IN BRIEF

GENOME EVOLUTION

Natural selection shapes genome wide patterns of copy number polymorphism in *D. melanogaster*.

Emerson, J. J. *et al. Science* 5 Jun 2008 (doi:10.1126/science.1158078)

Using high-density tiling arrays to survey copy number polymorphisms (CNPs) in an unbiased way across the whole *Drosophila melanogaster* genome, these authors identified 2,658 CNPs, more than half of which contain genic sequence. Further analysis revealed that purifying selection has a strong influence on the distribution of *D. melanogaster* CNPs; those located in exons and introns and on the X chromosome are most strongly affected, with deletions being affected more strongly than duplications.

MODEL ORGANISMS

Targeted gene inactivation in zebrafish using engineered zinc-finger nucleases.

Meng, X. *et al. Nature Methods* 25 May 2008 (doi:10.1038/nbt1398)

Heritable targeted gene disruption in zebrafish using designed zinc-finger nucleases.

Doyon, Y. *et al. Nature Methods* 25 May 2008 (doi:10.1038/nbt1409)

Zinc-finger nucleases (ZFNs) are chimeric enzymes that combine targeted DNA binding with DNA cleavage; the result is double-strand-break repair that can lead to mutations at a site of interest. These papers show that ZFNs can be used for efficient, heritable, targeted gene disruption in zebrafish by injecting mRNAs encoding ZFNs into one-cell embryos. This method can potentially be applied to any organism in which fertilized eggs can be injected.

GENETIC VARIATION

Genome-wide survey of allele-specific splicing in humans.

Nembaware, V. *et al. BMC Genomics* **9**, 265 (2008)

Links between allele-specific splicing differences and disease susceptibility mean that being able to predict the effect of a polymorphism on splicing is an important goal. These authors carried out a genome-wide scan for SNPs that potentially affect splicing and compared the results with publicly available EST and exon-array data, confirming splicing effects for 1,185 out of 30,977 polymorphisms and enabling them to develop a new method for inferring allele-specific splicing from EST data. These data provide a resource for investigating the influence of splicing variation in human disease.

HUMAN DISEASE

Towards a transgenic model of Huntington's disease in a non-human primate.

Yang, S-H., Cheng, P-H., Banta, H. *et al. Nature* 18 May 2008 (doi:10.1038/nature06975)

Although mouse and fly models of Huntington disease (HD) have helped to understand the molecular basis of this triplet-repeat disorder, these models do not replicate all the behavioural changes seen in humans. The first non-human primate model of HD has now been created by inserting an expanded exon of the human huntingtin gene into unfertilized macaque eggs. The animals show many clinical features of HD and so could be used to explore disease pathogenesis and potential treatments.