Articles of Significant Interest in This Issue

Linking Herpes Simplex Virus 1 MicroRNAs to Reactivation

During herpes simplex virus 1 (HSV-1) latency, viral gene expression is limited to select noncoding RNAs (ncRNAs), including microRNAs (miRNAs). Barrozo et al. (e00639-20) found that deletion of seed sequences corresponding to the antisense viral miRNAs miR-H1/H6 results in decreased reactivation from explanted murine dorsal root ganglia and latently infected rabbits. The antisense nature of these miRNAs, as well as their differential effects on expression of other ncRNAs in the latent region of the HSV-1 genome, shed light on the complex interactions that regulate the balance between latency and reactivation.

Deletion of HSV-1 miR-H1/H6 miRNAs severely impairs reactivation.

Phosphomimetic Recombinant Rotavirus Shows Early Interaction between Virus Protein vNSP2 and Cellular Lipid Droplets

Lipid droplets (LDs) are critical for replication of many pathogens. Rotavirus replication factories (viroplasm) composed of vNSP2 and NSP5 associate with LDs. Criglar et al. (e00972-20) used reverse genetics to generate a recombinant NSP2 S313D phosphomimetic rotavirus that exhibits slower viroplasm formation, allowing analysis of early assembly events. vNSP2 S313D colocalized with LDs without NSP5, and subsequent viroplasm development correlated with delayed NSP5 hyperphosphorylation. These findings suggest that NSP2 may be the viral protein that induces LD biogenesis and NSP5 hyperphosphorylation. Understanding the mechanism of NSP2-mediated viroplasm/LD biogenesis and assembly will lead to new insights into this host-pathogen interaction.

Electron microscopy of cells infected with recombinant wild-type rotavirus (left) compared to rNSP2 S313D rotavirus (right) shows that the single NSP2 mutation results in loosely assembled, less-dense viroplasms.

First Isolation of a Novel Aquatic Flavivirus from Chinook Salmon and Its *In Vivo* Replication in a Piscine Animal Model

The flaviviruses include important pathogens of humans and terrestrial animals. Soto et al. (e00337-20) reported the first isolation of a fish flavivirus, the salmon flavivirus (SFV), from the brains of moribund Chinook salmon (*Oncorhynchus tshawytscha*). SFV assumes a basal phylogenetic position among a new subgroup of recently discovered aquatic and bat flaviviruses, distinct from established flavivirus groups. Animal challenge experiments demonstrated *in vivo* replication of SFV in the brain; the virus remained infectious up to 15 days postinjection, fulfilling Rivers' postulates. As a prototype aquatic flavivirus, the discovery of SFV warrants future research on flavivirus evolution, pathogenicity, and disease potential in aquatic animals.

Phylogeny of the salmon flavivirus.

Comparison of Severe Acute Respiratory Syndrome Coronavirus 2 Spike Protein Binding to ACE2 Receptors from Human, Pets, Farm Animals, and Putative Intermediate Hosts

Effective binding to the host receptor angiotensin-converting enzyme 2 (ACE2) is the first prerequisite for severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) to infect cells. Zhai et al. (e00831-20) compared ACE2 proteins from species known to support and not support SARS-CoV-2 infection to identify amino acids essential for binding. Twenty amino acids in ACE2 contact the viral spike protein, and up to seven can be replaced without disrupting ACE2 function as the receptor. These findings illuminate a reason that the species barrier is low for SARS-CoV-2 and indicate that monitoring should be strengthened to prevent generation of a new animal reservoir.

Amino acids in ACE2 important for binding to the spike protein of SARS-CoV-2.

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Two Prophage Genes Block DNA Entry of Superinfecting Bacteriophages

Prophages may “instruct” their bacterial hosts to exclude superinfecting phages, a feature that confers selective advantages to both the resident prophage and its host. *Pseudomonas aeruginosa* usually achieves exclusion by preventing adsorption by altering the superinfecting phage receptors in the cell envelope. Carballo-Ontiveros et al. (e00953-20) discovered that two B3-like prophage genes instruct the host to block DNA entry of an adsorbed phage. These findings highlight a novel mechanism for preventing superinfection.

Note Added after Publication

In the originally published version, the figure legends for the second and final paragraphs, pages 1 and 2, respectively, were switched. The legends now appear under the correct figures.

The prophage (Ps56) or a construct harboring genes 9 and 10 (pHERD30T/ORF5_10_9) prevents K⁺ efflux, a hallmark of DNA entry into the cell, after incubation with virulent phage PaMx53.