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IN BRIEF

VIRAL PATHOGENESIS

Taking a bite

Aedes spp. mosquitoes are important vectors of disease as they inject pathogen-containing saliva into a vertebrate host. Schmid *et al.* showed that salivary gland extract (SGE) augmented dengue virus (DENV) infection in the presence of enhancing serotype cross-reactive antibodies (which are non-neutralizing antibodies that do not protect against a different serotype and instead enhance subsequent infection), leading to severe disease and death. Furthermore, during antibody-dependent enhancement, SGE increased viral titres in the skin, augmented infection of dermal dendritic cells and macrophages, and accelerated the migration and recruitment of immune cells to the skin and lymph nodes. The authors also found that SGE disrupts endothelial barrier function *in vitro* and endothelial permeability *in vivo*. Finally, surgically removing the body site of inoculation rescued mice from severe disease, whereas this protective effect was abolished in the presence of SGE, which suggests that SGE affects systemic infection with DENV. In a second study, Liu *et al.* reported that the flavivirus non-structural protein 1 (NS1) has a crucial role in the acquisition of DENV and Japanese encephalitis virus (JEV) by mosquito vectors. During infection, NS1 is abundantly present in sera of patients and has been implicated in viral pathogenesis. The authors of this study now report that NS1 increases virus transmission from infected mice and that this effect is blocked following the inoculation of infected hosts with anti-NS1 antibodies. Importantly, the authors found that NS1 suppresses the expression of immune-related genes in the mosquito midgut, thereby enabling the virus to overcome the gut immune barrier. Finally, immunization of mice with a modified DENV2 NS1 in which immunogenic regions were deleted led to decreased DENV acquisition by mosquitoes and increased survival rates in infected mice compared with full-length NS1, which suggests that this approach might provide a new vaccination strategy.

ORIGINAL ARTICLES Schmid, M. A. *et al.* Mosquito saliva increases endothelial permeability in the skin, immune cell migration, and dengue pathogenesis during antibody-dependent enhancement. *PLoS Pathog.* **12**, e1005676 (2016) | Liu, J. *et al.* Flavivirus NS1 protein in infected host sera enhances viral acquisition by mosquitoes. *Nat. Microbiol.* **1**, 16087 (2016)

BACTERIAL PHYSIOLOGY

Recognizing yourself

Contact-dependent growth inhibition (CDI) systems have been implicated in the killing of susceptible competing bacteria on cell–cell contact. In *Burkholderia* spp., this system involves two-partner secretion proteins that are encoded by the *bcpAIOP* locus: a bacterial cell delivers a toxic effector domain that is derived from the carboxyl terminus of the exoprotein BcpA (BcpA-CT) to a neighbouring cell, which is protected from CDI if it produces the immunity protein, BcpI. Garcia *et al.* show that CDI systems also have a role in cell-to-cell communication and cooperation between bacteria that produce the toxin–antidote protein pair. The delivery of catalytically active BcpA-CT results in changes in gene expression in immune recipient cells that affect community behaviour, such as the formation of biofilms. The authors termed this form of interbacterial communication contact-dependent signalling (CDS). The findings of this study suggest that the *bcpAIOP* locus has a dual function: antagonism of non-self cells as well as cell-to-cell communication between *Burkholderia* species that express the same *bcpA–bcpI* allele.

ORIGINAL ARTICLE Garcia, E. C. *et al.* Interbacterial signaling via *Burkholderia* contact-dependent growth inhibition system proteins. *Proc. Natl Acad. Sci. USA* http://dx.doi.org/10.1073/pnas.1606323113 (2016)