IN BRIEF

FUNGAL IMMUNE EVASION

An LHC1 shield for *Cryptococcus*

Polysaccharide capsules surround many fungal pathogens and contribute to virulence by inhibiting complement-mediated phagocytosis. By purifying polysaccharide capsule-associated proteins, Park *et al.* found a new *Cryptococcus neoformans* enzyme — the lactonohydrolase LHC1 — and showed that it is involved in the higher-order assembly of this structure. Mutant strains that lack LHC1 have a larger capsule than wild-type strains and have altered polysaccharide branching; the larger capsule is more permeable to anti-capsular antibodies and more susceptible to opsonization by both mouse and human complement. These alterations led to increased phagocytosis of fungi that lacked LHC1, which correlated with decreased virulence of the mutant strains in mice. These results establish LHC1 as a new *C. neoformans* virulence factor that functions by introducing modifications in capsular structure that prevent fungal detection by the immune system.

ORIGINAL RESEARCH PAPER Park, Y.-D. *et al.* A role for *LHC1* in higher order structure and complement binding of the *Cryptococcus neoformans* capsule. *PLoS Pathog.* http://dx.doi.org/10.1371/journal.ppat.1004037 (2014)

STRUCTURAL BIOLOGY

Switching on *Plasmodium* SUB1

As part of its life cycle, the malarial parasite *Plasmodium falciparum* replicates in host erythrocytes, and daughter merozoites are released via a highly regulated and calcium-dependent process. The serine protease SUB1 is discharged shortly before egress to cleave parasite proteins that are required for erythrocyte exit and invasion. Withers-Martinez *et al.* now solve the crystal structure at 2.25 Å of the enzymatically active core domain of *P. falciparum* SUB1 partially bound to its prododomain and complexed with a monoclonal antibody Fab fragment. The structure reveals the architecture of the substrate cleavage site and also identifies a labile, redox-sensitive disulphide bridge near the active site that regulates SUB1 activity. Thus, although not the native protein, this structure provides mechanistic insights that could be useful for the design of new antimalarial drugs.

ORIGINAL RESEARCH PAPER Withers-Martinez, C. *et al.* The malaria parasite egress protease SUB1 is a calcium-dependent redox switch subtilisin. *Nature Commun.* http://dx.doi.org/10.1038/ncomms4726 (2014)

BACTERIAL TRANSCRIPTION

A consensus pausing sequence

Transcriptional pausing by RNA polymerase has diverse gene regulatory roles; however, the determinants and distribution of these pauses were not well established. The authors of this study sequenced nascent elongating transcripts (NETs) and identified 20,000 new pause sites in known *Escherichia coli* genes. They defined a 16-nucleotide consensus sequence that is conserved across several bacterial lineages. Investigating the minimal requirements for pausing, the authors found that interactions of RNA polymerase with the DNA template and the transcript that inhibit nucleotide addition were sufficient for pausing *in vivo*. Finally, the conserved sequence is enriched at translational start sites in both *E. coli* and *Bacillus subtilis*, which might cause transcriptional pausing to facilitate RNA folding and ribosome access.

ORIGINAL RESEARCH PAPER Larson, M. H. *et al.* A pause sequence enriched at translation start sites drives transcription dynamics *in vivo*. *Science* http://dx.doi.org/10.1126/science.1251871 (2014)