

HIV/AIDS and Zika Virus Co-Infection: A Comprehensive Review of Transmission Dynamics, Pathogenesis, and Treatment Strategies

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Abstract

The Human Immunodeficiency Virus (HIV) was first identified in humans in 1959 and is believed to have originated from chimpanzees, with transmission to humans occurring through contact with infected blood, likely during hunting or butchering. HIV/AIDS remains a significant global health challenge, affecting millions of individuals across diverse regions and populations. Acquired Immunodeficiency Syndrome (AIDS) is a condition in which the immune system progressively weakens, making the body vulnerable to severe infections.

HIV exists in bodily fluids as free viral particles and within infected immune cells, primarily targeting critical immune cells such as CD4⁺ T-helper cells and macrophages. Antiretroviral therapy (ART) effectively reduces viral load, preserving immune function and significantly lowering transmission risk.

This review examines the transmission dynamics of HIV and Zika Virus (ZIKV) co-infection using a mathematical model that incorporates both vertical and horizontal transmission. Two sub-models were developed: one for HIV-only infection and another for ZIKV-only infection. The HIV-only sub-model reaches a globally asymptotically stable disease-free equilibrium when the reproduction number is below unity. Conversely, the ZIKV-only sub-model exhibits a backward bifurcation phenomenon, where both stable disease-free and stable endemic equilibria coexist when the reproduction number is less than unity. This phenomenon complicates the effective control of ZIKV infection.

Keywords

HIV (Human Immunodeficiency Virus), AIDS (Acquired Immunodeficiency Syndrome), Retrovirus, Zika Virus, HIV Transmission, Antiretroviral Therapy, Acute Immunity, AIDS Syndrome, Drug Resistance

I. Introduction

Acquired Immunodeficiency Syndrome (AIDS) was first reported in medical literature in 1981 by the Centers for Disease Control and Prevention (CDC), initially affecting specific "risk groups" such as gay men and intravenous drug users. However, by 1983, cases were identified in men and women in Africa, signifying the transition of HIV into an epidemic within the heterosexual population, impacting both developed and developing nations.

HIV/AIDS is a global health crisis, affecting individuals in every country and emerging as one of the leading causes of death among young adults. Beyond its medical implications, HIV poses significant social challenges, including stigma, discrimination, and isolation due to its primary mode of sexual transmission and association with certain high-risk behaviors.

Addressing the multifaceted challenges of HIV management requires enhanced healthcare accessibility, community-driven initiatives, and strong policy frameworks. Strengthening collaboration among healthcare providers, community organizations, and policymakers is crucial in reducing barriers, improving care delivery, and enhancing the quality of life of people living with HIV. ART remains the cornerstone of HIV management, preventing disease progression to AIDS and enabling individuals to maintain a functional immune system.

II. Structure of HIV

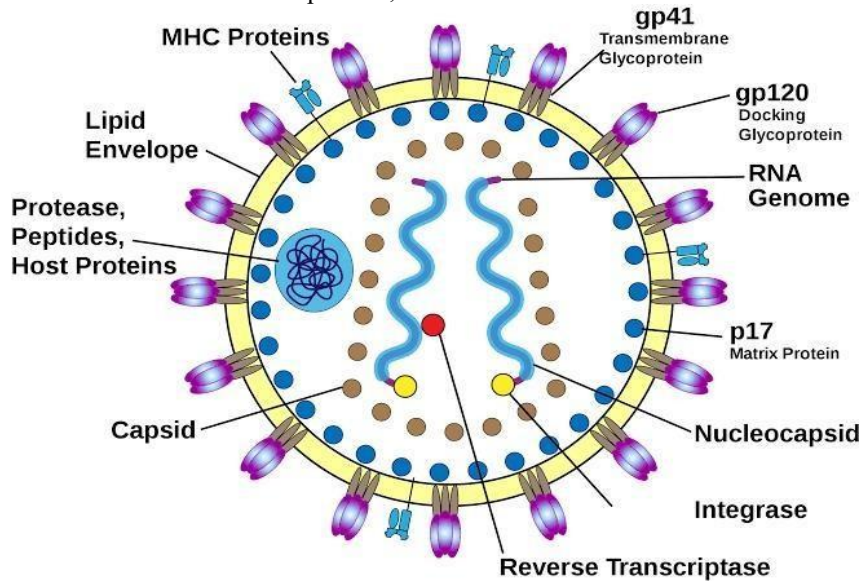
The structural components of HIV play a crucial role in its infectivity and replication cycle:

- **Gp41:** A sub unit of the envelope protein complex, essential for membrane fusion and viral entry into host cells.
- **Gp120:** A glycoprotein involved in binding to CD4 receptors and co-receptors (CCR5 or CXCR4) on host cells, facilitating viral entry.

- **P17 (Matrix Protein):** Forms part of the inner shell of the virus and plays a role in viral assembly.
- **P24 (Capsid Protein):** Constitutes the viral capsid and is critical for viral genome encapsulation and replication.
- **Protease:** An enzyme necessary for processing viral polyproteins into functional viral proteins,

facilitating viral maturation.

- **Integrase:** An enzyme responsible for incorporating viral DNA into the host genome, ensuring the persistence of HIV within infected cells.



Structure of HIV(human immunodeficiency virus)

III. Modes of HIV Transmission

HIV is transmitted through the exchange of bodily fluids from an infected person, including:

1. **Unprotected Sexual Contact:** Transmission occurs during vaginal, anal, or oral sex without condom use or pre-exposure prophylaxis (PrEP).
2. **Bloodborne Transmission:** Sharing contaminated needles, syringes, or exposure to infected blood increases the risk of infection.
3. **Mother-to-Child Transmission (MTCT):** HIV can be transmitted during pregnancy, childbirth, or breastfeeding.
4. **Contaminated Tattoo or Piercing Instruments:** Use of unsterilized needles for tattoos or piercings can result in HIV transmission.
5. **Concurrent Sexually Transmitted Infections (STIs):** STIs such as syphilis, herpes, or gonorrhea increase susceptibility to HIV due to mucosal damage and immune activation.

- Swollen lymph nodes
- Sore throat
- Rash
- Muscle aches
- Fatigue
- Diarrhea
- Oral ulcers

4.2 AIDS (Late-Stage HIV Infection)

AIDS represents the final stage of untreated HIV infection, characterized by severe immunosuppression and opportunistic infections such as:

- Chronic weight loss (wasting syndrome)
- Persistent diarrhea
- Night sweats
- Opportunistic infections (oral thrush, pneumonia, tuberculosis, Kaposi's sarcoma)

IV. Clinical Manifestations of HIV

4.1 Acute HIV Infection (Primary Stage)

Symptoms appear 2–4 weeks post-exposure and mimic flu-like symptoms, including:

- Fever
- Headache

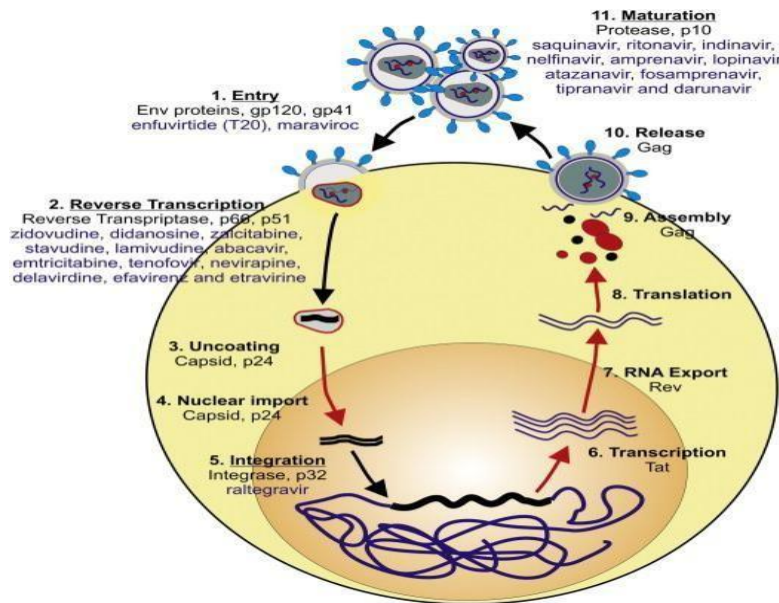
V. Life Cycle of HIV

HIV follows a multi-step replication cycle:

1. **Entry:** HIV binds to CD4+ T cells using gp120, facilitated by CCR5 or CXCR4 co-receptors.
2. **Reverse Transcription:** Reverse transcriptase

- converts viral RNA into complementary DNA (cDNA).
- Integration:** Integrase incorporates HIV cDNA into the host genome.
 - Replication:** The host cell synthesizes viral proteins and RNA.
 - Assembly:** Viral components assemble at the host

- cell membrane.
- Budding:** New HIV particles are released from the host cell.
 - Maturation:** Protease processes viral polyproteins, rendering the virus infectious.



VI. Diagnosis and Treatment of HIV

6.1 Diagnostic Methods

- **HIV Antigen/Antibody Test:** Detects p24 antigen and HIV antibodies.
- **Home HIV Test:** An FDA-approved rapid test for self-screening.
- **CD4 Count:** Measures immune function and progression risk.
- **Viral Load Test:** Quantifies HIV RNA in plasma to monitor disease severity.

6.2 Antiretroviral Therapy (ART)

ART suppresses viral replication, preserves immune function, and reduces transmission risk. Key drug classes include:

- **Nucleoside Reverse Transcriptase Inhibitors (NRTIs):** Zidovudine, Lamivudine
- **Non-Nucleoside Reverse Transcriptase Inhibitors (NNRTIs):** Efavirenz, Nevirapine
- **Protease Inhibitors (PIs):** Lopinavir, Ritonavir
- **Integrase Inhibitors:** Dolutegravir, Raltegravir
- **Entry Inhibitors:** Maraviroc (CCR5 antagonist)

VII. Conclusion

HIV/AIDS remains a global challenge, with significant medical and socio-economic implications. Advances in ART have transformed HIV from a fatal disease into a manageable chronic condition. However, co-infections such as Zika virus pose additional challenges in disease management. Continued research, improved access to ART, and preventive strategies are essential to controlling the HIV epidemic.

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