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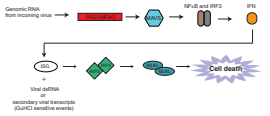
SPOTLIGHT

Articles of Significant Interest Selected from This Issue by the Editors

Reovirus Induces Necroptosis

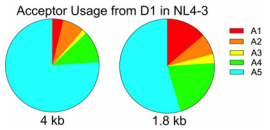
Death of host cells following mammalian reovirus infection is a determinant of viral disease. Berger et al. (e02404-16) report that infection of some cell types by reovirus results in cell death via RIP3 kinase-dependent necroptosis. Necroptosis following reovirus infection requires the detection of genomic RNA within incoming virions to produce type I inteferons. Furthermore, *de novo* synthesis of viral double-stranded RNA within infected cells also is required for the induction of necroptosis. In addition to describing a new pathway for reovirus-induced cell death, this work highlights a link between innate recognition of RNA viruses and necroptotic cell death.



Model for reovirus-induced necroptosis.

Efficient Deep-Sequencing Quantification of HIV-1 Splicing

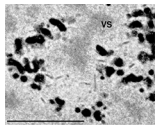
Alternate splicing of HIV-1 transcripts produces over 50 conserved RNAs that can be quantified using a new assay that combines transcript deep sequencing with Primer ID template indexing. Emery et al. (e02515-16) developed an assay to quantify temperature- and structure-dependent splicing changes and compare splicing among viral strains. Differences in the two transcript size classes (4 kb and 1.8 kb) suggest that Rev-dependent transcripts experience a different splicing environment than that for completely spliced transcripts. The relative ease of this three-primer assay paired with the capacity of next-generation sequencing provide a new tool for studying the regulation of the HIV-1 splicing program.



Splicing in 4-kb and 1.8-kb mRNAs.

New Insights into Baculovirus Nucleocapsid Assembly

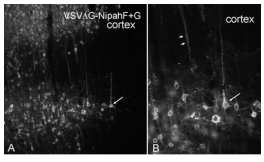
The baculovirus *vp39* gene encodes a major nucleocapsid protein, but many critical domains and residues of the VP39 protein product have not been identified. Katsuma and Kokusho (e02253-16) report the isolation of a *vp39* mutant and identify Gly-276 as a conserved residue essential to the function of VP39 in nucleocapsid assembly. This work also provides evidence for a link between nucleocapsid formation and transcription of the baculovirus *polyhedrin* and *p10* very late genes.



Large and aberrant nucleocapsid-like structures produced by a *vp39*-mutated baculovirus.

Chikungunya, Influenza, Nipah, and Semliki Forest Chimeric Viruses with Vesicular Stomatitis Virus: Actions in the Brain

Vesicular stomatitis virus (VSV) shows promise as a vaccine vehicle and an oncolytic agent. However, VSV-G glycoprotein targets neurons, which can generate adverse effects in the brain. van den Pol et al. (e02154-16) compare chimeric viruses expressing combinations of VSV genes together with genes from chikungunya, influenza, Nipah, or Semliki Forest viruses. A chimeric chikungunya virus-VSV appeared safe in the rodent brain. In contrast, Nipah F+G-VSV was more lethal than wild-type VSV. These results suggest that while chimeric VSVs show promise, each must be tested for neural injury.

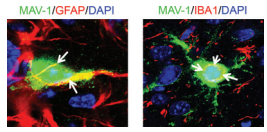


VSVΔG-NipahF+G infects neurons in brain.

https://doi.org/10.1128/JVI.00101-17

Increased Matrix Metalloproteinase Activity Associated with Mouse Adenovirus-Induced Encephalitis

Blood-brain barrier (BBB) disruption can accompany viral encephalitis, and matrix metalloproteinases (MMPs) degrade BBB components. Ashley et al. (e01412-16) discovered that mouse adenovirus type 1 (MAV-1) induces MMP activity in susceptible mouse brains. Astrocytes and microglia infected *ex vivo* secrete activated MMPs and may directly contribute to BBB breakdown. Conditioned medium from these cells also induces MMP activity from MAV-1-infected endothelial cells, suggesting an indirect mechanism of BBB disruption in encephalitic virus infections.



MAV-1 infects GFAP$^+$ astrocytes and IBA1$^+$ microglia.