EVOLUTION

Flower colour power

Image courtesy of D. Schemske and T. Bradshaw.

Turning down flowers on the basis of their colour is unlikely to raise your popularity stakes. Yet, some animal pollinators are adapted for this particular choosiness, which enables them to single out the specfic plant species that they help to propagate by their colour. For example, the red-flowered species of the monkeyflower genus *Mimulus* is visited exclusively by hummingbirds, whereas its pink sister species is selectively pollinated by bumblebees. Such fastidiousness ensures the reproductive isolation of the two flower species in overlapping habitat ranges. By experimentally switching the colour of the two types of *Mimulus,* Toby Bradshaw and Doug Schemske have now shown that the adaptation of each plant species to a specific pollinator might be due, in large measure, to a mutation at a single locus that affects flower colour.

The *YUP* (yellow upper) gene controls whether yellow pigment is deposited in flowers: the pigment is present in the red, hummingbird-pollinated *M. cardinalis*, but absent in the pink, bumblebee-pollinated *M. lewisii* (shown in the figure). The authors substituted the *YUP* allele of one species by crossing it into the near-isogenic line of the other, to create pale, yellow-orange *M. lewisii* and dark-pink *M. cardinalis*. The effect of this colour change on pollinator presence was striking: bumblebees preferred the 'mutant' pink *M. cardinalis* — the species normally selected by birds — over the red, wild-type variety by 74-fold, and hummingbirds visited the 'mutant' yellow-orange *M. lewisii* — the species normally favoured by bees — 68 times more than the pink, wild-type one. That the effect was so large and symmetrical reinforces the idea that mutations in *YUP* have a marked effect on pollinator preference and that this locus alone — under the right ecological conditions — could initiate an adaptive shift in pollinator choice.

POPULATION GENETICS

Evolution, the passive way

From the prokaryotic mayhem of the primordial soup emerged the multicellular eukaryotes, which occupy more space, do cleverer tasks and have bigger and more complex genomes. But what route did phenotypic evolution take in going from one to the other? Did adaptive diversification depend on having a more complex genome or was it the other way around? Mike Lynch and John Conery provide statistical evidence that the more complex genomes of multicellular eukaryotes arose passively and therefore without much adaptive purpose. They propose that only once the new genomic features were in place would they have been exploited for adaptive purposes.

The genomes of multicellular eukaryotes are not only much bigger than unicellular ones, but they also have more genes, more introns and more mobile elements. Although there are plausible advantages that these features would bring, it is also possible that more complex genomes arose because there was nothing to prevent them from arising. The 'something' that could prevent this is purifying selection, which purges undesirable variants from a population.

Purifying selection is less powerful in smaller populations, in which traits have a better chance of spreading through the population owing to random forces. The authors have calculated that as organisms get larger, on average their population size gets smaller. They show that the effective population size (N_e) — the number of individuals that actually contribute to the next generation — can vary by several orders of magnitude between the largest and smallest organisms. So, multicellular species, with their much smaller N_e — which, in turn, is probably caused by their larger cell and body sizes — would be freer to accumulate non-selected changes to their genome.

The authors tested their theoretical expectations against the characteristic features of multicellular genomes. For example, they show that multicellular species probably have more genes because they retain duplicated genes longer than do unicellular species, as mutations take longer to erode them. So, rather than one copy of the duplicate pair degenerating out of existence, both could survive by splitting between them the role of the ancestral locus (through a process known as 'subfunctionalization'). A similar sort of reasoning can be proposed to explain the emergence of a large number of introns and mobile elements.

Of course, the idea is not that all complex traits arose by chance — rather, it is that a non-adpative expansion in the genome provided the genetic raw material for selection to work on. For example, it is perfectly feasible that once a large number of introns were present, they would be put to use in alternative splicing, thereby paving the way for more adaptive evolutionary changes. As the authors point out, more directed experiments are needed to prove their model and that 'exceptional species' within each group should be good testing ground for their theories.

Tanita Casci

References and links

ORIGINAL RESEARCH PAPER Lynch, M. & Conery, J. S. The origins of genome complexity. *Science* **302**, 1401–1404 (2003)

WEB SITES

Mike Lynch's laboratory:
http://www.bio.indiana.edu/facultyresearch/faculty/Lynch.html

John Conery's home page:
http://www.cs.uoregon.edu/~conery

Could Ronald A. Fisher — the innovative twentieth-century statistician and evolutionary biologist — possibly have got it wrong? His theory that adaptation proceeds by the gradual accumulation of an infinite number of infinitesimally small steps might now have to give way to the increasingly popular view that the same result can be achieved by taking just a few, perhaps even one, big step. How close this study of pollinator shifts has come to demonstrating such a paradigm shift will depend on identifying additional mutations in the 'adaptive walk' from bumblebee to hummingbird pollination.

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References and links

ORIGINAL RESEARCH PAPER Bradshaw, H. D. Jr & Schemske, D. W. Allele substitution at a flower colour locus produces a pollinator shift in monkeyflowers. *Nature* **426**, 176–178 (2003)

WEB SITES

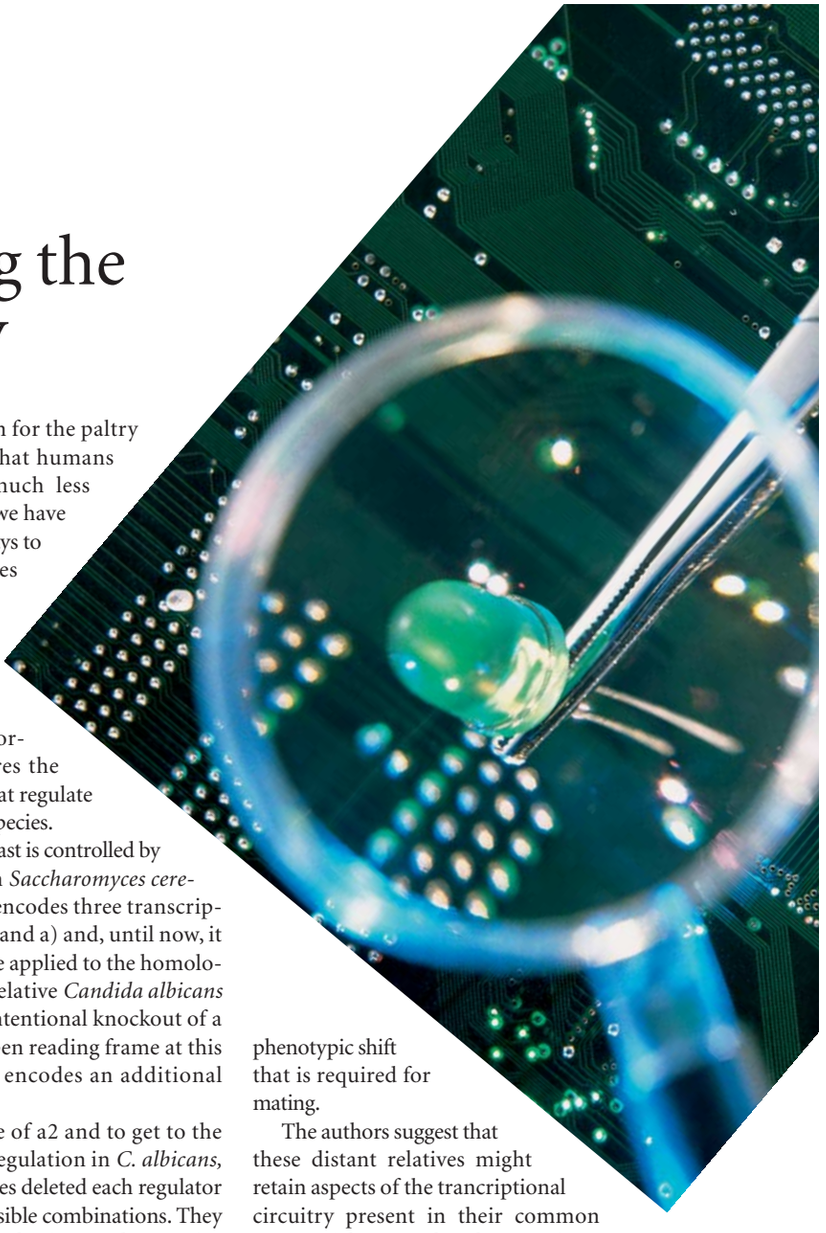
Toby Bradshaw's laboratory:
http://faculty.washington.edu/toby

Doug Schemske's laboratory:
http://www.plantbiology.msu.edu/schemske.shtm

***Mimulus* page:**
http://www.ex.ac.uk/~MRMacnai/mimulus.html

GENE REGULATION

Rewiring the circuitry



One possible explanation for the paltry number of extra genes that humans have, compared with much less complex animals, is that we have evolved many different ways to regulate the same genes during development. Just how important evolutionary rewiring of the regulatory circuitry can be is now evident from a new and thorough study that compares the transcriptional circuits that regulate mating type in two yeast species.

Mating behaviour in yeast is controlled by the mating-type locus. In *Saccharomyces cerevisiae*, this locus (*MAT*) encodes three transcriptional regulators (α1, α2 and a) and, until now, it was thought that the same applied to the homologous locus in its distant relative *Candida albicans* (*MTL*). However, an unintentional knockout of a previously overlooked open reading frame at this locus showed that *MTL* encodes an additional regulator (a2).

To investigate the role of a2 and to get to the bottom of mating-type regulation in *C. albicans*, Annie Tsong and colleagues deleted each regulator individually and in all possible combinations. They then analysed the mating behaviour and transcriptional profiles of the 16 possible mutant strains.

Their complete mating-behaviour data set allowed the authors to determine that a2 and α1 are positive regulators of their respective mating types, whereas a1 and α2 each contribute one half of a heterodimer that negatively regulates the ability to switch from the white phase to the opaque phase that is needed to mate efficiently. By contrast, in *S. cerevisiae*, which lacks a2, cells default to the a-mating-type when *MAT* does not contribute anything, and when α2 is present, it acts as a negative regulator of a-type-mating.

Together with gene-expression data, these mating experiments provide some unique insights into the regulation of mating. Even more interesting than the differences in the control of individual genes in *C. albicans* and *S. cerevisiae* were the differences in transcriptional circuitry. For example, a-specific genes are under negative control in *S. cerevisiae*, but in *C. albicans* they are under positive control. There is also an extra level of mating control present in *C. albicans* that is reflected in the white-to-opaque phenotypic shift that is required for mating.

The authors suggest that these distant relatives might retain aspects of the trancriptional circuitry present in their common ancestor. They postulate that negative regulation of a-specific genes in the *S. cerevisiae* lineage replaced the ancestral positive regulation (still retained in *C. albicans*), whereas white-opaque switching in the latter is likely to be a recent adaptation to life in mammalian hosts, now coupled to a much more ancient regulatory circuit, also for adaptive reasons.

So, mixing and matching regulatory-circuit elements seems to be a viable evolutionary means of increasing complexity and adapting to new environments. The remaining question seems to be one of scale: can such transcriptional rewiring explain differences in complexity as large as those that are found between humans and yeast?

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References and links

ORIGINAL RESEARCH PAPER Tsong, A. E. *et al.* Evolution of a combinatorial transcriptional circuit: a case study in yeast. *Cell* **115**, 389–398 (2003)

FURTHER READING Johnson, A. The biology of mating in *Candida albicans*. *Nature Rev. Micro.* **1**, 106–116 (2003)

WEB SITE

Sandy Johnson's laboratory:
http://itsa.ucsf.edu/%7Emicro/Faculty/Johnson/johnson_index.html