EVOLUTION

From organelle to nucleus

In a reconstruction of events that took place early in the evolution of eukaryotic cells, a new study has shown how the transfer of DNA from an organelle to the nucleus can give rise to functional genes.

After the initial uptake of endosymbiotic bacteria that led to eukaryotic evolution, DNA transfer took place between newly formed organelles and host nuclear genomes. Stegemann and Bock used cultured tobacco cells carrying a construct that represents the transfer of a plastid gene to a position next to a nuclear gene. The construct was moved from transgenic chloroplasts to the nuclear genome and consists of one gene, *aadA*, in a cassette with sequences that drive its expression only in plastids, plus an upstream gene, *nptII*, which is flanked by eukaryotic promoter and termination signals.

By growing large numbers of cells in the presence of the two antibiotics to which *aadA* encodes resistance, the authors selected lines in which *aadA* had become functional, with expression from the nuclear genome. In all cases, rearrangements had placed *aadA* under the control of the *nptII* promoter; however, the signals that were needed for mRNA 3′ end formation had been provided by the plastid 3′ flanking sequences. The authors postulate that the AT-richness of plastid non-coding regions might make it easier for chloroplast genes to take on eukaryotic-type mRNA cleavage and polyadenylation.

Although other outcomes and functionalization processes are possible, this study provides an important insight into how organellar gene functions can be taken over by the nucleus — a process that might have fundamentally shaped the biology of eukaryotic cells.

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ORIGINAL RESEARCH PAPER Stegemann, S. & Bock, R. Experimental reconstruction of functional gene transfer from the tobacco plastid genome to the nucleus. *Plant Cell* 3 November 2006 (doi:10.1105/tpc.106.046466)

GENE THERAPY

Getting into mitochondria

> …this approach could potentially be used to treat several other diseases that are caused by mitochondrial tRNA mutations.

Gene therapy options for diseases that are caused by mutations in mitochondrial DNA are limited for several reasons, not least the difficulty of getting therapeutic molecules into the organelle. A recent study shows how this problem can be overcome by co-opting a mitochondrial import mechanism from protozoan parasites.

Leishmania parasites use a specialized protein complex to import tRNAs into mitochondria. Mahata and colleagues investigated whether this complex, RIC (RNA import complex), could be used to restore mitochondrial function in cells from patients with myoclonic epilepsy with ragged red fibres (MERFF) and Kearns–Sayre syndrome (KSS). In these conditions, the *MTTK* gene, which encodes mitochondrial $tRNA^{Lys}$, is mutated or deleted, resulting in decreased translation of mitochondrial mRNAs.

The authors used 'cybrid' cell lines that were made by fusing cytoplasts (enucleated cells) from MERFF and KSS patients with a cell line that lacks mitochondrial DNA. When these cells were incubated with RIC, the complex was taken up and transported into mitochondria. Using RT-PCR to detect $tRNA^{Lys}$ in different cellular compartments, Mahata and colleagues showed that, as hoped, RIC mediates mitochondrial import of cytoplasmic $tRNA^{Lys}$.

Is RIC-mediated tRNA import sufficient to restore mitochondrial

EVOLUTION

Good mothers have bad sons

When a gene affects a trait that has an optimum value that differs between the sexes, its alleles can have opposing effects on fitness in males and females. Such intralocus sexual conflict predicts that the sons of high-fitness females and the daughters of high-fitness males will be of low fitness, undermining the effect of sexual selection. Pischedda and Chippindale directly measured fitness in *Drosophila melanogaster* to demonstrate the extent of intralocus sexual conflict and showed that it occurs mainly in X-chromosomal genes.

Many genes that affect sexually dimorphic phenotypes have evolved to be imprinted, expressed in a sex-limited manner, or inherited preferentially by the same sex. This reduces the problem of alleles that increase the fitness of one sex interfering with the fitness of the other. However, many loci are not

> … intralocus sexual conflict … can occur in any trait that is differentially selected between the sexes but is genetically constrained from diverging.

modified in these ways and there is mounting evidence that some are the focus of intralocus sexual conflict.

After screening the *D. melanogaster* genome for genetic variation in fitness, the authors selected lines of high-fitness and low-fitness males and females, and directly measured the fitness of their male and female offspring. High-fitness females produced fitter daughters but less fit sons than low-fitness females, and high-fitness males produced less fit daughters than low-fitness males. However, the sons of high-fitness males did not inherit the fitness of their fathers, which is consistent with most of the sexually antagonistic loci being on the X chromosome.

These results help explain why strong sexual selection does not eliminate variation for fitness in populations. Moreover, they might explain why the results of sexual selection are more pronounced