

PHYLOGENETICS IN MOLECULAR EPIDEMIOLOGY OF ZIKA VIRUS

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Zika virus infection acquired devastating phenomenon globally because of its ability to cause birth defects such as microcephaly and other neurological manifestations like Guillain-Barré syndrome. The zika epidemic in Brazil turned into a public health emergency and spread to several countries. The Latin American country of Colombia also reported 42 cases of zika associated congenital syndrome followed by U.S.A. with 26 cases as on October 2016. The genetic evolution of the virus in the Americas was the objective of the study which helped to throw light on its pathogenicity.

Mutation of a highly conserved threonine to alanine was observed at carboxy terminal of NS5 gene protein sequence coding for RNA polymerase. The hydrophilic amino acid was substituted with hydrophobic amino acid which is predicted to bring about change in interaction of the virus. The polymorphism found at the phylogenetically conserved amino acid position of 581 was useful in genotyping the zika virus. This led to the classification of a Latin American genotype of zika virus isolated from countries of Colombia, Mexico, Panama and Martinique. (Adiga, 2016 J. Med. Virol. 88:1821–1826).

Further computational analysis of the protein sequence of NS5 led to focus attention on the plausible benefits accrued to the virus in mutating the particular residue. Viruses have been known to use host-like amino acid motifs to interact with host proteins as part of molecular mimicry. The viral strategies promote host modulation and facilitate viral-host interaction. (Hagai et al, 2014 Cell Reports 7: 1729–1739). Motif analysis led to the identification of a conserved PVA motif which belongs to the family of chorion S19 gene. The chorion gene is developmentally regulated, has a role in cell proliferation and is secreted as middle stage autosomal chorion cluster from the chorion, which is the fetal part of the placenta. The presence of zika virus in amniotic fluid and its capacity to cross placental barrier spreading from the decidua to chorionic membrane has been reported. The interaction with host-like motifs may help gain entry of the virus and is predicted to interact with the chorion. Other motifs predicted are the THAP zinc finger which have DNA binding activity. Though clinical co-relation with the Latin American genotype has not been found, the NS5 gene is predicted to facilitate host- viral interaction.