IN BRIEF

TUMORIGENESIS

PCBP1 suppresses the translation of metastasis-associated PRL3 phosphatase

Wang, H. *et al. Cancer Cell* **18**, 52–62 (2010)

Phosphatase induced in regenerating liver 3 (PRL3) is overexpressed in human cancer. Zeng and colleagues identified that PRL3 is negatively regulated at a translational level by binding of polyC-RNA-binding protein 1 (PCBP1) to GC-rich motifs in the 5′ untranslated region of the *PRL3* transcript. Overexpression of PCBP1 decreased the ability of one cancer cell line to form tumours in nude mice, whereas knocking down PCBP1 promoted tumorigenesis. These data present the PCBP1–PRL3 interaction as a possible route to control tumorigenesis.

THERAPY

Mitigation of hematologic radiation toxicity in mice through pharmacological quiescence induced by CDK4/6 inhibition

Jonhson, S. *et al. J. Clin. Invest.* **120**, 2528–2536 (2010)

The efficacy of radiotherapy is limited by its severe toxic effects that mainly affect cells transitioning from the G1 phase to S phase of the cell cycle, but which do not affect cells arresting in G1. On the premise that the G1/S transition is regulated by the cyclin-dependent kinases CDK2 and CDK4, Sharpless and colleagues showed that specific inhibitors for these kinases can cause reversible G1 arrest in RB-positive human cells from bone marrow but not in RB-deficient cells. Administration of these inhibitors prior to radiation effectively protected mice from lethal myelosuppression.

CYTOSKELETON

Adenomatous polyposis coli protein nucleates actin assembly and synergizes with the formin mDia1

Okada, K. *et al. J. Cell Biol.* **189**, 1087–1096 (2010)

Loss of the effects of adenomatous polyposis coli (APC) on the cytoskeleton may contribute to cancer. Okada ***et al.*** showed that the carboxy-terminal region of APC induces actin assembly and triggers rapid polymerization (nucleation), regulating cell motility and polarity. For these effects, APC needs to synergistically cooperate with another nucleator, mouse formin mDia1, which elongates the polymer started by APC by interacting with other actin assembly-promoting factors.

SIGNALLING

NANOG regulates glioma stem cells and is essential *in vivo* acting in a cross-functional network with GLI1 and p53

Zbinden, M. *et al. EMBO J.* 25 Jun 2010 (doi:10.1038/emboj.2010.137)

Glioblastoma can be characterized by the expression of an embryonic stem cell-like gene signature (including the homeobox transcription factor *NANOG*) and the activation of the Hedgehog (HH) pathway. Zbinden and colleagues show that NANOG and the HH pathway operate in a feedback loop whereby NANOG is a target of the HH pathway and NANOG activity is required for the activity of GLI1, a transcription factor in the HH pathway.