THE ROLE OF CHOLESTEROL AND GLYCOSPHINGOLIPIDS IN HUMAN CELLULAR INFECCION BY DENGUE VIRUS

Germer, R. [1]; Scovino, A.M. [1]; Rust, N.M.[4]; Peçanha, L.M.T.[2]; Nimrichter, L.[3]; Arruda, L.B.[1]

Departamentos de [1]Virologia, [2]Imunologia e [3]Microbiologia Geral, Instituto de Microbiologia Prof. Paulo de Góes. [4]Instituto de Biofísica Carlos Chagas Filho. UFRJ. Rio de Janeiro – RJ

Dengue is the most prevalent arthropod-borne viral illness in human being and is caused by any of the four serotypes of dengue virus (DV 1–4). The virus biosynthesis in mammalian cells is initiated by receptor-mediated endocytosis. After membrane fusion, protein synthesis and genome replication, the viruses bud from cellular membranes. Lipid rafts are liquid ordered membrane microdomains enriched with sphingolipids and cholesterol, which may participate in some events of cell-virus interaction, such as attachment, fusion and budding from the host cell. To study the functions of the rafts, human brain microvascular endothelial cells (BMECs) and glial cells (U87) were infected with DV-2 and DV-3 in the presence of either beta-ciclodextrin (β–CD) or threo-1-phenyl-2-decanoylamino-3-morpholino-1-propanol (PDMP). These are raft-disrupting drugs responsible for cholesterol depletion and inhibition of glycosphingolipid synthesis, respectively. The infection was evaluated by imunofluorescence microscopy and plaque forming units (PFU). We observed that treatment of both U87 and BMECs with β–CD and PDMP inhibited DV-2 and DV-3 infection, affecting cell attachment and virus entry. Taken together, our data evidence the importance of cholesterol and glycosphingolipids to DV infection in human cells, suggesting a crucial role of lipid rafts.

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