

IN BRIEF

FUNCTIONAL GENOMICS

Functional genomic analysis of *C. elegans* molting.

Frand, A. R. *et al. PLoS Biol.* **3**, e312 (2005)

These authors use a genome-wide RNA-interference screen to discover the genes that are involved in nematode moulting, including the endocrine control of the process and the synthesis and degradation of the exoskeleton. They used a GFP reporter to assess the timing of expression and the epistatic interactions of the key genes, allowing them to propose regulatory networks that govern moulting. They note that these genes are conserved in parasitic nematodes and are therefore potential therapeutic and pesticide targets.

GENE NETWORKS

A network-based analysis of systemic inflammation in humans.

Calvano, S. E. *et al. Nature* 31 August 2005 (doi:10.1038/nature03985)

Microarray analyses notoriously produce data sets that are too large to extract meaningful information about specific biological processes. The authors tackle this problem by combining their microarray time-course with an interactome database of published data, thereby limiting possible hypotheses to those that are biologically plausible. By using existing knowledge of protein–protein and transcriptional interactions in this way, they deduce from the microarray data that human leukocytes dysregulate their bioenergetics, protein synthesis and protein degradation in response to systemic inflammation.

GENOMICS

Genome streamlining in a cosmopolitan oceanic bacterium.

Giovannoni, S. J. *et al. Science* **309**, 1242–1245 (2005)

The bacterium with the smallest genome has been identified as *Pelagibacter ubique*, an α -proteobacterium that makes up 25% of all microbial cells. Although it is small (1.3 Mb), this genome codes for all 20 amino acids and for most basic metabolic functions, and owes its svelte nature to the lack of non-coding DNA such as transposons and pseudogenes. The genome might have been streamlined by pressure to reduce the amount of non-functional DNA that is replicated, as would be expected in large populations.

GENOME BIOLOGY

Inactivation of the SR protein splicing factor ASF/SF2 results in genomic instability.

Li, X. & Manley, J. L. *Cell* **122**, 365–378 (2005)

SR proteins are ubiquitous pre-mRNA splicing factors, but the authors suggest a surprising role for the prototypical SR protein, ASF/SF2 (alternative splicing factor/splicing factor 2), in genome stability in animals. Depletion of ASF/SF2 causes a hypermutation phenotype that depends on genome rearrangements, which are caused by hybrid molecules forming between a nascent RNA and a non-template DNA strand. The authors show that ASF/SF2 might normally be recruited to nascent transcripts to prevent the formation of such mutagenic hybrids.