The main structural protein VP1 is the product of late polyomaviral genes and it is the largest and the most abundant protein of the whole polyomaviral capsid. Because of the low coding capacity of the polyomaviral genomes, it is considered that in addition to its structural role the VP1 protein might have some additional functions in the late phase of the infectious cycle. This diploma thesis is exactly on these additional functions. In the case of the VP1 protein of mouse polyomavirus, it was observed that the protein is capable of binding to the structure of cellular microtubules. The first objective of this work was to test whether pentamers of the VP1 protein are able of this binding without the participation of other cellular (or viral) proteins. Based on an *in vitro* experiment, we showed that protein VP1 binds to the structure of microtubules very inefficiently. The second objective of this work was to prepare a detection system that would allow an identification of potential interaction partners of BK polyomavirus VP1 protein. Therefore, expression plasmids producing the N and C-terminally tagged VP1 protein were prepared. These tagged proteins had the property of being biotinylated whilst being produced in the transfected cells. By using affinity chromatography, the entire protein complexes containing the modified protein were isolated. Using mass spectrometry, individual isolated proteins were identified and after a subsequent analysis and data filtering, a list of 128 potential interaction partners of BK polyomavirus VP1 protein was compiled.

Keywords: mouse polyomavirus, BK polyomavirus, VP1 protein, VLPs, microtubules, interactions partners