HCMV Latency Locus

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An Epistatic Relationship between the Viral Protein Kinase UL97 and the *UL133-UL138* Latency Locus during the Human Cytomegalovirus Lytic Cycle

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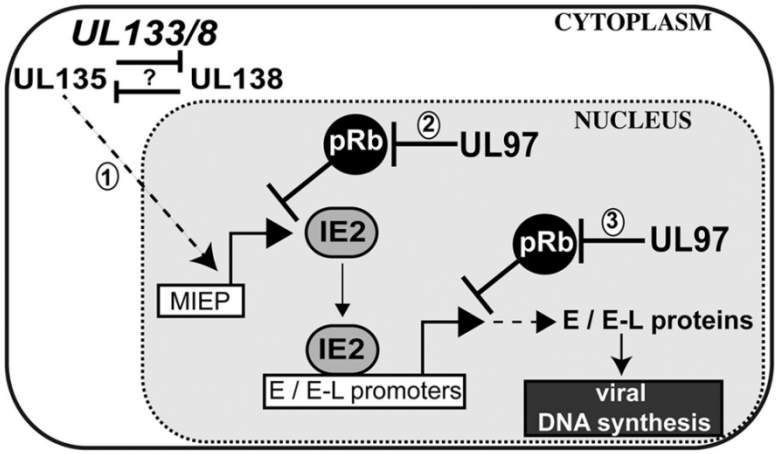
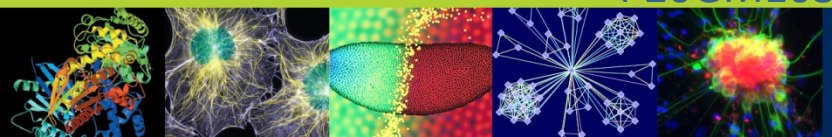
K. Frueh, Editor

- 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, *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.

Jeremy Kamil is an assitant professor at LSU, identified as faculty in Dr. O'Callaghan's COBRE grant.



Model for roles of UL97 and *UL133/8* in the viral lytic cycle. In step 1, proteins expressed from *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 *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.



NIH National Institute of General Medical Sciences