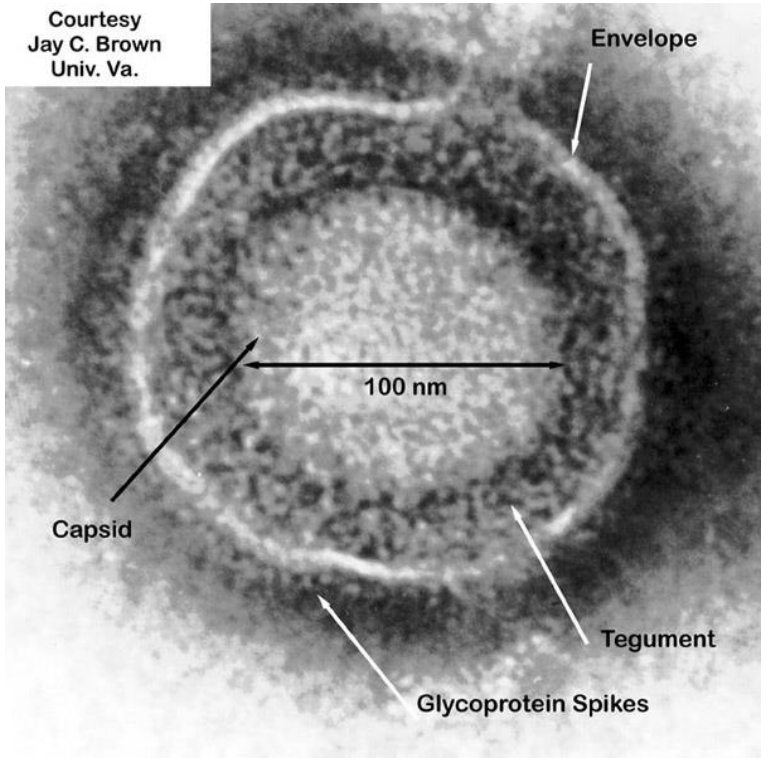
Lesson 24

Microbiological diagnosis of DNA virus infections (Herpesviridae, Adenoviridae, Papillomavidiae, Poxviridae)

Family Herpesviridae (herpesviruses)

- The first representative of this family, the herpes simplex virus, causes vesicular rashes on the skin and mucous membranes, followed by their transition to erosion.
- The name of the family reflects the pathological effect produced by viruses (from the Greek "herpes" - "creeping")

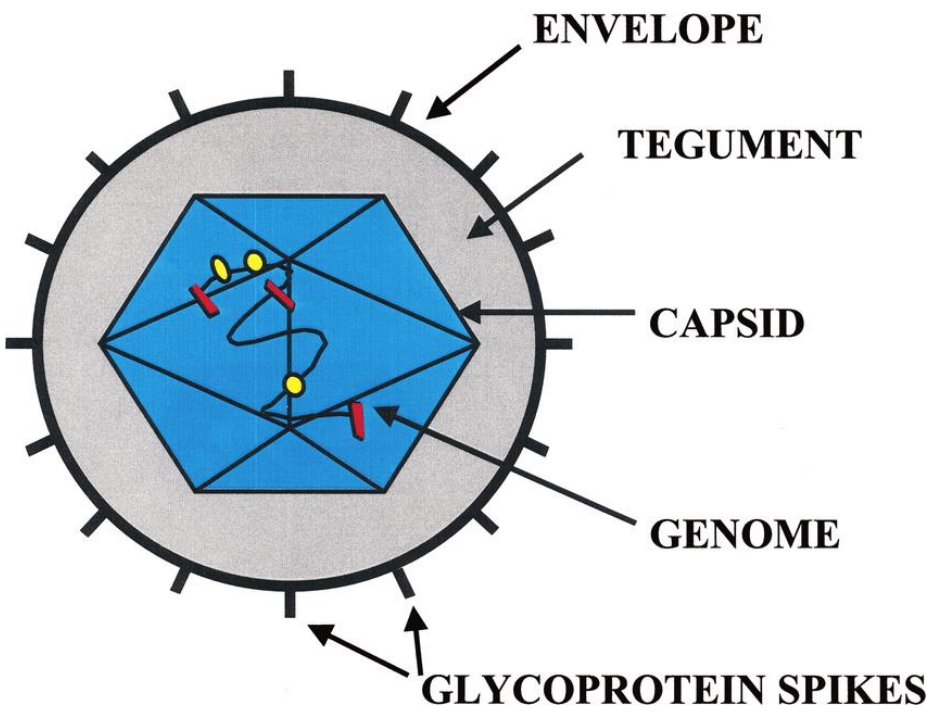


Classification of herpes viruses

- There are 8 types of herpes viruses pathogenic for humans:
- 1- herpes simplex virus - HSV type 1 (Herpes simplex virus 1 - HSV-1);
- 2- herpes simplex virus - HSV type 2 (Herpes simplex virus 2 - HSV-2);
- 3-Varicella zoster virus (VZV), or human herpes virus GVCh-3;
- 4-Epstein-Barr virus - EBV (Epstein-Barr virus - EBV), or human herpes virus GVCh-4;
- 5-Cytomegalovirus - CMV, or human herpes virus HVV-5;
- 6-Human herpes virus type 6 - HVV-6 (Human herpesvirus 6 - HHV-6)
- 7-Human herpes virus type 7 - HVV-7 (Human herpesvirus 7 - HHV-7) 8-
- Human herpes virus type 8 - HVV-8 (Human herpesvirus 8 - HHV-8)

Structure of herpes viruses

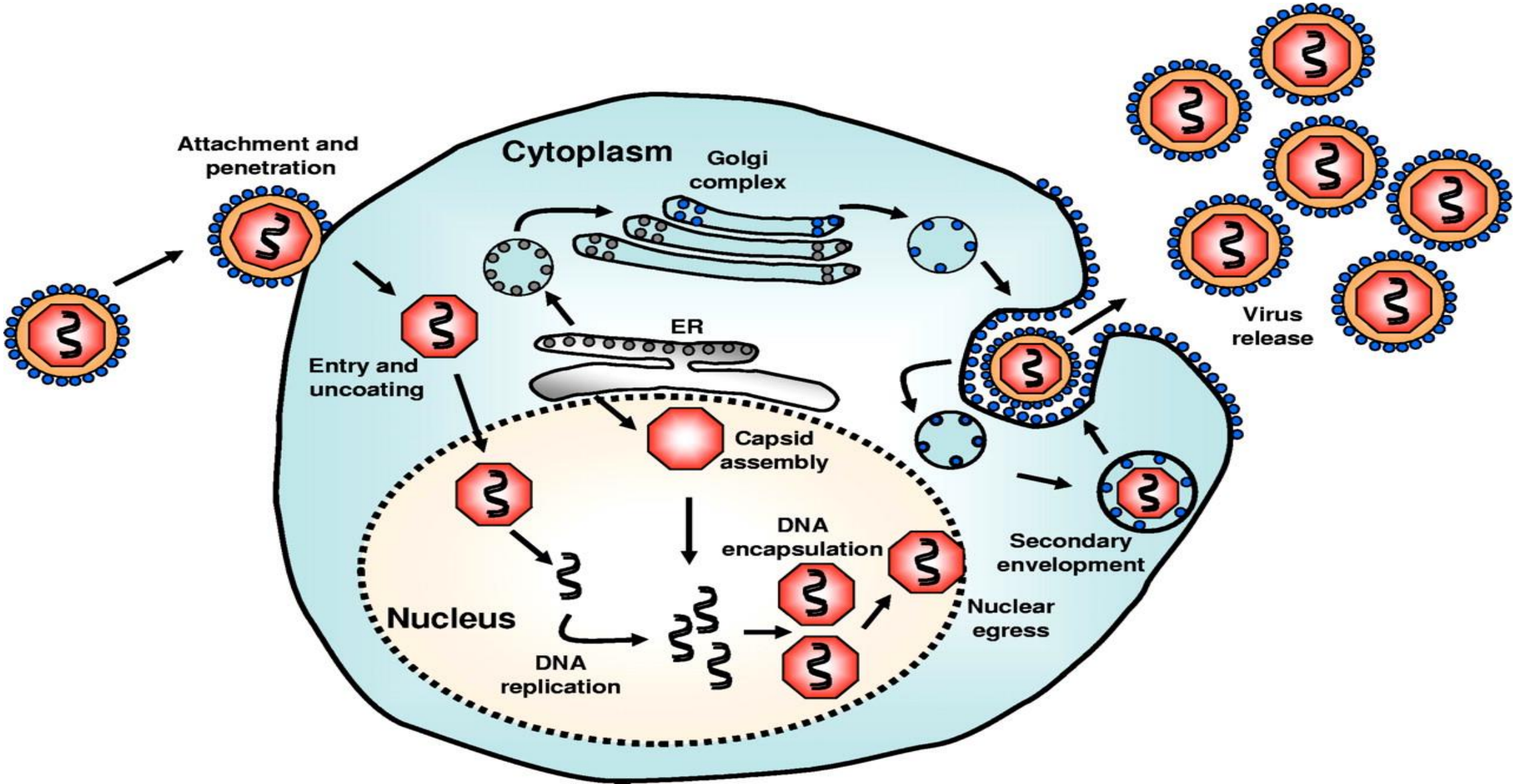
- Herpes viruses are large enveloped DNA-containing viruses, 150-200 nm in diameter.
- The virion is oval in shape. In the central part of the virion is DNA, surrounded by an icosahedral capsid, consisting of 162 capsomeres.
- Outside, the virus is surrounded by a sheath with glycoprotein spikes. The space between the capsid and the envelope is called the tegument, which contains the viral proteins and enzymes needed to initiate viral replication.
- The genome is double-stranded linear DNA.



Replication of herpesviruses

- The surface glycoproteins of the virion bind to host cell receptors.
- The transcription of the viral genome (DNA) occurs in the nucleus, the resulting mRNAs penetrate into the cytoplasm, where not only proteins with regulatory activity are synthesized, but also structural proteins, including capsid and glycoproteins.
- The emerging capsid is filled with viral DNA, the synthesized glycoproteins and nucleocapsid diffusely adhere to the nuclear envelope, where the supercapsid is formed.
- Virions bud through the modified membranes of the nuclear envelope and then, moving through the Golgi apparatus, exit the cell by exocytosis or cell lysis.
- Most of the mature virions leave the cell, while some remaining inside the cells cause a cytopathic effect characteristic of some herpesviruses, that is, the formation of multinucleated cells - symplasts.

Scheme of reproduction of herpes viruses

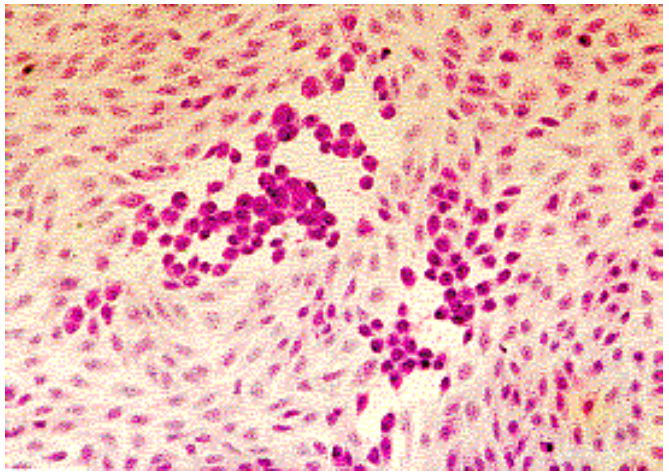


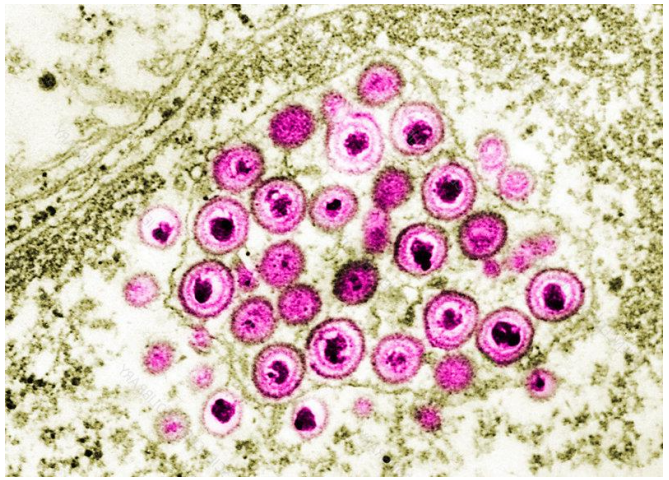
Herpes simplex virus

- Herpes simplex virus belongs to the Herpesviridae family, genus Simplexvirus. There are two types of herpes simplex virus:
- 1st type of herpes simplex virus (HSV-1) - more often affects the mucous membranes of the oral cavity, eyes, central nervous system;
- Herpes simplex virus type 2 (HSV-2) - affects the genitals.

Cultivation of herpes simplex virus

- For the cultivation of HSV, a chicken embryo is used (small dense plaques form on the chorion-allantoic membrane).
- In cell culture (HeLa, Hep-2, human embryonic fibroblasts) cause a cytopathic effect in the form of the appearance of giant multinucleated cells with intranuclear inclusions - Caudry inclusions.



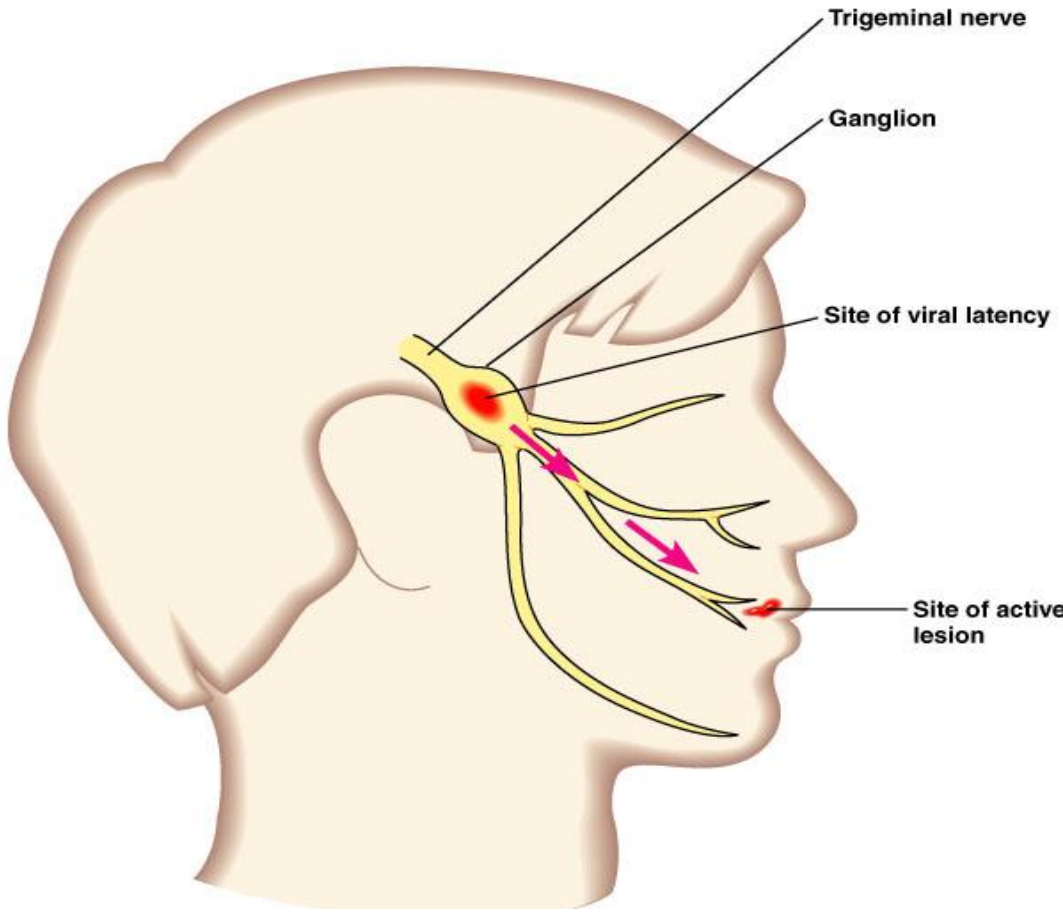


Pathogenesis

- HSV-1 is transmitted mainly by contact, less often by airborne droplets. HSV-2 is transmitted by contact, during sexual contact. Infection with HSV-2 is possible when the fetus passes through the birth canal, less often - transplacental.
- Viruses penetrate through mucous membranes and damaged skin. HSV-1 replicates on the mucous membranes of the oral cavity and pharynx, HSV-2 - on the skin and mucous membrane of the genital tract.
- Primary infection is mild, mostly asymptomatic. The viruses then migrate into neurons by retrograde axonal transport and cause latent infection (HSV-1 in the trigeminal ganglion, HSV-2 in the lumbar and sacral ganglia). In latently infected neurons, about 1% of the cells in the affected ganglion carry the viral genome. At the same time, viral DNA exists in the form of free circular episomes (about 20 copies per cell).
- Most people (about 80%) are lifelong carriers of the virus, which persists in the ganglia, causing a latent persistent infection in neurons.

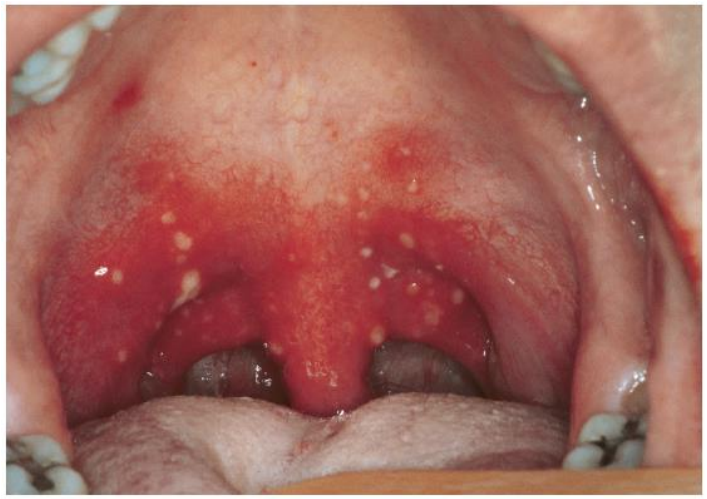
Pathogenesis

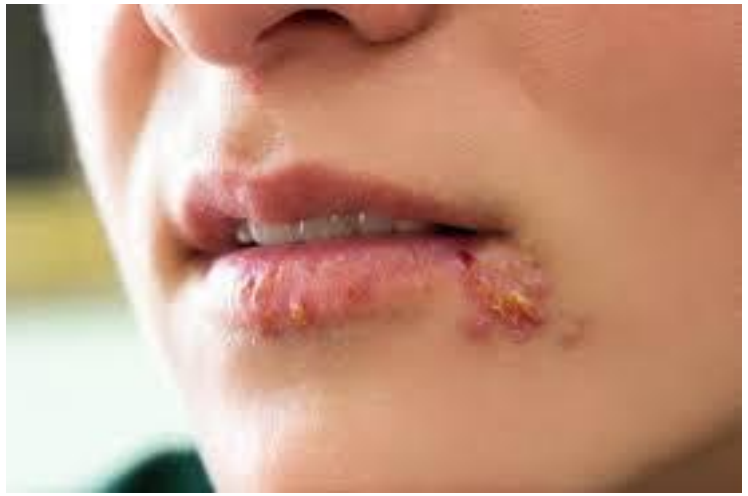
- Viruses in latently infected ganglia persist throughout life. Reactivation of herpes viruses is caused by various factors (hypothermia, fever, trauma, stress, concomitant diseases, UV exposure, etc.) that reduce immunity.
- The DNA of herpes viruses passes along the axon back to the nerve ending, where infection can develop with the reproduction of the virus in epithelial cells.

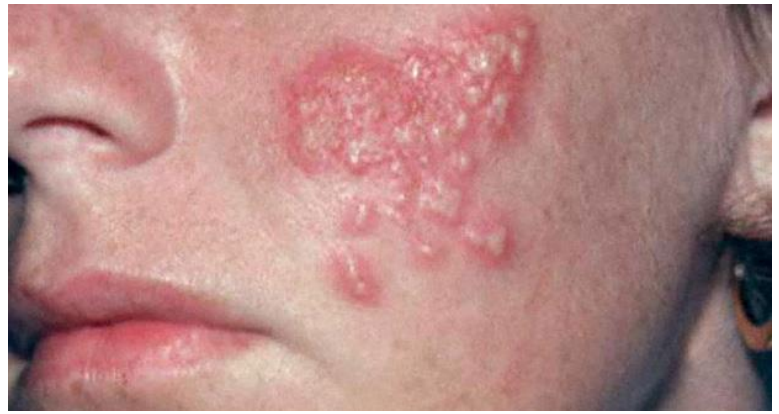


Clinical manifestations of diseases caused by herpes simplex viruses

- Diseases caused by viruses are varied. There are primary and recurrent herpes simplex.
- Oropharyngeal herpesvirus infections cause HSV-1. The primary infection is often asymptomatic, but in children aged 1-5 years it manifests itself with vesicular and ulcerative lesions of the oral mucosa and gums (gingivostomatitis).
- The recurrence of the disease manifests itself in the form of vesicles, mainly in the area of the lips - therefore it is called labial herpes (herpes labialis)

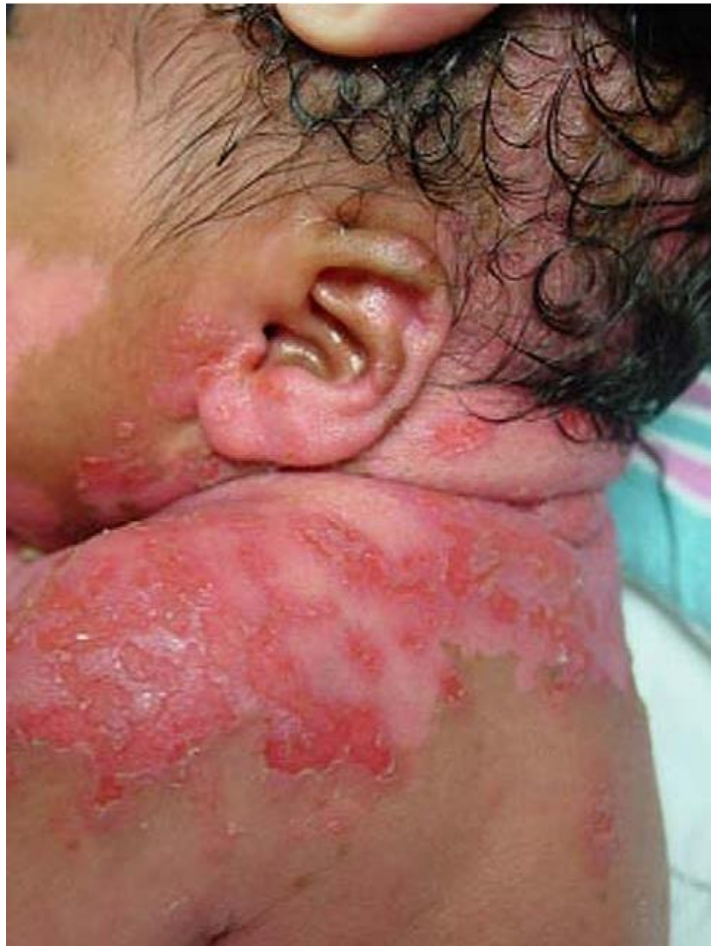






Clinical manifestations of infections caused by HSV

- Genital herpes is mainly caused by HSV-2. The lesion is manifested by the formation of vesicles, which subsequently ulcerate.
- Neonatal herpes, or herpes of the newborn, is caused mainly by HSV-2. Infection can occur transplacentally, during childbirth, and after childbirth. Untreated neonatal herpes is fatal in about 50% of cases.
- Immunocompromised individuals are most likely to develop HSV infections.



Microbiological diagnostics

- For diagnosis, the contents of herpetic vesicles, saliva, scrapings from the cornea of the eyes, blood, cerebrospinal fluid are used.
- Express diagnostics reveals multinucleated cells with intranuclear inclusions (Tzank cells) in Giemsa-stained smears from rashes.
- To identify the virus, amplification of the viral DNA genes in the PCR reaction is used.
- To isolate the virus, cultures of HeLa, Hep-2, and human embryonic fibroblasts are infected with the test material. Viruses are identified in RIF and ELISA using monoclonal antibodies.
- Serodiagnostics is carried out using ELISA by increasing the titer of specific antibodies. Basically, specific antibodies are determined on the 4th-7th day, on the 2nd-4th week there is a significant increase in antibody titer. IgG in the blood persist throughout life.

Treatment and prevention

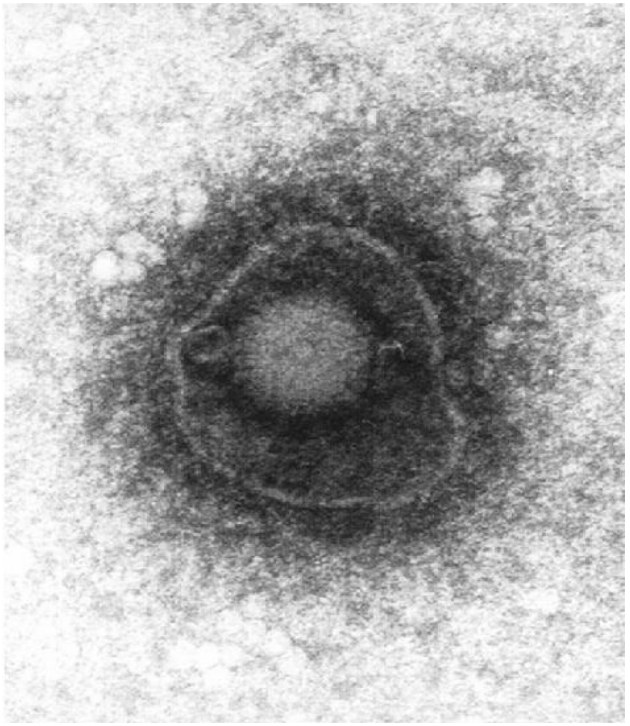
- Chemotherapeutic antiviral drugs (acyclovir, valaciclovir, vidarabine, etc.) that inhibit viral DNA synthesis are used to treat herpesvirus infections. The standard therapeutic drug is acyclovir (zovirax).
- An effective genetically engineered vaccine containing glycoprotein antigens of the outer envelope of viruses. The vaccine helps prevent primary infection.

Varicella-zoster virus and herpes zoster (VZV)

- Human herpesvirus type 3 or Varicella-zoster virus (VZV) belongs to the Herpesviridae family, genus Varicellovirus.
- VZV causes two diseases. Primary infection leads to the development of chicken pox (varicella). After primary infection, the viruses persist for life in the ganglia of the cranial nerves. Subsequent activation of viruses leads to the development of herpes zoster (herpes zoster). Therefore, the pathogen was named the varicella-zoster virus and varicella-zoster virus.

Varicella-zoster virus and herpes zoster (VZV)

- The structure of VZV is similar to that of other herpesviruses.
- The virus multiplies in human embryonic fibroblasts with the formation of intranuclear inclusions.
- The virus is unstable in the environment; at a temperature of 600C it dies within 30 minutes. , sensitive to fat solvents and disinfectants .



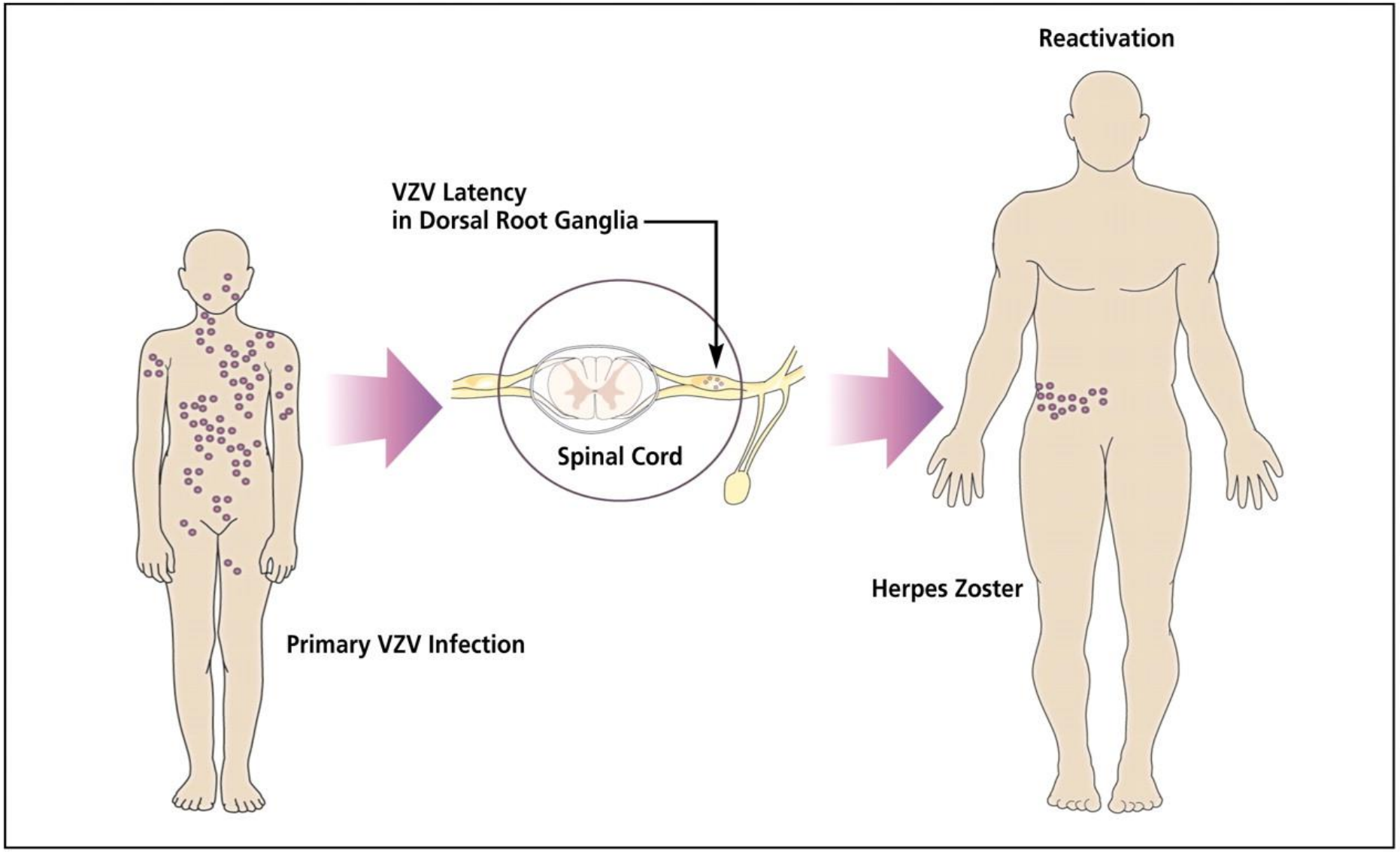
Varicella zoster virus and herpes zoster

- Chickenpox is an anthroponotic disease that most often affects children under the age of 10 years. The source of infection is a patient with chickenpox or a virus carrier; a patient with herpes zoster is sometimes contagious. The virus is transmitted by airborne droplets and contact (through detachable vesicles) by. Transplant transmission is possible.
- After an illness (chicken pox), the virus persists for a long time in the ganglia of the cranial nerves, causing a latent infection. Herpes zoster mainly affects adults, the disease develops as a result of reactivation of the virus that persists in the body, i.e. a virus that has survived after childhood chicken pox.

Pathogenesis

- The causative agent penetrates through the mucous membranes of the upper respiratory tract, possibly through the conjunctiva.
- After primary reproduction in the regional lymph nodes, the pathogen enters the bloodstream and spreads throughout the body, causing primary viremia.
- After replication in the liver and spleen, the viruses are introduced into various organs and tissues, but mainly into the epithelium of the skin (dermatotropic action) and mucous membranes, causing secondary viremia. Epithelial cells undergo dystrophy, accumulation of interstitial fluid with the formation of vesicles - vesicles.
- After primary infection, the virus persists for a long time in the posterior spinal nerve root or trigeminal ganglion. The activated virus, which is located in the nerve cells of the spinal cord, reaches the skin through the nerve trunks, causing herpes zoster.

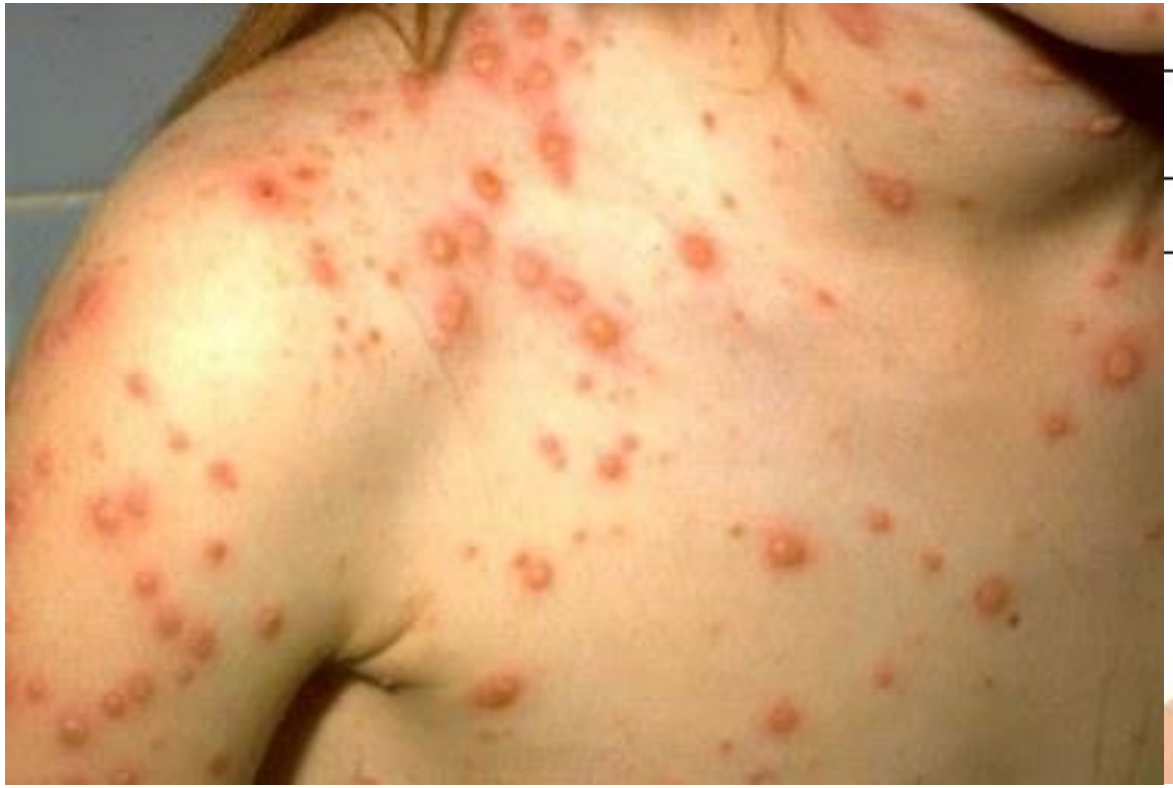
Pathogenesis

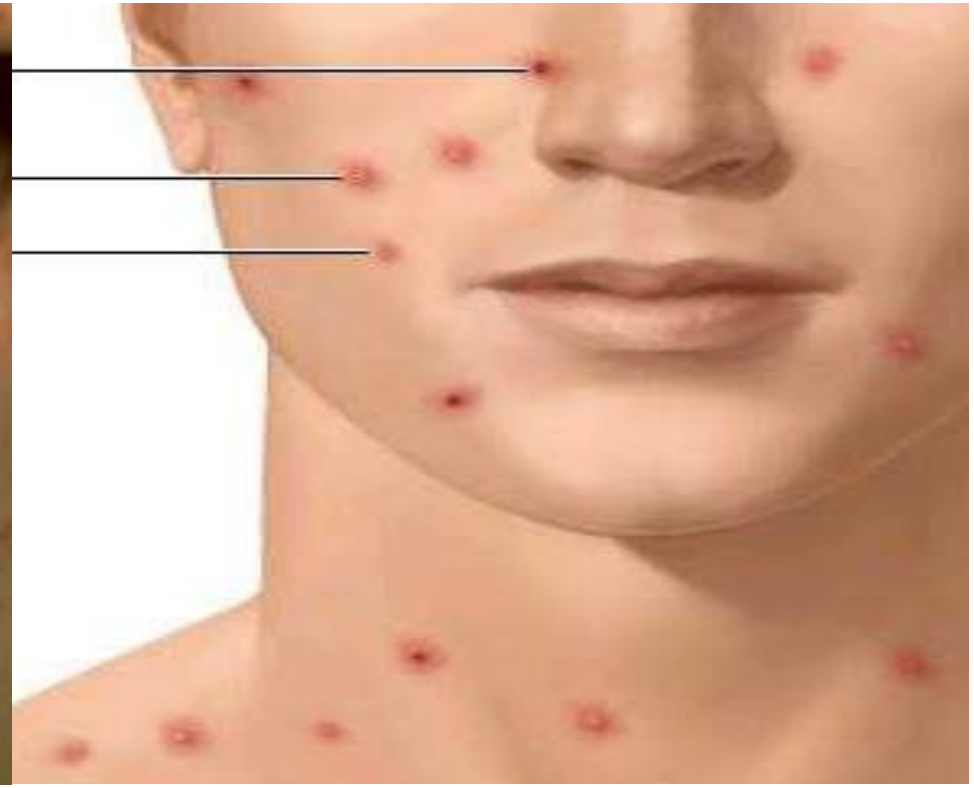


Clinical manifestations of chickenpox

- The incubation period for chickenpox (varisella) is 10-21 days. The disease is characterized by fever, the appearance of a maculopapulovesicular rash on the skin of the trunk, neck, face and extremities. Initially, the rash appears in the form of a macula, papule, and then - in the form of vesicles with transparent contents.
- The disease in infants, the elderly and those with immunodeficiencies is severe, complicated by pneumonia, hepatitis, encephalitis, otitis, pyoderma.

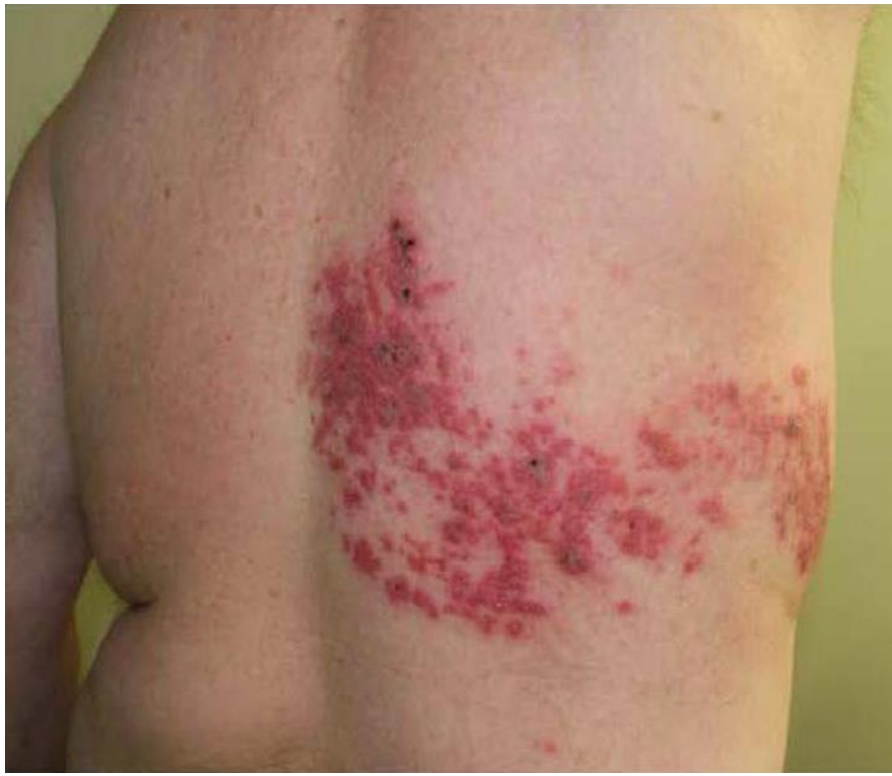
Clinical manifestations of chickenpox





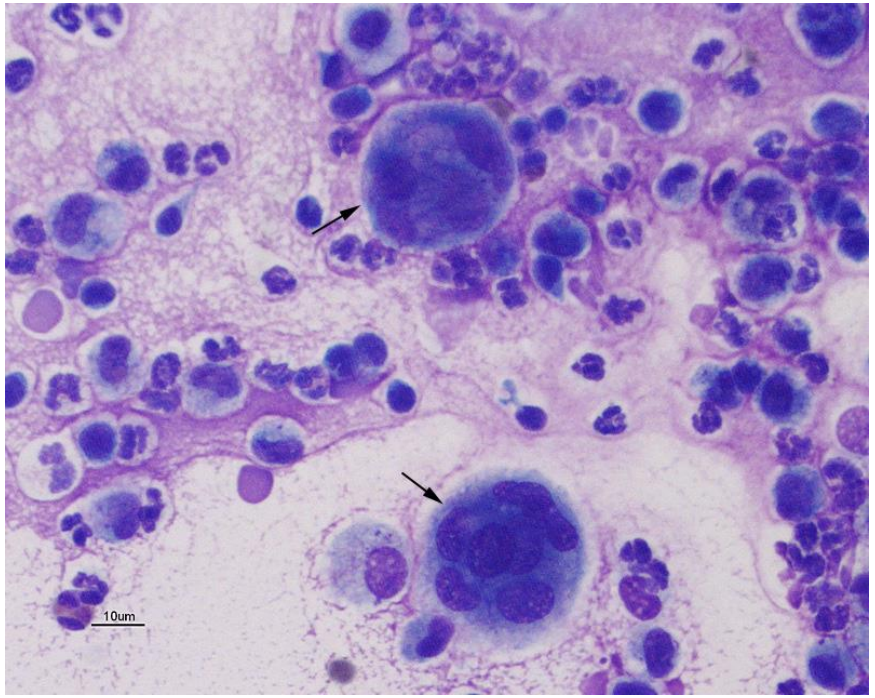
Clinical manifestations of herpes zoster

- Herpes zoster (zoster) is an endogenous infection that develops in people who have had chickenpox in childhood.
- The infection develops as a result of the activation of viruses that persist in the ganglia of the posterior roots of the spinal nerves and in the ganglia of the trigeminal nerve. Activation of the virus is facilitated by various factors that weaken the immune system - diseases, colds, injuries, etc. The virus enters the skin along damaged nerves (often intercostal nerves), causing rashes that cover the surface of the body in the form of a hoop (hence the name of the disease).
- The disease is accompanied by severe pain. The most common complication of herpes zoster, postherpetic neuralgia, can last for many months.



Microbiological diagnostics

- The diagnosis is exposed on the basis of clinical data.
- To confirm the diagnosis, the contents of the rash, the discharge of the nasopharynx and blood are taken. The virus can be detected in smears-prints, stained according to Romanovsky-Giemsa, by the formation of intranuclear inclusions.
- The virus does not replicate well in cell cultures and is therefore unimportant.
- In serodiagnosis, specific antibodies are detected by RIF and ELISA.
- HSV infections promote synthesis of cross-immunity against VZV



Treatment

- In children with normal immunity, there is no need for antiviral therapy for chickenpox.
- Treatment of newborns and immunocompromised individuals is essential. Gamma globulin, which contains high titers of antibodies against VZV, is considered effective in treatment.
- For treatment, acyclovir, vidarabine, interferon preparations, and other immunomodulators are used.

Epstein-Barr virus

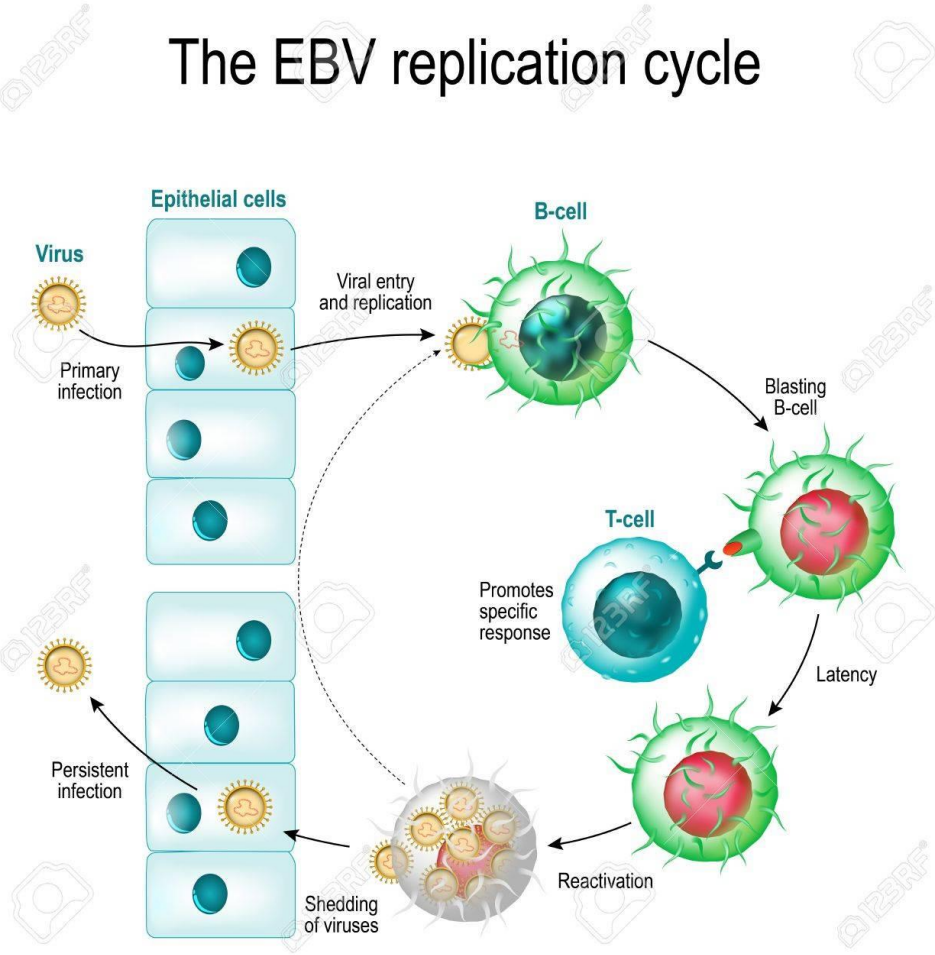
- Epstein-Barr virus (EBV, human herpes virus type 4) belongs to the Herpesviridae family, genus Limphocryptovirus.
- The virus was isolated in 1964 by M. Epstein and I. Barr using electron microscopy of a biopsy of Burkitt's lymphoma.

Epstein-Barr virus

- Structure and antigens. EBV is structurally and morphologically similar to other herpesviruses. According to latent nuclear antigens (EBNA and EBER), they are divided into two types - VEB-1 and VEB-2.
- EBNA has EBNA antigen, which is divided into six types (EBNA 1, 2, 3A, 3B, 3C, LP).
- EBV-2 has an EBER antigen. These are small Epstein-Barr-encoded RNA molecules - EBER1 and EBER 2.
- The virus has latent membrane proteins LMP 1 and LMP 2. These antigens are expressed by infected B lymphocytes.

The pathogenesis of infections caused by EBV

- The source of infection is a sick person or a virus carrier. The virus is transmitted by airborne droplets, by contact through saliva.
- Primary EBV replication occurs in the nasopharynx or epithelial cells of the salivary glands.
- The virus has a tropism for B-lymphocytes, interacts with receptors for the C3 complement component located on the surface of B-lymphocytes and, having penetrated into them, spreads throughout the body.

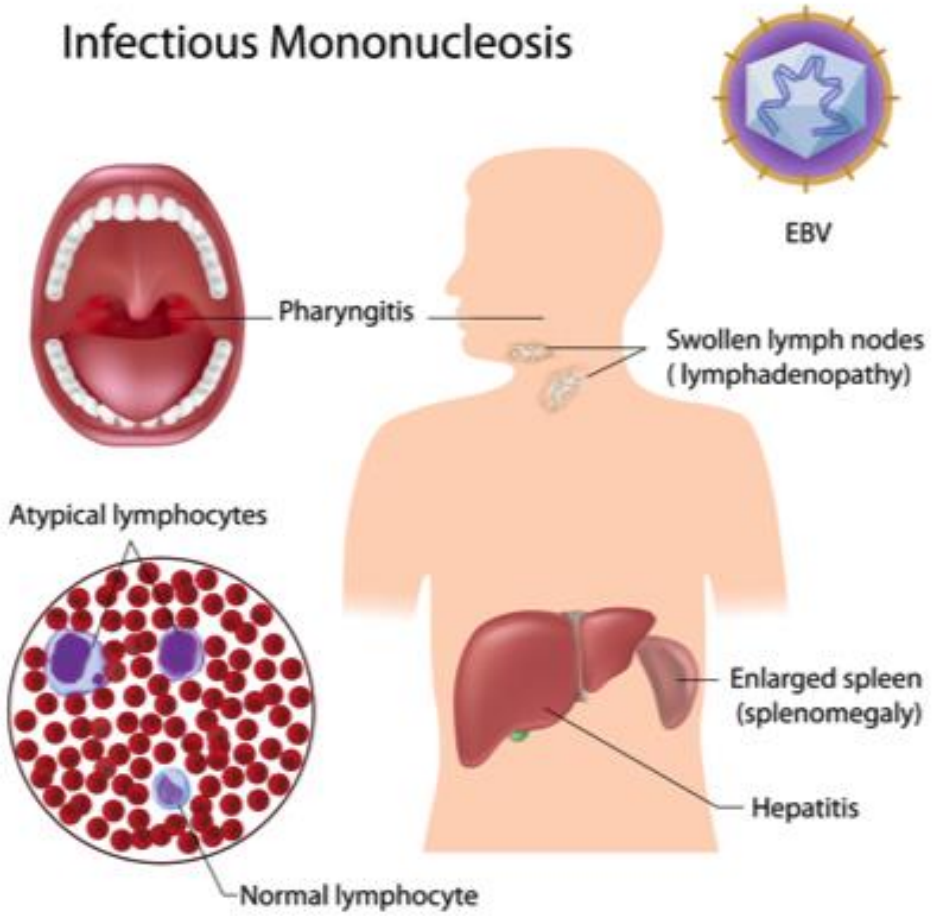


The pathogenesis of infections caused by EBV

- Viruses do not replicate in B-lymphocytes, but persist as copies of extrachromosomal DNA, causing latent infection.
- EBV has a transforming effect, increases the ability of B-lymphocytes to proliferate, thereby making them "immortal".
- Polyclonal stimulation of infected B-lymphocytes promotes the synthesis of various immunoglobulins, including heterophile antibodies (for example, to ram erythrocytes). These cells become targets for cytotoxic T-lymphocytes, which suppress their proliferation.
- This is why the weakening of cellular immunity for various reasons (taking immunosuppressants, AIDS, etc.) increases susceptibility to EBV-associated lymphomas.

Clinical manifestations of infections caused by EBV (infectious mononucleosis)

- EBV causes infections mononucleosis and lymphoproliferative diseases, as well as some carcinomas.
- Infections mononucleosis is characterized by intoxication, damage to the palate and tonsils, lymphadenopathy, and splenomegaly.



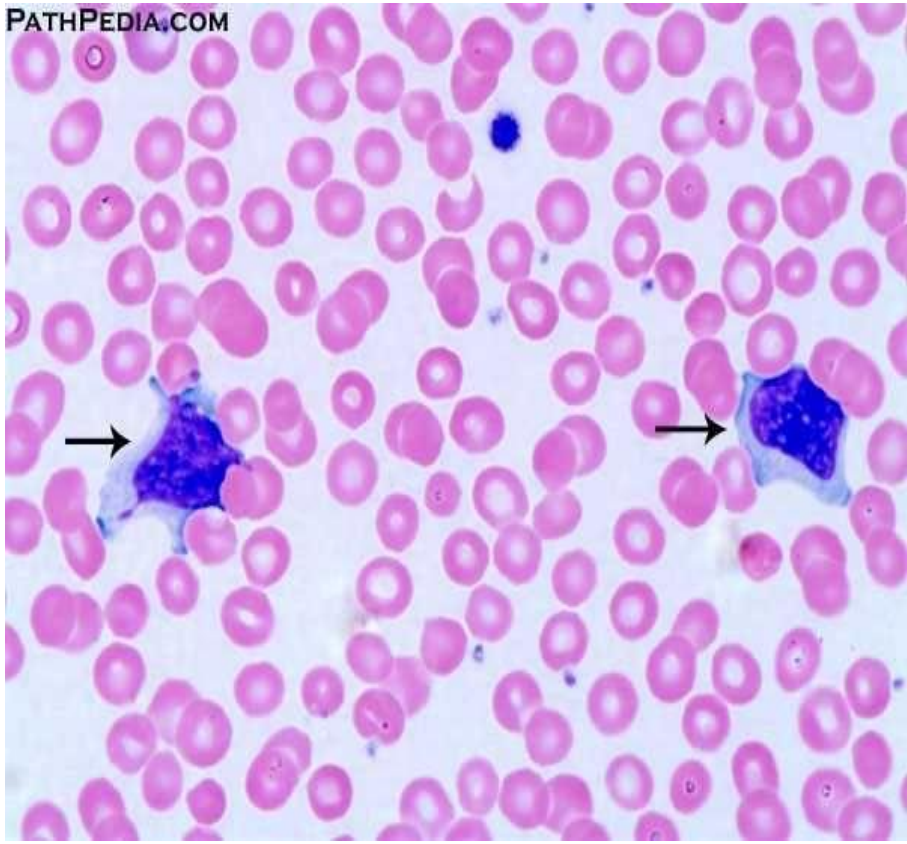
Clinical manifestations of diseases caused by EBV
(lymphoproliferative diseases)

- Hairy oral leukoplakia is an AIDS-specific lesion of the oral mucosa.
- Burkitt's lymphoma is associated with malaria in Africa and is a malignant, rapidly progressive tumor. It develops mainly in children of 5-8 years of age, accompanied by destruction of the upper jaw. Metastases to other organs are possible.
- Nasopharyngeal carcinoma is an endemic disease in China, most common among the male population. Tumor cells have an epithelial origin and contain EBV DNA sequences.
- EBV DNA is also found in a significant number of patients with Hodgkin's lymphoma.
- Lymphoproliferative diseases in immunocompromised individuals can also be induced by EBV.

Burkitt's lymphoma

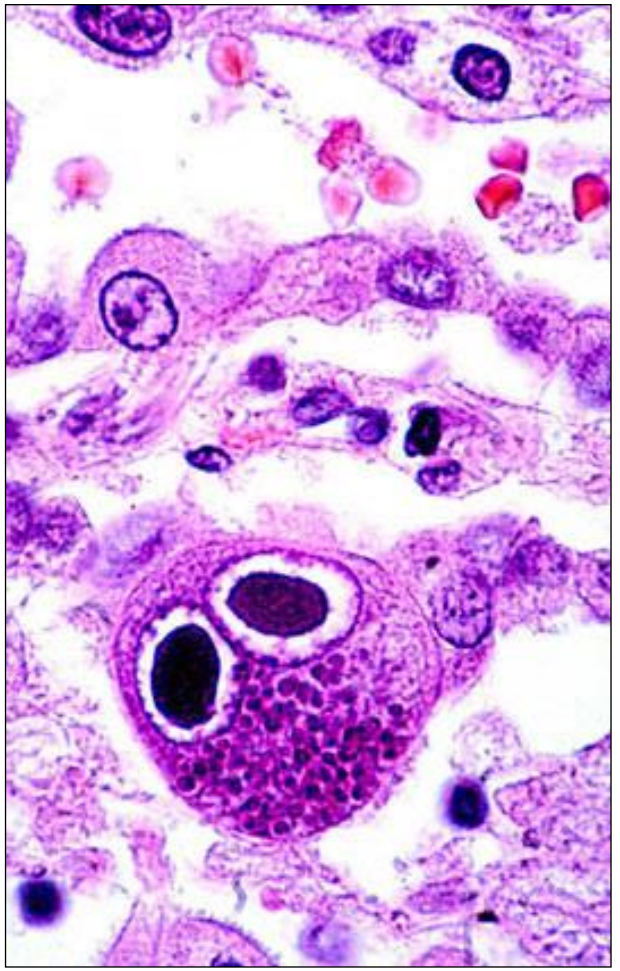
Microbiological diagnosis of infections caused by EBV

- Infectious mononucleosis is documented by the detection of atypical lymphocytes, lymphocytosis (monocytes make up 60-70% of white blood cells with 30% of atypical lymphocytes)
- Auxiliary reactions are also used to detect heterophile antibodies (agglutination of sheep erythrocytes with the patient's blood serum, etc.)
- In the early stages of the disease, they are determined against the capsid antigen, then IgG antibodies are formed, which persist throughout life. A few weeks after an acute infection, antibodies to EBNA and membrane antigens are detected and persist throughout life.



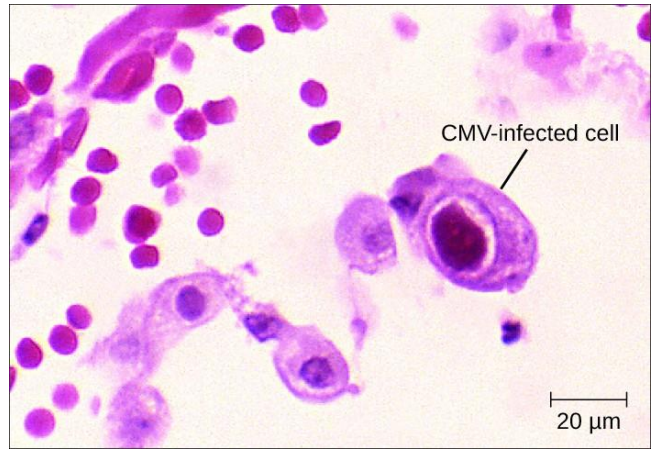
Cytomegalovirus

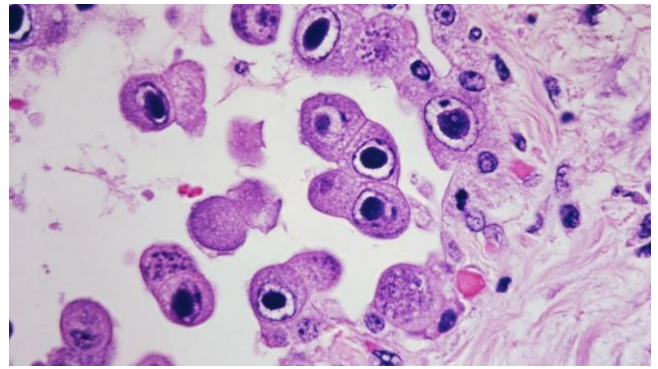
- Cytomegalovirus (CMV), or cytomegalovirus, or herpesvirus type 5, belongs to the Herpesviridae family, genus Sytomegalovirus.
- The name of the virus refers to pathological changes in cell culture morphology resulting from cytopathic action.
- In the affected foci, giant cells 25-40 microns in size (from the Greek cytos - cell, meqas - large) containing inclusion bodies are found. Intranuclear inclusions are separated from the nuclear membrane by a transparent unstained zone and are shaped like an "owl's eye".



Cytomegalovirus (features)

- CMV has the largest DNA genome among herpes viruses. The virus is cultivated in human fibroblast culture.
- The virus causes a cytopathic effect with the formation of giant, or cytomegalic cells with intranuclear inclusions, which is detected 30-50 days after infection.
- The virus is unstable, thermolabile, sensitive to disinfectants and fat solvents.





Ways of transmission of CMV infection

- According to some reports, all people under the age of 50 are infected with CMV.
- The source of infection is a person with an acute or latent form.
- Infection occurs through blood, saliva, urine, semen, breast milk, etc. by contact-household, airborne, sometimes fecal-oral.
- Infection can be through sexual contact, blood transfusion and organ transplantation.

Pathogenesis and clinical manifestations of CMV infection

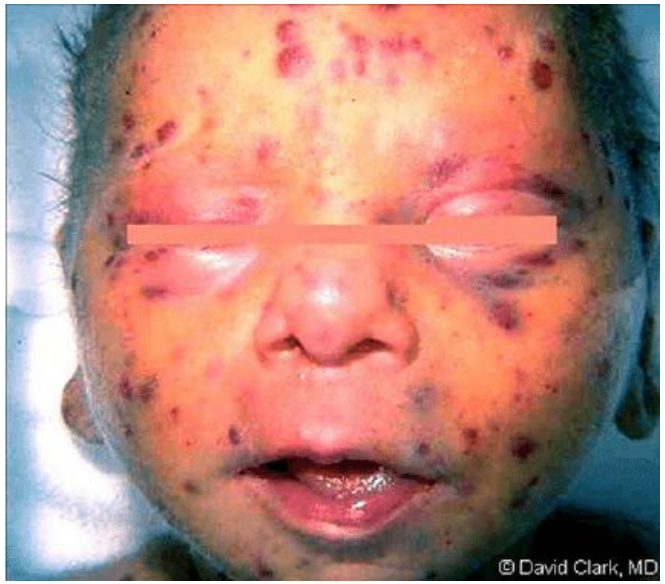
- The entry gates of infection are the skin, mucous membranes, respiratory tract and placenta (congenital cytomegaly).
- The virus enters the body and causes a systemic infection with damage to the salivary glands, lungs, liver, kidneys, monocytes, T- and B-lymphocytes. Incubation period 1-2 months
- The disease is accompanied by symptoms similar to infectious mononucleosis, but in most cases it proceeds subclinically. The main clinical symptoms include prolonged fever, weakness, muscle pain, hepatic dysfunction, and lymphocytosis.
- The exact localization of CMV persisting in the body is unknown, presumably in monocytes and macrophages.
- Most often, the activation of the virus is observed in pregnant women and patients with immunodeficiencies.

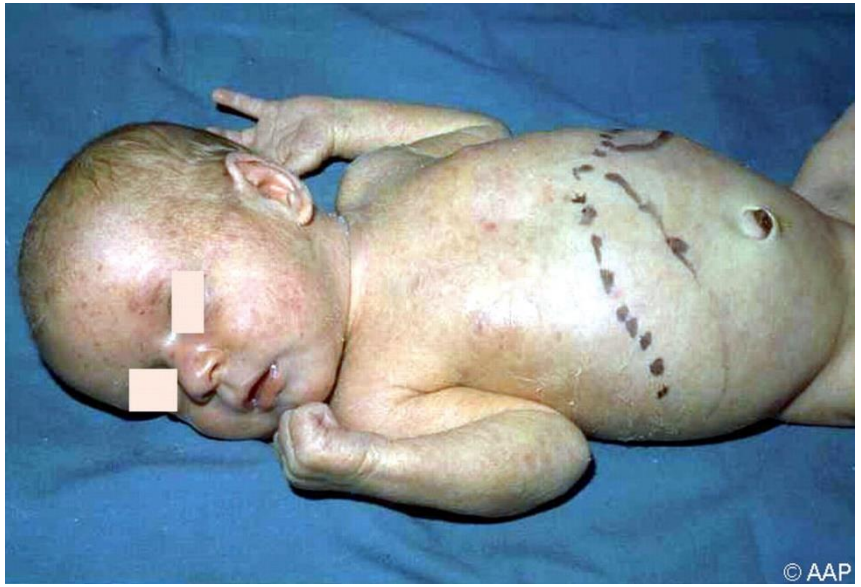
CMV infection in immunocompromised individuals

- In immunocompromised patients, CMV infection is more severe.
- Disseminated CMV infection in the form of various complications (most often pneumonia) can develop after organ transplantation, in patients with malignant tumors and in patients with AIDS.
- The virus can potentially cause tumors (prostate adenocarcinoma, etc.)

Congenital CMV infection

- During primary infection, as well as during reactivation of the virus in pregnant women, intrauterine infection of the fetus may occur. Newborns develop hepatosplenomegaly, jaundice, cachexia, microcephaly and other malformations, leading to death in about 20% of cases.
- The majority of viable children under 2 years of age, along with a weakening of vision and hearing (sometimes deafness), have various pathologies of the central nervous system.





Microbiological diagnosis of CMV infection

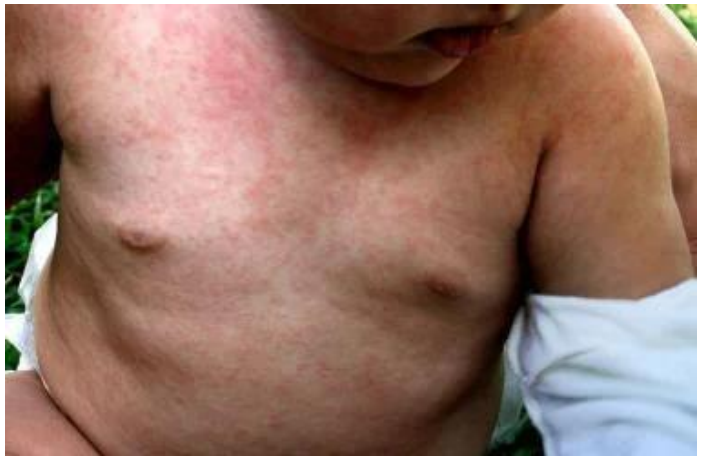
- Antibodies (IgM and IgG) to the virus in the patient's blood serum are determined by ELISA. Detection of IgG indicates a past infection and the possibility of potential activation of the virus, while IgM indicates a current infection (or activation of the infection).
- The virus in the blood and urine is detected by PCR.
- Virus-positive leukocytes in patients can be determined by RIF using monoclonal antibodies.
- Due to the long periods of cultivation in the clinical laboratory, the cultivation of the virus in cell culture is not carried out.

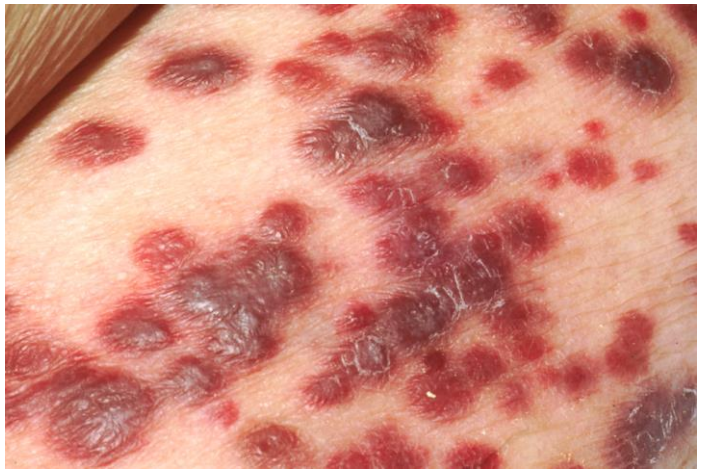
Treatment of cytomegalovirus infection

- Nucleoside analogues, ganciclovir, are used for treatment.
- The appointment of acyclovir and valaciclovir alleviates the course of CMV infection that developed after bone marrow and kidney transplantation
- Due to the obtained conflicting results, the use of cytomegalovirus immunoglobulin containing high titers of antibodies against CMV, used to alleviate the course of CMV infections after organ transplantation, is limited.

Other human herpesviruses

- HHV type 6 is a lymphotropic virus that infects T-lymphocytes. HHV-6 infection occurs in early childhood. Causes a sudden exanthema (roseola infantum) or "sixth disease", accompanied by a sudden increase in temperature on the background of exanthema.
- HHV type 7 infects in early childhood, persists in salivary glands, and is excreted in saliva. Presumably, HHV-7 causes chronic fatigue syndrome with low-grade fever, sweating, arthralgia and weakness.
- HHV-8 type was isolated in the study of tissue of patients with Kaposi's sarcoma in HIV-infected and was called herpes virus associated with Kaposi's sarcoma

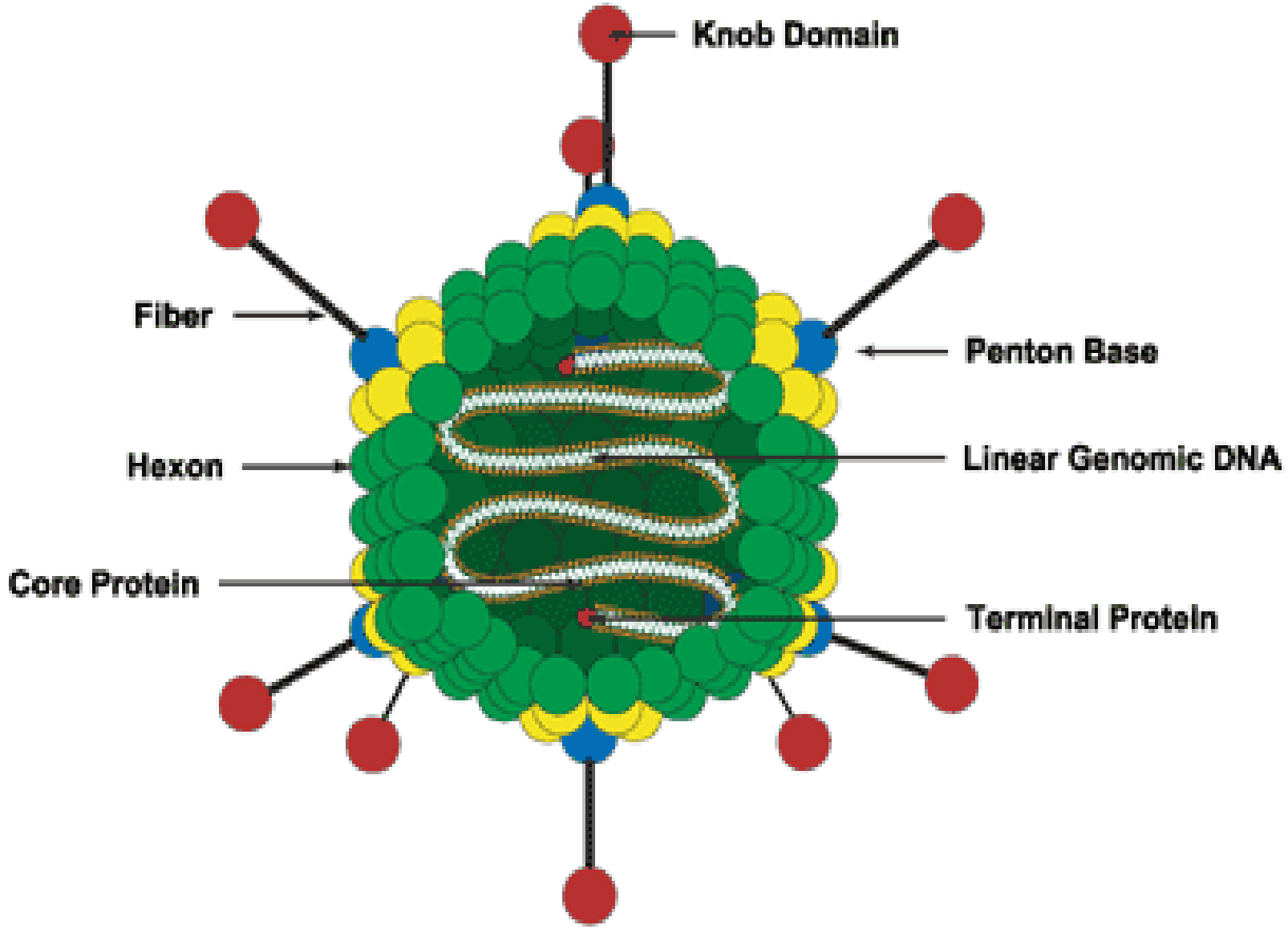




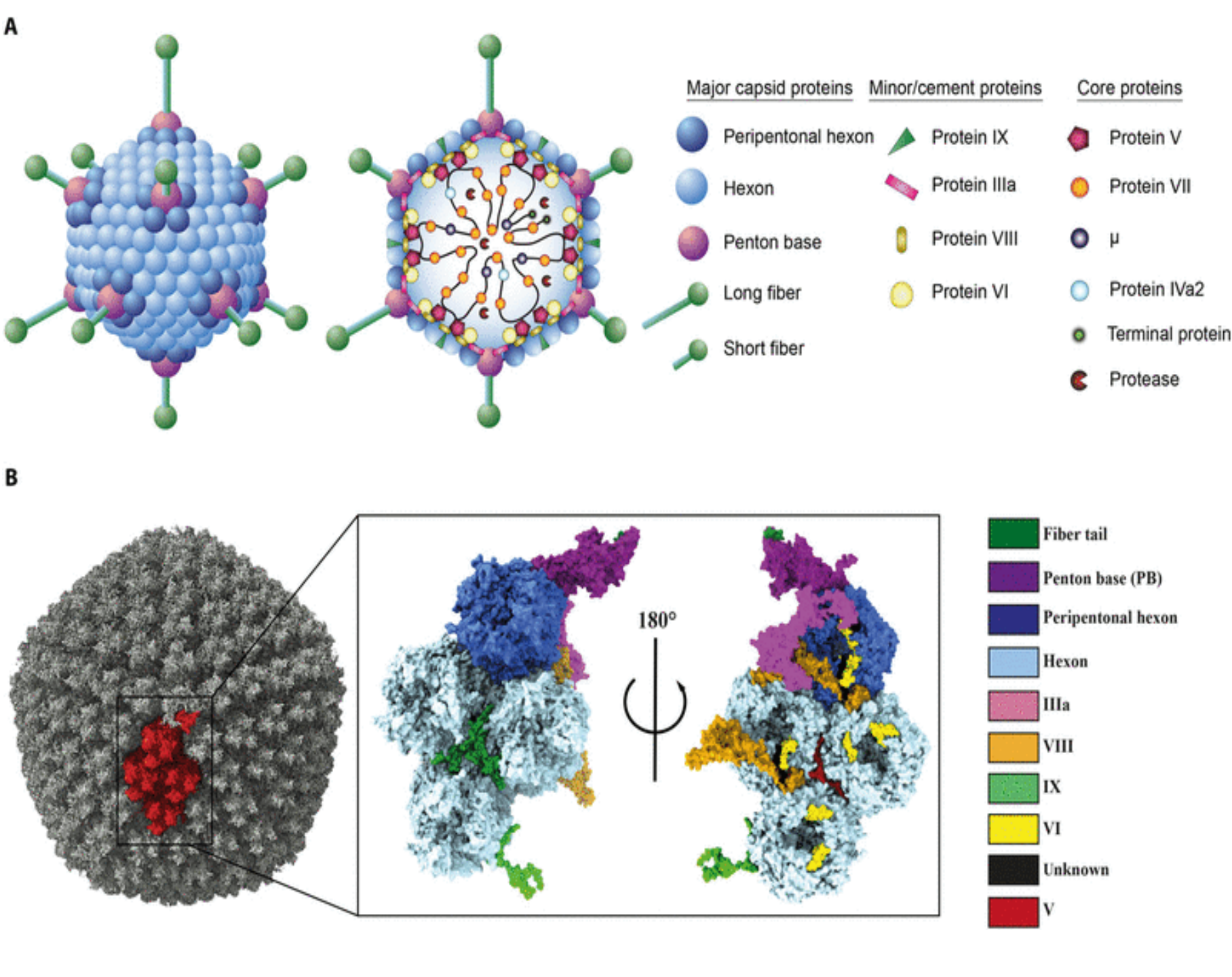
Adenoviruses

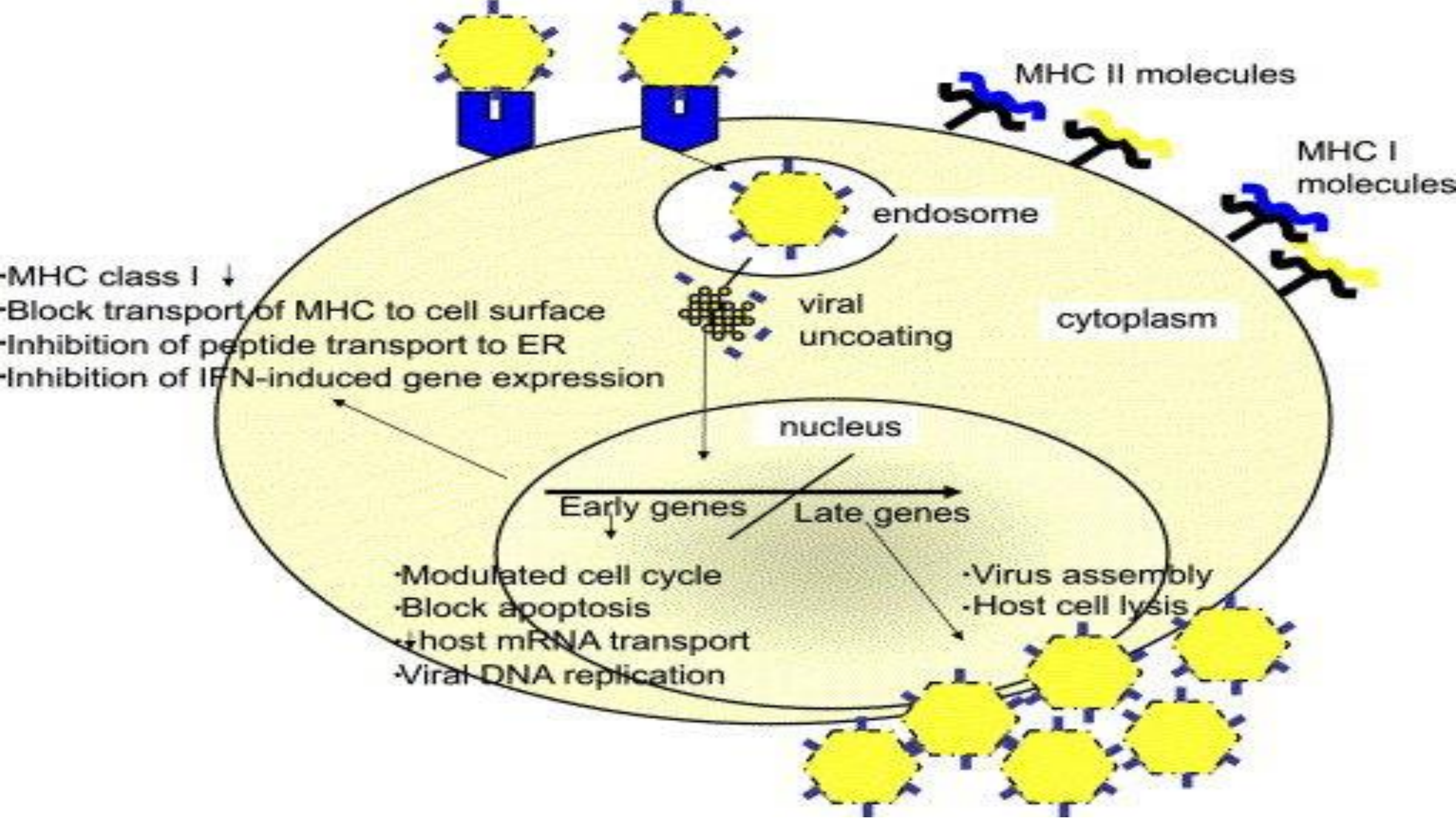
- The family includes 4 genera:
- Mastadenovirus (mammalian viruses)
- Aviadenovirus (avian viruses)
- Atadenovirus (genome enriched in A-T pairs)
- Siadenovirus (contains the sialidase gene)
- 49 out of 100 serotypes are pathogenic for humans
- Cause damage to the respiratory, gastrointestinal and visual systems

The virion does not have a lipid envelope, the icosahedral capsid consists of 252 capsomeres. The genome is the linear double-stranded DNA that forms the core of the virus. Adenoviruses cause latent or lytic infection



The capsid consists of hexons and 12 pentons. Penton consists of 5 protein molecules. A glycoprotein thread (fibril) departs from each penton, which performs the function of hemagglutinin and an attachment protein. The virus enters the cell by endocytosis.




MHC II molecules
MHC I molecules
endosome
viral uncoating
cytoplasm
nucleus
-MHC class I ↓
-Block transport of MHC to cell surface
-Inhibition of peptide transport to ER
-Inhibition of IFN-induced gene expression
Early genes
Late genes
·Modulated cell cycle
·Block apoptosis
·↓host mRNA transport
·Viral DNA replication
·Virus assembly
·Host cell lysis

Epidemiology

BOX 53-3. Epidemiology of Adenoviruses

Disease/Viral Factors

- Capsid virus is resistant to inactivation by gastrointestinal tract and drying.
- Disease symptoms may resemble those of other respiratory virus infections.
- Virus may cause asymptomatic shedding.

Transmission

- Direct contact via respiratory droplets and fecal matter, on hands, on fomites (e.g., towels, contaminated medical instruments), close contact, and inadequately chlorinated swimming pools.

Who Is at Risk?

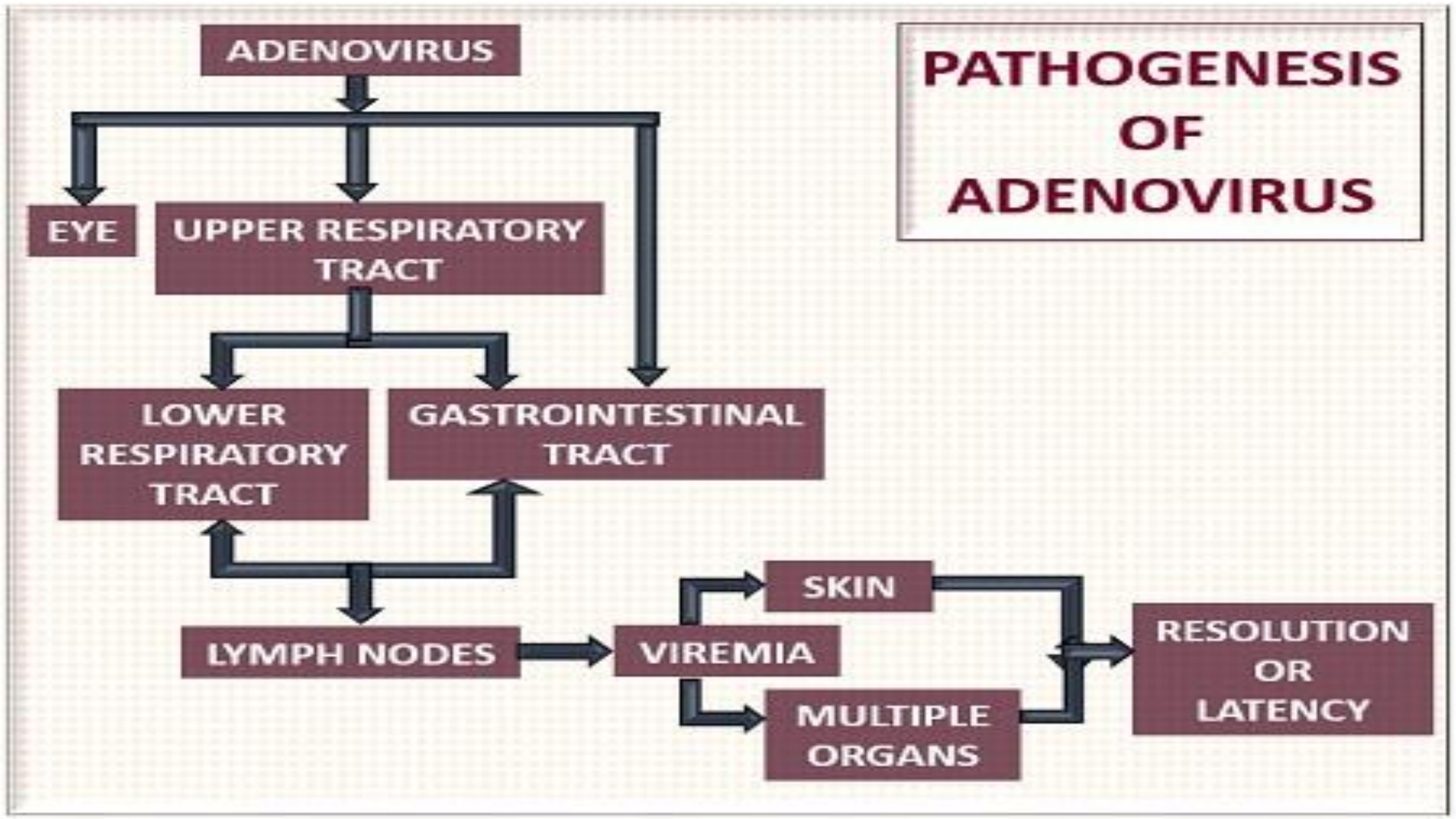
- Children younger than 14 years of age.
- People in crowded areas (e.g., daycare centers, military training camps, swimming clubs).

Geography/Season

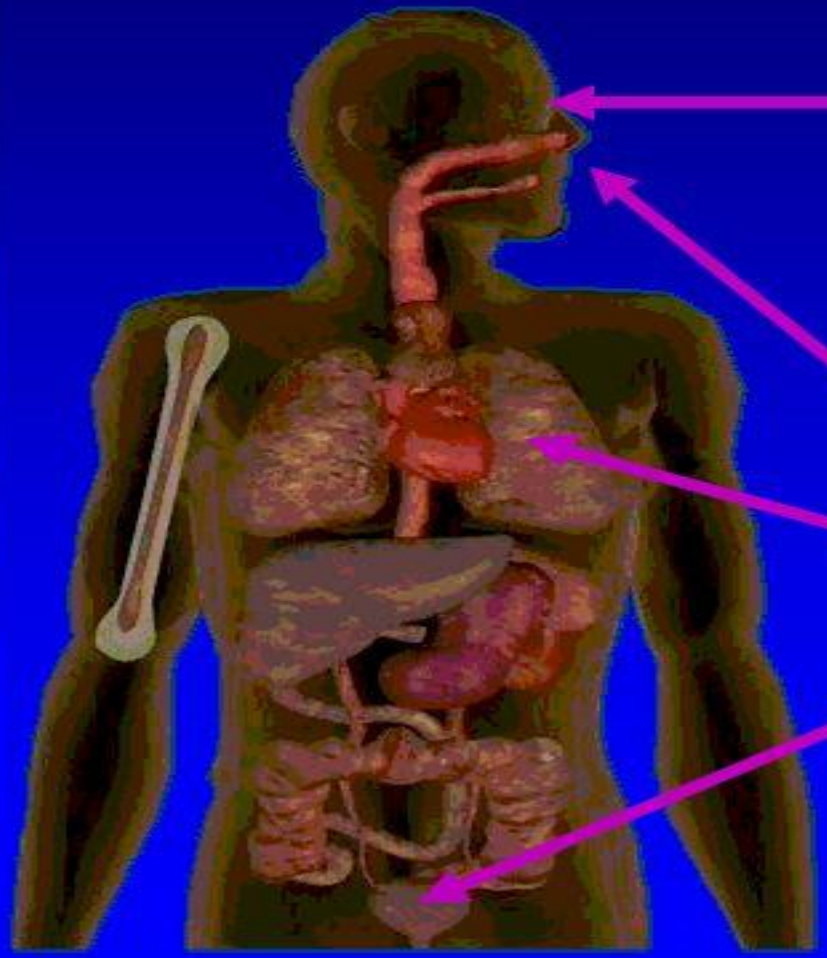
- Virus is found worldwide.
- There is no seasonal incidence.

Modes of Control

- Live vaccine for serotypes 4 and 7 is available for military use.


ADENOVIRUS
PATHOGENESIS OF ADENOVIRUS
EYE
UPPER RESPIRATORY TRACT
LOWER RESPIRATORY TRACT
GASTROINTESTINAL TRACT
LYMPH NODES
VIREMIA
SKIN
MULTIPLE ORGANS
RESOLUTION OR LATENCY

Clinical Manifestations



- ☑ Conjunctivitis
  - ↗ acute folicular conjunctivitis
  - ↗ epidemic keratoconjunctivitis
- ☑ Pharyngitis- acute febrile
- ☑ Pneumonia- sequalae
- ☑ Cystitis
- ☑ Infantile Gastroenteritis

CLINICAL SYNDROMES

- Adenoviruses primary infect children.
- Adults are also infected.
- More than one serotypes of virus may produce the same clinical syndrome and one serotype of virus may cause clinically different diseases.
- Adenoviruses 1-7 are the common serotypes worldwide and are responsible for most cases of adenovirus-associated infections.
- Incubation period varies from 5 to 8 days.
- The major clinical syndromes caused by the human adenoviruses are as follows.

RESPIRATORY INFECTIONS - cough, runny nose, fever, sore throat, pneumonia (10-20% of all pneumonias)

EYE INFECTIONS - serotypes 3 and 7 - pharyngoconjunctival fever, epidemic conjunctivitis, keratitis

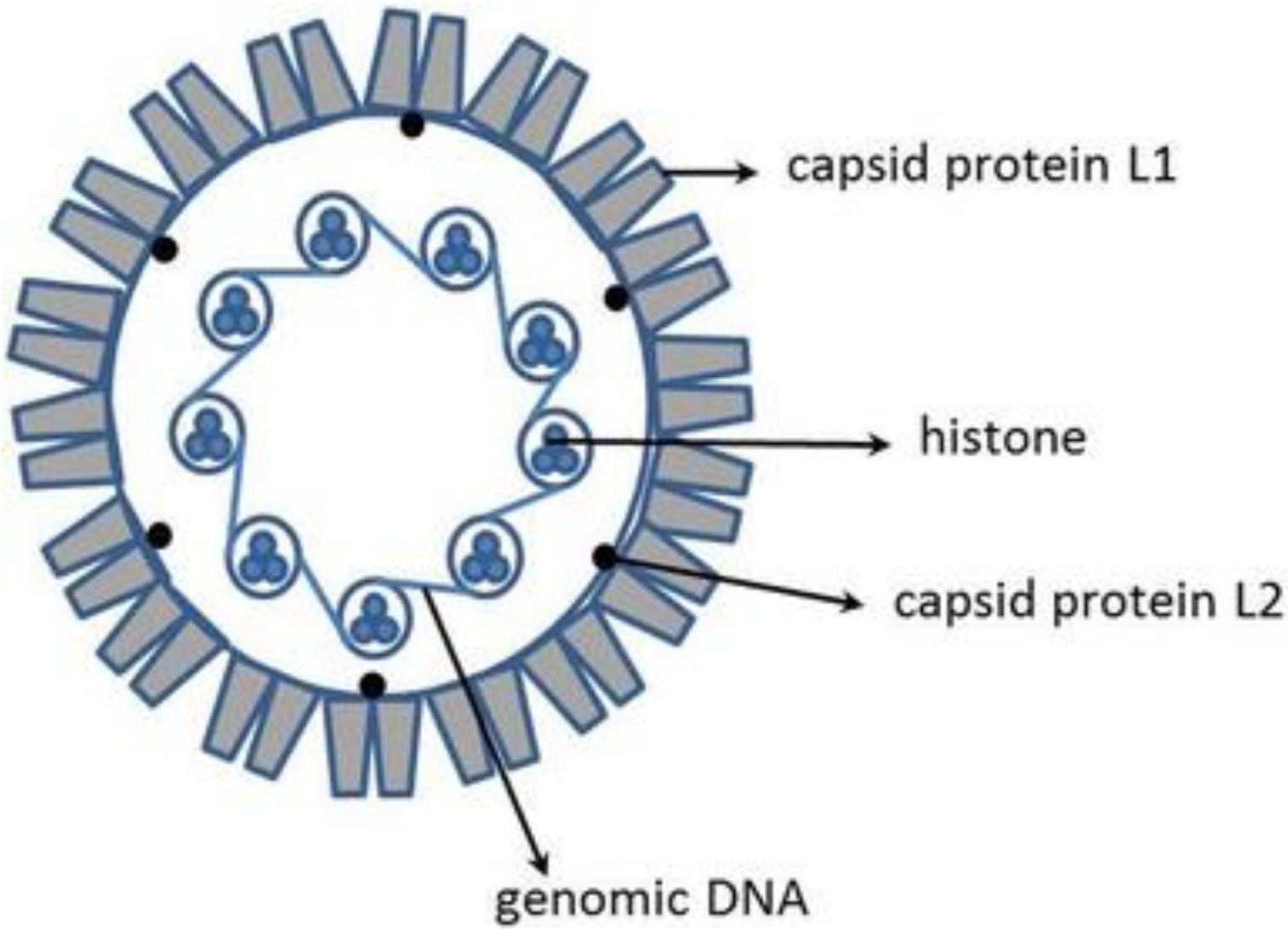
GASTROENTERITIS -40 and 41 serotypes

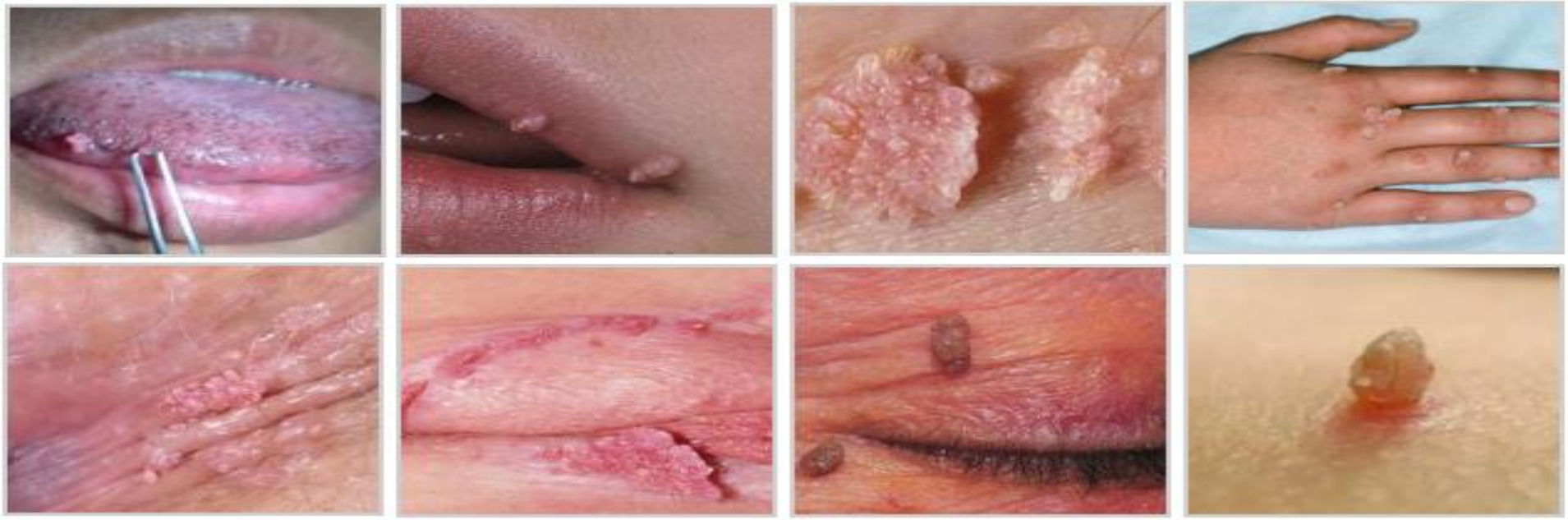
OTHER INFECTIONS - after transplantation, pneumonia may develop, in patients with AIDS, gastrointestinal infections

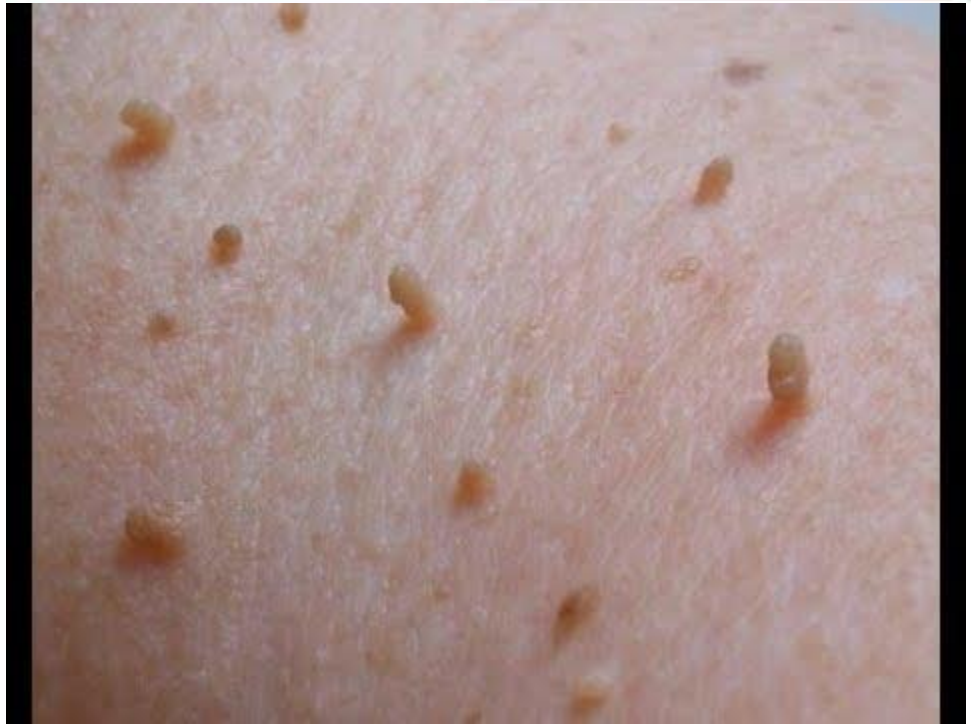
MICROBIOLOGICAL DIAGNOSIS

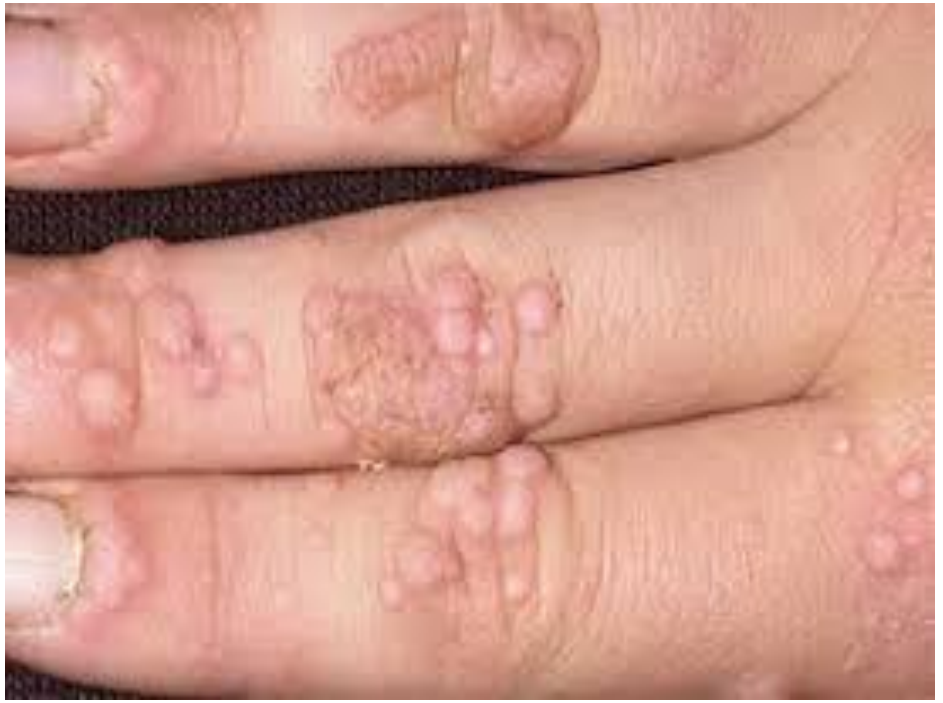
- Virological method: discharge of the nasopharynx, pharynx, conjunctiva, feces are cultivated in a culture of human epithelial cells (CPE, intranuclear inclusions - indication).
- Identification of viruses is carried out by RIF, ELISA, RSK, RN
- Serological method: determine the increase in the titer of antibodies in the blood serum using RSK, RN, RNGA
- Virus DNA is detected by PCR

Family Papillomavidiae

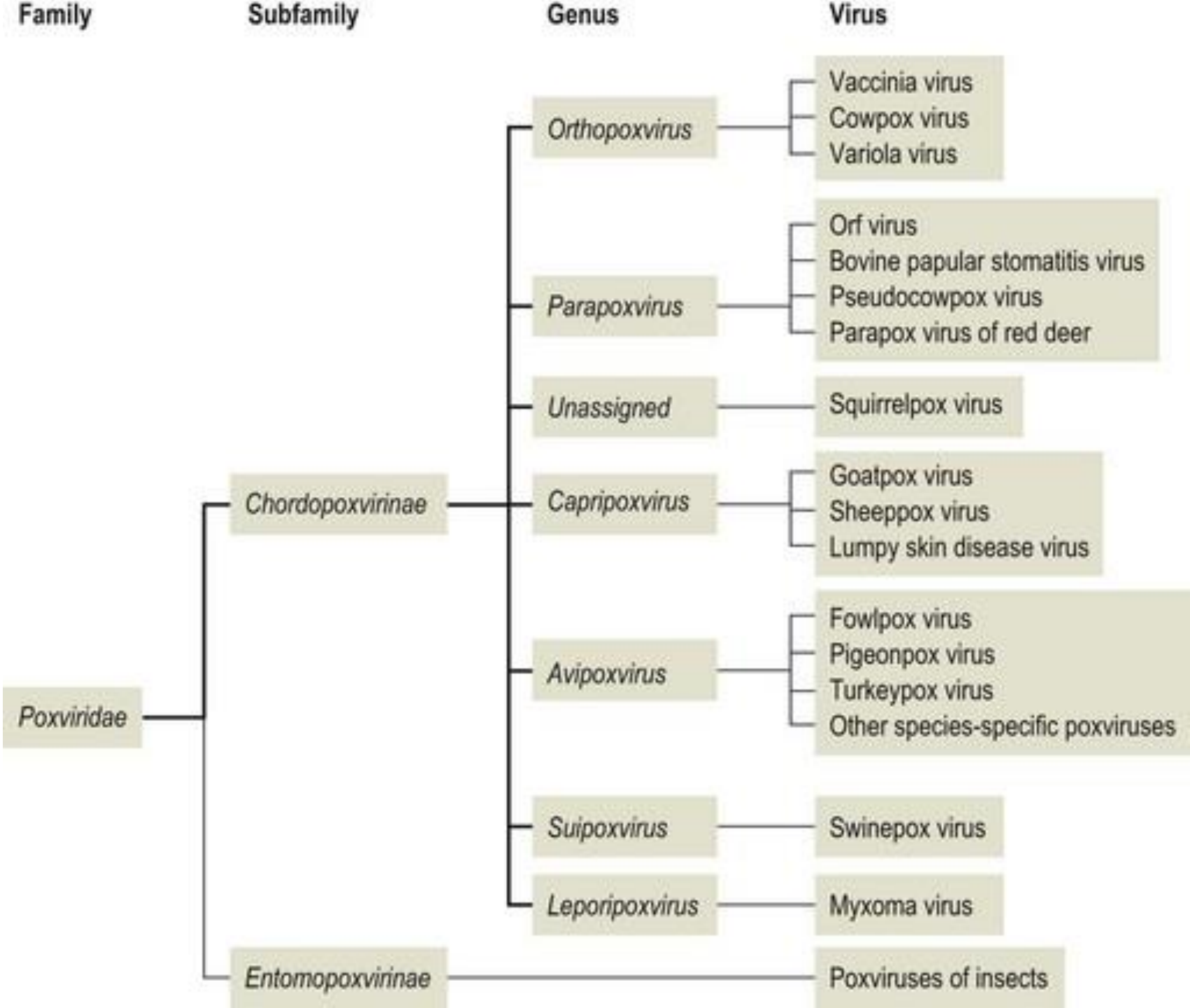






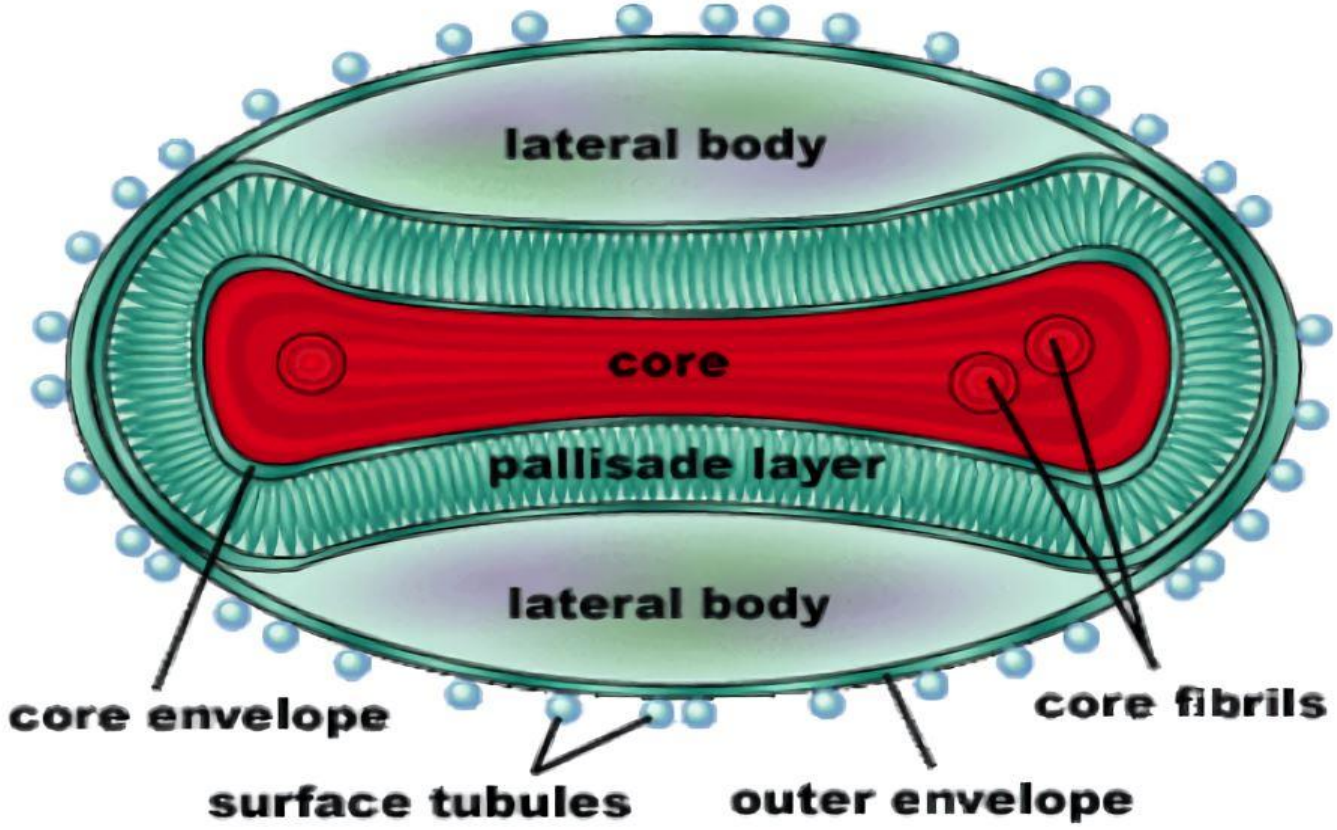


Poxviruses (Poxviridae from English pox - smallpox + viruses) - a family of large DNA-containing viruses, including two subfamilies: Chordopoxvirinae - Vertebrate pox viruses (Table 16.6), Entomopoxvirinae - insect pox viruses. Family contains variola viruses, vaccines, monkeypox and etc

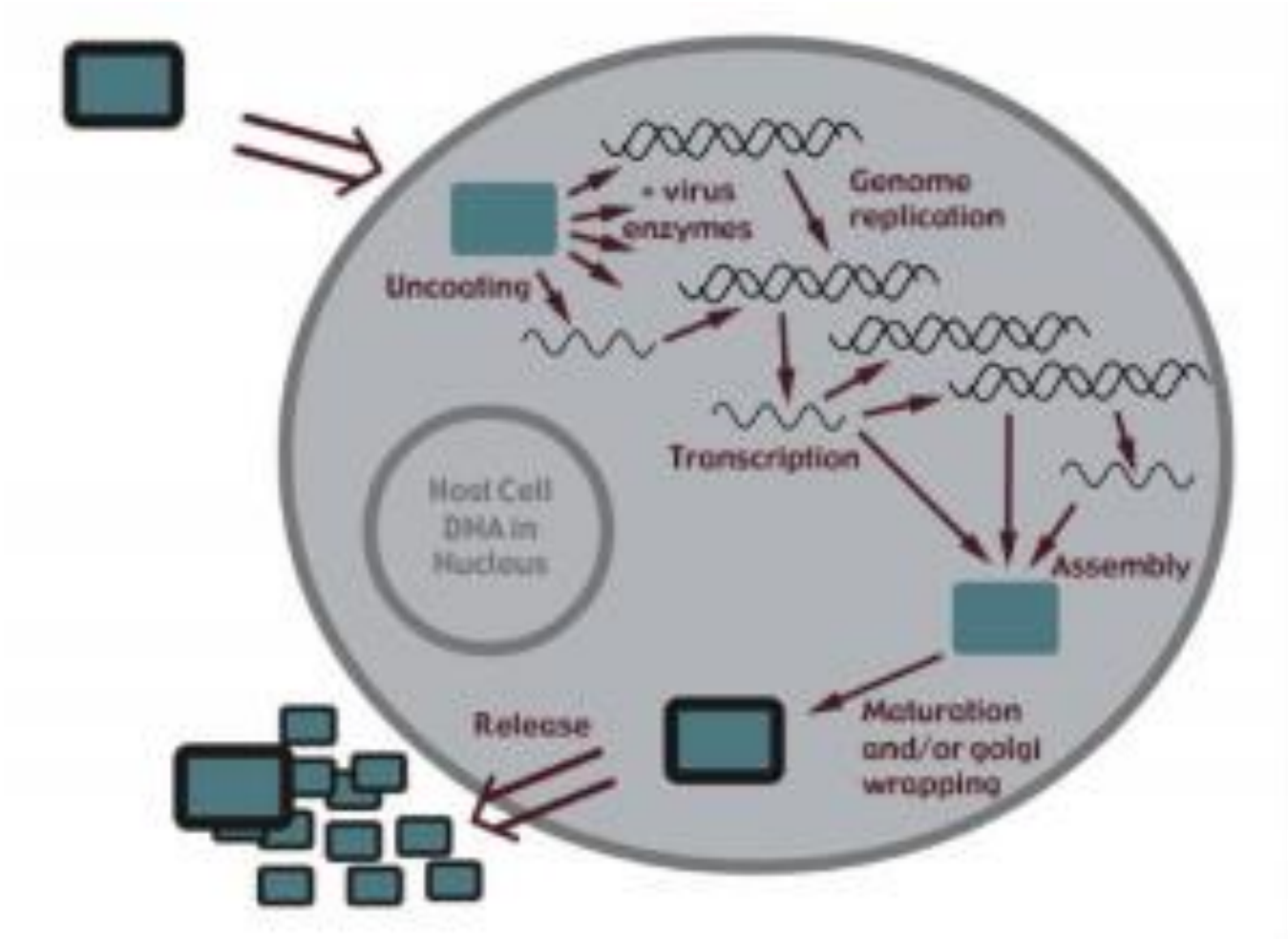


Smallpox

The structure of a virus particle

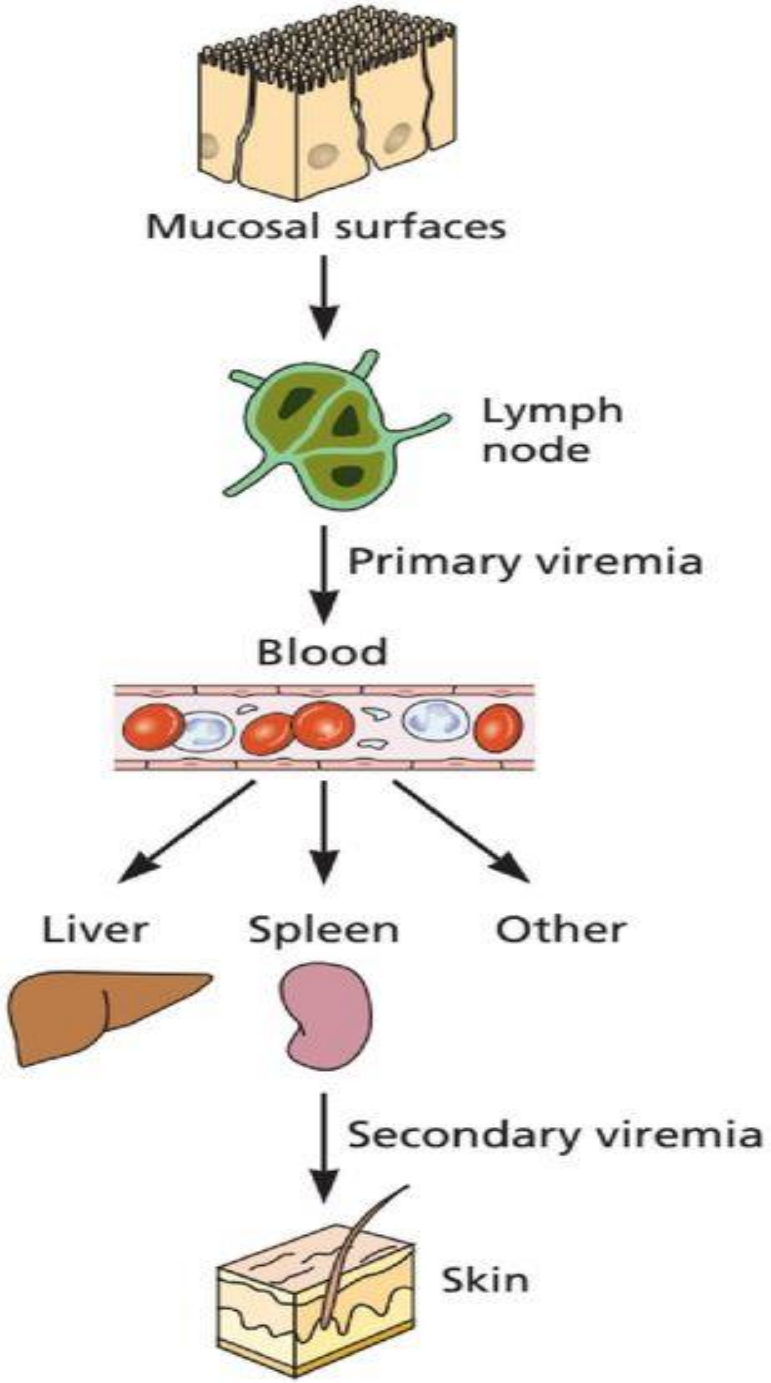


Smallpox virus life cycle



Pathogenesis

- After being inhaled, smallpox virus replicates in the upper respiratory tract.
- Dissemination occurs via lymphatic and cell-associated viremic spread.
- Internal and dermal tissues are inoculated after a second viremia, causing the simultaneous eruption of the characteristic pocks.
- Molluscum contagiosum and the other poxviruses, however, are acquired through direct contact with lesions.



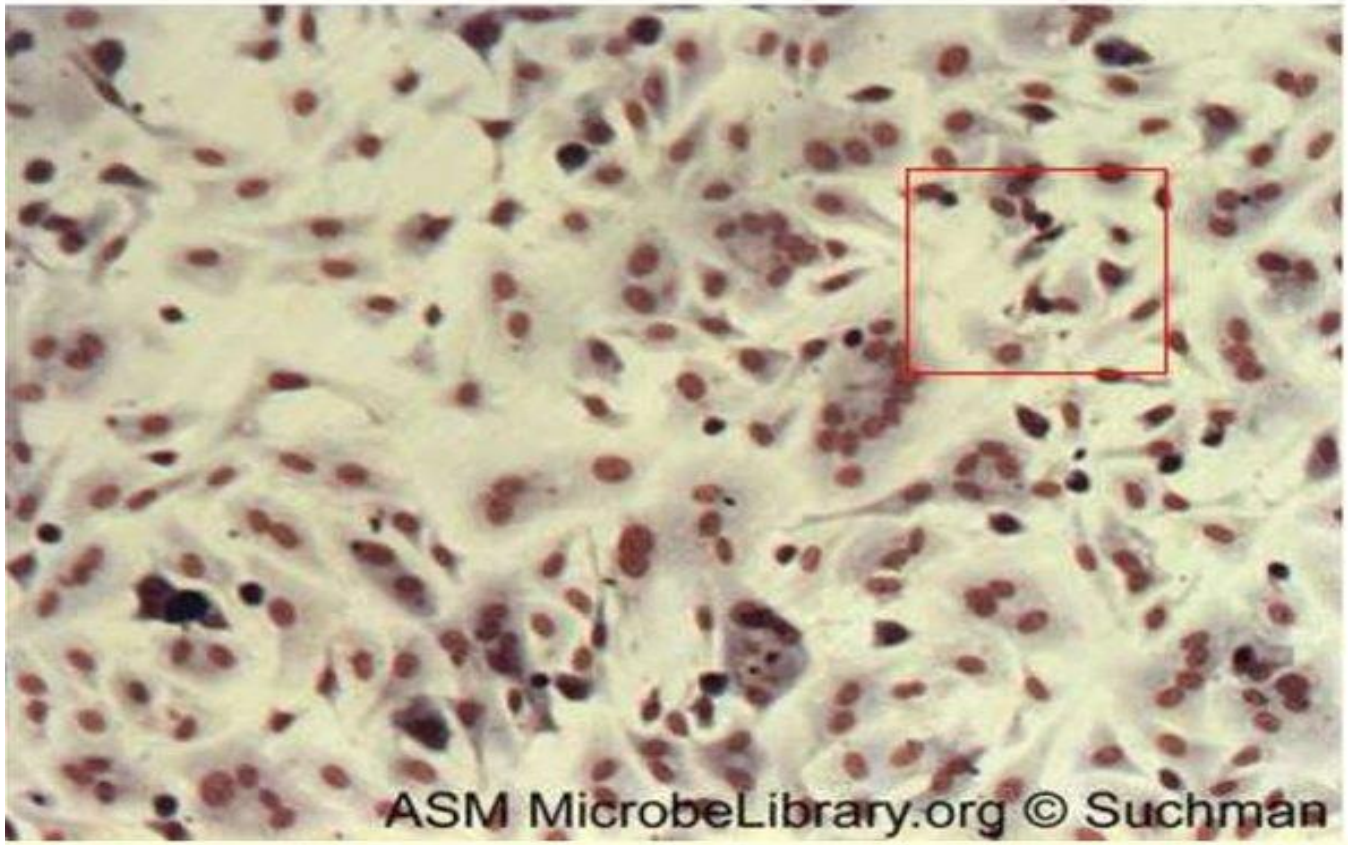
Smallpox Prodrome

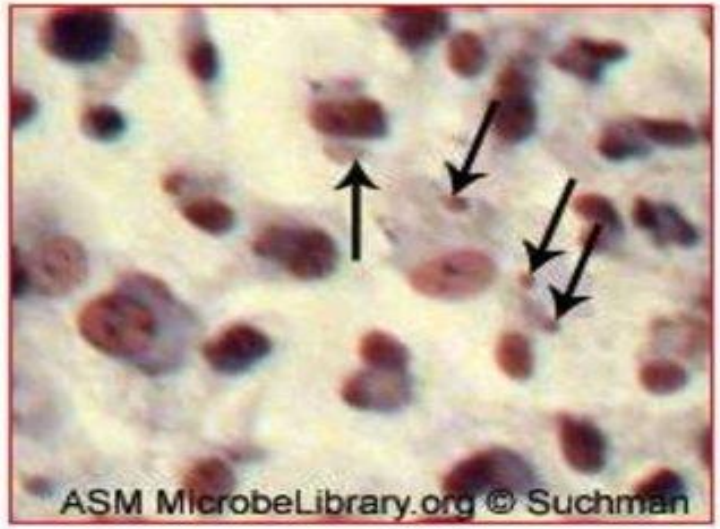
- Incubation period 12 days (range 7-19 days)

- Prodrome
  - abrupt onset of fever ≥101°F
  - malaise, headache, muscle pain, nausea, vomiting, backache
  - lasts 1-4 days

MINNESOTA MDH DEPARTMENT OF HEALTH

Taurus Guarnieri





Area of red callout box has been enlarged via Photoshop to better view CPE.

Monkeypox virus

The infectious disease caused by the monkeypox virus is characterized by intoxication, fever, and a pustular-papular rash.

The virus was isolated in 1958 from sick monkeys, and in 1970 from a sick child. The virus mainly infects squirrels, porcupines and rodents in Africa. The disease occurs in Africa in villagers who come into contact with animals.

Monkeypox virus is pathogenic to humans, although human susceptibility is relatively low.

Monkeys are the source of infection for humans.

The contagiousness of a sick person is low. The pathogen transmission mechanism is airborne.

A variant of this virus was introduced into the United States in 2003 not with monkeys, and with popular exotic animals - Gambian rats and so called prairie dogs.

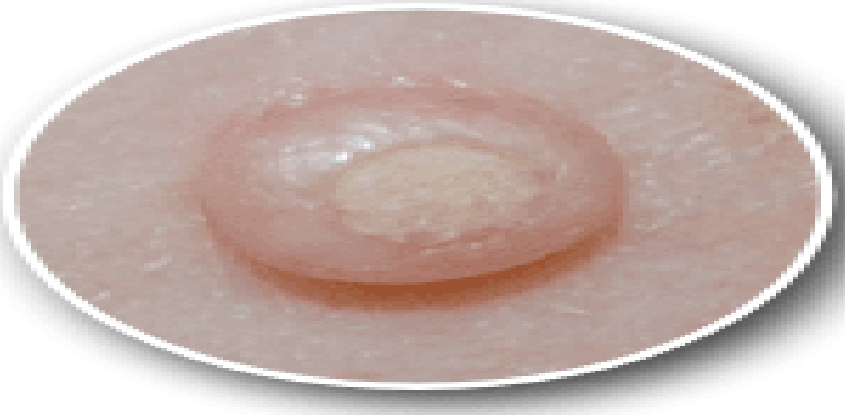
The virus was transmitted by contact these animals.

Monkeypox virus

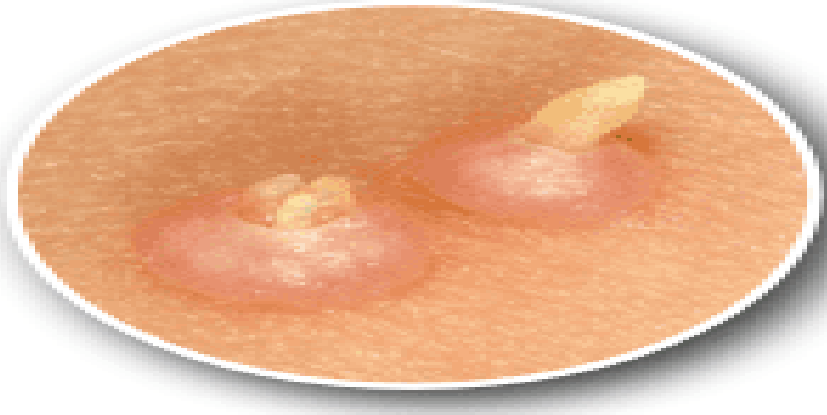
Molluscipoxvirus

- Существует 4 типа вируса контагиозного моллюска (MCV-1, MCV-2, MCV-3, MCV-4). Из них наиболее распространены MCV-1 и MCV-2 (только половой путь передачи)
- Заражение через воду (например, бассейн), общие предметы быта, игрушки у детей
- Инкубационный период болезни продолжается от 2 недель до 2-9 месяцев
- Лечение – использование комплексной противовирусной терапии, удаление высыпаний.

Molluscum Contagiosum



Umbilicated



Central keratin plug

Poxvirus

Clinical

- Most common in school-age children
- Transmitted by direct contact
- Painless, no systemic symptoms
- Typically affects face, torso, extremities
- Spares the palms and soles

Management

- Resolves spontaneously (no therapy needed)
- Cryotherapy
- Curettage
- Cantharidin
- Podophyllotoxin