IN BRIEF

GENOMIC INSTABILITY

HCLK2 is essential for the mammalian S-phase checkpoint and impacts on Chk1 stability

Collis, S. J. *et al. Nature Cell Biol.* **9**, 391–401 (2007)

Simon Boulton and colleagues have shown that CDC-like kinase 2 (CLK2), homologous to the *Caenorhabditis elegans* biological clock protein CLK2, functions in the DNA damage response to replication stress. Cells deficient in *CLK2* accumulate DNA damage and fail to activate the S-phase checkpoints as well as key members of the Fanconi anaemia and homologous recombination DNA repair pathways, which resulted from the defective regulation of CHK1 stability. This indicates that *CLK2* mutations could account for a subset of patients with Fanconi anaemia and possibly other DNA-damage-response disorders, such as Seckel syndrome.

SIGNALLING

c-Src activates endonuclease-mediated mRNA decay

Peng, Y. & Schoenberg, D. R. *Mol. Cell* **25**, 779–787 (2007)

Peng and Schoenberg have now shown that the *Drosophila melanogaster* mRNA endonuclease, polysomal ribonuclease 1 (PMR1), is activated by phosphorylation and that activated SRC is the kinase responsible. SRC is commonly overactive in cancer cells, and the authors showed that the epidermal growth factor-mediated activation of SRC induced the phosphorylation of exogenously expressed PMR1, and therefore increased mRNA decay. This potentially provides a new arm to the oncogenic receptor tyrosine kinase signalling networks and requires further investigation to identify the target mRNAs.

INNOVATION

High-throughput telomere length quantification by FISH and its application to human population studies

Canela, A. *et al. Proc. Natl Acad. Sci.* **104**, 5300–5305 (2007)

Understanding the influence of telomere length on cancer and other diseases associated with ageing has been limited, in part, by the inability to use telomere length as a readout for high-throughput assays. Maria Blasco and colleagues have now developed a quantitative telomere fluorescence *in situ* hybridization (FISH) platform that can sensitively and accurately measure telomere length and the proportion of short telomeres within a population of samples. This innovative technique could provide substantial information about the true association of telomere length and human disease.

TUMORIGENESIS

14-3-3σ controls mitotic translation to facilitate cytokinesis

Wilker, E. W. *et al. Nature* **446**, 329–332 (2007)

The σ isoform of the 14-3-3 proteins, which have numerous roles in cell-cycle regulation and apoptosis, is expressed in response to p53 activation, and so is frequently downregulated in cancer cells and early in tumorigenesis. Michael Yaffe and colleagues now show that the tumour-suppressor function of 14-3-3σ might derive from its ability to regulate translation factors in mitosis. The loss of 14-3-3σ impaired mitotic exit and led to the accumulation of binucleated cells, indicating a route by which tumorigenesis might occur.