IN BRIEF

PARASITE BIOLOGY

Divide and conquer

Different mechanisms have been proposed to explain how *Toxoplasma gondii* crosses the blood–brain barrier and infects the brain, including parasite transmigration through endothelial tight junctions and the 'Trojan horse' mechanism, by which infected monocytes carry parasites into the brain. Now, using intravital microscopy to image fluorescence-labelled parasites in transgenic mice that express endothelial cells labelled with GFP, Konradt *et al.* show that free parasites in the vascular compartment can infect brain endothelial cells. Furthermore, by comparing non-replicating *T. gondii* strains with replicating strains, the authors found that replication within endothelial cells and lysis of these cells was necessary for parasite entry into the central nervous system.

ORIGINAL ARTICLE Konradt, C. *et al.* Endothelial cells are a replicative niche for entry of *Toxoplasma gondii* to the central nervous system. *Nat. Microbiol.* **1**, 16001 (2016)

BACTERIAL PHYSIOLOGY

A new way to die

The envelope of Gram-negative bacteria includes an inner membrane (IM), a peptidoglycan cell wall in the periplasm and an outer membrane (OM). Furthermore, the OM is asymmetric, with phospholipids in the inner leaflet and lipopolysaccharide (LPS) in the outer leaflet. Now, Sutterlin, Shi *et al.* show that a gain-of-function mutation in MlaA, a lipoprotein that is thought to prevent phospholipid accumulation in the outer leaflet of the OM, disrupts the balanced synthesis of the cell envelope and induces cell lysis by a novel mechanism. The *mlaA** mutation increased the level of LPS in the outer leaflet of the OM; this altered the permeability of the OM and induced vesiculation and blebbing at division sites, which resulted in the loss of lipids from the OM. As the rigid peptidoglycan cell wall prevents cell shrinkage, lipids lost from the OM must be replaced, which occurs through using lipids from the IM; this causes the IM to shrink, increasing the density of the cytoplasm, which leads to mechanical rupture of the IM and slow leakage of the cytoplasmic components, culminating in cell death.

ORIGINAL ARTICLE Sutterlin, H. A., Shi, H. *et al.* Disruption of lipid homeostasis in the Gram-negative cell envelope activates a novel cell death pathway. *Proc. Natl Acad. Sci. USA* http://dx.doi.org/10.1073/pnas.1601375113 (2016)

HOST RESPONSE

Amoebae cast a wider net

Innate immune cells, particularly neutrophils, are known to produce DNA-based extracellular nets that can trap and kill extracellular pathogens. Now, Zhang, Zhuchenko *et al.* show that the social amoeba *Dictyostelium discoideum* can also produce nets, either following stimulation with lipopolysaccharide (LPS) or in response to *Klebsiella pneumoniae*. Under starvation conditions, individual amoebae form a migrating multicellular slug community that includes phagocytic sentinel cells, and only sentinel cells were capable of producing extracellular nets. Furthermore, by deleting genes that have been previously associated with the phagocytic capacity of sentinel cells, the authors could demonstrate that the production of extracellular nets requires the Toll/interleukin-1 receptor domain-containing protein TirA and the generation of reactive oxygen species. These data increase the arsenal of defence systems used by *D. discoideum* and suggest an ancient origin for extracellular nets.

ORIGINAL ARTICLE Zhang, X., Zhuchenko, O. *et al.* Social amoebae trap and kill bacteria by casting DNA nets. *Nat. Commun.* **7**, 10938 (2016)