Abstract

Coevolution of pathogens and their hosts leads to selection of series mutations. From the hosts point of view we can these accumulated mutations characterize as restriction factors preventing effecive replication of target pathogen, for example retrovirus. These factors acts on level of virion entry into cell, its intergation, expression and maturation. On the other hand, from the viruses point of view these mutations manifests the ability to overcome restriction factors mentioned earlier. One of the mechanizms is for example shift in receptor specifity, which allows to virus infect hosts in case, where original specific receptor is for some reason unavailable. This thesis focus on effect point mutation L154S in envelope glycoprotein of virus MAV-B and its influence in replication and pathogenesis *in vitro* and *in vivo*. For this purpose we infected and analyzed chicken breed of white and brown leghorns and different cell cultures. Results of infection of chickens displayed increased incidence of osteopetrosis, disease displaying the hyperthrophy of bone matter. In consistence with literature, we shown polyclonal origin of this neoplasia. With series of experiments we discovered abnormal behavior of envelope glycoprotein coded by virus causing this disease. Abnormality lies in termal lability and neutralization by low pH. Based on our observation we suggest model of premature activation of glycoprotein, which cause nonspecfic fusion with target membrane in a receptor independent way.

Key words: retrovirus, ASLV, receptor, env, gallus, osteopetrosis, receptor interference, MAV-B, extended host range