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tures of human mtDNA disease, were not found. Mice with a high mtDNA load died after 200 days from kidney failure, a condition not normally associated with human mitochondrial disease.

Further differences between the mouse and human phenotypes, as discussed by Eric Shoubridge in a News and Views article, indicate there may be species-specific differences in the accumulation of mutant mtDNA in certain tissues — a feature of human mtDNA disease not seen in this mouse model. MtDNA deletions are also rare in humans — point mutations and duplications predominate. The authors' data indicate that this deletion may be transmitted in the mouse germline as a partially duplicated structure, consisting of a full-length mtDNA and the deleted mtDNA, which then rearranges in somatic tissues during development to leave the mutant form.

These differences might only be resolved with the generation of a mouse that models human mtDNA point mutations. Meanwhile, many questions about the transmission and segregation of deleted mtDNA, and its role in kidney disease, ageing and age-related neurodegeneration, could be addressed in these mice.

Jane Alfred

References and links

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WEB SITE MITOMAP — A human mitochondrial genome database

human mitochondria imported the yeast $tRNA^{Lys}_{CUU}$ and its derivatives, provided that the human cytosolic extracts were supplemented with the yeast pre-MSK. The foreign tRNA was functional on the translational apparatus of human mitochondria, just as in yeast.

This recent innovation might be useful for replacing non-functional tRNAs or for suppressing nonsense mutations in mtDNA.

Tanita Casci

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ORIGINAL RESEARCH PAPER Kolesnikova, O. A. *et al*. Suppression of mutations in mitochondrial DNA by tRNAs imported from the cytoplasm. *Science* **289**, 1931–1933 (2000)
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WEB SITES Thomas Fox's lab | Ivan Tarassov's lab

IN BRIEF

EVO-DEVO

Spatial expression of *Hox* cluster genes in the ontogeny of a sea urchin.

Arenas-Mena, C. et al. Development ***127***, *4631–4643 (2000).*

The patterning function of the *Hox* gene cluster has mostly been studied in animals, such as chordates and arthropods, which have two-fold symmetry around the adult anterior–posterior axis. The embryo and larva of the sea urchin are bilaterally symmetrical, but develop into five-fold symmetrical adults. Phylogenetic studies suggest that the five-fold axis of symmetry evolved from a bilateral ancestor. The theory is now clearly supported by the expression of five sea urchin *Hox* genes during the development of the adult body plan.

GENE EXPRESSION

A calmodulin-related protein that suppresses posttranscriptional gene silencing in plants.

Anandalakshmi, R. et al. Science ***290***, *142–144 (2000).*

A viral movement protein prevents spread of the gene silencing signal in *Nicotiana benthamiana*.

Voinnert, O. et al. Cell ***103***, *157–167 (2000).*

Post-transcriptional gene silencing (PTGS) in plants is an anti-viral defence system that directs the sequence-specific degradation of target RNAs. Using a yeast two-hybrid assay, with a viral suppressor of PTGS (HC-Pro) as a bait, Anandalakshmi and colleagues have discovered the first cellular suppressor of silencing. This protein, called rgs-CaM, might point to a role for calcium in regulating the PTGS pathway. PTGS is non-cell-autonomous, as the signal of gene silencing (probably a nucleic acid) can move long distances through the plant vasculature, to potentiate RNA sequence-specific virus resistance in as yet uninfected tissues. Voinnert and colleagues report the finding of a 25 kDa viral protein (p25) encoded by potato virus X, which can suppress the anti-viral effect of local and systemic PTGS.

DEVELOPMENT

Combinatorial signaling in the specification of unique cell fates.

Flores, G. V. et al. Cell ***103***, *75–85 (2000).*

Overlapping activators and repressors delimit transcriptional response to receptor tyrosine kinase signals in the *Drosophila* eye.

Xu, C. et al. Cell ***103***, *87–97 (2000).*

How do multiple signals specify unique cell fates during development? These papers address this issue, in the *Drosophila* eye. In this system, cell-type specific transcription factors have been shown to be important in cell fate specification. The two papers demonstrate how signals (such as Notch and EGF) are interpreted in a combinatorial fashion and lead to the expression of the appropriate transcription factor. The implication is that a small number of signals can be integrated to generate a variety of cell-specific outcomes.