

IN BRIEF

DOI:

10.1038/nrg2156

URLs

RNA INTERFERENCE

RNA interference-mediated suppression and replacement of human rhodopsin *in vivo*.

O'Reilly, M. *et al. Am. J. Hum. Genet.* **81**, 127–135 (2007)

Mutational heterogeneity presents a challenge to the therapeutic correction of genetic disease. In mice carrying a dominant mutation in the rhodopsin gene, which is mutated in retinitis pigmentosa, the authors suppressed wild-type and mutant rhodopsin expression by RNAi. The simultaneous expression of a replacement rhodopsin gene that was refractory to RNAi owing to a modified codon composition resulted in the expression of functional rhodopsin. The same therapeutic molecules could be used to correct a range of rhodopsin mutations.

DEVELOPMENT

Transcription factor modularity in a gene-centered *C. elegans* core neuronal protein–DNA interaction network.

Vermeirssen, V. *et al. Genome Res.* 22 May 2007 (doi:10.1101/gr.6148107)

This study describes the mapping of a core network of transcription factors and their target genes in *C. elegans* sensory neurons, providing insights into how the architectures of such networks relate to their functions. The network consists of two distinct modules: one contains transcription factors that are involved in reproduction and target genes that are expressed in both neurons and other tissues, whereas the other is enriched for transcription factors with targets that are mainly expressed in neurons.

EPIGENETICS

DNA damage, homology-directed repair and DNA methylation.

Cuozzo, C. *et al. PLoS Genet.* 22 May 2007 (doi:10.1371/journal.pgen0030110.eor)

This work provides a link between DNA repair and DNA methylation. The authors showed that, in mouse and human cells, DNA double-strand breaks are repaired through homology-directed repair and half of the repaired molecules are marked by *de novo* DNA methylation. This occurs independently of the methylation status of the template DNA, and can silence the repaired gene. This mechanism can alter the overall expression of genetic information; therefore, if expressing the repaired gene is harmful, cells that inherit the silenced repaired gene have selective advantages.

TECHNOLOGY

Genome-wide mapping of *in vivo* protein–DNA interactions.

Johnson, D. S., Mortazavi, A., Myers, R. M. & Wold, B. *Science* 31 May 2007 (doi:10.1126/science.1141319)

A new method, ChIPSeq, involving chromatin immunoprecipitation and ultra-high-throughput DNA sequencing allows high-resolution genome-wide mapping of protein–DNA interactions. ChIPSeq has several advantages over methods that couple ChIP to microarray analysis: for example, it can be performed on any sequenced genome and single-copy sites that might be under-represented in microarrays are accessible. Through ChIPSeq, the authors identified 1,946 binding sites for the neuron-restrictive silencer factor (NSFR) and several previously unknown NSFR targets.