IN BRIEF

MHC MOLECULES

Tweaking developing synapses

This study by Carla Shatz and colleagues offers a fascinating insight into the non-immune functions of MHC class I molecules. MHC class I molecules (and other immunological mediators) have been implicated in synapse elimination in the central nervous system (CNS). However, it has been unclear if these molecules regulate developing synapses, and whether a neuronal or immune function is involved. The authors found that the mouse MHC class I molecule H2-D^b is essential for synapse elimination during the development of the visual system. Mice that lacked both H2-D^b and H2-K^b showed defects in synapse elimination and in the formation of eye-specific layers in visual processing areas of the brain, but this could be rescued by restoring H2-D^b expression exclusively in CNS neurons. Polymorphisms in the MHC locus have been linked to schizophrenia, and the authors propose that differential MHC class I expression during brain development could affect synaptic pruning and lead to long-lasting changes in human brain circuits and behaviour.

ORIGINAL RESEARCH PAPER Lee, H. *et al.* Synapse elimination and learning rules co-regulated by MHC class I H2-D^b. *Nature* http://dx.doi.org/10.1038/nature13154 (2014)

ASTHMA AND ALLERGY

Diagnostic methods are moving on…

Here, Sackmann *et al.* describe a new technique that can be used to diagnose asthma from only a single drop of whole blood. The authors have developed a handheld microfluidic device that sorts neutrophils by pumping the blood cells through P-selectin-coated microchannels. The selected neutrophils are then exposed to a hydrogel that contains a neutrophil chemoattractant, and their movement is measured using time-lapse microscopy and automated tracking software. Using this method, the authors found that neutrophils from patients with asthma showed significantly reduced chemotaxis velocities compared with neutrophils from control patients. They suggest that the decreased chemotaxis velocity of neutrophils from patients with asthma may reflect the increased adhesion of these cells to the P-selectin molecules that coat the microchannels. Importantly, the technique is easy to perform and obtains a rapid diagnostic result.

ORIGINAL RESEARCH PAPER Sackmann, E. K.-H. *et al.* Characterizing asthma from a drop of blood using neutrophil chemotaxis. *Proc. Natl Acad. Sci. USA* http://dx.doi.org/10.1073/pnas.1324043111 (2014)

B CELLS

Signal strengths BOB up and down

Mice deficient in the transcription factor BOB1 (also known as POU2AF1) have defective B cell responses and lack germinal centres and marginal zone B cells. These findings have led to the suggestion that BOB1 potentiates B cell receptor (BCR) signalling. By identifying novel transcriptional targets of BOB1, Lindner *et al.* offer an alternative explanation for the immunodeficiency seen in BOB1-deficient mice. They show that BOB1 regulates the expression of the microRNAs miR-146a and miR-210, which are known to negatively regulate B cell signalling pathways. Thus, they propose that B cells that lack BOB1 may receive excessively strong BCR signals during their development, which leads to the induction of an anergic-like state. In support of this idea, they found that peripheral B cells in BOB1-deficient mice have an anergic phenotype.

ORIGINAL RESEARCH PAPER Lindner, J. M. *et al.* Cutting edge: The transcription factor Bob1 counteracts B cell activation and regulates miR-146a in B cells. *J. Immunol.* http://dx.doi.org/10.4049/jimmunol.1303022 (2014)