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IN BRIEF

PARASITE BIOLOGY

New insights into *Plasmodium* hepatocyte entry

Liver infection by *Plasmodium* spp. sporozoites is a crucial initial step in the life cycle of malaria parasites; however, the underlying molecular mechanisms are still poorly understood. Now, a new study provides molecular insights into hepatocyte invasion by *Plasmodium* spp. sporozoites. The authors showed that the human parasites *Plasmodium falciparum* and *Plasmodium vivax* use two distinct host cell receptors, CD81 and scavenger receptor class B type I (SR-BI), respectively, to enter hepatocytes. By contrast, the same receptors were functionally redundant for the entry of the rodent parasite *Plasmodium berghei*. In addition, they found that the parasite protein P36, which is a member of the 6-cysteine domain protein family, is a key determinant of receptor use. This study sheds light on the pathways that are used by malaria parasites to enter liver cells and reports new functional receptor–ligand interactions on hepatocytes.

ORIGINAL ARTICLE Manzoni, G. *et al. Plasmodium* P36 determines host cell receptor usage during sporozoite invasion. *eLife* **6**, e25903 (2017)

VIRAL EVOLUTION

Phage infection strategies

The functional consequences of divergent phage evolution on infection have not been analysed until now. By using transcriptomics analyses, Blasdel *et al.* compared the infection strategies of two evolutionarily related phages, PAK_P3 and PAK_P4, against the same *Pseudomonas aeruginosa* host strain. They showed that despite their limited DNA sequence similarity, the global infection strategies of these phages were conserved and relied on the temporal regulation of gene expression. This included the use of specific antisense transcripts and the rapid degradation of host mRNA. Moreover, specific adaptations had evolved in each virus. Indeed, the authors identified distinct core gene expression patterns and the manipulation of host gene expression, as exemplified by the specific manipulation of iron metabolism by PAK_P4. Altogether, this study shows that despite their genomic divergence, phages rely on conserved ancestral infection mechanisms.

ORIGINAL ARTICLE Blasdel, B. G. *et al.* Comparative transcriptomics analyses reveal the conservation of an ancestral infectious strategy in two bacteriophage genera. *ISME J.* http://dx.doi.org/10.1038/ismej.2017.63 (2017)

FUNGAL PHYSIOLOGY

Acidic pH interferes with *Candida* persistence

Candida albicans is an opportunistic fungal pathogen that can colonize host niches at varying pH. A study by Sherrington *et al.* shows that the growth of *C. albicans* in acidic environments results in structural changes to the cell wall that enhance its recognition by factors of the host innate immune system. At low pH, exposure of chitin and β-glucan is enhanced, which facilitates their recognition by macrophages and neutrophils and stimulates the production of pro-inflammatory cytokines. The increased exposure of chitin was dependent on the decreased expression of chitinase 2 (Cht2), which is regulated through the biofilm and cell wall regulator 1 (Bcr1)–Rim101 signalling cascade. By contrast, the enhanced exposure of β-glucan was regulated through a non-canonical signalling pathway. Thus, mucosal acidic environments may stimulate an antifungal immune response and contribute to the immunopathology of candidiasis.

ORIGINAL ARTICLE Sherrington, S. L. *et al.* Adaptation of *Candida albicans* to environmental pH induces cell wall remodelling and enhances innate immune recognition. *PLoS Pathog.* **13**, e1006403 (2017)