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SPOTLIGHT

Articles of Significant Interest Selected from This Issue by the Editors

Parvovirus B19 Genome Replicates in Nonpermissive Cells with Help from Adenovirus

Human parvovirus B19 (B19V) replicates only in permissive erythroid progenitor cells. Guan et al. (p. 9541–9553) now show that replication of the B19V genome can be achieved in nonpermissive 293 cells coinfected with adenovirus or transfected with the necessary adenovirus genes, resulting in production of progeny virus. A minimal replication origin of B19V that supports efficient hairpin-independent replication of B19V in permissive cells was identified. These data provide essential clues to the unique replication mechanism of B19V, in which specific factors or a unique cellular microenvironment may be required.

Phosphorylation of Flaviviruses Is Arthropod Specific

Most flaviviruses are transmitted by arthropods, and every flavivirus that has been studied to date undergoes serine/threonine phosphorylation of its essential replication protein NS5. Bhattacharya et al. (p. 9195–9205) have found that protein kinase G phosphorylates a residue critical for dengue virus replication. This phosphorylation event is likely common to all mosquito-borne, but not tick-borne, flaviviruses. This work suggests that acquisition of phosphorylation sites may be an important adaptation in viral evolution.

Coxsackievirus Persistence and Chronic Damage to the Central Nervous System

The lasting consequences of coxsackievirus infection in the neonatal central nervous system (CNS) have not been fully evaluated. Feuer et al. (p. 9356–9369) demonstrate that coxsackievirus B3 (CVB3) in neonatal mice establishes viral persistence characterized by a low-level, noncytolytic infection in the adult CNS of surviving animals. Persistent CVB3 infection and the associated chronic inflammatory response may contribute to the observed immunopathology in the CNS that is apparent in adulthood. Hence, the potential for long-term damage to the CNS following coxsackievirus infection in infants should be reconsidered.

Gene Expression Changes in Bovine Spongiform Encephalopathy Agent-Infected Cattle

The pathogenesis of bovine spongiform encephalopathy agent is still poorly understood. In a gene expression study using microarray technology, Tang et al. (p. 9464–9473) identify a large number of differentially regulated genes prior to the detection of prion infectivity in central nervous system tissues of orally infected cattle. Moreover, evidence suggests that it is possible to predict the infectious status of animals using expression profiles derived from this study.