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IN BRIEF

MICROENVIRONMENT

Stromal metabolism has paracrine effects

Valencia *et al.* investigated the role of p62 in tumour-associated stromal cells, in which its expression is frequently reduced in tumour samples. The growth of prostate cancer cells in syngeneic p62-knockout mice was significantly increased compared to growth in control mice. p62 loss in cancer-associated fibroblasts led to increased expression of interleukin-6 (IL-6), the loss of which reversed the effects of p62 ablation, indicating that p62 suppresses the expression of IL-6, which, when expressed by fibroblasts, has paracrine effects on tumour cells. Notably, p62 repressed mTOR complex 1, MYC and the production of reactive oxygen species through metabolic reprogramming, which in turn repressed the expression of IL-6.

ORIGINAL RESEARCH PAPER Valencia, T. *et al.* Metabolic reprogramming of stromal fibroblasts through p62-mTORC1 signaling promotes inflammation and tumorigenesis. *Cancer Cell* http://dx.doi.org/10.1016/j.ccr.2014.05.004 (2014)

GENOMIC INSTABILITY

Suppressing CIN promotes growth

Ertych, Stolz *et al.* found an increase in microtubule assembly rates in colorectal cancer cells, and reducing microtubule assembly to the rate of normal cells suppressed chromosomal instability (CIN). The increased microtubule assembly rates were associated with transient spindle geometry defects and promoted lagging chromosomes. Additionally, CHK2–BRCA1 negatively regulated aurora kinase A (AURKA) to modulate microtubule assembly rates, and *CHK2* loss or *AURKA* overexpression frequently occurred in colorectal cancer samples. Unexpectedly, they found that restoring microtubule assembly rates to normal in colorectal cancer cells increased their growth in soft agar and as xenografts *in vivo*, even though the suppression of CIN was maintained.

ORIGINAL RESEARCH PAPER Ertych, N. *et al.* Increased microtubule assembly rates influence chromosomal instability in colorectal cancer cells. *Nature Cell Biol.* http://dx.doi.org/10.1038/ncb2994 (2014)

TUMOUR DETECTION

Molecular detection to improve surgery

Using desorption electrospray ionization (DESI) mass spectrometry to detect the oncometabolite 2-hydroxyglutarate (2-HG) in surgically resected gliomas, Santagata, Eberlin and colleagues showed that those gliomas with mutant isocitrate dehydrogenase (IDH) could be rapidly identified during surgery. Detection of 2-HG revealed the tumour margins and could thus be used to improve surgery and patient care.

ORIGINAL RESEARCH PAPER Santagata, S. *et al.* Intraoperative mass spectrometry mapping of an onco-metabolite to guide brain tumor surgery. *Proc. Natl Acad. Sci. USA* http://dx.doi.org/10.1073/pnas.1404724111 (2014)

THERAPEUTIC RESISTANCE

Inducing differentiation

Wang, Liu and colleagues examined the pathways associated with tyrosine kinase inhibitor (TKI) resistance of BCR–ABL T315I-mutant chronic myeloid leukaemia (CML) cells and found that the expression of genes associated with myeloid differentiation was reduced. Treating BCR–ABL-expressing CML cells with all-*trans* retinoic acid induced differentiation and prevented acquisition of the T315I mutation and TKI resistance.

ORIGINAL RESEARCH PAPER Wang, Z. *et al.* ATRA-induced cellular differentiation and CD38 expression inhibits acquisition of BCR-ABL mutations for CML acquired resistance. *PLoS Genet.* http://dx.doi.org/10.1371/journal.pgen.1004414 (2014)