(RESEARCH ARTICLE)

A primer on immune responses and mechanisms

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Abstract:

The immune response is how your body recognizes and defends itself against bacteria, viruses, and substances that appear foreign and harmful. Innate, or nonspecific, immunity is the defense system with which you were born. It protects you against all antigens. Innate immunity involves barriers that keep harmful materials from entering your body.

An antigen-presenting cell (APC) is a cell that displays antigen bound by major histocompatibility complex (MHC) proteins on its surface; this process is known as antigen presentation. T cells may recognize these complexes using their T cell receptors (TCRs). APCs process antigens and present them to T-cells. They are found in a variety of tissue types. Professional antigen-presenting cells, including macrophages, B cells and dendritic cells, present foreign antigens to helper T cells, while virus-infected cells (or cancer cells) can present antigens originating inside the cell to cytotoxic T cells

There are two broad classes of immune responses—antibody responses and cell-mediated immune responses, and they are carried out by different classes of lymphocytes, called B cells and T cells, respectively. The way the body defends itself against substances it sees as harmful or foreign. In an immune response, the immune system recognizes the antigens (usually proteins) on the surface of substances or microorganisms, such as bacteria or viruses, and attacks and destroys, or tries to destroy, them.

Keywords: Immune Response; Innate; Adaptive Immunity; Mechanisms

1. Introduction

1.1. History

The term immunity is derived from the Latin word *immunitas* which referred to the protection from legal prosecution offered to Roman Senators during their tenures in office.

An immune response is a reaction which occurs within an organism for the purpose of defending against foreign pathogens. These pathogens include a wide variety of different microorganisms including viruses, bacteria, parasites, and fungi which could cause serious problems to the health of the host organism if not cleared from the body

There are two distinct aspects of the immune response, the innate and the adaptive, which work together to protect against pathogens.

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The innate branch—the body's first reaction to an invader—is known to be a non-specific and quick response to any sort of pathogen. Components of the innate immune response include physical barriers like the skin and mucous membranes, immune cells such as neutrophils, macrophages, and monocytes, and soluble factors including cytokines and complement

On the other hand, the adaptive branch is the body's immune response which is catered against specific antigens and thus, it takes longer to activate the components involved. The adaptive branch include cells such as T cell, B cells „as well as molecules e.g antibodies—also known as immunoglobulin's—which directly interact with antigen and are a very important component for a strong response against a pathogen..

2. Innate immunity

Innate immunity represents the first line of defense to an intruding pathogen. It is an *antigen-independent (nonspecific) defense mechanism* that is used by the host immediately or within hours of encountering an antigen. The innate immune response has no immunologic memory and, therefore, it is unable to recognize or "memorize" the same pathogen should the body be exposed to it in the future. Adaptive immunity, on the other hand, *is antigen-dependent and antigen-specific* and, therefore, involves a lag time between exposure to the antigen and maximal response. The hallmark of adaptive immunity is the capacity for memory which enables the host to mount a more rapid and efficient immune response upon subsequent exposure to the antigen. Innate and adaptive immunity are not mutually exclusive mechanisms of host defense, but rather are complementary, with defects in either system resulting in host vulnerability or inappropriate response.

Innate immunity can be viewed as comprising four types of defensive barriers: anatomic (skin and mucous membrane), physiologic (temperature, low pH and chemical mediators), endocytic and phagocytic, and inflammatory. Cells and processes that are critical for effective innate immunity to pathogens that evade the anatomic barriers have been widely studied. Innate immunity to pathogens relies on pattern recognition receptors (PRRs) which allow a limited range of immune cells to detect and respond rapidly to a wide range of pathogens that share common structures, known as pathogen associated molecular patterns (PAMPs). Examples of these include bacterial cell wall components such as lipopolysaccharides (LPS) and double-stranded ribonucleic acid (RNA) produced during viral infection. An important function of innate immunity is the rapid recruitment of immune cells to sites of infection and inflammation through the production of cytokines and chemokines (small proteins involved in cell– cell communication and recruitment). Cytokine production during innate immunity mobilizes many defense mechanisms throughout the body while also activating local cellular responses to infection or injury.

These cytokines are critical for initiating cell recruitment and the local inflammation which is essential for clearance of many pathogens. Inflammation is the process of recruitment of leukocytes and plasma proteins from the blood, their accumulation in tissues, and their activation to destroy microbes. Among the most important plasma proteins of innate immunity are the components of the alternative pathway of the complement system.

The complement system is a biochemical cascade that functions to identify and opsonize (coat) bacteria and other pathogens. It renders pathogens susceptible to phagocytosis, a process by which immune cells engulf microbes and remove cell debris, and also kills some pathogens and infected cells directly.

The phagocytic action of the innate immune response promotes clearance of dead cells or antibody complexes and removes foreign substances present in organs, tissues, blood and lymph. It can also activate the adaptive immune response through the mobilization and activation of antigen-presenting cells (APCs). Numerous cells are involved in the innate immune response such as phagocytes (macrophages and neutrophils), dendritic cells, mast cells, basophils, eosinophils, natural killer (NK) cells and innate lymphoid cells. Phagocytes are sub-divided into two main cell types: neutrophils and macrophages. Both of these cells share a similar function: to engulf (phagocytose) microbes and kill them through multiple bactericidal pathways. In addition to their phagocytic properties, neutrophils contain granules and enzyme pathways that assist in the elimination of pathogenic microbes.

Dendritic cells also phagocytose and function as APCs, initiating the acquired immune response and acting as important messengers between innate and adaptive immunity. Mast cells and basophils share many salient features with each other, and both are instrumental in the initiation of acute inflammatory responses, such as those seen in allergy and asthma. Mast cells also have important functions as immune "sentinel cells" and are early producers of cytokines in response to infection or injury. Unlike mast cells, which generally reside in the connective tissue surrounding blood vessels and are particularly common at mucosal surfaces, basophils reside in the circulation.

Eosinophils are granulocytes that possess phagocytic properties and play an important role in the destruction of parasites that are often too large to be phagocytosed. Along with mast cells and basophils, they also control mechanisms associated with allergy and asthma. Natural killer (NK) cells play a major role in the rejection of tumors and the destruction of cells infected by viruses. Destruction of infected cells is achieved through the release of perforins and granzymes (proteins that cause lysis of target cells) from NK-cell granules which induce apoptosis (programmed cell death) [4]. NK cells are also an important source of another cytokine, interferon-gamma (IFN-γ), which helps to mobilize APCs and promote the development of effective antiviral immunity.

Innate lymphoid cells (ILCs) play a more regulatory role. Depending on their type (i.e., ILC-1, ILC-2, ILC-3), they selectively produce cytokines such as IL-4, IFN-γ and IL-17 that help to direct the appropriate immune response to specific pathogens and contribute to immune regulation in that tissue.

3. Adaptive immunity

The development of adaptive immunity is aided by the actions of the innate immune system, and is critical when innate immunity is ineffective in eliminating infectious agents. The primary functions of the adaptive immune response are: the recognition of specific "non-self" antigens, distinguishing them from "self" antigens; the generation of pathogen-specific immunologic effector pathways that eliminate specific pathogens or pathogen-infected cells; and the development of an immunologic memory that can quickly eliminate a specific pathogen should subsequent infections occur

Adaptive immune responses are the basis for effective immunization against infectious diseases. The cells of the adaptive immune system include: antigen-specific T cells, which are activated to proliferate through the action of APCs, and B cells which differentiate into plasma cells to produce antibodies.

3.1. T cells and APCs

Red cells, granulocytes, monocytes, dendritic cells, platelets, B and T cells as well as NK cells all originate from a common hematopoietic stem cell (HSC) in the bone marrow. T cells following migration, mature in the thymus. These cells express a series of unique antigen-binding receptors on their membrane, known as the T-cell receptor (TCR). Each T cell expresses a single type of TCR and has the capacity to rapidly proliferate and differentiate if it receives the appropriate signals. T cells require the action of APCs (usually dendritic cells, but also macrophages, B cells, fibroblasts and epithelial cells) to recognize a specific antigen.

The surfaces of APCs express a group of proteins known as the major histocompatibility complex (MHC). MHC are classified as either class I (also termed human leukocyte antigen [HLA] A, B and C) which are found on all nucleated cells, or class II (also termed HLA DP, DQ and DR) which are found only on certain cells of the immune system, including macrophages, dendritic cells and B cells. Class I MHC molecules present endogenous (intracellular) peptides, while class II molecules on APCs present exogenous (extracellular) peptides to T cells. The MHC-antigen complex activates the TCR and the T cell secretes cytokines which further control the immune response.

The antigen presentation process stimulates T cells to differentiate primarily into either cytotoxic T cells (CD8+ cells) or T-helper (T) cells (CD4+ cells). . CD8+ cytotoxic T cells are primarily involved in the destruction of cells infected by foreign agents, such as viruses, and the killing of tumor cells expressing appropriate antigens. They are activated by the interaction of their TCR with peptide bound to MHC class I molecules. Clonal expansion of cytotoxic T cells produces effector cells which release substances that induce apoptosis of target cells. Upon resolution of the infection, most effector cells die and are cleared by phagocytes. However, a few of these cells are retained as memory cells that can quickly differentiate into effector cells upon subsequent encounters with the same antigen.

CD4+ T cells play an important role in establishing and maximizing the immune response. These cells have no cytotoxic or phagocytic activity, and cannot directly kill infected cells or clear pathogens. However, they "mediate" the immune response by directing other cells to perform these tasks and regulate the type of immune response that develops. T cells are activated through TCR recognition of antigen bound to class II MHC molecules. Once activated, T cells release cytokines that influence the activity of many cell types, including the APCs that activate them.

The T2 response is characterized by the release of cytokines (IL-4, 5 and 13) which are involved in the development of immunoglobulin E (IgE) antibody producing B cells, as well as the development and recruitment of mast cells and eosinophils that are essential for effective responses against many parasites. In addition, they enhance the production of certain forms of IgG that aid in combatting bacterial infection.

T17 cells have been more recently described. They are characterized by the production of cytokines of the IL-17 family, and are associated with ongoing inflammatory responses, particularly in chronic infection and disease.

A subset of the CD4+ T cell, known as the regulatory T cell (Treg), also plays a role in the immune response. Treg cells limit and suppress immune responses and, thereby, may function to control aberrant responses to self-antigens and the development of autoimmune disease. Treg cells may also help in the resolution of normal immune responses, as pathogens or antigens are eliminated. These cells also play a critical role in the development of "immune tolerance" to certain foreign antigens, such as those found in food.

3.1.1. B cells

B cells arise from hematopoietic stem cells in the bone marrow and, following maturation, leave the marrow expressing a unique antigen-binding receptor on their membrane. Unlike T cells, B cells can recognize antigens directly, without the need for APCs, through unique antibodies expressed on their cell surface. The principal function of B cells is the production of antibodies against foreign antigens which requires their further differentiation

Under certain circumstances, B cells can also act as APCs. When activated by foreign antigens to which they have an appropriate antigen specific receptor, B cells undergo proliferation and differentiate into antibody-secreting plasma cells or memory B cells. Memory B cells are "long-lived" survivors of past infection and continue to express antigen-binding receptors. These cells can be called upon to respond quickly by producing antibodies and eliminating an antigen upon re-exposure. Plasma cells, on the other hand, are relatively short-lived cells that often undergo apoptosis when the inciting agent that induced the immune response is eliminated. However, these cells produce large amounts of antibody that enter the circulation and tissues providing effective protection against pathogens. Given their function in antibody production, B cells play a major role in the humoral or antibody-mediated immune response (as opposed to the cell-mediated immune response, which is governed primarily by T cells).

3.2. Antibody-mediated vs. Cell-mediated immunity

Antibody-mediated immunity is the branch of the acquired immune system that is mediated by B-cell antibody production. The antibody-production pathway begins when the B cell's antigen-binding receptor recognizes and binds to antigen in its native form. Local T cells secrete cytokines that help the B cell multiply and direct the type of antibody that will be subsequently produced. Some cytokines, such as IL-6, help B-cells to mature into antibody-secreting plasma cells. The secreted antibodies bind to antigens on the surface of pathogens, fagging them for destruction through complement activation, opsonin promotion of phagocytosis and pathogen elimination by immune effector cells. Upon elimination of the pathogen, the antigen–antibody complexes are cleared by the complement cascade.

3.2.1. T cell receptor and function

T cells play a central role in the immune system as effectors and regulators. They become activated upon antigen recognition by their T-cell receptors (TCR). The TCR repertoire is established by developmentally regulated TCR gene rearrangements and shaped by predominantly intrathymic selection processes. Failure of this system can lead to autoimmune disease.

The TCR, a defining structure of T cells, is a transmembrane heterodimer consisting of either an alpha and beta chain or delta and gamma chain linked by a disulphide bond. Within these chains are complementary determining regions (CDRs) which determine the antigen to which the TCR will bind.

The T-cell receptor (TCR) is a protein complex found on the surface of T cells, or T lymphocytes,[] that is responsible for recognizing fragments of antigen as peptides bound to major histocompatibility complex (MHC) molecules. The binding between TCR and antigen peptides is of relatively low affinity and is degenerate: that is, many TCRs recognize the same antigen peptide and many antigen peptides are recognized by the same TCR.

The TCR is composed of two different protein chains (that is, it is a heterodimer). In humans, in 95% of T cells the TCR consists of an alpha (α) chain and a beta (β) chain (encoded by TRA and TRB, respectively), whereas in 5% of T cells the TCR consists of gamma and delta (γ/δ) chains (encoded by TRG and TRD, respectively).

When the TCR engages with antigenic peptide and MHC (peptide/MHC), the T lymphocyte is activated through signal transduction, that is, a series of biochemical events mediated by associated enzymes, co-receptors, specialized adaptor molecules, and activated or released transcription factors. Based on the initial receptor triggering mechanism, the TCR belongs to the family of non-catalytic tyrosine-phosphorylated receptors (NTRs).

Signal transduction is the process by which a chemical or physical signal is transmitted through a cell as a series of molecular events, most commonly protein phosphorylation catalyzed by protein kinases, which ultimately results in a cellular response. Proteins responsible for detecting stimuli are generally termed receptors, although in some cases the term sensor is used. The changes elicited by ligand binding (or signal sensing) in a receptor give rise to a biochemical cascade, which is a chain of biochemical events known as a signaling pathway.

The major histocompatibility complex (MHC) is a large locus on vertebrate DNA containing a set of closely linked polymorphic genes that code for cell surface proteins essential for the adaptive immune system. These cell surface proteins are called MHC molecules.

This locus got its name because it was discovered via the study of transplanted tissue compatibility. Later studies revealed that tissue rejection due to incompatibility is only a facet of the full function of MHC molecules: binding an antigen derived from self-proteins, or from pathogens, and bringing the antigen presentation to the cell surface for recognition by the appropriate T-cells.[2] MHC molecules mediate the interactions of leukocytes, also called white blood cells (WBCs), with other leukocytes or with body cells. The MHC determines donor compatibility for organ transplant, as well as one's susceptibility to autoimmune diseases.
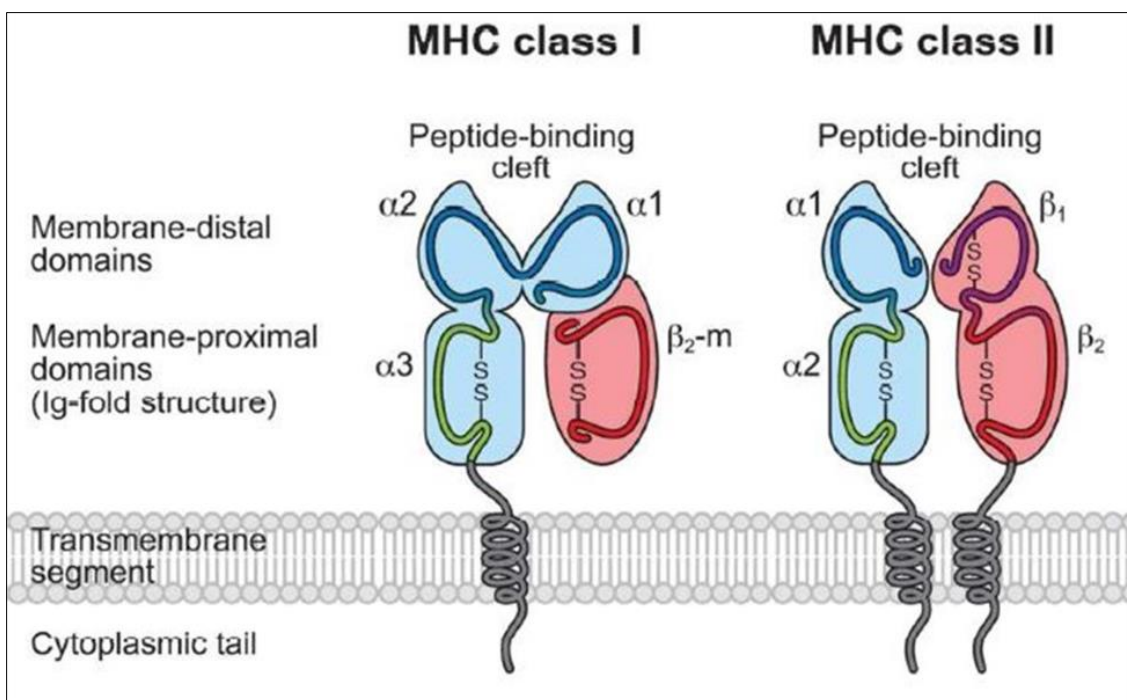
Figure 1 Major histocompatibility complex

In a cell, protein molecules of the host's own phenotype or of other biologic entities are continually synthesized and degraded. Each MHC molecule on the cell surface displays a small peptide (a molecular fraction of a protein) called an epitope.[3] The presented self-antigens prevent an organism's immune system from targeting its own cells. The presentation of pathogen-derived proteins results in the elimination of the infected cell by the immune system.

Protein phosphorylation is a reversible post-translational modification of proteins in which an amino acid residue is phosphorylated by a protein kinase by the addition of a covalently bound phosphate group. Phosphorylation alters the structural conformation of a protein, causing it to become either activated or deactivated, or otherwise modifying its function.[1] Approximately 13000 human proteins have sites that are phosphorylated.[2]

The reverse reaction of phosphorylation is called dephosphorylation, and is catalyzed by protein phosphatases. Protein kinases and phosphatases work independently and in a balance to regulate the function of proteins.[3]

The amino acids most commonly phosphorylated are serine, threonine, tyrosine in eukaryotes, and also histidine in prokaryotes[4] and plants (though it is now known to be common in humans). These phosphorylations play important and well-characterized roles in signaling pathways and metabolism.

Tyrosine phosphorylation is the addition of a phosphate (PO_4^{3-}) group to the amino acid tyrosine on a protein. It is one of the main types of protein phosphorylation. This transfer is made possible through enzymes called tyrosine kinases. Tyrosine phosphorylation is a key step in signal transduction and the regulation of enzymatic activity.
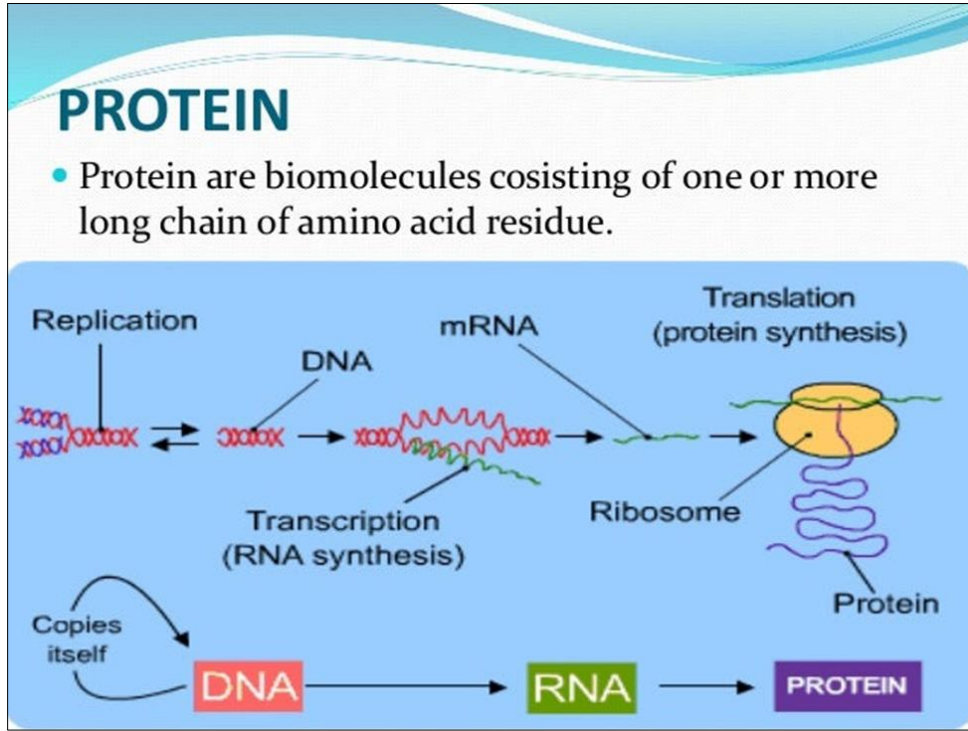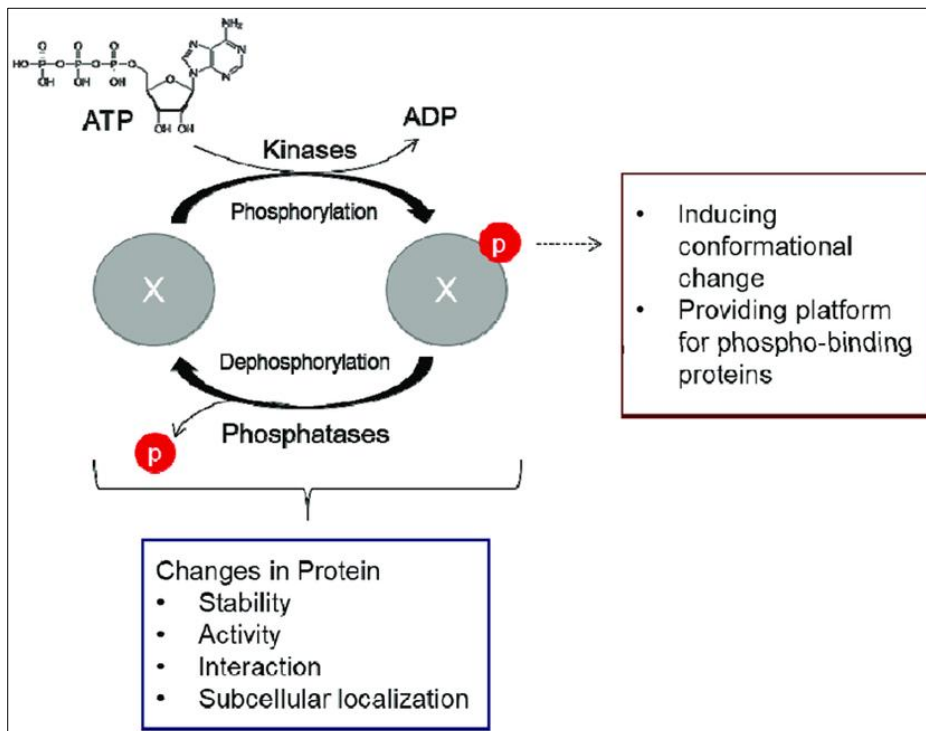

Figure 2 Proteins

Figure 3 Protein phosphorylation and dephosphorylation

MHC class I molecules are expressed in some nucleated cells and also in platelets—in essence all cells but red blood cells. It presents epitopes to killer T cells, also called cytotoxic T lymphocytes (CTLs). A CTL expresses CD8 receptors, in addition to T-cell receptors (TCR)s. When a CTL's CD8 receptor docks to a MHC class I molecule, if the CTL's TCR fits the epitope within the MHC class I molecule, the CTL triggers the cell to undergo programmed cell death by apoptosis. Thus, MHC class I helps mediate cellular immunity, a primary means to address intracellular pathogens, such as viruses and some bacteria, including bacterial L forms, bacterial genus *Mycoplasma*, and bacterial genus *Rickettsia*. In humans, MHC class I comprises HLA-A, HLA-B, and HLA-C molecules.

MHC class II can be conditionally expressed by all cell types, but normally occurs only on "professional" antigen-presenting cells (APCs): macrophages, B cells, and especially dendritic cells (DCs). An APC takes up an antigenic protein, performs antigen processing, and returns a molecular fraction of it—a fraction termed the epitope—and displays it on the APC's surface coupled within an MHC class II molecule (antigen presentation). On the cell's surface, the epitope can be recognized by immunologic structures like T-cell receptors (TCRs). The molecular region which binds to the epitope is the paratope.

On surfaces of helper T cells are CD4 receptors, as well as TCRs. When a naive helper T cell's CD4 molecule docks to an APC's MHC class II molecule, its TCR can meet and bind the epitope coupled within the MHC class II. This event primes the naive T cell. According to the local milieu, that is, the balance of cytokines secreted by APCs in the microenvironment, the naive helper T cell (Th_0) polarizes into either a memory Th cell or an effector The cell of phenotype either type 1 (Th_1), type 2 (Th_2), type 17 (Th_{17}), or regulatory/suppressor (T_{reg}), as so far identified, The cell's terminal differentiation

3.2.2. Antigen presentation

Antigen presentation is a vital immune process that is essential for T cell immune response triggering. Because T cells recognize only fragmented antigens displayed on cell surfaces, antigen processing must occur before the antigen fragment, now bound to the major histocompatibility complex (MHC), is transported to the surface of the cell, a process known as presentation, where it can be recognized by a T-cell receptor. If there has been an infection with viruses or bacteria, the cell will present an endogenous or exogenous peptide fragment derived from the antigen by MHC molecules.

There are two types of MHC molecules which differ in the behavior of the antigens: MHC class I molecules (MHC-I) bind peptides from the cell cytosol, while peptides generated in the endocytic vesicles after internalization are bound to MHC class II (MHC-II)

Cellular membranes separate these two cellular environments - intracellular and extracellular. Each T cell can only recognize tens to hundreds of copies of a unique sequence of a single peptide among thousands of other peptides presented on the same cell, because an MHC molecule in one cell can bind to quite a large range of peptides.

3.2.3. B-cell receptor

The B cell receptor (BCR) is a transmembrane protein on the surface of a B cell. A B cell receptor is composed of a membrane-bound immunoglobulin molecule and a signal transduction moiety. The former forms a type 1 transmembrane receptor protein, and is typically located on the outer surface of these lymphocyte cells. Through biochemical signaling and by physically acquiring antigens from the immune synapses, the BCR controls the activation of the B cell.

A transmembrane protein (TP) is a type of integral membrane protein that spans the entirety of the cell membrane. Many transmembrane proteins function as gateways to permit the transport of specific substances across the membrane. They frequently undergo significant conformational changes to move a substance through the membrane. They are usually highly hydrophobic and aggregate and precipitate in water.

The B cell receptor is composed of two parts:

A membrane-bound immunoglobulin molecule of one isotype (IgD, IgM, IgA, IgG, or IgE). With the exception of the presence of an integral membrane domain, these are identical to a monomeric version of their secreted forms.

Signal transduction moiety: a heterodimer called Ig-α/Ig-β (CD79), bound together by disulfide bridges. Each member of the dimer spans the plasma membrane and has a cytoplasmic tail bearing an immunoreceptor tyrosine-based activation motif (ITAM)
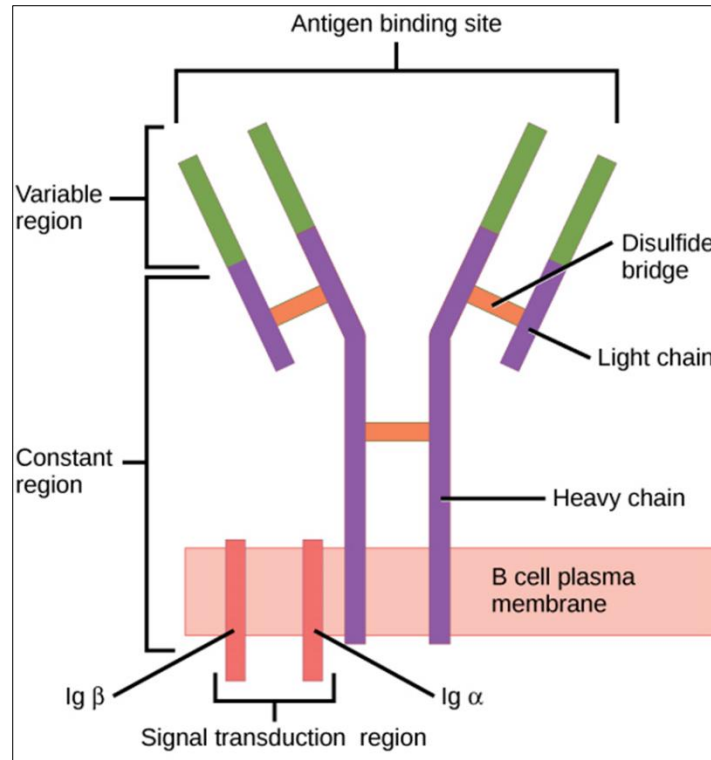
Figure 4 B CELL RECEPTOR: The general structure of the B cell receptor includes a membrane-bound immunoglobulin molecule and a signal transduction region. Disulfide bridges connect the immunoglobulin isotype and the signal transduction region

4. Antibody-mediated vs. Cell-mediated immunity

Five major types of antibodies are produced by B cells: IgA, IgD, IgE, IgG and IgM. IgG antibodies can be further subdivided into structurally distinct subclasses with differing abilities to fix complement, act as opsonins, etc. The major classes of antibodies have substantially different biological functions, recognize, and neutralize specific pathogens.

NB: Fab = Fraction antibody binding.

       Fc = Complement fixing fraction
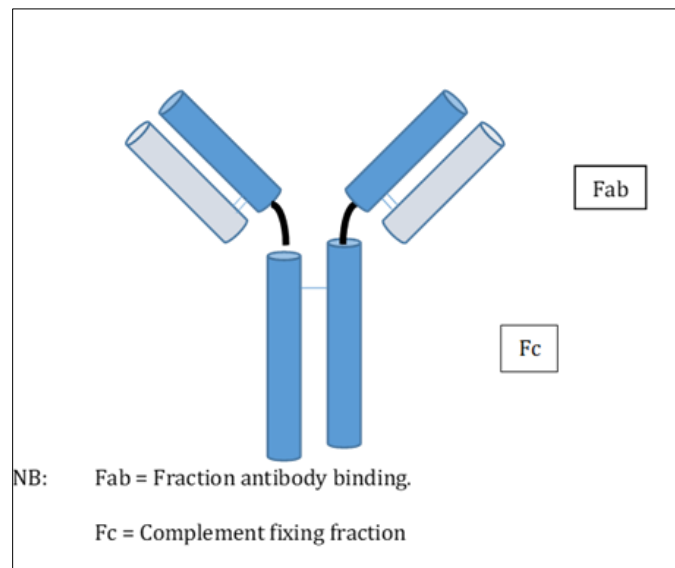
Figure 5 Prototype of Immunoglobulin G structure

5. Immunoglobulin superfamily

The **immunoglobulin superfamily** (**IgSF**) is a large protein superfamily of cell surface and soluble proteins that are involved in the recognition, binding, or adhesion processes of cells.

Molecules are categorized as members of this superfamily based on shared structural features with immunoglobulins (also known as antibodies); they all possess a domain known as an immunoglobulin domain or fold. Members of the IgSF include cell surface antigen receptors, co-receptors and co-stimulatory molecules of the immune system, molecules involved in antigen presentation to lymphocytes, cell adhesion molecules, certain cytokine receptors and intracellular muscle proteins. They are commonly associated with roles in the immune system.

Proteins of the IgSF possess a structural domain known as an immunoglobulin (Ig) domain. Ig domains are named after the immunoglobulin molecules. They contain about 70-110 amino acids and are categorized according to their size and function.[2] Ig-domains possess a characteristic Ig-fold, which has a sandwich-like structure formed by two sheets of antiparallel beta strands. Interactions between hydrophobic amino acids on the inner side of the sandwich and highly conserved disulfide bonds formed between cysteine residues in the B and F strands, stabilize the Ig-fold.

Members of the Immunoglobulin superfamily include vascular and neural cell adhesions molecules (VCAM and NCAM), intercellular adhesion molecules (ICAM) and the Nectins and nectin-like (Necl) proteins.

Members of the Ig superfamily resemble each other in their three-dimensional structure as well as their amino acid sequence.

5.1. Cell adhesion molecules

Cell adhesion molecules (**CAMs**) are a subset of cell surface proteins that are involved in the binding of cells with other cells or with the extracellular matrix (ECM), in a process called cell adhesion. In essence, CAMs help cells stick to each other and to their surroundings. CAMs are crucial components in maintaining tissue structure and function. In fully developed animals, these molecules play an integral role in generating force and movement and consequently ensuring that organs are able to execute their functions normally.[3] In addition to serving as "molecular glue", CAMs play important roles in the cellular mechanisms of growth, contact inhibition, and apoptosis.

CAMs are typically single-pass transmembrane receptors and are composed of three conserved domains: an intracellular domain that interacts with the cytoskeleton, a transmembrane domain, and an extracellular domain.

There are four major superfamilies or groups of CAMs: the immunoglobulin super family of cell adhesion molecules (IgCAMs), Cadherins, Integrins, and the Superfamily of C-type of lectin-like domains proteins (**CTLDs**). Proteoglycans are also considered to be a class of CAMs.

5.2. -Transmembrane proteins

A **transmembrane protein** (**TP**) is a type of integral membrane protein that spans the entirety of the cell membrane. Many transmembrane proteins function as gateways to permit the transport of specific substances across the membrane. They frequently undergo significant conformational changes to move a substance through the membrane. They are usually highly hydrophobic and aggregate and precipitate in water.
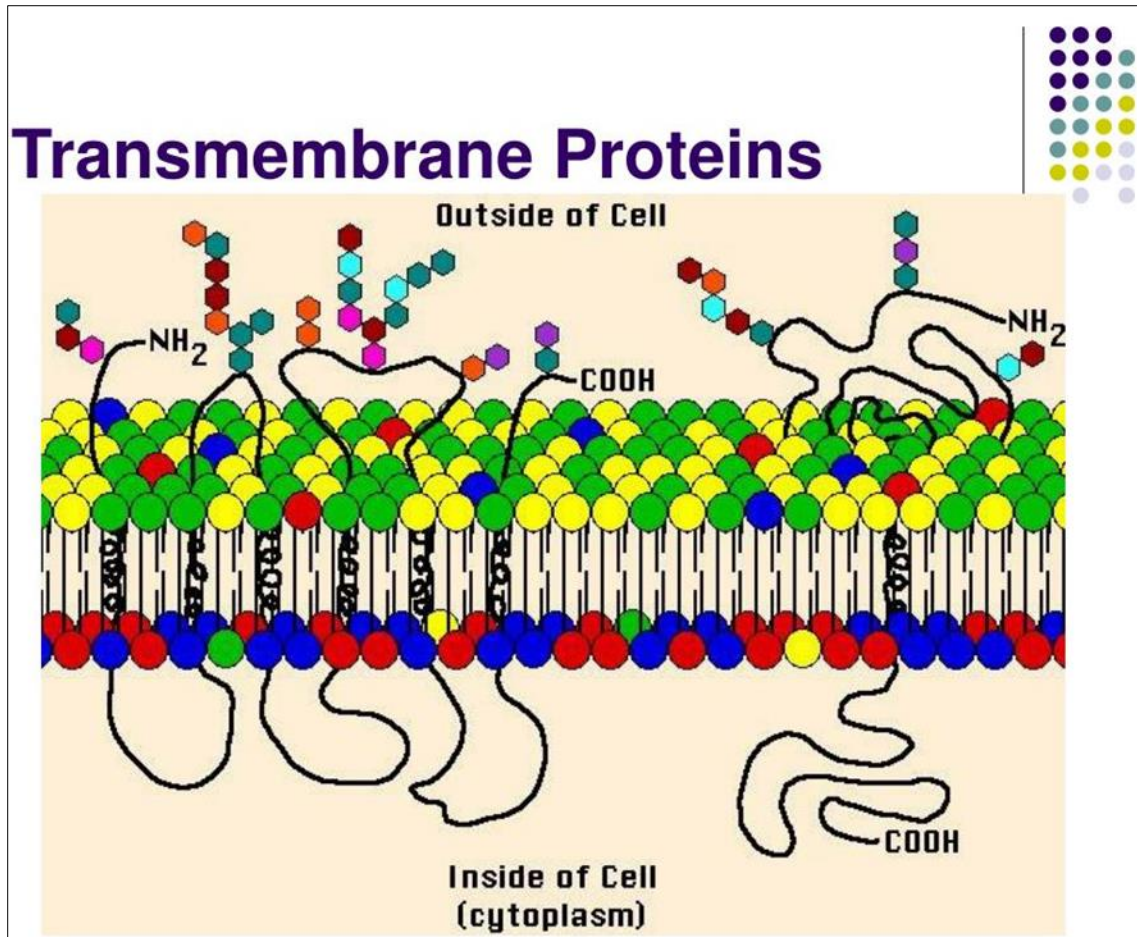
Figure 5 Transmembrane protein

5.3. Transcription factors

In molecular biology, a **transcription factor** (**TF**) (or **sequence-specific DNA-binding factor**) is a protein that controls the rate of transcription of genetic information from DNA to messenger RNA, by binding to a specific DNA sequence.

The function of TFs is to regulate—turn on and off—genes in order to make sure that they are expressed in the desired cells at the right time and in the right amount throughout the life of the cell and the organism. Groups of TFs function in a coordinated fashion to direct cell division, cell growth, and cell death throughout life; cell migration and organization (body plan) during embryonic development; and intermittently in response to signals from outside the cell, such as a hormone. There are 1500-1600 TFs in the human genome.

Transcription factors are essential for the regulation of gene expression and are, as a consequence, found in all living organisms. The number of transcription factors found within an organism increases with genome size, and larger genomes tend to have more transcription factors per gene.

6. Conclusion

An immune response is generally divided into innate and adaptive immunity.

Innate immunity occurs immediately, when circulating innate cells recognize a problem. Adaptive immunity occurs later, as it relies on the coordination and expansion of specific adaptive immune cells. Immune memory follows the adaptive response, when mature adaptive cells, highly specific to the original pathogen, are retained for later use.

Immune dysregulation can manifest as autoimmunity, auto-inflammation, allergy, or lympho-proliferation.

Vaccination, or immunization, is a way to train your immune system against a specific pathogen. Vaccination achieves immune memory without an actual infection, so the body is prepared when the virus or bacterium enters. Immune deficiencies may be temporary or permanent. Temporary immune deficiency can be caused by a variety of sources that weaken the immune system. Common infections, including influenza and mononucleosis, can suppress the immune system. When immune cells are the target of infection, severe immune suppression can occur. For example, HIV specifically infects T cells, and their elimination allows for secondary infections by other pathogens.

Allergies are a form of hypersensitivity reaction, typically in response to harmless environmental allergens like pollen or food. Hypersensitivity reactions are divided into four classes. Class I, II, and III are caused by antibodies, IgE or IgG, which are produced by B cells in response to an allergen.

Overproduction of these antibodies activates immune cells like basophils and mast cells, which respond by releasing inflammatory chemicals like histamine. Class IV reactions are caused by T cells, which may either directly cause damage themselves or activate macrophages and eosinophils that damage host cells.

Auto-immune diseases occur when self-tolerance is broken. Self-tolerance breaks when adaptive immune cells that recognize host cells persist unchecked. B cells may produce antibodies targeting host cells, and active T cells may recognize self-antigen. - Autoimmunity is either organ-specific or systemic, meaning it affects the whole body.

Auto inflammatory diseases (AIDs) are a group of rare disorders caused by dysfunction of the innate immune system. They are characterized by periodic or chronic systemic inflammation usually without the involvement of adaptive immunity.

Compliance with ethical standards

Disclosure of conflict of interest

No conflict of interest is declared by the Authors.

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