

# Viral Infections of the Urogenital Tract

Dr. Hala Mahmoud Altarawneh



# Outlines

- Definition, Etiology, transmission, clinical features, diagnosis and treatment of :
  - Herpes simplex virus (HSV)
  - Human papilloma virus (HPV)
  - Molluscum contagiosum virus (MCV)



# Genital herpes



# Genital herpes: **Introduction**

- Herpes simplex virus (HSV) infections can be caused by herpes simplex virus type 1 (HSV-1) and herpes simplex virus type 2 (HSV-2).
- **Epidemiology:**
  - More than 90% of the world's population over the age of 40 years carries HSV.

# Genital herpes: Etiology

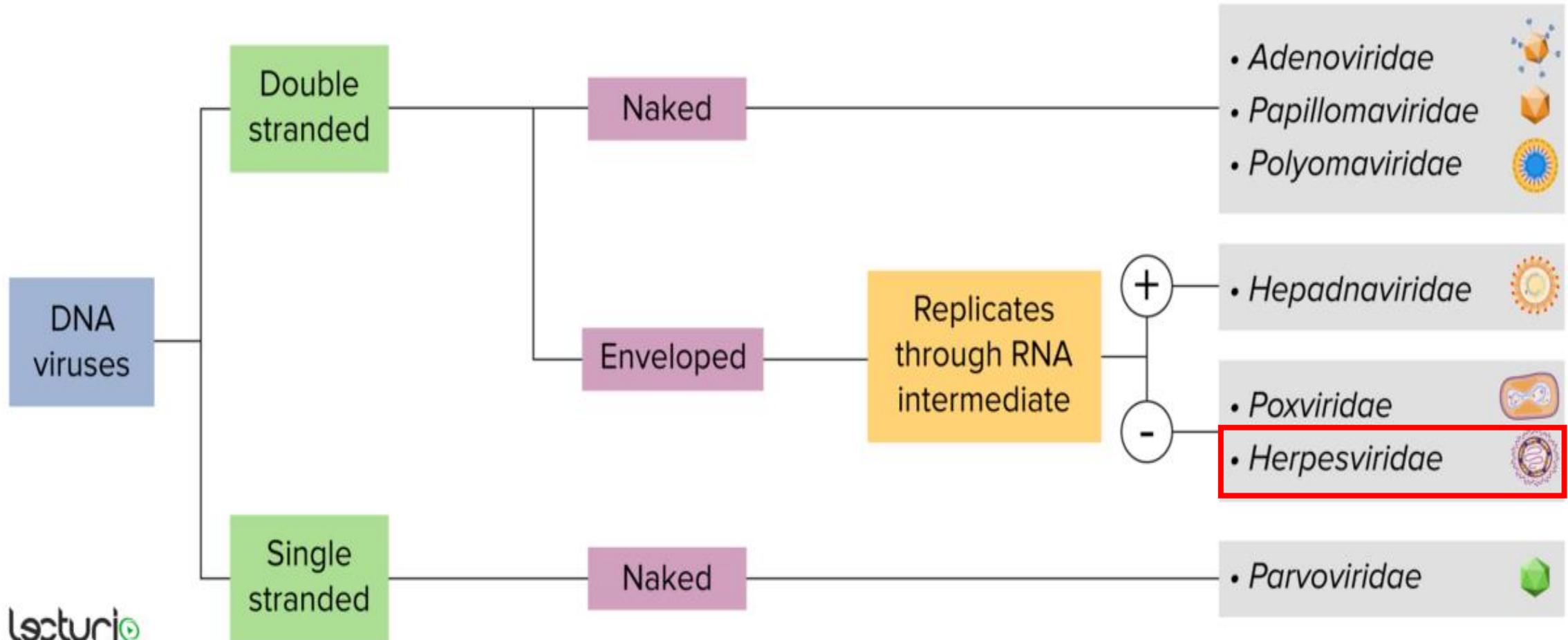
- **Basic features of herpes simplex virus:**

- Family: **Herpesviridae**
- **Double stranded DNA virus**
- **Envelope** and glycoprotein spikes
- **Icosahedral nucleocapsid**

- **Types:**

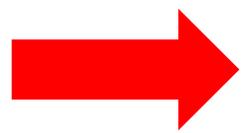
- Herpes simplex virus type 1 (HSV-1), (tropism for **oral epithelium**)
- Herpes simplex virus type 2 (HSV-2), (tropism for **genital epithelium**)

# Genital herpes: Etiology



# Genital herpes: Etiology- Transmission

- Direct contact with mucosal tissue or secretions of another infected person
- Infection with **HSV-1** usually is acquired in **childhood via saliva**.
- **HSV-2** is mostly spread through **sexual contact**



Therefore, the detection of **HSV-2** in children should **↑ raise suspicion** for **sexual abuse**

# Genital herpes: Etiology- Pathophysiology

1. Inoculation: The virus enters the body through mucosal surfaces or small dermal lesions.
2. Neurovirulence: The virus invades, spreads, and replicates in **nerve cells**.
3. Latency: After primary infection, the virus remains dormant in the ganglion neurons.
  - Trigeminal ganglion: HSV-1
  - Sacral ganglion: HSV-2
4. Reactivation: triggered by various factors (e.g., immunodeficiency, stress, trauma) → clinical manifestations

# Genital herpes: Etiology- Pathophysiology



Inoculation

Neurovirulence:

The virus invades, spreads, and replicates in nerve cells.

Latency:  
the virus remains dormant in the ganglion neurons (Sacral ganglion)

Reactivation:  
triggered by various factors (e.g., stress, trauma) → clinical manifestations.

# Genital herpes: **Clinical features**

- Affected individuals are **often asymptomatic or have mild symptoms** but may still be at risk of transmission.
- **Primary infection:**
  - **Prodromal symptoms:** redness, swelling, tingling, pain, pruritus
  - Genital tract: skin lesions in the anogenital area, cervicitis, white, thick, and/or foul-smelling vaginal discharge
  - Grouped **erythematous vesicles** that progress to **painful ulcers** in the anogenital area
  - **Associated symptoms:** fever, headaches, myalgias, malaise, tender bilateral inguinal lymphadenopathy

# Genital herpes: **Clinical features**

- **Recurrent infection:**
  - Prodromal symptoms (lasting hours to days): pain or tingling in the genitals, legs, buttocks, and/or hips
  - Skin lesions are usually unilateral, less painful, and of shorter duration than in the initial infection.

# Genital herpes: **Diagnosis and Treatment**

- **Diagnostics:** clinical diagnosis of HSV infection or reactivation.
  - Confirm diagnosis with PCR and/or viral culture in patients with suspected infection or reactivation regardless of symptoms.
- **Treatment:** Acyclovir
  - Antiviral treatment effect: Decrease in duration and severity of infection, Reduction of viral shedding, However, recurrence **cannot be prevented.**





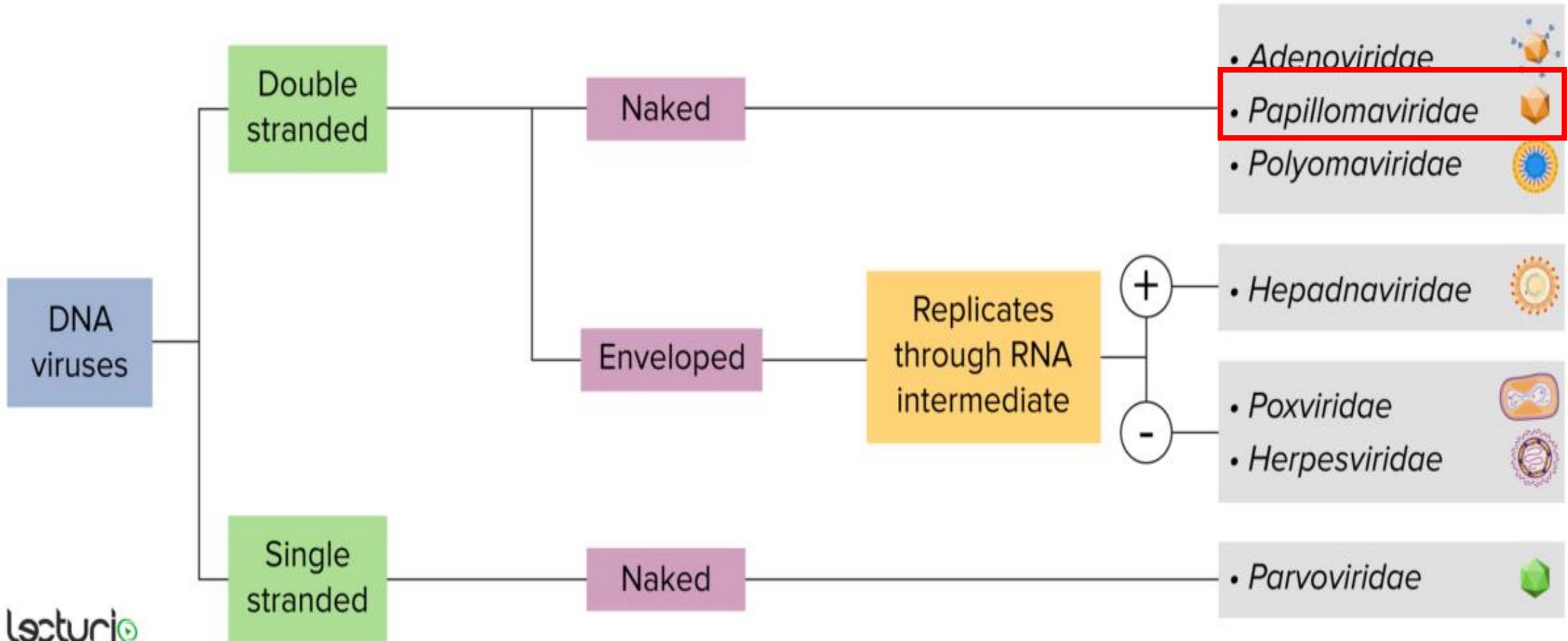
# Human papillomavirus infection (HPV)



# HPV: Introduction

- The human papillomavirus (HPV) is a nonenveloped DNA virus that **infects** the **cutaneous and mucosal epithelium**.
- Basic features of human papillomavirus:
  - Family: **Papillomaviridae**
  - **Double stranded DNA** virus, Circular genome
  - Structure: **Nonenveloped**, **Icosahedral capsid**

# HPV: Etiology

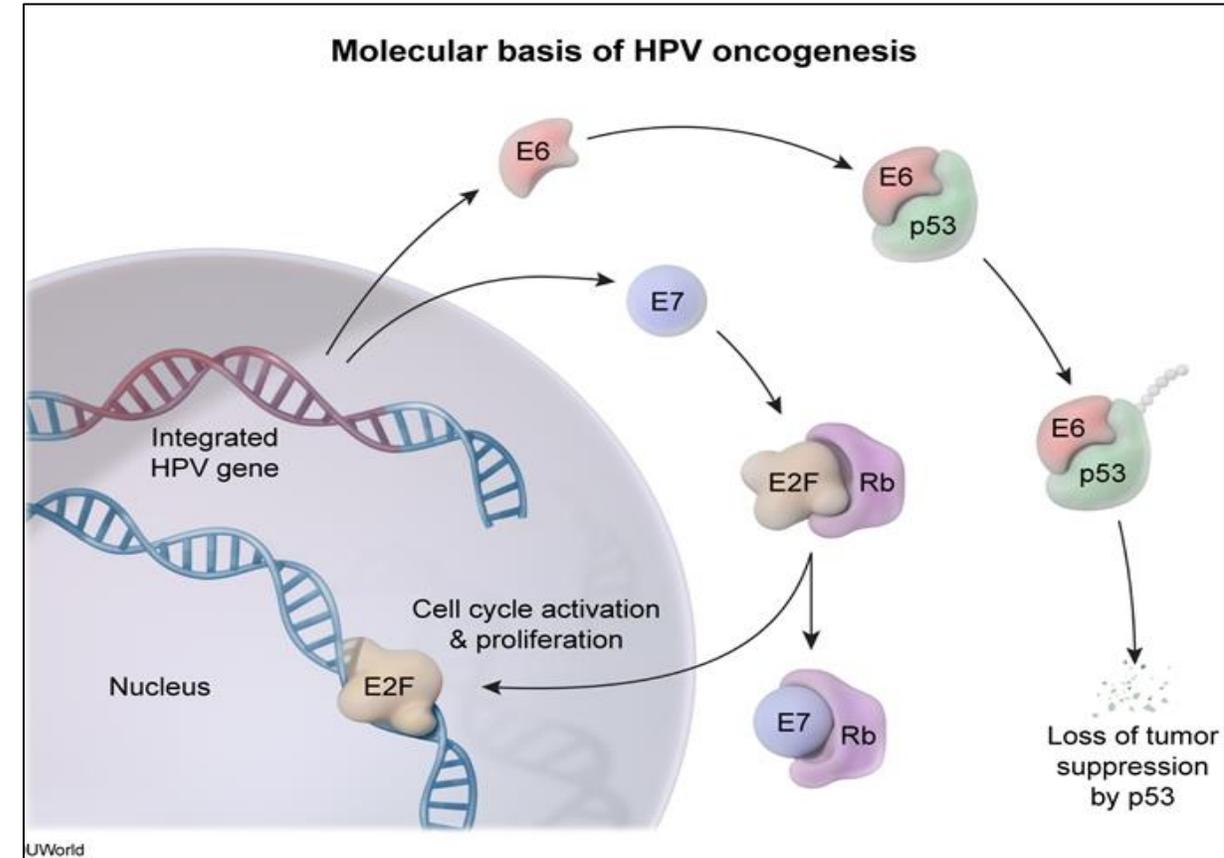


# HPV: Etiology

- **Routes of transmission:**
  - Direct contact (e.g., sexual activity, autoinoculation)
  - Fomites
- **Clinically relevant species**
  - There are > 200 HPV serotypes, some infect the cutaneous epithelium, and others infect the mucosal epithelium.

# HPV: Pathophysiology

- HPV DNA integrate into the host genome and subsequently produce viral proteins E6 and E7
- E6 and E7 interact with cell cycle regulatory proteins p53 and retinoblastoma protein (Rb), respectively
- Without p53, the cell is unable to stop cell growth to repair damaged DNA or trigger apoptosis when DNA is damaged beyond repair
- Similarly, E7 binds Rb promoting unregulated DNA replication.
- The collective effects of E6 and E7 lead to inhibition of cell cycle regulation and evasion of apoptosis, consequently increasing malignant potential.



# Human papillomavirus

- Low-risk HPV types 6 and 11:
  - Anogenital warts (condylomata acuminata)
  - Mild cervical cell abnormalities
  - Tumors of non-genital mucosal membranes (e.g., respiratory tract, oral cavity)
- High-risk HPV types 16, 18, 31, and 33
  - Cervical cancer (responsible for 70% of cases)
  - High risk of anogenital, oral, and oropharyngeal squamous cell carcinoma
- HPV types 1, 2, and 4: cause skin warts, such as common warts and plantar warts

# Non-anogenital manifestations:

- **Common warts:** Lesions are plaques or papules, Skin-coloured or whitish usually firm, often with a rough and scaly surface, located on the elbows, knees, fingers, and/or palms.
- **Plantar warts:** Rough, hyperkeratotic lesions on the sole of the foot often grow inwardly and cause pain while walking.
- **Flat warts:** Multiple small, flat patches or plaques localized on the face, hands, and shins.

# Non-anogenital manifestations:



# HPV: Diagnosis and Treatment

- **Diagnosis:** Cutaneous warts and anogenital warts are usually diagnosed clinically.
- **Management:**
  - There is **no cure for HPV** infections.
  - Most HPV infections in immunocompetent individuals resolve spontaneously within 2 years.
  - Management is based on clinical manifestations of HPV; options include observation, topical pharmacotherapy, cryotherapy, laser therapy, and surgical excision.

# HPV: Vaccination

- HPV vaccine: The human papillomavirus 9-valent vaccine protects against HPV types which cause anogenital warts and HPV-related cancers.
- All individuals between 11 and 12 years of age, preferably before first sexual intercourse in USA



# Molluscum contagiosum



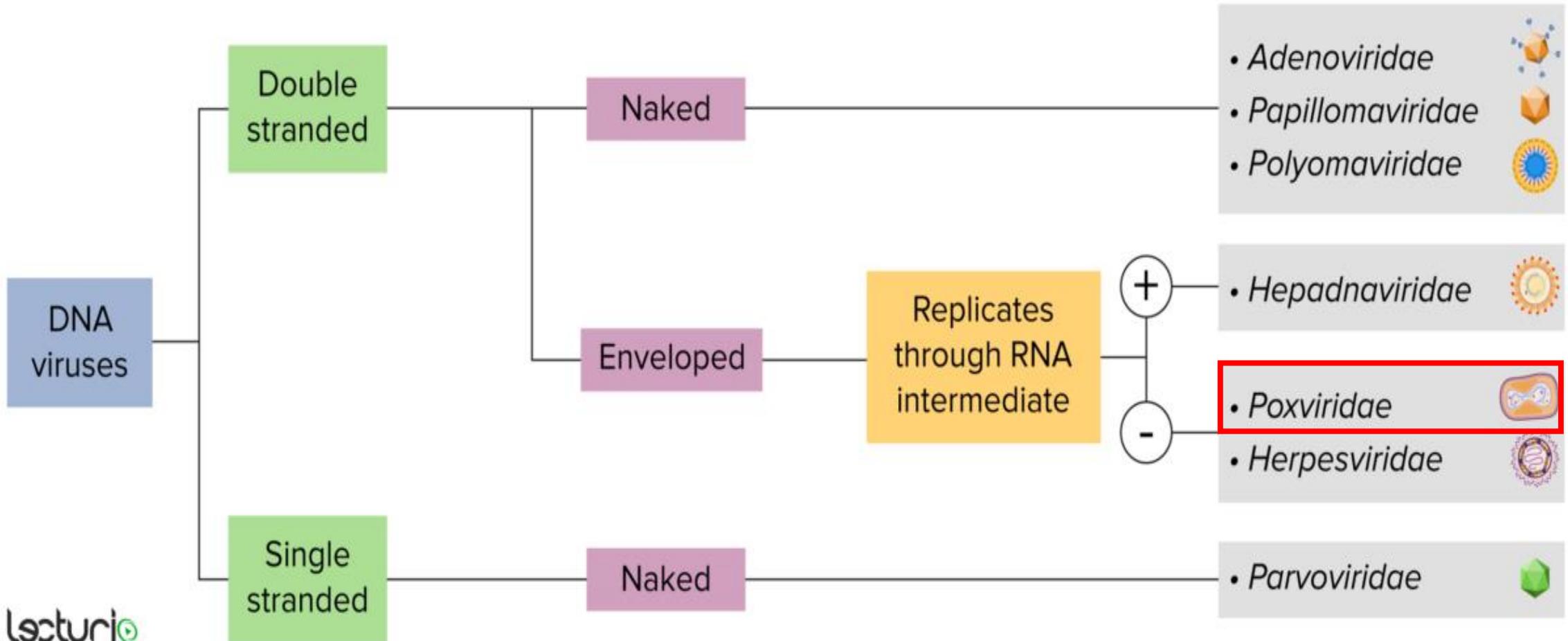
# MCV: Introduction

- Molluscum contagiosum is a common skin infection caused by the molluscum contagiosum virus (MCV).
- **Epidemiology:**
- Age: most common in childhood (peak incidence < 5 years of age) and early adolescence

# MCV: Etiology

- **Pathogen:** Molluscum contagiosum virus (MCV) is an enveloped double-stranded, linear, DNA poxvirus.
- **Transmission:**
  - Direct skin contact (e.g., through contact sports, sexual contact)
  - Autoinoculation (from scratching, shaving, or touching)
  - Fomites (e.g., on bath sponges or towels)
- Incubation period: typically, **2–7 weeks**

# MCV: Etiology



# MCV: Clinical features

- **Appearance:**

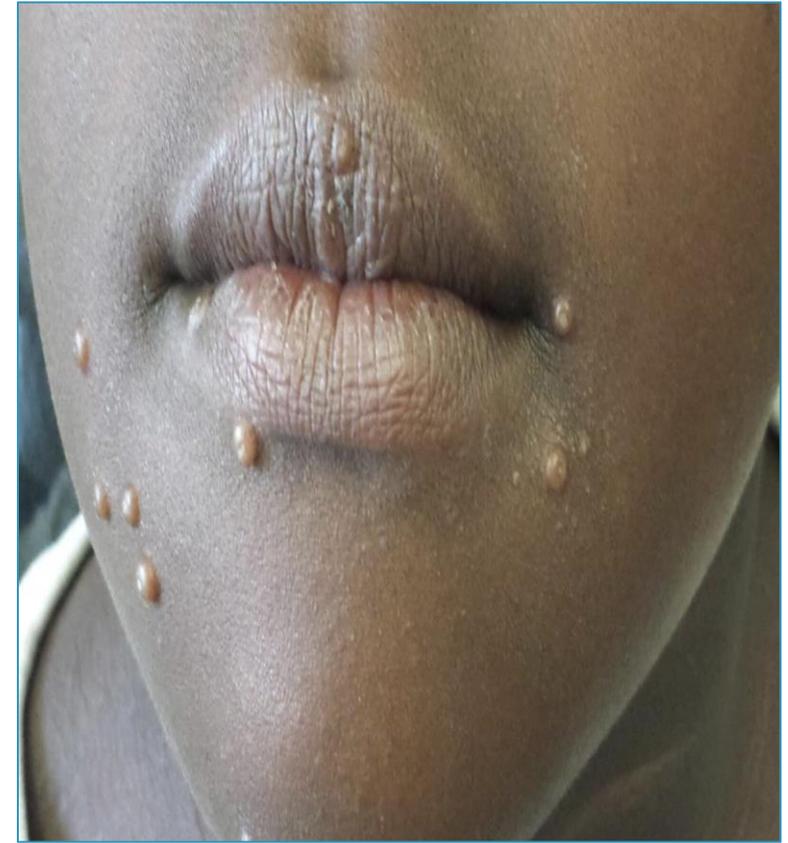
- Solitary or multiple nontender, skin-colored, pearly, dome-shaped papules with central umbilication
- Usually 2–5 mm in diameter

- **Typical distribution:**

- In children: face, trunk, and extremities
- In adults or in sexually transmitted cases: lower abdomen, groin, genitalia, and proximal thighs



# MCV: Clinical features



# MCV: Diagnosis and Treatment

- **Diagnosis:** Clinical diagnosis is sufficient.
- **Treatment:**
  - **No treatment is necessary** for healthy individuals as it is a self-limiting disease.
  - Complete resolution can take up to 1 year.
  - Management for **cosmetic reasons** or to **reduce transmission**:
    - Physical destruction: cryotherapy, curettage
    - Topical agents for chemical destruction: topical cantharidin

# MCV: Infection control measures

- Measures that can reduce the possibility of spread to others include **avoidance of:**
  - Scratching, shaving over, or picking at lesions
  - Sharing towels or bed linen
  - Skin-to-skin contact (cover lesions during contact sports and swimming)

Thank you