

Impact of HCMV proteins on viral replication and cellular signaling pathways

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The human cytomegalovirus (CMV) is a member of the herpesvirus family. CMV causes severe disease in immunocompromised individuals and is a leading cause of birth defects worldwide. Upon infection of a host cell, CMV must overcome the cell's innate ability to prevent viral replication. The long-term goal of this project is to determine how HCMV proteins manipulate cellular proteins in order establish a permissive environment for replication. Recently, we have identified a network of virus-host protein interactions involving chromatin remodeling factors and the cellular tumor suppressor protein, p53. Along with viral proteins, these cellular factors participate in regulating viral gene expression from the CMV major immediate early promoter (MIEP). Using the technologies developed by the Wisconsin CEGS program, we are working to identify the specific changes in protein composition on the MIEP due to these regulatory factors.

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