EVOLUTION

The right background for sex

Why sex has evolved is an age-old problem. The prevalence of sexually reproducing species indicates that this mode of reproduction is more successful than the asexual, non-recombining kind, but none of the proposed models for why sex evolved — that sexual recombination brings together beneficial mutations, purges the genome from harmful mutations or frees mutations from the burden of their genetic backgrounds — have been entirely satisfactory. The relative merits of each model have largely been fought on theoretical grounds; however, now Rice and Chippindale provide some welcome experimental backing for the last of these theories. By following the fate of a beneficial mutation in *Drosophila* strains, they have found that it is much more successful in recombining than in non-recombining lines, indicating that sexual recombination is required to bring new beneficial mutations into genetic backgrounds in which they can thrive.

The thinking and the experimental design behind the experiments in this paper are as follows. A mutation that arises on a non-recombining background has little say on the genetic company in which it finds itself. This is bad news for a mildly advantageous mutation, which can only hope to be fixed (that is, achieve a frequency of 100%) if it arises in a genome at the higher end of the reproductive fitness scale. If it doesn't, the advantage conferred by the mutation will be swamped by the overall low fitness of the genome in which it occurs. The same mutation occurring in a sexually reproducing genome, however, has more opportunities to sample different genetic environments — those produced through recombination — and to land in ones in which its success is unhindered by an unfavourable genetic background. This idea predicts that the frequency of a beneficial mutation will increase when propagated in a recombining versus a non-recombining lineage.

Using *Drosophila*, the authors followed the success of a beneficial *white*$^{+}$ allele (conferring red eyes) as it was passed down through ten generations of *white*$^{-}$ (white-eyed) flies that were either recombining or non-recombining. Non-recombining flies were obtained by manipulating the female genome so that it propagated itself clonally (*Drosophila* males normally do not recombine). The experiment — repeated 34 times — invariably showed that the *white*$^{+}$ allele accumulated more rapidly in the sexual population, supporting the idea that sex is advantageous because it accelerates adaptive evolution.

Needless to say, the story doesn't end here. As Richard Lenski points out in an accompanying Perspective, two puzzles surround why sex evolved: why sexual reproduction came about in the first place and why it is maintained. This paper has found an answer (although probably not the only one) to the second of these questions, and also shows how incisive experimental design can complement theoretical studies of long-standing evolutionary issues.

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References and links

ORIGINAL RESEARCH PAPER Rice, W. M. & Chippindale, A. K. Sexual recombination and the power of natural selection. *Science* **294**, 555–559 (2001)

FURTHER READING Lenski, R. E. Come fly, and leave the baggage behind. *Science* **294**, 533–534 (2001)

WEB SITE

William Rice's lab: http://lifesci.ucsb.edu/EEMB/faculty/rice/research.html

IN BRIEF

GENE REGULATION

Identification of novel genes coding for small expressed RNAs.

Lagos-Quintana, M. *et al. Science* **294**, 853–858 (2001)

An abundant class of tiny RNAs with probable regulatory roles in *Caenorhabditis elegans*.

Lau, N. C. *et al. Science* **294**, 858–862 (2001)

An extensive class of small RNAs in *Caenorhabditis elegans*.

Lee, R. C. *et al. Science* **294**, 862–864 (2001)

Keen to expand the number of known small RNAs that are involved in developmental timing in the worm (so-called small temporal RNAs, stRNAs) and in RNA interference (small intermediate RNAs, siRNAs), the authors of these papers have together identified and cloned 91 novel 22–25-nucleotide RNAs from worm, fly and human. Genome sequence and expression studies indicate that these hitherto elusive micro (mi)RNAs are processed from larger RNA molecules and that some are conserved, even in vertebrates, and probably function in translational repression. The task ahead is to find the precise function and potential targets of the new miRNAs (see Sean Eddy's review on p 919).

EVOLUTION

Pattern and timing of gene duplication in animal genomes.

Friedman, R. & Hughes, A. L. *Genome Res.* **11**, 1842–1847 (2001)

The existence of numerous multigene families in vertebrates has often been explained in terms of whole-genome duplication that might have occurred early in vertebrate evolution. Friedman and Hughes tested this hypothesis by analysing homologous multigene families from the genomes of human, worm, fly and yeast, and found little evidence of whole-genome duplication. Instead, their results support several independent duplications of individual genes and the occasional duplication of chromosomal blocks.

EPIGENETICS

Lsh, a member of the SNF2 family, is required for genome-wide methylation.

Dennis, K. *et al. Genes Dev.* **15**, 2940–2944 (2001)

This paper adds to the growing evidence that chromatin-remodelling proteins can influence DNA methylation, when once the opposite was believed to be true (see also accompanying Highlight on histone methylation). Dennis *et al.* knocked out *Lsh* — a member of the SNF2 family of chromatin remodelling proteins — in mice to create animals that develop normally but have genome-wide hypomethylation, indicating that altering chromatin structure can affect global genomic methylation, which appears not to be essential for mouse embryogenesis.