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ER Stress involved in replication and pathogenesis of Dengue virus

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Abstract

Dengue virus is a single standard positive RNA virus that responsible for developing mysterious dengue fever. Dengue virus (DENV) associated with endoplasmic reticulum for replication and assembling. ER Stress response generated due to the accumulation of unfolded proteins in the endoplasmic reticulum lumen and unfolded protein response (UPR). UPR significantly reduced dengue virus due to the involvement of three different branches having a role in cellular activity. Moreover, ER Stress responsible for the induction of autophagy response. Dengue virus-induced autophagy response which represents an inter-linked reaction for the virus replication and modification. We demonstrated that CHOP and IRE-JNK signaling pathways increased cell migration response with autophagy response with elevated expression level of dengue virus. ER Stress pathway interlinked with apoptosis. These findings suggest that the invitro trail represents the dengue virus titration level with symptoms development through connected with ER Stress and apoptosis pathway. The present study filled a gap between dengue virus-induced ER Stress autophagy and pathogenesis through the UPR signaling pathway.

Keywords:

Dengue virus, ER Stress, autophagy

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