IN BRIEF

RNA LOCALIZATION

Spatial regulation of β-actin translation by Src-dependent phosphorylation of ZBP1.

Hüttelmaier, S. *et al. Nature* **438**, 512–515 (2005)

The protein ZBP1 is known to promote the translocation of the β-actin transcript to actin-rich cell compartments at the cell periphery, but how it does this has been unclear. Hüttelmaier *et al.* now show that ZBP1 binds to β-actin mRNA in the nucleus and prevents premature translation by blocking translation initiation. Once the complex reaches its destination, Src kinase induces translation by phosphorylating a key tyrosine residue of ZBP1 that is required for RNA binding. So, the regulated repression and subsequent derepression of translation of an mRNA is coupled to its transport around the cell.

DNA REPLICATION

Recruitment of ORC or CDC6 to DNA is sufficient to create an artificial origin of replication in mammalian cells.

Takeda, D. Y. *et al. Genes Dev.* **19**, 2827–2836 (2005)

Takeda *et al.* hypothesized that the recruitment of replication-initiation factors to DNA might be sufficient to mimic origin-of-replication function by initiating DNA replication. This is indeed the case, as human replication-initiation factors fused to a GAL4 DNA-binding domain could stimulate the replication of plasmids that contained GAL4 DNA-binding sites. Replication occurred once per cell cycle and was regulated accurately. So, the artificial recruitment of replication-initiation factors suffices to create a functional origin of replication. This *in vitro* replication assay is likely to become an invaluable tool in DNA-replication studies.

TELOMERES

Functional human telomeres are recognized as DNA damage in G2 of the cell cycle.

Verdun, R. E. *et al. Mol. Cell* **20**, 551–561 (2005)

Telomeres differ from double-stranded DNA breaks, yet proteins of the DNA-damage-response machinery have been found to localize to telomeres. Verdun *et al.* provide new insight into this apparent paradox by showing that the telomeric structure changes in G2 phase, as indicated by the partial release of the telomeric protein POT1. This coincides with a DNA-damage response, which, surprisingly, is necessary for telomere function. The authors suggest that telomeres use the homologous-recombination DNA-repair pathway to process telomere ends after DNA replication.

MECHANISM OF DISEASE

Polycystin-1 and polycystin-2 regulate the cell cycle through the helix–loop–helix inhibitor Id2.

Li, X. *et al. Nature Cell Biol.* **7**, 1202–1212 (2005)

Autosomal-dominant polycystic kidney disease (ADPKD) is the most common hereditary kidney disease, and is caused by mutations in *PKD1* or *PKD2*, which encode polycystin-1 (PC1) and polycystin-2 (PC2), respectively. The authors show that PC1 or PC2 expression induces the phosphorylation of PC2 and its binding to the helix–loop–helix protein Id2. The PC2–Id2 interaction retains Id2 in the cytosol and prevents repression of the cyclin-dependent-kinase inhibitor p21, thereby controlling the cell proliferation that is a key feature of the disease.