I. Introduction to Immune System

The immune system is composed of two major subdivisions:

1. the innate (nonspecific immune system)

And

2. The adaptive (specific immune system) (Figure 1).

The innate immune system is our first line of defense against invading organisms while the adaptive immune system acts as a second line of defense and also affords protection against re-exposure to the same pathogen.

Each of the major subdivisions of the immune system has both cellular and humoral components

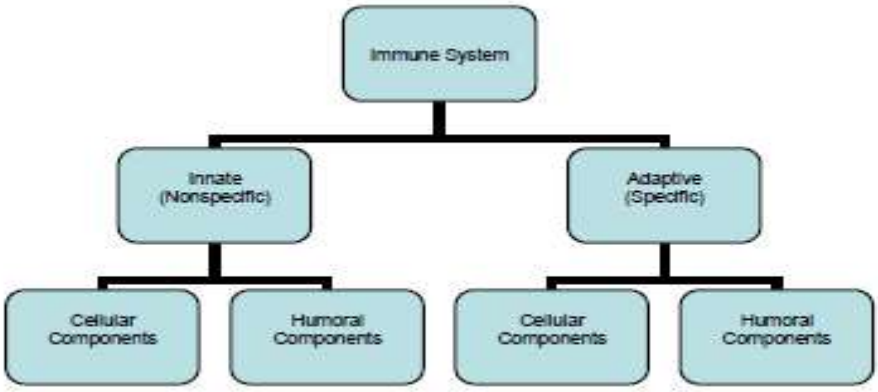


Figure 1. Overview of the Immune System

Although the innate and adaptive immune systems both function to protect against invading organisms, they differ in a number of ways.

1.The adaptive immune system requires some time to react to an invading organism, whereas the innate immune system ready to be mobilized upon infection.

2. the adaptive immune system is antigen specific and reacts only with the organism that induced the response. In contrast, the innate system is not antigen specific and reacts equally well to a variety of organisms.

3. the adaptive immune system demonstrates immunological memory. In contrast, the innate immune system does not demonstrate immunological memory.

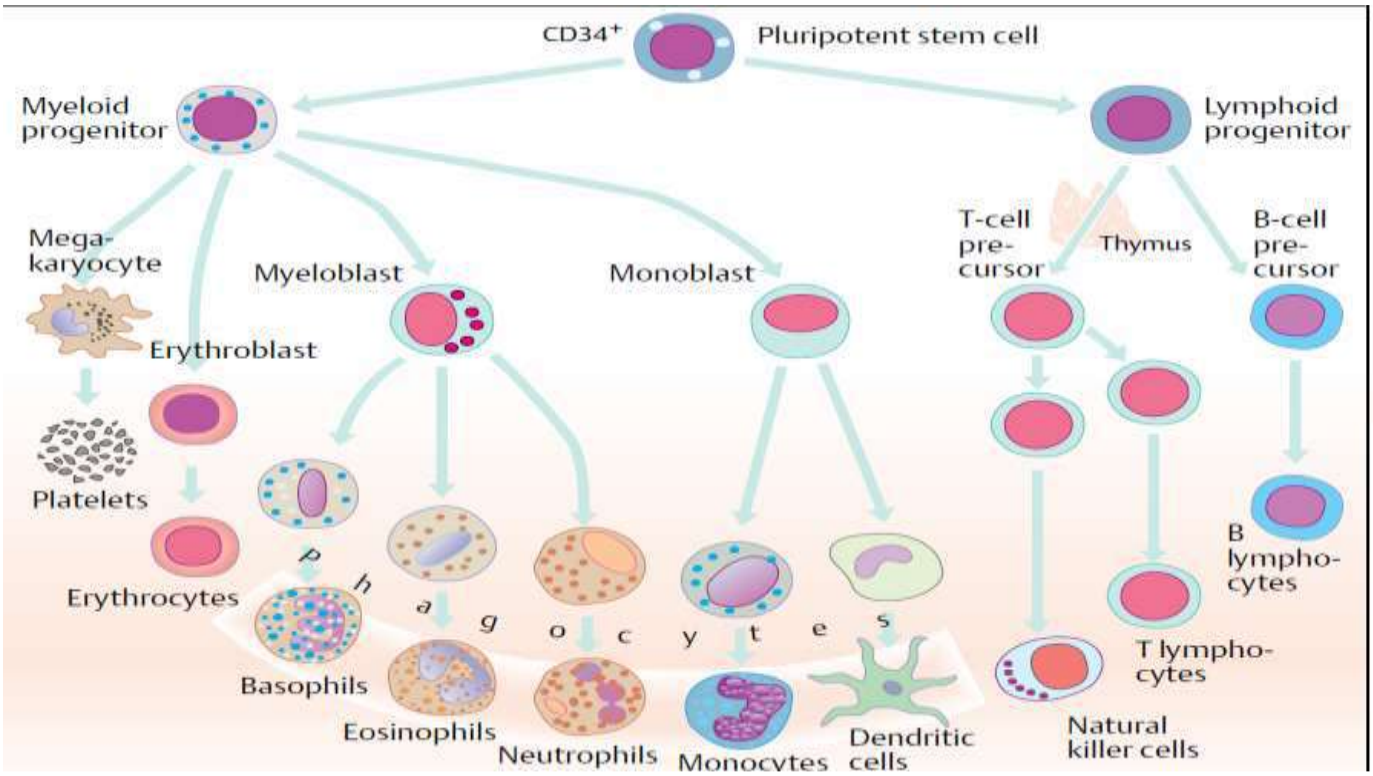
The main function of the immune system is self/non-self discrimination. This ability to distinguish between self and non-self is necessary to protect the organism from invading pathogens and to eliminate modified or altered cells (e.g. malignant cells). Since pathogens may replicate intracellularly (viruses and some bacteria and parasites) or

extracellularly (most bacteria, fungi and parasites), different components of the immune system have evolved to protect against these different types of pathogens.

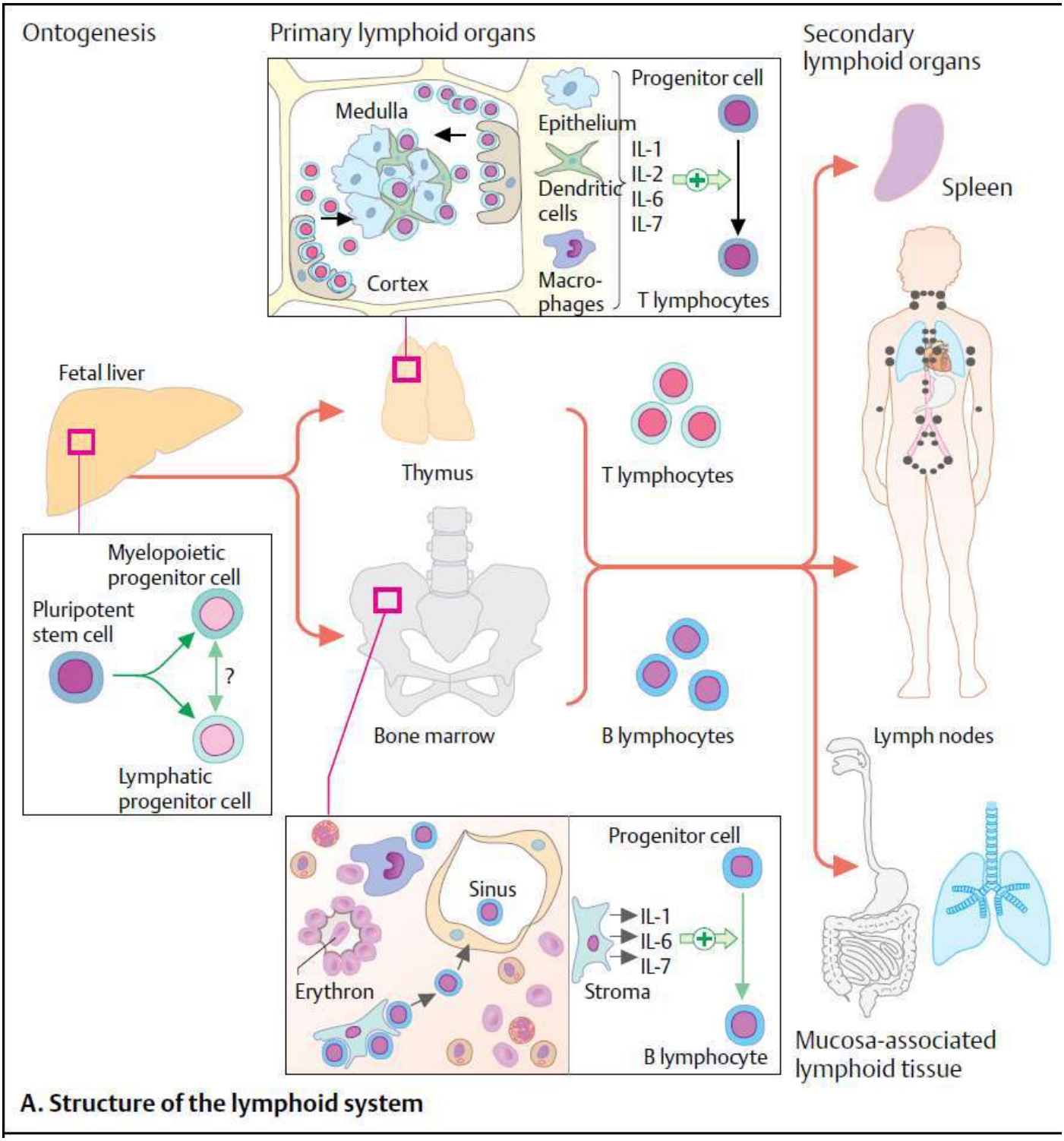
All cells of the immune system have their origin in the bone marrow and they include 1.myeloid (neutrophils, basophils, eosinpophils, macrophages and dendritic cells)

&

2. lymphoid (B lymphocyte, T lymphocyte and Natural Killer) cells (Figure 2), which differentiate along distinct pathways (Figure 3). For T cell development the precursor T cells must migrate to the thymus where they undergo differentiation into two distinct types of T cells, the CD4+ T helper cell and the CD8+ pre-cytotoxic T cell. Two types of T helper cells are produced in the thymus the TH1 cells, which help the CD8+ pre-cytotoxic cells to differentiate into cytotoxic T cells, and TH2 cells, which help B cells, differentiate into plasma cells, which secrete antibodies.



Origin of cells of the immune system



A. Structure of the lymphoid system

INNATE (NONSPECIFIC) IMMUNITY
II. Innate host defenses

A. Anatomical barriers to infections
- **Mechanical factors**

- The skin acts as our first line of defense against invading organisms. The desquamation of skin epithelium also helps remove bacteria and other infectious agents that have adhered to the epithelial surfaces.
- Movement due to cilia or peristalsis helps to keep air passages and the gastrointestinal tract free from microorganisms.
- The flushing action of tears and saliva helps prevent infection of the eyes and mouth.
- The trapping affect of mucus that lines the respiratory and gastrointestinal tract helps protect the lungs and digestive systems from infection.

- Chemical factors

- Fatty acids in sweat inhibit the growth of bacteria.
- Lysozyme and phospholipase found in tears
- saliva and nasal secretions can breakdown the cell wall of bacteria and destabilize bacterial membranes.
- -The low pH of sweat and gastric secretions prevents growth of bacteria.
- Defensins (low molecular weight proteins) found in the lung and gastrointestinal tract have antimicrobial activity.
- Surfactants in the lung act as **opsonins**(substances that promote phagocytosis of particles by phagocytic cells).

- Biological factors

The normal flora of the skin and in the gastrointestinal tract can prevent the colonization of pathogenic bacteria by secreting toxic substances or by competing with pathogenic bacteria for nutrients or attachment to cell surfaces.

B. Humoral barriers to infection

- **Complement system –** The complement system is the major humoral nonspecific defense mechanism (complement can lead to increased vascular permeability, recruitment of phagocytic cells, and lysis and opsonization of bacteria).

- **Lactoferrin and transferrin :** By binding iron, an essential nutrient for bacteria, these proteins limit bacterial growth.
- **Interferons** :Interferons are proteins that can limit virus replication in cells.
- **Lysozyme** : Lysozyme breaks down the cell wall of bacteria.

C. Cellular barriers to infection

Which include **Neutrophils – Polymorphonuclear cells (PMNs)** , **Macrophages** ,**Natural killer (NK) and lymphokine activated killer (LAK) ,Eosinophils**

INNATE & ADAPTIVE IMMUNITY

Our immune host defenses can be divided into two major categories: **innate (natural)** and **adaptive (acquired)**.

1. Innate Immunity

Innate immunity is resistance that exists **prior to exposure** to the microbe (antigen). It is **nonspecific** andSeveral components of the innate arm recognize what is foreign by detecting certain carbohydrates or lipids on the surface of microorganisms that are different from those on human cells. Components of the innate arm have receptors called **pattern-recognition receptors(PRR)**that recognize a molecular pattern called a **pathogen-associated molecular pattern (PAMP)** that is present on the surface of many microbes but—very importantly—is not present on human cells.
There are two classes of receptors on the surface of cells (Toll-like receptors and mannan-binding lectin receptors) that recognize microbes outside of cells and two classes of receptors in the cytoplasm of cells (NOD receptors and RIG-I helicase receptors) that recognize microbes within cells.

The most important of these pattern-recognition receptors are the **Toll-like receptors (TLR)**. This is a family of 10 receptors found mainly on the surface of three types of cells:macrophages, dendritic cells, and mast cells. TLRs recognize various microbial components

Four important examples of this pattern recognition are as follows:

(1) Endotoxin is a lipopolysaccharide (LPS) found on the surface of most gram negative bacteria .

(2) a polysaccharide called mannan on their surface.A pattern-recognition receptor called **mannan-binding lectin (MBL)** binds to the mannan on the surface of the microbes.

(3) Peptidoglycan (cell wall) of bacteria is recognized by **NOD receptors**.

(4) RIG-I helicase receptors recognize the nucleic acids of viruses in the cytoplasm of infected cells.

2. Adaptive (Acquired) Immunity

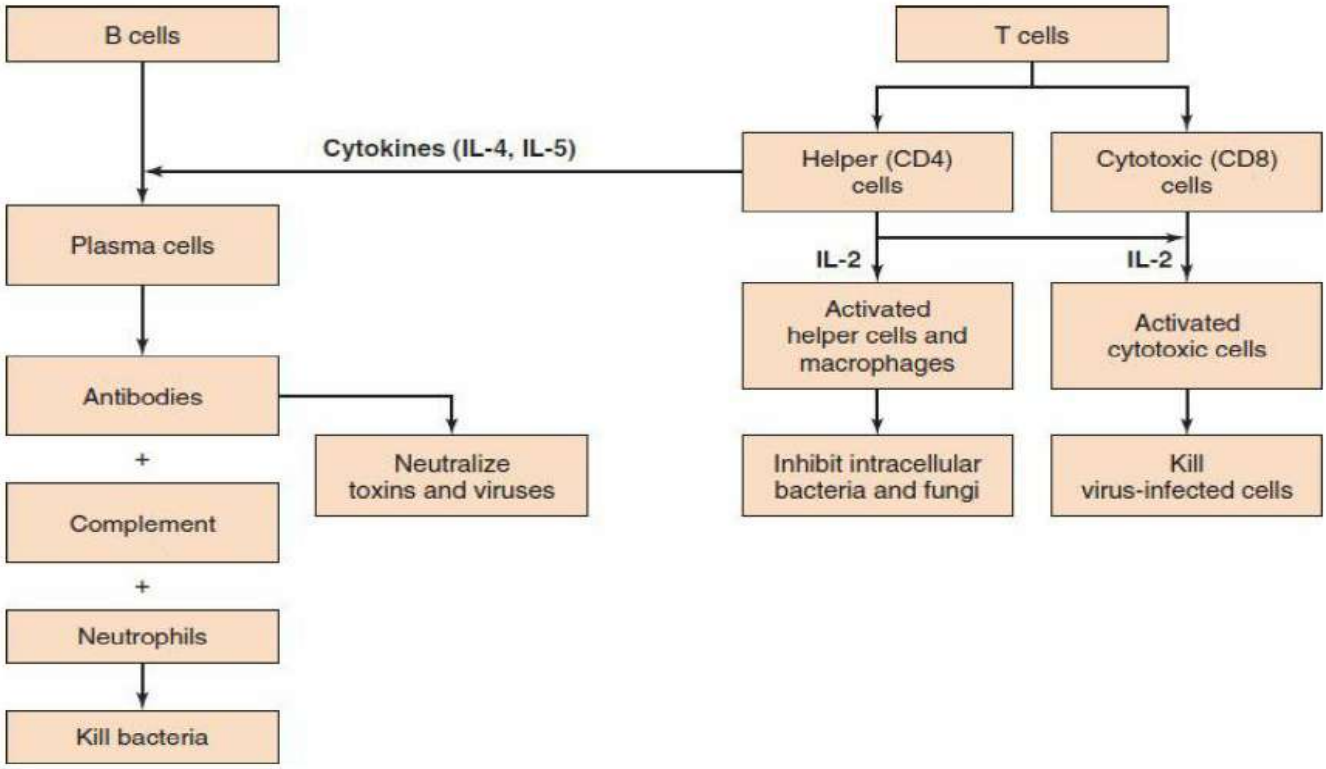
Adaptive immunity occurs **after exposure** to an agent, **improves upon repeated exposure,** and is **specific**. It is mediated by antibody produced by B lymphocytes and by two types of T lymphocytes, namely, helper T cells and cytotoxic T cells. The cells responsible for adaptive immunity have **long-term memory** for a specific antigen. Adaptive immunity can be active or passive. Macrophages and other antigen-presenting cells such as dendritic cells play an important role in both the innate and the adaptive arms of the immune system .

Adaptive immunity characterized by three important features:

(1) **diversity**(i.e., they can respond to millions of different antigens);

(2) **memory**(i.e., they can respond many years after the initial exposure because memory T cells and memory B cells are produced).

(3)**specificity**(i.e., their actions are specifically directed against the antigen that initiated the response).



Figure(1):The interactions and functions of the major components of the immune system. **Left:** Antibody- mediated (humoral) immunity.

Right: Cell-mediated immunity.

The cell-mediated arm consists primarily of **T lymphocytes** (e.g., helper T cellsand cytotoxic T cells), whereas the antibody-mediated arm consists of antibodies(immunoglobulins) and **B lymphocytes** (and plasma cells).

The main functions of antibodies are:

(1) to**neutralize toxins and viruses** and (2) to **opsonize bacteria,** making them easier to phagocytize. **Opsonization**is the process by which immunoglobulin G (IgG) antibody and the C3b component of complement enhance phagocytosis .

Cell-mediated immunity, on the other hand, inhibits organisms such as fungi, parasites, and certain intracellular bacteria such as *Mycobacterium tuberculosis*; it also kills **virus-infected cells** and **tumor cells**.

Macrophages and certain other phagocytic cells such as dendritic cellsparticipate in both the innate and adaptive arms of the immune response. They are, ineffect, a bridge between the two arms. As part of the innate arm, they ingest and killvarious microbes. They also present antigen to helper T cells, which is the essentialfirst step in the activation of the adaptive arm (see later). It is interesting to note thatneutrophils, which are also phagocytes and have excellent microbicidal abilities, do*not* present antigen to helper T cells and therefore function in innate but not acquiredimmunity.

Adaptive immunity consist of cell-mediated immunity and antibody –mediated immunity, as bellow:

1. Cell-Mediated Immunity

In the following example, a bacterium (e.g., *Mycobacterium tuberculosis*) enters the body and is ingested by a macrophage. The bacterium is broken down, and fragments of it called **antigens** or **epitopes** appear on the surface of the macrophage in association with **class II major histocompatibility complex (MHC)** proteins. The antigen−class II MHC protein complex interacts with an antigen-specific receptor on the surface of a **helper T lymphocyte**. Activation and clonal proliferation of this antigen-specific helper T cell occur as a result of the production of **interleukins,** the most important of which are interleukin-2 (T cell growth factor) and **gamma interferon** (activates macrophages).

Cytotoxic (cytolytic) T lymphocytes are also specific effectors of the cellular immune response, particularly against virus-infected cells. In this example, a virus (e.g., influenza virus) is inhaled and infects a cell of the respiratory tract. Viral envelope glycoproteins appear on the surface of the infected cell in association with **class I MHC proteins**. A cytotoxic T cell binds via its antigen-specific receptor to the viral antigen−class I MHC protein complex and is stimulated to grow into a clone of cells by interleukin-2 produced by helper T cells. These cytotoxic T cells specifically kill influenza virus−infected cells (and not cells infected by other viruses) by recognizing viral antigen−class I MHC protein complexes on the cell surface and releasing perforins that destroy the membrane of the infected cell.

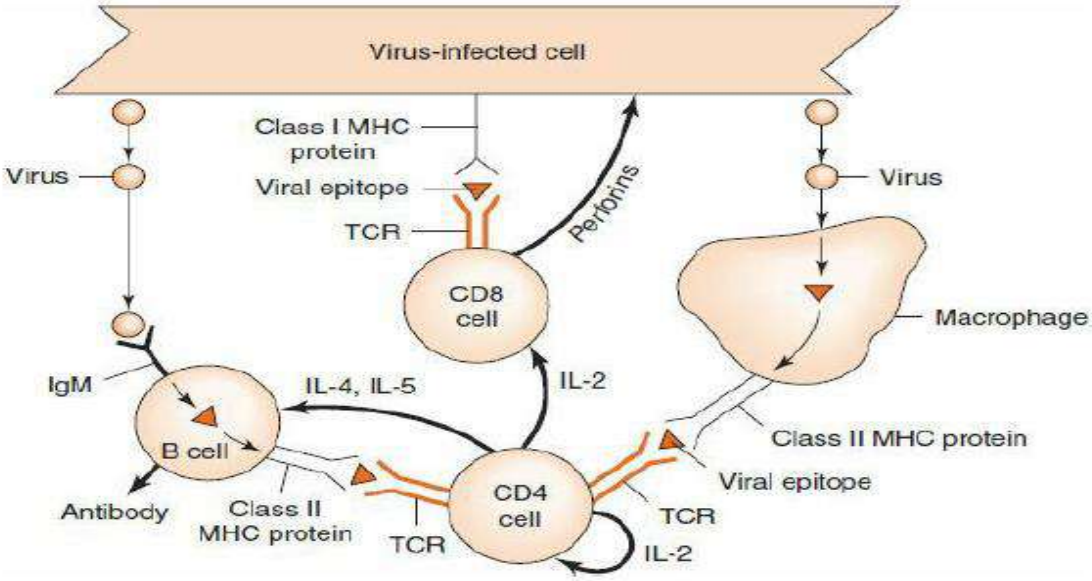


FIGURE 57–3 Induction of cell-mediated immunity and antibody against a viral infection. **Right:** Virus released by an infected cell is ingested and processed by an

2. Antibody-Mediated Immunity

Antibody synthesis typically involves the cooperation of three cells: **antigen presenting cells** (e.g., **dendritic cells** and **macrophages**), **helper T cells,** and **B cells**. After processing by an antigen-presenting cell, fragments of antigen appear on the surface of that cell in association with **class II MHC** proteins. The antigen–class II MHC protein complex binds to receptors on the surface of a helper T cell specific for that antigen. This activates the helper T cells to produce interleukins such as interleukin-2 (IL-2), IL-4, and IL-5. These interleukins activate the B cell to produce antibodies specific for that antigen.

ACTIVE & PASSIVE IMMUNITY

Active immunity is resistance induced after **contact** with foreign antigens (e.g., microorganisms). This contact may consist of clinical or subclinical infection, immunization with live or killed infectious agents or their antigens, or exposure to microbial products (e.g., toxins and toxoids). In all these instances, the host actively produces an immune response consisting of antibodies and activated helper and cytotoxic T lymphocytes.The main advantage of active immunity is that resistance is **long-term** .

Passive immunity is resistance based on antibodies **preformed** in another host.Administration of antibody against diphtheria, tetanus, botulism, etc., makes large amounts of antitoxin immediately available to neutralize the toxins. Likewise, preformed antibodies to certain viruses (e.g., rabies and hepatitis A and B viruses) can be injected

during the incubation period to limit viral multiplication. Other forms of passive immunity are IgG passed from mother to fetus during pregnancy and IgA passed from mother to newborn during breast feeding.

The main advantage of passive immunization is the **prompt availability** of large amounts of antibody; disadvantages are the **short life span** of these antibodies and possible hypersensitivity reactions if globulins from another species are used.

Passive–active immunity

involves giving both preformed antibodies (immune globulins) to provide immediate protection and a vaccine to provide long-term protection. These preparations should be given at different sites in the body to prevent the antibodies from neutralizing the immunogens in the vaccine. This

approach is used in the prevention of tetanus , rabies ,

, and hepatitis B .

Table:**Characteristics of Active and Passive Immunity**

	Mediators	Advantages	Disadvantages
Active Immunity	Antibody and T cells	Long duration (years)	Slow onset
Passive Immunity	Antibody only	Immediate availability	Short duration (months)

ANTIGENS

Antigens are molecules that react with antibodies, whereas immunogens are molecules that induce an immune response. In most cases, antigens are immunogens, and the terms are used interchangeably. However, there are certain important exceptions (e.g., haptens). A **hapten** is a molecule that is not immunogenic by itself but can react with specific antibody. Haptens are usually small molecules, but some high-molecular-weight nucleic acids are haptens as well. Many drugs (e.g.,penicillins) are haptens, and the catechol in the plant oil that causes poison oak and poison ivy is a hapten.

Haptens are not immunogenic because they cannot activate helper T cells. The failure of haptens to activate is due to their inability to bind to MHC proteins; they cannot bind because they are not polypeptides and only polypeptides can be presented by MHC proteins. Furthermore, haptens are univalent and therefore cannot activate B cells by themselves.

Although haptens cannot stimulate a primary or secondary response by themselves, they can do so when covalently bound to a “carrier” protein . In this process, the hapten interacts with an IgM receptor on the B cell and the hapten–carrier protein complex is internalized. A peptide of the carrier protein is presented in association with class II MHC protein to the helper T cells. The activated helper T cell then produces interleukins, which stimulate the B cells to produce antibody to the hapten .

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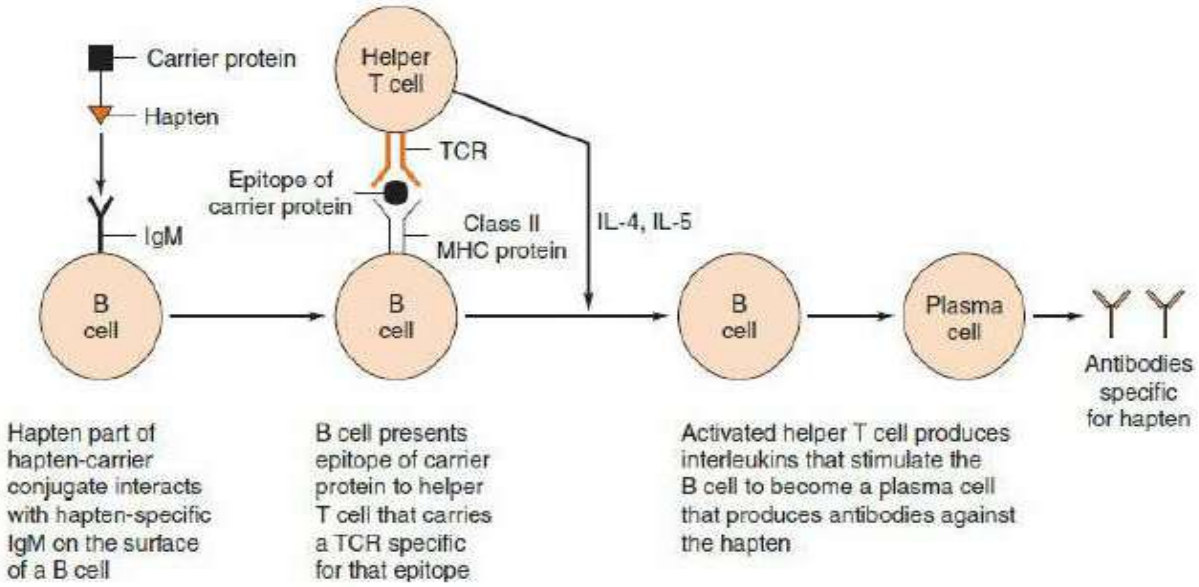


The interaction of antigen and antibody is highly specific, and this characteristic is frequently used in the diagnostic laboratory to identify microorganisms. Antigen and antibody bind by **weak forces** such as hydrogen bonds and van der Waals'forces rather than by covalent bonds. The affinity (The strength of the binding) of antibodies increases with successive exposures to the specific antigen .

The features of molecules that determine immunogenicity are as follows.

1-Foreignness

In general, molecules recognized as "self" are not immunogenic (i.e., we are tolerant to those self-molecules) . To be immunogenic, molecules must be recognized as "nonself" (i.e., foreign).

2-Molecular Size

The most potent immunogens are proteins with high molecular weights (i.e., above 100,000). Generally, molecules with molecular weight below 10,000 are weakly immunogenic, and very small ones (e.g., an amino acid) are non immunogenic. Certain small molecules (e.g., haptens) become immunogenic only when linked to acarrier protein.

3-Chemical–Structural Complexity

A certain amount of chemical complexity is required (e.g., amino acid homopolymers are less immunogenic than heteropolymers containing two or three different amino acids).

4-Antigenic Determinants (Epitopes)

Epitopes are small chemical groups on the antigen molecule that can elicit and react with antibody. An antigen can have one or more determinants (epitopes). Most antigens have many determinants (i.e., they are multivalent). In general, a determinant is roughly five amino acids or sugars in size. The overall three dimensional structure is the main criterion of antigenic specificity.

5-Dosage, Route, and Timing of Antigen Administration

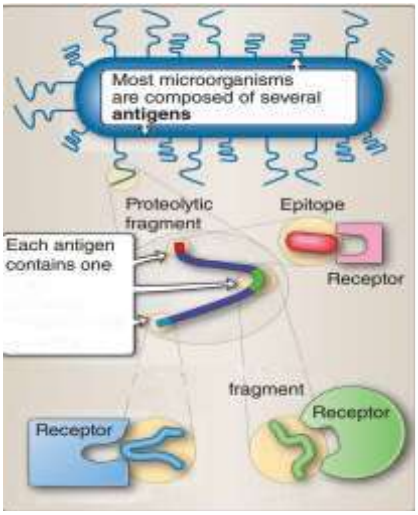
These factors also affect immunogenicity. In addition, the genetic constitution of the host (HLA genes) determines whether a molecule is immunogenic. Different strains of the same species of animal may respond differently to the same antigen.

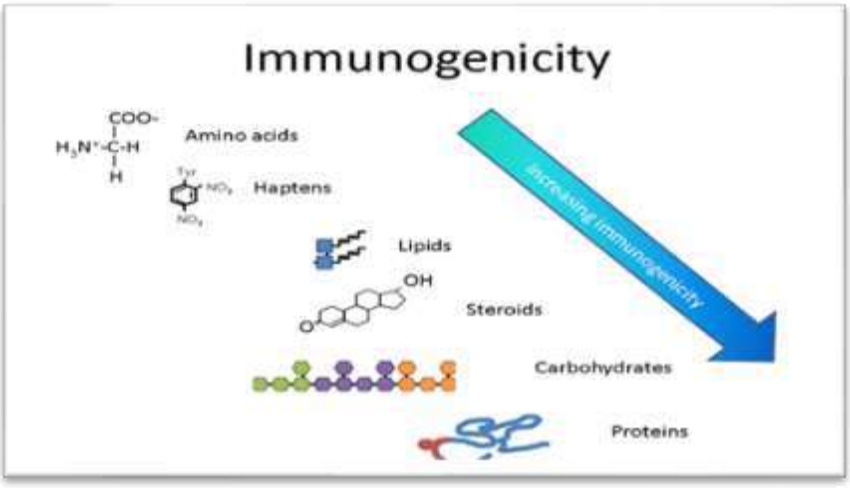
Adjuvants

Adjuvants enhance the immune response to an immunogen. They are chemically unrelated to the immunogen and differ from a carrier protein because

the adjuvant is not covalently bound to the immunogen, whereas the carrier protein is. Adjuvants can act in a variety of ways;1- they can cause slow release of immunogen, thereby prolonging the stimulus; 2-enhance uptake of immunogen by antigen-presenting cells;

3-and induce costimulatory molecules ("second signals"). Another important mechanism of action of some adjuvants is to stimulate Toll-like receptors on the surface of macrophages, which results in cytokine production that enhances the response of T cells and B cells to the immunogen (antigen).Some human vaccines contain adjuvants such as aluminum hydroxide or lipids.





AGE & THE IMMUNE RESPONSE

Immunity is less than optimal at both ends of life (i.e., in the newborn and the elderly). Newborns appear to have less effective T-cell function than do adults. In newborns, antibodies are provided primarily by the transfer of maternal IgG across the placenta. Because maternal antibody decays over time (little remains by 3–6 months of age), the risk of infection in the child is high. Colostrum also contains antibodies, especially secretory IgA, which can protect the newborn against various respiratory and intestinal infections.

The response to protein antigens is usually good; hence hepatitis B vaccine can be given at birth and poliovirus immunization can begin at 2 months of age. However, young children respond poorly to polysaccharide antigens unless they are conjugated to a carrier protein.

In the elderly, immunity generally declines. There is a reduced IgG response to certain antigens, fewer T cells, and a reduced delayed hypersensitivity response. As in the very young, the frequency and severity of infections are high.

The frequency of autoimmune diseases is also high in the elderly, possibly because of a decline in the number of regulatory T cells, which allows autoreactive T cells to proliferate and cause disease.

COMPLEMENT

The complement system consists of approximately 20 proteins that are present innormal human (and other animal) serum. The term *complement* refers to the abilityof these proteins to complement (i.e., augment) the effects of other components of the immune system (e.g., antibody). Complement is an important component of our innate host defenses.

There are three main effects of complement:

(1) lysis of cells such as bacteria,allografts, and tumor cells;

(2) generation of mediators that participate in inflammation and attract neutrophils; and

(3) opsonization (i.e., enhancement of phagocytosis). Complement proteins are synthesized mainly by the liver.

يتكون النظام التكميلي من حوالي 20 بروتينًا موجودًا في مصل الإنسان غير الطبيعي (والحيواني الآخر). يشير مصطلح مكمل إلى قدرة هذه البروتينات على استكمال (أي زيادة) تأثيرات المكونات الأخرى لجهاز المناعة (مثل الجسم المضاد). التكملة عنصر مهم في دفاعات مضيفنا الفطرية. هناك ثلاثة تأثيرات رئيسية للمكملات: (1) تحلل الخلايا مثل البكتيريا والطعوم الخيفية والخلايا السرطانية. (2) جيل من الوسطاء التي تشارك في الالتهاب وتجذب العدلات. و (3) طهارة (أي تعزيز البلعمة). يتم تصنيع البروتينات المكملة بشكل رئيسي عن طريق الكبد.

Complement activation

Several complement components are proenzymes, which must be cleaved to form active enzymes. Activation of the complement system can be initiated either by antigen-antibody complexes or by a variety of non immunologic molecules (e.g.,endotoxin).

Sequential activation of complement components occurs via one of three pathways: the classic pathway, the lectin pathway, and the alternative pathway. Of these pathways, the lectin and the alternative pathways are more important the first time we are infected by a microorganism because the antibody required to trigger the classic pathway is not present. The lectin pathway and the alternative pathway are, therefore, participants in the innate arm of the immune system.

العديد من المكونات التكميلية عبارة عن إنزيمات طليعية ، والتي يجب شقها لتكوين إنزيمات نشطة. يمكن أن يبدأ تنشيط النظام المتمم إما عن طريق معقدات مستضد - جسم مضاد أو عن طريق مجموعة متنوعة من الجزيئات غير المناعية (على سبيل المثال ، الذيفان الداخلي). يحدث التنشيط المتسلسل للمكونات التكميلية عبر أحد المسارات الثلاثة: المسار الكلاسيكي ، ومسار المحاضرة ، والمسار البديل. من بين هذه المسارات ، يكون المحاضرة والمسارات البديلة أكثر أهمية في المرة الأولى التي نصاب فيها بكائن حي مجهري لأن الجسم المضاد المطلوب لتحريك المسار الكلاسيكي غير موجود. وبالتالي ، فإن مسار الليكتين والمسار البديل هما مشاركان في الذراع الفطرية لجهاز المناعة.

All three pathways lead to the production of **C3b, the central molecule** of the complement cascade. The presence of C3b on the surface of a microbe marks it as foreign and targets it for destruction. C3b has two important functions:

(1) It combines with other complement components to generate C5 convertase, the enzyme that leads to the production of the membrane attack complex; and (2) it opsonizes bacteria because phagocytes have receptors for C3b on their surface.

على سطح C3b ، الجزيء المركزي في السلسلة التكميلية. إن وجود C3b تؤدي المسارات الثلاثة جميعها إلى إنتاج C5 له وظيفتان مهمتان: (1) يتحد مع مكونات مكملة أخرى لتوليد C3b .الميكروب يجعله غريبًا ويستهدفه للتدمير ، الإنزيم الذي يؤدي إلى إنتاج مركب هجوم الغشاء ؛ و (2) يطهر البكتيريا لأن البالعات لديها مستقبلات لـ convertase على سطحها C3b.

(1) In the **classic** pathway, antigen‑antibody complexes1 activate C12 to form a protease, which cleaves C2 and C4 to form a C4b,2b complex. The latter is C3 convertase, which cleaves C3 molecules into two fragments, C3a and C3b. C3a, an **anaphylatoxin,** is discussed later. C3b forms a complex with C4b,2b, producing a new enzyme, C5 convertase(C4b,2b,3b), which cleaves C5 to form C5a and C5b. C5a is an anaphylatoxin and a chemotactic factor . C5b binds to C6 and C7 to form a complex that interacts with C8 and C9 to produce the **membrane attack** complex (C5b,6,7,8,9), which causes cytolysis. Note that the "b" fragment continues in the main pathway, whereas the "a" fragment is split off and has other activities.

(1)في المسار الكلاسيكي ، تعمل مجمعات المستضد والأجسام المضادة 1 على تنشيط C12 لتشكيل بروتياز ، والذي يشق C2 و C4 لتكوين مركب C4b ، 2 .bالأخير هو C3 convertase ، الذي يشق جزيئات C3 إلى جزأين ، C3aو .C3b تمت مناقشة C3a ، anaphylatoxin، لاحقًا. يشكل C3b مركبًا مع C4b ، 2 b، ينتج إنزيمًا جديدًا ، (C4b C5 convertase، 2 b، 3 (b، والذي يشق C5 ليشكل C5a و C5a .C5b هو عامل تأقلي وعامل كيماوي. يرتبط C5b بـ C6 و C7 لتشكيل معقد يتفاعل مع C8 و C9 لإنتاج مركب هجوم الغشاء (C5b ، 6 ، 7 ، 8 ، 9 (، والذي يسبب انحلال الخلايا. لاحظ أن الجزء "b" يستمر في المسار الرئيسي ، بينما يتم تقسيم الجزء "a" وله أنشطة أخرى

(2) In the **lectin** pathway, mannan-binding lectin (MBL) (also known as mannose binding protein) binds to the surface of microbes bearing mannan (a polymer of the sugar, mannose). This activates proteases associated with MBL that cleave C2 and C4 components of complement and activate the classic pathway. Note that this process bypasses the antibody-requiring step and so is protective early in infection before antibody is formed.

(3) In the **alternative** pathway, many unrelated cell surface substances (e.g.,bacterial lipopolysaccharides [endotoxin], fungal cell walls, and viral envelopes)can initiate the process by binding C3(H2O) and factor B. This complex is cleavedby a protease, factor D, to produce C3b, Bb. This acts as a C3 convertase togenerate more C3b.

(2)في مسار الليكتين ، يرتبط الليكتين المرتبط بالمانان) (MBL) المعروف أيضًا باسم بروتين ربط المانوز) بسطح الميكروبات التي تحمل المنان (بوليمر السكر ، المانوز). هذا ينشط البروتياز المرتبط بـ MBL الذي يشق مكونات C2 و C4للمكملات وينشط المسار الكلاسيكي. لاحظ أن هذه العملية تتجاوز الخطوة التي تتطلب الجسم المضاد وبالتالي فهي وقائية في وقت مبكر من العدوى قبل تكوين الجسم المضاد. (3) في المسار البديل ، يمكن للعديد من مواد سطح الخلية غير المرتبطة (على سبيل المثال ، عديدات السكاريد الدهنية البكتيرية [الذيفان الداخلي] ، وجدران الخلايا الفطرية ، والمغلفات الفيروسية) بدء العملية عن طريق ربط C3 (H2O) والعامل ب. عامل د لانتاجC3b ، .Bbيعمل هذا كمحول C3لتوليد المزيد من.C3b

BIOLOGIC EFFECTS OF COMPLEMENT

1.Opsonization

Microbes, such as bacteria and viruses, are phagocytized much better in the presence of C3b because there are C3b receptors on the surface of many phagocytes.

2.Chemotaxis

C5a and the C5,6,7 complex attract neutrophils. They migrate especially well toward C5a. C5a also enhances the adhesiveness of neutrophils to the endothelium.

3.Anaphylatoxin

C3a, C4a, and C5a cause degranulation of mast cells with release of mediators (e.g.,histamine), leading to increased vascular permeability and smooth muscle contraction, especially contraction of the bronchioles leading to bronchospasm.Anaphylatoxins can also bind directly to smooth muscle cells of the bronchioles and cause bronchospasm. C5a is, by far, the most potent of these anaphylatoxins.Anaphylaxis caused by these complement components is less common than anaphylaxis caused by type I (IgE-mediated) hypersensitivity .

-1تبلعم الميكروبات ، مثل البكتيريا والفيروسات ، بشكل أفضل بكثير في وجود C3b لأن هناك مستقبلات C3b على سطح العديد من البالعات. 2- يجذب المحور الكيميائي C5a والمركب C5،6،7 العدلات. يهاجرون بشكل جيد نحو .C5a يعزز C5a أيضًا التصاق العدلات بالبطانة. 3- يسبب Anaphylatoxin C3a و C4a و C5a تحلل الخلايا البدينة مع إطلاق الوسطاء (على سبيل المثال ، الهيستامين) ، مما يؤدي إلى زيادة نفاذية الأوعية الدموية وتقلص العضلات الملساء ، وخاصة تقلص القصيبات مما يؤدي إلى تشنج قصبي. وتسبب خلايا عضلات القصيبات تشنج قصبي. يعتبر C5a ، إلى حد بعيد ، أقوى أنواع التأقيدات هذه. الحساسية المفرطة التي تسببها هذه المكونات التكميلية أقل شيوعًا من الحساسية المفرطة الناتجة عن فرط الحساسية من النوع الأول(IgE)

4.Cytolysis

Insertion of the C5b,6,7,8,9 complex into the cell membrane forms a “pore” in the membrane. This opening in the membrane results in the killing (lysis) of many types of cells, including erythrocytes, bacteria, and tumor cells. Cytolysis is not an enzymatic process;

rather, it appears that insertion of the complex results in disruption of the membrane and the entry of water and electrolytes into the cell.

5.Enhancement of Antibody Production

The binding of C3b to its receptors on the surface of activated B cells greatly enhances antibody production compared with that by B cells that are activated by antigen alone.

.4انحلال الخلايا يشكل إدخال مركب C5b ، 6،7،8،9 في غشاء الخلية "مسامًا" في الغشاء. ينتج عن هذا الفتح في الغشاء قتل (تحلل) العديد من أنواع الخلايا ، بما في ذلك خلايا الدم الحمراء والبكتيريا وخلايا الورم. التحلل الخلوي ليس عملية إنزيمية. بدلاً من ذلك ، يبدو أن إدخال المركب يؤدي إلى تمزق الغشاء ودخول الماء والإلكتروليتات إلى الخلية. 5- تعزيز إنتاج الجسم المضاد: إن ارتباط C3b بمستقبلاته على سطح الخلايا البائية المنشطة يعزز بشكل كبير إنتاج الجسم المضاد مقارنة بالخلايا البائية التي يتم تنشيطها بواسطة المستضد وحده.

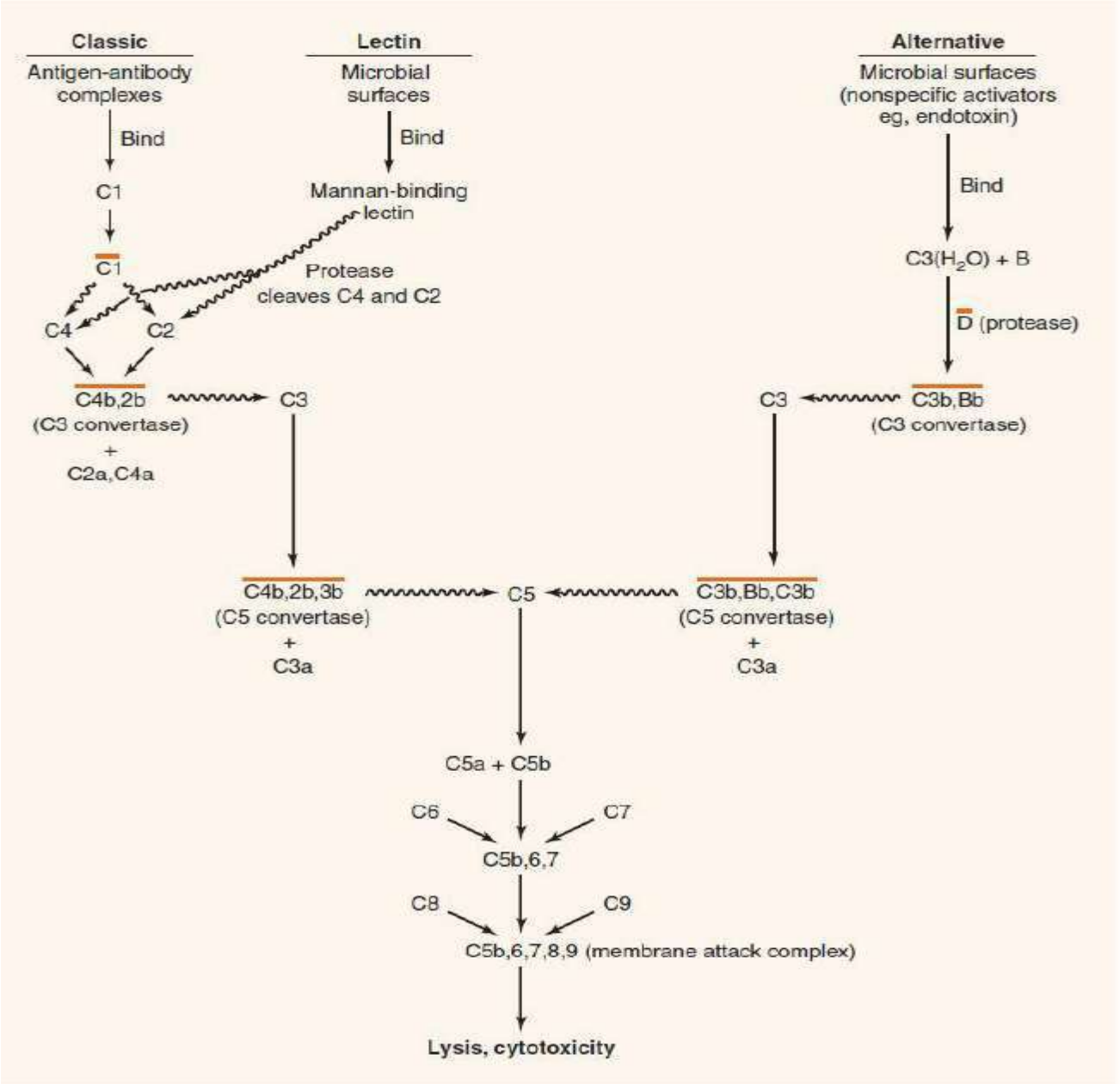


FIGURE 63–1 The classic and alternative pathways of the complement system.