Prospects for the golden monkey

Photo courtesy of Xiao-feng Ma

***Rhinopithecus roxellana*, the iconic golden monkey of mainland China, is an endangered species with an estimated population size of 10,000–20,000. Although efforts to assess its population history and remaining genetic diversity have been hampered by a scarcity of samples, Haipeng Li and colleagues now have evidence for at least one severe population bottleneck in the *R. roxellana* population, possibly within the last 15,000 years (*Genetics* 164, 269–275; 2003). After two years of effort, Li *et al.* collected 32 blood samples throughout the primary habitat in Sichuan province. They examined 44 loci and found that none were polymorphic, and overall *R. roxellana* has less genetic diversity than the endangered giant panda. Based on the current census size, the generation time and the estimated mutation rate, the authors argue that this lack of heterozygosity is most compatible with a bottleneck within the last 15,000 years, when the effective population size probably dropped to fewer than 1,000. Despite the relatively large current population, Li *et al.* propose continued vigilance in conservation efforts, particularly focused on the promotion of genetic diversity.** *AP*

The versatile semaphorins

Semaphorins, a family of secreted and cell-surface molecules, have an important role in guiding neural growth cones to their targets. Wataru Shoji and colleagues have now provided direct evidence that Semaphorin3a1 is also required for angioblast migration and formation of the dorsal aorta in zebrafish (*Development* **130**, 3227–3236; 2003). Angioblasts normally arise in the lateral mesoderm during gastrulation and migrate to the midline where they differentiate into blood and endothelial cells. Induced ubiquitous expression of *Sema3a1* in zebrafish embryos inhibited the migration of angioblasts, as did knockdown of gene expression with antisense morpholinos. Shoji *et al.* also observed severe restriction of the dorsal aorta and a complete absence of circulation. They propose two possible models for the effect of Sema3a1 on migrating angioblasts. The first involves direct repulsion by Sema3a1—a possibility because angioblasts express neuropilin 1, a component of the Sema3a1 receptor. As neuropilin 1 is also part of the vascular endothelial growth factor (VEGF) receptor, Sema3a1 may also interfere with the chemoattractant activity of VEGF by competing for binding to neuropilin 1. *AP*

Some salient genetics

To survive, a fruit fly has to be able to selectively orient its attention toward some conspicuous visual stimulus that might, for example, represent a source of food. A recent study by van Swinderen and Greenspan reports the first physiological marker for this behavior in flies and a first attempt at elucidating the underlying molecular mechanisms (*Nat. Neurosci.* **6**, 579–586; 2003). The authors measured voltage differences between two electrodes placed in *Drosophila melanogaster* brains in response to various visual and other stimuli. They identified a specific response, in the 20–30 Hz range, that correlates with the conspicuousness (or 'salience') of objects. Several features of the *D. melanogaster* 20–30 Hz response are similar to the physiological correlates of selective attention in monkeys and humans, suggesting that flies and primates may use analogous mechanisms. By testing fly strains that carry temperature-sensitive mutations in membrane conductance or synaptic release targeted to specific regions of the brain, the authors were then able to determine that the 20–30 Hz response originated from the mushroom bodies—structures in fly brains implicated in olfactory learning. By providing the first physiological assay for salience, this study may open the way to the molecular genetic dissection of perception in the fly. *LB*

A function for mammalian miRNA

In mammals, the role of the more than 200 known micro RNAs (miRNAs) has remained opaque. A recent paper in *Nature* (advance online publication 8 June 2003; doi: 10.1038/nature01730) from Hiroaki Kawasaki and Kazunari Taira identifies a role for a human miRNA in translational regulation. The authors identified *HES1*, encoding a transcriptional repressor found in undifferentiated cells, as a potential target of miR-23 by searching for sequence complementarity. In NT2 cells, which differentiate into neural cells on addition of retinoic acid, miR-23 was absent in undifferentiated cells but present in differentiated cells—corresponding with the presence and absence, respectively, of HES1. Introduction of synthetic miR-23 into NT2 cells corresponded with a drop in HES1 protein levels, but *HES1* mRNA levels remained constant. siRNA knockdown of miR-23 in differentiated NT2 cells resulted in an increase in HES1 levels—again without affecting *HES1* mRNA levels. When this experiment was carried out in undifferentiated NT2 cells, the cells did not differentiate after treatment with retinoic acid. Thus, the authors conclude that miR-23 regulates *HES1* at the translational, rather than transcriptional, level and has a role in regulating retinoic acid–induced neuronal differentiation. Identifying how miR-23 accomplishes this represents the next challenge. *DG*

Mouse model of lupus

People with systemic lupus erythematosus often have antibodies against a 60-kDa RNA-binding protein called Ro. The protein has been shown to be specifically associated with a class of small RNAs of unknown function called Y RNAs, as well as with presumably misfolded 5S ribosomal RNAs. Thus it has been proposed that Ro has the role of quality controller during ribosomal biogenesis. In a recent paper, Dahai Xue and colleagues show that mice lacking Ro protein develop an autoimmune syndrome characterized by antibodies to ribosomes and chromatin as well as glomerulonephritis and, when backcrossed, sensitivity to ultraviolet radiation—all features common in individuals with lupus (*Proc. Natl. Acad. Sci. USA* advance online publication 3 June 2003; doi: 10.1073/pnas.0832411100). The authors speculate that subtle structural changes in ribosomes caused by the inclusion of misfolded 5S RNAs that would normally be targeted for degradation by Ro could result in autoimmunity. As the human gene encoding Ro maps to chromosome 1q31, which has been linked to lupus, it will be interesting to see if Ro has a role in the etiology of the disease in humans. *MS*

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