IN BRIEF

MICROBIOME

Interkingdom suppression of virulence

To understand the bacterial–fungal interactions between *Pseudomonas aeruginosa* and *Candida albicans*, Lopez-Medina *et al.* used a mouse model of microbial gastrointestinal colonization and dissemination. They found that co-colonization with *C. albicans* inhibited the virulence of *P. aeruginosa*, although the fungus did not interfere with the levels of bacterial colonization. Notably, proteins secreted by *C. albicans* were sufficient to inhibit the bacterial production of pyochelin and pyoverdine, which are two siderophores that are essential for *P. aeruginosa* virulence. In agreement, oral supplementation of iron was sufficient to rescue bacterial virulence in mice co-colonized with *P. aeruginosa* and *C. albicans*. These data reveal that interkingdom interactions can modulate the virulence of polymicrobial infections.

ORIGINAL RESEARCH PAPER Lopez-Medina, E. *et al. Candida albicans* inhibits *Pseudomonas aeruginosa* virulence through suppression of pyochelin and pyoverdine biosynthesis. *PLoS Pathog.* **11**, e1005129 (2015)

VIRAL INFECTION

Growing our knowledge of HCV replication

The study of hepatitis C virus (HCV) has been hampered by the inability to grow viral clinical isolates in cell culture. Saeed *et al.* postulated that this was due to the lack of essential factors in cell lines, and transduced human hepatoma cells with a lentivirus-based human complementary DNA (cDNA) library to identify the missing factors. They found a gene product, SEC14L2, which was sufficient to enable replication of HCV replicons and HCV isolates from patient sera in cell culture. HCV replication is known to induce lipid peroxidation, which inhibits viral replication; SEC14L2 seems to counteract this inhibition by enhancing vitamin E-mediated protection against lipid peroxidation, therefore promoting viral replication. The ability to grow HCV isolates from patients in cell culture is expected to further our understanding of HCV biology.

ORIGINAL RESEARCH PAPER Saeed, M. *et al. SEC14L2* enables pan-genotype HCV replication in cell culture. *Nature* **524**, 471–475 (2015)

BACTERIAL PATHOGENESIS

Ironing out *Legionella* infection

Following infection, *Legionella pneumophila* is able to hijack several cellular processes in order to establish the *Legionella*-containing vacuole (LCV), which enables bacterial replication within the host cell. However, how the bacteria acquire essential nutrients, such as iron, within the LCV is unknown. Isaac *et al.* found that the MavN protein, which is secreted by the Dot/Icm type IV secretion system (T4SS), is required for intracellular growth of *L. pneumophila*. Interestingly, MavN integrates into the host LCV membrane, and growth of a bacterial mutant lacking MavN was rescued following iron supplementation. Furthermore, the authors identified a putative iron-binding motif in MavN, and mutation of this motif recapitulated the growth defects observed for the mutant lacking MavN. Collectively, these data suggest that MavN is a bacterial protein that inserts into the LCV membrane and facilitates iron transport into the vacuole, thereby promoting bacterial virulence.

ORIGINAL RESEARCH PAPER Isaac, D. T. *et al.* MavN is a *Legionella pneumophila* vacuole-associated protein required for efficient iron acquisition during intracellular growth. *Proc. Natl Acad. Sci. USA* http://dx.doi.org/10.1073/pnas.1511389112 (2015)

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