Toll-like receptors (TLRs) are important receptor family of innate immunity. They enable fast recognition of infection through so called pathogen associated molecular patterns (PAMPs). In this thesis, we studied interaction of mouse polyomavirus (MPyV) with TLRs of mouse embryonic fibroblasts (MEF cells). We observed that inhibition of TLR4 signaling abolished response of MEF cells to MPyV. This suggested that TLR4 plays a role in MEF cells recognition of MPyV. To detect response of MEF cell to MPyV, we measured IL-6 production by ELISA.

Next, we investigated effect of TLR4 signalization on MPyV infection. Inhibition of TLR4 signaling with CLI-095 inhibitor did not affect number of infected cells. Presence of TLR4 antagonist, LPS-RS, led to significant decrease in quantity of infected cells 20 hours post infection. Decrease in number of infected cells was also observed in presence of LPS.

Viral infection was also inhibited by TLR9 antagonist ODN 2088.

We also investigated role of MAP kinases in MPyV infection. We tested, whether inhibition of selected MAP kinases would affect number of infected cells. Inhibition of kinase p38 did not affect infection. On the other hand, inhibition of MEK kinase or JNK resulted in decrease of number of cells infected by MPyV.