

What is adaptive immunity?

What are the primary lymphoid tissues?

What are secondary lymphoid tissues?

What is an epitome?

What is spermine?

What is innate immunity?

What is a hapten?

What is a carrier?

What is an adjuvant?

The lymph nodes tonsils, adenoids, spleen, and mucosa-associated lymphoid tissues (MALT) are examples of this type of tissue.

The bone marrow, spleen, & thymus are examples of this type of tissue.

This type of immunity is characterized by lymphocytes. It is slower but is specific and has memory.

It is the "first line" of defense to invading pathogens. The cells and molecules involved provide rapid host protection without memory or specificity.

It is a component of semen that is a chemical barrier that prevents the growth of gram positive bacteria.

It is a unique portion of an antigen.

It is a substance that enhances immune responses to antigens (especially T-dependent ones).

It is a large molecule that binds to a hapten to make it immunogenic.

Small molecules that are nonimmunogenic until coupled to a larger molecule.

What are class II major histocompatibility proteins?

What are natural killer (NK) cells?

What are interleukin-2 & interleukin-12?

What is secondary response?

What are the major antigen presenting cells (APCs)?

What are the major cells involved in the inflammatory response?

What are monoclonal activators?

What are polyclonal activators?

What is primary response?

These are two cytokines that enhance the ability of NK cells to kill their targets.

These are cells whose main role is destruction of virally infected self cells.

These cell surface proteins when complexed with an antigenic peptide are recognized by T lymphocytes.

Basophils (Mast cells in tissues) & Eosinophils share this common role in the immune system.

Dendritic cells, macrophages, & B cells share this common role in the immune system.

This is the general term for the action the adaptive immune system takes on rechallenge by a pathogen the body has previously been exposed to.

This is the general term for the action the adaptive immune system takes on first exposure to a pathogen.

These are molecules that activate most or all clones of responding cells.

These are molecules that activate a single (antigen-specific) clone of responding cells.

What is the "bursa equivalent"?

What is tolerance induction?

What is death by neglect?

What are T helper (Th) cells?

What are the CD8+ subset of T cells?

What are interleukin-3 & interleukin-7?

What is interleukin-2?

What are plasma cells?

What are the CD4+ subset of T cells?

This is the deletion of T-cells that are of no value to the host.

This is the process by which potentially autoreactive cells are deleted or inactivated.

This is the site of antigen-independent B-cell maturation.

These are two interleukins that play a role in hematopoiesis.

This subset of cells recognizes antigen fragments presented in association with class I MHC expressed on the surface of autologous cells and are capable of destroying that cell.

These are cells that express the CD4 marker and whose primary role is the secretion of cytokines required for both adaptive & innate immunity.

This subset of cells recognize antigen fragments presented in association with class II MHC expressed on the surface of antigen presenting cells.

These are clonally expanded B cells that secrete antibody.

This is a cytokine that when present in high concentrations activates natural killer (NK) cells to lymphokine activated killer cells.

What is the marginal zone?

What is mucosa associated lymphoid tissue (MALT)?

What is follicle associated epithelium (FAE)?

What are secondary follicles?

What are the cells located in the medulla of a lymph node?

What is the periarteriolar lymphatic sheath (PALS)?

What is negative selection?

What is the thymus?

What are primary follicles?

These are regions in the gut where there are no goblet cells and the mucus layer is sparse or absent.

These are unencapsulated lymphoid tissues present in regions underlying mucosal areas.

This is the predominant B-cell region of the spleen.

This is the predominant T-cell region of the spleen.

Macrophages, dendritic cells, & plasma cells are located in this region of a lymph node.

These are antigen activated B-cells (germinal centers) in lymph nodes, the marginal zone of the spleen or, MALT tissue.

These are mature resting B-cells in lymph nodes, the marginal zone of the spleen or, MALT tissue.

This is the organ in which T-cells mature.

This is the deletion of T-cells that are potentially autoreactive.

What are oligocolnal activators?

What is an immunogen?

What are T-independent antigens?

What pathway do IgM & IgG activate?

What is IgE?

What pathway does the deposition of spontaneously generated fragments on microbial cell surface activate?

What are M cells?

What are Peyer's patches?

What is "complement"?

These are molecules that can activate B cells in the absence of T cell help. Examples include:  
(1) Polysaccharides (e.g. pneumococcal polysaccharide)  
(2) Lipopolysaccharide (e.g. cell wall component of gram negative bacteria)

This is a molecule that induces the activation of T cells or B cells.

These are molecules that activate more than one clone, but not all clones.

This process activates alternative pathway of complement (innate immunity).

This immunoglobulin is essential for the activation of eosinophils.

These immunoglobulins activate the classical pathway of complement (adaptive immunity).

This is a term used to describe a family of regulatory proteins that is mainly synthesized by the liver & circulate through the body in dormant form. They facilitate elimination of microorganisms, particularly bacteria.

These are MALTs that are abundant in the ileum and are also present in the jejunum.

These cells are located in the FAE and transport microorganisms from the luminal surface to the lamina propria to follicles termed Peyer's patches.

What is C3b?

What are C3a, C4a, & C5a?

What is C4b2a?

What is V $\beta$ ?

What are Type II T-independent antigens?

What are Type I T-independent antigens?

What are T-dependent antigens?

What is aluminum hydroxide?

What are superantigens?

This is the C3 convertase in the classic complement pathway. It cleaves C3 to C3a & C3b.

They are anaphylatoxins generated during the alternative & complement pathways. They bind to cognate receptors on mast cells and basophils stimulating anaphylaxis.

This is a complement protein that is involved.  
(1) Opsonization when deposited on microbial surfaces  
(2) Facilitates the elimination of immune complexes when deposited on immune complexes  
(3) Component of C3 convertase in the alternative pathway  
(4) Component of C5 convertase in the terminal pathway (classic & alternative)

These are molecules that can activate B cells in the absence of T cell help. The prototypic example of this type of antigen is the lipopolysaccharide that is a component of gram negative bacteria.

These are molecules that can activate B cells in the absence of T cell help. The prototypic example of this type of antigen is the pneumococcal polysaccharide.

It is a T-cell receptor (~200 different ones) that are shared by the entire T-cell population. They bind to superantigens leading to T cell clonal expansion of many by not all clones.

These are often bacterial products that activate a subset of T cells in a nonantigen specific & oligoclonal fashion.

This is a chemical mixed with antigen in human vaccines to enhance immunogenicity, presumably by slowing the release of antigen, so that presentation to T cells is prolonged.

These are molecules that require the T cell help for the activation of B cells. Examples include:  
(1) Proteins

What is Factor H?

What is properdin?

What is C5b?

What is C3bBbC3b (Properdin)?

What is anaphylatoxin inhibitor?

What is Factor I?

What is C1?

What is C3bBb (Properdin)?

What is C4b2a3b?

This complement fraction activates the formation of the membrane attack complex?

This is a tetrameric protein that stabilizes C3 convertase and extends its half 2 life by 6-10 times.

This is a cofactor that along with Factor I inactivates free C3b.

This cofactor also causes dissociation of the C3 convertase (C3bBb) of the alternative pathway.

This inactivates C3b using Factor H.

Also inactivates C4b using cofactors CR1, MCP, or C4 binding protein (C4bp)

This inactivates C3a.

This is the C5 convertase in the alternative complement pathway. It cleaves C5 to C5a & C5b.

This is the C5 convertase in the classic complement pathway. It cleaves C5 to C5a & C5b.

This is the C3 convertase in the alternative complement pathway. It cleaves C3 to C3a & C3b.

This is a protein that binds to antibodies (IgM & IgG) that are bound to bacteria & then cleaves C2 to C2a & C2b and cleaves C4 to C4b and C4a.

What are CD59 & HRF  
(homologous restriction factor)?

What is kininogen?

What is bradykinin?

What is C1 INH (C1 esterase  
inhibitor)?

What is kallikrein?

What is S-protein?

What is DAF?

What is CR1 (complement receptor  
1)?

What is anaphylatoxin

It is activated by kallikrein through cleavage of kininogen. It is important in the establishment of an inflammatory response.

It is activated by kallikrein and is a potent vasodilator that increases vascular permeability.

This is a MAC inhibitor that works by binding C8, preventing the binding and polymerization of C9 and formation of MAC.

This is a MAC inhibitor that works by binding soluble C5b, C6, & C7 complexes preventing their insertion into autologous membranes.

This is a protein that links the complement & intrinsic coagulation pathway.

This protein is inhibited by C1 INH (C1 esterase inhibitor).

It is activated from its inactive form by Factor XII.

It activates Factor XII to XIIa enhancing coagulation.

Uses C5 as a substrate and

This forms a complex with C1 preventing the spontaneous activation of the classical complement pathway.

This inactivates kallikrein.

This binds to C3a, C4a, & C5a and inhibits them binding to cognate receptors on mast cells and basophils.

This is a cofactor for Factor I.

This binds membrane bound C4b and C3b, blocking the formation of the alternative and classical pathway C3 convertases. If the C3 convertases have already formed it binds to C3b or C4b promoting their dissociation.

This binds membrane bound C3bi and C4bi preventing their degradation to biologically active

This binds membrane bound C4b and C3b, blocking the formation of the alternative and classical pathway C3 convertases. If the C3 convertases have already formed it binds to C3b or C4b promoting their dissociation.