IN BRIEF

⟹ ASTHMA AND ALLERGY

Real-time assessment of inflammation and treatment response in a mouse model of allergic airway inflammation

Cortez-Retamozo, V. *et al. J. Clin. Invest.* 6 Nov 2008 (doi:10.1172/JCI36335)

Eosinophils promote airway inflammation in response to allergens by expressing various mediators and proteinases. Based on the finding that matrix metalloproteinases (MMPs) were mainly expressed by eosinophils in a mouse model of allergic airway inflammation, the authors of this study used a specific MMP-targeted optical sensor to track these cells *in vivo*. When non-invasive molecular imaging techniques were used to track eosinophils with the MMP-targeted sensor in live mice, it was found that the amount of MMP activity correlated with the severity of airway inflammation. In addition, this system was successfully used to measure the treatment efficacy of glucocorticoids, which are commonly used to treat patients with asthma, and to identify a new compound that reduced airway inflammation. These findings provide hope that such non-invasive molecular imaging techniques can be translated to the clinical setting to assess patients with asthma and other chronic inflammatory diseases of the airways.

⟹ B CELLS

Tonic B cell antigen receptor signals supply an NF-κB substrate for prosurvival BLyS signaling

Stadanlick, J. E. *et al. Nature Immunol.* 2 Nov 2008 (doi:10.1038/ni.1666)

This study explains why B cells need continuous signalling through both the B-cell receptor (BCR) and the B lymphocyte stimulator receptor 3 (BR3; also known as BAFFR and TNFRSF13C) for their survival. The authors show that BCR signalling activates the classical nuclear factor-κB (NF-κB) pathway to generate p100, which in turn serves as a substrate for BR3-mediated signalling that leads to the accumulation of nuclear p52 (a hallmark of the non-classical NF-κB pathway) and consequently B-cell survival. The capacity to produce p100 constitutively through tonic BCR signalling was acquired during the late transitional stage of B-cell differentiation, when BCR signals promote survival rather than negative selection. These findings support a model in which receptor signalling crosstalk ensures that B cells gain a gradual resistance to negative selection as they mature.

⟹ INFLAMMATION

Cutting Edge: IFN-γ enables APC to promote memory Th17 and abate Th1 cell development

Kryczek, I. *et al. J. Immunol.* **181**, 5842–5846 (2008)

In this study, the authors examined the effect of interferon-γ (IFNγ) on the development and maintenance of human T helper 1 (T_H1) and T_H17 cells. Contrary to what was previously thought, they found that IFNγ-conditioned antigen-presenting cells (APCs) suppressed IFNγ production by co-cultured T cells by upregulating their expression of the co-inhibitory ligand B7-H1. By contrast, IFNγ-conditioned APCs induced interleukin-17 (IL-17) production and proliferation of human memory T_H17 cells by upregulating IL-1 and IL-23 expression. So, based on these *in vitro* observations, the authors propose that IFNγ attenuates T_H1-cell-mediated inflammation and induces memory T_H17-cell expansion, suggesting a new regulatory role for IFNγ. However, the implications of these observations in controlling the progression of inflammation *in vivo* have yet to be analysed.