

IN BRIEF

ANGIOGENESIS**Augmentation of tumour angiogenesis by a MYC-activated microRNA cluster**

Dews, M. *et al. Nature Genet.* 30 July 2006 (doi:10.1038/ng1855)

MYC expression in tumour cells increases tumour neovascularization. Dews *et al.* show that the transduction of mouse colonocytes that express KRAS with a MYC-encoding retrovirus stimulates angiogenesis and the concomitant downregulation of anti-angiogenic thrombospondin 1 (TSP1). Antisense inhibition of the *mir-17-92* microRNA cluster, which is upregulated in the transformed colonocytes, partly restores TSP1 expression, and transduction with an miR-17-92-encoding retrovirus mimics the effect of MYC transduction, establishing a role for this microRNA in tumour angiogenesis.

TUMOUR SUPPRESSORS**A CK2-dependent mechanism for degradation of the PML tumour suppressor**

Scaglione, P. P. *et al. Cell* **126**, 269–283 (2006)

The loss of expression of PML in human cancers correlates with tumour progression. Scaglione *et al.* demonstrate that casein kinase 2 (CK2) phosphorylates the PML protein and promotes its ubiquitin-mediated degradation. The tumour-suppressor function of PML is increased in cells that express PML mutants that are resistant to CK2 phosphorylation or CK2 inhibition. This regulatory mechanism explains the inverse correlation between CK2 kinase activity and PML protein levels in human lung cancer cells.

METASTASIS**Interaction of KAI1 on tumour cells with DARC on vascular endothelium leads to metastasis suppression**

Bandyopadhyay, S. *et al. Nature Med.* **12**, 933–938 (2006)

KAI1 is a cell-surface protein associated with suppressing metastasis in various human tumours. To investigate how KAI1 achieves this, the authors used a yeast two-hybrid screen to identify KAI1-interacting proteins. Duffy antigen receptor for chemokines (DARC) is one such protein and is expressed on endothelial cells. The authors found that KAI1-expressing tumour cells that enter a blood vessel (intravasate) bind DARC and undergo senescence. Furthermore, cells that express KAI1 were able to metastasize in DARC-knockout mice, showing that the KAI1–DARC metastasis-suppression pathway is activated once tumour cells have intravasated.

CELL DEATH**DRAM, a p53-induced modulator of autophagy, is critical for apoptosis**

Crighton, D. *et al. Cell* **126**, 121–134 (2006)

The transcription factor and tumour suppressor p53 can induce cell-cycle arrest and apoptosis. Kevin Ryan and colleagues have identified a new p53 gene target, damage-regulated autophagy modulator (DRAM), which induces autophagy and contributes to p53-mediated apoptosis. The expression of DRAM mRNA is decreased in a various squamous-cell tumours with functional p53 expression, indicating that the modulation of both apoptosis and autophagy might be important for p53-mediated tumour suppression.