IN BRIEF

IMAGING

Polyps in technicolour

Colorectal cancer (CRC) can be prevented by colonoscopy using white light to detect polyps that are then removed. However, small and flat polyps are difficult to detect using this technique. Burggraaf *et al.* have tested fluorescence-guided colonoscopy using the agent GE-137, a cyclic peptide that binds the extracellular receptor MET, which is overexpressed in early stages of CRC. Intravenous administration of GE-137 conjugated to a fluorescent dye enabled visualization of all neoplastic polyps that were visible, as well as additional polyps that were not visible, with white light in patients with CRC.

ORIGINAL RESEARCH PAPER Burggraaf, J. *et al.* Detection of colorectal polyps in humans using an intravenously administered fluorescent peptide targeted against c-Met. *Nat. Med.* **21**, 955–961 (2015)

TUMOUR IMMUNOLOGY

T_{Reg} cells, more than a suppression problem

Chronic intestinal inflammation is associated with the development of colorectal cancer. Regulatory T (T_{Reg}) cells suppress inflammatory responses and maintain intestinal homeostasis, and therefore they might mitigate the formation of inflammation-associated tumours. Surprisingly, Geis *et al.* have found that in mice colonized with enterotoxigenic *Bacteroides fragilis* (ETBF) — a human colonic bacterium associated with inflammatory intestinal diseases — T_{Reg} cells produce interleukin-17, which promotes the earliest stages of colon carcinogenesis. Depletion of T_{Reg} cells in ETBF-colonized mice enhanced colitis but diminished tumorigenesis.

ORIGINAL RESEARCH PAPER Geis, A. L. *et al.* Regulatory T cell response to enterotoxigenic *Bacteroides fragilis* colonization triggers IL-17-dependent colon carcinogenesis. *Cancer Discov.* http:/dx.doi.org/10.1158/2159-8290.CD-15-0447 (2015)

GENETICS

The evil twin

Some cancers have an abundance of mutations at cytosines on one DNA strand, which is consistent with the mutagenic properties of APOBEC cytidine deaminases. *APOBEC3B* mRNA expression has been found in various cancers, whereas the role of APOBEC3A as a mutator is less clear. By analysing 15 cohorts of recently published whole-genome sequencing samples that included 5 different types of cancer with mutation signatures resembling those of either APOBEC, Chan *et al.* have found that APOBEC3A is the predominant mutagenic deaminase. Although *APOBEC3B* mRNA tends to be more abundant than that of *APOBEC3A*, APOBEC3A is a much more potent inducer of mutations than APOBEC3B.

ORIGINAL RESEARCH PAPER Chan, K. *et al.* An APOBEC3A hypermutation signature is distinguishable from the signature of background mutagenesis by APOBEC3B in human cancers. *Nat. Genet.* http:/dx.doi.org/10.1038/ng.3378 (2015)

THERAPY

Reinventing the wheel

Huang *et al.* have identified that the potassium channel ether-a-go-go 2 (EAG2) promotes tumour growth and metastasis in multiple fly and mouse brain tumour models. In a xenograft model of medulloblastoma, EAG2 knockdown impaired metastasis, and pharmacological inhibition of EAG2 with an FDA-approved antipsychotic drug, thioridazine, showed potent efficacy in reducing intracranial tumour growth and metastasis.

ORIGINAL RESEARCH PAPER Huang, X. *et al.* EAG2 potassium channel with evolutionarily conserved function as a brain tumor target. *Nat. Neurosci.* http:/dx.doi.org/10.1038/nn.4088 (2015)