Infections and diseases

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Describe the process of antigen presentation

- APC engulfs pathogen (bacterial cell) via phagocytosis where pseudopodia are formed and extended outwards to engulf bacteria when it encounters a pathogen
- Ends of pseudopodia fuse and vesicle pinched off and enters into cytoplasm , forming a phagosome
- The phagosome fuses with lysosomes to form phagolyosome
- The hydrolytic enzyme, lysosome breaks down the antigens on the surface of bacterial cell into short peptides
- The peptides are transported via vesicles to the cell surface of the antigen-presenting cell where they are presented on major histocompatibility complexes (MHC) which is synthesised in the endoplasmic reticulum
- Other components of the bacterial cell that are not presented are released from the cell via exocytosis.

Explain how affinity maturation occurs

- Affinity maturation is the result of somatic hypermutation of immunoglobulin genes in B cells.
- B cells with B cell receptors that bind to introduced antigen with higher affinity are fed survival signals and so they undergo clonal expansion in a process called clonal selection

- B cells with B cell receptors showing lower affinity are not fed survival signals and so they die from apoptosis.
- Plasma cells differentiated from selected B cells produced antibodies with higher affinity to the antigen.

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explain how somatic recombination during B cell development results in the formation of millions of different antibody molecules

- Different segments of V, D, J being ligated to form gene encoding different variable regions in heavy chain of the antibody
- refer to only one segment from each V, D and J segments is chosen and remaining gene segments removed / excised
- refer to different arrangement / combination of V, D, J segments being transcribed to give different mRNA sequences
- leading to different amino acid sequences, and subsequently different specific 3D conformation of variable region / code for different antibody molecules
- Tip: Describe the process in the DNA,RNA and then protein level

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How do influenza virus damage's epithelial cells

- Virus destroys epithelial cells after budding newly formed viruses leads to extensive plasma membrane loss
- Replication of influenza virus leads to usage of RNA nucleotides to make viral genome and amino acids used to make viral proteins, depriving host cell of these monomers for their own use

How does this damage lead to increase risk of bacterial infection

- Influenza virus infect the epithelial cells and destroy them, causing build up of dead epithelial cells and mucus in airways causing symptoms of running nose and sore throat
- Weakening of epithelial layer caused by viral replication can make respiratory passage more susceptible to secondary bacterial infections as it creates

environment for bacteria to grow

- With secondary bacterial infections, this can lead to diseases like pneumonia which can be fatal

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How does latent TB occur

- Once bacteria enters the lungs through airborne transmission, alveolar macrophages phagocytosise the bacteria
- Inside the phagosome of the macrophage, alveolar macrophages are unable to kill the bacteria they engulfed due to bacterium inhibiting the fusion of phagosome with lysosome to form phagolysosome
- Lysosomal enzymes are not available to break down the bacteria, M. Tuberculosis survives amd continue to multiply inside the macrophages
- Infected macrophages secretes cytokines to attract other immune cells such as T cells, resulting in clustering of immune cells and leads to formation of granuloma where macrophages, T cells and B cels form a cluster around undestroyed bacteria at the site of infection
- Necrosis of infected macrophages occurs in the centre of the granuloma, where the cell membrane of infected cells rupture and bacterium is released in the cavity, allowing it to replicate outside the macrophage

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how do vaccinations provide long term immunity (for people with severe symptoms)

- plasma cells are short lived and hence will decrease over time
- Vaccination exposé's individuals to a harmless form of a pathogen to induced specific adaptive immune responses to form memory B and T cells
- Upon vaccination, memory B cells are also produced along with plasma cells where they are long lived
- Memory cells able to proliferate and differentiate into effector/plasma cells upon re-exposure to the same antigen/virus resulting in a faster and stronger and longer lasting secondary response to provide immunity

- Earlier appearance of cytotoxic T cells will remove virus- infected cells by apoptosis
- Earlier production of antibodies by plasma B cells will allow virus particles to be neutralsied and removed by phagocytes through agglutination and opsonization, preventing virus from replicating and causing severe symptoms

Treatment of TB

- Since TB is caused by Mycobacterium tuberculosis bacterial infections, antibiotics can be used such as bacteriocidal
- Vaccination programmes can be introduced so that country can have herd immunity, preventing spread of TB and reduce number of deaths
- Vaccination programme can also produce memory B cells in individuals so that there is a faster, larger secondary immune response

How do antibodies prevent virus from infecting cell

- Binding of antigen binding site (Ig G) to the epitope of antigen causes neutralisation of antigen and prevents the antigen from binding to host cell surface receptor via receptor mediated endocytosis
- It also allows for oponisation, recruiting macrophages or phagocytes via the Fc region, increasing frequency of phagocytosis by macrophages

How affinity maturation occurs

- Activated B cells divide rapidly by mitosis during clonal expansion
- Some of these B cells can undergo somatic hypermutation, mutation occurs on Ig genes or genes that code for antibodies
- Each of these. Cells will acquire slight amino acid difference in variable regions on antigen binding site of BCR
- B cells that contain BCR with higher affinity selected for and undergo further clonal expansion and differentiation to form plasma cells

Outline roles of antibodies

- two antigen binding sites per antibody molecules allows agglutination of pathogen, able to bind to two antigens at the same time, pathogens clump to facilitate clearance
- Neutralisation of pathogen by antibodies which prevent entry of pathogen into host cell
- Fc region bind to Fc Fc receptors on phagocytes to promote phagocytosis which is opsonisation

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How are plasma cells formed from B lymphocytes

- Each B lymphocytes has one specific type of BCR that recognise an epitope which is complementary in conformation and bind to the specific antigen
- BCR together with bound antigen taken into B lymphocytes by endocytosis
- Antigen is processed into shirt peptides and attached to MHC complex
- Transported to CSM where peptide is recognised by antigen-specific helper T cell
- Interaction stimulates cytokines secretion from helper T cell
- In turn, these cytokines activate antigen specific B lymphocytes to undergo clonal expansion and differentiation into antibody secreting plasma cells