IN BRIEF

METASTASIS

The clot thickens

Metastases that form in the bone constitute an essentially untreatable disease, so understanding the mechanisms that enable this is crucial. This paper indicates that platelets might function as messengers that exchange information between the primary tumour and the bone microenvironment. Specifically, primary tumours in mice can induce bone formation. The depletion of platelets in three mouse cancer models prevented bone formation, and the authors found that platelets can sequester proteins such as transforming growth factor-β that result in changes to the bone microenvironment.

ORIGINAL RESEARCH PAPER Kerr, B. A. *et al.* Platelets govern pre-metastatic tumour communication to bone. *Oncogene* 15 Oct 2012 (doi:10.1038/onc.2012.447)

THERAPEUTICS

BET spreading

Bromodomain and extraterminal domain (BET) proteins are transcriptional co-activators through their effects on histone modification. Inhibitors that target BET proteins have therapeutic efficacy in mouse models of leukaemia and lymphoma through the suppression of *MYC* expression. William Lockwood and colleagues show that the BET inhibitor JQ1 also has antiproliferative effects in a subset of lung adenocarcinoma cell lines. Gene expression profiling indicates that this occurs through reduced expression of the transcription factor FOSL1. Thus, depending on the genes expressed, BET inhibitors might prove useful for the treatment of certain solid tumours.

ORIGINAL RESEARCH PAPER Lockwood, W. W. *et al.* Sensitivity of human lung adenocarcinoma cell lines to targeted inhibition of BET epigenetic signaling proteins. *Proc. Natl Acad. Sci. USA* 5 Nov 2012 (doi:10.1073/pnas.1216363109)

LEUKAEMIA

Supporting role

Work by Nina Reinart and colleagues has indicated a role for macrophage migration inhibitory factor (MIF) in the survival of chronic lymphocytic leukaemia (CLL) cells. Crossing a transgenic mouse model of CLL with *Mif*-knockout mice delayed leukaemogenesis, and this correlated with reduced numbers of macrophages in lymphoid organs. *In vitro* studies showed that the absence of MIF increases CLL cell sensitivity to apoptosis, and a MIF-specific antibody reduced the interaction between CLL cells and macrophages. Thus, MIF seems to promote the interaction between CLL cells and macrophages, increasing CLL cell survival.

ORIGINAL RESEARCH PAPER Reinart, N. et al. Delayed development of chronic lymphocytic leukemia in the absence of macrophage migration inhibitory factor. Blood 1 Nov 2012 (doi:10.1182/blood-2012-05-431452)

SIGNALLING

All together now

CD133 is a membrane glycoprotein that is expressed by progenitor and stem cell-like cells, but it has unclear biological functions. This paper indicates that CD133 interacts with histone deacetylase 6 (HDAC6), preventing its degradation, and that these proteins also form a complex with β-catenin. This interaction stabilizes β-catenin through its deacetylation and enables its translocation to the nucleus where it promotes gene expression and proliferation. Downregulation of either CD133 or HDAC6 prevents this and delays the growth of tumour xenografts.

ORIGINAL RESEARCH PAPER Mak, A. B. *et al.* Regulation of CD133 by HDAC6 promotes β-catenin signaling to suppress cancer cell differentiation. *Cell Rep.* **2**, 951–963 (2012)