**Abstract** - The phosphorylation state of proteins is tightly controlled by cellular protein kinases and protein phosphatases. More than 70% of all eukaryotic cellular proteins are regulated by protein phosphorylation. The Ser/Thr protein phosphatase 1 (PP1) is responsible for much of the phosphatase activity in eukaryotic cells. Several viruses exploit the importance of PP1 for the host cell, including innate immune signaling, for the benefit of viral replication by the expression of viral PP1 binding proteins or inhibition of PP1 activity. In contrast, Human Cytomegalovirus (CMV) activates PP1 expression in target cells and additionally carries cellular PP1 in its virion for a rapid supply of the enzyme after viral entry. However, the mechanism and consequences behind CMV-mediated PP1 activation remain unknown.

**Human cytomegalovirus** kidnaps the human protein phosphatase 1 from infected cells and carries it in the tegument layer of the virion.

**PP1** has different binding sites, the most important of which is the hydrophobic pocket binding to the “RVxF” motif present on almost all PP1-binding proteins.

Using a novel approach specifically targeting the regulatory “RVxF” binding site of PP1 rather than catalytic activity, we aim to inhibit the virus without harming the cell. All life stages of the virus are investigated (qPCR, Western blot, plaque assay) to see which stage is specifically targeted by the inhibitor.