IN BRIEF

HUMAN GENETIC VARIATION

The diversity present in 5,140 human mitochondrial genomes

Pereira, L. *et al. Am. J. Hum. Genet.* 7 May 2009 (doi:10.1016/j.ajhg.2009.04.013)

High-throughput sequencing is rapidly increasing the amount of data on human mitochondrial genetic variation. These authors developed a new computational tool, mtDNA-GeneSyn, which analyses diversity among mitochondrial genomes. Using this tool to analyse all mitochondrial DNA (mtDNA) data currently in GenBank, the authors provide an overall picture of human mtDNA diversity. The software is free to download, allowing other researchers to perform similar analyses as more data is deposited, and to assess mtDNA diversity in specific populations.

TRANSLATION

Bases in the anticodon loop of tRNA prevent misreading

Murakami, H. *et al. Nature Struct. Mol. Biol.* **16**, 353–358 (2009)

A sequence element that tunes *Escherichia coli* $tRNA^{Ala}_{GGC}$ to ensure accurate decoding

Ledoux, S. *et al. Nature Struct. Mol. Biol.* **16**, 359–364 (2009)

Using *in vitro* assays for peptide synthesis these papers show that the interaction between the anticodon on a tRNA molecule and its cognate mRNA codon is labile, as some variants lead to relaxed constraints on codon–anticodon interactions and to the insertion of a wrong amino acid. Such translational infidelity is caused by particular combinations of interacting base pairs (positions 32 and 38) on either side of the anticodon loop. The structural basis for the infidelity is unknown, but the suggestion that mutations at positions 32 or 38 are causal to several progressive human diseases is intriguing.

GENE REGULATION

Progressive lengthening of 3′ untranslated regions of mRNAs by alternative polyadenylation during mouse embryonic development

Ji, Z. *et al. Proc. Natl Acad. Sci. USA* **106**, 7028–7033 (2009)

This paper reveals that an important means of regulating gene expression post-transcriptionally might be provided by the progressive lengthening of the 3′UTR of mRNAs. Such lengthening — which was observed *in vivo* and *in vitro* — is attributed to alternative adenylation, permitted by weak polyadenylation signals; the resulting AU-rich 3′UTRs would make them better targets for microRNAs, among other regulators.

COMPLEX DISEASE

Multilocus Bayesian meta-analysis of gene-disease associations

Newcombe, P. J. *et al. Am. J. Hum. Genet.* 30 Apr 2009 (doi:10.1016/j.ajhg.2009.04.001)

In meta-analyses of gene–disease association studies the widely used approach of pooling data for each SNP is inefficient because, often, only a subset of studies provide data about a particular marker. This study reports a generally applicable Bayesian, multimarker approach to meta-analysis that uses all data for a region or gene, irrespective of the specific markers that have been typed, to make efficient use of data from all constituent studies.