IN BRIEF

BACTERIAL GENOMICS

Three centuries of plague

Spearheaded by the Black Death in the fourteenth century, the second plague pandemic was a formative influence on European societies in the medieval and early modern periods. Bos *et al.* sequenced five osteoarcheological samples from an outbreak — the Great Plague of Marseille (1720–1722) — dated towards the end of the second pandemic. A SNP-based phylogenetic analysis showed that the Marseille outbreak was caused by a *Yersinia pestis* strain descended from the Black Death strain but not ancestral to any known extant strain, which suggested that this pathogenic lineage had persisted for three centuries before becoming extinct. Based on the new phylogeny, the authors argue that an uncharacterized host reservoir local to Europe or West Asia may have sustained the repeated outbreaks of the second pandemic.

ORIGINAL ARTICLE Bos, K. I. *et al.* Eighteenth century *Yersinia* pestis genomes reveal the long-term persistence of an historical plague focus. *eLife* http://dx.doi.org/10.7554/eLife.12994 (2016)

PARASITE GENETICS

Putting a stop to kinetoplastid transcription

As early divergers from other eukaryotes, kinetoplastids have several unusual features of gene regulation. For example, genes are post-transcriptionally processed from polycistronic transcription units and contain a hypermodified base, base J (β-D-glucosyl-hydroxymethyluracil), that demarcates the sites of transcription initiation and termination. In *Leishmania* spp., the loss of base J results in genome-wide defects in the termination of transcription, but only a partial defect is observed in *Trypanosoma brucei*. Two new studies now establish that the histone mark H3.V is required in addition to base J to terminate transcription by RNA polymerase II in *T. brucei*. Furthermore, the studies found that H3.V, but not base J, mediates the termination of transcription by RNA polymerase I at telomeric loci that encode variant surface glycoproteins. Therefore, H3.V may contribute to the antigen switching that occurs at these loci, which hides *T. brucei* from the host immune system.

ORIGINAL ARTICLES Reynolds, D. *et al.* Histone H3 variant regulates RNA polymerase II transcription termination and dual strand transcription of siRNA loci in *Trypanosoma brucei*. *PLoS Genet.* **12**, e1005758 (2016) | Schulz, D. *et al.* Base J and H3.V regulate transcriptional termination in *Trypanosoma brucei*. *PLoS Genet.* **12**, e1005762 (2016)

CELLULAR MICROBIOLOGY

Damage limitation after friendly fire

Restriction–modification (R–M) systems are commonly used by bacteria to defend against invading DNA. In these systems, a methyltransferase methylates endogenous DNA as a marker of 'self' and a cognate restriction enzyme cleaves invading DNA that lacks this modification. Guet and colleagues asked whether R–M systems ever err in the discrimination between self and non-self DNA, and whether such an error would incur a fitness cost. Testing the EcoRI and EcoRV R–M systems in *Escherichia coli*, they found that EcoRI, but not EcoRV, measurably, and stochastically, targets self DNA, albeit at a low frequency. The difference between EcoRI and EcoRV was attributed to a more efficient restriction enzyme, which makes for a more potent defence against foreign DNA but also increases the chance of cleaving self DNA prior to modification. However, fitness costs were minimal; *E. coli* cells growing at steady-state efficiently repaired EcoRI-mediated DNA damage and the induced SOS response was transient and did not affect cell viability.

ORIGINAL ARTICLE Pleška, M. *et al.* Bacterial autoimmunity due to a restriction-modification system. *Curr. Biol.* http://dx.doi.org/10.1016/j.cub.2015.12.041 (2016)