IN BRIEF

VIRAL INFECTION

Too big to be ignored

Phages with genomes >200 kb and >500 kb are referred to as jumbophages and megaphages, respectively, and are rarely isolated using conventional methods. Al-Shayeb, Sachdeva et al. performed metagenomic sequencing of DNA isolated from diverse ecosystems (for example, humans, animals, oceans, sediments, soils, deep subsurface habitats and the built environment) and found hundreds of phage genomes >200 kb in length, including a 735 kb genome, which the authors claim is the largest phage genome reported to date. The phylogeny of the phages was constructed, and ten 'huge phage' clades were defined, which are distributed across a broad bacterial host range. Thirty five of the huge phage genomes were manually curated, and large inventories of genes were found, including novel CRISPR–Cas systems and genes involved in translation, which the authors suggest may intercept host translation to redirect protein synthesis towards viral replication.

ORIGINAL ARTICLE Al-Shayeb, B., Sachdeva, R. et al. Clades of huge phages from across Earth's ecosystems. *Nature* https://doi.org/10.1038/s41586-020-2007-4 (2020)

MICROBIOME

Passing on pesticide resistance

After ingestion, the gut microbiota can metabolize xenobiotics, and xenobiotic exposures can lead to changes in the microbiota. Wang et al. exposed the model wasp species *Nasonia vitripennis* to subtoxic levels of atrazine, a widely used pesticide, and observed changes in the structure and function of the gut microbiome that confer multigenerational host resistance to the pesticide. The authors performed multi-omics analyses on laboratory populations of *N. vitripennis* and observed changes in dominant members of the microbiota that were also inherited by subsequent, unexposed generations. Two members of the gut microbiota — *Pseudomonas protegens* and *Serratia marcescens* — became enriched after atrazine exposure, were found to metabolize atrazine and were sufficient to confer resistance in wasps. As *Nasonia* species maternally inherit their microbiota, this study suggests an inability to return to an ancestral-like microbiome pre-atrazine exposure.

ORIGINAL ARTICLE Wang, G.-H. et al. Changes in microbiome confer multigenerational host resistance after sub-toxic pesticide exposure. *Cell Host Microbe* **27**, 213–224 (2020)

SYMBIOSIS

Viscosity regulates a competitive strategy

Symbiotic bacteria use numerous strategies to compete for their host niches, but how intraspecific competition is regulated by environmental cues during host colonization was unknown. Speare et al. found that liquid viscosity regulates type VI secretion system (T6SS)-mediated killing in *Vibrio fischeri* during habitat transition. Using a liquid hydrogel medium that mimics the high viscosity of the mucus-rich host light organ of the Hawaiian bobtail squid *Euprymna scolopes*, the authors found that exposure to high viscosity increases T6SS expression and sheath formation in vitro and activates T6SS-mediated killing after only 30 minutes. High viscosity was also found to promote co-aggregation of different genotypes that naturally compete for the same niche, thereby facilitating the cell–cell contacts that are required for interbacterial competition.

ORIGINAL ARTICLE Speare, L. et al. Environmental viscosity modulates interbacterial killing during habitat transition. *mBio* **11**, e03060–19 (2020)

MICROBIOME

Protecting bee health

Honey bees (*Apis mellifera*) are threatened by synergistic interactions between the parasitic mite *Varroa destructor* and RNA viruses, such as deformed wing virus (DWV), for which it is a vector. In an effort towards combating this threat, Leonard et al. have engineered *Snodgrassella alvi* wkB2 (a symbiont in the honey bee gut microbiota) to express double-stranded RNA (dsRNA) in honey bees that can reduce the expression of bee genes, enhance bee survival in response to DWV infection and kill *V. destructor*.

After establishing that engineered *S. alvi* could colonize bee guts, the authors inoculated bees with *S. alvi* expressing plasmid-encoded dsRNA against green fluorescent protein (GFP; pDS-GFP), noting that dissected bees expressed GFP RNA in the head, gut and haemolymph. In these bees, the expression of genes implicated in the bee immune response and of *dicer* (the RNA interference (RNAi) gene) was upregulated compared with bees colonized with *S. alvi* expressing empty plasmid (pNR). Thus, *S. alvi* can produce dsRNA, and trigger an immune response, in bees.

The authors next colonized bees with *S. alvi* expressing dsRNA against the insulin receptor gene *InR1* (pDS-InR1). pDS-InR1 reduced *InR1* expression across body regions, increased the sensitivity of bees to low sucrose concentrations and increased the weight of bees (likely owing to increased feeding) compared with pDS-GFP-expressing bees. Thus, *S. alvi*-produced dsRNA can silence bee genes to influence bee behaviour. Importantly, unlike the injection of dsRNA into bees, which only transiently reduces gene expression, pDS-InR1 reduced *InR1* expression levels for at least 15 days.

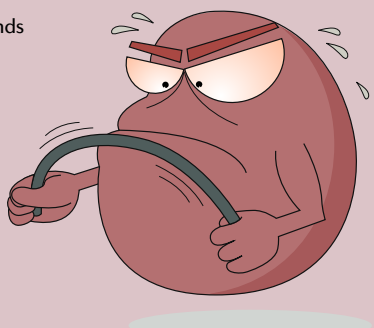
BACTERIAL PATHOGENESIS

Bend out of shape

Chlamydia pneumoniae are obligate intracellular bacteria that cause respiratory infections in humans. The pathogen has evolved several mechanisms to modulate host cell processes to facilitate entry. The most common entry strategies are the zipper mechanism, whereby bacteria hijack the endocytic pathway through adhesin–receptor interactions, and the trigger mechanism, whereby entry is facilitated by the release of bacterial effectors. It was previously shown that a *C. pneumoniae* membrane protein binds to epidermal growth factor receptor (EGFR) and activates a cell signalling cascade that leads to the endocytic uptake of the pathogen. Moreover, it was also shown that *C. pneumoniae* secrete the CPn0572 effector via its type III secretion system (T3SS), which induces actin polymerization and promotes its uptake.

In this study, Hänsch, Spona et al. report that secretion of an early effector induces curvature of the host plasma membrane, which is sensed by a key regulator of endocytosis that is subsequently recruited to promote internalization of *C. pneumoniae* elementary bodies (which are the infectious particles).

First, the authors identified a new *C. pneumoniae* effector, CPn0678 (SemC from hereafter), that is expressed during the early phase of infection and is secreted via the T3SS.



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