IN BRIEF

VIROLOGY

Efficient hepatitis C virus particle formation requires diacylglycerol acyltransferase-1

Herker, E. *et al. Nature Med.* 10 Oct 2010 (doi:10.1038/nm.2238)

Hepatitis C virus (HCV) uses the host triglyceride-synthesizing enzyme diacylglycerol *O*-acyltransferase 1 (DGAT1) to assemble its virions, according to a new study. DGAT1 resides in the endoplasmic reticulum and, following fatty acid uptake, localizes to lipid droplets, which have a role in the production of infectious HCV virions. Loss of DGAT1 activity through chemical inhibition or RNA interference suppressed the production of HCV particles in cell culture. This was not due to reduced production of lipid droplets, which could be compensated for by DGAT2. Instead, in the absence of DGAT1 the viral core protein was retained in the endoplasmic reticulum and could not associate with lipid droplets. Thus, HCV virion assembly requires trafficking of the viral core protein to lipid droplets, which is mediated by DGAT1.

BIOFILMS

Bacteria use type IV pili to walk upright and detach from surfaces

Gibiansky, M.L. *et al. Science* **330**, 197 (2010)

Many bacteria form structured multicellular communities, known as biofilms, that have roles in infection. *Pseudomonas aeruginosa* uses type IV pili (T4P) for the 'twitching' motility mode used in biofilms, but how they adapt their motility while they transition from planktonic to surface-associated states was unclear. In this study the authors analysed movies of flagellum-deficient bacteria (which rely on T4P for migration) and identified two T4P-driven surface-motility modes. Horizontally oriented bacteria crawled with high directional persistence, whereas vertically oriented bacteria walked diffusively and frequently changed direction, which could enable local exploration. The T4P were required for both types of motility, for detachment and motility following cell division, and for the maintenance of biofilm morphology. Indeed, T4P-deficient bacteria formed morphologically different biofilms (containing heterogeneous bacterial clusters) to those formed by T4P-competent bacteria.

PARASITOLOGY

Wolbachia stimulates immune gene expression and inhibits *Plasmodium* development in *Anopheles gambiae*

Kambris, Z. *et al. PLoS Pathog.* **6**, e1001143 (2010)

The *Wolbachia pipientis* strain wMelPop had been shown to upregulate immune genes in *Aedes aegypti* and to inhibit *Plasmodium gallinaceum* transmission. Homologues of these genes in *Anopheles gambiae* have roles in regulating *Plasmodium* spp. development, so the authors tested whether infection with *W. pipientis* wMelPop decreases *Plasmodium berghei* transmission. Because the parasite replicates in somatic tissues, female *A. gambiae* were transiently infected in these tissues with *W. pipientis* wMelPop; this upregulated the expression of six immune genes compared with expression in uninfected controls and in *Escherichia coli*-infected mosquitoes. Furthermore, *W. pipientis* wMelPop-infected *A. gambiae* had fewer *P. berghei* oocysts than controls. This was linked to immune gene upregulation, as knockdown of the immune gene *TEP1*, which has an effect on *Plasmodium* spp. development, led to more oocysts than in controls infected with *W. pipientis* wMelPop and double-stranded *lacZ* RNA. These findings could be used to develop new malaria control strategies.