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A Mathematical Model for HIV Transmission with Screening, Treatment, Viral Suppression and Loss of Suppression

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ABSTRACT

Human Immunodeficiency Virus (HIV) remains a major global public health challenge, particularly in sub-Saharan Africa where late diagnosis, treatment delay, and incomplete viral suppression continue to sustain transmission. In this study, we formulate and analyze a deterministic compartmental model incorporating screening, diagnosis, treatment, viral suppression, and AIDS progression dynamics. The force of infection is modeled using a frequency-dependent transmission mechanism with differential infectivity contributions from diagnosed, treated, and AIDS individuals. Qualitative analysis of the model establishes positivity and boundedness of solutions. The disease free equilibrium is obtained and its local and global stability are rigorously analyzed using the Routh–Hurwitz criteria and comparison theorem. The basic reproduction number R_0 is derived using the next-generation matrix approach, and its sensitivity indices with respect to key epidemiological parameters are computed. Results show that strengthening early screening and sustained viral suppression through consistent treatment significantly reduces R_0 and drives the system toward disease elimination. The findings provide useful insights for optimizing HIV control strategies.

1. Introduction

Human Immunodeficiency Virus (HIV) remains one of the most serious global public health threats despite significant advances in treatment and prevention. Since the beginning of the epidemic, over 85 million people have been infected worldwide, with sub-Saharan Africa bearing a disproportionate burden of disease [1]. The introduction of antiretroviral therapy (ART) has dramatically improved survival among people living with HIV and transformed the disease from a fatal infection into a manageable chronic condition [2]. Beyond its therapeutic benefits, ART plays a crucial role in reducing transmission by lowering viral load. A major scientific breakthrough in HIV prevention is the recognition of the Undetectable = Untransmittable (U=U) paradigm, which states that individuals who maintain sustained viral suppression do not transmit HIV sexually. Large multicenter studies such as HPTN 052 and the PARTNER trials reported zero genetically linked transmissions among serodiscordant couples when the infected partner achieved viral suppression [3, 4]. Mathematical modeling has long been an essential tool for understanding HIV transmission dynamics and evaluating intervention strategies [5]. Early compartmental models provided insights into epidemic thresholds and persistence, while later studies incorporated treatment effects and behavioral changes. However, many existing HIV models fail to explicitly distinguish between treated individuals who remain infectious and those who are fully virally suppressed. Furthermore, treatment failure, AIDS relapse into care, and differential disease induced mortality are often neglected, potentially leading to biased estimates of transmission dynamics. The present study develops a comprehensive deterministic model that integrates screening, diagnosis, treatment initiation, viral suppression, treatment failure, progression to AIDS, and disease-induced mortality. By aligning mathematical structure with modern clinical evidence, the model provides a robust framework for evaluating HIV control strategies.

2. Literature Review

Several HIV-specific models have since been developed: Ndelwa, Luboobi, and Mugisha (2015) developed a mathematical model on the Role of Diagnosis and Treatment in HIV/AIDS Dynamics with the aim of assessing the impact of screening and diagnosis on HIV transmission. Employing a deterministic compartmental framework that distinguishes undiagnosed and diagnosed infected individuals, they conducted equilibrium and stability analysis supported by numerical simulations. Their results showed that increased screening significantly reduces HIV prevalence by shortening the highly infectious undiagnosed period; however, treatment success and viral suppression were not explicitly incorporated.

Huo and Chen (2015), in their study on Stability and Bifurcation Analysis of an HIV/AIDS Epidemic Model with Treatment, aimed to investigate the effects of antiretroviral therapy on HIV transmission dynamics. They formulated a nonlinear HIV model that incorporates treatment and applied stability and bifurcation theory to analyze system behavior. The findings revealed that ART substantially reduces the basic reproduction number and can lead to disease elimination under sufficient treatment coverage, although treated individuals were assumed to remain partially infectious.

Bashiru and Fasoranbaku (2019), worked on Mathematical Modeling of HIV/AIDS Transmission Dynamics with Treatment, where they examined the role of treatment and disease progression to AIDS in HIV dynamics. Using a compartmental model that included an AIDS class, they analyzed equilibrium solutions and stability properties of the system. Their results highlighted the significant influence of AIDS-related mortality on disease dynamics, but the contribution of AIDS individuals to transmission was treated inconsistently.

Cohen et al. (2016), through the clinical trial: Antiretroviral Therapy for the Prevention of HIV-1 Transmission, aimed to determine whether early ART initiation could prevent sexual transmission of HIV. Using a large randomized controlled trial involving serodiscordant couples, the study demonstrated a near-complete elimination of HIV transmission among individuals receiving effective ART. This landmark finding provided strong empirical support for treatment-as-prevention strategies.

Rodger et al. (2016), in their study on Sexual Activity without Condoms and the risk of HIV Transmission in Serodifferent Couples, investigated the risk of HIV transmission among individuals with sustained viral suppression. Using a prospective cohort design that tracked thousands of condomless sexual encounters, the study reported zero genetically linked HIV transmissions when viral load was undetectable, leading to the widely accepted *Undetectable = Untransmittable (U = U)* principle.

Silva and Torres (2018), in their work on Modeling Treatment and Prevention Strategies for HIV/AIDS, aimed to evaluate the combined effects of prevention and treatment interventions on HIV transmission. They formulated a mathematical model that incorporates reduced infectivity due to ART and analyzed it using optimal control techniques. Their findings confirmed that treatment significantly reduces transmission, although viral suppression was modeled implicitly rather than as a distinct epidemiological class.

Nsuami and Witbooi (2018), developed A Mathematical Model of HIV/AIDS with ART and Adherence, which investigated the impact of treatment adherence on HIV dynamics. By developing a compartmental model that included adherence-related parameters and conducting stability analysis, they demonstrated that poor adherence undermines the effectiveness of ART. However, individuals with durable viral suppression were not explicitly represented as a separate class.

More recently, Odebiyi et al. (2024) developed a mathematical model assessing the impact of screening on HIV/AIDS transmission dynamics using a compartmental model. Their analysis showed that the importance of screening is evident in its ability to detect and reduce asymptomatic infectious individuals, which in turn leads to an increase among the symptomatic population, highlighting the importance of early detection of their status and preventing the spread of HIV/AIDS.

Overall, the reviewed literatures demonstrate substantial progress in modeling HIV screening, treatment, and disease progression. However, most existing models do not explicitly distinguish between treated individuals who are virally suppressed and those who are not, nor do they consistently incorporate frequency-dependent transmission. These gaps motivate the development of the present model, which extends the existing screening–treatment models (specifically the model by Odebiyi et al. (2024)) by explicitly incorporating viral suppression in line with contemporary clinical evidence.

3. Methodology

We formulate a deterministic compartmental model to describe the transmission dynamics of HIV using the model of (Odebiyi et al.,2024) as the basis model.

3.1 Model assumptions

The formulation of the model is based on the following assumptions:

- The total population is stratified into mutually exclusive epidemiological classes according to infection status, diagnosis, treatment, and viral suppression.
- Recruitment and natural death occur at constant per capita rates. Also, all parameters are positive constant.
- The population mixes homogeneously and susceptible individuals acquire HIV infection through effective contact with infectious individuals.
- Newly infected individuals enter the undiagnosed infected class before screening. Screening leads to diagnosis, after which individuals may initiate ART.
- Individuals on ART may achieve durable viral suppression and become epidemiologically non-infectious (U = U), hence, we assume that virally suppressed individuals do not contribute to new HIV infections.
- Viral suppression may be lost due to treatment interruption, poor adherence, or drug resistance, returning individuals to the treated but unsuppressed class.
- Disease-induced deaths occur in all infectious classes (with different magnitude) except the suppressed class whose life expectancy is approximately that of the general population.
- AIDS patients can still initiate treatment.

3.2 Model variables and parameters

The variables and parameters used in the model, together with their descriptions are given in Table 1 and 2 respectively.

Table 1: Description of Model Variables

Variable	Description
$S(t)$	Susceptible individuals
$I(t)$	HIV-infected but undiagnosed individuals
$D(t)$	Diagnosed HIV-infected individuals

$T(t)$	Individuals on ART but not virally suppressed
$V(t)$	Virally suppressed individuals (U = U)
$A(t)$	Individuals with advanced HIV/AIDS

Table 2: Description of model parameters

Parameter	Description
π	Recruitment rate into the susceptible population
μ	Natural death rate
β	Effective HIV transmission rate
η_1	Relative infectivity of diagnosed individuals
η_2	Relative infectivity of treated individuals
η_3	Relative infectivity of AIDS individuals
θ	Diagnosis rate of undiagnosed individuals
σ	Progression rate from undiagnosed to AIDS
τ	Treatment initiation rate
α	Progression rate from diagnosed to AIDS
ω	Viral suppression rate
ϵ	Loss of viral suppression rate
ρ	Treatment failure rate leading to AIDS
ϕ	Treatment rate for AIDS individuals
δ_I	Disease-induced death rate for undiagnosed individuals
δ_D	Disease-induced death rate for diagnosed individuals
δ_T	Disease-induced death rate for treated individuals
δ_A	Disease-induced death rate for AIDS individuals

3.3 Model Description and Equations

The total human population at time t is divided into six epidemiological compartments: susceptible individuals $S(t)$, undiagnosed infected individuals $I(t)$, diagnosed infected individuals $D(t)$, individuals receiving treatment $T(t)$, virally suppressed individuals $V(t)$, and individuals with AIDS $A(t)$. Thus,

$$N(t) = S(t) + I(t) + D(t) + T(t) + V(t) + A(t). \quad (1)$$

Susceptible individuals are recruited into the population at rate π and become infected at the force of infection λ. Natural mortality occurs in all compartments at rate μ, while disease induced mortality occurs in infected classes. Transmission occurs through effective contact with infectious individuals in the classes I, D, T, and A, with modification parameters η_1, η_2, and η_3 representing relative infectiousness of diagnosed, treated, and AIDS individuals respectively.

Infected undiagnosed individuals move to the diagnosed class through screening at rate θ, progress to AIDS at rate σ, and experience disease-induced death at rate δ_I. Diagnosed individuals initiate treatment at rate τ and may progress to AIDS at rate α. Individuals treated become virally suppressed at rate ω, but may lose suppression at rate ϵ. Treatment failure and disease progression may also lead to the AIDS class through parameter ρ. AIDS patients may re-enter treatment at rate ϕ.

The relationship between compartments in the model is schematically represented in Figure 1.

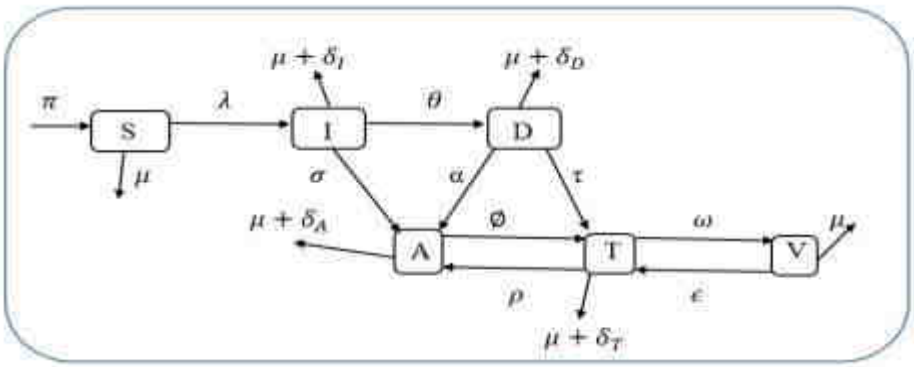

Figure 1: Model Schematic Diagram.

Although individuals with AIDS often experience reduced sexual activity due to illness, their elevated viral loads may still contribute to transmission. Therefore, a reduced but non-zero infectivity parameter is assigned to this class.

The force of infection is therefore defined as

$$\lambda = \beta\left(\frac{I+\eta_1 D+\eta_2 T+\eta_3 A}{N^*}\right). \quad (2)$$

where the modification parameters satisfy $0 < \eta_i < 1$ depending on the relative infectiousness, and $0 < \eta_3 < \eta_2 < \eta_1 < 1$.

Virally suppressed individuals V are excluded from transmission in accordance with the Undetectable = Untransmittable (U=U) principle.

The resulting model is governed by the following system of nonlinear differential equations:

$$\frac{dS}{dt} = \pi - \lambda S - \mu S,$$

$$\frac{dI}{dt} = \lambda S - (\theta + \sigma + \mu + \delta_I)I,$$

$$\frac{dD}{dt} = \theta I - (\tau + \alpha + \mu + \delta_D)D,$$

$$\frac{dT}{dt} = \tau D + \phi A + \epsilon V - (\omega + \rho + \mu + \delta_T)T,$$

$$\frac{dV}{dt} = \omega T - (\epsilon + \mu)V,$$

$$\frac{dA}{dt} = \sigma I + \alpha D + \rho T - (\phi + \mu + \delta_A)A. \quad (3)$$

with initial condition:

$S(0) > 0, I(0) \geq 0, D(0) \geq 0, T(0) \geq 0, V(0) \geq 0, A(0) \geq 0.$

We are going to perform qualitative analysis on the model to determine its ability to perform the intended task.

4. Findings

Here, we present the results of our model analysis, together with their discussions.

4.1 Positivity and Boundedness of Solutions

For an epidemiological model to be mathematically and biologically well-posed, it is necessary to show that all state variables remain non–negative for all time and that the total population is bounded within a feasible region. This guaranties that the model does not produce unrealistic negative population sizes or unbounded growth.

4.1.1 Positivity of Solutions

Theorem 1. *Let the initial conditions*

$S(0), I(0), D(0), T(0), V(0), A(0) \geq 0.$

Then all state variables remain positive for all $t > 0$.

Proof. We show that for non–negative initial conditions, the system solutions remain non–negative for all $t > 0$, where all parameters are assumed positive. For instance, we consider the Susceptible Population:

$$\frac{dS}{dt} = \pi - (\lambda + \mu)S \geq -(\lambda + \mu)S.$$

Using the differential inequality theorem,

$S(t) \geq S(0)e^{-(\lambda+\mu)t} \geq 0.$ (4)

Thus, the susceptible population remains non-negative. Similar argument applies to the remaining equations, showing that all state variables remain non-negative for all time $t > 0$ whenever the initial conditions are non-negative. Hence, the model preserves positivity. □

4.1.2 Invariant Region and Boundedness

Let the total population be:

$N(t) = S + I + D + T + V + A.$

Summing the model equations gives:

$$\frac{dN}{dt} = \pi - \mu N - \delta_I I - \delta_D D - \delta_T T - \delta_A A. \quad (5)$$

Since disease-induced deaths are non-negative,

$$\frac{dN}{dt} \leq \pi - \mu N. \quad (6)$$

Using the comparison theorem, consider $\frac{dN}{dt} = \pi - \mu N$. By using the integrating factor method, this linear differential equation has solution

$$\mathrm{N(t)} = \frac{\pi}{\mu} + \left(\mathrm{N}(0) - \frac{\pi}{\mu}\right)\mathrm{e}^{-\mu \mathrm{t}}. \quad (7)$$

As $t \longrightarrow \infty, N(t) \longrightarrow \frac{\pi}{\mu}$. Thus, $N(t) \leq \frac{\pi}{\mu}$.

The biologically feasible region is given as

$$\Omega = \{(S, I, D, T, V, A) \in \mathbb{R}_+^6 : N(t) \leq \frac{\pi}{\mu}\}. \quad (8)$$

Therefore, all solutions that start in Ω remain in Ω for all $t > 0$. Hence, the model is mathematically well-posed and epidemiologically realistic.

4.2 Disease-Free Equilibrium and Basic Reproduction Number

In this section, we determine the disease-free equilibrium (DFE) of the model and derive the basic reproduction number using the next-generation matrix approach of van den Driessche and Watmough (2002). To reflect heterogeneous transmission risks across infected classes, the force of infection is defined in (2).

4.2.1 Disease-Free Equilibrium (DFE)

The disease-free equilibrium corresponds to a state where no infection persists in the population. Setting all infected compartments equal to zero: $I = D = T = V = A = 0$.

The susceptible equation becomes $\frac{dS}{dt} = \pi - \mu S$.

At equilibrium, $0 = \pi - \mu S \implies S^0 = \frac{\pi}{\mu}$.

Therefore, the Disease-Free Equilibrium is

$$E_0 = \left(\frac{\pi}{\mu}, 0, 0, 0, 0, 0\right). \quad (9)$$

4.2.2 Basic Reproduction Number

The basic reproduction number, denoted by R_0, is defined as the expected number of secondary infections produced by a single infectious individual introduced into a completely susceptible population. The basic reproduction number is computed using the next-generation matrix approach proposed by van den Driessche and Watmough [15].

The Infectious Compartments are: (*I,D,T,V,A*). However, new infections, according to the undetectable = untransmittable principle, are generated only by: *I, D, T, A*. Hence, the New Infection Matrix at disease free - equilibrium, where $N_0 = S_0$ and $\lambda S = \beta(I + \eta_1 D + \eta_2 T + \eta_3 A)$ is:

$$\mathcal{F} = \begin{bmatrix} \beta(I + \eta_1 D + \eta_2 T + \eta_3 A \\ 0 \\ 0 \\ 0 \end{bmatrix} \quad (10)$$

The Jacobian of new infection becomes

$$F = \begin{bmatrix} \beta & \beta\eta_1 & \beta\eta_2 & \beta\eta_3 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix} \quad (11)$$

For the Transition Matrix V, we define removal constants:

$G_1 = \theta+\sigma+\mu+\delta_I$, $G_2 = \tau+\alpha+\mu+\delta_D$, $G_3 = \omega+\rho+\mu+\delta_T$, $G_4 = \epsilon+\mu$, *and* $G_5 = \phi+\mu+\delta_A$. (12)

The Jacobian of the transition matrix $\mathcal{V}$ is

$$V = \begin{bmatrix} G_1 & 0 & 0 & 0 \\ -\theta & G_2 & 0 & 0 \\ 0 & -\tau & G_3 & -\phi \\ -\sigma & -\alpha & -\rho & G_5 \end{bmatrix}. \quad (13)$$

Rather than explicitly computing V^{-1}, we evaluate the expected number of secondary infections produced along all infection pathways.

Contribution from Undiagnosed Individuals:

Average infectious period: $\frac{1}{G_1}$, and Contribution: $\frac{\beta}{G_1}$.

Contribution from Diagnosed Individuals:

Probability of progression: $\frac{\theta}{G_1}$, Duration: $\frac{1}{G_2}$, and Contribution: $\frac{\beta\ \eta_1\theta}{G_1G_2}$.

Contribution from Treated Individuals:

Progression probability: $\frac{\theta}{G_1} \cdot \frac{\tau}{G_2}$, Duration: $\frac{1}{G_4}$, and Contribution: $\frac{\beta\ \eta_2\theta\tau}{G_1G_2G_4}$.

Contribution from AIDS Individuals:

There are three pathways into AIDS: $\frac{\sigma}{G_1}$, $\frac{\theta\alpha}{G_1G_2}$, $\frac{\theta\tau\rho}{G_1G_2G_3}$.

Duration in AIDS: $\frac{1}{G_5}$, and Contribution: $\beta\eta_3\left(\frac{\sigma}{G_1G_5} + \frac{\theta\alpha}{G_1G_2G_5} + \frac{\theta\tau\rho}{G_1G_2G_3G_5}\right)$.

The Basic Reproduction Number is therefore given as:

$$R_0 = \beta\left[\frac{1}{G_1} + \frac{\eta_1\theta}{G_1G_2} + \frac{\eta_2\theta\tau}{G_1G_2G_3} + \eta_3\left(\frac{\sigma}{G_1G_5} + \frac{\theta\alpha}{G_1G_2G_5} + \frac{\theta\tau\rho}{G_1G_2G_3G_5}\right)\right] \quad (14)$$

Biologically, this formulation reveals that early screening reduces transmission by shortening the infectious period, effective treatment lowers progression into AIDS, preventing treatment failure significantly suppresses R_0. Although AIDS patients may have reduced contact rates, their contribution remains epidemiologically important.

4.3 Stability Analysis of the Model

Here, we investigate the local stability of the disease–free equilibrium (DFE) and the endemic equilibrium (EE) of the model using the Routh–Hurwitz (RH) stability criterion.

4.3.1 Local Stability of the Disease free equilibrium

Theorem 2. *The disease–free equilibrium E_0 of system (3) is locally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$.*

Proof.

Recall that the disease–free equilibrium of system (3) is given by

$E_0 = (S_0, I_0, D_0, T_0, V_0, A_0) = (\frac{\pi}{\mu}, 0,0,0,0,0)$.

Let $X = (I,D,T,V,A)^T$ denote the vector of infected compartments. Linearizing system (3) about E_0 yields

$\frac{dX}{dt} = J(E_0)X$,

where $J(E_0)$ is the Jacobian matrix evaluated at the disease–free equilibrium.

Recall the composite parameters from (12), the Jacobian matrix of the infected subsystem at E_0 is

$$J(E_0) = \begin{bmatrix} -G_1 & 0 & 0 & 0 & 0 \\ \theta & -G_2 & 0 & 0 & 0 \\ 0 & \tau & -G_3 & \epsilon & 0 \\ 0 & 0 & \omega & -G_4 & 0 \\ \sigma & \alpha & \rho & 0 & -G_5 \end{bmatrix} \quad (15)$$

The characteristic equation of $J(E_0)$ is

$$\det(\lambda I - J(E_0)) = 0, \quad (16)$$

which yields the fifth degree polynomial,

$$\lambda^5 + a_1\lambda^4 + a_2\lambda^3 + a_3\lambda^2 + a_4\lambda + a_5 = 0, \quad (17)$$

According to the Routh - Hurwitz criterion, all eigenvalues of the characteristic polynomial have negative real parts if and only if the following conditions hold: $a_i > 0$, for $i = 1,...,5$, $a_1a_2 > a_3$, $a_1a_2a_3 > a_3^2 + a_1^2a_4$, and $a_1a_2a_3a_4 > a_4^2 + a_1^2a_5$.

Since all parameters are positive, we have

$a_1 > 0$, $a_2 > 0$, $a_3 > 0$, $a_4 > 0$. (18)

Moreover,

$a_5 > 0 \iff R_0 < 1$. (19)

Thus, all Routh–Hurwitz conditions are satisfied whenever $R_0 < 1$. Hence, by Theorem 2, the model is locally asymptomatically stable at the DFE. □

4.3.2 Existence of Endemic Equilibrium

When $R_0 > 1$, the disease–free equilibrium becomes unstable and a unique endemic equilibrium exists. By continuity arguments and the Routh–Hurwitz conditions applied at the endemic equilibrium, the endemic equilibrium is locally asymptotically stable whenever it exists.

Assume $R_0 > 1$. Let the endemic equilibrium be

$E^* = (S^*,I^*,D^*,T^*,V^*,A^*)$. (20)

We solve for the endemic equilibrium points E^* as follows:

From $0 = \pi - (\lambda^* + \mu)S^*$, we obtain

$S^* = \frac{\pi}{\lambda^* + \mu}$ (21)

Recall that: $G_1 = \theta + \sigma + \mu + \delta_I$., then we have:

$I^* = \frac{\lambda^* S^*}{G_1}$ (22)

Let

$r_1 = \frac{\theta}{G_2}$, $r_2 = \frac{\tau}{G_3}$, $r_3 = \frac{\omega}{G_4}$ (23)

Then,

$D^* = r_1 I^*$, $T^* = r_2 r_1 I^*$, $V^* = r_3 r_2 r_1 I^*$, $A^* = \frac{\sigma I^* + \alpha D^* + \rho T^*}{G_5}$. (24)

By appropriate substitution, we obtain $A^* = kI^*$, where

$k = \frac{\sigma + \alpha r_1 + \rho r_1 r_2}{G_5}$ (25)

We then determine λ^*. Using the force of infection,

$\lambda^* = \beta\left(\frac{I^* + \eta_1 D^* + \eta_2 T^* + \eta_3 A^*}{N^*}\right)$. (26)

After substitution and simplification, we obtain

$\lambda^* = \mu(R_0 - 1)$. (27)

We can see that $R_0 > 1 \Rightarrow$ unique endemic equilibrium exists.

4.3.3 Local Stability of the Endemic Equilibrium

The Jacobian evaluated at E^* yields a characteristic polynomial of degree five:

$\lambda^5 + a_1\lambda^4 + a_2\lambda^3 + a_3\lambda^2 + a_4\lambda + a_5 = 0$. (28)

Because all transition parameters are positive when $R_0 > 1$, the Routh–Hurwitz criteria are satisfied. Thus, E^* is locally asymptotically stable whenever it exists.

4.3.4 Global Stability of the Disease–Free Equilibrium

We establish the global asymptotic stability of the disease–free equilibrium (DFE) using the approach of Castillo–Chavez and Song (2002), together with the LaSalle Invariance Principle.

Theorem 3. *The disease-free equilibrium E_0 of system (3) is globally asymptotically stable whenever $R_0 < 1$ and unstable whenever $R_0 > 1$.*

Proof.

Let

$X = S, \quad Y = (I,D,T,V,A)^T.$

The system can be rewritten as

$$\frac{dX}{dt} = F(X,Y), \quad \frac{dY}{dt} = G(X,Y). \tag{29}$$

Recall that the disease-free equilibrium is $E_0 = \left(\frac{\pi}{\mu}, 0, 0, 0, 0, 0\right)$.

According to Castillo–Chavez and Song (2002), the disease-free equilibrium is globally asymptotically stable if the following conditions hold:

(H1) The subsystem

$$\frac{dS}{dt} = \pi - \mu S \tag{30}$$

has a globally asymptotically stable equilibrium.

(H2) The infected subsystem satisfies

$$\mathrm{G}(\mathrm{S},\mathrm{Y}) = \mathrm{BY} - \widehat{\mathrm{G}}(\mathrm{S},\mathrm{Y}). \tag{31}$$

Where $B = D_Y G(E_0)$ is an M-matrix and $\hat{G}(S,Y) \geq 0$.

We now check to verify the conditions as follows:

First Condition (H1): Consider the susceptible equation in the absence of infection: $\frac{dS}{dt} = \pi - \mu S$.

This linear differential equation has solution

$S(t) = S_0 e^{-\mu t} + \frac{\pi}{\mu}(1 - e^{-\mu t})$. Hence, $lim_{t\to\infty} S(t) = \frac{\pi}{\mu}$.

Therefore, the subsystem is globally asymptotically stable, implying that condition (H1) holds.

Second Condition (H2): Linearizing the infected subsystem at E_0 yields

$$\mathrm{B} = \begin{bmatrix} -\mathrm{G}_1 & 0 & 0 & 0 & 0 \\ \theta & -\mathrm{G}_2 & 0 & 0 & 0 \\ 0 & \tau & -\mathrm{G}_3 & \epsilon & 0 \\ 0 & 0 & \omega & -\mathrm{G}_4 & 0 \\ \sigma & \alpha & \rho & 0 & -\mathrm{G}_5 \end{bmatrix} \tag{32}$$

Observe that: all diagonal entries of B are negative, and all off-diagonal entries are non-negative.Thus, matrix B is a Metzler matrix and hence an M-matrix. Furthermore, nonlinear infection terms satisfy

$$\hat{G}(S,Y) = \lambda S \geq 0. \tag{33}$$

Therefore, condition (H2) holds.

Since both conditions (H1) and (H2) are satisfied, it follows that the disease-free equilibrium is globally asymptotically stable whenever $R_0 < 1$. □

This implies that when the basic reproduction number is less than unity, the infection cannot invade the population regardles s of the initial number of infected individuals. Consequently, strengthening screening, treatment initiation, and viral suppression reduces R_0 and guarantees eradication of HIV from the population.

4.3.5 Sensitivity Analysis of the Basic Reproduction Number

To determine the relative importance of epidemiological parameters in disease trans mission, the normalized forward sensitivity index of R_0 with respect to a parameter p is defined as

$$\Upsilon_p^{R_0} = \frac{\partial R_0}{\partial p} \cdot \frac{p}{R_0}. \tag{34}$$

We are going to check the sensitivity of the basic reproduction number with respect to some key parameters.

Sensitivity with respect to transmission rate β:

Given that, $R_0 = \beta\Psi$, where Ψ represents the remaining parameter expression, we obtain

$\frac{\partial R_0}{\partial \beta} = \Psi = \frac{R_0}{\beta}$. Hence, $\Upsilon_\beta^{R_0} = \frac{R_0}{\beta} \cdot \frac{\beta}{R_0} = 1$.

Thus, a 10% increase in transmission rate leads to a 10% increase in R_0.

Sensitivity with respect to screening rate θ:

Differentiating R_0 with respect to θ gives

$$\frac{\partial R_0}{\partial \theta} = -\beta\left[\frac{1}{\mathrm{G}_1^2} + \frac{\eta_1}{\mathrm{G}_1^2 G_2} + \frac{\eta_2 \tau}{\mathrm{G}_1^2 G_2 G_3} + \eta_3\left(\frac{\sigma}{\mathrm{G}_1^2 G_5} + \frac{\alpha}{\mathrm{G}_1^2 G_2 G_5} + \frac{\tau\rho}{G_1 G_2 G_3 G_5}\right)\right]$$

Therefore $\Upsilon_\theta^{R_0} = \frac{\partial R_0}{\partial \theta} \cdot \frac{\theta}{R_0} < 0$.

Hence increasing screening decreases R_0.

Similarly, Sensitivity with respect to treatment initiation rate τ is obtained thus:

$\Upsilon_{\tau}^{R_0} = \frac{\partial R_0}{\partial \tau} . \frac{\tau}{R_0} < 0,$

Indicating that expanding treatment reduces transmission potential. By similar computation, we obtain the table of sensitivity indices as follows;

Table 3: Normalized sensitivity indices of R_0

Parameter	Description	Sensitivity Sign
β	Transmission rate	+1
θ	Screening rate	Negative
T	Treatment initiation	Negative
Σ	Progression to AIDS	Negative
Ω	Viral suppression rate	Negative
η_3	AIDS infectivity	Positive

4.3.6 Discussion of Sensitivity Analysis

The sensitivity analysis of the basic reproduction number R_0 provides quantitative insight into the relative importance of epidemiological parameters in HIV transmission dynamics. The transmission rate β has a positive sensitivity index equal to unity, indicating that R_0 changes proportionally with the effective contact rate. Thus, behavioral interventions such as condom use, awareness programs, and reduction of risky sexual practices can substantially lower transmission potential. Screening rate θ exhibits a negative sensitivity index, implying that increased HIV testing and early diagnosis reduce the number of undiagonized infectious individuals and consequently decrease R_0. This highlights the importance of expanding voluntary counseling and testing programs. The treatment initiation rate τ also shows a negative sensitivity index, demonstrating that rapid enrollment of diagnosed individuals into treatment programs reduces disease spread. Similarly, the viral suppression rate ω negatively influences R_0, emphasizing the epidemiological significance of treatment adherence and sustained viral suppression. The infectivity modification parameter of AIDS individuals η_3 has a positive sensitivity index, suggesting that increased infectiousness among late-stage patients substantially increases the transmission potential. This result underscores the need for early treatment initiation before progression to advanced disease stages. Therefore, the sensitivity results indicate that prevention strategies focusing on reducing transmission probability, expanding early screening, improving treatment uptake, and maintaining viral suppression are the most effective measures for lowering the reproduction number and controlling the epidemic.

5. Conclusion and Recommendations

In this study, a deterministic compartmental model for HIV transmission dynamics incorporating screening, diagnosis, treatment, viral suppression, and AIDS progression was formulated and rigorously analyzed. The model accounts for differential infectivity among undiagnosed, diagnosed, treated, and AIDS individuals, thereby providing a realistic representation of HIV epidemiological processes. The qualitative analysis established the fundamental mathematical properties of the model. It was shown that all solutions of the system remain positive for all time and that the feasible epidemiological region is positively invariant, ensuring biological well-posedness of the model. The disease-free equilibrium was derived and analyzed, and the basic reproduction number R_0 was obtained using the next-generation matrix approach. Local and global stability analysis demonstrated that the disease-free equilibrium is locally and globally asymptotically stable whenever $R_0 < 1$, indicating that the infection can be eliminated from the population if transmission potential is sufficiently reduced. Conversely, when $R_0 > 1$, the system admits an endemic equilibrium corresponding to persistent HIV transmission. Sensitivity analysis of the reproduction number revealed that the effective contact rate and infectivity of advanced-stage individuals contribute positively to disease transmission, while screening, treatment initiation, and viral suppression parameters contribute negatively to R_0. These findings highlight the critical importance of early diagnosis, rapid treatment enrollment, sustained adherence to therapy, and prevention of disease progression in reducing the long-term transmission potential of HIV. Altogether, the theoretical results emphasize that integrated intervention strategies combining expanded screening programs, timely treatment initiation, improved treatment adherence, and strengthened prevention measures are essential for achieving long-term control and eventual elimination of HIV transmission, and hence recommended. The analytical framework developed in this study provides a useful foundation for future investigations involving optimal control, parameter estimation, and policy evaluation in HIV epidemic management.

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