World Academy of Science, Engineering and Technology
International Journal of Medical and Health Sciences
Vol:11, No:09, 2017

Alleviation of Endoplasmic Reticulum Stress in Mosquito Cells to Survive Dengue 2 Virus Infection

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Abstract : Dengue viruses (DENVs) are naturally transmitted between humans by mosquito vectors. Mosquito cells usually survive DENV infection, allowing infected mosquitoes to retain an active status for virus transmission. In this study, we found that DENV2 virus infection in mosquito cells causes the unfolded protein response (UPR) that activates the protein kinase RNA-like endoplasmic reticulum kinase (PERK) signal pathway, leading to shutdown of global protein translation in infected cells which was apparently regulated by the PERK signal pathway. According to observation in this study, the PERK signal pathway in DENV2-infected C6/36 cells alleviates ER stress, and reduces initiator and effector caspases, as well as the apoptosis rate via shutdown of cellular proteins. In fact, phosphorylation of eukaryotic initiation factor 2α (eIF2α) by the PERK signal pathway may impair recruitment of ribosomes that bind to the mRNA 5′-cap structure, resulting in an inhibitory effect on canonical cap-dependent cellular protein translation. The resultant pro-survival “byproduct” of infected mosquito cells is undoubtedly advantageous for viral replication. This finding provides insights into elucidating the PERK-mediated modulating web that is actively involved in dynamic protein synthesis, cell survival, and viral replication in mosquito cells.

Keywords : cap-dependent protein translation, dengue virus, endoplasmic reticulum stress, mosquito cells, PERK signal pathway

Conference Title : ICVVIHIS 2017 : International Conference on Viruses, Viral Infections and Human Immune System

Conference Location : San Francisco, United States

Conference Dates : September 28-29, 2017