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IN BRIEF

BACTERIAL PATHOGENESIS

Yersinia RNA feels the heat

The foodborne enteropathogen *Yersinia pseudotuberculosis* uses transcriptional and post-transcriptional thermoregulation to respond to the increase in temperature that occurs during infection of warm-blooded hosts. Previously, one *Y. pseudotuberculosis* transcript had been shown to be thermoregulated by the presence of an 'RNA thermometer' (RNAT) upstream of the coding region. RNATs regulate translation by altering the accessibility of the ribosome-binding site to the ribosome, based on temperature-dependent changes to the RNA structure. Righetti *et al.* took advantage of recent methodological advances to structurally probe the *Y. pseudotuberculosis* transcriptome at environmental (25 °C), host (37 °C) and heat-shock (42 °C) temperatures. Twenty candidate RNATs were validated using a heat-stable reporter protein, and these included RNATs that regulated genes that are involved in oxidative stress or virulence, including the adhesin gene *ailA*.

ORIGINAL ARTICLE Righetti, F. *et al.* Temperature-responsive *in vitro* RNA structurome of *Yersinia pseudotuberculosis*. *Proc. Natl Acad. Sci. USA* http://dx.doi.org/10.1073/pnas.1523004113 (2016)

BACTERIAL PHYSIOLOGY

Cell stiffness beyond the wall

Auer *et al.* developed the high-throughput 'GRABS' assay to study cell stiffness. Based on the concept that agarose provides mechanical resistance to cell elongation, GRABS measures the growth, and thus cell stiffness, of agarose-embedded cells in 96-well microplates. In a GRABS screen of 3,844 *Escherichia coli* deletion mutants, the 46 strongest hits — several of which were validated using a microfluidic bending assay — included not only genes with functions that are related to the cell envelope, as expected, but also 37 genes with other functions, including energy production and conversion; DNA replication, recombination and repair; and amino-acid transport and metabolism. The diversity of these functions suggests that cell stiffness is an emergent property that depends on processes beyond those that maintain the cell envelope.

ORIGINAL ARTICLE Auer, G. K. *et al.* Mechanical genomics identifies diverse modulators of bacterial cell stiffness. *Cell Syst.* http://dx.doi.org/10.1016/j.cels.2016.05.006 (2016)

MICROBIAL ECOLOGY

Broad-spectrum anti-CRISPRs

Anti-CRISPR proteins inhibit CRISPR–Cas systems through various mechanisms. Each anti-CRISPR protein examined to date is associated with *Pseudomonas aeruginosa* and is encoded by an operon that also encodes a homologue of Aca1 or Aca2, which are putative transcriptional regulators. Pawluk *et al.* used a guilt-by-association approach to identify candidate anti-CRISPR genes located in proximity to *aca1* and *aca2* homologues. Five new families of anti-CRISPR proteins were identified, all of which were able to inhibit type I-F CRISPR–Cas systems in *P. aeruginosa*; however, the genes that encode these anti-CRISPR proteins were not restricted to *Pseudomonas* spp. but instead were widely distributed throughout the Proteobacteria. Thus, these anti-CRISPR proteins were able to promiscuously inhibit CRISPR–Cas systems from species other than their natural host. Finally, one anti-CRISPR protein had acquired a carboxy-terminal region that enabled the protein to inhibit type I-E CRISPR–Cas systems in addition to type I-F CRISPR–Cas systems.

ORIGINAL ARTICLE Pawluk, A. *et al.* Inactivation of CRISPR–Cas systems by anti-CRISPR proteins in diverse bacterial species. *Nat. Microbiol.* http://dx.doi.org/10.1038/nmicrobiol.2016.85 (2016)