IN BRIEF

EPIGENETICS

SATB1 defines the developmental context for gene silencing by *Xist* in lymphoma and embryonic cells

Agrelo, R. *et al. Dev. Cell* **16**, 507–516 (2009)

Inactivation of the X chromosome is mediated through the binding of the non-coding RNA *Xist*. Using lymphoma cells and xenografts in nude mice, Anton Wutz and colleagues showed that *Xist* expression prevented tumour growth and induced cell death. By analysing genome-wide expression profiles they found that special AT-rich sequence-binding protein 1 (SATB1) — a chromatin modifier and regulator of gene expression — was downregulated in *Xist*-resistant cells. Moreover, SATB1 probably recruited *Xist* indirectly through the recruitment of polycomb group proteins.

METABOLISM

Glioma-derived mutations in *IDH1* dominantly inhibit IDH1 catalytic activity and induce HIF-1α

Zhao, S. *et al. Science* **324**, 261–265 (2009)

Monoallelic mutations in isocitrate dehydrogenase 1 (*IDH1*) are associated with various types of brain tumour, and Kun-Liang Guan, Yue Xiong and colleagues show that these mutations act in a dominant-negative manner to prevent IDH1 activity. Enforced expression of IDH1 mutants showed that α-ketoglutarate — the product of IDH1 activity — was reduced. This correlated with increased expression of hypoxia-inducible factor 1α (HIF1α) — a subunit of the HIF transcription factor that mediates cell survival. Therefore, the authors propose that IDH1 is a tumour suppressor.

BIOMARKERS

A compendium of potential biomarkers of pancreatic cancer

Harsha, H. C. *et al. PLoS Med.* **6**, e1000046 (2009)

Akhilesh Pandey and colleagues have analysed the published literature to produce a database of potential biomarkers for pancreatic cancer. Of 2,516 genes reportedly overexpressed in pancreatic cancer they sorted the list according to different criteria. For example, they examined whether a gene product was secreted or membrane-bound and would therefore make a useful biomarker. This approach has highlighted genes worthy of further investigation, and the methodology could be applied to any type of cancer to produce databases of potential biomarkers.

RESISTANCE

Targeting autophagy potentiates tyrosine kinase inhibitor-induced cell death in Philadelphia chromosome-positive cells, including primary CML stem cells

Bellodi, C. *et al. J. Clin. Invest.* **119**, 1109–1123 (2009)

Using chronic myeloid leukaemia (CML) cell lines and primary cells, Paolo Salomoni, Bruno Calabretta and colleagues found that treatment with the tyrosine kinase inhibitor (TKI) imatinib induced autophagy. Inhibition or knockdown of key regulators of autophagy increased cell death and almost entirely eradicated CML stem cells isolated from peripheral blood samples of patients with CML. This suggests that targeting autophagy might increase the potency of TKIs and reduce resistance.