well as Zika virus antibodies in the same group. The significant association between antibody responses to these viruses shows the presence of cross-reactive immunity caused by common antigenic determinants (Parker et al., 2025). Cross-reactive immune responses have also been reported in paediatric arboviral infections. Comparative investigations of immune responses to dengue, Zika, and chikungunya viruses indicated that these infections produce similar immunological markers, such as B-cell proliferation and activation of adaptive immune responses. In contrast to dengue infection, Zika virus infection was linked with delayed activation of B cells and lower inflammatory responses, show unique immunological dynamics despite antigenic similarities (Tomalin et al., 2025). These data show that cross-reactive immunity among flaviviruses can affect both immune protection and disease severity during consecutive infections.

Challenges in Vaccine Development

Understanding immunological imprinting and ADE is crucial to developing safe and effective dengue vaccines. One of the most difficult challenges is that vaccines must establish protective immunity against all four dengue serotypes while preventing the production of antibodies that could exacerbate infection. The experience with Dengvaxia, the first licensed

dengue vaccine, illustrated the difficulty of the undertaking. Trials in clinics showed that production of vaccine in which caused neutralising antibodies, people who were not previously attacked by dengue virus were at high risk of diseases following vaccination (Shukla et al., 2020). The data shows that immune protection and severity of disease during consective infections can be affected by cross-reactive immunity among flaviviruses. According to research, vaccinations of dengue at first produce antibodies that target the viral envelope protein's fusion loop epitope and the prM protein.

These antibodies have weak neutralising activity but are generally cross-reactive increases the risk of ADE (Shukla et al., 2020). Antibodies prescribed against the envelope domain III (EDIII) region have greater neutralizing activity, they are more serotype-specific and are less likely to promote. New vaccine techniques have serotype-specific immunity and seek to elicit balanced. Protective responses against all four dengue serotypes at the same time can be stimulated by Tetravalent vaccines formulations it lowers the possibility of predisposal of individuals to severe disease during subsequent infections because of partial immunity (Balingit et al., 2025). Carefully developed live-attenuated vaccines can be both successful and safe and it was done in vitro studies of the experimental KD-382 tetravalent vaccination it showed long lasting cross-genotype neutralizing antibody responses. It is necessary to test neutralizing and boosting antibody responses as it is a very important aspect of vaccine development when doing vaccine trials. Evaluation of traditional vaccine generally relied on evaluating neutralising antibody titers but this alone is not enough to predict safety and protection (Balingit et al., 2025). During vaccine development long-term immunological monitoring and integrating ADE prior to large scale deployment can help in identification of possible dangers. The important thing is vaccine plans must consider the whole environment of flavivirus. Vaccination can elicit cross-binding antibodies without boosting infection enhancement (Vista et al., 2025). These findings showed that pathogenic enhancement always is not the result of cross-reactive immune responses and cross-reactivity can be tackle by vaccine design in a protective and regulated manner.

Conclusion

Zika virus and dengue virus interaction is very complex immunological mediated disease. Clinical evidences, experimental outcomes and epidemiological studies has revealed that subsequent infection can occur in organism with previous exposure to flavivirus. In some cases, cross reactive immunity may be a mild protection. On the other hand, immune imprinting, entry of virus through B-cell receptor (BCR), disease of antibody-dependent enhancement can promote the pathogenesis. These processes showed that immune memorization during previous infection does not help but prerequisite the environment in which virus proliferate and causes more severe inflammation reactions. These immunological conditions have terrible effect on public health, especially in regions where many flavivirus co-exist. Vaccine development is complicated because of strong immune response. There is need to make ways with balance immunity which is neither too weak nor too strong and does not instigate increasing titers or sub-neutralizing antibodies, which causes infection. In future detailed immunological studies, suitable animal models and longitudinal cohort studies should be performed to check whether cross reactive immunity is pathogenic or protective. Future advance studies are very important for the creation of effective vaccines, better treatment strategies and globally control of flavivirus.

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