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Adaptive Immunity is a Very Important for Maintaining the Health

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Abstract

The adaptive immune system, known as specific resistance, is a type of immunity that an organism develops throughout its life in response to contact with different foreign agents, including pathogens. This system possesses the capability to adjust to unfamiliar infections, which enables it to establish immunity against each distinct pathogen. Specific resistance is generally categorized into two distinct types, depending on the method by which immunity is obtained. Within the adaptive immunity framework, there are two forms of immunity: naturally acquired immunity and artificially acquired immunity. Naturally acquired immunity develops through exposure to an infectious agent, resulting from unintentional contact, while artificially acquired immunity comes solely from deliberate actions, such as vaccinations.

Keywords: Immunity, Immune System, Adaptive Immunity, Antigens, Health

1. Introduction

The main function of the immune system is to safeguard the organism from pathogens that constantly evolve [1]. Healthy immune operations are marked by the precise identification and removal of microbial pathogens, cancers, and toxins without causing harm to the host. Disruptions in this intricate system can lead to a weakened defense against infections or inappropriate damage to host tissues, evident in allergic reactions or autoimmune conditions.

2. Components

The immune system can be classified into innate and adaptive parts [1]. The innate immune system, which is encoded in the germline, represents a more ancient evolutionary aspect. The activation of pattern-recognition receptors results in a swift release of proinflammatory cytokines and the activation of immune cells. Adaptive immunity is defined by the formation of antigen-specific primary and memory responses, which arise through random rearrangements of somatic genes. Malfunctions in either the innate or adaptive responses can have serious consequences for the host, such as making them more prone to infections (immune deficiency), causing autoimmunity (loss of self-tolerance), or triggering hypersensitivity reactions.

3. Tolerance

The adaptive immune system has developed to enhance the survival chances of long-lived and not very prolific vertebrates in the ongoing fight against rapidly replicating and ever-evolving microbial pathogens [2]. The quick generation times of pathogenic microorganisms enable them to mutate and change far more swiftly than any germ-line-encoded defense mechanisms in higher organisms. As a result, it can be expected that microbes will evade the innate immune system, leading to the evolution of the adaptive immune system that does not rely on a 'mutational race.' The adaptive immune system generates a large array of receptor specificities at random, which helps it account for future pathogen changes. Although this approach addresses the issue of pathogen evasion, it introduces another problem: some of the randomly generated specificities are likely to react to the host's own structures and provoke autoimmune damage. Consequently, an additional somatic mechanism is necessary to eliminate self-reactivity from the receptor repertoire, a process known as the acquisition of self-tolerance.

4. Barriers

The interface between the host and the environment consists of protective barriers, including the skin and mucosal layers, which feature enzymes and mucus that prevent microbial adherence

or have direct antimicrobial properties [1]. Surfaces with cilia, particularly in the respiratory system, function to expel foreign materials from the host. Additional physiological defenses that create an unfriendly environment for certain pathogens involve increased body temperature and the acidic conditions present in the stomach. Pathogens have adapted various strategies to bypass these defenses, necessitating further protective measures for the host to survive.

5. Development

The development of a human fetus represents a distinct phase during which a functional immune system arises amidst potential graft versus host reactions [1]. Between the fourth and fifth weeks of gestation, the fetal liver serves as the primary location for lymphoid cell development, followed by lymphocyte generation in the bone marrow. The thymus, a lymphoid organ formed from the third and fourth pharyngeal pouches of the embryo, descends into the mediastinum by the seventh week of gestation. This organ is critical for T-cell maturation and acts as a checkpoint to ensure proper antigen recognition while preventing self-reactivity. During the sixth to eighth weeks of gestation, numerous T-lymphocyte precursors traverse the thymic layers, with the potential to transform into circulating immunocompetent T cells. However, only about 5% of these developing T cells endure the rigorous selection process, while the remaining cells undergo programmed cell death or apoptosis.

By 10.5 weeks of gestation, the human fetus begins to produce IgM antibodies, followed by IgG at 12 weeks, and IgA antibodies by week 30. Because of the absence of antigen exposure in utero, the immunocompetent newborn has limited levels of circulating IgA and IgM. Nearly all IgG present originates from the mother, transferred through active and selective transport across the placenta. The effectiveness of fetal immunoglobulins is restricted, showing limited capacity to switch to IgG and IgA and a failure to respond to specific capsular polysaccharides. The serum concentrations of immunoglobulins and their functional capabilities rise as the individual ages.

There is minimal passage of complement components C1q, C2, C4, C3, and C5 through the placenta, resulting in low overall hemolytic complement levels in newborns. These shortcomings may account for the relative deficiency in opsonization observed in newborns, since the complement system reaches adult levels of functionality only after 3 to 6 months of life. Phagocytic cells appear in the human fetus around 8 weeks of gestation as myelocytes, while histiocytes emerge during the early stages of yolk sac hematopoiesis. Monocytes make their first appearance in the spleen and lymph nodes between 16 and 20 weeks of gestation, with macrophage functionality gradually improving as the fetus matures.

6. Innate Immunity

The body's innate immune system serves as the primary barrier against harmful pathogens and toxins entering the host [1]. Weaknesses in the innate immune response can lead to a higher risk of infections, and the triggering of innate inflammatory mechanisms by allergens can exacerbate allergic reactions. Immune cells such as natural killer cells, NKT cells, macrophages, dendritic cells, neutrophils, eosinophils, and mast cells are vital in initiating inflammatory responses, in conjunction with structural cells like epithelial cells.

7. Adaptive Immunity

A significant characteristic of the adaptive immune response is its capability to develop immunologic memory [1]. B and T cells serve as the main types of cells involved in adaptive immunity. These cells originate from the bone marrow and constitute around 40% of the overall white blood cells circulating (with B cells being 10% to 15% and T cells making up 70% to 80%). Together with various immune cell types, B and T cells are marked by surface indicators classified as cluster of differentiation (CD). T lymphocytes display CD3 and further classify into CD4-positive and CD8-positive cells. The surface markers for B cells include CD19, CD20, and B220. Unlike other lymphocyte variants, such as NK cells, progenitors of B and T cells undergo several recombination events leading to specificity and diversity in their receptors, which are essential for their functions as effector cells within the adaptive immune framework.

8. Antigens

The term immune repertoire encompasses both humoral and cellular immunity [3]. It refers to the extensive diversity in the immune system's ability to recognize and differentiate among millions of distinct epitopes found on various molecules and organisms. The immune repertoire evolves progressively over time. In fact, it is estimated that more than 1600 genes play a role in forming the human immune system. Even prior to birth, our immune systems can identify countless antigenic epitopes due to several physiological processes that contribute to the development of tolerance. Through positive and negative selection, our immune system learns to eliminate harmful antigens while tolerating safe ones, including self-antigens and those from maternal and environmental sources. The capacity to boost immune responses towards specific antigens via these selections and the mechanisms behind central and peripheral tolerance ultimately define an individual's immune repertoire.

As our immune systems evolve and we gradually encounter novel antigens, our cells differentiate into specialized variants that can remember past exposures (as in memory cells) and those that exhibit effector capabilities. A wide array of antigens can be recognized by our immune system through biochemical mechanisms such as V(D)J recombination and somatic hypermutation. V(D)J recombination aids in constructing the variable region of immunoglobulin and T cell receptor genes. This mechanism is precisely governed by the cleavage of DNA at short conserved sequences managed by Recombination-activating gene (RAG)

proteins. Somatic hypermutation leads to additional modifications in immunoglobulin genes by creating mutations in variable gene segments, which drives affinity maturation. This fulfills the need for amplification, resulting in a more potent response within adaptive immunity.

Isotype class switching, however, modifies the constant region of the heavy chain in immunoglobulins to create antibodies besides IgM. This alteration determines the functional response of the antibody molecule. Both somatic hypermutation and heavy chain isotype (class) switching are regulated by activation-induced cytidine deaminase (AID), which induces DNA deamination of the targeted transcribed DNA. All these mechanisms collectively contribute to the defining traits of adaptive immunity—resulting in a more potent response characterized by significant diversity and specificity.

9. Inflammation

Inflammation is typically a biological reaction that adapts to infections from pathogens and injuries to tissues, aiming to activate the immune system and facilitate tissue repair mechanisms [4]. The initial recognition of pathogens and tissue harm is often accomplished by pattern recognition receptors, particularly toll-like receptors, which are primarily present in cells involved in the innate immune response. When these receptors are activated, they send out signals that lead to the activation of transcription factors such as NF-kB and AP-1, which in turn regulate numerous genes that enhance the early inflammatory reaction, produce antimicrobial activities, and kickstart adaptive immunity. Furthermore, sterile inflammation induced by endogenous substances, including elements from dead cells and protein clumps, can also provoke inflammation via pattern recognition receptors like TLRs. To sustain normal tissue homeostasis and respond effectively to infections and injuries, there is a necessary coordination between the innate and adaptive immune systems; any disruption in these processes may lead to chronic inflammatory conditions across various organ systems.

Various negative feedback systems work to diminish inflammatory responses. These mechanisms encompass the activation of proteins that block signaling pathways (for instance, Suppressors of Cytokine Signaling or SOCS proteins), the generation of soluble or cell surface mediators with anti-inflammatory properties (such as IL-10, TGFb, resolvins, and ligands for TAM receptor tyrosine kinases), and the transcriptional inhibition of genes related to inflammation by certain members of the nuclear receptor transcription factor family (including glucocorticoid receptors, liver X receptors, and peroxisome proliferator-activated receptors). The growing understanding that inflammation might play a role in the development of various neurodegenerative disorders has sparked increased interest in how nuclear receptors might regulate inflammatory responses in the nervous system. This review discusses how nuclear receptors act to manage inflammation, the inflammatory components associated with selected neurodegenerative diseases, and the effects of nuclear receptor activity on these mechanisms.

10. Infections

Infectious diseases are triggered by various pathogens, including viruses, bacteria, fungi, and parasites, and they have historically been a significant source of human suffering, contributing to both illness and death [5]. The spread of these infectious diseases has been influenced by different periods in human history. For instance, the rise of parasitic and zoonotic infections became more common following the domestication of animals, while airborne infections caused by viruses and bacteria became widespread along with the establishment of large settlements and urban life. Across the ages, humanity has been impacted by major pandemics such as the plague, smallpox, cholera, influenza, and COVID-19, in addition to more persistent infections like tuberculosis and syphilis. Despite modern advances in diagnosis, treatment, and management strategies, infectious diseases continue to result in significant morbidity. The World Health Organization has reported an estimated 300 to 500 million cases of malaria, 333 million instances of sexually transmitted diseases (STDs) like syphilis, gonorrhea, chlamydia, and trichomonas, 33 million cases of HIV/AIDS, 14 million individuals afflicted with tuberculosis, and between 3 to 5 million cholera cases globally.

The body's defense mechanisms against infectious organisms can be divided into two primary categories: innate and adaptive immune responses. These two types of reactions are interconnected. For example, the triggering of innate receptors, such as toll-like receptors, results in the release of cytokines, which subsequently activates the adaptive immune system. The initial reaction of the innate immune system to a pathogen commences when pathogen-associated molecular patterns from bacterial agents stimulate pattern recognition receptors found in the innate immune system. The innate immune response instigates antigen-specific actions that are formed by the adaptive immune system. This process is followed by the release of both pro-inflammatory and anti-inflammatory substances that assist in managing the infection. Consequently, pathogens exploit host defense mechanisms, such as evading phagocytosis, to ensure their survival and multiplication. Adaptive immunity encompasses antigen-specific reactions that are finely tuned to particular pathogens and are carefully regulated through the interactions of innate immune cells.

It has developed to offer a flexible and well-adapted collection of receptors that can distinguish between self and foreign antigens. Throughout their development, naïve B and T lymphocytes undergo genetic rearrangement of antigen receptors to establish a varied collection of antigen-specific receptors capable of recognizing all possible antigens. Upon the defeat of a pathogen, long-lasting memory lymphocytes specific to that pathogen are created. These memory cells respond quickly and effectively to future encounters with the same invading microorganism, producing new effector cells to manage the infection. Whether the infectious

microorganism is previously encountered or a new threat, the immune system's battle is usually the first line of defense it meets. In circumstances where vaccines and efficient treatments are often lacking, the immune system's efforts to eliminate infectious agents or cells infected by them frequently represent the sole method available for eradication. Understanding the immune system, along with the strategies infectious microorganisms employ to undermine it, holds significant importance for researchers and healthcare providers.

Timely and precise identification of infections is essential for effective and focused treatment. Nevertheless, standard microbiological identification often proves inefficient and frequently experiences delays that render it impractical in a clinical setting. The immune system possesses the ability to swiftly recognize a wide array of microbes with high sensitivity and specificity, a capability honed over millions of years of evolution. As a result, initial immune responses are likely to offer significantly better information regarding the actual nature and severity of microbial infections compared to traditional testing methods.

11. Viruses

Innate immunity primarily serves to slow down viral infections rather than eliminate them, giving the adaptive immune response time to activate [6]. The adaptive immune system can be divided into two key categories: antibody-mediated immunity and T-cell-mediated immunity, each targeting different aspects of infection. Antibodies work by binding to free viral particles, effectively preventing those viruses from infecting host cells. In contrast, T cells mainly function by identifying and eliminating cells that are infected by viruses and by coordinating an inflammatory response that includes various antiviral mechanisms. Since viruses replicate within host cells and many can transfer directly between cells without needing to exit into the extracellular space, the resolution of infection primarily hinges on the activity of T cells over that of antibodies. Nevertheless, broadly neutralizing antiviral antibodies hold promise as treatments for a wide range of human infections, including those caused by HIV, influenza, and Ebola virus. Recent breakthroughs in research have enabled the isolation and identification of human monoclonal antibodies against various pathogens, providing hope for new treatment options as well as valuable insights for vaccine development. Antiviral antibodies also play a crucial role as a protective defense against reinfection. The presence of antibodies at entry points into the body, primarily mucosal surfaces, is particularly significant in the context of infections like influenza, HSV, and HIV. However, creating vaccines that elicit strong antibody responses, particularly those that generate broadly neutralizing antibodies, continues to be a major challenge.

In certain individuals, the body produces highly effective or cross-reactive antibodies in response to viral infections. These broadly neutralizing antibodies, often referred to as super-antibodies, present numerous therapeutic advantages, especially if modified to have a prolonged lifespan in the body. The current techniques that focus on single-cell methods for identifying and isolating B cells are crucial for utilizing broadly cross-reactive human monoclonal antibodies as treatments, including those targeting emerging and pandemic viruses like SARS-CoV-2, which is responsible for the recent COVID-19 pandemic.

The initiation of adaptive immunity is heavily reliant on the early innate mechanisms that activate antigen-presenting cells, mainly specific subsets of dendritic cells. Antigen-presenting cells and lymphocytes are attracted to lymphoid tissues through signals from chemokines and cytokines and remain there for several days to promote effective cell interactions. The structure of secondary lymphoid tissues facilitates coordinated interactions among the cells involved in the adaptive immune response via supportive stromal cells and local chemokine gradients. The activation events typically take place in lymph nodes that drain the site of infection or in the spleen when the virus enters the bloodstream. Viral antigens usually move to lymph nodes within dendritic cells. Certain viruses, including HSV and measles virus, can interfere with the functioning of antigen-presenting cells, hindering the maturation of dendritic cells.

B-cell activation takes place when antigens are encountered within B-cell follicles, and possibly in T-cell areas, located in the spleen or lymph nodes. Upon activation, some B cells transition into short-lived plasma cells, while others migrate to the periphery of B-cell follicles where they engage with antigen-specific CD4 helper T cells through the display of antigenic peptides on MHC class II molecules of B cells. These CD4 T follicular helper (Tfh) cells, which depend on Bcl6, are essential for supporting and modulating responses from B cells. The activation of B cells triggers germinal center (GC) reactions in collaboration with CD4 Tfh cells, facilitating somatic hypermutation and affinity maturation necessary for the selection of high-affinity antibody-secreting plasma cells that are long-lived, along with memory B cells. At the molecular level, the increase of transcription factors Blimp-1, XBP-1, and IRF-4 regulates the development of plasma cells, while the expression of Pax-5 indicates B cells that are on the path to undergoing germinal center reactions and differentiating into memory B cells.

12. Immune Response

Antigen contact triggers a series of actions involving the immune system, notably the engagement of lymphocytes, which are white blood cells that combat infections [7]. Upon encountering an antigen, B cells and T cells—two kinds of lymphocytes—undergo differentiation into effector cells (such as antibody-producing B cells and both cytotoxic and helper T cells) and memory cells. Within a standard immune reaction to an antigen encounter, the interval between the initial exposure (primary) and the formation of the primary response is divided into three phases: a lag phase, a logarithmic phase, and a plateau phase. The lag phase marks the

early stimulation of B and T cells when they first meet their specific antigen, leading to their transformation into effector and memory cells. Generally, this lag time lasts between 4 and 7 days from the primary exposure to the start of the logarithmic phase, though this duration can change based on how the antigen was encountered and its specific characteristics. In B cells, the logarithmic phase signifies a rise in serum antibody levels that is typically logarithmic in nature. The plateau phase is when antibody levels stabilize at their peak for a period, after which there is a reduction in serum antibody levels. For many antigens, this latency period (lag phase) from initial exposure to the onset of the primary antibody response is often between 7 to 10 days. The presence of memory B and T cells formed during the primary immune response generally decreases the time taken between subsequent exposures to the same antigen and the immune response. The lag phase in these cases is usually between 1 to 3 days, with the logarithmic phase of the secondary antibody response occurring in the following 3 to 5 days. As noted for primary immune responses, these durations can also be influenced by factors like the exposure way, timing of the next exposure, the specific antigen, and the amount of antigen.

Cells typically associated with the innate immune system, such as macrophages and dendritic cells, also facilitate the activation of B and T cells and the start of the adaptive immune response. These innate immune cells are essential at each of the phases mentioned earlier and are often the first immune cells to interact with the antigen. When they encounter an antigen, macrophages and dendritic cells uptake the antigen, which subsequently activates them to act as antigen-presenting cells. These antigen-presenting cells, as their name implies, show the antigen to T cells, and they also produce inflammatory substances (like cytokines and chemokines) that assist in recruiting, activating, and proliferating B and T cells. Activated B and T cells then release further inflammatory substances that help recruit and activate additional immune cells, which intensifies the immune response with the release of inflammatory signals. Although not covered in this report, regulatory cells and soluble immunoregulatory substances help to suppress the immune response.

13. Conclusion

The adaptive immune response is triggered when a particular pathogen successfully bypasses the innate immune defense, resulting in a decrease in antigen levels within the body. This system plays a crucial role in preserving overall health and carries out various essential tasks. Its main task is to identify specific foreign antigens, or harmful agents, through the antigen presentation process. Furthermore, the adaptive immune system is tasked with developing the body's response to the invading pathogens. This bodily response aims to eliminate the particular pathogen or any infected cells as swiftly and effectively as possible.

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