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Immune response to infection

- Defense against microbes is mediated by the effector mechanisms of innate and adaptive immunity.
- The survival and pathogenicity of microbes in a host are critically influenced by the ability of the microbes to evade or resist the effector mechanisms of immunity.
- Inherited and acquired defects in innate and adaptive immunity are important causes of susceptibility to infections.

Bacterial immunity :-

The principal mechanisms of innate immunity to extracellular bacteria are complement activation, phagocytosis, and the inflammatory response.

There are five basic mechanisms by which the adaptive immune responses combat bacterial infections:
(1) neutralization of toxins or enzymes by antibody
(2) killing of bacteria by the classical complement pathway
(3) opsonization of bacteria by antibodies and complement, resulting in their Phagocytosis and destruction
(4) destruction of intracellular bacteria by activated macrophages
(5) direct killing of bacteria by cytotoxic T cells and NK cells.

Bacteria evade Immune System by :

1- Presence of capsule
2- Multiplication of bacteria inside the macrophages, like salmonella, Brucella, Listeria, and Mycoplasma.
3- Changing of surface antigens: some bacteria like *Campylobacter fetus*.
4- Suppression of T lymphocytes: some bacteria have the ability to inhibit T cells, like *Mycoplasma mycoides*
5- Production of aphlatoxins: This toxin inhibits immune responses.
6- Release of cAMP (cyclic adenine monophosphate): This prevents fusion of lysosomes with phagosomes.

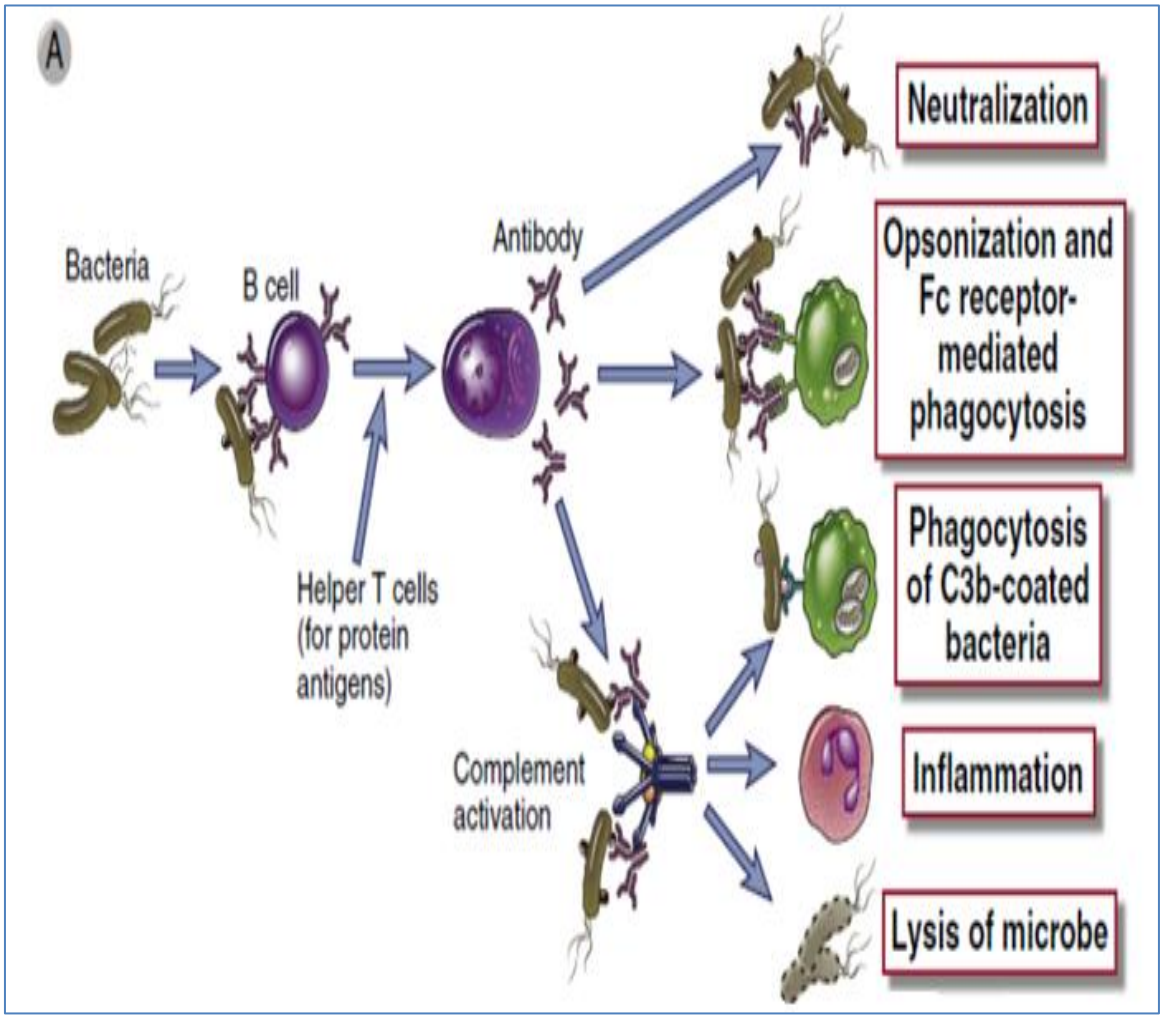
Summary of defense mechanisms

- The bacterial cell wall proteoglycan can be attacked by lysozyme.
- Bacteria release peptides, which are chemotactic for polymorphs.
- Polymorphs and macrophages use receptors for bacterial sugars to bind and slowly phagocytose them.
- Bacteria induce macrophages to release inflammatory cytokines such as interleukin-1 (IL-1) and IL-6 and tumour necrosis factor-α (TNF-α).
- Bacterial lipopolysaccharides and endotoxins activate the alternative complement pathway, generating opsonizing C3b on the bacterial surface.
- Bacterial polysaccharides (e.g., pneumococcal) with multiple repeated epitopes may activate B cells independently of T-helper cells.

▪ Exogenous processing of phagocytosed bacteria by macrophages results in presentation of peptide epitopes in the context of major histocompatibility complex (MHC) to T_H cells. These induce macrophage activation for efficient bacterial killing.

■ Processing of bacterial antigens by B cells induces TH2 responses and high-affinity antibody production:

IgG antibodies neutralize soluble bacterial products such as toxins; IgA antibodies protect mucosal surfaces from bacterial attachment. Immune complexes activate the classical complement pathway. Phagocytic uptake of bacteria coated with C3b/iC3b and antibody is rapid and efficient.



IMMUNITY TO VIRUSES

Innate immunity to virus by interferon while Adaptive immunity against viral infections is mediated by antibodies, which block virus binding and entry into host cells neutralizing them, and Cell-mediated responses are primarily responsible for antiviral immunity. The major mechanism involved is the killing of virus infected cells by cytotoxic T cells. • Because viruses are obligate intracellular parasites, they employ a wide variety of methods of evading the immune response .

Viruses evade Immune system by :

1- Changing of viral surface antigens: like influenza viruses.
2- Changing of cellular surface anti gens: like measles viruses.
3- Integration of viral N.A. with cellular N.A.: like HIV virus in AIDS.
4- Immuno-suppression due to lymphatic tissue infection.
5- Stress factors and Steroids

Summary of defense mechanisms

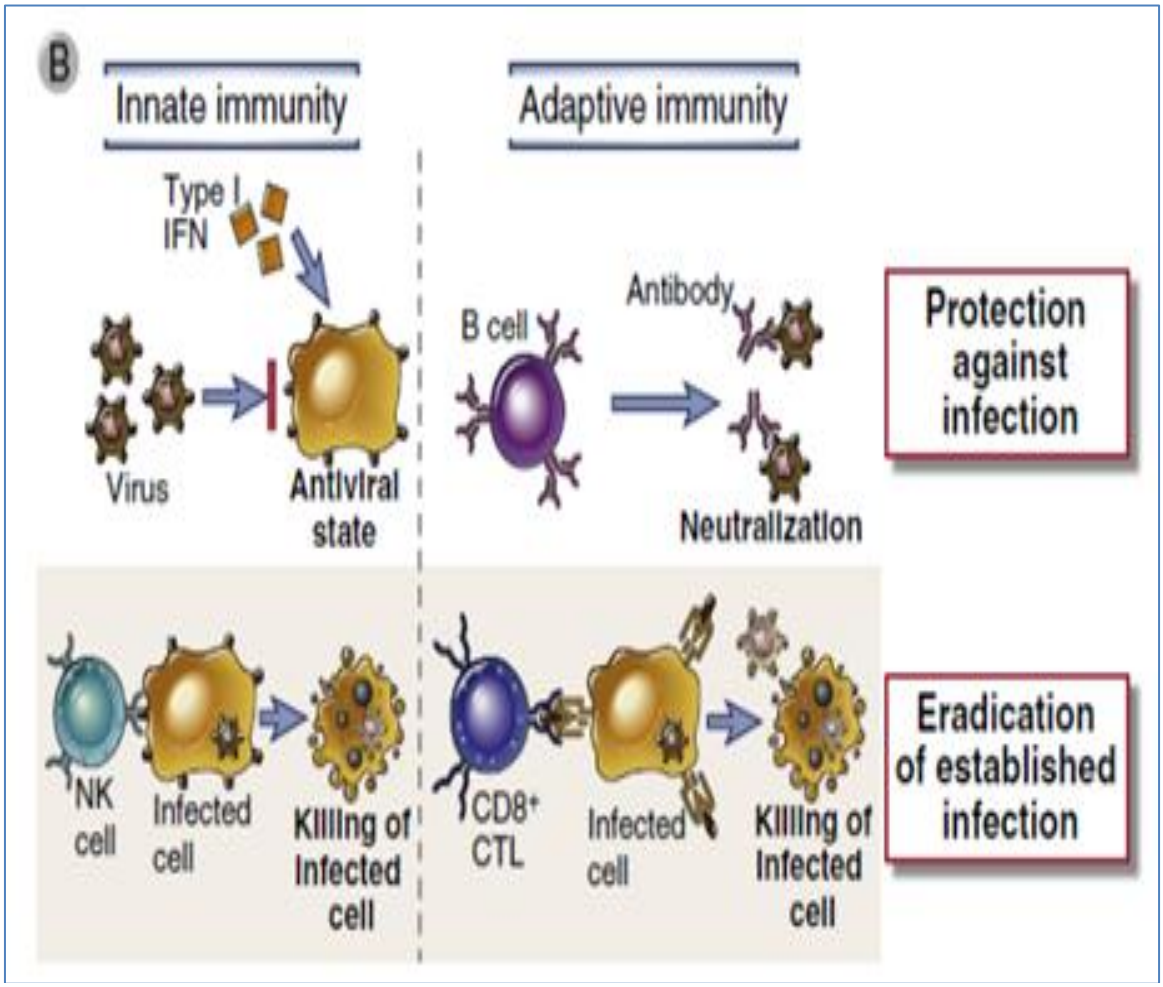
▪ Viral proliferation induces infected cells to produce interferons (IFN) α and β, which protect neighbouring cells from productive infection.

Interferons induce enzymes that inhibit messenger RNA translation into proteins and degrade both viral and host cell messenger RNA, effectively preventing the host cell from supporting replication of the virus or replicating itself.

▪ Some viruses, notably Epstein–Barr virus, bind Cl and activate the classical complement pathway.

▪ Macrophages readily take up viruses non-specifically and kill them. Some viruses, however, are able to survive and multiply in macrophages.

■ Processing of viral antigens by B cells and presentation to TH2 cells induces high-affinity antibody production. Antibodies are effective against free rather than cell-associated viruses. Antibody-coated viruses may be destroyed by the classical complement activation



Parasite immunity

In general antibody-mediated immune responses protect against extracellular protozoa, whereas cell-mediated responses control intracellular protozoa..

Helminth parasites have a unique ability to trigger T responses and immunoglobulin E (IgE) production. IgE may have evolved as an antiparasite antibody.

Parasitic worms have a thick cuticle that protects them against damage caused by most protective cells. However, Eosinophils appear to be uniquely able to damage and kill helminths.

Summary of defense mechanisms

▪ Protozoan parasites such as *Plasmodium* (malaria), *Leishmania* (leishmaniasis) and *Trypanosoma* (Chagas disease, sleeping sickness) induce macrophages to release inflammatory IL-1, IL-6 and TNF-α.

▪ Protozoa that survive within macrophages (e.g., *Trypanosoma cruzi*, *Leishmania*) can be killed following macrophage activation by T_H1 cells.

▪ IgG and IgM antibodies are effective against parasites that circulate freely in blood (e.g., *Trypanosoma brucei*, plasmodium sporozoite and merozoite stages) and against parasite-infected cells that display parasite antigens on the surface. Complement activation leads to target cell lysis and opsonization for phagocytosis.

■ IgE antibodies are of major importance against helminths such as *Schistosoma, Trichinella, Strongyloides* .antigens leads to release of eosinophil chemotactic factor.