IN BRIEF

MICRORNA

Silencing of microRNAs *in vivo* with 'antagomirs'.

Krutzfeldt, J. *et al*. *Nature* 30 Oct 2005 (doi:10.1038/nature04303)

Regulation of gene expression by non-coding microRNAs (miRNAs) is important for many biological and disease processes. Jan Krutzfeldt and colleagues now report the development of antagomirs: a class of single-stranded RNA analogues that are complementary to miRNAs, and that are chemically modified and cholesterol conjugated to improve their stability and delivery to cells. They show that antagomirs can specifically silence miRNAs *in vivo*, and could therefore prove a powerful tool for miRNA study as well as provide a possible therapeutic strategy.

METABOLISM

Logic of the yeast metabolic cycle: temporal compartmentalization of cellular processes.

Tu, B. P. *et al*. *Science* 27 Oct 2005 (doi:10.1126/science.1120499)

Steven McKnight and colleagues show that budding yeast undergoes a robust ultradian metabolic cycle of ~4–5 hours in length, which persists indefinitely when cultures are continuously supplemented with glucose. The metabolic cycle is accompanied by a highly organized transcriptional cycle, which involves over half the organism's genes. Key cellular processes occur in synchrony over this cycle, and are therefore compartmentalized in time.

ENDOCYTOSIS

A modular design for the clathrin- and actin-mediated endocytosis machinery.

Kaksonen, M., Toret, C. P. & Drubin, D. *Cell* **123**, 305–320 (2005)

David Drubin and co-workers carried out a comprehensive analysis of the endocytosis machinery in budding yeast. The dynamics of the previously defined budding yeast endocytosis pathway were examined in 61 deletion mutants, and 15 proteins that function in this pathway were identified, 8 of which the authors analysed in detail. These proteins can be grouped into four cooperative, functional modules based on their dynamic behaviour.

TELOMERES

A telomeric repeat sequence adjacent to a DNA double-strand break produces an anticheckpoint.

Michelson, R. J., Rosenstein, S. & Weinert, T. *Genes Dev.* **19**, 2546–2559 (2005)

Telomeres protect the ends of chromosomes and stop them acting like DNA double-strand breaks (DSBs) and triggering the DNA-damage checkpoint. Now, Michelson *et al.* have taken a step towards clarifying how telomeres achieve this with their discovery that a telomeric repeat sequence might inhibit this checkpoint. DSBs up to 600 bp away from the repeat sequence produced a severely limited checkpoint response, which indicates this locus might have 'anticheckpoint' activity.