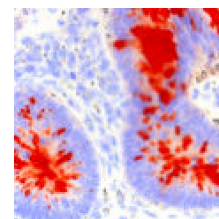




Articles of Significant Interest in This Issue

Guinea Fowl Coronavirus Diversity Has Phenotypic Consequences for Glycan and Tissue Binding

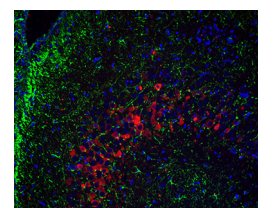
Guinea fowl coronavirus (GfCoV) causes fulminating enteritis in guinea fowl flocks. Bouwman et al. (e00067-19) studied the phenotypic consequences of GfCoV spike protein diversity and found that spike-receptor interactions determine tropism and pathogenicity. GfCoV/2014 spike has an increased affinity for these glycans compared to GfCoV/2011 spike, which is consistent with the increased avidity of GfCoV/2014 spike for gastrointestinal tract tissues. These differences in affinity for glycan receptors and altered avidity for intestinal tract tissues suggest consequences for tissue tropism and pathogenesis of GfCoV in guinea fowls.



Binding of GfCoV S1 protein to guinea fowl colon using spike histochemistry.

Interferon Beta Contributes to Astrocyte Activation during Reovirus Encephalitis

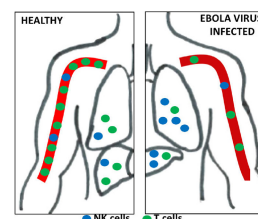
The role of neuroinflammation in the pathogenesis of viral encephalitis is not well understood. Clarke et al. (e02027-18) discovered that reovirus infection of the brain results in astrogliosis with increased expression of glial fibrillary acidic protein (GFAP) and upregulation of genes associated with activated astrocytes. Reovirus does not infect astrocytes. Instead, interferon beta, likely produced by reovirus-infected neurons, contributes to astrocyte activation. Activated astrocytes in reovirus-infected brains later die by Bak-mediated apoptosis. These findings shed light on neuroinflammatory responses during viral encephalitis and may facilitate the development of new treatment options.



Astrogliosis in the brain (hippocampus) following reovirus infection (GFAP, green; reovirus, red; Hoechst, blue).

NK Cells Accumulate in Infected Tissues and Contribute to Ebola Virus Disease Progression

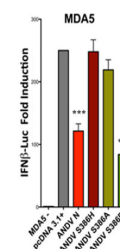
During lethal Ebola virus (EBOV) infection, there are reduced levels of several immune cells, including natural killer (NK) cells, in the blood of infected individuals. It is thought that this decrease results from massive cell death. Fausther-Bovendo et al. (e01703-18) discovered that NK cells relocate to infected tissues rather than undergo cell death. Relocated NK cells contribute to EBOV pathogenicity. These data shed light on the properties and fates of tissue-resident immune cells and their blood counterparts during viral infection.



NK cells accumulate in Ebola virus-infected tissues.

Phosphorylation of the Andes Virus Nucleocapsid Protein Regulates Interferon Responses

Andes virus (ANDV) is the only hantavirus known to be transmitted person to person, and it causes highly lethal disease in immunocompetent Syrian hamsters. ANDV expresses a nucleocapsid protein that inhibits interferon-signaling pathways. Simons et al. (e00338-19) found that interferon regulation is conferred by the phosphorylation of S386, a serine residue specific to the ANDV nucleocapsid protein. The ANDV nucleocapsid protein is a phosphoprotein, and S386 is an ANDV-specific, interferon-regulating virulence determinant. These findings suggest targets for ANDV attenuation and potential therapeutic approaches for ameliorating ANDV disease outcomes.



ANDV N:S386 and phosphomimetic D386 inhibit interferon induction.

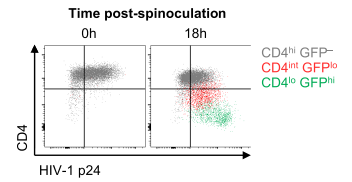
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Killing a Moving Target

HIV-specific antibodies capable of antibody-dependent cellular cytotoxicity (ADCC) could kill early-stage infected cells in the process of downregulating CD4, providing a mechanism for ADCC antibodies to prevent and control HIV infection. Lee et al. (e01901-18) discovered that soluble envelope glycoprotein (Env) and virions from the viral inoculum sensitize uninfected cells to ADCC prior to *de novo* Env expression and allow ADCC against early-stage infected cells. These results provide a new mechanism for antibody-mediated protection against HIV.



HIV virions can be detected on the surface of uninfected cells prior to *de novo* Env expression.