UNIWERSYTET MEDYCZNY
IM. PIASTÓW ŚLĄSKICH WE WROCŁAWIU

Subject: Faculty Lectures on Virology
Topic: Sexually Transmitted Viruses and Their Adverse Effects on the Mother, Fetus, and Newborn

Academic Year 2024/2025

Faculty: Medicine
Field of study: Virology
Level of study (uniform MA):
Form of study (full time):
Year of study: III

Academic title/professional title: professor
Name, last name of the lecturer: Beata Sobieszczańska
Position of person conducting classes: teacher
Wroclaw Medical University

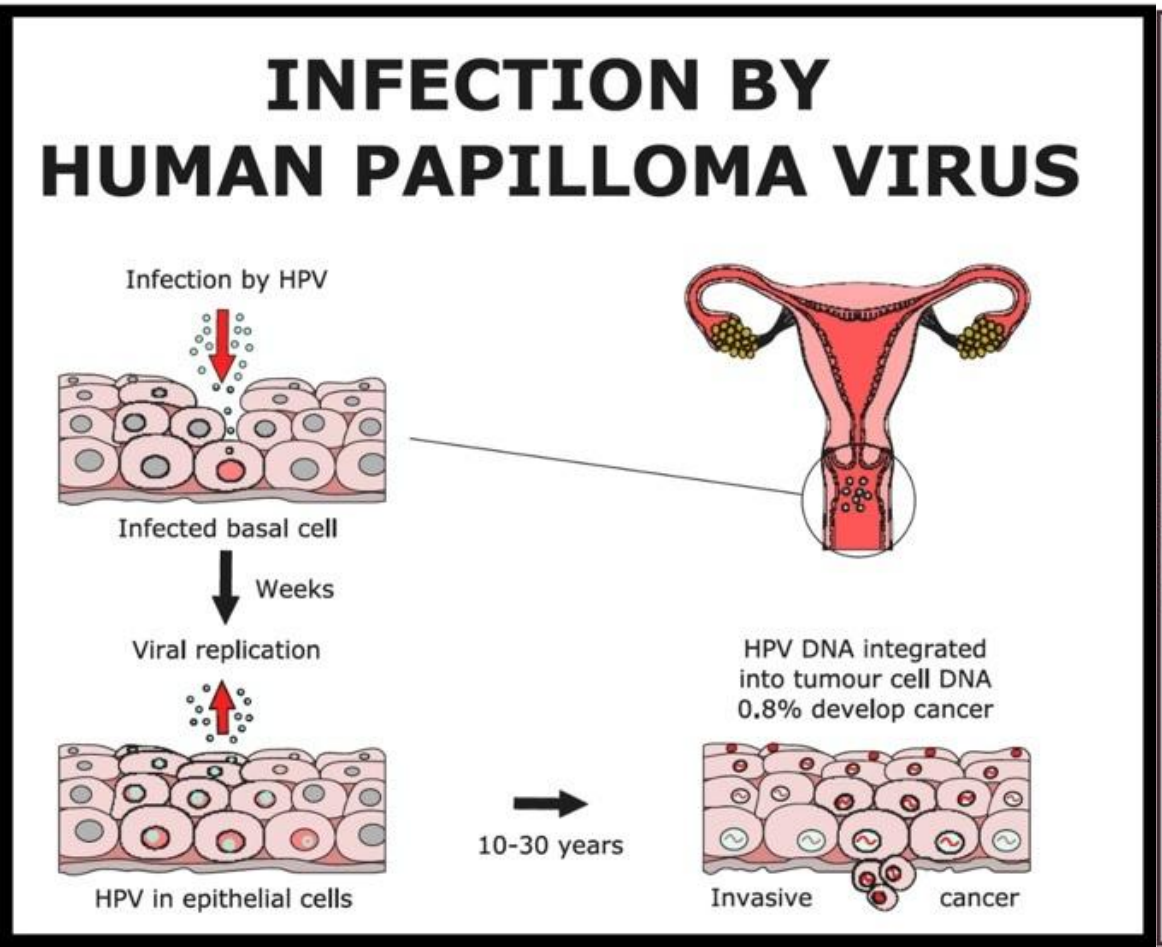
Human Papilloma Virus (HPV)

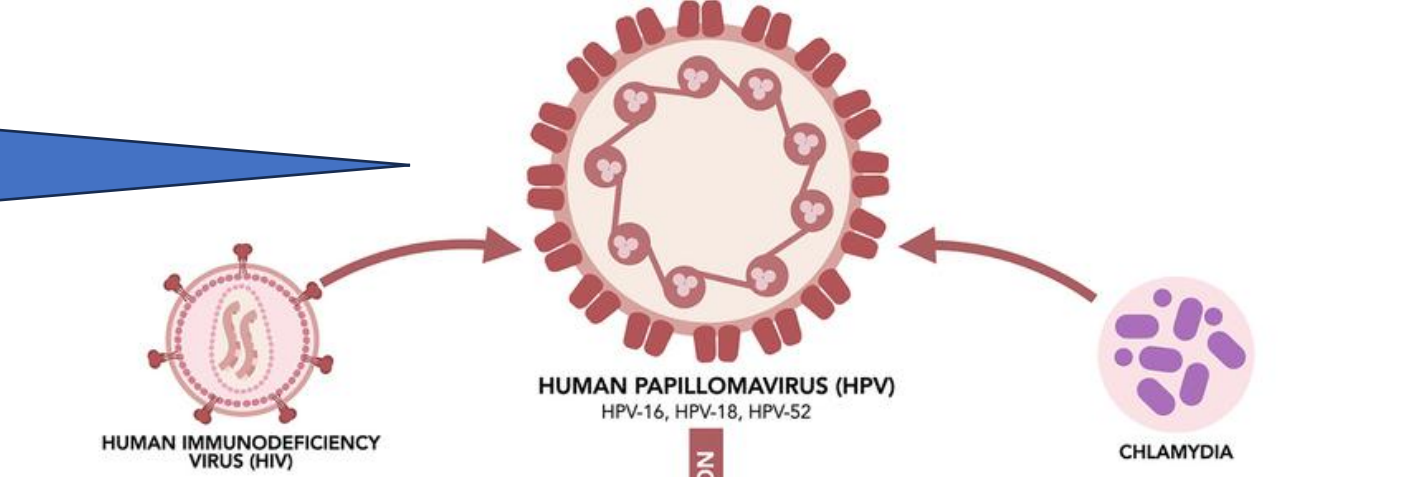
You don't have to have sex to get an HPV-associated STD - skin-to-skin contact is enough to spread HPV

Vaccines can protect against some of the most dangerous types of HPV

Signs: Pink or flesh-colored warts raised, flat, or shaped like cauliflower - often, there are no symptoms

Coinfection of HPV with chlamydia and HIV exacerbates the risk of cervical cancer





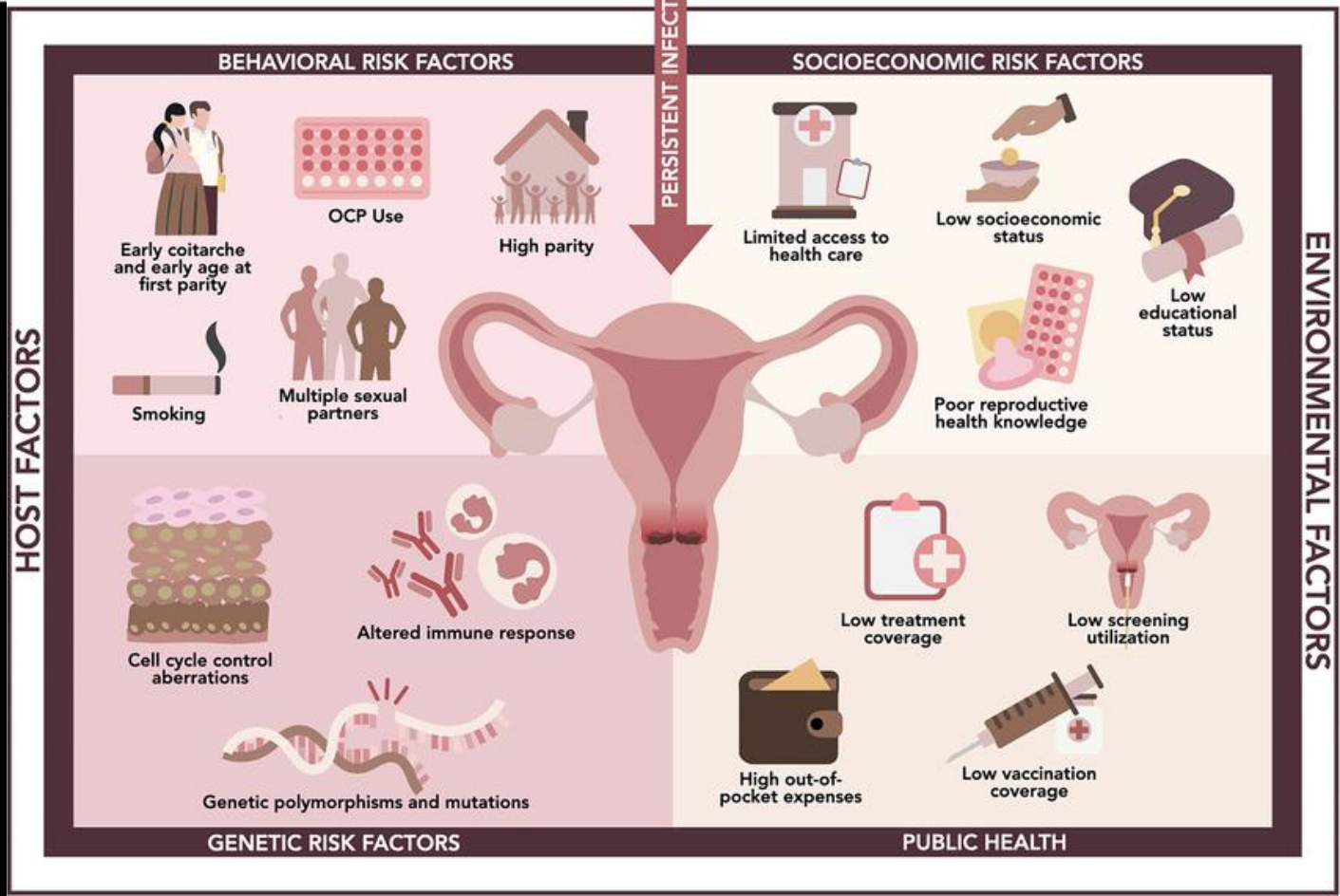
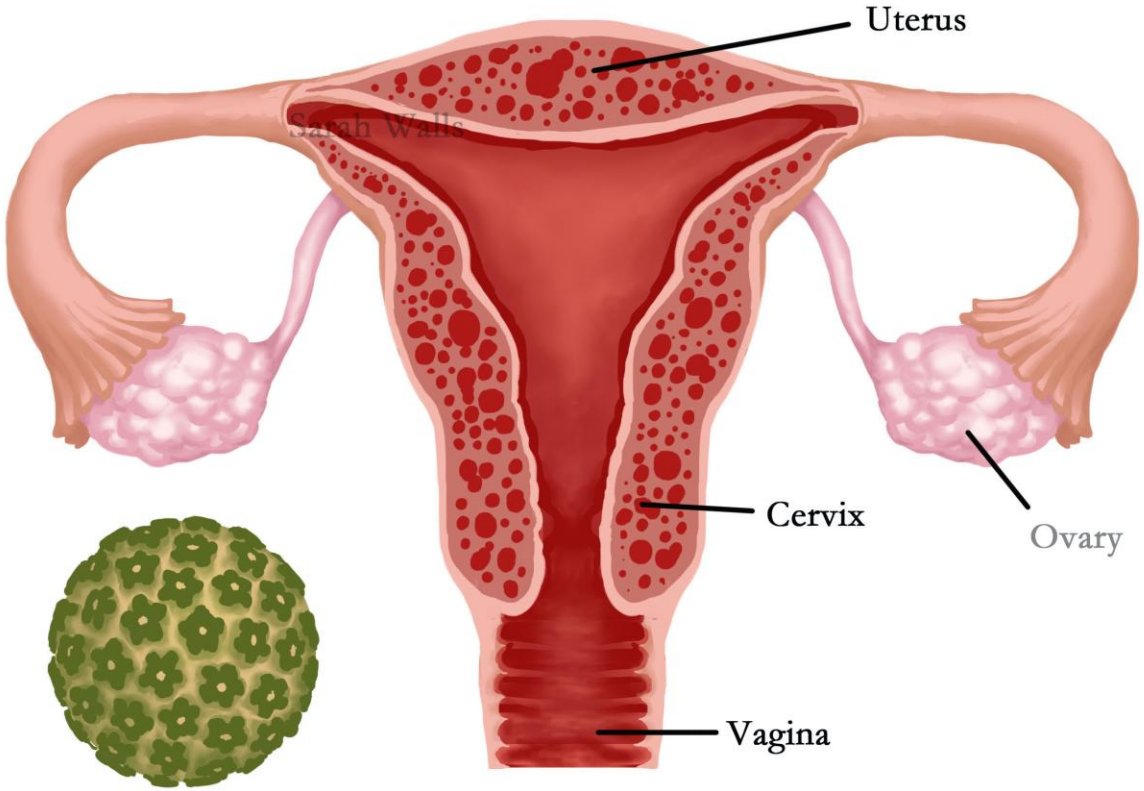
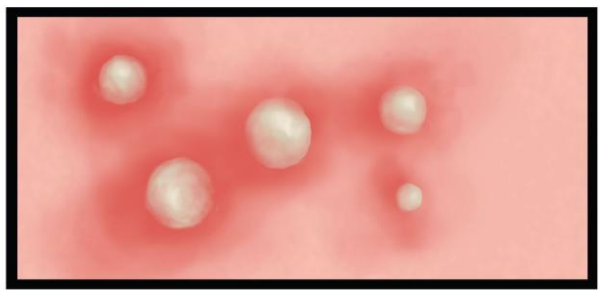


Image by Sarah Walls

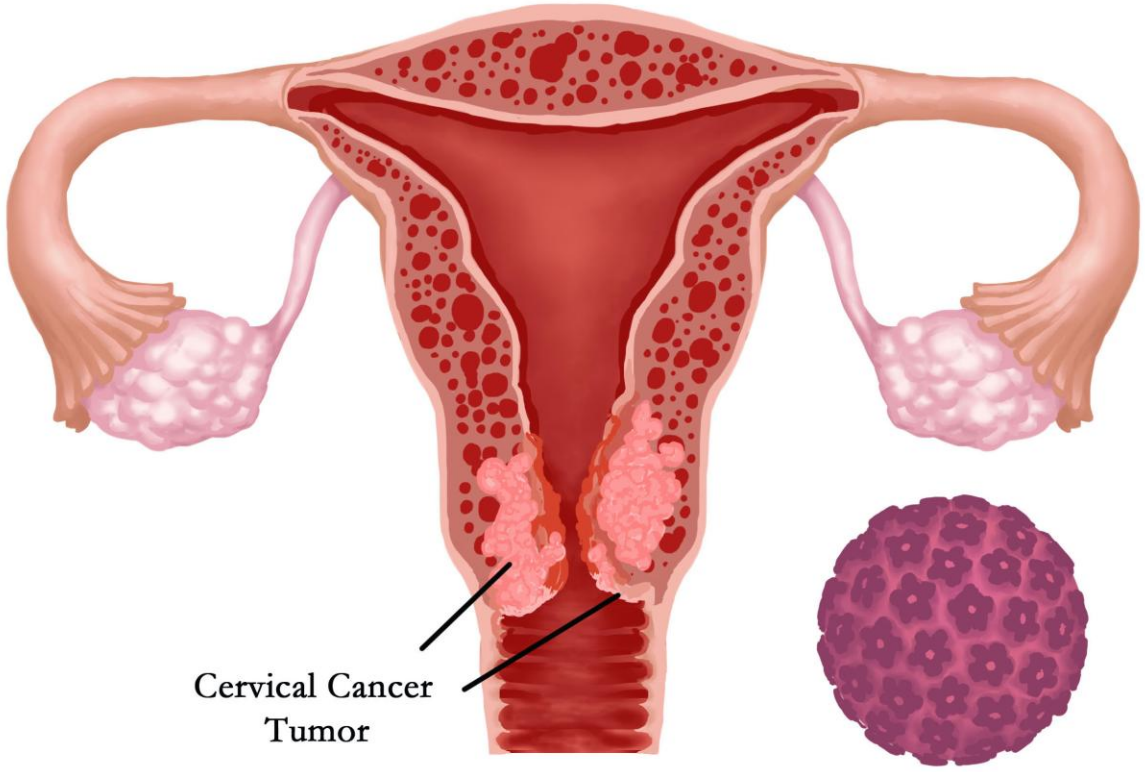
HPV 8 and HPV 11

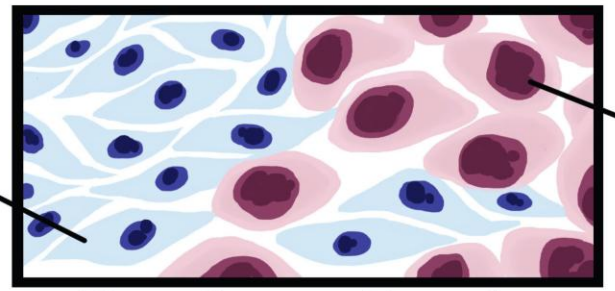




Genital Warts

HPV 16 and HPV 18

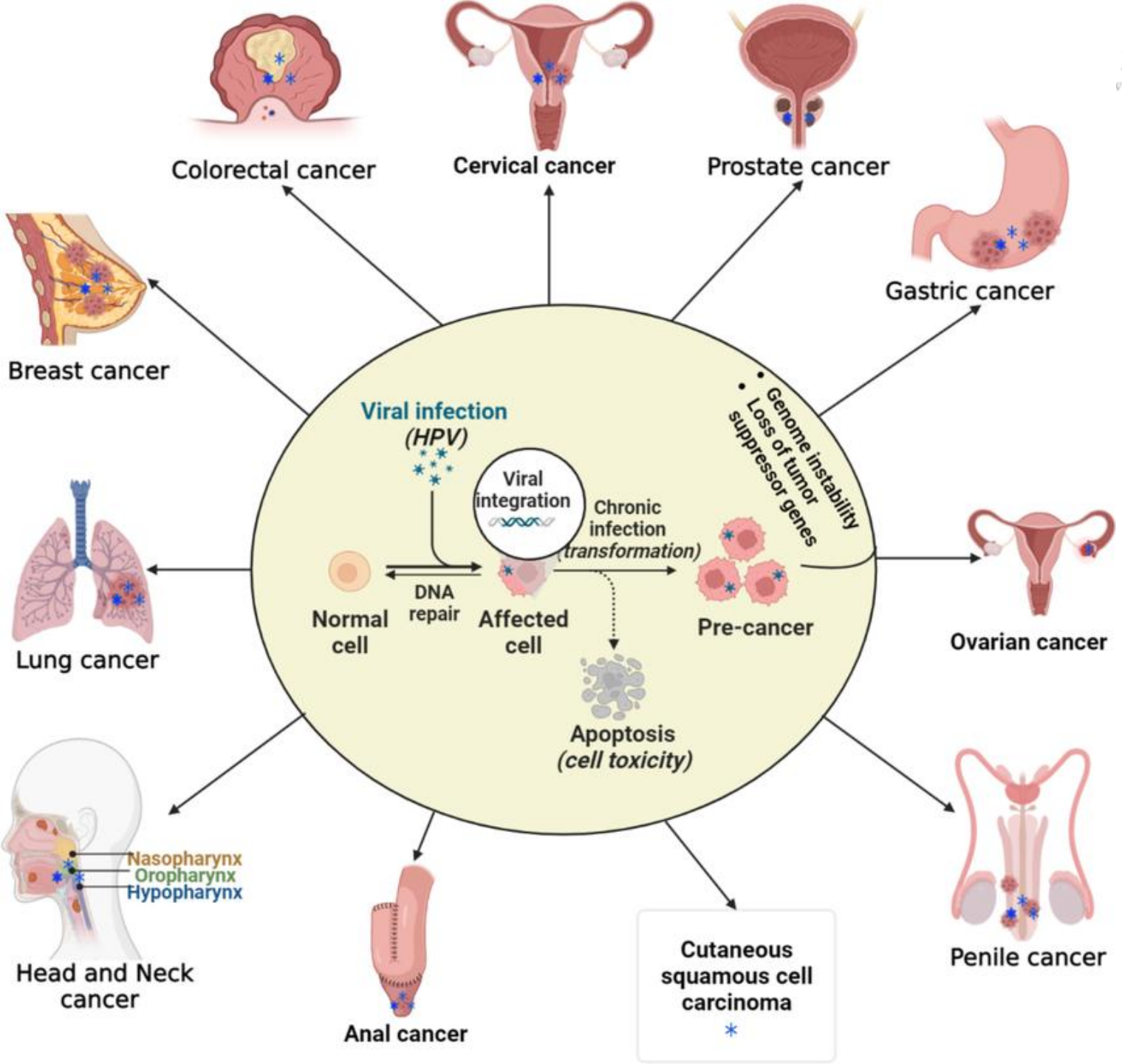




Pap Smear Results

Scientific research is slowly revealing the dark side of HPV that we didn't know about

So, the vaccine is not only for girls, but also for boys!



HPV impact on pregnancy and offspring

Generally, HPV is not associated with pregnancy complications like miscarriage, premature birth, or other serious outcomes

The risk of transmitting HPV (even HPV with carcinogenic potential) from mother to the baby is low

Genital warts in pregnant women: grow faster and larger – rarely big warts may cause vaginal obstruction

A baby born to a mother with genital warts can get warts in the pharynx or develop recurrent respiratory papillomatosis (wart-like growth, or papillomas, in the larynx, on vocal cords, in the trachea, and bronchi, often leading to hoarseness, breathing difficulties, or airway obstruction)

HPV impact on pregnancy and offspring

BUT! What does science say?

In pregnant women, HPV DNA has been detected in the placenta, amniotic fluid, and umbilical cord, fetal membranes

There are studies linking HPV with
- premature birth
- premature rupture of membranes (PROM)
- low birth weight
- intrauterine growth restriction (IGUR)

In vitro studies in 2021

Human trophoblasts possess receptors for HPV and facilitate the virus replication

Infection of trophoblasts with HPV leads to:

a) decreased number of trophoblasts
b) reduced the ability of trophoblasts to adhere to endometrial cells
c) HPV with carcinogenic potential (E6 and E7 genes) promotes trophoblast apoptosis

Result: problem getting pregnant and embryo expulsion in pregnant women

HPV impact on pregnancy and offspring

HPV induces placental distress, contributing to preterm delivery

What does science say?

The risk of HPV-positive pregnant women delivering preterm was twice as great as the risk of those who were HPV-negative

The impact of HPV on miscarriage – controversial results, yet not established

Preeclampsia – pregnancy-induced hypertension (PIH)

The impact of HPV? Opposing results

1) DNA of HPV prevalence in placental samples from preeclampsia cases was similar to the control (108 cases; 311 cases; 15,357 cases) = HPV does not influence the risk of pregnant women developing preeclampsia

2) HPV contributes to a twofold increase in preeclampsia risk (942 cases)

HPV impact on pregnancy and offspring

What does science say?

HPV impacts on intrauterine growth restriction (IUGR)

Several studies confirmed the role of HPV in IUGR
A 31,827-case study confirmed that mothers with HPV were at an increased risk of giving birth to babies below the third percentile, with very low birth weight, independent of other factors

HPV impacts on premature rupture of membranes (PROM)

A 400,583-case study demonstrated that 24.3% of HPV-positive women experienced PROM compared with 14.2% of HPV-negative women
($p<0.05$, statistically significant result)

Does HPV impact fetal death? Too few studies, but 81% (13 from 16 cases) of fetal deaths were from an HPV-positive mother with no prior medical condition

The HPV vaccine is contraindicated in pregnant women

Herpes simplex virus (HSV)

First and only contact forever

Latency

In sensory neurons in the trigeminal ganglia for HSV-1 or in dorsal root ganglia for HSV-2, but also in autonomic branches (e.g., superior cervical ganglia)

Herpes Simplex Virus Type 2

The HSV-2 virus causes most cases of genital herpes

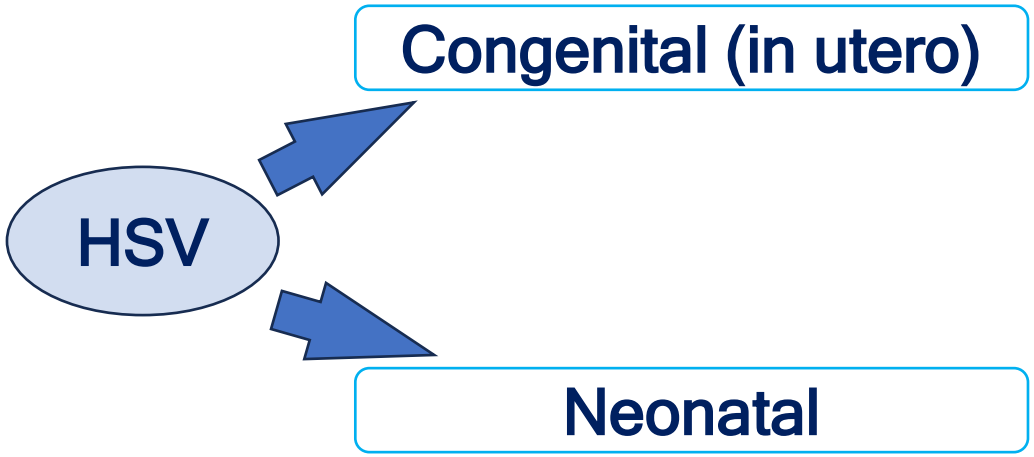
It's highly contagious and can spread through intercourse or direct contact with a herpes sore

Treatment: acyclovir, valacyclovir, famciclovir, and docosanol

Antiviral drugs can reduce the frequency of outbreaks and help alleviate symptoms more quickly

Symptoms: Fluid-filled blisters that form painful, crusted sores on the genitals, anus, thighs, or buttocks
It can spread to the lips through oral contact

Herpes Simplex Virus



Rare: 5% of cases
50% of cases occur when the mother develops disseminated infection, but 70% occur when caused by HSV-2

85 – 90% of all cases
50% of cases from the mothers with primary infection
< 3% of cases from mothers with recurrent infection
70% of cases – exposure to asymptomatic genital HSV infection near delivery
10-50% of cases are caused by early postnatal HSV acquisition
70 – 80% of cases are caused by HSV-2 (graver prognosis), the remaining by HSV-1

Congenital herpes

First 20 weeks of gestation
Abortion, stillbirth, and congenital anomalies (intrauterine fetal growth restriction)
Mortality rate: 50%

Skin vesicles or scarring
Eye lesions (chorioretinitis, microphthalmia, cataract)
Neurologic damage (intracranial calcifications, microcephaly, seizures, encephalomalacia)
Growth retardation, and psychomotor development (IUGR)

Both primary and recurrent infections can result in congenital disease, although in recurrent maternal infection, the risk is small

Neonatal (intrapartum, postnatally)

Localized HSV diseases (SEM)

(skin, eye, and/or mouth) – low mortality, but significant morbidity – may progress (60-70%) to encephalitis or disseminated diseases if left untreated

50% of the affected neonates

HSV encephalitis with or without

SEM involvement – neurologic morbidity (majority of survivors)

33% of the affected neonates

Disseminated HSV disease

(multiple organ dysfunction: CNS, lung, liver, adrenal glands, skin, eye, mouth)

Mortality risk is > 80% if untreated

17% of the affected neonates

Herpes Simplex Virus Type 2

At diagnosis:

Skin vesicles 68% (not always present!)
Fever 39%
Lethargy 38%
Seizures 27%
Conjunctivitis 19%
Pneumonia 13%
DIC11%

Symptoms:

Occasionally present at birth
In 60%, later than 5 days after birth
Sometimes after 4-6 weeks of life

The risk (30-50%) of neonatal infection is higher during the third trimester of pregnancy (no or low antibody level)

Herpes and Autism?

One study: High levels of antibody to HSV-2 in pregnant women are associated with an increased risk that their baby will later be found to have autism… true or false?

Cytomegalovirus (CMV) – the most common cause of congenital infection globally

A leading cause of congenital infections: Rate of CMV infection in pregnant women: **1 – 2% per year**

Sources of maternal CMV infection:
Sexual activity and close contact with CMV-infected children

About 1 in 5 babies with congenital CMV infection will have birth defects or other long-term health problems

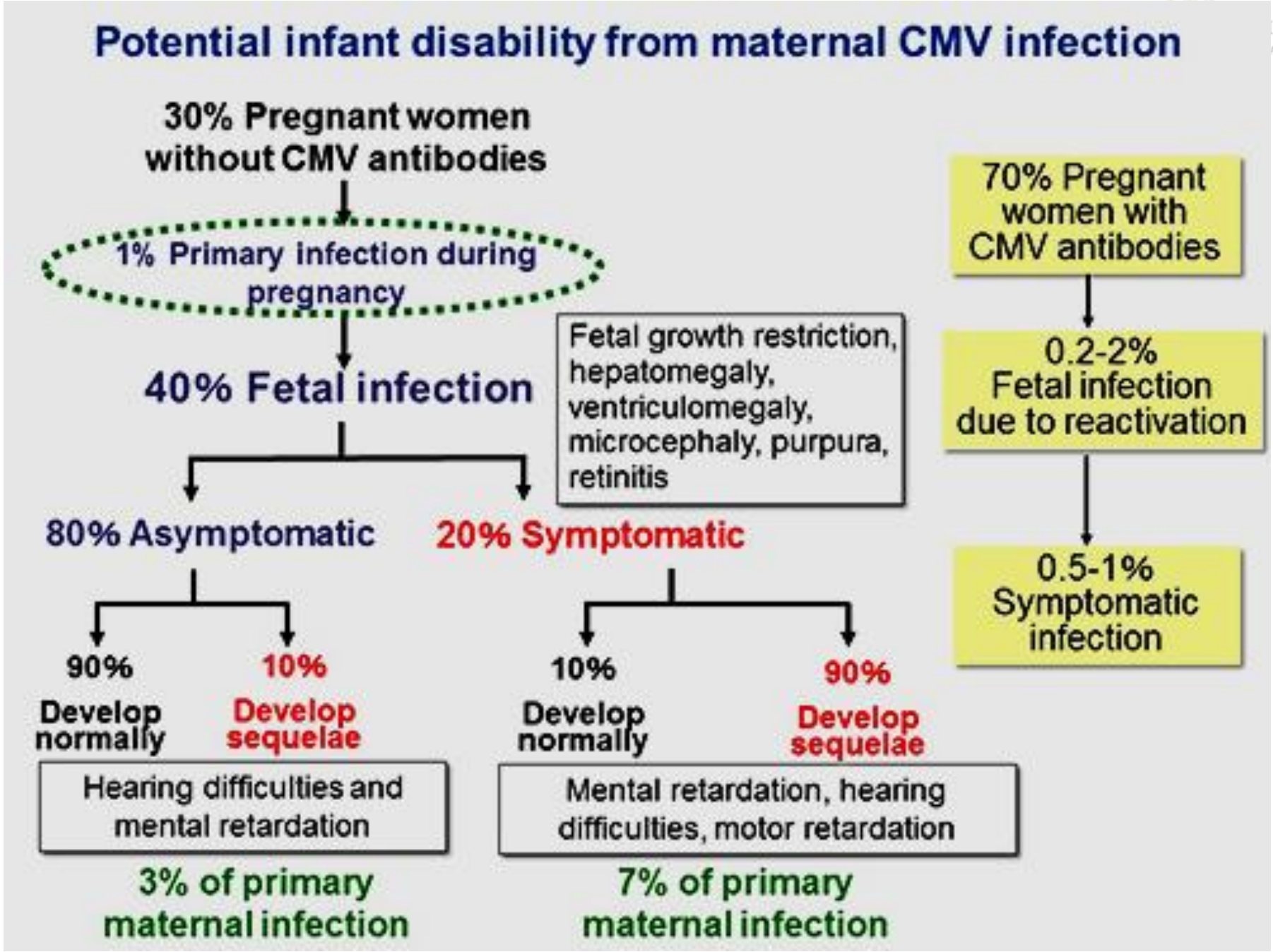
- **Maternal immune response** = crucial determinant of the transplacental transmission of CMV
- **Primary infection** = acquisition of CMV during pregnancy, identified by conversion from serum antibody-negative to antibody-positive status or presence of IgM to CMV
- **Nonprimary maternal infection** = reactivation of endogenous latent CMV infection or reinfection with a new virus strain

The risk of fetal transmission is approximately 30%-35% after maternal primary CMV infection and lower with non-primary maternal CMV infection (1%-3%)

First trimester (first 12 weeks):
Organogenesis (the development of major organs and body systems)
CMV infection = the highest risk of fetal anomalies

Second trimester:
The severity of fetal anomalies tends to be less pronounced compared to the first trimester

Third trimester:
The highest rates of transmission to the fetus, but the severity of fetal anomalies is the lowest



Transmission of CMV from mother to child

Transplacentally
Intrapartum
Postnatally

Congenital CMV infection – clinical findings

Petechiae	76%
Jaundice	67%
Hepatosplenomegaly	60%
Microcephaly	53%
Small for gestational age	50%
Chorioretinitis	20%
Purpura	13%
Seizures	7%

Intracranial calcifications
Blindness
Deafness
Mental retardation

Congenital CMV infection rates:

- related to maternal seroprevalence
- associated with young maternal age
- Lower socioeconomic status

Range from 0.48% to 1.42%

Intrapartum CMV transmission

- Occurs in around 50% of infants born to mothers shedding CMV from cervix or vagina at time of delivery
- Almost all infants infected in this fashion are asymptomatic, but some may demonstrate CMV pneumonitis

Postnatal CMV transmission

Breast milk – principal route of transmission

27 – 70% of seropositive women shed CMV in breast milk

All infants infected in this fashion are asymptomatic

Hepatitis viruses transmitted via sexual intercourse: HBV, HCV

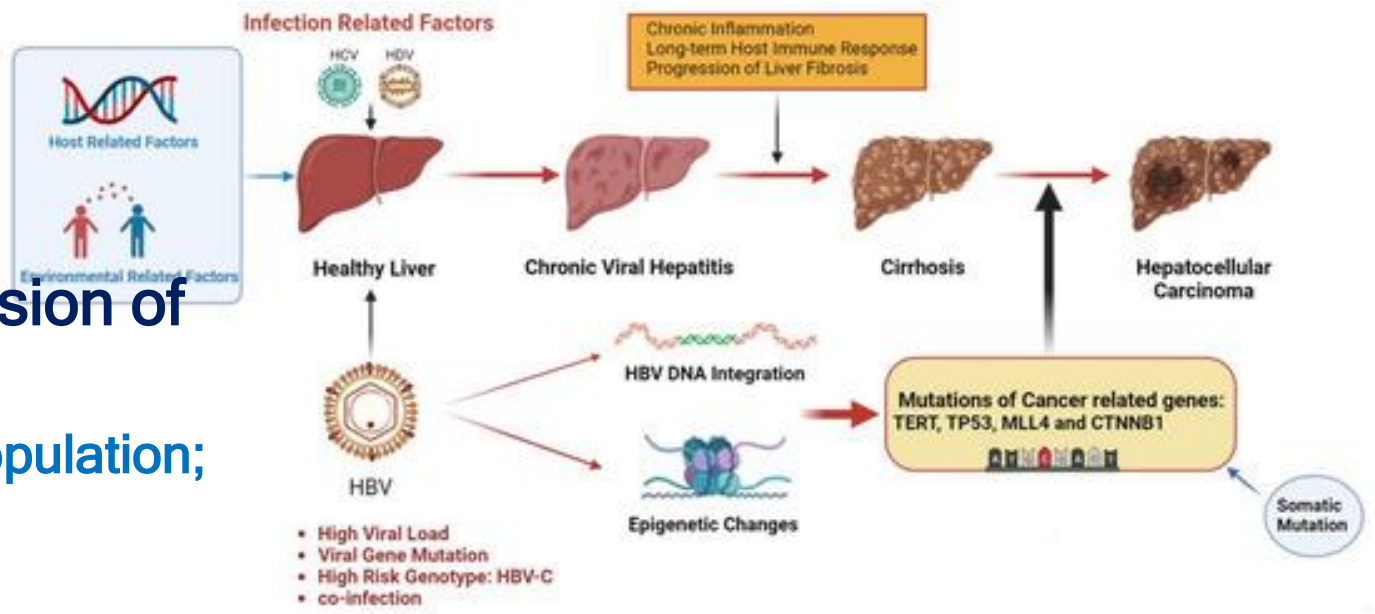
A stable liver equals a safe pregnancy

Both are transmitted transplacentally, perinatally, and postnatally
What is the outcome?

HBV infection of the fetus or neonate predisposes to carriage, liver cirrhosis, and hepatocellular carcinoma in young adults

The main risk factor for vertical transmission of HBV is the mother's viral load

Global prevalence is ca. 3.5% of the human population; 750,000 deaths each year



A pregnant women with HBV infection have a poor prognosis (increased incidence of PROM and neonatal asphyxia, gestational diabetes mellitus, intrahepatic cholestasis, preterm birth)

Invasive procedures (amniocentesis, villus sampling) break the maternal-fetal barrier, increasing the risk of HBV and HCV vertical transmission!

Questions

- List viruses that can have an impact on the offspring of infected pregnant women.
- What is the impact of HPV on pregnancy and offspring?
- Does the HSV reactivation in pregnant women have any impact on the offspring? If so, what?
- What is the impact of intrauterine HSV infection on the baby? And what if the child contracts HSV during labor?
- What are the factors influencing the outcome of offspring infection with CMV in pregnant women?
- Do hepatitis viruses have any impact in any way on the children born to infected mothers?

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