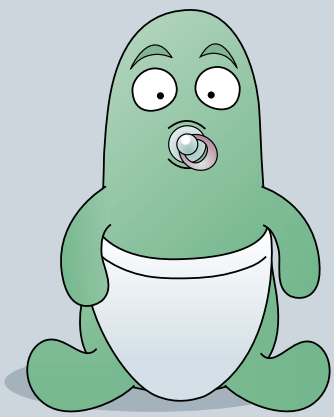
Credit: Charlotte Allen/Alamy Stock Photo

The adjuvants had no effect on drug resistance and on low-tolerance isolates but they reduced the tolerance of all tested isolates that had mid to high tolerance levels, with all adjuvants leading to the death of cells inside the inhibition zone. Notably, the adjuvant fluphenazine improved survival of *Galleria mellonellae* larvae that were infected with *C. albicans* and treated with fluconazole. Furthermore, isolates from patients that had persistent candidemia despite fluconazole treatment had higher tolerance than isolates from patients in which one course of fluconazole had cleared the infection.

Taken together, these results show that *C. albicans* can form a subpopulation of drug-tolerant cells, which contribute to persistent infection but which might be tackled with adjuvants.

Ursula Hofer

ORIGINAL ARTICLE Rosenberg, A. et al. Antifungal tolerance is a subpopulation effect distinct from resistance and is associated with persistent candidemia. *Nat. Commun.* **9**, 2470 (2018)
FURTHER READING Brauner, A. et al. Distinguishing between resistance, tolerance and persistence to antibiotic treatment. *Nat. Rev. Microbiol.* **14**, 320–330 (2016)



Credit: Philip Patenall/Springer Nature Limited

also found evidence that maternally acquired strains are more likely to adapt and persist in the infant gut compared with strains from other sources.

In the second study, Yassour, Jason, Hogstrom et al. analysed the stool of 44 mother–infant pairs in the first 3 months of life. They observed low species-level complexity and striking differences between maternal and infant microbiomes on day 1 of birth, but found evidence of transmission of a number of species between mothers and infants. By analysing strain-level variation, they observed two patterns of inheritance across multiple bacterial species in which either dominant strains colonized infants or, occasionally, secondary (less dominant) maternal strains were successful in colonization. Remarkably, in cases where a secondary strain of *Bacteroides uniformis* was transmitted, a specific starch utilization (Sus) module was absent in the mother's dominant strain, but was encoded in the secondary strain of *B. uniformis*. The authors suggest that this strain provides a selective advantage in metabolizing breast milk.

Together, these studies provide further evidence that mother-to-infant microbiome transmission is important in the establishment and development of the infant microbiome.

Ashley York

ORIGINAL ARTICLES Ferretti, P. et al. Mother-to-infant microbial transmission from different body sites shapes the developing infant gut microbiome. *Cell Host Microbe* **24**, 133–145 (2018) | Yassour, M., Jason, E., Hogstrom, L. et al. Strain-level analysis of mother-to-child bacterial transmission during the first few months of life. *Cell Host Microbe* **24**, 146–154 (2018)

IN BRIEF

BACTERIAL PHYSIOLOGY

Bearing the load

The cell envelope of Gram-negative bacteria, which is composed of the inner membrane, the peptidoglycan cell wall and the outer membrane, enables cells to withstand mechanical loads. It was previously thought that the mechanical integrity of the cell envelope is conferred by the cell wall; however, Rojas et al. report that the outer membrane has load-bearing capacity. Genetic or chemical perturbations to the outer membrane increased deformation of the cell envelope in response to different types of load, suggesting that the mechanical properties of the envelope are dependent on the composition and integrity of the outer membrane. Compromising the outer membrane reduced cell survival during osmotic fluctuation and proliferation of L-form bacteria, which lack a cell wall. Thus, both the cell wall and the outer membrane contribute to the mechanical properties of the cell envelope.

ORIGINAL ARTICLE Rojas, E. R. et al. The outer membrane is an essential load-bearing element in Gram-negative bacteria. *Nature* **559**, 617–621 (2018)

PHAGE BIOLOGY

Teamwork pays off

To overcome CRISPR–Cas defence systems, phages have evolved anti-CRISPR proteins (Acrs). Although insights into the structure, function and mechanisms of action of Acrs have been gained, their deployment and effect on phage epidemiology was elusive. Two studies now report that phages cooperate to supress CRISPR–Cas-mediated immunity. Borges et al. show that infections fail when phage population numbers fall below a critical threshold. However, although initial phage infection might fail owing to the rapid degradation of the phage genome by CRISPR–Cas systems, Acr proteins that were deployed during the first infection process render the host cell immunocompromized and thus more susceptible to subsequent infections. Landsberger et al. show that this cooperation leads to an epidemiological tipping point: if the initial densities of phages are high enough, densities of immunosuppressed hosts reach a critical threshold, whereby the numbers of new phages that are produced from successful infections are higher than the number of phages that are eliminated, leading to an epidemic.

ORIGINAL ARTICLES Borges, A. L. et al. Bacteriophage cooperation suppresses CRISPR-Cas3 and Cas9 immunity. *Cell* https://doi.org/10.1016/j.cell.2018.06.013 (2018) | Landsberger, M. et al. Anti-CRISPR phages cooperate to overcome CRISPR-Cas immunity. *Cell* https://doi.org/10.1016/j.cell.2018.05.058 (2018)

VIRAL INFECTION

Continued risk of Ebola virus outbreak

Although widespread transmission of Ebola virus disease in West Africa was mostly controlled following the devastating epidemic in 2014–2015, subsequent clusters of infection have occurred in the region. In this study, Dokubo, Wendland, Mate, Ladner et al. investigated the cluster of Ebola virus disease cases identified in November 2015 in Liberia. Based on serological profiles and genomic and epidemiological data, they report that the possible source of infection in this cluster was a female patient who survived Ebola virus disease in 2014 and was persistently infected with the virus or suffered recurrent disease and subsequently transmitted the virus to family members a year later. These findings highlight that Ebola virus persistence poses a continued risk for disease resurgence.

ORIGINAL ARTICLE Dokubo, E. K., Wendland, A., Mate, S. E., Ladner, J. T. et al. Persistence of Ebola virus after the end of widespread transmission in Liberia: an outbreak report. *Lancet Infect. Dis.* https://doi.org/10.1016/S1473-3099(18)30417-1 (2018)