

## DENGUE VIRUS: A BASIC STUDY

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**Abstract**– Female Aedes mosquitoes transmit the acute viral infection dengue which is spread by an RNA virus belonging to the Flaviviridae family. In tropical and subtropical regions, it is more prevalent. The causative agent of dengue fever is dengue virus (DENV) with four serotypes (DENV 1-4). An estimated 25 to 100 million cases of dengue fever (DF) are caused by DENV.

### INTRODUCTION

Dengue fever is a mosquito-borne viral illness that can cause a range of symptoms, from mild to severe. Dengue fever (DF), the most prevalent viral illness in humans, is caused by the dengue virus (DENV) and it is basically arthropod borne disease. The four serotypes of DENV (DENV 1-4), slightly antigenically distinct are primarily transmitted to humans through the bite of infected Aedes mosquitoes, particularly *Aedes aegypti* (Murugesan and Manoharan, 2020). However, the other two vectors such as *Ae. polynesiensis* and *Ae. niveus* have been identified as the secondary vectors in some regions throughout the world (Zahoor *et al.*, 2018). DENV is a member of the Flaviviridae family and has similarities to the viruses that cause yellow fever and the Japanese, St. Louis, and West Nile encephalitides. The Flaviviridae branched into four subgroups: (1) the insect-specific viruses that have only been isolated from various mosquito species; (2) the vertebrate viruses that have no known arthropod vector, and which have been isolated only from rodents and bats; (3) the mosquito-borne viruses; and (4) the tick-borne viruses. DENV causes an estimated 25 to 100 million cases of dengue fever (DF) and 250,000 cases of DHF (dengue hemorrhagic fever) per year worldwide, with 2.5 billion people at risk of infection (Ross, 2010).

#### **Virology and molecular basis of pathogenesis related to Dengue virus:**

DENV is an enveloped virus with a single-stranded, spherical shape with 11kb in length and positive-sense 10.7 kilobase RNA genome as genetic material

which has a 5-methyl cap with a single open reading frame. Its outer surface is covered with envelope proteins surrounding a lipid bilayer envelope. The genetic material is translated as a single polyprotein. This polyprotein was then cleaved into 3 structural proteins (capsid [C] protein, premembrane/membrane [prM/M] protein, and envelope [E] protein) and 7 nonstructural (NS) proteins by the virus- and host-encoded proteases. The 3 structural components are required for capsid formation (C) and assembly into viral particles (prM and E) to form complete virus progeny. The NS proteins contain a serine protease and ATP-dependent helicase (NS3), which is required for virus polyprotein processing, a methyltransferase and RNA-dependent RNA polymerase (NS5), and a cofactor for the NS3 protease (NS2B). The NS protein is very much essential for viral replication purposes, as per a recent study NS4B has been implicated in blocking the interferon (IFN) response.

In primary DENV infection, the virus enters target cells after the E protein adheres to cell surface receptors. Viral uptake occurs by receptor-mediated endocytosis. Endosomal acidification induces a conformational change in the E protein, due to the changes in E protein conformation fusion of the viral and endosomal membranes occurs and the viral nucleocapsid is released into the cytoplasm (Ross, 2010; Monath, 1994). Virus genome replication and assembly of viral particles occurs in discrete domains within the endoplasmic reticulum (ER) of the host cell (Mackenzie *et al.*, 1999).

#### **Viral particle Assembly**

Viral RNA is synthesized in multiple steps. First, the

ends of the viral RNA fold up to form a circle. The RNA then attaches to the replication complex system to initiate the first round of synthesis. Viral RNA's positive-sense RNA act as a template to make a negative-sense copy. The pair of RNA strands form a double helix of the same and the RNA becomes a circle again. Now, the negative-sense strand acts as a template to make a positive-sense strand. Many copies of the positive-sense RNA strand are made by repeated cycles of RNA synthesis. Some of these positive sense RNA strands are involved in translation to make more viral proteins. The aggregation of envelope proteins occurs in the lumen of the endoplasmic reticulum and the capsid proteins aggregate on the cytoplasmic side. A viral RNA binds to the capsid protein for the packaging into a new virus particle as it buds off into the endoplasmic reticulum. Though the virus is not mature. Its pre-membrane proteins cover the tips of the envelope proteins to prevent premature fusion back into the cell. The virus buds off to travel through the Golgi apparatus and continues toward the cell surface. Before reaching the surface, the premembrane protein is processed and a virus becomes mature. New dengue viruses are released from the cell ready to infect other cells (<https://www.biointeractive.org>).

### **Complications related to Dengue virus transmitted disease**

#### **Skin**

In the case of 20% of Dengue patients, skin flushing including the face, chest, neck may be seen (Murugesan and Manoharan, 2020).

#### **Eye**

Musculoskeletal symptoms are very common aspect of dengue. Nearly all adults have some degree of myalgia, and about one-third of patients also have arthralgia. These characteristics can help distinguish dengue from other febrile infections (Murugesan and Manoharan, 2020).

#### **GI Tract**

Many people with dengue fever reported experiencing anorexia, nausea, vomiting, diarrhea, and abdominal pain. The first five days of an illness are typically characterized by nausea, vomiting, and anorexia, while abdominal pain tends to develop slightly later, between days 3 and 6 of illness (Murugesan and Manoharan, 2020).

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