HIV/AIDS Lecture.

فيروس العوز

المناعي البشري

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1443 / 2022

HIV / AIDS

- History.
- Epidemiology.
- Human immunodeficiency virus.
- Transmission
- Pathogenesis and life cycle & replication.
- Clinical features.
- Laboratory diagnosis.
- Complications.
- Option of treatment.
- Natural history.

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MMWR

Weekly

June 5, 1981 / 30(21);1-3

Epidemiologic Notes and Reports

http://www.cdc.gov/mmwr/preview/mmwrhtml/june_5.htm

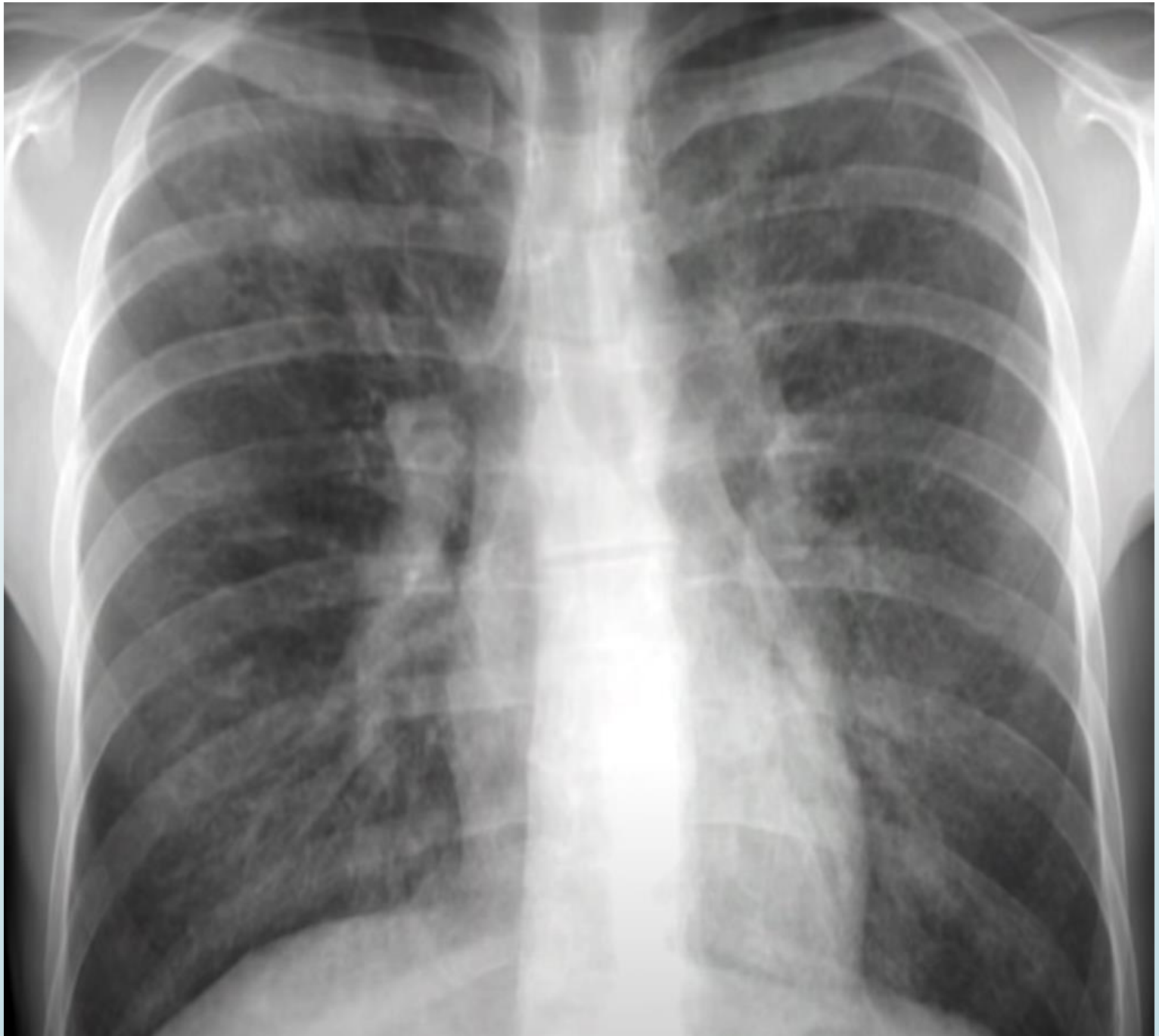
Pneumocystis Pneumonia --- Los Angeles

In the period October 1980-May 1981, 5 young men, all active homosexuals, were treated for biopsy-confirmed *Pneumocystis carinii* pneumonia at 3 different hospitals in Los Angeles, California. Two of the patients died. All 5 patients had laboratory-confirmed previous or current cytomegalovirus (CMV) infection and candidal mucosal infection. Case reports of these patients follow.

Patient 1: A previously healthy 33-year-old man developed *P. carinii* pneumonia and oral mucosal candidiasis in March 1981 after a 2-month history of fever associated with elevated liver enzymes, leukopenia, and CMV viruria. The serum complement-fixation CMV titer in October 1980 was 256; in may 1981 it was 32.* The patient's condition deteriorated despite courses of treatment with trimethoprim-sulfamethoxazole (TMP/SMX), pentamidine, and acyclovir. He died May 3, and postmortem examination showed residual *P. carinii* and CMV pneumonia, but no evidence of neoplasia.

Patient 2: A previously healthy 30-year-old man developed *p. carinii* pneumonia in April 1981 after a 5-month history of fever each day and of elevated liver-function tests, CMV viruria, and documented seroconversion to CMV, i.e., an acute-phase titer of 16 and a convalescent-phase titer of 28* in anticomplement immunofluorescence tests. Other features of his illness included leukopenia and mucosal candidiasis. His pneumonia responded to a course of intravenous TMP/.SMX, but, as of the latest reports, he continues to have a fever each day.

Editorial Note: *Pneumocystis* pneumonia in the United States is almost exclusively limited to severely immunosuppressed patients (1). The occurrence of pneumocystosis in these 5 previously healthy individuals without a clinically apparent underlying immunodeficiency is unusual. The fact that these patients were all homosexuals suggests an association between some aspect of a homosexual lifestyle or disease acquired through sexual contact and *Pneumocystis* pneumonia in this population. All 5 patients described in this report had laboratory-confirmed CMV disease or virus shedding within 5 months of the diagnosis of *Pneumocystis* pneumonia. CMV infection has been shown to induce transient abnormalities of *in vitro* cellular-immune function in otherwise healthy human hosts (2,3). Although all 3 patients tested had abnormal cellular-immune function, no definitive conclusion regarding the role of CMV infection in these 5 cases can be reached because of the lack of published data on cellular-immune function in healthy homosexual males with and without CMV antibody. In 1 report, 7 (3.6%) of 194 patients with pneumocystosis also had CMV infection' 40 (21%) of the same group had at least 1 other major concurrent infection (1). A high prevalence of CMV infections among homosexual males was recently reported: 179 (94%) had CMV viruria; rates for 101 controls of similar age who were reported to be exclusively heterosexual were 54% for seropositivity and zero fro viruria (4). In another study of 64 males, 4 (6.3%) had positive tests for CMV in semen, but none had CMV recovered from urine. Two of the 4 reported recent homosexual contacts. These findings suggest not only that virus shedding may be more readily detected in seminal fluid than urine, but



miologic Notes and Reports

MMWR

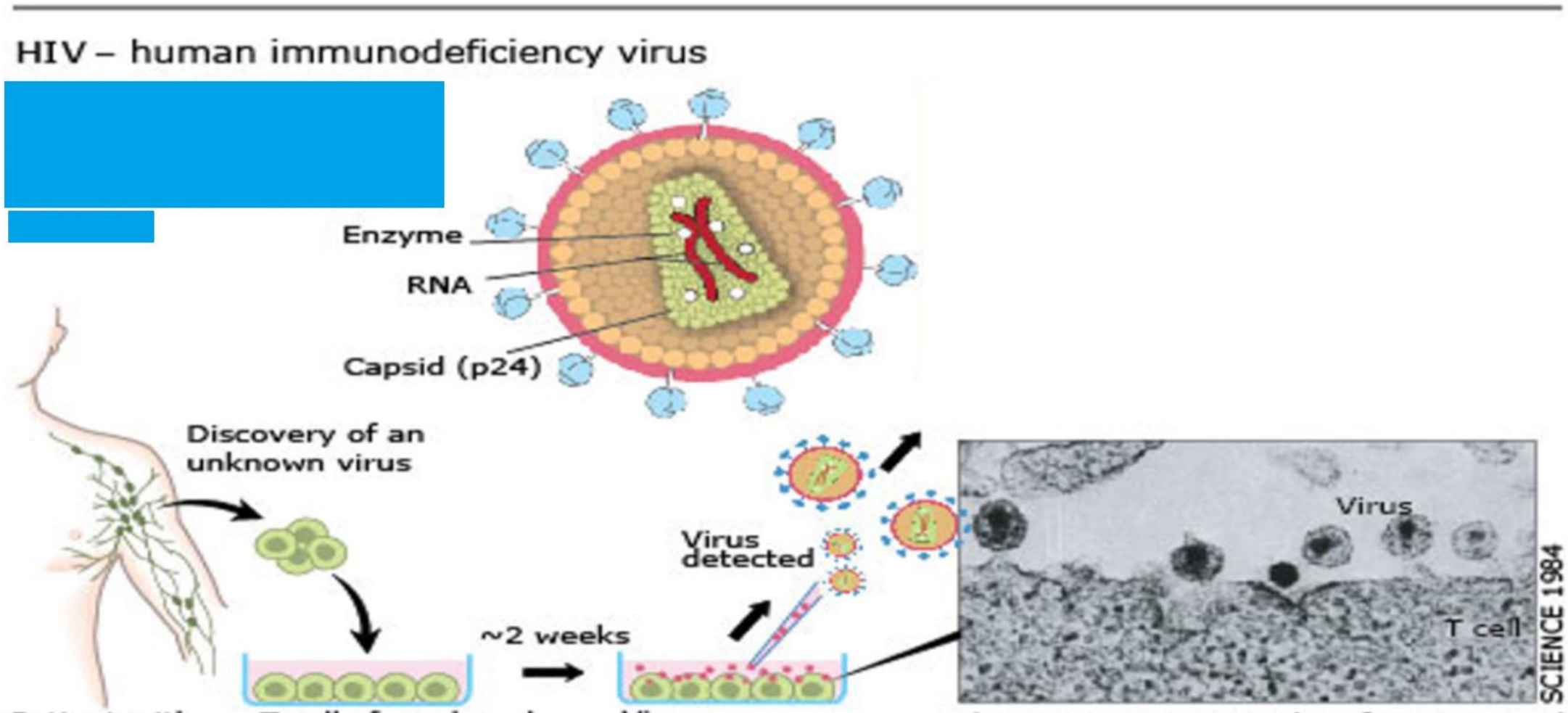
Kaposi's Sarcoma and *Pneumocystis* Pneumonia Among Homosexual Men – New York City and California

ing the past 30 months, Kaposi's sarcoma (KS), an uncommonly repo
in the United States, has been diagnosed in 26 homosexual men (20 in

History

- **By the end of 1981**, total of **270 reported cases** of severe immune deficiency among gay men, and **121 of those individuals had died**.
- **In 1983**, **Luc Montagnier and Françoise Barré-Sinoussi** reported the discovery of a new virus (later called HIV) that is the cause of AIDS.
- **1984**: Blood test was developed.
- Electron microscopy and genome sequence analysis revealed that this virus (HIV) to be a lentivirus, known group of **retroviruses.**
- **HIV reported first in Human in USA (1981) and then spread rapidly to all over the world**.

1983- Identification of the HIV virus- Françoise Barré-Sinoussi and Luc Montagnier (Shared 2008 Nobel Prize in Physiology or Medicine)



Epidemiology

WORLDWIDE STATISTICS

HIV : the worst epidemic of the twentieth century and has reached every country .

With more than 35 million fatalities,

The AIDS epidemic now ranks alongside the:

A] Bubonic Plague of the 14th century : (1347 to 1352) **75 to 100 million death**

[Flea-borne zoonosis caused by the bacterium Yersinia pestis.].

April 1, 2015, a total of 11 cases of human plague have been reported IN Western USA. (3 Patients died).

B] Influenza pandemic (1957–1958) 20 to 50 million death

Epidemiology

- **By 2020:**

- **38.0 million** people globally were **living with HIV .**
- **1.5 million people** became **newly infected.**
- **680 000 people died from AIDS-related illnesses.**

- **~ 100 million people have become infected** with HIV since the start of the epidemic.

- **~ 36 million people have died** from AIDS-related illnesses since the start of the epidemic.

An estimated 0.7% of adults aged 15–49 years worldwide are living with HIV,

Epidemiology

Summary of the global HIV epidemic, 2020

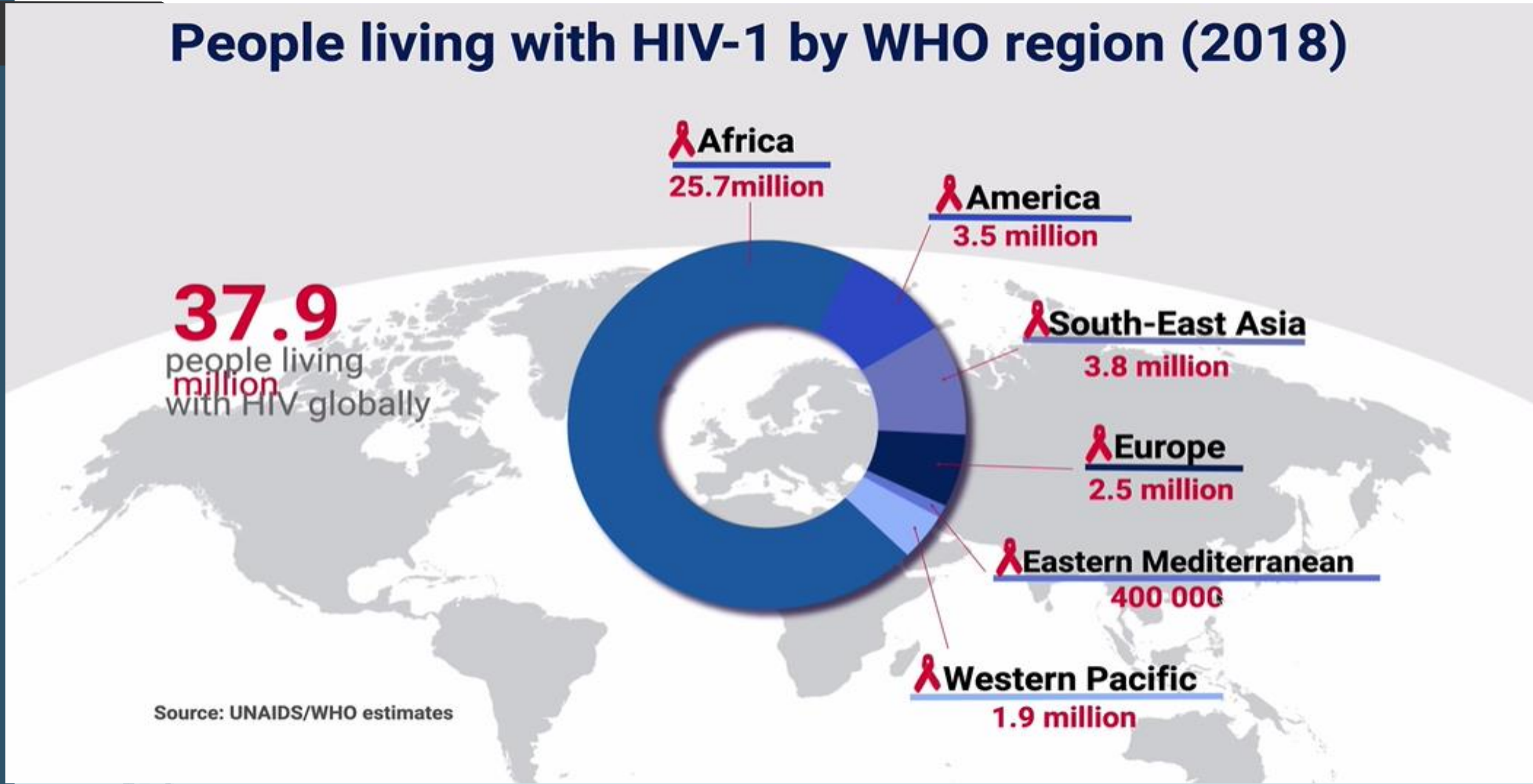
	People living with HIV in 2020	People acquiring HIV in 2020	People dying from HIV-related causes in 2020
Total	37.7 million [30.2–45.1 million]	1.5 million [1.0–2.0 million]	680 000 [480 000–1.0 million]
Adults (15+ years)	36.0 million [28.9–43.2 million]	1.3 million [910 000–1.8 million]	580 000 [400 000–850 000]
Women (15+ years)	19.3 million [15.5–23.1 million]	660 000 [450 000–920 000]	240 000 [170 000–360 000]
Men (15+ years)	16.7 million [13.3–20.1 million]	640 000 [460 000–890 000]	340 000 [230 000–490 000]
Children (<15 years)	1.7 million [1.2–2.2 million]	150 000 [100 000–240 000]	99 000 [68 000–160 000]

Source: UNAIDS/WHO estimates

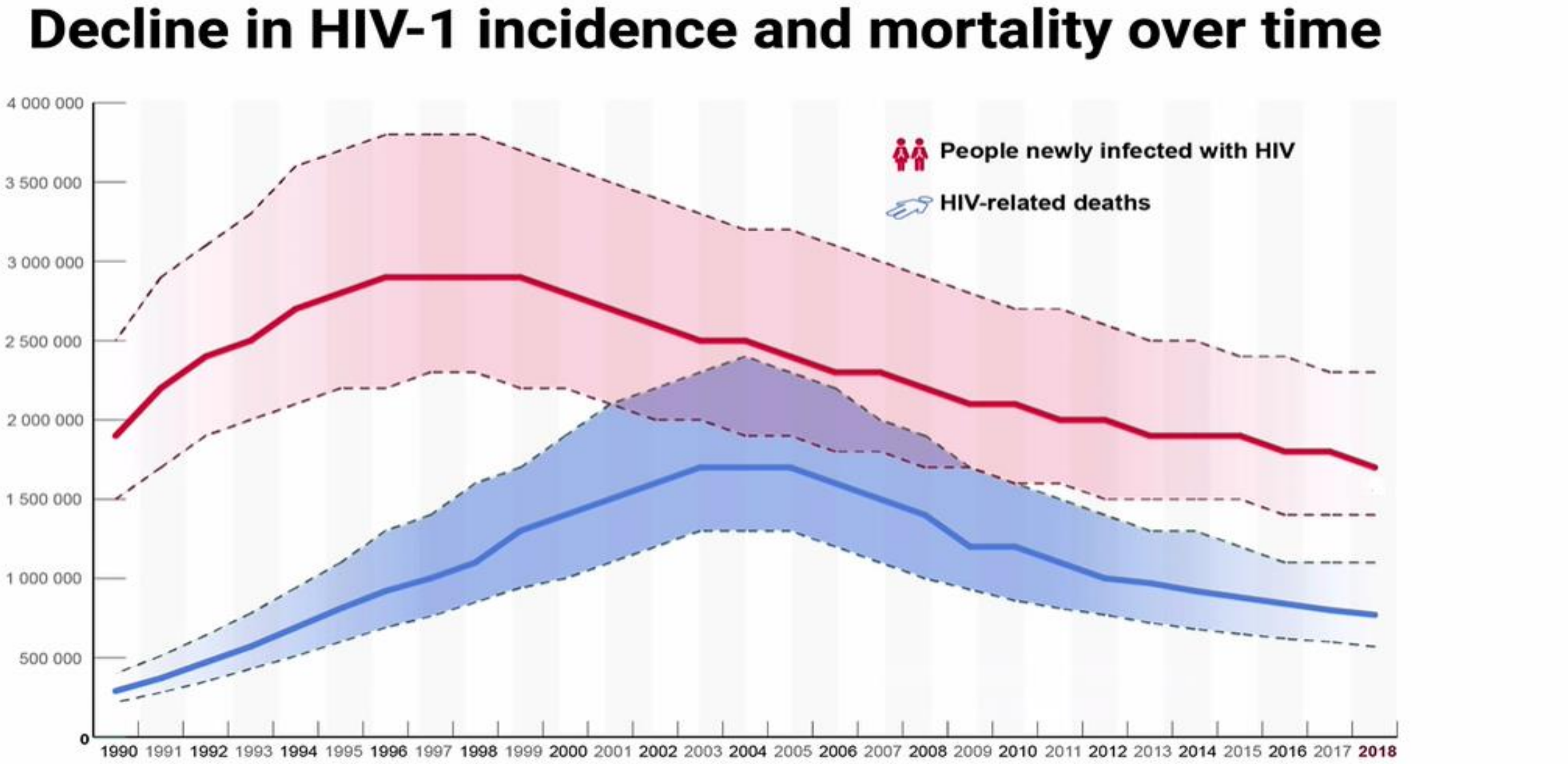
Updated: July 2021

World Health Organization

Epidemiology



Epidemiology



Source: UNAIDS/WHO estimates

Human immunodeficiency virus.

- Is a member of the lentivirus family, a subgroup of **retroviruses**, and it is an **RNA virus**.

- **It targets the cells of the immune system and**
- **Replicates within T-lymphocyte expressing the CD4 antigen**

Progressive depletion in CD4 cell counts with increased risk of:

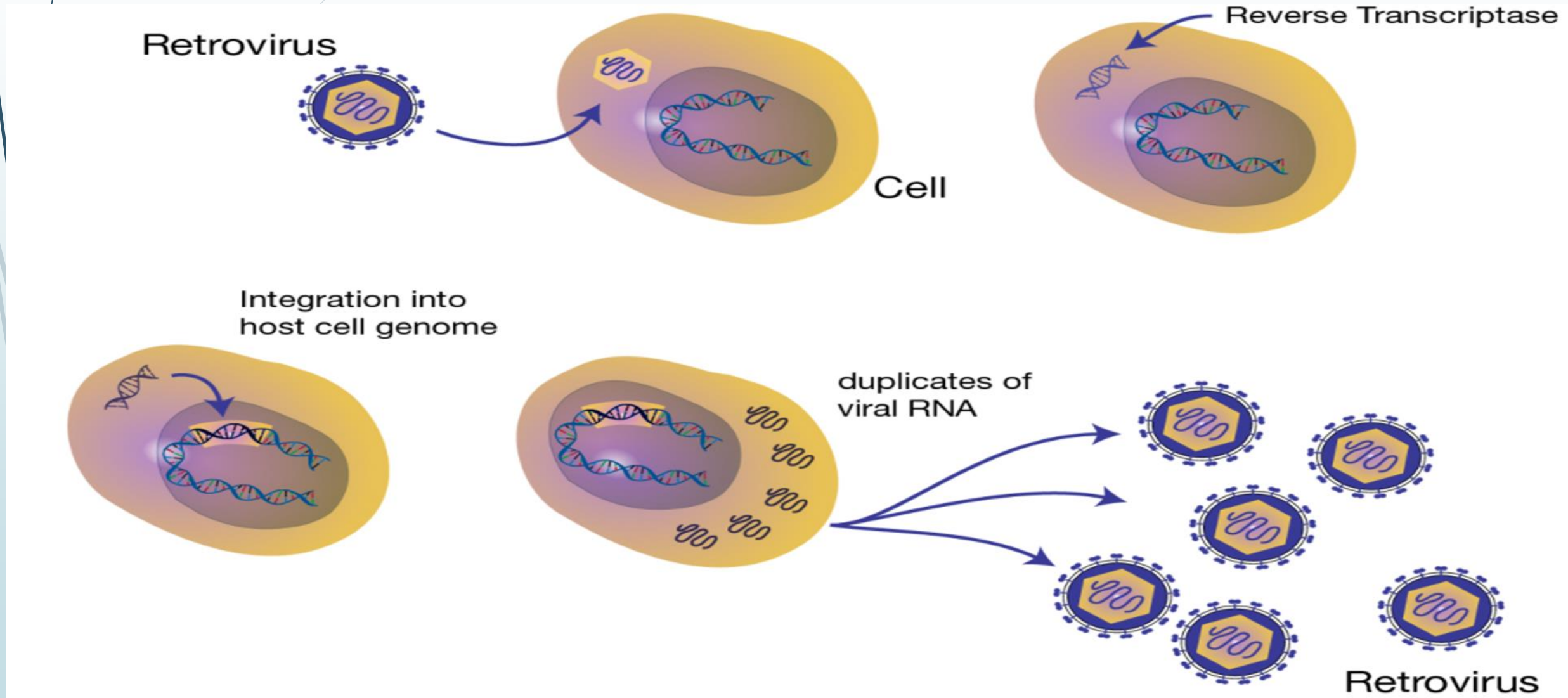
1] Opportunistic infections (OIs) such as Pneumocystis Jiroveci, CMV….etc

2] Malignancy such as Lymphoma and Kaposi sarcoma

This is called:

- **AIDS (Acquired immunodeficiency syndrome)**
- **It is defined by a loss of CD4 T lymphocytes (< 200 cell/) or the occurrence of opportunistic infections or cancers in HIV infected Patient.**

Retroviruses: a **virus that uses RNA as its genetic material and it makes a DNA copy of its genome that is inserted into the DNA of the host cell.**



There are two HIV viruses :

HIV1 : **<u>Predominate world wide</u>**

HIV2 : Closely resemble HIV-1, but is a **much slower progression** to AIDS than HIV1.

It Predominate in **western Africa** and Southern Asia.

HIV STRCTURE.

It is an RNA virus

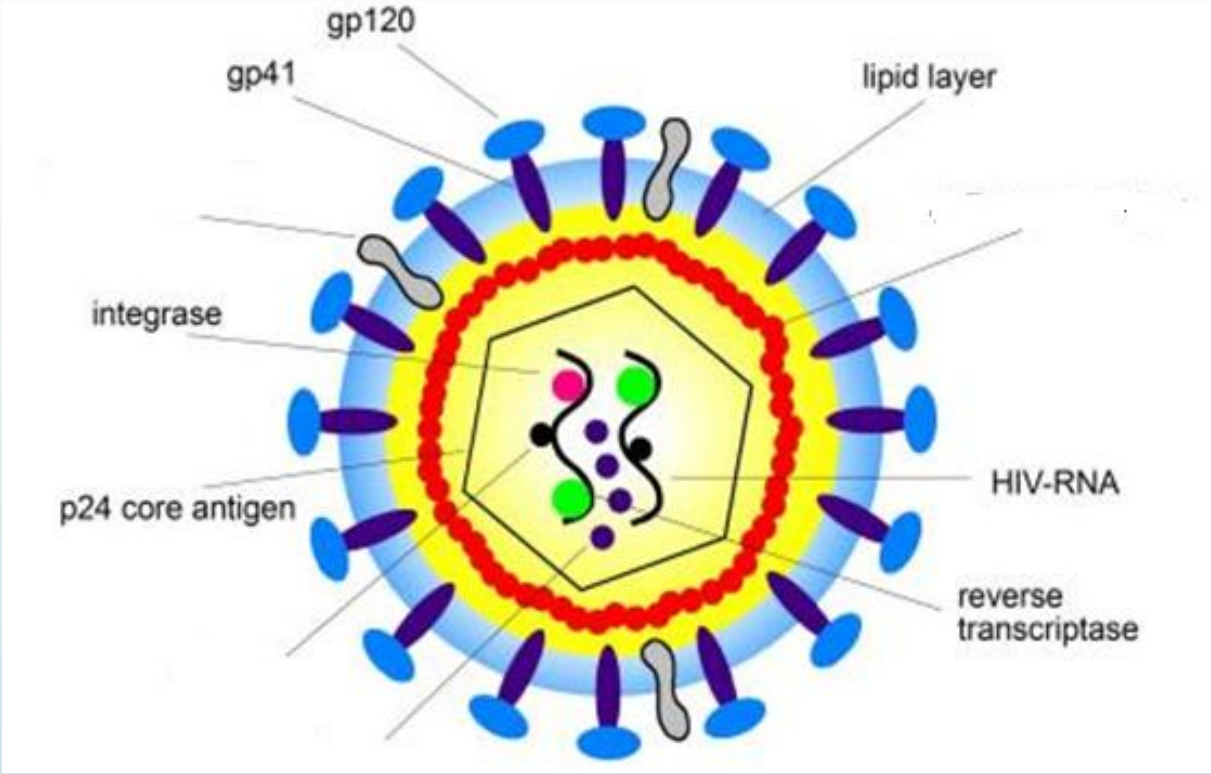
1)The core: contain the **genetic material** [RNA] and Reverse transcriptase [enzyme]

2) The capsid: outer protein coat.

3) Lipid Envelope (env) derived from infected cell, containing numerous external spikes formed by two major envelope proteins :

a) **The external gp 120**

b) **The trans membrane gp 41**



Transmission

- A person gets HIV when a body fluid :
- Blood , semen , vaginal fluid , Breast milk : enters his /her blood stream.
- The virus can enter the blood via:
- 1] the lining of the male/female genital tract (sexual activity).
- 2] The lining of the mouth (breast feeding from infected mother).
- 3] Broken skin .

Epidemiology: Transmission

HIV is fragile virus and cant live outside the human body

- **1] Sexual transmission**: unprotected sexual contact.

  [semen, vaginal fluid and blood]…. ***Repeated exposure↑ Risk***.

- Heterosexual transmission :

  More than 80 percent of infections worldwide.

  Sub-Saharan Africa

  A) **Houses the majority of the world's HIV-infected population**

  B) **Heterosexual transmission is the main contributor to the HIV epidemic.**

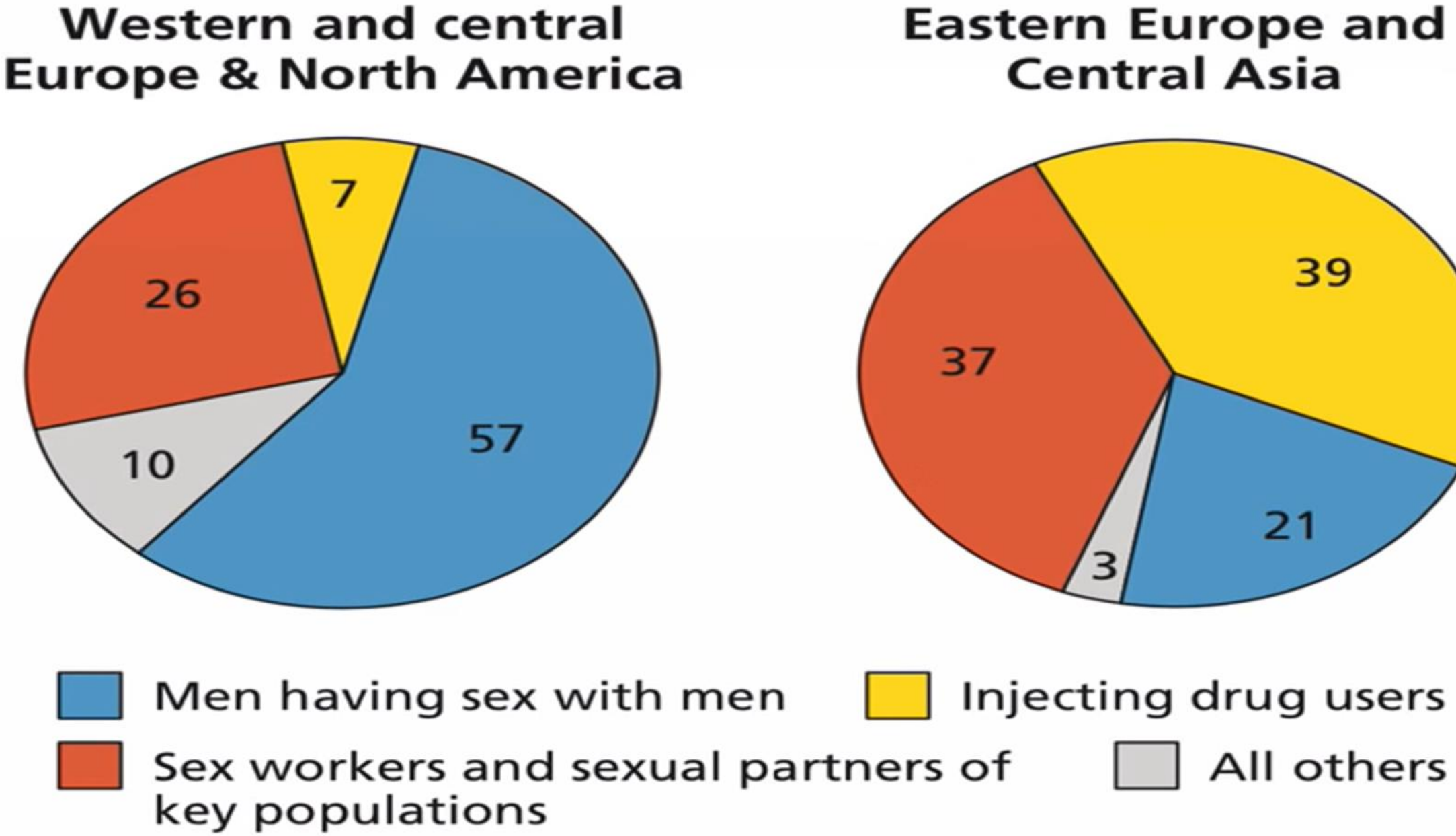
United States, Since 2009:

Newly diagnosed HIV infections attributed to

MSM sexual contact **increased** , **68% of newly diagnosed HIV,** while

Injection drug use and **heterosexual contact** **decreased**.

Epidemiology: Transmission



Epidemiology: Transmission

- **2] Injection drug use** :
- **Outside of sub-Saharan Africa**, injection drug use **(IDU)** accounts for approximately **30 percent of new HIV infections** in **Central and Eastern Europe** and in some countries of Asia.
- **Unfortunately, 40 percent of countries** with documented injecting drug use do not have needle-syringe programmes in place.

Transmission

- **3] Mother-to-child transmission: [MTCT]**
- **Over two million infants** are born to HIV-infected women annually.
- These children are vulnerable to HIV transmission:

in utero (30%) , **at birth (60%)** , or through **breastmilk (10%)**…..and Without antiretroviral preventive interventions, the risk of perinatal HIV transmission has varied between **15 and 45 percent**.

Mother-to-child transmission accounts for 90% of HIV infections among children worldwide.

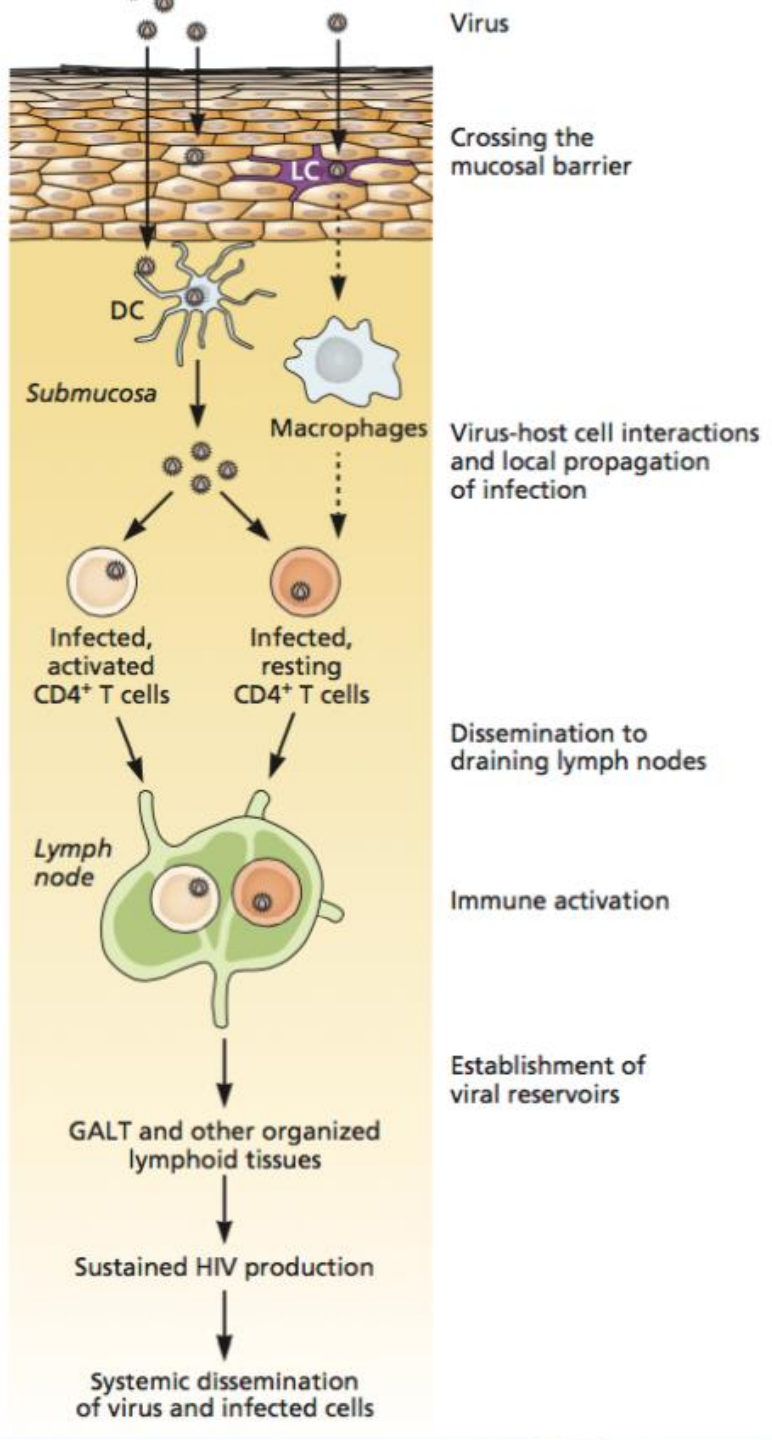
- Certain countries in sub-Saharan Africa, **20 to 40 %** of pregnant women are HIV-infected, and **one-third of their babies become infected.**

Transmission

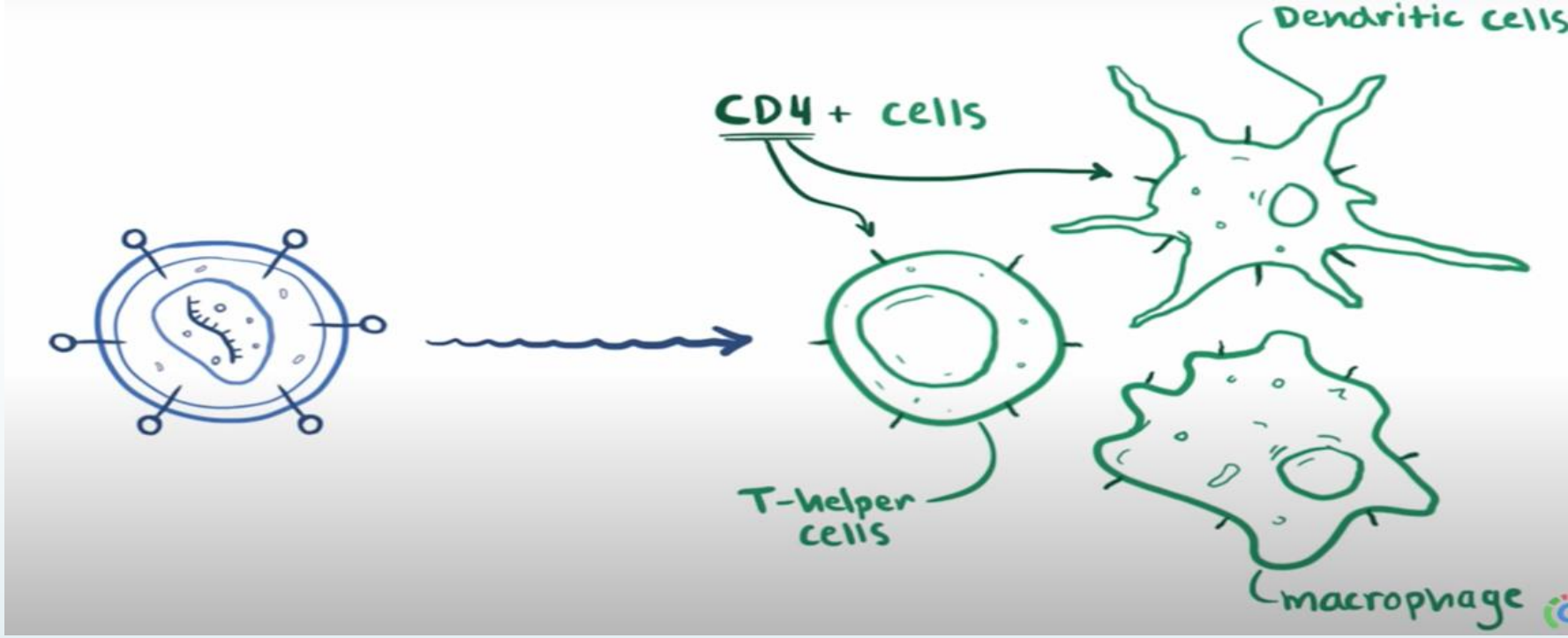
- **Blood product recipients**.
- **Health care workers with a needle-stick exposure.**

- **Risk factors for HIV transmission:**
- High viral load.(**Acutely infected or chronically untreated patient**)
- Certain sexual behaviours.(MSM is more)
- Presence of ulcerative sexually transmitted infections.
- lack of circumcision.

Pathogenesis:



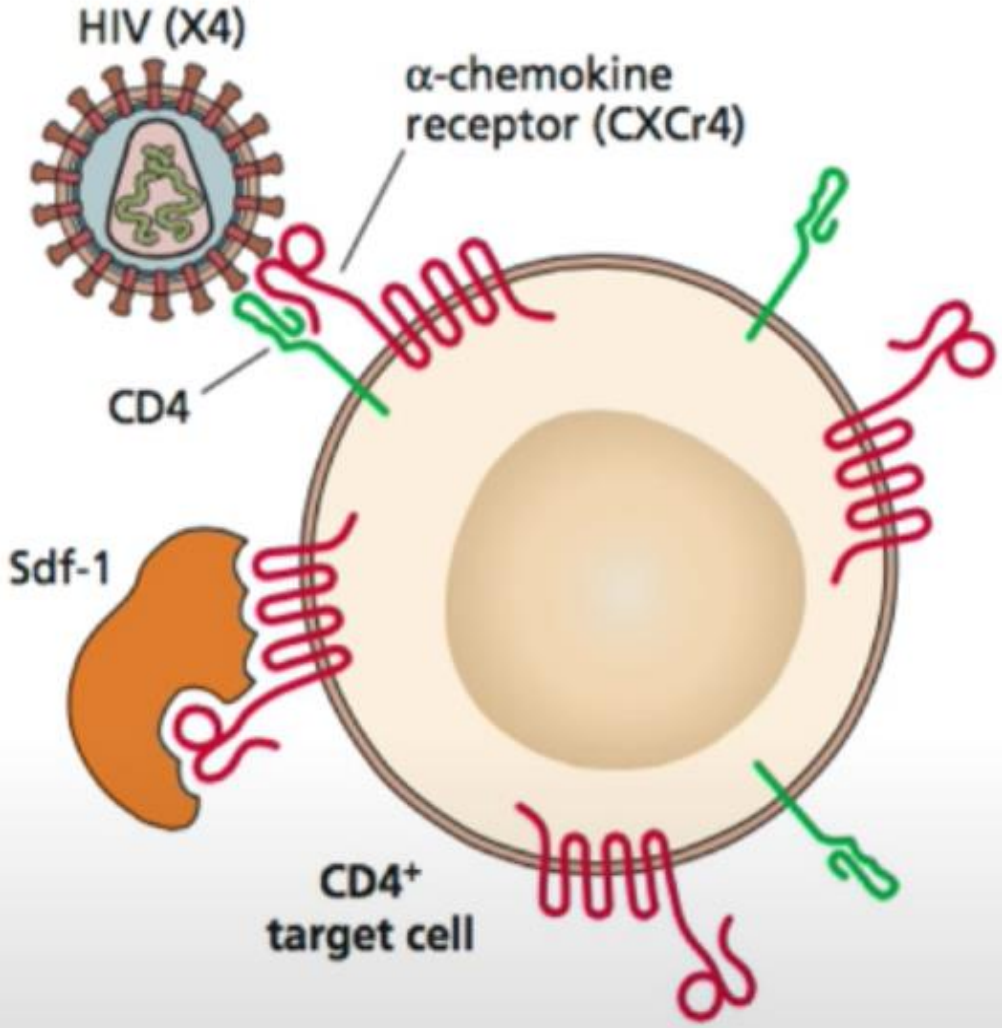
PATHOGENESIS

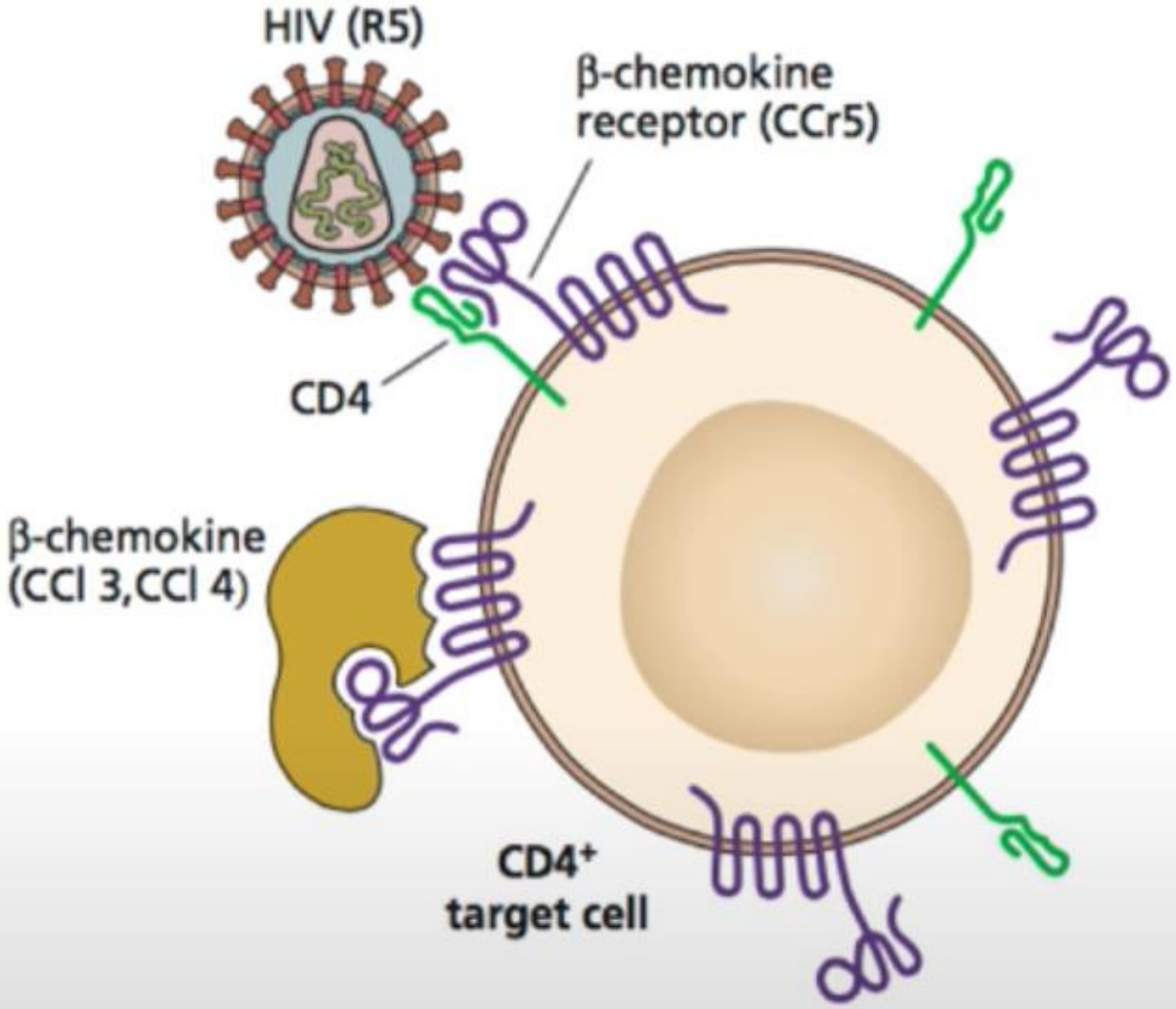


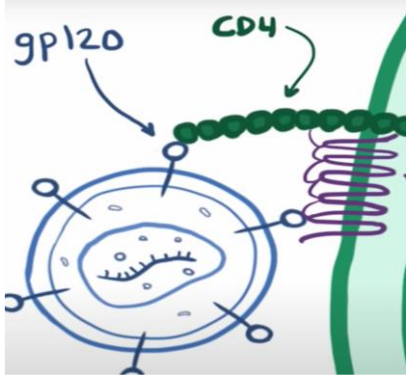
- **Target cells**: **CD4+ cells**.
  - Has several targets including:
  - Dendritic cells.
  - Macrophages.
  - CD4+ T cells.

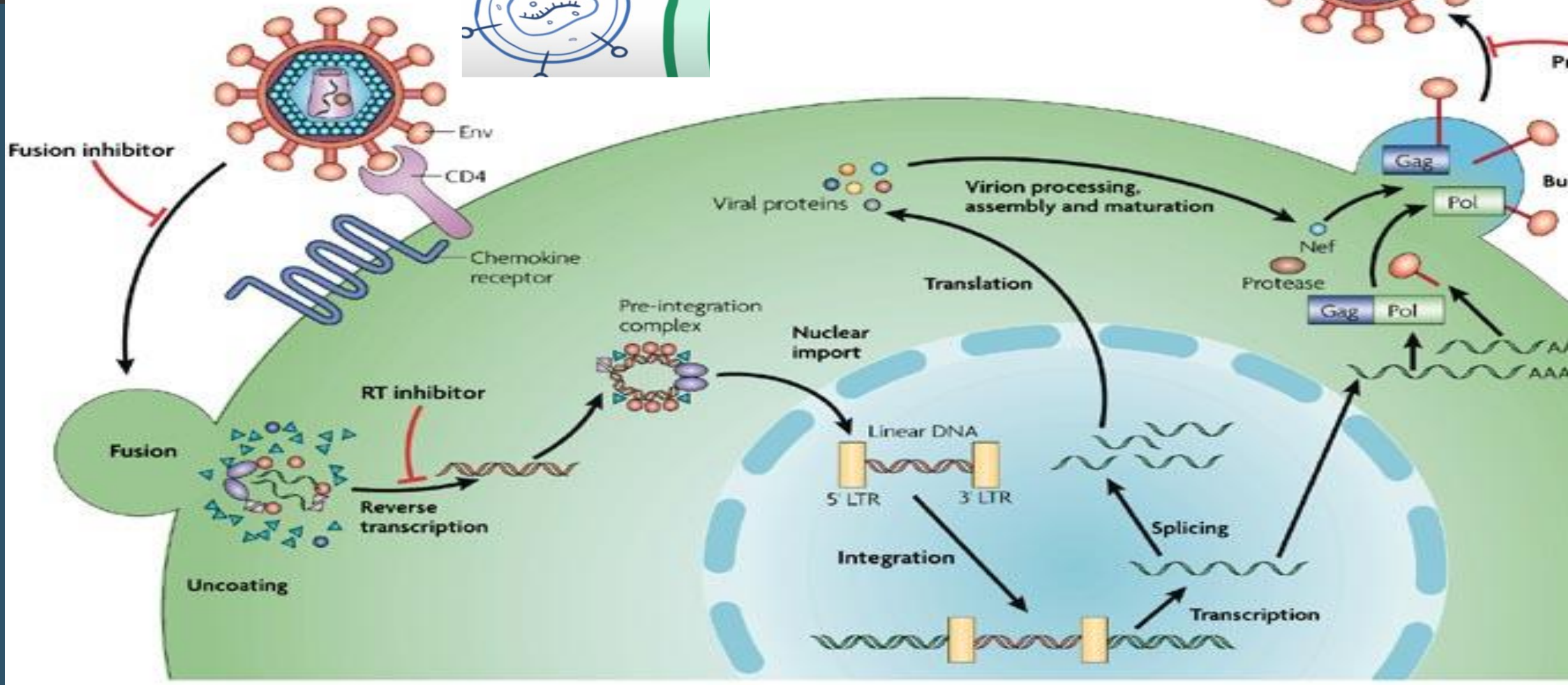
CO-RECEPTORS:

Viral entry into these cells is mediated by different receptors. **GP-120 must bind to CD4+ receptor as well as to the chemokine receptor CCr5 or CXCr4**






gp120
CD4


Fusion inhibitor
Env
CD4
Chemokine receptor
Viral proteins
Virion processing, assembly and maturation
Gag
Pol
Nef
Protease
Translation
Pre-integration complex
Nuclear import
RT inhibitor
Fusion
Linear DNA
5' LTR
3' LTR
Reverse transcription
Uncoating
Integration
Splicing
Transcription

Pathogenesis

- HIV RNA levels rapidly increase to a peak level that usually **coincides** with seroconversion.

- **Cellular immune response**
- At the time of initial infection with HIV, patients have a large number of susceptible CD4+ T cells and no HIV-specific immune response. **Therefore**

- Viral replication is rapid; plasma HIV RNA levels may climb to more than 107 COPIES/mL.

- Concomitant with the evolution of HIV specific immunity (**virus-specific CD8+ cytotoxic T lymphocytes**) This will lead to:

  a fall in plasma RNA levels precipitously by 2 to 3 logs, and symptoms of the acute retroviral syndrome resolve.

Pathogenesis

- **Elite controllers**:

A few individuals with HIV may, even in the absence of antiretroviral therapy, retain normal CD4 counts and low or undetectable plasma viremia.

- **Genetic susceptibility**:

The most extensively studied of these genetic factors is the C-C chemokine receptor 5 (CCR5), **a major receptor for HIV……and**

CCR5 (delta) 32 homozygotes genotype: people who inherited the Delta 32 mutation, resulting in the genetic deletion of a portion of the CCR5 gene are **highly resistant** to HIV infection.

- During the initial period, the effector memory $CD4^+$ T cells in the gut mucosa are preferentially depleted.
- followed by the progressive generalized loss of naïve T cells.

- A latent HIV reservoir is a group of immune system cells in the body that are infected with HIV but are not actively producing new HIV.
- Finding ways to target and destroy latent reservoirs is a major challenge facing HIV researchers who are exploring different strategies for clearing out reservoirs.

- HIV medicines prevent HIV from multiplying, which reduces the amount of the virus in the body (called the viral load). Because the HIV-infected cells in a latent reservoir are not producing new copies of the virus, HIV medicines have no effect on them.

- **Stage 1: Acute HIV infection:**
  Flu – like infection OR no symptoms.
  Large viral load / highly contagious.

Stage 2: chronic HIV infection.
Clinical latency / asymptomatic
Viral load increases a lot and
CD4 cell count decreases.

Stage 3: Immune system is seriously damaged

Clinical manifestations.

- **Acute HIV infection** : **(two to four weeks after exposure)**
- Flu – like infection OR no symptoms.
  Large viral load / highly contagious.

Mononucleosis syndrome : Diagnosis can frequently be **missed by clinicians.**

Fever, fatigue, and myalgia/arthralgia : the most common symptoms.

lymphadenopathy, sore throat, rash,, diarrhoea, weight loss.

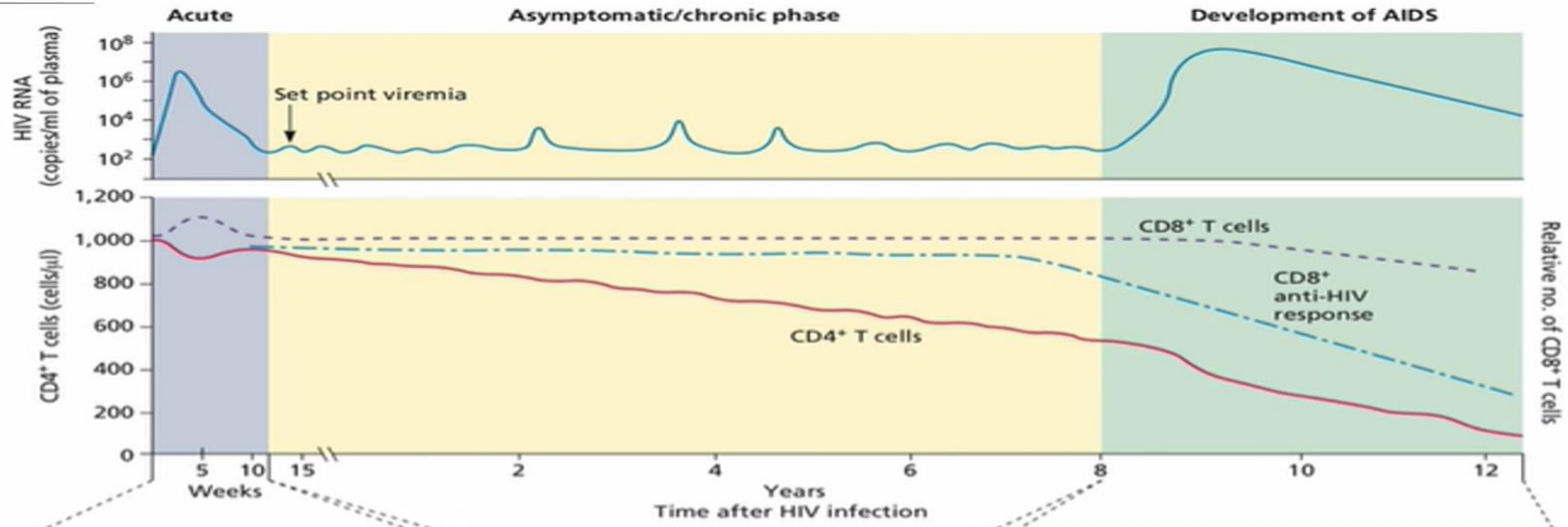
None OF THESE SYMPTOMEs is specific **But**:

Prolonged duration of symptoms. are suggestive of the diagnosis.

Most of the symptoms are self-resolving.

Asymptomatic infection : 10 to 60 percent .

Progression of HIV infection



Acute phase.

Symptoms

Fever.

Diarrhoea.

Lymphadenopathy.

At cellular level

$CD4^+$ T cell decline temporarily.

$CD8^+$T cell increase temporarily.

Chronic phase

Symptomes

Usually: No symptoms

Sporadically: fatigue, weight loss, Lymphadenopathy. Shingles.

At cellular level:

CD4+ T cells: gradually decline.

CD8+T cells: remain high.

Symptomatic phase

CD4+ T cells count:

200-500 CD4+ T cell/ml

Reactivation of latent tuberculosis

Kaposi Sarcoma.

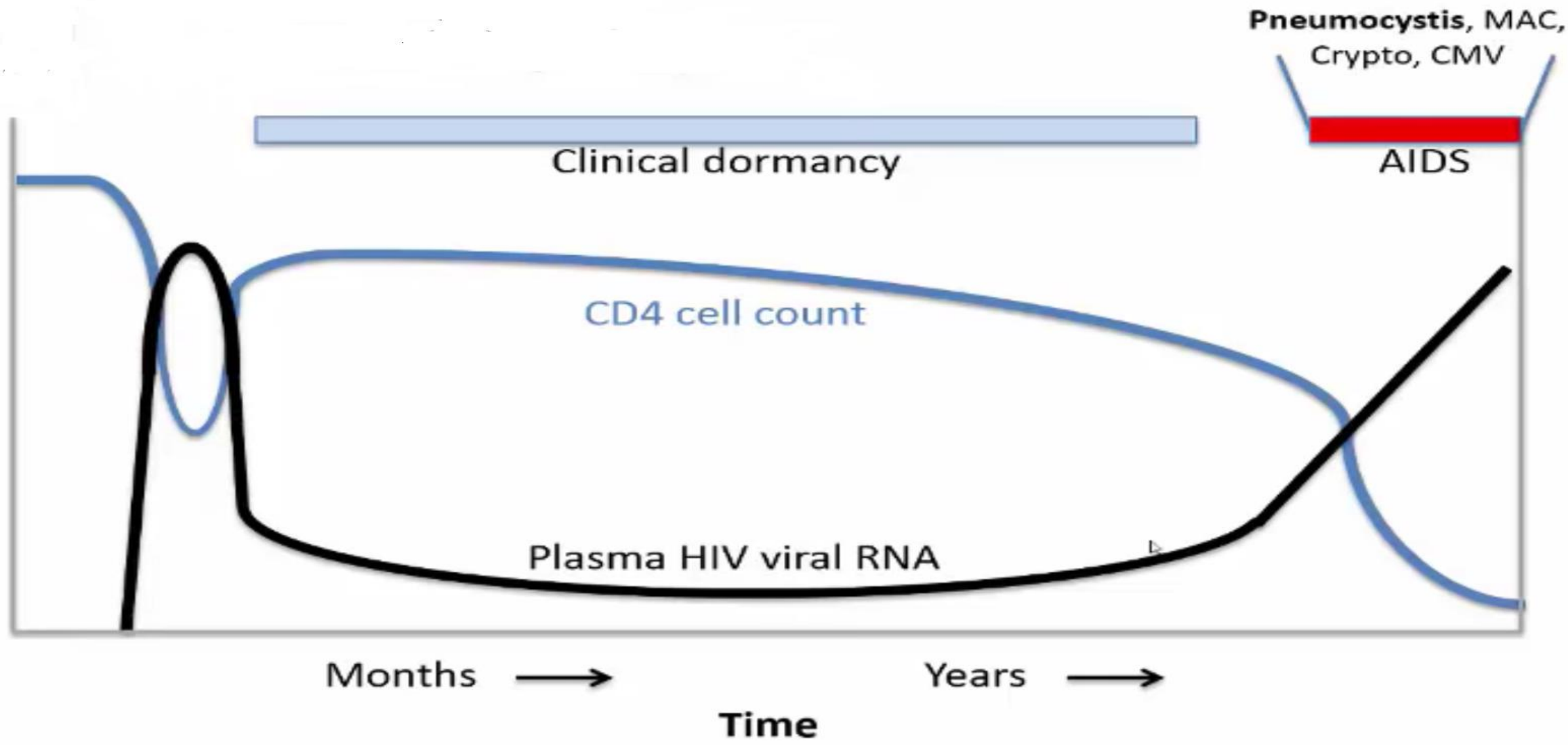
< 200 CD4+ Tcell/ml

Opportunistic infections.

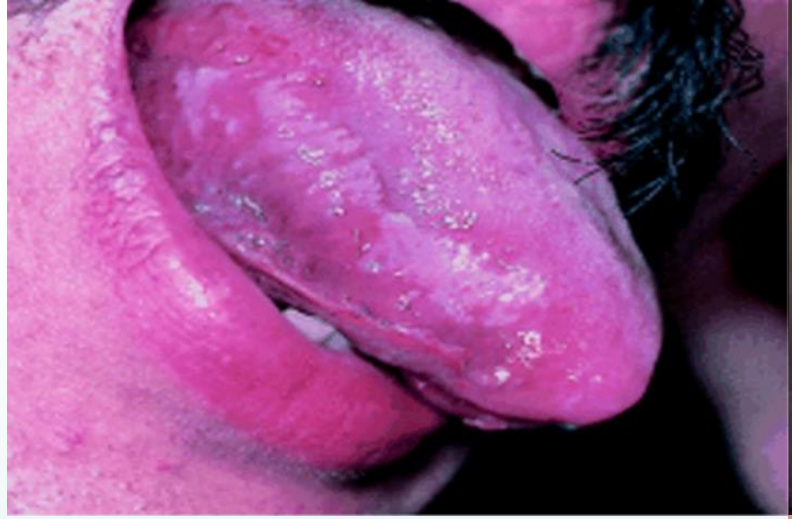
Malignancies

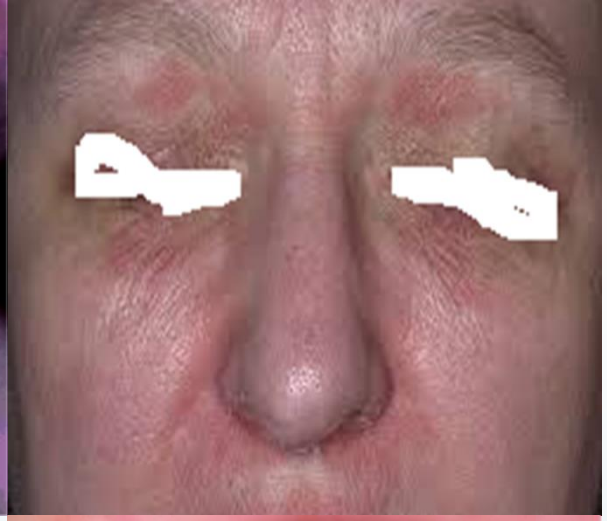
Neurological symptoms.

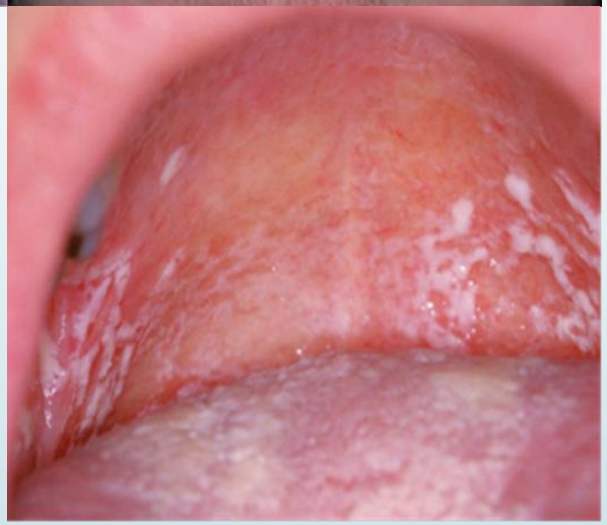
Natural history.



Clinical features:







Physical examination:

- Skin: condition associated with HIV

  Seborrheic dermatitis,

- Oropharynx:

  1) **Oral trush** 2) **Hairy leukoplakia**

  3) **Mucosal kaposi sarcoma**

- Lymph node:

  Generalized lymphadenopathy.

- Eyes:

  Fundoscopy : **CMV retinitis.**

- Génital exam:

  Condylomatous lesions :

  Condyloma acuminatum: A wart, found on the genitals It is caused by human papilloma virus. (STD).

  Condyloma latum: wart-like lesions on the genitals due to syphilis (STD).

Complication.

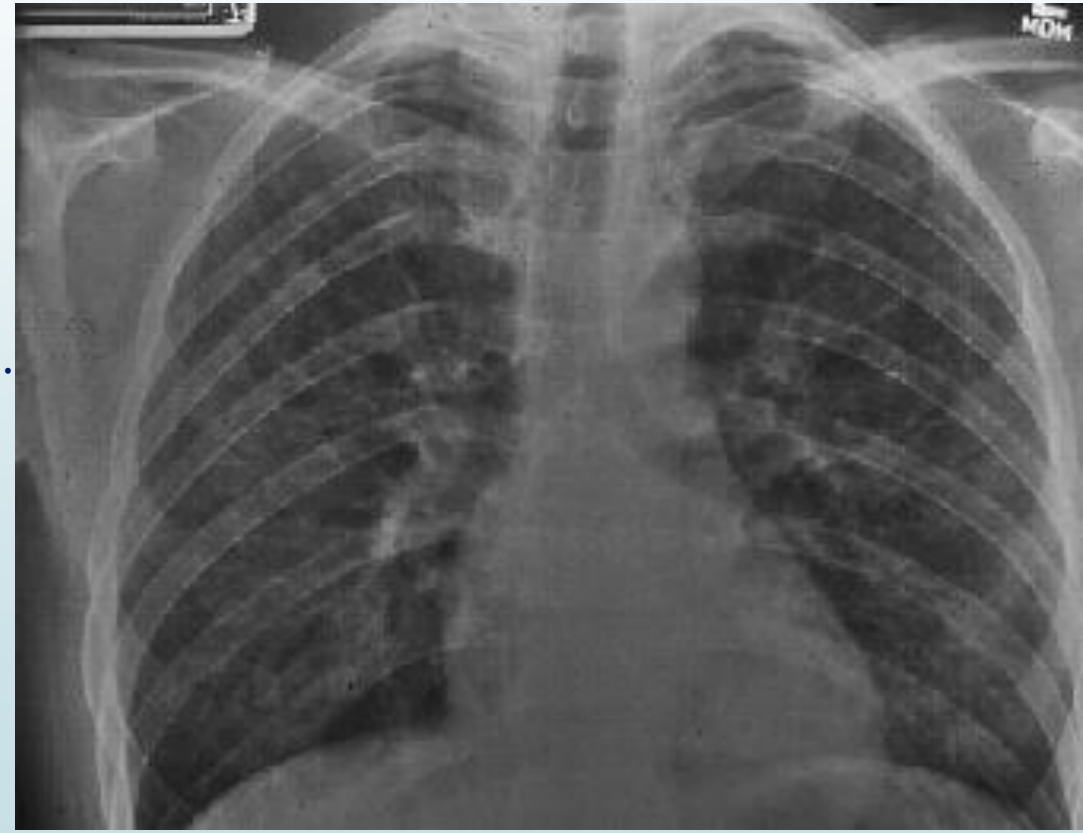
1] Infections:

- **Pneumocystis jirovecii Pneumonia.**
- One of the leading causes of **opportunistic infections** among persons with HIV and low CD4 cell counts.
- Occur in those who are unaware of their HIV diagnoses or are not receiving medical care.
- Pneumocystis is currently recognized as a fungus.(atypical fungi). based upon ribosomal RNA and other gene sequence homologies.
- Transmission of P. jirovecii is via the airborne route.
- It causes lower respiratory tract infection in severely immunosuppressed patients.

Case discussion

- **22 years old young male who presented with** :
- Progressive shortness of breath, cough and dyspnoea for 2 wks.
- Examination: looks in respiratory distress with RR : 28/m
- No focal lung findings
- Oral thrush
- Decreased oxygen saturation.
- Chest x-ray: diffuse lung infiltrate:
- HIV antibody: Reactive . CD4: 27cells/microL.
- Elevated LDH.

- DX: AIDS with Pneumonia. Likely to be Pneumocystis jirovecii.



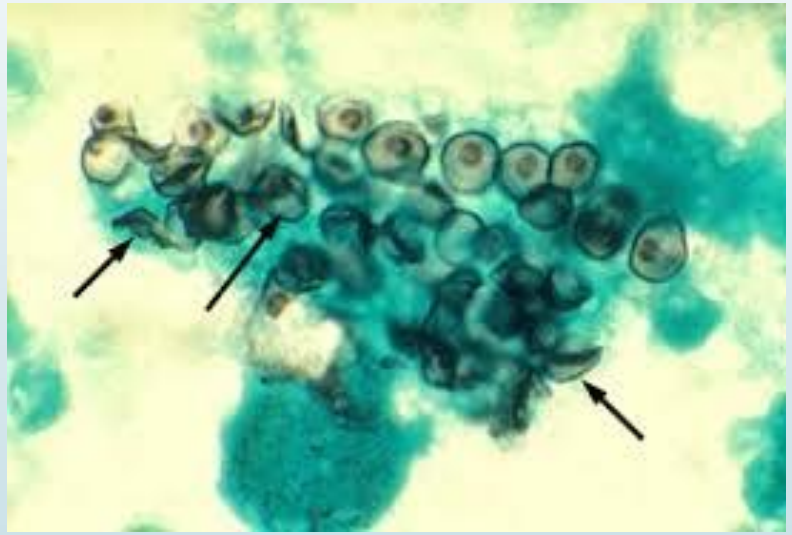
- **Consider pneumocystis pneumonia in certain populations**:
- 1] AIDS with CD4 < 200 cells.
- 2] Organ transplant
- 3] High dose of corticosteroid

- Laboratory DX: Definitive diagnosis of PCP requires **visualization of the cystic or trophic** forms in respiratory secretions by:

  methenamine silver stain.

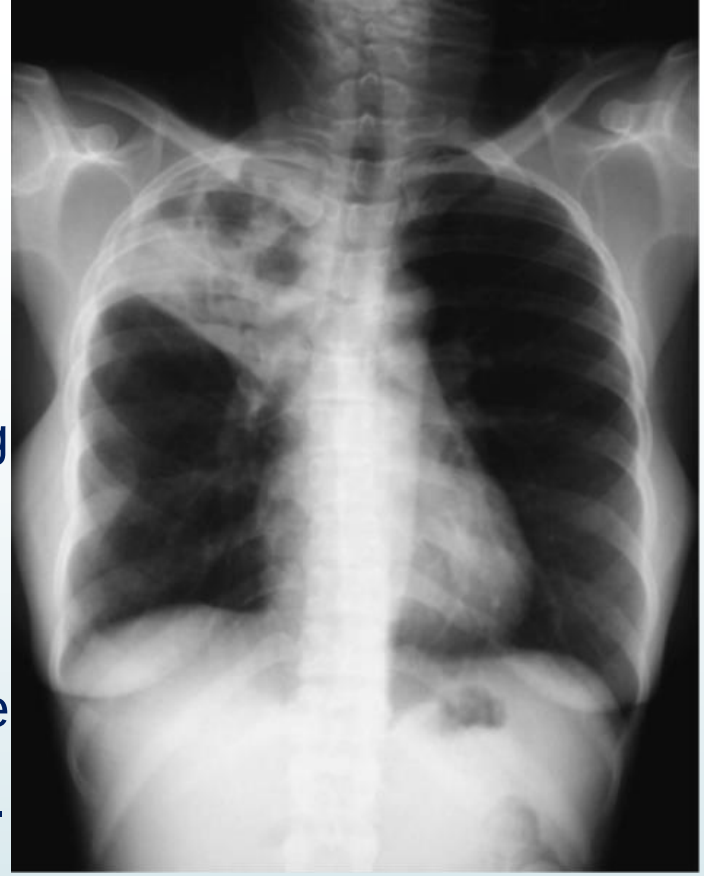
- Treatment:

  Trimethoprim-sulfamethoxazole.



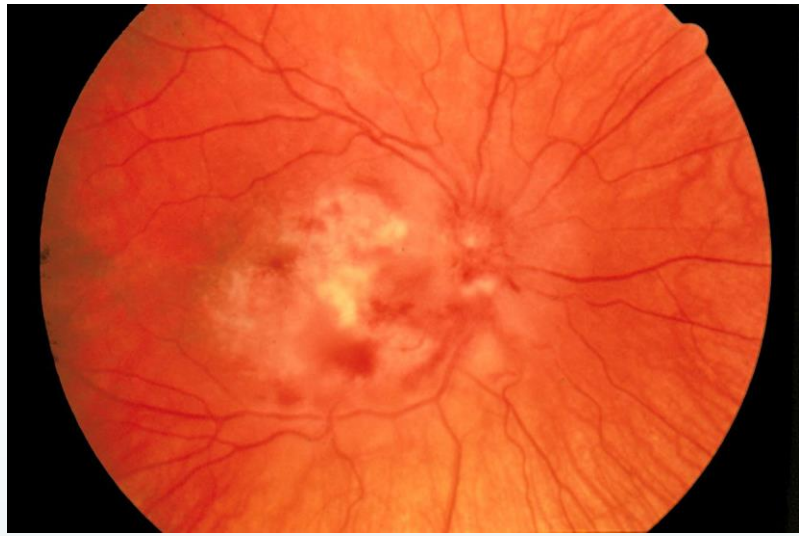
Pneumocystis cysts.

Tuberculosis



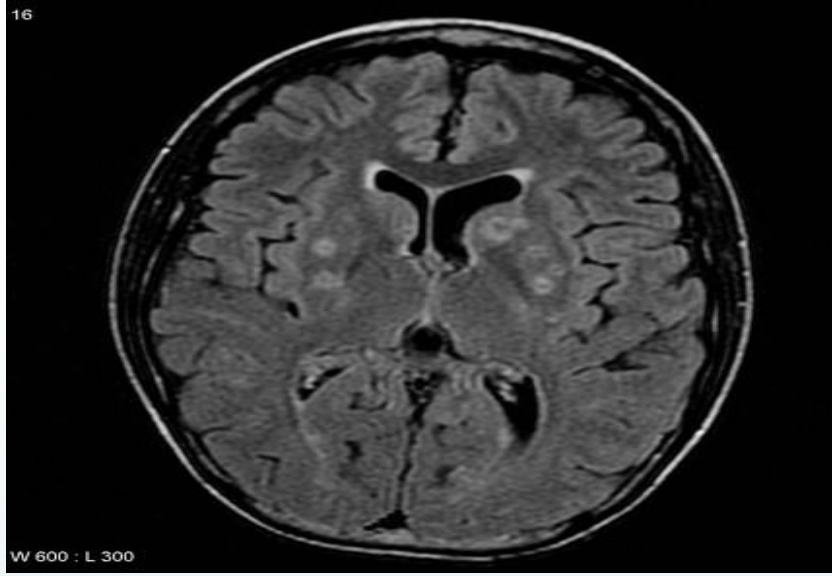
- Among people with **latent TB infection**, HIV infection is the strong known risk factor for progressing to TB disease.
- Patient infected with both HIV and TB is at least 10 times more like develop active TB, especially when their CD4 count is under 200.
- Worldwide, TB is a leading cause of death for people with HIV.
- It can be : 1] **Pulmonary** or 2] **Extrapulmonary**

Cytomegalovirus infection .(CMV)



- Cytomegalovirus (CMV) is a double-stranded DNA virus in the herpesvirus family that can cause disseminated or localized end-organ disease in people with HIV with advanced immunosuppression.
- Most clinical disease occurs in individuals previously infected with CMV experiencing **reactivation of latent infection.**
- End-organ disease occurs in patients with advanced immunosuppression, typically those with CD4+ T lymphocyte cell (CD4) counts <50 cells/mm^3.
- **CMV retinitis** is the **most common CMV end-organ disease in HIV patients**.(30% without ART).
- **CMV Colitis**. The most frequent clinical manifestations are weight loss, fever, anorexia, abdominal pain, diarrhea, and malaise.
- **CMV Esophagitis** occurs in a **small percentage** of patients with AIDS and causes odynophagia, nausea, and occasionally fever.
- It Can be treated with Antiviral drugs : Ganciclovir (IV) OR Valganciclovir (Orally).

Toxoplasmosis

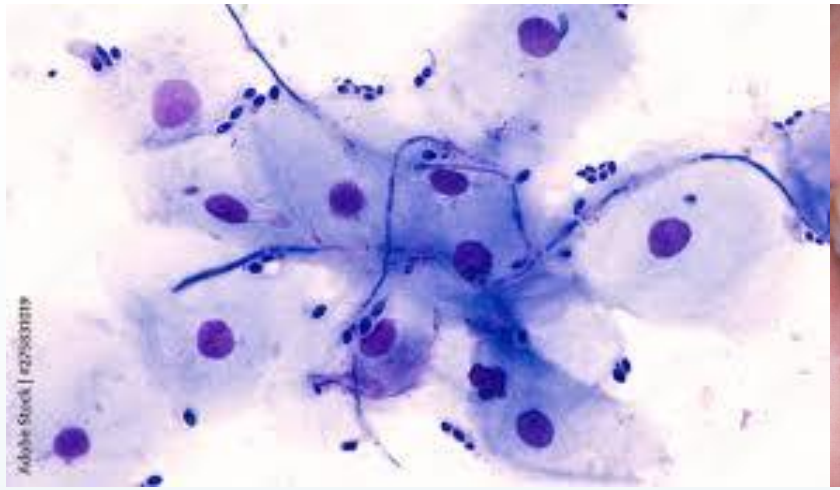


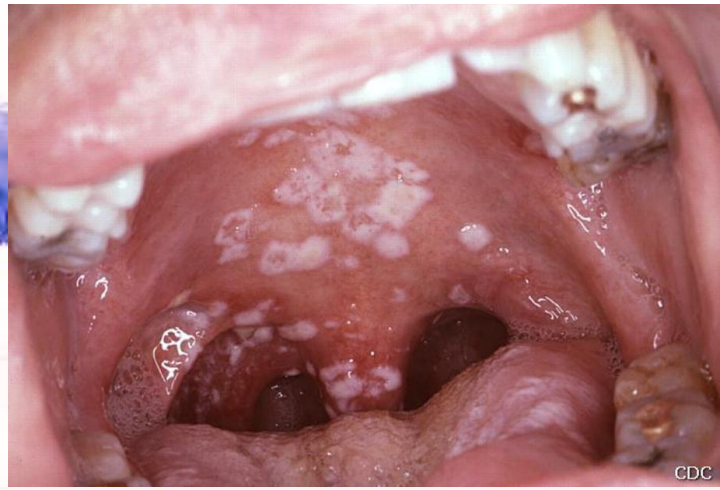
- ***Toxoplasma gondii*** **is an obligate intracellular protozoan parasite presenting as a zoonotic infection distributed worldwide.**
- **Symptomatic disease most often occurs as a complication of reactivation of latent infection.**
- **It causes severe opportunistic infections as a result of reactivation of the parasite if the CD4 counts fall below 100 cells/µl.**
- **The most common site of reactivation is the central nervous system.**
- **CNS toxoplasmosis begins with encephalitis :**

Headache. Later, confusion and drowsiness, seizures, focal weakness. Fever is usually but not always present.

- **CT-SCAN or MRI : Single or multiple hypodense or hypointense lesions in white matter and basal ganglia.**
- **Standard therapy consists of:**
- **pyrimethamine, sulfadiazine. or**
- **Trimethoprim-sulfamethoxazole an alternative regimen.**

Candidiasis.





The infection can be :

Mucosal and disseminated

- Mucosal candidiasis, in the form of oral, esophageal or vaginal:
- **Thrush or oropharyngeal candidiasis** : White patches on the inner cheeks, tongue, roof of the mouth, and throat

- **Candida esophagitis** : is one of the most common infections in people living with HIV/AIDS and present with dysphagia, **odynophagia**, and retrosternal pain.

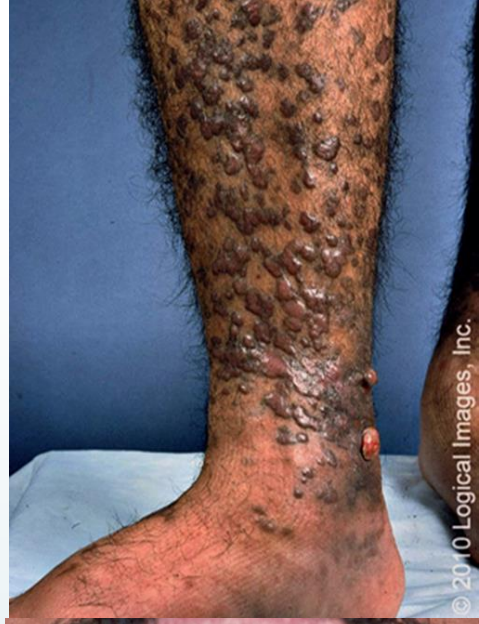
Diagnosis: Endoscopy shows white-yellow plaques.

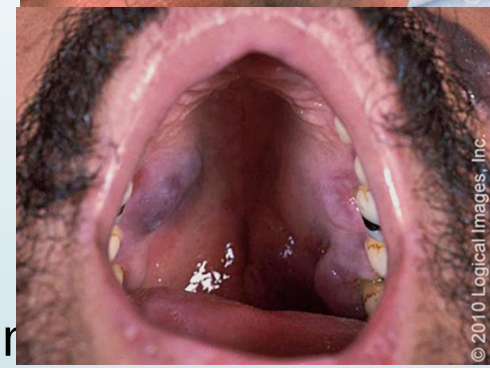
Confirmation: Histologic examination using Hematoxylin and eosin stain of biopsies or brushing of esophageal plaques showing pseudohyphae

Treatment : Antifungal : Oral fluconazole.

2] Malignancy:...AIDS-defining conditions

- Kaposi sarcoma: **HIV is 500 times more likely to be diagnosed with KS.**
- Non-Hodgkin lymphoma.
- Cervical cancer.





Kaposi sarcoma(KS):

Is a vascular tumour that is associated with human herpesvirus 8 (HHV-8).

It is the **most common tumour** arising in HIV-infected persons.

It is most common in **homosexual** or bisexual men, and is much less common among injection drug users, transfusion recipients, women or children, and haemophiliacs.

KS is considered an AIDS-defining illness and is predominantly a disease of men.

Incidence has declined substantially since widespread use of potent antiretroviral therapy.

Diagnosis.

- **Nearly 15 percent** of HIV-infected persons in the United States remain unaware of their HIV infection.
- **WHOM TO TEST:**

1] Symptoms of HIV infection:

Signs and symptoms of acute or chronic HIV infection should be tested. testing for HIV RNA may be needed.

2] Possible HIV exposure:

Patients after a known high-risk exposure to HIV (e.g., sexual or percutaneous).

3] Patient with sexually transmitted disease (STD).

4] Pregnant women should be tested for HIV early in each pregnancy. [Screening]

Tests:

- **1] HIV antibody tests** only look for antibodies to HIV . Detect HIV infection **23 to 90 days** after an exposure.

- **2] Antigen/antibody test:** looks for both HIV antibodies and antigens.

  detect HIV infection **18 to 45 days after an exposure.**

- **3] A NAT (PCR)** looks for the actual virus in the blood. **10 to 33 days after an exposure.**

- **Rapid tests:**
- **1) The rapid antigen/antibody test with a finger prick and takes 30 minutes.**
- **2) The Oral fluid antibody self-test provides results within 20 minutes.**

Time of positivity of HIV diagnostic tests

Test	Target of detection	Approximate time to positivity (days)
Enzyme-linked immunoassay		
First generation	IgG antibody	35 to 45
Second generation	IgG antibody	25 to 35
Third generation	IgM and IgG antibody	20 to 30
Fourth generation	IgM and IgG antibody and p24 antigen	15 to 20
Western blot		
	IgM and IgG antibody	35 to 50 (indeterminate)
		45 to 60 (positive)
HIV viral load test		
Sensitivity cutoff 50 copies/mL	RNA	10 to 15
Ultrasensitive cutoff 1 to 5 copies/mL	RNA	5

- **Positive result** with **third**-(HIV- antibody only) and/or **fourth**-generation (HIV antigen and antibody) HIV serologic assays should be confirmed by :

  Confirmatory HIV-1/HIV-2 antibody differentiation immunoassay.

Treatment

- **HIV can be suppressed by treatment regimens:**
- **Current ART does not cure HIV infection but highly suppresses viral replication and allows an individual's immune system recovery to strengthen and regain the capacity to fight off infections.**
- **HIV/AIDS has become a manageable chronic health condition, enabling people living with HIV to lead long and healthy lives.**
- **The standard of care today is to treat nearly all HIV-infected individuals with ART, regardless of CD4 count.**

- **RATIONALE FOR UNIVERSAL TREATMENT**:

- **Reduce HIV infection–related morbidity and prolong duration and quality of life**
- **Restore and preserve immunologic function.**
- **Maximally and durably suppress viral load (plasma HIV RNA)**
- **Prevent HIV transmission.**

Treatment

- Patients with a CD4 count ≤350 :
  Initiating ART results in a significant decline in the risk of AIDS-related morbidity and mortality .

- Patients CD4 count <200 cells **[AIDS] :** ART improves survival and delays disease progression.

Medication

- **Antiretroviral drugs:**
- Fusion / Entry inhibitors: not used currently :parentral inj.
- Reverse transcriptase inhibitor:
- A] Nucleoside Analogue RTI.

  Abacavir(ABC)

  Emtricitabine(FTC)

  Lamivudine(3TC)

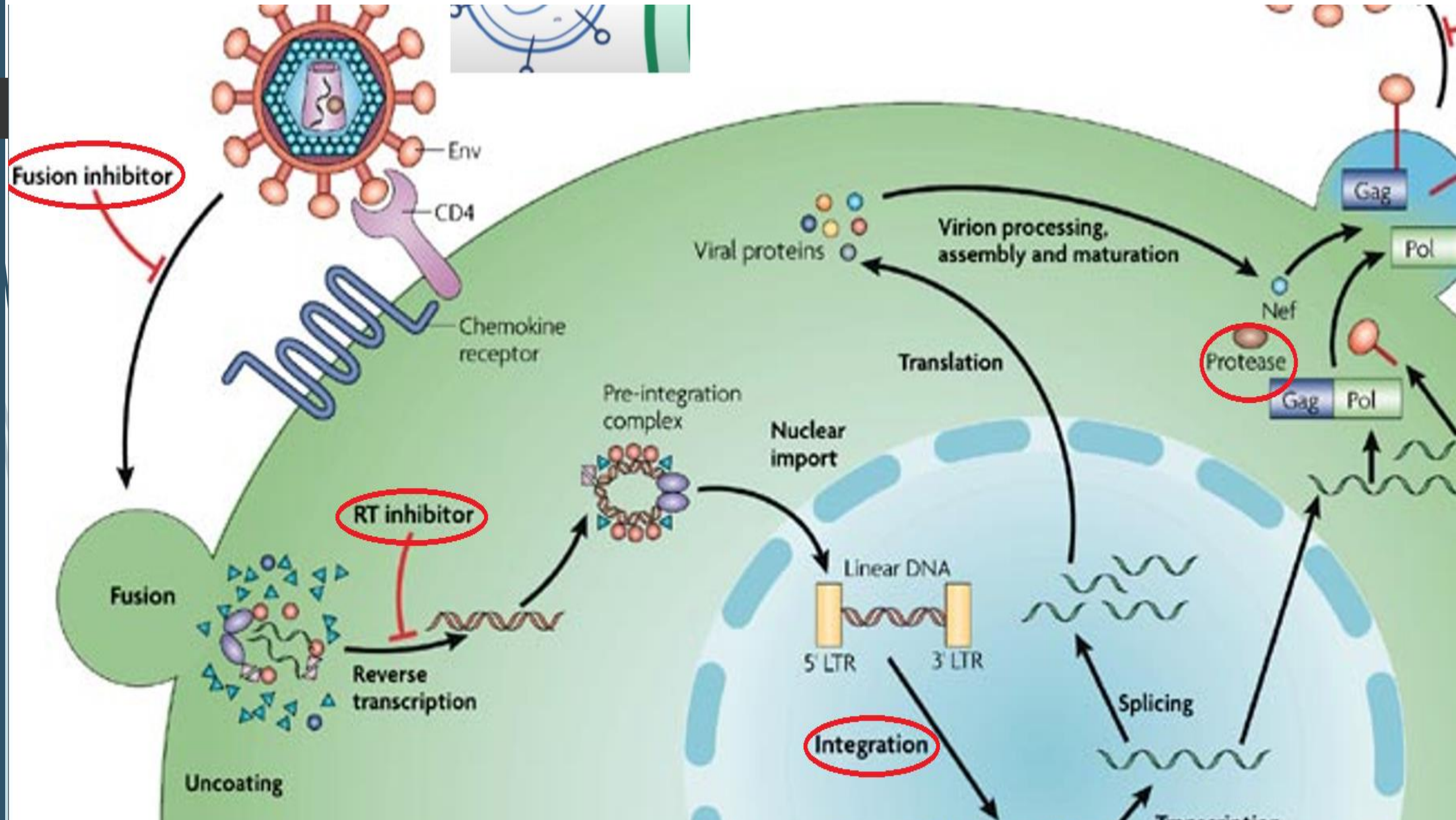
  Tenofovir

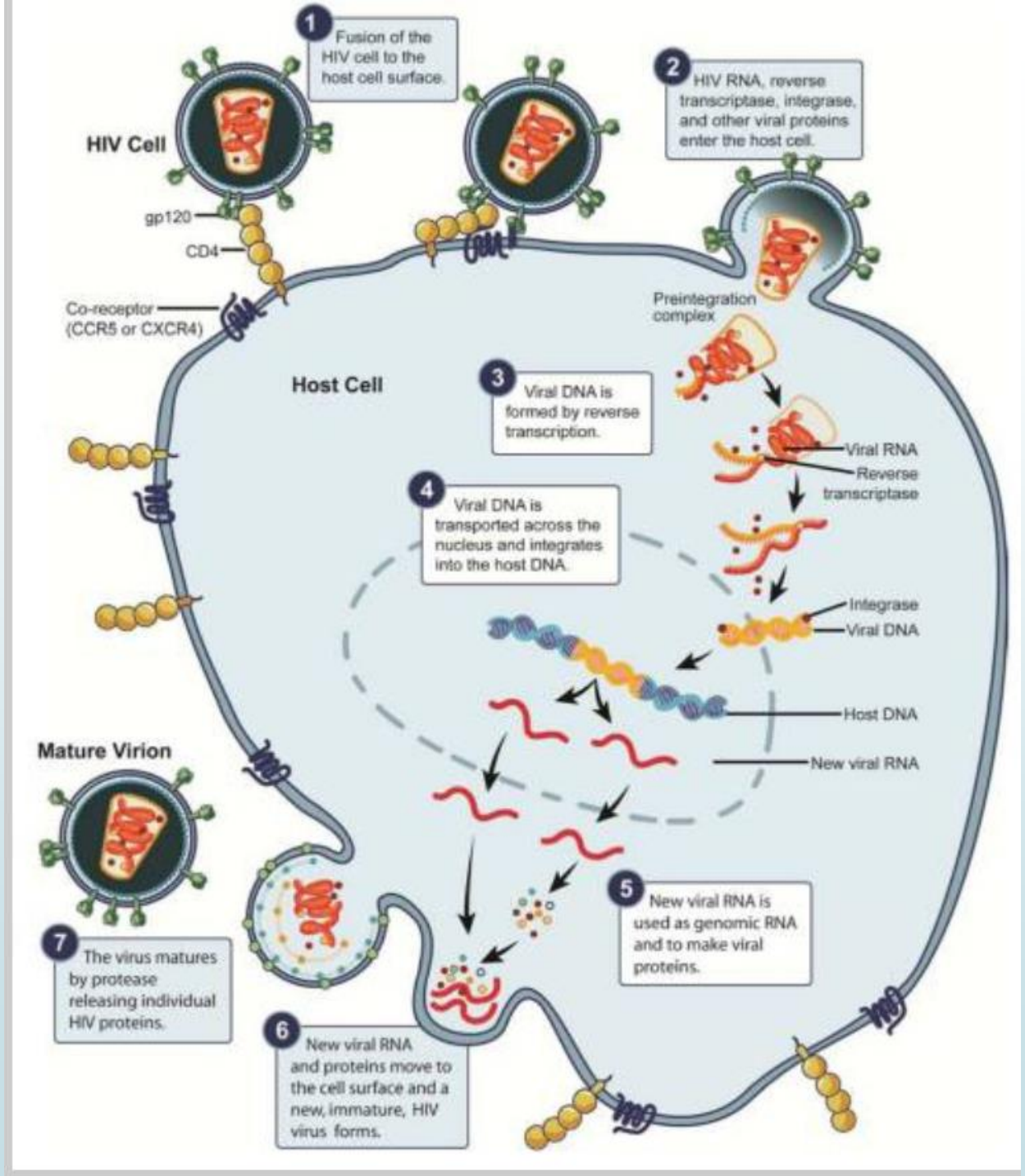
- B] Non-nucleoside RTI

  Efavirenz

  Nevirapine

- Integrase inhibitors : raltegravir, dolutegravir, elvitegravir, bictegravir
- Protease inhibitors : Atazanavir & Darunavir


Env
CD4
Fusion inhibitor
Chemokine receptor
Viral proteins
Virion processing, assembly and maturation
Gag
Pol
Nef
Protease
Translation
Gag Pol
Pre-integration complex
Nuclear import
RT inhibitor
Linear DNA
Fusion
5' LTR
3' LTR
Splicing
Reverse transcription
Integration
Uncoating


1 Fusion of the HIV cell to the host cell surface.
HIV Cell
gp120
CD4
Co-receptor (CCR5 or CXCR4)
2 HIV RNA, reverse transcriptase, integrase, and other viral proteins enter the host cell.
Preintegration complex
Host Cell
3 Viral DNA is formed by reverse transcription.
Viral RNA
Reverse transcriptase
4 Viral DNA is transported across the nucleus and integrates into the host DNA.
Integrase
Viral DNA
Host DNA
New viral RNA
Mature Virion
5 New viral RNA is used as genomic RNA and to make viral proteins.
7 The virus matures by protease releasing individual HIV proteins.
6 New viral RNA and proteins move to the cell surface and a new, immature, HIV virus forms.

Prevention

- The only absolute way to **Prevent sexual transmission** of HIV infection is

- **A] Abstinence from sexual relation completely**

- **B] Safer sexual contact** :

  Correct and consistent use of condoms during sexual contact have an **85%** or greater protective effect against HIV and other STIs .[**10 – 15 % failure rate**] .

- **C] CIRCUMCISION*** : results in 50 - 60% reduction of HIV acquisition and other STD.
- **D] Stop using IDUs :**
- **Needle exchange programs has resulted in reducing the number of new HIV infections by up to 70 percent like in Australia.**

- **E] Screen all blood** and blood products

- * Longitudinal studies among HIV discordant couples suggest that male circumcision.

Prevention:

- **F] Use of ARVs for prevention [Secondary prevention benefits of ART]**

  Several studies confirmed that if an HIV-positive person is taking ART and is virally suppressed [**Durably undetected**] they do not transmit HIV to other partner.

- **G] Pre-exposure prophylaxis (PrEP)** for HIV-negative partner.
- Oral PrEP of HIV is the daily use of ARVs by HIV-negative people to block the acquisition of HIV. **Its very effective in reducing HIV transmission.**

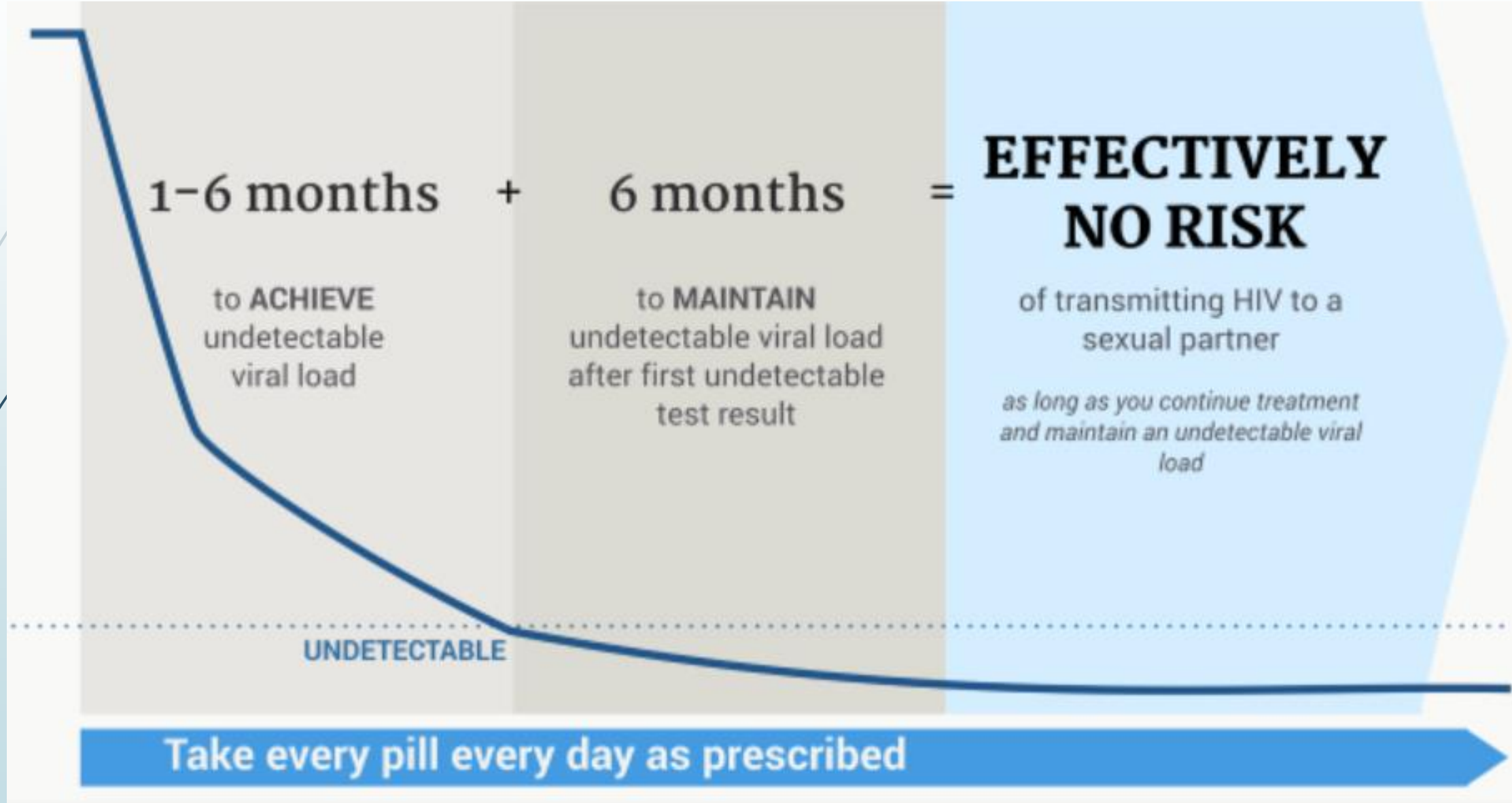
- **H] Post exposure Prophylaxis**:

  Sexual contact (unprotected)

  Health care associated percutaneous exposure.

  PEP is not recommended when care is **sought > 72 hours** after potential exposure.

Treatment as Prevention



Prevention:
Elimination of mother-to-child transmission of HIV

Pregnant women infected with HIV infection caries risk to infect her baby by:

1) In utero …25-40%
2) **Intrapartum …60-75%**
3) Breast feeding : 1) Established infection 14%
   2) Primary infection 29%

In the **absence of any interventions** during these stages, rates of HIV transmission from mother-to-child can be between **15% and 45%**

- **Today the risk of perinatal transmission is :**

  Less than 2% with :

✓ **Effective antiretroviral therapy (ART)**

✓ **Formula feeding**.