Non MHC immune linked immune response -

Lipid Antigens — Lipid Antigens are presented to CD_4 cells by cell surface molecules designated cluster of differentiation (CD). Antigen presenting cells express several different forms of CDs at their surface. Each is probably specialized to bind a particular type of lipid Antigen (eg lipopeptide V/s glycolipid) The exposed surface of CD_4 molecules form an antigen binding groove much like that of MHC molecule except that the amino acids in the groove are more hydrophobic than these MHC like protein antigens lipid antigens are also presented as small antigen fragments.

Polysaccharide Antigens — Some bacterial polysaccharides ingested by antigen presenting cells (APCs) can be degraded in their lysosomes and presented to T cells by MHC class II molecules. Nitric oxide appears to be essential per this process.

The antigen after entry into tissues finishes up being trapped and drained into lymph nodes. Draining of antigens is done by macrophages. Antigens are also trapped by other APCs such as B cells and dendritic cells. The lipopolysaccharide (LPS) antigens of bacterial microbes are recognised and trapped through MHC class II antigens on B-cells Antigen sensitized macrophages stimulate T and B cells both.

The dendritic cells such as langerhans cells of skin trap and process peripheral antigens and then migrate in lymph ~~node~~ to settle as interdigitating cells in paracortical region of lymph node

Dendritic cells lack Ig receptors and complement protein -1 (CI) and they express high level of MHC class I and class II molecules for T cells and B cells respectively. In Paracortical region of lymph node, maturation of ITD cells is completed. ITD cells convey the antigen along with stimulatory factors to stimulate the specific T cells. T lymphocyte cells take benefit of large surface area of MHC peptide complex on ITD cells effectively bind with it. Secondary responses would be initiated the secondary antigen responses are well taken care by memory B cells.

Immunologically, the brain, testis and anterior chamber of eye are expressed as privilege sites These privilege sites are very strongly protected by blood tissue Barrier.

The above sites have very insignificant level of complements which reduces inflammatory reaction. In the words of Lesley Brent

"It may be supposed that it is beneficial to the organism not to turn the anterior chamber of the cornea of the eye or the brain into an inflammatory battle field for immunological response is sometime more damaging than the antigen insults that provokes it.

Mechanism of defence from Viral diseases —

Professional antigen presenting cells (APCs) like dendritic cells can use the class I as well as the class II Pathways of antigen presentation. This is

useful because (i) most viruses infect cells other than APC (antigen presenting cells) (ii) Viral antigens displayed on the surface of infected cells can serve as targets for cytolytic T lymphocytes (CTL's) (iii) The lack of any co-stimulatory molecules on the cell surface makes them poor stimulants for the development of clones of CTL's.

At least two mechanisms exist for transferring viral antigens from any infected cell to a professional APC. first- when an infected cell dies it can be engulfed by a a professional APC and the viral antigens within it can enter the class I pathway. The dead cell is engulfed by phagocytosis as discribed above. The endosome that fuses with a lysosome and degradation of dead cell begins. Viral antigens then pass into the cytosol and are degraded in proteasomes. The viral peptides formed are then packed up by TAP and inserted into class I MHC mole and displayed at the cell surface along with the co-stimulatory molecules needed to start a vigorous clonal expansion of CD8 cells. Second the cells infected with viruses can transfer viral peptides directly from their cytosol to an adjacent cell like a professional APC able to present the peptide with needed co-stimulatory molecules up to CTL's. The viral peptides can be transferred to a cell of the same type and can then present it on a class I molecule and be killed by CTL before infection. This mechanism provides a way of preventing spread of infection. In both cases the transfer occurs through gap junctions linking the adjacent cells.

Antigen presentation can be diverted from class I to class II pathway. Autophagy provides a mechanism by which cells can transfer intracellular antigens into the class II pathway in addition to class I. In this way viral infection can generate CD4 cells as well as cytotoxic CD8 cells.

B lymphocytes processes antigens by the class II pathway. They act as phagocytic cells as B cells engulf antigen by receptor mediated endocytosis and as B cell receptors for antigen (BCRs) are antibodies anchored in the plasma membrane. The affinity of BCRs for epitope on an antigen may be so high that B cell can bind and internalise the antigen when it is present in body fluids in concentration thousands of times smaller than needed by microphages. The remaining steps of antigen processing occurs by the same class II pathway described above. for macrophages producing fragments of antigen displayed at the cell surface nestled in the groove of class II histocompatibility molecules. A CD_4 cell that recognizes the displayed antigen is stimulated to release lymphokines. These in turn, stimulate the B cell to enter the cell cycle. Because of the part they play in stimulating B-cells, these CD_4 cells are called 'helper T-cells (T_H)'. The B-cells grow into a clone of cells called plasma cells. These cells synthesized BCRs with the identical binding site for the epitope but without the transmembrane tail. These antibodies are secreted into the surroundings.