

## IN BRIEF

## MICROBIOME

## Sharing the work

The microbial gut communities of social bees are dominated by five bacterial groups. Bees feed on pollen, which contains diverse polysaccharides. Moran and colleagues analysed the genomes of bacterial isolates from honey bees and bumble bees and used transcriptomic and metabolomic experiments to investigate the ability of individual members of the gut microbiota to digest polysaccharides. They identified *Bifidobacterium* and *Gilliamella* as the main degraders of hemicellulose and pectin, whereas other gut bacterial species cannot degrade polysaccharides. Moreover, the ability to digest pollen varied between different bacterial strains as well as within and between host bee species. Thus, strain composition within hosts may determine their metabolic capabilities. The authors suggest that core bacteria of the bee microbiota occupy distinct metabolic niches to promote efficient substrate metabolism and their co-existence.

**ORIGINAL ARTICLE** Zheng, H. et al. Division of labor in honey bee gut microbiota for plant polysaccharide digestion. *Proc. Natl Acad. Sci. USA* <https://doi.org/10.1073/pnas.1916224116> (2019)

## VIRAL INFECTION

## Hiding from defence systems in a shell

To efficiently replicate, phages must avoid bacterial defence systems that target nucleic acids. Two studies report that a nucleus-like structure protects jumbo phages against DNA-targeting immune pathways. Bondy-Denomy and colleagues showed that jumbo phage  $\phi$ KZ, which infects *Pseudomonas aeruginosa*, is resistant to CRISPR systems that are found in its natural host (types I-C and I-F), to those that are not naturally present in *P. aeruginosa* species (types II-A and V-A) and to restriction endonucleases (types I and II). Phage  $\phi$ KZ assembles a proteinaceous compartment in which phage DNA replication occurs, which led the authors to hypothesize that this structure provides a physical protective barrier against DNA-targeting enzymes. Indeed, Cas proteins and restriction enzymes were excluded from the structure, whereas targeting the type II restriction enzyme EcoRI inside the compartment decreased phage titres and protected host cells. Finally, the authors showed that phage  $\phi$ KZ is susceptible to Cas13a, which targets RNA, possibly as mRNA is exported into the cytoplasm for translation. Fineran and colleagues report that a *Serratia* jumbo phage, PCH45, which is distinct from other described jumbo phages such as *Pseudomonas* phages, evades type I CRISPR–Cas systems independently of DNA modification or anti-CRISPR. By contrast, the type III-A system provided phage resistance by targeting phage RNA in the cytoplasm. As the *Serratia* jumbo phage encodes a tubulin homologue and a potential shell protein, the authors also investigated the possibility that the phages produce a protective nucleus-like compartment. They showed that shell proteins assembled into a spherical structure enclosing the phage DNA upon infection and that native CRISPR–Cas complexes in *Serratia* (types I-E, I-F and III-A) were excluded from this compartment. In sum, the studies suggest that the formation of a nucleus-like structure in the bacterial cytoplasm may be a widespread defence strategy against DNA-targeting immune pathways among jumbo phages.

**ORIGINAL ARTICLES** Mendoza, S. D. et al. A bacteriophage nucleus-like compartment shields DNA from CRISPR nucleases. *Nature* <https://doi.org/10.1038/s41586-019-1786-y> (2019) | Malone, L. M. et al. A jumbo phage that forms a nucleus-like structure evades CRISPR–Cas DNA targeting but is vulnerable to type III RNA-based immunity. *Nat. Microbiol.* <https://doi.org/10.1038/s41564-019-0612-5> (2019)

## MARINE MICROBIOLOGY

## New fungal relative hiding in diatoms

Microbial eukaryotes are exceedingly diverse and often poorly defined, despite their important ecological roles, for example, in environmental nutrient cycling. One such enigmatic group is the Opisthokonta, which are related to fungi and only patchily characterized, although they have a few well-studied members such as the microsporidia. A new study now characterizes the life cycle of one member of this group, the novel chytrid-like-clade-1 (NCLC1), and shows that they infect marine diatoms.

Although NCLC1 sequences had been detected previously, the phylogenetic position of these organisms was controversial. Chambouvet et al. now confirm with additional small subunit (SSU) ribosomal RNA (rRNA) sequences that the NCLC1 lineage belongs to the Opisthokonta, and that it

is related to other fungus-like marine microorganisms in a branch close to the base of the fungal radiation.

Not much had been known about the life cycle of NCLC1, except that NCLC1-like sequences are abundant in samples from some European coastal waters. To identify NCLC1 cells, the authors developed fluorescent in situ hybridization (FISH) probes that are specific for NCLC1 rRNA regions. In samples from a Norwegian fjord they found several different NCLC1 cells, including free-living single cells, multi-nucleate aggregates and cells in association with diatom hosts. NCLC1 associated with several different diatom taxa, specifically, also with the most abundant diatom groups in the samples. Interestingly, the authors found NCLC1 inside diatom cells without a nucleus, suggesting that NCLC1 infected

## BACTERIAL SECRETION

## Poking holes in your competitor

The type VI secretion system (T6SS) of Gram-negative bacteria has a crucial function in bacterial competition: upon contact with neighbouring competitors, the T6SS releases toxic effector proteins into target cells to kill or inhibit the recipient bacterial cell. To avoid self-intoxication, donor cells express a cognate immunity protein, which directly binds to the effector to inactivate it. Although numerous effector proteins have been described, the identity and mechanism of action of many effector proteins remain unknown. In this study, Mariano et al. characterized a new family of antibacterial effectors that are delivered by the T6SS and mediate their toxic effect by depolarization of the inner membrane in target cells.

Previous studies have described an effector termed Ssp6 in the

opportunistic pathogen *Serratia marcescens*, but its mode of action was not determined. The authors first confirmed that Ssp6 is secreted in a T6SS-dependent manner and then went on to identify the cognate immunity protein, Sip6, which was able to neutralize the toxicity of Ssp6. Co-immunoprecipitation assays suggested that the inhibitory effect of the immunity protein is conferred by direct Sip6–Ssp6 interactions rather than by protection or modification of the Ssp6 target.

But what is the mode of action of Ssp6? The authors showed that the effector does not cause cell lysis and instead inhibits bacterial growth. They hypothesized that the membrane was the target of Ssp6. Indeed, Ssp6 intoxication led to depolarization of the inner