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IN BRIEF

BACTERIAL PHYSIOLOGY

Raising the alarm

The targeted degradation of foreign nucleic acids by type III CRISPR–Cas systems is mediated by the multisubunit Csm interference complex. In addition, the CRISPR-associated protein Csm6 ribonuclease, which does not form part of the Csm complex, degrades foreign RNAs to provide full immunity, but how these two processes are linked is unknown. Now, two new studies reveal the mechanistic link between invader sensing and the activity of Csm6. Both studies by Kazlauskiene *et al.* and Niewoehner, Garcia-Doval *et al.* discovered that RNA binding by the Csm complex triggers its Cas10 subunit to synthesize cyclic oligoadenylate from ATP, which then binds to Csm6 and allosterically activates its ribonuclease activity. Together, these studies reveal a signalling pathway in bacteria that is reminiscent of signalling in mammalian innate immunity to control viral infection.

ORIGINAL ARTICLES Kazlauskiene, M. *et al.* A cyclic oligonucleotide signaling pathway in type III CRISPR–Cas systems. *Science* http://dx.doi.org/10.1126/science.aao0100 (2017) | Niewoehner, O., Garcia-Doval, C. *et al.* Type III CRISPR–Cas systems produce cyclic oligoadenylate second messengers. *Nature* http://dx.doi.org/10.1038/nature23467 (2017)

VIRAL PATHOGENESIS

Finding the enemy within

Ebola virus (EBOV) can persist for many months after survivors have recovered from acute EBOV disease and cause post-EBOV syndrome. Understanding EBOV persistence *in vivo* has been challenging owing to the lack of an animal model, but now Zeng *et al.* report the pathological characterization of persistent asymptomatic infection in rhesus monkeys. The authors used fluorescence *in situ* hybridization and immunofluorescence to detect viral RNA and proteins in infected tissues, and documented the progressive spread of EBOV into the eyes, brain and testes. They identified $CD68^+$ monocytes as the viral reservoir in the vitreous humour of the eye, the epididymis, and in inflamed regions of the brain, but not in organs that are typically affected during acute infection. This study suggests that rhesus monkeys could provide an animal model to study EBOV persistence and to test new antiviral strategies.

ORIGINAL ARTICLE Zeng, X. *et al.* Identification and pathological characterization of persistent asymptomatic Ebola virus infection in rhesus monkeys. *Nat. Microbiol.* **2**, 17113 (2017)

PARASITE GENOMICS

Screening for the essentials

Functional genome-wide screens in *Plasmodium* spp. parasites, the causative agents of malaria, have been lacking, as they are refractory to genetic manipulation. Now, a new study by Bushell *et al.* reports the findings of an *in vivo* genetic screen that identified essential genes and pathways that are required for parasite growth. The authors created pools of knockout *Plasmodium berghei* mutants covering 2,578 genes (representing >50% of the parasite genome), and infected mice with these pools of mutants. They used next-generation sequencing to measure the relative growth rate of the genetically modified parasites and observed that approximately 63% of *P. berghei* genes were required for normal asexual blood stage growth *in vivo*. By contrast, they found that many genes at the host–pathogen interface, such as genes that are involved in erythrocyte invasion and immune evasion, were redundant, which suggests that opposing evolutionary pressures have shaped the *Plasmodium* spp. genome.

ORIGINAL ARTICLE Bushell, E. *et al.* Functional profiling of a *Plasmodium* genome reveals an abundance of essential genes. *Cell* **170**, 260–272.e8 (2017)