IN BRIEF

AUTOIMMUNITY

SCARF1 helps clean up the dead

A failure of the immune system to remove apoptotic cells can lead to autoimmune disease, such as systemic lupus erythematosus (SLE). This study identifies a key scavenger receptor that is used by phagocytes to recognize and to engulf apoptotic cells. Similar to the *Caenorhabditis elegans* homologue CED-1, SCARF1 (scavenger receptor class F member 1) was found to bind to apoptotic cells but not to live cells. This interaction did not involve direct binding to known 'eat-me' signals such as phosphatidylserine, but rather involved specific recognition of the complement component C1q, which bound phosphatidylserine on dying cells. Dendritic cells, and to a lesser extent macrophages and endothelial cells, from *Scarf1*$^{-/-}$ mice had impaired uptake of apoptotic cells. Consequently, dying cells accumulated in *Scarf1*$^{-/-}$ mice and the mice developed a lupus-like disease, which was associated with autoantibody production, nephritis and dermatitis.

ORIGINAL RESEARCH PAPER Ramirez-Ortiz, Z. G. *et al.* The scavenger receptor SCARF1 mediates the clearance of apoptotic cells and prevents autoimmunity. *Nature Immunol.* http://dx.doi.org/10.1038/ni.2670 (2013)

NEUROIMMUNOLOGY

Linking immune and emotional health

Indirect evidence suggesting a link between immune function and mood disorders is supported by the anxiety-like behaviour that is characteristic of recombination-activating gene 1 (*Rag1*)$^{-/-}$ mice. This study showed that the presence of CD4^{+} T cells but not of CD8^{+} T cells in *Rag1*$^{-/-}$ OT-II-transgenic mice but not in *Rag1*$^{-/-}$ OT-I-transgenic mice reverts the increased digging and marble-burying behaviours of *Rag1*$^{-/-}$ mice. Transient depletion or reconstitution of CD4^{+} or CD8^{+} T cells did not affect these activites, which indicates that life-long immunodeficient conditions are required to affect behaviour. There were no differences in systemic factors or in brain anatomy that could be an explanation for the altered emotional behaviour. Whole-brain microarray analysis showed that *Rag1*$^{-/-}$ OT-II mice have a genetic fingerprint more similar to wild-type mice than to *Rag1*$^{-/-}$ mice. Nine main signalling pathways (including genes involved in various neuropsychological conditions) were significantly altered in *Rag1*$^{-/-}$ mice compared with wild-type mice.

ORIGINAL RESEARCH PAPER Rattazzi, L. *et al.* CD4^{+} but not CD8^{+} T cells revert the impaired emotional behavior of immunocompromised *RAG-1*-deficient mice. *Trans. Psych.* **3**, e280 (2013)

IMMUNE REGULATION

Long non-coding RNAs in the immune system

Studies from the past few years have shown a role for long non-coding RNAs (lncRNAs) in regulating a range of physiological processes. Two studies now report a role for lncRNAs in the immune system. Rapicavoli *et al.* describe the induction of Lethe, a pseudogene lncRNA, by tumour necrosis factor and interleukin-1β. Lethe negatively regulates nuclear factor-κB signalling by binding directly to RELA. Lethe expression decreases with age, which might be associated with a decreased ability to control the inflammatory response. Carpenter *et al.* describe the induction of lincRNA-*Cox2* downstream of Toll-like receptor signalling, which mediates the activation and repression of distinct sets of immune target genes. Transcriptional repression involves the interaction of lincRNA-*Cox2* with heterogeneous nuclear ribonucleoproteins.

ORIGINAL RESEARCH PAPERS Rapicavoli, N. A. *et al.* A mammalian pseudogene lncRNA at the interface of inflammation and anti-inflammatory therapeutics. *eLIFE* **2**, e00762 (2013) | Carpenter, S. *et al.* A long noncoding RNA mediates both activation and repression of immune response genes. *Science* http://dx.doi.org/10.1126/science.1240925 (2013)