Immunity is the body's ability to defend itself against various diseases, infections and contagions.

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Abstract

Immunity is a medical term used to describe the body's ability to resist and defend itself against infections, diseases or other biological or chemical hazards. Immunity functions like a mechanism whose role is to establish barriers against pathogens, cancer cells, harmful molecules, and toxins. The body's overall defense system is called the immune system. The basic tasks of the immune system are the destruction and expulsion of pathogens such as bacteria, viruses, parasites or fungi, the recognition and destruction of harmful substances from the environment, as well as the fight against their own cells that have changed due to disease. For the defense to be effective, it is important for the system to recognize the difference between its own and foreign cells. In healthy people, the system will not attack what is its own. But when it comes to autoimmune diseases, then the immune system turns against the tissues in your own body. Tissue-attacking antibodies, called autoantibodies, are also produced. On the other hand, the immune system can react excessively violently to one or more otherwise harmless substances. Then we talk about allergens, or allergies.

Keywords: Immune System, Innate Immunity, Adaptive Immunity, Autoimmunity.

Introduction

The immune system evolved so on defend our bodies against infectious microorganisms like viruses, bacteria, fungi and parasites [1]. Throughout history it's been observed that people who survive an infectious disease acquire protection against that disease, which is otherwise referred to as immunity. As far back as the fifteenth century attempts are made to induce immunity against infectious diseases, a process said as vaccination. The realisation that immunity will be transferred from one person to a different demonstrated that soluble factors exist within the blood and body fluids that protect against pathogens. It's now known that cellular components of the immune system also are present throughout the complete body which these immune cells engage with any harmful substance or microorganism so as to preserve the integrity of host tissues. The defense against microorganisms is fought on many fronts and there are immune cells and innate components of the immune system within every tissue and organ. There are a mess of cells and soluble factors which will be considered a part of the immune system. as an example, the barrier function of the outer layers of the skin, the mucus produced within the airways, the antibodies secreted into the gut lumen or the circulating lymphocytes that destroy virus-infected cells. The immune system comprises variety of various cell types and a multitude of secreted factors and surface bound molecules.

The system features a multi-layered organization that has immunity to infectious organisms. Each layer of the system also can be considered to possess an increasing complexity. The primary layer is provided by physical barriers like the skin and therefore the mucosal epithelium of the respiratory and gastrointestinal tracts. These barriers aim to stop pathogens gaining access to underlying tissue. The following layer is that the non-specific chemical barrier that consists of antimicrobial compounds and factors of the humoral system (soluble factors found in body fluids). Other chemical immune defense mechanisms include the acidic environment of the stomach and therefore the proteolytic enzymes produced within the intestines. The third layer consists of all the cells of the system. Therefore, if a pathogen breaches the physical barriers and chemical barriers then the system utilizes its immune cells.

The human immune system has evolved over millions of years from both invertebrate and vertebrate organisms to develop sophisticated defense mechanisms to protect the host from microbes and their virulence factors [2]. From invertebrates, humans have inherited the innate immune system, an ancient defense system that uses germ line–encoded proteins to recognize pathogens. Cells of the innate immune system, like macrophages, dendritic cells, and NK lymphocytes, recognize pathogen molecular motifs that are highly conserved among many microbes (PAMPs) and use a various set of receptor molecules (PRRs). Important components of the recognition of microbes by the innate immune system are: (1) recognition

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by germ line– encoded host molecules, (2) recognition of key microbe virulence factors but not recognition of self-molecules, and (3) nonrecognition of benign foreign molecules or microbes. Upon contact with pathogens, macrophages and NK cells may kill pathogens directly or may activate a series of events that both slows the infection and recruits the more recently evolved arm of the human immune system, the adaptive immune system.

Technology

In the distant past, several societies practiced a form of "empirical vaccination," but it's only recently that we are ready to "rationally design" vaccines [3]. Molecular engineering allows us to synthesize subunits of certain vaccines. Armed with novel synthetic adjuvants, and modern delivery methods, we now can potentiate the specified immune reaction in some vaccines. However, we've not yet discovered all the principles of induction of protective immune responses. So as to shield us from microbes, the cells of the system have to make the correct decisions, which can only be achieved by proper "education." Thus, cells receive their first education within the thymus school and learn the way to discriminate "self from 'nonself'." They continue teaching within the periphery and find out how to tolerate self. During evolution, we learned to create use of two protective systems: innate and adaptive immunities. Innate immunity operates under the control of germ-line genes; thus, it's very quick and crude, lacks fine specificity, and has no memory. This method recognizes the "common" structures of microbes by means of pattern recognition molecules like Toll-Like Receptors (TLR), NLRs, RIG-I, etc. On the contrary, adaptive immunity is slow, but specific, and generates memory cells. In order to recognize "uncommon" microbial structures, this method must generate enough diversity so it can cope with rapidly growing, and unpredictably changing microbes. During coevolution, we survived because our adaptive immunity has learned to get from limited sets of germ-line genes, more diversity than microbes can ever generate. In short, self-tolerance, specificity, diversity, and memory are the hallmarks of the immune system. A well-coordinated collaboration of those rapid and slow systems may be a prerequisite for the success of vaccines (and for that matter, our survival). Also, design of a vaccine depends on what quantity we all know about the "invasion strategy" of every infective agent. Only then can we induce appropriate B-cell (antibody) and T-cell responses (T-helper-1 (TH1), TH2, TH17, T-regulatory cells, and T-killer cells). Because the immune cells integrate a multitude of signals at a given time, these T-cell subsets are induced in distinct conditions and might be reinforced or destabilized by other conditions.

Innate Immunity

The innate immune system provides the primary line of immunological defence against infection [1]. The innate immune system is distinct form the adaptive system and is characterized by several key factors. Innate immunity relies on generic protection using molecules and receptors that are somatically expressed and phylogenetically conserved. In other words, the molecules and receptors of the innate immune system are considered to be non-specific, providing a broad range of protection. For this reason the innate immune system is usually stated as providing natural immunity. Elements of natural immunity are found throughout the whole animal kingdom, from simple invertebrates to the more complex vertebrates. This differs from the adaptive system, which is just found in higher vertebrates and utilizes receptors that are highly specific for a specific antigen and permit adaptation and increased specificity to an infectious pathogen. Unlike the adaptive immune system, the innate immune system doesn't afford immunological memory or provide long lasting protection against infection. The principal components of the innate immune system include physical barrier defence (e.g. skin and mucosal epithelia), chemical barriers (e.g. antimicrobial peptides and reactive oxygen species), innate immune cells (e.g. granulocytes, monocytes, DCs and NK cells), components of humoral immunity (e.g. complement factors and innate antibodies) and associated cytokines. Although innate and adaptive immunity are readily separated on the premise of differing functionality, there's considerable interaction between the two systems.

The functions of the innate immune system are therefore diverse. This includes the prevention of pathogens from entering the body through the formation of physical barriers and therefore the release of antimicrobial mediators; the prevention of the spread of infections by the activation of the complement cascade and other humoral factors; the removal of pathogens from the body through mechanisms of phagocytosis and cytotoxicity; and at last the activation of the adaptive immune system through the synthesis of cytokines and also the presentation of antigens to T cells and B cells. These innate immune functions can vary between different tissues.

Adaptive Immunity

As opposed to innate immunity, adaptive immunity is specific [3]. So as to acknowledge "uncommon" microbial structures, this technique must generate enough diversity (at the level of both B- and T-cell receptors, BCR (B-cell receptors), TCR (T-cell receptors)) that it can deal with rapidly growing, and unpredictably changing microbes. During coevolution, we survived because our adaptive immunity learned to generate, from limited sets of germ-line genes, more diversity than microbes can ever generate (this is taken into account as one of the foremost sophisticated biological phenomena). In other words, since the system had to predict the unpredictable (microbial behavior), it had to own the capacity to get ~1014 antibodies and ~1018 TCRs. If you're thinking that that adaptive immunity is exaggerating this issue consider these facts: In humans, the microbiota comprises an oversized population of diverse bacterial species present within the oral cavity, within the upper respiratory and digestive tracts, within the vagina, and on the skin. Approximately 1014 microorganisms are present within the colon alone. This number is one order of magnitude over the combined number of somatic and germinal cells that compose the human body. Thus, this metagenome is 100 times superior to the human

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genome. Exhaustive genomic analysis recently unraveled the wealth of genomic diversity of the human gut microbiota. Most recent findings stress the dynamics of genomes in their acquisition and loss of virulence genes and gene clusters like pathogenicity islands. No doubt, the adaptive power of immunity has got to match that of microbes.

The adaptive immunity evolved two major recognition systems for non-self-antigens which are expressed by T cells and B cells [4]. Both lymphocyte types express individual-specific antigen recognition molecules that aren't determined within the germ line. T cells are responsible for the cellular arm of adaptive immunity and are the main protagonists of immune regulation. T cells expressing α/β TCRs recognize short peptide antigen molecules displayed in non-covalent association of MHC on the surface of antigen-presenting cells and target cells. Antigenic peptides of 9 or 13–17 amino acids length are generated by proteolytic cleavage of proteins from either intracellular or extracellular origin presented in MHC class I or II, respectively. The TCR binding to the MHC–antigen complex is of low affinity and is supported by the co-receptors CD8 or CD4. These co-receptors don't influence the antigen specificity of the TCR complex but enhance its signal over three orders of a magnitude, such the recognition of about 100 antigen: MHC complexes is sufficient to activate the T cell. B cells are the most important players of the humoral arm of the adaptive immunity. In contrast to TCRs, antibodies recognize a broad spectrum of antigens independent of a cellular or MHC context and sometimes with very high affinity. Despite their fundamental different antigen recognition, antibodies and TCR are very similar regarding their molecular structure. They're formed by two different disulfide-linked chains, light (L) and heavy (H) chains for antibodies and α/β chains for TCR. Each chain consists of immunoglobulin domains forming a constant (C) and a variable (V) region. The V region is answerable for the antigen recognition of antibodies as well as of TCRs. The C domains stabilize the antigen recognition domain, assemble the various chains, mediate effector or signal functions, and, if so, the transmembrane association. Immunoglobulin and TCR gene loci are similarly organized. During the first phases of T- or B-cell development both antibody and TCR chains are joined from separate genetic germ line V(D)J elements organized in large and diverse clusters by somatic recombination. This mechanism provides the key a part of the tremendous repertoire of antigen recognition of the adaptive immunity. However, in contrast to TCRs, immunoglobulin genes additionally undergo a process of somatic hypermutation and antigen-driven affinity maturation.

Autoimmunity

Autoimmunity is often broadly defined as a specific immune effector response against self-components that inflicts harm on the host [5]. The self-damaging response is often of inflammatory nature, but other effector mechanisms, e.g., complement activation by autoantibodies bound to self-structures, may also be the main reason for pathology. Autoimmunity should be distinguished from autoreactivity, the latter denoting the presence of self-specific antigen receptors and antibodies within the body that remain without harmful consequences. The manifestations of autoimmunity are called autoimmune diseases (AIDs), which represent a rather large assemblage of various illnesses with distinct symptoms, locations and pathomechanisms. The only commonality of AIDs on which most participants of the sphere agree is that the pathology is that the consequence of a failure in one or another mechanism of self-tolerance. Therefore, autoimmunity is considered the down-side of self-tolerance, as well as the most likely selective pressure that has driven self–non-self-discrimination. During this context, the frequency of AIDs within the population (up to 5%) may reflect the limit of natural selection, i.e., that the latter cannot operate to perfection, only to adequacy defined because the level of harm that not threatens the procreation of the species.

Naturally, many of the diseases now known or assumed to be of autoimmune origin, particularly the more frequent ones, are known since ancient times, and an oversized body of data has been gathered on their symptoms and pathology. But that an response against self-components may be the cause of disease was first recognized at the beginning of the twentieth century. The arrival of immunobiology ('the immunological revolution') was a crucial impetus also for autoimmunity research, and thus a large number of diseases are added to the list of AIDs from the 1950s on.

Disorders

The system provides effective defence against invading microorganisms and potentially harmful substances [1]. However, aberrations within the normal functioning of the system can often result in disease. Immune disorders be three broad categories. The primary category includes those diseases that result from an excessive or overactive immune reaction, like allergies and asthma. The second are due to the generation of immune responses directed against self-antigens and are called autoimmune diseases. The third are characterized by an abnormal system resulting from an inherited disease or mutation and are referred to as immunodeficiency diseases. All three groups of immune-mediated diseases can affect the mucosal system in some way.

The immune mechanisms responsible for mediating allergic responses and autoimmune reactions often share a typical feature, which may be a lack of proper immune regulation (immunodeficiencies are different, as they're caused by genetic defects). Without appropriate regulatory signals, immune cells have the potential to proliferate and exert excessive immunological effector functions. Excessive inflammation generally leads to tissue damage and disease. Moreover, the immune-mediated tissue damage is caused by the inappropriate induction of a selected adaptive response to an antigen that might normally be considered harmless. At the heart of immunoregulation, and therefore immunological disease, may be a mechanism referred to as tolerance. Central tolerance occurs within the thymus and bone marrow, where T cells and B cells that are reactive to self-antigens are deleted from the system. Peripheral tolerance occurs in secondary lymphoid tissues or the periphery and acts to take care of central tolerance and make sure that T cells and B cells don't respond

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to harmless antigens. When this mechanism of tolerance is disregulated, lymphocytes respond inappropriately to harmless antigens. Within the case of allergies, lymphocytes respond to environmental antigens, while autoimmune reactions are related to lymphocyte responses to self-antigens.

Immunodeficiency

Molecular anomalies within the genes of some receptors, cytokines, signaling molecules, and enzymes of lymphocytes and other cells transmitted by heredity result in extremely decreased functions of innate and adaptive immunity, primary immunodeficiency's, and major primary immunodeficiency's [6]. However, some gene's anomalies don't result in fatal consequences and should be termed as "minor" primary immunodeficiency's. Primary immunodeficiency diseases number a minimum of 176 hereditary disorders that are thought to be individually rare. The frequency of occurrence of primary immunodeficiency's is estimated to be 104, but the actual prevalence and incidence of those diseases and syndromes remain unclear. for instance, for Europe, only about 15,000 cases were registered (2.27%) to 2013, whereas the upper estimate was 638,000 cases. Therefore, proper epidemiologic studies are required. For the precise diagnosis of any primary immunodeficiency, the correct molecular biological and genetic tests might must be ordered because certain gene anomalies would need to be revealed. These techniques are the Northern blot, Restriction Fragment Length Polymorphism (RFLP), Polymerase Chain Reaction (PCR), etc.

Minor primary immunodeficiencies are relatively benign and not life-threatening. At birth, a baby has 100% of maternal IgG. The maternal antibodies degrade between the third and fifth months during a period called physiological hypogammaglobulinemia. By the sixth month most babies have already synthesized about 1/3 of their self IgG, then they progressively save more. In some cases, the IgG synthesis is retarded up to 4-6 years to develop transient hypogammaglobulinemia of infancy. Gene mutations are unknown. Such babies and toddlers may suffer from recurrent infections including severe abscesses and must need a correct therapy, which includes antibiotics, immune enhancement medications, and sometimes surgical manipulations and operations. Meanwhile, the transient hypogammaglobulinemia of infancy is benign and at last leads to recovery when those children are at the age of 6 years.

Secondary immunocompromised conditions may end up from HIV infection, malnutrition, post-traumatic stress disorder, aging and immunosenescence, radiation therapy, particular medications (e.g., immunosuppressive drugs after graft transplantation, disease-modifying antirheumatic drugs, chemotherapy in malignancies, prolonged corticosteroid therapy, etc.), many sorts of cancer (leukemias, lymphomas, etc.), protein-losing enteropathy, burns, uremia, loss of lymphoid organs (e.g., splenectomy, appendectomy, resection of the little intestine containing Peyer's patches, etc.), and a few autoimmune diseases and other disorders. Interestingly, the highperformance sports are in danger of occurrence of the secondary immunocompromised condition.

Vaccines

Vaccines function by stimulating the immune system and prompting a primary response to an infecting pathogen or to molecules derived from a selected pathogen [7]. The reaction elicited by this primary exposure to vaccine pathogen creates immunological memory, which involves the generation of a pool of immune cells that may recognize the pathogen and mount a more robust or secondary response upon subsequent exposure to the virus or bacterium. In successful immunization, the secondary reaction is sufficient to stop disease within the infected individual, still as prevent the transmission of the pathogen to others. For communicable diseases, immunizations protect not only the individual who receives the immunization, but also others with whom he or she has contact. High levels of vaccination in an exceedingly community increase the amount of individuals who are less susceptible or resistant to illness and propagation of the infective agent. Unvaccinated individuals or people who haven't developed immunity to the current pathogen are afforded an indirect measure of protection because those with immunity reduce the spread of the pathogen throughout the whole population. The larger the proportion of individuals with immunity, greater the protection of these without immunity. This effect is named "herd immunity." Herd immunity is a very important phenomenon as immunization programs rarely achieve 100 percent immunization during a population; and in some cases, previously vaccinated persons might not exhibit effective immunity and disease may result from exposure to the pathogen. for protection, immunization of not only ourselves but also our neighbors is very important.

The overwhelming safety and effectiveness of vaccines in current use in preventing serious disease has allowed them to gain their preeminent role within the routine protection of health. Before an immunization is introduced for population-wide use, it's tested for efficacy and safety. However, immunization isn't without risks. As an example, it's well established that the oral polio vaccine on rare occasion causes paralytic polio which vaccines sometimes result in anaphylactic shock. Given the widespread use of vaccines; state mandates requiring vaccination of children for entry into school, college, or day care; and also the importance of ensuring that trust in immunization programs is justified, it's essential that safety concerns receive assiduous attention.

Herd immunity describes the collective immunological status of a population of hosts, as opposition a personal host, with relevancy a given pathogen [8]. Herd immunity may be thought of as a collective biological state of a population of hosts. Herd immunity of a population is high if many of us are immunized or have recovered from infection with immunity or be low if most people are susceptible. The level of herd immunity can decrease if the proportion of susceptibles increases or vaccinated protection wanes in individuals. The term herd immunity is usually somewhat incorrectly used to refer to the threshold at which circulation of an agent is essentially eliminated. We prefer the definition of herd immunity that considers it a continuum instead of a threshold.

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If herd immunity is high enough, then a threshold could also be reached at which infectious hosts now not contact enough susceptible hosts to take care of transmission.

Conclusion

Immunity encompasses specific and nonspecific components. Nonspecific components act as barriers or eliminators of a large number of pathogens regardless of their antigenic ability. Other components of the immune system adapt to each new unknown infection, which means that they have the potential ability to create immunity for each individual type of pathogen. Creating immunity is the body's ability to defend itself against various diseases, infections and contagions. Every person has certain immunity, someone stronger, someone weaker, it is important to further strengthen it or occasionally activate it, especially at a certain season.

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