Poster Session I

Retroviruses

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Short sequences of the matrix- and the capsid-protein of the *Human Immunodeficiency Virus* (HIV) influence viral assembly and inhibit production of infective particles

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Besides the approach to interfere with the enzyme activities of the *Human Immunodeficiency Virus* (HIV) other mechanisms of the HIV replication cycle may also be inhibited. Especially the viral assembly and budding of mature virus particles represent a worthwile target to prevent the production of infectious viruses. During assembly the p55*gag*-precursor-molecules are transported to the inner side of the cell membrane of the host cell where they are inserted into the lipid bilayer by means of the cotranslationally added fatty acid myristate. After release of the virus particles the precursor proteins are cleaved by a virus-encoded protease into the p17 matrix (MA), the p24 capsid (CA), the p9 nucleocapsid (NC) and three smaller proteins (p6, p2, p1). The structurally highly ordered arrangement of the membrane bound polyproteins is a necessary prerequisite for the budding and maturation process. Distinct sections of the p17 and the p24 are thereby functionally active and are highly conserved between individual isolates. In order to clearly determine such regions we exchanged amino acid tripletts in the p17-region (amino acids 46-60) and in the p24-region (amino acids 341-352) for alanine and also deleted the whole regions. The effect of the mutations was investigated by *gag*-particle formation in the Vacciniavirus-, the Baculovirus- and the HIV-proviral system. So far two p24 mutants turned out to be deficient in the intracellular virus assembly and/or budding since much less virus-like particles were detectable in the cell supernatant. The results of the infection experiments performed with the p17 mutants will be shown. Taken together they may represent the experimental basis for a previously shown inhibitory effect of synthetic peptides on the production of infectious HIV-particles derived from those regions. Additionally performed experiments indicated that those sequences are not involved in cyclophilin binding of the *gag*-protein as recently demonstrated.