IN BRIEF

METABOLISM

Repressing glycolysis

Liu *et al.* have identified zinc finger and BTB domain-containing protein 7A (ZBTB7A; also known as POKEMON) as a novel tumour suppressor that acts by binding to the promoter and suppressing the transcription of several crucial glycolytic genes including *GLUT3*, *PFKP* and *PKM*. Furthermore, analysis of The Cancer Genome Atlas data showed that *ZBTB7A* is frequently deleted in several tumour types, including colon cancer, in which *ZBTB7A* loss correlated with poor survival. Xenograft tumours in mice derived from ZBTB7A-deficient cells decreased in size when treated with glycolytic inhibitors, highlighting both a functional role for ZBTB7A in glycolysis and a potential use of glycolytic inhibitors to treat ZBTB7A-deficient tumours.

ORIGINAL RESEARCH PAPER Liu, X.-S. *et al.* ZBTB7A acts as a tumor suppressor through the transcriptional repression of glycolysis. *Genes Dev.* **28**, 1917–1928 (2014)

COMPUTATIONAL MODELLING

A computational crystal ball

A new study outlines a computational method, termed DAISY, that statistically infers synthetic lethal interactions from cancer genomic data derived from cell lines and clinical samples. Jerby-Arnon *et al.* then used this method to construct genome-wide synthetic lethal interaction networks and exploited this network to predict the response of various cancer cell lines to anticancer drugs. The authors suggest that their approach can be used to triage current approaches based on the genetic makeup of a patient's tumour, to repurpose currently available drugs, to identify novel drug targets, and to predict prognosis.

ORIGINAL RESEARCH PAPER Jerby-Arnon, L. *et al.* Predicting cancer-specific vulnerability via data-driven detection of synthetic lethality. *Cell* **158**, 1199–1209 (2014)

BREAST CANCER

Driving postpartum metastasis

Lyons *et al.* have shown that human postpartum breast cancers, defined as cancers that arise up to five years after childbirth, have increased peritumour lymphatic vessel density. This characteristic may underlie increased rates of metastasis in women with postpartum breast cancers compared with breast cancers in other cohorts. The authors showed that in rodent models of this cancer, increased peritumour vessel density is due to overexpression of cyclooxygenase 2 (COX2) by tumour cells and treatment of these tumours with COX2 inhibitors caused a significant reduction in tumour cell invasion and metastasis. These results suggest that COX2 inhibitors are promising therapeutics for postpartum breast cancers.

ORIGINAL RESEARCH PAPER Lyons, T. R. *et al.* Cyclooxygenase-2–dependent lymphangiogenesis promotes nodal metastasis of postpartum breast cancer. *J. Clin. Invest.* **124**, 3901–3912 (2014)

THERAPEUTICS

Disrupting an oncogenic relationship

It is widely appreciated that MYCN is a potent oncogene but efforts to inhibit it have been hampered by the lack of surfaces to which small molecules can bind. Now, Gustafson *et al.* have shown that targeting and destabilizing Aurora kinase A (AURKA), a binding partner of MYCN, is sufficient to inhibit MYCN activity in a variety of tumour types. This result highlights AURKA inhibitors as potential anticancer therapeutics in a variety of MYCN- and, possibly, MYC-driven tumours.

ORIGINAL RESEARCH PAPER Gustafson, W. C. *et al.* Drugging MYCN through an allosteric transition in aurora kinase A. *Cancer Cell* **26**, 414–427 (2014)