IN BRIEF

MICROENVIRONMENT

p53 takes on a new role in macrophages

Lowe *et al.* found that in primary human monocytes and macrophages, the transcription factors p53 and nuclear factor-κB (NF-κB) co-regulate genes that are associated with inflammation, such as interleukin-6 (*IL6*). Furthermore, tumour cell-derived factors activated p53 in tumour-associated macrophages and induced the expression of IL-6. This may be a tumour-promoting role of p53, in cooperation with NF-κB, but further work is required to understand precisely what effect this has on tumours.

ORIGINAL RESEARCH PAPER Lowe, J. M. *et al.* p53 and NF-κB coregulate proinflammatory gene responses in human macrophages. *Cancer Res.* **74**, 2182–2192 (2014)

TRANSCRIPTION

Mediating tumorigenesis

Somatic mutations in mediator complex subunit 12 (*MED12*) occur in approximately 70% of uterine leiomyomas. Turunen, Spaeth *et al.* showed that the *MED12* mutations disrupted the binding of MED12 with cyclin C, which is required for the stimulation of cyclin C–cyclin-dependent kinase 8 (CDK8) activity. This resulted in a loss of CDK activity that is associated with the mediator complex.

ORIGINAL RESEARCH PAPER Turunen, M. *et al.* Uterine leiomyoma-linked MED12 mutations disrupt mediator-associated CDK activity. *Cell Rep.* **7**, 654–660 (2014)

IMMUNOLOGY

Predicting immunogenicity

Using RNA-sequencing data from The Cancer Genome Atlas, Brown *et al.* used tools to predict whether missense mutations found in tumours from 515 patients were likely to result in neoantigens that could be presented by major histocompatibility complex (MHC) class I. Tumours producing neoantigens that were predicted to be bound by MHC class I correlated with *CD8A* expression (a surrogate for the presence of $CD8^+$ tumour-infiltrating lymphocytes) and patient survival. The potentially immunogenic tumours had increased expression of RNAs encoding the immune checkpoint proteins PDCD1 and CTLA4, indicating that these tumours might benefit from immunotherapy that suppresses immune checkpoint signalling.

ORIGINAL RESEARCH PAPER Brown, S. D. *et al.* Neo-antigens predicted by tumor genome meta-analysis correlate with increased patient survival. *Genome Res.* **24**, 743–750 (2014)

TUMORIGENESIS

Allergic dermatitis resists skin cancer

Some allergic responses, such as atopic dermatitis, are inversely correlated with the occurrence of some types of cancer. To more clearly understand this correlation, Cipolat *et al.* induced tumours using DMBA–TPA in mice that lack three barrier proteins (EPI mice) and thereby model what occurs in some cases of atopic dermatitis. They found that these mice were resistant to developing benign tumours and this was associated with an exaggerated immune response on TPA administration. The authors suggest that this protective affect might arise from keratinocytes signalling to the immune system, which induces a protective immune response.

ORIGINAL RESEARCH PAPER Cipolat, S. *et al.* Epidermal barrier defects link atopic dermatitis with altered skin cancer susceptibility. *eLife* **3**, e01888 (2014)