

The Exposition of Antigenic Determinants of the Semliki Forest Virus Envelope Glycoprotein E₂ can be Altered by Carbohydrate Chains

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On E₂ of the viral envelope, 6 non competing epitopes a-f can be discerned by means of monoclonal antibodies. Blocking tests revealed that carbohydrate side chains of the viral glycoproteins compete with at least one of them (f). Epitopes b and e cannot be detected, if intramolecular disulfide bonds in the viral envelope are cleaved, or in E₂ of virus particles released in the presence of MdN (such particles are devoid of complex carbohydrate chains), or in infected cells after a 15 min - pulse labelling. These two epitopes seem to be located on a critical structural point specific for the final conformation in the spike complex. Antibodies directed to epitope b have neutralizing properties. The data confirm earlier findings, that the final structure of the envelope glycoprotein E₂ is acquired upon particle release.

Molecular Biological Aspects of Virus-Induced Subacute Encephalomyelitis in Lewis Rats

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In order to define parameters leading to persistent measles virus (MV) infection of the CNS, the expression of MV specific RNA's and the related proteins were investigated in our model of measles virus induced subacute encephalomyelitis (SAME) in Lewis rats. In vivo results suggest that synthesis of measles virus H, F and M protein is reduced or absent in the brain of SAME animals already 2-3 weeks following infection. The virus specific mRNAs, however, have been shown to be present for all six structural proteins in the subacute type of infection by Northern blot analysis and in situ hybridization with strand specific probes. In contrast, in *in vitro* experiments the matrix protein and probably the H proteins cannot be translated from the corresponding mRNA. - These results indicate specific restriction of the expression of viral M protein in CNS infections supporting measles virus persistence.

Selection of Mutations in the Primary Immunogenic Proteins of Sendai-Virus in Persistently Infected Mice

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C129 mice were inoculated with Sendai-Wildtype-Virus (Strain D52) or ts-mutants ts1-5 transcription-defective at 39°C. Infectious virus could be shown in the sera of all infected mice on PID 22. Brain cells of the ts4 infected mice were cocultivated with uninfected mice