Herpesviridae

Presented by:

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**The herpesvirus family contains several of the most important human viral pathogens.

**Clinically, the herpesviruses exhibit a spectrum of diseases.

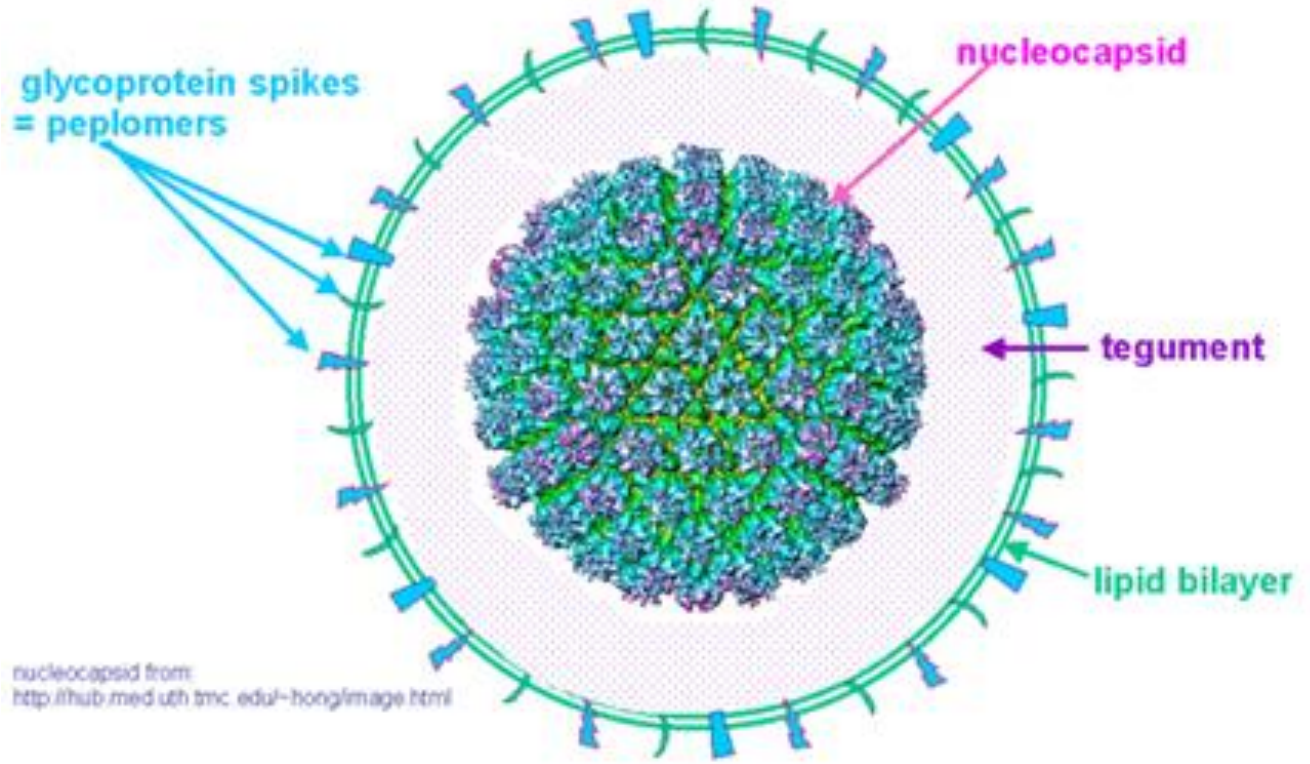
**Some have a wide host-cell range, and others have a narrow host-cell range.

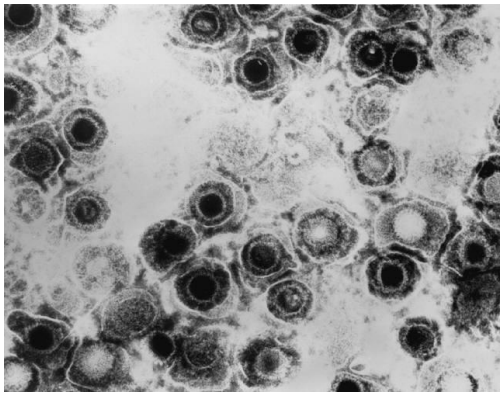
**their ability to establish lifelong persistent infections.

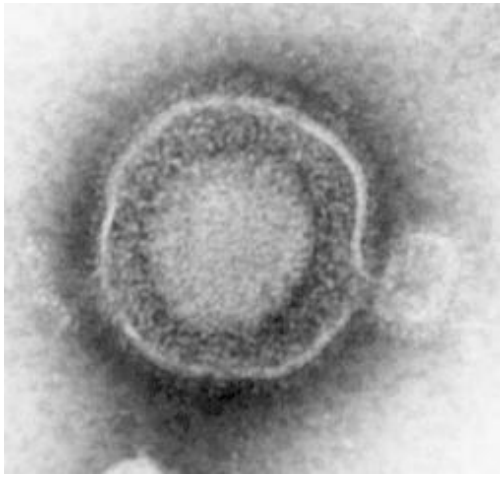
**The herpesviruses that commonly infect humans include herpes Simplex virus types 1 and 2 , Varicella-zoster virus, cytomegalovirus (CMV), Epstein-Barr virus (EBV), herpesviruses 6 and 7, and herpesvirus 8 (Kaposi sarcoma-associated herpesvirus [KSHV]).

Structure and Composition

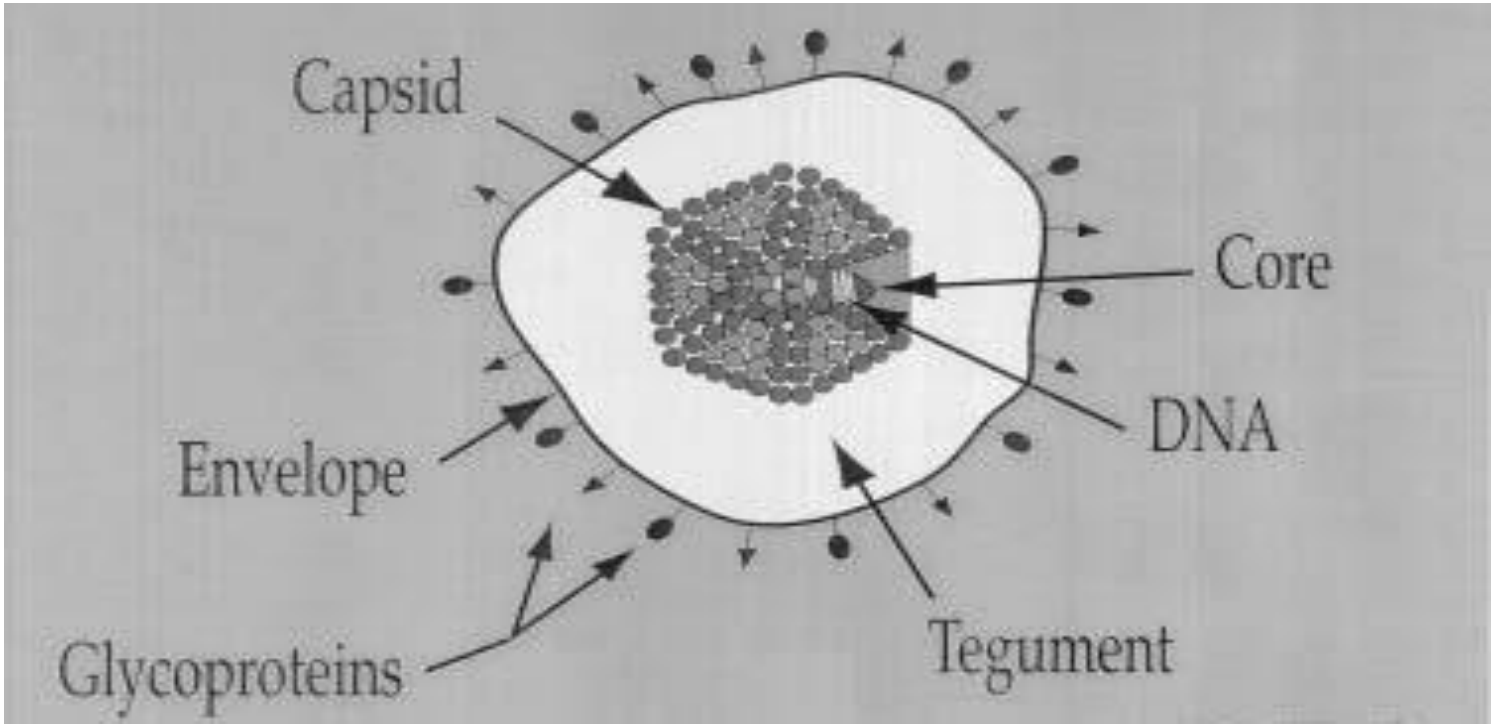
Herpesviruses are large viruses, Ds-DNA, envelope, **spikes**







The virions of different members of the Herpesviridae family are indistinguishable and consist of four distinct components: the core, capsid, tegument, and envelope (Fig. 1) (1). The core contains a double-stranded DNA genome arranged in an unusual torus shape that is located inside an icosadeltahedral capsid that is approx 100 nm in size and contains 162 capsomeres (2). Located between the capsid and the viral envelope is an amorphous structure termed the tegument that contains numerous proteins. The tegument structure is generally asymmetrical. The outermost structure of the herpes virion is the envelope, which is derived from cell nuclear membranes and contains several viral glycoproteins. The size of mature herpesviruses ranges from 120 to 300 nm owing to differences in the size of the individual viral teguments.



Schematic drawing of a typical herpesvirus particle.

The life cycle of all herpesviruses in their natural host can be divided into lytic (resulting in the production of infectious progeny)

and latent (dormant) infections.

During a lytic infection the virus is replicated and newly synthesized particles are released into the surrounding medium.

During a latent infection viral replication is suppressed.

The establishment of viral latency is a hallmark of all known herpesviruses.

Herpesviridae- Classification

β herpesviruses

- Slowly replicating
- Restricted host range
- Infected cells enlarge (*cytomegalia*)
- Latency established in secretory glands, lymphoreticular cells, kidneys

Cytomegalovirus (CMV)
Human Herpesvirus-6 and 7 (HHV-6/HHV-7)

Herpesviridae- Classification

γ herpesviruses

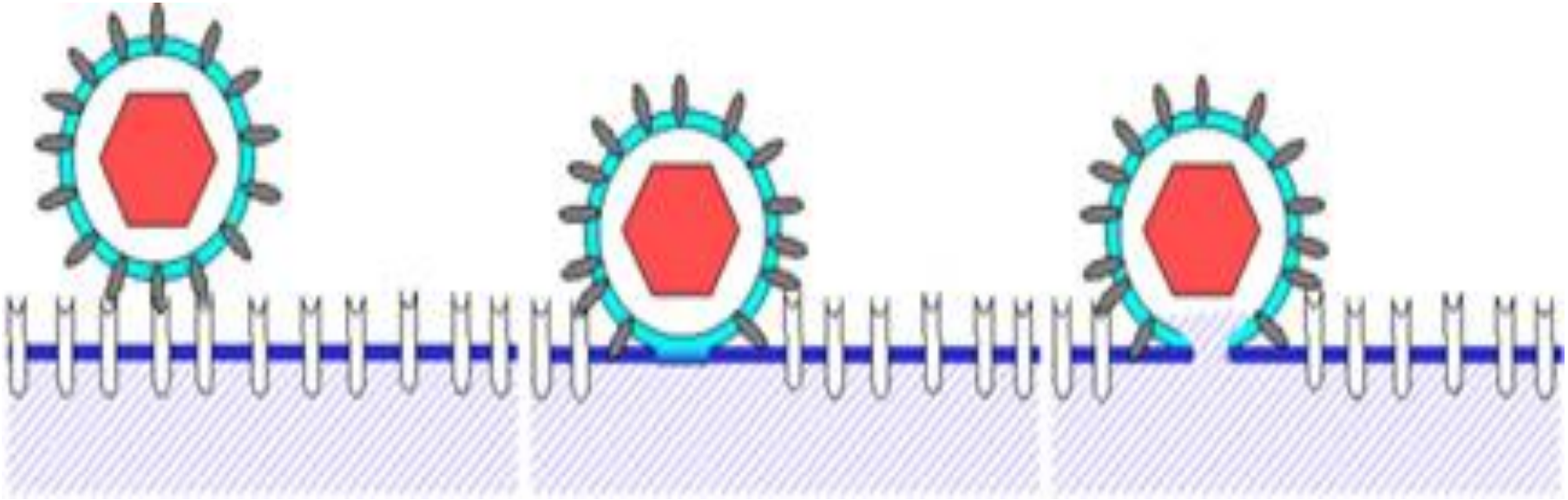
- Replicate poorly
- Highly restricted host range
- Latency established in lymphoid tissue
  (T-cell or B-cell specific)

Epstein-Barr Virus (EBV), a B-cell transforming virus
Human Herpesvirus-8 (HHV-8, KSHV)

Herpesviridae- Replication

ADSORPTION

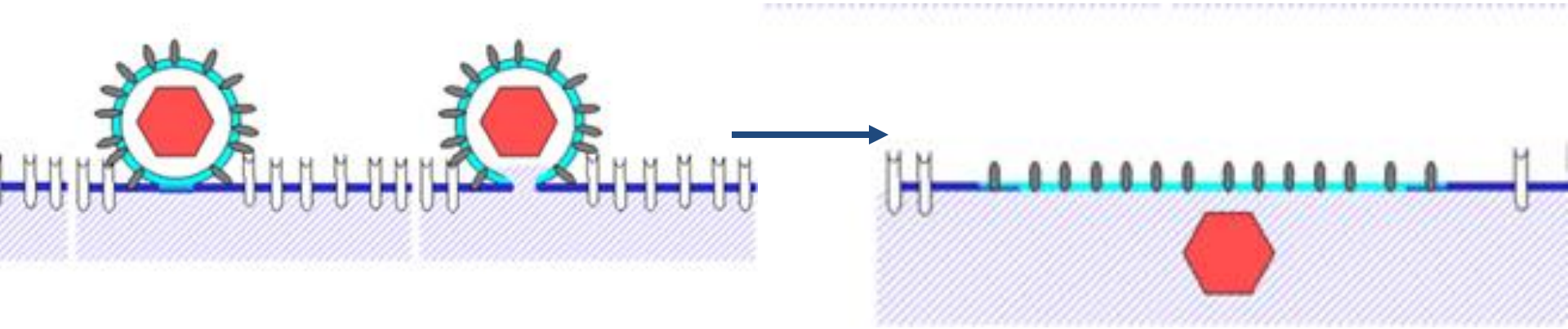
Envelope glycoproteins are required for binding and penetration



Herpesviridae- Replication

PENETRATION

The nucleocapsid enters the cell by direct membrane fusion with the cell plasma membrane

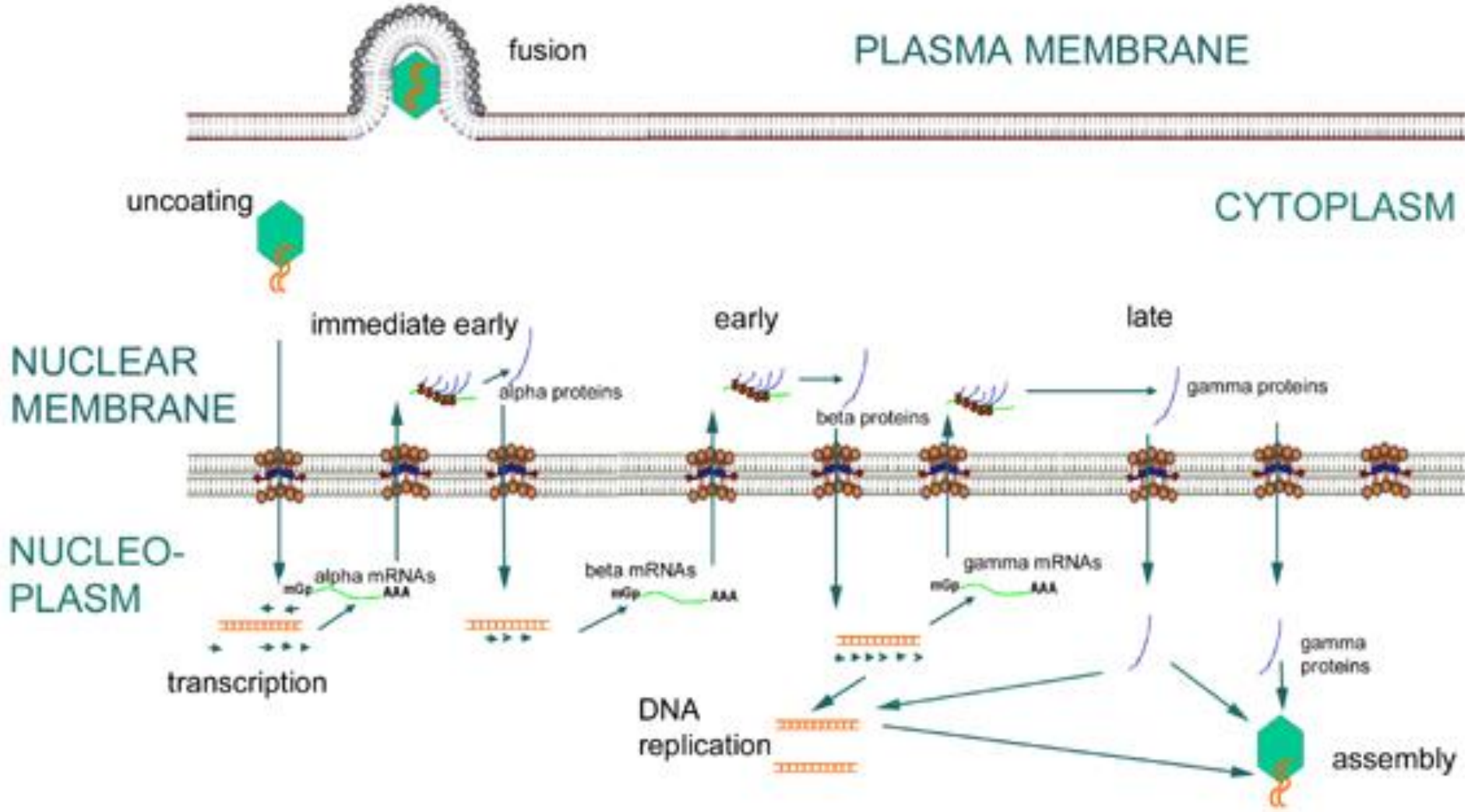


Capsids are transported to the nucleus

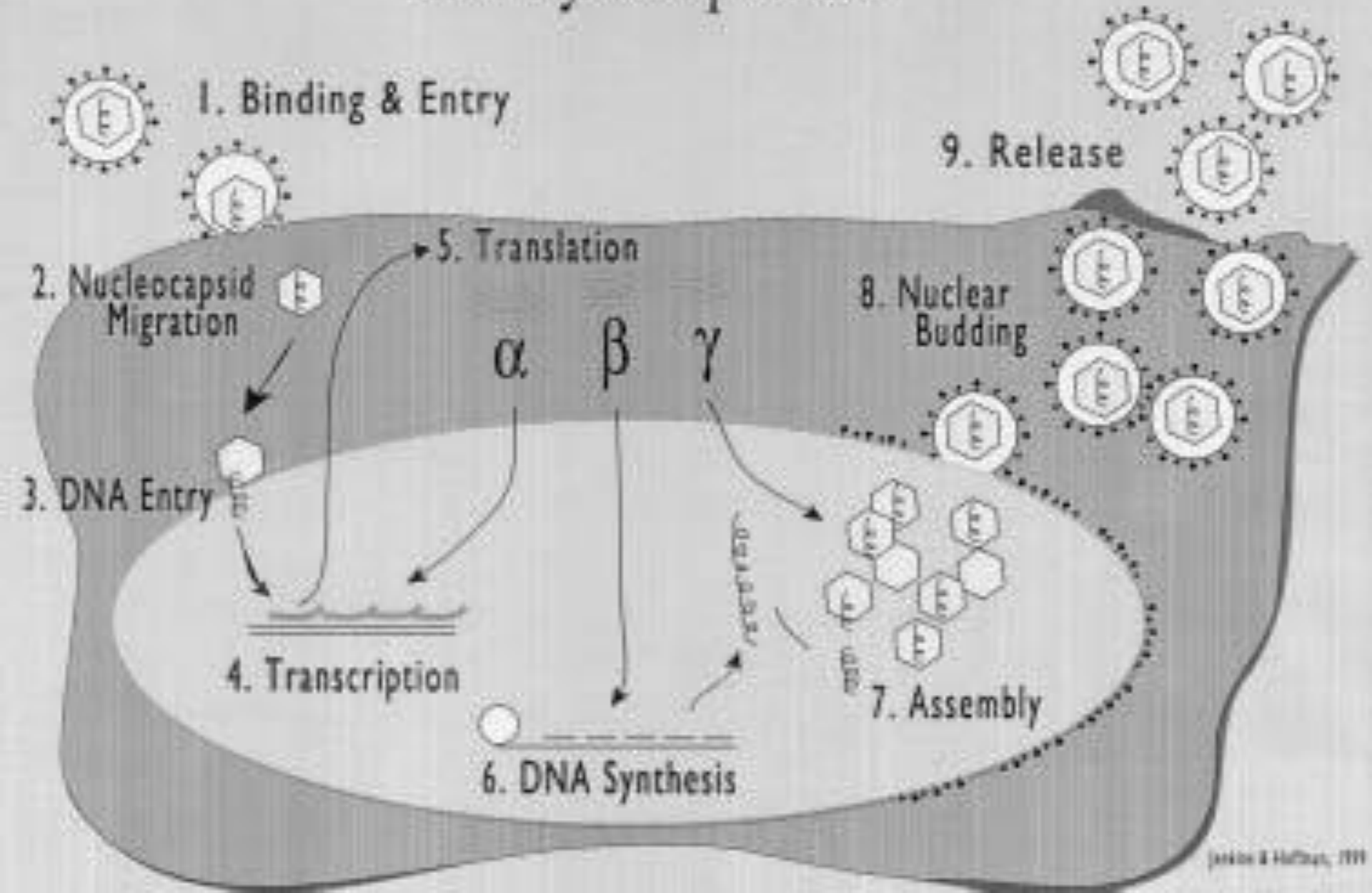
DNA passes into the nucleus, probably via nuclear pores

Herpesviridae- Replication

Herpesvirus replication is a carefully regulated, multi-step process



HSV Lytic Replication

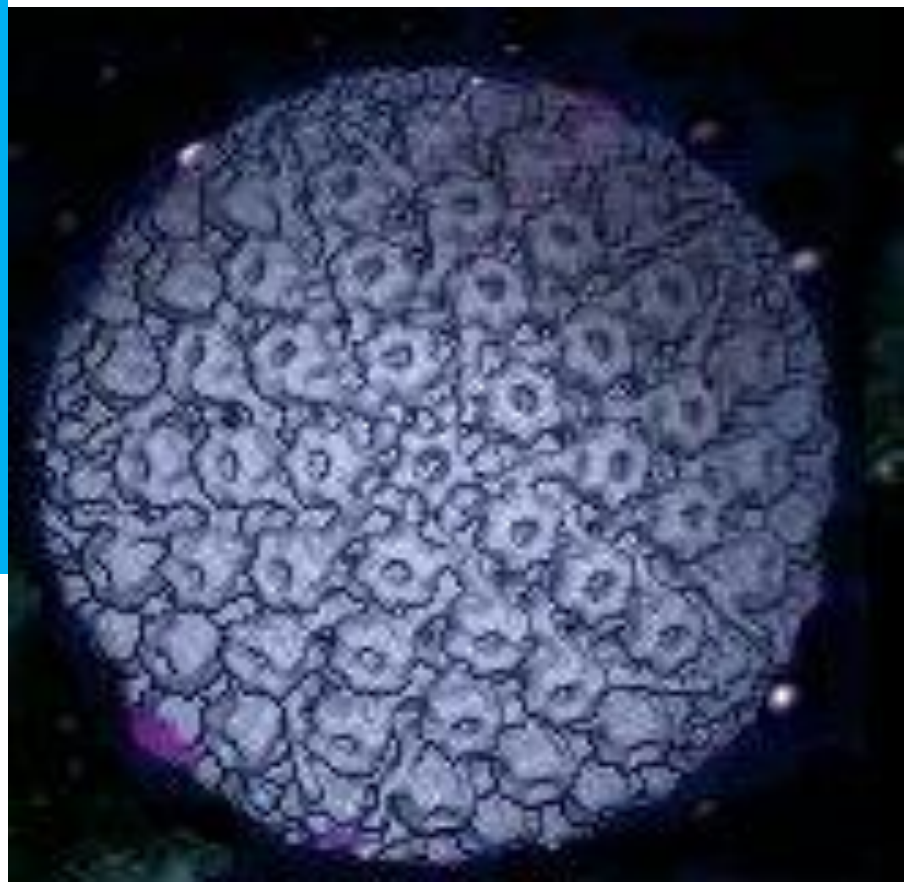


Herpesviridae- Replication

Virus Assembly

Assembly of the *nucleocasid* occurs in the nucleus

The nucleocapsid “buds” through intracellular membranes ultimately taking up tegument proteins beneath the envelope



Herpesviridae- Infection and Disease

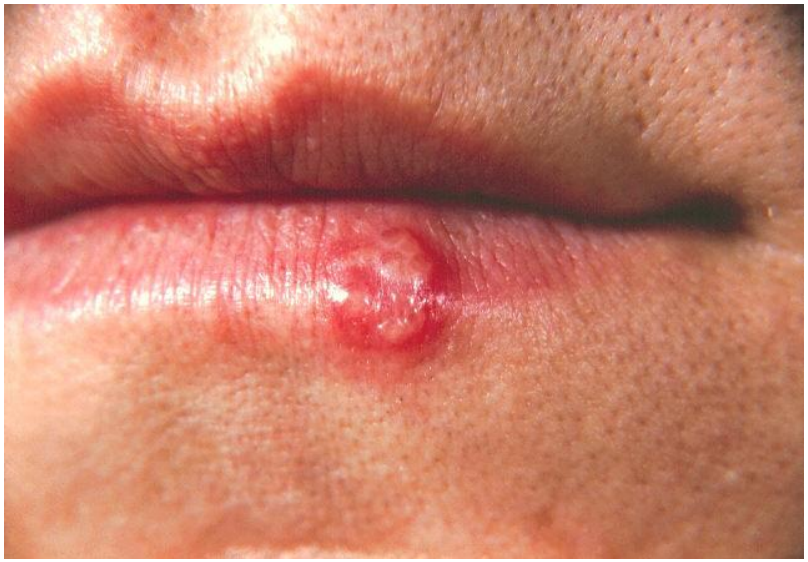
Designation	Common Name	Subfamily	Associated Diseases
HHV-1	HSV-1	Alpha	Oral Herpes (cold sore), Genital Herpes
HHV-2	HSV-2	Alpha	Genital Herpes
HHV-3	VZV	Alpha	Chicken Pox, Shingles
HHV-4	EBV	Gamma	Mononudeosis, Lymphoma, Carcinoma
HHV-5	CMV	Beta	Mononucleosis, Retinitis, Transplant Rejection
HHV-6	HHV-6	Beta	Roseola infantum, Mononucleosis syndrome, Chronic fatigue syndrome, Multiple Sclerosis?
HHV-7	HHV-7	Beta	Roseola infantum?, Mononucleosis syndrome?
HHV-8	KSHV	Gamma	Kaposis Sarcoma

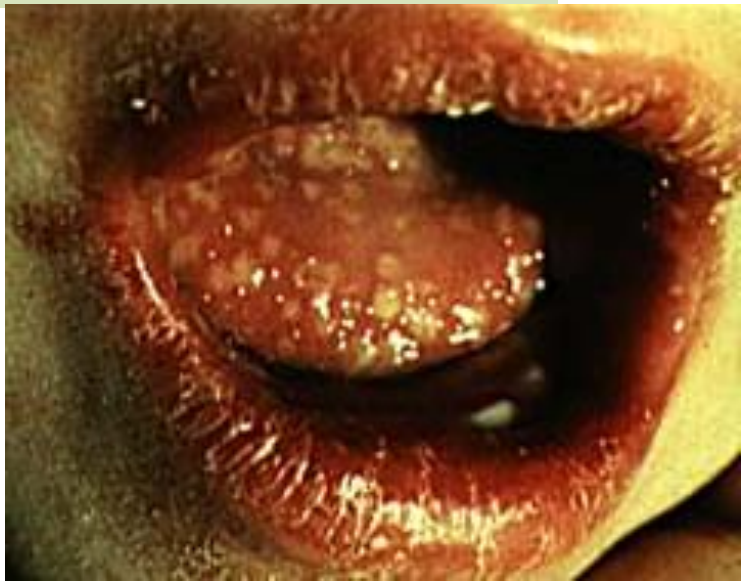
HSV- Pathology

The virus replicates in the epithelial tissue yielding a characteristic “fever blister” or “cold sore”

The fluid in this blister is full of infectious virus

The blister ulcerates and forms a crusted lesion that heals without a scar





HSV- Reactivation

Several agents may trigger recurrence

- stress
- exposure to strong sunlight
- fever

The virus can travels back down the nerve axon and arrives at the mucosa that was initially infected

Vesicles containing infectious virus are formed on the muscosa and the virus spreads

Recurrent infections are usually less pronounced than the primary infection and resolve more rapidly

HSV Infections

Oral Herpes

Both HSV-1 and HSV-2

Genital Herpes

Primarily HSV-2 (10% cases HSV-1)

Involve a transient viremia (fever, myalgia, glandular inflammation in the groin area)

Secondary infections are frequently less severe

Herpes Keratitis

An infection of the eye

Primarily HSV-1

Sometimes recurrent

Leading cause of corneal blindness in the US

HSV- Treatment

Nucleoside Analogs

Acyclovir (Zovirax®)
Valacyclovir (Valtrex®; L-valyl ester of acyclovir)
Famciclovir (Famvir®; diacetyl ester of 6-deoxy penciclovir)

All suffer from the appearance of resistant HSV mutants

Fortunately, the mutant strains are less virulent

The drugs are ineffective against latent virus

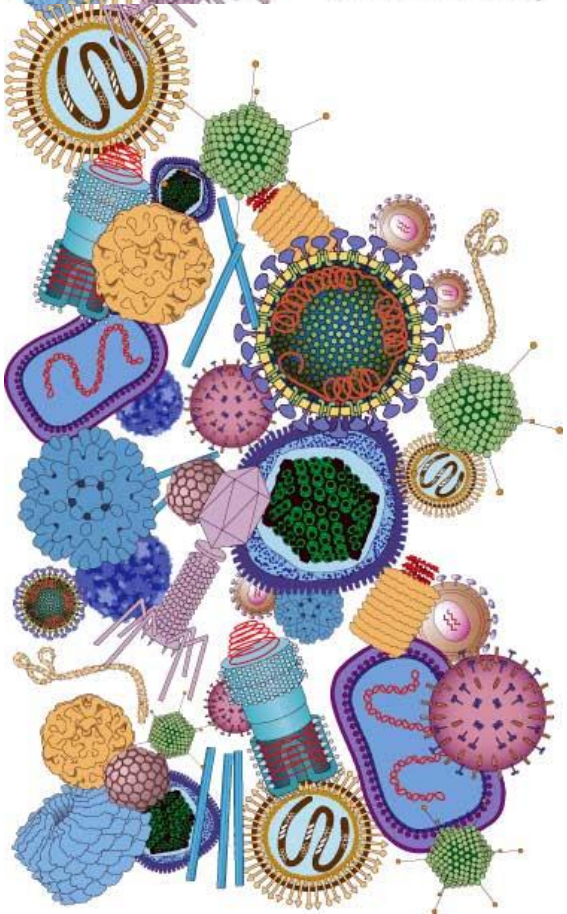
SUMMARY

#The human herpesviruses induce a variety of illnesses ranging from asymptomatic to life-threatening infections and cancer.

#The majority of these viruses are acquired during childhood and persist for life.

#The ability of the herpesviruses to establish and maintain a latent infection adds an additional layer of complexity to the viruses' life cycle and complicates all attempts to eradicate the virus from infected individuals.

#While much has been learned about the human herpesviruses over the last 30 yr, there is still much more to be learned before they are no longer a serious health threat.



Questions?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Herpesviridae

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Varicella-Zoster Virus (VZV)

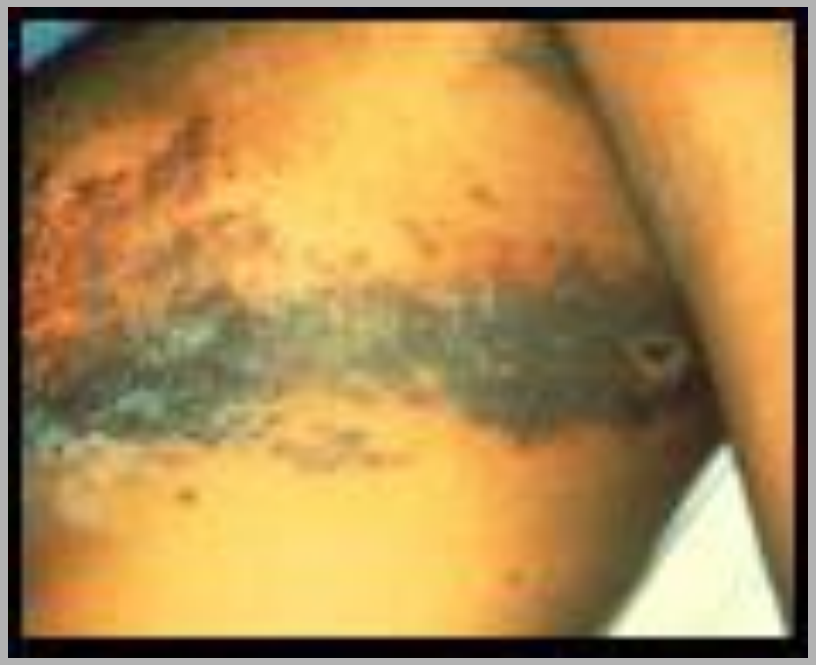
- Initial infection usually in childhood with Varicella virus ***Chicken Pox,*** is a mild,
- characterized clinically by a generalized vesicular eruption of the skin and mucous membranes.
- The disease may be severe in adults and in immune-compromised individuals.

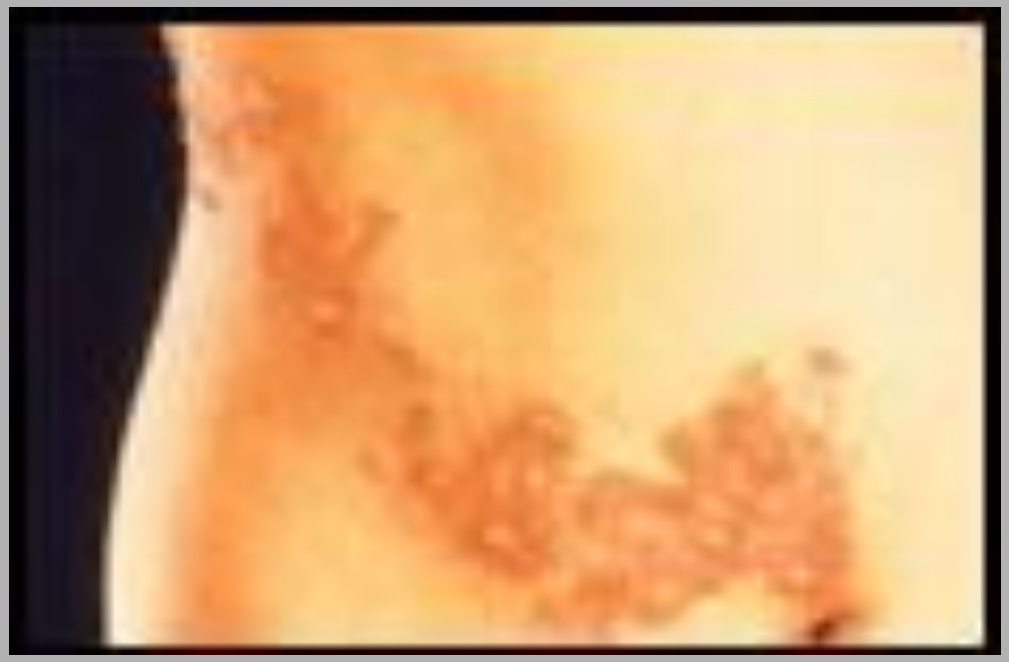
- Zoster (shingles) is a sporadic, incapacitating disease of elderly or immune-compromised individuals that is characterized by pain and a rash limited in distribution to the skin innervated by a single sensory ganglion.

Years or decades later, the virus (Herpes zoster) may reactivate -> ***Shingles***

Varicella-Zoster Virus (VZV)

Both diseases are caused by the same virus. Whereas varicella is the acute disease that follows primary contact with the virus, zoster is the response of the partially immune host to reactivation of varicella virus present in latent form in neurons in sensory ganglia.





Rash along dermatomes

Pathogenesis and Pathology

A. Varicella

The route of infection is the mucosa of the upper respiratory tract or the conjunctiva . After initial replication in regional lymph nodes,
primary viremia spreads virus and leads to replication in the liver and spleen.
Secondary viremia involving infected mononuclear cells transports virus to the skin, where the typical rash develops. Swelling of epithelial cells, ballooning degeneration, and the accumulation of tissue fluids result in vesicle formation.
Varicella-zoster virus replication and spread are limited by host humoral and cellular immune responses.

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VZV- Pathology

B. Zoster

The skin lesions of zoster are histopathologically identical to those of varicella. There is also an acute inflammation of the sensory nerves and ganglia. Often only a single ganglion may be involved. As a rule, the distribution of lesions in the skin corresponds closely to the areas of innervation from an individual dorsal root ganglion.

It is not clear what triggers reactivation of latent varicella- zoster virus infections in ganglia.

It is believed that waning immunity allows viral replication to occur in a ganglion, causing intense inflammation and pain. Virus travels down the nerve to the skin and induces vesicle formation.

#Cell-mediated immunity

#Reactivations are sporadic and recur infrequently.

Clinical Findings

A. Varicella

-Subclinical varicella is unusual.

-Incubation period of typical disease is 10–21 days.

-Complications are rare in normal children, and the mortality rate is very low.

Immunity

#Varicella and zoster viruses are identical,
the two diseases being the result of differing host responses. Previous infection with varicella is believed to confer lifelong immunity to varicella. Antibodies induced by varicella vaccine persist for at least 20 years.

##Zoster occurs in the presence of neutralizing antibody to varicella. Increases in varicella antibody titer may occur in persons with HSV infections.

#The development of varicella-zoster virus-specific cell-mediated immunity is important in recovery from both varicella and zoster.

Laboratory Diagnosis

Rapid diagnostic procedures are clinically useful for varicella- zoster virus. **PCR** assays are preferred for **sensitivity, specificity, and rapidity**. Varicella-zoster virus DNA can be detected in saliva in many patients, including those with zoster without rash. Viral DNA can be detected in vesicle fluid, skin scrapings, and biopsy material.

In stained smears of scrapings or swabs of the base of vesicles (Tzanck smear), multinucleated giant cells. Varicella-zoster virus in vesicle fluid is very labile, and cell cultures should be inoculated promptly.

A rise in specific antibody titer can be detected in the patient's serum by various tests, including **fluorescent antibody** and **enzyme immunoassay**. The choice of assay to use depends on the purpose of the test and the laboratory facilities available.

Epidemiology

- Varicella and zoster occur worldwide.
- Varicella (chickenpox) is highly communicable and is a common epidemic disease of childhood
- Zoster occurs sporadically, chiefly in adults and without seasonal prevalence.
- About 10–20% of adults will experience at least one zoster attack during their lifetime, usually after the age of 50 years.
- Varicella spreads readily by airborne droplets and by direct contact.
- Zoster patients can be the source of varicella in susceptible children, perhaps because viral DNA is often present in their saliva.

Treatment

Several antiviral compounds provide effective therapy for varicella, including acyclovir, valacyclovir, famciclovir, and foscarnet. Acyclovir can prevent the development of systemic disease in varicella-infected immunosuppressed patients and can halt the progression of zoster in adults

Prevention and Control

A **live attenuated** varicella vaccine was approved in 1995 for general use in the United States.

A similar vaccine has been used successfully in Japan for about 30 years.

A zoster (shingles) vaccine was licensed in the United States in 2006.

The zoster vaccine is recommended for those with chronic medical conditions and for persons older than 60 years of age.


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Herpesviridae
CMV. EBV, Herpes 6,7,8

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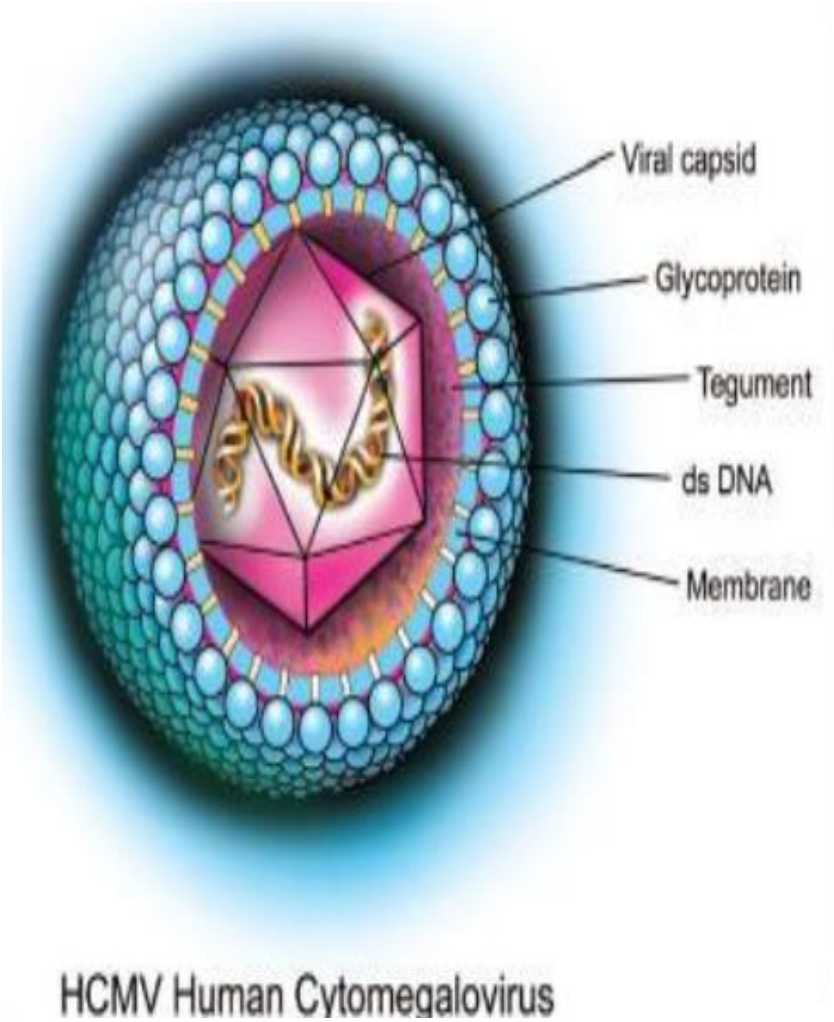
Cytomegalovirus

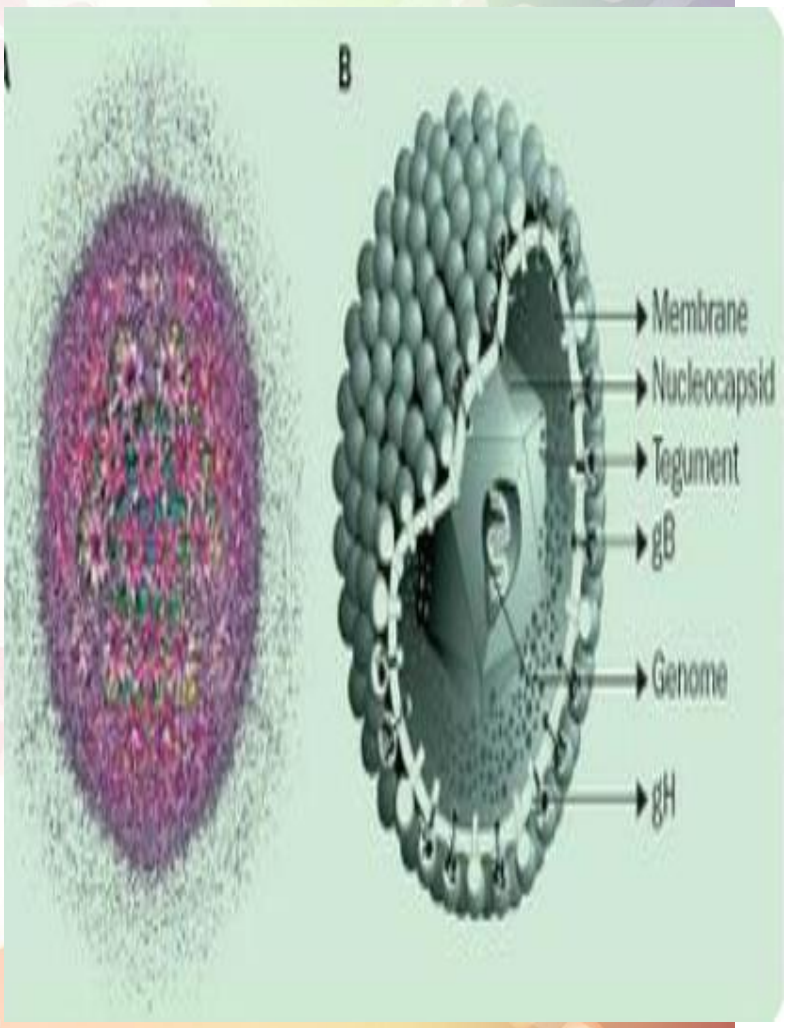
- ***Cytomegalovirus*** (from the Greek *cyto-*, "cell", and *-megalo-*, "large") is a herpes viral genus of the Herpesviruses group: in humans it is commonly known as HCMV or **Human Herpesvirus 5** (HHV-5).
- CMV belongs to the *Betaherpesvirinae* subfamily of *Herpesviridae*,

- HCMV infections are frequently associated with salivary glands, though they may be found throughout the body. HCMV infection can also be life threatening for patients who are immunocompromised (*e.g.* patients with HIV, organ transplant recipients, or neonates)



HCMV Human Cytomegalovirus

Properties of Virus.



- Spherical in shape.
- 150-200 nm in diameter.
- Genome ds DNA.
- Species specific.
- Cell type specific.

- **Transmission**
  - Transmission of HCMV occurs from person to person through bodily fluids. Infection requires close, intimate contact with a person excreting the virus in their saliva, urine, or other bodily fluids. CMV can be sexually transmitted and can also be transmitted via breast milk, transplanted organs, and rarely from blood transfusions.

- The name for the classic cytomegalic inclusion disease derives from the propensity for massive enlargement of CMV-infected cells. CMV poses an important **public health problem** because of its high frequency of congenital infections, which may lead to **severe congenital anomalies**.

Pathogenesis and Pathology.

- **Pathogenesis and Pathology.**
  • Spreads because of close contact between human to human.
  • Isolated from Lungs, Liver, oesophagus, colon kidney, Monocytes,
  T and B lymphocytes,
  Salivary glands
  Cell mediate immunity is depressed.
- Incubation period 4 – 8 weeks
- Manifest as Infectious mononucleosis syndrome

Pathogenesis

- Sub clinical infections are common
- Latent infections are common
- Virus are shed from Pharynx, and excreted in urine.
- Produce severe infections in Immunosuppressed patients.
- Pneumonia most important complication.

Congenital and Perinatal Infections.

- 1% are infected congenitally in USA.
  - Produce birth defects,
  - 1% maternal transmission,
  - Genital tract helps in spread during delivery.
  - Blood transfusion can spread to virus to recipient.

Part of the To RCH panel

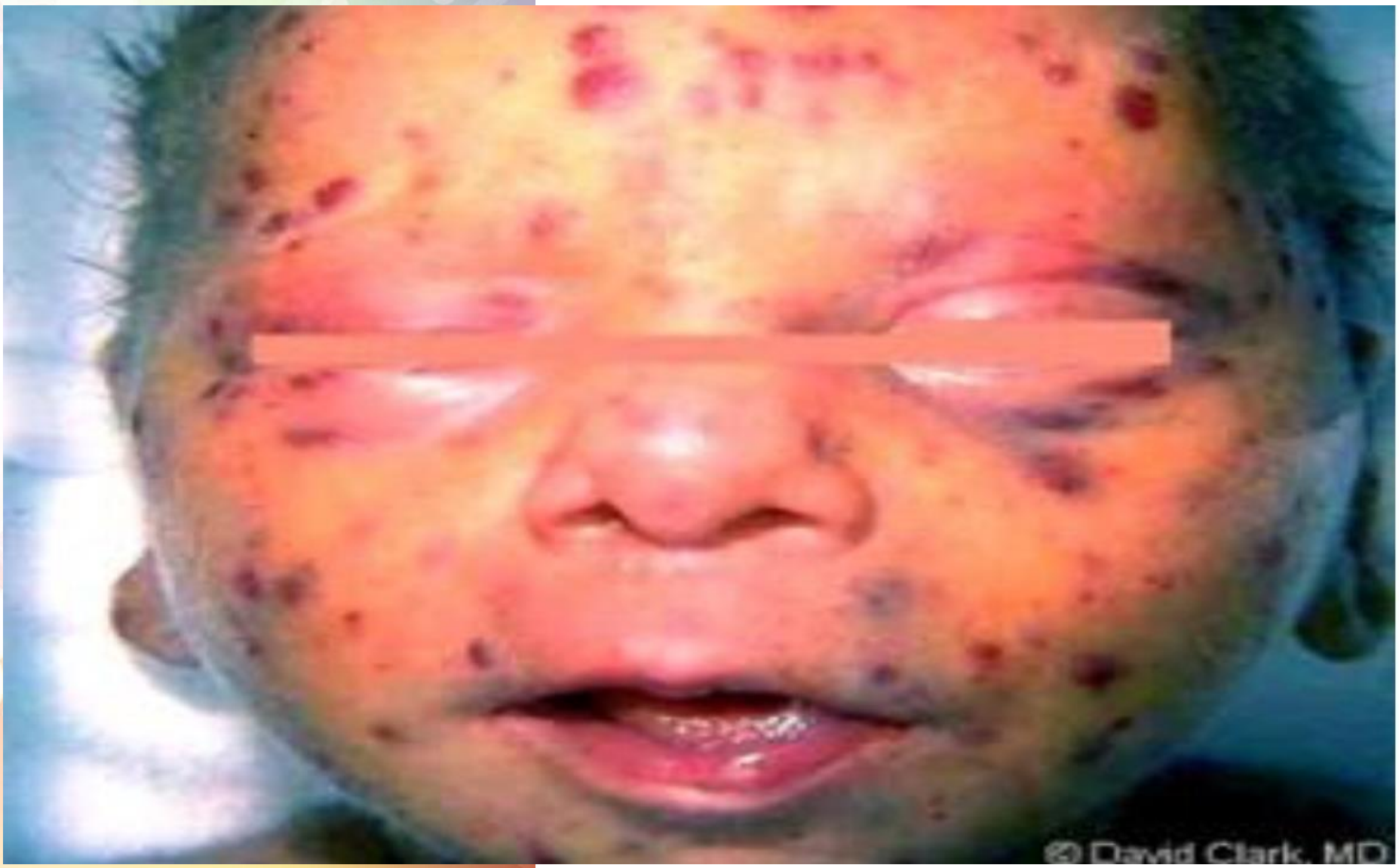
- • HCMV is one of the TORCH infections that lead to congenital abnormalities. These are: toxoplasmosis, rubella, herpes simplex, and cytomegalovirus. Congenital HCMV infection occurs when the mother suffers a primary infection (or reactivation) during pregnancy.
  • HCMV is one of the TORCH infections that lead to congenital abnormalities. These are: toxoplasmosis, rubella, herpes simplex, and cytomegalovirus. Congenital HCMV infection occurs when the mother suffers a primary infection (or reactivation) during pregnancy.

Clinical Findings

- Asymptomatic in majority,
  - Infectious Mononucleosis.
  - Fever, Myalgia and Liver dysfunction,
  - In Immune compromised,

  Increased Morbidity, and Mortality,
  Pneumonia, Bone marrow transplantation,
  Disseminate Disease in AIDS.
  Gastro Enteritis and Chorioretinitis,lead to Blindness

A New Born Child with CMV



Laboratory Diagnosis.

- Cell cultures Too slow,
  - PCR, Replication of virus,
  - Isolation of virus- Human Fibroblasts,
  - Serology,

  Ig M current infection

  Ig G Potential reactivation, past infection

Laboratory Tests

- The enzyme-linked immunosorbent assay (or ELISA) is the most commonly available serologic test for measuring antibody to CMV. The result can be used to determine if acute infection, prior infection, or passively acquired maternal antibody in an infant is present. Other tests include various fluorescence assays, indirect haemagglutination, (PCR) and latex agglutination.

Epidemiology.

- Endemic world wide.
  - Developing countries 90%.
  - Person to Person spread.
  - Urine, Saliva, Semen,
  - Breast Milk Cervical secretions,
  - Mother to Child spread congenital infections.
  - Risk with Blood transfusions 1-5%

Epstein Barr Virus
DNA Group ds DNA

Commonly Called as Kissing Disease

Properties of Virus.

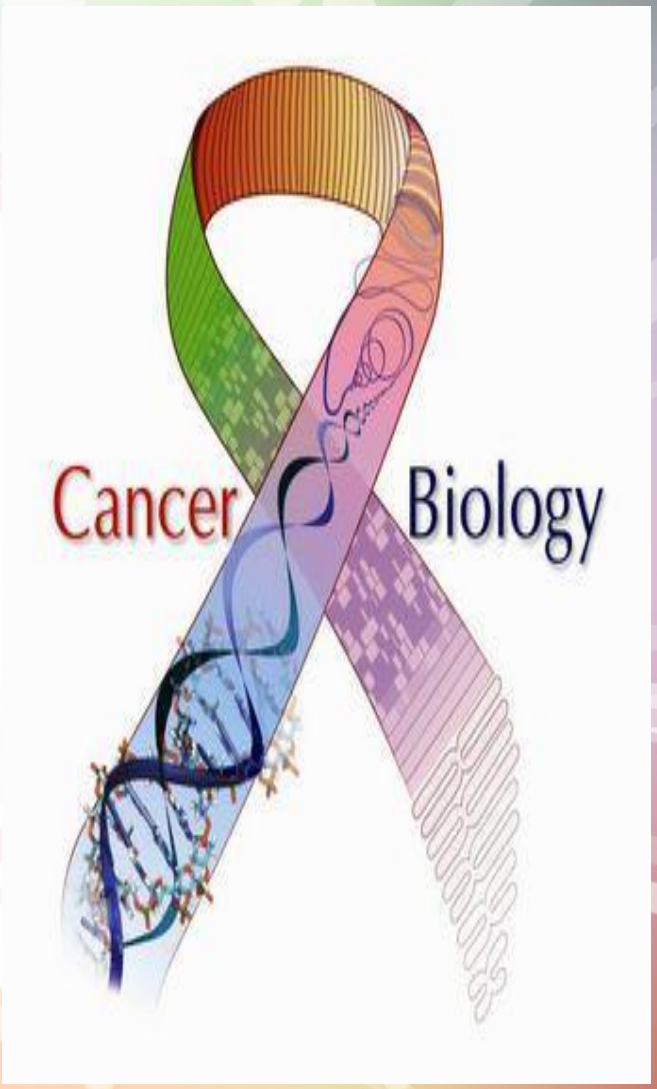
- Size 150 -200 nm
- Ds DNA
- Two antigenic types EBV1,and EBV2.
- Targets B lymphocytes.
- Causes Immortalization of B lymphocytes.
- Binds on B lymphocytes,
- Causes viral persistence.

- EBV **directly enters a latent state in the lymphocyte** without undergoing a period of complete viral replication.
- -The hallmarks of latency are viral persistence, restricted virus expression, and the potential for reactivation and lytic replication.

Herpes Group Of Viruses.

- Causative Agent
  1. Acute Infections Mononucleosis.
  2. Nasopharyngeal Carcinoma.
  3.Lymphoma.
  4.Burkitts Lymphoma.
  5.Lymphoproliferative disorders.

- (Immunosuppressed)
  Transplant recipients
  AIDS Patients

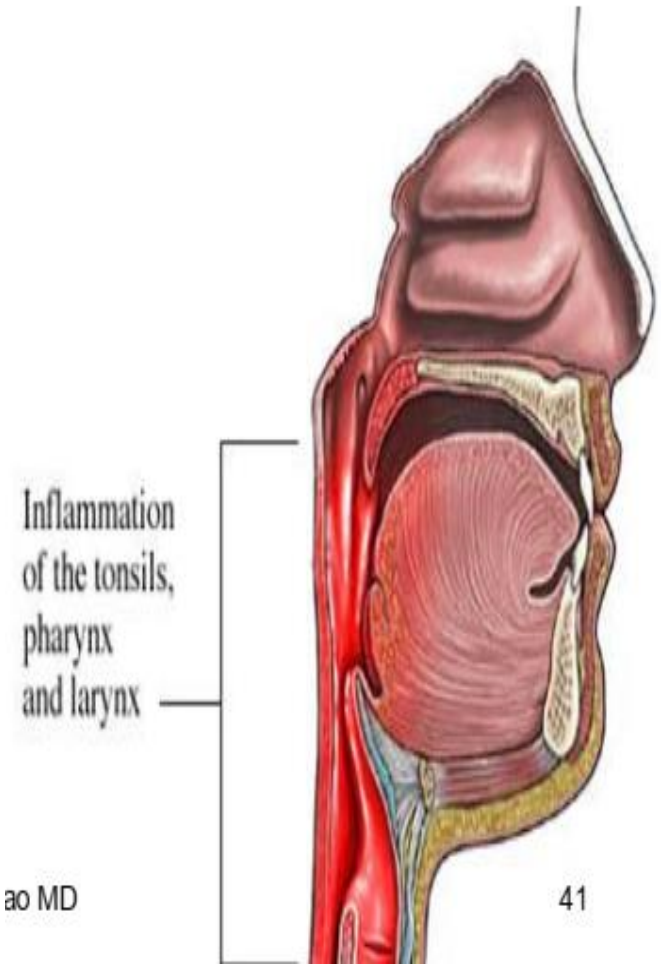


The kiss of cancer

- Named after its discoverers, Epstein–Barr virus (EBV) was first isolated in 1964 from patients with hematologic pathology.
- It is a lymphocytic human herpesvirusthat is carried, like some other pathogenic herpesviruses, by the majority of the world's population as a persistent, latent contagious agent.
- Many children become infected with EBV but do not usually show symptoms.
- Causes lifelong, persistent infections – majority are benign

EBV replication occurs in

- Epithelial cells of Oropharynx.
- Parotid Gland.
- Uterine cervix
- Nasopharynx



Symptoms

- Incubation 30-50Days
  Head ache, fever, Malaise Fatigue. Sore throat. Enlargement of Lymph nodes and Spleen
  Hepatitis,Lymphocytosis.Large Large Atypical T Lymphocytes

- Other symptoms include:
  - Rash
  - Muscle aches
  - Abdominal pain
  - Occasional jaundice

TUMOURS

- Burkett's Lymphoma
- Nasopharyngeal Carcinoma.
- Hodgkin's Diseases
- Other Lymphomas.
- Complicates immune suppressed

- Diagnosis
  - Isolation of infectious virus from peripheral blood mononuclear cells is the most definitive method of diagnosing primary infection. However special cell culture techniques need to be applied. Not often performed.

Laboratory Diagnosis
- Nucleic Acid Hybridization.
- Saliva, Peripheral Blood Cells.
- PCR

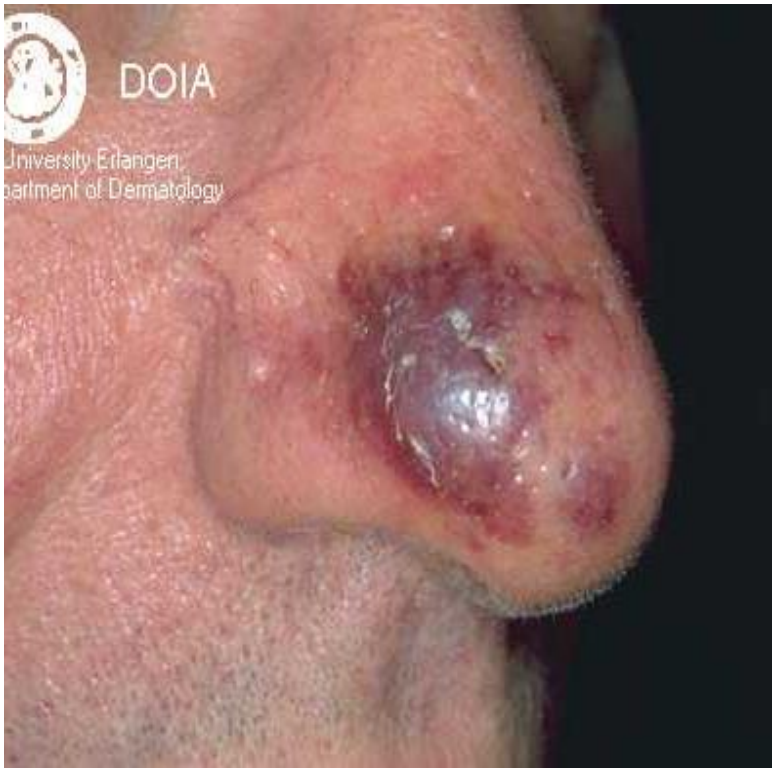
Serology
ELISA Ig M , Ig G
HeterophileAgglutination tests
Paul Bunnell Test uses sheep cells.
Titers are estimated

Human Herpes Viruses 6 &7

- T-lymphotropic viruses of world wide distribution
  - Found in most of adults saliva
  - Infection is acquired by the age of 2 years
  - HHV-6 infects T cells, epithelial cells, NK cells & monocytes
  - Causes childhood disease – Exanthem subitum (Roseola infantum or Sixth disease)
  - Primary infection in adults can result in hepatitis, mononucleosis,
  - No disease has been established with HHV-7

Human Herpesvirus 8

- Human Herpesvirus 8 (HHV-8) , or **Kaposi Sarcoma Herpes Virus** (KSHV), is associated with the development of Kaposi's Sarcoma in AIDS patients.
- Kaposi's sarcoma is a type of cancer that affects men and is rarely seen in women.
- Although KS mainly affects the skin, the mouth, and the lymph nodes, it can also involve the bowels and lungs.
- HHV 8 is sexually transmitted.

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Paramyxoviruses

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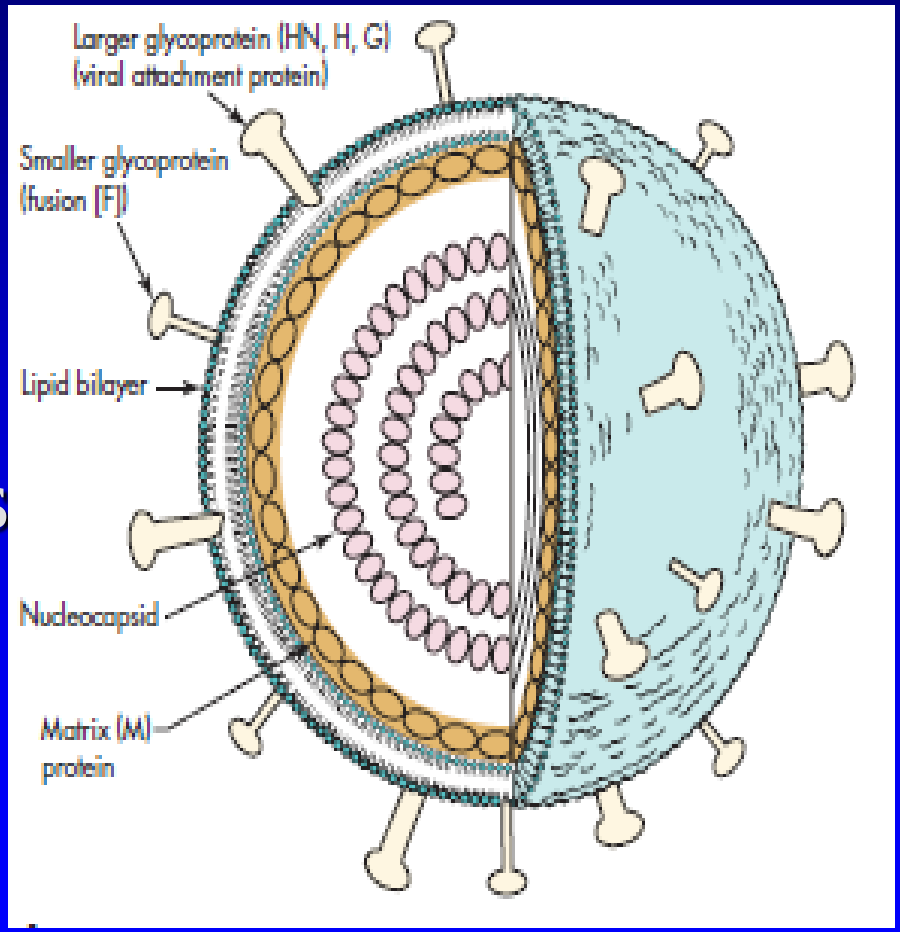
Paramyxoviruses

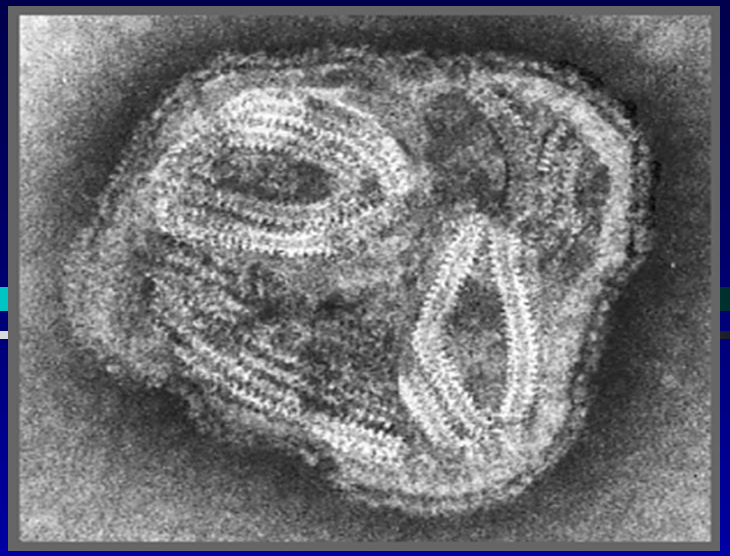
- ☑Structure
- ☑Classification
- ☑Multiplication
- ☑Clinical manifestations
- ☑Epidemiology
- ☑Diagnosis
- ☑Control



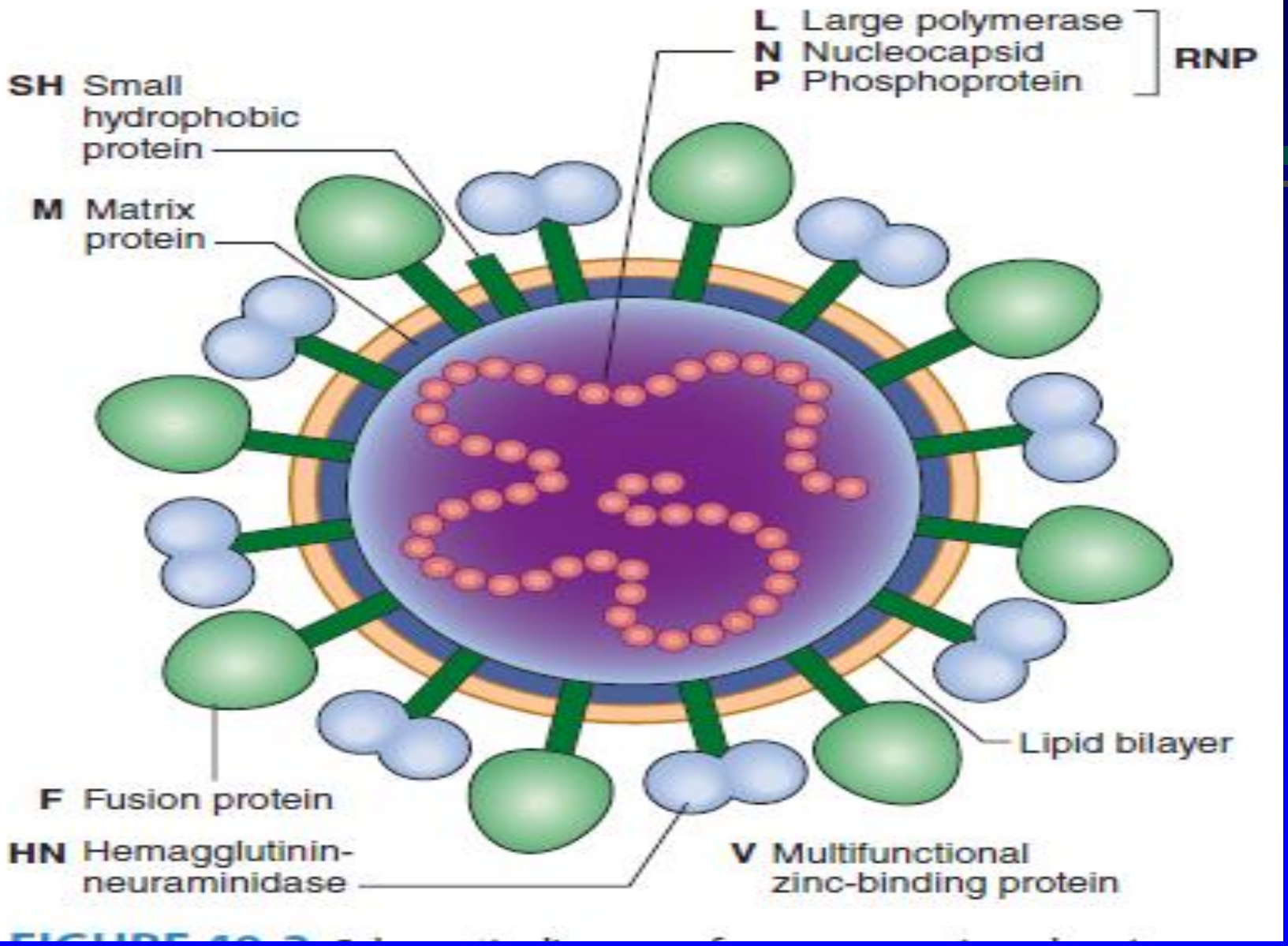
General Concepts

- The family Paramyxoviridae consists of three genera:
- Paramyxovirus, parainfluenza viruses and mumps virus;
- Pneumovirus, which includes respiratory syncytial virus;
- Morbillivirus, which includes the measles virus.

Structure



- All paramyxoviruses are enveloped particles 150 to 300 nm in diameter.
- The tube like, helically symmetrical nucleocapsid,
- single-stranded RNA genome protein (M) at the base of a double-layered lipid envelope.
- The spikes on the envelope
- In parainfluenza viruses, the viral protein spikes have hemagglutinating and neuraminidase activities (HN).
- Respiratory syncytial virus lacks both these activities and measles virus lacks neuraminidase but has hemagglutinating activity.


L Large polymerase
N Nucleocapsid
P Phosphoprotein
RNP
SH Small hydrophobic protein
M Matrix protein
Lipid bilayer
F Fusion protein
HN Hemagglutinin-neuraminidase
V Multifunctional zinc-binding protein

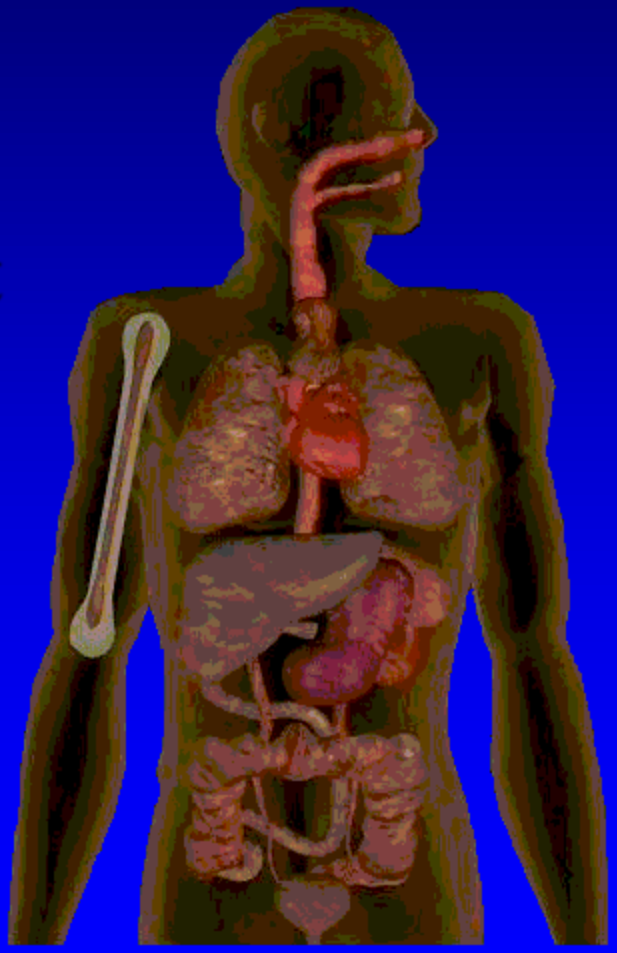
Viral Proteins

- RNA-directed RNA polymerase
- Hemagglutinin
  - parainfluenza
  - measles
- Neuraminidase
  - parainfluenza

Parainfluenza Viruses

Paraflu:Clinical manifestions

- ☑mild or severe infections
- ☑lower and upper respiratory tract
- ☑particularly in children



Paraflu: Classification

- types 1,2,3,4 in humans
- type 4 subtypes A & B

Pathogenesis

- Transmission is by droplets or direct contact. The virus disseminates locally in the ciliated epithelial cells of the respiratory mucosa.

Host Defenses

- Nonspecific defenses, including interferon, are followed by the appearance of secretory and humoral antibodies and cell mediated immune responses.

Paraflu: Epidemiology

- ☑occurs worldwide
- ☑usually endemic
- ☑primarily in young children
- ☑reinfections common

Paraflu: Diagnosis

- ☑clinical symptoms nonspecific
- ☑Isolate virus
- ☑Detect viral antigens
- ☑Detect rise in specific antibodies

No vaccine is available for Parainfluenza

Mumps virus

Clinical Manifestations:

mumps is a common acute disease of children and young adults that is characterized by non purulent inflammation of the salivary glands, especially the parotids.

Severe manifestations may include pancreatitis, meningitis and encephalitis with hearing loss or deafness at any age.

Both symptomatic and asymptomatic mumps virus infections usually induce life long immunity.

Rarely, reinfections with wild-type virus leading to typical mumps may occur.

☑Classification and Antigenic Type:

It occurs only in a single serotype

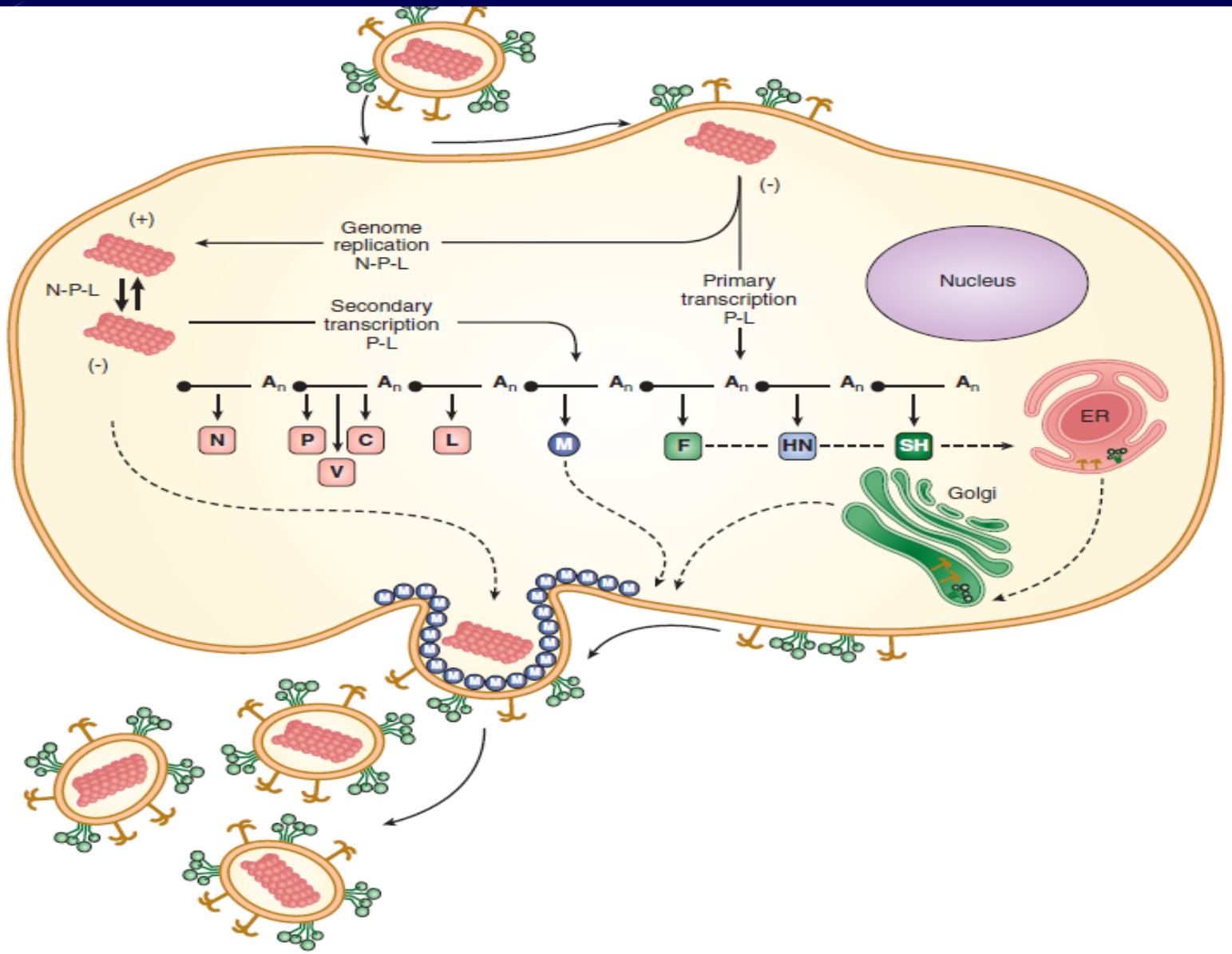
Multiplication:

mumps virus initiates infection by attachment of the HN protein to sialic acid on the cell-surface glycolipids and works together with the F protein to promote fusion with the plasma membrane.

Following uncoating, the negative-sense viral RNA is transcribed by the RNA-dependent RNA polymerase to mRNAs followed by the synthesis of viral proteins which are essential for the continuation of the replication process.

After assembly of the nucleocapsids (RNA,N, L, and P protein) in the cytoplasm, the maturation of the virus is completed by budding.

- **Paramyxovirus life cycle.**
- The infecting virus particle fuses with the plasma membrane and releases the viral nucleocapsid into the cytoplasm. *Solid lines* represent transcription and genome replication. *Dotted lines* indicate transport of newly synthesized viral proteins to plasma membrane. Progeny virions are released from the cell by a budding process. The entire paramyxovirus replication cycle takes place in the cell cytoplasm. ER, endoplasmic reticulum


(+)
(-)
N-P-L
Genome replication N-P-L
Secondary transcription P-L
Primary transcription P-L
Nucleus
ER
Golgi
N
P
C
V
L
M
F
HN
SH

Mumps:Pathogenesis

- Mumps virus causes a systemic generalized infection that is spread by viremia with involvement of glandular and nervous tissues as target organs.
- The incubation period usually is 18 to 21 days
- The main target organs (various salivary glands, testes, ovaries, pancreas, and brain).
- It is not known how the virus spreads to the central nervous system.

Mumps:Clinical manifestions

- systemic febrile infection
- children & young adults
- swelling of salivary glands
  - Parotid gland
- meningitis common
- encephalitis can occur
- orchitis oophoritis in adults

Single mumps serotype

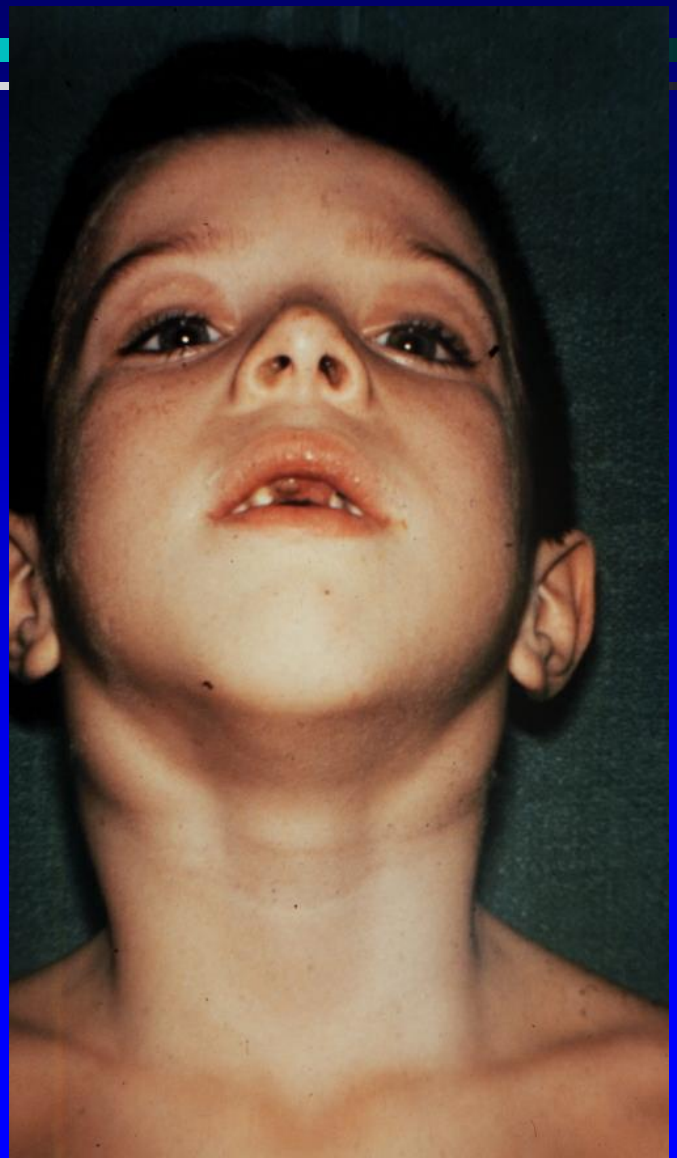
- **Classification and Antigenic Type**: It occurs only in a single serotype and shares minor common envelope antigens with other Paramyxovirus species.

Mumps:Epidemiology

- worldwide
- endemic in urban areas
- intermittant in rural areas
  - epidemic 2-7 years
- peak incidence Jan-May
- Active vaccination in the United States has reduced the incidence of reported mumps and mumps complications by more than 90 percent.

Mumps:Diagnosis

- ***TYPICAL***
- clinical diagnosis
- ***ATYPICAL***
- isolate virus
- viral antigen in saliva of CSF
- Detect specific IgM
- Detect rising titer of IgG



Mumps: Defenses

- Interferon
- humoral immunity (IgM class-specific antibodies to mumps antigens develop rapidly within the first 3 days. The IgG antibodies appear a few days later and persist for life. Circulating antibodies are responsible for the lifelong protection against recurrent disease, but re-infection may occur.
- cell mediated immunity
- lifelong protection

Mumps:Control

- ☑live attentuated vaccine
- ☑long term protection
- ☑reinfections can occur

Morbillivirus

Measles virus

Measles: Clinical manifestions

- coryza, conjunctivitis, fever rash
- maculopapular rash 1-3 days later
- Complications
- otitis, pneumonia, encephalitis
- SSPE (subacute sclerosing panencephalitis)- rare

Measles: Pathogenesis

- viremia
- multiples in cells of :
  - lymphatic system
  - respiratory system
  - skin
  - brain

Measles:Host Defenses

- ☑Interferon
- ☑Humoral immunity
- ☑Cell mediated immunity
- ☑Life long protection

Measles: Epidemiology

- ☑worldwide
- ☑endemics & epidemics
- ☑mainly late winter-early spring

Measles: Diagnosis

Typical

- ☑clinical manifestations

Atypical

- ☑Isolate virus
- ☑Detect specific IgM
- ☑Detect increase in IgG

Measles: Control

Active vaccination

- ☑Live attentuated virus vaccine
- ☑long lasting protection

Passive immunity

- ☑measles hyperimmunoglobulin

WHO Measles Vaccination Strategy

- "catch-up" everyone aged 1-14 years
- "keep-up" 90% of children at age 12 months;
- "follow-up" 3-5 years

Pneumovirus

Respiratory syncytial virus

RSV:Clinical manifestions

- ☑upper & lower respiratory tract infection
- ☑frequent in young children
- ☑significant in elderly

RSV: Pathogenesis

- ☑droplets
- ☑direct contact
- ☑infects ciliated epithelium of respiratory mucosa
- ☑localized

Antibody Dependent Cytotoxicity

RSV: Host Defenses

- ☑interferon
- ☑cell mediated immunity
- ☑Humoral immunity
- ☑Secretory immunity (sIgA)
- ☑reinfection possible

RSV: Epidemiology

- ☑worldwide
- ☑temperate climates
- ☑epidemic winter and early spring
- ☑infants & young children

RSV: Diagnosis

- ☑nonspecific clinical symptoms
- ☑Isolate virus
- ☑Detect viral antigen
- ☑Detect IgM, IgA
- ☑Detect increase in IgG

RSV: Control

- ☑no vaccine
- ☑ribavrin as aerosol
- ☑isolate patients in hospitals

Unique Features of the Paramyxoviridae

- Large virion consists of a negative RNA genome in a helical
- nucleocapsid surrounded by an envelope containing a viral
- attachment protein (hemagglutinin-neuraminidase [HN], parainfluenza virus and mumps virus; hemagglutinin [H], measles virus; and glycoprotein [G], respiratory syncytial virus [RSV]) and a fusion glycoprotein (F).

The three genera can be distinguished by the activities of the viral attachment protein: HN of parainfluenza virus and mumps virus has hemagglutinin and neuraminidase, and H of measles virus has hemagglutinin activity, but G of RSV lacks these activities.

- ☑Virus replicates in the cytoplasm.
- ☑Virions penetrate the cell by fusion with and exit by buddingfrom the plasma membrane.

- Viruses induce cell-cell fusion, causing multinucleated giantcells.
- Paramyxoviridae are transmitted in respiratory droplets and initiate infection in the respiratory tract.
- Cell-mediated immunity causes many of the symptoms but is essential for control of the infection.

QUESTIONS?


Thank You!
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