IN BRIEF

MICRORNA

p53-mediated activation of miRNA34 candidate tumour-suppressor genes

Bommer, G. T. *et al. Curr. Biol.* **17**, 1298–1307 (2007)

Bommer *et al.* found that p53 can directly regulate transcription of the miR-34 family of microRNAs (miRNAs) in cell lines and mice. Transcriptional arrays on colon cancer cell lines showed that these miRNAs primarily downregulate cell-cycle-associated genes. Although miR-34s induced cell-cycle arrest, they also decreased the levels of the anti-apoptotic protein BCL2, and therefore seem to mediate two key p53 functions. The expression of two miR-34s was significantly reduced in 6 of 14 non-small-cell lung cancer (NSCLC) samples, and ectopic expression of miR-34 in NSCLC cell lines inhibited their growth.

PROSTATE CANCER

A secreted isoform of ERBB3 promotes osteonectin expression in bone and enhances the invasiveness of prostate cancer cells

Chen, N. *et al. Cancer Res.* **67**, 6544–6548 (2007)

Prostate cancer preferentially metastasizes to bone. Previously, a secreted isoform of ERBB3 (p45-sERBB3) was detected in metastatic prostate cancer cells isolated from patients and was shown to interact with osteoblasts. Chen *et al.* now show that p45-sERBB3 stimulates mouse bone to secrete factors including osteonectin, which increases the invasiveness of prostate cancer cells *in vitro*. Invasion induced by p45-sERBB3 was also blocked by osteonectin antibodies. Thus, p45-sERBB3 and osteonectin might mediate the interactions between metastasizing prostate cancer cells and bone.

STEM CELLS

Tumour growth need not be driven by rare cancer stem cells

Kelly, P. N. *et al. Science* **317**, 337 (2007)

Kelly *et al.* challenge the hypothesis that growth of acute myeloid leukaemia is always sustained by a rare cancer stem cell. By titrating numbers of mouse pre-B/B lymphoma cells into non-irradiated histocompatible recipient mice, they showed that all mice developed disseminated lymphoma regardless of the number of cells injected. They suggest that mouse lymphomas can be maintained by a relatively large proportion of tumour cells, and that results of previous studies might reflect the need to co-transfer essential human support cells when using immunocompromised mice as a model.

ONCOGENES

Wild-type NRAS and KRAS perform distinct functions during transformation

Fotiadou, P. P. *et al. Mol. Cell. Biol.* 16 July 2007 (doi:10.1128/MCB.00234-07)

Why do cells express two or more wild-type Ras isoforms? Fotiadou *et al.* used a genetically-defined system of mouse embryo fibroblasts to show that both wild-type KRAS and NRAS are essential for transformation. This is because they have distinct functions: KRAS coordinates motility by signalling through Akt and CDC42, whereas NRAS regulates adhesion by signalling through Raf and RHOA.

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