Nistatina um macrolídeo polienólico é viricida

Requirement of cholesterol in the viral envelope for dengue virus infection.


Source

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Abstract

The role of cholesterol in the virus envelope or in the cellular membranes for dengue virus (DENV) infection was examined by depletion with methyl-beta-cyclodextrin (MCD) or nystatin. Pretreatment of virions with MCD or nystatin significantly reduced virus infectivity in a dose-dependent manner. By contrast, pre-treatment of diverse human cell lines with MCD or nystatin did not affect DENV infection. The four DENV serotypes were similarly inactivated by cholesterol-extracting drugs and infectivity was partially rescued when virion suspensions were treated with MCD in the presence of bovine serum. The addition of serum or exogenous water-soluble cholesterol after MCD treatment did not produce a reversion of MCD inactivating effect. Furthermore, virion treatment with extra cholesterol exerted also a virucidal effect. Binding and uptake of cholesterol-deficient DENV into the host cell were not impaired, whereas the next step of fusion between virion envelope and endosome membrane leading to virion uncoating and release of nucleocapsids to the cytoplasm appeared to be prevented, as determined by the retention of capsid protein in cells infected with MCD inactivated-DENV virions. Thereafter, the infection was almost completely inhibited, given the failure of viral RNA synthesis and viral protein expression in cells infected with MCD-treated virions. These data suggest that envelope cholesterol is a critical factor in the fusion process for DENV entry.

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