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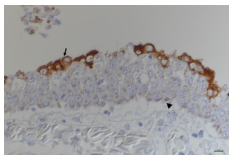
Articles of Significant Interest Selected from This Issue by the Editors

New Nidovirus Targets the Respiratory Tract of Pythons

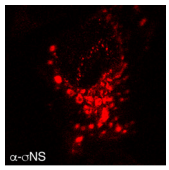
Nidoviruses have been identified in boid snakes and cultured in primary ophidian tissue cultures. Dervas et al. (e00718-17) isolated and identified Morelia viridis nidovirus (MVNV) using *in situ* hybridization and immunohistochemistry and determined full genome sequences. MVNV infection correlates with chronic proliferative pneumonia in the green tree python (*Morelia viridis*). Infected lungs exhibit severe hyperplasia of pulmonary epithelial cells with mucin production. These findings suggest that nidoviruses cause fatal pneumonia in pythons.



Viral antigen expression in pulmonary epithelia in proliferative pneumonia.

Stress Granule Modulation by Mammalian Orthoreovirus Factories

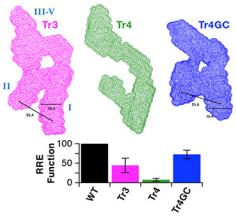
Stress granules (SGs) are part of the host innate immune response and disrupted by many viruses. Mammalian orthoreovirus (MRV) induces SGs but interferes with their formation as infection proceeds. Choudhury et al. (e01298-17) discovered that MRV disrupts canonical SGs by altering the localization of SG effector protein G3BP1 to the periphery of viral factories via association of nonstructural protein σNS with G3BP1 and factory matrix protein μNS. G3BP1 inhibition of MRV replication correlates with strain-specific host translational shutoff, suggesting that G3BP1 localization to viral factories is a strategy for overcoming immune-triggered translational shutoff.



G3BP1 accumulation at the periphery of viral factories in MRV-infected cells.

Functional Contributions of Individual Domains in the HIV-1 Rev Response Element

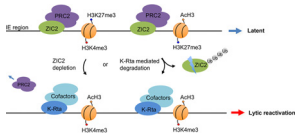
Retroviral replication requires that some viral RNAs be exported to the cytoplasm without being spliced. For HIV-1, Rev protein binds to the Rev response element (RRE), a highly structured element within unspliced viral RNA, and escorts this RNA from the nucleus. RRE architecture is "A" shaped, and the two legs opposite one another allow for the specificity of Rev for RRE. O'Carroll et al. (e00746-17) analyzed the functional contributions of individual RRE domains and found that several domains contribute to maintenance and stabilization of the overall RRE shape. Thus, the opposed placement of the two legs is essential for RRE function.



Mutations in Tr4GC stabilizing the truncated RRE stem rescue the A shape and RRE function.

Regulation of Latency-Lytic Switching by Kaposi's Sarcoma-Associated Herpesvirus

Key developmental genes in embryonic stem cells are modified by bivalent histone marks that enable the genomic region to rapidly respond to stimulation. These genomic domains are coordinated and maintained through recruitment of histone enzymes by specific transcriptional factors, such as ZIC2. Lyu et al. (e00980-17) discovered that ZIC2 occupies the immediate early (K-Rta) promoter region and maintains Kaposi's sarcoma-associated herpesvirus (KSHV) latency by tethering polycomb repressive complex 2 (PRC2). KSHV K-Rta targets ZIC2 via the ubiquitin-proteasome pathway and thus releases PRC2-mediated gene repression. This study illustrates a herpesvirus strategy to regulate the latency-lytic switch.

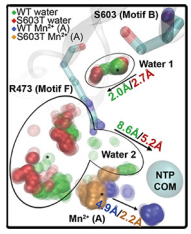


Proposed model of a regulatory loop between ZIC2-mediated repression and K-Rta-dependent activation of KSHV lytic replication.

https://doi.org/10.1128/JVI.01550-17

Escape Strategy of Tick-Borne Flavivirus from Nucleoside Antivirals

Specific therapies are not available to treat tick-borne encephalitis (TBE), a severe and potentially fatal viral infection in humans. Eyer et al. (e01028-17) report that a nucleoside analog, 7-deaza-2′-*C*-methyladenosine (7-deaza-2′-CMA), is highly effective in treating TBE in mice. However, TBE virus rapidly develops resistance to 7-deaza-2′-CMA and various other 2′-*C*-methylated nucleoside antivirals. The resistance is conferred by a single mutation that causes a subtle atomic effect within the active site of viral NS5 RNA-dependent RNA polymerase and is associated with strong attenuation of the virus.



TBE virus resistance to nucleoside antivirals is conferred by a single mutation (S603T) in the active site of the viral polymerase.