Humoral Immunity and Cell-Mediated Immunity

Humoral immunity, or antibody-mediated beta cellular system, is a type of immunity which is mediated by macromolecules found in fluids such as the secreted antibodies, complement proteins, and bound antimicrobial peptides. In contrast to cell-mediated immunity, the term 'humoral' describes the non-cellular compositions of the blood, such as plasma and lymphatics. However, the cellular components of the blood, such as lymphocytes and antigen-presenting cells, are important for mediating antigen-specific antibody reaction.

Primary Immune Response

This refers to the first encounter of the body-host with an antigen, in which naïve B cells activate and proliferate to induce an effective immune response against the antigen. The primary immune response is relatively slow in protecting against invasive pathogens, mostly because it induces the release of polyspecific, natural antibodies. These antibodies have a low affinity, but the body uses them in its initial defense against the pathogen.
Secondary Immune Response

The secondary immune response occurs upon the activation of certain types of cells, called ‘memory B-cells’. In contrast to the primary response, the secondary immune response is relatively faster and more effective in suppressing infection progress because of the increased antibody binding affinities. This is how vaccines work. As vaccines induce the initial primary immune response, the body responds more quickly and effectively upon re-exposure to the same antigen (secondary response) from which the vaccine was made.

The study of the molecular and cellular parts comprising this humoral system is the central science of humoral immunity, along with their operations and interactions. Humoral immunity depends on B cells, while cell immunity depends on T cells.

Humoral immunity refers to protein production; therefore, the processes that accompany it include Th2 activation and protein production, germinal center formation and isotope changing, affinity maturation, and memory cell generation. Jointly, it refers to the effective functions of antibodies, such as infectious agent and poisonous substance neutralization, classical complement activation, antibody promotion of phagocytosis, and infectious agent elimination.

T-Dependent, T-independent Antigens and Class Switching

A B cell response area is classified as T-dependent (T-D) or T-independent (T-I) depending on whether the antigen depends on T cells to activate the B cells. The T-D antigen area unit protects that area unit, processes and gives on MHC category II molecules for recognition by its cognate T cells helper.

T-I antigen area units are divided into sort I and sort II. The previous area unit of the mutagenic stimuli, like LPS, Chg., or poly-IC, elicits polyclonal B lymphocyte activation via Toll-like receptors. The latter area unit polysaccharides interact with the B lymphocyte receptor and, therefore, induce antigen-specific B lymphocyte responses.

T-I sort II antigens cause strong and long primary protein responses. Sugar vaccines, like Menomonee, confer, semi-permanent body substance, protect adults.
However, T-I sort II antigens do not cause a recall response, i.e. a lift in protein production upon secondary protection. Help from unsusceptible with T-I sort II antigens will answer the secondary challenge once adoptively transferred into naïve, irradiated recipients, and the injection of immune bodily fluid into naïve recipients before the adoptive transfer suppresses this response.

T-D antigens cause memory B cells that develop in T-D germinal centers and might be known by physical mutations in their immune serum globulin loci, or by surface expression of secondary immune serum globulin isotopes.

T-I sort II antigens stimulate extrafollicular foci of lymph cell production and fugacious presumptively unsuccessful T-I germinal centers. It is not known whether or not T-I sort II immune responses can generate memory B cells. Extremely low levels of physical hypermutation and low frequency of shifts to secondary immune serum globulin isotopes through T-I sort II responses hinders the identification of T-I memory B cells. It is widely accepted that memory B cell area units are derived solely from T-D responses.

Structure of Antibodies and Immunoglobulin Domains

Antibody molecules are square measured, roughly Y-shaped molecules, consisting of three equal-sized parts loosely connected by a versatile tether, as shown in figure 1.

This structure permits protein molecules to complete their twin tasks, i.e. binding to a good kind of antigen and to a restricted variety of effector molecules and cells.

Every task is disbursed by dissociable elements of the molecule. Both arms of the Y finish in regions that fluctuate between totally different protein molecules, called the V regions. These square measures are concerned with matter binding, whereas the stem
of the Y, or the **C region**, is less variable and only interacts with effector cells and molecules.

The serious and light-weight chains of square measures are composed of a series of distinct supermolecule domains. These supermolecule domains have the same pleated structure.

Among these basic three-dimensional structures, there are **distinct measure variations between the V and C domains**. The structural similarities and variations are shown in the lightweight chain diagram.

Every domain is made from **2 β sheets**. The sheets are elements of a protein structure consisting of polypeptide chain strands (**β strands**) packed together. The sheets are joined together by a type of disulfide bridge in a roughly barrel-shaped structure, referred to as a **β barrel**.

Every domain can be a barrel-shaped structure with strands. Each one forms the **essential similarity out of V and C domains**, and the **vital distinction** between them. Wherever the cylindrical domains open, the peptide chain folds and forms every part of the β sheets. They form **versatile loops** because they change direction. The distinction between the V and C domains is that the V domain is larger and has an additional loop.

**Antibody Polymers and Antibody Binding to Antigens**

The antigen-antibody reaction is widely used in laboratory diagnostics, including immunohematology. It is a reversible chemical reaction:

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\text{Antigen} + \text{antibody} \rightarrow \text{antigen-antibody complex}
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The forces which connect the antigen-antibody are not sturdy valence bonds; however,
weaker bonds are fittingly named ‘weak interactions’. The first vital issue is governing the antibodies’ behavior. It depends on the genetic system and how mature the response is (affinity maturation). The low-affinity antibody area unit is notably sensitive to substances such as protein concentrations and a substance called zygosity.

The high-affinity antibody area unit is sensitive to the serum/cell quantitative relation. The second most vital issue is the activity at low ionic strength: the Rh antibody area unit notably increases, and most alternative antibody area unit decreases; however, Kell antibodies are not increased.

An appropriate technique for all clinically important red cell antibodies will meet a series of needs.

- Incubation at low ionic strength (I=0.03–0.04) or within the presence of PEG
- Period of incubation no longer than 20 minutes
- The best serum/cell quantitative relation
- The minimum quantity of red cells
- Homozygous red cells

Volume limitation forces a compromise between low ionic strength and, therefore, the quantity of serum/plasma. To succeed in ionic strength, 0.4 humor should be diluted to a minimum of 1:4 with a coffee ionic strength answer. Moreover, the minimum quantity of red cells depends on the detection system; tube tests need a lot of red cells than gel or solid part tests.

The Function of Antibodies

Activated B cells differentiate into either antibody-producing cells, referred to as plasma cells, or memory cells that survive in the body years later, for the system to recollect an Associate in Nursing substance and respond more rapidly to future exposures. At the antenatal and infant stages of life, antibodies are provided by passive protection from the mother. Early endogenous protein production varies from various sorts of antibodies, typically showing the primary years of life.

Since antibodies exist freely within the blood, they are said to be a part of the body’s substance system. The current antibody area unit is created by organism B cells that specifically respond to only one substance.

Antibodies contribute to immunity in three ways: they prevent pathogens from entering or damaging cells by binding to them, stimulatingly removing of pathogens by macrophages and alternative cells by coating the pathogen, and triggering the destruction of the offending pathogens by stimulating alternative immune responses like the complement pathway.

Classes of Antibodies (Immunoglobulin)
Autoimmune Disease

Autoimmune diseases can affect the human immune system, so the primary goal is to create self-immunity. However, if a person’s body hasn’t developed an adequate immune system, some autoimmune diseases can appear, such as rheumatic fever, rheumatoid arthritis, ulcerative colitis, myasthenia gravis, Lyme disease, microbial etiology, Guillain-Barre syndrome, Reiter’s syndrome or reactive arthritis, or insulin-dependent diabetes mellitus (IDDM), etc.

Conclusion of Humoral Immunity and Cell-Mediated Immunity

Antigens are any foreign substance that elicits an immune response when introduced into the tissues of a susceptible animal, and capable of combining with the specific antibodies formed.

They are generally of high molecular weight and consist of proteins or polysaccharides. One of the most familiar antigenic is microbes, which contain and produce many antigens. Antigens consist of many specific sites that connect and bind to antibodies called epitopes. Immunity and immune responses consist of two cellular
systems: humoral or circulating antibody system (B cells) and cell-mediated immunity (T cells).

The immune system itself identifies antigens (unfamiliar proteins or polysaccharides) like its components of microbes, or likewise their partially degraded by-products and other unfamiliar proteins and polysaccharides, which includes nucleic acids. On the other hand, the host can be human or animal antigens and are not made by the individual, so it can result in a graft or transplant rejection, etc.

The human immune system begins since birth, as it develops from the beginning (embryo stage) and starts with hematopoietic stem cells (Greek: ‘Blood making’). Those stem cells are developing and differentiating into bigger cells in the immune system (granulocytes, monocytes, and lymphocytes).

Stem cells differentiate throughout human life and they can also differentiate into cells in the blood which are not involved in the human immune function (erythrocytes and megakaryocytes).

Humoral Immune Responses to antigens begin by exposing the human immune system to an antigen. While exposing to antigen ‘A’ for the first time, the immune system begins to make low levels of antibody, approximately in a week. During the second exposure to the antigen ‘A’, the human immune system produces a much faster response, several orders of magnitude higher levels of antibody, and the ability of these antibodies to bind, the antigen increases dramatically during the secondary response to the exposure.

Whilst injecting a new antigen “B” with “A” can only elicit a primary response, and it shows that memory or prior exposure is required for a fast response.

The role of a selection of new microbial strains in susceptibility to infection and illness

Antigenic changes in microbes can overcome immunity, which increases the risks of re-
infection or illness. Antigenically different strains of microbes appear and are selected for over time and space. The constant selections of new strains are selected by antigenic shift and drift. It is partly driven by herd immunity and genetic recombination, re-assortment, bacterial conjugation, bacteriophage infection, and point mutations.

The difference between antigenic shift and antigenic drift

The antigenic shift is a bigger change in virus genetic composition by gene substitution or replacement (e.g., re-assortment).

The antigenic drift is small changes in the virus genetic composition, often by a mutation involving specific codons in existing genes like point mutations.

A single point mutation can greatly alter microbial virulence.

References


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