Abstract:

Viruses, etiological agents of many infectious diseases, are small noncellular particles that run their replication process solely inside host cells. It is generally assumed that the posttranslational modifications of viral capsid proteins are responsible for their infectivity (e.g. phosphorylation catalysed by kinases of host cells). The appropriate model for study of the viral phosphorylation "profile" relation with its infectivity is mouse polyomavirus of the *Polyomaviridae* family.

By comparison of virions, as well as the major capsid protein VP1 of wild type mouse polyomavirus with viral mutant created by deletion of the part of the genome coding regulatory proteins of virus and produced in two different cell lines WOP and 3T6 was found by mass spectrometry a major phosphorylation in the three specific amino acids of VP1. These are considered important for proper morphogenesis of virions and their ability to infect host cells. The qualitative representation was not affected by the cell line selection.

Furthermore, in case of VP1 "dimerization" detected on SDS-PAGE electrophoretogram the double phosphorylation of VP1 pThr63, pSer66 was confirmed in our experimental *in vivo* approach. Therefore, posttranslational modifications, specifically phosphorylation, could probably affect structural properties of viral proteins.

The integral part of the work was a successful optimalization of many used methodical approaches: the purification of intact viral particles, determination of protein concentration of virions as well as protein enrichment of viral particles also in a case of the low concentration of the purified virions.

Key words: mouse polyomavirus, posttranslational modifications of proteins, mass spectrometry