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IN BRIEF

HIV

Fc receptor but not complement binding is important in antibody protection against HIV.

Hessell, A. J. *et al. Nature* **449**, 101–104 (2007)

Neutralizing antibodies block viral entry into host cells, but it is not their only function. b12 is one of the best-known neutralizing antibodies against HIV but how it functions *in vivo* is not clear. Here, Hessell *et al.* examine the ability of passive administration of b12 or two variant b12 antibodies — one that has abrogated complement-binding activity, and one that cannot bind to Fc receptors or complement proteins — to protect macaques against SHIV (an HIV–SIV chimaera) infection. They found that the loss of complement binding had no effect on protection, whereas the loss of Fc-receptor binding resulted in reduced protection against infection. This indicates that, in addition to blocking viral entry, the ability of this neutralizing antibody to activate immune cells by binding to their Fc receptors has a role in maximal protection against HIV.

DENDRITIC CELLS

BDCA2/FcεRIγ complex signals through a novel BCR-like pathway in human plasmacytoid dendritic cells.

Cao, W. *et al. PLoS Biol.* **5**, e248 (2007)

Human plasmacytoid dendritic cells (pDCs) express a C-type lectin called BDCA2 (blood DC antigen 2), which lacks an identifiable signalling motif. BDCA2 is known to be a potent regulator of pDC function, although how it transduces signals is unknown. Here, Lanier, Liu and colleagues show that BDCA2 signals in pDCs by forming a complex with the transmembrane adaptor protein FcεRIγ (high-affinity Fc receptor for IgE, γ-chain). The association of FcεRIγ with BDCA2 led to the activation of an immunoreceptor-based tyrosine activation motif (ITAM)-dependent signalling cascade, which was similar to that downstream of the B-cell receptor (BCR). This cascade suppressed the production of type I interferons and other cytokines in response to Toll-like receptor activation. Therefore, by associating with FcεRIγ, BDCA2 activates a BCR-like signalling pathway to regulate the immune function of pDCs in humans.

T CELLS

Ghrelin promotes thymopoiesis during aging.

Dixit, V. D. *et al. J. Clin. Invest.* 6 September 2007 (doi:10.1172/JCI30248)

Several reasons have been evoked to explain the loss of thymic function with age, and now Dixit *et al.* propose that the hormone ghrelin, better known as a stimulator of food intake, and its receptor may contribute to thymic involution. First, ghrelin and ghrelin receptor expression in the thymus were shown to decline with age. Second, infusion of ghrelin into old mice could partly reverse age-associated changes in thymic architecture and thymocyte numbers, increasing the thymic export of naive T cells and the diversity of T cells in the periphery. Third, mice deficient in ghrelin and ghrelin receptor showed enhanced thymic involution. Based on these observations, the authors warn against the proposed use of ghrelin inhibitors in the treatment of obesity and instead suggest the use of ghrelin in boosting T-cell numbers in elderly or immunocompromised individuals.