Polyomaviruses are small non-enveloped DNA viruses infecting birds and mammals, including human. Their capsid consists of the major capsid protein, VP1, and two minor capsid proteins, VP2 and VP3. The VP2 and VP3 proteins are supposed to have an important function in the transport of viral genome into the cell nucleus, which is a key step to facilitate viral replication. VP2 and VP3 proteins of mouse polyomavirus and SV40 have an ability to bind and disrupt cellular membranes. This feature is believed to be involved in the transport of viral genome into the nucleus. Plasmids carrying genes of the minor capsid proteins of Merkel cell polyomavirus were prepared in order to produce and visualize these proteins in mammalian cells. These proteins are known to have very unusual sequences compared to other human polyomaviruses or related mouse polyomavirus. When produced alone, the minor capsid proteins of Merkel cell polyomavirus did not significantly interact with cellular membranes, unlike the minor proteins of the mouse polyomavirus. The second goal of this work was to prepare mouse polyomavirus mutants with deletion in hydrophobic domains of VP2 and VP3 proteins. These domains are likely responsible for the mentioned membrane interactions. Prepared mutants were non-infectious. The loss of infectivity was not only due to missing hydrophobic domains, since the overall production of virus structural proteins seemed to be affected.