IN BRIEF

VIRAL PATHOGENESIS

Travelling in clusters

Host-cell entry and exit are traditionally viewed as processes that viruses undergo as independent units. However, Chen *et al.* now describe a novel intercellular transport pathway, in which clusters of approximately 20 mature enterovirus particles, such as poliovirus, are packaged into phosphatidylserine (PS)-enriched vesicles and are transmitted in unison between cells in a non-lytic manner. Compared with free virus particles, transmission via vesicles led to a substantial increase in infection efficiency, even though in both cases cell entry was dependent on the receptor CD155. The increased infection efficiency might be due to interactions between the vesicles and host cell PS receptors, as vesicles in which PS was masked were not infectious. Aside from casting doubt on the paradigm that viruses enter and exit cells as independent units, these findings suggest that genetic cooperation might occur between individual enterovirus genomes.

ORIGINAL RESEARCH PAPER Chen, Y.-H. *et al.* Phosphatidylserine vesicles enable efficient en bloc transmission of enteroviruses. *Cell* **160**, 619–630 (2015)

BACTERIAL EVOLUTION

Bugs-to-bunny in a single-hit tropism shift

The genetic changes that enable pathogens to jump from one host species to another, which often results in epidemic disease, are poorly understood. To identify the molecular basis of host adaptation, Penadés and colleagues traced the evolutionary history of *Staphylococcus aureus* ST121, which switched its host preference from humans to rabbits more than 40 years ago. Using whole-genome sequencing of both present-day and historical human and rabbit isolates, the authors found that only a single nonsynonymous mutation in the core genome was required and sufficient for host switching. Interestingly, the mutated gene, *dltB*, had also acquired nonsynonymous mutations in seven other *S. aureus* strains that had switched host specificity from humans to rabbits, as well as in a soil-borne strain of *Bacillus amyloliquefaciens* that had adapted from a plant-root niche. This convergent evolution suggests that *dltB* has an important function in defining host tropism, but establishing the mechanism that links the gene to host specificity requires further study.

ORIGINAL RESEARCH PAPER Viana, D. *et al.* A single natural nucleotide mutation alters bacterial pathogen host tropism. *Nature Genet.* http://dx.doi.org/10.1038/ng.3219 (2015)

VIRAL INFECTION

Better protection against HIV

Adeno-associated virus (AAV) vectors that express HIV-1 broadly neutralizing antibodies (bNAbs) have had limited success owing to the large proportion of HIV-1 isolates that remain partially or wholly resistant to bNAbs. Gardner *et al.* now present a new approach in which bNAbs are replaced with a fusion protein that mimics the virus receptor CD4 and its co-receptor CCR5, which bind to the two most conserved epitopes in the HIV-1 Env protein, thereby facilitating host-cell entry. Compared with a CD4 mimic alone, the fusion protein resulted in a 20–200-fold increase in viral neutralization, and the targeting of only the most conserved epitopes resulted in the neutralization of a diverse panel of HIV-1, HIV-2 and SIV isolates. A 40-week trial of this new AAV vector in rhesus macaques demonstrated durable protection against SHIV with no adverse effects.

ORIGINAL RESEARCH PAPER Gardner, M. R. *et al.* AAV-expressed eCD4-Ig provides durable protection from multiple SHIV challenges. *Nature* http://dx.doi.org/10.1038/nature14264 (2015)