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IN BRIEF

NATURAL KILLER CELLS

Dendritic cells prime natural killer cells by *trans*-presenting interleukin 15.

Lucas, M. *et al. Immunity* **26**, 503–517 (2007)

Natural killer (NK) cells have an important role in the control of infections, but the signals that activate these cells *in vivo* are still not fully understood. Using a transgenic mouse model that allows for the inducible ablation of all conventional CD11c^{hi} dendritic cells (DCs), Lucas *et al.* showed that CD11c^{hi} DCs are essential for NK-cell priming *in vivo*. Further studies showed that naive NK cells are recruited to the draining lymph nodes following local DC activation where they interact with DCs and emerge as effector cells. Priming of NK cells depended on type I interferon production and signalling and the subsequent *trans*-presentation of interleukin-15 (IL-15) by DCs to NK cells. DC-derived IL-15 was necessary and sufficient for NK-cell priming *in vivo*. The data show that NK cells acquire their effector functions in an IL-15-dependent manner by interacting with DCs.

AUTOIMMUNITY

T_H17 cells contribute to uveitis and scleritis and are expanded by IL-2 and inhibited by IL-27/STAT1.

Amadi-Obi, A. *et al. Nature Med.* 13 May 2007 (doi:10.1038/nm1585)

This study identifies an important role for interleukin-17 (IL-17)-producing T helper cells (T_H17 cells) in human uveitis and scleritis. The authors found that the blood of patients with uveitis or scleritis contained more T_H17 cells than the blood of healthy individuals. This expansion was driven by IL-2 but inhibited by interferon-γ (IFNγ). In a mouse model, treatment with an IL-17-specific antibody reduced the severity of ocular inflammation. However, IFNγ or IL-27 may also be potential therapeutic targets because IFNγ was shown to inhibit T_H17-cell proliferation by upregulating IL-27 expression in mouse retinal cells. Finally, the authors suggest that T_H17 cells may mediate disease by inducing the production of tumour-necrosis factor, as large amounts of this cytokine were found in retinal cells of mice with ocular inflammation.

IMMUNE REGULATION

Type I interferons protect neonates from acute inflammation through interleukin 10-producing B cells.

Zhang, X. *et al. J. Exp. Med.* **204**, 1107–1118 (2007)

Newborns and infants are highly vulnerable to infection, even though their immune systems are fairly well developed. As shown here, this might be because a CD5$^+$ B-cell subset, which is prevalent in neonates but rare in adults, limits the production of pro-inflammatory cytokines by dendritic cells (DCs) in response to microorganisms by producing copious amounts of the regulatory cytokine interleukin-10 (IL-10). In addition, IL-10 production by CD5$^+$ B cells protected neonatal mice from overexuberant responses following an acute inflammatory challenge that was otherwise lethal in adult mice and in neonatal mice that lack CD5$^+$ B cells or IL-10. Interestingly, rather than promoting inflammation, DC-derived type I interferons stimulated the secretion of IL-10 by CD5$^+$ B cells, thereby acting to reinforce the regulatory loop in neonatal mice.