# **IN BRIEF**

## NEUROIMMUNOLOGY

#### Interferon signalling in neuronal subtypes

Cho et al. characterized the distinct susceptibility of neuronal subtypes to infection with neurotropic viruses and associated this susceptibility with the differential expression of interferon (IFN)-stimulated genes (ISGs) and other IFN signalling components. Granule cells (which are neurons of the cerebellum) were more responsive to stimulation with IFN $\beta$  and less susceptible to infection with West Nile virus (WNV) than neurons of the cerebral cortex. Moreover, both basal expression and IFN $\beta$ -induced expression of IFN-related genes were higher in granule cells than in cortical neurons. Transfection of cortical neurons with granule cell-expressed ISGs identified a role for *Ifi27, Irg1* and *Rsad2* in protection against WNV infection. Interestingly, the differential expression of ISGs and other IFN signalling components in neuronal subtypes seem to involve the miRNA-132-dependent regulation of histone acetylation.

**ORIGINAL RESEARCH PAPER** Cho, H. *et al.* Differential innate immune response programs in neuronal subtypes determine susceptibility to infection in the brain by positive-stranded RNA viruses. *Nature Med.* 3 Mar 2013 (doi:10.1038/nm.3108)

### TUMOUR IMMUNOLOGY

# AIRE-dependent $T_{Reg}$ cells in tumours

Malchow et al. report a central role for the autoimmune regulator (AIRE)-dependent thymic expression of self antigens in the development of natural regulatory T ( $T_{Red}$ ) cells, and they indicate that natural  $T_{Req}$  cells are the most prevalent type of T<sub>Reg</sub> cells in at least some tumours. T cell receptor (TCR) repertoire analysis of  $T_{Reg}$  cells from autochthonous prostate tumours of transgenic mice identified selective enrichment of a  $T_{Reg}$  cell clone (referred to as an MJ23  $T_{Reg}$  cell clone), which expressed a characteristic V $\alpha$ 2 TCR $\alpha$  chain that was absent in non-T<sub>Ren</sub> cell TCRs. Next, studies in male and female transgenic mice, in which all T cells expressed the MJ23 TCR, showed that this TCR was specific for a prostate self antigen. MJ23 T<sub>Rep</sub> cells did not differentiate from naive T cells intratumourally but developed in the thymus of both male and female mice in an AIRE-dependent manner. So, besides its central role in negative thymic selection, AIRE is involved in natural  $\rm T_{\rm Reg}$  cell development. The authors suggest that it is such self antigen-specific natural  $T_{Reg}$  cells (rather than tumour antigen-specific induced  $T_{Reg}$  cells) that accumulate in tumours.

ORIGINAL RESEARCH PAPER Malchow, S. et al. Aire-dependent thymic development of tumor-associated regulatory T cells. Science **339**, 1219–1224 (2013)

#### **IMMUNOMETABOLISM**

#### CD4<sup>+</sup> T cell activation by adipocytes in obesity

A recent report suggests that adipocytes activate CD4<sup>+</sup> T cells to initiate inflammation in obese tissue. Deng *et al.* observed that obesity promotes MHC class II expression on adipocytes in both humans and mice. MHC class II expression seemed to be triggered by the leptin-induced secretion of interferon- $\gamma$ (IFN $\gamma$ ) by adipose tissue-resident CD4<sup>+</sup> T cells. Adipocytes that had been isolated from obese mice or stimulated with IFN $\gamma$  activated CD4<sup>+</sup> T cells in an antigen- and cell contact-dependent manner. Strikingly, CD4<sup>+</sup> T cell activation by adipocytes preceded macrophage accumulation in the adipose tissue of mice fed a high-fat diet. As obese MHC class II-deficient mice showed lower levels of adipose tissue inflammation and insulin resistance than obese wild-type mice, CD4<sup>+</sup> T cell activation by adipocytes might be one of the early events that initiate adipose tissue inflammation in obesity.

ORIGINAL RESEARCH PAPER Deng, T. et al. Class II major histocompatibility complex plays an essential role in obesity-induced adipose inflammation. Cell Metab. 17, 411–422 (2013)