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Perspective

Transmission and Aetiology of Dengue Virus

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DESCRIPTION

One third of the world's population is at danger of contracting dengue fever, which is the most common human arbovirus disease. Members of the family Flaviviridae and the genus *Flavivirus*, dengue viruses divide into four closely related serotypes that share 60%–75% amino acid similarity. The search for effective antiviral drugs is made more difficult by this genetic variation. Therefore, there aren't any licenced therapeutics for dengue at the moment. The development of a group of 19 dengue viruses, which represent the genotypic variation within the four serotypes, was done in order to provide an effective tool for the drug discovery process for the dengue virus.

With around one third of the world's population at risk of infection, the dengue virus (DENV) is a serious hazard to human health. The etiological agent of dengue fever, dengue shock syndrome, and the more severe dengue haemorrhagic fever (DHF), is DENV (DSS). It is a member of the *Flavivirus* genus (*Flaviviridae* family), which also includes other clinically significant human viruses like the West Nile virus, the recently discovered Zika virus, and the yellow fever virus. DENV is a virus carried by arthropods that is spread via the bite of infected *Aedes* mosquitoes (Stegomyia).

DENV's epidemiological transmission is only found in urban and peri-urban areas, with *Aedes aegypti* and *Ae albopictus* mosquitoes serving as the main carriers of the disease. The 10.7 kb genome of the positive-sense single-stranded RNA virus known as dengue encodes a single polyprotein that is post-translated into three structural proteins, including the capsid, pre-membrane, and envelope, as well as seven non-structural proteins (NS1, NS2A, NS2B, NS3, NS4A, NS4B and NS5). There are four DENV serotypes (1-4) that are antigenically similar and have 60%–75% amino acid homology.

As a result, several DENV diagnostic instruments struggle to differentiate between DENV serotypes. Additionally, the difficulty of virus identification is increased by the co-circulation of many serotypes during DENV epidemics. A potential additional consequence for patients receiving effective treatment is antibody dependent exacerbation of the disease, which occurs when patients get a heterotypic secondary DENV infection. Therefore, it is a challenge for researchers to create Directly Active Antivirals (DAA) that can inhibit the complete range of genetically varied DENV serotypes and/or genotypes. To treat dengue infections, however, there are still no licenced medications on the market despite the unceasing efforts to develop an antiviral therapy. Currently, there are just supportive treatments available.

CONCLUSION

The lack of a well-defined panel of viruses that specifically captures the genetic variety of all known DENV isolates poses a significant obstacle to evaluating the activity range of prospective DENV-inhibitory compounds. We have created a library of DENV with sequences that comprise typical genotypes from within the four DENV serotypes in order to provide a tool for DENV research, with which to evaluate the antiviral activity of possible inhibitory compounds.

A full reference virus panel must be used to determine the antiviral efficacy against each of the reported DENV genotypes in order to evaluate anti-dengue candidate compounds effectively in cellulo.

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