

3. Processing of the antigen involves cleavage of the antigen by proteolytic enzymes into short stretches of amino acids known as a peptide. The peptide then binds within a cleft of the MHC class II molecule, which is upregulated in order to facilitate presentation of the peptides, and is presented on the surface of the dendritic cell to T-cell receptors (TCR) on the surface of T-cells. CD4 on the surface of the T-cell binds to an invariant site on the MHC complex.
4. The co-receptors CD80 and CD86 are also upregulated in order to provide costimulation by engaging CD28 on the T-cells in the lymph nodes. The expression of B7 family proteins is controlled by NFκB, which is activated downstream of many PRRs. Naive T-cells require both the MHC class II plus peptide presented to them and costimulation from co-receptors to become successfully activated.
5. In addition, when a particular PRR is stimulated, the DC can produce particular cytokines that can help drive Th cells down the Th1 or Th2 differentiation pathways. For example, IL-12 production by DCs favours the development of Th1 cells.
6. In addition to activating T-cells, DCs can also inactivate T-cells. This occurs if DCs take up self-antigens. In the absence of microbial danger signals, the CD80 and CD86 costimulatory molecules are not expressed at significant levels. Costimulation is therefore not provided and self-reactive T-cells are made tolerant.

Activation of B-cells

1. FDCs have on their cell surface complement receptors and receptors for the Fc region of the antibody (FcγR).
2. FDCs capture whole antibody antigen complexes (sometimes including complement), rather than processed antigens at their surface for long periods and then present this to B-cell receptors (BCR), which are the transmembrane version of the antibody molecule.
3. Efficiency is increased because FDCs have many of the complement and Fc receptors on their cell surface and therefore, multiple copies of the antibody antigen complexes are presented to BCRs, making it more efficient.
4. If a B-cell recognises that complex with high affinity, it would engulf the antigen and present it to T-cells, allowing clonal expansion of T-cells. The low affinity B-cells however would die via apoptosis and are phagocytosed by macrophages.

Natural Killer (NK) cells

NK cells kill infected or abnormal (cancer) cells and can recognise the target cell in a number of different ways. They can recognise upregulated self-proteins on infected or tumour cells, as well as cells that lack the normal expression of MHC class I molecules on their surface and can also recognise antibody-coated cells. NK cells possess PRR-like receptors that can be categorised under killer activating receptors and the inhibitory receptors. The effect of a signal sent from one receptor is counterbalanced by the signal sent from the other in a normal cell. These activating and inhibitory receptors can both be of the lectin-like family or the immunoglobulin-like family.