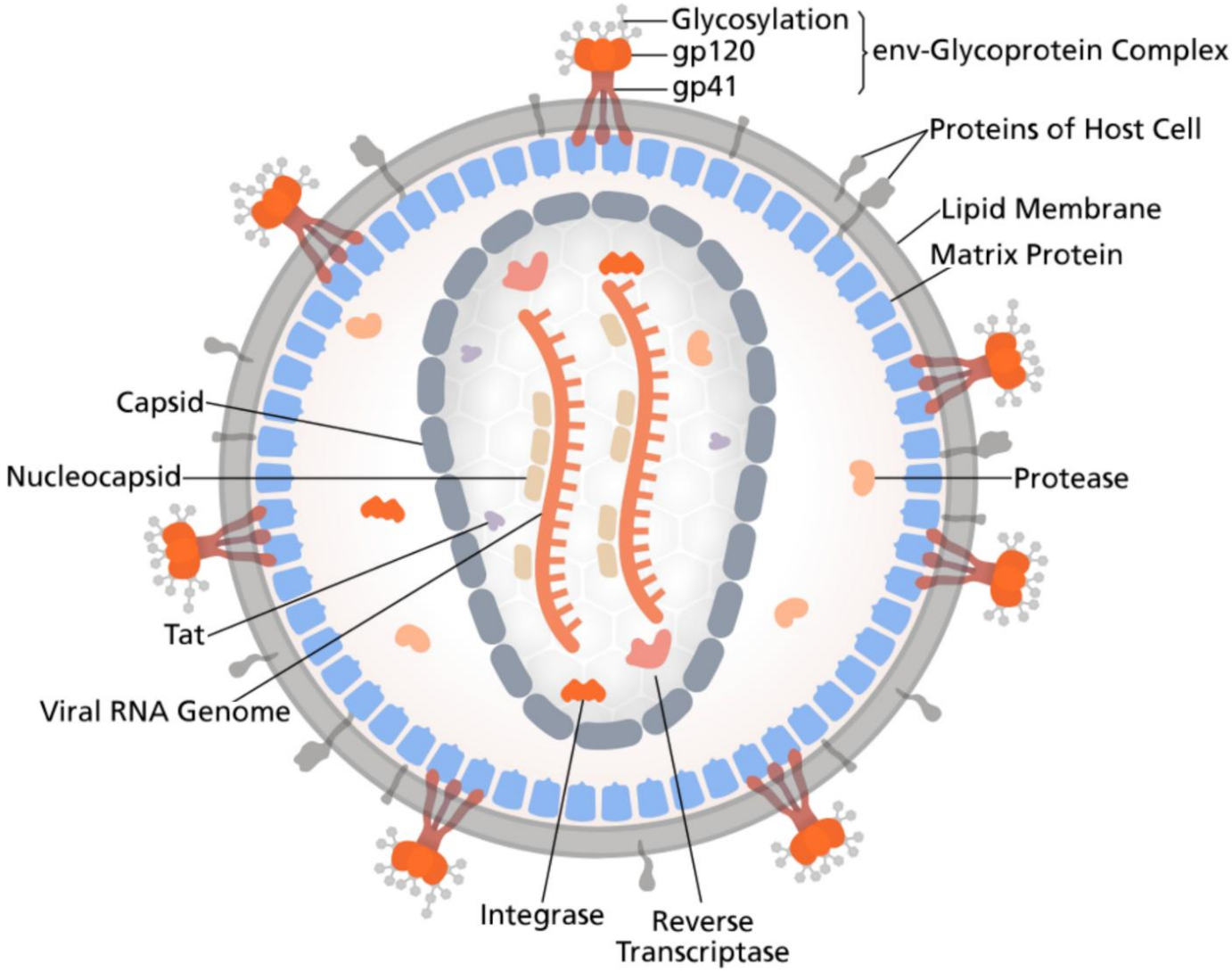
Sexually transmitted diseases

HIV (Human Immunodeficiency Virus)

- HIV is a **retrovirus** that attacks the **immune system**, specifically targeting **CD4+ T lymphocytes**, leading to progressive immune deficiency.
- If untreated, HIV can progress to **AIDS (Acquired Immunodeficiency Syndrome)**, the most advanced stage of HIV infection.
- HIV is a **lifelong infection**, but it is **treatable** with antiretroviral therapy (ART), which can suppress the virus and prevent disease progression and transmission.

Structure of HIV



A. Envelope Glycoproteins:

- **gp120**: Binds to CD4 receptor
- **gp41**: Mediates fusion with host cell membrane

B. Core Protein:

- **p24**: Important for diagnostics

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C. Genome:

- Two copies of single-stranded RNA

D. Enzymes:

- **Reverse Transcriptase**: RNA → DNA
- **Integrase**: Integrates viral DNA into host genome
- **Protease**: Maturation of viral particles

Causes

1. Viral Transmission Routes:

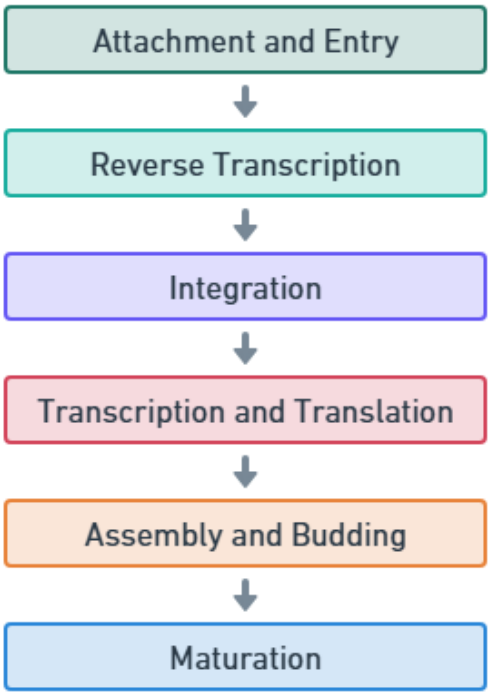
- **Sexual contact** (most common): Unprotected vaginal, anal, or oral sex with an infected person.
- **Blood exposure:** Sharing needles, transfusion with infected blood (rare due to screening).
- **Mother-to-child transmission (MTCT):** During pregnancy, childbirth, or breastfeeding.
- **Occupational exposure:** Needlestick injuries in healthcare settings.

2. Risk Factors:

- Multiple sexual partners.
- Presence of other sexually transmitted infections (STIs).
- Intravenous drug use.
- Unprotected sex with high-risk individuals.
- Infants born to HIV-positive mothers.

HIV Life Cycle

The HIV life cycle includes several key stages:



A. Attachment and Entry:

- HIV attaches to the CD4 receptor on CD4 cells using its surface glycoprotein, gp120.
- The virus binds to a co-receptor (CCR5 or CXCR4), enabling fusion with the host cell membrane.
- The viral capsid containing RNA and enzymes enters the host cell.

B. Reverse Transcription:

- The viral enzyme reverse transcriptase converts single-stranded viral RNA into double-stranded DNA.

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C. Integration:

- The viral DNA is transported to the nucleus, where integrase incorporates it into the host cell's genome, forming a provirus.

D. Transcription and Translation:

- The host cell machinery transcribes the integrated viral DNA into RNA, which is then translated into viral proteins.

E. Assembly and Budding:

- Viral RNA and proteins assemble at the host cell membrane, forming immature virus particles.
- These particles bud off from the host cell, acquiring a lipid envelope.

F. Maturation:

- The viral enzyme protease cleaves viral polyproteins into functional proteins, maturing the virus particles, making them infectious.

Stages of HIV Infection

1. Acute HIV Infection:

- **Timeline:** 2-4 weeks after exposure.
- **Symptoms:** Flu-like symptoms (fever, swollen glands, sore throat, rash, muscle/joint aches, mouth ulcers).
- **Significance:** High viral load, highly infectious.

2. Clinical Latency Stage:

- **Timeline:** Can last for years or decades.
- **Symptoms:** Often asymptomatic or mild symptoms. Virus continues to replicate at low levels.
- **Significance:** HIV is still active and transmissible.

3. AIDS:

- **Timeline:** Progresses when the immune system is severely weakened.
- **Symptoms:** Severe weight loss, fever, night sweats, extreme fatigue, swollen lymph glands, prolonged diarrhea, sores (mouth, anus, genitals), pneumonia, neurological issues (memory loss, depression).
- **Diagnosis Criteria:** CD4 count below 200 cells/mm^3, or presence of specific opportunistic infections or cancers (e.g., Kaposi's sarcoma, non-Hodgkin's lymphoma).

Symptoms

A. Acute HIV Infection (Primary Stage):

- Occurs 2–4 weeks after exposure.

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- Often resembles flu or mononucleosis.
- Symptoms include:
  - Fever.
  - Fatigue.
  - Sore throat.
  - Rash.
  - Lymphadenopathy.
  - Headache.
  - Myalgia.

B. Chronic HIV (Clinical Latency):

- Asymptomatic or mild symptoms.
- Can last several years.
- Virus replicates at low levels; CD4 count slowly declines.

C. AIDS (Advanced HIV):

- Defined by **CD4 count < 200 cells/mm^3** or presence of **AIDS-defining illnesses**.
- Symptoms:
  - Persistent fever.
  - Weight loss.
  - Chronic diarrhea.
  - Opportunistic infections (e.g., Pneumocystis jirovecii pneumonia, tuberculosis, candidiasis).
  - Neurological manifestations (e.g., HIV-associated dementia).
  - Malignancies (Kaposi sarcoma, lymphoma, cervical cancer).

Diagnosis

1. Screening Tests:

- **4th-generation ELISA** (detects both HIV antibodies and p24 antigen).
- **Rapid antibody tests** (point-of-care).

2. Confirmatory Tests:

- **Western blot** (historically used, now replaced by more modern tests).
- **HIV-1/HIV-2 differentiation assay.**

3. Nucleic Acid Testing (NAT):

- Detects HIV RNA (viral load), useful for early infection or neonatal diagnosis.

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4. Monitoring Tests:

- **CD4 count:** Assesses immune status.
- **HIV viral load:** Monitors treatment response.

Pathophysiology of HIV

1. Entry and Infection:

- HIV targets **CD4+ T cells** via binding to **CD4 receptor** and **co-receptors (CCR5 or CXCR4)**.
- Virus enters the cell and converts its RNA into DNA via **reverse transcriptase**.

2. Integration and Replication:

- Viral DNA integrates into host genome via **integrase enzyme**.
- New viral particles are synthesized and assembled, then released to infect other cells.

3. Immune System Damage:

- Progressive **loss of CD4+ T cells** impairs immune responses.
- Chronic immune activation contributes to immune exhaustion and dysfunction.

4. Opportunistic Infections and Cancer:

- As immune function declines, the body becomes susceptible to infections and certain cancers that do not typically occur in immunocompetent individuals.

Treatment

1. Antiretroviral Therapy (ART):

- Combination of at least three drugs from two different classes.
- Goals:
  - Suppress viral load to undetectable levels.
  - Restore and preserve immune function.
  - Reduce HIV-related morbidity and mortality.
  - Prevent transmission.
- **Main drug classes:**
  - **NRTIs (Nucleoside Reverse Transcriptase Inhibitors):** e.g., tenofovir, emtricitabine.
  - **NNRTIs (Non-Nucleoside RTIs):** e.g., efavirenz.
  - **PIs (Protease Inhibitors):** e.g., lopinavir/ritonavir.
  - **INSTIs (Integrase Inhibitors):** e.g., dolutegravir, bictegravir.
  - **Entry/Fusion Inhibitors:** e.g., maraviroc, enfuvirtide.

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