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Opinion Article

Signatures of Dengue Virus Receptor Proteins in Human Cells

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ABOUT THE STUDY

Dengue fever is a mosquito-borne infection spread by *Aedes* mosquitos. In humans and nonhuman primates, the dengue virus causes fever and hemorrhagic diseases. Dengue fever is one of India's most serious health issues. One of the earliest processes in Dengue virus pathogenesis is interaction with particular receptors on the cell surface. These receptors, on the other hand, are poorly understood.

The viral infection primarily targets cellular receptors in human monocytes and mice brain cells. The virus's envelope protein (E-protein) is necessary for virus attachment to target cells and interaction with cellular receptors. Modulation of receptor genes and/or proteins can be utilized to block virus entrance, and hence could become a novel approach of disease prevention. Purification of receptors using affinity chromatography and E-protein as a ligand seems possible. The addition of highly sulfated heparan sulphate impairs E-protein binding to target cells, implying that the dengue envelope protein uses heparan sulphate to bind to target cells.

The presence of non-neutralizing antibody increased dengue virus infection in human peripheral blood leukocytes. Fcγ receptors expressed on leukocytes were responsible for the increase. These data suggested that Fcγ receptor-mediated entry is implicated in secondary infection, particularly infection with a different serotype from that which caused primary infection. Mammalian cells have been used to study primary infection and the virus's first contact with host cells in order to uncover receptor molecules. Several investigations on dengue virus receptors in mammalian cells have been undertaken earlier. Four major groups of potential compounds can be identified. To begin with, carbohydrate molecules like sulfated glycosaminoglycans (GAGs) and Glyco Sphingo Lipid (GSL) are hypothesized to act as co-receptor molecules, enhancing virus entry efficiency.

Heparan sulphate is a sulfated GAG that is required for virus adsorption to host cells. Another form of carbohydrate molecule has recently been discovered to have a role in virus attachment neolactotetraosylceramide (nLc4Cer), a non-sulfated GSL, may also operate as a co-receptor on host cells. DENV virus responsible for causing dengue infection of many cell types was successfully suppressed by native and semi synthetic carbohydrate compounds generated from GAG and GSL structures.

These data suggest that extracellular carbohydrate molecules play an important role in DENV proliferation in target cells. Second, lectins, which are carbohydrate-binding proteins expressed on Dendritic Cells (DCs) and macrophages beneath the human skin, are implicated in the initial contact of DENV transmitted *via* mosquito bite. Dendritic cell-specific intercellular adhesion molecule-3-grabbing non-integrin (DC-SIGN) has been the most well studied of these lectins in virus-DC interactions.

Recombinant lectin protein binds directly to N-glycans at position 67 of E protein produced on viral particles, according to cryoelectron microscopic examination. DENV may proliferate in DC attributable to DC-SIGN-mediated entry, indicating that DC is the virus's principal target. Another lectin, mannose receptor, was recently discovered to have a role in DENV entrance into macrophages. Taken together, the findings suggest that carbohydrate recognition events are linked to DENV transmission in the human body.

Third, molecules involved in protein folding, such as heat shock proteins and chaperones, may play a role in DENV serotype 2 (DENV-2) interactions with host cells. A single serotype, DENV-2, has been found to bind these compounds. Fourth, additional research has revealed that other proteins, such as the high-affinity laminin receptor, CD14-associated protein, and unidentified proteins, may be involved in DENV-host cell contact. Some of the proteins described thus far may have the same features, such as molecular mass.

Some of the suggested receptors appear to interact with several DENV serotypes, whereas others appear to connect with a specific DENV serotype. These data strongly suggest that DENV binds various molecules that may form complexes on host cells, and that DENV enters different types of cells through specific combinations of receptor possibilities. The nature of cellular

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receptors and molecular pathways for dengue viral entrance, on the other hand, is yet unknown.

CONCLUSION

The immune system is the body's primary defense against the dengue virus. When someone is infected with dengue, the innate and adaptive immune responses combine forces to fight the virus. B cells produce antibodies that specifically recognize and neutralize the foreign viral particles, and cytotoxic T cells recognize and kill cells that are infected with the dengue virus. People who are infected a subsequent time with a different type of the dengue virus may experience something called antibody-dependent enhancement. This condition occurs when the immune response actually makes the clinical symptoms of dengue worse, increasing the risk of severe dengue. In contrast, a comprehensive differential gene expression investigation of *Leishmania* constitutively expressed genes revealed just a small number of genes that showed variable expression during development. Survival may not be dependent on the induction or control of gene expression for differentiation, virulence, or pathogenicity. Rather, by exploiting a needed set of genes/proteins for each dramatically different environment, constitutive expression may enable *Leishmania* to be constitutively adapted for survival and reproduction in either the *aedes* mosquito's vector or host macrophages.