IMMUNE SUPRESSION

① Upon encountering self-antigens, some of reactive T-cells may develop into regulatory T-cells which prevent the activation of other T-cells. The regulatory T-cells naturally may generate in Thymus or in the periphery due to several conditions

② Regulatory T-cells, $CD4^+$, $CD25^+$, $ForP3^+$ which supresses immune function in multiple cases

③ Some regulatory T-cells produce cytokines like interleukin (IL-10), TGFβ1 (Transforming Growth Factor β-1) which blocks the activation of lymphocytes & macrophages.

Regulatory T-cells may also directly interact with and supresses other lymphocytes & APCs by certain unidentified mechanisms.

It has been found that mice, lacking $CD25^+$ T-cells are better off compared to animals where $CD25^+$ T-cells were injected artificially. These mice develops disseminated autoimmune disease involving multiple

2021

B- Cell Tolerance:

Polysaccharides, lipids & nucleic acids are thymus independent antigens which are not recognized by T-cells. These antigens need to induce tolerance w.r.t. B- lymphocyte in order to prevent autoantibody formation. The principles of central and peripheral B- cell tolerance is similar to that of T- cell Tolerance.

NOTE: T- Independent Ag are those antigens which are not recognized by T-cell generally and constitute one of the significant antigenic repertoire where B- cells predominately play significant role.

Central B- cell Tolerance -

(1) Immature B- cells ~~c~~ interact with self antigen in bone marrow are deleted by negative selection. In certain instances, these cells may change their receptor specificity in order to avoid disastrous effect.

(2) The process of negative selection in B- cells is similar to that of T- cells. Like T- cells, B- cells with high affinity for self- antigens are deleted.

(3) Immature B- cells ~~that~~ when recognize self antigen in bone-marrow, they reactivate immunoglobulin (Ig) gene recombination machinary and begin to express new Ig like

light chain. This new light chain associate with previously expressed Ig heavy chain to produce a new antigenic receptor. This process is called as receptor editing.

(2) If central tolerance in B-cells fails, then chances of autoimmunity ~~occures~~ appears.

Peripheral B-cell Tolerance :-

Mature B-lymphocyte when encounter self-antigen(s) then they got anergy and can't respond to those antigen(s) (2) If a B-cell recognized a self-antigen but doesn't get T-cell help then also, B-cell become anergic.

For humans - IgG, G_2, G_3, G_{15}

mouse - IgG, IgG2a, 2b, IgG3

In our body IgE is very low 0.001 %.

On beting bee IgE is needed immediately for producing inflammatory response which can by stimulation of cytokines. Otherwise person will collapse the receptor editing.

T-independent antigen(s) activates B lymphocyte without T-cell help and can trigger strong signal in B-cell.

(3) Anergic B-cells may leave lymphoid follicle and are subsequently excluded from the follicles. These excluded B-cells doesn't get sufficient survival stimuli and eventually gets deleted.

(5) Autoimmune disease like Systemic lupus erythromatosus is caused due to defective tolerance both in B lymphocytes as well as helper T-cells.