IN BRIEF

CELLULAR MICROBIOLOGY

Acetyl phosphate revealed as a global acetyl donor

Lysine acetylation is a common post-translational modification in bacteria, but the mechanisms regulating this process and its link to metabolism are poorly understood. Weinert *et al.* now show that growth arrest results in a global, low-level increase in acetylation in *Escherichia coli* owing to the accumulation of acetyl phosphate (AcP), an intermediary metabolite of glycolysis. This effect was shown to be independent of YfiQ, the only known acetyltransferase in *E. coli*, suggesting that AcP donates its acetyl group in a non-enzymatic manner. Indeed, AcP was shown to chemically acetylate lysine residues *in vitro*, and AcP levels correlated with acetylation levels *in vivo*. Furthermore, the indiscriminate acetylation pattern that was observed is inconsistent with site-specific mechanisms that are typical of enzyme-catalysed reactions. However, future work is needed to rule out the potential involvement of an unknown acetyltransferase and to understand the functional consequences of such widespread acetylation.

ORIGINAL RESEARCH PAPER Weinert, B. T. *et al.* Acetyl-phosphate is a critical determinant of lysine acetylation in *E. coli*. *Mol. Cell* **51**, 265–272 (2013)

FUNGAL PHYSIOLOGY

Converting to commensalism

Candida albicans can exist in the mammalian gut as either a commensal or an invasive pathogen. Pande *et al.* show that passage of wild-type *C. albicans* through the mouse gastrointestinal tract elicits expression of the transcription factor Wor1, which promotes commensalism. Overexpression of Wor1 enhanced fitness, whereas cells lacking the encoding gene were rapidly depleted. Wor1 was shown to trigger a heritable developmental switch that converted wild-type *C. albicans* from a white phenotype to a morphologically distinct dark phenotype (termed the gastrointestinally induced transition (GUT)). Transcriptomics also revealed that the GUT cells were functionally specialized for the metabolism of local nutrients in the digestive tract. These data highlight the fact that microorganisms can use distinct developmental programmes to switch between pathogenic and commensal states.

ORIGINAL RESEARCH PAPER Pande, K. *et al.* Passage through the mammalian gut triggers a phenotypic switch that promotes *Candida albicans* commensalism. *Nature Genet.* http://dx.doi.org/10.1038/ng.2710 (2013)

BIOFILMS

Flagella function as mechanosensors

To begin building a biofilm, motile cells must adhere to a surface structure. However, the mechanisms by which cells sense and respond to surfaces are unclear. In *Bacillus subtilis*, several signalling pathways control biofilm formation, including the two-component system DegS–DegU. This study reveals that inhibition of flagellar rotation, which is likely to occur when a motile cell encounters a surface, activates DegS–DegU signalling. Mutation or deletion of the flagellar stator gene *motB* (which is required for flagellar rotation), as well as inhibition of flagellar rotation by tethering with antibodies or by inducing the flagellar clutch, EpsE, induced several cellular processes driven by phosphorylated DegU, including synthesis of the biofilm exoplymer poly-γ-DL-glutamic acid. These findings suggest that the arrest of flagellar rotation acts as a mechanosensory signal to facilitate the transition from a motile to a sessile state.

ORIGINAL RESEARCH PAPER Cairns, L. S. *et al.* A mechanical signal transmitted by the flagellum controls signalling in *Bacillus subtilis*. *Mol. Microbiol.* http://dx.doi.org/10.1111/mmi.12342 (2013)