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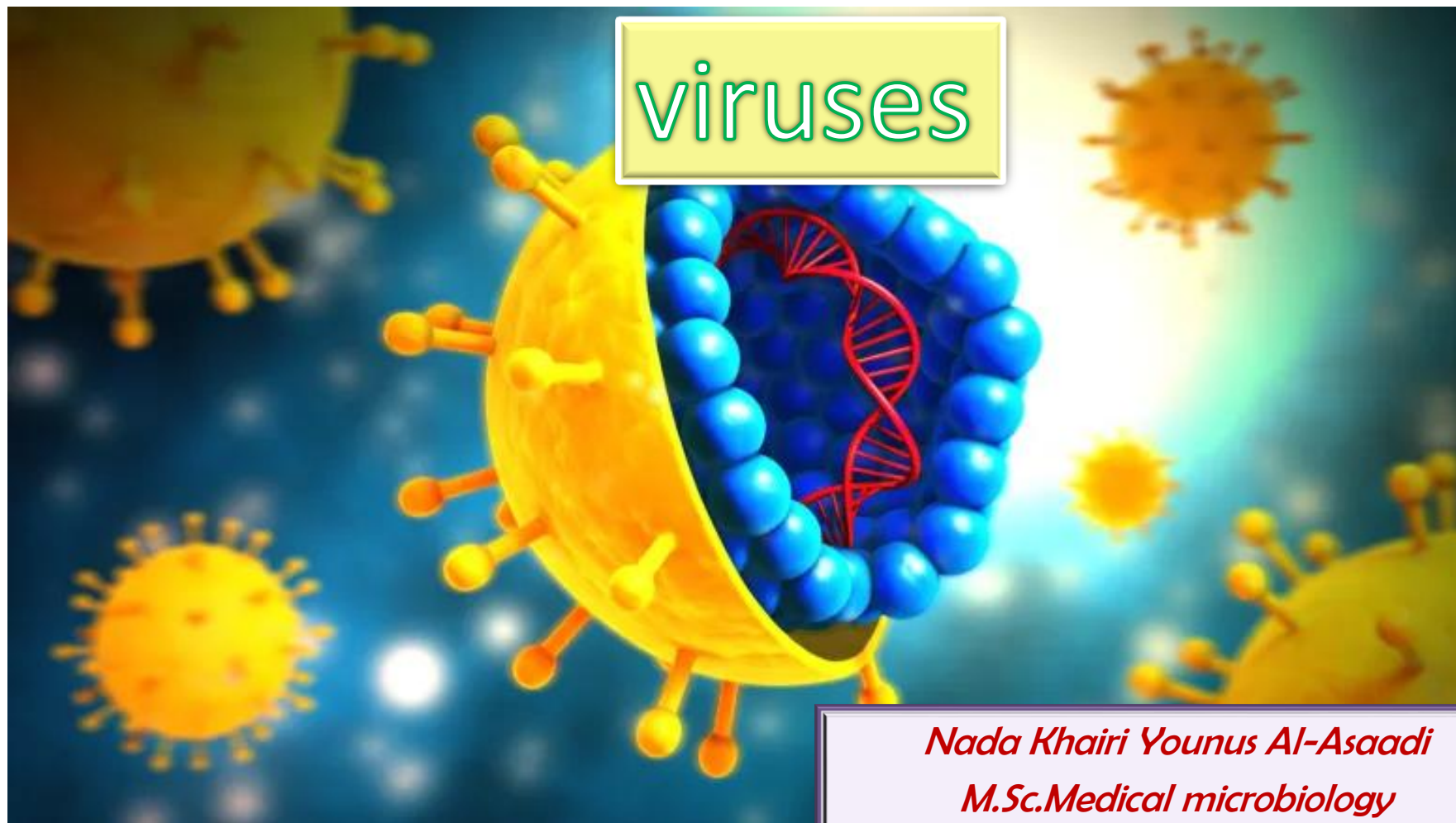
Pharmacy Department

قسم الصيدلة

Medical Microbiology II



viruses



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M.Sc. Medical microbiology

Introduction

A virus can be simply defined as an obligate intracellular parasite.

Each **viral particle**, or **virion**, consists of a single nucleic acid, RNA **or** DNA, encoding the viral genome surrounded by a protein coat, and is capable of replication only within the living cells of bacteria, animals or plants.

Viruses are classified into different orders and families by consideration of the type of nucleic acid present (RNA or DNA), whether the nucleic acid is single- or double-stranded, and the presence or absence of an envelope.

Structure: A typical virus consists of a protective protein coat, known as a **capsid**.

The capsid shape varies from simple helical and icosahedra forms to more complex structures with tails. The capsid provides protection for the viral genome against the environment and functions in receptor recognition, targeting the virus to a susceptible host and cell type.

Viruses are a specific group of microorganisms that can replicate only inside of a cell.

These are extremely small organisms and visible only under an electron microscope. Viral infections can occur in all types of life forms including plants, animals and other microorganisms (bacteria).

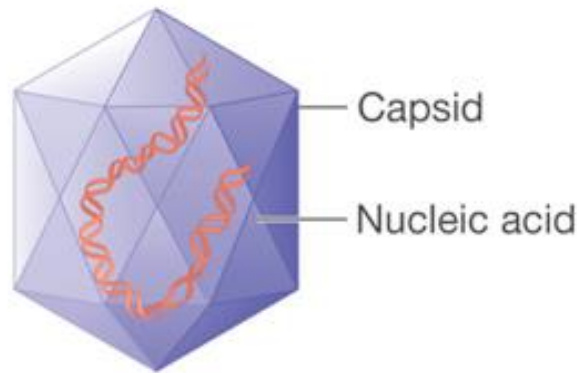
A virus generally contains DNA or RNA as a genome which is protected by a viral protein coat. Some enzymes are often attached to the genome of the virus.

A complete viral particle is known as a **virion**. At the time of infection, viruses insert their genome inside of a host cell, which is then incorporated in the host genome. As a result, the host genome codes the viral proteins important for viral replication.

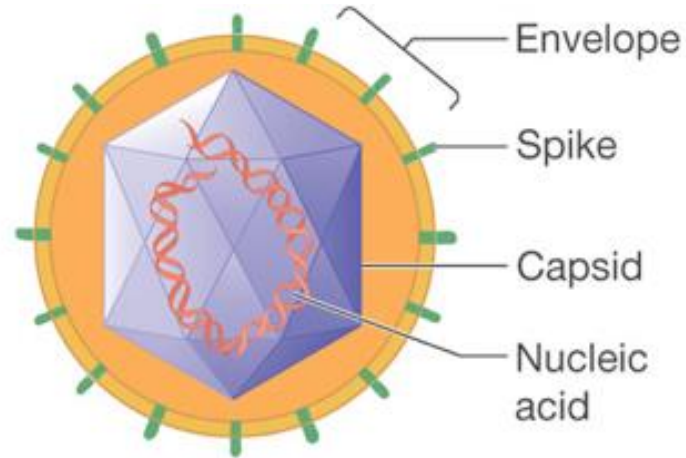
Some viruses have a phospholipid envelope, derived from the infected host's cell membrane, that surrounds the protein capsid.

Inserted into the lipid envelope there are usually viral encoded proteins known as **spike projections** – these are typically glycoproteins and are also involved in receptor recognition.

Virus structure



(a) Naked virus

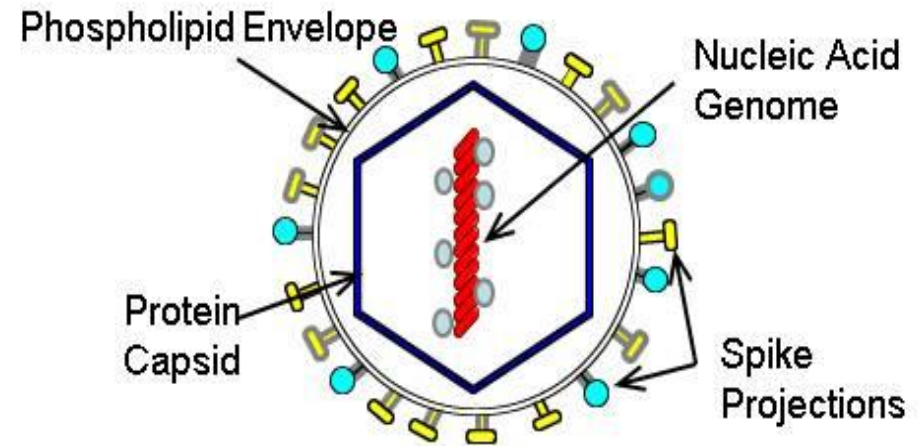


(b) Enveloped virus

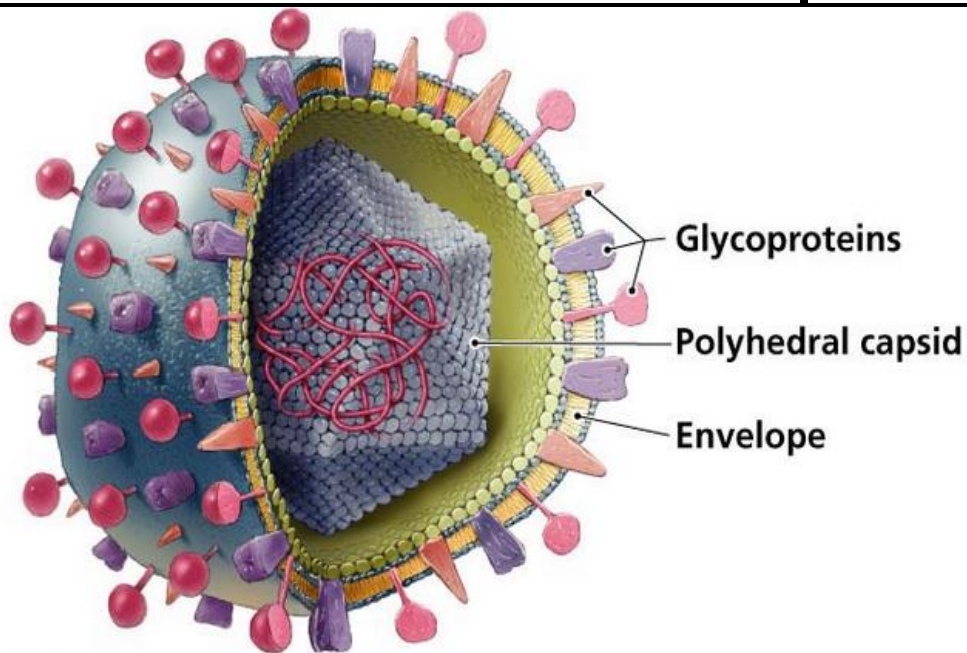
Viruses consist of:

- 1- A nucleic acid genome either DNA or RNA.
- 2- A protein coat (capsid) that enclosed the genome.
- 3- In some types a lipid membrane enveloped virus particle.

The structure of the virus



A typical enveloped virus



(b) Enveloped virus with polyhedral capsid

Varicella- zoster virus

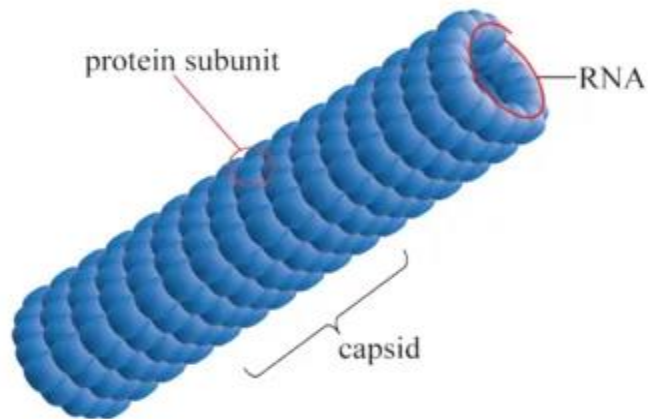
Viral shape structure

Nucleocapsid is the arrangement between the viral nucleic acid genome with the capsid, this connection controlled by specific NA genetic information leading to different types of symmetry.

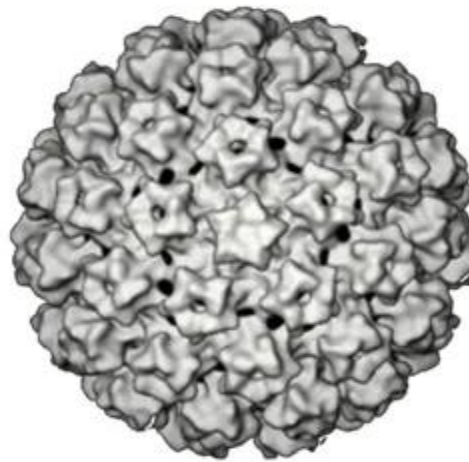
Accordingly, viruses can be classified into four symmetry structures:

- 1- Helical symmetry
- 2- Cubical symmetry
- 3- Binal symmetry
- 4- Complex symmetry

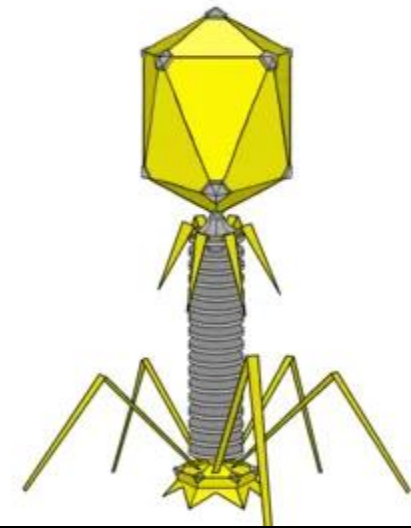
Helical



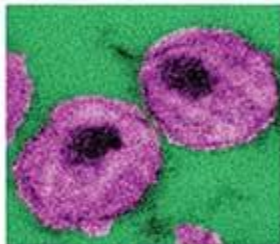
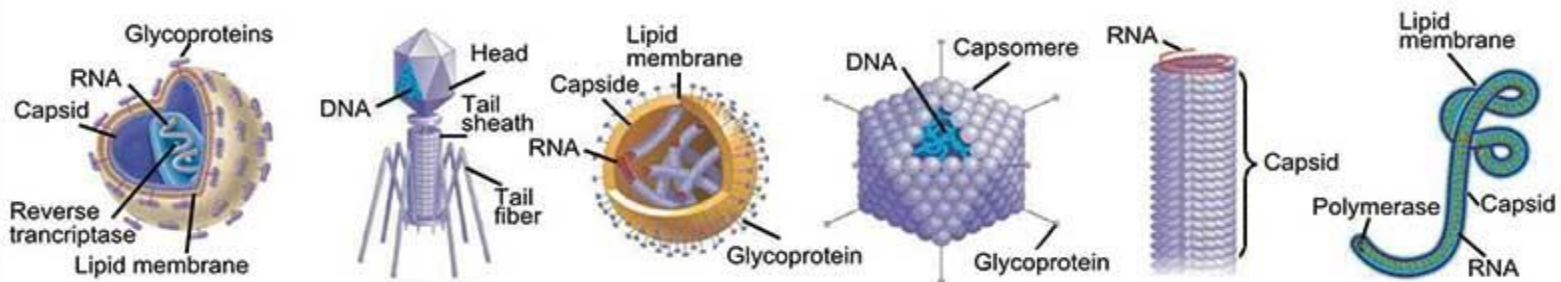
Icosahedral



Complex



Virus Classification on the basis of morphology and replication



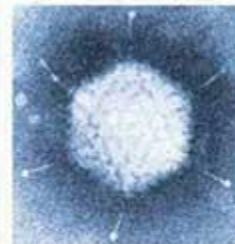
Human immunodeficiency virus



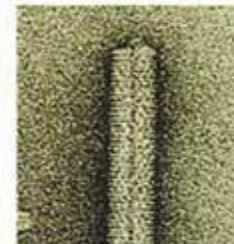
Bacteriophage



Influenza virus



Adenovirus



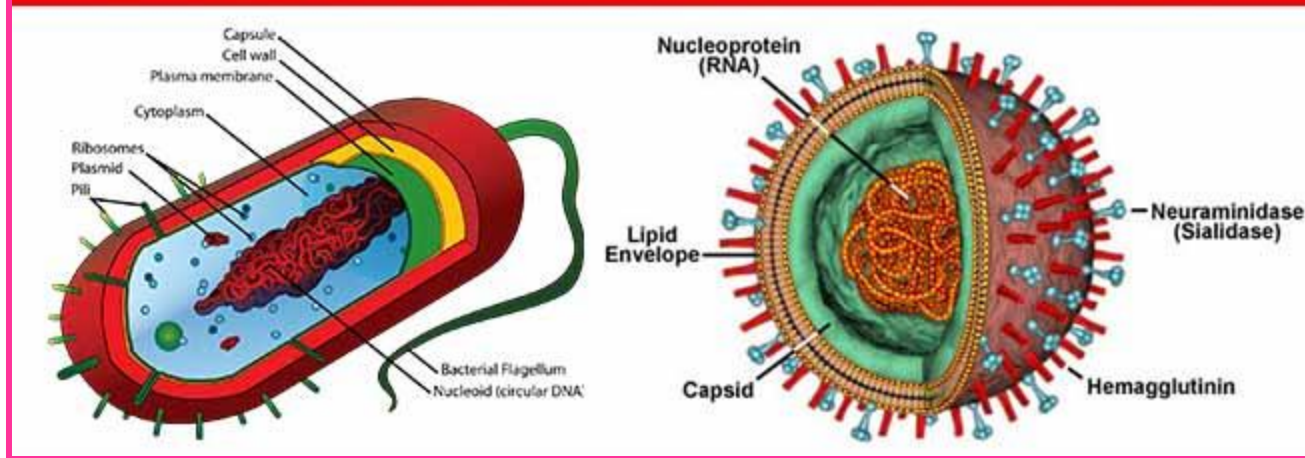
Tobacco mosaic virus



Ebola virus

What are the main differences between bacteria and viruses?

Differences Between Bacteria and Viruses

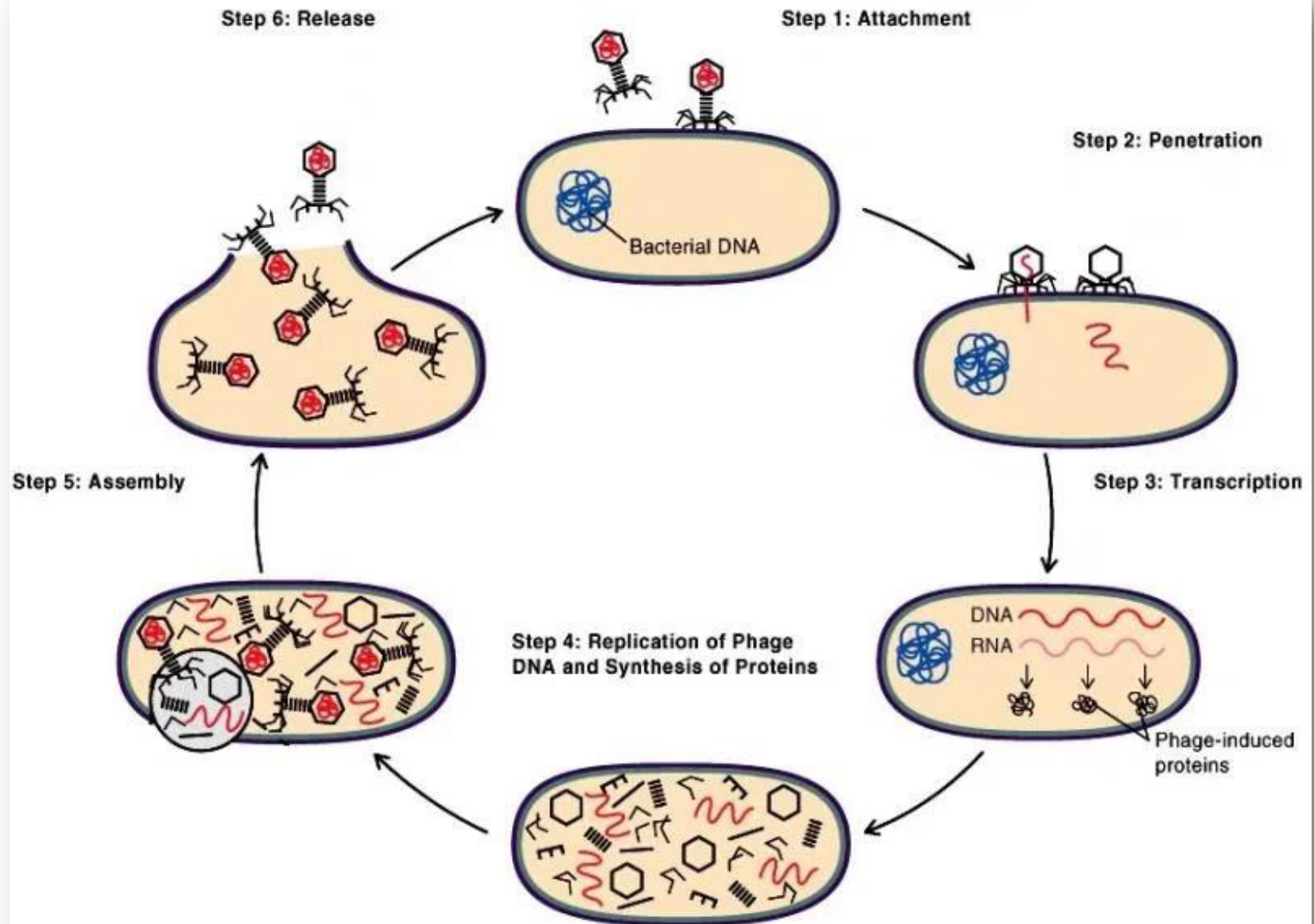


	<u>Characteristics</u>	<u>Bacteria</u>	<u>Viruses</u>
1	Size	Larger (1000 nm)	Smaller (20-400 nm)
2	Cell Wall	Peptidoglycan or Lipopolysaccharide	No cell wall. Protein coat present instead.
3	Ribosomes	Present	Absent
4	Number of cells	One cell (Unicellular)	No cells
5	Living/Non-Living	Living organisms	Between living and non-living things.
6	DNA and RNA	DNA and RNA floating freely in cytoplasm.	DNA or RNA enclosed inside a coat of protein.
7	Infection	Localized	Systemic
8	Reproduce	Able to reproduce by itself	Need a living cell to reproduce
9	Reproduction	Fission- a form of asexual reproduction	Invades a host cell and takes over the cell causing it to make copies of the viral DNA/RNA. Destroys the host cell releasing new viruses.
10	Duration of illness	A bacterial illness commonly will last longer than 10 days.	Most viral illnesses last 2 to 10 days.
11	Fever	A bacterial illness causes a fever.	A viral infection may or may not cause a fever.
12	Cellular Machinery	Possesses a cellular machinery	Lack cellular machinery
13	Under Microscope	Visible under Light Microscope.	Visible only under Electron Microscope.
14	Benefits	Some bacteria are beneficial (Normal Flora)	Viruses are not beneficial. However, a particular virus may be able to destroy brain tumors. Viruses can be useful in genetic engineering.
15	Treatment	Antibiotics	Virus does not respond to antibiotics.
16	Examples	<i>Staphylococcus aureus</i> , <i>Vibrio cholerae</i> , etc	HIV, Hepatitis A virus, Rhino Virus, etc
17	Diseases/Infections	Food poisoning, gastritis and ulcers, meningitis, pneumonia, etc	AIDS, common cold, influenza, chickenpox, etc

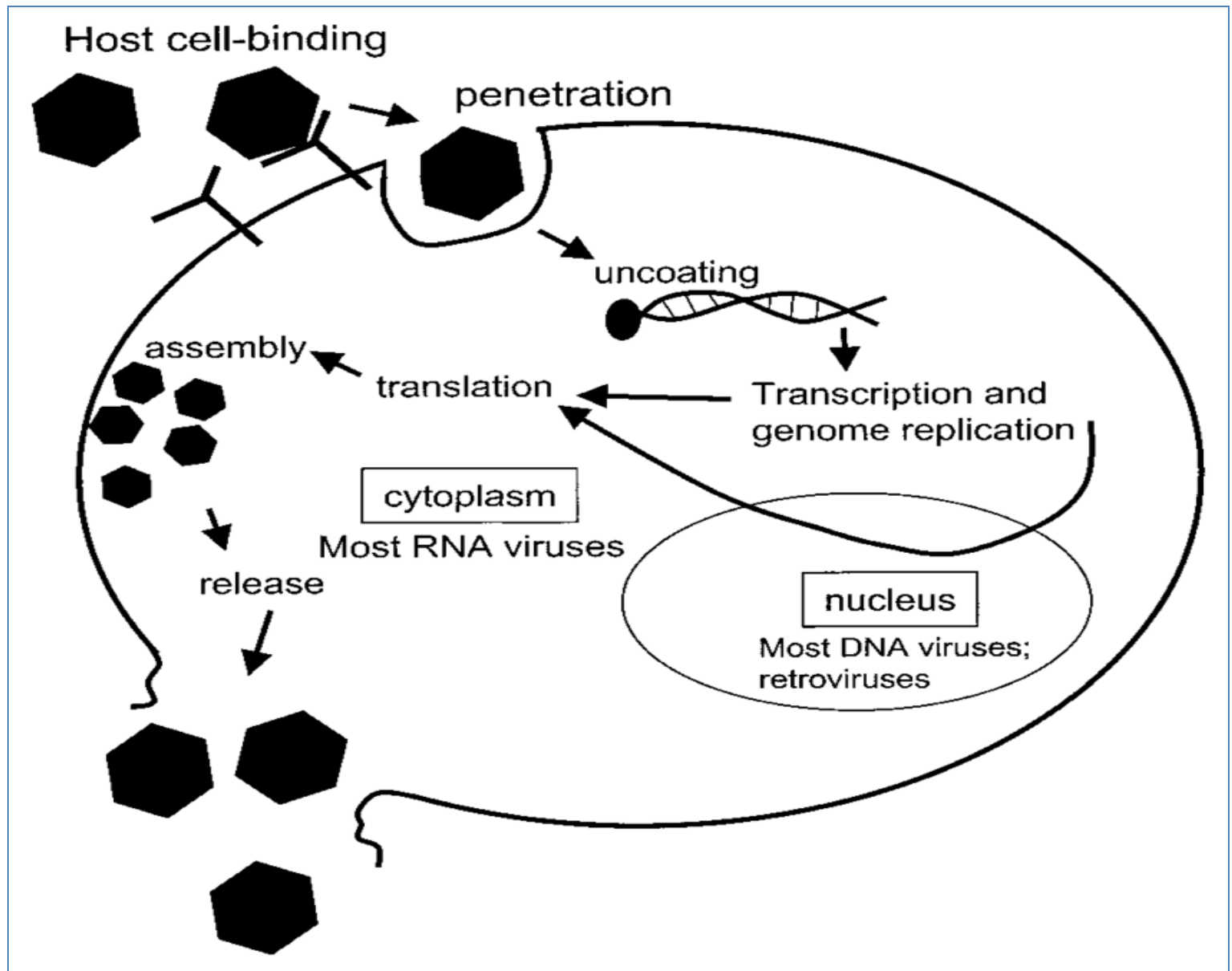
Viral replication

- The basic steps in virus replication are
 - 1- **Adsorption or attachment** of the virus particle to the specific receptors of the host cell plasma membrane.
 - 2- **Penetration or uptake**. The process by which the virus or its genome enters the host cell cytoplasm.
 - 3- **Uncoating and eclipse**. As uncoating proceeds, the viral nucleic acid becomes free to act as a template for the synthesis of virus mRNA.
 - 4- **Transcription**. The virus mRNA codes for the synthesis of enzymes necessary to complete the process of uncoating itself and also to initiate early steps in viral replication.
 - 5- **Synthesis of viral components**. Viral proteins are of two types:
 - a) Structural proteinmake up the virus particles.
 - b) Non-structural protein ...enzymes required for virus genome replication.
 - 6- **Assembly**. May occur in the cell nucleus, cytoplasm, or at the plasma membrane.
 - 7- **Release**. May occur by sudden rupture or through gradual budding.

Bacteriophage replication

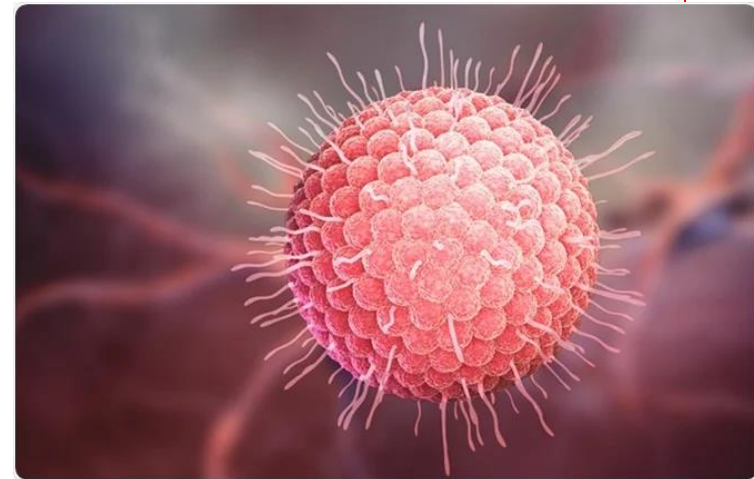
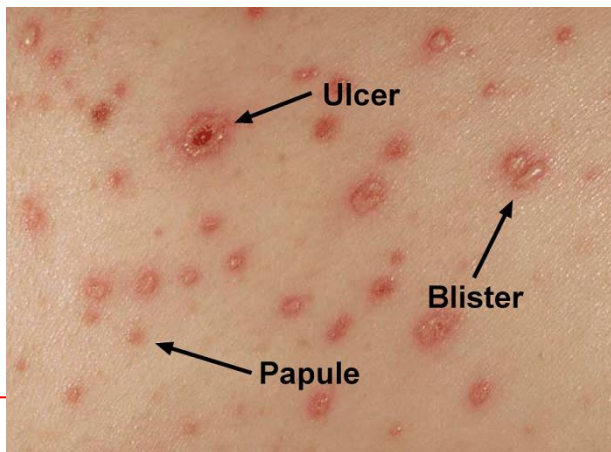


General model of eukaryotic viral replication

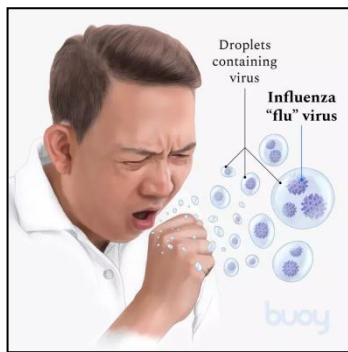


What are viral diseases?

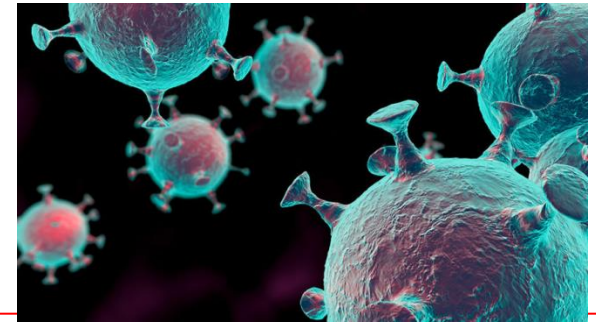
- Chickenpox. Chickenpox, also known as varicella, is a highly contagious disease caused by the initial infection with varicella zoster virus (VZV). The disease results in a characteristic skin rash that forms small, itchy blisters, which eventually scab over. It usually starts on the chest, back, and face.



varicella-zoster virus (VZV)



Influenza virus



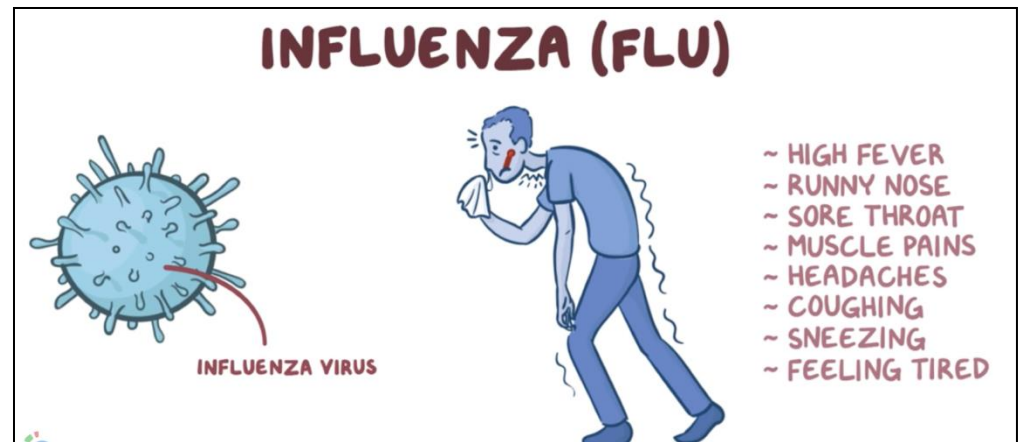
Flu is a contagious respiratory illness caused by influenza viruses that infect the nose, throat, and sometimes the lungs. It can cause mild to severe illness, and at times can lead to death. The best way to prevent flu is by getting a flu vaccine each year.

Flu Symptoms

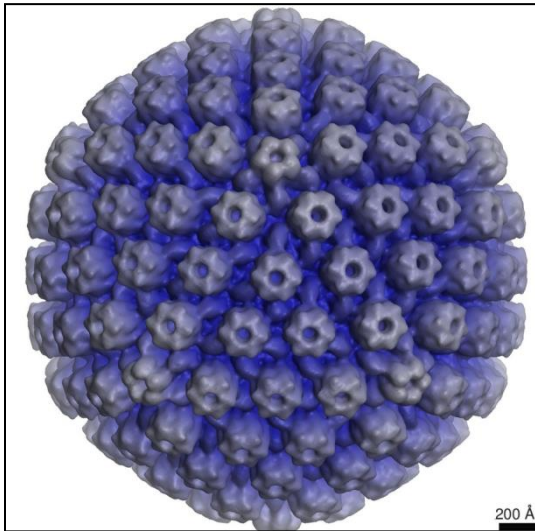
Influenza (flu) can cause mild to severe illness, and at times can lead to death. Flu is different from a cold. Flu usually comes on suddenly. People who have flu often feel some or all of these symptoms:

fever* or feeling feverish/chills
cough
sore throat
runny or stuffy nose
muscle or body aches
headaches
fatigue (tiredness)

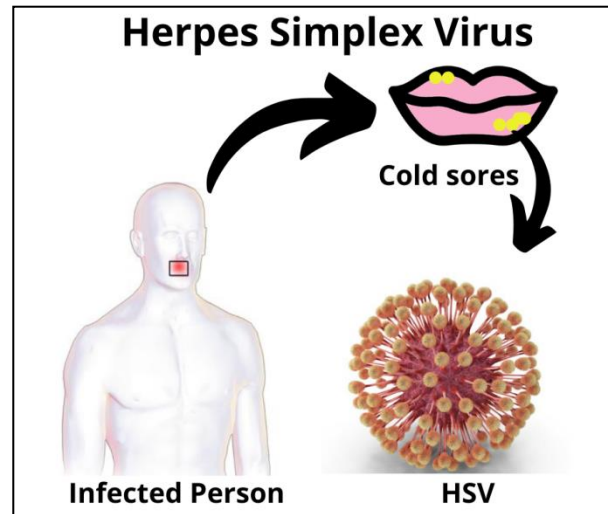
some people may have vomiting and diarrhea, though this is more common in children than adults.



Herpes. Herpes results from infection with the herpes simplex virus (HSV). It causes sores or blisters to form in or around the mouth or genitals, as well as other symptoms. HSV-1 resides in cranial nerve ganglia, generally the trigeminal ganglia, in a latent state and reactivates periodically, causing painful oral and labial ulcerations.



Herpes simplex virus



labial ulcer

Mumps, measles and rubella

Mumps is a viral infection that primarily affects saliva-producing (salivary) glands that are located near your ears. Mumps can cause swelling in one or both of these glands.

Mumps can be serious, but most people with mumps recover completely within two weeks. While infected with mumps, many people feel tired and achy, have a fever, and swollen salivary glands on the side of the face.



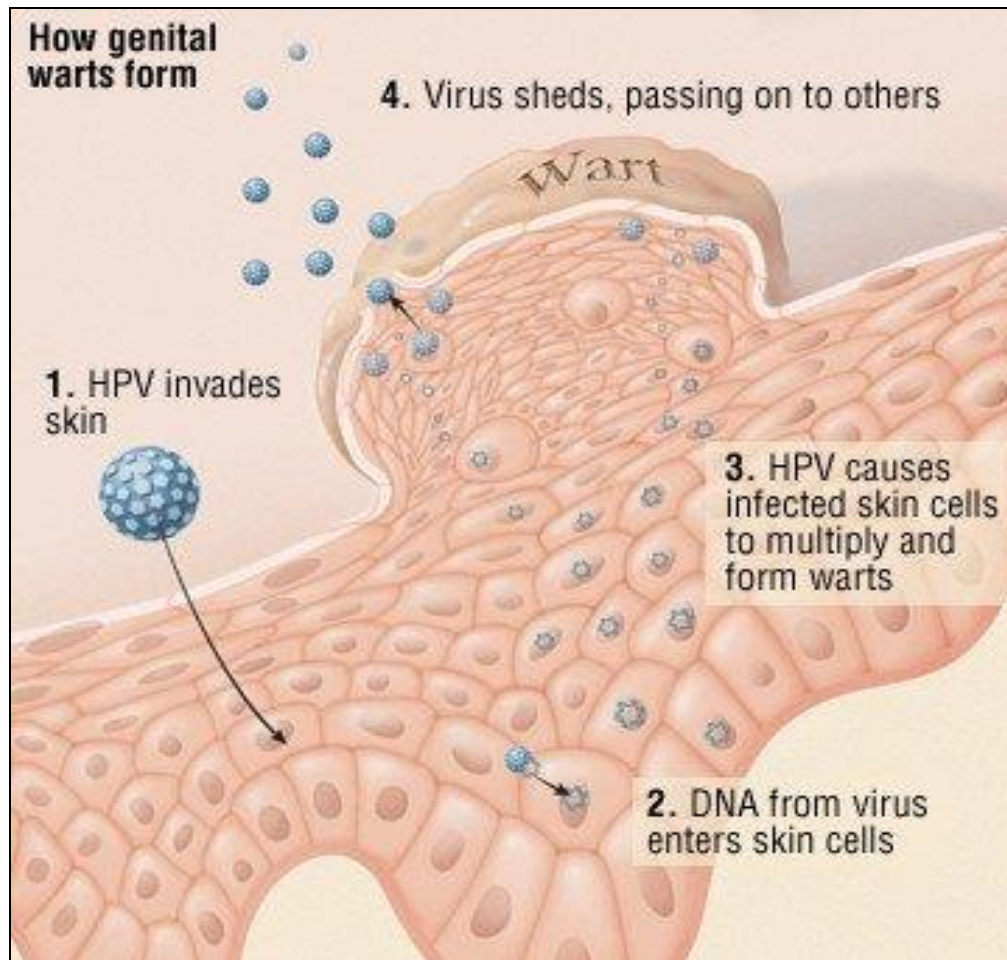
Measles is a highly contagious infectious disease caused by measles virus. Symptoms usually develop 10–12 days after exposure to an infected person and last 7–10 days. Initial symptoms typically include fever, often greater than 40 °C (104 °F), cough, runny nose, red, watery eyes (conjunctivitis), and rash (3-5 days after symptoms begin).



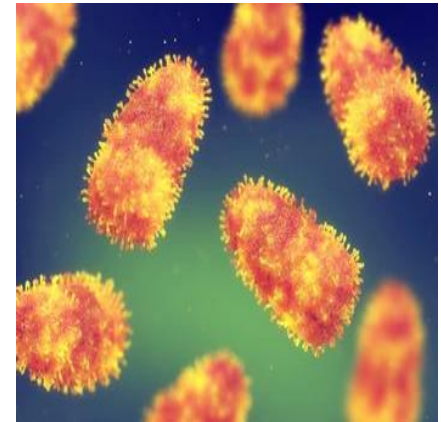
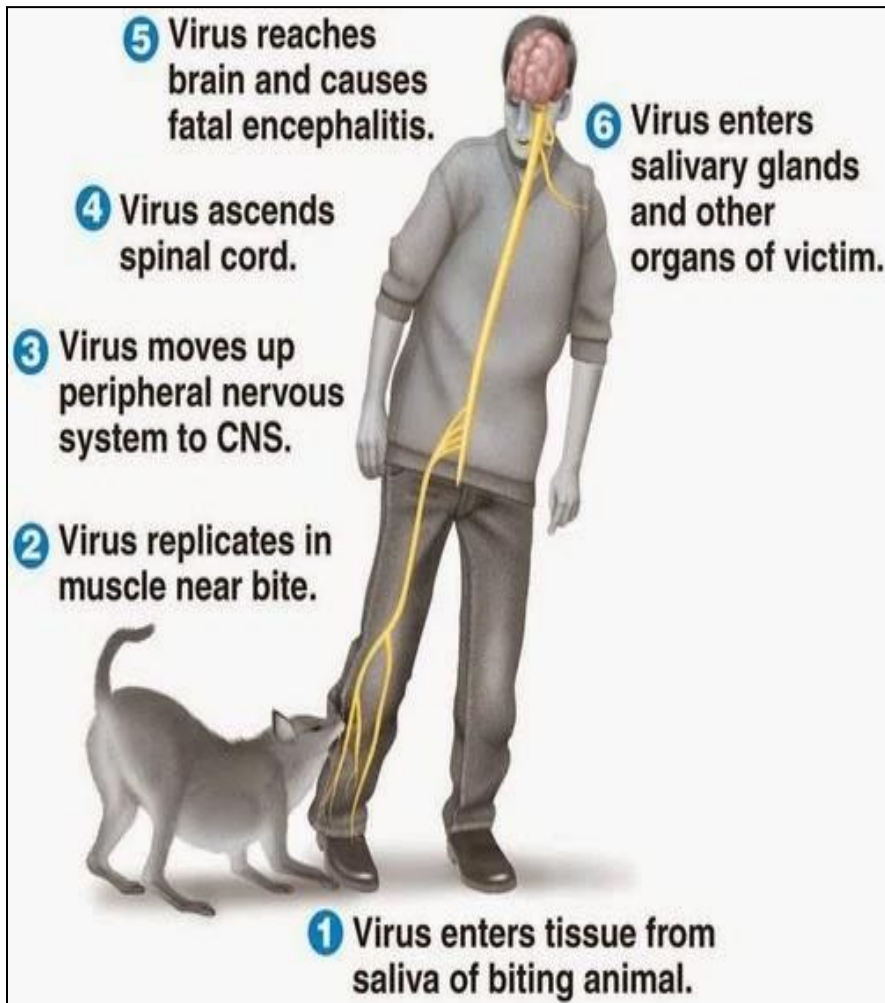
Rubella is caused by a virus that's passed from person to person. It can spread when an infected person coughs or sneezes. It can also spread by direct contact with an infected person's respiratory secretions, such as mucus. It can also be passed on from pregnant women to their unborn children via the bloodstream.



Human papilloma virus (HPV) causes common warts, the small, white, beige or brown skin growths that can appear almost anywhere on the body and on the moist mucous membranes near the mouth.



Rabies is a deadly virus spread to people from the saliva of infected animals. The rabies virus is usually transmitted through a bite. Animals most likely to transmit rabies in the United States include bats, coyotes, foxes, raccoons and skunks



Rhabdovirus bullet-shaped

Treatment

Antiviral Chemotherapy

There are three types of antiviral agents:

- (1) **virucidal agents**, which directly inactivate viruses. Virucidal agents are chemical substances that attack and inactivate viral particles outside the cell (virions). In general this is accomplished by damaging their protein shells (capsid) or the substance penetrates the core itself, where it destroys the genetic material..e.g bleach.
- (2) **Antiviral agents**, which inhibit viral replication. ..e.g acyclovir (aciclovir).
- (3) **Immunomodulators**, which boost the host immune response. Immunomodulators are a group of drugs that mainly target the pathways that treat multiple myeloma and a few other cancers. They have many ways to work, including working on the immune system directly by turning down some proteins and turning up others. e.g Alferon N.





Nada.k. younus

Cytokine

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Medical Microbiology II

2023

Introduction

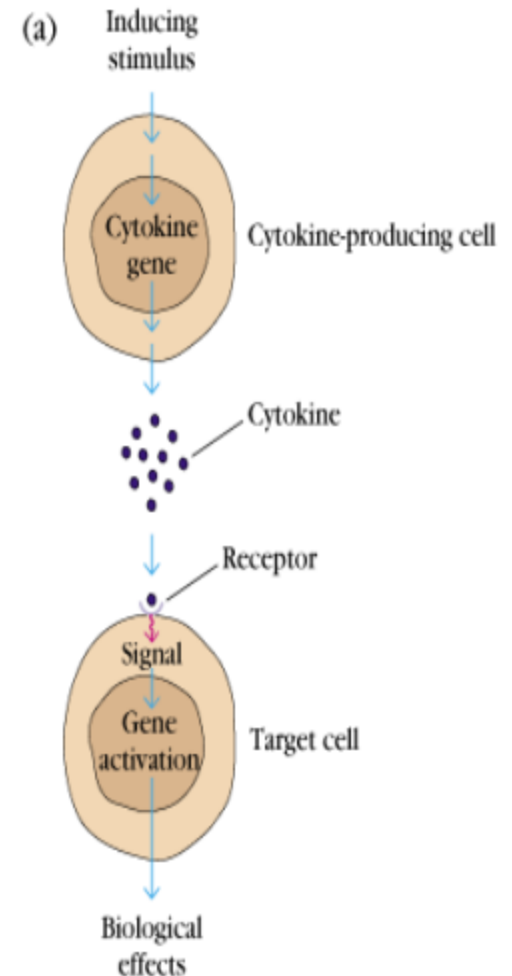
- “Cytokine” is word that comes from cyto- (cell) and –kinin (hormone).
- Cytokines are potent, low-molecular weight protein, cell regulators, produced transiently and locally by numerous cell types, involved in both innate & adaptive immunity
- Play a key role in hematopoiesis, immunity, infectious disease, tumorigenesis, homeostasis, tissue repair, and cellular development and growth.
- Their effects are mediated by binding to specific receptors on target cells. Thus, cytokines are like other hormones in that their effects are mediated through receptors that signal target cells respond to.
- Cytokines can be categorized into groups based on their common functions. Examples of functional categories are immunoregulatory, proinflammatory, anti-inflammatory, and growth and differentiation.

Family

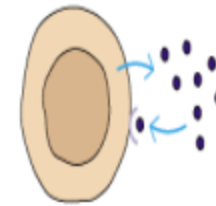
- grouped by structures into families
- Interferon family(IFN): type I (IFN α and IFN β), type II (IFN γ)
- Interleukin family : IL-1, IL-2
- Chemokine family: CXCL and CCL chemokines
- Tumor necrosis factor family(TNF α): TNF α , FasL, CD40L...
- Hematopoietic family: erythropoietin (EPO), colony- stimulation factors (CSF)

Properties of Cytokines

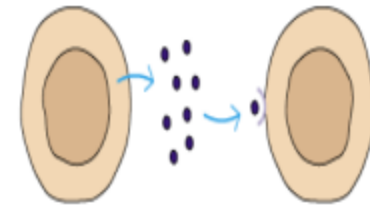
- produced in response to immune stimuli
 - not store pre-formed
 - synthesis: DNA → mRNA → protein → secretion
 - slow cellular response
- Cytokines usually act as signaling molecules by binding to their own glycoprotein receptors on cell membranes. This initial interaction is followed by a relay of the signal to the cell nucleus.
- Signal transduction is mediated as in many hormone-receptor systems via kinase-mediated phosphorylation of cytoplasmic proteins. In fact, tyrosine kinase activity is intrinsic to many cytokine receptors.



- can act on the cells that produce them (**autocrine** action)
- can act on nearby cells (**paracrine** action)
- can act on distance cells (**endocrine** action)

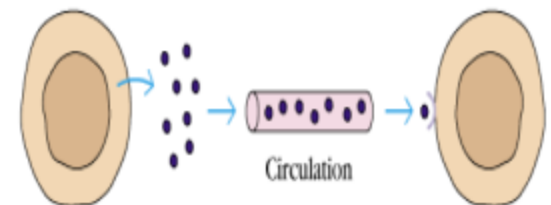


Autocrine action



Paracrine action

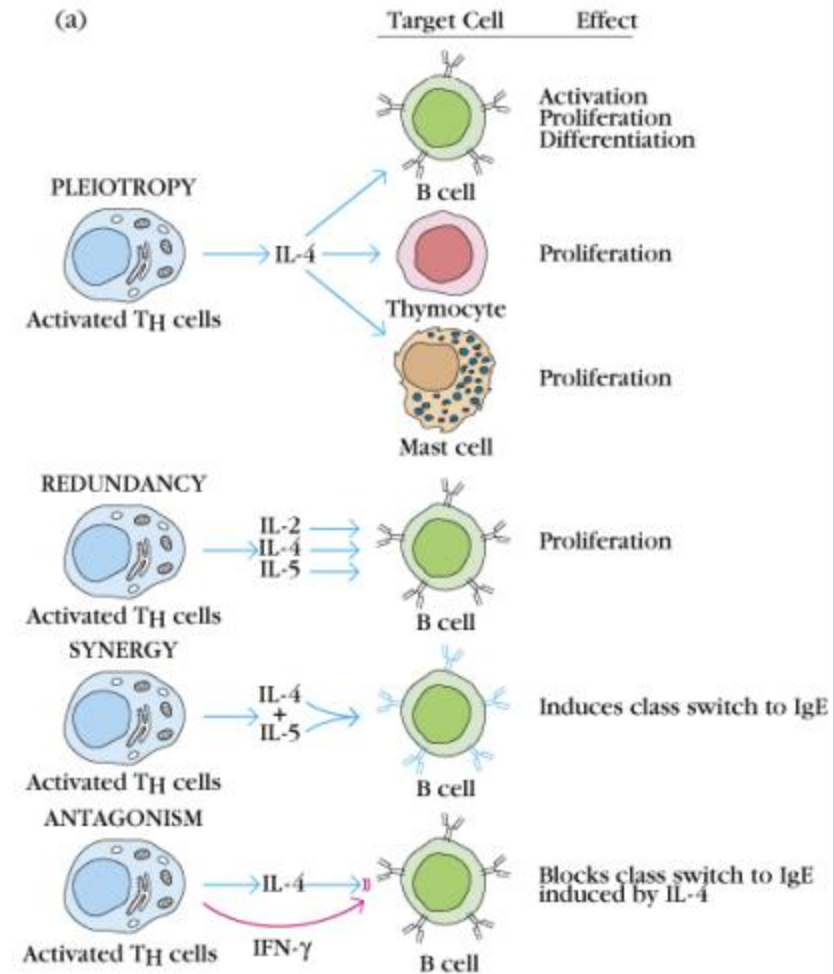
Nearby cell



Endocrine action

Distant cell

- can be produced by many cell types and act on many cell types (**pleiotropic**)
- different cytokines can have similar actions (**redundant**)



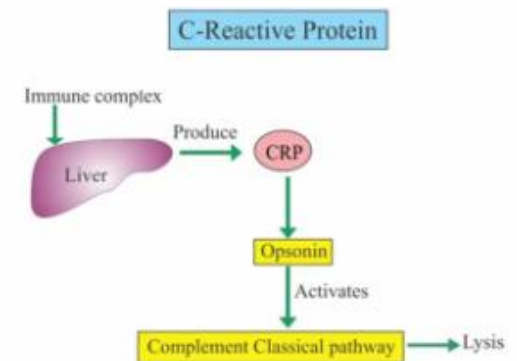
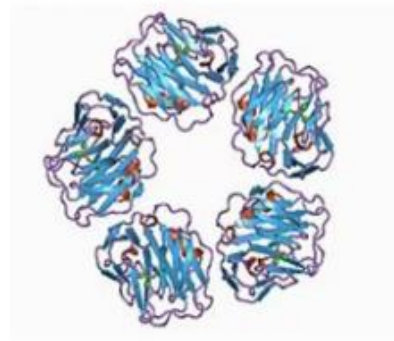
Cytokines and Immunoregulation

A) Mediators of Innate Immunity

- $\text{TNF}\alpha$
- IL-1
- IL-10
- IL-12
- $\text{IFN}\alpha$, $\text{IFN}\beta$
- $\text{IFN}\gamma$
- Chemokines

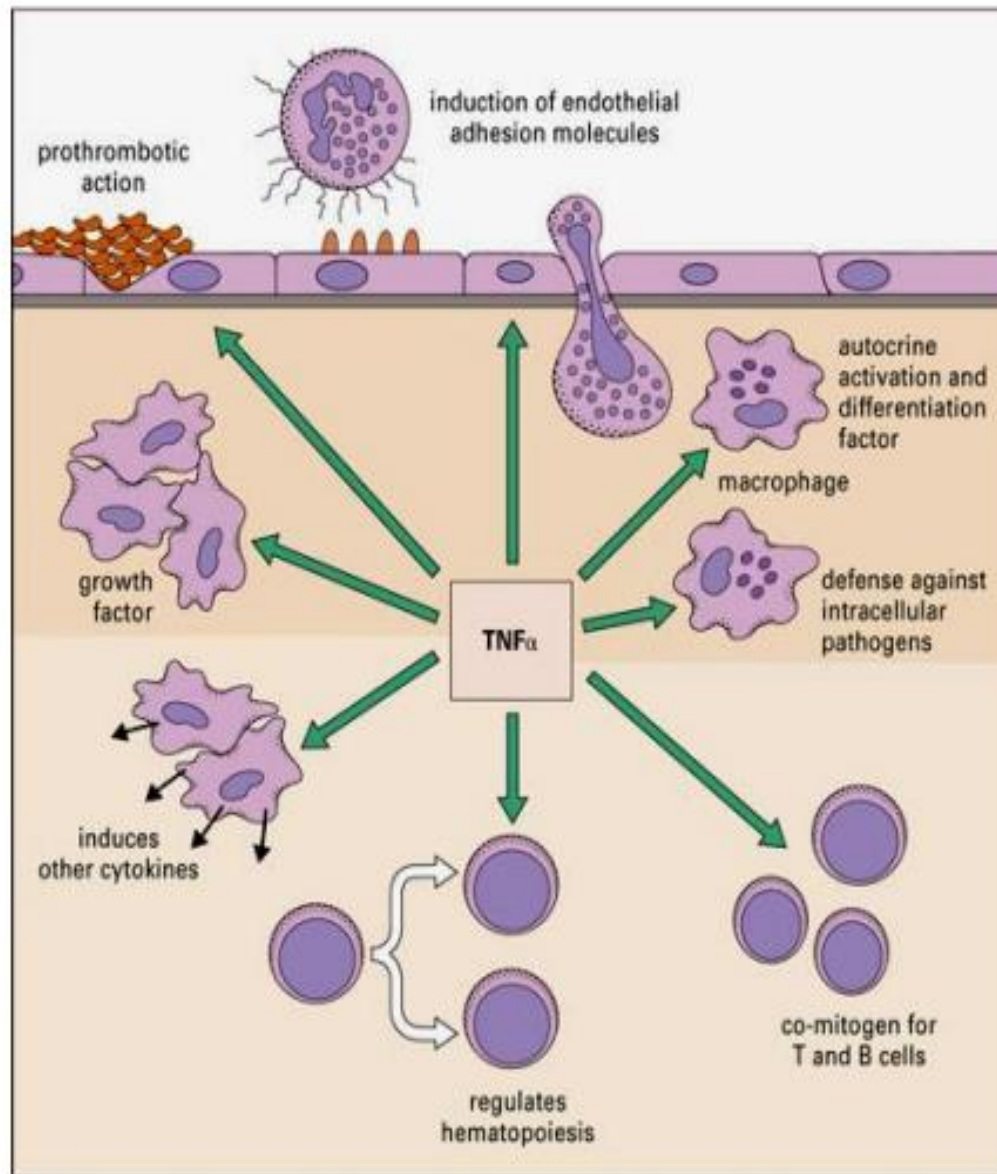
Tumor Necrosis Factor α (TNF α)

- Produced by activated macrophages and T cells
- Most important mediator of acute inflammation in response to microbes, such as LPS
- Induces production of myeloid CSFs, IFN- γ , IL-6, IL-8 and other chemokines
- Mediate recruitment of neutrophils and macrophages to site of inflammation by stimulating cells to produce adhesion molecules
- Stimulates endothelial cells and macrophages to produce chemokines
- A potent pyrogen causing fever by direct action or via IL-1
- Promotes production of acute phase proteins, such as CRP
- Roles in rheumatoid arthritis, psoriasis, tuberculosis, ...



C-Reactive Protein [CRP]

prothrombotic



hematopoiesis

Interleukin 1 (IL-1)

- Produced by activated macrophages, stimulated lymphocytes, keratinocytes, fibroblasts
- Helps activate T cells
- Can be induced by inflammation, injury, and infection

Interleukin 10 (IL-10)

- Produced by macrophages, B cells, Th2 cells
- Originally identified as cytokine synthesis inhibitory factor
- **Suppresses inflammatory responses**
- Inhibits production of IFN-g, IL-2, IL-3, TNF α , GM-CSF
- Stimulate thymocytes, mast cells, B cells
- Inhibits expression of class II MHC and co-stimulatory molecules on macrophages

Interleukin 12 (IL-12)

- Produced by macrophages, dendritic cells, Tc cells, NK cells
- Belongs to the IL-6 cytokine family
- Has immunoregulatory effect on NK cells and T cells
- Stimulates production of IFN-g
- Promotes Th cells → Th1
- Enhances differentiation of Cytotoxic T Lymphocytes (with IL-2)
- Enhances cytolytic functions of T cells and NK cells

Type I Interferon (IFN- γ , IFN- β)

- Produced by macrophages and virus-infected cells
- Inhibits viral replication in cells
- Increases expression of MHC I and Tc mobilization
- Stimulates production of IFN-g by activated T cells
- Activate NK cells

Type II Interferon (IFN- γ)

- Produced primarily by Th1
- Activate NK cells
- Increase MHC I and MHC II expression to help Th cell and APC interaction
- Promotes B cell differentiation to plasma cell
- Promotes cytotoxic T cell differentiation

Chemokines

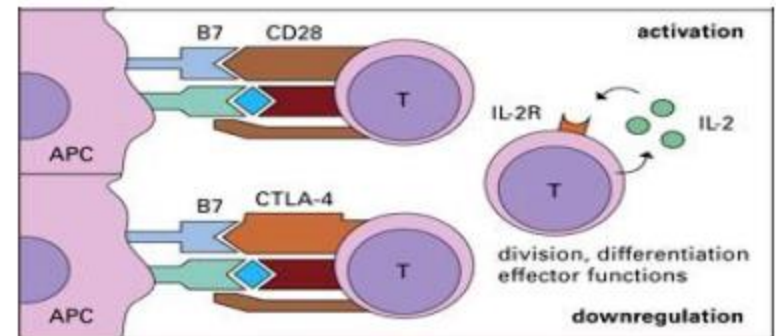
- Produced by many leukocytes and other types of cells
- Large family of molecules (over 50)
- Have significant structural homology and overlapping functions
- Chemotactic for leukocytes, such as PMN, T and B cells
- Recruit leukocytes to sites of infection and inflammation
- Involved in lymphocytes trafficking, wound healing, metastasis, angiogenesis, lymphoid organ development....

B) Mediators of adaptive immunity

- IL-2
- IL-4
- IL-5
- TGF- β
- IL-10
- IL-12
- IFN-g

Interleukin 2 (IL-2)

- Produced by antigen-activated Th cells
- Powerfully immunoregulatory lymphokine
- Main growth factor for both T and B lymphocytes
- Activates NK cells and monocytes



Interleukin 4 (IL-4)

- Produced by macrophages, Th2 cells, activated B cells
- Has complex biological actions via cytokine production
- Enhances antigen-presenting activity of B to T cells
- Stimulates development of Th2 cells
- Stimulates Ig class switch from IgG1 to IgE (allergy)

Interleukin 5 (IL-5)

- Produced by Th2 cells
- Originally identified as a B cell differentiation factor
- Aids in the growth and differentiation of eosinophils and late-developing B cells to plasma cells

Transforming growth factor β (TGF β)

- Produced by T cells, macrophages, other cell types
- 30 members
- Have effect on many cell types
- Have pro- and anti-inflammatory effect
- Inhibits proliferation of T cells and activation of B cells
- Acts on PMNs and endothelial cells to block the effects of pro-inflammatory cytokines

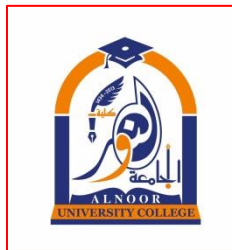
C) Stimulators of hematopoiesis (Colony Stimulating Factors)

- GM-CSF: promotes differentiation of bone marrow progenitors
- M-CSF: promotes growth and differentiation of monocytes and macrophages
- G-CSF: promotes production of PMNs

Clinical Applications

Today there are at least four major clinical applications of cytokines.

- First, cytokines can serve as biomarkers of disease and provide clues for mechanisms of disease. For example, the proinflammatory cytokines TNF- α , IL-1, and IL-6 can be detected in the sera of patients with septic shock.
- Second, the measurement of cytokine production in vitro is a useful monitor of immune status. T-cell function can be monitored by the ability of the T cells to produce IFN- γ . This is currently being used to identify tuberculosis (TB) reactivity.
- Third, recombinant cytokines are key therapeutic agents. An example of this is seen with the IFN molecules. The FDA has approved the use of IFN- α for hepatitis C infections, IFN- β for multiple sclerosis.
- Fourth, cytokines can be targets of therapeutics. Recently, cytokine receptor antagonists and anti-cytokine monoclonal antibodies both which downregulate pathogenic responses to exaggerated cytokine production have been used as effective treatments. Examples of this approach are the inhibitors of TNF- α used to manage rheumatoid arthritis (RA) and inhibitors of IL-2 and IL-15 used in transplantation and cancer.



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Immunology

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Medical Microbiology II

Oncogenic immunity

What is the definition of oncogenic?

- Definition of oncogenic
 - 1 : relating to tumor formation.
 - 2 : tending to cause tumors.

Does oncogenic mean cancer?

The name of oncogene suggests it is a gene that can cause cancer. Initially, oncogenes were identified in viruses, which could cause cancers in animals. Later, it was found that oncogenes can be mutated copies of certain normal cellular genes also called proto-oncogenes.

What are examples of oncogenes?

An example of an oncogene is the HER2 gene that makes HER2 protein. This protein helps control healthy breast cell division and growth. Extra copies of this gene may lead to an excess of HER2 protein, which causes cells to grow more quickly. The HER2 oncogene is found in some breast cancer and ovarian cancer cells.

What viruses are oncogenic?

Oncogenic DNA viruses include EBV, hepatitis B virus (HBV), human papillomavirus (HPV), human herpesvirus-8 (HHV-8), and Merkel cell polyomavirus (MCPyV). Oncogenic RNA viruses include, hepatitis C virus (HCV) and human T-cell lymphotropic virus-1 (HTLV-1).

What cells play a role in stopping cancers?

T-cells work in both direct and indirect ways to fight cancer. Killer T-cells kill cancer cells directly. These cells first find cancer cells and can also be stimulated to kill cancer cells. Helper T-cells fight cancer indirectly.

What causes cells to become cancerous?

Cancer cells have **gene mutations** that turn the cell from a normal cell into a cancer cell. These gene mutations may be inherited, develop over time as we get older and genes wear out, or develop if we are around something that damages our genes, like cigarette smoke, alcohol or ultraviolet (UV) radiation from the sun.

What activates the immune system to destroy a tumor?

Immunotherapy is a type of cancer treatment. It uses substances made by the body or in a laboratory to boost the immune system and help the body find and destroy cancer cells. Immunotherapy can treat many different types of cancer. It can be used alone or in combination with chemotherapy and/or other cancer treatments.

How does the immune system fight cancer?

The immune system consists of a complex process that your body uses to fight cancer. This process involves cells, organs, and proteins. Cancer can commonly get around many of the immune system's natural defenses, allowing cancer cells to continue to grow.

Different types of immunotherapy work in different ways. Some immunotherapy treatments help the immune system stop or slow the growth of cancer cells. Others help the immune system destroy cancer cells or stop the cancer from spreading to other parts of the body.

The different types of immunotherapy include:

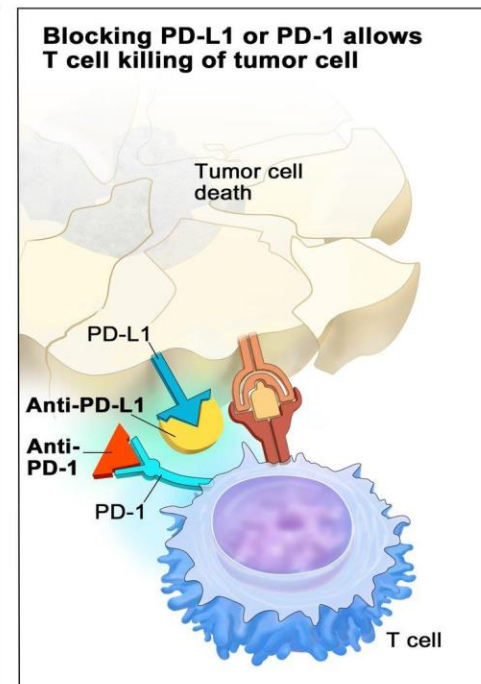
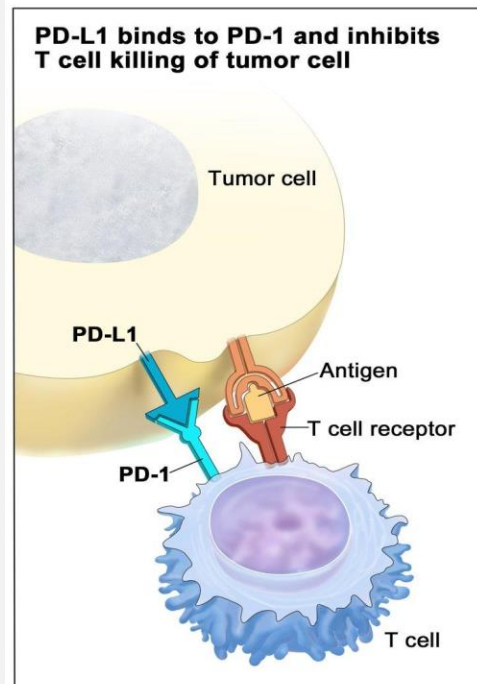
Monoclonal antibodies and immune checkpoint inhibitors

Non-specific immunotherapies

Oncolytic virus therapy

T-cell therapy

Cancer vaccines



Strategies to combat tumors based on immunotherapy,

passive.....

Active.....

What are cancer vaccines? (passive)

- A cancer vaccine can also help your body fight disease. A vaccine exposes your immune system to a foreign protein, called an antigen. This triggers the immune system to recognize and destroy that antigen or related substances. There are 2 types of cancer vaccine: prevention vaccines and treatment vaccines.
- One example of a cancer prevention vaccine is **Gardasil**, the vaccine to protect against the human papillomavirus (HPV).
- An example of a treatment vaccine includes **Spuleucel-T (Provenge)**, which treats advanced prostate cancer that does not respond to hormone therapy.
- T-VEC is also considered a cancer treatment vaccine. Side effects for both of these cancer vaccines are flu-like symptoms.



Which of the following are strategies for immunotherapy?

- Several different immunotherapy strategies are currently being studied or used as cancer treatments, including the following.
- Adoptive T-cell Transfer (T-cell Therapy) ...
- Immune Checkpoint Inhibitors. ...
- Monoclonal Antibodies. ...
- Nonspecific Immune Stimulation. ...
- Oncolytic virus immunotherapy. ...
- Vaccinations.

Auto-immune diseases.

What is an autoimmune disease?

- An autoimmune disease is a condition in which your immune system attacks your body.
- The immune system usually guards against bacteria and viruses. When it senses these foreign invaders, it sends out an army of fighter cells to attack them.
- Usually, the immune system can tell the difference between foreign cells and your own cells.
- In an autoimmune disease, the immune system mistakes part of your body, like your joints or skin, as foreign. It releases proteins called autoantibodies that attack healthy cells.
- Some autoimmune diseases target only one organ. Type 1 diabetes damages the pancreas. Other diseases, like systemic lupus erythematosus (SLE), or lupus, can affect the whole body.

Autoimmune responses are directed against self antigen

Autoimmune disease occurs when a specific adaptive immune response is mounted against self antigens.

The normal consequence of an adaptive immune response against a foreign antigen is the clearance of the antigen from the body.

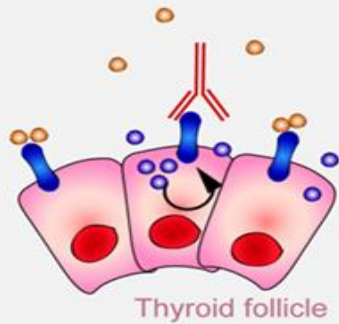
Virus-infected cells, for example, are destroyed by cytotoxic T cells, whereas soluble antigens are cleared by formation of immune complexes of antibody and antigen, which are taken up by cells of the mononuclear phagocytic system such as macrophages.

When an adaptive immune response develops against self antigens, however, it is usually impossible for immune effector mechanisms to eliminate the antigen completely, and so a sustained response occurs.

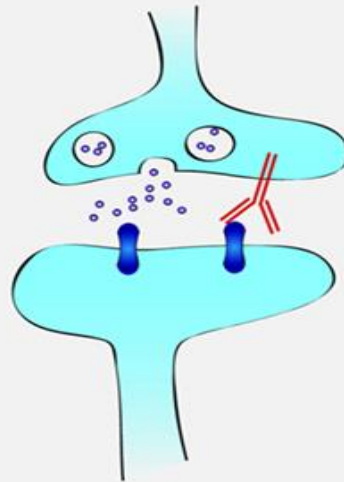
The consequence is that the effector pathways of immunity cause **chronic inflammatory injury to tissues**, which may **prove lethal**.

The mechanisms of tissue damage in autoimmune diseases are essentially the same as those that operate in protective immunity and in hypersensitivity diseases

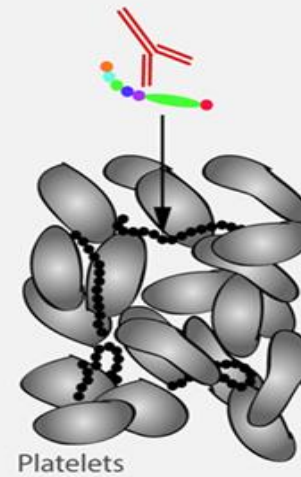
Mimickry of receptor activation by hormone



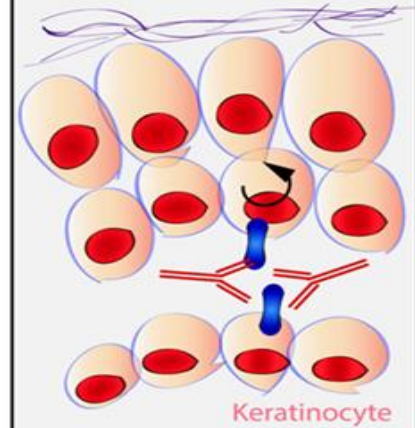
Blocking of neural transmission



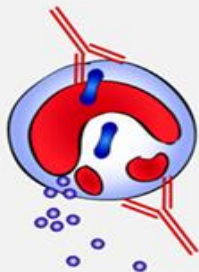
Blocking of enzymes/coagulation



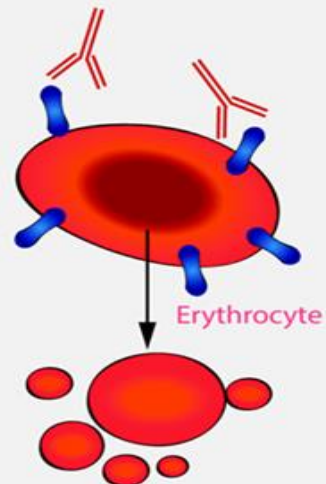
Altered cell signaling/acantolysis



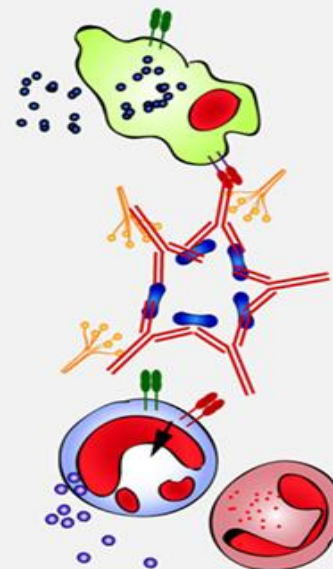
Neutrophil activation



Direct cell lysis



Induction of inflammation



- | | |
|--|-------------|
| | Antibodies |
| | Autoantigen |
| | Neutrophil |
| | Macrophage |
| | Basophil |
| | TSH |
| | ADAMTS13 |
| | Complement |

MULTIPLE PATHWAYS LEAD TO AUTOANTIBODY-INDUCED PATHOLOGY.

1. Depending on the targeted auto-antigen : antibodies against the thyrotropin receptor (TSHR) mimic hormone stimulation of the TSHR receptor leading to hyperthyroidism.
2. Blockade of neural transmission by autoantibody binding to the corresponding receptors may lead to severe neurological diseases such as anti-N-methyl-d-aspartate encephalitis.
3. Autoantibody-mediated blockade of enzymes of the primary hemostasis may trigger uncontrolled microthrombosis.
4. In pemphigus, autoantibodies induce an altered signaling in keratinocytes, which either reflects or leads to, a loss of cell–cell adhesion, resulting in severe skin blistering.
5. Autoantibodies to antigens expressed by neutrophils can lead to their uncontrolled activation, resulting in severe tissue injury.
6. In autoimmune idiopathic thrombocytopenia autoantibodies trigger thrombocytopenia and severe bleeding.
7. Fc γ -mediated functions may trigger tissue inflammation in many autoimmune diseases, e.g., rheumatoid arthritis and pemphigoid disease.

Key words

Word & Definition	Example
Immunocompromised <i>An overarching term that means the immune system is weakened or not working properly.</i>	People who are taking certain medications to slow down the immune system or those who have conditions that impact the body's ability to fight off infections like HIV or genetic immunodeficiencies may be considered immunocompromised.
Immune dysregulated <i>This term means that the immune system isn't working properly, but doesn't have the connotation that you're at higher risk for infection.</i>	People with autoimmune disease may be considered immune dysregulated. For example, with T1D, your immune system isn't working properly because it's attacking your pancreas. But that doesn't necessarily increase your risk of infection.
Immunodeficiency <i>This means that your immune system isn't working properly, typically because important components (like cells) aren't working right, there's not enough of them or they're missing altogether.</i>	Conditions like HIV where T cells are severely damaged and genetic conditions where people are born with low levels of certain immune cells are immunodeficient. These conditions typically put people at higher risk for infections.
Immunosuppression/ Immunosuppressive therapies <i>Immunosuppression is the process of slowing down the immune system.</i>	Immunosuppressive therapies can also slow down the immune system in autoimmune diseases, which happen when the immune system mistakenly attacks healthy tissue. Stronger immunosuppressive therapies may be used with organ transplants to help keep the immune system from attacking a new organ.
Immunomodulatory/ Immunomodulating therapies <i>Immunomodulatory means changing or altering the immune system by speeding it up or slowing it down.</i>	Immunomodulatory therapies may slow down the immune system for conditions like autoimmune disease. They may also speed up the immune system. Some immunotherapy drugs can amp up the immune system to fight cancer.

14 common autoimmune diseases

1. Type 1 diabetes

The pancreas produces the hormone insulin, which helps regulate blood sugar levels. In type 1 diabetes mellitus, the immune system attacks and destroys insulin-producing cells in the pancreas.

High blood sugar results can damage the blood vessels and organs, including the heart, kidneys, eyes, and nerves.

2. Rheumatoid arthritis (RA)

In rheumatoid arthritis (RA), the immune system attacks the joints. This attack causes redness, warmth, soreness, and stiffness in the joints.

Unlike osteoarthritis, which commonly affects people as they get older, RA can start as early as your 30s or sooner.

3. Psoriasis/psoriatic arthritis

Skin cells grow and then shed when they're no longer needed. Psoriasis causes skin cells to multiply too quickly. The extra cells build up and form inflamed, red patches, commonly with silver-white scales of plaque on lighter-toned skin. On darker skin, psoriasis can appear purplish or dark brown with gray scales.

4. Multiple sclerosis

Multiple sclerosis (MS) damages the myelin sheath, the protective coating surrounding nerve cells in your central nervous system. Damage to the myelin sheath slows the transmission speed of messages between your brain and spinal cord to and from the rest of your body.

5. Systemic lupus erythematosus (SLE)

Although doctors in the 1800s first described lupus as a skin disease because of the rash it commonly produces, the systemic form, which is most common, actually affects many organs, including the joints, kidneys, brain, and heart.

6. Inflammatory bowel disease

Inflammatory bowel disease (IBD) describes conditions that cause inflammation in the lining of the intestinal wall. Each type of IBD affects a different part of the GI tract.

Ulcerative colitis affects only the lining of the large intestine (colon) and rectum.

7. Addison's disease

Addison's disease affects the adrenal glands, which produce the hormones cortisol and aldosterone as well as androgen hormones. Too little cortisol can affect how the body uses and stores carbohydrates and sugar (glucose). Deficiency of aldosterone will lead to sodium loss and excess potassium in the bloodstream. Symptoms include weakness, fatigue, weight loss, and low blood sugar.

8. Graves' disease

Graves' disease attacks the thyroid gland in the neck, causing it to produce too much of its hormones. Thyroid hormones control the body's energy usage, known as metabolism.

Having too much of these hormones revs up your body's activities, causing symptoms like nervousness, a fast heartbeat, heat intolerance, and weight loss.

9. Sjögren's syndrome

This condition attacks the glands that provide lubrication to the eyes and mouth. The hallmark symptoms of Sjögren's syndrome are dry eyes and dry mouth, but it may also affect the joints or skin.

10. Hashimoto's thyroiditis

In Hashimoto's thyroiditis, thyroid hormone production slows to a deficiency. Symptoms include weight gain, sensitivity to cold, fatigue, hair loss, and swelling of the thyroid (goiter).

11. Myasthenia gravis

Myasthenia gravis affects nerve impulses that help the brain control the muscles. When the communication from nerves to muscles is impaired, signals can't direct the muscles to contract.

The most common symptom is muscle weakness, which worsens with activity and improves with rest. Muscles that control eye movements, eyelid opening, swallowing, and facial movements are often involved.

12. Autoimmune vasculitis

Autoimmune vasculitis happens when the immune system attacks blood vessels. The inflammation that results narrows the arteries and veins, allowing less blood to flow through them.

13. Pernicious anemia

This condition causes a deficiency of a protein made by stomach lining cells, which is an intrinsic factor needed for the small intestine to absorb vitamin B12 from food. Without enough of this vitamin, one will develop anemia, and the body's ability for proper DNA synthesis will be altered.

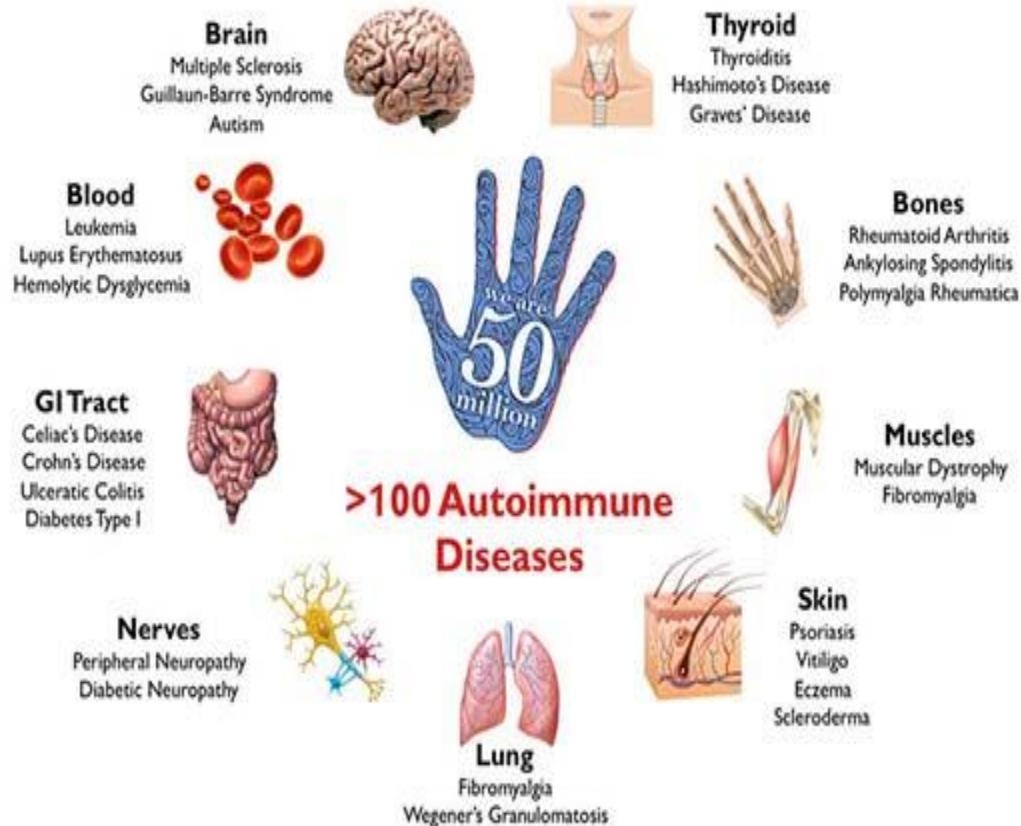
14. Celiac disease

People with celiac disease can't eat foods containing gluten, a protein found in wheat, rye, and other grain products. When gluten is in the small intestine, the immune system attacks this part of the gastrointestinal tract and causes inflammation.

How are autoimmune diseases treated?

- Treatments can't cure autoimmune diseases, but they can control the overactive immune response and bring down inflammation or at least reduce pain and inflammation. Drugs used to treat these conditions include:
 - Non-steroidal anti-inflammatory drugs (NSAIDs), such as ibuprofen (Motrin, Advil) and naproxen (Naprosyn)
 - immune-suppressing drugs
 - Treatments are also available to relieve symptoms like pain, swelling, fatigue, and skin rashes.
- Eating a well-balanced diet and getting regular exercise may also help you feel better.

Autoimmune Diseases



thank
you!

قسم الصيدلة



كلية النور الجامعة

2023

Brain-eating amoeba

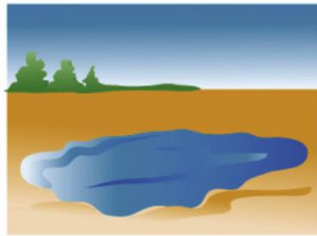
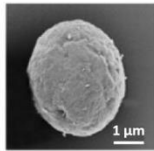


Free living amoeba

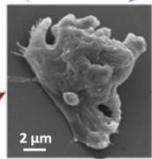
Naegleria fowleri

- It is a free-living amoeba that can be pathogenic, causing a fulminant brain infection called naegleriasis.
- Found in warm freshwater and in the soil near warm-water discharges of industrial plants, and in un-chlorinated or minimally-chlorinated swimming pools.
- It can be seen in either an amoeboid or temporary flagellate stage. *N. fowleri* is inhaled through the nose, where it then enters the nasal and olfactory nerve tissue, travelling to the brain.
- *N. fowleri* normally eat bacteria, but when it enters humans, it uses the brain as a food source.
- It does not form a cyst in human tissue, where only the amoeboid trophozoite stage exists. The flagellate form can exist in the cerebrospinal fluid.
- Disease = PAM (primary amoebic meningoencephalitis)
- -Early stages of infection may be similar to bacterial meningitis.
- -Initial PAM symptoms start 1-14 days after infection.
- -Early Symptoms = headache, fever, nausea, vomiting, stiff neck -Later Symptoms = confusion, lack of attention to environment, loss of balance, seizures, & hallucinations.
- -Rapid progression of disease after symptoms start, death within 3-7 days.
- Only Amphotericin B has been used to successfully treat means -Almost all cases are diagnosed during autopsies due to the rapid progression of the disease.

Cyst



Promitosis



Amoebic trophozoite



Flagellated trophozoite

Olfactory bulb

Cribriform plate

Olfactory nerves

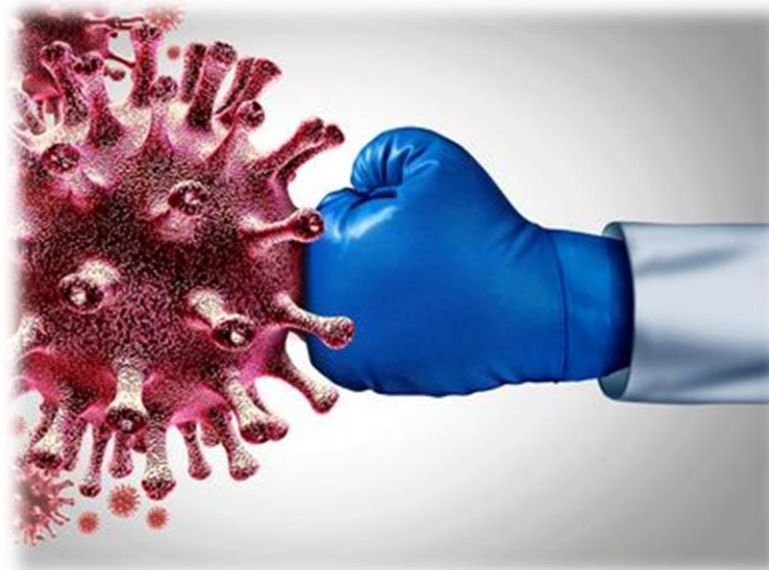
They attack the olfactory bulb and reach the brain

Trophozoites adhere to olfactory nerves and cross the cribriform plate

Trophozoites enter the nasal cavity with water



Alnoor University Collage
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قسم الصيدله
Medical Microbiology II



Introduction

Antiviral therapy is one of the most exciting aspects of virology, since it has successfully employed basic science to generate very effective treatments for serious viral infections.

Antiviral drugs are a class of medication used for treating viral infections. Most antivirals target specific viruses, while a broad-spectrum antiviral is effective against a wide range of viruses. Unlike most antibiotics, antiviral drugs do not destroy their target pathogen; instead they inhibit their development.

Most antivirals are considered relatively harmless to the host, and therefore can be used to treat infections.

They should be distinguished from **viricides**, which are not medication but deactivate or destroy virus particles, either inside or outside the body. Natural viricides are produced by some plants such as eucalyptus and Australian tea trees.

How do antiviral drugs fight viral diseases?

Unlike other antimicrobials, antiviral drugs do not deactivate or destroy the microbe (in this case, the virus) but act by inhibiting replication. In this way, they prevent the viral load from increasing to a point where it could cause pathogenesis, allowing the body's innate immune mechanisms to neutralize the virus.

What do antiviral drugs do?

Antiviral drugs are a class of medication used specifically for treating viral infections rather than bacterial ones. ... Unlike most antibiotics, antiviral drugs do not destroy their target pathogen; instead they inhibit their development.

Antivirals

Antiviral medications

- 1- Help the body **fight off** harmful viruses.
- 2- The drugs can **ease** symptoms and shorten the length of a viral infection.
- 3- Antivirals also **lower** the risk of getting or spreading viruses that cause herpes and HIV.

What are antivirals?

Antivirals are medications that help your body fight off certain viruses that can cause disease. Antiviral drugs are also preventive. They can protect you from getting viral infections or spreading a virus to others.

What are viruses?

Viruses are tiny (microscopic) infectious agents that grow and multiply only inside living cells of an organism. Viruses have receptors that allow them to attach to healthy (host) cells in your body. Once a virus attaches to and enters a host cell, it can replicate (make copies of itself). The host cell dies, and the virus infects other healthy cells.

Sometimes, viruses remain in a host cell without replicating or damaging it. The virus is still there (which means you could be contagious), but you don't have symptoms. This latent, or inactive, virus can become active at any time and cause symptoms or be passed on to others.

The way viruses spread depends on the type of virus.

Viruses can spread through: modes of viral spread (differs by type of virus):

- 1- Contaminated bodily products like blood, urine, feces, vomit, and saliva.
- 2- Bug bites (transfer of a virus from a bug's saliva into a person's blood).
- 3- Skin-on-skin contact, including sex.

How do antiviral medications work?

Antiviral medicines work differently depending on the drug and virus type. Antivirals can:

- 1- Block receptors so viruses can't bind to and enter healthy cells.
- 2-Boost the immune system, helping it fight off a viral infection.
- 3-Lower the viral load (amount of active virus) in the body.

What do antivirals treat?

Most viruses clear up without antiviral medications. Healthcare providers prescribe antivirals to treat chronic or life-threatening viral infections, including:

Coronaviruses like COVID-19.

Ebola.

Flu, including H1N1 (swine flu).

Genital herpes.

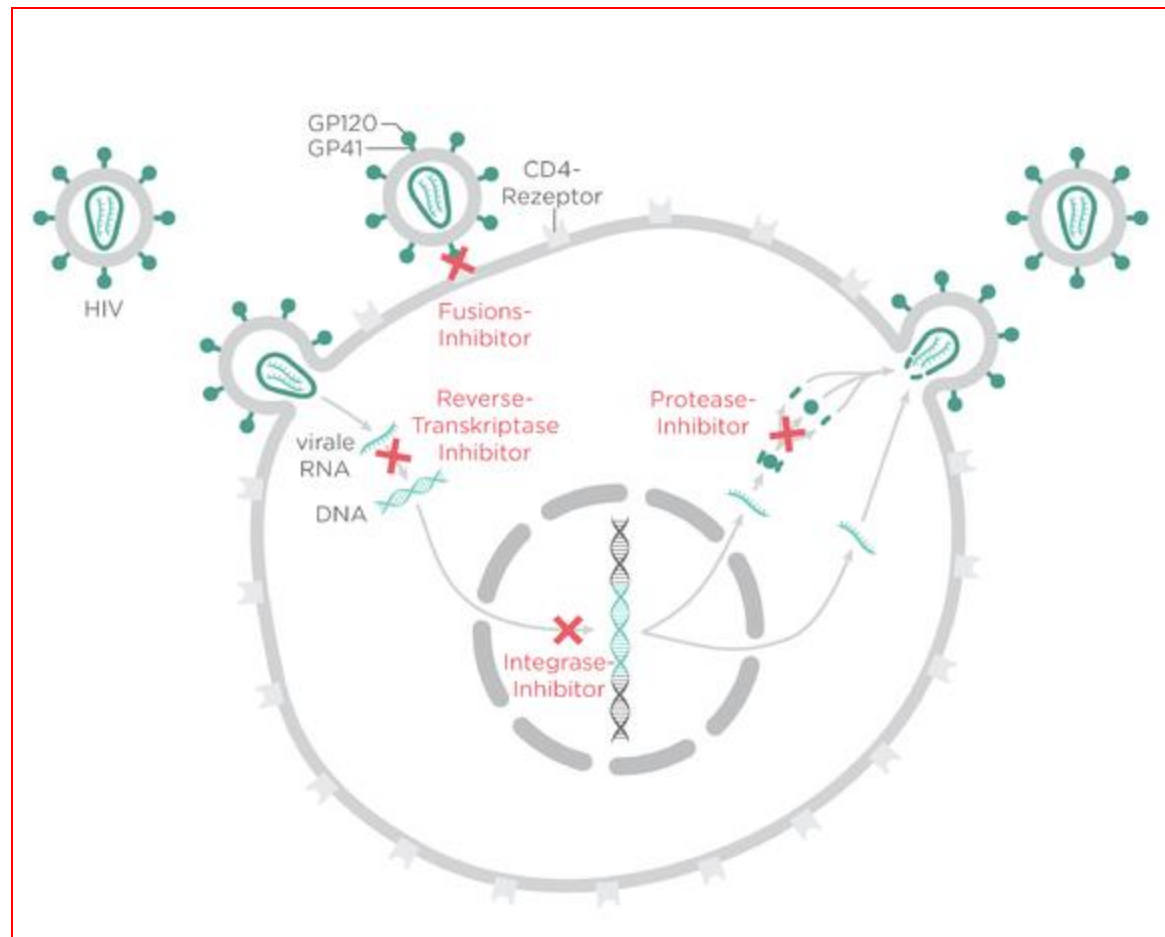
Hepatitis B and hepatitis C.

Human immunodeficiency virus (HIV).

Key Points about antiviral drugs:

Mechanism of Action (in combination or single action)

- 1- Inhibit viral attachment
- 2- Prevent genetic copying of virus
- 3- Prevent viral protein production, vital for reproduction of virus.



Can antivirals cure viral infections?

Antiviral drugs can ease symptoms and shorten how long you are sick with viral infections like the flu and Ebola. They can rid your body of these viruses.

Viral infections like HIV, hepatitis and herpes are chronic.

Antivirals can't get rid of the virus, which stays in your body. However, antiviral medicines can make the virus latent (**inactive**) so that you have few, if any, symptoms. Symptoms that develop while you take antivirals may be less severe or go away faster.

How do you take antiviral medications?

Most antivirals are oral drugs that you swallow. But you may also receive antiviral medications as:

- Eye drops.
- Inhaled powder.
- Injection (shot) into a muscle.
- IV into a vein.
- Topical (skin) ointments or creams.

What's the difference between antibiotics and antivirals?

Antibiotics help the immune system fight off bacterial infections. Bacteria typically reproduce outside of cells, making it easier for medicines to target them. An antibiotic can usually treat many different types of bacterial infections. But the drugs do not affect viruses.

Each antiviral only works against a specific virus. Because viruses inside cells are harder to target, antiviral drugs are more challenging to develop. There are more viruses than antiviral drugs to treat them.

What are the potential side effects of antivirals?

Side effects from antivirals vary depending on the drug type and strength (dosage). You may experience:

- Cough.
- Dry mouth.
- Diarrhea.
- Dizziness.
- Fatigue.
- Headaches.
- Insomnia.
- Joint pain or muscle pain.
- Nausea and vomiting.
- Skin rash.



What is antiviral resistance?

Skipping doses or starting and stopping an antiviral medicine can allow a virus to change/adapt so that the antiviral is no longer effective. This is antiviral resistance.

People who take antivirals for extended periods are more prone to antiviral resistance.

Who shouldn't take antiviral medications?

Antivirals are relatively safe medicines. Children as young as two weeks, as well as pregnant and breastfeeding individuals, can take certain antiviral medications. Guidelines for who shouldn't take antivirals vary depending on the drug. Your healthcare provider can determine whether an antiviral medicine is safe for you.

Examples of antiviral drugs



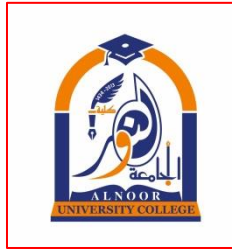
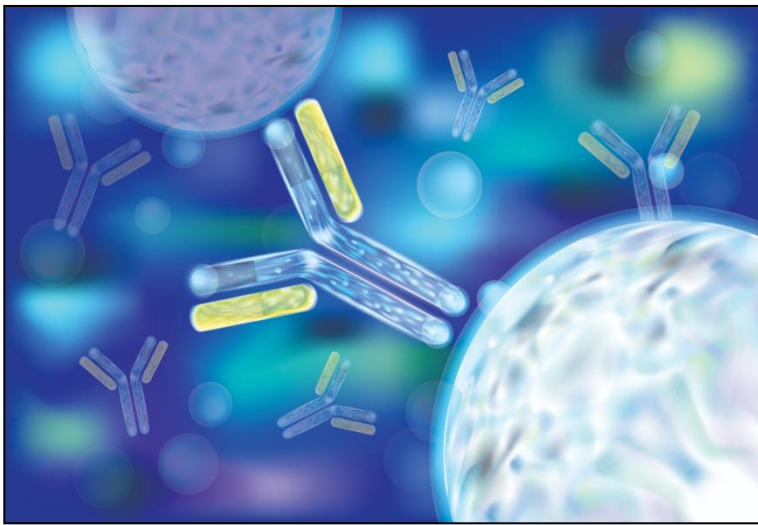
Virus	Antiviral Drug	Target
Herpes viruses	Vidarabine	Virus polymerase
Herpes simplex	Acyclovir	Virus polymerase
Cytomegalovirus	Gancyclovir	Virus polymerase
Retroviruses (HIV)	Zidovudine, Didanosine, Zalcitabine, Lamivudine	Reverse transcriptase
Retroviruses (HIV)	Saquinavir, Ritonavir, Indinavir, Nelfinavir	HIV protease
HCV, HSV	Ribavirin	RNA mutagen
Influenza A	Amantadine, Rimantadine	Haemagglutinin protein
Influenza B	Relenza and Tamiflu	Neuraminidase Inhibitor
Picorna viruses	Pleconaril	Blocks attachment
Hepatitis B & C	Interferons	Cell defence proteins

Note:-

- Antibiotics don't treat the viral infection



Discuss this sentence to explain why antibiotics have no effect on viral infection??? & what is the differences between antibiotics and antiviral drugs?



Alnoor University Collage

كلية النور الجامعة □

Pharmacy Department

قسم الصيدلة □

Medical Microbiology II

Immunology

Nada Khayri Younus Al-Asaadi

MSc. Medical microbiology

Immunology

1. General introduction, innate & adaptive immunity, B& T cells.
2. Antigen characteristics, Complements, Hypersensitivity types.
3. Oncogenic immunity.
4. Auto-immune diseases.

Introduction

- The body has developed defense mechanisms to control with constant attack of microorganisms.
 - The basic terms, the immune system has two lines of defense: **innate immunity** and **adaptive immunity**.
 - Human have innate (inborn) resistance to certain pathogens, it is a rapid immune response, occurring within minutes or hours after aggression, that has no immunological memory. as well as three overlapping lines of defense.
 - The first two lines of defense are nonspecific and protect the body against a wide variety of potential pathogens.(The skin has thick layer of dead cells in the epidermis which provides a physical barrier. Periodic shedding of the epidermis removes microbes). The third is a specific immune response to particular pathogen.
-
- The body has three lines of defense
 - 1. Physical barriers
 - 2. Defensive cells & proteins, Inflammation, and fever
 - 3. The immune system
 - These are a combination of physical and chemical barriers that prevent all types of foreign agents from penetrating the outer layer of the body.

1. The first line of defense:

- Skin, composed of an outer epidermis and a deeper dermis. Dendritic cells of the epidermis devour (tuck or eat) pathogens. Sweat glands of the skin produce salty sweat containing an antimicrobial protein called lysozyme.
- **Sebum** is an oily substance of the skin that lowers pH, which deters (stop) the growth of many pathogens. (Sebum is an oily, waxy substance produced by your body's sebaceous glands. It coats, moisturizes, and protects our skin) .
- The mucous membranes, part of the body's first line of defense, are composed of tightly packed cells often coated with sticky mucous secreted by goblet cells.
- Tears contain antibacterial lysosome and also flush invaders from the eyes. Saliva similarly protects the teeth. The low pH of the stomach inhibits most microbes that are swallowed.

2.

The second line of defense

1) The second line of defense include cells (especially phagocytes), antimicrobial chemicals (complement, interferon), and processes (inflammation and fever).

2) Blood is composed of erythrocytes, leucocytes and platelets. Basophils function to release histamine during inflammation, whereas eosinophil and neutrophils phagocytize pathogens, when monocytes leave the blood they become macrophage.

3) Chemotactic factors, such as chemicals called **cytokines**, attract phagocytic leukocytes to the site of damage or invasion. The phagocytes attach to pathogens via a process called adherence. Leukocytes can distinguish between the body's normal cells and foreign cells because they have receptor molecules for foreign cells components.

4) Opsonization, the coating of pathogens by proteins called opsonin, makes those pathogens more vulnerable to phagocytes (Antibody opsonization is the process by which the pathogen is marked for ingestion and eliminated by the phagocytes) .

A phagocyte's pseudopodia then surround the microbe to form a sac called phagosome, which fuse with a lysosome to form a phagolysosome, in which the pathogen is digested.

5) Eosinophils and Natural killer (NK) lymphocytes attack extracellular, especially in the case of helminth infections and cancerous cells. Eosinophilia- an abnormally high number of eosinophils in the blood- typically indicates such a helminth infection.

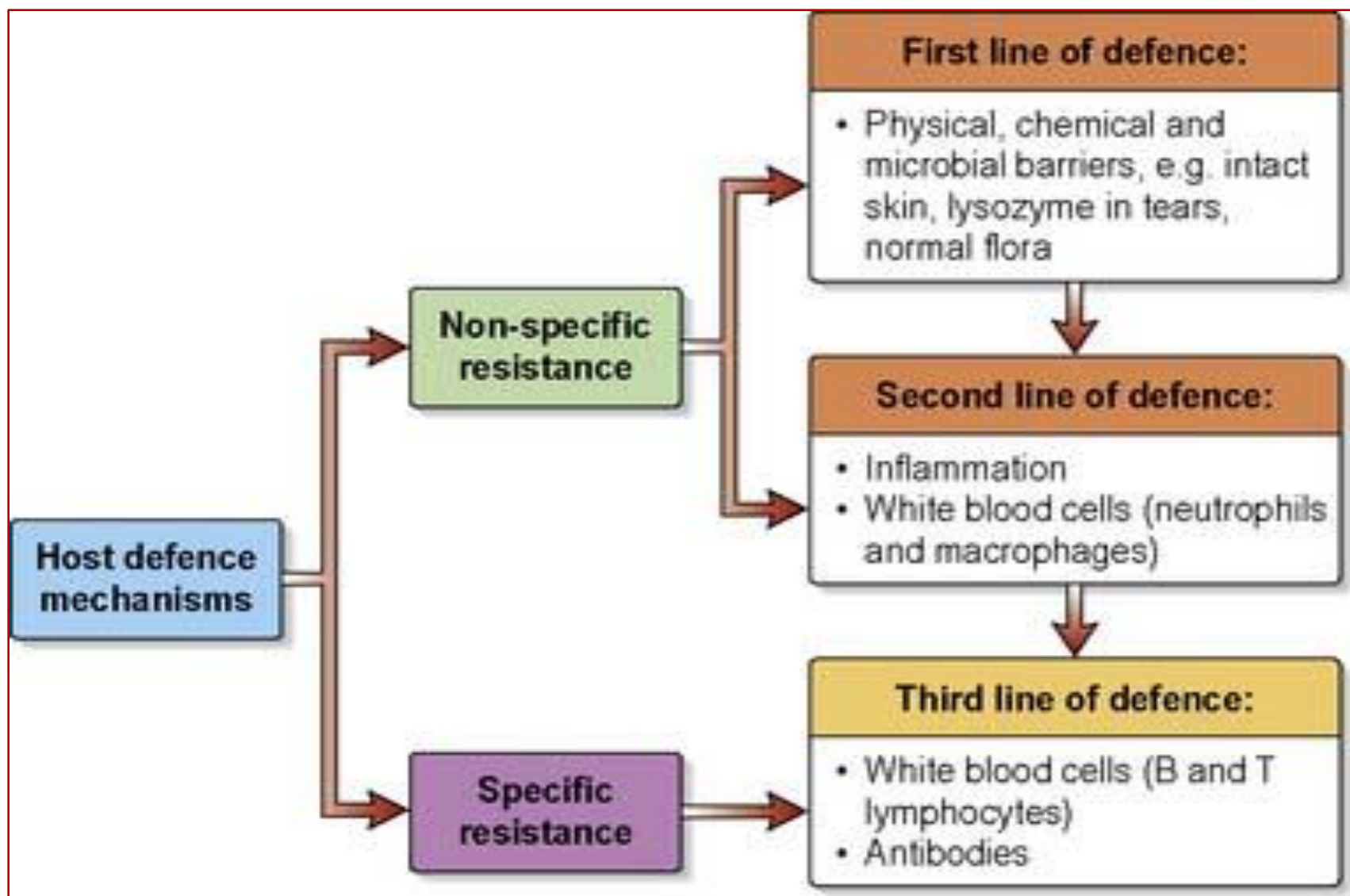
6) The complement system is a set of proteins that act as chemotactic attractants, trigger inflammation and fever. Complement is activated by classical pathway involving antibodies.

7) Interferons are protein molecules that inhibit the spread of viral infections. Alpha-interferons & Beta-interferons, which are released within hours of infection, trigger antiviral proteins to prevent viral reproduction in neighboring cells. Gamma-interferons, reproduced days after initial infection, activate macrophages and neutrophils.

8) Acute inflammation develops quickly and damages pathogens, whereas chronic inflammation develops slowly and can cause bodily damage that can lead to disease. Signs and symptoms of inflammation include: rubor (redness), calor (heat), tumor (swellaw), and dolor (pain).

9) Infection causes damaged cells to release chemicals such as histamine, which trigger vasodilation, and prostaglandins & leukotrienes (LTs), which increase permeability of blood vessels.

10) Fever results when chemicals called pyrogens, including interleukin 1, affect the hypothalamus in a way that causes it to reset body temperature at higher level.



The first line
of defense
(non-specific
immunity)

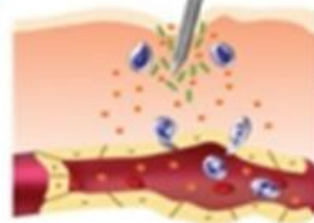


Skin

Mucous
membrane

The second line
of defense
(non-specific
immunity)

Phagocyte



inflammation

The third line
of defense
(specific
immunity)



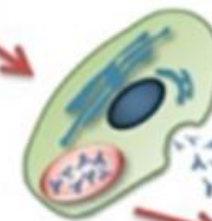
Helper
T cell



Cytotoxic T cell



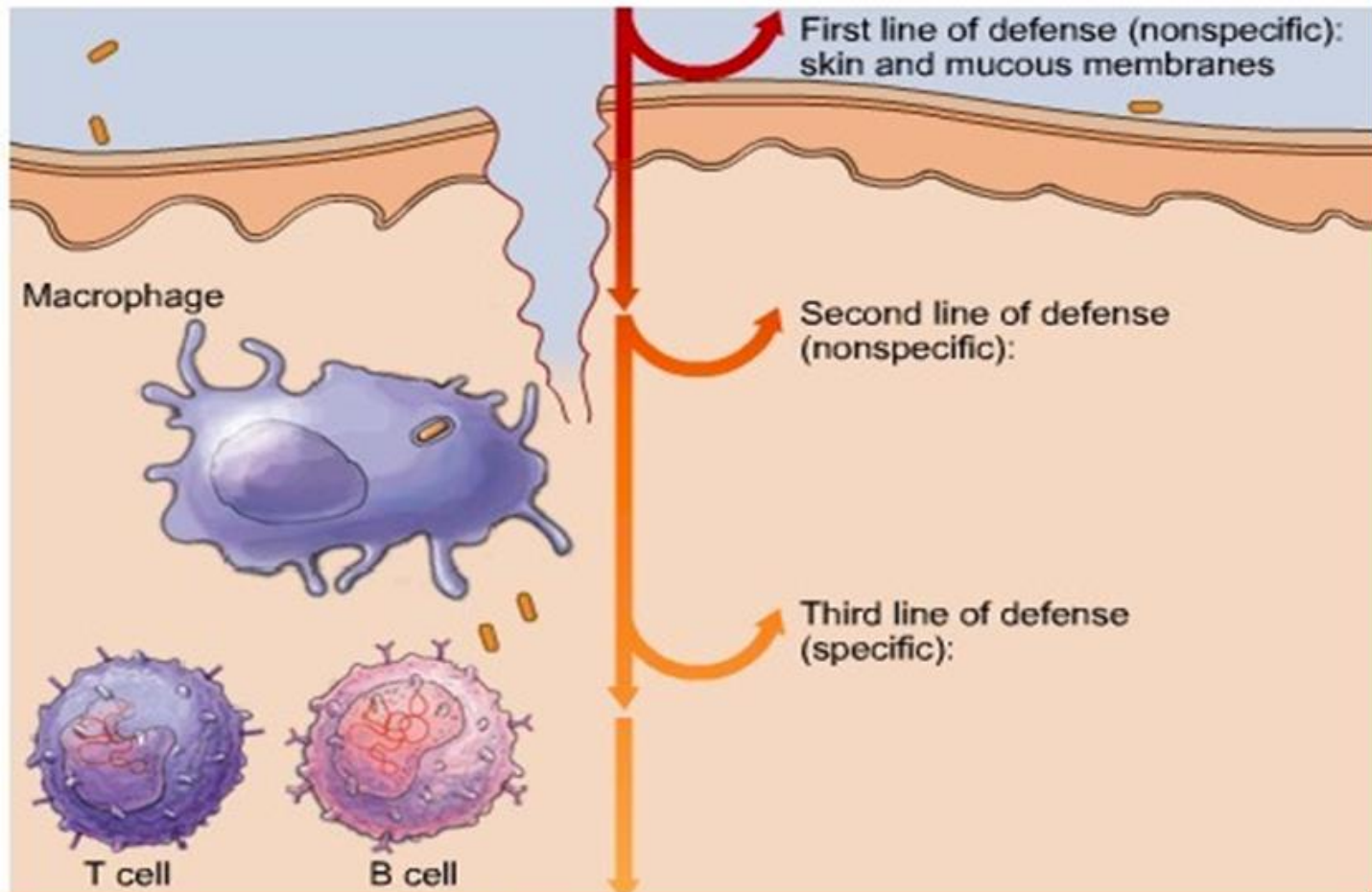
B cell



Antibody



The Three Lines of Defense



NONSPECIFIC DEFENSE MECHANISMS		SPECIFIC DEFENSE MECHANISMS (IMMUNE SYSTEM)
First line of defense	Second line of defense	Third line of defense
<ul style="list-style-type: none"> • Skin • Mucous membranes • Secretions of skin and mucous membranes 	<ul style="list-style-type: none"> • Phagocytic white blood cells • Antimicrobial proteins • The inflammatory response 	<ul style="list-style-type: none"> • Lymphocytes • Antibodies

Components of immune system

1. Cells

- Phagocytes: Macrophages & Granulocytes
- Lymphocytes: T- cells & B-cells
- Antigen presenting cells: Dendritic cells

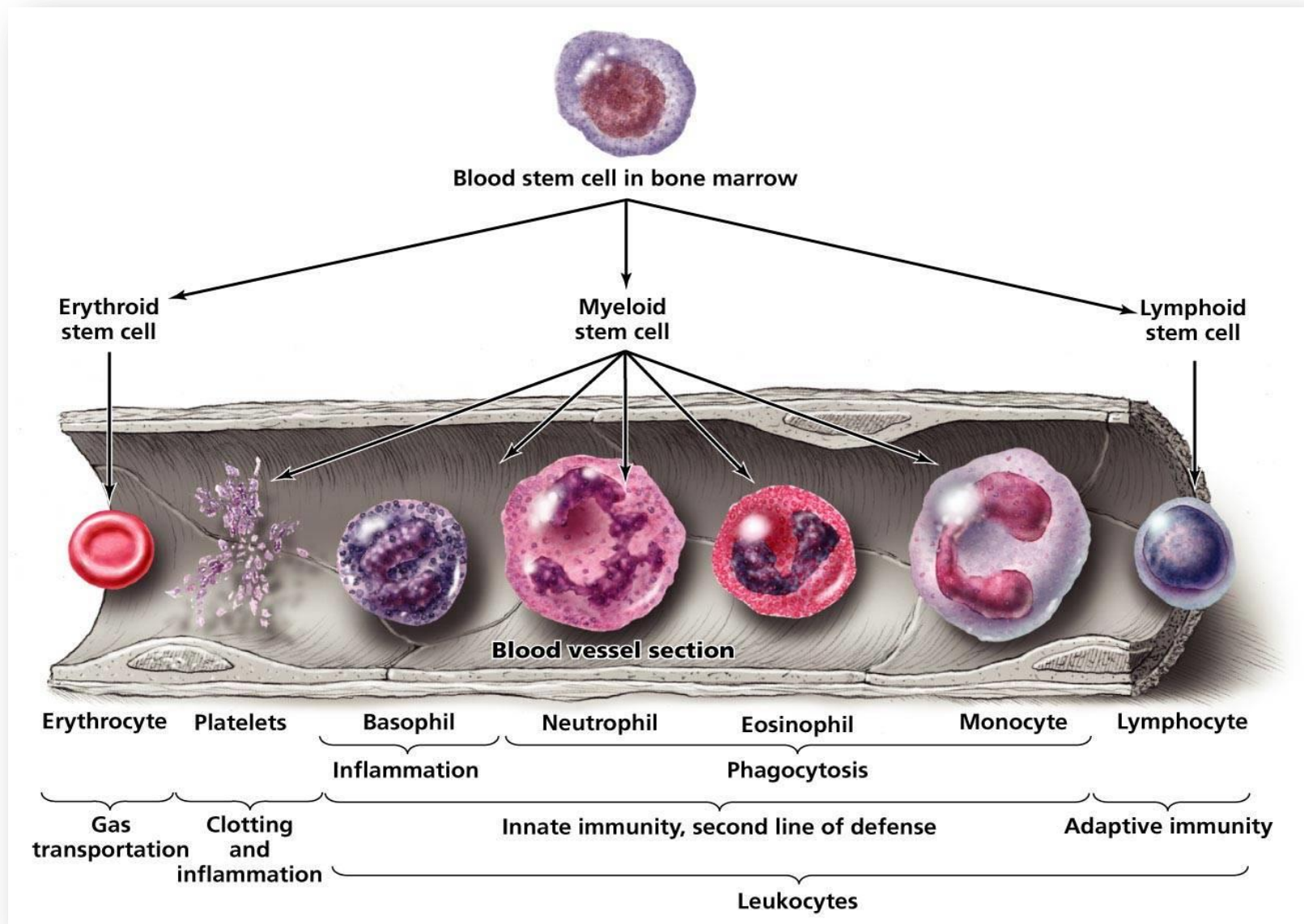
2. Organs

- **Primary organs:** immune cells develop and mature.
 - Bone marrow: differentiation of blood cells.
 - Thymus: T- cell maturation.
 - Bursa of fabricius: B-cell maturation.
- **Secondary immune organs:**
 - Spleen
 - Lymph nodes.
 - Mucosa associated lymphoid tissue.

3. Soluble mediators:

- Signaling molecules like [cytokines, complement, antibodies, chemokines and interleukins.
- Cytotoxic molecules

Types of bloods cells & there functions

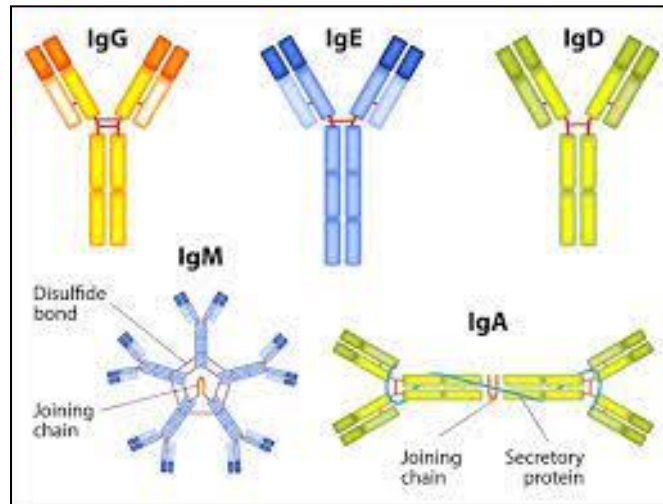


Antibodies

- What are antibodies?

Antibodies are proteins that protect you when an unwanted substance enters your body. Produced by your immune system, antibodies bind to these unwanted substances in order to eliminate them from your system. Another word for antibody is immunoglobulin.

Its function: they work to destroy disease-causing organisms (such as viruses or bacteria) and block them from infecting human cells.



Antibodies are glycoproteins, termed as **immunoglobulins (Igs)**, which are produced in response to an immune reaction and specifically bind to antigens responsible for initiating the reaction.

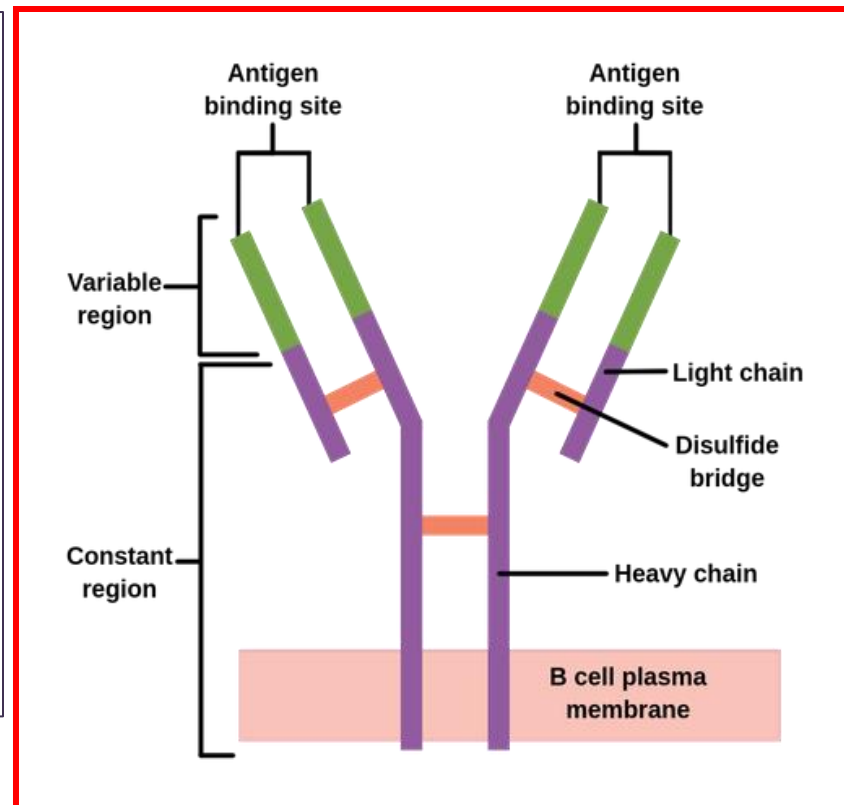
Functions of antibodies (immunoglobulins)

The most important function of antibodies is to provide protection against microbial pathogens. Some of the main functions of antibodies are:

- They reduce the virulence of microbes by neutralizing toxins and viruses.
- They opsonize microbes so they are more easily phagocytosed, also, they activate the complement,
- Antibodies will prevent the attachment of microbes to mucosal surfaces.

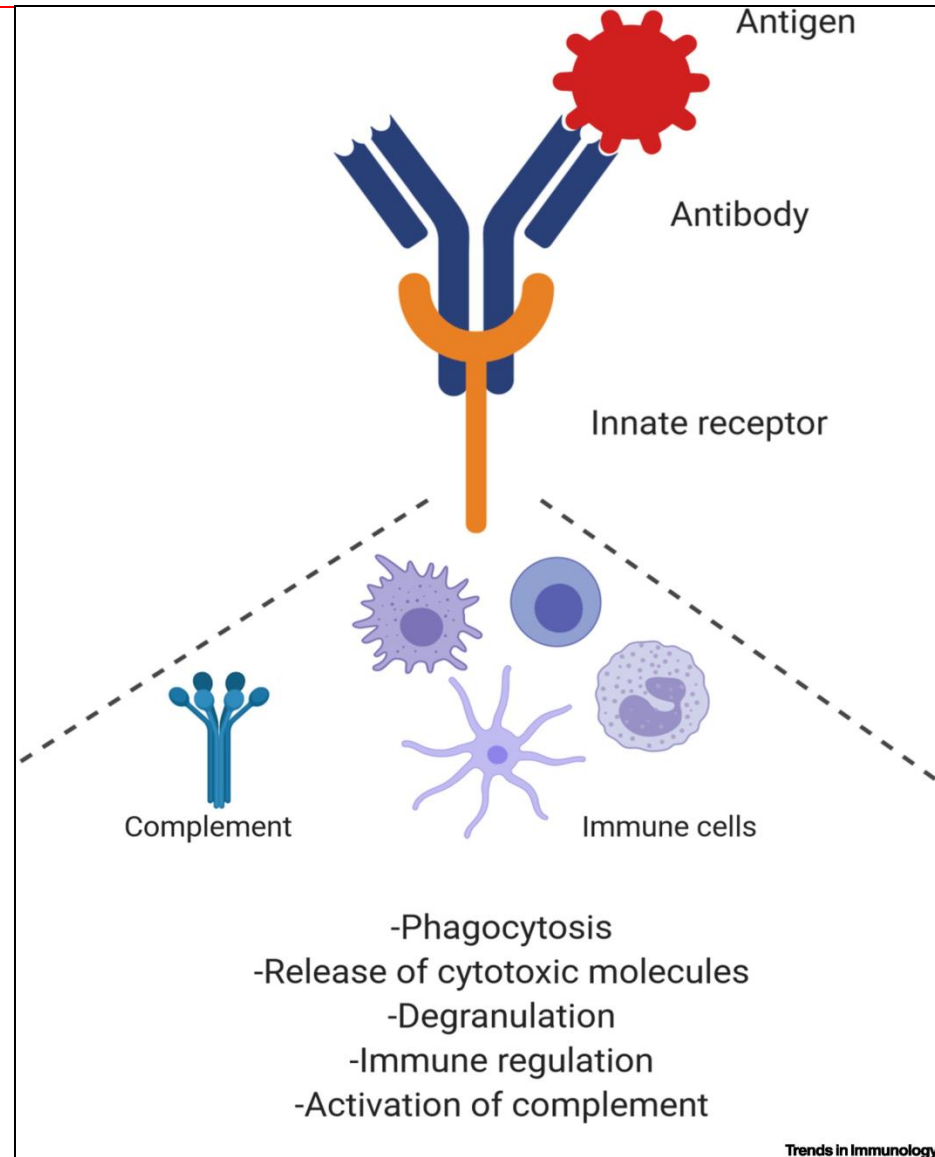
STRUCTURE OF ANTIBODIES/IMMUNOGLOBULINS

Immunoglobulins are glycoproteins comprises of four polypeptide chain: two identical light (L) and two identical heavy (H) chains. Further, L and H chains are subdivided into variable and constant regions. The terms light and heavy refer to molecular weight. **The heavy chains are longer** whereas **light chains are shorter**. Light chains have a molecular weight of about **25,000 Da** whereas heavy chains have a molecular weight of **50-70,000 Da**.



Variable and constant region

- Each polypeptide chain of an immunoglobulin molecule contains an amino-terminal part and a carboxy-terminal part. The amino terminal part is called the variable region (**V** region) whereas the carboxy-terminal part is called the constant region (**C** region).
- The variable regions of both the light and heavy chain are responsible for antigen binding whereas the constant region of the heavy chain is responsible for various biologic functions.
- This structure allows antibody molecules to carry out their dual functions: antigen binding and biological activity mediation.



Types of antibodies and properties

- There are **five** immunoglobulin classes (isotypes) of antibody molecules found in serum: IgG, IgM, IgA, IgE, and IgD. They are distinguished by the type of heavy chain they contain.
- IgG molecules possess heavy chains known as γ -chains [gamma-chains]. Secreted by plasma cells in the blood. Able to cross the placenta into the fetus.
- IgMs have μ -chains [mu-chains]. May be attached to the surface of a B-cell or secreted into the blood. Responsible for early stages of immunity.
- IgAs have α -chains [alpha-chains] Found in mucous, saliva, tears, and breast milk. Protects against pathogens.
- IgEs have ϵ -chains [epsilon-chains]. Protects against parasitic worms. Responsible for allergic reactions.
- IgDs have δ -chains [delta-chains]. Part of the B-cell receptor. Activates basophils and mast cells.

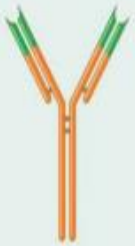

The biological and chemical characteristics of antibodies

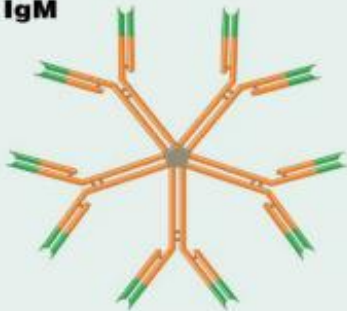
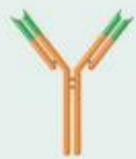
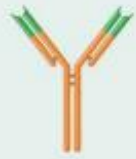


PROPERTIES OF IMMUNOGLOBULINS

	IgG	IgM	IgA	IgE	IgD
Structure	Monomer	Pentamer	Dimer	Monomer	Monomer
Location	blood, lymph, intestines	Secretions	Blood, lymph & B-cell surfaces	Bound to mast & basophil cells	B-cell surfaces, blood, lymph
Molecular weight in (Da) Dalton	1,50,000	9,00,000	1,60,000	1,90,000	1,80,000
Percentage of total serum	80%	5-8%	10-13%	0.002%	0.2%

5 Types of antibodies & their functions

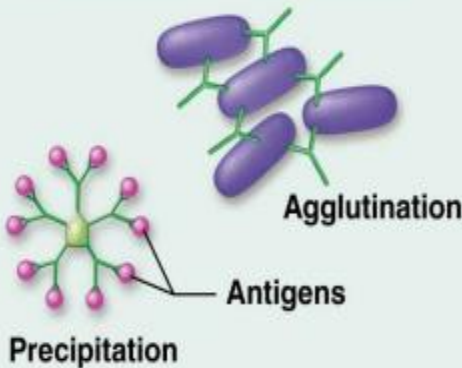
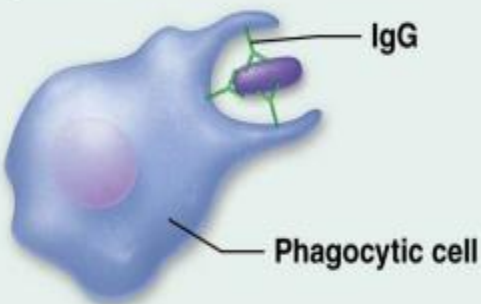
CLASS	FUNCTION
IgG 	<ul style="list-style-type: none"> • Makes up the majority of antibodies in serum • The only antibody that can cross the placenta from mother to fetus • Functions in opsonization, neutralization, and complement fixation
IgA 	<ul style="list-style-type: none"> • Found in secretions such as breast milk and saliva • Functions in agglutination and neutralization

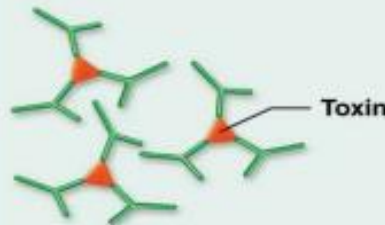
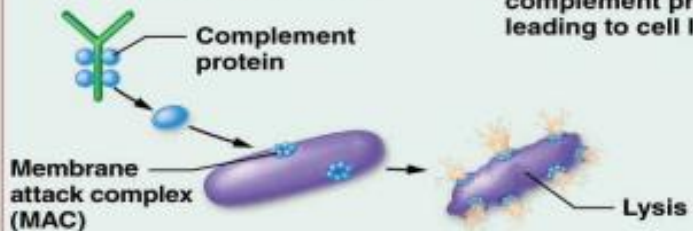
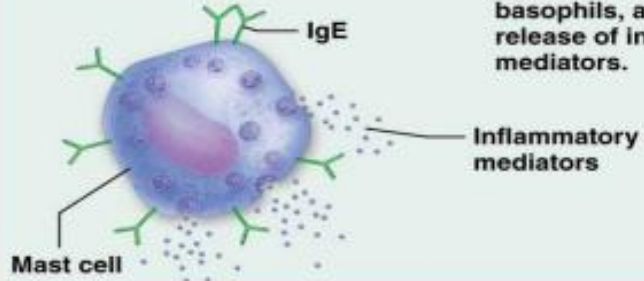
CLASS	FUNCTION
IgM 	<ul style="list-style-type: none"> • The first antibody secreted on exposure to an antigen • Potent agglutinating and precipitating agent • Functions in complement fixation
IgE 	<ul style="list-style-type: none"> • Binds mast cells and basophils and triggers their degranulation, facilitating inflammation, particularly in the allergic response
IgD 	<ul style="list-style-type: none"> • Antibody found exclusively on the surface of B cells • Has a role in B cell sensitization and activation

The main functions of antibodies are:

Functions of Antibodies.

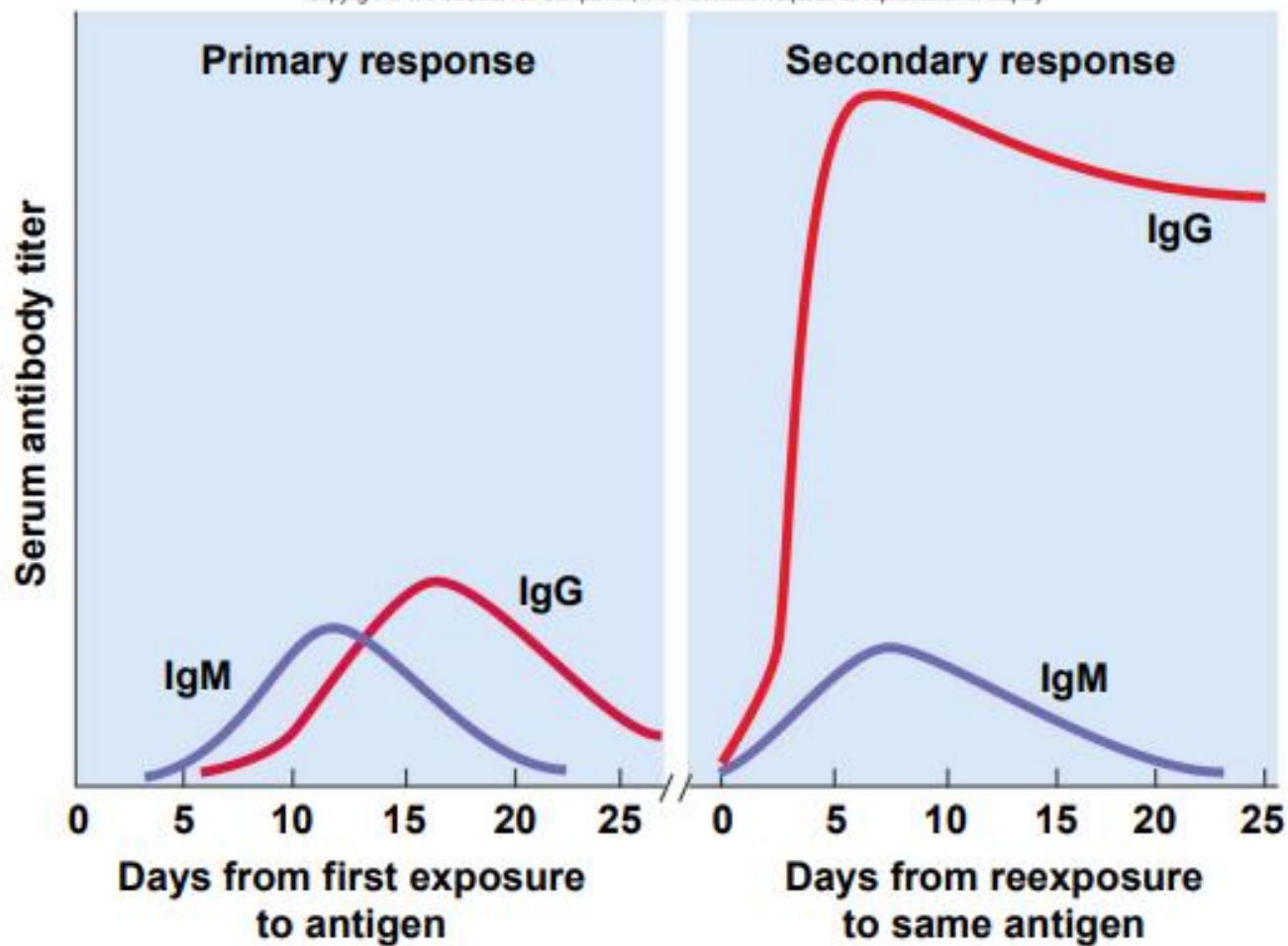
- Agglutination/Precipitation
- Opsonization
- Neutralization
- Complement Activation
- Stimulation of Inflammation

FUNCTION	DESCRIPTION
Agglutination/precipitation 	Antibodies clump antigens together to enhance phagocytosis.
Opsonization 	IgG coats antigens and binds phagocytes, enhancing phagocytosis.

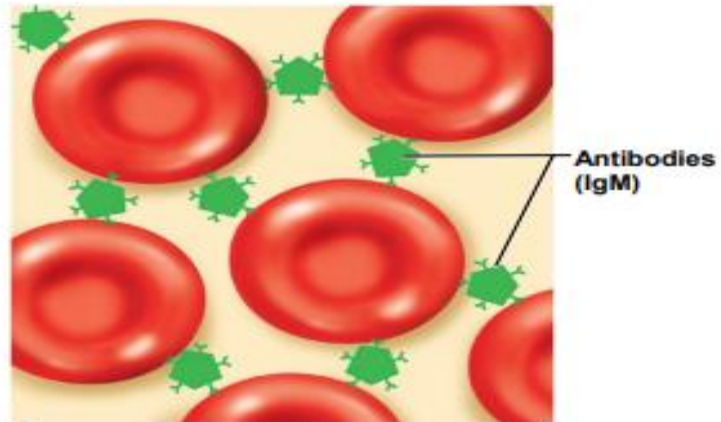
FUNCTION	DESCRIPTION
Neutralization 	Antibodies bind pathogenic components of toxins and block toxic effects.
Complement activation 	Antibodies activate complement proteins, leading to cell lysis.
Stimulation of inflammation 	IgE binds mast cells and basophils, and triggers release of inflammatory mediators.

Humoral Immunity Responses

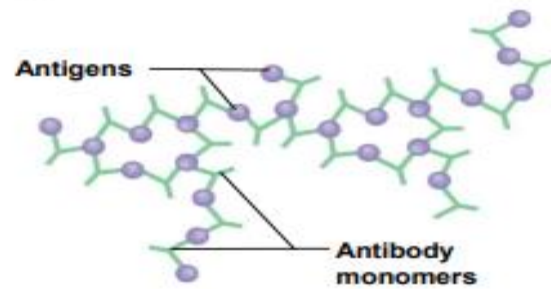
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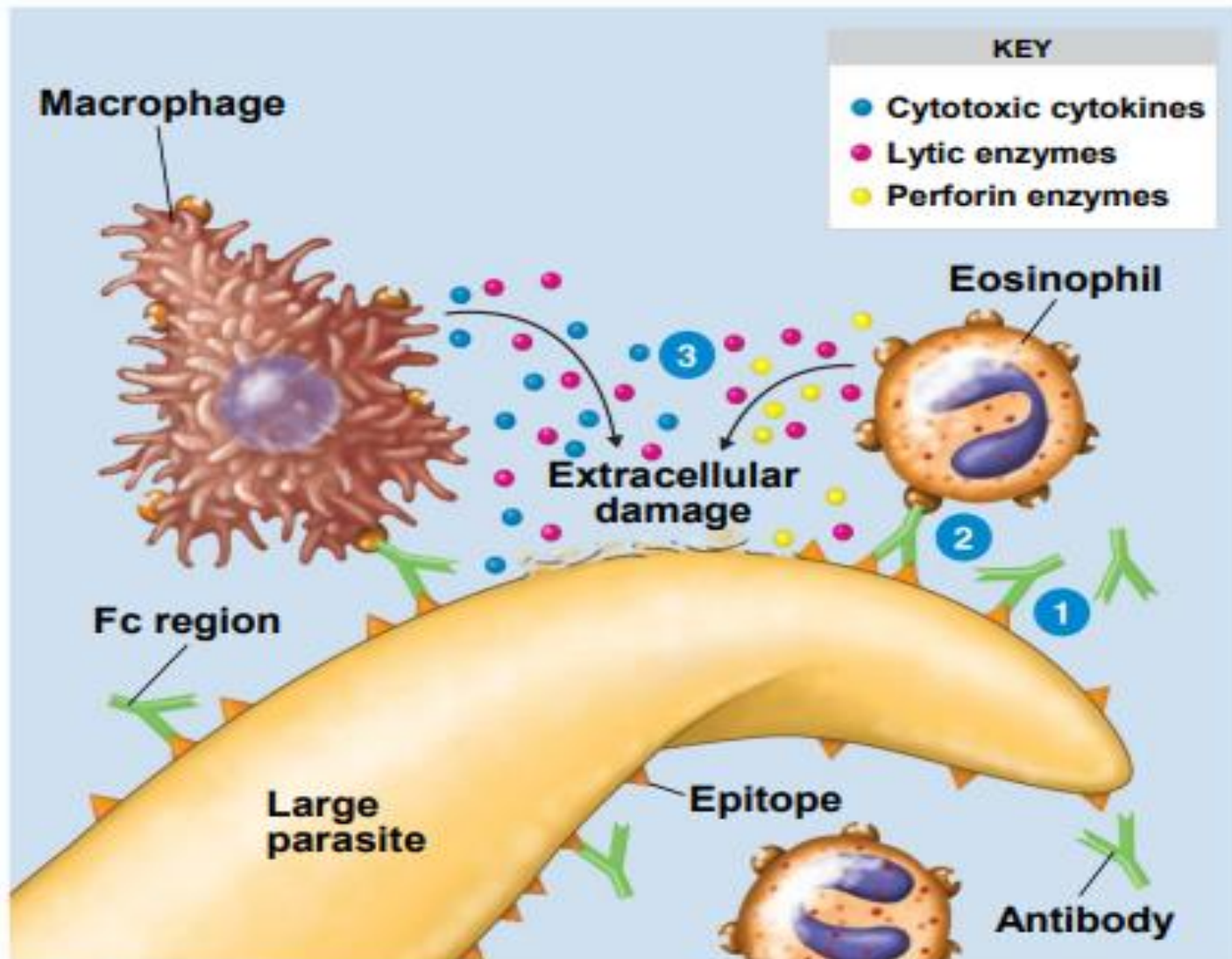
Agglutination



(a)



Antibody-dependent cell-mediated cytotoxicity (ADCC).



(a) Organisms, such as many parasites, that are too large for ingestion by phagocytic cells must be attacked externally.

Antigen

Antigen, substance that is capable of stimulating an immune response, specifically activating lymphocytes, which are the body's infection-fighting white blood cells.

Antigens can be grouped into different types based on different factors. Some of the common classifications are based on the origin of the antigen and its immunogenicity.

1. Types of antigen-based on their origin

Antigens can be classified into two groups on the basis of their origin;

a. Exogenous Antigens

Exogenous antigens are the antigens that are originated outside the body of the host and, thus, are foreign to the host.

These antigens might enter the body through inhalation, ingestion, or injection and then circulate throughout the body via bodily fluids.

The uptake of exogenous antigens is primarily mediated by phagocytosis via Antigen Processing Cells (APCs) like macrophages, dendritic cells, etc.

Many antigens like intracellular viruses might begin as exogenous antigens and later become endogenous.

b. Endogenous Antigens

Endogenous antigens are antigens that originate within the body of the host during metabolism or as a result of intracellular viral or bacterial infection.

Endogenous antigens are usually the cells of the body or fragments, compounds, or antigenic products of metabolism.

These are usually processed in the macrophages and are later detected by cytotoxic T-cells of the immune system.

Endogenous antigens include antigens that are xenogenic or heterologous, autologous, and idiotypic or allogenic.

Endogenous antigens might result in autoimmune diseases as the host immune system detects its own cells and particles as immunogenic

2. Types of antigens on the basis of immune response

Antigens can be classified into two distinct groups on the basis of immune response;

a. Complete antigens/ Immunogens

Complete antigens or Immunogens are antigens that elicit a specific immune response. These antigens can induce an immune response by themselves without any carrier particles.

These are usually proteins, peptides, or polysaccharides with high molecular weight (greater than 10,000 Da).

b. Incomplete antigens/ Haptens

Incomplete antigens or haptens are antigens that cannot generate an immune response by themselves.

These are usually non-protein substances that require a carrier molecule to form a complete antigen.

Haptens have a low molecular weight (usually less than 10,000 Da) and fewer antigenic determinant sites.

The carrier molecule bonded to the hapten is considered a non-antigenic component and is a protein or a polysaccharide molecule.

Characteristics of Antigens

- **Immunogenicity** – property of substance (**immunogens** or **antigens**) to induce a detectable immune response
- **Antigenic specificity** – property of antigen molecule (or its part) to react with the **specific antibody**.
- **Antigenicity** – given by a surface structure of immunogen - **antigenic determinants**. The organism responds only to those that are foreign to him.
- **The number of antigenic determinants** – usually varies with the size and chemical complexity of macromolecule (egg ovalbumin, MW 42 000, has 5 antigenic determinants and thyroglobulin, MW 700 000, has many as 40).

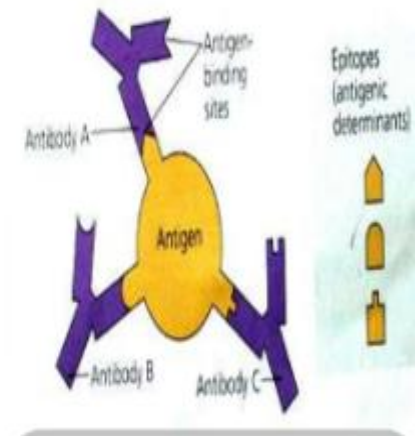
5/2013

Dr TV Rao MD








Characteristics of Antigens

- **Chemical nature of antigens:**

- proteins
- polysaccharides
- lipopolysaccharides
- nucleoproteins
- glycoproteins
- steroid hormones
- bacterial cells, viruses
- synthetic polypeptides
- synthetic polymers



Antigen Examples

<u>ABO Blood Types</u>				
	A	B	AB	O
Antigens present on erythrocytes	 A antigen	 B antigen		
Antibodies present in plasma	 Anti-B antibodies	 Anti-A antibodies	No antibodies	 Anti-A antibodies Anti-B antibodies

- **1. Blood group antigens**
- Blood group antigens are proteins or sugars present on the surface of different components in the red blood cell membrane.
- The type of sugar in the red blood cell is determined by the type of enzyme involved, which in turn is determined by the person's DNA.
- The antigens of the Rh blood group are proteins that are also determined by the host's DNA. The RhD gene encodes the D antigen, which occurs as a large protein on the red blood cell.
- These antigens can be distinguished by antigen-antibody reactions that help determine different blood groups in humans.

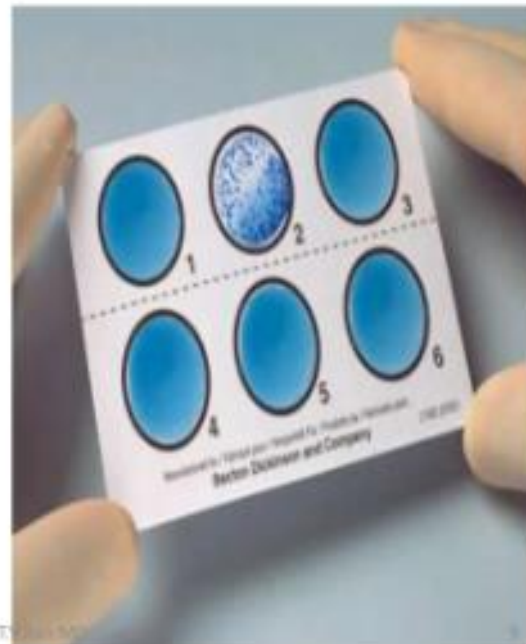
To determine the reaction between Ag-Ab

Serology

- **Serology** is the scientific study of blood serum. In practice, the term usually refers to the diagnostic identification of antibodies in the serum

We can detect antigens too

1/5/2013



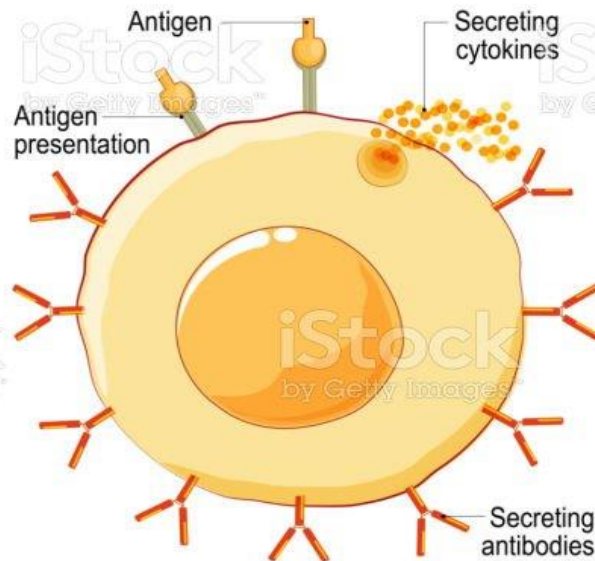
Dr. T. J. M. M.



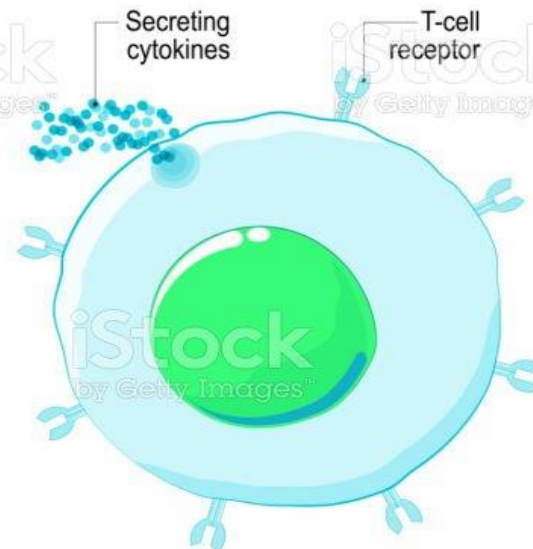
Adaptive immune response

T-cell & B-cell

Cells of the adaptive immune system



B-cell



T-cell

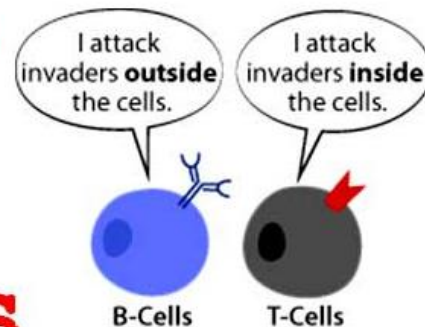
T-cell & B-cell

- ◆ The T and B lymphocytes (T and B Cells) are involved in the acquired or antigen-specific immune response given that they are the only cells in the organism able to recognize and respond specifically to each antigenic epitope.

The B-Cells have the ability to transform into plasmocytes and are responsible for producing antibodies (Abs). Thus, humoral immunity depends on the B-Cells while cell immunity depends on the T-Cells.

- ◆ From the morphological point of view, T and B lymphocytes are indistinguishable since they are both small cells (8–10 microns in diameter) and each possesses a large nucleus with dense hetero-chromatin and a cytoplasmic border that contains few mitochondria, ribosomes, and lysosomes. When they are activated by the antigenic stimulus, they may enlarge, thus increasing their cytoplasm and organelle number.

**Differences Between
B-Cells
and
T-Cells**



Some of the differences between B Cells and T Cells are as follows:

S.N.	Properties	B-Cells	T-Cells
1	Name	B-lymphocytes	T-lymphocytes
2	Origin	Bone Marrow	Thymus
3	Position	Outside Lymph Node	Interior of Lymph Node
4	Membranereceptor	BCR (= immunoglobulin) for antigen	TCR for antigen
5	Connections	B-cells can connect to antigens right on the surface of the invading virus or bacteria.	T-cells can only connect to virus antigens on the outside of infected cells.
6	Tissue Distribution	Germinal centres of lymph nodes, spleen, gut, respiratory tract; also subcapsular and medullary cords of lymph nodes	Parafollicular areas of cortex in nodes, peri-arteriolar in spleen
7	Life Span	Life span is short	Life span is long
8	Surface Antibodies	Surface Antibodies present	Absence of surface antibodies
9	Secretion	They secrete antibodies	They secrete Lymphokines
10	Function	B-cells form humoral or antibody-mediated immune system (AMI). مناعة خلطية	T-cells form cell-mediated immune system (CMI). مناعة خلوية

S.N.	Properties	B-Cells	T-Cells
11	Blood	20% of lymphocytes	80% of lymphocytes; CD4 > CD8
12	Formation	They form plasma cells and memory cells.	They form killer, helper and suppressor cells.
13	Movement to Infection Site	Plasma cells do not move to the site of infection.	Lymphoblasts move to the site of infection.
14	Function	Plasma cells do not react against transplants and cancer cells.	Killer T-cells react against transplants and cancer cells.
15	Function	Plasma cells have no inhibitory effect on immune system.	Suppressor cells inhibit immune system.
16	Function	They defend against viruses and bacteria that enter the blood and lymph.	They defend against pathogens including protists and fungi that enter the cells.

Complement

- <https://youtu.be/2jjyiXq8toc>
- <https://youtu.be/mLnBqxltTf8>

Complement

The complement system consists of a number of small proteins found in the blood, normally circulating as inactive [zymogens](#). When stimulated by one of several triggers, [proteases](#) in the system cleave specific proteins to release [cytokines](#) and initiate an amplifying cascade of further cleavages. The end result of this activation cascade is massive amplification of the response and activation of the cell-killing [membrane attack complex](#).

What are the 3 main functions of the complement system?

- At the basic level the broad functions of the complement system can be split into three areas:
 - (1) the activation of inflammation.
 - (2) the opsonization (labeling) of pathogens and cells for clearance/destruction.
 - (3) the direct killing of target cells/microbes by lysis.

What are complements in blood?

Complement is a blood test that measures the activity of certain proteins in the liquid portion of your blood. The complement system is a group of nearly 60 proteins that are in blood plasma or on the surface of some cells.

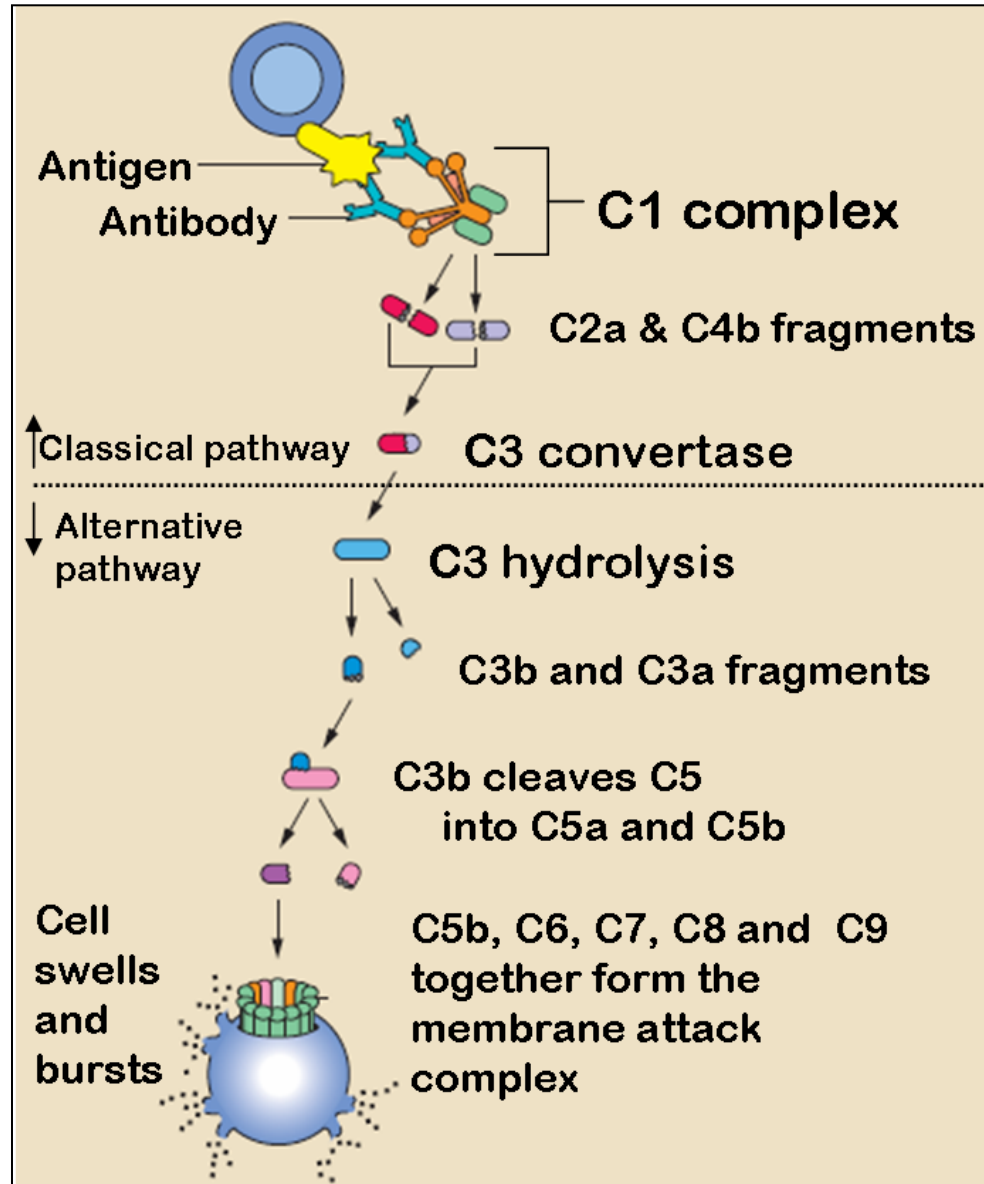
What are the three main effects of complement activation?

Its activation results in three major potential outcomes for microbes: cell lysis upon assembly and insertion of the terminal membrane attack complex (MAC), complement mediated opsonization, and the release of anaphylatoxins that enhance local inflammation

The complement cascade

What does the complement cascade do?

The complement cascade is a process where, once an antibody recognizes a foreign particle and binds to it, the component proteins become activated by each other in complex mechanisms, leading to the removal of the pathogen or foreign particle by phagocytosis at the end of the process.

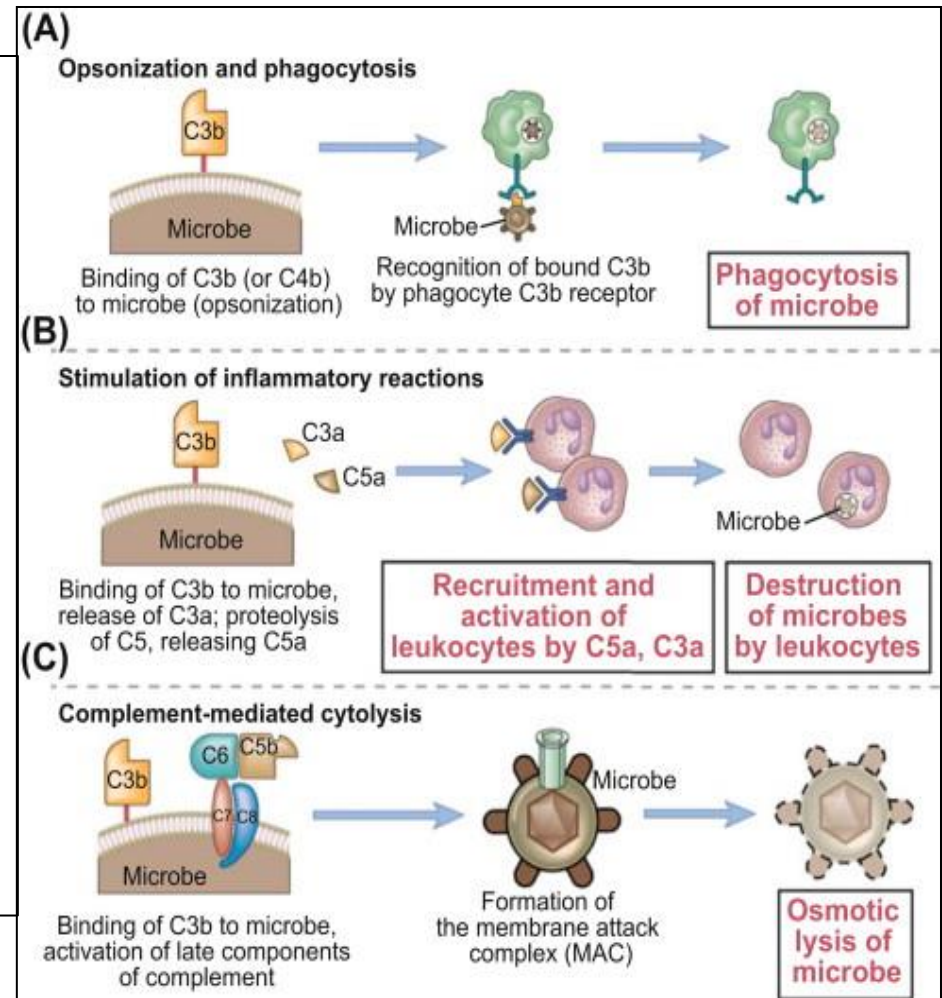


The major functions of complement.

(A) Complement fragment C3b on the surface of microbes (or other cells) promotes phagocytosis (also called opsonization).

(B) C3a and C5a proteolytic fragments increase vascular permeability and cause vasodilation by releasing histamine from mast cells; C5a is also chemotactic and enhances leukocyte binding to endothelium (shown here is a neutrophil), while stimulating leukotriene synthesis and the production of reactive oxygen species.

(C) The C5b–C9 complex forms a membrane attack complex (MAC) that punches holes in microbes (and other cells) leading to osmotic rupture.

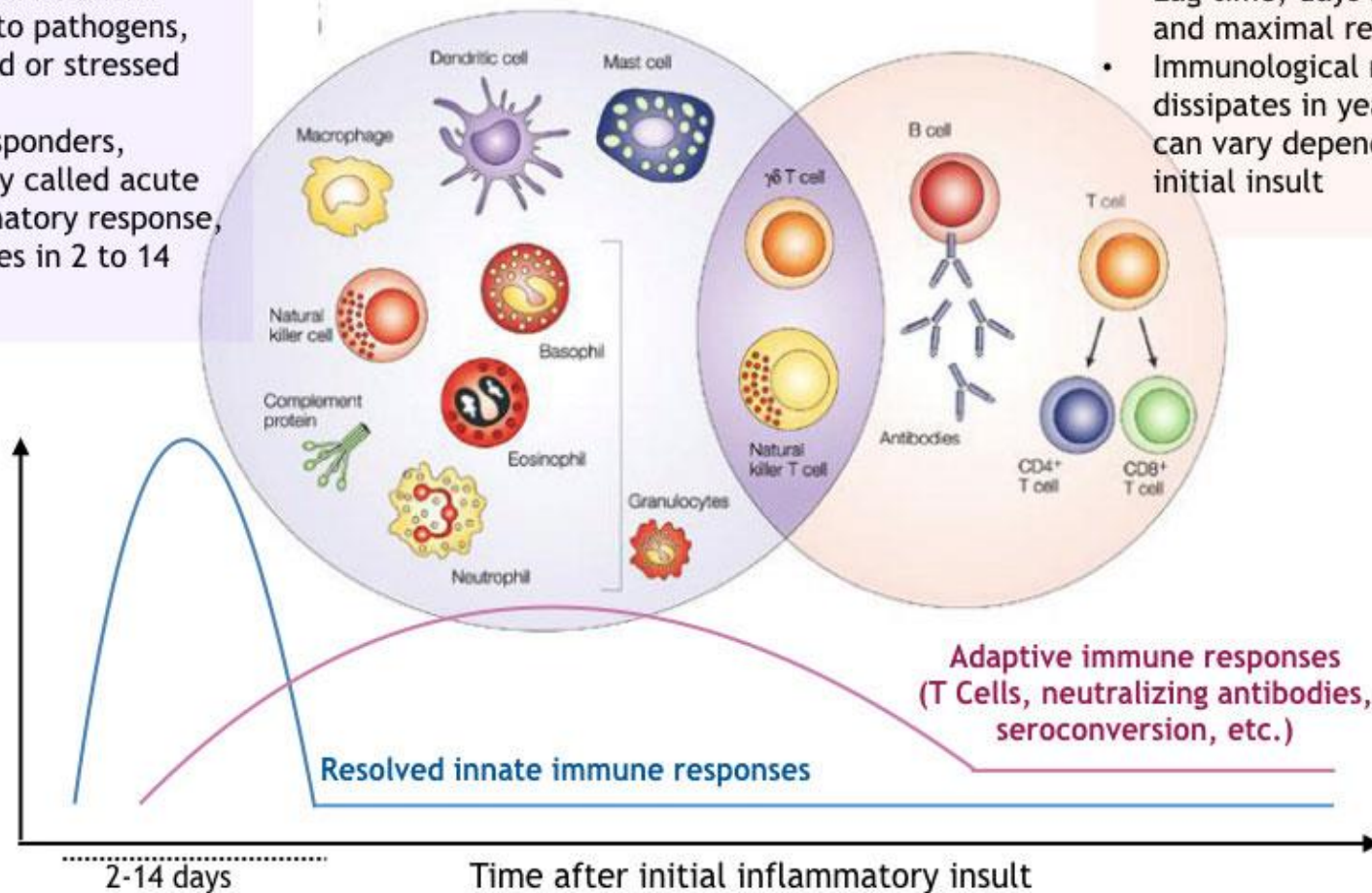


Innate

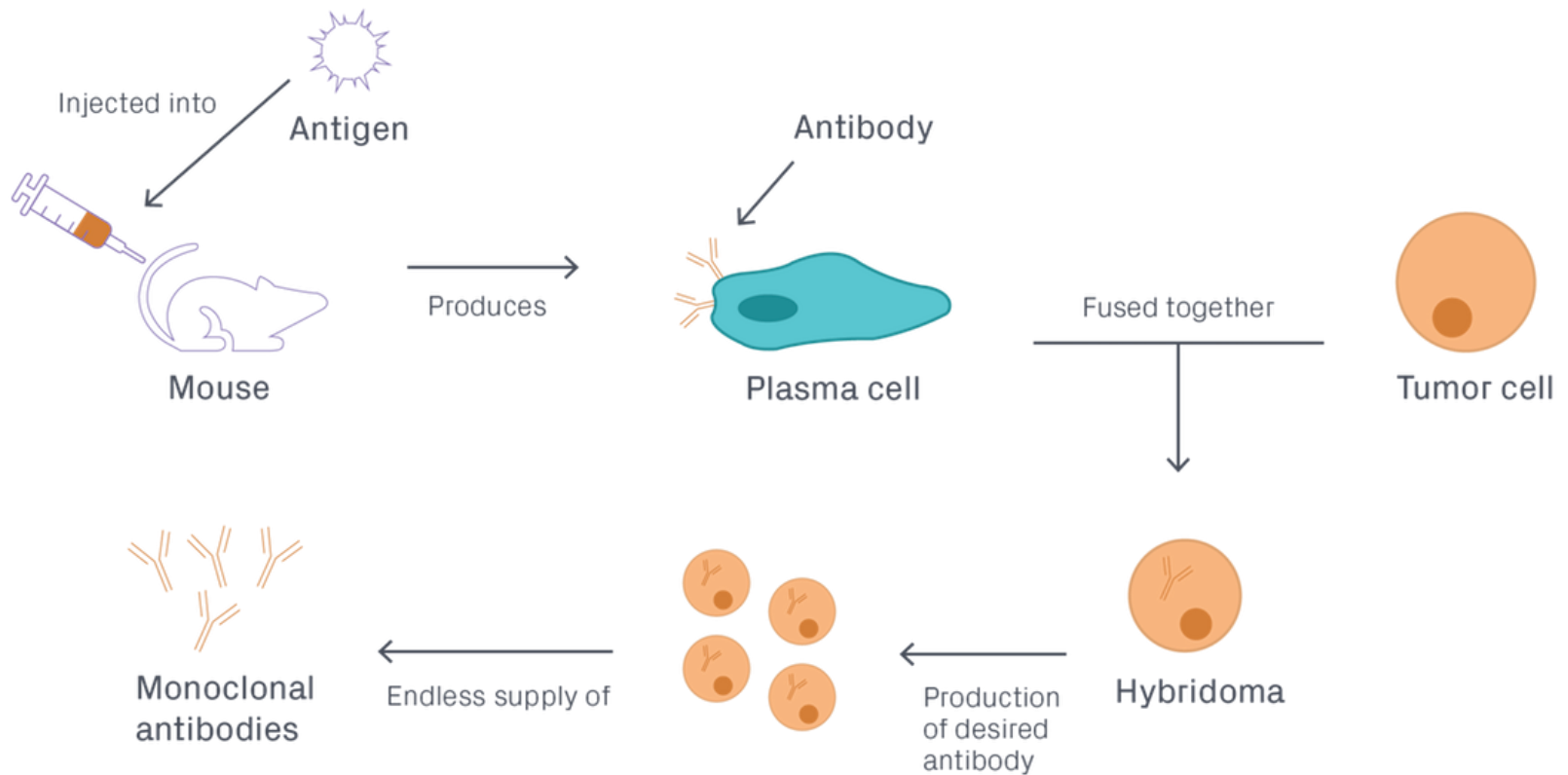
- Non-Specific alarm system to pathogens, damaged or stressed cells
- First responders, clinically called acute inflammatory response, dissipates in 2 to 14 days

Adaptive

- Lag time, days between exposure and maximal response
- Immunological memory, response dissipates in years not days, and can vary dependent upon the initial insult



Monoclonal antibodies



Antibodies are specialized proteins produced by the immune system that bind and neutralize foreign invaders such as viruses, bacteria, fungi, or parasites. **Monoclonal antibodies (mAbs)**, composed of unique pairs of heavy and light chains, have been widely used by researchers to target antigens with high specificity.

They have various applications in the diagnosis and treatment of a wide range of diseases including cancers, autoimmune disorders, and sexually transmitted infections.

In recent years, technological improvements in antibody design have significantly expanded mAb development. Decreasing immunogenicity in humans, improving bioavailability, optimizing affinity and antigen-binding specificity, and other advances in protein engineering have contributed to better profiles for therapeutic antibodies.

The amount of mAb needed and the importance of factors such as cost, turnaround time, and regulatory compliance depends on the purpose. Hybridoma technology, which uses an animal-based approach, has long been the industry standard for monoclonal antibody production. However, increased commercial demands and quality requirements have caused many companies to explore recombinant antibody production as an alternative. To help you decide which method of antibody production is the most appropriate for your needs, we will look at the process, benefits, and challenges of each one.

Hypersensitivity reactions

The four types of hypersensitivity are:

- Type I: reaction mediated by IgE antibodies.
- Type II: cytotoxic reaction mediated by IgG or IgM antibodies.
- Type III: reaction mediated by immune complexes.
- Type IV: delayed reaction mediated by cellular response.

Introduction

- The human **immune system** is vital for **defense against pathogens**, but at times it can ‘overreact’ causing undesirable consequences. The effects on the body from these reactions are not due to antigens directly but from the **inflammatory processes** generated via immune cells.
- When there is an overreaction to **exogenous** or ‘non-self’ antigens this can lead to **allergy**, and with **endogenous** or ‘self’ antigen this can cause **autoimmunity**.
- By these mechanisms, **hypersensitivity reactions** are implicated in the pathogenesis of many diseases, and appreciating the basic cellular processes helps to understand many conditions.
- There can be several factors that lead an individual to develop hypersensitivity. There may be a **genetic susceptibility** to these reactions or a **triggering event** of another kind on the immune system such as an infection.

Type I hypersensitivity

- Type I hypersensitivity is an **immediate reaction** (within minutes) mediated by **IgE antibody**, which results in **allergy, anaphylaxis and atopic disease**.
- When an individual first encounters an antigen, their immune system may produce large amounts of IgE antibodies against this specific substance. These IgE molecules attach themselves to **mast cells and basophils**. The individual is now 'sensitized' to the antigen.
- When this antigen is encountered again, it will cause cross-linking of the bound IgE and **degranulation** of mast cells and basophils, releasing potent vasoactive molecules such as **histamine**. This leads to the signs and symptoms of **allergy**, and if severe can cause **anaphylaxis**.

Type I hypersensitivity



anaphylaxis



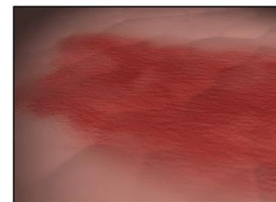
Atopic dermatitis



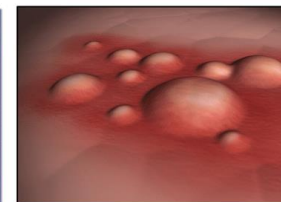
Eczema



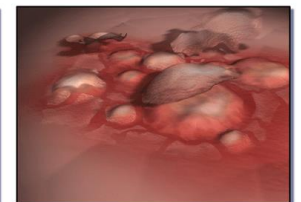
Redness



Blisters



Flaking



Common allergens associated with type I hypersensitivity

Proteins

Foreign serum
Vaccines

Plant pollens

Rye grass
Ragweed
Timothy grass
Birch trees

Drugs

Penicillin
Sulfonamides
Local anesthetics
Salicylates

Foods

Nuts
Seafood
Eggs
Peas, beans
Milk

Insect products

Bee venom
Wasp venom
Ant venom
Cockroach calyx
Dust mites

Mold spores

Animal hair and dander
Latex

IgE-mediated diseases in humans

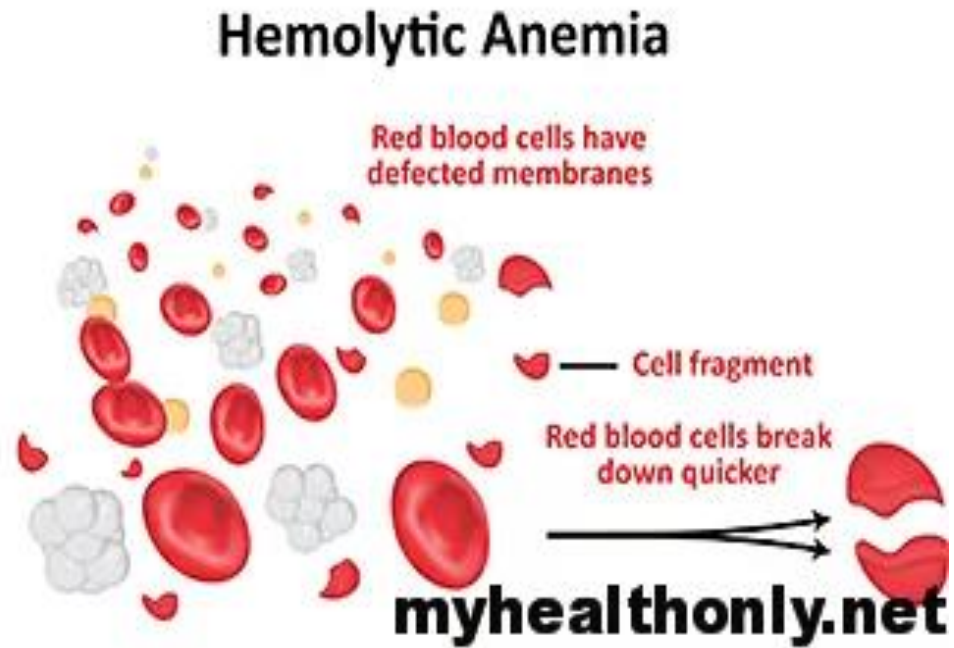
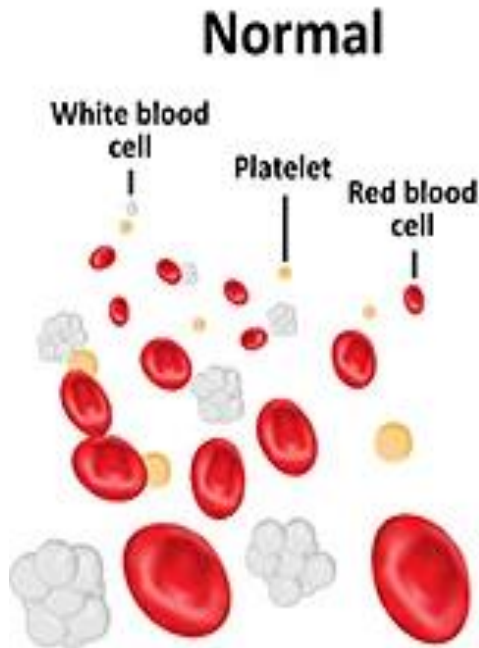
- Systemic (anaphylactic shock)
- Asthma
 - Classification by immunopathological phenotype can be used to determine management strategies
- Hay fever (allergic rhinitis)
- Allergic conjunctivitis
- Skin reactions
- Food allergies

Type II hypersensitivity

- Type II hypersensitivity is an **IgG or IgM** antibody-mediated **cytotoxic** reaction occurring in **hours to days**, which results in pathologies such as haemolytic disease of the newborn, autoimmune haemolytic anaemia and Good-pasture's syndrome.
- An individual may possess or develop IgG and IgM antibodies directed against **cell surface** or **extracellular matrix antigen**.
- These antibodies can cause damage to cells or tissues (**cytotoxicity**) either directly by cell surface **receptor binding**, via activation of the **complement pathway** or by antibody-dependent **cellular cytotoxicity**.
- Pathology is dependent on the target of the antibody. If antibodies are directed to cell surface antigen on red blood cells this can cause **haemolytic anaemia**, if they are targeted to type IV collagen in the basement membrane this can cause **Goodpasture's syndrome**.

Type II hypersensitivity

Hemolytic Anemia

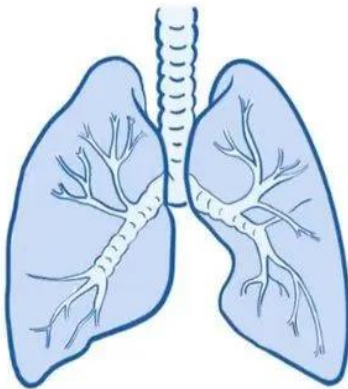


Type II hypersensitivity

GOODPASTURE SYNDROME

an AUTOIMMUNE DISEASE that AFFECTS

LUNGS



INFLAMMATION
& BLEEDING

* HEMOPTYSIS *
(coughing up blood)

KIDNEYS



* HEMATURIA *

blood urine

Type III hypersensitivity

- Type III hypersensitivity is an **antigen-antibody** immune complex-mediated reaction, which can occur over **hours, days or weeks**. Examples include serum sickness, rheumatoid arthritis (RA), [systemic lupus erythematosus](#) (SLE) and post-streptococcal glomerulonephritis.
- Soluble antigen in the circulation is **bound to by antibodies** (most commonly IgG and IgM) forming **immune complexes**. These complexes can precipitate out of the circulation and **deposit in certain tissues**, notably blood vessels, synovial joints and the glomerular basement membrane.
- These complexes trigger the **classical complement pathway**, leading to **the recruitment of inflammatory cells** including neutrophils that release enzymes and free radicals causing tissue damage. These inflammatory processes cause pathology in diseases such as rheumatoid arthritis, where immune complexes damage the filtration systems vital in synovial fluid formation.

Type III hypersensitivity

Signs And Symptoms Of Lupus



Poststreptococcal Glomerulonephritis

RoshReview



Group A Beta-Hemolytic
Streptococcus
(nephritogenic strain)

Strep pharyngitis

Strep skin infection

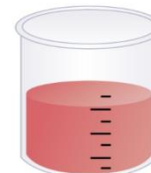


1–2 weeks



3–6 weeks

Glomerulonephritis



Hematuria



Hypertension



Periorbital edema

Type IV hypersensitivity

- Type IV hypersensitivity is also known as **delayed hypersensitivity**, as the reaction typically occurs **24 to 72 hours** after antigen exposure. Unlike types I to III, it is not antibody-mediated but **T cell-mediated**. It is involved in the processes of **contact dermatitis** and the **tuberculin skin test (Mantoux)**.
- When an individual first encounters an antigen, it can be processed by antigen-presenting cells and lead to **sensitisation of T helper cells**.
- On subsequent exposure to this antigen, these T helper cells will become activated and lead to an **inflammatory response** involving several immune cells such as macrophages, though there will be a **delay of 24 to 72 hours** as cells are recruited to the site of antigen exposure.
- This can cause local tissue inflammation and damage as seen in **contact dermatitis** when substances such as nickel or poison ivy contact the skin, or in the **Mantoux test** where proteins from *M. tuberculosis* are injected intradermally and an indurated area forms in individuals who have been previously exposed to the bacteria.

Mantoux Skin Test

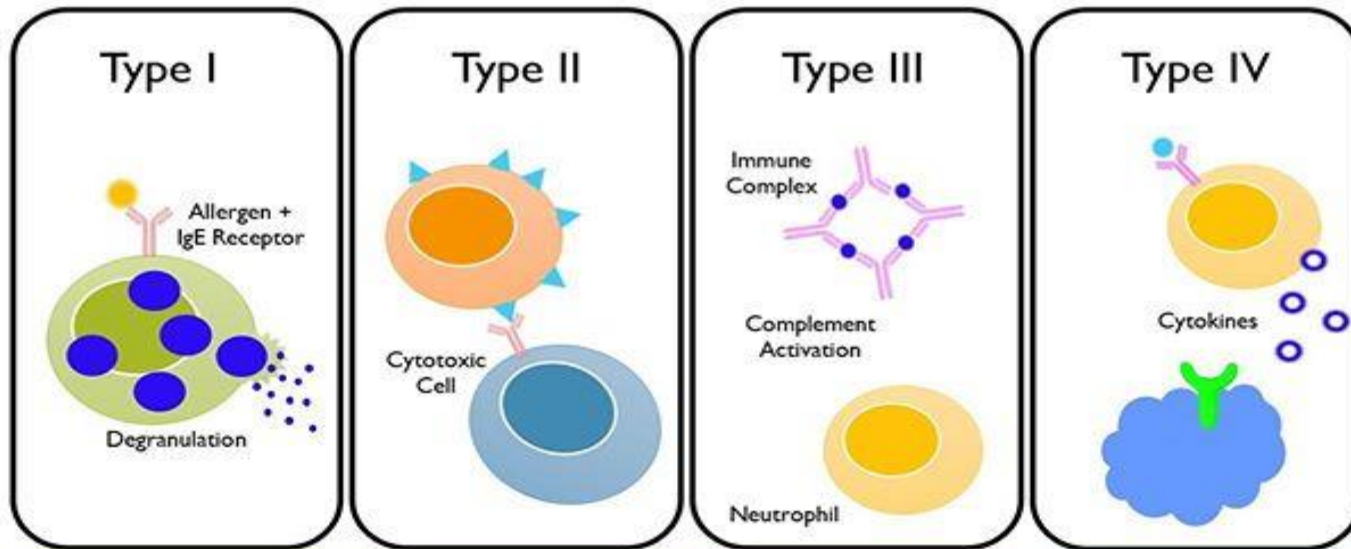


Key points

- The key features of the four types of hypersensitivity reaction.

Type	Mediated by	Timeframe	Examples
Type I hypersensitivity	IgE antibody	Immediate (minutes)	Allergy, anaphylaxis, atopy
Type II hypersensitivity	IgG or IgM antibody (cytotoxic)	Hours to days	Haemolytic disease of the newborn, autoimmune haemolytic anaemia, Goodpasture's syndrome
Type III hypersensitivity	Antigen-antibody immune complexes	Hours to days/weeks	Serum sickness, RA, SLE, post-streptococcal glomerulonephritis
Type IV hypersensitivity	T cells	Delayed (24 to 72 hours)	Contact dermatitis, tuberculin skin test

Types of Hypersensitivity Reactions



Mediators Onset Examples				
	IgE-Mediated	IgG or IgM Cytotoxic	Immune Complex-Mediated	T-Cell-Mediated
	Within 1 Hour	Hours to Days	1-3 Weeks	Days to Weeks
Examples	Anaphylaxis	Hemolytic Anemia	Serum Sickness SLE	Rash SJS

Factors in the development of allergic diseases

- Geographical distribution
- Environmental factors - climate, air pollution, socioeconomic status
- Genetic risk factors
- “Hygiene hypothesis”
 - Older siblings, day care
 - Exposure to certain foods, farm animals
 - Exposure to antibiotics during infancy
- Cytokine milieu



Oncogenic immunity

What is the definition of oncogenic?

- Definition of oncogenic
 - 1 : relating to tumor formation.
 - 2 : tending to cause tumors.

Does oncogenic mean cancer?

The name of oncogene suggests it is a gene that can cause cancer. Initially, oncogenes were identified in viruses, which could cause cancers in animals. Later, it was found that oncogenes can be mutated copies of certain normal cellular genes also called proto-oncogenes.

What are examples of oncogenes?

An example of an oncogene is the HER2 gene that makes HER2 protein. This protein helps control healthy breast cell division and growth. Extra copies of this gene may lead to an excess of HER2 protein, which causes cells to grow more quickly. The HER2 oncogene is found in some breast cancer and ovarian cancer cells.

What viruses are oncogenic?

Oncogenic DNA viruses include EBV, hepatitis B virus (HBV), human papillomavirus (HPV), human herpesvirus-8 (HHV-8), and Merkel cell polyomavirus (MCPyV). Oncogenic RNA viruses include, hepatitis C virus (HCV) and human T-cell lymphotropic virus-1 (HTLV-1).

What cells play a role in stopping cancers?

T-cells work in both direct and indirect ways to fight cancer. Killer T-cells kill cancer cells directly. 2 These cells first find cancer cells and can also be stimulated to kill cancer cells. Helper T-cells fight cancer indirectly.

What causes cells to become cancerous?

Cancer cells have **gene mutations** that turn the cell from a normal cell into a cancer cell. These gene mutations may be inherited, develop over time as we get older and genes wear out, or develop if we are around something that damages our genes, like cigarette smoke, alcohol or ultraviolet (UV) radiation from the sun.

What activates the immune system to destroy a tumor?

Immunotherapy is a type of cancer treatment. It uses substances made by the body or in a laboratory to boost the immune system and help the body find and destroy cancer cells. Immunotherapy can treat many different types of cancer. It can be used alone or in combination with chemotherapy and/or other cancer treatments.

How does the immune system fight cancer?

The immune system consists of a complex process that your body uses to fight cancer. This process involves cells, organs, and proteins. Cancer can commonly get around many of the immune system's natural defenses, allowing cancer cells to continue to grow.

Different types of immunotherapy work in different ways. Some immunotherapy treatments help the immune system stop or slow the growth of cancer cells. Others help the immune system destroy cancer cells or stop the cancer from spreading to other parts of the body.

The different types of immunotherapy include:

Monoclonal antibodies and immune checkpoint inhibitors

Non-specific immunotherapies

Oncolytic virus therapy

T-cell therapy

Cancer vaccines

Strategies to combat tumors based on immunotherapy,

passive.....

Active.....

What are cancer vaccines? (passive)

- A cancer vaccine can also help your body fight disease. A vaccine exposes your immune system to a foreign protein, called an antigen. This triggers the immune system to recognize and destroy that antigen or related substances. There are 2 types of cancer vaccine: prevention vaccines and treatment vaccines.
- One example of a cancer prevention vaccine is **Gardasil**, the vaccine to protect against the human papillomavirus (HPV).
- An example of a treatment vaccine includes **Spuleucel-T (Provenge)**, which treats advanced prostate cancer that does not respond to hormone therapy.
- T-VEC is also considered a cancer treatment vaccine. Side effects for both of these cancer vaccines are flu-like symptoms.



Which of the following are strategies for immunotherapy?

- Several different immunotherapy strategies are currently being studied or used as cancer treatments, including the following.
- Adoptive T-cell Transfer (T-cell Therapy) ...
- Immune Checkpoint Inhibitors. ...
- Monoclonal Antibodies. ...
- Nonspecific Immune Stimulation. ...
- Oncolytic virus immunotherapy. ...
- Vaccinations.

Auto-immune diseases.

What is an autoimmune disease?

- An autoimmune disease is a condition in which your immune system attacks your body.
- The immune system usually guards against bacteria and viruses. When it senses these foreign invaders, it sends out an army of fighter cells to attack them.
- Usually, the immune system can tell the difference between foreign cells and your own cells.
- In an autoimmune disease, the immune system mistakes part of your body, like your joints or skin, as foreign. It releases proteins called autoantibodies that attack healthy cells.
- Some autoimmune diseases target only one organ. Type 1 diabetes damages the pancreas. Other diseases, like systemic lupus erythematosus (SLE), or lupus, can affect the whole body.

Autoimmune responses are directed against self antigen

Autoimmune disease occurs when a specific adaptive immune response is mounted against self antigens.

The normal consequence of an adaptive immune response against a foreign antigen is the clearance of the antigen from the body.

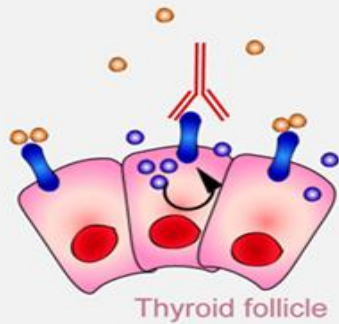
Virus-infected cells, for example, are destroyed by cytotoxic T cells, whereas soluble antigens are cleared by formation of immune complexes of antibody and antigen, which are taken up by cells of the mononuclear phagocytic system such as macrophages.

When an adaptive immune response develops against self antigens, however, it is usually impossible for immune effector mechanisms to eliminate the antigen completely, and so a sustained response occurs.

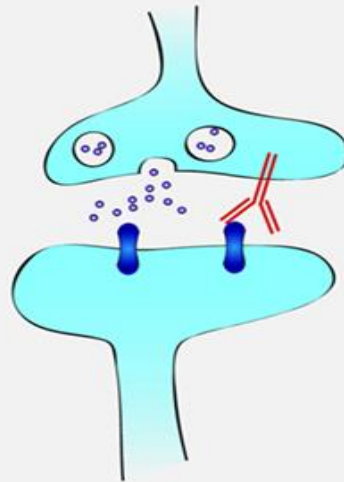
The consequence is that the effector pathways of immunity cause **chronic inflammatory injury to tissues**, which may **prove lethal**.

The mechanisms of tissue damage in autoimmune diseases are essentially the same as those that operate in protective immunity and in hypersensitivity diseases

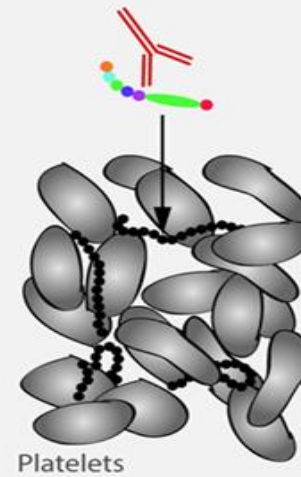
Mimickry of receptor activation by hormone



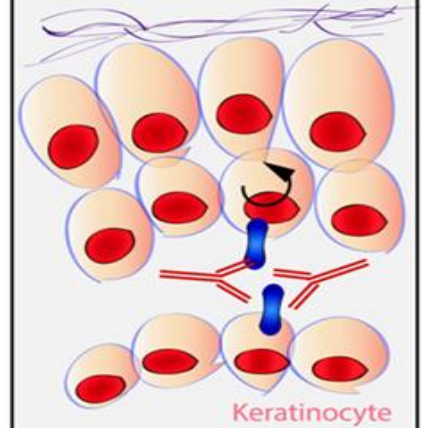
Blocking of neural transmission



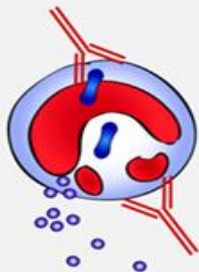
Blocking of enzymes/coagulation



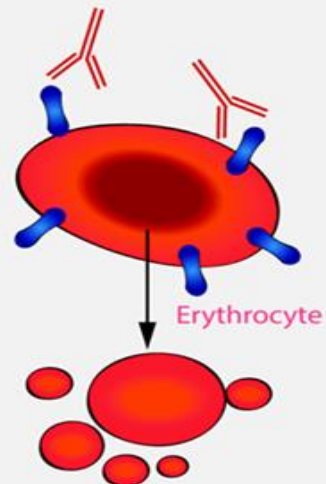
Altered cell signaling/acantolysis



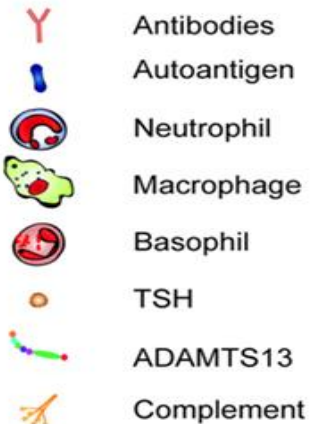
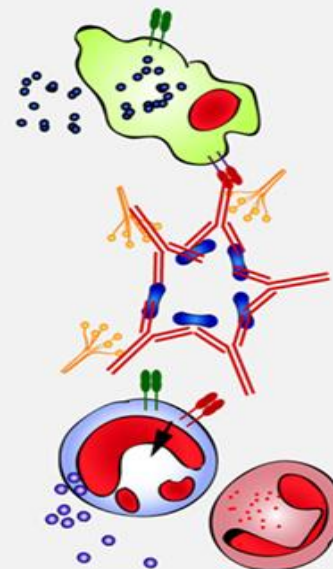
Neutrophil activation



Direct cell lysis



Induction of inflammation



MULTIPLE PATHWAYS LEAD TO AUTOANTIBODY-INDUCED PATHOLOGY.

1. Depending on the targeted auto-antigen : antibodies against the thyrotropin receptor (TSHR) mimic hormone stimulation of the TSHR receptor leading to hyperthyroidism.
2. Blockade of neural transmission by autoantibody binding to the corresponding receptors may lead to severe neurological diseases such as anti-N-methyl-d-aspartate encephalitis.
3. Autoantibody-mediated blockade of enzymes of the primary hemostasis may trigger uncontrolled microthrombosis.
4. In pemphigus, autoantibodies induce an altered signaling in keratinocytes, which either reflects or leads to, a loss of cell–cell adhesion, resulting in severe skin blistering.
5. Autoantibodies to antigens expressed by neutrophils can lead to their uncontrolled activation, resulting in severe tissue injury.
6. In autoimmune idiopathic thrombocytopenia autoantibodies trigger thrombocytopenia and severe bleeding.
7. Fc γ -mediated functions may trigger tissue inflammation in many autoimmune diseases, e.g., rheumatoid arthritis and pemphigoid disease.

Key words

Word & Definition	Example
Immunocompromised <i>An overarching term that means the immune system is weakened or not working properly.</i>	People who are taking certain medications to slow down the immune system or those who have conditions that impact the body's ability to fight off infections like HIV or genetic immunodeficiencies may be considered immunocompromised.
Immune dysregulated <i>This term means that the immune system isn't working properly, but doesn't have the connotation that you're at higher risk for infection.</i>	People with autoimmune disease may be considered immune dysregulated. For example, with T1D, your immune system isn't working properly because it's attacking your pancreas. But that doesn't necessarily increase your risk of infection.
Immunodeficiency <i>This means that your immune system isn't working properly, typically because important components (like cells) aren't working right, there's not enough of them or they're missing altogether.</i>	Conditions like HIV where T cells are severely damaged and genetic conditions where people are born with low levels of certain immune cells are immunodeficient. These conditions typically put people at higher risk for infections.
Immunosuppression/ Immunosuppressive therapies <i>Immunosuppression is the process of slowing down the immune system.</i>	Immunosuppressive therapies can also slow down the immune system in autoimmune diseases, which happen when the immune system mistakenly attacks healthy tissue. Stronger immunosuppressive therapies may be used with organ transplants to help keep the immune system from attacking a new organ.
Immunomodulatory/ Immunomodulating therapies <i>Immunomodulatory means changing or altering the immune system by speeding it up or slowing it down.</i>	Immunomodulatory therapies may slow down the immune system for conditions like autoimmune disease. They may also speed up the immune system. Some immunotherapy drugs can amp up the immune system to fight cancer.

14 common autoimmune diseases

1. Type 1 diabetes

The pancreas produces the hormone insulin, which helps regulate blood sugar levels. In type 1 diabetes mellitus, the immune system attacks and destroys insulin-producing cells in the pancreas.

High blood sugar results can damage the blood vessels and organs, including the heart, kidneys, eyes, and nerves.

2. Rheumatoid arthritis (RA)

In rheumatoid arthritis (RA), the immune system attacks the joints. This attack causes redness, warmth, soreness, and stiffness in the joints.

Unlike osteoarthritis, which commonly affects people as they get older, RA can start as early as your 30s or sooner.

3. Psoriasis/psoriatic arthritis

Skin cells grow and then shed when they're no longer needed. Psoriasis causes skin cells to multiply too quickly. The extra cells build up and form inflamed, red patches, commonly with silver-white scales of plaque on lighter-toned skin. On darker skin, psoriasis can appear purplish or dark brown with gray scales.

4. Multiple sclerosis

Multiple sclerosis (MS) damages the myelin sheath, the protective coating surrounding nerve cells in your central nervous system. Damage to the myelin sheath slows the transmission speed of messages between your brain and spinal cord to and from the rest of your body.

5. Systemic lupus erythematosus (SLE)

Although doctors in the 1800s first described lupus as a skin disease because of the rash it commonly produces, the systemic form, which is most common, actually affects many organs, including the joints, kidneys, brain, and heart.

6. Inflammatory bowel disease

Inflammatory bowel disease (IBD) describes conditions that cause inflammation in the lining of the intestinal wall. Each type of IBD affects a different part of the GI tract.

Ulcerative colitis affects only the lining of the large intestine (colon) and rectum.

7. Addison's disease

Addison's disease affects the adrenal glands, which produce the hormones cortisol and aldosterone as well as androgen hormones. Too little cortisol can affect how the body uses and stores carbohydrates and sugar (glucose). Deficiency of aldosterone will lead to sodium loss and excess potassium in the bloodstream. Symptoms include weakness, fatigue, weight loss, and low blood sugar.

8. Graves' disease

Graves' disease attacks the thyroid gland in the neck, causing it to produce too much of its hormones. Thyroid hormones control the body's energy usage, known as metabolism.

Having too much of these hormones revs up your body's activities, causing symptoms like nervousness, a fast heartbeat, heat intolerance, and weight loss.

9. Sjögren's syndrome

This condition attacks the glands that provide lubrication to the eyes and mouth. The hallmark symptoms of Sjögren's syndrome are dry eyes and dry mouth, but it may also affect the joints or skin.

10. Hashimoto's thyroiditis

In Hashimoto's thyroiditis, thyroid hormone production slows to a deficiency. Symptoms include weight gain, sensitivity to cold, fatigue, hair loss, and swelling of the thyroid (goiter).

11. Myasthenia gravis

Myasthenia gravis affects nerve impulses that help the brain control the muscles. When the communication from nerves to muscles is impaired, signals can't direct the muscles to contract.

The most common symptom is muscle weakness, which worsens with activity and improves with rest. Muscles that control eye movements, eyelid opening, swallowing, and facial movements are often involved.

12. Autoimmune vasculitis

Autoimmune vasculitis happens when the immune system attacks blood vessels. The inflammation that results narrows the arteries and veins, allowing less blood to flow through them.

13. Pernicious anemia

This condition causes a deficiency of a protein made by stomach lining cells, which is an intrinsic factor needed for the small intestine to absorb vitamin B12 from food. Without enough of this vitamin, one will develop anemia, and the body's ability for proper DNA synthesis will be altered.

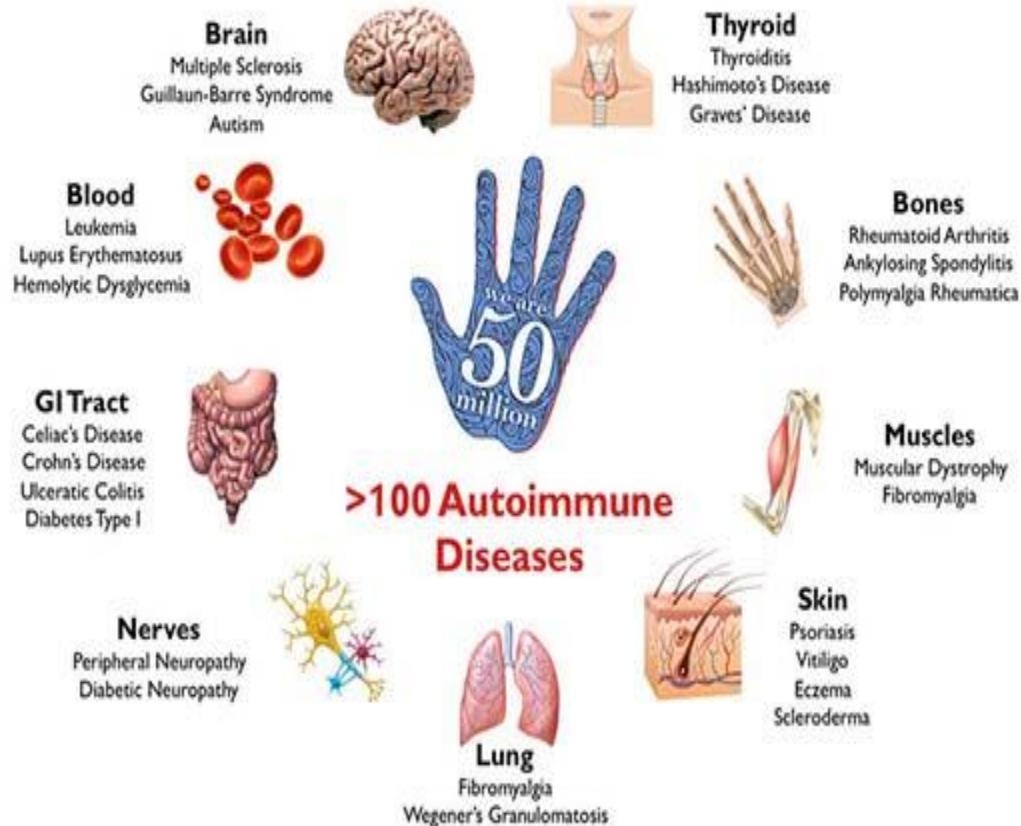
14. Celiac disease

People with celiac disease can't eat foods containing gluten, a protein found in wheat, rye, and other grain products. When gluten is in the small intestine, the immune system attacks this part of the gastrointestinal tract and causes inflammation.

How are autoimmune diseases treated?

- Treatments can't cure autoimmune diseases, but they can control the overactive immune response and bring down inflammation or at least reduce pain and inflammation. Drugs used to treat these conditions include:
 - nonsteroidal anti-inflammatory drugs (NSAIDs), such as ibuprofen (Motrin, Advil) and naproxen (Naprosyn)
 - immune-suppressing drugs
 - Treatments are also available to relieve symptoms like pain, swelling, fatigue, and skin rashes.
- Eating a well-balanced diet and getting regular exercise may also help you feel better.

Autoimmune Diseases



thank
you!

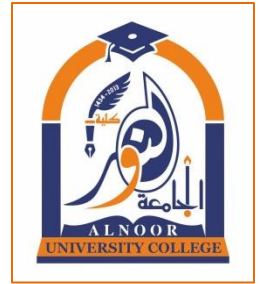
Alnoor University Collage

□ كلية النور الجامعة

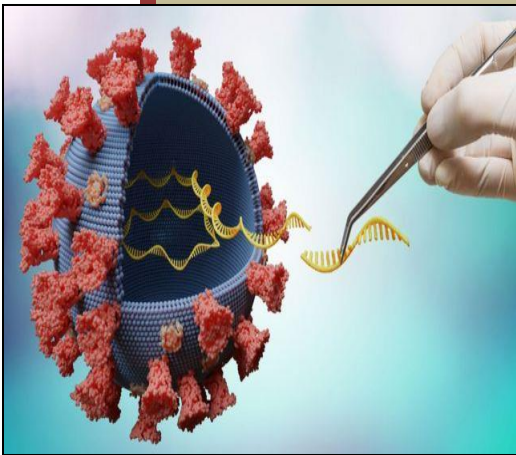
Pharmacy Department

□ قسم الصيدلة

Medical Microbiology II



فائروسات DNA

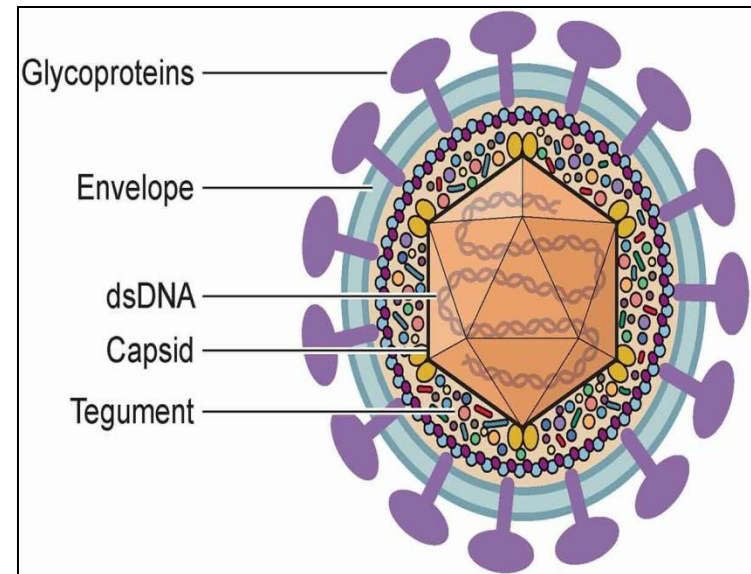


Nada Khairi Younus Al-Asaadi

M.Sc. Medical microbiology

Herpesviridae

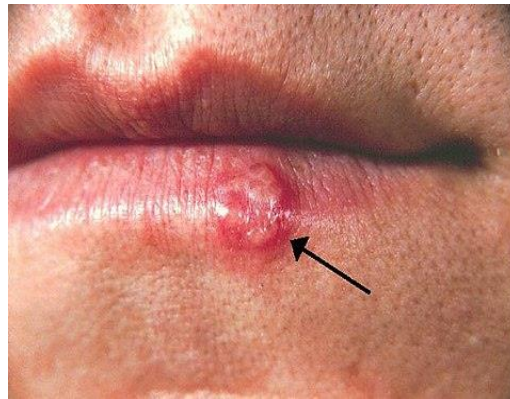
- is a large family of DNA viruses that cause infections and certain diseases in animals, including humans.
- The members of this family are also known as **herpes-viruses**. The family name is derived from the Greek word referring to spreading cutaneous lesions, usually involving blisters, seen in flares of herpes simplex 1, herpes simplex 2 and herpes zoster ([shingles](#))



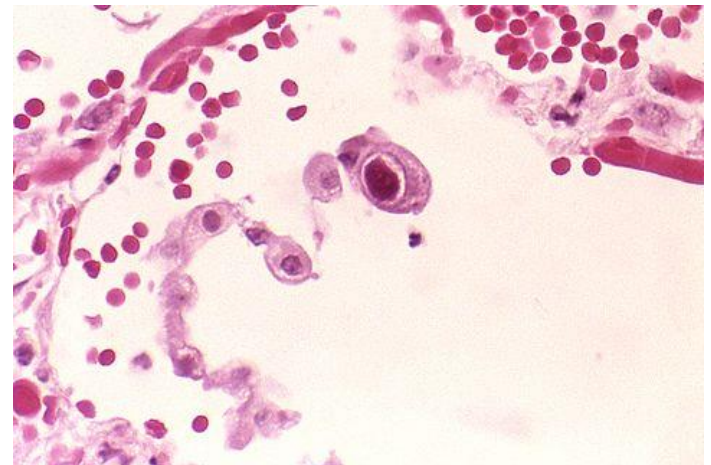
- Herpesvirus types are known to primarily infect humans and cause common diseases:
 1. Herpes simplex 1 and 2 (HSV-1 and HSV-2, also known as HHV-1 and HHV-2; both of which can cause orolabial and genital herpes).
 2. Varicella zoster (or HHV-3; the cause of chickenpox and shingles).
 3. Epstein–Barr (EBV or HHV-4; implicated in several diseases, including mononucleosis and some cancers).
 4. Human cytomegalovirus (HCMV or HHV-5).
 5. Human herpesvirus 6A and 6B (HHV-6A and HHV-6B).
 6. Human herpesvirus 7 (HHV-7).
 7. Kaposi's sarcoma-associated herpesvirus (KSHV, also known as HHV-8).



Shingles
Zoster
Herpes zoster



Herpes labialis
Cold sores
Fever blisters



Human beta herpes virus
HCMV
Human cytomegalovirus



Kaposi's sarcoma-associated herpes virus
(KSHV, also known as HHV-8)

Herpes virus

Virus	Disease	Signs/ Symptoms
HSV1, HSV2	Herpes simplex	Sores or ulcerations, fever blisters, flu-like discomfort
VSZ	Chickenpox, Shingles	Skin rash, blisters, fever, pain, sore throat, headache, stomach ache
CMV	CMV infections, Mononucleosis	Fever, rash, sore throat, nausea, muscle aches, swollen glands, fatigue
EBV	EBV infectious mononucleosis, associated with Burkitt's lymphoma and other malignancies	Fever, rash, sore throat, nausea, muscle aches, pain, swollen lymph nodes, fatigue, weight loss, vomiting
HHV6, HHV7	Roseola (exanthem subitum)	Fever, swollen glands, runny nose, mild diarrhea, swollen eyelids, fatigue, rash
KSHV	Kaposi's sarcoma (KS)	Characteristic skin lesions, lymphoma, non-specific symptoms (fever, weight loss, etc.)

Transmission

Most human herpes-viruses are transmitted from person-to-person when a susceptible individual has direct physical contact with an infected person. Some herpes-viruses may also spread through airborne transmission. The transmission of human herpes-viruses is summarized in the following table.

Virus	Transmission
HHV-1 (HSV1)	Contact with lesions and body fluids.
HHV-2 (HSV2)	Sexual, infection at birth by a genitally-infected mother.
HHV-3 (VZV)	Contact, respiratory route.
HHV-4 (CMV)	Saliva, sexual (probable), transplacental.
HHV-5 (EBV)	Infected body fluids (urine, saliva), transplacental, transplantation, blood transfusion.
HHV-8 (KSHV)	Saliva, sexual.

- **Treatment**

- Herpesviruses, which are able to remain latent, cause recurring infections and cannot be completely eliminated from the host. Although herpes infections cannot be cured, antiviral drug treatments are available to reduce viral shedding.
- Infections are treated with various antiviral drugs such as **acyclovir**, **valacyclovir**, **famciclovir**, **ganciclovir**, etc.



Poxviridae

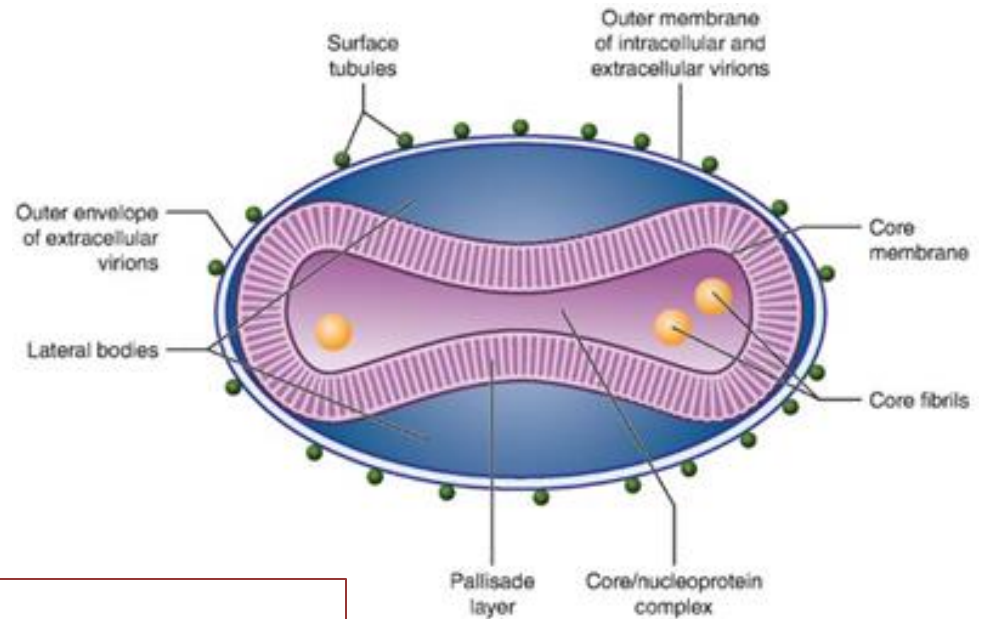
- Poxviridae is a family of double-stranded DNA viruses. Vertebrates and arthropods serve as natural hosts.
- The largest and most complex viruses that occur in humans, birds, animals, and insects.
- Poxviruses (family Poxviridae) are large, brick-shaped or ovoid double-stranded DNA viruses of about 200–300 nm in diameter with a complex structure.
- Include a large group of DNA viruses that are morphologically similar and share a common nucleocapsid protein
- They cause primarily vesicular lesions in the host.
- They replicate in the cytoplasm of vertebrate or invertebrate cells
- These viruses are of special interest because of their unique biologic properties and impact on human health.
- Smallpox is a major disease among all. It is also known as variola virus.
- The primary reason for infection in humans is due to its ability to evade the host immune responses and avoid complement activation.
- The name smallpox is derived from the Latin word “spotted” and refers to the raised bumps that appear on the face and body of an infected person.
- The disease provides at least three ‘firsts’: the first vaccine, the first disease to be totally eradicated by immunization, and the first virus infection against which chemotherapy was clinically effective.

Both الجذري وجذري الماء chicken pox and small pox

- Chickenpox and smallpox are both diseases that produce rashes on the skin, but they are different. For one thing, smallpox is a much more serious disease, causing severe illness and death. They are caused by different viruses.

S.NO.	SMALLPOX	CHICKENPOX
1.	Smallpox is defined as a disease that begins with fever resulting in blisters on the skin.	On the other hand, chickenpox is a contagious disease that results in rashes on the skin.
2.	Smallpox is a rare disease that ended in the year 1980.	Chickenpox is still seen in various parts of the world. The vaccine for chickenpox first came in the year 1995.
3.	The causative agent of smallpox is the variola virus.	The causative agent of chickenpox is the varicella-zoster virus.
4.	The incubation period of smallpox is 7-17 days.	The incubation period of chickenpox is 14-21 days.
5.	Smallpox is a fatal disease than chickenpox.	Chickenpox is less threatening as compared to smallpox.
6.	The blisters first appear in the throat, mouth, and face.	The blisters first appear on the face, chest, and back.
7.	The initial symptoms of smallpox are seen after 2-3 days of serious illness.	The initial symptoms of chickenpox are seen after two days of mild illness.
8.	The scabs in smallpox can be infectious.	The scabs in chickenpox are not infectious.
9.	The development of rashes leads to fever.	Fever occurs due to the collapse of vesicles.

جدري الماء Small pox



What caused smallpox?

Before smallpox was eradicated, it was a serious infectious disease caused by the variola virus. It was contagious—meaning, it spread from one person to another. People who had smallpox had a fever and a distinctive, progressive skin rash.

Treatment

Treatment for Smallpox



- ▶ No cure or treatment against smallpox exists
- ▶ Antiviral drugs may help treat smallpox
- ▶ Routine vaccination may help prevent smallpox

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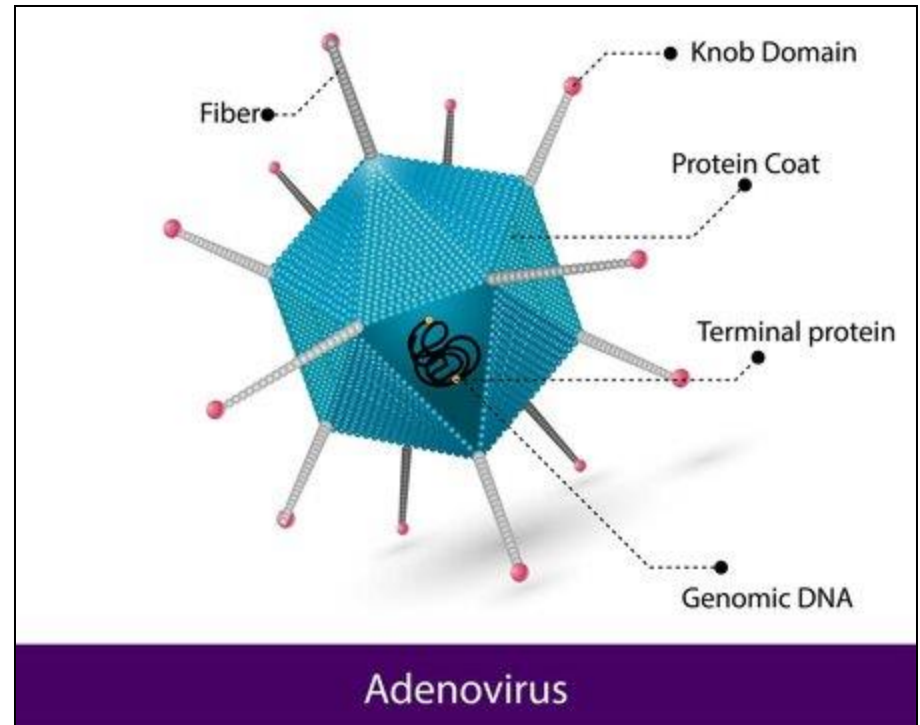


Adenoviridae

- Adenoviruses are a group of viruses that typically cause respiratory illnesses, such as a common cold, conjunctivitis (an infection in the eye that is sometimes called pink eye), croup, bronchitis, or pneumonia.
- These viruses typically cause mild cold- or flu-like illness.
- There is no specific treatment for people with adenovirus infection. Most adenovirus infections are mild and may require only care to help relieve symptoms, such as over-the-counter pain medicines or fever reducers. Always read the label and use medications as directed.

- Adenoviruses are medium-sized (90-100 nm), non-enveloped icosahedral viruses with double-stranded DNA.

Disease
Acute respiratory disease
Acute febrile pharyngitis
Endemic
Epidemic
Pneumonia
Pharyngoconjunctival fever
Acute follicular conjunctivitis
Epidemic keratoconjunctivitis
Gastroenteritis and diarrhea
Intussusception
Acute hemorrhagic cystitis
Disseminated infection





Adenovirus

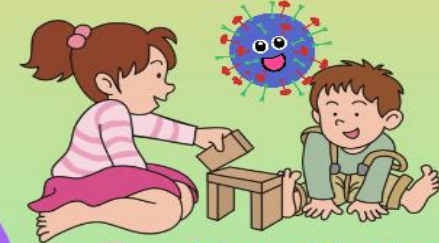


Infection can occur at any age.

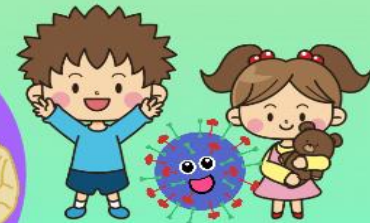
Severe infection can occur in infants and patients with weakened immune system or previous heart or lung disease



Close contact:
Touching or shaking hands



Touching objects with adenovirus on them, then touching mouth, nose or eyes,



Air: Coughing and sneezing

SYMPTOMS



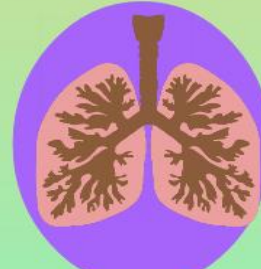
Common cold



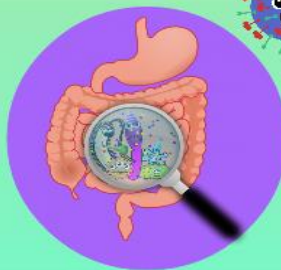
Bronchitis



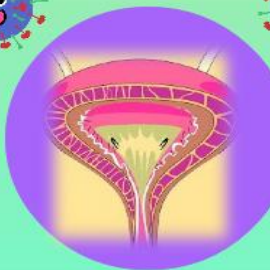
Fever



Pneumonia



Gastroenteritis:
Inflammation of stomach and intestine



Bladder inflammation



Conjunctivitis



Neurologic disease:
Affecting brain and spinal cord

#roypath

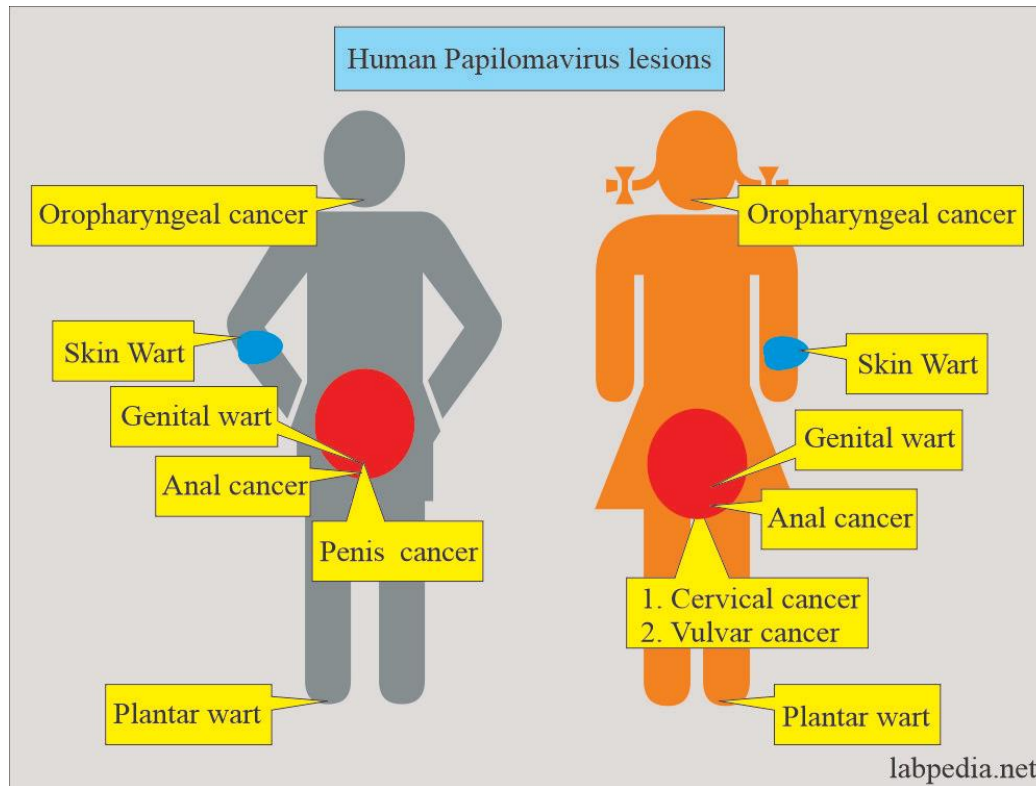
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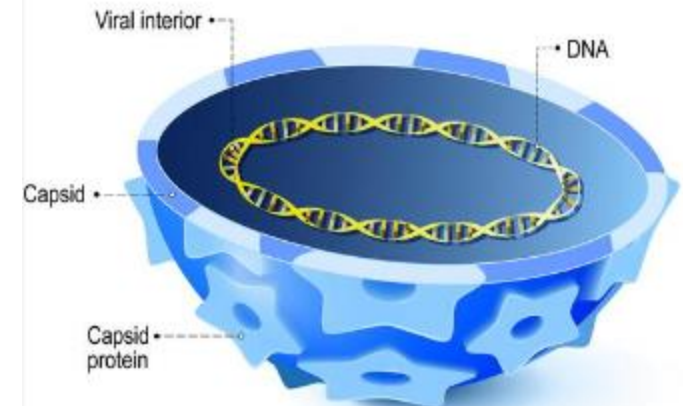
Is there a vaccine for adenovirus?

- There is currently no adenovirus vaccine available to the general public. Adenovirus vaccine contains live adenovirus Type 4 and Type 7. It will prevent most illness caused by these two virus types. The vaccine comes as two tablets, taken orally (by mouth) at the same time.

Papoviridae - Human Papillomavirus (HPV)



HPV
human papillomavirus



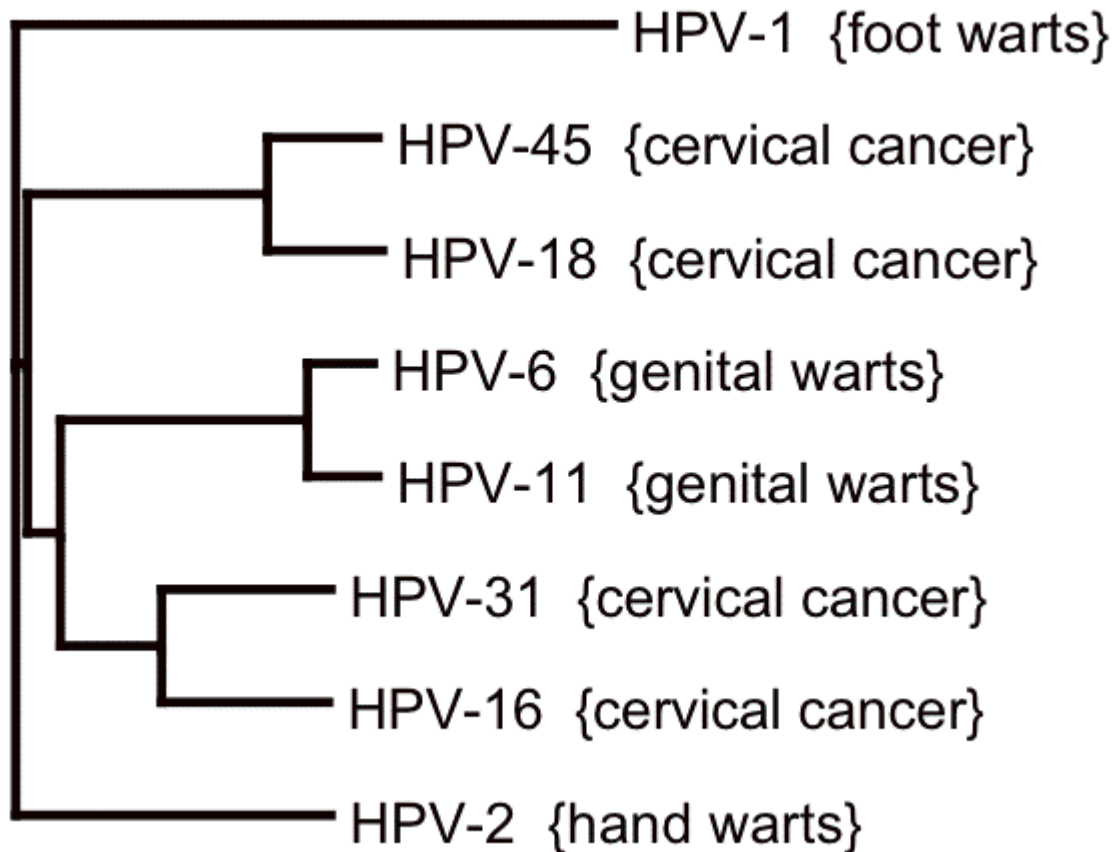
- **What Is HPV?**

- HPV is a sexually transmitted infection. HPV stands for human papillomavirus. It's very common. Many people don't have any symptoms, and the infection might go away on its own. But some types of HPV can lead to cervical cancer, head and neck cancer, or cancer of the anus or penis.
- HPV isn't just one virus. There are more than 100 kinds, and some are riskier than others.

- **Types of HPV**

- Each human papillomavirus has its own number or type. The term "papilloma" refers to a kind of wart that results from some HPV types.
- HPV lives in thin, flat cells called epithelial cells. These are found on the skin's surface. They're also found on the surface of the vagina, anus, vulva, cervix and head of the penis. They're also found inside the mouth and throat.
- About 60 of the 100 HPV types cause warts on areas like the hands or feet. The other 40 or so enter the body during sexual contact. They're drawn to the body's mucous membranes, such as the moist layers around the anus and genitals.
- **High-risk HPVs** can cause several types of cancer. There are about 14 high-risk HPV types including HPV 16, 18, 31, 33, 35, 39, 45, 51, 52, 56, 58, 59, 66, and 68. Two of these, **HPV16** and **HPV18**, are responsible for most HPV-related cancers.

HPV types and associated diseases



- The symptoms of a low-risk HPV infection are **warts**. The kind of warts you get will depend on which kind of HPV you have.
- Genital warts. These are either flat spots or raised bumps. In women, they usually grow on the vulva, but can also show up on the anus, cervix, or vagina. Men get them on the penis, scrotum, or anus.
- Common warts. These rough bumps typically show up on the hands and fingers.
- Plantar warts. Plantar warts are hard, grainy, painful bumps that affect the bottom of your feet.
- Flat warts. These are slightly raised spots with a flat top. You can get them anywhere, but they're common on the face and legs.
- HPV Causes and Risk Factors
- The human papillomavirus infects you by entering your body through a cut, scrape, or tear in your skin. You get it from skin-to-skin contact, or vaginal, anal, or oral sex. You can pass HPV to your baby if you have genital warts when you're pregnant. In rare cases, this can cause a noncancerous growth in your baby's voice box (larynx).
- The warts are **contagious**. You can get them by touching someone else's wart, or by touching a surface that came into contact with one.

Certain things make your chances of getting HPV go up. They include:

- **Damaged skin**. Places on your skin that have been cut a lot or have holes are more likely to get common warts.
- **Direct contact**. If you touch someone's warts or come into contact with surfaces warts have touched, you can get HPV.
- **Number of sexual partners**. The more sexual partners you have, the higher your risk of getting HPV. If you have sex with someone who has many partners, that increases your risk, too.
- **Age**. Children are more likely to get common warts. Genital warts are more common in adolescents and young adults.
- **Weak immune system**. If you have a condition such as HIV or AIDS, or are on treatment that weakens your immune system, you're more likely to get HPV.

HPV Treatments

- Warts may go away without treatment, especially in kids. But there are also medications that treat them, including:
 - **Salicylic acid.** You put treatments with this ingredient directly on the wart. They destroy the wart one layer at a time. You shouldn't use it on your face.
 - **Imiquimod.** This is a prescription cream that helps your immune system get rid of HPV. It can cause some redness and swelling around the area you apply it.
 - **Podofilox.** You apply this gel directly to genital warts to destroy their tissue. You may get some burning and itching from it.
 - **Trichloroacetic acid.** This burns off warts on your palms, soles of your feet, and genitals. It may irritate your skin.

Usually your doctor will recommend medication first. If that doesn't work, they can remove them with:

- Cryotherapy (freezing with liquid nitrogen)
- Electrocautery (burning with an electric current)
- Surgery
- Laser surgery (using intense light to destroy warts and abnormal cells)

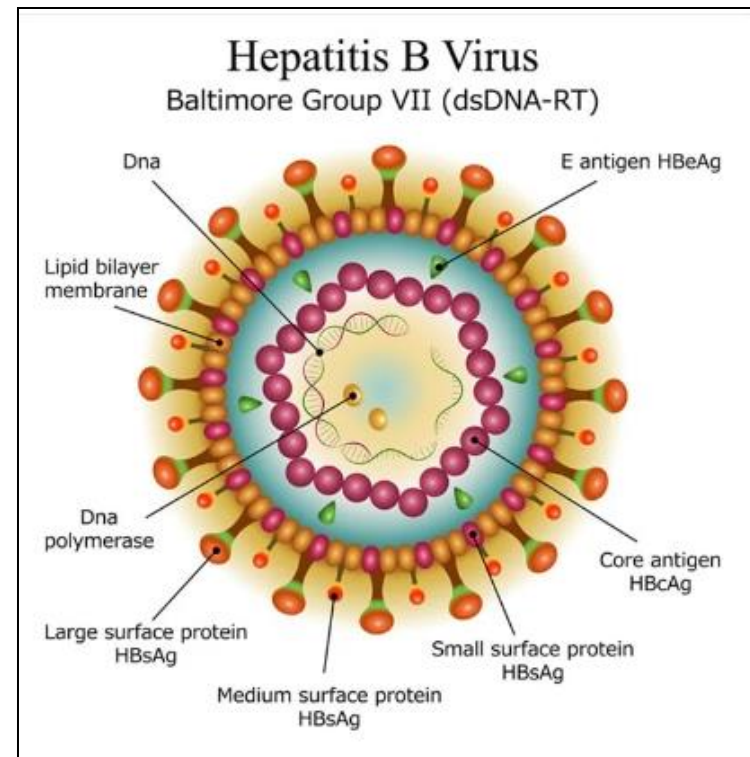
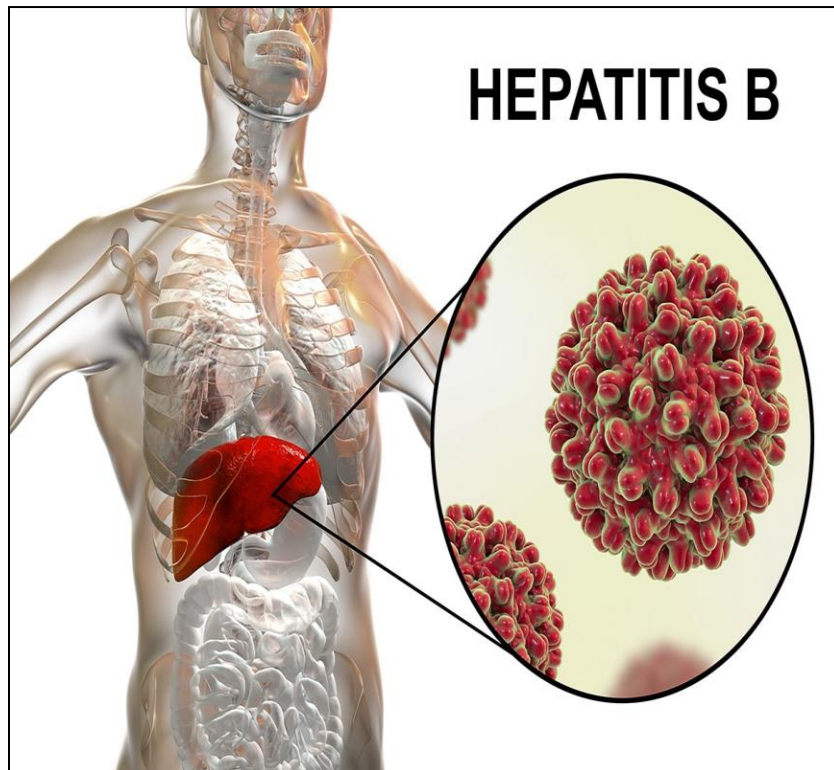


Hepadna virus

HBV

Hepatitis B is a potentially life-threatening liver infection caused by the hepatitis B virus (HBV). It is a major global health problem. It can cause chronic infection and puts people at high risk of death from cirrhosis and liver cancer.

A safe and effective vaccine that offers 98% to 100% protection against hepatitis B is available. Preventing hepatitis B infection averts the development of complications including chronic disease and liver cancer.



What is hepatitis B?

- Hepatitis B is a viral infection that affects your liver. It causes inflammation in your liver tissues, which is what “hepatitis” means. It begins as an acute infection that’s usually short-lived. But in some people, it turns into a chronic infection that never goes away.
- Long-term inflammation does serious damage to your liver over time. It can lead to **cirrhosis** and **liver failure**. Like other chronic liver diseases, hepatitis B infection can do this damage without causing symptoms.

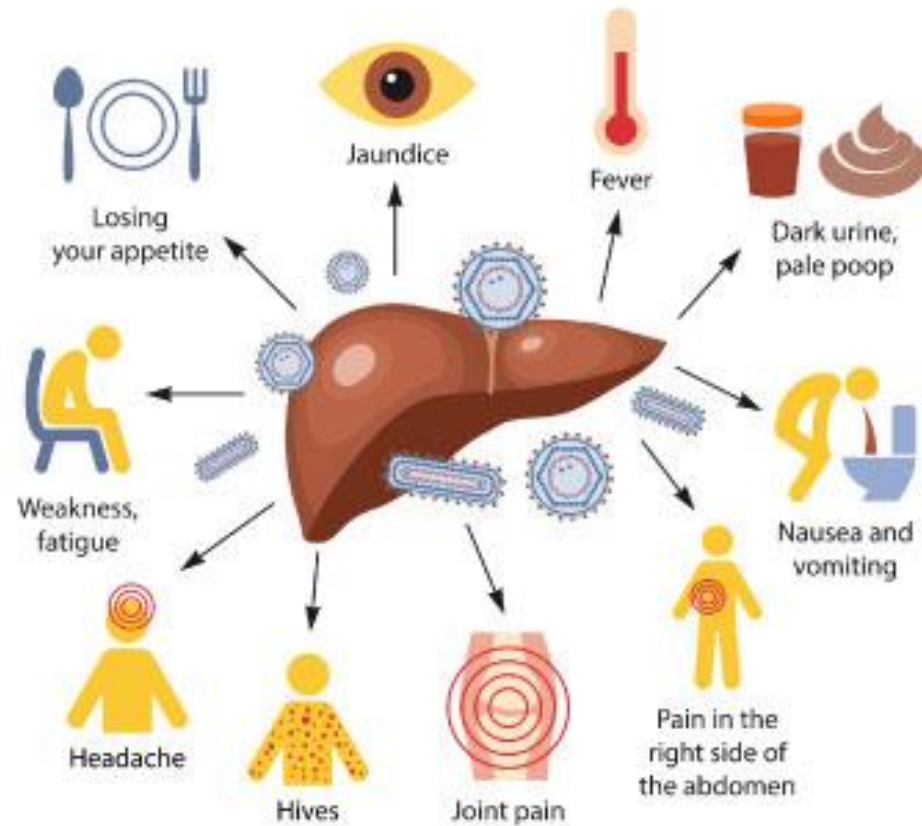
Why is it called “hepatitis B”?

- There are several different viruses that can infect your liver and cause inflammation (hepatitis). They include **hepatitis A, B, C, D** and **E**.

What are the symptoms of hepatitis B infection?

- Typical symptoms of infection include:
- Fever, Loss of appetite, Nausea and vomiting, Abdominal pain, Weakness and fatigue, Joint pain.
- Also there are symptoms of **liver disease**, including:
 - 1) Jaundice (yellowing of your skin and the whites of your eyes).
 - 2) Dark-colored urine.
 - 3) Light or clay-colored poop.
 - 4) Swelling with fluid in your belly or arms and legs.

SYMPTOMS OF HEPATITIS B



How do you get hepatitis B?

- Hepatitis B infection comes from the hepatitis B virus (HBV). The virus spreads through bodily fluids. Transmission occurs when fluids from the body of a person who's infected enter the body of a person who's uninfected. This might happen through:
 - 1) Childbirth.
 - 2) Sexual contact.
 - 3) Contact with an open wound.
 - 4) Sharing needles or syringes.
 - 5) Sharing a toothbrush or razor.
 - 6) Accidental stick from an infected sharp instrument.

Who is more likely to get hepatitis B?

- 1) People with HIV [AIDS (acquired immunodeficiency syndrome)].
- 2) People who use intravenous drugs.

Complications of chronic hepatitis B can include:

- 1) **Hepatitis D.** Hepatitis D, or delta virus, is another viral hepatitis infection that only affects people with hepatitis B.
- 2) **Cirrhosis.** Chronic liver inflammation leads to cirrhosis in some people. Cirrhosis happens when injured liver tissues are gradually replaced with scar tissue. The scar tissue stops your liver from functioning, which leads to chronic liver failure.
- 3) **Chronic liver failure.** Chronic liver failure is a gradual process where the liver loses its ability to function over time
- 4) **Liver cancer.** For reasons that aren't entirely clear, people with chronic hepatitis are more likely to develop primary liver cancer.

These treatments include:

- 1) Vaccination.
- 2) Hepatitis B immune globulin (HBIG).
- 3) Antiviral medications. Several antiviral medicines — including entecavir (Baraclude), **tenofovir** (Viread), **lamivudine** (Epivir), adefovir (Hepsera) and telbivudine — can help fight the virus and slow its ability to damage your liver. These drugs are taken by mouth.

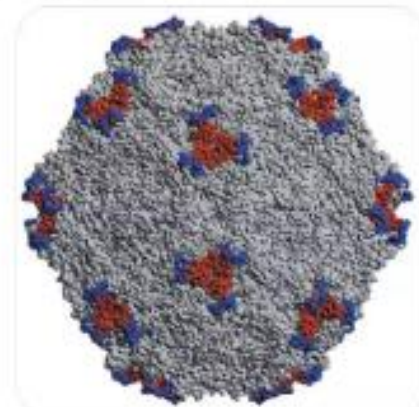


Parvoviridae

B19

Parvovirus B19

- Family: *Parvoviridae*
 - Latin *parvus* means small
- ~20 nm in diameter
 - (0.02 μm)
- Single-stranded DNA virus
- Icosahedral capsid
- No envelope
- Only known human parvovirus



X-ray crystallographic image of parvovirus

Body source & Transmission

- Replication in human cells restricted to erythroid progenitor cells
 - Adult bone marrow
 - Fetal liver
- Transmission by close contact
 - Airborne droplets
 - 50% of a household may become infected
 - 10-60% of students in school outbreaks

Environmental Habitat

- Wide distribution among warm-blooded animals
- B19 is only known HUMAN parvovirus



What is the pathogenesis of parvovirus B19?

Parvovirus B19 is a non-enveloped virus that binds to host cell receptors in the respiratory tract and enters the cell. It then translocate its genome to the host nucleus, whereby DNA replication, RNA transcription, and assembly of the virus occurs. Lastly, the cells lyse and release the mature virions.

How does parvovirus B19 cause anemia?

Parvovirus B19 infection can trigger an acute cessation (stop) of red blood cell production, causing **transient aplastic crisis**, chronic red cell aplasia, hydrops fetalis, or congenital anemia.

Pathogenicity

B19

Pathogenicity

- Fifth disease
 - Erythema infectiosum
- Aplastic crisis
 - Patients with hemoglobinopathies
 - Immunosuppressed, immunodeficient, immunocompromised
- Congenital parvovirus
 - Hydrops fetalis

Symptoms – Fifth Disease

- Incubation 7-10 days
- Lasts 5-7 days
- Three Phases
 - First phase – peak level of virus and RBC destruction
 - Fever
 - Malaise
 - Chills
 - Bright red, raised “slap cheek” rash

Erythema infectiosum



“Slap cheek” rash on the face, lacy rash on the extremities.

Henoch-Schönlein purpura (HSP)



Treatment

- Mainly supportive care
- Acetaminophen or Ibuprofen for fever
- Topical anesthetic or antihistamine for itching
- Intravenous Immunoglobulin (IVIG) in chronic parvovirus
- Aplastic crisis may require packed RBC transfusion
- Vaccine is in trials



وَقُلِ الْحَمْدُ لِلَّهِ



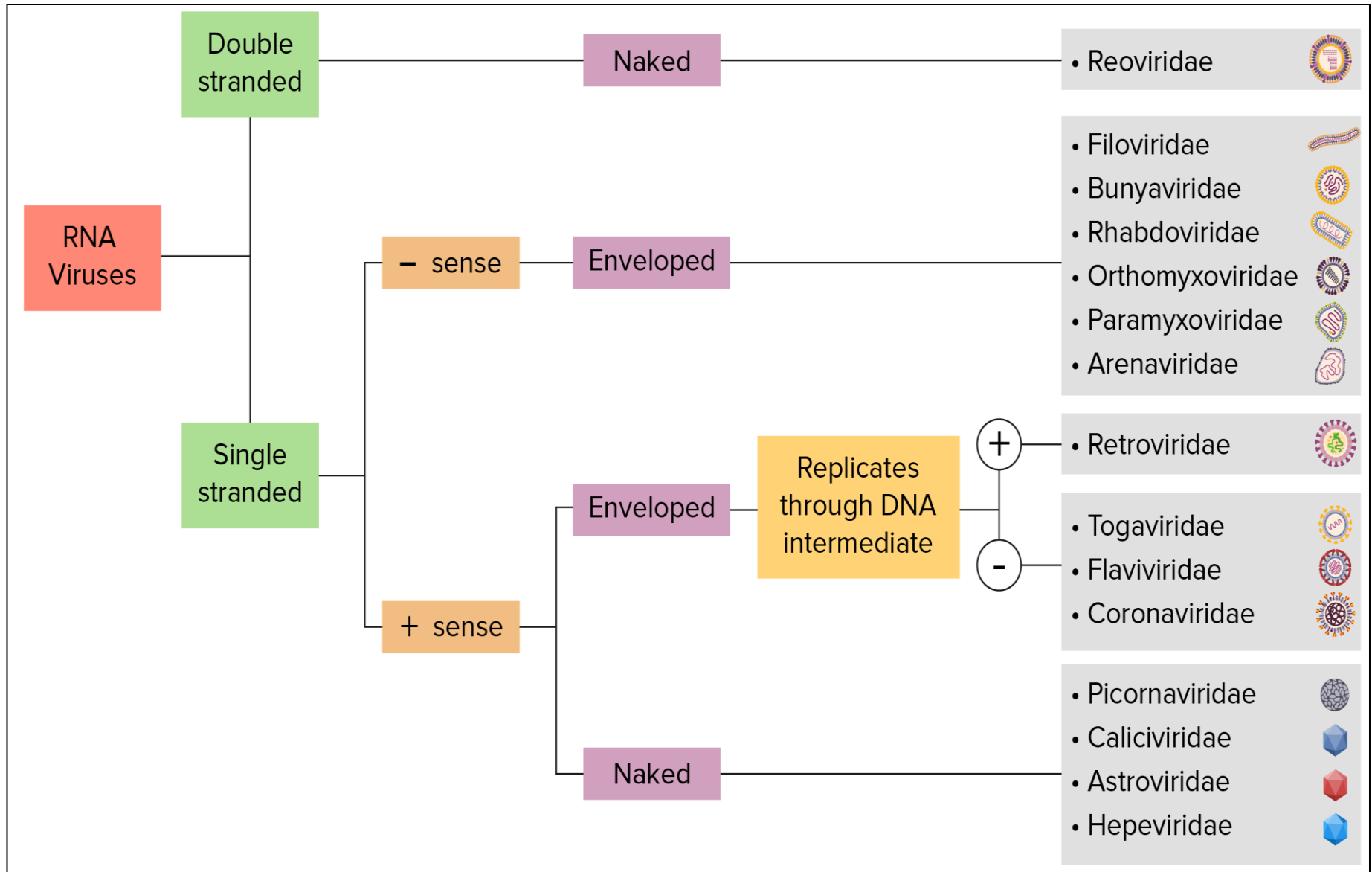
كلية النور الجامعة
قسم الصيدلة
المرحلة الثانية
احياء مجهرية طبية 2



RNA viruses

Nada Khairi Younus
M.Sc Medical Microbiology

RNA virus classification



ss+ve sense & ss-ve sense RNA viruses ??????????

- Viruses can be differentiated based on how they store their genomic information, such as by DNA or double-stranded RNA. Positive-sense single-stranded RNA (+ssRNA) viruses are one such way and it is a key aspect of the infectious cycle of the virus.
- Two important examples of +ssRNA viruses are severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) and Hepatovirus A, which cause coronavirus and hepatitis A, respectively.

DSRNA

The RNA molecules with
two complementary strands

Occurs in double-stranded
RNA viruses

RNA-dependent RNA
polymerase transcribes
dsRNA

Examples: Rotavirus

SSRNA

The RNA molecules with a
single strand

Occurs in single-stranded
RNA viruses

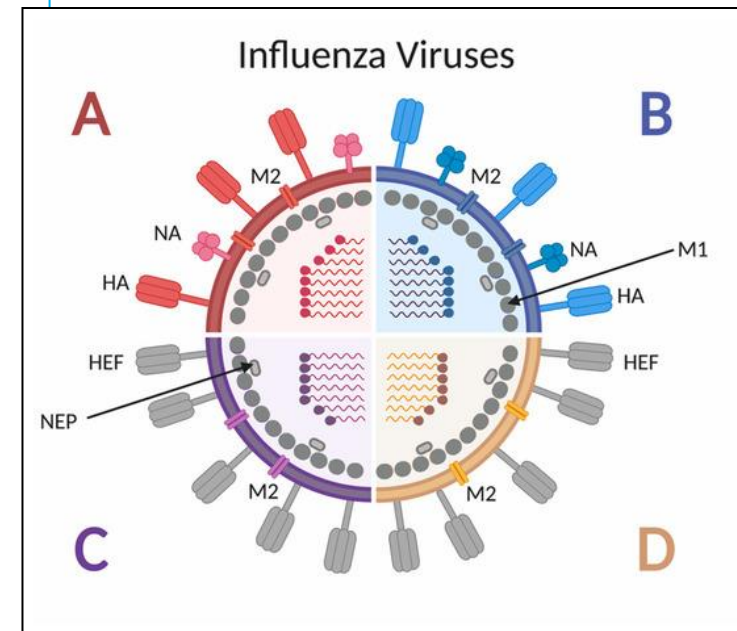
Only the negative sense
RNA undergoes transcription

Examples: Hepatitis C virus,
West Nile virus, dengue
virus, and MERS

1. Enveloped segmented single-stranded RNA viruses

- **Influenza A,B,C**

- Schematic of influenza A, B, C and D virus structure.
- Influenza A and B viruses express surface glycoproteins hemagglutinin (HA) and neuraminidase (NA), as well as the M2 ion channel.
- Both A and B viruses have 8 genomic segments coding for at least 10 proteins.
- Influenza C and D viruses express the surface glycoprotein hemagglutinin-esterase fusion (HEF), as well as the M2 ion channel.
- Both C and D viruses have 7 genomic segments coding for 9 proteins.
- All four types of influenza viruses express the M1 protein along the inner surface on the envelope, adjacent to the nuclear export protein (NEP).



What Are the Different Types of Flu?

- There are three types of flu viruses: A, B, and C.
- Type A and B cause the annual influenza epidemics that have up to 20% of the population sniffing, aching, coughing, and running high fevers. Type C also causes flu; however, type C flu symptoms are much less severe.
- What Is Type A Flu Virus?
- Type A flu or influenza A viruses are capable of infecting animals, although it is more common for people to suffer the ailments associated with this type of flu. Wild birds commonly act as the hosts for this flu virus.
- **Type A flu virus** is constantly changing and is generally responsible for the large flu epidemics. The influenza A2 virus (and other variants of influenza) is spread by people who are already infected. The most common flu hot spots are those surfaces that an infected person has touched and rooms where they have been recently, especially areas where they have been sneezing.
- **Type B Flu Virus**
- Unlike type A flu viruses, type B flu is found only in humans. Type B flu may cause a less severe reaction than type A flu virus, but occasionally, type B flu can still be extremely harmful. Influenza type B viruses are not classified by subtype and do not cause pandemics.

How Is Type C Flu Virus Different From the Others?

- Influenza C viruses are also found in people. They are, however, milder than either type A or B. People generally do not become very ill from the influenza type C viruses. Type C flu viruses do not cause epidemics.

There are four types Trusted Source of influenza virus.

Influenza A viruses cause seasonal flu epidemics practically every year in the United States. They can infect humans and animals. Influenza A is the only type that can cause a pandemic, which is a global spread of disease. Bird flu and swine flu pandemics both Trusted Source resulted from influenza A viruses. An influenza A virus has two surface proteins: hemagglutinin and neuraminidase. These help doctors with classification.

Influenza B viruses can also cause seasonal epidemics that typically only affect humans. There are two lineages of influenza B: Victoria and Yamagata. Influenza B viruses mutate more slowly than influenza A viruses.

Influenza C viruses cause mild illnesses — they do not appear to cause epidemics.

Symptoms

Flu symptoms can range from mild to severe, and they vary from person to person. Common symptoms of the flu include:

Fatigue, nasal congestion, a cough, headaches, a sore throat, body aches, chills, a fever, vomiting or diarrhea, which are more common in children

Antiviral medications

- Antiviral medications are available by prescription only. They can shorten the duration of symptoms or prevent complications, such as pneumonia.
- Antivirals can especially benefit people with a greater risk of flu complications, including young children, older adults, pregnant women, and people with certain chronic illnesses.
- Antiviral medications work best when a person takes them within 1–2 days of symptoms starting.

There are a few different types of antivirals for the flu, including:

- Oseltamivir, zanamivir, peramivir, baloxavir marboxil,

These can come in pill, liquid, inhalable powder, or intravenous forms.

Influenza (flu) vaccines (often called “**flu shots**”) are vaccines that protect against the four influenza viruses that research indicates will be most common ...



2. Enveloped **Non** - segmented single-stranded RNA viruses

Para influenza, mumps, measles, RSV

Human parainfluenza viruses (HPIVs)

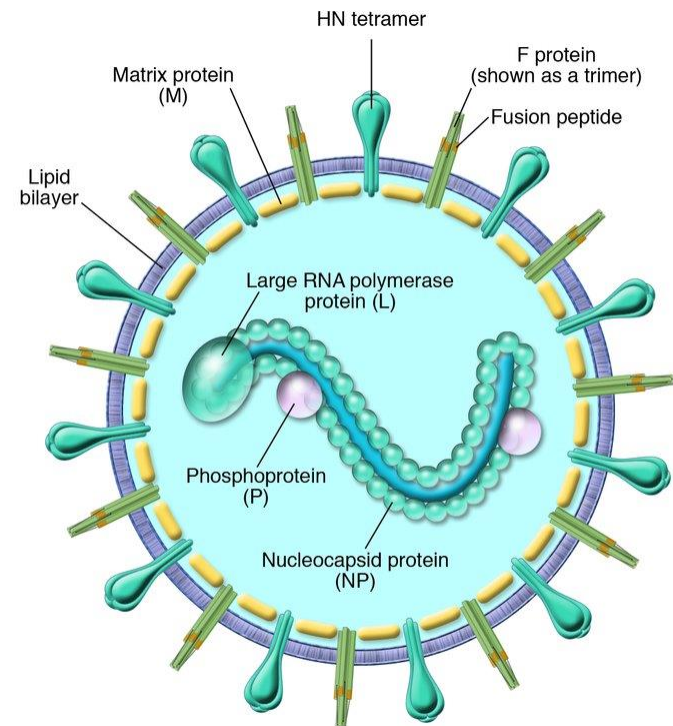
commonly cause respiratory infections in infants and young children. Patients usually recover on their own. Symptoms & Illnesses ·

Is parainfluenza the same as the flu?

Parainfluenza viruses are also called **human parainfluenza viruses (HPIVs)**. They aren't the same as flu (influenza) viruses. The flu is more common in winter. In the U.S., parainfluenza viruses are more common in the spring, summer, and fall.

There are four commonly recognized types of parainfluenza viruses:

HPIV-1, HPIV-2, HPIV-3, HPIV-4.



Human parainfluenza viruses (HPIVs)

Treatment

- There is no cure for HPIV. Once your child is infected, the virus needs to run its course. Antibiotics are not useful. Instead, treatment is aimed at reducing the symptoms.

What medication is used for parainfluenza?

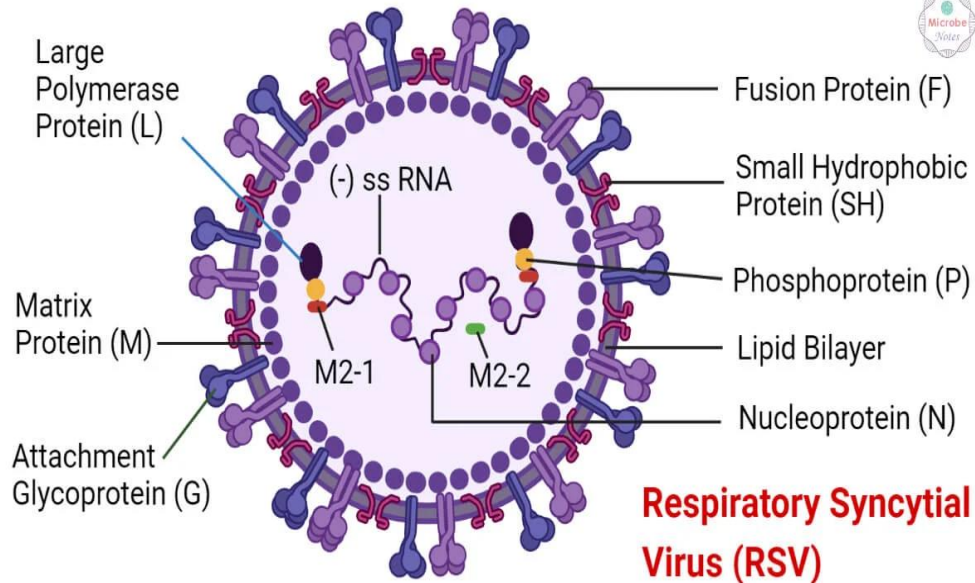
- Take acetaminophen, ibuprofen, and other over-the-counter medications for pain and fever (Caution: Aspirin should not be given to children.)
- use a room humidifier or take a hot shower to help ease a sore throat and cough.

How long does parainfluenza last in adults?

- Parainfluenza virus type 3 is one of a group of common viruses known as human parainfluenza viruses (HPIV) that cause a variety of respiratory illnesses. Symptoms usually develop between 2 and 7 days from the time of exposure and typically resolve in 7-10 days. Symptoms may include fever, runny nose, and cough.

Respiratory syncytial virus

RSV



Respiratory Syncytial Virus or **RSV** in short, is classified as a **Pneumovirus** which is a viral infection virus that can spread quickly through respiratory droplets when an infected person sneezes or coughs. The RSV can cause infection in the lungs and respiratory tract, especially in infants and young children because children's lungs are not adequately developed as well as their immune systems are not as efficient as adults so they can get infected more easily. Children are most likely to be infected by RSV when they reach their 2nd birthday.

However, adults are also susceptible to RSV infection.

The common symptoms of RSV include:

Fever, Dry or Wet Cough, Sneezing, Runny nose, Loss of appetite or Refuse when breastfeed or bottle-feed, Breathing difficulty or Wheezing (in severe cases).

Typically, the symptoms will show within 4 – 6 days after the virus infection. In addition, the infection and inflammation in the respiratory tract and lungs can lead to complications such as Laryngitis, Bronchitis, Bronchiolitis, and Pneumonia in severe cases. Thus, people who have severe symptoms may need to be admitted to the hospital to receive special medical treatment, such as a bronchodilator to relax the lung's muscle and widen the airway. However, most mild cases do not require treatment and will naturally clear up on their own within 5 – 7 days. In moderate cases, the patients are infected in the lower respiratory tract or have a chronic cough with mucus. The patients may need percussion and postural drainage to drain mucus out of the respiratory tract, and the symptoms may take around 2 – 3 weeks until it gets better.

Currently, there is **no vaccination** for RSV. Therefore, prevention is the best option for everyone. We can prevent RSV infection by breastfeeding to increase the immunity naturally and avoid taking children to crowded areas or visiting respiratory patients. We can also prevent the spread of the virus by separating the infected patient from others, and by frequently washing hands with soap or alcohol-based hand sanitizer.

Rhabdovirus

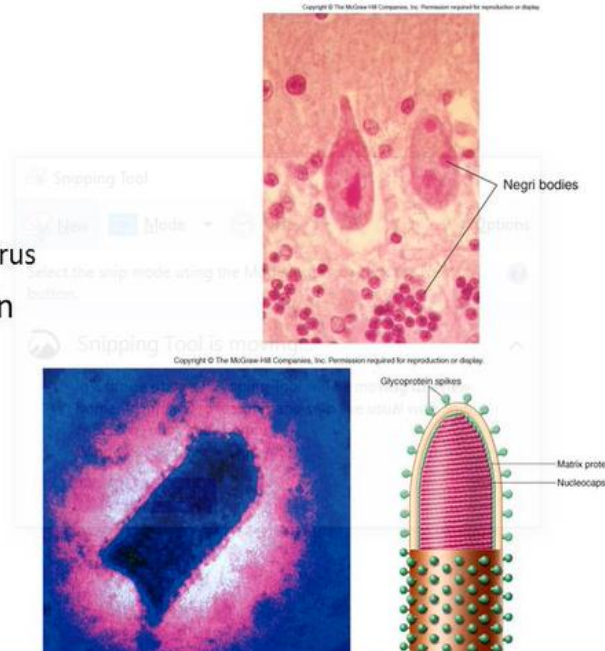
Lassa virus (Rabies)



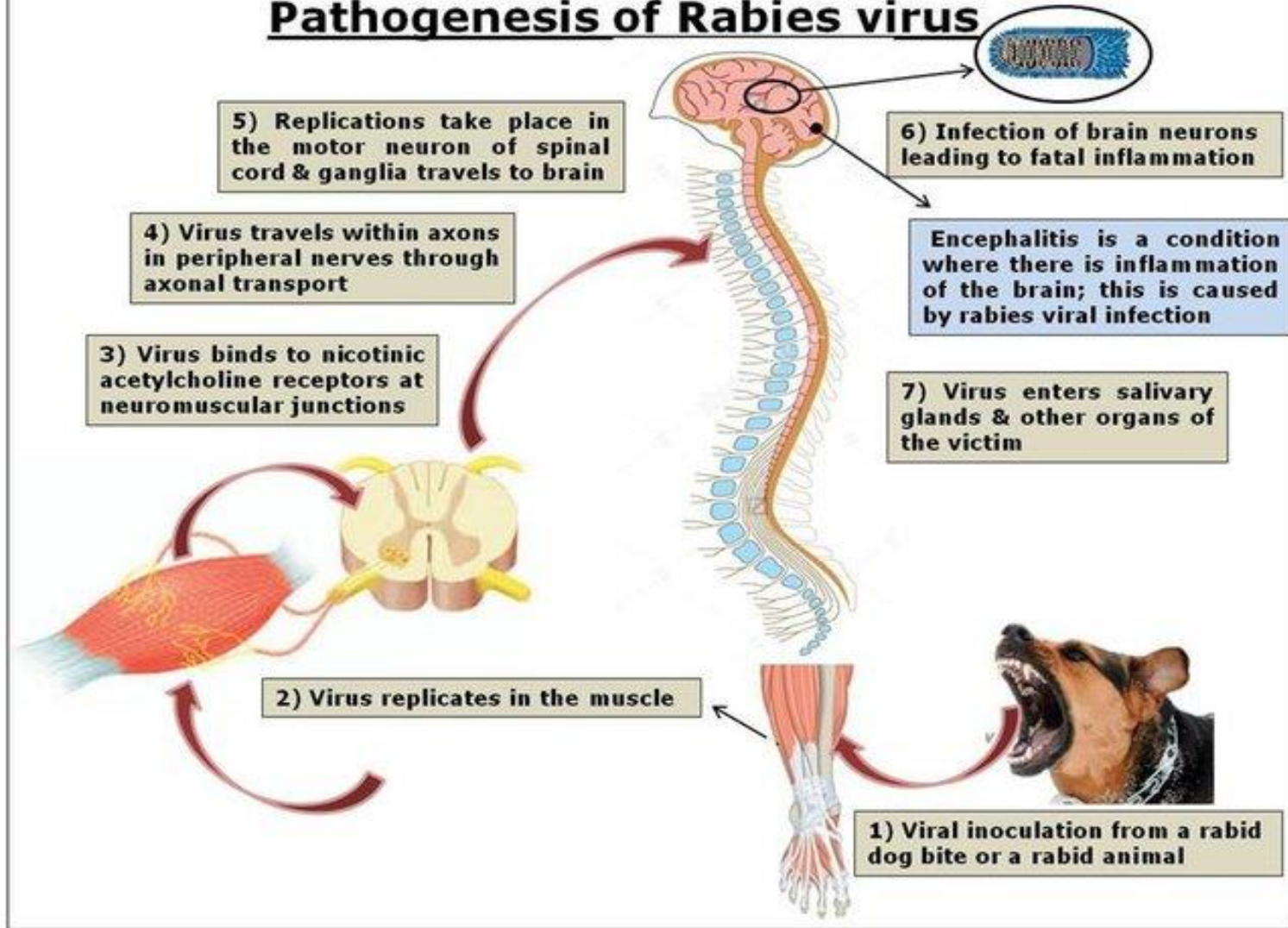
Does rhabdovirus cause rabies?

- Rabies virus, scientific name Rabies lyssavirus, is a neurotropic virus that causes rabies in humans and animals. Rabies transmission can occur through the saliva of animals and less commonly through contact with human saliva. Rabies lyssavirus, like many rhabdoviruses, has an extremely wide host range.

- Causative agent:
 - Rabies virus
 - Lyssavirus
 - Enveloped ssRNA
 - Spiked bullet shaped virus
 - Virus multiplies in brain forming Negri bodies



Pathogenesis of Rabies virus



- Signs & Symptoms

- Pain and itching at site of infection
- Fever, headache, myalgia, sore throat, fatigue
- Progress rapidly to secondary symptoms
 - Encephalitis, agitation, confusion, hallucinations, seizure, increased sensitivity to light and touch, coma
 - Increased salivation and difficulty swallowing
 - Results in frothing of mouth
 - Hydrophobia occurs in 50% of cases
 - About 50% of patients die within 4 days

Why can't rabies be cured?

- There's no cure for rabies once it's moved to your brain because it's protected by your blood-brain barrier. Your blood-brain barrier is a layer between your brain and the blood vessels in your head.

Why do rabies victims fear water?

- Rabies affects parts of the brain that controls speaking, swallowing, and breathing. It alters the saliva production process and causes painful muscle spasms that discourage swallowing.
- Two types of vaccines to protect against rabies in humans exist - **nerve tissue** and **cell culture vaccines**. **HDCV vaccine (Imovax, Sanofi Pasteur)**.

Flavivirus

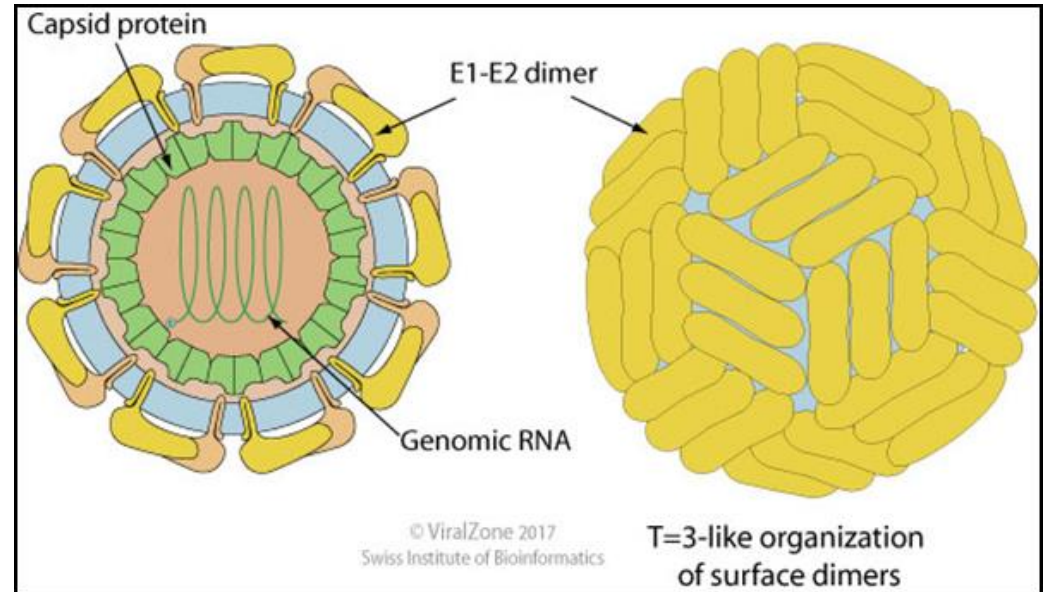
What disease is caused by flavivirus?

- Members of this family belong to a single genus, Flavivirus, and cause widespread morbidity and mortality throughout the world. Some of the mosquitoes-transmitted viruses include: **Yellow Fever, Dengue Fever, Japanese encephalitis, West Nile viruses, and Zika virus.**

Why is it called the flavivirus?

- Flaviviruses are named for the yellow fever virus; the word flavus means 'yellow' in Latin, and yellow fever in turn is named from its propensity to cause yellow jaundice in victims.
- **Signs and Symptoms:** Infection with these viruses usually presents as a self-limiting febrile illness characterized by headache, arthralgia المفاصل, myalgia الم العضلات and lethargy الخمول, and sometimes accompanied by a rash الطفح الجلدي. Full recovery however can take several months.

- There are vaccines only for few flaviviruses while no effective treatments are available.



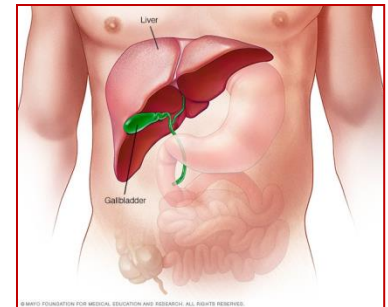
Examples of ssRNA + sense flavivirus are

1. HCV التهاب الكبد الفيروسي C
2. HIV الايدز

HCV virus (hepatitis C virus)

ssRNA+ve sense

- Hepatitis C is a liver infection caused by the hepatitis C virus (HCV). Hepatitis C is spread through contact with **blood** from an infected person. Today, most people become infected with the hepatitis C virus by sharing needles or other equipment used to prepare and inject drugs.
- Hepatitis C is a viral infection that causes liver inflammation, sometimes leading to serious liver damage. The hepatitis C virus (HCV) spreads through contaminated **blood**.
- Until recently, hepatitis C treatment required weekly injections and oral medications that many HCV-infected people couldn't take because of other health problems or unacceptable side effects. Today, chronic HCV is usually curable with oral medications taken every day for two to six months.



Symptoms

- Long-term infection with the hepatitis C virus is known as chronic hepatitis C. **Chronic hepatitis C** is usually a "silent" infection for many years, until the virus damages the liver enough to cause the signs and symptoms of liver disease.

Signs and symptoms include:

- Bleeding easily نزيف, Bruising easily كدمات, Fatigue ارهاق, Poor appetite, Yellow discoloration of the skin and eyes (jaundice), Dark-colored urine, Itchy skin حكة, Fluid buildup in your abdomen (ascites) تراكم السوائل في البطن الاستسقاء, Swelling in your legs, Weight loss, Confusion ارتباك, drowsiness and slurred speech (hepatic encephalopathy), Spiderlike blood vessels on your skin (spider angiomas). وتداخل الكلام. (اعتلال الدماغ الكبدي) ، أو عية دموية تشبه العنكبوت على الجلد (أورام وعائية عنكبوتية).

Signs and Symptoms

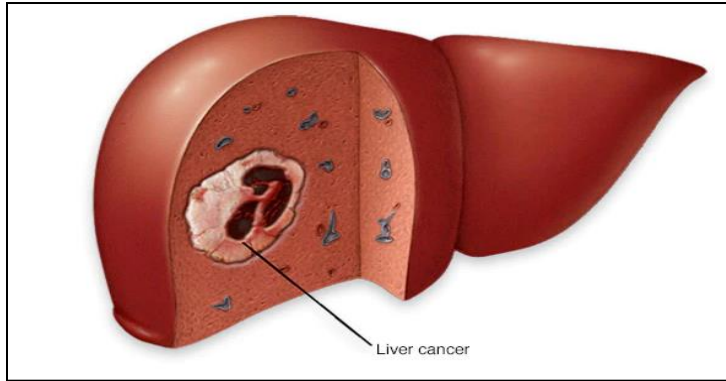
While most people with hepatitis C do not have any signs of the disease, some people can experience mild to severe symptoms soon after being infected, including:

- Jaundice (yellow color in the skin or eyes)
- Loss of appetite
- Nausea
- Vomiting
- Abdominal pain
- Dark urine
- Clay-colored bowel movements

- Fatigue
- Fever

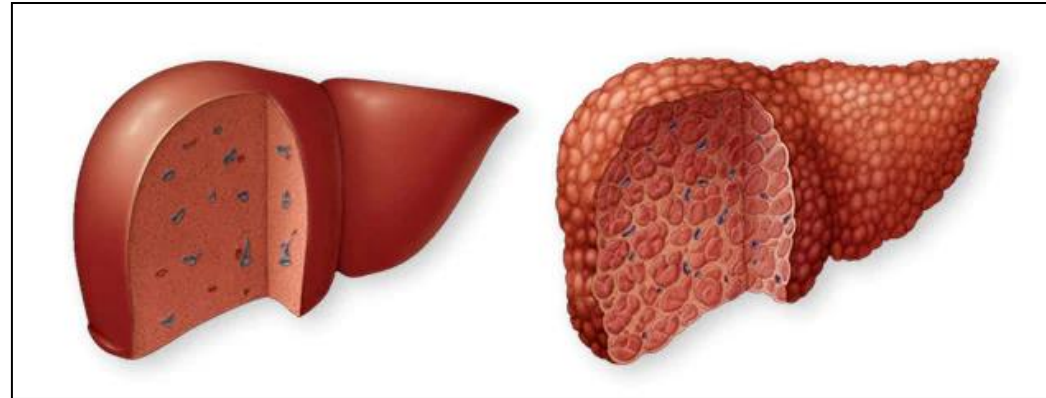
- Joint pain





Liver cancer

Liver cancer begins in the cells of the liver. The most common form of liver cancer begins in cells called hepatocytes and is called hepatocellular carcinoma.



A typical liver (left) shows no signs of scarring. In cirrhosis (right), scar tissue replaces healthy liver tissue.

Complications

1. **Scarring of the liver (cirrhosis).** After decades of hepatitis C infection, cirrhosis may occur. Scarring in your liver makes it difficult for your liver to function.
2. **Liver cancer.** A small number of people with hepatitis C infection may develop liver cancer.
3. **Liver failure.** Advanced cirrhosis may cause your liver to stop functioning.

- Prevention

1. Stop using illicit drugs, particularly if you inject them.
2. Be cautious about body piercing and tattooing.
3. Practice safer sex.

Right now, there is **no vaccine for the hepatitis C virus**. Researchers have been trying to make a vaccine against hepatitis C for many years, but they have not succeeded yet. The main reason there is no vaccine for hepatitis C is because this virus has many strains, called genotypes, and many subtypes.

Hepatitis C medicines

sofosbuvir.

a combination of ledipasvir and sofosbuvir.

a combination of ombitasvir, paritaprevir and ritonavir.

a combination of elbasvir and grazoprevir.

a combination of sofosbuvir and velpatasvir.

a combination of sofosbuvir, velpatasvir and voxilaprevir.



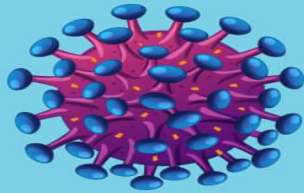
HIV VIRUS

HIV (human immunodeficiency virus)

AIDS ايدز

- HIV (human immunodeficiency virus) is a virus that attacks cells that help the body fight infection, making a person more vulnerable to other infections and diseases. It is spread by contact with certain bodily fluids of a person with HIV, most commonly during unprotected sex. If left untreated, HIV can lead to the disease **AIDS** (acquired immunodeficiency syndrome).
- The human body can't get rid of HIV and no effective HIV cure exists. So, once the human have HIV, he has it for life.
- Luckily, however, effective treatment with HIV medicine (called **antiretroviral therapy or ART**) is available. If taken as prescribed, HIV medicine can reduce the amount of HIV in the blood (also called the viral load) to a very low level. This is called viral suppression. If a person's viral load is so low that a standard lab can't detect it, this is called having an undetectable viral load. People with HIV who take HIV medicine as prescribed and get and keep an undetectable viral load can live long and healthy lives and will not transmit HIV to their HIV-negative partners through sex.

WHAT IS THE DIFFERENCE BETWEEN HIV AND AIDS?



HIV



AIDS

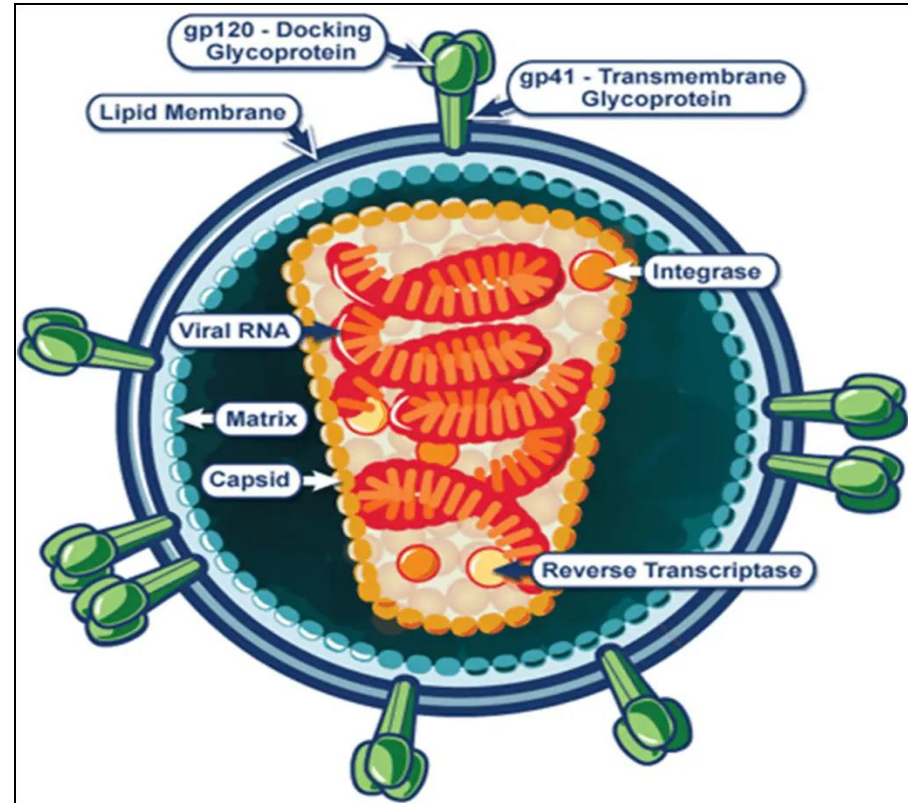
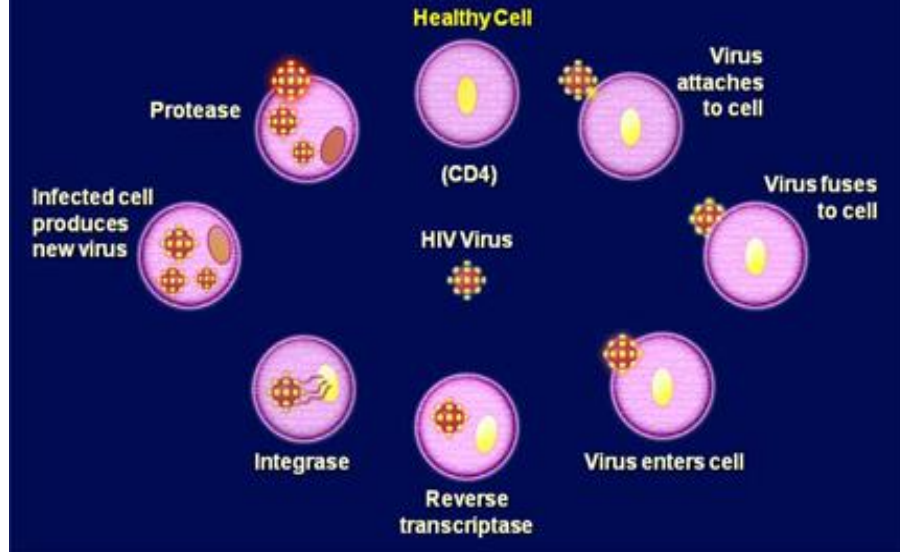
- In the simplest of terms, HIV is the beginning stage of a disease that can progress into AIDS. HIV is transmitted through bodily fluids, including blood, breastmilk, seminal, vaginal, or rectal fluids. It is commonly transmitted through unprotected sex, shared needles, during the birthing process, or through breastfeeding.

Another major difference between HIV and AIDS are the symptoms and signs.

During the first stage of HIV – which typically occurs 2 to 4 weeks after contraction – a person will have flu-like symptoms, such as:

- Body aches, Chills and fever, Fatigue, Mouth sores, Sore throat, Rashes, Swollen lymph nodes.
- If they are not tested for HIV or **do not take ART**, these symptoms will eventually subside as they progress into stage 2.
- Once HIV progresses into the third stage and becomes AIDS, a person will start to experience far more serious symptoms, such as:
- Rapid weight loss, Extreme fatigue, Recurring fever, Neurological issues such as memory loss
- Chronic diarrhea, Sores in the mouth, genitals, and rectum, Pneumonia.
- AIDS may lead to additional health issues, including cancer, tuberculosis, meningitis, kidney disease, and liver disease.

Targets for HIV Meds



What type of virus is HIV?

- HIV belongs to a class of viruses known as retroviruses.
- HIV targets CD4+ T cells by binding to the CD4 molecule on the cell surface.
- Left untreated, HIV infection goes through the following stages:
 1. Seroconversion illness. Some people experience a short illness soon after they contract HIV. ...
 2. The asymptomatic stage of HIV. Once seroconversion is over, most people feel fine and don't experience any symptoms.
 3. Symptomatic HIV. ...
 4. Late-stage HIV.



Is there a vaccine for HIV patients?

- No. There is currently no vaccine available that will prevent HIV infection or treat those who have it.
- What is the HIV vaccine called?
- The vaccine, currently dubbed eOD-GT8, is being developed by Professor William.

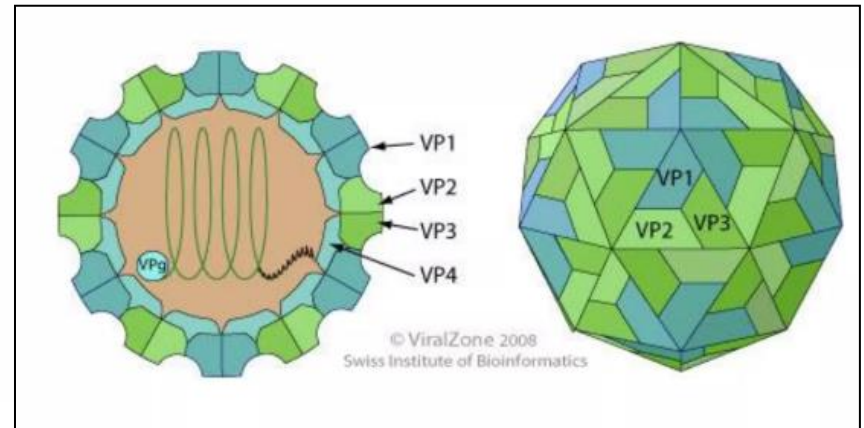


Non- enveloped Non- segmented ss RNA Viruses

Picornavirus & Claviviruses (Picornavirus HAV)

Introduction

- The picornaviruses are small (22 to 30 nm) **nonenveloped, single-stranded RNA viruses** with cubic symmetry
- The virus capsid is **composed of 60 protein** subunits, each consisting of four polypeptides VP1–VP4
- Because they contain **no essential lipids, they are ether resistant**
- They replicate in the cytoplasm
- The picornaviruses that affect humans are
 - **The enteroviruses**, found primarily in the gut
 - **the rhinoviruses**, found in the upper respiratory tract
 - **hepatovirus (Hepatitis A virus)** in the intestine and liver.

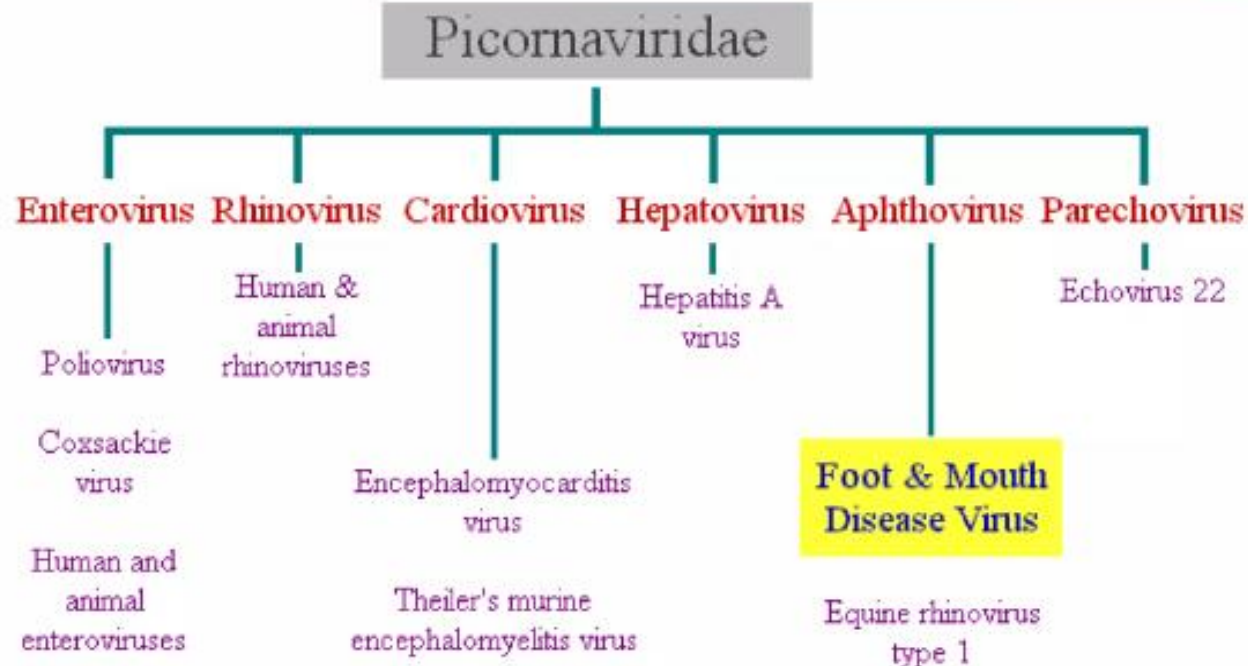


Classification

The family Picornaviridae comprises **five genera**:

- **Enterovirus**
- **Hepatovirus**
- **Rhinovirus**, which infect humans
- **Aphthovirus** (foot-and-mouth disease virus), which infects cloven-hoofed animals and occasionally humans;
- **Cardiovirus**, which infects rodents

Classification



Hepatitis A virus

- Hepatitis A virus (HAV), classified as hepatovirus, is a small, **unenveloped symmetrical RNA virus** which shares many of the characteristics of the picornavirus family, and is the cause of infectious or epidemic hepatitis **transmitted by the fecal-oral route**
- virus enters the body by ingestion and intestinal infection
- The virus then spreads, **probably by the bloodstream, to the liver, a target organ**
- Large numbers of virus particles are detectable in feces during the incubation period, beginning as early as 10–14 days after exposure and continuing
- **Antibody to hepatitis A virus that persists is also detectable late in the incubation period, coinciding approximately with the onset of biochemical evidence of liver damage**

Hepatitis A virus

- Fatigue
- Sudden nausea and vomiting
- Abdominal pain or discomfort, especially on the upper right side beneath your lower ribs (by your liver)
- Clay-colored bowel movements
- Loss of appetite
- Low-grade fever
- Dark urine
- Joint pain
- Yellowing of the skin and the whites of your eyes (jaundice)
- Intense itching

Hepatitis A virus

- Unlike other types of viral hepatitis, **hepatitis A does not cause long-term liver damage**, and it doesn't become chronic
- In rare cases, **hepatitis A can cause a sudden loss of liver function**, especially in older adults or people with chronic liver diseases
- **Acute liver failure requires a stay in the hospital for monitoring and treatment.** Some people with acute liver failure may need a liver transplant
- The **hepatitis A vaccine can prevent infection with the virus**
- The vaccine is typically given in **two shots**. The first one is followed by a **booster shot six months later**

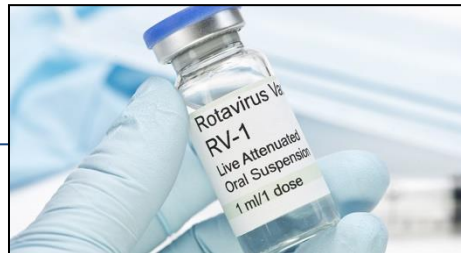
Control

- Control of picornavirus diseases depends **largely on mass education of the public on the mode of virus transmission, stressing the importance of good personal hygiene, and on provision of a good sewage disposal system and uncontaminated water supply**
- **Fecal and pharyngeal discharges are infectious**; hence, they must be handled with care and disposed of safely
- **Vaccine is commercially available for poliomyelitis and hepatitis A**
- There is **no established specific therapy**
- Treatment is symptomatic and supportive
- Clinical studies show that ribavirin shortens respiratory illnesses and interferon nasal sprays have prophylactic value for common colds

Non- enveloped Segmented ds RNA Viru

Reo viruses (rota & reo)

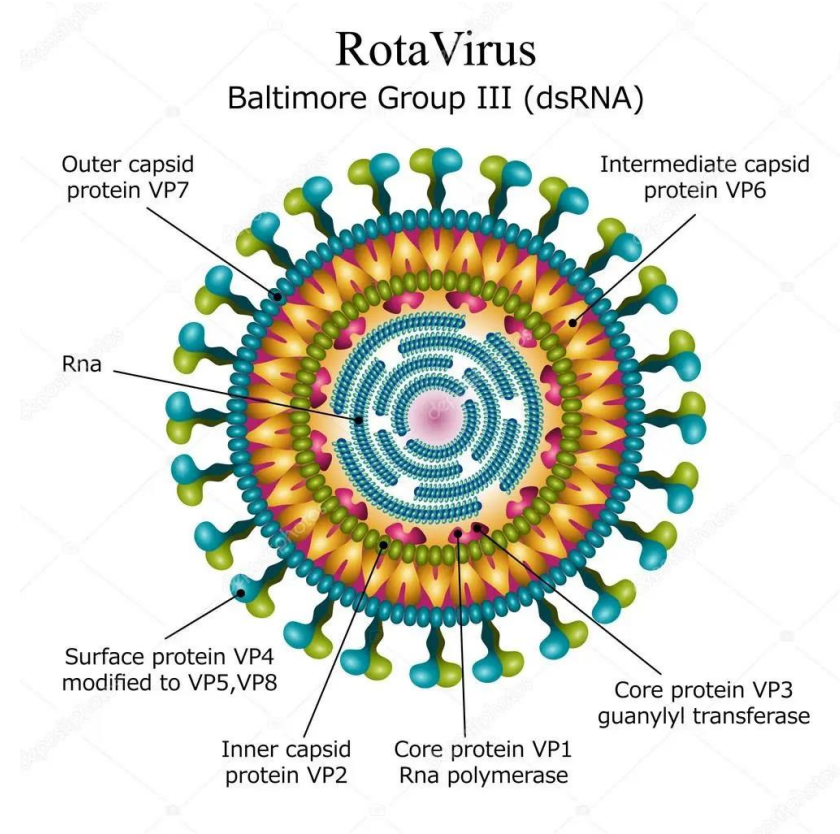
- Rotavirus is a genus of double-stranded RNA viruses in the family Reoviridae. Rotaviruses are the most common cause of diarrhoeal disease among infants and young children.
- Nearly every child in the world is infected with a rotavirus at least once by the age of five.
- Immunity develops with each infection, so subsequent infections are less severe; adults are rarely affected.
- There are ten species of the genus, referred to as A, B, C, D, E, F, G, H, I and J. Rotavirus A, the most common species, causes more than 90% of rotavirus infections in humans.
- The virus is transmitted by the **faecal-oral route**. It infects and damages the cells that line the small intestine and causes gastroenteritis (which is often called “**stomach flu**” despite having no relation to influenza).
- Rotaviral enteritis is usually an easily managed disease of childhood, but in 2013, rotaviruses caused 37 % of deaths of children from diarrhea and 215,000 deaths worldwide and almost two million more became severely ill. Most of these deaths occurred in developing countries.
- Public health campaigns to combat rotavirus focus on providing oral rehydration therapy for infected children and vaccination to prevent the disease.
- The incidence and severity of rotavirus infections has declined significantly in countries that have added **rotavirus vaccine** to their routine childhood immunisation policies.



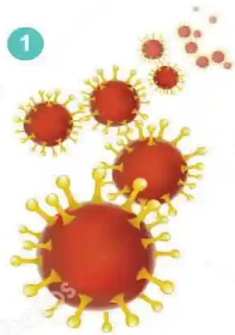
There are six viral proteins (VPs) that form the virus particle (virion). These structural proteins are called VP1, VP2, VP3, VP4, VP6 and VP7.

VP1 is located in the core of the virus particle and is an RNA-dependent RNA polymerase enzyme.

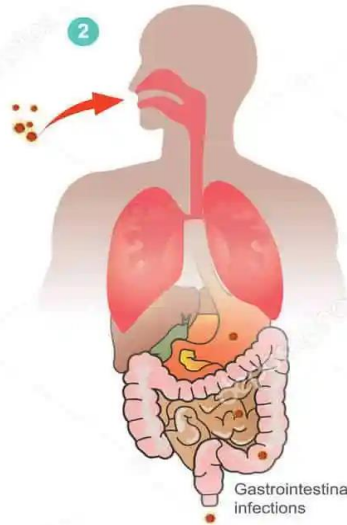
In an infected cell this enzyme produces mRNA transcripts (enzymes) for the synthesis of viral proteins and produces copies of the rotavirus genome RNA segments for newly produced virus particles.



Rota Virus Infection



Rotavirus is transmitted by the accidental ingestion damages the lining of the small intestine causing intense diarrhea, which can lead to dehydration and eventually death

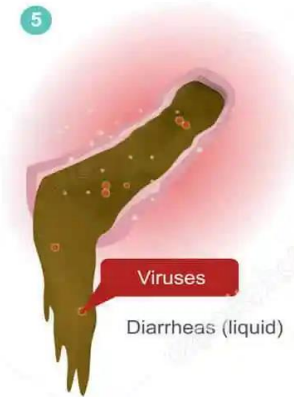


Baby 6-15 months

Rotavirus can also spread through contaminated water, food, toys, or sometimes even through droplets coughed into the air



1. Pentavalent human-bovine reassortant rotavirus vaccine
2. Monovalent rotavirus vaccine



ROTAVIRUS

Rotavirus is a viral illness that causes diarrhea. It is **most serious in kids under 2 years of age**.

Most common in **winter and spring**, rotavirus **spreads easily** through contact with infected stool.

SYMPTOMS

- ✓ Fever
- ✓ Upset stomach/vomiting
- ✓ Diarrhea
- ✓ Black or bloody stools
- ✓ Slow to move or does not respond



COMPLICATIONS

- Lack of water in the body (which can cause dry mouth, crying without tears, little or no urine, sleepiness, lack of movement or response). This may require a trip to the hospital.
- In some cases, the illness can be deadly.

TREATMENT/PREVENTION

- ✓ Drink plenty of fluids.
- ✓ Antibiotics don't work against rotavirus.
- ✓ Wash hands thoroughly, especially after using the toilet or changing a diaper. But washing hands won't always stop the virus' spread.



BE WISE — IMMUNIZE

Vaccination prevents about 40,000 to 50,000 hospitalizations among infants and young children each year.

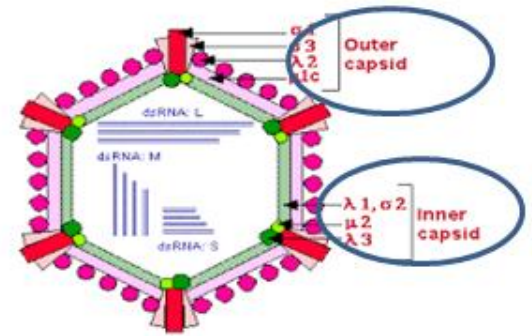
No. 1 way to prevent rotavirus is by getting the vaccination.

Two types of vaccines protect infants: One requires doses at 2 and 4 months, the other requires them at 2, 4, and 6 months.

Ask your doctor which vaccine is best for your child.

Reo virus

- Reoviruses (which also are called orthoreoviruses to avoid confusion with the family Reoviridae) are nonenveloped viruses.
- Reovirus particles are composed of an inner protein shell (ie, core) of a diameter of 60 nm, which is surrounded by an outer protein shell (ie, outer capsid) that measures 81 nm in diameter.
- **Reo** virus is a **nonenveloped double-stranded RNA virus**. This virus was initially not known to be related to any specific disease, and so was named **Respiratory Enteric Orphan virus**.
- However, some members of the reovirus family have been shown to cause mild illnesses such as diarrhea.
- ds-RNA viruses are found in human, animals, fishes, birds, insects (invertebrates) plants and fungi. In human, virus attacks mucosal membrane of respiratory and gastrointestinal system. The viruses are made up of double stranded (ds) RNA protein complex. The virus is not covered by lipid capsules. There are 87 subtypes. Some of the subtype of reoviruses are linked to symptoms of few unknown diseases. The reovirus viral infection in human is often subclinical.



Clinical Manifestation of Reovirus in Humans

- Infection caused by reovirus is often subclinical and disappears within 3 to 7 days.
- In few cases serotype 1 and 2 of reovirus enters into mucosal epithelial cells of respiratory and gastrointestinal system resulting in inflammatory viral infection. Rarely serotype 1 and 3 of reovirus were found with diseases like meningitis and encephalitis involving central nervous system. The virus infection rarely causes life threatening conditions.
- The most common symptomatic viral infection involving reovirus is upper respiratory tract infection known as **common cold**, which is mostly observed in children age 3 to 7 years attending kindergarten and elementary school.
- One of the subtype of reovirus known as coltivirus causes Colorado Tick Fever. The virus is transmitted from tick bite into human.

Diseases Caused By Reovirus

- **Upper Respiratory Illness Caused by Reovirus:**

1. Common Cold
2. Tonsillitis
3. Bronchiolitis Obliterans
4. Pneumonia

- **Gastrointestinal Illnesses Caused by Reovirus:**

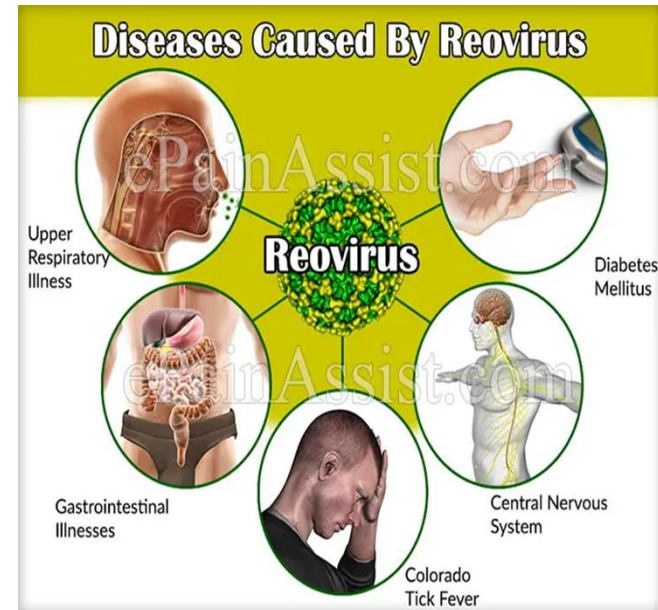
1. Gastroenteritis
2. Biliary Atresia (liver infection in infants)

- **Central Nervous System**

1. Encephalitis
2. Meningitis

- **Diabetes Mellitus**

- **Colorado Tick Fever**



Treatment of Reovirus Infection

- Treatment of reovirus infection depends on symptoms. Symptoms are appropriately treated for rapid recovery. Persistent infection and prolonged symptoms are treated with antiviral medications.
- **Cough Suppressant-** Dry cough is treated with cough suppressants. Cough suppressant medication like dextromethorphan are sold over the counter as syrup or tablets.
- **Cough Expectorant-** The cough syrup containing medication like guaifenesin helps to soften cough as well release the mucosal secretion from mucosal epithelial cells. The treatment helps to cough out mucosal secretions as sputum.
- **Anti-Fever Medication-** Fever due reovirus infection is treated with tylenol or NSAIDs.
- **Pain Medications-** Acute pain associated with reovirus infection is treated with tylenol or NSAIDs like motrin or naproxen.
- **Muscle Pain-** Muscle and body ache responds to NSAIDs or tylenol. If body ache is associated with severe muscle spasm, then muscle relaxants can be tried.
- **Dehydration-** Oral repeated fluid intake. If one cannot take oral fluid then consider intravenous fluid.
- **Persistent Vomiting-** Severe vomiting often does not respond to conservative therapy. In that case anti-histaminic or anti-vomiting medications should be tried under the supervision of physician.
- **Diarrhea-** Diarrhea is initially treated with over the counter medication like kaopectine or stool hardener. If diarrhea continues and get worse then one should see physician and get prescription of antidiarrheal medication like Imodin.
- **Dyspnea or Short of Breath-** Treatment may involve hospital or ICU (intensive care unit) admission depending on severity of symptoms. Treatment includes nasal oxygen, intravenous fluid and anti-viral medications. Serious cases may need insertion of endotracheal tube in trachea and assisted breathing.

Prions & Spongiform Encephalopathies

- Transmissible spongiform encephalopathies (TSEs) or prion diseases are a family of rare progressive neurodegenerative brain disorders that affect both humans and animals. They have long incubation periods, progress rapidly once symptoms develop and are always fatal.

Do prions cause spongiform encephalopathies?

- BSE (**bovine spongiform encephalopathy**) is a progressive neurological disorder of cattle that results from infection by an unusual transmissible agent called a prion. The nature of the transmissible agent is not well understood.

Why are diseases caused by prions called spongiform diseases?

- Prion diseases, because they cause spongelike holes in brain tissue, are also called transmissible spongiform encephalopathies. They are not curable, though symptoms can be treated. Creutzfeldt-Jakob disease, or CJD, is the most common prion disease

What are 5 diseases caused by prions?

- Identified Prion Diseases
- Creutzfeldt-Jakob Disease (CJD)
- Variant Creutzfeldt-Jakob Disease (vCJD)
- Gerstmann-Straussler-Scheinker Syndrome.
- Fatal Familial Insomnia.
- Kuru.

What prion causes bovine spongiform encephalopathy?

- Image result for Prions and Spongiform Encephalopathies
- BSE possibly originated as a result of feeding cattle meat-and-bone meal that contained BSE-infected products from a spontaneously occurring case of BSE or scrapie-infected sheep products. Scrapie is a prion disease of sheep.





What is a prion simple definition?

- The term “prions” refers to abnormal, pathogenic agents that are transmissible and are able to induce abnormal folding of specific normal cellular proteins called prion proteins that are found most abundantly in the brain.

What is an example of a prion?

- A prion is a type of **protein** that can trigger normal proteins in the brain to fold abnormally. Prion diseases can affect both humans and animals and are sometimes spread to humans by infected meat products.
- The most common form of prion disease that affects humans is Creutzfeldt-Jakob disease (CJD). مرض كروتزفيلد جاكوب .

Is prions a bacteria or virus?

- Prions are virus-like organisms made up of a prion protein. These elongated fibrils (green) are believed to be aggregations of the protein that makes up the infectious prion. Prions attack nerve cells producing neurodegenerative brain disease. "Mad cow" symptoms include glazed eyes and uncontrollable body tremor.

What are the similarities and differences between viruses and prions?

- Prions and viruses are both replicating infectious particles that are not considered "alive". They differ in that prions are misfolded proteins, while viruses carry DNA and RNA.

Spongiform encephalopathy

How do humans get spongiform encephalopathy?

- Transmissible Spongiform Encephalopathies (TSEs) cannot be transmitted through the air or through touching or most other forms of casual contact. However, they may be transmitted through contact with infected tissue, body fluids, or contaminated medical instruments.

What are the symptoms of spongiform encephalopathy?

- The clinical signs in humans vary, but commonly include personality changes, psychiatric problems such as depression, lack of coordination, and/or an unsteady gait (ataxia). Patients also may experience involuntary jerking movements called myoclonus, unusual sensations, insomnia, confusion, or memory problems.

What is the treatment for spongiform encephalopathies?

- There is currently no treatment that can halt progression of any of the TSEs. Treatment is aimed at alleviating symptoms and making the patient as comfortable as possible.



Alnoor University College
Pharmacy Department
كلية النور الجامعة ---قسم الصيدلة
Medical Microbiology II
2022

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ماجستير احياء مجهرية طبية
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M.Sc - medical microbiology



Parasitology

Introduction

Parasitism, relationship between two species of plants or animals in which one benefits at the expense of the other, sometimes without killing the host organism.

Parasitology: is the study of parasites, their hosts, and the relationship between them. In medical parasitology, you study medical parasites including their morphology, life cycle, and the relationship with host and the environment.

What is a parasite?

Is an organism which lives in or upon another organism (its host) and benefits by deriving nutrients directly from it. Parasites rely on their hosts for food and shelter and they can cause harm to their hosts.

Types of parasites

1. **Endoparasites**: are those parasites which live within the host (inside the body of its host). Usually, the endoparasites cause most human diseases e.g worms

Endoparasites: are also divided into:

- **Obligate parasites** where these parasites can only survive in a host hence usually cannot survive if kept isolated from their hosts and therefore go directly from one host to another e.g. hookworms
- **Facultative parasites** which are normally free-living organisms and infect a host only by accident depending on the surrounding conditions e.g. Fungi such as *Candida Spp.*

Ectoparasites: are those parasites which live outside the body of the host e.g. ticks, mosquitoes, lice

What is host?

Host: is defined as an organism which harbors the parasite and provides the nourishment.

Types of hosts

Definitive host: It is an organism that hosts the adult form or sexually mature stages of the parasite e.g. human a definitive host for *Taenia saginata* (tapeworm).

Intermediate host: an organism that supports the immature (larvae stages) or non-reproductive (asexual form) of a parasite e.g. *trypanosomes*, the cause of sleeping sickness, humans are the intermediate host, while the tsetse fly is the definitive host.

Reservoir host: An animal which harbors the parasite and serve as an important source of infection to other susceptible hosts. It serves as a source of infection and potential reinfection of humans and as a means of sustaining a parasite when it is not infecting humans e.g. water buffalo is the reservoir host for schistosomiasis.

What is a vector?

It is any agent (person, animal or microorganism) that carries and transmits an infectious pathogen into another living organism- mostly are arthropods e.g. female mosquito.

Vectors can be divided into two different types:

Biological vectors: examples-haematophagous arthropodes such as mosquitoes and other biting insects.

Mechanical vectors: Examples: flie for transport of amoebal cysts.

What is infection?

Infection: is the invasion of a host organism's body tissues by disease-causing agents and their multiplication.

A person gets a disease if he or she carries an infection. For example a person gets malaria if he carries an infection caused by the bite of the female Anopheles mosquito.

Transmission:

It is the passing of a disease from an infected host individual or group or environment to another individual or group.

Mode of transmission

1) ***Oral route***- by ingestion of food, water or vegetables e.g. contaminated by the faeces that contain the infective stages of the parasite (fecal-oral route) or ingestion of raw or undercooked meat e.g. pork containing *Cysticercus cellulosae* the larval stage of *Taenia solium*.

2) ***Penetration of the skin and mucous membranes*** e.g. penetration of the skin by hook worms larvae.

- 3) *Inoculation by an arthropod vector* e.g. inoculation of Plasmodium parasites by female Anopheles mosquitoes, tsetse flies transmit sleeping sickness.
- 4) *Sexual contact* e.g. Trichomonas.

What makes parasites different from other microorganisms?

Multiple life stages.

1. Different immune response.
2. Difficult to formulate vaccine.
3. Difficult to control.

Medically important parasites (Classification)

1. Protozoology (study of protozoa "primitive single cell")
2. Helminthology (study of helminthes "worms")
3. Entomology (study of arthropods "insects")

Classification of pathogenic protozoans according to type of movement:

1. Amoebas (move using pseudopodia).
2. Ciliates (move using one or more cilia).
3. Flagellates (move using one or more flagella).
4. Apicomplexans (non-motile, intracellular parasites).

Classification of pathogenic protozoa

Site of infection	Mode of locomotion	Species
Intestinal tract	Pseudopodia	<u>Amoeba</u> <i>Entamoeba histolytica</i>
	Cilia	<u>Intestinal ciliate</u> <i>Balantidium coli</i>
Urogenital tract	Flagella	<u>Urogenital flagellate</u> <i>Trichomonas vaginalis</i>
Blood and tissue	Flagella	<u>Hemoflagellates</u> <i>Trypanosoma</i> spp.
	Non-motile sporozoa	<u>Blood Sporozoans</u> <i>Plasmodium</i> spp. <u>Tissue Sporozoans</u> <i>Toxoplasma gonidii</i>

Species:

1. *Entamoeba histolytica*

Medical Definition of Entamoeba histolytica

Entamoeba histolytica: cause amoebic dysentery, & Amoebiasis a disorder with inflammation of the intestine and ulceration of the colon.

Entamoeba histolytica is a single-celled parasite that is transmitted to humans via contaminated water and food.

Entamoeba histolytica is well recognized as a pathogenic ameba, associated with intestinal infections.

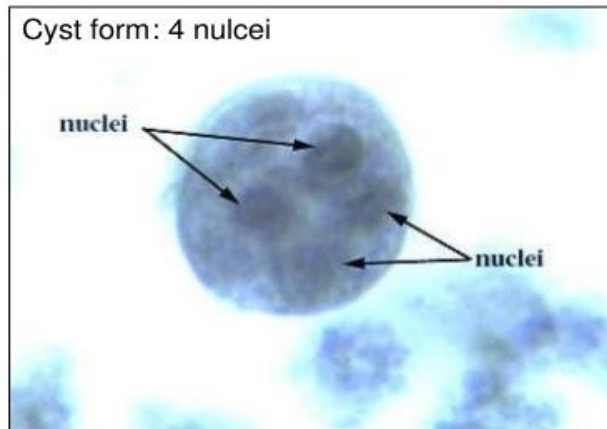
Entamoeba histolytica



Trophozoite form

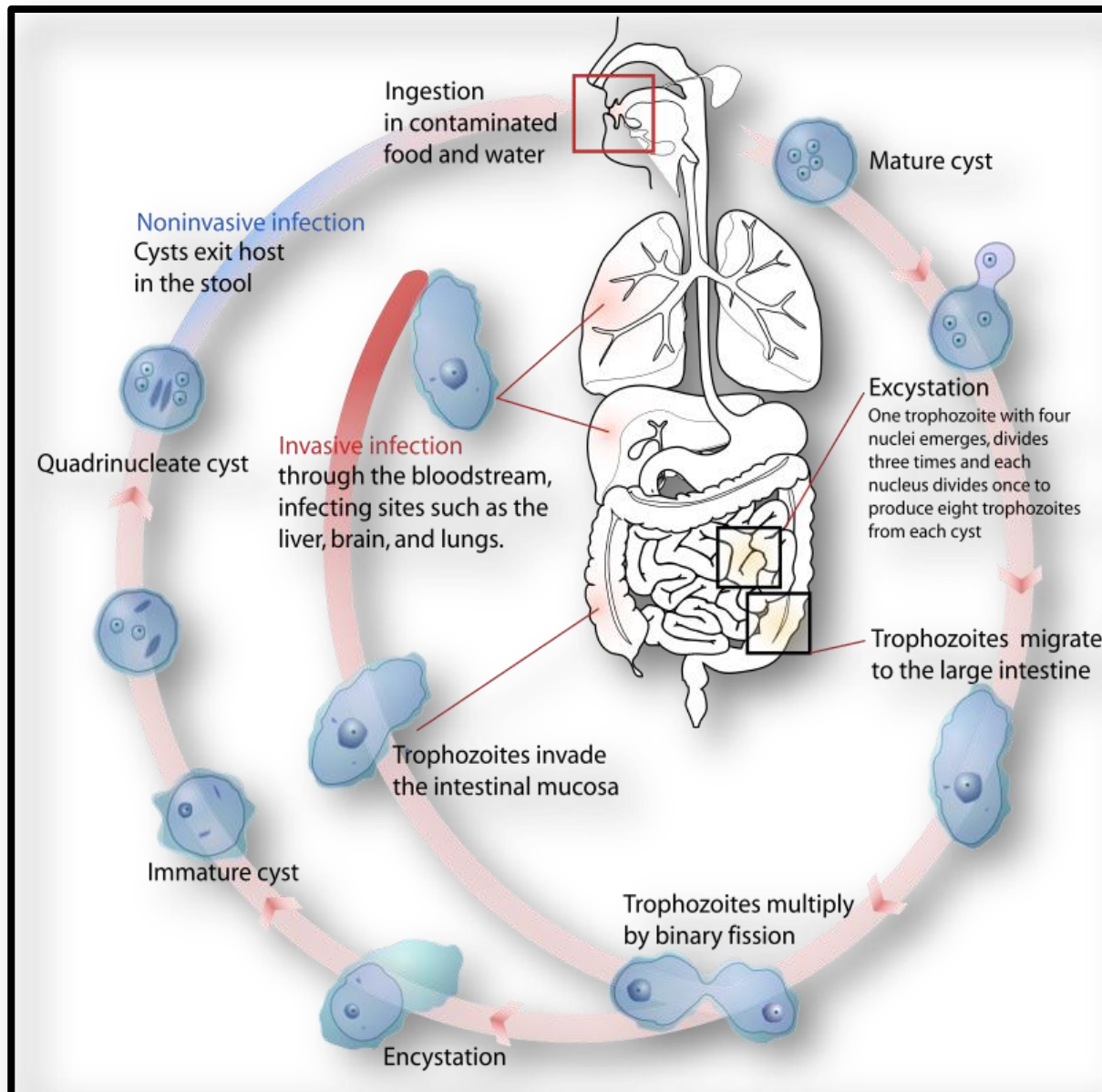
Bull eye shape nucleus

Contain ingested RBCs



Cyst form: 4 nuclei

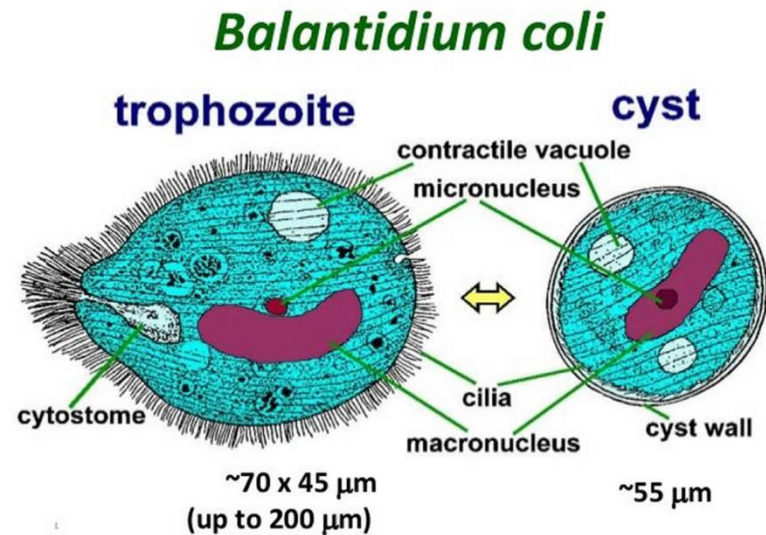
Life cycle of *Entamoeba histolytica*



Genus and species	<i>Entamoeba histolytica</i>
Etiologic agent of:	Amoebiasis; amoebic dysentery; extra intestinal amoebiasis, usually amoebic liver abscess, amoebic lung abscess.
Infective stage	Tetra nucleated cyst (having 2-4 nuclei)
Definitive host	Human
Portal of entry	Mouth
Mode of transmission	Ingestion of mature cyst through contaminated food or water
Habitat	Colon and cecum
Pathogenic stage	Trophozoite
Locomotive apparatus	Pseudopodia ("false foot")

Treatment

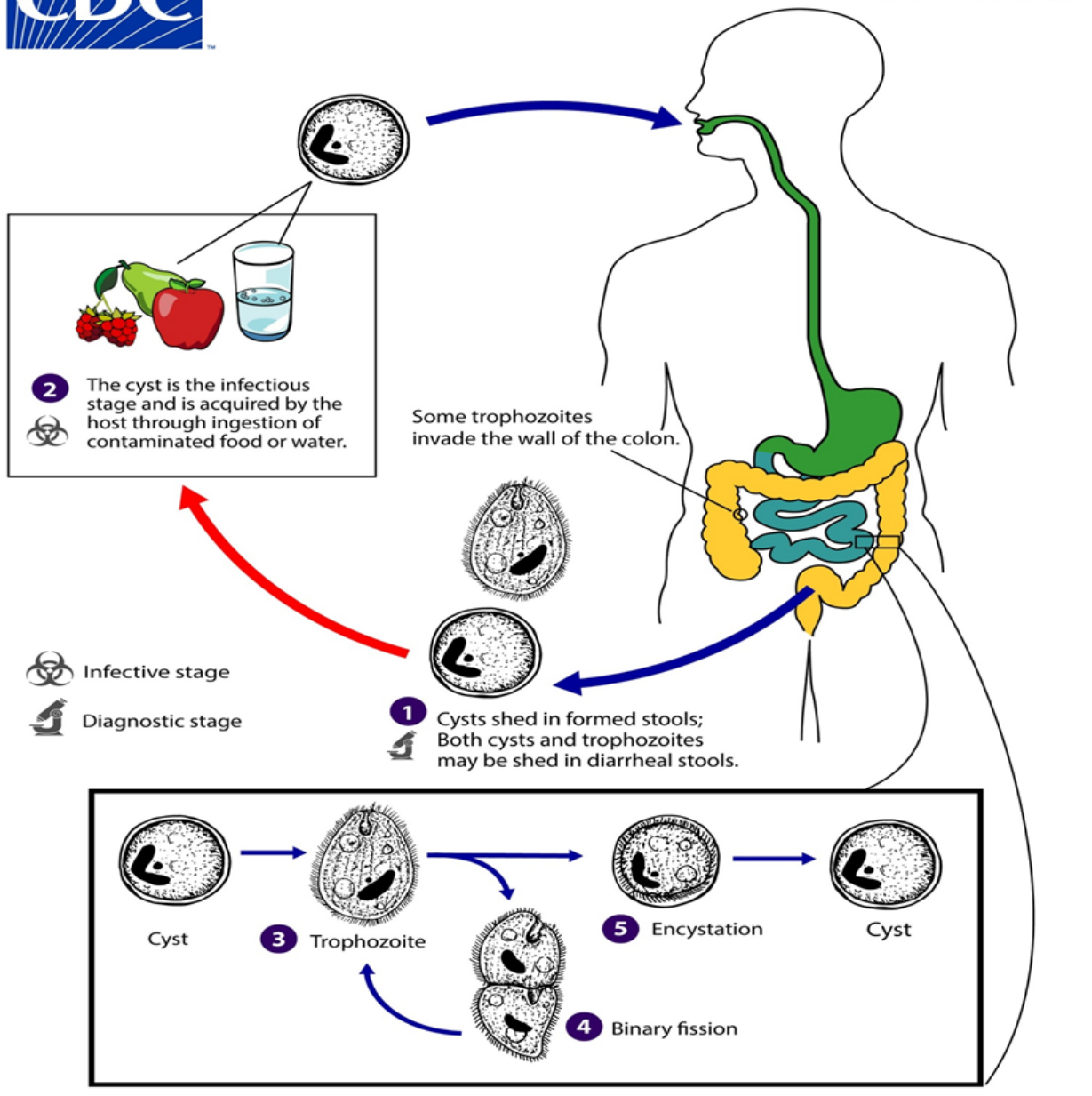
Metronidazole or Tinidazole




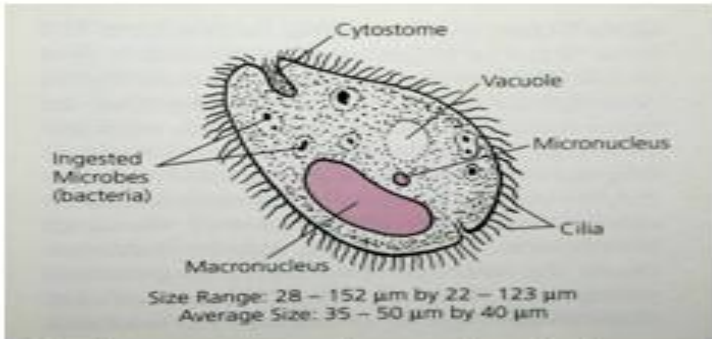
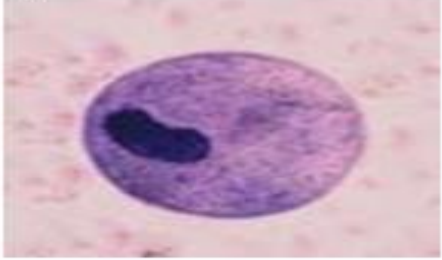
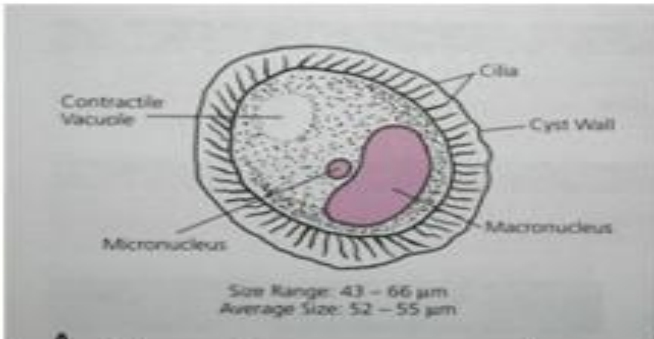
2. *Balantidium coli*

B. coli is a parasitic species of ciliate alveolates that causes the disease balantidiasis & balantidial dysentery.

Balantidiasis is caused through contamination and transmitted through fecal-oral route. *Balantidium coli* primarily are found in the lumen of the large intestines. It can penetrate into the mucosa layer of the large intestine and start to cause ulcers.



Balantidium coli primarily is found in the lumen of the large intestines. It can penetrate into the mucosa layer of the large intestine and start to cause ulcers.

Trophozoite	Cyst
 	 
They have two nuclei such as kidney shape macro nucleus and spherical shape micro nucleus.	Only kidney shape macronucleus is visible under microscope.
It is covered with cilia.	It is covered with thick hard cyst wall. Sometime cilia can be seen within the cyst.
It has pointed anterior called cystosome for feeding	It is spherical in shape.
It is non-infective stage.	It is the infective stage.

Symptoms of Blalantidiasis

- Diarrhea, Weight loss, Dysentery.

Treatment

Three medications are used most often to treat *Balantidium coli*: tetracycline, metronidazole.

*Thank
you*





Alnoor University College

Pharmacy Department

كلية النور الجامعة ---- قسم الصيدلة

Medical Microbiology II

2022

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M.Sc - medical microbiology

Helminths

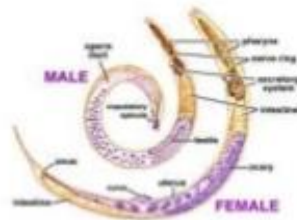
Helminths: are **macro**scopic invertebrates characterized by elongated, flat or round bodies.

The parasitic intestinal helminths can be divided into three groups:

- **Nematodes** (roundworms) are long, thin, un-segmented, cylindrical worms.
- **Cestodes** (tapeworms) are long, flat, & segmented.
- **Trematodes** (flukes) are flat, un-segmented, & leaf-shaped.

Classification of Helminths

	Nematodes (roundworms)	Cestodes (tapeworms)	Trematodes (flukes)
Shape	Cylindrical	Segmented plane	Unsegmented plane
Body cavity	Yes	No	No
Digestive tract	Complete	No	Incomplete
Sex	Male or female	hermaphrodites	hermaphrodites (except <i>Schistosoma</i>)



The life cycle of parasitic helminths are quite complex. For many helminths, intermediate hosts are required to support larval (immature) stages needed for the worms to reach maturity.

Adult worms are either *diocious*, meaning that the male and female sex organs are in separate worms. Or *monoecious*, meaning that each worm has both sex organs.

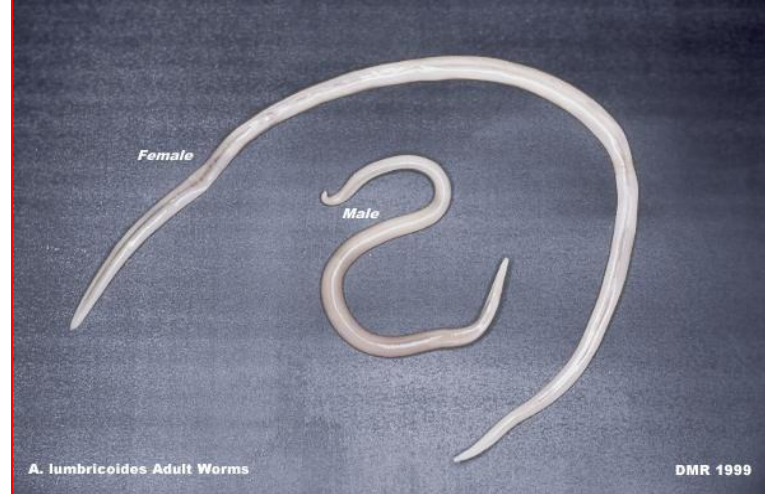
1) Nematodes (roundworms).e.g. *Ascaris lumbricoides*

Ascariasis is an infection of the small intestine caused by *Ascaris lumbricoides*, which is a species of roundworm.

The roundworm is the largest intestinal parasite and can reach up to 40 cm in length. The infection is common in regions with poor sanitation, particularly in poorer countries in the tropics and subtropics.

The worms live in the intestine of man, but can move to other parts. The larvae migrate to the lungs. The infestation can contribute significantly to malnutrition & diminished work capacity, though it is usually not fatal.

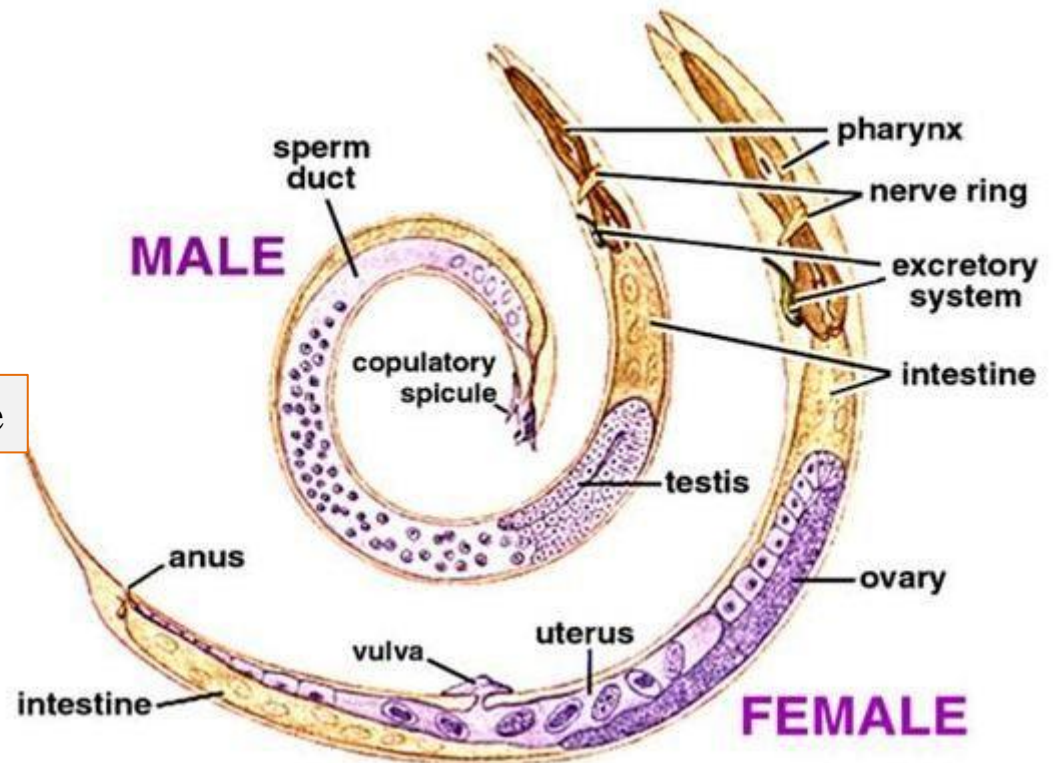
The human can become infected with ***ascariasis*** after accidentally ingesting the eggs of the *A. lumbricoides*. The eggs can be found in soil contaminated by human feces or uncooked food contaminated by soil that contains roundworm eggs.

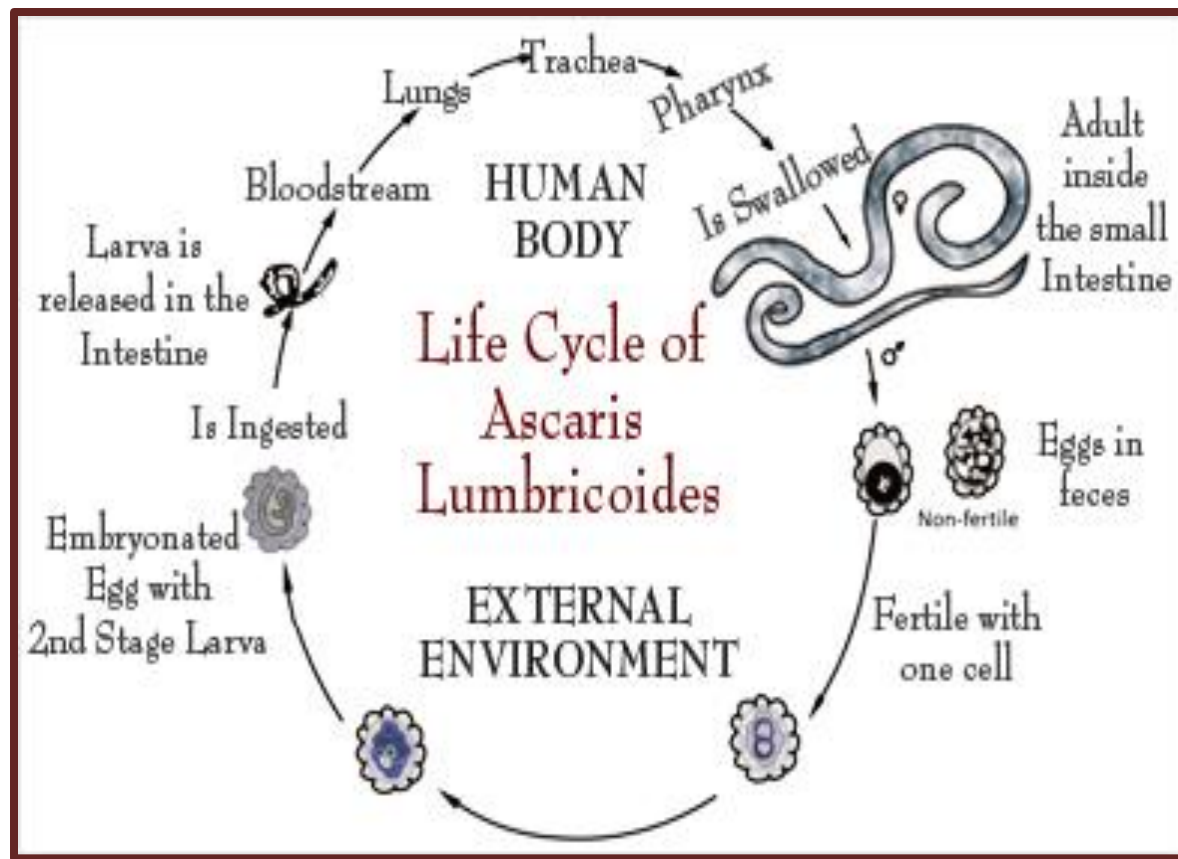


Ascaris lumbricoides (large intestinal roundworm) adult worms live in the small intestine. The female grows to reach 22 to 35 cm in length and the male reaches 10 to 31 cm in length.



A. Lumbricoides egg under microscope





Life cycle of *Ascaris lumbricoides*

After ingestion the egg, the *A. lumbricoides* roundworm reproduces inside intestine. The worm goes through several stages:

Swallowed eggs first hatch in the intestine. The larvae then move through the bloodstream to the lungs.

After maturing, the roundworms leave the lungs and travel to throat. The human either cough up or swallow the roundworms in the throat. The worms that are swallowed will travel back to the intestine

Once they're back in the intestine, the worms will mate and lay more eggs.

The cycle continues. Some eggs are excreted through feces. Other eggs hatch and return to the lungs.

Symptoms:

Ascaris lumbricoides in the lungs can cause:

Coughing, wheezing or shortness of breath, blood in mucus, chest discomfort & fever. Pulmonary symptoms called **Loeffler's syndrome**.

Ascaris lumbricoides in intestines can cause:

Nausea, vomiting, irregular stools or diarrhea, intestinal blockage, which causes severe pain and vomiting, loss of appetite, visible worms in the stool & abdominal pain.

Diagnosis is made by the microscopic identification of eggs in the stool, larvae in sputum or adult worm passed in the stool.

How is ascariasis treated?

Any one of the following anti-helminthic drugs can be used in the treatment of ascariasis:

Ivermectin

Albendazole

Mebendazole



Enterobius vermicularis (pin-worm)

Pinworms are tiny, narrow worms. They're white in color and less than a half-inch long. Pinworm infections are also known as *enterobiasis* or *oxyuriasis*.

The worm is named after the characteristic pin-like tail present on the posterior part of female worms. Pinworm infections can spread easily. They're most common in children between the ages of 5 and 10, people who live in institutions, and those who have regular, close contact with individuals in these groups.

Enterobius vermicularis is a worm that primarily lives in ileum and cecum. Once *E. vermicularis* eggs are ingested, they take about 1 to 2 months to develop into adult worms which happen in the small intestine.

The female adult worms and ova migrate to the anal area mostly at night time and deposit thousands of eggs in the perianal area.

This migration causes a lot of itching. This leads to contamination of the fingers and results in ingestion of the eggs (autoinfection) and restarting of the life cycle of the worm.

Occasionally, the larvae migrate back into the rectum and to the small intestine and begin the life cycle (retro infection).

The eggs of *Enterobius vermicularis* measure 50—60 μm by 20—30 μm . They are transparent, elongate to oval in shape (D-shape), and slightly flattened on one side. Microscopy is used for diagnosis.

Treatment is with an initial dose of mebendazole followed by a second treatment 2 weeks later to kill any newly acquired worms.



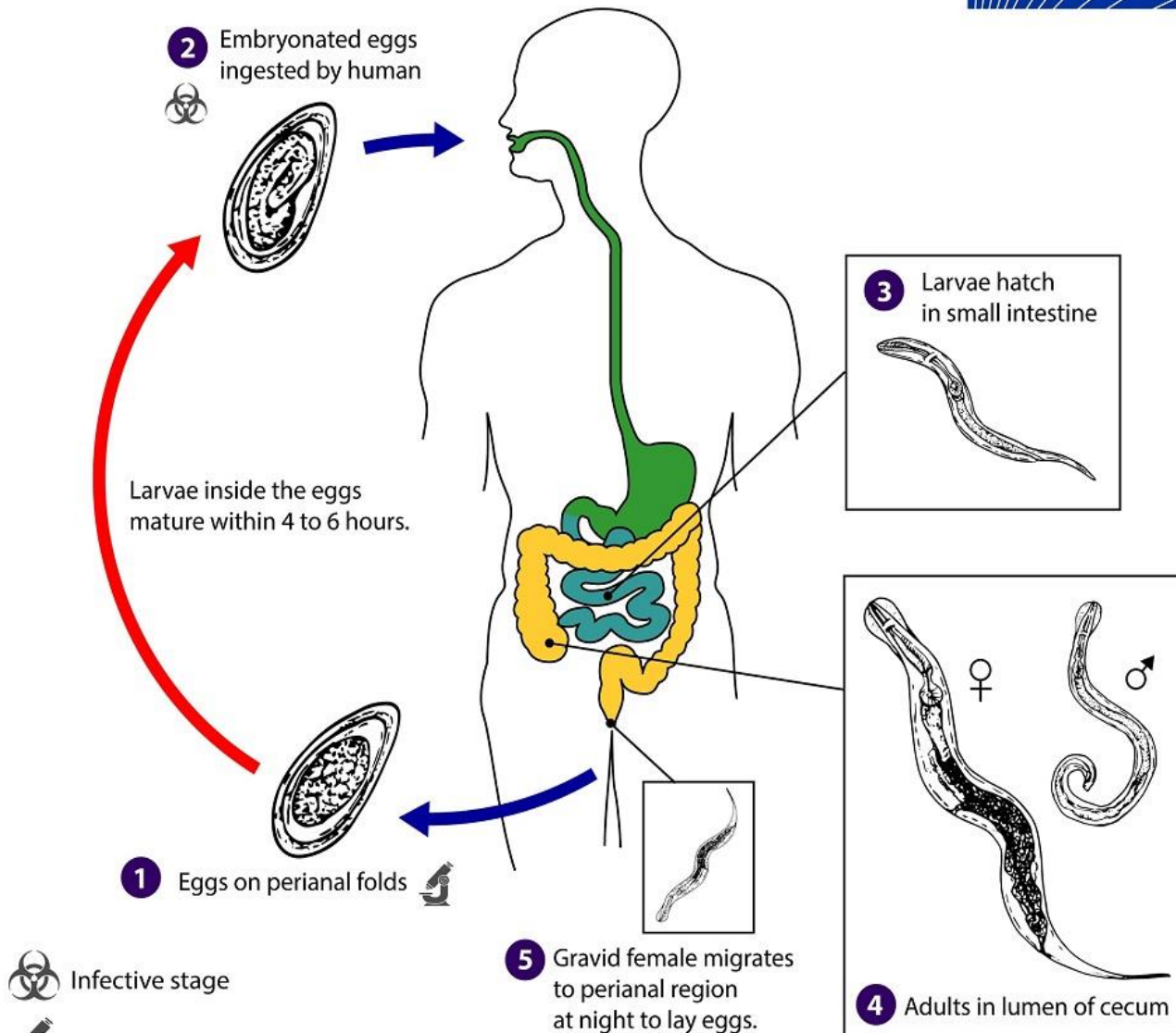
Female

Male

Enterobius vermicularis adult



Enterobius vermicularis egg



Enterobius vermicularis life cycle

2) Cestodes (tapeworms)

1) *Echinococcus granulosus*

2) *Tenia spp*

Echinococcus granulosus----dog tapeworm



Echinococcus platyhelminth worm belonging to the Cestoda class.

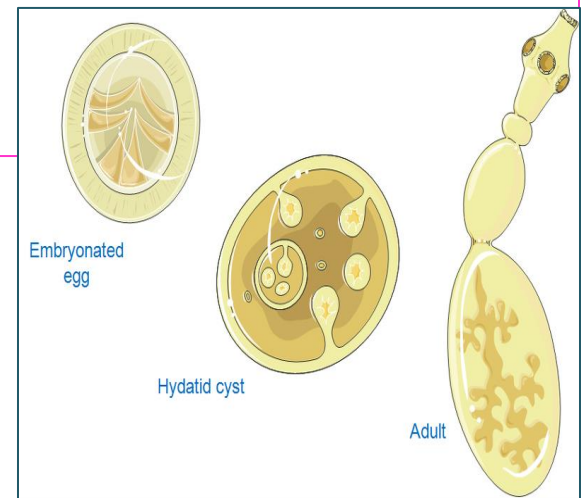
- 1) *E. granulosus*
- 2) *E. multilocularis* are the two species responsible of the **hydatid disease**, an infection that can affect humans and other intermediate hosts.

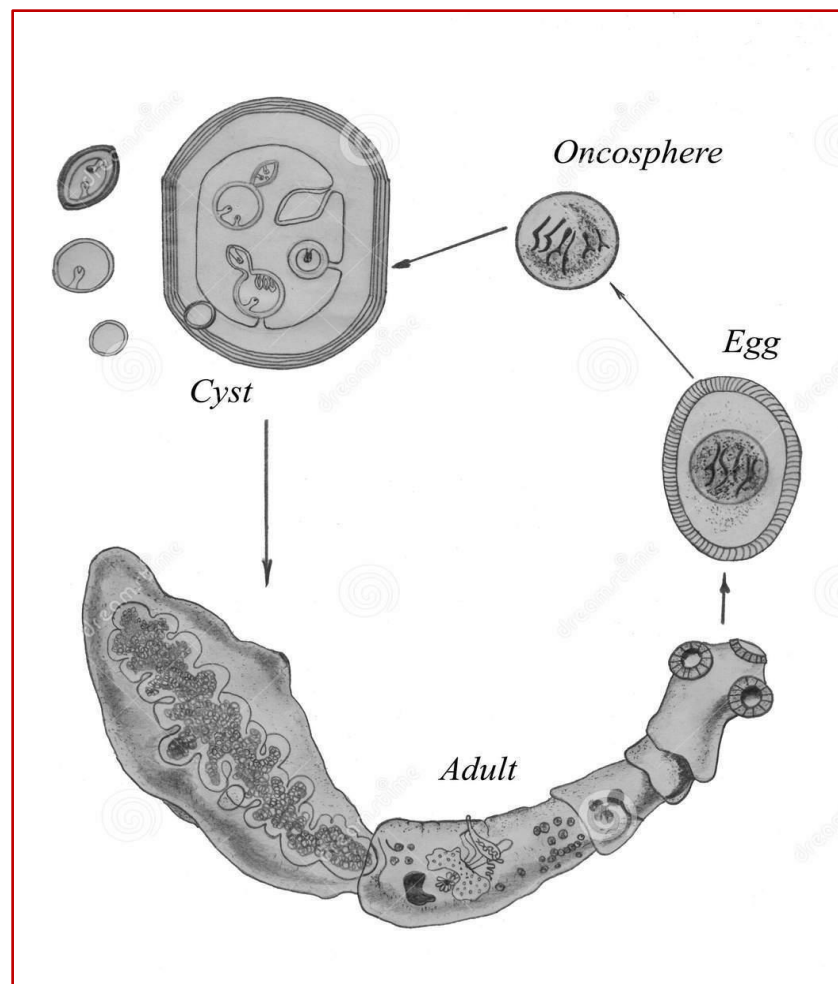
Human echinococcosis (hydatidosis, or hydatid disease) is caused by the larval stages of cestodes (tapeworms) of the genus *Echinococcus granulosus*.

The definitive host is dog. Sheep and cattle are intermediate hosts that get infected by ingesting contaminated feces. In humans the infection mainly spreads in the liver, but it can also affect lungs, spleen, peritoneum and other parts of the body.

Echinococcus reaches humans by the ingestion of food containing the eggs of the parasite and, after digestion; the embryo arrives to the liver through lymphatic vessels or blood.

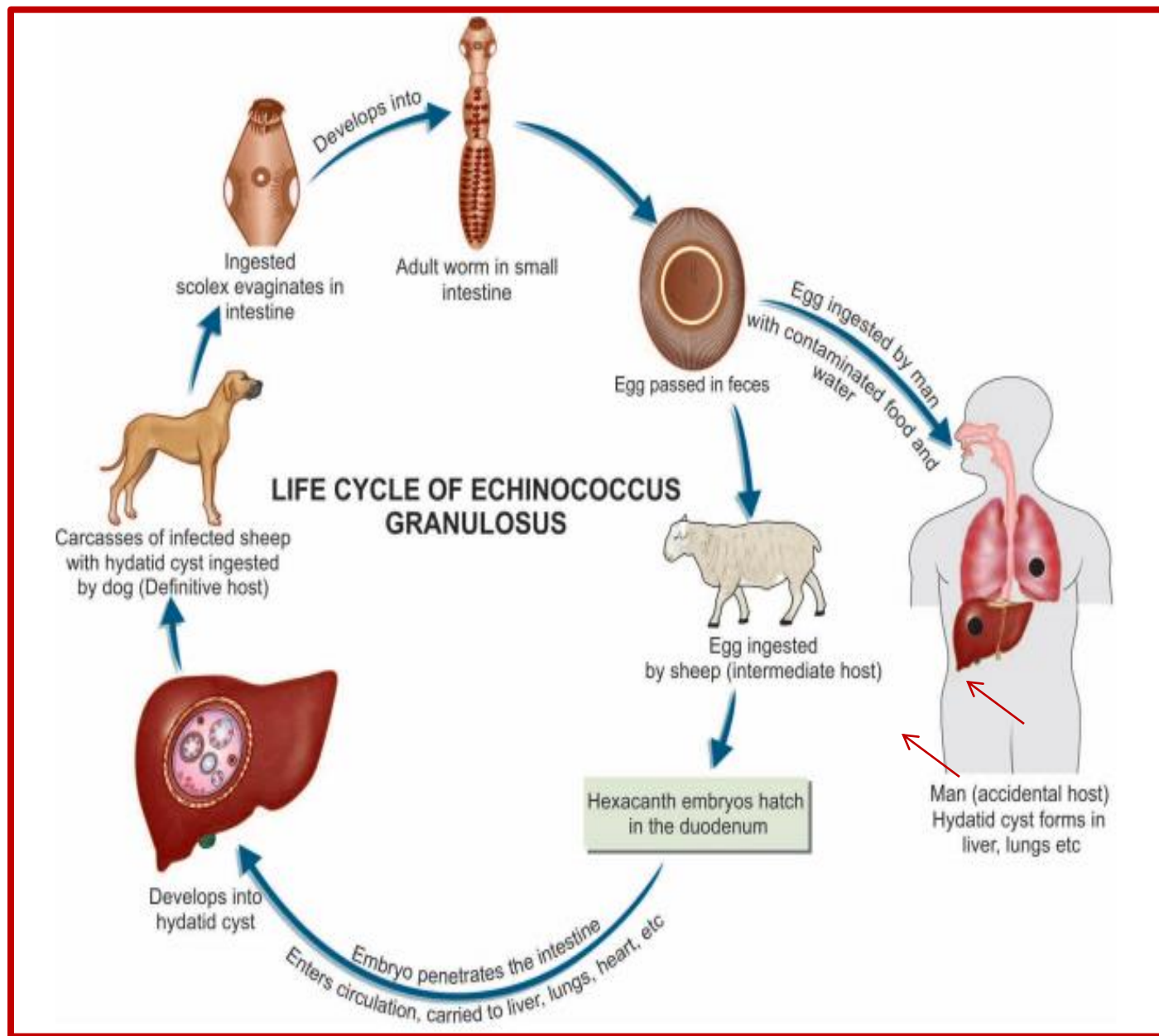
The cyst is already visible in the liver three weeks after its arrival; the cyst contains hydatid fluid which highly stimulates the allergic reactions of the patient.





Adults *Echinococcus granulosus* worm

The average adult *Echinococcus granulosus* is only 4.5 mm in length; relatively small. The worm is made up of a scolex, a small neck, and three proglottids, one at each stage of development — immature, mature, and gravid. The scolex has four suckers and about 36 hooks. Typically, this form is not seen in humans, but is commonly found in canines that serve as definitive hosts.



***Echinococcus granulosus* life cycle**

- 1) The adult *Echinococcus granulosus* resides in the small intestine of the definitive host.
- 2) Gravid ***proglottids*** release eggs that are passed in the feces, and are immediately infectious.
- 3) After ingestion by a suitable intermediate host, eggs hatch in the small intestine and release six-hooked ***oncospheres*** that penetrate the intestinal wall and migrate through the circulatory system into various organs, especially the liver and lungs.
- 4) In these organs, the ***oncosphere*** develops into a thick-walled ***hydatid cyst*** that enlarges gradually, producing ***protoscolices***. The definitive host becomes infected by ingesting the cyst-containing organs of the infected intermediate host.
- 5) After ingestion, the ***protoscolices*** evaginate, attach to the intestinal mucosa, and develop into adult stages in 32 to 80 days.

Humans are accidental hosts, and become infected by ingesting eggs. Oncospheres are released in the intestine, and hydatid cysts develop in a variety of organs. If cysts rupture, the liberated protoscolices may create secondary cysts in other sites within the body (secondary echinococcosis).

Infection is mostly asymptomatic and discovered accidentally.

Clinical disease only develops when the hydatid cyst has grown large enough to cause obstructive symptoms. Disease mainly results from pressure effects caused by the enlarged cysts.

The primary hydatid cyst occurs in the liver. The usual manifestations are hepatomegaly, pain, and obstructive jaundice. The next common site is the lung.

Treatment

Surgical removal of the hydatid cyst has been considered the treatment of choice for *Echinococcus* if it is located in an appropriate area for surgery.

In situations where the hydatid cyst was inoperable, the medications mebendazole, albendazole, and praziquantel were particularly useful.



Alnoor University Collage

كلية النور الجامعة

Pharmacy Department

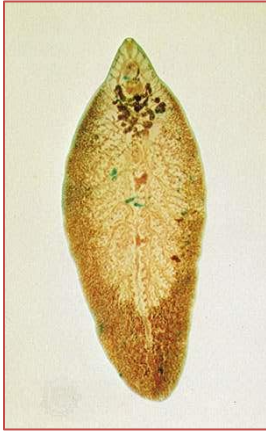
قسم الصيدلة

Medical Microbiology II

2022

م.م ندى خيرى يونس

MS.c Medical microbiology



Taenia spp

1- *T. saginata* ...The beef tapeworm

2- *T. solium* ... The pork tapeworm

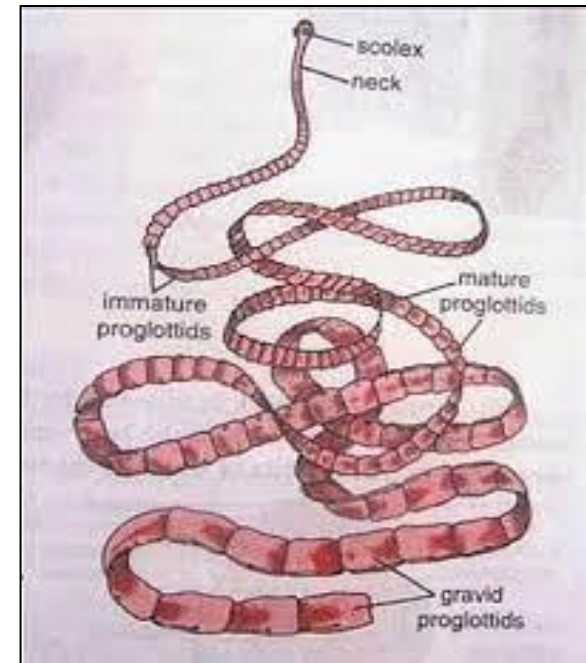
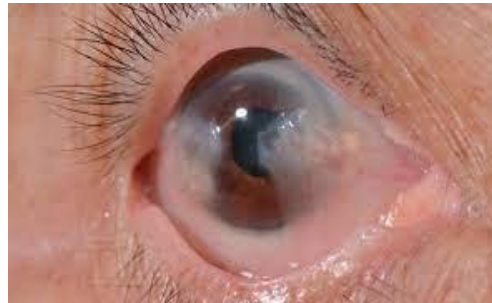
- Both are so named because cattle and swine (pig), respectively, serve as intermediate host & both tapeworm species are distributed worldwide in areas where beef and pork are eaten.

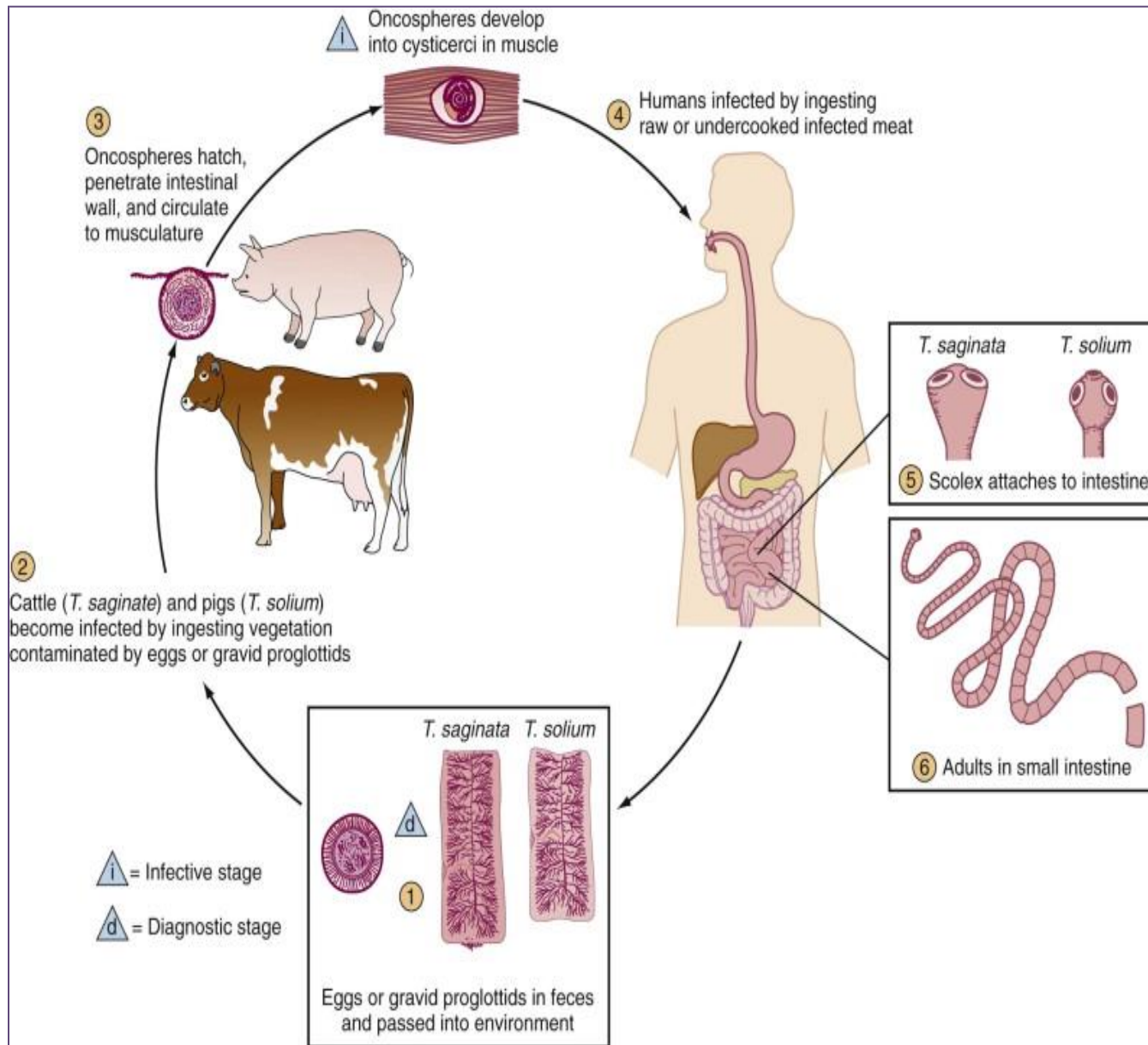
- *T. solium* tapeworm infections can lead to human cysticercosis.

- *T. solium* contains four large suckers and a rostellum containing two rows of large and small hooks. There are usually 13 hooks of each size. The scolex of *T. saginata* has four large suckers but lacks the rostellum and rostellar hooks.

- **Which is more harmful to human in *Taenia Solium* or *Taenia Saginata*?**



- Infection with *T. solium* tapeworms can result in human cysticercosis, which can be a very serious disease that can cause seizures and muscle or eye damage. *Taenia saginata* does not cause cysticercosis in humans.


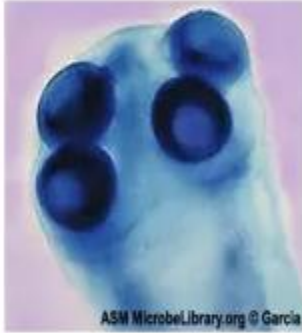


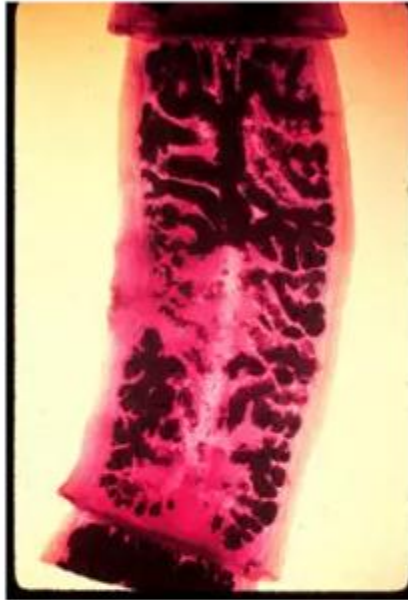
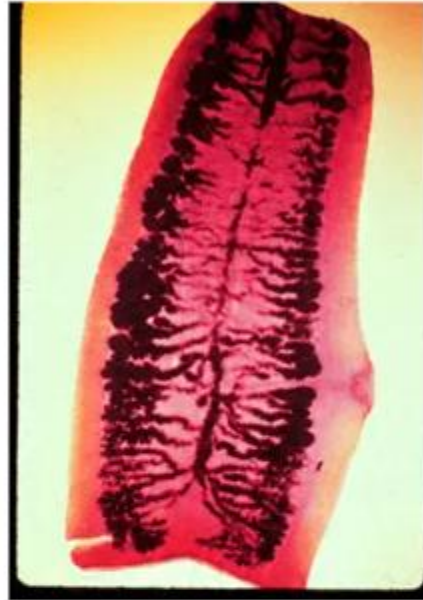



Life cycle of both *T. saginata* & *T. solium*

The major differences between *T. solium* and *T. saginata* are summarized in this

Properties	<i>Taenia solium</i>	<i>Taenia saginata</i>
Common Name	 <p><i>Taenia solium</i> (Pork Tapeworm)</p>	 <p><i>Taenia saginata</i> (Beef Tapeworm)</p>
Definitive host	Human	Human
Intermediate Host	Pig	Cow/Cattle
Disease	Taeniasis and Cysticercosis	Taeniasis only
Transmission	Infection is common among those eating raw or insufficiently cooked measly pork containing the cysticerci.	Human beings are infected through the eating of undercooked beef containing the cysticerci ("measly" beef)
Size	Size of adult worm: 2-7 m	Size of adult worm: 5 m or less (sometime up to 25 m)

Properties	<i>Taenia solium</i>	<i>Taenia saginata</i>
Scolex	 <ul style="list-style-type: none"> ▪ The scolex head is globular in outline and has four circular suckers. ▪ The head is provided with the rostellum armed with double row of alternating large and small hooklets. The rostellar hooklets are shaped like daggers or Arabian poniards (armed scolex) 	 <p>ASM MicroLibrary.org © Garcia</p> <ul style="list-style-type: none"> ▪ The scolex ("head") is quadrate in outline and has four circular suckers. ▪ Rostellum and hooklets are absent (i.e. unarmed scolex)

Properties	<i>Taenia solium</i>	<i>Taenia saginata</i>
Proglottids	 <ul style="list-style-type: none"> ▪ The total number of proglottids (segments) is an average of 1000. ▪ The gravid uterus consists of a median longitudinal stem with 5- 13 compound lateral branches on each side ▪ The gravid segments are expelled passively , in chains of 5-6 at a time, and not singly. ▪ <i>T. solium</i> may produce 50,000 eggs per proglottid. 	 <ul style="list-style-type: none"> ▪ The number of proglottids varies from 1000- 2000. ▪ The gravid uterus consists of a central longitudinal stem with 15-30 lateral branches on each side; these in turn sub-branch leaving practically no space in between. ▪ The gravid segments are expelled singly ▪ <i>T. saginata</i> may produce up to 100,000 eggs per proglottid.

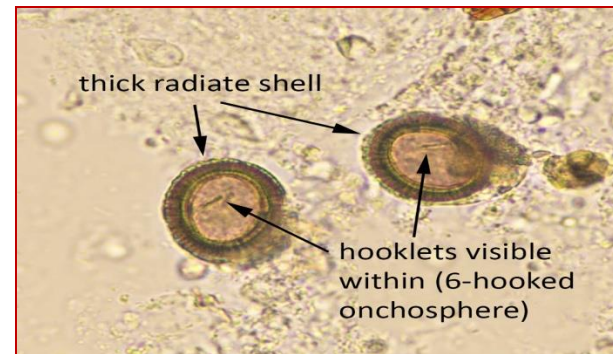
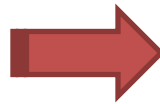
Properties	<i>Taenia solium</i>	<i>Taenia saginata</i>
Eggs	 <ul style="list-style-type: none"> ▪ The eggs are about 30-35 micrometers in diameter and are bile stained. ▪ The internal oncosphere contains six refractile hooks. ▪ The eggs are not floated in the saturated solution of NaCl. ▪ Eggs of <i>Taenia solium</i> and <i>Taenia saginata</i> are morphologically indistinguishable. 	Eggs of <i>Taenia solium</i> and <i>Taenia saginata</i> are morphologically indistinguishable.

Diagnosis & Treatment:

Diagnosis of *Taenia* tapeworm infections is made by examination of stool samples; individuals should also be asked if they have passed tapeworm segments.

Stool specimens should be collected on three different days and examined in the lab for *Taenia* eggs using a microscope.

Medications for the treatment of taeniasis include albendazole (Albenza). This drug is anti-helminthic, which means that they kill parasitic worms and their eggs. In most cases, this medication is provided in a single dose. They can take a few weeks to fully clear an infection.



Helminths

3. (Flukes)

Trematodes, commonly known as flukes, are flat, leaf-shaped worms, lack complete digestive system as they have no anus.

Oral and ventral suckers enable the parasite to attach to host tissues and obtain nutrients.

The geographical distribution of most flukes is limited, because the geographical distribution of the specific species of snails they require as intermediate hosts is also limited.

There are many types Flukes (worms) such as:

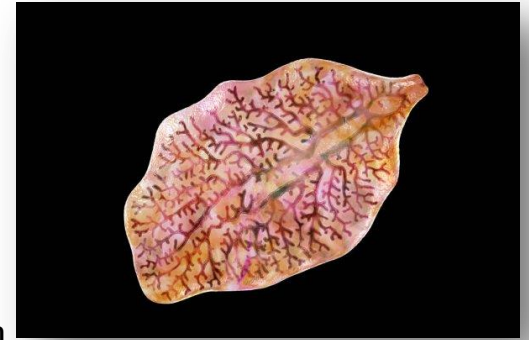
1. Blood flukes (schistosoma)
2. Liver flukes (fasciola)

Blood flukes (schistosoma)

Schistosomiasis is also known as **bilharzia**, is a disease caused by parasitic worms. However, the disease causes continued inflammation, which damages the affected organs and so the problem becomes more severe. Schistosomiasis can affect various organs such as the liver, spleen, intestine, and kidneys, and can lead to death.

Three types of schistosoma infect human:

1. *Schistosoma mansoni*
2. *S. haematobium*
3. *S. japonicum*



Schistosoma haematobium

Schistosoma haematobium (urinary blood fluke) is species of trematode, belonging to a group (genus) of blood flukes (*Schistosoma*). It is found in Africa and the Middle East. It is the major agent of schistosomiasis, the most prevalent parasitic infection in humans.

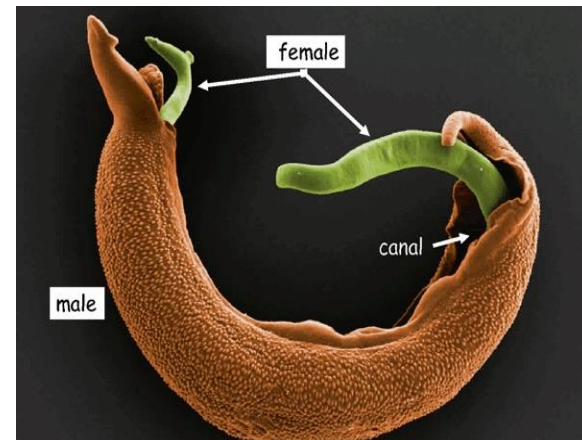
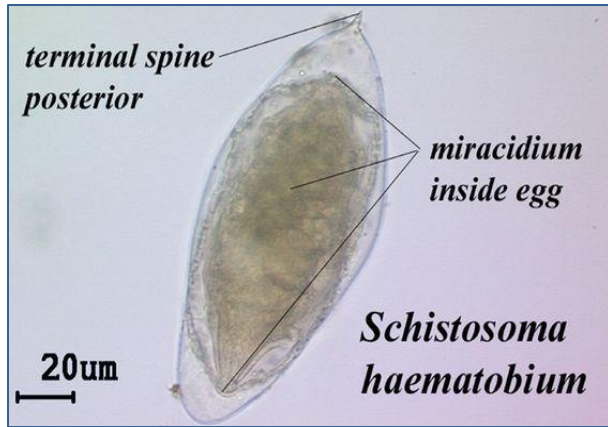
S. haematobium causes urinary schistosomiasis. Urinary schistosomiasis is often chronic and can cause pain, secondary infections, kidney damage, and even cancer.

In 1851, Theodor Bilharz described a parasitic infection (bilharzia) that would later be termed schistosomiasis. Currently, 200 million people in 74 countries have this disease.

The parasites enter the body when a person is swimming, washing, or paddling in contaminated water. They can also become infected by drinking the water or eating food that a person has washed in untreated water. The infective form of the fluke is known as *cercariae*

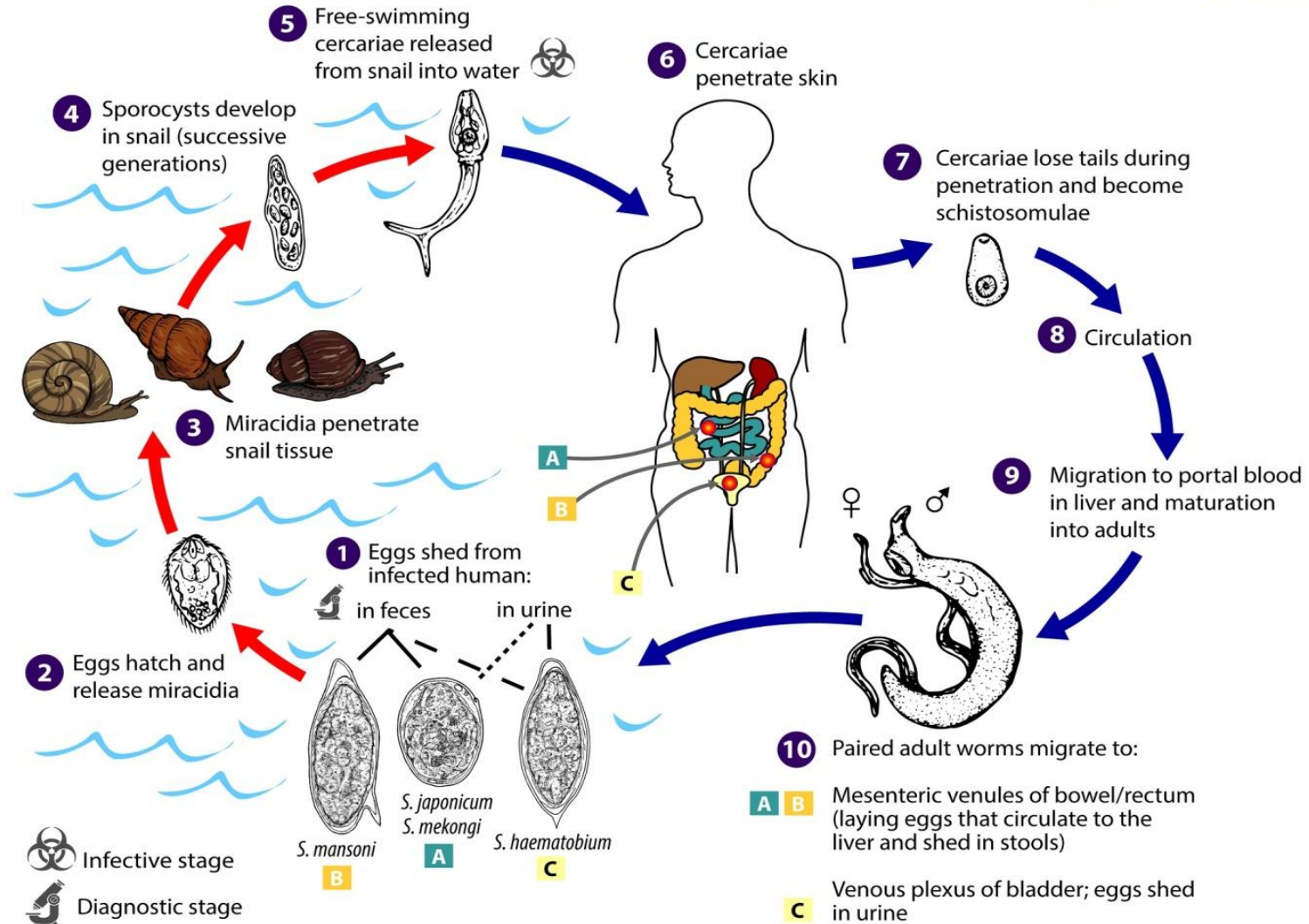


Schistosoma haematobium



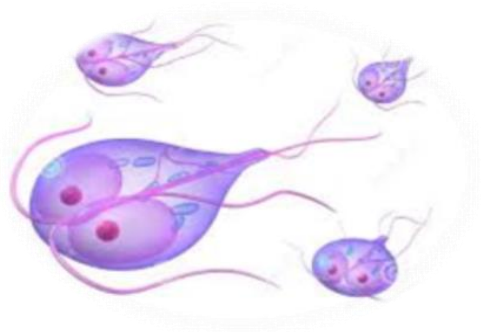
Schistosoma haematobium adult





Life cycle of schistosoma spp.





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Pharmacy Department
كلية النور الجامعة --- قسم الصيدلة
Medical Microbiology II
2022

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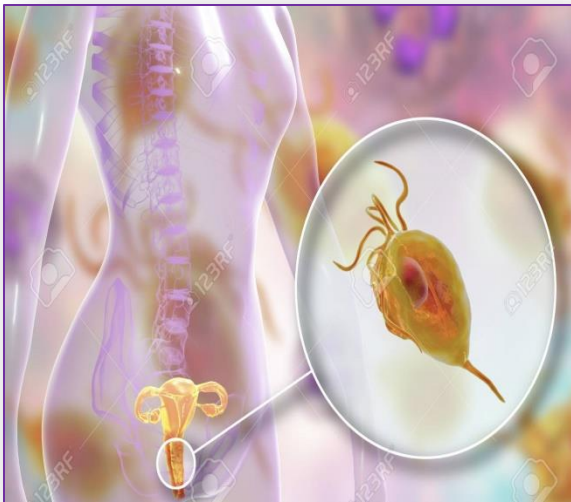
3. *Trichomonas virginalis*

A flagellated protozoan parasite and the causative agent of trichomoniasis.

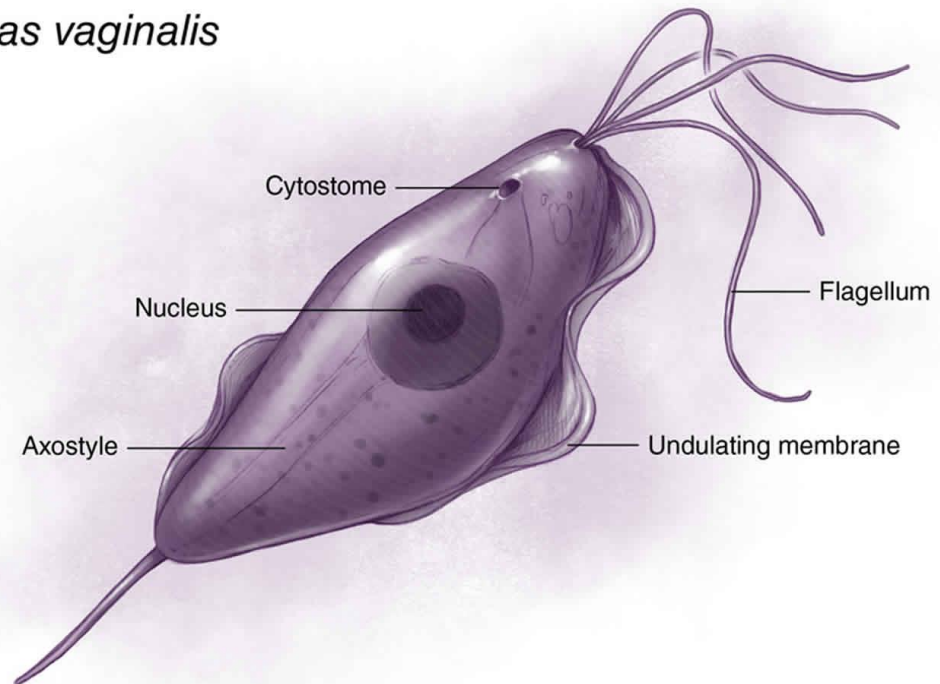
The organism has four free, anterior flagella and a fifth flagellum embedded in an undulating membrane that extends around the anterior two-thirds of the cell.

Trichomonads occurring in Humans belong to 3 species:

1. *Trichomonas. Vaginalis* (inhabits vagina)
2. *Trichomonas. Tenax* (inhabits oral cavity)
3. *Trichomonas hominis* (inhabits large intestine)



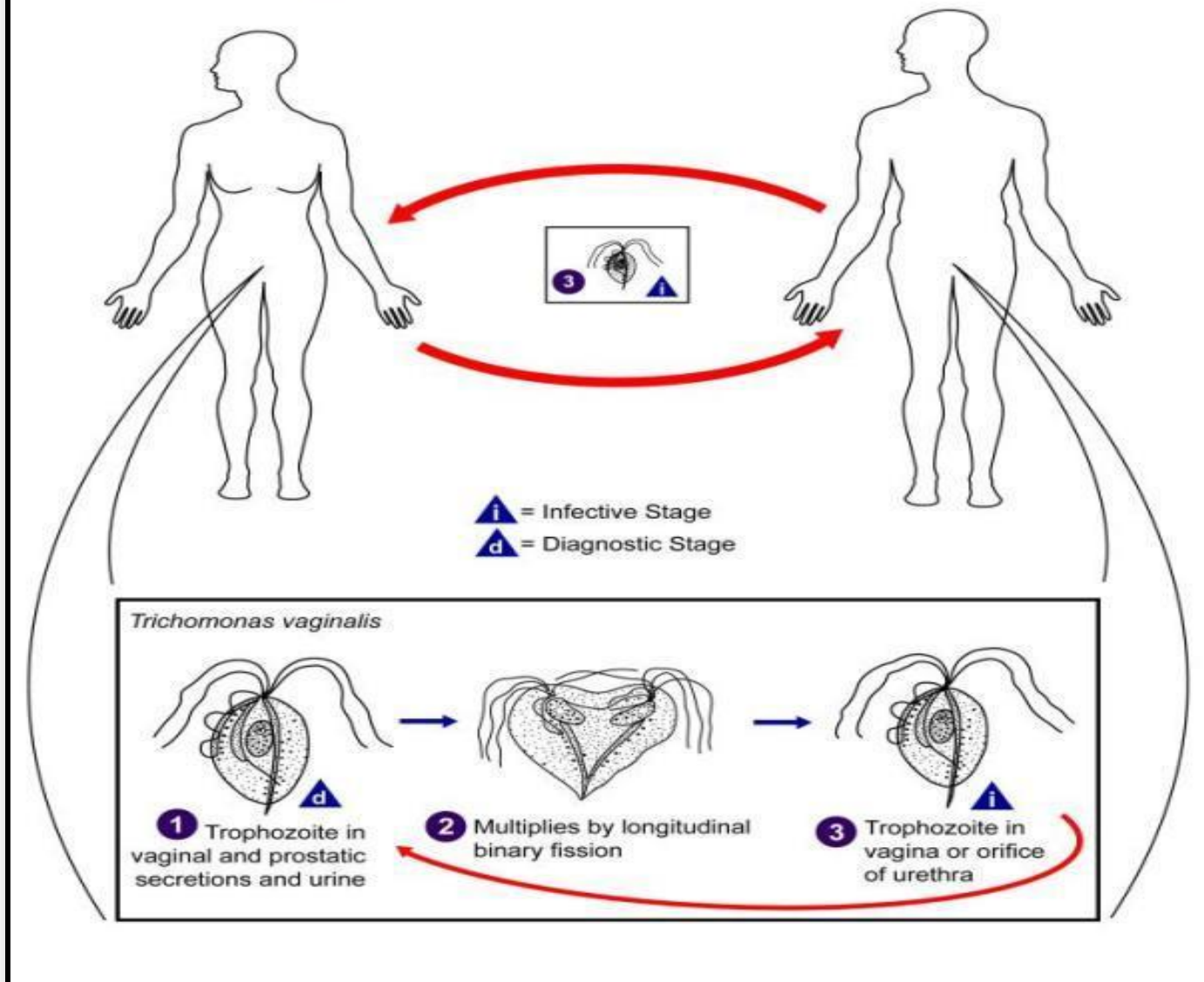
Trichomonas vaginalis



- *Trichomonas vaginalis* is anaerobic, flagellated protozoan parasite and the causative agent of **trichomoniasis**. It is the most common pathogenic protozoa infection of humans.
- *Trichomonas vaginalis* is usually transmitted among humans by sexual intercourse. No animal reservoirs exist.
- *T. vaginalis* occurs only as the trophozoite.
- No cystic form.
- Treatment for trichomoniasis:
- Metronidazole is the drug of choice for treatment.

Trichomoniasis

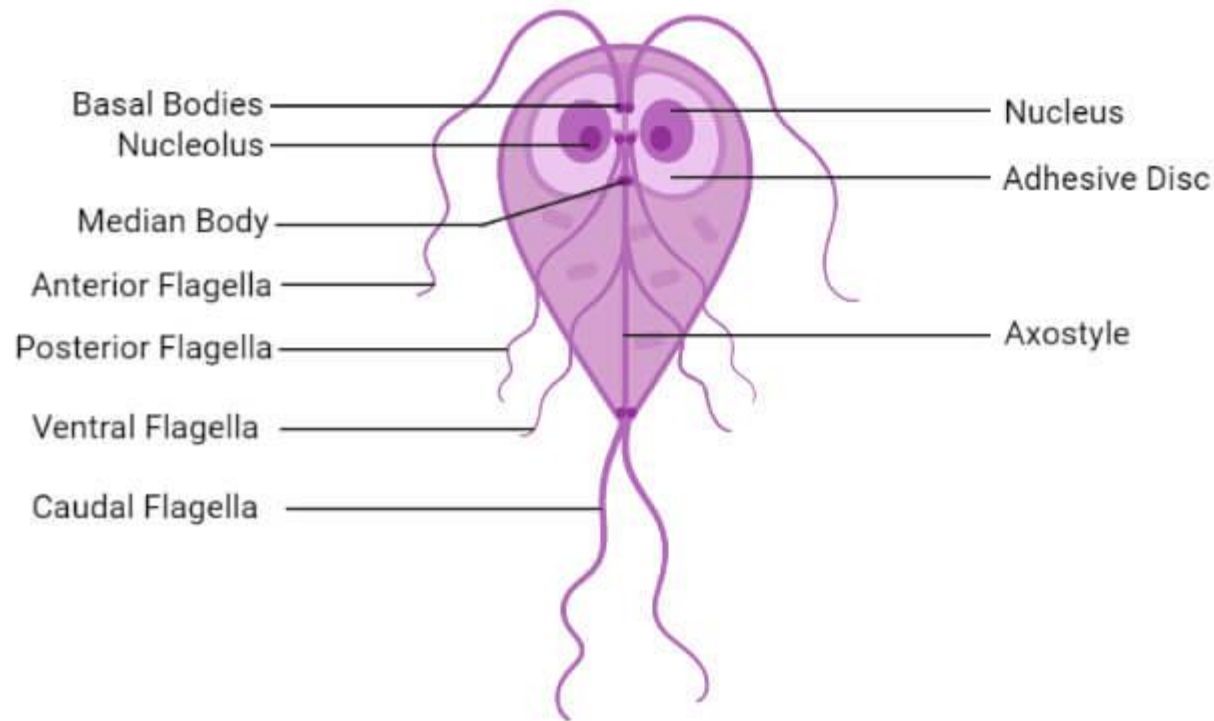
(*Trichomonas vaginalis*)



Life cycle of *Trichomonas vaginalis*

Giardia lamblia

Giardia duodenalis (Giardia lamblia or Giardia intestinalis)

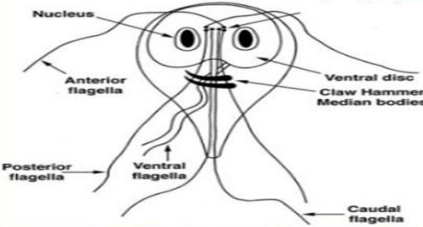
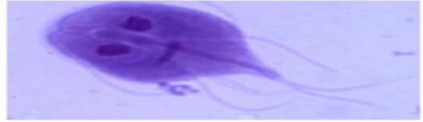


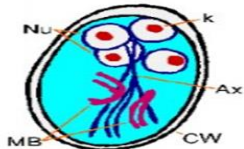


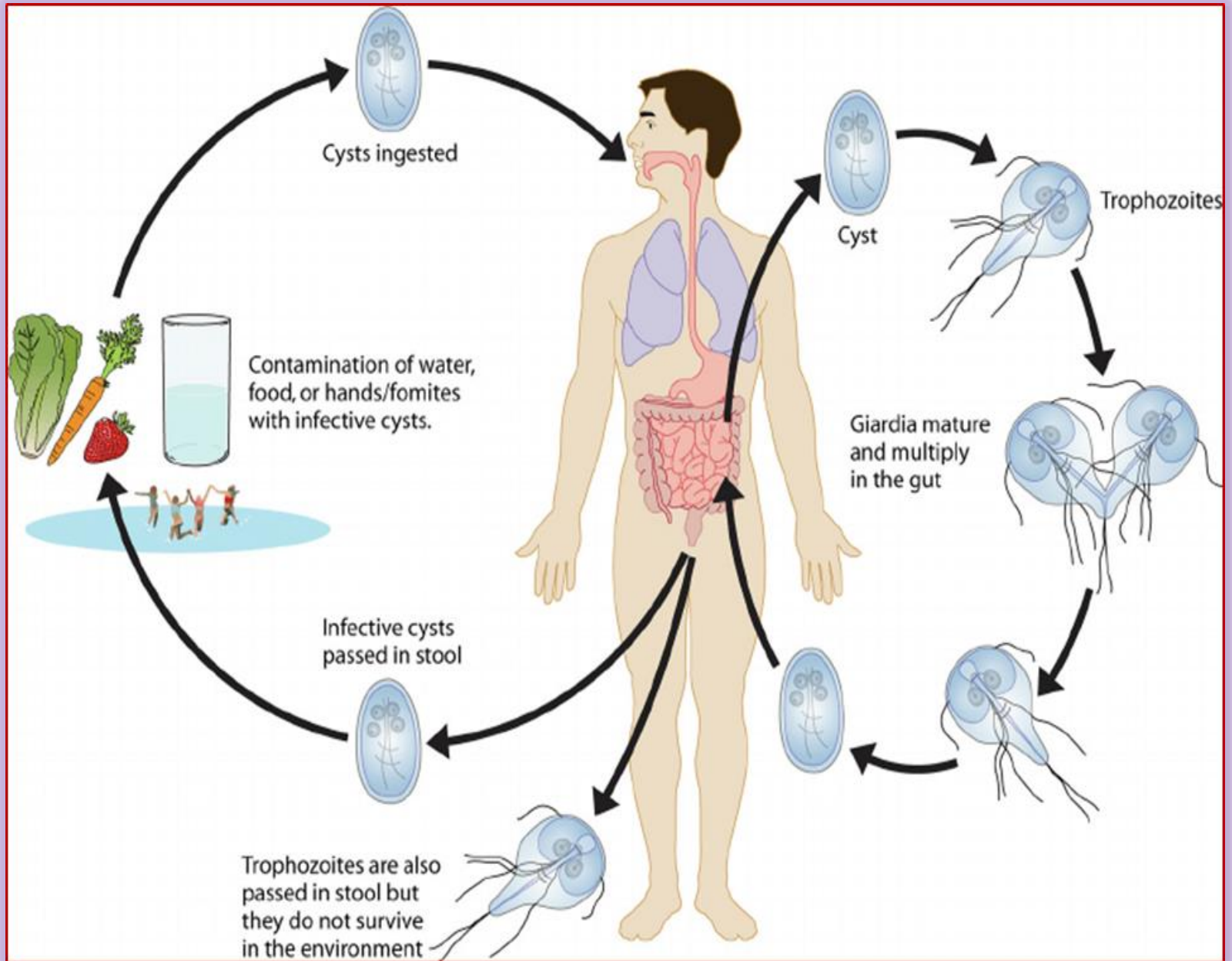
4. *Giardia lamblia*

- *Giardia lamblia*, is a flagellated parasitic microorganism that colonizes and reproduces in the small intestine, causing giardiasis.
- The parasite attaches to the epithelium by a ventral adhesive disc or sucker, and reproduces via binary fission.
- Giardiasis is an infection in a small intestine. It's caused by a microscopic parasite called *Giardia lamblia*. Giardiasis spreads through contact with infected people. And can get giardiasis by eating contaminated food or drinking contaminated water.
- For those who do get sick, signs and symptoms usually appear one to three weeks after exposure and may include:
 - Watery diarrhea.
 - Nausea.
 - Weight loss.

Treatment

Metronidazole (Flagyl). Metronidazole is the most commonly used antibiotic for giardia infection. ..

Morphology	Trophozoite
 	<p>Trophozoites are 10-20 μm long, teardrop shaped and have:</p> <ul style="list-style-type: none">. Two nuclei with central karyosome. 4 pairs of flagella. 2 longitudinal axostyles crossed by 2 parabasal bodies.o Convex dorsal surface & flat/concave ventral surface with an adhesive disc
Morphology	Cyst
 	<p>Mature cysts are 8–12 μm long, ovoid, with a granular cytoplasm containing:</p> <ul style="list-style-type: none">. 4 nuclei. median bodies. longitudinally oriented fibrils. 



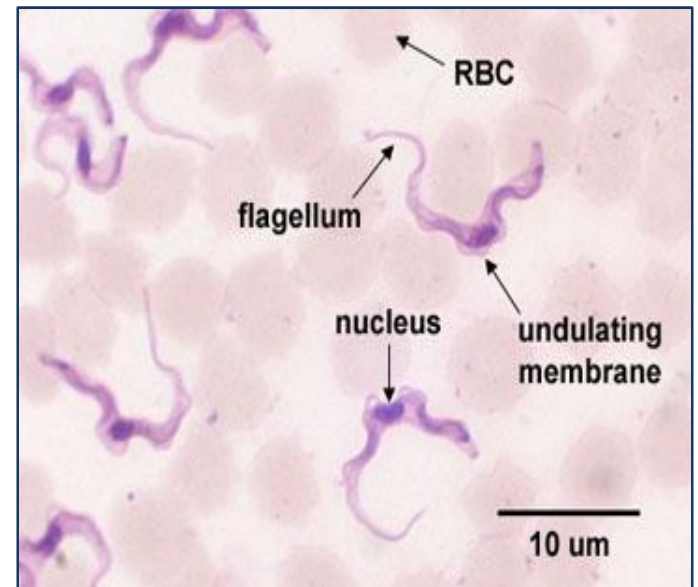
Life cycle of *Giardia lamblia*

Blood & Tissue Flagellates

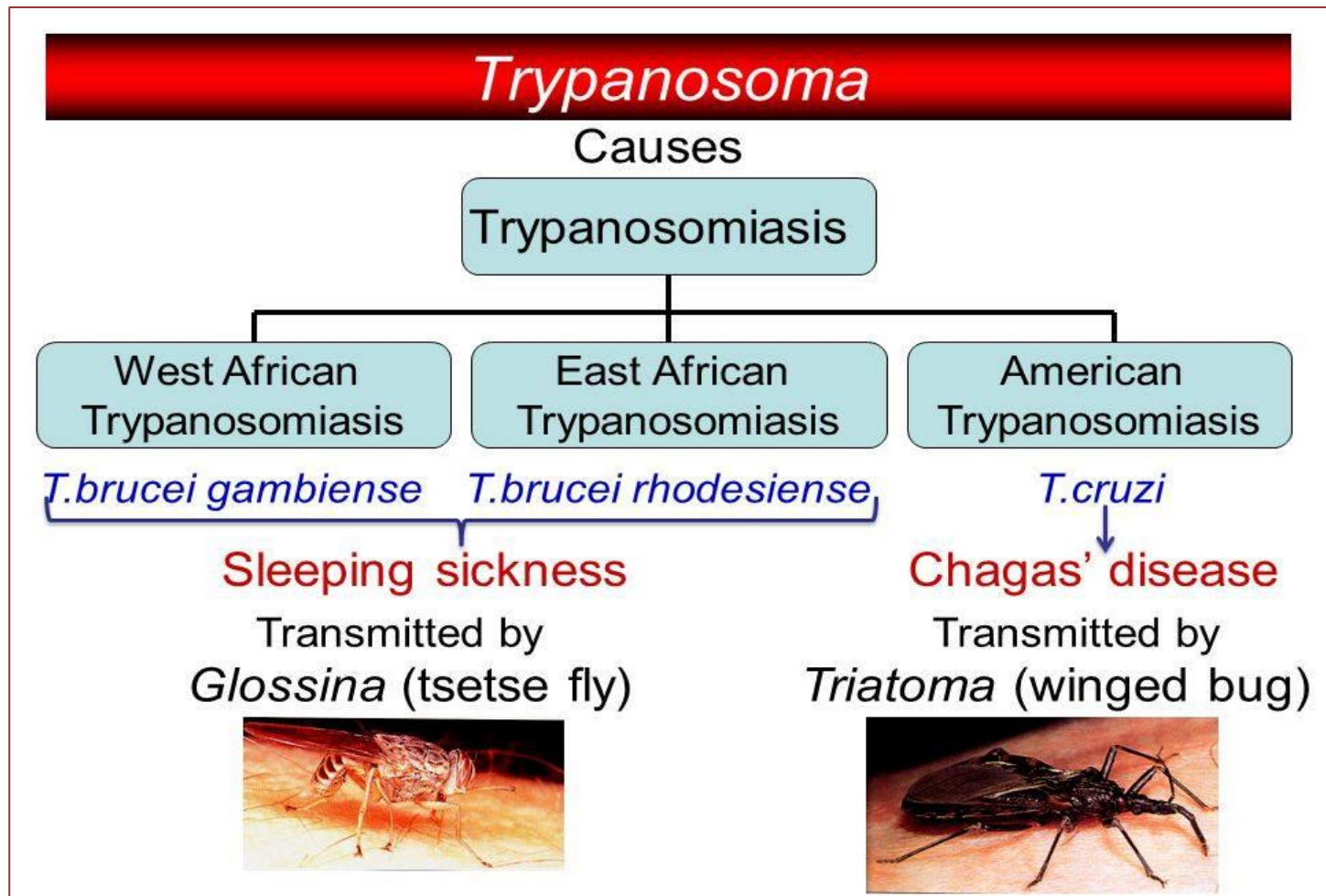
- 1) Flagella “Hemoflagellates”.e.g. *Trypanosoma spp* & *Leishmania spp*
- 2) Non motile sporozoan
 - a- Blood sporozoan.e.g. *Plasmodium spp*
 - b- Tissue sporozoan.e.g *Toxoplasma gonidii*

Types of *Trypanosoma spp*

1. *Trypanosoma cruzi*
2. *Trypanosoma gambiense*
3. *Trypanosoma rhodesiense*



Classification



Plasmodium spp

Plasmodium spp. is a genus of protozoal (single-celled) parasites that are usually transmitted by mosquitoes.

✚ Four species of *plasmodium* infect humans:

1. *Plasmodium viva*
2. *Plsmodium ovale*
3. *Plasmodium malariae*
4. *Plasmodium falciparum*



and trigger different forms of malaria.

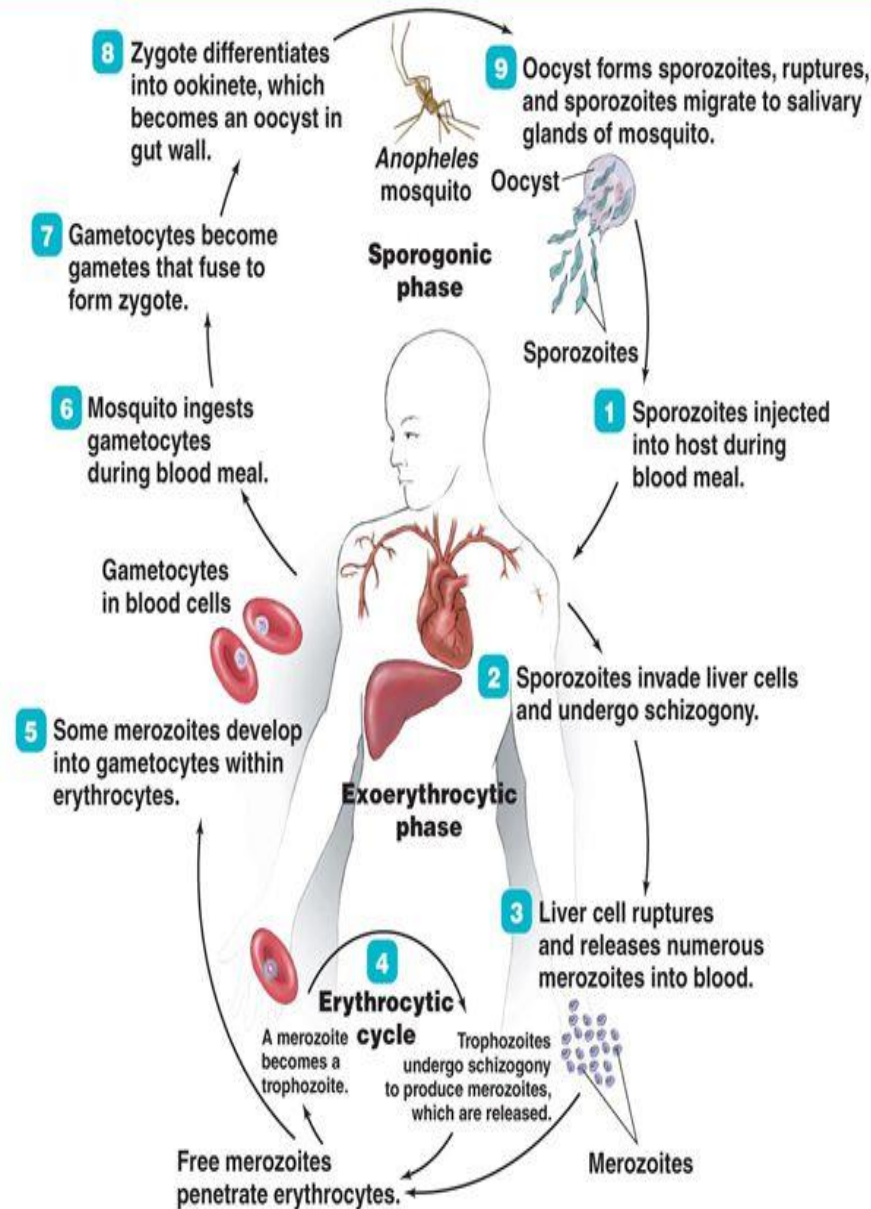
✚ Over 400 million people are infected with *plasmodium* and 2-3 million (usually children) die annually.

✚ Females of sixty different species of mosquito genus *Anopheles* serve as vectors of *plasmodium*. (Adult male mosquitoes do not feed).

✚ The life cycle of *plasmodium* has three stages:

- 1- The exo-erythrocytic cycle
- 2- The erythrocytic cycle
- 3- The sporogenic cycle

The life cycle of *Plasmodium*

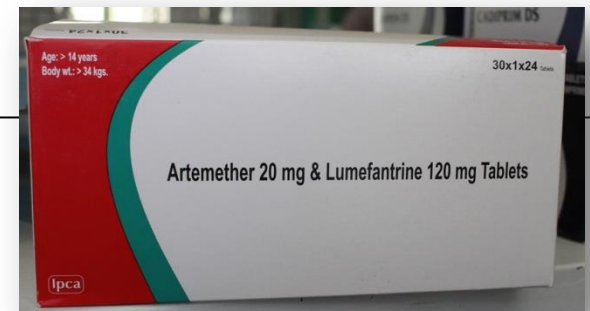


- Malaria infection begins when an infected female *Anopheles* mosquito bites a person, injecting *Plasmodium* parasites, in the form of sporozoites, into the bloodstream.
- The sporozoites pass quickly into the human liver.
- The sporozoites multiply asexually in the liver cells over the next 7 to 10 days, causing no symptoms.
- In all types of malaria the periodic febrile response is caused by rupture of mature schizonts. In *P.vivax* and *P.ovale* malaria, a brood of schizonts matures every 48 hr.
- Where as in *P.malariae* disease, fever occurs every 72 hours.
- Some people who have malaria symptoms starts with:
Shivering and chills, followed by a high fever, followed by sweating and a return to normal temperature. Malaria signs and symptoms typically begin within a few weeks after being bitten by an infected mosquito.

Treatment

The most common antimalarial drugs include:

- Artemisinin-based combination therapies (ACTs). ACTs are, in many cases, the first line treatment for malaria.
- Chloroquine phosphate.



Toxoplasma gondii



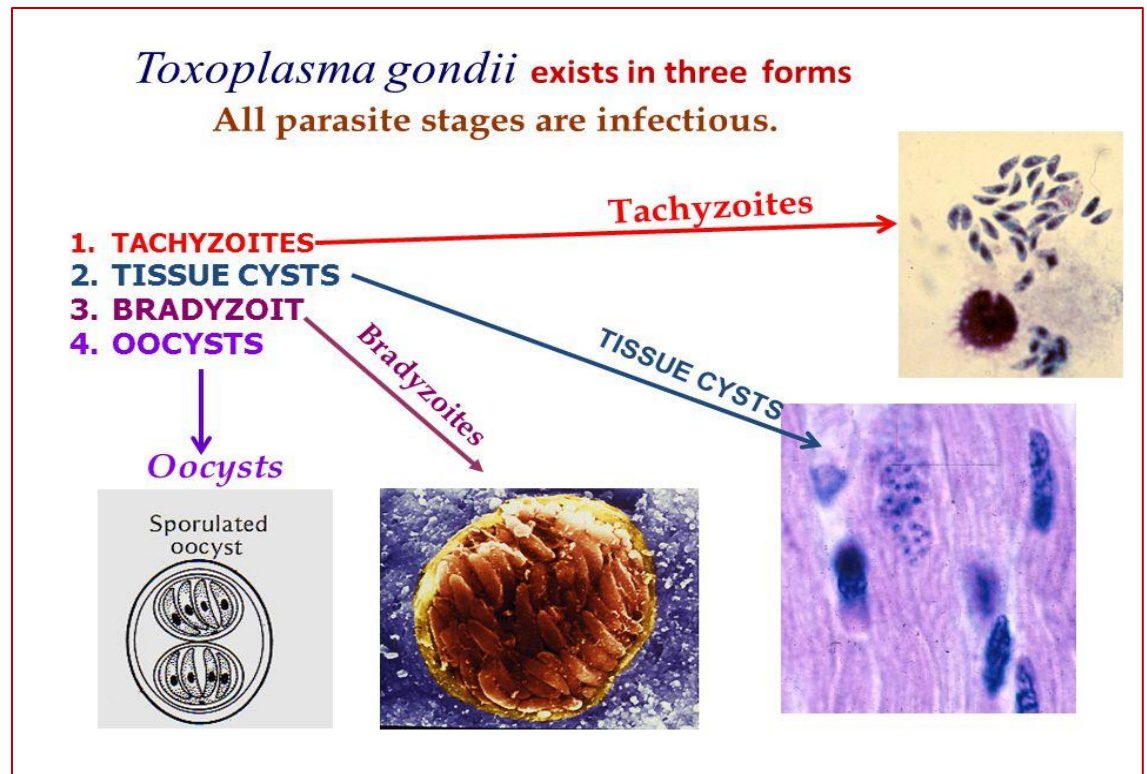
- *Toxoplasma gondii* is an obligate intracellular parasitic one-celled eukaryote that causes the infectious disease toxoplasmosis.
- Toxoplasmosis is an infection caused by a single-celled protozoan parasite called *Toxoplasma gondii*, which usually affects warm-blooded animals, including humans. The infection is most commonly acquired from contact with cats and their feces or undercooked meat.
- Wild & domestic mammals and birds are the major reservoir for the protozoan, and cats are the definitive host, in which the protozoan reproduces sexually.
- Humans typically become infected by ingesting under-cooked meat containing the parasite.
- The protozoa can also cross the human placenta to infect the fetus. kissing or owning a cat, working in the garden contaminated with cat feces or receiving an organ transplant or blood transfusion from an infected donor are among the common ways in which people can become infected with the disease.
- The majority of people infected with *T.gondii* show fever-producing illness with other symptoms resembling pneumonia, hepatitis, or myocarditis.
- Toxoplasmosis is more severe in two populations: AIDS patients and fetuses.

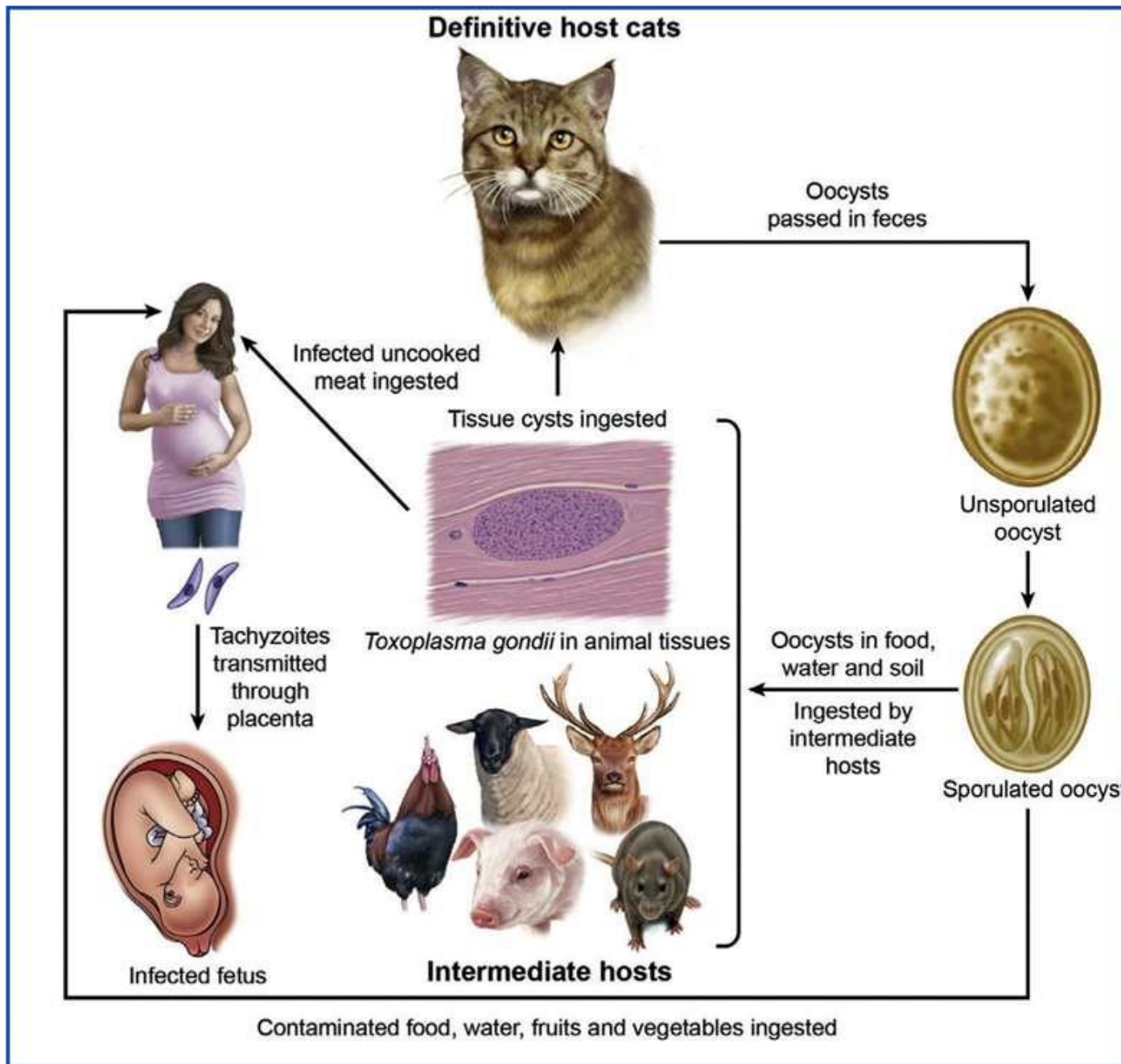
- Transplacental transfer from pregnancy; it can result in spontaneous abortion.

Treatment

Sulphadiazine and pyrimethamine are two drugs widely used for 3-4 weeks.

Controlling the incidence of *T. gondii* is difficult because so many hosts harbor the parasite. A vaccine for cats is important to prevent and reduce the chance of pet-to-owner transmission. The best prevention is to cook or deep-freeze meats and avoid contact with contaminated soil.

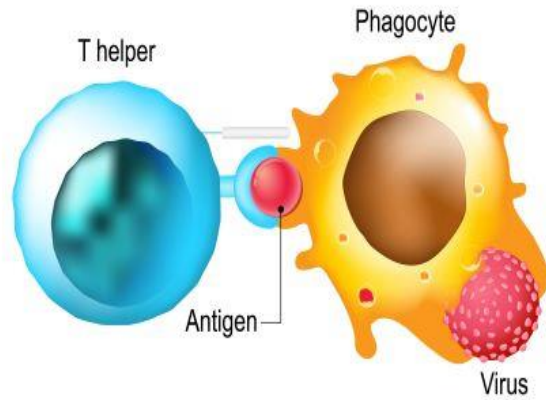




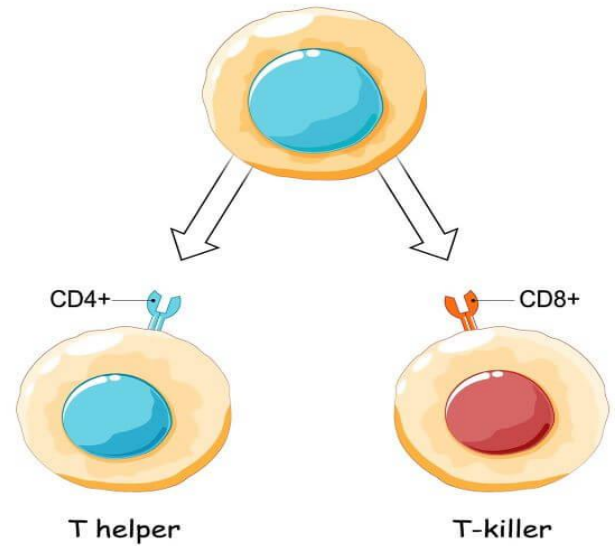
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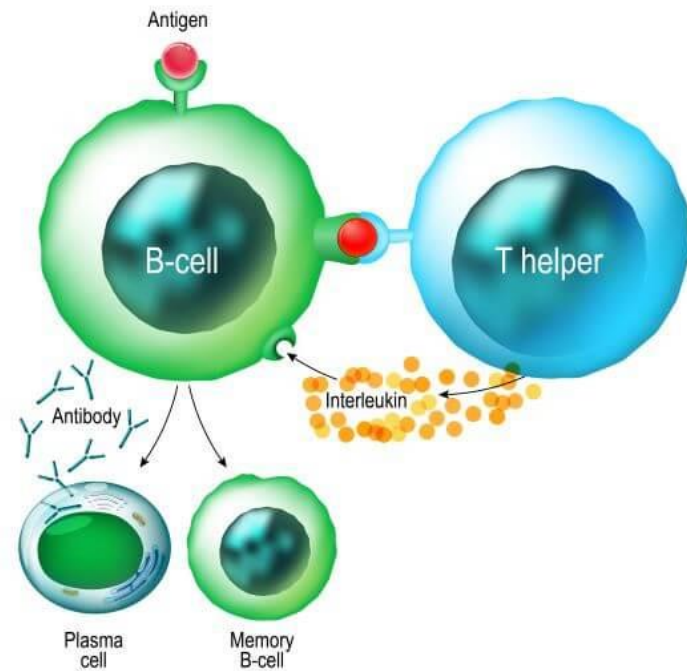
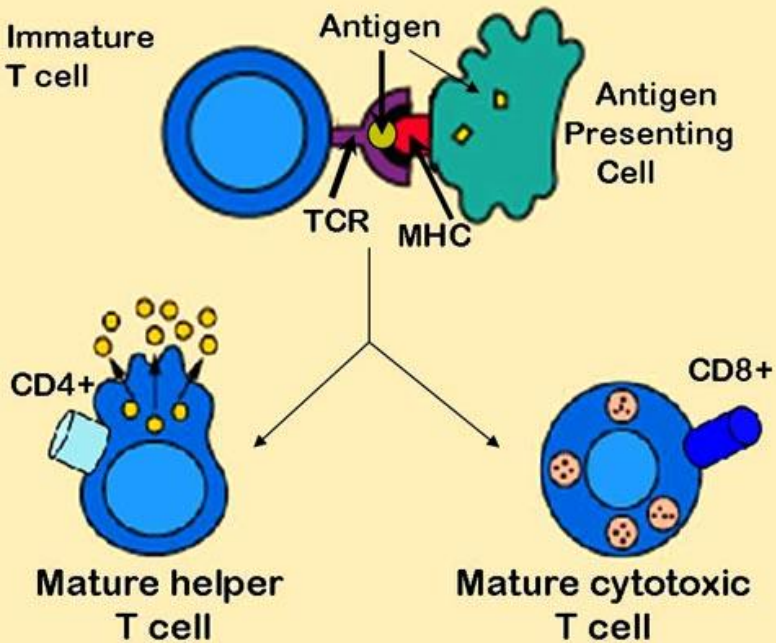
ACTIVATION



T cell
(adaptive immune response)



Immature
T cell



pathways of complement activation

The main function of complement proteins is to aid in the destruction of pathogens by piercing their outer membranes (cell lysis) or by making them more attractive to phagocytic cells such as macrophages (a process known as opsonization).

Some complement components also promote inflammation by stimulating cells to release histamine and by attracting phagocytic cells to the site of infection.

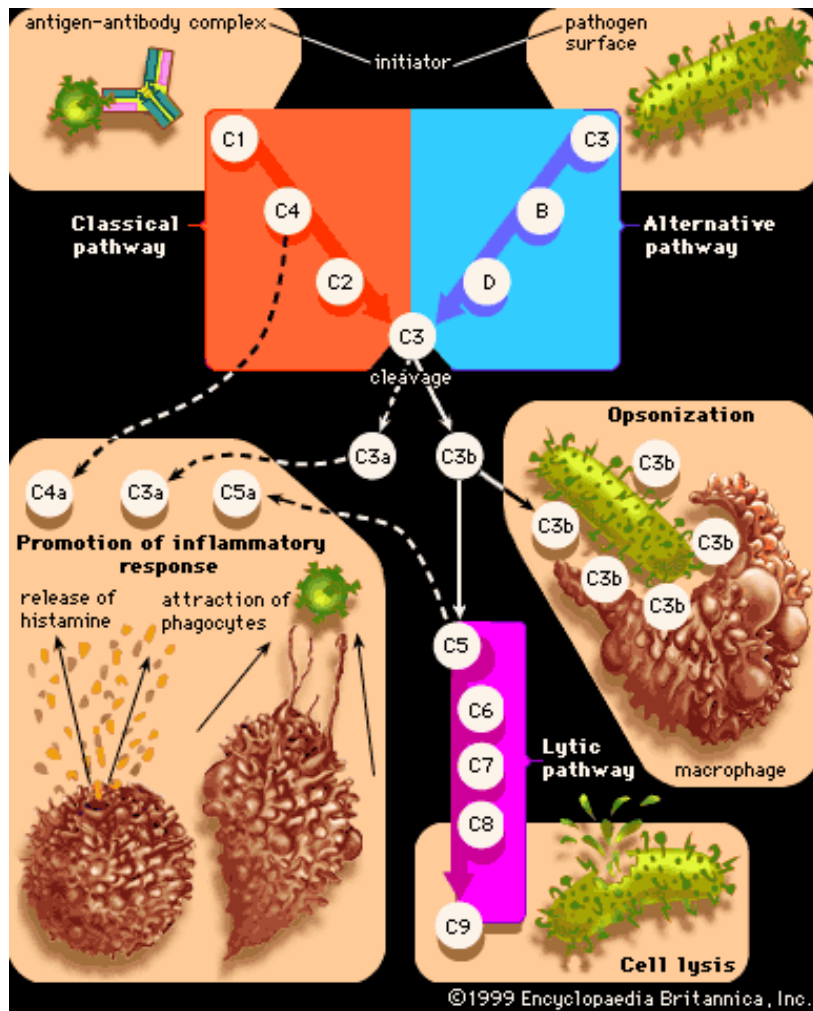
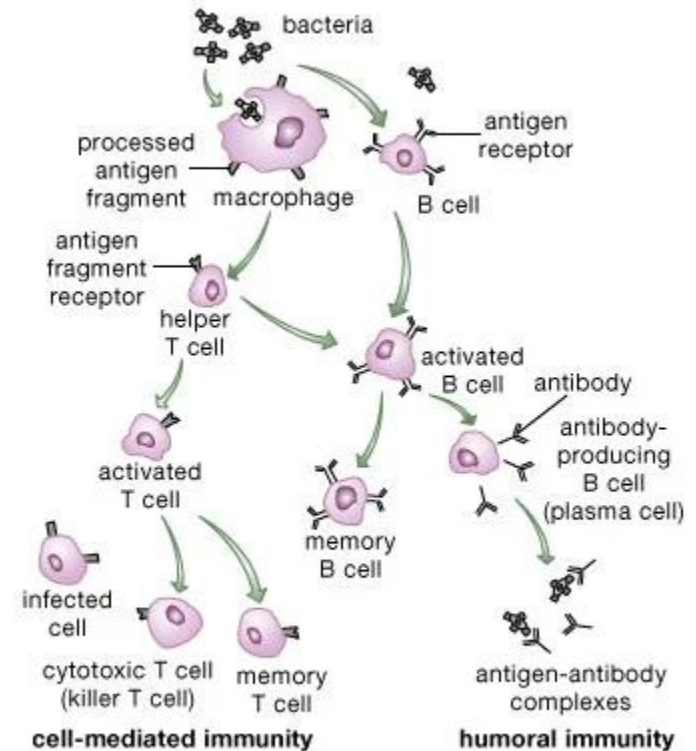


diagram of the activities of T and B cells

Acquired immunity depends on the activities of T and B lymphocytes (T and B cells). One part of acquired immunity, humoral immunity, involves the production of antibodies by B cells. The other part, cell-mediated immunity, involves the actions of T cells. When an antigen (such as a bacterium) enters the body, it is attacked and engulfed by macrophages, which process and display parts of it on their cell surface. A helper T cell, recognizing the antigen displayed, initiates maturation and proliferation of other T cells. Cytotoxic (killer) T cells develop and attack foreign and infected cells. B cells stimulated by the presence of antigen are activated by helper T cells to divide and form antibody-producing cells (plasma cells). Released antibody binds to antigen, marking the cell for destruction. Helper T cells also induce the development of memory T and B cells needed to mount future immune responses on reinfection with the same pathogen.



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