IN BRIEF

FUNGAL PATHOGENESIS

Copy and cut to evade host defence

Progressive, systemic fungal infections are associated with host immune dysregulation; however, how fungi impair host immune responses is unclear. Sterkel *et al.* now report that the fungal serine protease di-peptidlyl peptidase IVA (DppIVA) inhibits leukocyte recruitment, differentiation and activation at sites of inflammation in a mouse model of pulmonary infection with *Blastomyces dermatitidis*. Importantly, they showed that DppIVA mimics the host ectopeptidase CD26 by cleaving CC chemokines and granulocyte–macrophage-colony stimulating factor (GM-CSF) to evade host defences. In agreement with this, decreasing the expression of fungal DppIVA or treatment with selective DppIVA inhibitors restored leukocyte recruitment and function, and control of disease. These data establish the fungal virulence factor DppIVA as a potential therapeutic drug target.

ORIGINAL ARTICLE Sterkel, A. K. *et al.* Fungal mimicry of a mammalian aminopeptidase disables innate immunity and promotes pathogenicity. *Cell Host Microbe* http://dx.doi.org/10.1016/j.chom.2016.02.001 (2016)

BACTERIAL PATHOGENESIS

How pneumococci lose their capsule

Pathogenic bacteria produce surface-associated complex carbohydrate capsules that prevent complement-mediated and antibody-mediated neutralization by the host. However, the capsule is also a major immunogen and a target of antimicrobial peptides (AMPs), so in different host tissues (such as the epithelium), pneumococci shed the capsule by an unknown mechanism. In a recent study, Kietzman *et al.* showed that exposure to cationic AMPs (CAMPs) on epithelial surfaces leads to the rapid loss of the *Streptococcus pneumoniae* capsule. This process was dependent on the hydrolytic activity of the pneumococcal cell wall hydrolase LytA, which is involved in both cell density-dependent and β-lactam antibiotic-induced autolysis. Decreased encapsulation in the epithelium was associated with increased bacterial resistance to microbicidal innate defence molecules and promoted invasion of epithelial cells, whereas enhanced encapsulation in the blood protected bacteria from phagocytosis.

ORIGINAL ARTICLE Kietzman, C. C. *et al.* Dynamic capsule restructuring by the main pneumococcal autolysin LytA in response to the epithelium. *Nat. Commun.* **7**, 10859 (2016)

ARCHAEA

A novel DNA import system

Although DNA exchange through cellular contact has been described in archaea, the mode of DNA transport has not been studied at the molecular level. Based on the assumption that DNA transport has a role in DNA repair and involves membrane proteins, Albers and colleagues looked at published microarray data describing changes in gene expression in *Sulfolobus acidocaldarius* following exposure to UV light. They identified three clustered genes that had increased expression, which they termed *cedA* (crenarchaeal system for exchange of DNA A), *cedA1* and *cedA2*. Another highly upregulated gene, *cedB*, encodes a homologue of the ATPase VirB4, a bacterial conjugation protein. In agreement with their role in DNA exchange, DNA transfer following UV treatment was blocked in mutants lacking CedA or CedB. Furthermore, the authors showed that this system works as a DNA importer in the UV-damaged cell.

ORIGINAL ARTICLE van Wolferen, M. *et al.* The archaeal Ced system imports DNA. *Proc. Natl Acad. Sci. USA* **113**, 2496–2501 (2016)