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Principals of innate and adaptive immunity. Immunity to microbes & fundamental concepts in immunology

Andrew Kiboneka *

Department of Paediatrics, Case Hospital, Kampala, Uganda.

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Abstract

Microorganisms such as bacteria that penetrate the epithelial surfaces of the body for the first time are met immediately by cells and molecules that can mount an innate immune response. Phagocytic macrophages conduct the defense against bacteria by means of surface receptors that are able to recognize and bind common constituents of many bacterial surfaces. Bacterial molecules binding to these receptors trigger the macrophage to engulf the bacterium and also induce the secretion of biologically active molecules. Activated macrophages secrete cytokines, which are defined as proteins released by cells that affect the behavior of other cells that bear receptors for them. They also release proteins known as chemokines that attract cells with chemokine receptors such as neutrophils and monocytes from the bloodstream. Macrophages in response to bacterial constituents initiate the process known as inflammation. Antigen-presenting cells (APCs) are a heterogeneous group of immune cells that mediate the cellular immune response by processing and presenting antigens for recognition by certain lymphocytes such as T cells. Classical APCs include dendritic cells, macrophages, Langerhans cells and B cells. Innate lymphoid cells (ILCs) are immune cells that belong to the lymphoid lineage but do not express antigen-specific receptors. These cells have important functions in innate immune responses to infectious microorganisms and in the regulation of homeostasis and inflammation.

Keywords: Cells; Innate; Adaptive; Immunity; Interleukin 2 cells

1. Introduction

Innate immunity is the immunity one is born with and is the initial response by the body to eliminate microbes and prevent infection. Adaptive (acquired) immunity refers to antigen-specific defense mechanisms that take several days to become protective and are designed to react with and remove a specific antigen. I n biology and genetics, the germline is the population of a multicellular organism's cells that pass on their genetic material to the progeny (offspring). In other words, they are the cells that form the egg, sperm and the fertilized egg, as well as the fertilized egg's future sperm or egg cells. They are usually differentiated to perform this function and segregated in a specific place away from other bodily cells. In sexually reproducing organisms, cells that are not in the germline are called somatic cells. According to this view, mutations, recombination and other genetic changes in the germline may be passed to offspring, but a change in a somatic cell will not be.

2. Material and methods

A literature review of articles published between January 2015 through April 2021 on PubMed and Hinari search engines. Search items included Innate and adaptive Immune system, cells and molecules, Immunity.

* Corresponding author: Andrew Kiboneka
Department of Paediatrics Case Hospital, Kampala, Uganda.

3. Results

3.1. Components of the innate Immune System

The innate immune system is one of the two main immunity strategies found in vertebrates (the other being the adaptive immune system). The innate immune system is an older evolutionary defense strategy, relatively speaking, and is the dominant immune system response found in plants, fungi, insects, and primitive multicellular organisms.

The major functions of the vertebrate innate immune system include:

- Recruiting immune cells to sites of infection through the production of chemical factors, including specialized chemical mediators called cytokines
- Activation of the complement cascade to identify bacteria, activate cells, and promote clearance of antibody complexes or dead cells
- Identification and removal of foreign substances present in organs, tissues, blood and lymph, by specialized white blood cells
- Activation of the adaptive immune system through a process known as antigen presentation.
- Acting as a physical and chemical barrier to infectious agents; via physical measures like skin or tree bark and chemical measures like clotting factors in blood or sap from a tree, which are released following a contusion or other injury that breaks through the first-line physical barrier (not to be confused with a second-line physical or chemical barrier, such as the blood-brain barrier, which protects the extremely vital and highly sensitive nervous system from pathogens that have already gained access to the host's body).

3.2. Toll like Receptors

Toll-Like Receptors (TLRs) are a class of proteins that play a key role in the innate immune system. They are single-pass membrane-spanning receptors usually expressed on sentinel cells such as macrophages and dendritic cells, that recognize structurally conserved molecules derived from microbes [1]. Once these microbes have breached physical barriers such as the skin or intestinal tract mucosa, they are recognized by TLRs, which activate immune cell responses. The TLRs include TLR1, TLR2, TLR3, TLR4, TLR5, TLR6, TLR7, TLR8, TLR9, TLR10, TLR11, TLR12, and TLR13, though the last three are not found in humans [1,2).

3.3. Immune cells

3.3.1. Immune cells-essentially are white blood cells

White blood cells (WBCs), also called leukocytes or leucocytes, are the cells of the immune system that are involved in protecting the body against both infectious disease and foreign invaders. All white blood cells are produced and derived from multipotent cells in the bone marrow known as hematopoietic stem cells. Leukocytes are found throughout the body, including the blood and lymphatic system.

All white blood cells have nuclei, which distinguishes them from the other blood cells, the anucleated red blood cells (RBCs) and platelets.

The different white blood cell types are classified in standard ways; two pairs of broadest categories classify them either by structure (granulocytes or agranulocytes) or by cell lineage (myeloid cells or lymphoid cells) [2].

These broadest categories can be further divided into the five main types: neutrophils, eosinophils (Acidophiles), basophils, lymphocytes, and monocytes. These types are distinguished by their physical and functional characteristics. Monocytes and neutrophils are phagocytic. Further subtypes can be classified; for example, among lymphocytes, there are B cells (named from bursa or bone marrow cells), T cells (named from thymus cells), and natural killer cells.

Phagocytosis (from Ancient Greek φαγεῖν (phagein) 'to eat', and κύτος, (kytos) 'cell') is the process by which a cell uses its plasma membrane to engulf a large particle (≥ 0.5 μm), giving rise to an internal compartment called the phagosome. It is one type of endocytosis.

3.3.2. The engulfing of a pathogen by a phagocyte

Phagocytosis is one main mechanisms of the innate immune defense. It is one of the first processes responding to infection, and is also one of the initiating branches of an adaptive immune response. Although most cells are capable of phagocytosis, some cell types perform it as part of their main function. These are called 'professional phagocytes.' Phagocytosis is old in evolutionary terms, being present even in invertebrates. (Animals without a backbone or bony skeleton).

3.4. Innate Lymphoid cells

ILCs are a family of lymphocytes comprising the innate counterparts of T cells. They are poised to secrete cytokines that respond swiftly to pathogenic tissue damage and shape subsequent adaptive immunity [3]. While lacking antigen-specific receptors, ILCs detect changes in the microenvironment through receptors for cytokines that are released upon tissue damage, as well as a broad range of receptors for nutrient components, microbial products, lipid mediators, and neuronal transmitters. Found in both lymphoid and non-lymphoid tissues, ILCs are primarily tissue resident cells and are particularly abundant at the mucosal surfaces of the intestine and lung, whereas they are extremely rare in peripheral blood [3]. Based on the signature cytokines they produce, their phenotype, and their developmental pathways, ILCs are divided into three major groups: ILC1s, ILC2s, and ILC3s. Two additional immune cell types, NK cells and lymphoid tissue inducer (LTi) cells, are generally included in the ILC family because their phenotypic, developmental and functional properties overlap considerably with those of ILC1s and ILC3s, respectively [4,5]

3.4.1. Group 1 ILCs

ILC1 and NK cell lineages diverge early in their developmental pathways and can be discriminated by their difference in dependence on transcription factors, their cytotoxicity, and their resident marker expression. NK cells are cytotoxic cells, circulating in the bloodstream, killing virus-infected, and tumor cells. ILC1s, are non- cytotoxic or weakly cytotoxic, tissue resident cells, functioning in the defense against infections with viruses and certain bacteria.

3.4.2. Group 2 ILCs

ILC2s are tissue resident and involved in the innate response to parasites such as the helminth infection, by helping repair tissue damage. They are abundant in tissues of the skin, lung, liver, and gut. They are characterized by the production of amphiregulin, and type 2 cytokines, including IL-4, IL-5, and IL-13, in response to IL-25, TSLP, and IL-33 [6]. Due to their cytokine signature, they are considered the innate counterparts of Th2 cells. Recently, there is a common hypothesis emphasizing epithelium-derived cytokines, namely IL-25, IL-33, and TSLP, as key regulatory factors that link in immune-pathogenic mechanisms of allergic rhinitis (AR), chronic rhinosinusitis (CRS), and asthma, mainly involving in type 2 inflammatory responses and linking innate and adaptive immunities. The upper and lower

airways are lined with respiratory epithelium that plays a vital role in immune surveillance and modulation as the first line of defense to various infective pathogens,allergens, and physical insults .

3.4.3. Group 3 ILCs

ILC3s are involved in the innate immune response to extracellular bacteria and fungi. They play a key role in homeostasis of the intestinal bacteria, and in regulating Th17 cell responses [17]. Human adult ILC3s, are primarily found in the lamina propria of the intestine, and the tonsils, however, they are also found in the spleen, endometrium, decidua, and skin.

ILC3s are dependent on the transcription factor RORγt for their development and function.

3.5. Lymphoid Tissue inducer (LTi) cells

LTi cells are considered a separate lineage due to their unique developmental pathway, however, they are often considered part of the ILC3 group as they have many similar characteristics. Like ILC3s, LTi cells are dependent on RORγt.

They are involved in the formation of secondary lymph nodes, and Peyer's patches, by promoting lymphoid tissue development, through the action of lymphotoxin, a member of the TNF superfamily [6]. They are critical during both the embryonic and adult stages of development of the immune system, and therefore LTi cells are present in organs and tissues early during embryonal development.[6] They have a pivotal role in primary and secondary lymphoid tissue organization, and in adult lymphoid tissue, regulating the adaptive immune response and maintaining secondary lymphoid tissue structures.

3.6. Signaling molecules

Cellular communication ensures regulation of biological processes within various environments from single-celled to multicellular organisms. The major types of signaling mechanisms that occur in multicellular organisms are paracrine, endocrine, autocrine, and direct signaling. Signaling molecules are often called ligands, a general term for molecules that bind specifically to other molecules (such as receptors). The message carried by a ligand is often relayed through a chain of chemical messengers inside the cell. Here are four categories of chemical signaling found in multicellular organisms: paracrine signaling, endocrine signaling, autocrine signaling, and direct signaling across gap junctions. The main difference between the different categories of signaling is the distance that the signal travels through the organism to reach the target cell [2,3].

3.7. Cell adhesion molecules

May be defined as a molecule on the cell surface that mediates binding of cell to another cell or to an acellular material (e.g. extracellular matrix) Cell adhesion molecules are transmembrane or membrane-linked glycoproteins that mediate the connections between cells or the attachment of cells to substrate (such as stroma or basement membrane).. Cell adhesion is also an integrated component of the immune system and wound healing.

3.8. Cell adhesion molecules: Structure

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. These proteins can interact in several different ways [2,3].

4. Families of CAMS

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 (2).

One classification system involves the distinction between calcium-independent CAMs and calcium-dependent CAMs. Integrins and the Ig-superfamily CAMs do not depend on Ca^{2+} while cadherins and selectins depend on Ca^{2+}.

In addition, integrins participate in cell–matrix interactions, while other CAM families participate in cell–cell interactions.

4.1. Signal transduction

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.

Cell Adhesion Molecules (CAMs) and junctional complexes are abundant in epithelial tissues. Tight junctions build a seal between adjacent cells and are connected to actin filaments [2]. Adherens junctions are plaques of classical cadherins linked to the actin cytoskeleton [3]. Desmosomes are formed by desmosome cadherins, linked to intermediate filaments [4]. Gap junctions connect the cytoplasm of two adjacent cells and are linked to microfilaments. [Selectins, Ig-superfamily CAMs, but also other CAMs not belonging to the classical families can promote homophilic adhesion outside of junctions. Integrins bind in a heterophilic manner [2]. Focal adhesions (linked to actin) and hemi-desmosomes (linked to intermediate filaments) are cell-matrix junctions that are formed by integrins.

5. Cell to cell communication

5.1. Signal transduction

Signal transduction (also known as cell signaling) is the transmission of molecular signals from a cell's exterior to its interior. Signals received by cells must be transmitted effectively into the cell to ensure an appropriate response. This step is initiated by cell-surface receptors. The four steps of signal transduction [2]. Signal molecule binds to receptor that [2] activates a protein that [3] creates second messengers that creates a response.

5.2. Functions of the innate and adaptive immune systems

The innate immune system provides an immediate response to foreign targets, with responses typically within minutes to hours. It consists of a number of soluble factors and proteins as well as a diverse set of cells, including granulocytes, macrophages, dendritic cells and natural killer cells. The second branch of the immune system is the adaptive or acquired immune system, which provides specific, long-lasting immune responses. The adaptive and innate immune systems are linked; for example, dendritic cells are important adaptive immune system cell activators. The adaptive immune system consists of antibodies, B cells, and CD^{4+} and CD8 + T cells, and these enable a highly specific response against a particular target. Natural killer T cells and γδ T cells are cytotoxic lymphocytes that overlap both innate and adaptive immunity. Cells from both arms of the immune system are in development as potential cellular immunotherapies.

6. Discussion

6.1. Components of the Adaptive Immune system

Adaptive or acquired immunity is the protection mechanism from an infectious disease agent as a consequence of clinical or subclinical infection with that agent or by deliberate immunization against that agent with products from it. This type of immunity is mediated by B and T cells following exposure to a specific antigen. It is characterized by specificity, immunological memory, and self/non-self-recognition. The response involves clonal selection of lymphocytes that respond to a specific antigen. T cells and B cells are the two major components of adaptive immunity. The first line of defense against non-self-pathogens is the innate, or non-specific, immune response. The innate immune response consists of physical, chemical and cellular defenses against pathogens. The main purpose of the innate immune response is to immediately prevent the spread and movement of foreign pathogens throughout the body.

The second line of defense against non-self-pathogens is called adaptive immune response. Adaptive immunity is also referred to as acquired immunity or specific immunity and is only found in vertebrates. The adaptive immune response is specific to the pathogen presented. The adaptive immune response is meant to attack non-self-pathogens but can sometimes make errors and attack itself. When this happens, autoimmune diseases can develop (e.g., lupus, rheumatoid arthritis). Acquired immunity creates immunological memory after an initial response to a specific pathogen, and leads to an enhanced response to subsequent encounters with that pathogen. This process of acquired immunity is the basis of vaccination. Like the innate system, the acquired system includes both humoral immunity components and cell-mediated immunity components.

The hallmark of the adaptive immune system is the clonal expansion of lymphocytes. Clonal expansion is the rapid increase of T and B lymphocytes from one or a few cells to millions. Each clone that originates from the original T or B lymphocyte has the same antigen receptor as the original and fights the same pathogen [7].

While the innate immune response is immediate, the adaptive immune response is not. However, the effect of the adaptive immune response is long-lasting, highly specific, and is sustained long-term by memory T cells.

Table 1 Differences between innate and adaptive immunity

	Line of Defence	Timeline	Cells	Antigen Dependency	Examples
Innate (non-specific)	First	Immediate response (0 -96 hours)	Natural killer cells, macrophages, neutrophils, dendritic cells, mast cells, basophils, eosinophils	Independent	Skin, hair, cough, mucous membranes, phagocytes, granulocytes
Adaptive (specific)	Second	Long term (>96 hours)	T and B lymphocytes	Dependent	Pus, swelling, redness, pain, T and B lymphocyte response

6.2. Cells of the adaptive immune system

The adaptive defense consists of antibodies and lymphocytes, often called the humoral response and the cell mediated response. The term 'adaptive' refers to the differentiation of self from non-self, and the tailoring of the response to the particular foreign invader. The ability to shape the response in a virus-specific manner depends upon communication between the innate and adaptive systems. This communication is carried out by cytokines that bind to cells, and by cell-cell interactions between dendritic cells and lymphocytes in lymph nodes. This interaction is so crucial that the adaptive response cannot occur without an innate immune system. The cells of the adaptive immune system are lymphocytes – B cells and T cells. B cells, which are derived from the bone marrow, become the cells that produce antibodies. T cells, which mature in the thymus, differentiate into cells that either participate in lymphocyte maturation, or kill virus-infected cells.

6.3. Immunity to microbes

The development of an infectious disease in an individual involves complex interactions between the microbe and the host. The key events during infection include entry of the microbe, invasion and colonization of host tissues, evasion of host immunity, and tissue injury or functional impairment. Microbes produce disease by directly killing the host cells they infect, or by liberating toxins that can cause tissue damage and functional derangements in neighboring or distant cells and tissues that are not infected (8,9).

The interaction of the immune system with infectious organisms is a dynamic interplay of host mechanisms aimed at eliminating infections and microbial strategies designed to permit survival in the face of powerful defenses. Different types of infectious agents stimulate distinct types of immune responses and have evolved unique mechanisms for evading immunity. In some infections, the immune response is the cause of tissue injury and disease (10).

Here we consider the main features of immunity to four major categories of pathogenic microorganisms: extracellular and intracellular bacteria, fungi, viruses, and protozoan as well as multicellular parasites.

7. Immunity to Bacteria

7.1. Extracellular Bacteria

Extracellular bacteria are capable of replicating outside host cells, for example, in the blood, in connective tissues, and in tissue spaces such as the lumens of the airways and gastrointestinal tract. Many different species of extracellular bacteria are pathogenic, and disease is caused by two principal mechanisms. First, these bacteria induce inflammation, which results in tissue destruction at the site of infection. Second, bacteria produce toxins, which have diverse pathologic effects. The toxins may be endotoxins, which are components of bacterial cell walls, or exotoxins, which are secreted by the bacteria. The endotoxin of gram-negative bacteria, also called lipopolysaccharide (LPS), is a potent activator of macrophages, dendritic cells, and endothelial cells. Many exotoxins are cytotoxic, and others cause disease by various mechanisms. For instance, diphtheria toxin shuts down protein synthesis in infected cells, cholera toxin interferes with ion and water transport, tetanus toxin inhibits neuromuscular transmission, and anthrax toxin disrupts several critical biochemical signaling pathways in infected cells. Other exotoxins interfere with normal cellular functions without killing cells, and yet other exotoxins stimulate the production of cytokines that cause disease.

7.2. Innate Immunity

The principal mechanisms of innate immunity to extracellular bacteria are complement activation, phagocytosis, and the inflammatory response.

7.3. Complement activation

Peptidoglycans in the cell walls of Gram-positive bacteria and LPS in Gram-negative bacteria activate complement by the alternative pathway. Bacteria that express mannose on their surface may bind mannose-binding lectin, which activates complement by the lectin pathway. One result of complement activation is opsonization and enhanced phagocytosis of the bacteria. In addition, the membrane attack complex generated by complement activation lyses bacteria, Phagocytes and inflammation: Phagocytes (neutrophils and macrophages) use surface receptors, including mannose receptors and scavenger receptors, to recognize extracellular bacteria, and they use Fc receptors and complement receptors to recognize bacteria opsonized with antibodies and complement proteins, respectively. In addition, dendritic cells and phagocytes that are activated by the microbes secrete cytokines, which induce leukocyte infiltration into sites of infection (inflammation). The recruited leukocytes ingest and destroy the bacteria.

7.4. Adaptive Immunity

Humoral immunity is a major protective immune response against extracellular bacteria, and it functions to block infection, to eliminate the microbes, and to neutralize their toxins. Antibody responses against extracellular bacteria are directed against cell wall antigens and secreted and cell-associated toxins, which may be polysaccharides or proteins. The polysaccharides are prototypic T-independent antigens, and humoral immunity is the principal mechanism of defense against polysaccharide-rich encapsulated bacteria. The effector mechanisms used by antibodies to combat these infections include neutralization, opsonization and phagocytosis, and activation of complement by the classical pathway. The protein antigens of extracellular bacteria also activate CD4+ helper T cells, which produce cytokines that induce local inflammation, enhance the phagocytic and microbicidal activities of macrophages and neutrophils, and stimulate antibody production.

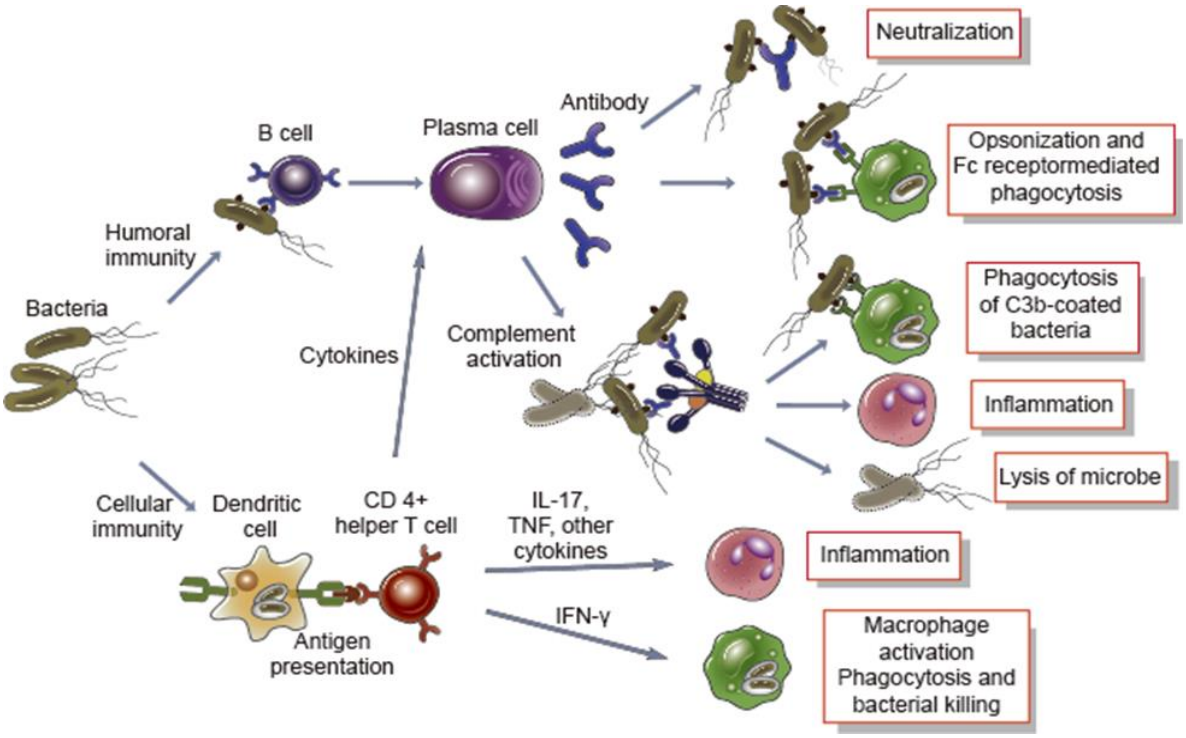

Figure 1 Adaptive immune responses to extracellular bacteria [2]

8. Intracellular Bacteria

A characteristic of facultative intracellular bacteria is their ability to survive and even to replicate within phagocytes. Because these microbes are able to find a niche where they are inaccessible to circulating antibodies, their elimination requires the mechanisms of cell-mediated immunity.

8.1. Innate Immunity

The innate immune response to intracellular bacteria is mediated mainly by phagocytes and natural killer (NK) cells. Intracellular bacteria activate NK cells by inducing expression of NK cell–activating ligands on infected cells and by stimulating dendritic cell and macrophage production of IL-12 and IL-15, both of which are NK cell activating cytokines. The NK cells produce IFN-γ, which in turn activates macrophages and promotes killing of the phagocytosed bacteria. Thus, the NK cells provide an early defense against these microbes, before the development of adaptive immunity.

8.2. Adaptive Immunity

The major protective immune response against intracellular bacteria is T cell–mediated recruitment and activation of phagocytes (cell-mediated immunity). Phagocytosed bacteria stimulate CD^{8+} T cell responses if bacterial antigens are transported from phagosomes into the cytosol or if the bacteria escape from phagosomes and enter the cytoplasm of infected cells. In the cytosol, the microbes are no longer susceptible to the microbicidal mechanisms of phagocytes, and for eradication of the infection, the infected cells have to be killed by CTLs. Thus, the effectors of cell-mediated immunity,

namely, CD^{4+} T cells that activate macrophages and CD^{8+} CTLs, function cooperatively in defense against intracellular bacteria.

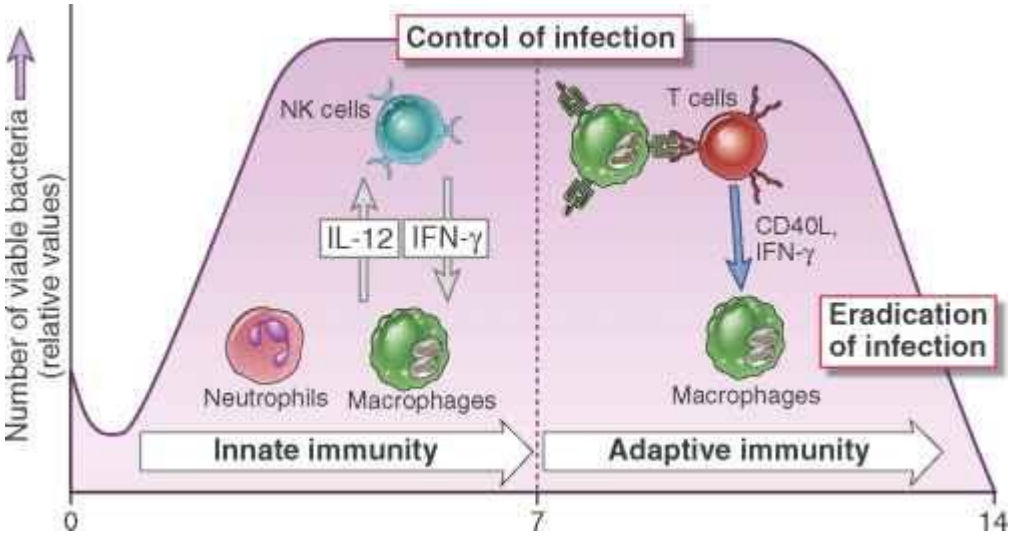

Figure 2 Innate and adaptive immunity to intracellular bacteria [2]

8.3. Immunity to viruses

Viruses are obligatory intracellular microorganisms that use components of the nucleic acid and protein synthetic machinery of the host to replicate and spread. Viruses typically infect various cell types by using normal cell surface molecules as receptors to enter the cells. After entering cells, viruses can cause tissue injury and disease by any of several mechanisms. Innate and adaptive immune responses to viruses are aimed at blocking infection and eliminating infected cells. Infection is prevented by type I interferons as part of innate immunity and neutralizing antibodies contributing to adaptive immunity. Once infection is established, infected cells are eliminated by NK cells in the innate response and CTLs in the adaptive response.

8.4. Innate Immunity to Virus

The principal mechanisms of innate immunity against viruses are inhibition of infection by type I interferons and NK cell–mediated killing of infected cells. NK cells kill other cells infected with a variety of viruses and are an important mechanism of immunity against viruses early in the course of infection, before adaptive immune responses have developed.

8.5. Adapted Immunity to Virus

Adaptive immunity against viral infections is mediated by antibodies, which block virus binding and entry into host cells, and by CTLs, which eliminate the infection by killing infected cells. Antibodies are effective against viruses only during the extracellular stage of the lives of these microbes. Viruses may be extracellular early in the course of infection, before they infect host cells, or when they are released from infected cells by virus budding or if the infected cells die. Antiviral antibodies bind to viral envelope or capsid antigens and function mainly as neutralizing antibodies to prevent virus attachment and entry into host cells. Elimination of viruses that reside within cells is mediated by CTLs, which kill the infected cells. The principal physiologic function of CTLs is surveillance against viral infection. Most virus-specific CTLs are CD^{8+} T cells that recognize cytosolic, usually endogenously synthesized, viral peptides presented by class I MHC molecules.

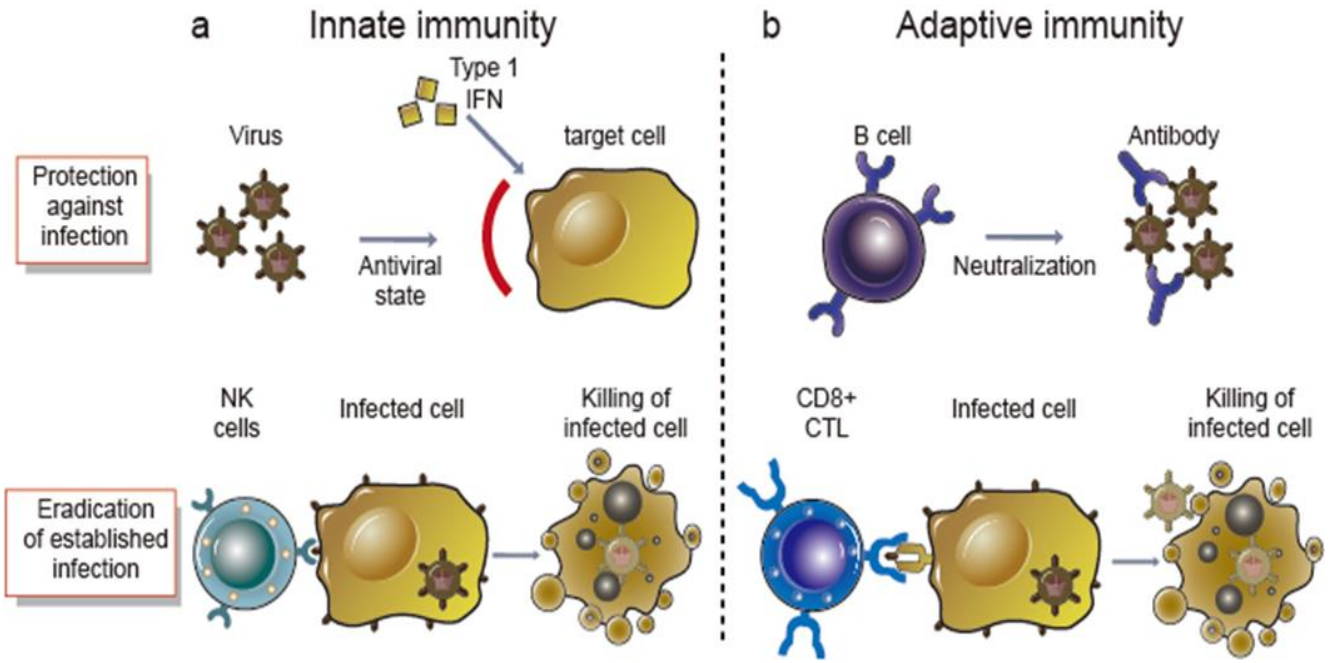

Figure 3 Innate and adaptive immune responses against viruses [2]

8.6. Immunity to parasites

In infectious disease terminology, parasitic infection refers to infection with animal parasites such as protozoa, helminths, and ectoparasites (e.g., ticks and mites). Most parasites go through complex life cycles, part of which occurs in humans (or other vertebrates) and part of which occurs in intermediate hosts, such as flies, ticks, and snails. Humans are usually infected by bites from infected intermediate hosts or by sharing a particular habitat with an intermediate host. Most parasitic infections are chronic because of weak innate immunity and the ability of parasites to evade or resist elimination by adaptive immune responses. Furthermore, many anti-parasitic drugs are not effective at killing the organisms.

9. Innate Immunity to Parasites

Although different protozoan and helminthic parasites have been shown to activate different mechanisms of innate immunity, these organisms are often able to survive and replicate in their hosts because they are well adapted to resist host defenses. The principal innate immune response to protozoa is phagocytosis, but many of these parasites are resistant to phagocytic killing and may even replicate within macrophages. Phagocytes may also attack helminthic parasites and secrete microbicidal substances to kill organisms that are too large to be phagocytosed. However, many helminths have thick teguments that make them resistant to the cytocidal mechanisms of neutrophils and macrophages, and they are too large to be ingested by phagocytes.

9.1. Adapted Immunity to Parasites

Different protozoa and helminths vary greatly in their structural and biochemical properties, life cycles, and pathogenic mechanisms. It is therefore not surprising that different parasites elicit distinct adaptive immune responses. Some pathogenic protozoa have evolved to survive within host cells, so protective immunity against these organisms is mediated by mechanisms similar to those that eliminate intracellular bacteria and viruses. In contrast, metazoan such as helminths to survive in extracellular tissues, and their elimination is often dependent on special types of antibody responses. The principal defense mechanism against protozoa that survive within macrophages is cell-mediated immunity, particularly macrophage activation by TH1 cell–derived cytokines. Defense against many helminthic infections is mediated by the activation of TH2 cells, which result in production of IgE antibodies and activation of eosinophils.

9.2. Immunity to fungi

Fungal infections, also called mycoses, are important causes of morbidity and mortality in humans. Some fungal infections are endemic, and these infections are usually caused by fungi that are present in the environment and whose spores enter humans. Other fungal infections are said to be opportunistic because the causative agents cause mild or no disease in healthy individuals but may infect and cause severe disease in immune-deficient persons. Compromised immunity is the most important predisposing factor for clinically significant fungal infections. Different fungi infect humans and may live in extracellular tissues and within phagocytes. Therefore, the immune responses to these microbes are often combinations of the responses to extracellular and intracellular bacteria. However, less is known about antifungal immunity than about immunity against bacteria and viruses.

10. Conclusion

Innate immunity is the immunity one is born with and is the initial response by the body to eliminate microbes and prevent infection. Adaptive (acquired) immunity refers to antigen-specific defense mechanisms that take several days to become protective and are designed to react with and remove a specific antigen. Adaptive immune responses develop later and require the activation of lymphocytes. The kinetics of the innate and adaptive immune responses are approximations and may vary in different infections.

Compliance with ethical standards

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Disclosure of conflict of interest

The author declares no conflict of interest.

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