Abstract

Most viruses can infect only a reduced range of organisms and an effective replication is possible only in selected hosts. These hosts are called permissive for the virus. Molecular principles of a nonpermissiveness and viral mechanisms of overcoming replication obstacles are still not clearly elucidated.

This thesis discusses the molecular causes of the cellular nonpermissiveness against a model retrovirus – Rous sarcoma virus. The research is conducted on duck cells which are semipermissive to the subgroup C of Rous sarcoma virus. The virus can enter those cells, but it is not able to produce enough infectious viral progeny.

Two blocks of the viral replication cycle in the duck cells are described in the thesis. The first one is the probably not optimal cellular receptor recognition. The second one is in the late phase of the replication cycle when the viral proteins are synthesized. The amount of the envelope glycoprotein coding mRNA is reduced due to the altered splicing ratios, and the virions produced from the duck cells are less infectious. This block is recessive and can be partially omitted by cell fusions with permissive chicken cells; therefore, the block is not caused by specific restriction factors in *sensu stricto*.

Additionally, the influence of mutations in duck adapted Rous sarcoma virus was studied. Acquired mutations in envelope glycoprotein do not have the expected effect on the virus replication in the duck cells.

Keywords: adaptation, cell fusion, duck, heterologous host, heterotransmission, mRNA splicing, permissiveness, RCAS, replication cycle, retrovirus, Rous sarcoma virus, RSV.