Abstract

Mouse polyomavirus (MPyV) is a model virus of the *Polyomaviridae* family. Polyomaviruses are small non-enveloped DNA viruses. They cause severe problems to immunocompromised patients. Their oncogenic potential is known in animals and humans. Trafficking of MPyV within the cell is not clear yet. The virus enters via smooth monopinocytic vesicles and continues to early and late endosomes. From there, the virus is transported to the ER by unknown mechanism. It bypasses Golgi aparatus (GA). One possible pathway is from late endosomes to *trans*-Golgi network (TGN) facilitated by Rab9 GTPase and then in COPI vesicles to the ER. In this thesis, the effect of inhibitors of retrograde transport (Brefeldin A, Golgicide A) on MPyV infection was evaluated. Brefeldin A is not completely specific; it has effect on whole endosomal system. Golgicide A causes specific disruption of transport via TGN and GA. Both inhibitors suppressed infection of MPyV. Confocal microscopy revealed colocalization of some MPyV virions with markers of TGN and COPI vesicles. MPyV didn't colocalize with *cis*-Golgi marker. Unfortunately, the effect of overexpression of Rab9 dominant negative mutant couldn't been evaluated due to its high cytotoxicity. However, overexpression of wild type Rab9 slightly increased infectivity. The results support hypothesis that MPyV enters TGN and from there it travels in COPI vesicles to the ER.

Keywords: mouse polyomavirus, retrograde transport, Brefeldin A, Golgicide A, colocalization, *trans*-Golgi network, COPI, Rab9