IN BRIEF

FUNGAL PHYSIOLOGY

A phospho-switch for pathogenesis

The opportunistic human fungal pathogen *Candida albicans* undergoes a morphological switch from the commensal budding yeast form to the pathogenic filamentous pseudohyphal and hyphal forms. Transcription factor-driven changes in gene expression are associated with this fungal polymorphism, ensuring the expression of genes required for pathogenesis. Greig *et al.* show that upon hyphal induction, the Cdc28 and Cbk1 kinases mediate changes in the phosphorylation status of the fork-head family transcription factor Fkh2 before it reverts back to the characteristic phosphorylation profile of the yeast form. Notably, although Fkh2 is known to be involved in cell-cycle regulation, this shift was independent of the cell cycle and promoted the expression of genes involved in tissue invasion, pathogenesis and biofilm formation. These data suggest a new function for Fkh2 in addition to its conserved housekeeping role, by which cell-cycle-independent Fkh2 phosphorylation drives the pathogenic switch in *C. albicans*.

ORIGINAL RESEARCH PAPER Greig, J. A. *et al.* Cell cycle-independent phospho-regulation of Fkh2 during hyphal growth regulates *Candida albicans* pathogenesis. *PLoS Path.* http://dx.doi.org/10.1371/journal.ppat.1004630 (2015)

BACTERIAL PATHOGENESIS

Crossing the barriers

Following infection, *Listeria monocytogenes* disseminates by crossing host barriers; binding of the *L. monocytogenes* surface protein InlA to host E-cadherin is sufficient for crossing the intestinal barrier, whereas interactions between InlA–E-cadherin as well as InlB and the host receptor cMet are required for crossing the placental barrier. Although both processes require PI3K activity in the host cell, the underlying mechanism for the differential contribution of InlA and InlB remained elusive. Previously, it was shown that InlB, but not InlA, triggers PI3K activation; now, this study shows that intestinal cells targeted by *L. monocytogenes* exhibit constitutive PI3K activity, thus rendering InlB dispensable for InlA-dependent crossing of this barrier. By contrast, PI3K activity was undetectable in uninfected placental cells, suggesting that InlB enables InlA-mediated invasion of placental cells by stimulating PI3K.

ORIGINAL RESEARCH PAPER Gessain, G. *et al.* PI3-kinase activation is critical for host barrier permissiveness to *Listeria monocytogenes*. *J. Exp. Med.* http://dx.doi.org/10.1084/jem.20141406 (2015)

SYMBIOSIS

Metabolites in differentiation

The cyanobacterium *Nostoc punctiforme* can grow in a free-living state as multicellular vegetative filaments or as a plant symbiont. Symbiosis requires differentiation into infectious motile filaments termed hormogonia and can be stimulated by environmental conditions and by plant-derived hormogonium-inducing factors and is repressed by an unknown *N. punctiforme* hormogonium-repressing factor (HRF). Liaimer *et al.* analysed the effect of *N. punctiforme* secondary metabolites in the differentiation process and identified the nonribosomal peptide nostopeptolide as a HRF. Nostopeptolide was constitutively expressed in the free-living state and downregulated during plant symbiosis, and its addition was sufficient to repress hormogonia formation, demonstrating a role for this secondary metabolite during cell differentiation.

ORIGINAL RESEARCH PAPER Liaimer, A. *et al.* Nostopeptolide plays a governing role during cellular differentiation of the symbiotic cyanobacterium *Nostoc punctiforme*. *Proc. Natl Acad. Sci. USA* http://dx.doi.org/10.1073/pnas.1419543112 (2015)