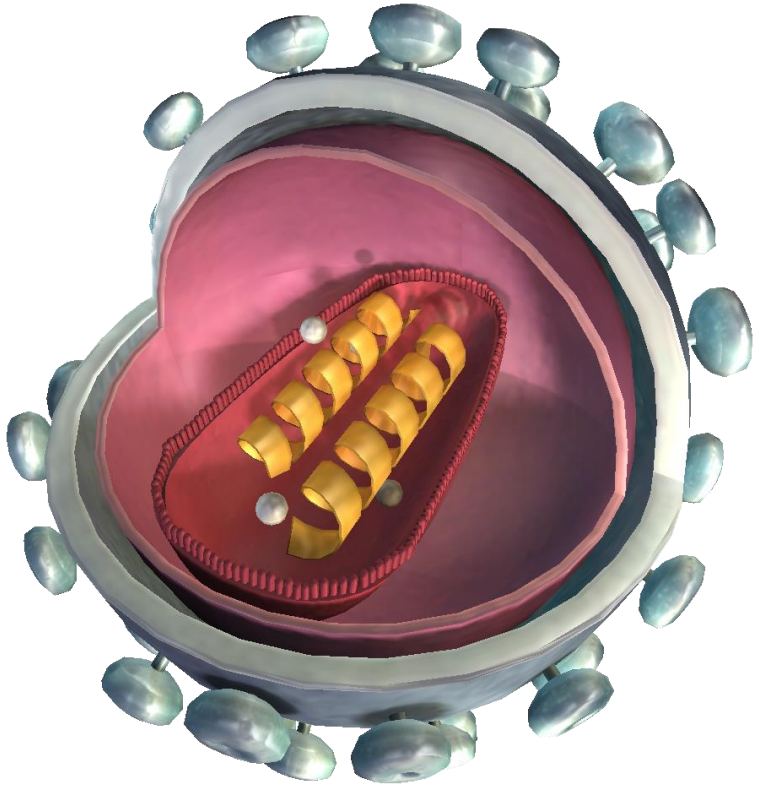
Lec1:

General properties of viruses

م.م ايهاب مجيد عباس

What are viruses?



WHAT ARE VIRUSES?

- 1. They are small size (20-300 nm in diameter) retaining infectivity after passage through filters able to hold back bacteria.
- 2. They are totally dependent upon a living cell, either eukaryotic or prokaryotic, for replication and existence. Viruses are obligate intracellular parasites.
- 3. They possess only one species of nucleic acid, either DNA or RNA.
- 4. They have a component - a receptor binding protein for attaching to cells.

COMPARISON BETWEEN VIRUSES AND BACTERIA

•	Viruses	Bacteria
1 -Size /	20-300 nm	1000 nm
2 –Genome/ (type of nucleic acid) : not both	DNA or RNA but	DNA and RNA
3 -Cell wall/	Envelope in some viruses	Cell wall
4- Ribosomes /	No ribosomes	Ribosomes
5 -Multiplication by binary fission/	-	+
6 /Sensitivity to antibiotics /	_	+
7 Growth in culture media /	Grow only in living host cell	Grow in culture media

Viral Structure - Overview

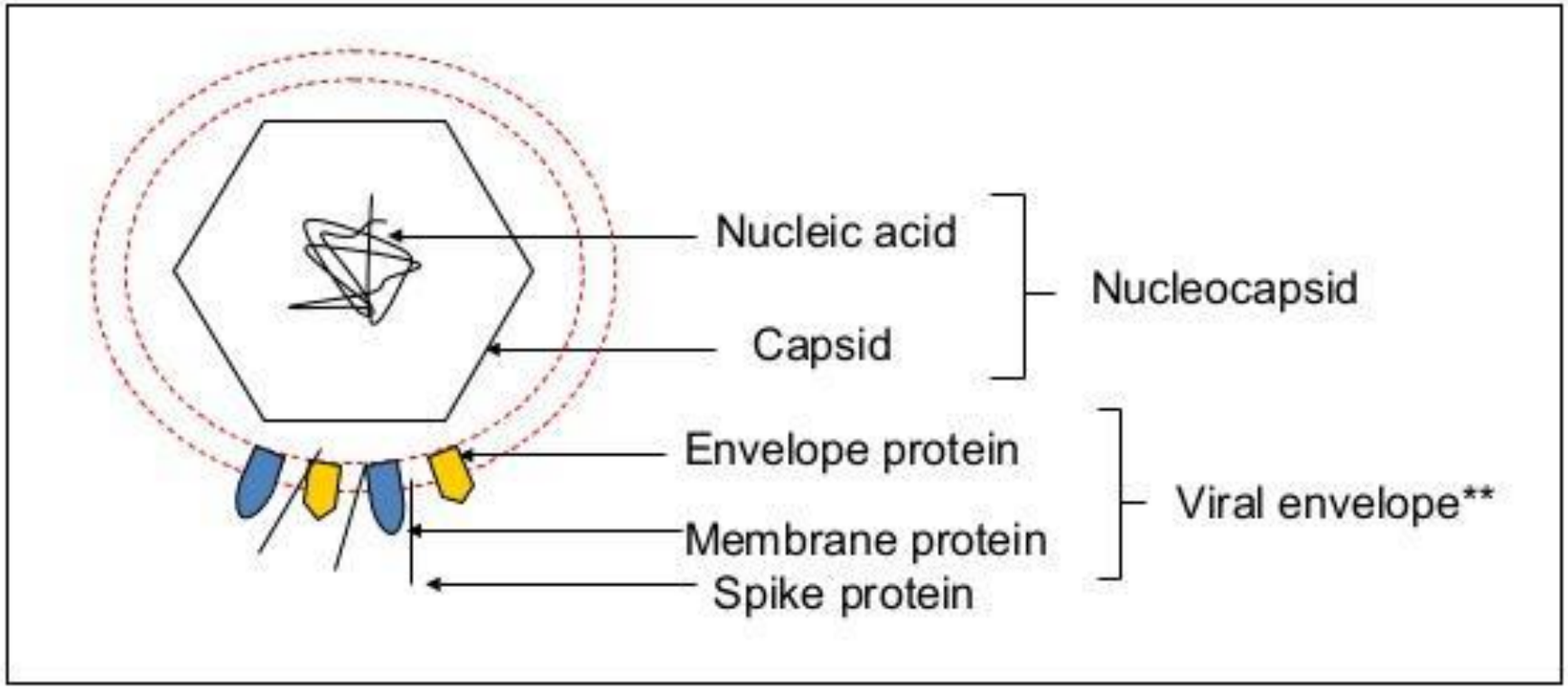


Fig 1. Schematic overview of the structure of animal viruses

** does not exist in all viruses

THE STRUCTURE OF VIRUSES:

1. Viral nucleic acid:

- The viral nucleic acid is located internally and can be either single- or double-stranded RNA or DNA. The nucleic acid can be either linear or circular.
- The DNA is always a single molecule, the RNA can exist either as a single molecule or in several pieces (segmented).
- Some RNA viruses are positive polarity and others are negative polarity.
- Positive polarity is defined as an RNA with same base sequence as the mRNA.
- Negative polarity has a base sequence that is complementary to the mRNA.

THE STRUCTURE OF VIRUSES:

2. Capsid:

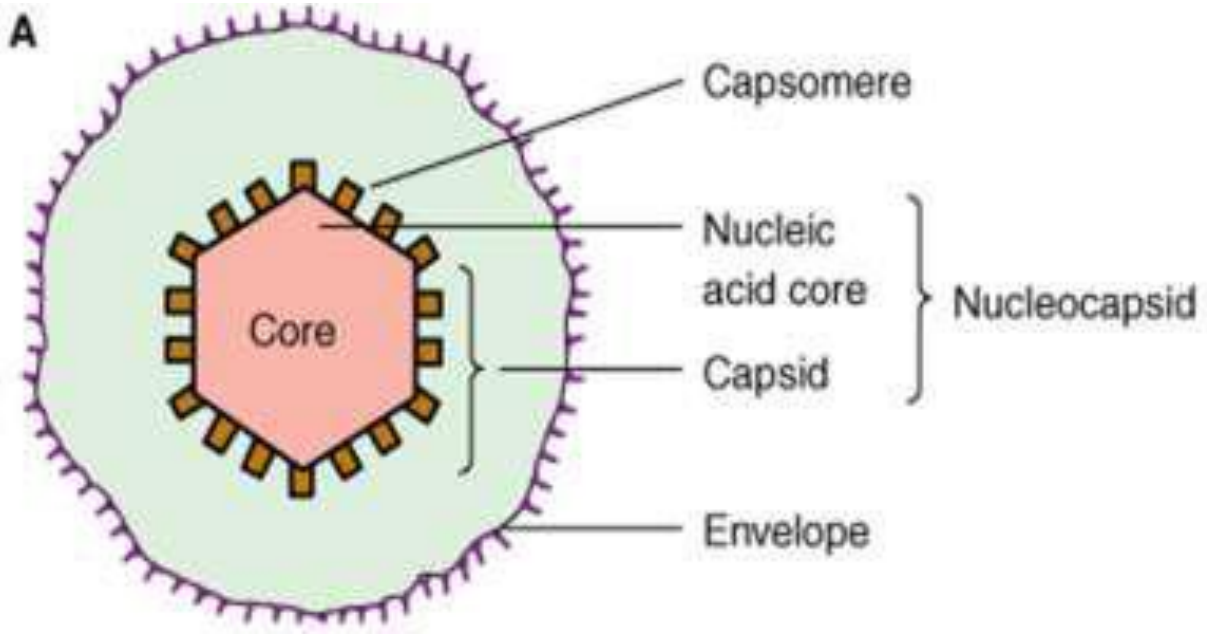
- The protein shell, or coat, that encloses the nucleic acid genome and mediates the attachment of the virus to specific receptors on the host cell surface. This interaction of the viral proteins with the cell receptor is the major determinant of species & organ specificity. The capsid proteins protect the genome from degradation by nucleases
- **3. Capsomeres:**
- Morphologic units seen in electron microscope. Each capsomere, consisting of one or several proteins.
- Naked viruses are composed of nucleic acid + capsid (nucleocapsid)

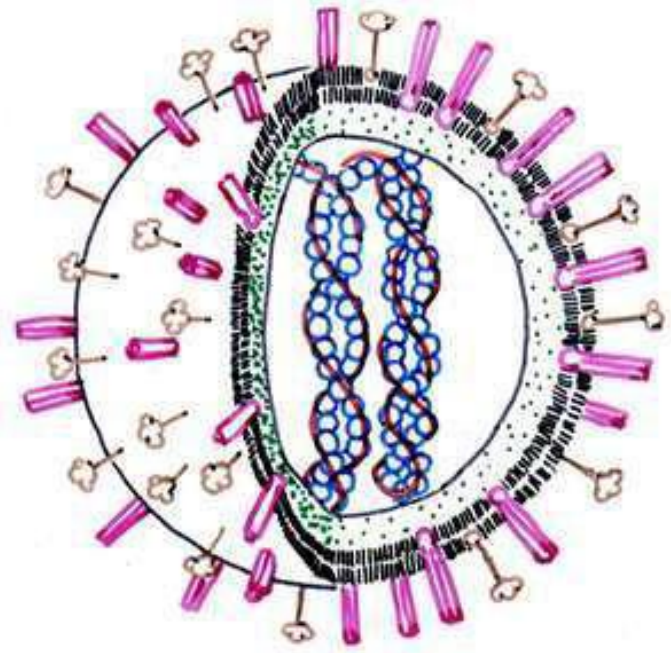
THE STRUCTURE OF VIRUSES:

- **4. Viral envelope**
- The envelope is a lipoprotein membrane composed of lipid derived from
- the host cell membrane and protein that is virus- specific.
- Furthermore, there are frequently **glycoproteins** in form of spike-like
- projections on the surface, which attach to host cell receptors.

THE STRUCTURE OF VIRUSES:

- The presence of an envelope confers instability on the virus.
- Enveloped viruses NA + capsid + envelope
- The whole virus particle is called **virion**.

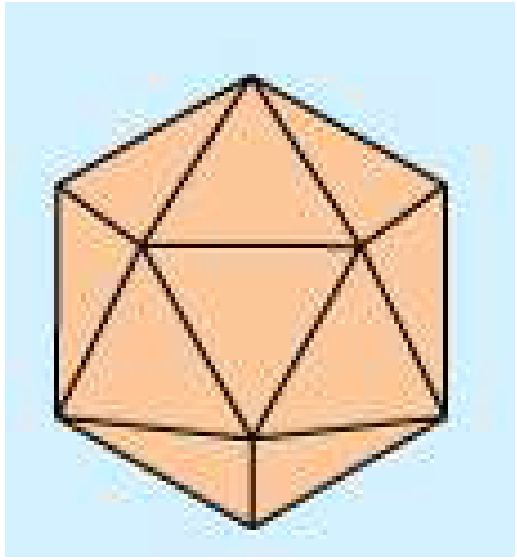




TYPES OF SYMMETRY OF VIRUS PARTICLES:

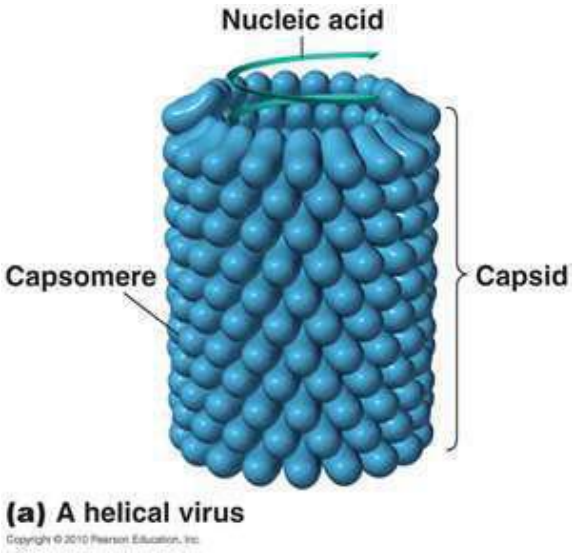
•1. Icosahedral symmetry

- Composed of 12 vertices, has 20 faces (each an equilateral triangle) with
- the approximate outline of a sphere.
- e.g. Herpesviruses , Adenoviruses



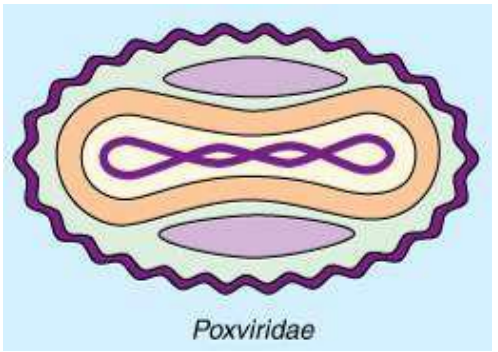
TYPES OF SYMMETRY OF VIRUS PARTICLES:

- **2. Helical symmetry**
- In which the capsomeres are arranged in a hollow coil that appears rodshaped.
- The helix can be either rigid or flexible.
- e.g. Influenza viruses



3. Complex structures

- e.g. Poxviruses



REACTION TO PHYSICAL AND CHEMICAL AGENTS:

- **1. Heat and cold**
- Viral infectivity is generally destroyed by heating at 50-60 C0 for 30 mint., hours at 20 C0, days at 4 C0. Viruses can be preserved at -90 C0 or -196 C0 (liquid nitrogens).
- **2. PH**
- Viruses can be preserved at physiological PH (7.3).
- **3. Ether susceptibility :**
- Ether susceptibility can be used to distinguish viruses that possess an envelope from those that do not.
- **4. Detergents:**
- Nonionic detergents solubilize lipid constituents of viral membranes. The viral proteins in the envelope are released. Anionic detergents also solubilize viral envelopes; in addition, they disrupt capsids into separated polypeptides.

REACTION TO PHYSICAL AND CHEMICAL AGENTS:

- **5. Salts**
- Many viruses can be stabilized by salt in concentrations of 1 mol/L.

  e.g. $MgCl_2$, $MgSO_4$, Na_2SO_4.
- **6. Radiation**
- Ultraviolet, X-ray, and high-energy particles inactivate viruses.
- **7. Formaldehyde**
- Destroys viral infectivity by reacting with nucleic acid.
- **8. Antibiotics**
- Antibacterial antibiotics have no effect on viruses.

CLASSIFICATION OF VIRUSES

- 1. Virion morphology, including size, shape, type of symmetry, presence or absence of envelope.
- 2. Virus genome properties, including type of nucleic acid (DNA or RNA), size of genome, strandedness (single or double), whether linear or circular, positive or negative sense (polarity), segments (number, size).
- 3. Physicochemical properties of the virion, including PH stability, thermal stability, and susceptibility to physical and chemical agents, especially ether and detergents.
- 4. Virus protein properties, including number, size and functional activities of structural and non-structural proteins, amino acid sequences, and special functional activities

CLASSIFICATION OF VIRUSES

- 5. Genome organization and replication, including gene order, strategy of replication (patterns of transcription, translation), and cellular sites (accumulation of proteins, virion assembly, virion release).
- 6. Antigenic properties
- 7. Biological properties, including natural host range, mode of transmission, vector relationships, pathogenicity, and pathology

Families of Animal Viruses That Contain Members Able to Infect Humans Summary

Nucleic Acid Core	Capsid Symmetry	Virion: Enveloped or Naked	Ether Sensitivity	Number of Capsomeres	Virus Particle Size (nm)[a]	Size of Nucleic Acid in Virion (kb/kbp)	Physical Type of Nucleic Acid[b]	Virus Family
DNA	Icosahedral	Naked	Resistant	32	18–26	5.6	ss	Parvoviridae
				72	45	5	ds circular	Polyomaviridae
				72	55	8	ds circular	Papillomaviridae
				252	70–90	26–45	ds	Adenoviridae
		Enveloped	Sensitive	180	40–48	3.2	ds circular[c]	Hepadnaviridae
				162	150–200	125–240	ds	Herpesviridae
	Complex	Complex coats	Resistant[d]		230 × 400	130–375	ds	Poxviridae
RNA	Icosahedral	Naked	Resistant	32	28–30	7.2–8.4	ss	Picornaviridae
					28–30	6.4–7.4	ss	Astroviridae
				32	27–40	7.4–8.3	ss	Caliciviridae
					27–34	7.2	ss	Hepeviridae
					60–80	16–27	ds segmented	Reoviridae
		Enveloped	Sensitive	42	50–70	9.7–11.8	ss	Togaviridae
	Unknown or complex	Enveloped	Sensitive		40–60	9.5–12.5	ss	Flaviviridae
					50–300	10–14	ss segmented	Arenaviridae
					120–160	27–32	ss	Coronaviridae
					80–110	7–11[e]	ss diploid	Retroviridae
	Helical	Enveloped	Sensitive		80–120	10–13.6	ss segmented	Orthomyxoviridae
					80–120	11–21	ss segmented	Bunyaviridae
					80–125	8.5–10.5	ss	Bornaviridae
					75 × 180	13–16	ss	Rhabdoviridae
					150–300	16–20	ss	Paramyxoviridae

SUMMARY

- 1-Viruses range in size from(20-300nm).
- 2-The genome of viruses either DNA or RNA single –stranded or double –stranded, linear or circular.
- 3-All viruses have protein coat called capsid .The capsid is composed of repeating subunits called capsomeres .Some viruses are naked while others possess envelope.
- 4-The capsomeres give the virus a symmetric appearance.Some have spherical (Icosahedral) symmetry, whereas others have helical symmetry.
- 5- Viral surface proteins mediate attachment to host cell receptors.
- 6-The viral envelope is acquired as the virus exits from the cell. Enveloped viruses are more sensitive to heat , detergent ,&lipid solvents.
- 7-The surface proteins are the targets of antibody .
- 8-The classification of viruses based on virion morphology, & virus genome properties.

Thank You

Lec 2 & 3

Atypical virus like agents

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ATYPICAL VIRUS LIKE AGENTS

- **1.Defective viruses** ar e composed of viral nucleic acid and proteins. Viruses usually have a mutation or a deletion of part of their genetic material.
- **2. Pseudovirions**: contain host cell DNA instead of viral DNA within the capsid.
- **3. Viroids:** Consist solely of a single molecule of circular RNA without a protein coat or envelope.
- **4. Prions:** are infectious particles that are composed solely of protein. No detectable nucleic acid.

ATYPICAL VIRUS LIKE AGENTS

- They are implicated as the cause of certain "slow" diseases called transmissible spongiform encephalopathies :
- Creutzfeldt-Jakob disease in humans.
- Scrapie in sheep
- The term spongiform refers to the sponge-like appearance of the brain seen in these diseases. The holes of the sponge are vacuoles resulting from dead neurons.
- **Prion proteins** are encoded by a cellular gene. When these proteins are in normal configuration, they are non pathogenic, butwhen their configuration changes, they aggregate into filaments, which disrupts neuronal function and results in the symptoms of disease.

ATYPICAL VIRUS LIKE AGENTS

- Prions are highly resistant to inactivation by UV light, heat, they are remarkably resistant to formaldehyde and nucleases. They are inactivated by hypochlorite, NaOH, and autoclaving.
- Prions transmitted by human growth hormone and neurosurgical instruments. Because they are normal human proteins, they do not elicit an inflammatory response or an antibody response in humans.
- **Bovine spongiform encephalopathy**
- {Mad cow} disease. Cattle eating brains obtained from sheep infected with scrapie prions.

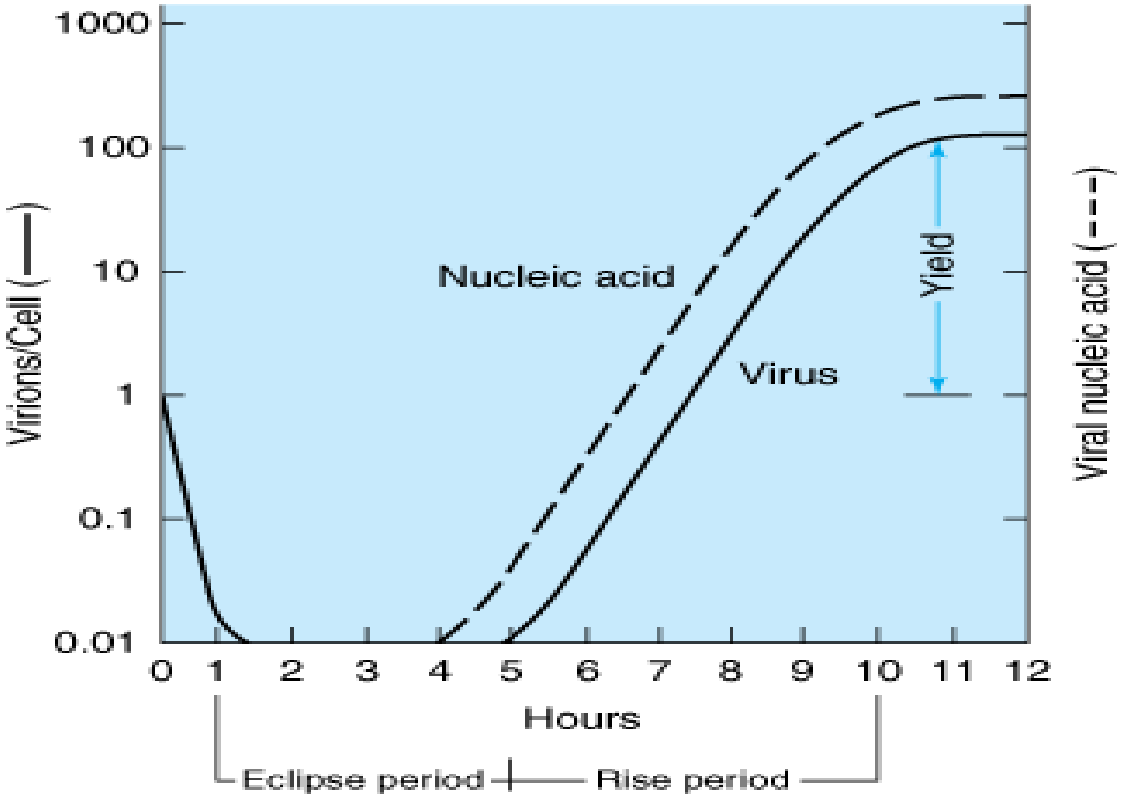
Thank You

Replication of viruses*:*is the formation of viruses during the infection process in the target host cells. Viruses must first get into the cell before viral replication can occur. Most DNA viruses assemble in the nucleus while most RNA viruses develop solely in cytoplasm .

Replication of viruses

The viral replication cycle is described below in two different ways:

1. Growth curve, which shows the amount of virus produced at different times after infection.



2. Stepwise description of the specific events within the cell during virus growth:

1. **Attachment** or interaction of a virion with a specific receptor siteon the surface of a cell.

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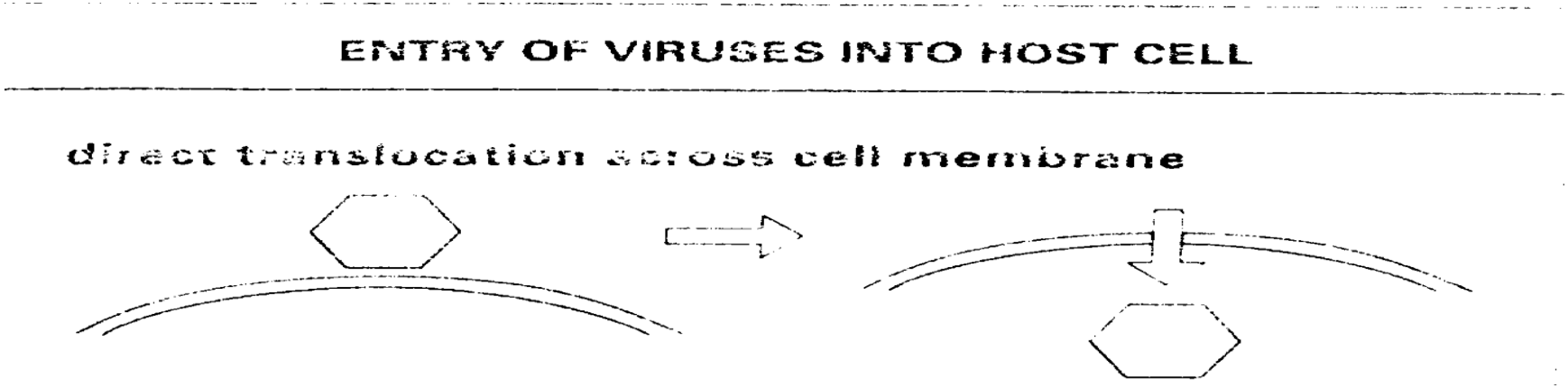
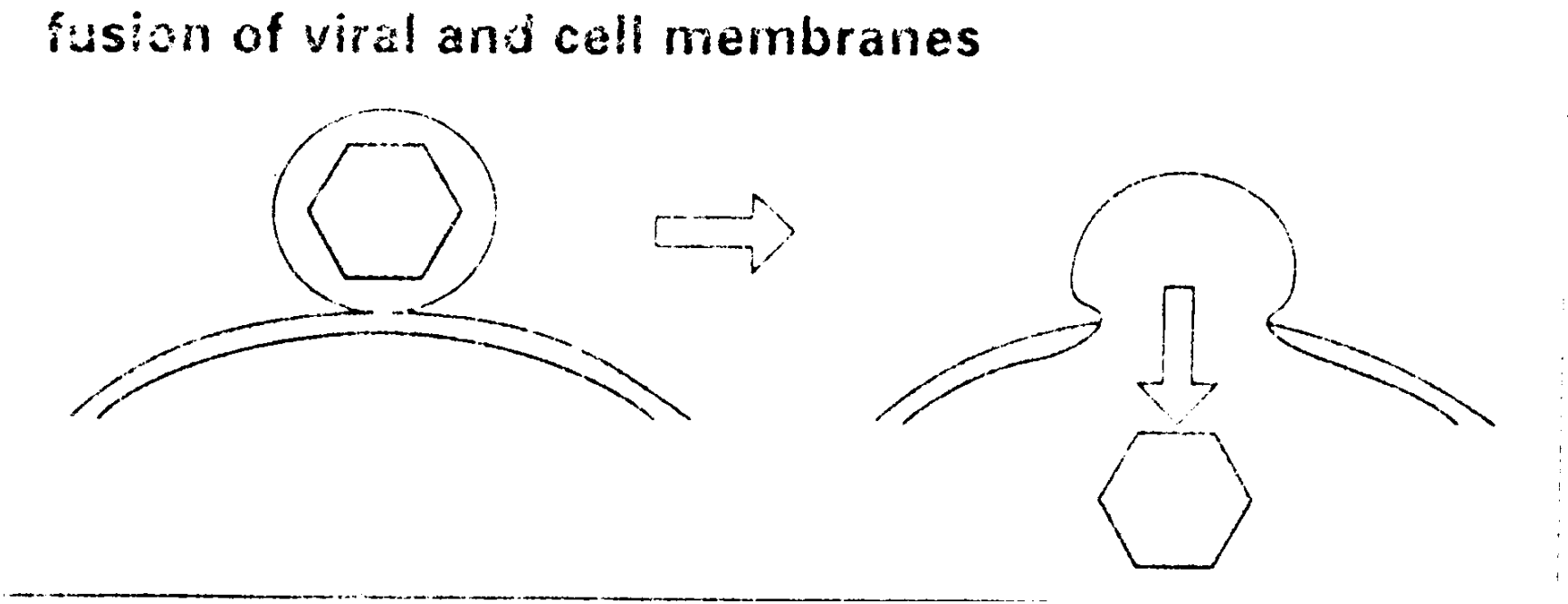
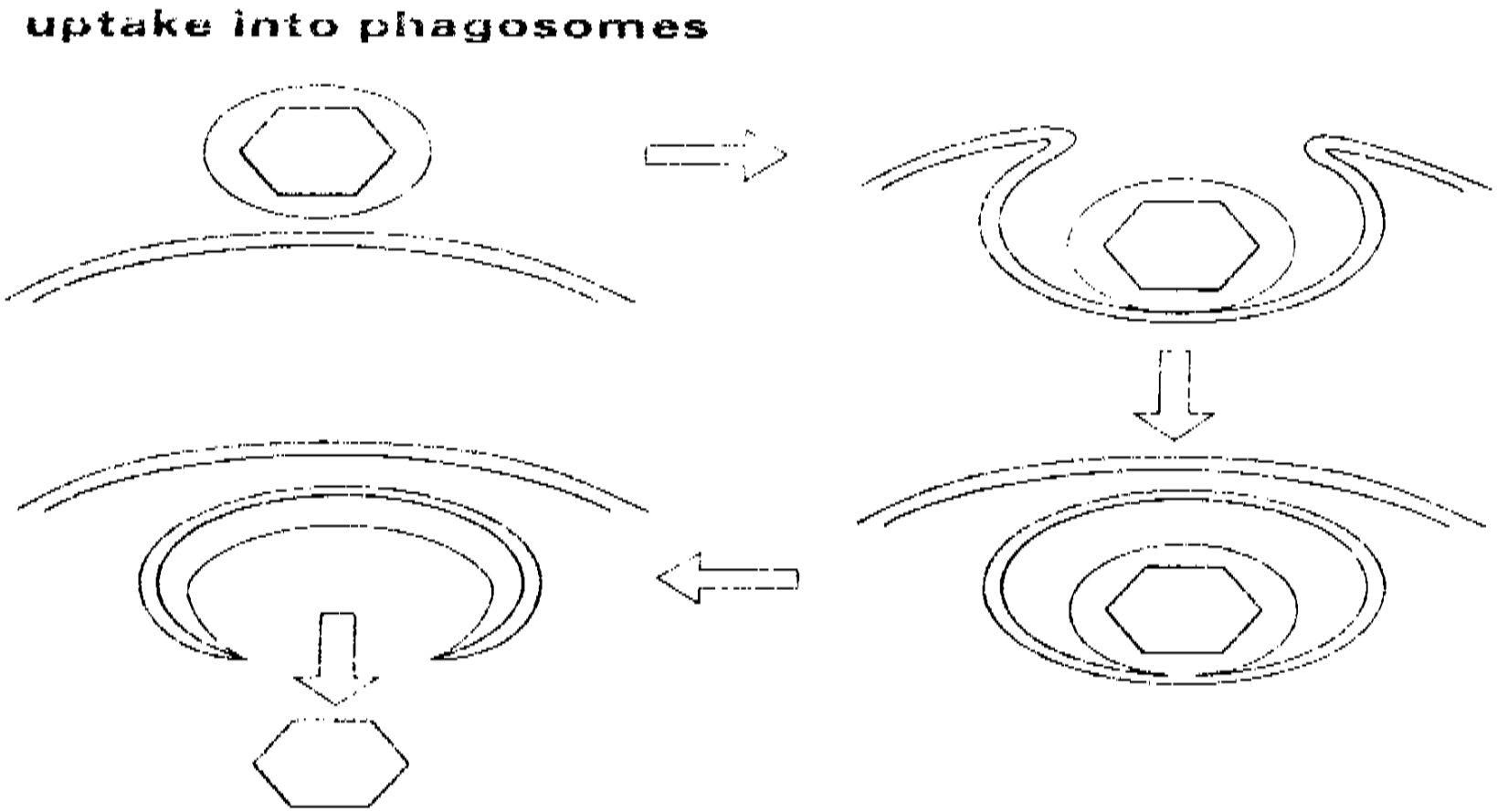
2.pentration ::
Direct translocation across cell membrane



B. Fusion of viral envelope and cell membrane



C. Engulfed in a pinocytotic vesicle



3. Uncoating

With some viruses ,the genome iscompletly released from the capsid after penetration . This is khnown uncoating .

4-Gene expression and genome replication:

1-Early viral mRNA synthesis (transcription)

A. DNA viruses:

Replicate in the nucleus and use the host cell **DNA-dependent RNApolymerase** to synthesize their mRNA.

The poxviruses are the exception because they replicate in the cytoplasm.

They carry their own polymerase within the virus particle.

The genome of all DNA viruses consists of double-stranded (ds) DNA, except for the **parvoviruses**, which have a single-stranded (ss) DNA genome.

B. RNA viruses:

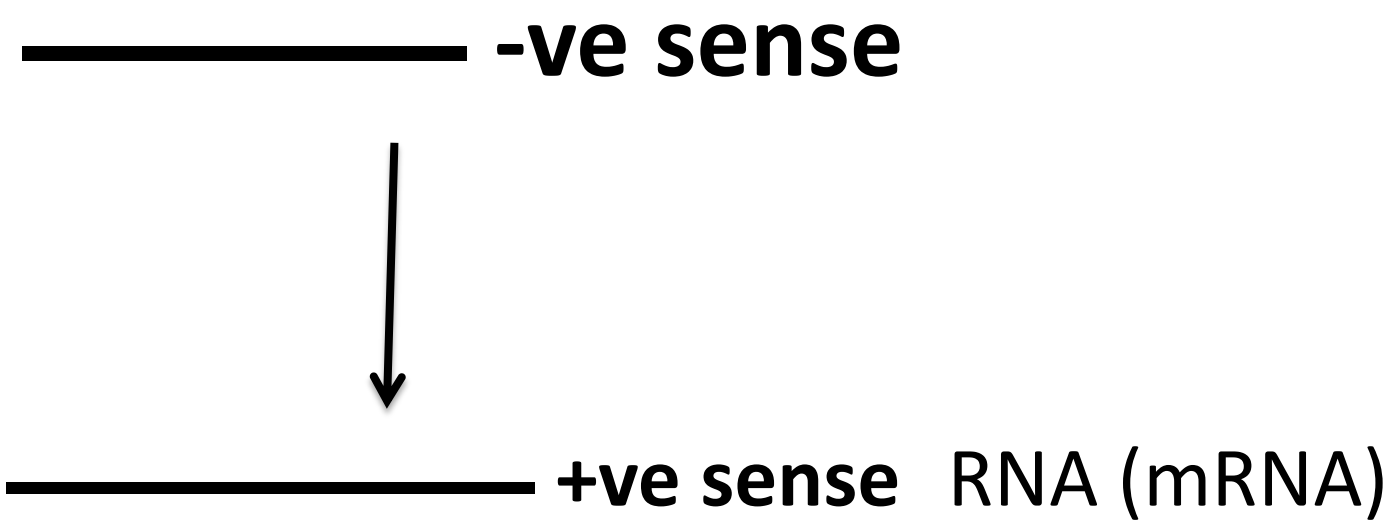
Fall into four groups with quite different strategies for synthesizing mRNA.

1. Single- stranded RNA of positive polarity.

These viruses use their RNA genome directly as mRNA (e.g. Poliovirus).

2. Single- stranded RNA of negative polarity.

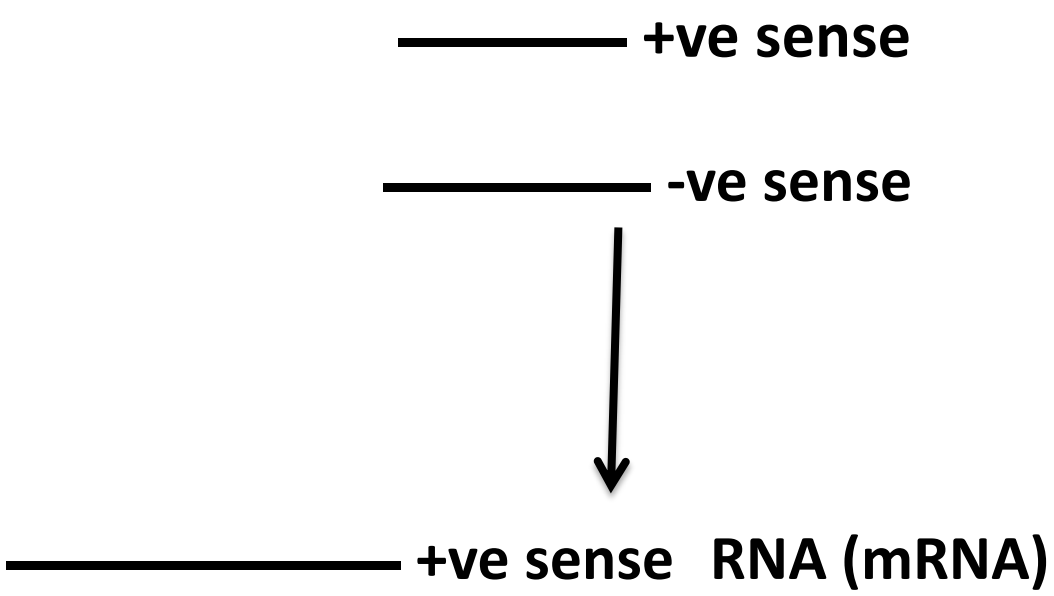
An mRNA must be transcribed by using the negative strand as a template. The virus carries its own **RNA- dependent RNA polymerase** (e.g. Influenza virus)



Single- stranded RNA of negative polarity.

3.Double-stranded-RNA :

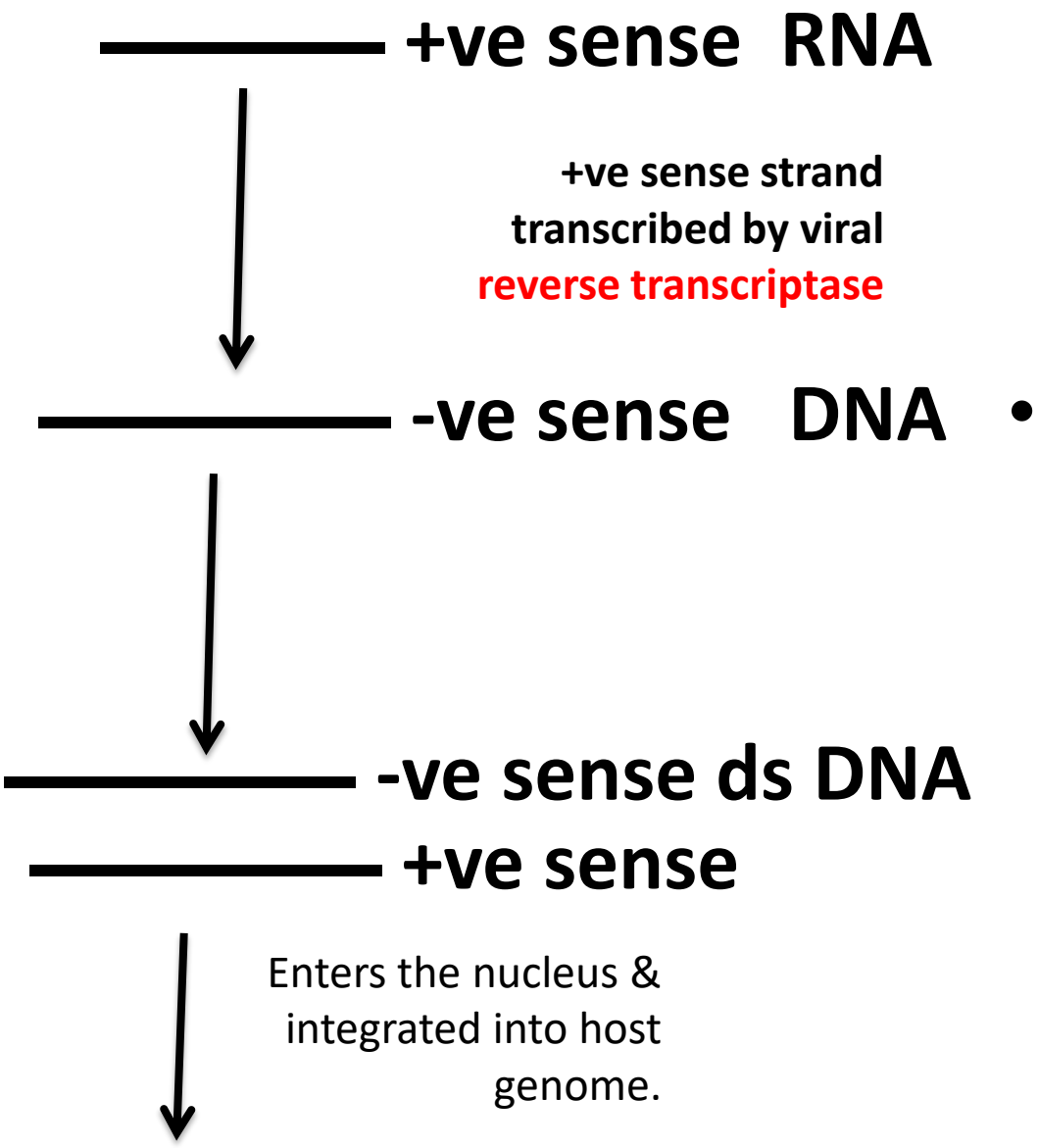
The virus carries its own **polymerase** for transcribing into mRNA (e.g. Reovirus).



4. Single- stranded RNA of positive polarity

The RNA transcribed into double- stranded DNA by the **RNA dependent DNA polymerase (reverse transcriptase), carried by** the virus. This DNA copy is then transcribed into viral mRNAby the regular host cell RNA polymerase. (e.g. Retroviruses).

Single- stranded RNA of positive polarity



Most RNA viruses undergo their entire replication cycle in cytoplasm. The two principle exceptions are retroviruses and influenza viruses, both of which have an important replication step-in-the-nucleus.

2-Early viral proteins synthesis (Translation):

Once the viral mRNA of either DNA or RNA viruses is synthesized, it is translated by host cell ribosomes into viral proteins. Some of which are early proteins, i.e. enzymes required for replication of viral genome, and others of which are late proteins, i.e. structural proteins of the progeny viruses.

Early proteins: occurring before the replication of the genome.

Late proteins: occurring after genome replication.

The most important of the early proteins for many RNA viruses is the polymerase that will synthesize many copies of viral genetic material for the progeny virus particles. Most viruses make a virus encoded- polymerase that replicates the genome.

Replication of viral genome(complementarity):Replication of the viral genome is governed by the principle ofcomplementarity, which requires that a strand with acomplementary base sequence be synthesized; this strand thenserves as the template for the synthesis of actual viral genome.

Replication of viral genome (complementarity)

Prototype Virus	Parental Genome[1]	Intermediate Form	Progeny Genome
Poliovirus	+ ssRNA	– ssRNA	+ ssRNA
Influenza virus, measles virus, rabies virus	– ssRNA	+ ssRNA	– ssRNA
Rotavirus	dsRNA	+ ssRNA	dsRNA
Retrovirus	+ ssRNA	dsDNA	+ ssRNA
Parvovirus B19	ssDNA	dsDNA	ssDNA
Hepatitis B virus	dsDNA	+ ssRNA	dsDNA
Papovavirus, adenovirus, herpesvirus, poxvirus	dsDNA	dsDNA	dsDNA

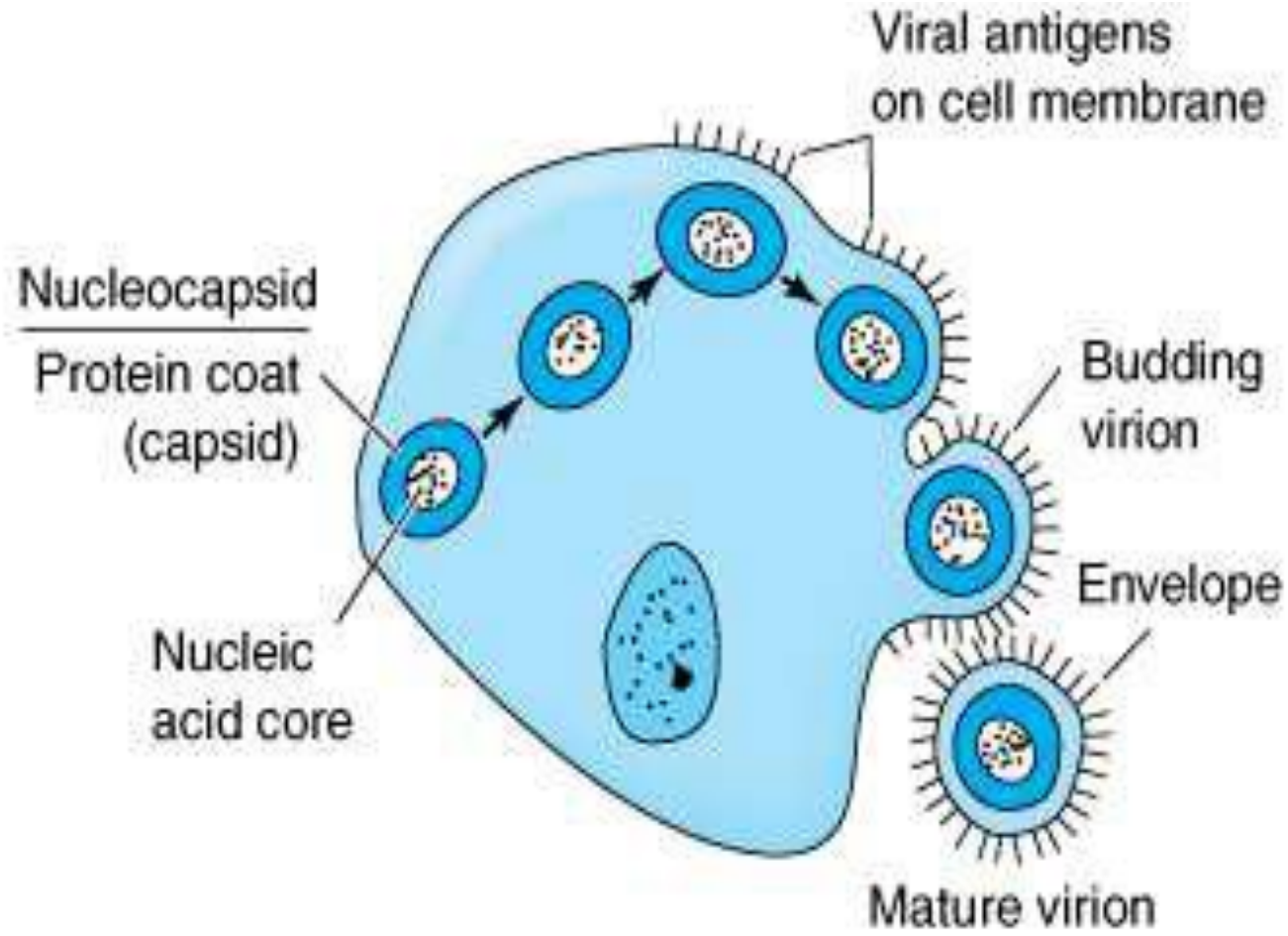
Late viral mRNA synthesis (transcription)
Late viral proteins synthesis (translation):
Capsid proteins

5-Assembly and release:

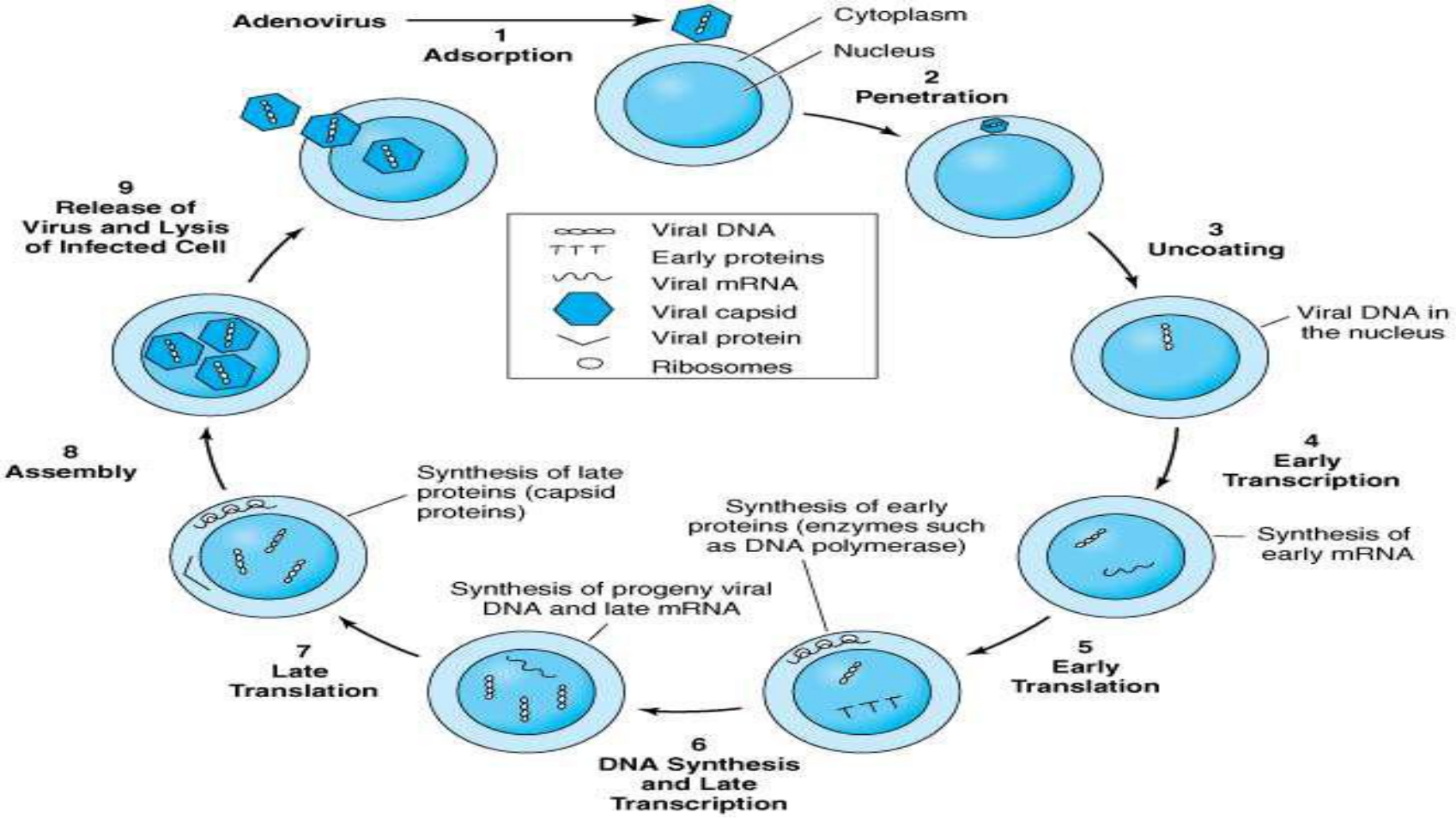
The progeny particles are assembled by packaging the viral nucleicacid within the capsid proteins.

Virus particles are released from the cell by either of two processes:

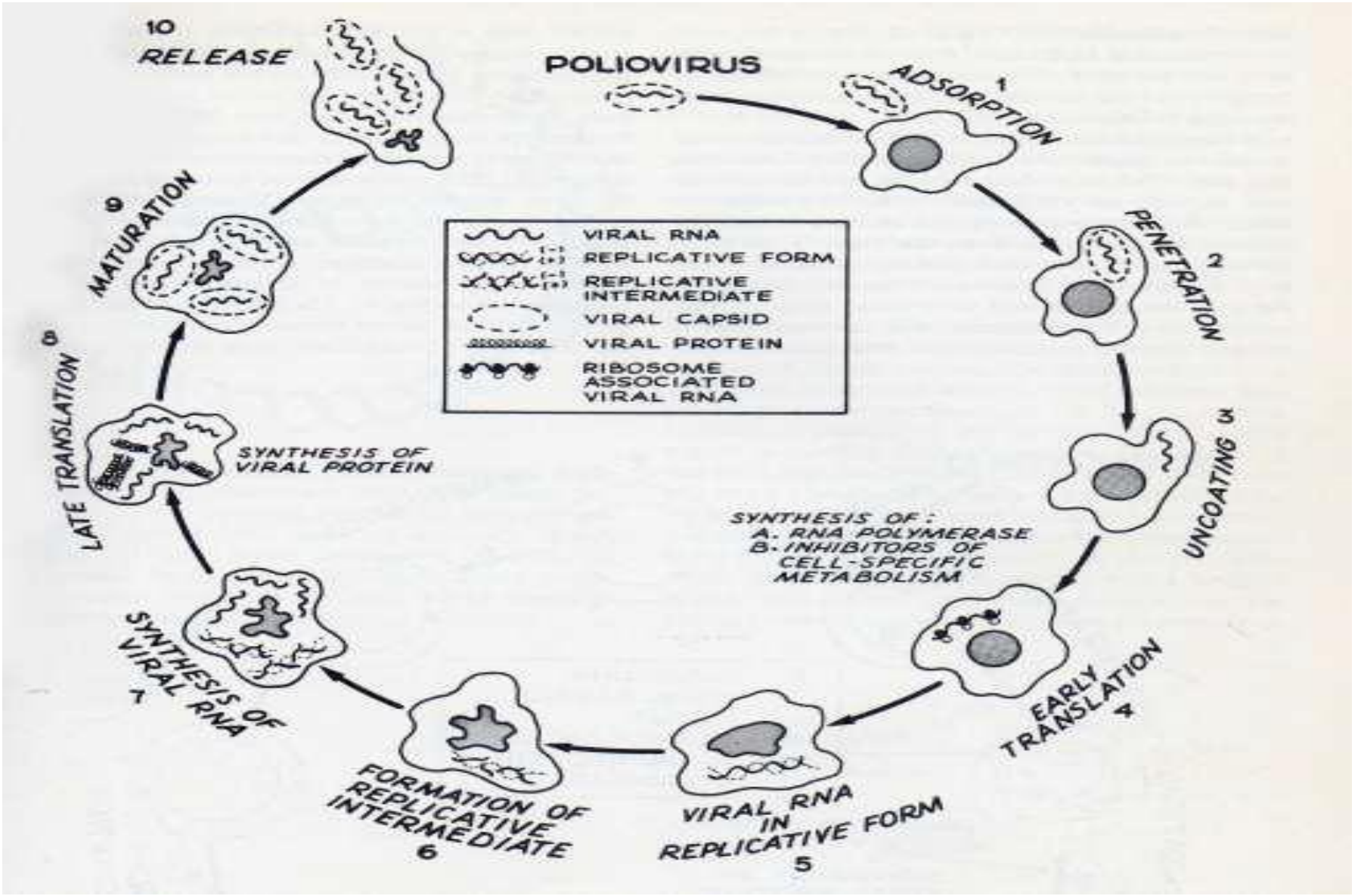
1. **Rupture of the cell membrane and release of the mature** particles (unenveloped viruses).
2. **Budding through the outer cell membrane (enveloped** viruses).


Viral antigens
on cell membrane
Nucleocapsid
Protein coat
(capsid)
Budding
virion
Envelope
Nucleic
acid core
Mature virion

The Growth cycle of DNA virus



The growth cycle of RNA virus



Pathogenesis of viral infections

Pathogenesis is the process by which an infection leads to disease.

Cellular Pathogenesis

Direct cell damage and death from viral infection may result from (1). diversion of the cell's energy, (2) competition of viral mRNA for cellular ribosomes, (3) inhibition of the interferon defense mechanisms. Indirect cell damage can result from integration of the viral genome, induction of mutations in the host genome, inflammation, and the host immune response.

Mode of transmission:

Horizontal: person- to -person (infl.v.,herpes v.,HIV) -

-Vertical: from mother to offspring (rubella,HIV,CMV,Hepatitis B &C,parvo B19)

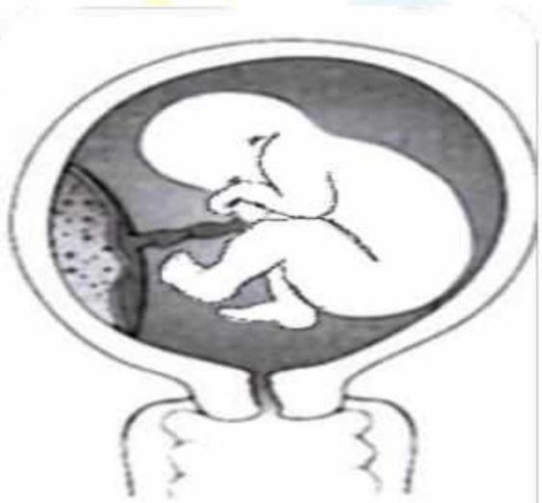
Zoonotic: from animal to man (rabies v.) -

- No transmission: due to reactivation of a latent, non-replicating virus can occurs within the individual infection (HSV1, HSV2 &CMV)

Mode of tranmission

Vertical transmission

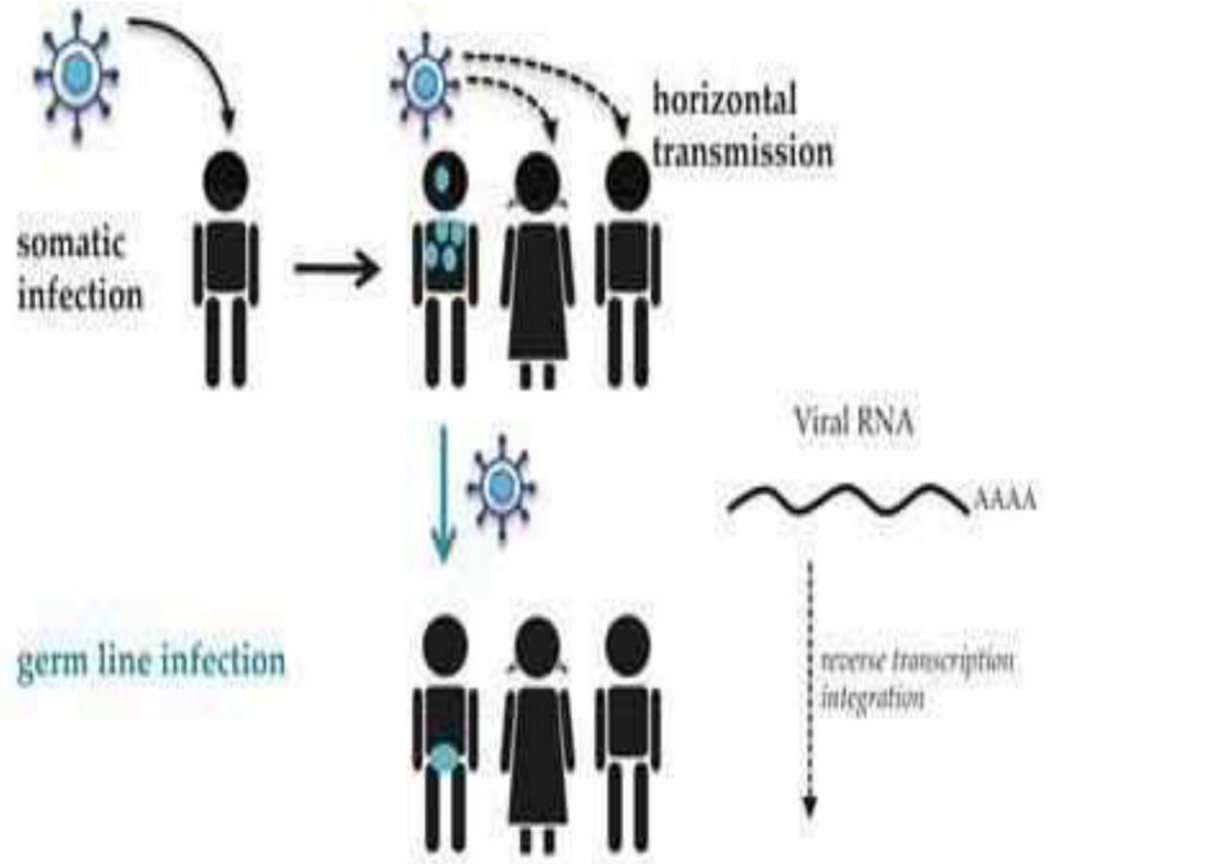
Transplacental



- **To R C H**
- **HIV**
- **HBV**

15

•32


horizontal
transmission
somatic
infection
Viral RNA
AAAA
germ line infection
reverse transcription
integration

Clinical illnesses

- Incubation Period: usually asymptomatic
- Prodromal period: associated with non-specific symptoms
- Specific illness period: characterized by sign &symptoms of the disease.
- Recovery period: illness wanes& patient regains good health.

Steps in viral pathogenesis:

1-Viral entry
2- viral replication
3-Viral spread
4-Cellular injury
5-Cell &tissue tropisms
6-Host immune response
7-Viral infection
8-Viral shedding

1.a-Viral entry: through the following systems:

-Respiratory Tract

-Gastrointestinal Tract most common routes of viral entry

-Skin

-Urogenital Tract

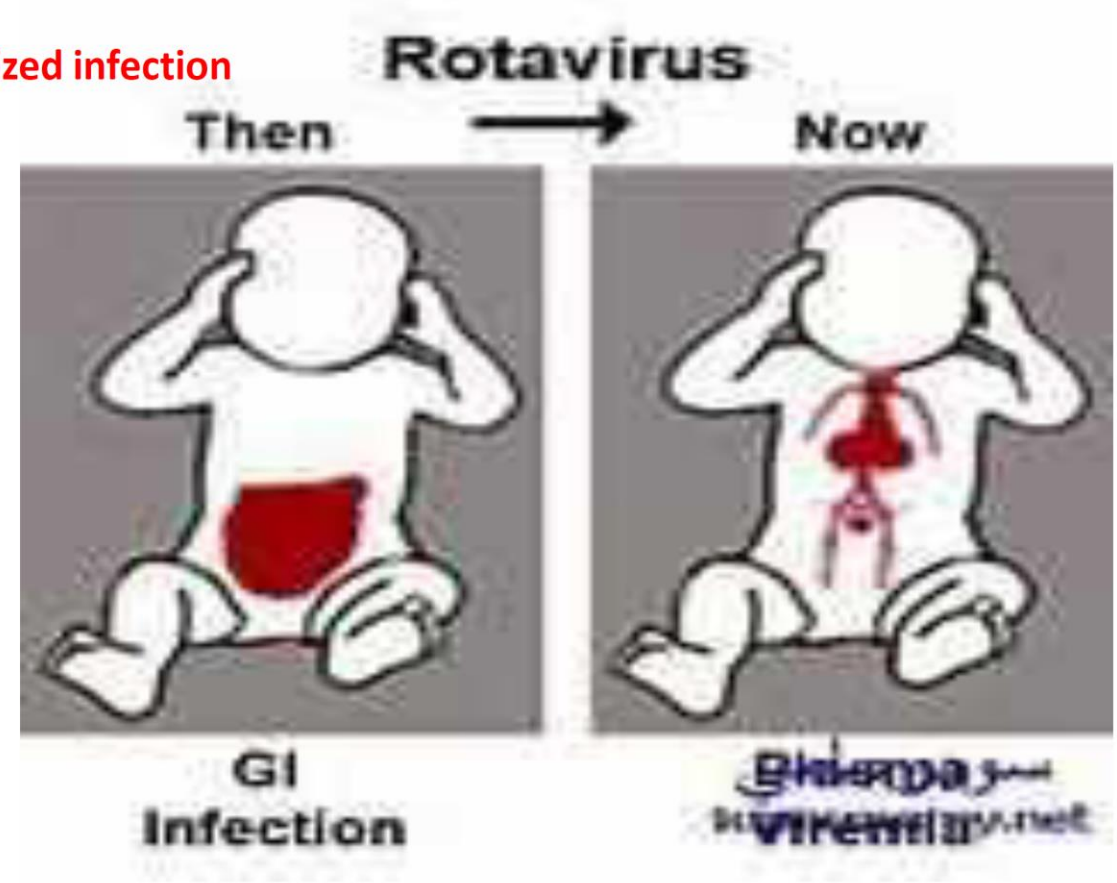
Conjunctiva

-blood

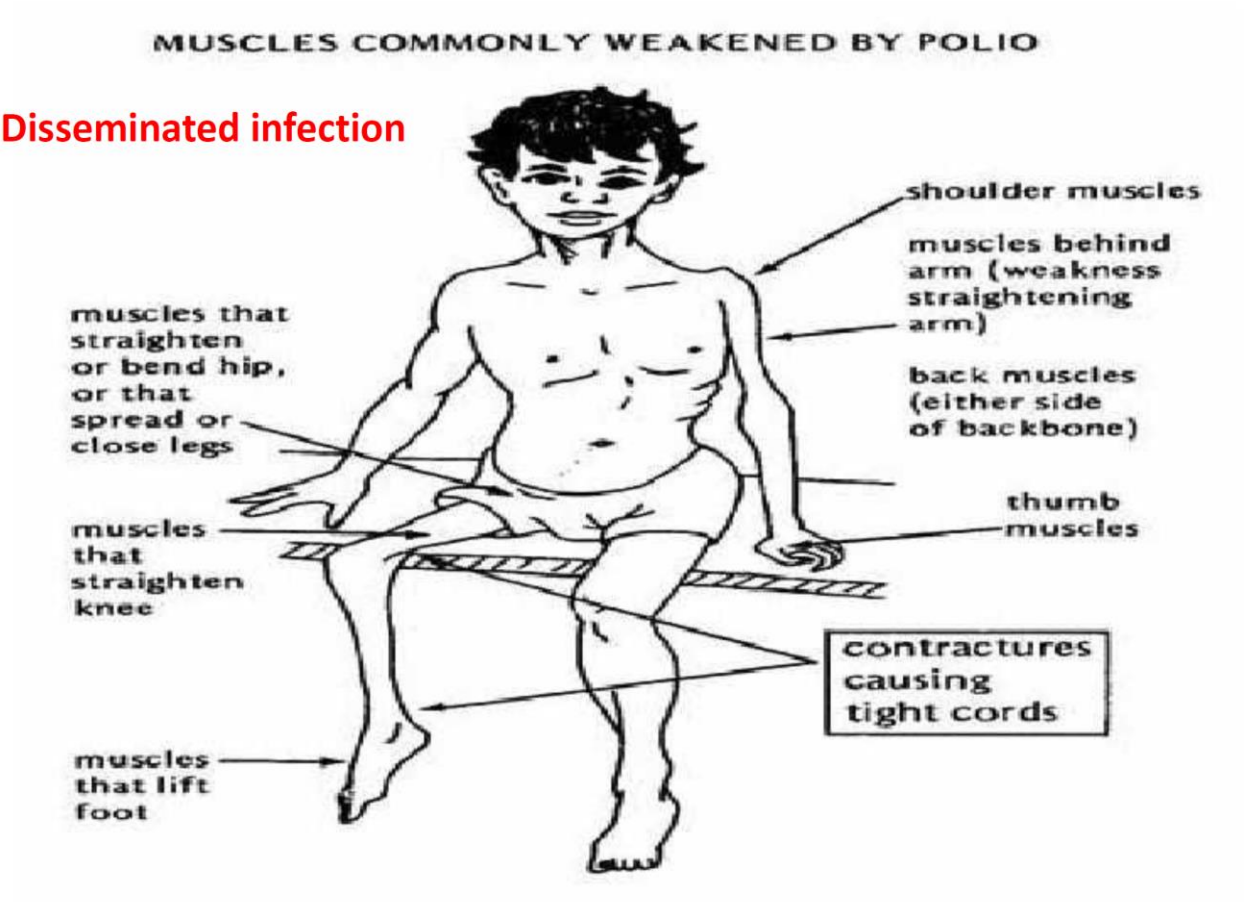
2-viral replication:

- *Local* Successful implantation may be followed by local replication and local spread of virus.
- Virus that replicates within the initially infected cell may spread to adjacent cells extracellularly or intracellularly.
- ***Localized infection**: virus replicate at primary site locally as: influenza v. & rota v.) these viruses spread locally over the epithelial surfaces, and there is no necessity for further systemic spread, no invasion to under lying tissues nor spread to distant site.*
- *• Disseminated infection: viruses that produce systemic manifestation distant from the site of entry as: polio v.& Measles v*

Localized infection



Disseminated infection



3-Viral spread Mechanisms:

Most common mechanisms for viral spread are through *blood and Lymphatic*

Neuronal spread (as: rabies v. and herpes v.) to ganglia to initiate latent infection.

Some virions are *free in plasma ….entero v.& toga v.*

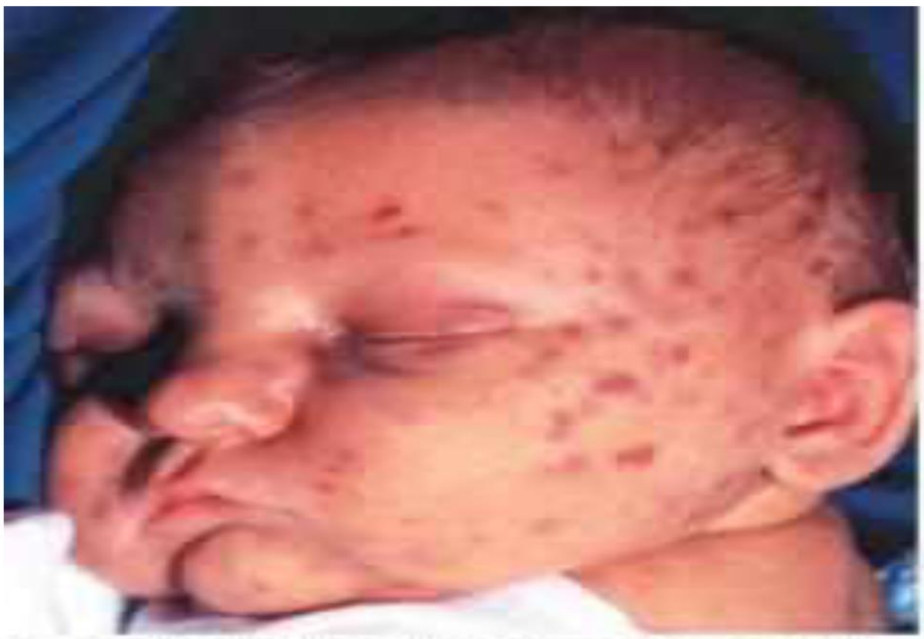
Or *associated with Particular types of cell (measles v.) and some viruses even multiply within these cells.*

4-Cell injury &clinicall illness:

-Cytopathic effect (CEP):

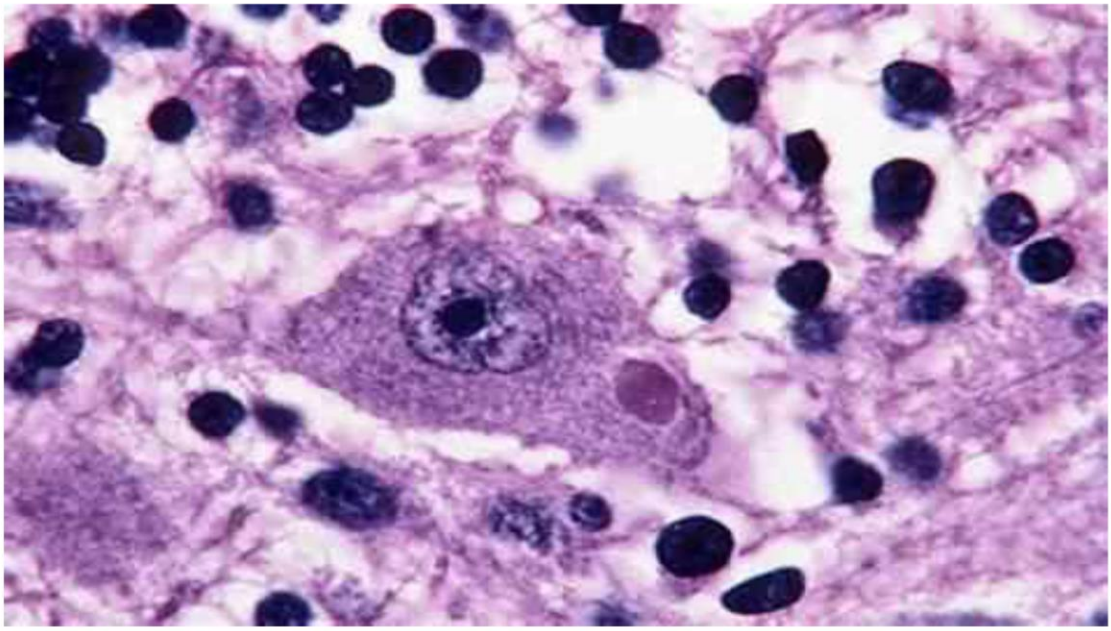
- a-Changes in the cell appearance; Rounding, darking and ballooning of cells.
- b-Multinucleated Giant cells: Due to fusion of virus infected cell as a result of cell membrane changes which caused by the insertion of viral proteins into the cell membrane(herpes v.& paramyxo v.)
- c- Inclusion bodies; discrete areas on cell cytoplasm or nucleus, containing viral proteins or viral particles e.g. Negri bodies (intracyoplasmic) by rabies v. and Owl's eye inclusion - Malignant transformation: uncontrolled cell growth &prolonged survival (oncogenic v.) (intranuclear) by CMV.

CMV



Owl eye in CMV

Negri bodies



- **5-Tissue Tropism**
- Viral affinity for specific body tissues (tropism) is determined by (1) cell receptors for virus, (2) cell transcription factors that recognize viral promoters and enhancer sequences, (3) digestive enzymes and bile in the gastrointestinal tract that may inactivate some viruses.

Cell Death: due to inhibition of macromolecular synthesis either by inhibition of host cell protein synthesis or inhibition of DNA and RNA synthesis.

6-Immunopathogenesis

*Direct effect: cell killing e.g. infection with *polio v.(kill motor neuron cells leading to paralysis of the muscles)*

**Ebola v. causing hemorrhage due to the damage in vascular endothelial cells which caused by enveloped of the virus

Indirect effect: (immunological attack)

Cytokines produced by rota –infected enterocytes that stimulate the enteric neurons resulting in excess fluid and electrolytes secretion into the bowel lumen…..causing diarrhea

7-Persistent viral infection:

Some time, the virus persists for long periods either intact or in the form of a subviral component (genome).

Mechanisms of Persistence:

1- Integration of a DNA provirus into host cell DNA e.g.: retroviruses

2-Virus –antibody complexes formation: which remain infectious

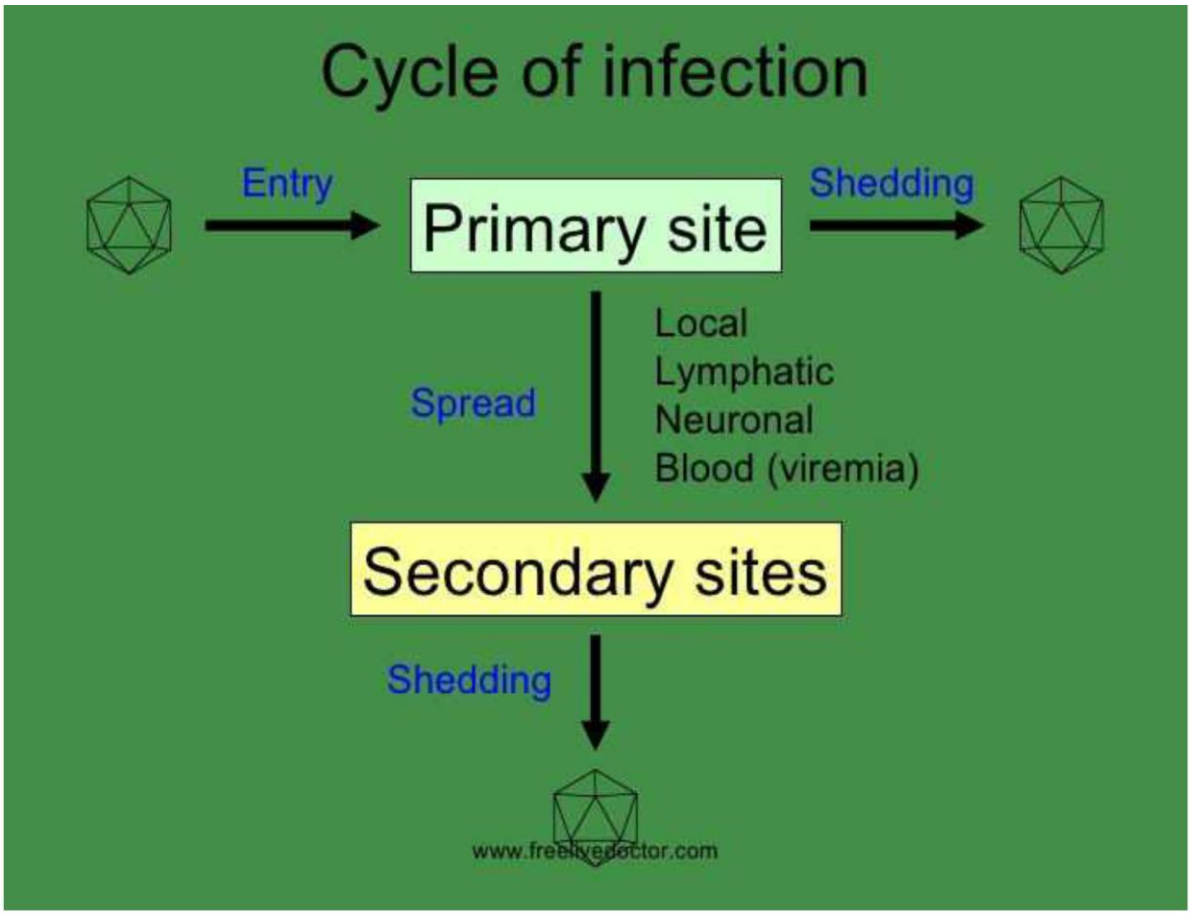
3- Immunosupression (e.g. AIDS)

8-Viral shedding:

Last stage that Maintain viral infection in population or in the environment.

Shedding occurs from body surface which involved in viral entry. Patient remains infectious to contacts.

Dead-end infection in human occurs only in Rabies, Poliomyelitis and SSPE in these cases there are no viral shedding.


Cycle of infection
Entry
Primary site
Shedding
Spread
Local
Lymphatic
Neuronal
Blood (viremia)
Secondary sites
Shedding
www.freeliveedoctor.com

HUMAN HERPESVİRUSES

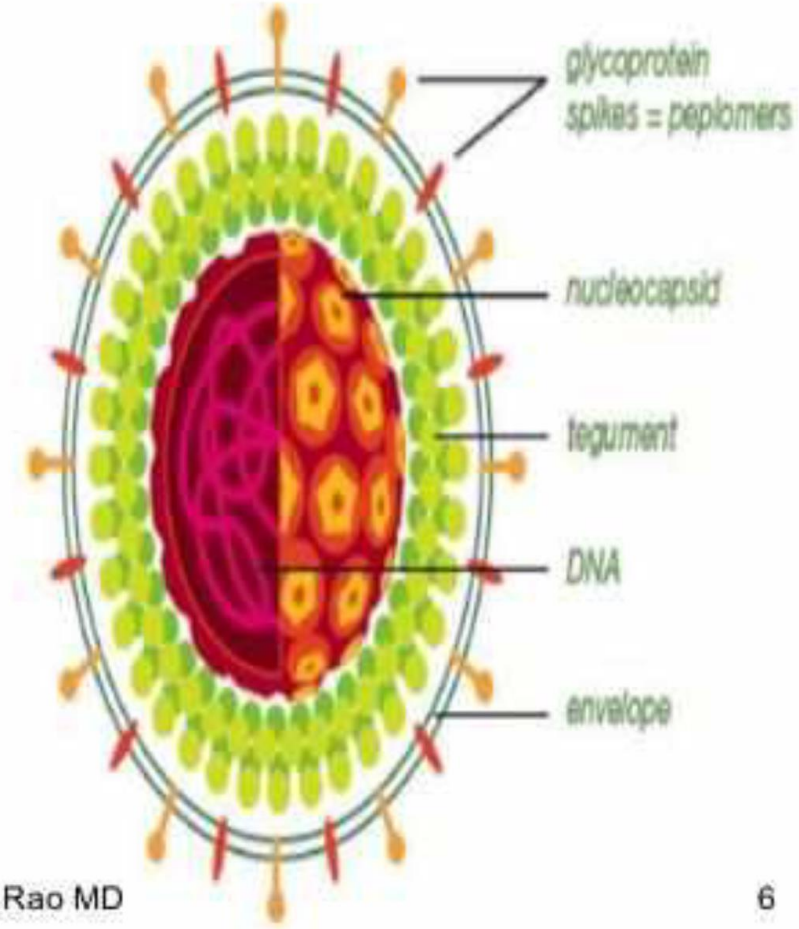
م.م إيهاب مجيد عباس

HERPESVİRUSES

- Herpesviruses have large, enveloped icosadeltahedral capsids containing double-stranded DNA genomes.
- Herpesviruses encode many proteins that manipulate the host cell and immune response.
- Herpesviruses encode enzymes (DNA polymerase) that promote viral DNA replication and that are good targets for antiviral drugs.
- DNA replication and capsid assembly occurs in the nucleus.
- Virus is released by exocytosis, cell lysis, and through cell-cell bridges.

Properties of Herpes Viruses.

- Spherical in Shape
- Icosahedral 150 to 200 nm in size
- Genome – Double stranded DNA Linear
- Envelope contains Glycoprotein's



HUMAN HERPESVİRUSES

- There are 9 herpes viruses which are known to infect humans

Human herpesvirus 1 Herpes simplex type 1 HSV-1

Human herpesvirus 2 Herpes simplex type 2 HSV-2

Human herpesvirus 3 Varicella-zoster virus VZV

Human herpesvirus 4 Epstein-Barr virus EBV

Human herpesvirus 5 Cytomegalovirus CMV

Human herpesvirus 6Herpes lymphotropic virus HHV-6

Human herpesvirus 7 Human herpesvirus 7 HHV-7

Human herpesvirus 8 Kaposi's sarcoma related virus HHV-8

Herpes B Virus/CercoPethicine Herpesvirus-1 (CHV-1)

HUMAN HERPESVİRUSES

They have common:

- Virion morphology
- Basic mode of replication
- Usually cause benign disease especially in children
- In immunosuppressed people they cause significant mortality

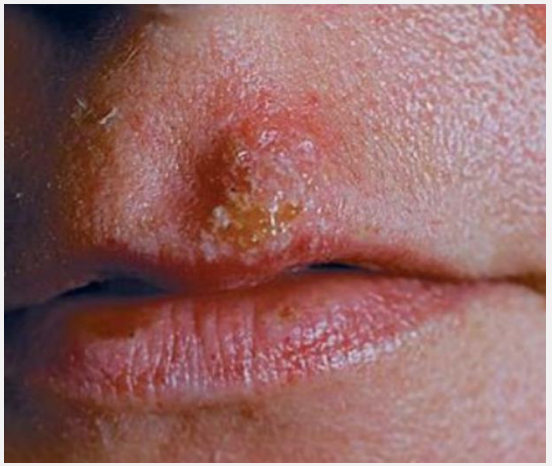
HERPES SIMPLEX VIRUSES

- Herpes simplex virus type 1 (**HSV-1**) and type 2 (**HSV-2**) are distinguished by two main criteria
- - Antigenicity
- - location of lesions.
- **HSV-1:above the waist, primarily in adults**
- Acute gingivostomatitis,
- Recurrent herpes labialis (cold sores),
- Keratoconjunctivitis (keratitis),
- Encephalitis
- **HSV-2: below the waist**
- herpes genitalis(genital herpes),
- **Neonatal encephalitis and other forms of neonatal herpes**
- Aseptic meningitis
- **Humans are the natural hosts of both.**

CLINICAL FINDINGS: HSV-I

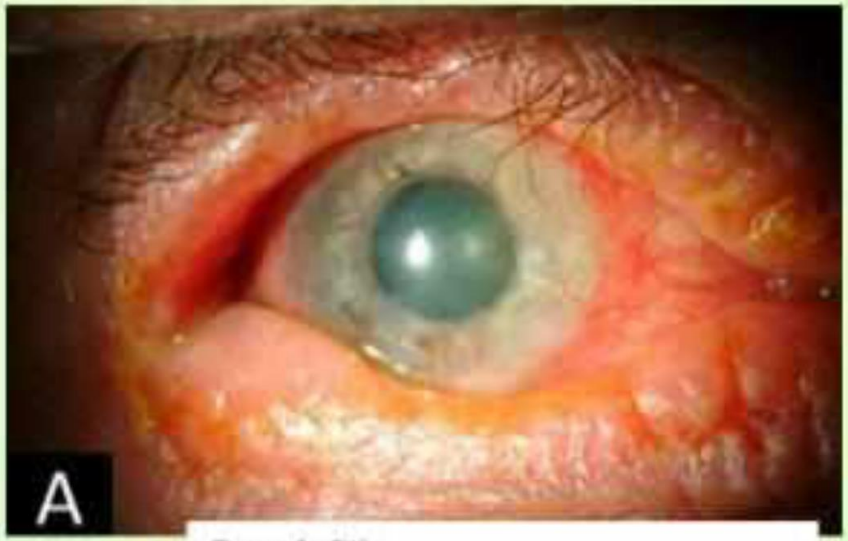
- causes several forms of primary and recurrent disease.
- **Gingivostomatitis**
- Occurs primarily in children and is characterized by fever, irritability, and vesicular lesions in the mouth.
- The primary disease is more severe and lasts longer than recurrences.
- The lesions heal spontaneously in 2 to 3 weeks.
- - Many children have asymptomatic primary infections
- **Herpes labialis**
- fever blisters or cold sores is the milder, recurrent form
- characterized by crops of vesicles, usually at

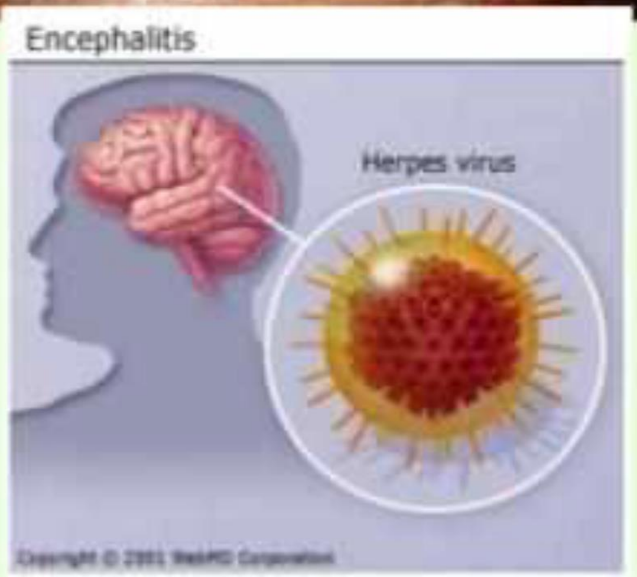
  the mucocutaneous junction of the lips or nose
- - Recurrences frequently reappear at the same site.



Clinical Findings: HSV-1

- **Keratoconjunctivitis**
  - characterized by corneal ulcers and lesions of the conjunctival epithelium.
  - Recurrences can lead to scarring and blindness
- **Encephalitis**
  - necrotic lesion in one temporal lobe.
  - Fever, headache, vomiting, seizures, and altered mental status





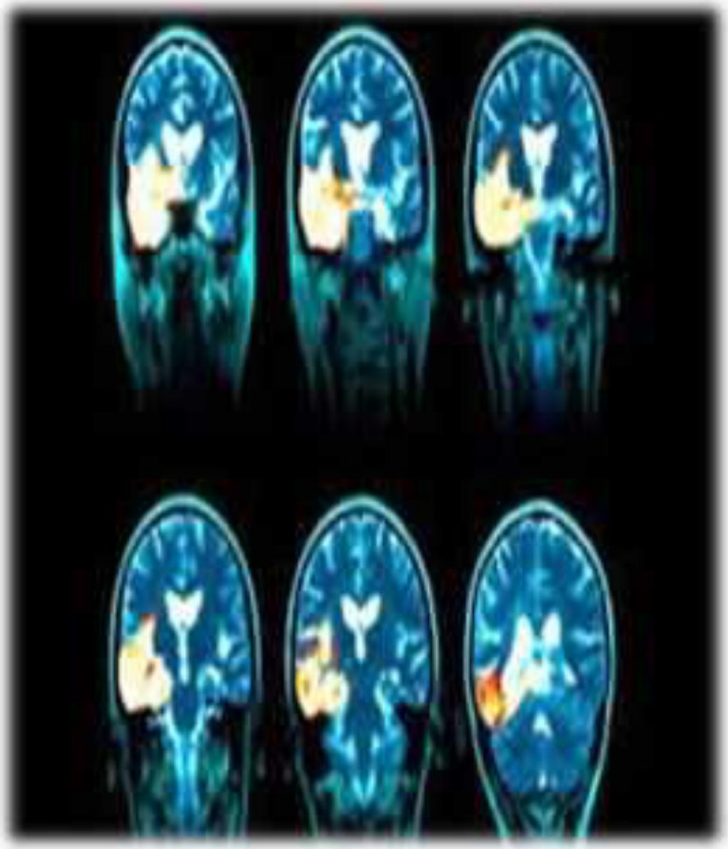
CLINICAL FINDINGS: HSV-2

- **Neonatal herpes**
- originates chiefly from contact with vesicular lesions within the birth canal.
- - varies from severe disease (e.g., disseminated lesions or encephalitis) to milder local lesions (skin, eye, mouth) to asymptomatic infection.
- - prevented by performing cesarean section on women with either active lesions or positive viral cultures.
- - neither HSV-1 nor HSV-2 causes congenital abnormalities to any significant degree.

NEONATAL HERPES

Other Manifestations.

- Meningitis,
- Encephalitis
- Multi organ Involvement
- Increased incidence in Immune compromised AIDS,
- Haematological Malignancies.



Laboratory Diagnosis

- Microscopy,
- Antigen Detection
- DNA detection PCR.
- Viral Isolation.
- Serology

Specimens for Diagnosis.

- Saliva.
- CSF
- Vesicle fluid.

Serology,

- ELISA Test
- Neutralization Tests
- Complement Fixation Tests

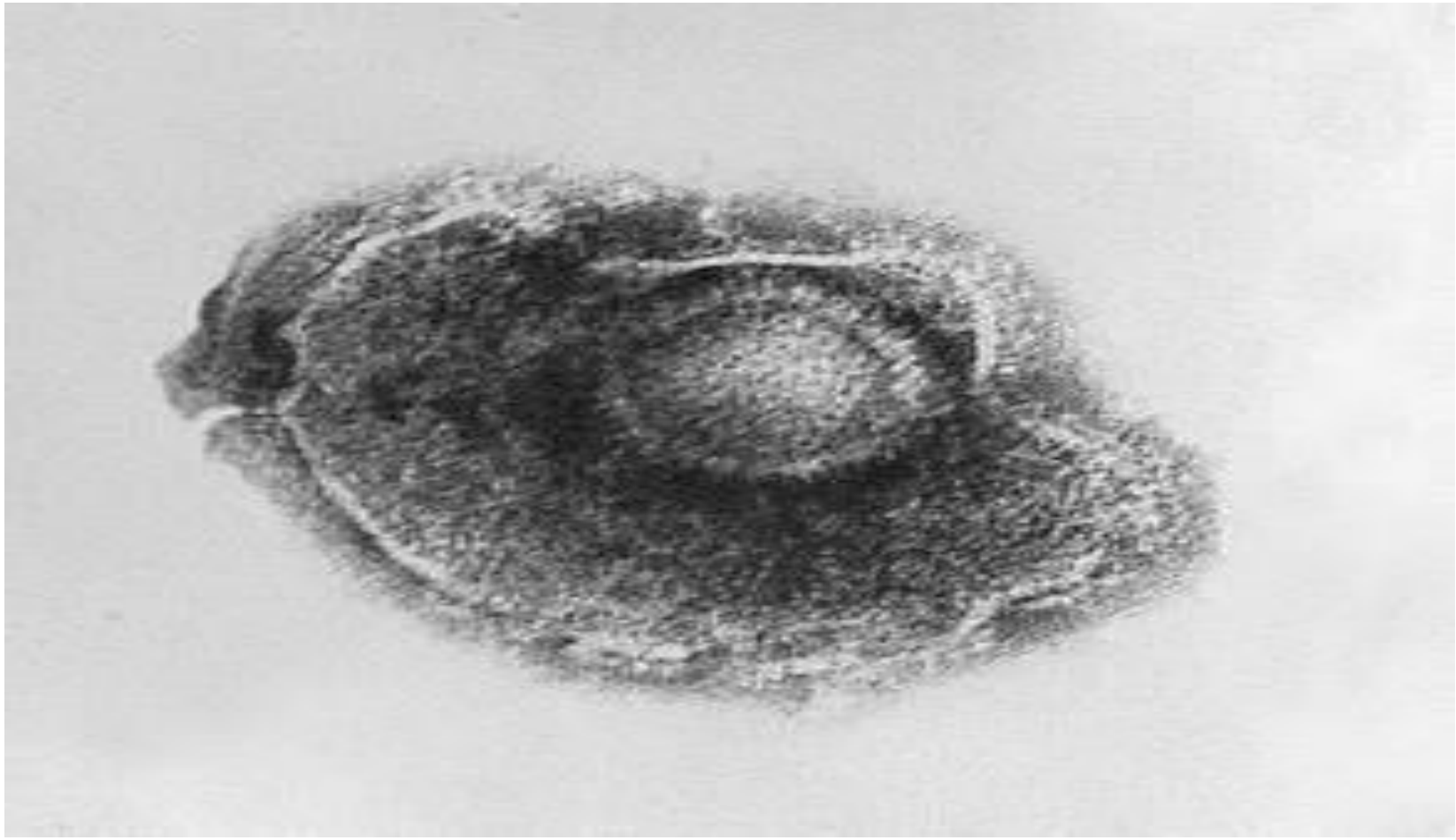
PREVENTION

- avoiding contact with the vesicular lesion or ulcer.
- Cesarean section is recommended for women who are at term and who have genital lesions or positive viral cultures.

Treatment

- Three antiviral drugs :Asyclovir
- Valacyclovir
- Famciclovir

varicella-zoster virus (VZV)



varicella-zoster virus (VZV)

Human herpesvirus 3 (HHV-3), usually referred to as the varicella-zoster virus (VZV), is one of nine herpes viruses known to infect humans. It causes **two diseases(1): chickenpox** (varicella),is highly infectious disease most commonly seen in children under 10 yearsand most healthy children

recover with no complications. Adults tend to suffer more severe disease than

children

and(2): shingles (herpes zoster) in adults; shingles is rare in children.

VZV infections are species-specific to humans but can survive in external environments for a few hours, maybe a day or two.

(1):Chickenpox

is the primary infection caused by the varicella-zoster virus, a highly contagious viral infection in which a person develops extremely itchy blisters all over the body and it is common childhood disease caused by a virus in the herpes family of viruses called the varicella virus.

The varicella virus can remain in the body for decades and become active again in adults, causing herpes zoster (shingles). Shingles involves the occurrence of painful skin sores along the distribution of nerves across the trunk or face

***the incidence of Chickenpox is seasonal reaching a peak from January to April when outbreaks of infection are common.

Transmission

- Chickenpox is highly contagious, The incubation period (the time from becoming infected until symptoms appear (ranges from 10 to 21 days although is usually from 14-16 days .
- individuals who have been in contact with a person with chickenpox should be considered potentially infectious from the 10th to the 21st day after exposure. The most infectious period is 1-2 days before the rash appears, but infectivity continues until all the vesicles have crusted over, at least 5 days after onset of the rash.

Chickenpox is transmitted by the following routes-:

.Airborne respiratory droplets**

. Direct contact with the vesicle fluid**

Indirect contact through contact with clothes, soiled by ** vesicle

fluid

Chickenpox can also be spread from people with shingles. A ** person with

shingles can spread the VZV virus to others who have never had

chickenpox.

The exposed person would need to come in contact directly or indirectly with the vesicle fluid of the person with shingles but .would develop chickenpox and not shingles

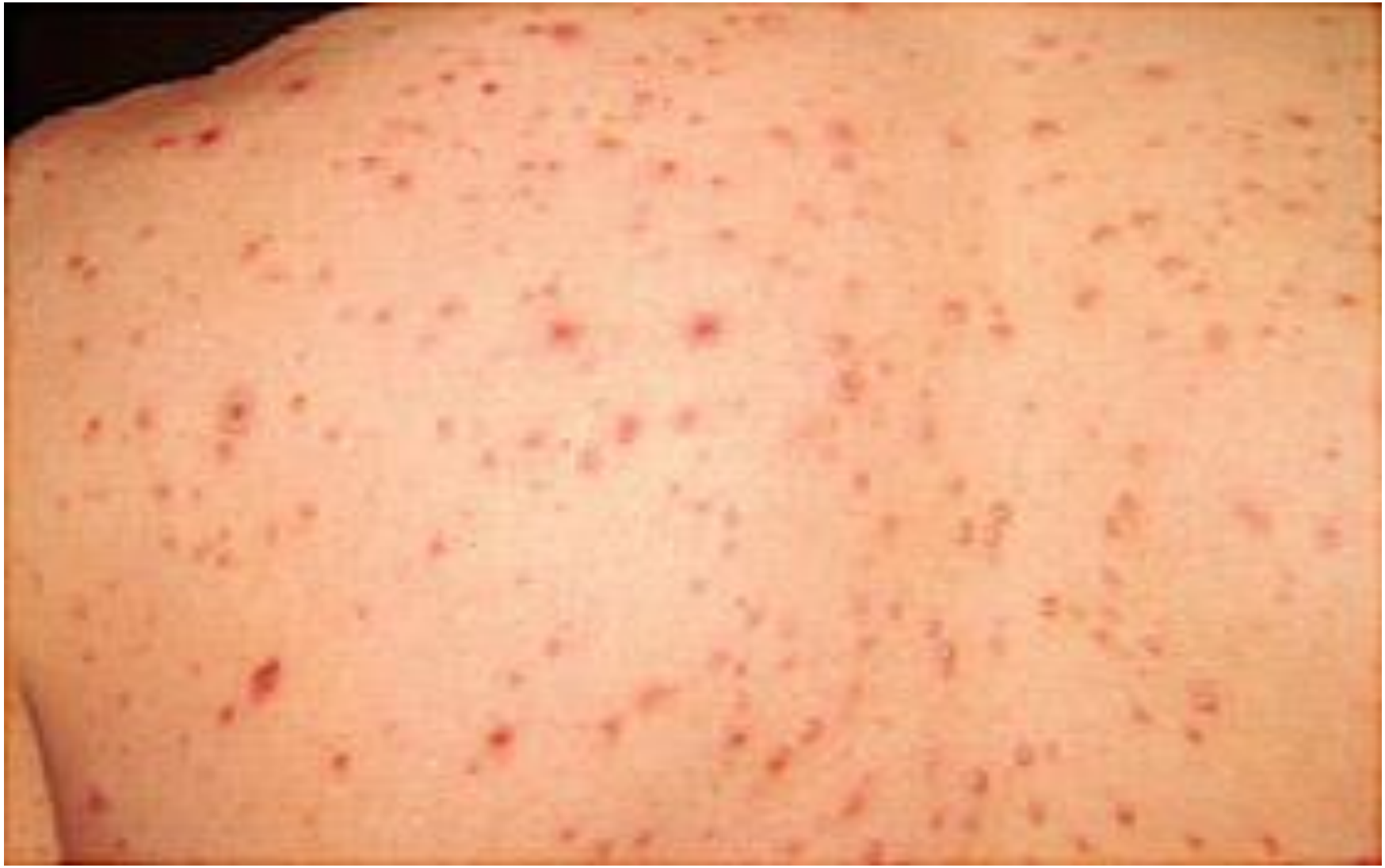
Signs and Symptoms

Chickenpox may initially begin with cold-like symptoms, This may be accompanied by fever, .mild headache and myalgia

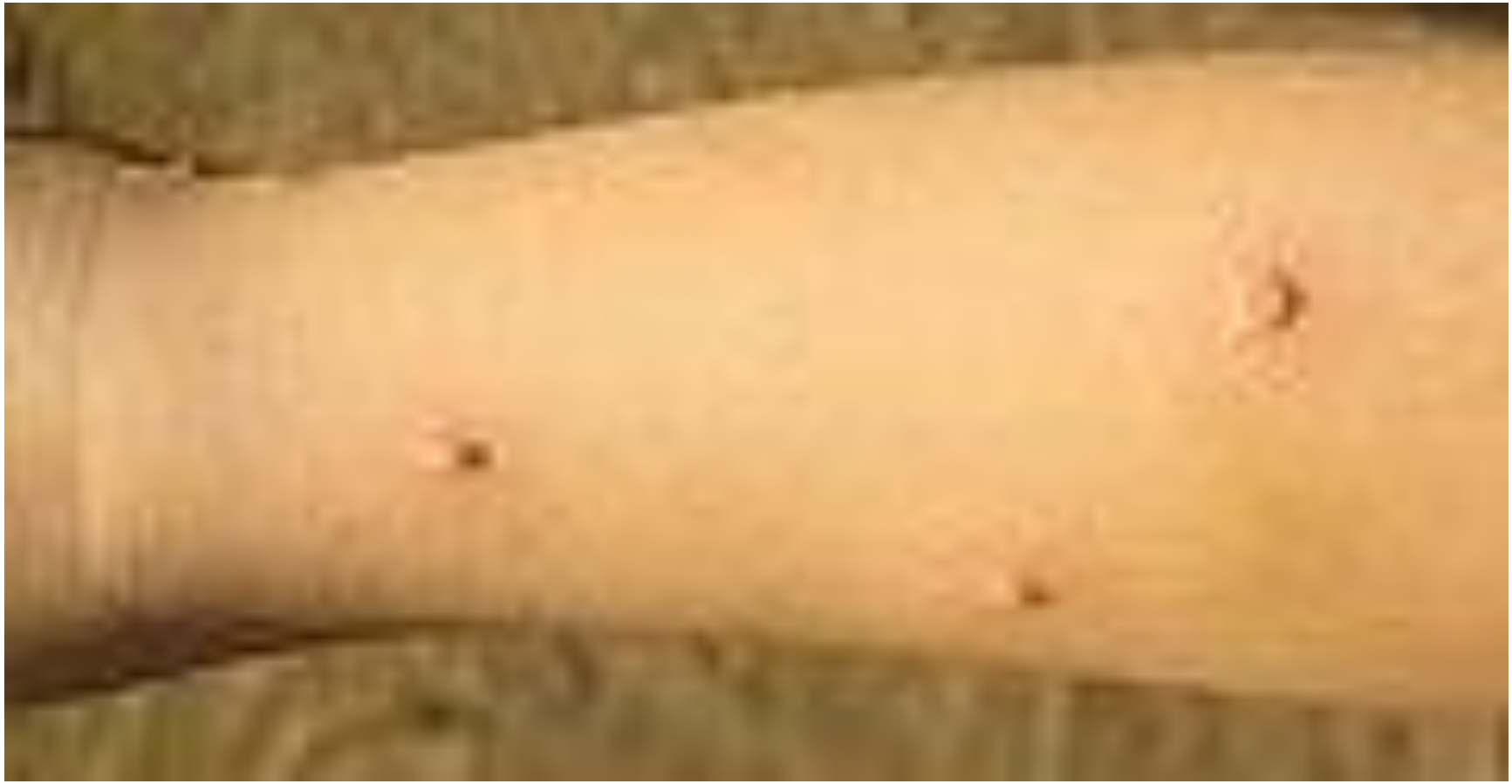
An itchy, vesicular (fluid-filled blister-like) rash - appears

vesicular spots appear, mostly over the trunk and to .a lesser extent the limbs

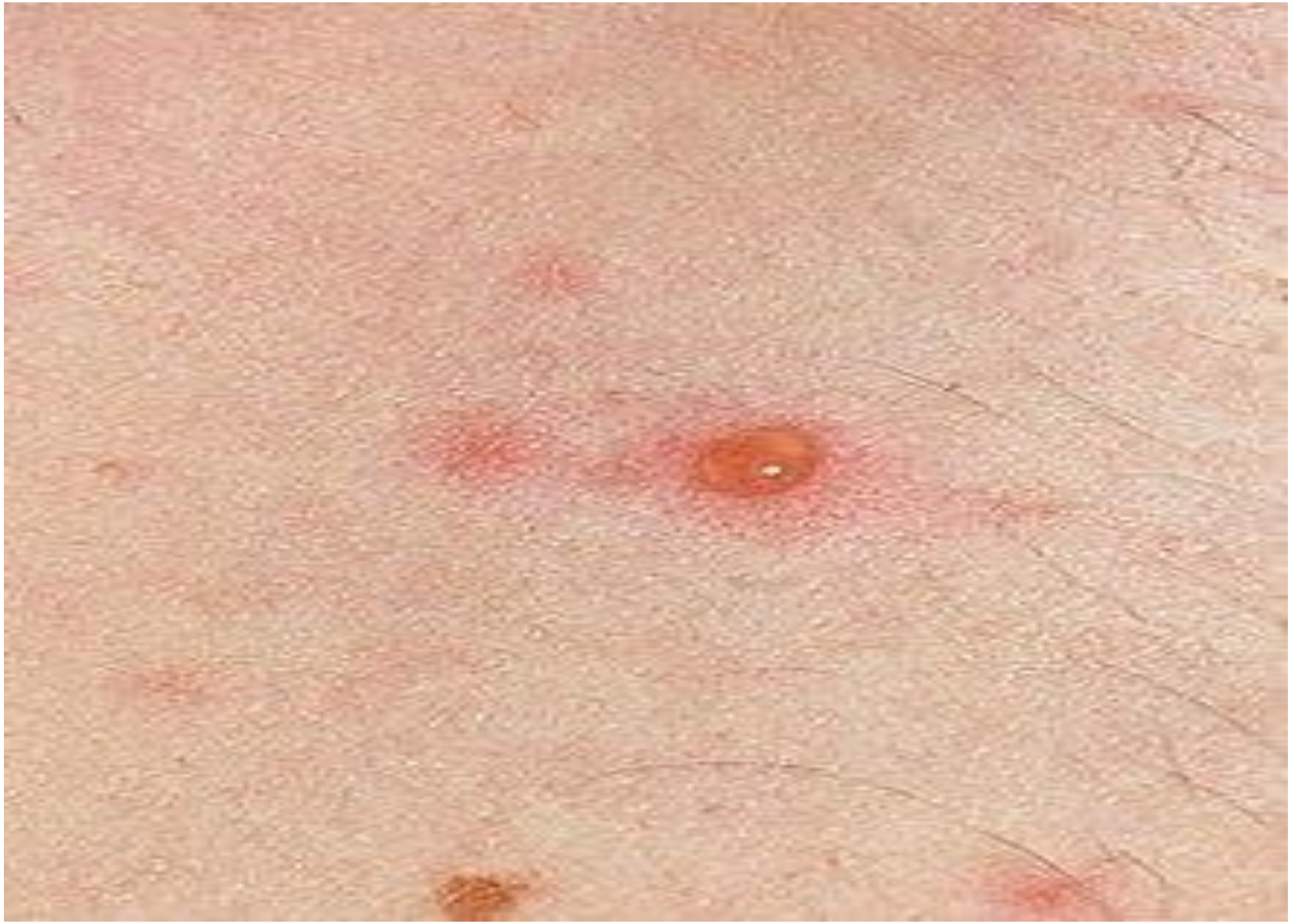
The severity of infection varies and it is possible to be infected but show no symptoms. Infectivity may be prolonged in people with altered .immunity



The back of a 30-year-old male after five days of the rash



Lower leg of a child with chickenpox



A single blister, typical during the early stages of the rash

Complications

The risk of complications varies with age and is higher ** .in infants under 1and in persons over 15 years

Nearly all children recover completely and have ** .detectable antibodies formany years

Complications in childhood may occur and include ** neurological complications (meningitis, encephalitis) and more rare glomerulonephritis and myocarditis

In children under 5, skin bacterial super infection is the ** most common complication. This manifests as a sudden high grade temperature (often after initial improvement

.

**adults with chickenpox may develope more severe disease with lung(varicella pneumonia.) , special in smokers

** infection in pregnancy (the risks to the foetus and neonate are related to the time of infection in the mother::

.a greater risk of severe varicella

**Varicella infection in the first 20 weeks of pregnancy can cause a variety of abnormalities in the foetus; low birth weight, underdevelopment of alimb(s), skin scarring, poor development of muscles or brain abnormality. The mortality rate ranges from 1-2%

Diagnosis

Chickenpox may be diagnosed by clinical signs and symptoms. The diagnostic

feature of chickenpox is the vesicular rash which starts as small papules, develop

.into clear vesicles that become pustules and then dry into crusts

The rash usually appears first on the trunk and appear after

several days over the hands and feet.

***Laboratory confirmation is rarely required but if necessary, by sending a microscopy slide with vesicle fluid to the Laboratory. and Serology is also available

Treatment

- There is no specific treatment for chickenpox. It is a viral infection that will therefore not respond to antibiotics. Treatment should be based on reducing symptoms such as fever and itchiness
- People at higher risk of developing serious complications from chickenpox may be given antiviral drugs such as acyclovir and/or immunoglobulin (a specialized preparation of antibodies taken from the plasma of blood donors), which may prevent severe illness from developing. These people include pregnant women, women who are not immune, neonates, immunosuppressed people

Prevention

**The spread of chickenpox can be prevented by isolating affected individuals.

**The varicella vaccine is recommended in many countries.

Refrences

- "Chickenpox (Varicella) Signs & Symptoms". Centers for Disease Control and Prevention (cdc.gov). 16 November 2011. Archived from the original on 4 February 2015. Retrieved 4 February 2015.
- ^ Jump up to: a b c d e f g h i j "Chickenpox (Varicella) Prevention & Treatment". cdc.gov. 16 November 2011. Archived from the original on 4 February 2015. Retrieved 4 February 2015.
- ^ Jump up to: a b "Chickenpox (Varicella) Overview". cdc.gov. 16 November 2011. Archived from the original on 4 February 2015. Retrieved 4 February 2015.
- ^ Jump up to: a b "Routine vaccination against chickenpox?". Drug Ther Bull. 50 (4): 42–5. 2012. doi:10.1136/dtb.2012.04.0098. PMID 22495050.
- ^ Jump up to: a b c GBD 2015 Mortality and Causes of Death, Collaborators. (8 October 2016). "Global, regional, and national life expectancy, all-cause mortality, and cause-specific mortality for 249 causes of death, 1980-2015: a systematic analysis for the Global Burden of Disease Study 2015". Lancet. 388 (10053): 1459–1544. doi:10.1016/s0140-6736(16)31012-1. PMC 5388903. PMID 27733281.
- ^ "Chickenpox (Varicella) Complications". cdc.gov. 16 November 2011. Archived from the original on 4 February 2015. Retrieved 4 February 2015.
- ^ Jump up to: a b c d e f g h i j k Atkinson, William (2011). Epidemiology and Prevention of Vaccine-Preventable Diseases (12 ed.). Public Health Foundation. pp. 301–323. ISBN 9780983263135. Archived from the original on 7 February 2015. Retrieved 4 February 2015.
- ^ "Chickenpox (Varicella) Interpreting Laboratory Tests". cdc.gov. 19 June 2012. Archived from the original on 4 February 2015. Retrieved 4 February 2015.
- ^ Breuer J (2010). VZV molecular epidemiology. Current Topics in Microbiology and Immunology. 342. pp. 15–42. doi:10.1007/82_2010_9. ISBN 978-3-642-12727-4. PMID 20229231.
- ^ Jump up to: a b c Flatt, A; Breuer, J (September 2012). "Varicella vaccines". British Medical Bulletin. 103 (1): 115–27. doi:10.1093/bmb/lds019. PMID 22859715.

Lec7
Viral hepatitis

Viral hepatitis

Hepatitis: is a general term referring to inflammation of the liver,by the way , The liver is essential for removing toxins from the blood, storing vitamins, and producing hormones. Hepatitis, however, can disrupt these processes.

hepatitis may result from various causes, may be

1/ **infectious** (ie, viral, bacterial, fungal, and parasitic organisms)

2/**noninfectious** (eg, alcohol, drugs, and autoimmune diseases).

Note ::this lecture focuses on viral hepatitis

- At least five viruses can cause hepatitis A, B, C, D, and E.. The three most common are hepatitis A, B, and C. Infection with any of these three viruses can lead to life threatening complications.
- Each type has different characteristics, and transmission happens in different ways, but the symptoms tend to be similar

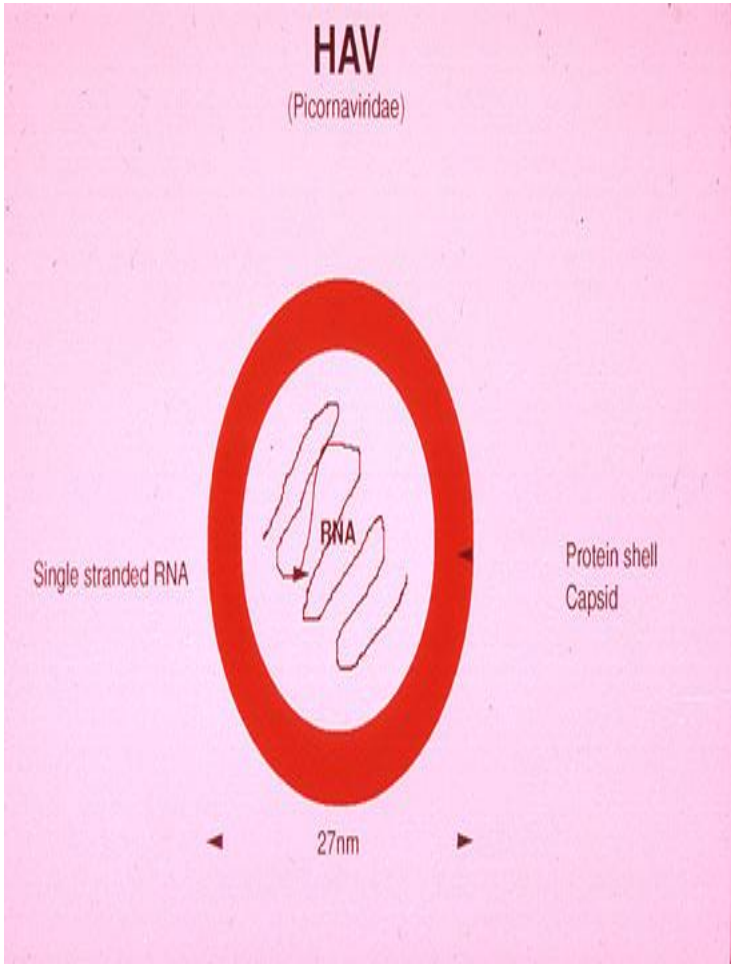
- Hepatitis can heal on its own with no significant consequence, or it can progress to scarring of the liver. Acute hepatitis lasts under six months, while chronic hepatitis lasts longer.
- Most liver damage is caused by 3 hepatitis viruses, called hepatitis A, B and C. However, hepatitis can also be caused by alcohol and some other toxins and infections, as well as from our own autoimmune process (the body attacks itself).

1) HEPATITIS A(infectious hepatitis)

BASICS structure:

- enterovirus 27nm , picornoviridae family,
- Naked ,genome SS RNA withIcosahedral nucleocapsid
- Replication occurs in the cytoplasm of the cell
- Single serotype
- Worldwide distribution.

Humans are the only reservoir



HAV

Transmission

- The hepatitis A virus is transmitted primarily by the faecal-oral route; that is when an uninfected person ingests food or water that has been contaminated with the faeces of an infected person. In families, this may happen though dirty hands when an infected person prepares food for family members.
- It's the least risky type because it almost always gets better on its own so It doesn't lead to long-term inflammation of your liver.
- In rare cases, hepatitis A can be fatal. However, there are safe and effective vaccines that protect against this virus.

Route of entry and Progress of infection:

- The virus is acquired by ingestion.
- It multiplies in the intestine and invades the blood, liver and saliva before any clinical manifestation of the disease appears. This period of incubation lasts an average of (14-28) days.
- The virus disappears soon after the peak of serum transaminase enzyme is reached at which time the immune response and the hepatocellular damage start,this indicates that the damage is immunologically mediated. Indeed at this time, CD8+ cytotoxic T lymphocytes that secrete gamma interferon infiltrate the field.

Symptoms

- The incubation period of hepatitis A is usually 14–28 days. Symptoms of hepatitis A range from mild to severe, and can include fever, malaise, loss of appetite, diarrhea, nausea, abdominal discomfort, and dark-coloured urine and jaundice (a yellowing of the skin and whites of the eyes). However, many people do not experience symptoms at all. Those who do usually make a full recovery within a few weeks to several months. After this, they have immunity to it. Children under 6 years do not usually show any symptoms.

How Jaundice Develops?

- Jaundice is the consequence of having too much bilirubin in the blood, Bilirubin is a yellow-pigmented substance derived from metabolized red blood cells. As old red blood cells enter the spleen, they are broken down and formed into bilirubins which the liver uses to create bile.

- The body avoids the accumulation of bilirubin by excreting any excess through urine or in stools. However, if the system is disrupted, there may be more bilirubin in the blood than the body can handle. If this happens, the accumulation can saturate cells and manifest with the yellowing we recognize as jaundice.

Diagnosis

- Hepatitis A is not clinically distinguishable from other types of acute viral hepatitis. Specific diagnosis is made by the detection of HAV-specific IgM and IgG antibodies in the blood. Additional tests include reverse transcriptase polymerase chain reaction (RT-PCR) to detect the hepatitis A virus RNA, but may require specialised laboratory

Treatment

- There is no specific treatment for hepatitis A. Recovery from symptoms following infection may be slow and may take several weeks or months. Therapy is aimed at maintaining comfort and adequate nutritional balance, including the replacement of f luids that are lost from vomiting and diarrhea.

Prevention

*Improved sewage

**food safety and immunization are the most effective ways to combat hepatitis A.

and **the spread of hepatitis A can be reduced by**:

1-supplies of safe drinking water;

2-proper disposal of sewage within communities; and

3-personal hygiene practices such as regular hand-washing before meals and after going to the bathroom.

*****Several injectable inactivated hepatitis A vaccines are available internationally. All are similar in terms of how well they protect people from the virus and their side-effects.

Q/Who are the people at risk of developing hepatitis A, so it is recommended to give them the vaccine ?

- Some countries also recommend the vaccine for people at increased risk of hepatitis A, including:

- users of recreational drugs;
- travellers to countries where the virus is endemic;
- men who have sex with men
- people with chronic liver disease (because of their increased risk of serious complications if they acquire hepatitis A infection).

THANK YOU •

LEC 8

Hepatitis B virus

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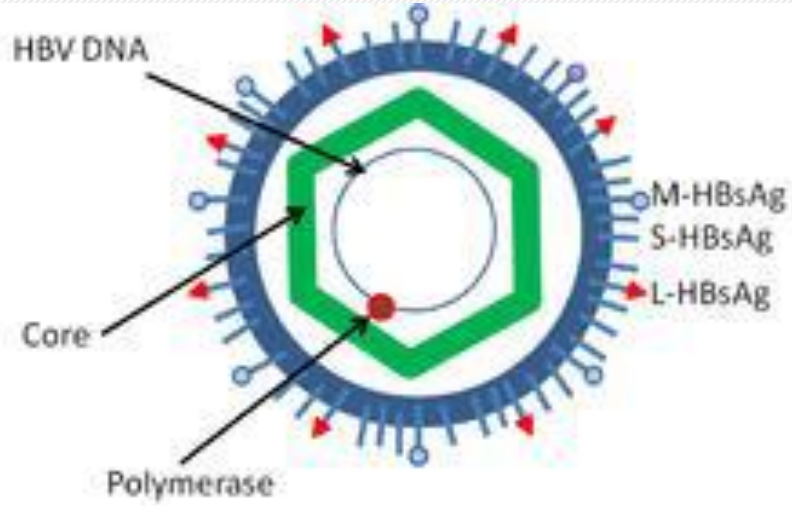
The structure of hepatitis B virus

- Hepatitis B virus (HBV) is a member of the hepadnaviridae family.
- The virus particle (virion) consists of an outer lipid envelope and an icosahedral nucleocapsid
- These virions are 30–42 nm in diameter.
- The nucleocapsid encloses the viral DNA and a DNA polymerase that has reverse transcriptase activity



The structure of hepatitis B virus

- In addition to the Dane particles, filamentous and spherical bodies lacking a core can be found in the serum of infected individuals. These particles are composed of the lipid and protein that forms part of the surface of the virion, which is called the surface antigens (HBsAg), and is produced in excess during the life cycle of the virus.
- Dane partcles :the 42nm virion which are capable of infecting liver cells .
- **HBsAg** (hepatitis B surface antigen)
  HBsAb or anti-HBs (hepatitis B surface antibody)
  HBcAb or anti-HBc (hepatitis B core antibody)

Transmission

- Transmission of hepatitis B virus results from exposure to infectious blood or body fluids.
- Possible forms of transmission include sexual contact,[blood transfusions and re-use of contaminated needles and syringes and
- vertical transmission from mother to child during childbirth. a mother who is positive for HBsAg has a 20% risk of passing the infection to her offspring at the time of birth. HBV can be transmitted between family members within households, possibly by contact of non intact skin or mucous membrane with secretions or saliva containing
- The virus may be detected within 30 to 60 days after infection and can persist and develop into chronic hepatitis B. The incubation period of the hepatitis B virus is 75 days on average but can vary from 30 to 180 days

Mechanisms

- Hepatitis B virus primarily interferes with the functions of the liver by replicating in hepatocytes.
- The virions bind to the host cell via the preS domain of the viral surface antigen.
- During HBV infection, the host immune response causes both hepatocellular damage and viral clearance. Although the innate immune response does not play a significant role in these processes, the adaptive immune response, in particular virus-specific cytotoxic T lymphocytes (CTLs), contributes to most of the liver injury associated with HBV infection. CTLs eliminate HBV infection by killing infected cells and producing antiviral cytokines, which are then used to purge HBV from viable hepatocytes

Symptoms

- **Acute infection** with hepatitis B virus is associated with acute viral hepatitis, an illness that begins with general ill-health, loss of appetite, nausea, vomiting, body aches, mild fever, and dark urine, and then progresses to development of jaundice. The illness lasts for a few weeks and then gradually improves in most affected people. The infection may be entirely asymptomatic and may go unrecognized.
- **Chronic infection** with hepatitis B virus either may be asymptomatic or may be associated with a chronic inflammation of the liver (chronic hepatitis), leading to cirrhosis over a period of several years. This type of infection dramatically increases the incidence of hepatocellular carcinoma (HCC; liver cancer).

Diagnosis

- The tests, called assays, for detection of hepatitis B virus infection involve serum or blood tests that detect either viral antigens (proteins produced by the virus) or antibodies produced by the host.
- PCR tests

Treatment

- Acute hepatitis B infection does not usually require treatment and most adults clear the infection spontaneously
- On the other hand, treatment of chronic infection may be necessary to reduce the risk of cirrhosis and liver cancer.
- Although none of the available medications can clear the infection, they can stop the virus from replicating, thus minimizing liver damage.

Prevention

- HBV vaccine is recommended:
- 1- for all children as part of their regular
- immunization schedule(Routine vaccination of 0-18 year olds)
- 2- Vaccination of risk groups of all ages
- Groups that screening is recommended for include those who have not been vaccinated and one of the following
- people from areas of the world where hepatitis B occurs in more than 2%
- those with HIV
- intravenous drug users, and those who live with someone with hepatitis B.

Thank you

LEC 9

Orthomyxovirus

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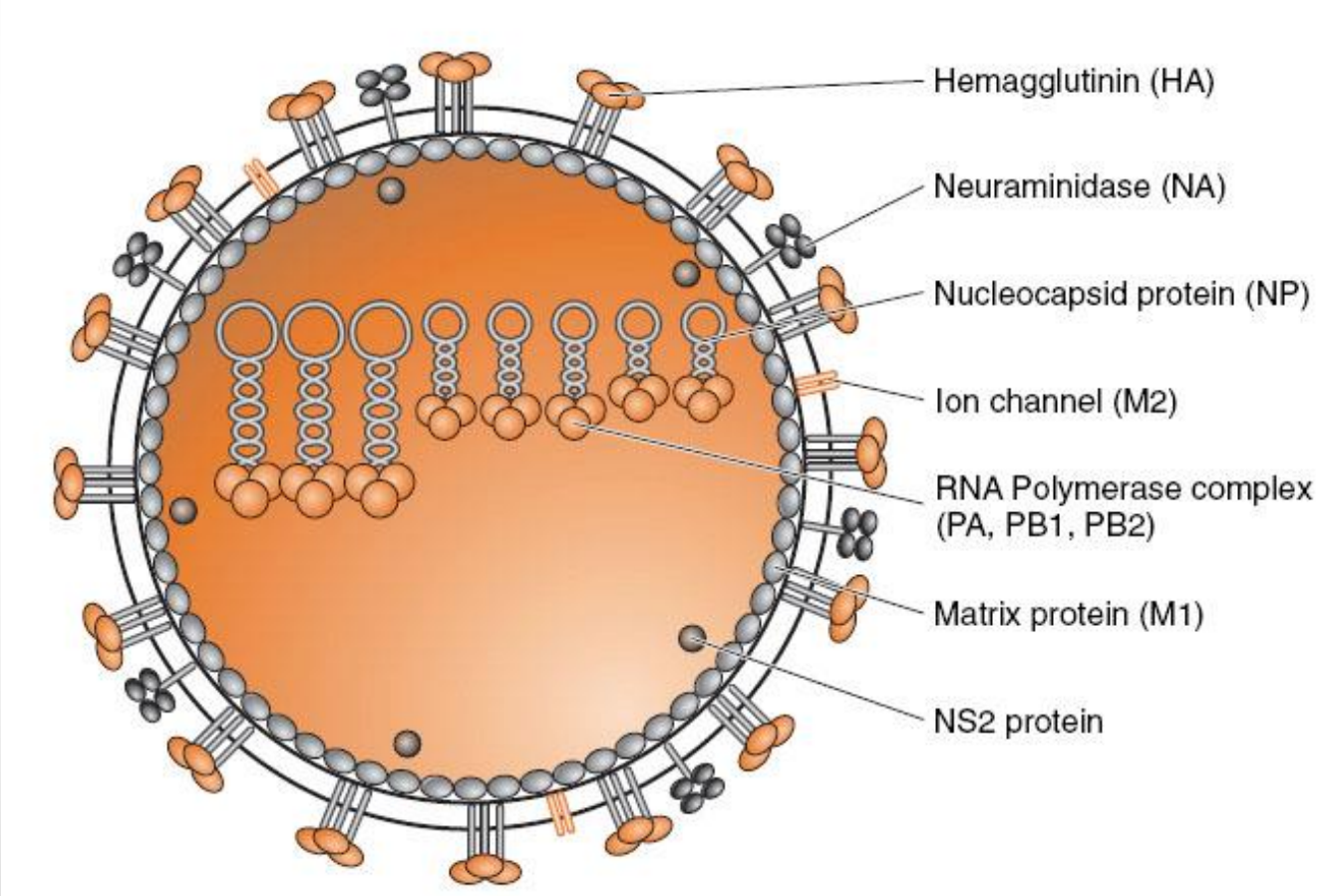
Introduction

- the orthomyxoviruses (influenza viruses) constitute the genus Orthomyxovirus, which consists of three types (species): A, B, and C. These viruses cause influenza, an acute respiratory disease with prominent systemic symptoms. Pneumonia may develop as a complication and may be fatal, particularly in elderly persons with underlying chronic disease.
- 3 Types: A, B and C
- Hosts: Birds, various mammals and humans
- Enveloped virion, 80-120 nm diameter

Structure

Influenza viruses are spherical and 80 to 120 nm in diameter, although filamentous forms may also occur. y. The nucleocapsid is enclosed in an envelope consisting of a lipid bilayer and two surface glycoproteins, a hemagglutinin and a neuraminidase. Because influenza viruses are enveloped.



Types

- **Influenza A**
- Influenza A viruses are further classified, based on the viral surface proteins hemagglutinin (HA or H) and neuraminidase (NA or N). Sixteen H subtypes (or serotypes) and nine N subtypes of influenza A virus have been identified.
- **Influenza B**
- Influenza B virus is almost exclusively a human pathogen, and is less common than influenza A. The only other animal known to be susceptible to influenza B infection is the seal. This type of influenza mutates at a rate 2–3 times lower than type A

Types

- **Influenza C**
- The influenza C virus infects humans and pigs, and can cause severe illness and local epidemics.[46] However, influenza C is less common than the other types and usually causes mild disease in children

Transmission / symptoms

- **Transmission**
- Typically, influenza is transmitted from infected mammals through the air by coughs or sneezes, creating aerosols containing the virus, and from infected birds through their droppings. Influenza can also be transmitted by saliva, nasal secretions, feces and blood
- **symptoms**
- Headache, chills, fever, malaise, myalgias, anorexia, and sore throat appear suddenly. The fever rapidly climbs to 101 to 104°F (38.3 to 40.0°C), and respiratory symptoms ensue. A nonproductive cough is characteristic. Sneezing, rhinorrhea, and nasal obstruction are common.

Pathogenesis

- Influenza virus is transmitted from person to person primarily in droplets released by sneezing and coughing. Some of the inhaled virus lands in the lower respiratory tract,. Infection of mucosal cells results in cellular destruction and desquamation of the superficial mucosa. Although the cough may be striking, the most prominent symptoms of influenza are systemic: fever, muscle aches, and general prostration. Viremia is rare, so these systemic symptoms are not caused directly by the virus.

Diagnosis

- A diagnosis of influenza is suggested by the clinical picture of sudden onset of fever, malaise, headache, marked muscle aches, sore throat, nonproductive cough, and coryza. When a syndrome resembling influenza occurs in the winter in an adult (the etiologies of illnesses of this type are more complex in children), an influenza virus is a likely cause.
- A rapid specific diagnosis of influenza may be obtained by demonstrating viral antigens in cells obtained from the nasopharynx in immunostaining tests such as immunofluorescence or in enzyme immunoassays (ELISA) employing respiratory secretions.
- PCR

Prevention

- Inactivated influenza virus vaccines have been used for about 40 years to prevent influenza. A given vaccine contains the strains of types A and B viruses that are judged most likely to produce epidemics during the following winter. The vaccine is administered parenterally in the fall; one or two doses are required, depending on the immune experience of the population with related antigens.

Treatment

- Amantadine and rimantadine are the only specific antiviral treatments available for influenza. As in the case of prophylaxis, they are effective only against type A virus. When administration is started early in the course of illness, drugs hasten the disappearance of fever and other symptoms. Emergence of viral resistance can occur during treatment

Medical Mycology

م.م ايهاب مجيد عباس

Medical Mycology

- **Medical mycology** is the study of mycoses of man and their etiologic agents.

  Mycoses are the diseases caused by fungi. Of the several thousands of species of fungi that are known, less than 300 are pathogenic to man.

  fungal invasion of human tissue was recognized in the early 1800s before the science of bacteriology was developed.

What is a Fungus ?

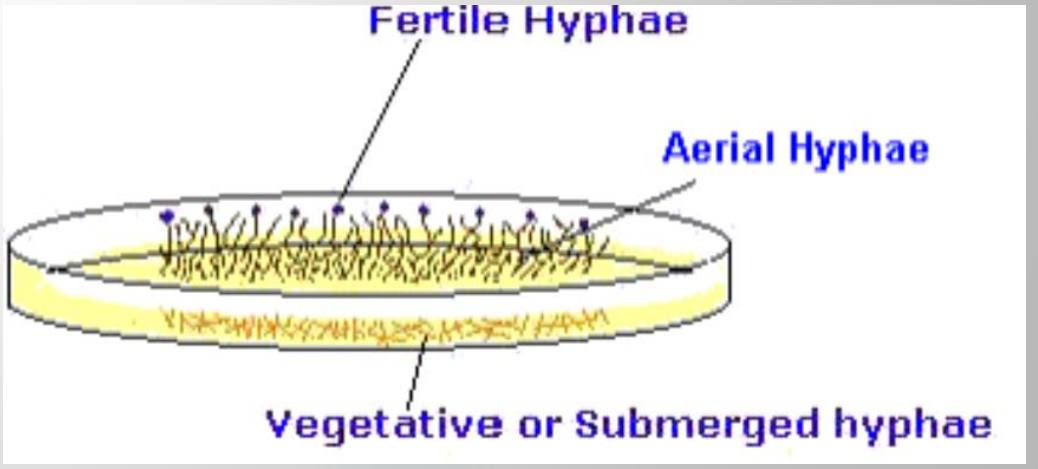
- **Eukaryotic** – a true nucleus , heterotrophic, do not contain chlorophyll
- Produce filamentous structures (hyphae)
- Produce spores (sexual & asexual reproduction)
- Saprophytic (on dead tissue).Parasitic (on living organism).
- All fungi required organic source of Carbon associated with decaying matter

Morphology of Fungi

- Filamentous fungi (molds) is long branching filaments. Mass of filaments called mycelium,
- Yeasts , round or oval bodies which reproduce by the formation of buds known as blastospores.
- Yeast-like fungi , this form of undetached budding yeast-cells which present the appearance of broad septate hyphae
- Dimorphic Fungi :These are fungi which exhibit a filamentous mycelial morphology (saprophytic phase) when grown at room temperature 27oC, but have a typical yeast morphology (parasitic phase) inside the body and when grown at 37oC in the laboratory.

Basic Structures

- **1. Hypha -** fundamental tube-like structural units of fungi.
- a. Septate - divided by cross walls
- b. Aseptate - lacking cross walls
- **2. Mycelium -** a mass / mat of hyphae forming the vegative portion of the fungus
- a. Aerial - growing or existing in the air
- b. Vegetative - absorbs nutrients
- c. Fertile – spores for reproduction



Spores

Sexual spores - fusion of nuclei

- 1. **Ascospore -** spore formed in a sac-like cell known as an ascus, the shape of which aids in identification of the fungus. Often eight (8) spores formed. (sexual). (Ascomycetes)
- 2. **Basidiospore -** sexual spore (union of two nuclei) produced on a specialized club-shaped structure, called a basidium. (Basidiomycetes)
- 3. **Zygospore -** a thick-walled spore formed during sexual reproduction in the Phycomycetes

Spores

Asexual spores - most common type

- 4. **Conidia** - asexual fungal spores borne externally in various ways from a conidiophore; often referred to a macro- and microconidia. Macroconidia are multicellular , Microconidia are unicellular.
- 5. **Arthroconidia** (Arthrospore) - special type of asexual spore formed by disarticulation of the mycelium.
- 6. **Blastoconidia / Blastospore** - asexual spore formed from a budding process along the mycelium or from another blastospore. (class Ascomycetes)

Asexual spores

- 7. **Chlamydospore -** thick-walled resistant asexual spore formed by direct differentiation of the mycelium
- 8. **Sporangiospore -** an asexual spore contained in a sporangium at the end of a sporangoiphore of the taxonomic class Phycomycetes
- 9. **Thallospore -** asexual spore produced on a thallus (hypha). (Deuteromycetes)

Epidemiology

- Normal habitat is the environment except *candida albicans* is part of normal human flora.
- Most mycotic agents are soil saprophytes.
- Mycotic diseases are **not contagious** except in few cases as in superfacial mycoses.
- Establishment of infection depends on inoculum size , resistance of the host rather than virulence of fungus

Categories of systemic disease:

- Those caused by truly pathogenic fungi with the ability to cause disease in the normal human host when the inoculum is of sufficient size (Histoplasma capsulatum, Blastomyces dermatitidis, Coccidioides immitis, Paracoccidioides braziliensis).
- Those caused by opportunistic fungi, low virulence organisms, which require the patient's defenses to be lowered before the infection is established (Aspergillus spp. Candida albicans, Cryptococcus neoformans).

Diagnosis

1. **Wet Mount (KOH)**
2. Skin test (dermal hypersensitivity)
3. **Serology** (Latex agglutination , Complement fixation ….)in systemic infection
4. Fluorescent antibody
5. Biopsy and histopathology(pyogenic , granulomatous ,or necrotic)
6. **Culture (Sabouraud dextrose agar)**
7. DNA probes

SUPERFICIAL MYCOSES

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CLASSIFICATION OF MYCOSES

- **Superficial mycoses**
- Superficial mycoses are limited to the outermost layers of the skin and hair.
- **Cutaneous mycoses**
- Cutaneous mycoses extend deeper into the epidermis, and also include invasive hair and nail diseases.
- **Subcutaneous mycoses**
- Subcutaneous mycoses involve the dermis, subcutaneous tissues, muscle.
- **Systemic mycoses**
- Systemic mycoses due to primary pathogens originate normally in the lungs and may spread to other organ systems.
- **opportunistic mycoses**
- opportunistic pathogens are infections of patients with immune deficiencies who would otherwise not be infected.

SUPERFICIAL MYCOSES

- The term “superficial mycosis” applies to diseases affecting the outermost layer of the skin (hair and nails).
- The most important types of these infections:
- 1-**Otomycosis** is a fungal ear infection, a superficial mycotic infection of the outer ear canal. The infection may be either sub acute or acute and is characterized by malodorous discharge, inflammation, pruritus, scaling, and severe discomfort. The mycosis results in inflammation, superficial epithelial masses of debris containing hyphae, suppuration, and pain

OTOMYCOSIS

- **Causes**
- Most fungal are infections are caused by *Aspergillus niger, Aspergillus fumigatus, Penicillium* and *Candida albicans*
- **Diagnosis**
- The fungal mass may appear white, brown or black Examined with an Otoscope
- Material taken for microscopic examination using a potassium hydroxide(KOH) preparation will reveal branching hyphae, budding cells, or both. Or the sporing head of *Aspergillus* infection. Culture fungal material on (SDA) (Sabouraud Dextrose Agar) will enable the species of the fungus involved to be identified.

TREATMENT

- Topical nystatin can be applied three times a day for two to three weeks.
- clotrimazole or econazole nitrate also give good result.

2-PIEDRA

- **2-Piedra** is a hair disease caused by a fungus, which causes formation of nodules on the hair shaft.
- • Types include:
- • **White piedra** (or tinea blanca): is a mycosis of the hair caused by several species of fungi in the genus ***Trichosporon***.
- • **Black piedra**: caused *by* ***Piedraia hortae*** is a superficial fungus that exists in the soils of tropical and subtropical environments and affects both sexes of all ages.This fungus grows very slowly, forming dark hyphae, which causes the formation of black nodules on the hair shaft and leads to progressive weakening of the hair.

2-PIEDRA

- **Diagnosis**
- a microscopic examination using potassium hydroxide KOH 10% where the small nodules formed on the hair are taken and placed with a drop of the solution above or a drop of lactophenol dye or by culture the specimen on SDA agar where an with - cycloheximide is added in the case the black piedra and without cycloheximide, in the case of the white piedra because it is sensitive to this
- **treatment**
- use formalin or use mercury solutions, Ketoconazole shampoo

TINEA VERSICOLOR

- **3- Tinea versicolor(pityriasis versicolor)**
- is a condition characterized by a skin eruption on the chest, back, , armpits, face and proximal extremities. The majority of tinea versicolor is caused by the fungus ***Malassezia furfur*** , These yeasts are normally found on the human skin and become only under certain circumstances, such as humid environment, although the exact conditions that cause initiation of the disease process are poorly understood.

TINEA VERSICOLOR

- **diagnosis**
- The diagnosis is in the direct microscopic examination, by placing the sample taken from the lesion area represented by the scrap present in the lesion area and mixed with a drop of potassium hydroxide solution or by using the culture method on Sabroud dextrose agar that added to it chloramphenicol
- **Treatment**
- for tinea versicolor include:
- Topical antifungal medications Ketoconazole (Nizoral ointment and shampoo) is another treatment. It is normally applied to dry skin and washed off after 10 minutes, repeated daily for two weeks

4- TINEA NIGRA

- **4- Tinea nigra**, is a superficial fungal infection that causes dark brown to black, painless patches called macules on the palms of the hands and the soles of the feet of otherwise healthy individuals. The macules can be anwywhere from a few mm to several cm in size
- **Causes** : *Exophiala werneckii*

DIAGNOSIS OF TINEA NIGRA

- • is made based on microscopic examination of stratum corneum skin scrapings obtained by using a scalpel. The scrapings are mixed with potassium hydroxide (KOH). The KOH lyses the nonfungal debris. The skin scrapings are cultured on Sabouraud's agar at 25°C and allowed to grow for about a week. *Exophiala* werneckii can generally be distinguished due to its two-celled yeast form and the presence of septate hyphae with thick, darkly pigmented walls.

TREATMENT

- Topical antifungal imidazoles such as ketoconazole, itraconazole, and miconazole may be used.

Cutaneous Mycoses

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Classification of mycoses

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- **opportunistic mycoses**
- opportunistic pathogens are infections of patients with immune deficiencies who would otherwise not be infected

Cutaneous Mycoses

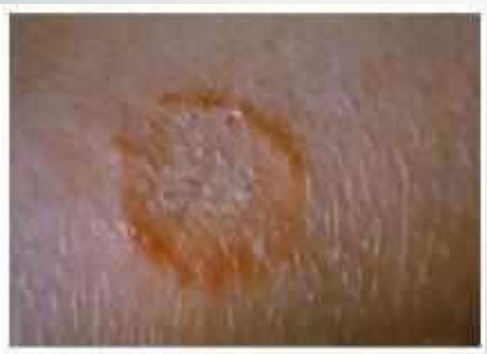
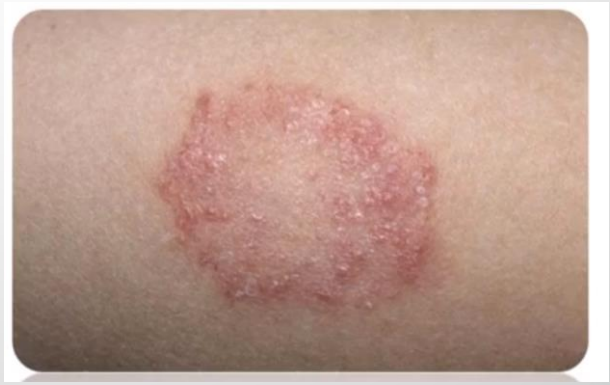
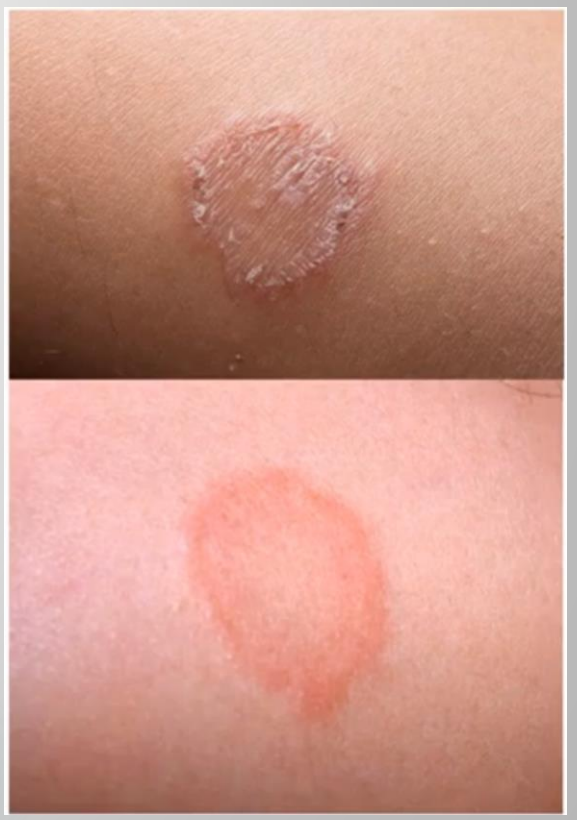
- These are superficial fungal infections of the skin, hair or nails. No living tissue is invaded, however a variety of pathological changes occur in the host because of the presence of the infectious agent and its metabolic products.
- **Dermatophytes Classification**
- Classify into three Groups according to
- • Clinical
- • Etiological
- • Ecological

Clinical manifestations of Dermatophytosis

- • Skin: Circular, dry, erythematous, scaly, itchy lesions
- • Hair: Typical lesions, ”kerion”, scarring, “alopecia”
- • Nail: Thickened, deformed, friable, discolored nails, debris accumulation

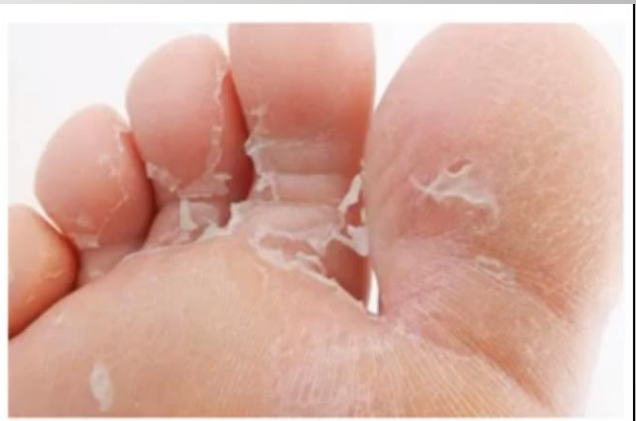
1-Clinical classification

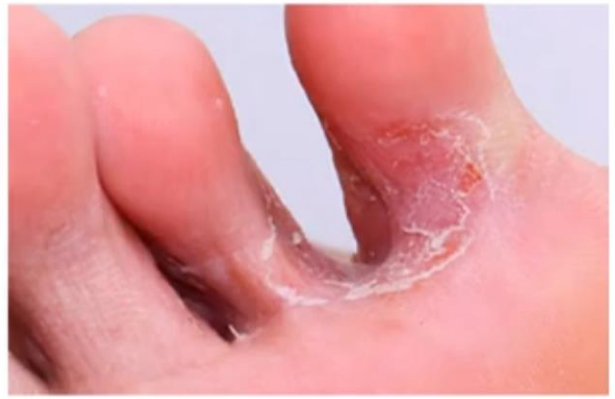
- • Infection is named according to the site of infection,
- **Tinea corporis**: This is small lesions occurring anywhere on the body.Itis a disease of childhood.It is also known as ringworm.It is commonly caused by all species of Dermatophytes

1-Clinical classification

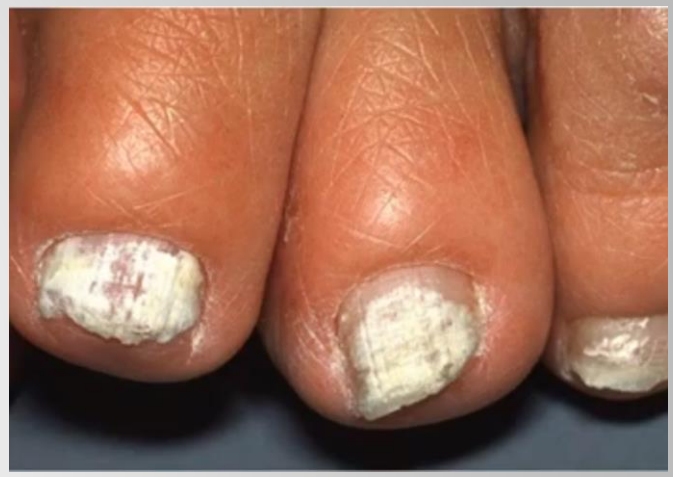
- **Tinea pedis**: athlete's foot". Infection of toe webs and soles of feet.
- This is seen in children and young adults. It is usually due to
- **1. Trichophyton mentagrophytes**
- **2. T. rubrum.**
- It causes toe-web fissures, maceration, scaling of soles, erythema,vesicles/pustules, and bullae.
- Bacterial organisms can produce an identical appearance.

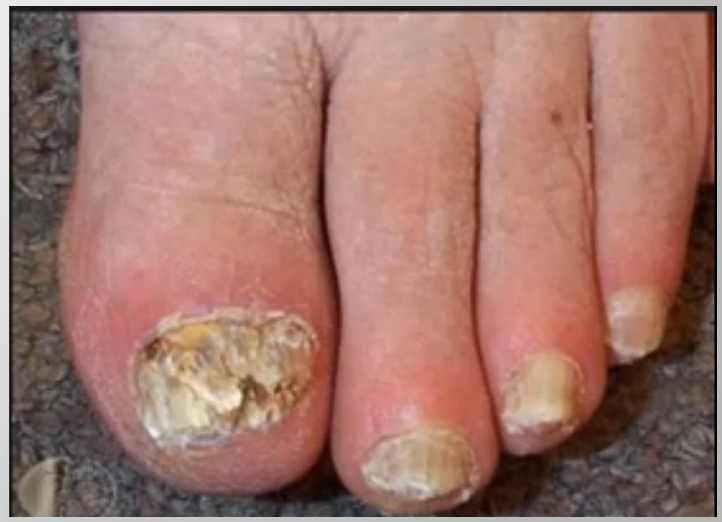




1-Clinical classification

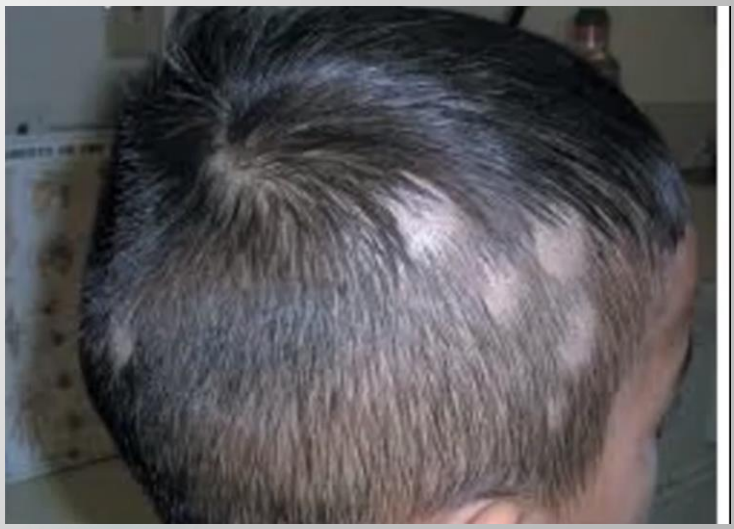
- **Tinea unguium (onychomycosis) :**
- The infections are on the nails.
- Sometimes, the infections are lifelong.
- In the commonest form, the nail is invaded from the nail bed.
- Thick, discolored (white, yellow, brown, black), dystrophic nails.
- It can occur at any age but is commoner with increasing age.





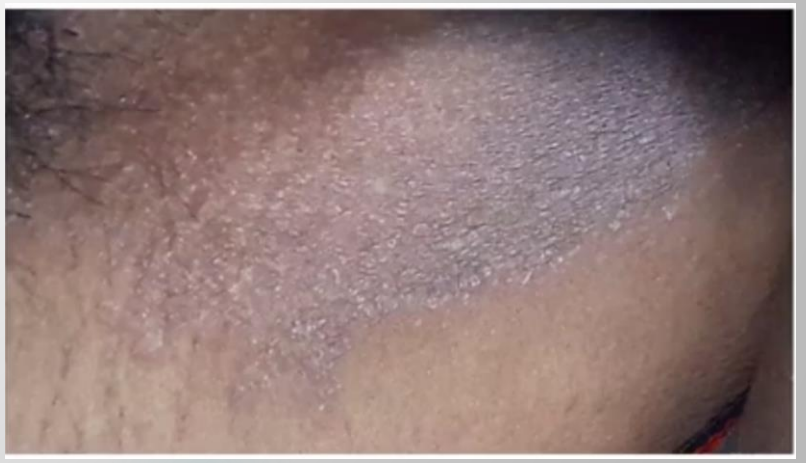
1-Clinical classification

- **Tinea Capitis-**This is fungal infection of the hair, mostly seen in children.
- Those infections in which the arthrospores are found on the hair surface are termed ectothrix infections while those with spores that develop inside the hair are called endothrix infections.
- In ectothrix infections, hair tends to break a few millimeters above the skin, unlike in endothrix infections where the hair breaks at the skin surface.
- Mainly, there is scalp scaling and hair loss.
- It may resemble dandruff.
- Inflammation is variable and may be severe with pustules and anexudative crest.



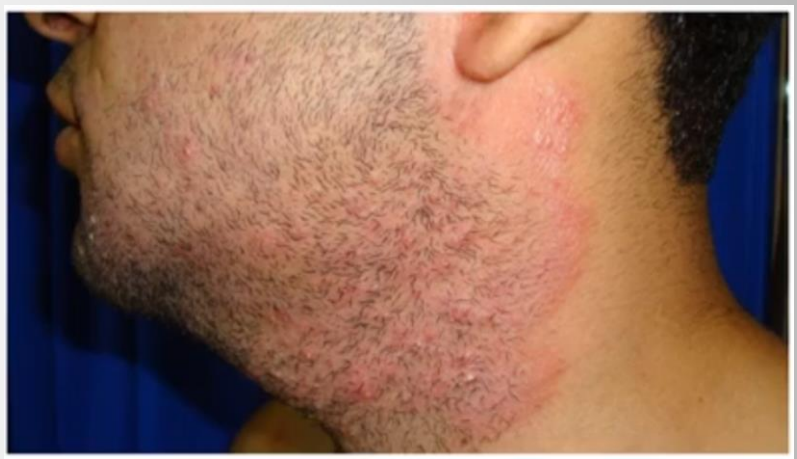
1-Clinical classification

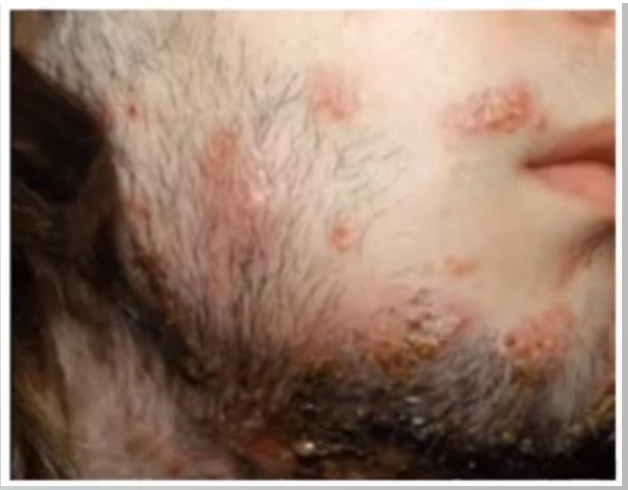
- **Tinea Cruris**
- "JOCK ITCH"-
- Infection of the groin, perineum or perianal area.
- These are erythematous lesions with central clearing and raised borders in the groinand less commonly, the scrotum.
- It is usually seen in young men.
- The type of fungus involved is usually
- **Trichophyton rubrum (Main cause)**
- **Trichophyton mentagrophytes**



1-Clinical classification

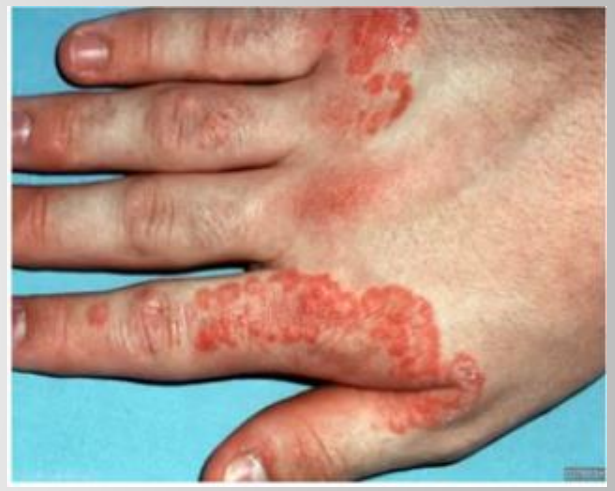
- **Tinea Barbae**
- Fungal infection of the bearded areas of the face and neck resulting into scaly plaques, pustules and vesicles.
- The most common causes are
- 1. Trichophyton mentagrophytes
- 2. T. verrucosum.
- It is endemic in parts of Southeast Asia, the South Pacific,Central America and South America

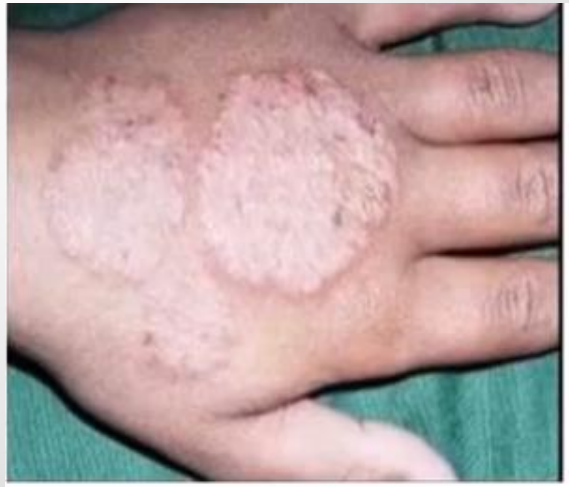


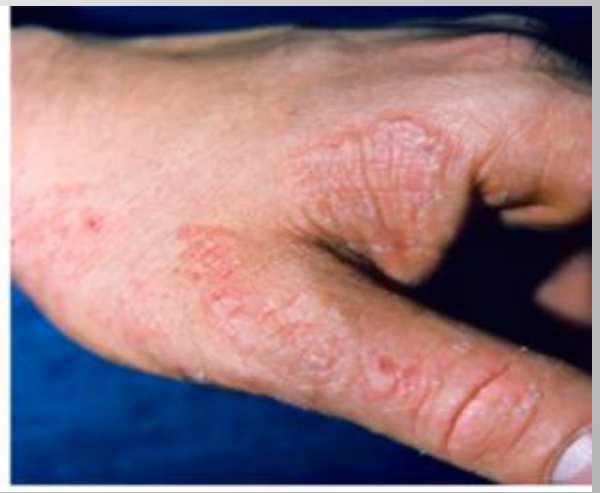


1-Clinical classification

- **Tinea Manuum (Manus)**
- Infection of the handsIt is typically more aggressive than **tinea pedis** but similar in look.
- ltching, burning, cracking, and scaling areobservable and may be transmitted by direct Contact.
- Caused by
- **1. Trichophyton rubrum,**
- **2. Trichophyton mentagrophyres**
- **3. Epidermophyton floccosum.**





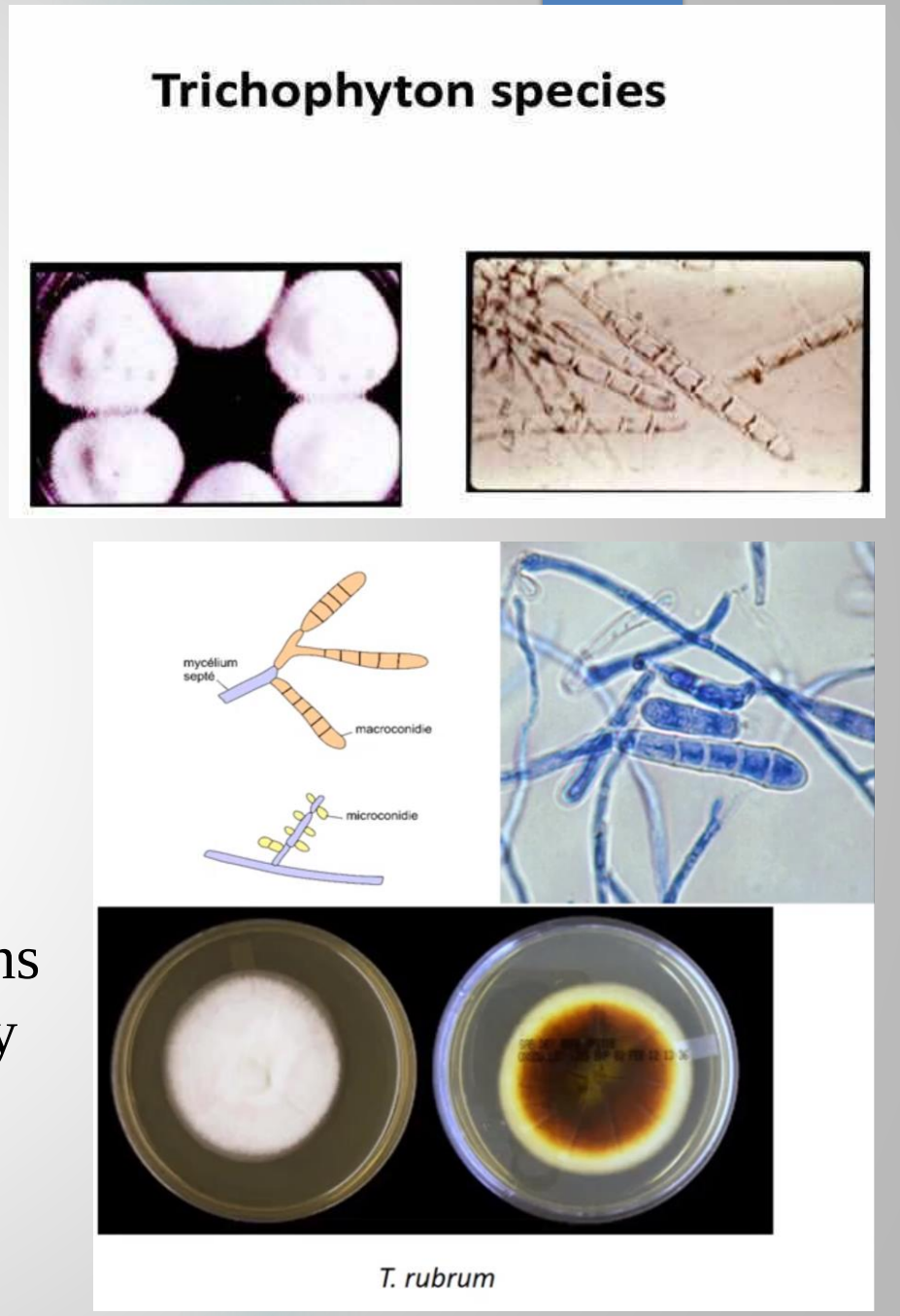


2- Etiology clssification

- • Etiology of Dermatophytes on the basis of their microscopic characteristics in to (3 Genera)
- • • ***Trichophyton***
- • • ***Microsporum***
- • • ***Epidermophyton***

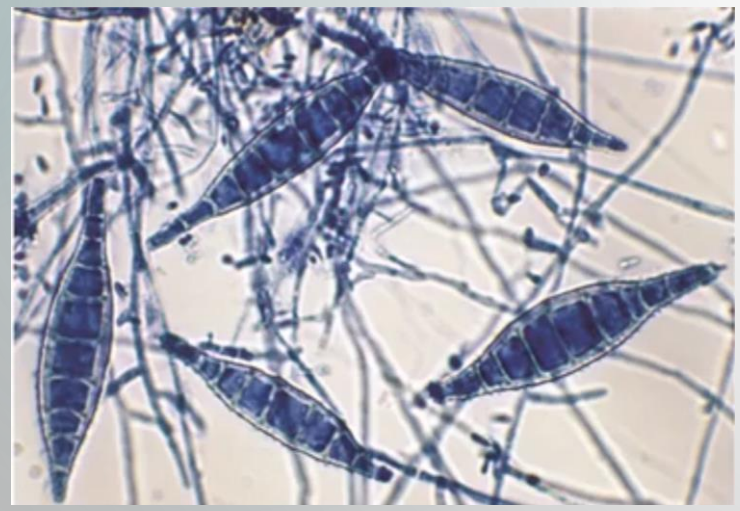
Trichophyton

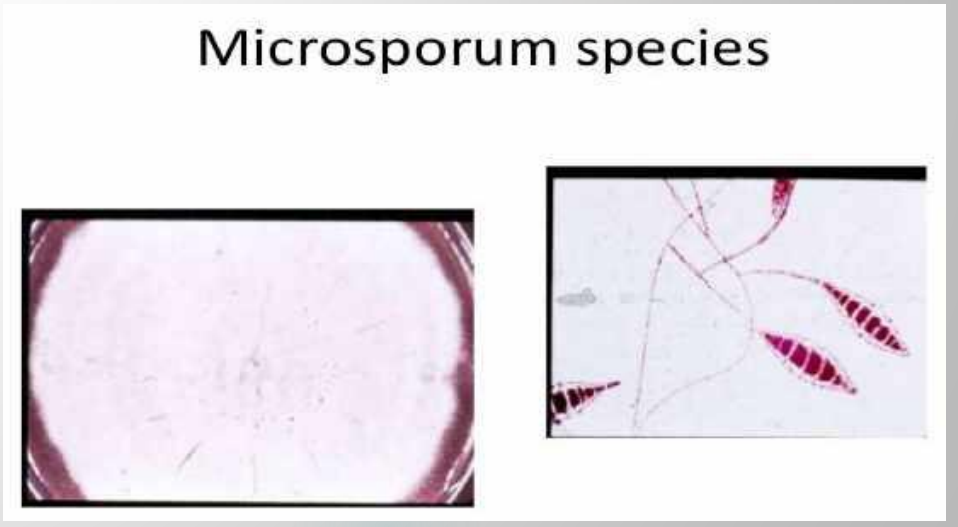
- infects skin, hair and nails.
- It takes about 2-3 weeks to grow in culture.
- It forms large conidia (macroconidia), which are smooth, thin-walled, septate, andpencil-shaped.
- The colonies have loose aerial mycelia, which produce a variety of pigments.
- **Trichophyton rubrum** may cause subcutaneous infections in immunocompromised individuals, although, this is very rare.



Microsporum

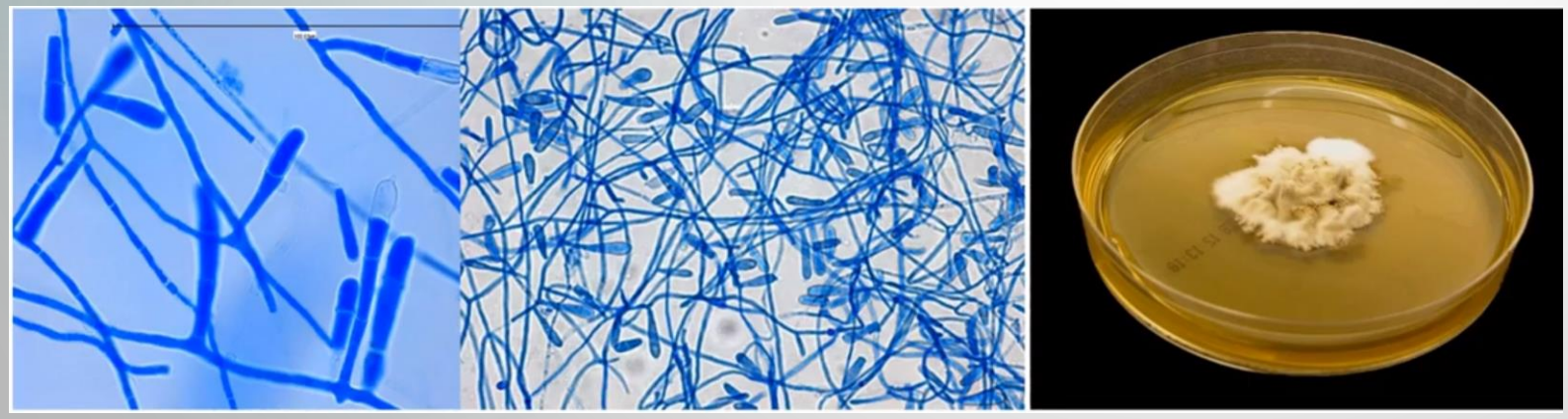
- It affects skin and hair.
- It causes infected hairs to fluoresce a bright green color when illuminated with a UVemitting wood's light.
- The loose, cottony mycelia produce macroconidia which are thick-walled, spindle shaped.

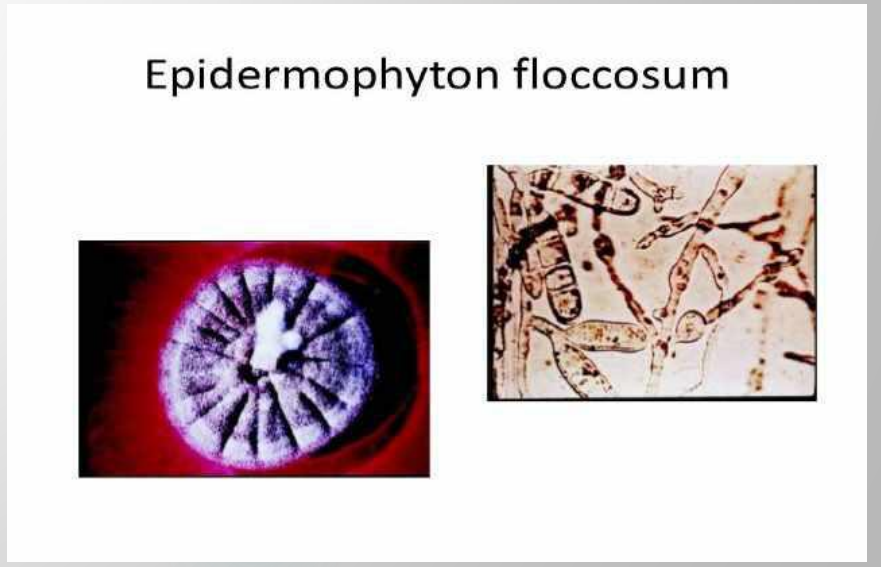




Epidermophyton

- Epidermophyton Floccosum
- This is the only one specie in this genus and it affects skin and nails.
- It appears as yellow-colored and cottony in culture.
- It is usually identified by the thick, hyphae with multiple, smooth, club shaped macroconidia.





3—Ecologyclssification

- *** Dermatophytes are classified based on their habitat as**
- **1. Anthropophilic, (humans)**
- **2. Geophilic (soil)**
- **3. Zoophilic (animals)**
- * **Anthropophilic:-** They are usually associated with humans only.
- * Transmission from man to man is by close contact or through contaminated objects.
- * Anthropophilic species, which cause the greatest number of human infections, cause relatively mild and chronic infections in humans.
- * They produce few conidia in culture, and may be difficult to eradicate.

Classitication

- **Geophilic**
- They are usually found in the soil, and are transmitted to man by direct exposure.
- This is less adapted to human hosts; they produce more acute inflammatory infections that tend to resolve more quickly.
- This also occurs in zoophilic cases.
- The most common geophilic species causing human infections is Microsporum gypseum.
- **Zoophilic**
- They are usually associated with animals and transmission to man is by close contactwith animals (cats, dogs, cows etc) or with contaminated products.

Thank you