IN BRIEF

HOST RESPONSE

Killer histones

Histones (the main protein components of chromatin in eukaryotes) have been shown to be bactericidal *in vitro*, so it had been proposed that they have a physiological role in antimicrobial defences, in particular against intracellular pathogens. This study identifies lipid droplet-bound histones as a new antibacterial defence system. Histones were released from lipid droplets (purified from *Drosophila melanogaster* embryos) in the presence of bacterial cell envelope components such as lipopolysaccharide (LPS) and lipoteichoic acid, and displayed bactericidal activity *in vitro*. Importantly, such bactericidal activity was also observed *in vivo*: *D. melanogaster* embryos lacking histone deposits on lipid droplets showed reduced survival compared with wild-type embryos when injected with either Gram-negative (*Escherichia coli* str. DH5a) or Gram-positive (*Staphylococcus epidermidis*) bacteria. Moreover, the levels of lipid droplet-bound histones in mice increased following LPS injection, suggesting that this antibacterial defence system might be conserved.

ORIGINAL RESEARCH PAPER Anand, P. *et al.* A novel role for lipid droplets in the organismal antibacterial response. *eLIFE* 13 Nov 2012 (doi:10.7554/eLife.00003)

BACTERIAL PHYSIOLOGY

Pass the (LPS) parcel

In *Escherichia coli*, lipopolysaccharide (LPS) is synthesized at the inner membrane and transported to the outer membrane for assembly. This is known to be mediated by LPS transport (Lpt) proteins: LptBFG forms a complex with the inner-membrane-bound LptC, which binds to LptA, and LptA interacts with LptD at the outer membrane, forming a trans-envelope bridge that connects the two membranes. Here, the authors elucidate the individual steps of LPS transport across the Lpt bridge. They found that LPS is extracted from the inner membrane by LptBFG, which then transports it to LptC; LPS is then transferred to LptA. Importantly, both transport events required ATP, so the authors propose that multiple rounds of ATP hydrolysis provide energy to push LPS from the inner to the outer membrane through the trans-envelope bridge.

ORIGINAL RESEARCH PAPER Okuda, A., Freinkman, E. & Kahne, D. Cytoplasmic ATP hydrolysis powers transport of lipopolysaccharide across the periplasm in *E. coli*. *Science* 8 Nov 2012 (doi:10.1126/science.1228984)

PARASITE PHYSIOLOGY

Moving like *Toxoplasma*

Motility, invasion of host cells and egress from host cells are mediated by calcium signalling in apicomplexan parasites. Here, the authors investigated the role of *Toxoplasma gondii* kinases in host cell invasion and egress. They observed that calcium-dependent protein kinase 1 (CDPK1) was required during invasion, consistent with previous findings, whereas CDPK3 was dispensable. By contrast, both enzymes were necessary for parasite egress. This distinction was also apparent when examining microneme secretion, a process required for apicomplexan motility; CDPK1 was necessary at all times, whereas inhibition of CDPK3 affected microneme secretion only under certain conditions. Further analysis revealed that cyclic GMP protein kinase (PKG) could partially compensate for the loss of CDPK3 during egress under certain conditions. Thus, these three kinases all seem to contribute to apicomplexan motility by regulating invasion and egress.

ORIGINAL RESEARCH PAPER Lourido, S., Tang, K. & Sibley, D. L. Distinct signalling pathways control *Toxoplasma* egress and host-cell invasion. *EMBO J.* 13 Nov 2012 (doi:10.1038/emboj.2012.299)