IN BRIEF

BACTERIAL PATHOGENESIS

More to CRISPR than adaptive immunity

A new study shows that the type II CRISPR–Cas (clustered, regularly interspaced short palindromic repeats–CRISPR-associated proteins) system of the intracellular pathogen *Francisella novicida* downregulates the expression of an immunostimulatory bacterial lipoprotein (BLP), which increases the integrity of the cell envelope, resulting in antibiotic resistance and inflammasome evasion. Sampson *et al.* found that a mutant that lacked the endonuclease Cas9 was resistant to the membrane-targeting antibiotic polymyxin B, and in combination with its partner RNAs (the tracrRNA and scaRNA), Cas9 directly enhanced envelope integrity *in vitro* and during intracellular infection, owing, at least in part, to reduced expression of BLP. Furthermore, evasion of the AIM2–ASC inflammasome (which is activated by Toll-like receptor 2 (TLR2)) was Cas9-dependent, and the reduced virulence of the *cas9*-null mutant was restored in the absence of both ASC and TLR2, which demonstrates the pivotal role of Cas9 in evading innate immunity. Further to its role in adaptive immunity, this study shows that CRISPR–Cas is an important determinant of gene expression and contributes to bacterial pathogenesis.

ORIGINAL RESEARCH PAPER Sampson, T. R. *et al.* A CRISPR–Cas system enhances envelope integrity mediating antibiotic resistance and inflammasome evasion. *Proc. Natl Acad. Sci. USA* **111**, 11163–11168 (2014)

VIRAL PATHOGENESIS

Influenza knows how to exploit its host

During budding from the plasma membrane, influenza A viruses (IAVs) incorporate host proteins, but the role of these proteins in the viral life cycle is poorly understood. Berri *et al.* now show that the membrane protein annexin V (A5), which is upregulated during IAV infection and is incorporated into viral particles, enables escape from immune detection. The authors found that A5 incorporation inhibited interferon-γ (IFNγ)-mediated STAT1 phosphorylation and the subsequent production of CXC-chemokine ligand 10 (CXCL10) in infected macrophages, whereas knockdown of A5 using a small interfering RNA restored IFNγ signalling and decreased viral replication in macrophages and in the lungs of mice. These findings demonstrate that IAV has evolved a strategy to exploit host proteins for the subversion of immunosurveillance.

ORIGINAL RESEARCH PAPER Berri, F. *et al.* Annexin V incorporated into influenza virus particles inhibits interferon-γ signaling and promotes viral replication. *J. Virol.* http://dx.doi.org/10.1128/JVI.01405-14 (2014)

MICROBIAL ECOLOGY

Turning up the heat on biofilms

Increased temperature as a result of climate change has a direct effect on the functional capacity of microbial communities, but community-wide responses are understudied. This study used tandem mass tag (TMT)-based proteomics to measure protein expression in acid mine drainage biofilms at 40 °C, 43 °C and 46 °C. The expression of proteins that are involved in amino acid metabolism increased substantially across the community at 46 °C, whereas proteins that are involved in carbon fixation showed differential responses, such that these proteins were repressed in two *Leptospirillum* spp., whereas their abundance increased in a third species. Thus, climate change is likely to have a considerable impact on the contribution of microbial communities to biogeochemical processes.

ORIGINAL RESEARCH PAPER Mosier, A. C. *et al.* Elevated temperature alters proteomic responses of individual organisms within a biofilm community. *ISME J.* http://dx.doi.org/10.1038/ismej.2014.113 (2014)