Credit: Old Visuals/Alamy Stock Photo

Only when the models included pioneer bacteria, that is, a subpopulation of cells that moved further than the average cells, irregular foci arose. Importantly, the simulations matched the experimental foci best when pioneer rates were relatively low, which translated to 1.4–12% of the bacterial population being or descending from pioneers.

The mechanisms underlying 'pioneering' motility are unclear; however, when the authors used a mutant *L. monocytogenes* strain that forms shorter actin comets and is less likely to form and enter long protrusions, the observed foci were more circular than the foci produced by the wild-type strain. This suggests that the pioneers use long protrusions to spread to cells that are not directly adjacent. Such behaviour might help sustain infection in the gut, as the epithelium, and with it the intracellular bacteria, is continuously shed at the tip of the villi. Maintaining infection in such an environment requires a fine balance between vigorous growth, which potentially overwhelms the host, and local growth, which risks elimination. Indeed, if the models took into account shedding, bacteria were more likely to persist in a steady state if pioneers were present.

Ursula Hofer

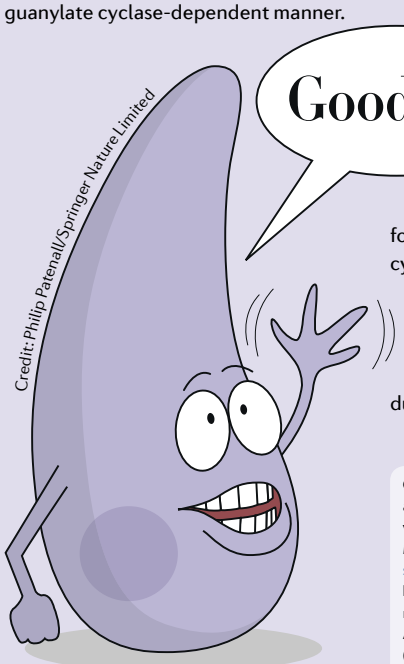
ORIGINAL ARTICLE Ortega, F. E., Koslover, E. F. & Theriot, J. A. *Listeria monocytogenes* cell-to-cell spread in epithelia is heterogeneous and dominated by rare pioneer bacteria. *eLife* **8**, e40032 (2019)
FURTHER READING Radoshevich, L. & Cossart, P. *Listeria monocytogenes*: towards a complete picture of its physiology and pathogenesis. *Nat. Rev. Microbiol.* **16**, 32–46 (2018)

messenger cGMP) that harbours a P4-ATPase phospholipid flippase domain. The authors found that this receptor is essential for microneme secretion and that ePA stimulates microneme secretion and egress in a guanylate cyclase-dependent manner. Moreover, the flippase activity was shown to be essential for guanylate cyclase activation, which suggests that ePA activates a signalling cascade that ultimately leads to egress.

Through co-immunopreciptation and mass spectrometry experiments, the authors also identified two associated cofactors — cell division control 50.1 and unique GC organizer — that are required for the assembly of the guanylate cyclase at the plasma membrane.

Altogether, these observations led to the proposal of a new model of *T. gondii* natural egress that relies on an intrinsic signal generated during infection.

Ashley York



Credit: Philip Patenall/Springer Nature Limited

ORIGINAL ARTICLE Bisio, H. et al. Phosphatidic acid governs natural egress in *Toxoplasma gondii* via a guanylate cyclase receptor platform. *Nat. Microbiol.* https://doi.org/10.1038/s41564-018-0339-8 (2019)
FURTHER READING Frénal, K. et al. Gliding motility powers invasion and egress in Apicomplexa. *Nat. Rev. Microbiol.* **16**, 645–660 (2017)

IN BRIEF

EPIDEMIOLOGY

A ghost from the past

Zika virus (ZIKV) and dengue virus (DENV) co-circulate in endemic regions and are genetically and antigenically similar, but whether pre-existing immunity to DENV promotes or supresses ZIKV emergence is unknown. A recent study finds that pre-existing immunity to DENV is associated with a reduction in ZIKV transmission. The authors prospectively followed a cohort of 1,453 individuals living in Salvador, Brazil — the epicentre of the 2015 outbreak in the Americas. By performing multiple serological assays that distinguish between ZIKV and DENV infections on samples collected before and after the outbreak, they found that ~73% of the cohort was infected with ZIKV during the outbreak. In individuals with immunity to DENV before the outbreak and high antibody titres, there was a reduction in the risk of ZIKV infection and symptoms. Together, these findings support the hypothesis that prior DENV infection had a role in driving ZIKV to local extinction by reducing transmission.

ORIGINAL ARTICLE Rodriguez-Barraquer, I. et al. Impact of preexisting dengue immunity on Zika virus emergence in a dengue endemic region. *Science* **363**, 607–610 (2019)

BACTERIAL PATHOGENESIS

Cut the fat

Lipases (lipid hydrolysing enzymes) that are secreted by bacteria enable the acquisition of host-derived fatty acids for bacterial growth and infection. This study found that the glycerol ester hydrolase (Geh) secreted by the opportunistic pathogen *Staphylococcus aureus* promotes immune evasion by cleaving *S. aureus* lipoproteins, which are major pathogen-associated molecular patterns (PAMPs) that activate innate immune responses. The Geh lipase prevented the activation of cultured innate immune cells and in mice, a *geh* mutant increased the proinflammatory cytokine response, promoted innate immune activity and accelerated clearance from infected tissues compared with wild-type. Geh was found to cleave microbial Toll-like receptor 2 ligands, which the authors propose masks the immune response and contributes to *S. aureus* persistence.

ORIGINAL ARTICLE Chen, X. & Alonzo, F. III. Bacterial lipolysis of immune-activating ligands promotes evasion of innate defenses. *Proc. Natl Acad. Sci. USA* https://doi.org/10.1073/pnas.1817248116 (2019)

VIRAL INFECTION

At the flick of a switch

Many RNA viruses cause both acute and persistent infections, but little is known of the mechanisms that underlie persistence. Using the parainfluenza virus type 5 (PIV5) as a model for paramyxoviruses, Young et al. found that the phosphorylation status of the P protein of the viral RNA polymerase complex determines whether viral transcription and replication become repressed at late times during infection. If repression occurs, a persistent infection is established, which fluxes between active and repressed states, and if not, the infected cell dies. The switch from acute to persistent infection was affected by single amino acid substitutions that prevent phosphorylation of the P protein. Viruses with a mutation at a specific phosphorylation site replicated to higher titres in mice, caused greater immune responses, but were cleared more quickly. The authors propose that during acute infection, lytic variants are selected, but later, the immune response selects for variants that promote persistence.

ORIGINAL ARTICLE Young, D. F. et al. The switch between acute and persistent paramyxovirus infection caused by single amino acid substitutions in the RNA polymerase P subunit. *PLOS Pathog.* https://doi.org/10.1371/journal.ppat.1007561 (2019)