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Regulation of memory CD4 T-cell pool size and function by natural killer T cells *in vivo*

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Abstract

To develop more effective vaccines and strategies to regulate chronic inflammatory diseases, it is important to understand the mechanisms of immunological memory. Factors regulating memory CD4⁺ T helper (Th)-cell pool size and function remain unclear, however. We show that activation of type I invariant natural killer T (iNKT) cells with glycolipid ligands and activation of type II natural killer T (NKT) cells with the endogenous ligand sulfatide induced dramatic proliferation and expansion of memory, but not naïve, CD4 T cells. NKT cell-induced proliferation of memory Th1 and Th2 cells was dependent largely on the production of IL-2, with Th2-cell proliferation also affected by loss of IL-4. Type II NKT cells were also required for efficient maintenance of memory CD4 T cells in vivo. Activation of iNKT cells resulted in up-regulation of IFN-γ expression by memory Th2 cells. These IFN-γ–producing memory Th2 cells showed a decreased capability to induce Th2 cytokines and eosinophilic airway inflammation. Thus, activated NKT cells directly regulate memory CD4 T-cell pool size and function via the production of cytokines *in vivo*.

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