Credit: Ikon Images / Alamy Stock Photo

and the neutralization fingerprint remained in a similar range.

Although this association between virus genetics and neutralization was highly statistically significant, the average effect size was moderate, similar to the effect size of virus genetics on $CD4^+$ T cell loss. HIV-1 is a hugely variable virus and some viral variants might be much stronger 'imprinters'. Remarkably, the authors found one transmission pair of so-called elite neutralizers; that is, the pair had neutralization levels in the top 1% of the original cohort in which the transmission pairs were identified. The authors confirmed that this pair had developed a broadly neutralizing response by testing 42 different HIV-1 strains and they determined that the probability of finding a pair with such a strong and similar bnAb response by chance is low at 0.017.

In summary, Kouyos et al. found that differences in HIV-1 genetics influence the development of antibody responses and that some viral variants can elicit bnAbs across individuals. Although such strong bnAb imprinting is likely to be rare, the viral strains and antigens that underlie this effect are prime candidates for vaccine development.

Ursula Hofer

ORIGINAL ARTICLE Kouyos, R. D. et al. Tracing HIV-1 strains that imprint broadly neutralizing antibody responses. *Nature* https://doi.org/10.1038/s41586-018-0517-0 (2018)

Gram-negative *Enterobacter cloacae*, *Proteus mirabilis*, *Salmonella enterica*, *Escherichia coli* and *Vibrio harveyi* in cystic fibrosis medium containing mucin on semi-solid agar plates. They observed the same basic physical characteristics of surfing motility in all species and that mucin was the only wetting agent (a substance that lowers the surface tension of a liquid) that was able to consistently induce surfing.

Next, the authors investigated the surfing motility of the different species in various medium viscosities. Compared to swarming and swimming motility, which occur in limited ranges of medium viscosities, surfing motility occurred over a broader range of viscosities and generally at a higher viscosity.

As *P. aeruginosa* exhibits increased resistance to antibiotics during surfing motility, the authors tested whether surfing cells of the other species were similarly resistant to antibiotics. All tested species exhibited broad-spectrum antibiotic resistance during surfing motility, but there was substantial variation in resistance to specific classes of antibiotics.

Surfing was first reported to be dependent on flagella but not pili in *P. aeruginosa*. This finding was corroborated for the other species as only flagella mutants, but not pili or fimbriae mutants, exhibited surfing defects. The authors also tested the importance of quorum sensing, which was reported to be required for *P. aeruginosa* surfing; however, this aspect was not conserved in the other tested species, including *V. harveyi* and *S. enterica*, as demonstrated by assaying quorum-sensing mutants. This finding suggests that the regulation of surfing is distinct among motile bacteria.

In summary, Sun et al. found that the physical characteristics of surfing motility are conserved among motile mucosa-associated pathogens and that this phenotype is associated with broad-spectrum antibiotic resistance.

Ashley York

ORIGINAL ARTICLE Sun, E., Liu, S. & Hancock, R. E. W. Surfing motility: a conserved yet diverse adaptation among motile bacteria. *J. Bacteriol.* https://doi.org/10.1128/JB.00394-18 (2018)

IN BRIEF

FUNGAL PATHOGENESIS

Making matters worse

Gut microbiota can have a distant effect on immune function in the lung through the gut–lung axis. This study observed that the specific expansion of the commensal fungus *Wallemia mellicola* in the gut (that is, without overgrowth of the total fungal community in the gut) enhances the severity of allergic airway disease in mice. Whereas mice with a healthy gut microbiota were able to resist a population expansion when gavaged with live spores of *W. mellicola*, mice that had been treated with antibiotics did not. Mice that had increased levels of *W. melliola* in the gut experienced alterations in a number of pulmonary immune responses to inhalation of the house dust mite antigen (an aeroallergen). Colonization of fungus-free altered Schaedler Flora mice with *W. mellicola* enhanced the severity of allergic airways disease, suggesting that any changes in bacterial community composition do not have a role in this phenomenon.

ORIGINAL ARTICLE Skalski, J. H. et al. Expansion of commensal fungus *Wallemia mellicola* in the gastrointestinal mycobiota enhances the severity of allergic airway disease in mice. *PLOS Pathog.* **14**, e1007260 (2018)

FURTHER READING Budden, K. F. et al. Emerging pathogenic links between microbiota and the gut–lung axis. *Nat. Rev. Microbiol.* **15**, 55–63 (2017)

VIRAL INFECTION

Exploiting peroxisomes

Peroxisomes perform important metabolic functions, including lipid metabolism. This study finds that human cytomegalovirus (HCMV) and herpes simplex virus 1 (HSV1) induce peroxisome biogenesis and changes to peroxisome morphology that support viral infection. Using proteomics, the authors observed a global increase in the abundance of peroxisomal proteins during HCMV and HSV1 infections. Microscopy analysis and mathematical modelling showed that infection caused peroxisome growth and fission, leading to an increase in the number of peroxisomes. On average, peroxisomes became larger in size and more elongated, which causes an increase in the peroxisome membrane-to-lumen ratio. HCMV-induced peroxisome biogenesis also enhanced the synthesis of the phospholipid plasmalogen, increasing virus production. Altogether, this suggests that these viruses exploit peroxisome lipid metabolism to enhance viral replication.

ORIGINAL ARTICLE Jean Beltran, P. M. et al. Infection-induced peroxisome biogenesis is a metabolic strategy for herpesvirus replication. *Cell Host Microbe* https://doi.org/10.1016/j.chom.2018.09.002 (2018)

BACTERIAL PATHOGENESIS

Stronger together

Uropathogenic *Escherichia coli* (UPEC) is a major cause of urinary tract infections. These infections are initiated by adhesion to the bladder epithelium, after which UPEC colonizes and persists in the bladder niche. Hollenbeck et al. find that the *E. coli* curli amyloid fibres promote bladder epithelial cell adhesion and that phosphoethanolamine (pEtN) cellulose enhances this interaction. The authors used a custom-built live cell monolayer rheometer to directly measure individual and combined contributions of type 1 pili, curli and pEtN cellulose to bladder cell adhesion during high-shear conditions. Using the UPEC strain UTI89, isogenic mutants and conditions that result in the production of the cell surface structures, curli-mediated adhesion was found to be stronger than type 1 pili and this was enhanced by pEtN cellulose, which the authors posit functions as a mortar, increasing the strength of the interaction.

ORIGINAL ARTICLE Hollenbeck, E. C. et al. Phosphoethanolamine cellulose enhances curli-mediated adhesion of uropathogenic *Escherichia coli* to bladder epithelial cells. *Proc. Natl Acad. Sci. USA* https://doi.org/10.1073/pnas.1801564115 (2018)