IN BRIEF

T CELLS

Critical regulation of CD4$^+$ T cell survival and autoimmunity by β-arrestin 1.

Shi, Y. *et al. Nature Immunol.* **8**, 817–824 (2007)

Tight regulation of the numbers and activity of effector T cells is crucial to protect us from developing autoimmune conditions. Here, Shi *et al.* show that β-arrestin 1 may contribute to autoimmunity by acting as a positive regulator of T-cell survival. In mice overexpressing β-arrestin 1 (*Arrb1*-transgenic mice), more T cells survived after activation than in wild-type mice, whereas fewer T cells survived in *Arrb1*$^{-/-}$ mice. This pro-survival effect of β-arrestin 1 was due to upregulated expression of the anti-apoptotic gene *Bcl2* (B-cell lymphoma 2), through a mechanism involving acetylation of histone H4 at the *Bcl2* locus. Furthermore, *Arrb1*-transgenic mice developed more severe experimental autoimmune encephalomyelitis, whereas *Arrb1*$^{-/-}$ mice were less susceptible to the disease. Finally, autoreactive T cells from patients with multiple sclerosis had higher levels of β-arrestin 1 than T cells from control individuals, making β-arrestin 1 a possible susceptibility factor for autoimmunity.

B CELLS

Decreased expression of Krüppel-like factors in memory B cells induces the rapid response typical of secondary antibody responses.

Good, K. L. & Tangye, S. G. *Proc. Natl Acad. Sci. USA* **104**, 13420–13425 (2007)

Good and Tangye investigated the poorly defined mechanisms that regulate B-cell memory and the rapid recall response to specific antigen rechallenge by comparing the responses of human splenic naive and memory B-cell subsets to defined stimuli *in vitro*. Gene-expression profiling showed downregulated expression of the cell-cycle regulatory genes Krüppel-like factor 4 (*KLF4*), *KLF9* and promyelocytic leukaemia zinc finger (*PLZF*) in memory B cells compared with naive B cells. Enforced expression of these genes in memory B cells delayed their entry into division and reduced their proliferation. So, memory B cells seem to have a considerably reduced activation threshold compared with naive B cells that allows them to enter division more rapidly and produce a more robust antibody response.

AUTOIMMUNITY

BAFF and MyD88 signals promote a lupuslike disease independent of T cells.

Groom, J. R. *et al. J. Exp. Med.* **204**, 1959–1971 (2007)

Transgenic mice that overexpress the cytokine B-cell-activating factor (BAFF) develop an autoimmune disease similar to systemic lupus erythematosus (SLE) and have impaired B-cell tolerance and altered differentiation of T cells. Here, the role of T cells in BAFF-driven autoimmune disease was investigated. *Baff*-transgenic mice that lacked T cells developed a disease indistinguishable from that of *Baff*-transgenic mice. BAFF also promoted Toll-like receptor 7 (TLR7) and TLR9 expression on and TLR-induced autoantibody production by B cells. Moreover, the induction of disease in *Baff*-transgenic mice required B-cell-intrinsic signals through the TLR signalling adaptor protein MyD88 (myeloid differentiation primary-response gene 88). The development of BAFF-induced SLE-like disease in *Baff*-transgenic mice, therefore, is T-cell independent, but B-cell activation and pathogenic autoantibody production requires MyD88-dependent signalling.

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