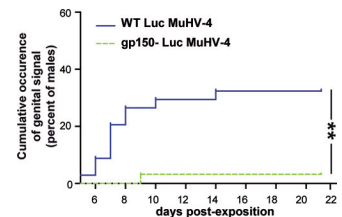




Articles of Significant Interest Selected from This Issue by the Editors

The Major Envelope Glycoprotein of Murid Herpesvirus 4 Promotes Sexual Transmission

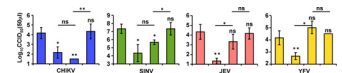
Gammaherpesviruses are important human and animal pathogens. Infection control has been difficult because the key process of transmission is not well understood. Murid herpesvirus 4 (MuHV-4), a gammaherpesvirus of rodents, transmits sexually in mice. Zeippen et al. (e00235-17) discovered that the major envelope glycoprotein, gp150, of MuHV-4 does not modulate viral entry or dissemination but rather promotes sexual transmission by facilitating virus release from infected vaginal epithelial cells. These data establish an important function of gp150, which also will be useful in investigating its homologs in human pathogens.



Absence of gp150 restricts sexual transmission of MuHV-4 from females to males.

Genome-Wide Screening Reveals Complexities of Chikungunya Virus Infection

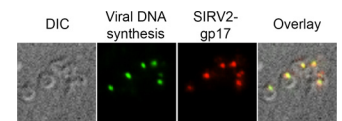
Various glycosaminoglycans (GAG) serve as attachment factors for microbes. Tanaka et al. (e00432-17) conducted genome-wide screening using human haploid HAP1 cells. N-Sulfate in heparin sulfate was found to be essential for efficient infection of HAP1 cells by a clinical chikungunya virus (CHIKV) strain. Chondroitin sulfate is essential for CHIKV infection but not required for binding. The mechanisms of CHIKV infection vary among host cell types. These data illustrate the complexity of the mechanisms of CHIKV propagation, cell tropism, and pathogenesis.



Susceptibility of various GAG-deficient HAP1 cells to CHIKV and other viruses.

Formation of a Viral Replication Focus by an Archaeal Virus

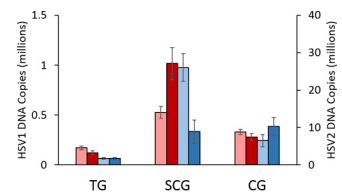
Many eukaryotic viruses and bacteriophages employ a strategy of compartmentalizing replication in viral replication organelles, i.e., viral factories or replication centers. Martínez-Alvarez et al. (e00486-17) discovered the formation of a peripheral replication focus by a crenarchaeal rudivirus, *Sulfolobus islandicus* rod-shaped virus 2 (SIRV2), in which viral DNA synthesis takes place and replication-related proteins are concentrated. This discovery reveals that the strategy of organization of viral replication in specialized microenvironments is common to viruses of all three domains of life.



Overlapping localization of viral DNA (green) and viral protein gp17 (red) in SIRV2-infected SsoTK cells.

Stress Hormones Modulate Herpes Simplex Virus Productive Infection

Stress exacerbates acute disease signs resulting from herpes simplex virus 1 (HSV-1) and HSV-2 infections. Stress hormones are thought to influence HSV-1 and HSV-2 replication through immune suppression. Ives and Bertke (e00582-17) show that neurons infected by these viruses express receptors for two stress hormones, epinephrine and corticosterone. Administration of these stress hormones modulates productive HSV-1 and HSV-2 infection in sympathetic but not sensory neurons. These findings show that stress has the potential to differentially affect HSV-1 and HSV-2 to produce divergent outcomes of infection and does so in autonomic rather than sensory neurons.



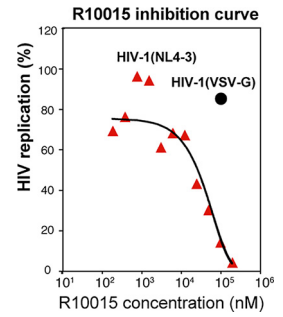
Stress hormones modulate HSV productive infection in sympathetic superior cervical ganglion (SCG) neurons.

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<https://doi.org/10.1128/JVI.00737-17>

New LIM Domain Kinase Inhibitors Block HIV-1 and Other Viruses

The LIM domain kinase (LIMK) regulates cofilin and actin dynamics that are required for efficient HIV entry, reverse transcription, nuclear migration, and virion release. Yi et al. (e02418-16) describe the design, discovery, and medicinal synthesis of classes of small-molecule LIMK inhibitors that block HIV infection. The LIMK inhibitor R10015 also inhibits herpes simplex virus, Rift Valley fever virus, Venezuelan equine encephalitis virus, and Zaire ebolavirus. This study initiates the development of these LIMK inhibitors as potential broad-spectrum antiviral drugs.



Dosage-dependent inhibition of HIV infection of human T cells by LIMK inhibitor R10015.