IN BRIEF

IMMUNOLOGY

Myeloid-derived suppressor cells are implicated in regulating permisiveness for tumor metastasis during mouse gestation

Mauti, L. A. *et al. J. Clin. Invest.* 6 Jun 2011 (doi:10.1172/JCI41936)

Breast tumours that arise during pregnancy are known to metastasize earlier than would be expected in non-pregnant women, indicating that pregnancy somehow permits tumour cell dissemination. Stamenkovic and colleagues showed that metastasis is enhanced in pregnant mice and that this correlated with decreased activity of natural killer (NK) cells. The suppression of NK cell activity seemed to be induced by myeloid-derived suppressor cells, which accumulated in premetastatic sites.

SIGNALLING

RNF20 inhibits TFIIS-facilitated transcriptional elongation to suppress pro-oncogenic gene expression

Shema, E. *et al. Mol. Cell* **42**, 477–488 (2011)

The E3 ubiquitin ligase BRE1A (also known as RNF20) monoubiquitylates histone H2B. Oren and colleagues showed that BRE1A activity suppresses the recruitment of the transcription elongation factor TFIIS, probably through H2B monoubiquitylation, and this impairs elongation by RNA polymerase II and proto-oncogene expression. As TFIIS is overexpressed in many types of cancer, these data indicate that H2B monubiquitylation and the suppression of elongation through TFIIS is a key target of BRE1A-mediated tumour suppression.

SIGNALLING

C-Raf is required for the initiation of lung cancer by K-Ras^{G12D}

Karreth, F. A. *et al. Cancer Disc.* 11 May 2011 (doi:10.1158/2159-8290.CD-10-0044)

The RAS–RAF pathway is often mutationally activated in cancer and so there are numerous efforts to target members of this pathway, such as BRAF. Investigating the roles of different RAF isoforms in RAS-driven tumours, Tuveson and colleagues found that CRAF (also known as RAF1) — rather than BRAF — is required for KRAS-G12D-mediated proliferation of primary epithelial cells. Moreover, CRAF was required for tumour formation in a mouse model of KRAS-G12D-driven lung cancer, whereas BRAF was not essential for this process. These data indicate that inhibitors of CRAF should be developed to target KRAS-driven tumours.

METABIOLISM

The androgen receptor fuels prostate cancer by regulating central metabolism and biosynthesis

Massie, C. E. *et al. EMBO J.* 20 May 2011 (doi:10.1038/emboj.2011.158)

The androgen receptor (AR) is an important therapeutic target in prostate cancer, but the AR-induced transcriptional network has not been characterized. Therefore, Mills and colleagues combined AR chromatin-binding profiles and transcription profiles to identify core AR binding sites and target genes. Subsequent metabolomic profiling revealed that AR regulates the expression of genes associated with anabolism, and further analysis showed that calcium/calmodulin-dependent protein kinase kinase 2 (CAMKK2) is a key target of AR that is overexpressed in prostate cancer and that regulates prostate cancer cell growth.