# IN BRIEF

## ALLERGY

### Allergic pulmonary inflammation in mice is dependent on eosinophil-induced recruitment of effector T cells.

Jacobsen, E. A. *et al. J. Exp. Med.* 3 March 2008 (doi:10.1084/jem.20071840)

The precise role of eosinophils during a pulmonary allergic immune response is controversial. To clarify this issue, Jacobsen *et al.* used transgenic mice that lack eosinophils, known as PHIL mice, and detected a reduction in T-cell accumulation and T helper 2 ($T_H2$)-type responses in the lungs of these mice following allergen challenge. The restoration of the $T_H2$-type inflammatory response in these mice required the transfer of both $T_H2$-polarized allergen-specific T cells and eosinophils; T cells alone were not sufficient. The production of the $T_H2$-type chemokines CC-chemokine ligand 17 (CCL17) and CCL22 was required for this recruitment by eosinophils. So, this study indicates that the primary role of eosinophils in allergic pulmonary responses is the recruitment of effector T cells to the lungs.

## REGULATORY T CELLS

### IRF9 and STAT1 are required for IgG autoantibody production and B cell expression of TLR7 in mice.

Thibault, D. L. *et al. J. Clin. Invest.* 14 March 2008 (doi:10.1172/JCI30065)

The production of high-affinity IgG autoantibodies that recognize nucleic-acid-associated antigens is a hallmark of systemic lupus erythematosus (SLE). Type I interferon (IFN) and nucleic-acid-sensing Toll-like receptors (TLRs; TLR7 and TLR9) are also involved in the pathogenesis of SLE but the exact relationship between these mediators is not fully understood. Using a pristane-induced mouse model of SLE, Utz and colleagues now show that the type I IFN-induced signalling molecules, IFN-regulatory factor 9 (IRF9) and signal transducer and activator of transcription 1 (STAT1), are required for the production of IgG autoantibodies. In addition, both IRF9 and STAT1 are required for the expression of TLR7 and TLR9 by B cells in response to type I IFN signalling. So, this study reveals a new role for type I IFN-induced signalling molecules in the autoimmune response in SLE.

## T CELLS

### Dual functions for the endoplasmic reticulum calcium sensors STIM1 and STIM2 in T cell activation and tolerance.

Oh-hora, M. *et al. Nature Immunol.* 9 March 2008 (doi:10.1038/ni1574)

The tight regulation of signalling pathways involving calcium ions ($Ca^{2+}$) is essential for the normal function of many immune cell types. Rao and colleagues have further characterized STIM1 (stromal interaction molecule 1) and STIM2, two proteins previously identified as important for monitoring $Ca^{2+}$ flow between the endoplasmic reticulum and the cytoplasm, in T cells. Conditional ablation of *Stim1* in T cells showed that this protein is required for proper $Ca^{2+}$ influx, interleukin-2 production, and nuclear translocation of NFAT (nuclear factor of activated T cells) following stimulation of $CD4^+$ T cells, whereas ablation of *Stim2* had a milder effect on these functions. Double-deficient mice developed a severe lymphoproliferative disease after 8 weeks, largely due to impaired development of and intrinsic defects in $FOXP3^+$ regulatory T cells in the absence of intact $Ca^{2+}$ signalling pathways. Therefore, STIM1 and STIM2 are essential not only for regulating $Ca^{2+}$ flux, but also for normal regulatory T-cell development.