IN BRIEF

MICROBIAL ECOLOGY

Colonization-induced host-gut microbial metabolic interaction

Claus, S. P. *et al. mBio* **2**, e00271-10 (2011)

To examine the phenomenon that gut microbiota can affect systemic metabolism at several distant bodily organs, Claus *et al.* probed the systemic metabolic changes that occur in response to gut colonization in germ-free mice over a period of 20 days using NMR spectroscopy to monitor metabolic profiles. Colonization resulted in a rapid increase in body weight over the first 5 days, with corresponding changes in the metabolic profiles of the urine, plasma, liver, kidney and colon. In particular, there was an increase in glycogenesis in the liver followed by an increase in hepatic triglyceride synthesis. Together with statistical modelling, 16S ribosomal RNA gene pyrosequencing of the gut microbiota during colonization revealed a strong association between colonization by members of the family Coriobacteriaceae and levels of triglyceride, glucose and glycogen in the liver. These data suggest that individual constituents of the gut microbiota can have profound effects on host metabolism.

EVOLUTION

Second-order selection for evolvability in a large *Escherichia coli* population

Woods, R. J. *et al. Science* **331**, 1433–1436 (2011)

Being optimally adapted to the current conditions does not ensure long-term survival, according to a new study. Woods *et al.* used samples from a decades-long *Escherichia coli* evolution experiment to show that the clones that will ultimately dominate the population prevail because of their potential for adaption, or their 'evolvability'. After only 500 generations, clones with two beneficial mutations that would eventually become fixed in the population (evolutionary winners) performed less well in competitive fitness assays than clones that lacked these mutations and which ultimately did not survive (evolutionary losers). The dominance of the evolutionary winners was reproducible and, over time, these winners showed a greater potential for further adaptation than the evolutionary losers, which fail owing to detrimental epistatic interactions that reduce the benefit of mutations.

BACTERIAL TOXINS

A novel mechanism of programmed cell death in bacteria by toxin-antitoxin systems corrupts peptidoglycan synthesis

Mutschler, H. *et al. PLoS Biol.* **9**, e1001033 (2011)

PezT is a kinase that forms part of the PezAT ε–ζ toxin–antitoxin system. Meinhart and colleagues have now found that PezT phosphorylates uridine diphosphate-*N*-acetylglucosamine (UNAG), which is a precursor of peptidoglycan. They isolated a mutant of the toxin that allows the cells to survive its presence for long enough to establish that the toxin kills the cell by inhibiting cell wall synthesis. The toxin is also active in eukaryotic cells, so the authors focused on UNAG as the potential target molecule, as it is present in eukaryotes and bacteria. Spectrometry and NMR confirmed that the toxin can add a phosphate group to the *N*-acetylglucosamine residue of UNAG, and *in vitro* experiments showed that UNAG that is phosphorylated by PezT inhibits MurA (which adds phospho-enolpyruvate to UNAG in one of the first steps of peptidoglycan synthesis), thereby inhibiting cell wall synthesis.