IN BRIEF

NATURAL KILLER CELLS

Development and function of murine B220$^+$CD11c$^+$NK1.1$^+$ cells identify them as a subset of NK cells.

Blasius, A. L. *et al. J. Exp. Med.* 8 October 2007 (doi:10.1084/jem.20070991)

CD11c^{lo}B220$^+$ interferon-producing killer dendritic cells are activated natural killer cells.

Vosshenrich, C. A. J. *et al. J. Exp. Med.* 8 October 2007 (doi:10.1084/jem.20071451)

Putative IKDCs are functionally and developmentally similar to natural killer cells, but not to dendritic cells.

Caminschi, I. *et al. J. Exp. Med.* 8 October 2007 (doi:10.1084/jem.20071351)

Interferon (IFN)-producing killer dendritic cells (IKDCs) are a recently described type of DC that have the functional features of both natural killer (NK) cells and DCs in that they can acquire the ability to present antigen and can lyse NK-cell targets. However, the ability of IKDCs to produce IFNα and IFNβ (a DC-like function) is controversial, and there have been conflicting reports on the specific cytokines required for IKDC development. Now, three papers published in *The Journal of Experimental Medicine* have reassessed IKDCs. Blasius *et al.* used specific markers to dissect the heterogeneous B220$^+$CD11c$^+$ population in mice and showed that Siglec-H$^-$NK1.1$^+$ cells correspond to IKDCs. Similar to NK cells, these cells do not produce IFNα and require the common cytokine receptor γ-chain (γ_c) and interleukin-15 (IL-15) for development. Vosshenrich *et al.* used a series of knockout mice that lack specific cytokines to show that IKDC development is dependent on IL-15 and that IKDCs express transcripts encoding the NK-cell-specific marker NK-cell protein 46 (NKp46). Caminschi *et al.* showed that mouse IKDCs can kill NK-cell targets but are unable to present antigen to T cells. IKDCs also lacked expression of the transcription factor PU.1 and were absent in mice that were deficient in the γ_c cytokines. These papers show that IKDCs, instead of being considered as a subset of DCs, should rather be considered as a subset of NK cells.

T-CELL RESPONSES

IL-13Rα2 and IL-10 coordinately suppress airway inflammation, airway-hyperreactivity, and fibrosis in mice.

Wilson, M. S. *et al. J. Clin. Invest.* **117**, 2941–2951 (2007)

Interleukin-10 (IL-10) has been shown to regulate immunity that is driven by T helper 2 (T_H2)-type cytokines, although its effects on T_H2-cell-mediated pathology can be paradoxical — IL-10 deficiency results in increased T_H2-cell-driven inflammation, but also in reduced airway hyperreactivity (AHR), mucous hypersecretion and fibrosis. IL-13 also has a crucial role in diseases such as asthma and in chronic helminth infections, which are characterized by persistent T_H2-type cytokine responses. Wilson *et al.* showed that increased expression of IL-13 receptor α-chain 2 (IL-13Rα2) is responsible for reduced AHR, mucous production and fibrosis in *Il10*$^{-/-}$ mice. Moreover, in models of allergic asthma and chronic helminth infection, IL-10 and IL-13Rα2 coordinately suppressed T_H2-cell-mediated inflammation and pathology, respectively. Therefore, both IL-10 and IL-13Rα2 are required to control chronic T_H2-cell-mediated pathological responses.