C-Terminal peptide of M protein from dengue virus (DVM-C) forms ion channels

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The dengue virus belongs to the family of Flaviviridae and causes an infectious disease carried by mosquitoes - dengue fever. There is no specific treatment for dengue fever, and most people recover completely within 2 weeks.

Like the alphaviruses and influenza viruses, the dengue virus enters cells in an endocytotic vesicle. The M, or membrane protein, of the dengue virus may have a similar function to that of the M2 protein of Influenza A and assist in the entry of the virus by functioning as an ion channel. The M protein is one of the structural proteins of the virus and is 75 amino acids in length. The C-terminal end of the protein from amino acids 48 to 70 contains a predicted transmembrane region, thought to anchor the protein in the lipid bilayer (Kuhn *et al.*, 2002). The function of the M protein is still unknown. To test the hypothesis that the M protein of dengue functions in a similar manner to the M2 protein of influenza A we tested the C-terminal end of the M protein containing the putative transmembrane region for channel activity.

The C-terminus of the M protein of the Dengue virus type 1 strain Singapore S275/90 (DVM-C) was chemically synthesised and tested for channel forming properties in artificial lipid bilayers.

We have found that the DVM-C peptide from Dengue virus forms ion channels in lipid bilayer membranes. The channels were selective for monovalent cations over monovalent anions but were relatively impermeable to calcium ions. Amantadine (10 μM) and 100 μM Hexamethylene amiloride (HMA) block channels formed by DVM-C. The dengue virus M protein can be added to an increasing list of virus proteins that have been shown to form ion channels in artificial lipid bilayers.

Kuhn, R.J., Zhang, W., Rossmann, M.G., Pletnev, S.V., Corver, J., Lenches, E., Jones, C.T., Mukhopadhyay, S., Chipman, P.R., Strauss, E.G., Baker, T.S., Strauss & J.H. 2002. *Cell* 108**:**717-25.