IN BRIEF

TECHNIQUES AND APPLICATIONS

Minimizing the risk

Recent work on H5N1 influenza A virus, in which strains have been genetically engineered to enable aerosol transmission between ferrets, has been criticized for potentially being dangerous. Here, Langlois *et al.* present an approach that could be used to improve the biosafety of such gain-of-function influenza virus experiments. Endogenous host microRNAs (miRNAs) have been shown to suppress the expression of viral genes, so the authors reasoned that modifying the viral genome to contain target sites for miRNAs present in humans and mice but absent in ferrets would block potential transmission to humans. miR-192 was identified as a candidate miRNA, as it is expressed specifically by human and mouse lung epithelial cells, but not ferret cells. Importantly, incorporation of the miR-192 target site into the viral genome did not interfere with transmission between ferrets, but it did attenuate pathogenicity in mice, which did not succumb to infection.

ORIGINAL RESEARCH PAPER Langlois, R. A. *et al.* MicroRNA-based strategy to mitigate the risk of gain-of-function influenza studies. *Nature Biotech.* http://dx.doi.org/doi:10.1038/nbt.2666 (2013)

FUNGAL PATHOGENESIS

SPRR spurs fungal growth

The highly conserved phosphatase calcineurin is required for hyphal growth and virulence in the filamentous fungus *Aspergillus fumigatus*. This study identifies a region (located in the linker between the catalytic domain (CnaA) and the regulatory domain (CnaB)) that has a key role in hyphal growth and virulence. This region, which is rich in serine and proline residues (and so was named SPRR), was phosphorylated by casein kinase 1 (CK1) and proline-directed kinases, and mutation of the serine residues led to defects in growth and virulence. Notably, SPRR is conserved in filamentous fungi but absent in humans, so could be targeted therapeutically.

ORIGINAL RESEARCH PAPER Juvvadi, P. R. *et al.* Phosphorylation of calcineurin at a novel serine-proline rich region orchestrates hyphal growth and virulence in *Aspergillus fumigatus*. *PLoS Pathog.* **9**, e1003564 (2013)

PARASITE BIOLOGY

Protective strategy for a trypanosome

Unlike *Trypanosoma brucei brucei*, *Trypanosoma brucei gambiense* resists killing by apolipoprotein L1 (apoL1), which associates with the serum complexes trypanolytic factor 1 (TLF1) and TLF2 and mediates killing after insertion into endosome–lysosome membranes. This study finds that *T. b. gambiense* resistance is conferred by TgsGP, which interacts with membrane lipids via its hydrophobic β-sheets and induces stiffening and increased curvature of endosomal membranes. Reduced sensitivity to apoL1 was also found to be due to increased activity of the parasite protease cathepsin D (which might accelerate apoL1 degradation) and downregulation of the TLF1 receptor HpHbR.

ORIGINAL RESEARCH PAPER Uzureau, P. *et al.* Mechanism of *Trypanosoma brucei gambiense* resistance to human serum. *Nature* http://dx.doi.org/doi:10.1038/nature12516 (2013)