# IN BRIEF

## GENETICS

### p53 variant increases cancer risk

The single nucleotide polymorphism P47S (rs1800371) in *TP53* is specific to populations of African descent. Jennis *et al.* reasoned that P47S might contribute to the increased risk of cancer in African American populations. P47S did not affect most p53 functions, but did impair transactivation of a subset of p53 target genes and inhibited cell death in response to cisplatin or agents that induce ferroptosis. Mice heterozygous or homozygous for P47S were predisposed to spontaneous cancers of various types, suggesting that this variant of p53 increases cancer risk.

**ORIGINAL ARTICLE** Jennis, M. *et al.* An African-specific polymorphism in the *TP53* gene impairs p53 tumor suppressor function in a mouse model. *Genes Dev.* http://dx.doi.org/10.1101/gad.275891.115 (2016)

## PROSTATE CANCER

### Connecting androgen receptor and immunity

Surgical androgen deprivation therapy (ADT) has activating effects on the immune system. However, whether medical ADT (with androgen receptor (AR) antagonists) has similar effects is not clear. Pu *et al.* report the unexpected finding that AR antagonists suppress immune responses by inhibiting T cell activation. Furthermore, surgical ADT synergized with immunotherapy in mouse models of prostate cancer, whereas medical ADT suppressed immunotherapy effects. These findings might improve prostate cancer immunotherapy.

**ORIGINAL ARTICLE** Pu, Y. *et al.* Androgen receptor antagonists compromise T cell response against prostate cancer leading to early tumor relapse. *Sci. Transl Med.* **8**, 333ra47 (2016)

## TUMOUR IMMUNOLOGY

### Another string to the MYC bow

Casey *et al.* show that MYC regulates the transcription of the immunoregulatory molecules CD47 and programmed cell death ligand 1 (PDL1). MYC bound to the promoters of *CD47* and *PDL1*, and suppression of MYC resulted in reduced expression of CD47 and PDL1 mRNA and protein in human and mouse tumour cells. MYC inactivation in mouse tumours promoted antitumour immune responses via the reduction in CD47 and PDL1 expression, adding regulation of immune evasion as another oncogenic activity that can be attributed to MYC.

**ORIGINAL ARTICLE** Casey, S. C. *et al.* MYC regulates the antitumor immune response through CD47 and PD-L1. *Science* **352**, 227–231 (2016)

## TRANSLOCATIONS

### Circular RNAs from translocations

Guarnerio *et al.* show that oncogenic translocations also give rise to fusion-circular RNAs (f-circRNAs). The authors went on to demonstrate that f-circRNAs derived from the mixed lineage leukaemia (*MLL*)–*AF9* (also known as *MLLT3*) fusion gene were able to induce transformation of immortalized cells *in vitro* and of haematopoietic stem cells expressing the MLL–AF9 fusion protein *in vivo*. Furthermore, they found that these f-circRNAs conferred resistance of *MLL–AF9*-expressing leukaemia cells to therapy, and knockdown of *MLL–AF9*-derived f-circRNAs increased apoptosis and the expression of p27 and p21 in THP1 cells (which have the *MLL–AF9* translocation), indicating that f-circRNAs are also important for cell viability.

**ORIGINAL ARTICLE** Guarnerio, J. *et al.* Oncogenic role of fusion-circRNAs derived from cancer-associated chromosomal translocations. *Cell* **165**, 289–302 (2016)