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IN BRIEF

VIRAL PATHOGENESIS

Ebola virus' shed GP activates immune cells

Cells infected with Ebola virus (EBOV) release large amounts of viral glycoproteins; however, how these proteins contribute to pathogenesis is unknown. Now, Escudero-Pérez *et al.* show that one of these proteins, termed shed GP, binds to uninfected dendritic cells (DCs) and macrophages, in a process dependent on the glycosylation pattern of shed GP and on cellular Toll-like receptor 4 (TLR4). The binding of shed GP resulted in the activation of DCs and macrophages, and induced the release of cytokines, which were sufficient to increase the permeability of endothelial barriers. These data suggest that the release of shed GP from infected cells leads to the dysregulation of the host immune response and the modulation of remote target cells, such as endothelial cells, resulting in increased inflammation and vascular permeability, which are two characteristics of fatal EBOV infection.

ORIGINAL RESEARCH PAPER Escudero-Pérez, B. *et al.* Shed GP of Ebola virus triggers immune activation and increased vascular permeability. *PLoS Pathog.* **10**, e1004509 (2014)

FUNGAL PATHOGENESIS

Good cop(per), bad cop(per)

Copper is required for virulence of the fungus *Cryptococcus neoformans*, but an excess of this metal can have an antimicrobial effect. Interestingly, *C. neoformans* must disseminate from the lungs (which have high copper levels) to the brain (which has low copper levels) where it causes lethal meningoencephalitis. Now, Sun, Ju, *et al.* show that two copper importers, Ctr1 and Ctr4, influence fungal survival at these two sites. They found that Ctr1 is rapidly degraded in the presence of high copper levels, and in agreement with this, deletion of *CTR1* had no impact on fungal virulence in mouse lungs. By contrast, deleting *CTR4* resulted in a hypervirulent phenotype. Furthermore, overexpressing *CTR1* or *CTR4* reduced fungal survival in the lungs, which suggests that high levels of copper in this organ are toxic to *C. neoformans*. By contrast, *CTR1* and *CTR4* were necessary for fungal replication in the mouse brain, suggesting that copper import is essential for *C. neoformans* survival in this environment. These data suggest that *C. neoformans* can sense copper levels and modulate the expression of Ctr1 and Ctr4 to switch from copper detoxification to copper acquisition as it disseminates from the lungs to the brain.

ORIGINAL RESEARCH PAPER Sun, T. -S., Ju, X., *et al.* Reciprocal functions of *Cryptococcus neoformans* copper homeostasis machinery during pulmonary infection and meningoencephalitis. *Nature Commun.* **5**, 5550 (2014)

SYMBIOSIS

Vibrio genes involved in squid colonization

One of the best-studied examples of symbiosis is the colonization of the light organ of the Hawaiian bobtail squid, *Euprymna scolopes*, by the bioluminescent bacterium *Vibrio fischeri*. Now, Brooks *et al.* have carried out a forward genetic screen to identify bacterial factors involved in this process. The authors mutagenized *V. fischeri* by insertion sequencing and analysed a library of over 41,000 unique insertions before and after colonization of 1,500 squid hatchlings. This strategy identified several genes involved in colonization, including the gene encoding the cytoplasmic chaperone DnaJ. Notably, a bacterial mutant lacking *dnaJ* was impaired in its ability to form biofilms, which are known to be crucial for squid colonization. Future studies are needed to clarify how this bacterial chaperone is involved in *V. fischeri* biofilm formation.

ORIGINAL RESEARCH PAPER Brooks, J. F. 2nd *et al.* Global discovery of colonization determinants in the squid symbiont *Vibrio fischeri*. *Proc. Natl Acad. Sci. USA* http://dx.doi.org/10.1073/pnas.1415957111 (2014)