# IN BRIEF

## DEVELOPMENTAL GENETICS

Genetic programs activated by proneural proteins in the developing *Drosophila* PNS.

Reeves, N. & Posakony, J. W. Dev. Cell 8, 413–425 (2005)

As their name suggests, proneural transcription factors promote neurogenesis. Their role is well established, unlike their mode of action, because few of their targets are known. Using whole-genome microarray analysis and *in situ* hybridization in proneural cell clusters, the authors identified a set of genes that are preferentially expressed in these cells. Loss of function of two of the candidates confirmed their role in PNS development. Sequence analysis and reporter studies allowed the authors to describe *cis*-regulatory elements that direct proneural gene expression.

#### EVOLUTION

Sex peptide causes mating costs in female *Drosophila melanogaster*.

Wigby, S. & Chapman, T. Curr. Biol. 15, 316–321 (2005)

Male fruitflies pass a cocktail of accessory-gland proteins (Acps) to females during mating. Not only do these elicit a range of post-mating behavioural changes in females, but they are toxic, reducing female survival and reproductive success. Chapman and Wigby found that a single Acp — the sex peptide — is responsible for most of this cost of mating, making the sex peptide, which also stimulates egg production and reduces female sexual receptivity, the first identified protein that is likely to have a role in sexual conflict.

#### HUMAN DISEASE

Inactivation of TGF $\beta$  signaling in neural crest stem cells leads to multiple defects reminiscent of DiGeorge syndrome.

Wurdak, H. et al. Genes Dev. 19, 530–535 (2005)

This paper provides a link between transforming growth factor- $\beta$  (TGFB) signalling and DiGeorge syndrome, which is typically associated with a small deletion of human chromosome 22. Mice that were engineered to lack the TGFB receptor in neural crest cells showed many features of DiGeorge syndrome. The chromosome 22 micro-deletion does not remove any members of the TGFB pathway, but it contains a gene, *CrkL*, that interacts with TGFB signalling.

#### FUNCTIONAL GENOMICS

A universal plasmid library encoding all permutations of small interfering RNA.

Chen, M. et al. Proc. Natl Acad. Sci. USA 102, 2356–2361 (2005)

Small interfering RNA (siRNA) libraries are limited to targeting predicted or known genes, restricting their usefulness in species for which the transcriptome is uncharacterized. Chen *et al.* circumvented this problem by constructing a library of 5 x 10<sup>7</sup> plasmids containing all permutations of siRNA sequences. The library should enable large-scale RNAi screens to be carried out in any organism or cell type.

## RECOMBINATION

# Two sexes, two tempos

A hundred years after it was found that male and female genomes differ in the rate at which they recombine, a cause has been found for this widespread phenomenon. The explanation lies not, as it was thought 80 years ago, in the nature of the sex chromosomes, but on the selective pressures acting on oocytes and sperm.

Sex differences in recombination rate are seen in animals and plants up and down the evolutionary scale - most famous, perhaps, is the lack of recombination in the male Drosophila melanogaster, which is used routinely to propagate mutations without the risk of them being shuffled about by recombination. Haldane and Huxley were the first to propose an explanation for this pattern: based on a small data sample they concluded that the heterogametic sex always recombines at a lower rate, and that this occurs because the lack of recombination between the sex chromosomes has spread to affect recombination between autosomes. However, this theory is lacking in many respects; perhaps the most convincing argument against it is that species that have no sex chromosomes also show differences in meiosis between males and females. Now, the availability of detailed linkage maps for a greater number of species has allowed us to revisit this long-standing enigma.

A dataset from 107 species of animals and plants reveals recombination patterns that would not have been available to Haldane or Huxley, and allow several new conclusions to be drawn. In the study, the rate of recombination in one species — as measured using chiasma number or map length — was correlated to the type of sex chromosomes present in that species and to the degree of competition that exists between gametes of the same sex (a measure of the selective force acting on gametes).

In plants at least, differences in the recombination rate between the sexes relate to the opportunity for selection on the haploid phase of an organism's life cycle. For example, in species where selection tends to be milder among male gametes than among female gametes (such as, in highly selfing compared with outcrossing species) then you would expect to see a higher recombination rate in males than in females. This is because of the advantage conferred by preserving, as much as possible, the existing gene combinations in both gametes. A second conclusion is that the lack of recombination seen in one sex of some species is not merely an extreme case of reduced recombination, but arises from qualitatively different evolutionary forces — this was deduced from the fact that absence of recombination in one sex is influenced by the nature of the sex chromosomes, whereas a lower rate of recombination is not.

The idea that differences in recombination rate between the sexes could be down to haploid selection is not new. However, this work has provided strong empirical evidence that although it does not provide a causal link — substantiates this idea, and also ties together many apparently contradictory observations.

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References and links ORIGINAL RESEARCH PAPER Lenormand, T. & Dutheil, J. Recombination difference between sexes: a role for haploid selection. *PLoS Biol.* 3, e63 (2005)

