Human cytomegalovirus (HCMV) is an important opportunistic pathogen. A crucial antiviral drug target is a protein kinase, UL97, which is encoded by the virus.

Study identified a relationship between UL97 and a cluster of viral genes, \textit{UL133-UL138} (UL133/8), best known for their roles in HCMV latency and reactivation from latency, the cause of viral persistence and a serious concern in immunocompromised patients.

Model for roles of UL97 and \textit{UL133/8} in the viral lytic cycle. In step 1, proteins expressed from \textit{UL133/8} promote transcription from the viral MIEP by an indirect mechanism that may involve a signal sent from the Golgi apparatus or cell surface, where \textit{UL133/8}-encoded proteins localize during infection. The signal requires UL135, which is antagonized by UL138. In steps 2 and 3, pRb negatively regulates transcription from the MIEP and viral E and E-L promoters. In step 2, UL97 inactivates pRb, which enables the signal from UL135 to result in increased expression of IE2. Viral E and E-L promoters are positively regulated by IE2. In step 3, UL97-mediated inactivation of pRb may also be important for high-level expression of viral E and E-L proteins that participate in viral DNA synthesis.