IN BRIEF

BACTERIAL PHYSIOLOGY

Buckling under stress

Bacteria can adapt in response to stressful environments. In this study, Phan et al. identify an emergent phenotype that enables *Escherichia coli* to escape near-lethal concentrations of antibiotics. When exposed to minimum inhibitory concentrations of cephalexin — a non-genotoxic antibiotic that impairs septum formation in bacteria — *E. coli* transforms into long helical filaments. The authors recorded swimming filaments using a high-speed resonant confocal microscope. Filaments were either right-handed or left-handed in conformation, but motile filaments were always right-handed. The helical bacteria were slightly past the critical torsional buckling point and had a twist angle of 2π radians, regardless of length. These cells do not swim by 'run and tumble' as observed in non-stressed *E. coli*, but rather synchronously flip their spin and velocity, causing them to reverse direction. The authors suggest that this motility increases the diffusivity of filamentous bacteria, enabling cells to more readily escape stress.

ORIGINAL ARTICLE Phan, T. V. et al. Emergence of *Escherichia coli* critically buckled motile helices under stress. *Proc. Natl Acad. Sci. USA* https://doi.org/10.1073/pnas.1809374115 (2018)

HOST RESPONSE

Chemical warfare against phages

The evolutionary arms race between prokaryotes and their viruses has resulted in the development of sophisticated anti-phage defence mechanisms, including restriction–modification and CRISPR–Cas systems. Now, Maxwell and colleagues report a new chemical anti-phage defence system that is widespread in *Streptomyces* spp. They show that *Streptomyces* spp. produce secondary metabolites (daunorubicin and doxorubicin) that intercalate into phage DNA and block phage replication, whereas other DNA-intercalating agents did not elicit this effect. Furthermore, these molecules did not affect bacterial growth. Daunorubicin was found to act very early in the phage life cycle, after DNA ejection but before DNA replication. This suggests that these molecules act at a stage that is unique to phage DNA replication, such as genome circularization. The authors also provide data that are consistent with a model in which metabolites can diffuse into bacteria and protect them from infection.

ORIGINAL ARTICLE Kronheim, S. et al. A chemical defence against phage infection. *Nature* https://doi.org/10.1038/s41586-018-0767-x (2018)

BACTERIAL PATHOGENESIS

Dissolving immune cell membranes

Quorum-sensing bacteria produce and release molecules that regulate community behaviours. Now, Song et al. report that a quorum-sensing molecule of *Pseudomonas aeruginosa* induces host immune cell death by causing cell surface lipid domain dissolution. They found that *N*-(3-oxododecanoyl) homoserine lactone (3OC12 HSL) — an autoinducer of the bacterial LasI–LasR circuitry — incorporates into mammalian plasma membranes and causes lipid domain disruption. This disruption subsequently leads to the spontaneous trimerization of tumour necrosis factor receptor 1 within the membrane, which drives caspase 3–caspase 8-mediated apoptosis. *P. aeruginosa* released 3OC12 HSL in mouse lung infection experiments, which induced neutrophil apoptosis and supressed host immunity, promoting the survival of *P. aeruginosa*. Remarkably, a caspase inhibitor diminished the severity of *P. aeruginosa* infection in mice.

ORIGINAL ARTICLE Song, D. et al. *Pseudomonas aeruginosa* quorum-sensing metabolite induces host immune cell death through cell surface lipid domain dissolution. *Nat. Microbiol.* https://doi.org/10.1038/s41564-018-0290-8 (2018)

IMMUNE EVASION

Changing your sugar coat

Methicillin-resistant *Staphylococcus aureus* (MRSA) is a leading cause of life-threatening infections associated with the failure of antibiotic therapy, and there is an ongoing need for the development of new treatment strategies. The cell envelope of Gram-positive bacteria such as *S. aureus* contains wall teichoic acid (WTA), which has an important role in pathogenicity and antibiotic resistance. Human antibodies are often directed against WTA and can confer protection against *S. aureus*; however, antibody titres and specificities vary greatly among humans, and the underlying mechanisms of this variability remained elusive. So far, no vaccination approaches, including WTA antigens, have been successful. In this study, Gerlach et al. report a mechanism whereby MRSA subverts antibody-mediated immunity by modifying its dominant WTA antigen.

Nascent WTA chains are composed of repeat units of ribitol phosphate (RboP) and are modified by the β-glycosyltransferase TarS, which attaches *N*-acetylglucosamine (GlcNAc) residues to *S. aureus* WTA. The authors hypothesized that *S. aureus* modifies WTA to promote immune evasion and pathogenicity. They screened *S. aureus* genomes for paralogues of WTA biosynthesis genes and identified TarP, which has 27% identity to TarS. Interestingly, TarP is exclusively encoded by prophages in health-care-associated and livestock-associated MRSA clones. Expression of *tarP* in a glycosylation-deficient strain restored WTA glycosylation as well as resistance to β-lactam antibiotics. Moreover, nuclear magnetic resonance analyses revealed that although TarS and TarP add GlcNAc to WTA in the β-configuration, TarS adds GlcNAc to the C4 position in RboP whereas TarP adds GlcNAc

VIRAL INFECTION

A rainbow of influenza virions

Influenza A virus (IAV) particles vary considerably in size, shape and composition, even among genetically identical virions. However, studying the consequences of this phenotypic variability has been hindered owing to technical limitations in labelling IAV proteins without disrupting their function. Now, Vahey and Fletcher labelled the most abundant proteins within virions with small tags and fluorophores, which enabled the quantification of IAV particle variability and its consequences.

Whereas the morphology of some other viruses is uniform, IAV is known to produce virions that range from small elliptical particles of a few hundred nanometres to filaments of up to several micrometres in length. In addition, not all particles contain all viral components, and the ratio of different components can differ substantially. To quantify these differences, the authors constructed IAV variants in which combinations of structural proteins were labelled, including one with labelled haemagglutinin (HA), neuraminidase (NA) and nucleoprotein (NP). They inserted tags of fewer than 12 amino acids into the proteins, which could then be visualized with small-molecule fluorophores. Importantly, the labelling minimally affected infectivity and replication kinetics, and the average numbers of HA and NA molecules per virion were similar to those previously determined by other methods.

Interestingly, the abundance of HA and NA varied by approximately