Credit: Loop Images Ltd/Alamy Stock Photo

Desulfosporosinus infrequens'. This sulfate-reducer is widespread in freshwater wetlands, although it only occurs at low abundance. The authors incubated anoxic peat microcosms and found constant but low levels of this bacterium during 50 days of incubation. However, minor additions of nutrients such as acetate led to an increase of the bacterium's ribosome content by up to one order of magnitude and an upregulation of the sulfate reduction pathway. However, genes involved in cell division and growth were not upregulated, and this finding, coupled with the stable abundance, suggests that no growth occurred. Instead, the bacterium upregulated stress responses, likely to deal with the acidic pH of the peat.

In summary, the two studies show that no or slow-growing bacteria are not simply biding their time in an inactive state; rather, they invest the scarce available resources in survival and maintenance, which likely puts them in an optimal position to resume growth in more favourable conditions.

Ursula Hofer

ORIGINAL ARTICLES Gray, D. A. et al. Extreme slow growth as alternative strategy to survive deep starvation in bacteria. *Nat. Commun.* **10**, 890 (2019) | Hausmann, B. et al. Long-term transcriptional activity at zero growth of a cosmopolitan rare biosphere member. *mBio* https://doi.org/10.1128/mBio.02189-18 (2019)
FURTHER READING Bergkessel, M., Basta, D. & Newman, D. K. The physiology of growth arrest: uniting molecular and environmental microbiology. *Nat. Rev. Microbiol.* **14**, 549–562 (2016)

M. globosa, the authors investigated the inflammatory activities of *M. restricta*. Treating pathogen-free mice with *M. restricta* exacerbated dextran sulfate sodium (DSS)-induced colitis, as judged by shortening of the colon, worsening of disease activity and increased intestinal inflammation. *M. restricta* appears to exacerbate disease without altering the gut microbiota as, in germ-free mice colonized with eight specific bacterial species, it did so without altering the relative level of any of the bacteria. As exposure of human or mouse dendritic cells to dead *M. restricta* evoked a stronger inflammatory response per organism than exposure to control fungi, *M. restricta* may directly activate immune responses in the gut.

Interestingly, the presence of *Malassezia* spp. in the gut of patients with Crohn's disease was strongly linked to the presence of *CARD9*^{S12N}. To better understand the inflammatory activity of *M. restricta*, the authors stimulated mouse dendritic cells lacking CARD9 or dectin 2 (an innate immune receptor that signals via CARD9) with *M. restricta*. The absence of CARD9 or dectin 2 reduced the level of inflammatory cytokines (namely TNF and IL-6) secreted. Furthermore, when stimulated with *M. restricta*, human cells homozygous for N at position 12 of CARD9 produced more TNF and IL-8 than cells homozygous for S at position 12 of CARD9. These data suggest that *M. restricta* exacerbates colitis by stimulating an innate immune response via *CARD9*^{S12N}. Indeed, *M. restricta* exacerbated DSS-induced colitis in wild-type mice but not in CARD9-null mice.

In sum, an innate immune response to changes in the intestinal mycobiome may contribute to the pathogenesis of Crohn's disease.

Katharine H. Wrighton

ORIGINAL ARTICLE Limon, J. J. et al. *Malassezia* is associated with Crohn's disease and exacerbates colitis in mouse models. *Cell Host Microbe* **25**, 1–12 (2019)
FURTHER READING Nilsson, R. H. et al. Mycobiome diversity: high-throughput sequencing and identification of fungi. *Nat. Rev. Microbiol.* **17**, 95–109 (2019)

IN BRIEF

VIRAL INFECTION

Take your coat off

Hepatitis A virus can be released from cells as quasi-enveloped virions (eHAVs); that is, enclosed in host membranes that lack virus-encoded surface proteins. Naked HAV virions have a role in faecal–oral transmission between individuals, whereas eHAVs mediate cell-to-cell spread within the infected hosts. How these two virion types enter cells was not well understood. Lemon and colleagues report that both naked virions and eHAVs enter cells through clathrin-dependent and dynamin-dependent endocytosis, and the entry process is mediated by integrin β1. They went on to show that both virion types are trafficked through the endosomal compartments and that uncoating of naked virions occurs in late endosomes. By contrast, eHAVs are trafficked to the lysosome, where the quasi envelope is degraded and the genome is uncoated. In sum, naked and quasi-enveloped virions enter cells via similar endocytic pathways, but uncoating of HAV and eHAV capsids is temporally and spatially different.

ORIGINAL ARTICLE Rivera-Serrano, E. E. et al. Cellular entry and uncoating of naked and quasi-enveloped human hepatoviruses. *eLife* https://doi.org/10.7554/eLife.43983 (2019)

PARASITE BIOLOGY

Blocking pathogen transmission

Tsetse flies transmit pathogenic African trypanosomes, which are the causative agents of African trypanosomiases. Current disease control strategies are aimed at controlling the size of tsetse populations, and novel strategies under development focus on reducing the ability of the flies to transmit trypanosomes. Weiss et al. stably colonized the gut of tsetse flies with the bacterium *Kosakonia cowanii* Zambiae (*Kco_Z*), which was isolated from *Anopheles gambiae* and has been shown to confer resistance against malaria parasites to the mosquitoes. In the presence of *Kco_Z* in the gut, tsetse flies were more refractory to infection with parasitic African trypanosomes compared with flies that did not harbour the bacterium. Next, the authors showed that *Kco_Z* acidifies the tsetse midgut, which inhibits trypanosome growth and establishment of infection. Finally, colonization of tsetse with *Kco_Z* conferred only a modest fitness cost to the fly, and thus this strategy could be used to reduce disease transmission.

ORIGINAL ARTICLE Weiss, B. L. et al. Colonization of the tsetse fly midgut with commensal *Kosakonia cowanii* Zambiae inhibits trypanosome infection establishment. *PLOS Pathog.* https://doi.org/10.1371/journal.ppat.1007470 (2019)

FUNGAL PATHOGENESIS

Defence and counter defence

Plant cells were previously shown to produce an extracellular matrix comprising DNA, protein and polysaccharides that, like animal neutrophil extracellular traps (NETS), captures and kills pathogens. In this study, Park et al. show that a plant pathogen uses extracellular DNases (exDNases) as a defence mechanism against host-secreted extracellular DNA in plant-produced NETs. The authors deleted a candidate secreted DNase-encoding gene in the maize pathogen *Cochliobolus heterostrophus* and showed that the fungal mutant exhibits reduced virulence on plant leaves and plant roots compared with the wild type. The phenotype was rescued by the addition of exogenous DNase I, and the authors confirmed the DNase activity of the enzyme in fungal culture filtrates, suggesting that the DNase is secreted. Last, secretion and activity of the DNase in the culture medium was induced in the presence of plant tissue. The authors hypothesize that the pathogen deploys the enzyme to degrade plant-secreted DNA.

ORIGINAL ARTICLE Park, H.-J. et al. A DNase from a fungal phytopathogen is a virulence factor likely deployed as counter defense against host-secreted extracellular DNA. *mBio* **10**, e02805–18 (2019)