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URLs

IN BRIEF

SMALL RNAs

MicroRNA pathways modulate polyglutamine-induced neurodegeneration.

Bilen, J. *et al. Mol. Cell* **24**, 157–163 (2006)

This paper demonstrates a role for miRNAs in protection from neurodegeneration, in both fly and human cells. Bilen *et al.* showed that the impairment of miRNA processing, but not that of siRNA, dramatically increases the neurodegeneration that is induced by pathogenic proteins such as the polyQ-containing Ataxin-3 or tau. In a parallel genetic screen in *Drosophila*, the authors identified one particular miRNA, *bantam* (*ban*), which has the ability to modulate both polyQ and tau toxicity, acting downstream of the pathogenic protein accumulation.

CLONING

Differentiated cells are more efficient than adult stem cells for cloning by somatic nuclear transfer.

Sung, L-Y. *et al. Nature Genet.* 1 October 2006 doi:10.1038/ng1895

The limited success of somatic cell nuclear transfer (SCNT) has been attributed to, among other things, the fact that some of the donor nuclei come from adult stem cells. Moreover, the efficiency of reproductive cloning is 5–10 times higher with embryonic stem cells than with somatic donors. The authors tested whether SCNT using fully differentiated cells can result in cloned animals. Using mouse haematopoietic stem cells at different differentiation stages, they show that cloning efficiency increases with more differentiated cell types, thereby revising the current thinking.

DEVELOPMENTAL GENETICS

Generation of robust left–right asymmetry in the mouse embryo requires a self-enhancement and lateral-inhibition system.

Nakamura, T. *et al. Dev. Cell* **11**, 495–504 (2006)

The bilateral symmetry of the mouse embryo is broken by the leftward flow of fluid in the node cavity — the nodal flow. This initial asymmetry is translated into subsequent left-sided expression of *nodal* and left–right asymmetry. By manipulating expression of *nodal* and *lefty* and by using mathematical modelling, these authors show that the nodal flow represents an initial small difference between the left and right side of the embryo; subsequent asymmetry is a result of self-enhancement and lateral-inhibition between *nodal* and *lefty*, which amplify the initial bias.

DISEASE GENETICS

Rescue of progeria in trichothiodystrophy by homozygous lethal *Xpd* alleles.

Andressoo, J. O. *et al. PLoS Biol.* **4**, e322 (2006)

This study indicates a contribution of inter-allelic complementation to genetic disease. Mutations in *XPD* cause three distinct recessive disorders: trichothiodystrophy (TTD), xeroderma pigmentosum (XP) and Cockayne syndrome (CS). The authors used mice that were compound heterozygotes for the TTD mutation and either the CS or XP mutations. These mice showed phenotypes that were closer to the wild type than the homozygous combinations. In light of these findings, recessive alleles that were previously identified as 'null' should be considered as contributors to organismal phenotype and disease outcome in compound heterozygous patients.

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