IN BRIEF

TUMOUR HETEROGENEITY

Growth rates and tumour evolution

Waclaw *et al.* sought to understand how tumours expand and become heterogeneous. First, they modelled expansion and tumour cell growth in a metastatic lesion and showed that stochastic growth most closely followed histopathological findings of metastatic lesions. Furthermore, they found that cell motility, and thus dispersal, within the tumour was important for determining cell division rates. Surprisingly, their data predict that without dispersal it could take 8 years for one cell to grow into one billion cells, whereas this would be 2 years for tumour growth involving dispersal. Next, they simulated tumour growth and mutation rates in response to therapy, and they showed that tumour cells that are able to migrate are likely to regrow more quickly. Finally, their model was also able to predict features of primary tumour growth that are observed histologically. Therefore, tumour cell migration within a tumour may expose cells to new environments that affect their fitness and thus their ability to divide.

ORIGINAL RESEARCH PAPER Waclaw, B. *et al.* A spatial model predicts that dispersal and cell turnover limit intratumour heterogeneity. *Nature* **525**, 261–264 (2015)

OBESITY

The supersized tumour microenvironment

The risk of certain types of breast cancer increases with obesity, and the density of the extracellular matrix (ECM) is also known to be a risk factor for breast cancer. Seo *et al.* found that obesity and ECM density are connected: they showed that the mammary fat pads of obese mice are enriched with myofibroblasts and ECM components that are associated with increased stiffness. In particular, adipose stromal cell characteristics were altered in obese mice such that they produced more myofibroblasts and generated dense and stiff ECMs. Caloric restriction reduced myofibroblast content in mice, indicating that obesity-associated fibrosis and the associated changes in tissue mechanics can be reversed.

ORIGINAL RESEARCH PAPER Seo, B. R. *et al.* Obesity-dependent changes in interstitial ECM mechanics promote breast tumorigenesis. *Sci. Transl Med.* **7**, 301ra130 (2015)

PANCREATIC CANCER

PDAC subtypes

By separating genomics data from the predicted cellular source (such as stromal cell versus pancreatic tumour cell), Moffitt *et al.* have identified subtypes of pancreatic adenocarcinoma (PDAC). They characterized a basal-like subtype that has a worse prognosis than the subtype with activated stroma.

ORIGINAL RESEARCH PAPER Moffitt, R. A. *et al.* Virtual microdissection identifies distinct tumor- and stroma-specific subtypes of pancreatic ductal adenocarcinoma. *Nat. Genet.* http://dx.doi.org/10.1038/ng.3398 (2015)

TUMOUR SUPPRESSION

PTEN gains new powers

Caserta *et al.* investigated the effects of a cancer-associated mutation in *PTEN* (*PTEN*^{FV}). They generated *Pten*^{FV} knock-in mice: the homozygous mice had intact AKT signalling and developed normally; the heterozyogus mice developed several types of carcinoma, none of which was usually associated with *Pten* deficiency. This mutation therefore exposes a new role for PTEN in tumour suppression that requires further investigation.

ORIGINAL RESEARCH PAPER Caserta, E. *et al.* Noncatalytic *PTEN* missense mutation predisposes to organ-selective cancer development *in vivo*. *Genes Dev.* **29**, 1707–1720 (2015)