IN BRIEF

T CELLS

Antigen specific T–T interactions regulate CD4 T cell expansion.

Helft, J. *et al. Blood* 6 June 2008 (doi:10.1182/blood-2007-09-114389)

The expansion of naive and memory T-cell populations in response to antigen is an important aspect of an immune response but, once the T cells have seen the antigen, how is this expansion controlled? Helft *et al.* now show that both naive and memory T cells arriving early in the draining lymph node initially proliferate in an antigen-dependent manner, but only late-arriving naive T cells and not memory T cells continue to proliferate. This loss of memory T-cell responsiveness was not due to the disappearance of antigen but was dependent on T-cell–T-cell interactions. Responding $CD4^+$ T cells capture and present their cognate peptide–MHC class II complexes, which when presented to memory or antigen-experienced T cells inhibit their proliferation and induce anergy. By contrast, naive T cells remain responsive to T-cell-presented antigen. So, this study describes a mechanism for controlling T-cell expansion in an antigen-specific manner while maintaining repertoire diversity.

IMMUNE REGULATION

Myeloid-derived suppressor cells accumulate in kidney allograft tolerance and specifically suppress effector T cell expansion.

Dugast, A. *et al. J. Immunol.* **180**, 7898–7906 (2008)

Myeloid-derived suppressor cells (MDSCs) have previously been identified as a population that can inhibit effector immune-cell responses in tumours or in inflamed tissues. Here, Dugast *et al.* characterize a population of $CD6^-CD80/CD86^+NKRP1^+$ MDSCs that accumulated in tolerant kidney allografts in rats and report that the capacity of these cells to induce nitric-oxide production *in vivo* was involved in long-term graft acceptance. MDSCs were found to suppress T-cell proliferation by inducing their apoptosis through a mechanism that was dependent on cell–cell contact and inducible nitric-oxide-synthase activity. Interestingly, MDSCs were also found at lower numbers in mice transplanted with syngeneic grafts, suggesting that MDSCs are not only an induced population but are naturally occurring immune modulators that might be mobilized by tumours or in response to inflammation.

B CELLS

A unique B2 B cell subset in the intestine.

Shimomura, Y. *et al. J. Exp. Med.* **205**, 1343–1355 (2008)

Here, the authors describe a unique subset of IgM^+ B cells that is present within the normal mucosa of the large intestine. These intestinal B cells are characterized by a unique $AA4.1^-CD21^-CD23^-$MHC class II^{hi} surface phenotype, which is distinct from that of recirculating B cells from the spleen, mesenteric lymph nodes or Peyer's patches. They are present in the steady state, originating from $AA4.1^+$ immature B2 cells, and are also rapidly recruited to the inflamed intestine from the recirculating IgD^{hi} naive B-cell pool. Unlike most small-intestine plasma cells, these cells can develop in the absence of both the spleen and organized lymphoid tissues in a T-cell- and antigen-independent but BAFF (B-cell-activating factor)-dependent manner. They readily produce high levels of interleukin-12p70 in response to the microbial product CpG-containing DNA, indicating that intestinal IgM^+ B cells might have a role in protecting against infection in the gut.