IN BRIEF

ANGIOGENESIS

p53-mediated inhibition of angiogenesis through upregulation of a collagen prolyl hydroxylase

Teodoro, J. G. *et al. Science* **313** 968–971 (2006)

It has been suggested that the tumour suppressor p53 has a role in suppressing angiogenesis, but the mechanism for this is unknown. Michael Green and colleagues report in *Science* that p53 transcriptionally activates α(II) collagen prolyl-4-hydroxylase (α(II) PH), which results in the formation of anti-angiogenic collagen fragments. The intracellular expression or exogenous delivery of α(II) PH also results in the specific inhibition of tumour growth in mice. This study shows an important link between p53 signalling and anti-angiogenic collagen-fragment production.

CHECKPOINTS

BRIT1 regulates early DNA damage response, chromosomal integrity, and cancer

Rai, R. *et al. Cancer Cell* **10**, 145–157 (2006)

The DNA damage response pathway promotes genomic stability and protects against tumorigenesis. Lin and colleagues have now identified that BRIT1 is a pivotal regulator in this pathway. It is required for the formation of DNA-damage foci. The authors show that lower BRIT1 levels correlate with greater numbers of chromosomal abnormalities and an increased incidence of several cancer types. The proposed regulatory role of BRIT1 in the DNA damage response pathway explains the correlation between BRIT1 levels and cancer, and indicates that it could function as a tumour suppressor.

TUMOUR SUPPRESSORS

Inhibition of the hyaluronan–CD44 interaction by merlin contributes to the tumour-suppressor activity of merlin

Bai, Y. *et al. Oncogene.* 4 September 2006 (doi: 10.1038/sj.onc.1209849)

The tumour suppressor merlin specifically prevents the formation of nervous system tumours. This happens through an interaction with the cell-surface receptor CD44, although the mechanism is not yet known. Yu and colleagues show that merlin prevents CD44 binding to its ligand, hyaluronan, which is known to promote tumorigenesis. The first 50 amino acids of merlin are responsible for this inhibitory effect on hyaluronan–CD44 binding.

ANGIOGENESIS

The Myc-dependent angiogenic switch in tumours is mediated by interleukin 1β

Shchors, K. *et al. Genes Dev.* 14 September 2006 (doi: 10.1101/gad.1455706)

The MYC oncoprotein is known to induce angiogenesis. To further explore the events that lead to this, Evan and colleagues used their mouse model in which MYC can be switched on in pancreatic β cells. They found that MYC-expressing β cells release a diffusible angiogenic signal, the cytokine interleukin 1β, which leads to the release of sequestered vascular endothelial growth factor A from the islet extracellular matrix. This results in endothelial cell proliferation and blood vessel leakiness.