IN BRIEF

TUMOUR SUPPRESSION

p53 suppresses retrotransposition

Wylie *et al.* show that p53 limits retrotransposition and interacts with members of the piwi-interacting RNA (piRNA) pathway in *Drosophila melanogaster* and zebrafish. Normal human *TP53* alleles suppressed retrotransposition but cancer-associated *TP53* mutants did not. Unrestrained retrotransposition was also observed in mouse and human cancers with p53 alterations, indicating that this ancestral function of p53 to suppress retrotransposition may also suppress tumorigenesis.

ORIGINAL ARTICLE Wylie, A. *et al.* p53 genes function to restrain mobile elements. *Genes Dev.* **30**, 64–77 (2016)

THERAPY

Targeting replication fork stalling

G-quadruplexes (G4) are naturally occurring DNA structures that stall replication forks. Homologous recombination (HR) stabilizes and restarts stalled replication forks and also repairs double-strand breaks (DSBs) that can form at these sites. Zimmer *et al.* show that the G4-stabilizing compound pyridostatin (PDS) induced DSB accumulation in HR-deficient cells, which in turn reduced their proliferation. HR-defective cells with certain types of resistance to the poly(ADP) ribose polymerase (PARP) inhibitor olaparib were also sensitive to PDS. This suggests that G4-stabilizing drugs could selectively target HR-defective tumour cells.

ORIGINAL ARTICLE Zimmer, J. *et al.* Targeting BRCA1 and BRCA2 deficiencies with G-quadruplex-interacting compounds. *Mol. Cell* http://dx.doi.org/10.1016/j.molcel.2015.12.004 (2015)

TUMORIGENESIS

Pathway to stemness

ID2 and hypoxia-inducible factor 2α (HIF2α) promote several characteristics of cancer stem cells, but the molecular pathways that connect the two are unclear. Lee *et al.* show that dual-specificity tyrosine-phosphorylation-regulated kinase 1A (DYRK1A) and DYRK1B phosphorylate ID2 in normoxia (but not in hypoxia). Phosphorylated ID2 is unable to bind to von Hippel–Lindau tumour suppressor (VHL), which consequently promotes HIF2α degradation. Thus, unphosphorylated ID2 promotes HIF2α activity, which can be reversed (and tumour growth inhibited) when DYRK1 kinases are overexpressed; DYRK1 expression in glioblastoma correlated with good prognosis.

ORIGINAL ARTICLE Lee, S. B. *et al.* An ID2-dependent mechanism for VHL inactivation in cancer. *Nature* **529**, 172–177 (2016)

TUMOUR MICROENVIRONMENT

Obesity promotes prostate cancer invasion

Obesity is associated with prostate cancer that is locally disseminated into the periprostatic adipose tissue (PPAT) that surrounds the prostate gland and Laurent *et al.* have found a possible explanation. Adipocytes secrete chemokine (C-C motif) ligand 7 (CCL7), which diffuses from PPAT into the prostate peripheral zone. This stimulates the migration of chemokine (C-C motif) receptor 3 (CCR3)-expressing tumour cells. This chemotaxis is increased in obesity and can be prevented when CCR3–CCL7 is blocked. CCR3 expression in human prostate cancer cells is associated with aggressive disease, local dissemination and higher risk of biochemical recurrence (denoted by changes in prostate-specific antigen (PSA) levels).

ORIGINAL ARTICLE Laurent, V. *et al.* Periprostatic adipocytes act as a driving force for prostate cancer progression in obesity. *Nat. Commun.* **7**, 10230 (2016)

CORRECTION

Obesity promotes prostate cancer invasion

Nature Reviews Cancer **16**, 70 (2016)

In the original version of this article the DOI number was incorrect. This error has been corrected in the HTML version of the article.