

## IN BRIEF

**DENDRITIC CELLS**

NOX2 controls phagosomal pH to regulate antigen processing during crosspresentation by dendritic cells.

Savina, A. et al. *Cell* **126**, 205–218 (2006)

A new study shows that the enzyme NADPH oxidase 2 (NOX2), which is an important component of the innate immune response mediated by neutrophils, also has a role in adaptive immunity, through the control of antigen presentation by dendritic cells (DCs). DCs can ‘cross-present’ exogenous antigens, in the form of proteolytically cleaved peptides, on MHC class I molecules to CD8<sup>+</sup> T cells. To ensure recognition by T cells, DCs have developed specialized ways to control the partial degradation of these exogenous antigens.

Savina et al. provide the first genetic evidence that a novel specific adaptation of the DC endocytic pathway is required for efficient cross-presentation. They show that NOX2-defective DCs have increased phagosomal acidification and antigen degradation, which causes defective antigen cross-presentation. Therefore, NOX2 is a crucial component in the phagocytic pathway of DCs, allowing DCs to process antigens rather than degrading them.

**ASTHMA AND ALLERGY**

Role of deficient type III interferon-λ production in asthma exacerbations.

Contoli, M. et al. *Nature Med.* 13 Aug 2006 (doi:10.1038/nm1462)

This study looks at the role of the recently discovered type III interferons IFNλ1 and IFNλ2/3 in the susceptibility of individuals with asthma to acute exacerbations associated with rhinovirus infections. The authors show that the production of IFNλ by primary bronchial epithelial cells and bronchoalveolar macrophages in response to rhinovirus replication is significantly lower for patients with asthma than for individuals without asthma. The decreased IFNλ production correlates with increased viral load and severity of symptoms *in vivo*. This indicates that therapies that aim to replace or augment IFNλ production might be a new approach to the treatment or prevention of asthma exacerbations. Further studies are required to determine the mechanism of deficient IFN production in patients with asthma.

**T-CELL RESPONSES**

Duration of the initial TCR stimulus controls the magnitude but not functionality of the CD8<sup>+</sup> T cell response.

Prlic, M. et al. *J. Exp. Med.* 14 Aug 2006 (doi:10.1084/jem.20060928)

The concept of T-cell programming describes how a brief encounter with antigen is sufficient to trigger a cell-autonomous programme that leads to proliferation and differentiation into memory T cells. Many previous studies of T-cell programming have been limited by the need to provide the timed antigen stimulus to T cells *in vitro* before *in vivo* transfer. This study used peptide-pulsed transgenic dendritic cells that were engineered to be susceptible to diphtheria toxin to allow timed antigen exposure entirely *in vivo*. The authors found that the duration of antigen exposure correlated with the magnitude of the primary response but that CD8<sup>+</sup> T-cell effector function and memory was independent of antigen timing above a certain minimum exposure time.