IN BRIEF

DENDRITIC CELLS

Distinct sources and targets of IL-10 during dendritic cell-driven T_H1 and T_H2 responses *in vivo*.

Perona-Wright, G. *et al. Eur. J. Immunol.* **36**, 2367–2375 (2006)

Interleukin-10 (IL-10) is known as a broadly immunosuppressive cytokine. When secreted by dendritic cells (DCs), it can function in both an autocrine and a paracrine manner, and it has been associated with both a T helper 2 (T_H2)-cell phenotype and T-cell tolerance. To clarify the role of IL-10 in shaping polarized T-cell responses to pathogen-derived antigens, the authors adoptively transferred pathogen-conditioned mouse DCs. They found that DC-derived IL-10 was not essential for the induction of DC-driven T_H1- and T_H2-cell responses *in vivo*. Interestingly, the regulation of emerging effector-cell populations was dominated by IL-10 produced by innate immune cells other than DCs or by non-haematopoietic cells. These data highlight the distinct actions of IL-10 during polarized DC-driven immune responses and emphasize that the function of IL-10 is context dependent.

INNATE IMMUNITY

A conserved role for a GATA transcription factor in regulating epithelial innate immune responses.

Shapira, M. *et al. Proc. Natl Acad. Sci. USA* **103**, 14086–14091 (2006)

An unbiased genome-wide study of *Caenorhabditis elegans* undertaken to improve our understanding of whole-organism host responses to infection has identified a conserved function for a transcription factor of the GATA family in the regulation of local innate immune responses in the epithelium. GATA-family transcription factors have indispensable roles in the development and the differentiation of diverse cell types in both vertebrates and invertebrates. The tissue-specific GATA-family transcription factor ELT-2 was found to be an important regulator in an intestinal innate immune response to the human bacterial pathogen *Pseudomonas aeruginosa*. The human homologue of ELT-2, GATA6, had a protective effect on lung epithelial cells exposed to the same pathogen, indicating that this transcription factor has a conserved role. Further work is required to determine which genes are regulated by GATA6 during infection, but a new mechanism involved in innate immunity has been uncovered.

SIGNALLING

The human adaptor SARM negatively regulates adaptor protein TRIF-dependent Toll-like receptor signaling.

Carty, M. *et al. Nature Immunol.* 10 Sep 2006 (doi:10.1038/ni1382)

Signalling through Toll-like receptors (TLRs) is an important process in immune-cell activation. So far, four adaptor proteins have been shown to mediate these signalling events, through two separate pathways. Now, new research shows that a fifth adaptor protein, SARM (sterile α- and Toll/interleukin-1 receptor (TIR)-motif-containing 1), is a negative regulator of TLR signalling. The inhibitory effect of SARM is focused on the TRIF (TIR-domain-containing adaptor protein inducing interferon-β)-dependent pathway and does not affect signalling through MyD88 (myeloid differentiation primary-response gene 88). Endogenous SARM expression is strongly increased by lipopolysaccharide, and SARM was shown to interact directly with TRIF. Small-interfering-RNA-mediated knockdown of *SARM* increased TRIF-dependent gene induction, confirming the inhibitory effect of SARM on this signalling pathway.