# Abstract

Mouse polyomavirus (MPyV) capsid is composed of the main capsid protein VP1 and minor capsid proteins VP2 and VP3. Minor proteins are not essential capsid assembly, but they are key for efficient viral infection. The first part of this thesis studies the modifications of VP2 and VP3, the deamidation of Asn at 253 of VP2 (137 of VP3) and N-terminal acetylation of Ala of VP3, which could be the cause of double bands for VP2 and VP3 on SDS-PAGE. Mutated genomes of MPyV N253D (Asn to Asp) and N253E (Asn to Glu) simulating deamidation and A117V (Ala to Val) with reduced acetylation were prepared previously. We prepared three isolations of the mutant viruses and we confirmed that the deamidation is the cause of the double bands. Mutant viruses were compared to the wild type in terms of efficiency of infection, but the role of deamidation could not be proven. Virus A117V is noninfectious either due to lowered acetylation or the substitution of amino acid at this position. This thesis also studies the role of α-tubulin acetylation in the infection of MPyV. The role of α-tubulin acetylation in viral infection is being investigated to find new antiviral strategies. Acetylation rises after MPyV infection, but this is not due to a change in mRNA expression of tubulin acetylating (αTAT1) or deacetylating enzyme (HDAC6). Inhibition of HDAC6 by tubacin, specific inhibitor of tubulin deacetylating activity, leads to higher efficiency of infection in early and late phase of MPyV infection.



**Key words:** mouse polyomavirus, minor proteins, microtubule acetylation, posttranslational modification, deamidation, acetylation, VP2, VP3, HDAC6