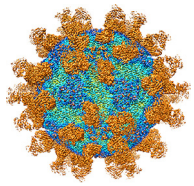
AMERICAN SOCIETY FOR MICROBIOLOGY | Journal of Virology®

Articles of Significant Interest in This Issue

High-Resolution Cryo-Electron Microscopy Reveals Insights into Parechovirus 3 Neutralization and Assembly

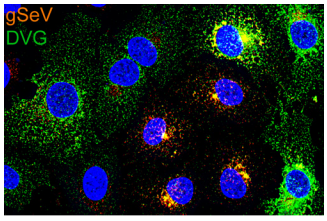
Human parechovirus 3 causes severe sepsis-like illness in neonates, and there are no antiviral treatments available. Domanska et al. (e01597-18) resolved a 2.8-Å structure of human parechovirus 3 in complex with antigen-binding fragments (Fabs) from a neutralizing antibody, which allows accurate modeling of virus-antibody interactions at the atomic level. In cultured human cells, antibody-bound virus is incapable of initial attachment. The structure provides new information about how the virion is organized, which may prove useful for developing strategies to combat human parechovirus 3 infections.



Cryo-EM structure of Fab-bound human parechovirus 3.

Defective Viral Genomes Drive Heterogeneity in Viral RNA Localization and Virion Production

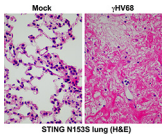
Defective viral genomes (DVGs) are formed during viral replication and modulate infection by inducing innate immunity and affecting viral replication. Using RNA fluorescence *in situ* hybridization, Genoyer and López (e01579-18) discovered heterogeneity in the accumulation and intracellular localization of defective and full-length Sendai virus genomes and the consequent differential production of standard and defective particles among infected cells. These findings highlight the functional dichotomy of cells as either producers and propagators of defective particles or inducers of antiviral immunity, depending on the content and intracellular localization of viral RNA.



Full-length Sendai virus genomes and DVGs accumulate in different intracellular locations during infection.

Gammaherpesvirus 68 Causes Pulmonary Fibrosis in STING Gain-of-Function Mice

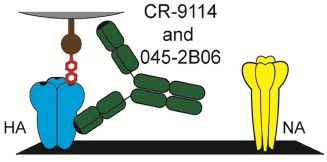
Autosomal dominant gain-of-function mutations in stimulator of interferon genes (STING) cause pulmonary fibrosis in humans. However, mice with the same mutations do not develop pulmonary fibrosis. Bennion et al. (e01806-18) found that intranasal inoculation of heterozygous STING N153S mice with gammaherpesvirus 68 (γHV68) causes severe pulmonary fibrosis and death. These results illustrate that a virus in conjunction with a human STING mutation causes pulmonary fibrosis and serves as an important example of a phenotype resulting from the interaction between a host gene and an environmental factor.



Histological sections of naive and infected lungs of STING N153S mice.

Hemagglutinin Stalk-Reactive Antibodies Interfere with Influenza Virus Neuraminidase Activity by Steric Hindrance

Hemagglutinin (HA) stalk-reactive antibodies are the basis of several universal influenza vaccine efforts. HA stalk antibodies inhibit HA stalk conformational rearrangements, blocking viral fusion and entry. Chen et al. (e01526-18) found that HA stalk-reactive antibodies also inhibit neuraminidase (NA) enzymatic activity, prohibiting viral egress by steric hindrance and thus providing an additional component of protection. These findings bring to light an additional mechanism of protection mediated by this broadly reactive and highly conserved class of antibodies.



Anti-hemagglutinin stalk-binding antibodies inhibit neuraminidase activity and viral egress.

https://doi.org/10.1128/JVI.02245-18
Published 5 February 2019