IN BRIEF

METAGENOMICS

Why stop here?

In all domains of life, three stop codons are thought to terminate translation: TAG (known as amber), TGA (known as opal) and TAA (known as ochre). Ivanova *et al.* analysed a global collection of 1,776 samples from the Integrated Microbial Genomes database (which includes both environmental and human-associated samples) and found extensive stop codon reassignment. Interestingly, bacteria only reassigned opal, eukaryotes only reassigned ochre, viruses reassigned amber and opal, and no reassignments were found in archaea. In the human microbiome samples, the authors also identified several DNA phages that have amber reassignments. As bacteria do not reassign amber, this questions the dogma that genetic code differences between phages and their bacterial hosts are a barrier to phage infection. Accordingly, the genomes of amber-recoded phages contain genes for peptide chain release factor 2 — which terminates translation at ochre and opal codons — as well as a non-canonical Gln-tRNA$_{CUA}$ — which reassigns amber to glutamine. This suggests that phages can actively interfere with translation of opal-recoded host genes and use their own, amber-recoded alternative genetic code.

ORIGINAL RESEARCH PAPER Ivanova, N. N. *et al.* Stop codon reassignments in the wild. *Science* **344**, 909–913 (2014)

VIRAL PATHOGENESIS

The pandemic potential of H10N8

H10N8 is the newest epidemic threat to human health from the avian influenza viruses, following in the footsteps of H7N9 and H5N1. To investigate the potential of H10 viruses to spread from birds to humans, Vachieri *et al.* compared H10 viruses with other influenza viruses for their ability to bind to human and avian receptor analogues. The avian H10 virus binds to both human and avian receptors with higher affinity than H7 avian and human viruses. Interestingly, although the binding of the H10 virus to human receptors is comparable with that of H1 and H3 viruses — the pandemic agents responsible for the 1918 Spanish flu and the 1968 Hong Kong flu, respectively — the H10 virus shows a strong preference for avian receptors, which is not shared by H1 and H3 viruses. These data suggest that, despite its ability to tightly bind to human receptors, the H10 virus would require mutations that switch its binding preference from avian to human receptors in order to efficiently spread among humans.

ORIGINAL RESEARCH PAPER Vachieri, S. G. *et al.* Receptor binding by H10 influenza viruses. *Nature* http://dx.doi.org/10.1038/nature13443 (2014)

BIOFILMS

Targeting (p)ppGpp disrupts biofilms

Biofilms are a major health concern worldwide, and no drugs are licensed to target them. Now, de la Fuente-Núñez *et al.* describe the identification of a new peptide that prevents the formation, and promotes the disruption, of biofilms that are formed by several pathogens, including *Pseudomonas aeruginosa*, methicillin-resistant *Staphylococcus aureus* and multiple Enterobacteriaceae. Peptide 1018 functions by directly interacting with guanosine tetra- or pentaphosphate, (p)ppGpp — a second messenger that is known to regulate the stringent response — and marking it for degradation. These data establish (p)ppGpp as a new drug target in the fight against biofilms.

ORIGINAL RESEARCH PAPER de la Fuente-Núñez, C. *et al.* Broad-spectrum anti-biofilm peptide that targets a cellular stress response. *PLoS Pathog.* **10**, e1004152 (2014)