

populations actually undergo an 'adaptive suicide'. A degrading environment can obstruct the evolutionary path of a dispersal trait towards more viable rescue states. From within such an 'adaptive trap', gradual evolution of dispersal can no longer prevent population extinction.

In this workshop, the recent rise of adaptive dynamics theory was very apparent, with many speakers using this tool to explore different aspects of dispersal evolution. In the real world, however, detailed knowledge about dispersal in many organisms remains scarce. Some contributions suggested that new techniques, such as those from molecular biology, might help to overcome this shortage. It will remain a challenge to integrate the various approaches presented, so that more theoretical predictions can be tested in the field. A forthcoming symposium* in France will provide the next opportunity to see how close we are to finding a unifying approach in the study of dispersal.

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Cloning *Odysseus* and the seed of speciation

The origin of new species, although a core subject in evolutionary biology, continues to mystify. One hundred and sixty years ago, John Herschel referred to speciation as 'that mystery of mysteries', and Darwin echoed him¹. In 1922, William Bateson said, 'that... bit of the theory of evolution which is concerned with the origin and nature of species remains utterly mysterious'². Even as recently as 1974, Richard Lewontin stated that 'we know virtually nothing about the genetic changes that occur in species formation'³.

Lamenting our ignorance about speciation appears to be a habitual penance for evolutionary biologists. Although Lewontin might have been overly pessimistic at the time, it is certainly true that our understanding of the fine workings of speciation has progressed only slowly. Over the past two decades, studies of natural and experimental hybridization have given some information on the

numbers and locations of genes involved in reproductive isolation. But only now has a study appeared that dissects speciation genes with molecular precision⁴, rewarding us with unexpected insights.

Better insights are desperately needed because, since the years that the New Synthesis was conceived, many different speciation models have accumulated in the literature. Although nature is so multifarious that no single unified speciation theory can exist, this plethora of models has sometimes caused more confusion than might be necessary. Fortunately, researchers are beginning to recognize that some speciation models are more plausible than others⁵, and most are best viewed as extreme points in a landscape of speciation possibilities, largely determined by two forces: gene flow and selection.

One corner of the speciation landscape is occupied by sympatric speciation, where strong selection acts on populations to diversify despite considerable

gene flow. The theory of sympatric speciation has only just started to be taken seriously, and attempts to identify the 'speciation genes' are barely under way⁶. Progress has been faster on the other edge of the speciation landscape, where classic allopatric speciation sits. In the complete absence of gene flow, any amount of selection will make populations diverge genetically, which might result in pre- and/or postzygotic isolation. The type and intensity of selection, however, have been controversial⁵, but might be understood if the speciation genes responsible are identified.

But identifying them is difficult. In principle, a speciation gene could be any sort of gene, or even an extranuclear factor, such as the incompatibility-causing symbiont *Wolbachia*⁷. Moreover, they cannot be found by simply pinpointing the genes responsible for reproductive isolation, because many of their differences could have accumulated after speciation took place. So, the best way to go about the tracking down of speciation genes is by studying sibling species that have attained only partial reproductive incompatibility. In dioecious animals, this incompatibility usually follows Haldane's Rule: sterile hybrids belong to the heterogametic sex only⁸.

The fruit flies *Drosophila simulans* and *D. mauritiana* are probably the best-studied pair of sibling species. The former is a cosmopolitan human commensal, whereas the latter is an island endemic, presumably isolated half a million years ago⁴ (Mya). When crossed, the males suffer spermatogenic defects and are sterile, whereas the females retain normal fertility. This has allowed geneticists to map the putative speciation genes by backcrossing hybrid females with parental males and thus introgressing portions of the genome of the one species into the genetic background of the other.

A particularly impressive series of such introgressions has been carried out by a group led by Chung-I Wu at the University of Chicago. Having located a range of speciation genes, they conclude that hybrid male sterility is caused by no less than 120 genes, scattered across the genomes of both species⁸. Most of these appear to have relatively small effects individually but exhibit strong epistatic interactions⁹. An X-linked locus called *Odysseus* is such an example. Like its mythological namesake, it causes havoc when introduced in the foreign genetic environment: it induces approximately a 40% reduction in male fertility. But its effect can be moderated or aggravated if it is accompanied by nearby co-introgressed genes.

Homeobox divergence

In a paper in *Science*, Ting *et al.* now report the cloning of *Odysseus*⁴. Having narrowed down the locus to an 8.4 kb region, they found it to possess three open reading frames, the 349 amino acids long transcript of which unexpectedly contained the 60 or so amino acids that characterize homeobox genes.

The group then sequenced part of the gene in all four species that make up the *melanogaster* clade, to which *simulans* and *mauritiana* belong. The homeobox turned out to be highly divergent among the four species, the largest difference being 15 amino acid substitutions between the sibling species *simulans* and *mauritiana*. Homologues of the gene are found in mouse, rat and the worm *Caenorhabditis elegans*, where it is expressed in neural, rather than reproductive, tissues, and where it is extremely conserved. For example, the sequence difference between *simulans* and *mauritiana* (diverged 0.5 Mya) is twice as large as that between mouse and *C. elegans* (diverged c. 700 Mya)!

The authors convincingly argue that the sequences compared in *Drosophila* are true homologues. For example, the difference in the introns between *simulans* and *mauritiana* is only 1.4%, exactly

conforming to the neutral rate. In addition, they sequenced multiple alleles per species, which means that the differences between the species are not the result of sampling within-species polymorphisms.

The gene (which Ting *et al.* refer to as *OdsH* – *Odysseus*-site Homeobox gene) thus appears to have undergone a dramatic increase in evolutionary rate in this species group. The increase has been driven by directional selection rather than a relaxation of stabilizing selection, as is proved by the overwhelming prevalence of substitutions that cause an amino-acid change over those that do not (at least in the lineage leading to *mauritiana*). What remains is the tantalizing question: what sort of selection could have propelled an otherwise strongly conserved regulatory gene across long mutational distances to cause hybrid male sterility?

Rapidly evolving homeobox genes that are expressed in male reproductive tissues have been found before. A gene called *Pem* is expressed in the testes of rat and mouse and its homeobox differs by 24 amino acids between these two species¹⁰. Sperm proteins in *Drosophila* and a mollusc are also known to evolve uncharacteristically fast^{11,12}.

These observations form a molecular extension of the taxonomists' old rule of thumb that male genitalia and secondary sexual traits are the best characters to distinguish otherwise similar species¹³. Such morphological traits are obviously the product of sexual selection by male competition and female choice. It is becoming increasingly clear, however, that sexual selection does not only act on gross reproductive morphology, but on all aspects related to competition for mates, including the chemistry of the ejaculate¹⁴.

Sexual selection can cause reproductive traits to change rapidly and unpredictably. In allopatric populations, this can lead to large differences in mate-recognition, which can cause behavioural incompatibility, such as in the various *Drosophila* species that have evolved prezygotic isolation before postzygotic isolation¹⁵. It seems likely that many speciation genes that cause hybrid-male sterility are regulators of aspects of sperm production that have somehow been under sexual selection^{4,8}.

At the moment, this hypothesis is still speculative, however attractively unifying it might be. The function of genes such as *OdsH* and *Pem* should be studied further. It would also be interesting to see their effects on male fertility in related species, which could be studied by transgenesis. In addition, experimental studies could be devised where populations are kept under the same external selection,

but with different opportunities for sperm competition. If this line of research is followed, it might bring us closer to solving that mystery of mysteries.

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