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IN BRIEF

MICROBIOME

Spore formation in the human gut microbiota

Spore formation by the human gut pathogen *Clostridium difficile*, a strict anaerobe, provides protection from oxygen during transmission between hosts. However, whether anaerobic bacteria of the commensal gut microbiota can also form spores for transmission is not known. Browne *et al.* developed a method to archive pure cultures of bacterial species of the human gut microbiota, including many species that were previously thought to be unculturable. By combining the method with selection for ethanol-resistant cultures, 66 candidate spore-forming bacterial species were isolated. Genes that were conserved among these species were also present in *C. difficile*, consistent with a shared genetic basis of spore formation and germination. The prevalence of these conserved genes in metagenomic datasets suggests that 60% of the bacterial genera present in the human gut have genes for spore formation and that many commensal anaerobic species may therefore form spores for transmission between hosts.

ORIGINAL ARTICLE Browne, H. P. *et al.* Culturing of 'unculturable' human microbiota reveals novel taxa and extensive sporulation. *Nature* http://dx.doi.org/10.1038/nature17645 (2016)

MICROBIOME

Autophagy genes link OMVs to IBD

Inflammatory bowel disease (IBD), which is associated with gut microbial dysbiosis, has been linked to variants of several human genes with functions in autophagy, such as *ATG16L1* and *NOD2*. How these genes should affect host–microbiota interactions is unclear. One possibility is that immunomodulation by commensal bacteria, such as by polysaccharide A (PSA) in outer membrane vesicles (OMVs) produced by *Bacteroides fragilis*, may involve autophagy. Chu *et al.* now show that the mouse homologues of *ATG16L1* and *NOD2* are required for an anti-inflammatory response to *B. fragilis* PSA–OMVs that is mediated by a non-canonical autophagy pathway. Furthermore, mouse cells that expressed an *ATG16L1* variant associated with IBD in humans were defective at inducing this response, as were cells from human donors that are homozygous for the risk allele.

ORIGINAL ARTICLE Chu, H. *et al.* Gene-microbiota interactions contribute to the pathogenesis of inflammatory bowel disease. *Science* http://dx.doi.org/10.1126/science.aad9948 (2016)

MICROBIOME

Why you're stuck with the skin you're in

Segre and colleagues report a two-year longitudinal survey of the bacteria, fungi and viruses that make up the human skin microbiome, using samples taken at three time points from 17 body sites of 12 healthy individuals. Metagenomic sequencing revealed a remarkable stability of the microbial community at each site for each individual over time, which contrasts with the variability seen between sites and individuals. Dry sites, such as palms, and sweat glands tended to have more stable communities than moist sites, such as feet. Stability also varied between individuals, with 4 of the 12 individuals exhibiting greater instability across all sites than the remainder of the cohort. Single-nucleotide variations were used to track individual strains of *Propionibacterium acnes* and *Staphylococcus epidermidis*, which suggested that stability arises from the maintenance of strains after colonization rather than repeated colonization by specific favoured species.

ORIGINAL ARTICLE Oh, J. *et al.* Temporal stability of the human skin microbiome. *Cell* **165**, 854–866 (2016)