IN BRIEF

INNOVATION

In vivo quantitation of rare circulating tumour cells by multiphoton intravital flow cytometry

He, W. *et al. Proc. Natl Acad. Sci. USA* **104**, 11760–11765 (2007)

Philip Low and colleagues describe a new method, intravital flow cytometry, which allows the detection of rare circulating tumour cells down to a concentration of ~2 per ml of whole blood. The method was validated in mice with metastatic tumours and whole blood samples from cancer patients. This method could provide important information about the development of metastatic disease, the primary cause of cancer death.

TUMORIGENESIS

Autophagy mitigates metabolic stress and genome damage in mammary tumorigenesis

Karantza-Wadsworth, V. *et al. Genes Dev.* **21**, 1621–1635 (2007)

Autophagy, a cell survival mechanism that is activated in response to starvation and can lead to cell death, is also a mammary tumour-suppressor mechanism. Eileen White and colleagues showed that beclin1-deficient mammary epithelial cells (beclin1 regulates autophagy and is commonly haploinsufficient in breast carcinomas) are sensitive to matabolic stress, and show gene amplifications and activation of the DNA damage response. Therefore, they propose that autophagy protects the genome, and defects in this pathway lead to genomic instability and mammary tumorigenesis.

RESISTANCE

The Myc-evoked DNA damage response accounts for treatment resistance in primary lymphomas *in vivo*

Reimann, M. *et al. Blood.* 11 June 2007 (doi: 10.1182/blood-2007-02-075614)

Clemens Schmitt and colleagues have investigated the role of a functional DNA damage response, a barrier to tumorigenesis, in response to anticancer therapeutics. Using the *Eμ-Myc* mouse model of B-cell lymphoma, they showed that the lymphomas that developed had inactivated the pro-apoptotic ataxia-telangiectasia (ATM)–p53 pathway. Their evidence indicates that ATM activates apoptosis in response to oncogenic (MYC) signalling in pre-neoplastic lesions, and the loss of this pathway could predetermine resistance to therapy.

GENETICS

Two variants on chromosome 17 confer prostate cancer risk, and the one in TCF2 protects against type 2 diabetes.

Gudmundsson, J. *et al. Nature Genet.* 1 July 2007 (doi; 10.1038/ng2062)

The recent identification by several groups of sequence variants at chromosome 8q24 that confer an increased risk for prostate cancer has now been followed up by the identification of another two variants on chromosome 17. Although these variants have only a modest affect on disease risk per individual, they are common within the populations in which these studies were undertaken, so the population-attributable risk is substantial. Moreover, the authors found that one of the variants occurs in *TCF2*, a gene associated with type 2 diabetes; however, the variant associated with prostate cancer seems to protect against diabetes.