Vaccinia virus is a typical member of the poxvirus family. It had been successfully used during the worldwide smallpox eradication campaign. Currently, it is used as vector for prophylactic, as well as experimental purposes.

This PhD thesis stems from the findings previously published in our lab. It is partially focused on a further characterization of vaccinia virus effects on the type of host cell death. The results point to the activation of apoptosis during vaccinia virus infection, but it cannot be completed and the cell dies by necrosis. Our attempts to shift the necrotic type of cell death induced by vaccinia virus infection towards apoptosis using a pharmacological inhibition of activity of a key enzyme PARP (Poly-(ADP-ribose) polymerase) remained unsuccessful. The effects of vaccinia virus-encoded anti-apoptotic factors appear superior to the inhibition of this single enzyme.

The second part of the thesis is focused on inhibitory effects of a redox-modulating compound, lipoic acid, on vaccinia virus infection. Our results demonstrated its inhibitory effects in cell lines of different embryonic origin. It appears that the lipoic acid inhibits vaccinia virus growth at the stage of late gene expression or possibly later, during virus morphogenesis. Lipoic acid could be potentially used as a supportive treatment in therapy of a poxvirus infection.