American Journal of Mathematical and Computer Modelling
2025; 10(2): 74-83
http://www.sciencepublishinggroup.com/j/ajmcm
doi: 10.11648/j.ajmcm.20251002.14
ISSN: 2578-8272 (Print); ISSN: 2578-8280 (Online)

Mathematical Analysis of an HIV/AIDS Epidemic Model with Defaulter Tracing Dynamics

Sammy Maingi*, Benjamin Kikwai, Mark Kimathi

Deparment of Mathematics and Statistics, Machakos University, Machakos, Kenya

Email address:

sammymaingi1@gmail.com (Sammy Maingi)
*Corresponding author

To cite this article:

Sammy Maingi, Benjamin Kikwai, Mark Kimathi. (2025). Mathematical Analysis of an HIV/AIDS Epidemic Model with Defaulter Tracing Dynamics. *American Journal of Mathematical and Computer Modelling, 10*(2), 74-83. 10.11648/j.ajmcm.20251002.14

Received: 22 May 2025; **Accepted:** 13 June 2025; **Published:** 30 June 2025

Abstract: The persistence of the HIV/AIDS epidemic is significantly challenged by difficulties in maintaining long-term adherence to antiretroviral therapy (ART), leading to treatment defaults hence hindering disease control. Defaulter tracing is a crucial intervention aimed at returning patients back to care. This paper develops and analyzes a deterministic mathematical model for HIV/AIDS transmission dynamics, explicitly incorporating defaulter tracing. The model utilizes a system of ordinary differential equations to describe the transitions between susceptible (S_p), infected (I_T), infected on ART (I_{ARV}), infected not on ART (I_{NARV}), and individuals under defaulter tracing (D_{TR}) compartments. Mathematical analysis includes establishing the positivity and boundedness of solutions, determination of the Disease-Free Equilibrium (DFE) and the existence of an Endemic Equilibrium (EE). The basic reproduction number (R_0) is also derived using the next-generation matrix method. Local stability analysis of the DFE shows it is asymptotically stable if $R_0 < 1$ and unstable otherwise. Sensitivity analysis identified parameters related to transmission (λ, c, π) as having a positive impact on R_0, while parameters associated with treatment, defaulter tracing, and mortality were found to negatively influence the R_0. This study shows that improving treatment uptake and retention to care, through defaulter tracing efforts, can contribute to reduction of R_0 and in turn controll the epidemic.

Keywords: HIV/AIDS, Mathematical Model, Defaulter Tracing, Basic Reproduction Number, Stability Analysis

1. Introduction

The human immuno-deficiency virus (HIV) infection, leading to acquired immuno-deficiency syndrome (AIDS), represents a significant global health challenge, causing millions of deaths and necessitating substantial resources for healthcare and control [17]. The HIV/AIDS epidemic persists as one of the most pressing health issues globally, carrying profound social, economic, and public health consequences [14]. Since its identification, HIV/AIDS has impacted countless communities worldwide, with regions like Sub-Saharan Africa bearing a disproportionate burden, although Asia, Eastern Europe, and Latin America also face significant epidemics.

Mathematical models have become instrumental in understanding the transmission dynamics of infectious diseases like HIV/AIDS [6]. These models are widely used in epidemiological research to gain insights into the major factors driving epidemics and to evaluate the potential impact of interventions [4, 5]. Studying the dynamics of HIV transmission is crucial for developing effective prevention and control strategies [8].

Antiretroviral therapy (ART) is a cornerstone of HIV management, suppressing viral replication and preventing disease progression [15]. Achieving viral suppression through ART not only improves individual health but also significantly reduces the risk of transmission. However, a major challenge in HIV care is default of treatment, where individuals discontinue ART [3]. Reasons for defaulting are multifaceted, including medication side effects, complex treatment regimens, stigma and discrimination, mental health issues, denial, and barriers to healthcare access. Discontinuation of ART leads to viral load increase, immune system decline, increased susceptibility to opportunistic

infections, development of drug resistance, and a higher risk of transmitting the virus to others [9, 16].

Defaulter tracing, the systematic process of locating and re-engaging individuals who have missed appointments or stopped treatment, has emerged as a crucial intervention to improve retention in care and treatment outcomes [2, 11]. It aims to minimize loss to follow-up, address barriers to adherence, reduce onward transmission, and optimize resource allocation.

Previous mathematical modeling studies have explored various aspects of HIV dynamics, including the impact of prevention and treatment strategies [6], co-infection dynamics [1], the role of stigma [8], and optimal control strategies [5, 7, 10]. Classic compartmental models, such as Susceptible-Infectious-Removed (SIR) frameworks, have been adapted to simulate HIV dynamics, incorporating parameters like transmission rates, treatment uptake, and viral load [13]. While studies have demonstrated the empirical effectiveness of defaulter tracing [2], relatively few mathematical models have explicitly incorporated defaulter tracing as a dynamic component or control mechanism within the system equations.

Thus the aim of this study is to develop and analyze a mathematical model for HIV/AIDS transmission that explicitly incorporates the dynamics of defaulter tracing for individuals who have dropped off care and treatment. The analysis will focus on understanding the fundamental mathematical properties of the model, including the existence and stability of equilibria and the derivation of the basic reproduction number, to provide theoretical insights into the impact of defaulter tracing on HIV/AIDS dynamics.

2. Model Formulation

We develop a compartmental mathematical model based on the Susceptible-Infectious (SI) framework to describe the transmission dynamics of HIV/AIDS, while introducing compartments for treatment status and defaulter tracing. The model divides the total human population, $N(t)$, into five mutually exclusive compartments based on their epidemiological status of HIV/AIDS and engagement in treatment and care.

2.1. Model Assumptions

The model is based on the following assumptions:

1. The population size N is constant, meaning births and deaths (except disease-related deaths) balance each other.
2. Individuals mix randomly with each other, leading to uniform exposure to HIV risks.
3. The infection spreads through contact between susceptible and infected individuals.
4. The force of infection is proportional to the number of infected individuals.
5. The transition rates between compartments are constant.
6. Some infected individuals receive ART while others do not.

2.2. Model Compartments and Flow Diagram

The model consists of the following compartments:

1. *Susceptible Population (S_p):* Individuals who are susceptible to HIV infection and have not yet been infected. They are at risk of acquiring HIV through contact with infectious individuals.
2. *Infected Population (I_T):* Individuals who have contracted HIV and are infectious to others. This compartment includes newly infected individuals transitioning from the susceptible class.
3. *Infected under Antiretroviral Therapy (I_{ARV}):* HIV-positive individuals who are currently receiving ART. ART suppresses viral replication, reducing infectiousness.
4. *Infected and Not under Antiretroviral Therapy (I_{NARV}):* Individuals diagnosed with HIV but not currently receiving ART, either due to barriers to care or personal reasons. They remain infectious.
5. *Infected and under Defaulter Tracing (D_{TR}):* Individuals who were previously on ART but have defaulted (missed appointments or stopped treatment) and are now targeted by defaulter tracing interventions aiming to return them back to care.

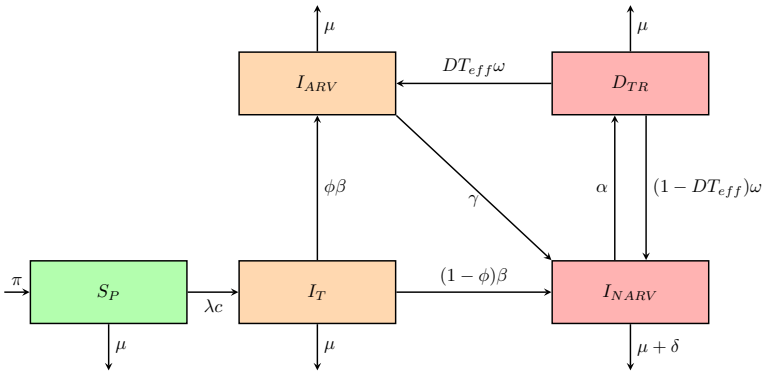

Figure 1. *Flow diagram of the HIV/AIDS model with defaulter tracing. Arrows indicate the transition of individuals between compartments S_p (Susceptible), I_T (Infected Total/Initial), I_{ARV} (Infected on ART), I_{NARV} (Infected not on ART), and D_{TR} (Defaulters under Tracing). Parameters governing the transitions are shown alongside the arrows.*

The flow of individuals between these compartments is depicted in Figure 1. New individuals enter the susceptible population at a rate πN. Susceptible individuals become infected at a rate $\lambda = \frac{cC}{N}(I_T + I_{NARV} + \eta I_{ARV})$ (where λ represents the force of infection, c is the contact rate, C is the total number of contacts, and η represent relative infectiousness of I_{ARV}). Infected individuals (I_T) may either start ART and move to I_{ARV} at a rate $\phi\beta$, or fail to enroll/default early and move to I_{NARV} at a rate $(1-\phi)\beta$. Individuals on ART (I_{ARV}) may abandon treatment and move to I_{NARV} at rate γ. Individuals not on ART (I_{NARV}) can be successfully traced at a rate α, and transition to the defaulter tracing compartment D_{TR} Individuals under tracing (D_{TR}) can successfully return to ART (I_{ARV}) at a rate proportional to tracing effectiveness DT_{eff}, or fail to get into care at a rate $(1 - DT_{eff})$. Natural death occurs in all compartments at rate μ, and there is an additional HIV-related death rate δ for individuals not virally suppressed (I_{NARV}).

2.3. Model Parameters

The parameters used in the model equations (1) are defined in Table 1.

Table 1. *Model Parameters and Descriptions.*

Parameter	Description
π	Recruitment rate
μ	Natural per capita death rate
λ	Rate of infection parameter
c	Effective contact rate parameter
β	Screening/treatment initiation rate from Infectious population (I_T)
ϕ	Proportion of I_T successfully enrolling in ART
α	Rate of progression from I_{NARV} into D_{TR} due to defaulter tracing
γ	Rate at which infected population under ART (I_{ARV}) drop off care
DT_{eff}	Defaulter tracing effectiveness
δ	Death rate due to HIV-related complications
ω	Intensity of awareness campaigns and media advertisements for ART uptake
N	Total population size

2.4. Model Equations

Based on the compartments, flow diagram, and assumptions, the dynamics of the HIV/AIDS epidemic with defaulter tracing are described by the following system of ordinary differential equations (ODEs):

$$\begin{aligned}
\frac{dS_p}{dt} &= \pi N - \frac{\lambda c S_p I_T}{N} - \mu S_p \\
\frac{dI_T}{dt} &= \frac{\lambda c S_p I_T}{N} - (\beta + \mu) I_T \\
\frac{dI_{ARV}}{dt} &= \phi\beta I_T - (\gamma + \mu) I_{ARV} + DT_{eff}\omega D_{TR} \\
\frac{dI_{NARV}}{dt} &= (1-\phi)\beta I_T + \gamma I_{ARV} - (\alpha + \mu + \delta) I_{NARV} + (1 - DT_{eff})\omega D_{TR} \\
\frac{dD_{TR}}{dt} &= \alpha I_{NARV} - (\omega + \mu) D_{TR}
\end{aligned} \tag{1}$$

The total population is $N(t) = S_p(t) + I_T(t) + I_{ARV}(t) + I_{NARV}(t) + D_{TR}(t)$. Initial conditions are $S_p(0) \geq 0, I_T(0) \geq 0, I_{ARV}(0) \geq 0, I_{NARV}(0) \geq 0, D_{TR}(0) \geq 0$.

3. Mathematical Analysis

In this section, we analyze the fundamental properties of the system of ODEs (1) describing the HIV/AIDS dynamics with defaulter tracing.

3.1. Positivity and Boundedness of Solutions

Since the model describes human populations, solutions must remain non-negative and bounded for all time $t \geq 0$.

Theorem 3.1: If the initial conditions of the model (1) are non-negative, i.e., $S_p(0) \geq 0, I_T(0) \geq 0, I_{ARV}(0) \geq 0, I_{NARV}(0) \geq 0, D_{TR}(0) \geq 0$, then the solution set $(S_p(t), I_T(t), I_{ARV}(t), I_{NARV}(t), D_{TR}(t))$ remains non-negative for all time $t \geq 0$.

Proof: To demonstrate the non-negativity of solutions, consider the first equation of the system (1):

$$\frac{dS_p}{dt} = \pi N - \frac{\lambda c S_p I_T}{N} - \mu S_p \tag{2}$$

We can re-write this to obtain:

$$\frac{dS_p}{dt} = \pi N - \left(\frac{\lambda c I_T}{N} + \mu\right) S_p \geq -\left(\frac{\lambda c I_T}{N} + \mu\right) S_p \tag{3}$$

Then let $K(t) = \frac{\lambda c I_T(t)}{N} + \mu$ such that $\frac{dS_p}{dt} \geq -K(t)S_p$. By separation of variables and integration from 0 to t:

$$\begin{aligned}
\int_{S_p(0)}^{S_p(t)} \frac{dS_p}{S_p} &\geq \int_0^t -K(\tau)d\tau \\
\ln\left(\frac{S_p(t)}{S_p(0)}\right) &\geq -\int_0^t K(\tau)d\tau \\
S_p(t) &\geq S_p(0)\exp\left(-\int_0^t K(\tau)d\tau\right)
\end{aligned}$$

Since $S_p(0) \geq 0$ and the exponential term is always positive, $S_p(t) \geq 0$ for all $t \geq 0$.

A similar analysis can be applied to the other compartments. For instance, from equation (1):

$$\frac{dI_T}{dt} = \left(\frac{\lambda c S_p}{N} - (\beta + \mu)\right) I_T \geq -(\beta + \mu) I_T$$

Integrating from 0 to t gives:

$$I_T(t) \geq I_T(0) \exp\left(-\int_0^t (\beta + \mu) d\tau\right)$$

Since $I_T(0) \geq 0$, $I_T(t) \geq 0$ for all $t \geq 0$.

$$I_{ARV}(t) \geq I_{ARV}(0) e^{-(\gamma+\mu)t} \geq 0 \tag{4}$$

$$I_{NARV}(t) \geq I_{NARV}(0) e^{-(\alpha+\mu+\delta)t} \geq 0 \tag{5}$$

$$D_{TR}(t) \geq D_{TR}(0) e^{-(1+\mu)t} \geq 0 \tag{6}$$

Therefore, all solutions remain non-negative for $t \geq 0$.

Theorem 1.1 (Invariant Region): The feasible region Ω defined by

$$\Omega = \left\{(S_p, I_T, I_{ARV}, I_{NARV}, D_{TR}) \in \mathbb{R}^5_+ : N(t) = S_p + I_T + I_{ARV} + I_{NARV} + D_{TR} \leq \frac{\pi N}{\mu}\right\} \tag{7}$$

is positively invariant for the system (1).

Proof: The total population is $N(t) = S_p(t) + I_T(t) + I_{ARV}(t) + I_{NARV}(t) + D_{TR}(t)$. Summing the equations in system (1), we obtain the rate of change of the total population:

$$\begin{aligned}
\frac{dN}{dt} &= \frac{dS_p}{dt} + \frac{dI_T}{dt} + \frac{dI_{ARV}}{dt} + \frac{dI_{NARV}}{dt} + \frac{dD_{TR}}{dt} \\
&= \left(\pi N - \frac{\lambda c S_p I_T}{N} - \mu S_p\right) + \left(\frac{\lambda c S_p I_T}{N} - (\beta + \mu) I_T\right) \\
&\quad + (\phi\beta I_T - (\gamma + \mu) I_{ARV} + DT_{eff}\omega D_{TR}) \\
&\quad + ((1 - \phi)\beta I_T + \gamma I_{ARV} - (\alpha + \mu + \delta) I_{NARV} + (1 - DT_{eff})\omega D_{TR}) \\
&\quad + (\alpha I_{NARV} - (\omega + \mu) D_{TR})
\end{aligned} \tag{8}$$

Simplifying the terms:

$$\begin{aligned}
\frac{dN}{dt} &= \pi N - \mu S_p - \beta I_T - \mu I_T + \phi\beta I_T - \gamma I_{ARV} - \mu I_{ARV} + DT_{eff}\omega D_{TR} \\
&\quad + (1 - \phi)\beta I_T + \gamma I_{ARV} - \alpha I_{NARV} - \mu I_{NARV} - \delta I_{NARV} + (1 - DT_{eff})\omega D_{TR} \\
&\quad + \alpha I_{NARV} - \omega D_{TR} - \mu D_{TR} \\
&= \pi N - \mu(S_p + I_T + I_{ARV} + I_{NARV} + D_{TR}) - \delta I_{NARV} \\
&= \pi N - \mu N(t) - \delta I_{NARV}
\end{aligned} \tag{9}$$

Since $\delta \geq 0$ and $I_{NARV}(t) \geq 0$, we have:

$$\frac{dN}{dt} \leq \pi N - \mu N(t) \tag{10}$$

This is a linear differential inequality. If $N(t) > \frac{\pi N}{\mu}$, then $\frac{dN}{dt} < 0$. If $N(t) = \frac{\pi N}{\mu}$, then $\frac{dN}{dt} \leq 0$. Therefore, $N(t)$ cannot grow beyond $\frac{\pi N}{\mu}$ if it starts at or below this value. Specifically, solving the inequality $\frac{dN}{dt} + \mu N \leq \pi N$ using an integrating factor $e^{\mu t}$:

$$\frac{d}{dt}(N(t)e^{\mu t}) \leq \pi N e^{\mu t}$$

Integrating from 0 to t:

$$N(t)e^{\mu t} - N(0) \leq \int_0^t \pi N e^{\mu\tau} d\tau = \frac{\pi N}{\mu}(e^{\mu t} - 1)$$

$$N(t) \leq N(0)e^{-\mu t} + \frac{\pi N}{\mu}(1 - e^{-\mu t})$$

As $t \to \infty$, $e^{-\mu t} \to 0$, thus $\limsup_{t\to\infty} N(t) \leq \frac{\pi N}{\mu}$. Thus, if the initial total population $N(0)$ is within the bound $\frac{\pi N}{\mu}$, the population $N(t)$ remains bounded by $\frac{\pi N}{\mu}$ for all $t \geq 0$. Therefore, the region Ω is positively invariant.

3.2. Equilibrium Points

Equilibrium points of the system (1) are obtained by setting the derivatives of all state variables to zero. We identify two possible equilibria: the Disease-Free Equilibrium (DFE) and the Endemic Equilibrium (EE).

3.2.1. Disease-Free Equilibrium (DFE)

The Disease-Free Equilibrium (DFE) occurs when there are no infected individuals in the population. This state is found by setting all infection-related compartments to zero:

$I_T = 0, I_{ARV} = 0, I_{NARV} = 0, D_{TR} = 0$ [6]. Substituting these into the first equation of system (1):

$$\frac{dS_p}{dt} = \pi N - \frac{\lambda c S_p \cdot 0}{N} - \mu S_p = \pi N - \mu S_p$$

Setting $\frac{dS_p}{dt} = 0$ yields:

$$\pi N - \mu S_{p0} = 0 \implies S_{p0} = \frac{\pi N}{\mu}$$

The remaining equations are identically zero when the infected compartments are zero. Therefore, the Disease-Free Equilibrium point, denoted by E_0, is given by:

$$E_0 = (S_{p0}, I_{T0}, I_{ARV0}, I_{NARV0}, D_{TR0}) = \left(\frac{\pi N}{\mu}, 0, 0, 0, 0\right) \tag{11}$$

3.2.2. Endemic Equilibrium (EE)

The Endemic Equilibrium (EE) represents a state where the disease persists in the population ($I_T^* > 0, I_{ARV}^* > 0, I_{NARV}^* > 0, D_{TR}^* > 0$, and $S_p^* > 0$). This equilibrium is found by setting all derivatives in system (1) to zero:

$$0 = \pi N - \frac{\lambda c S_p^* I_T^*}{N} - \mu S_p^* \tag{12}$$

$$0 = \frac{\lambda c S_p^* I_T^*}{N} - (\beta + \mu) I_T^* \tag{13}$$

$$0 = \phi\beta I_T^* - (\gamma + \mu) I_{ARV}^* + DT_{eff}\omega D_{TR}^* \tag{14}$$

$$0 = (1 - \phi)\beta I_T^* + \gamma I_{ARV}^* - (\alpha + \mu + \delta) I_{NARV}^* + (1 - DT_{eff})\omega D_{TR}^* \tag{15}$$

$$0 = \alpha I_{NARV}^* - (\omega + \mu) D_{TR}^* \tag{16}$$

From equation (13), assuming $I_T^* \neq 0$:

$$\frac{\lambda c S_p^*}{N} = \beta + \mu$$

Solving for S_p^*:

$$S_p^* = \frac{(\beta + \mu)N}{\lambda c} \tag{17}$$

From equation (12), we can express I_T^* in terms of S_p^*:

$$\frac{\lambda c S_p^* I_T^*}{N} = \pi N - \mu S_p^* \tag{18}$$

Substituting $\frac{\lambda c S_p^*}{N} = \beta + \mu$ in the (18) above:

$$(\beta + \mu) I_T^* = \pi N - \mu S_p^*$$

$$I_T^* = \frac{\pi N - \mu S_p^*}{\beta + \mu} \tag{19}$$

Substituting the expression for S_p^* from (17) into (19):

$$I_T^* = \frac{1}{\beta + \mu}\left(\pi N - \mu \frac{(\beta + \mu)N}{\lambda c}\right) = \frac{N}{\beta + \mu}\left(\pi - \frac{\mu(\beta + \mu)}{\lambda c}\right) \tag{20}$$

For the endemic equilibrium to exist with a positive number of infected individuals ($I_T^* > 0$), we require:

$$\pi - \frac{\mu(\beta + \mu)}{\lambda c} > 0 \implies \frac{\lambda c \pi}{\mu(\beta + \mu)} > 1$$

This condition is related to the basic reproduction number $R_0 > 1$.

Solving the equations (14), and (16), we can express I_{ARV}^* and I_{NARV}^*:

$$I_{ARV}^* = \frac{\phi\beta I_T^* + DT_{eff}\omega D_{TR}^*}{\gamma + \mu} \tag{21}$$

$$I_{NARV}^* = \frac{\omega + \mu}{\alpha} D_{TR}^* \tag{22}$$

Substituting these into (15) allows solving for D_{TR}^* in terms of I_T^*. The existence of a unique positive endemic equilibrium $(S_p^*, I_T^*, I_{ARV}^*, I_{NARV}^*, D_{TR}^*)$ is guaranteed when $R_0 >$

1, which is characterized by non-adherence to the ART programme and subsequent drop out of care.

3.3. Basic Reproduction Number (R_0)

The basic reproduction number, R_0, represents the average number of secondary infections produced by a single infected individual introduced into a completely susceptible population [12]. It is a crucial threshold parameter that determines whether an infectious disease can invade and persist in a population. We use the next-generation matrix (NGM) method as described by van den Driessche and Watmough [1] to calculate R_0 for our model (1).

Let $X = (I_T, I_{ARV}, I_{NARV}, D_{TR})^T$ be the vector of infected compartments. The system can be written as $\frac{dX}{dt} = \mathcal{F}(X) - \mathcal{V}(X)$, where $\mathcal{F}(X)$ represents the rate of new infections entering each compartment, and $\mathcal{V}(X)$ represents the rates of transfer between infected compartments and removal from the infected compartments (by recovery or death). From system (1), the vector of new infection rates is:

$$\mathcal{F}(X) = \begin{pmatrix} \frac{\lambda c S_p I_T}{N} \\ 0 \\ 0 \\ 0 \end{pmatrix} \tag{23}$$

And the vector of transfer and removal rates is:

$$\mathcal{V}(X) = \begin{pmatrix} (\beta+\mu)I_T \\ -\phi\beta I_T + (\gamma+\mu)I_{ARV} - DT_{eff}\omega D_{TR} \\ -(1-\phi)\beta I_T - \gamma I_{ARV} + (\alpha+\mu+\delta)I_{NARV} - (1-DT_{eff})\omega D_{TR} \\ -\alpha I_{NARV} + (\omega+\mu)D_{TR} \end{pmatrix} \tag{24}$$

The Jacobian matrices of $\mathcal{F}(X)$ and $\mathcal{V}(X)$ evaluated at the Disease-Free Equilibrium $E_0 = (\frac{\pi N}{\mu}, 0, 0, 0, 0)$ are denoted by F and V, respectively.

$$F = \left.\frac{\partial \mathcal{F}_i}{\partial X_j}\right|_{E_0} = \begin{pmatrix} \frac{\lambda c S_{p0}}{N} & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix} = \begin{pmatrix} \frac{\lambda c \pi}{\mu} & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix} \tag{25}$$

$$V = \left.\frac{\partial \mathcal{V}_i}{\partial X_j}\right|_{E_0} = \begin{pmatrix} \beta+\mu & 0 & 0 & 0 \\ -\phi\beta & \gamma+\mu & 0 & -DT_{eff}\omega \\ -(1-\phi)\beta & -\gamma & \alpha+\mu+\delta & -(1-DT_{eff})\omega \\ 0 & 0 & -\alpha & \omega+\mu \end{pmatrix} \tag{26}$$

The next-generation matrix is $K = FV^{-1}$. R_0 is defined as the spectral radius (dominant eigenvalue) of K, denoted by $\rho(K)$. Since F has only the first element non-zero, $R_0 = \rho(FV^{-1})$ simplifies considerably. Therefore, the next-generation matrix is:

$$FV^{-1} = \begin{pmatrix} \frac{\lambda c \pi}{\mu(\beta+\mu)} & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix} \tag{27}$$

The spectral radius is the largest absolute value of the eigenvalues, which is simply the expression.

$$R_0 = \rho(FV^{-1}) = \frac{\lambda c \pi}{\mu(\beta+\mu)} \tag{28}$$

This R_0 represents the number of secondary infections generated by a single individual in the initial infectious stage (I_T) before they either start treatment, default, or die. It depends on the infection rate (λc), the recruitment rate (π), the natural death rate (μ), and the rate of leaving the initial infected stage ($\beta + \mu$).

The value of R_0 determines the potential for the epidemic to spread. If $R_0 > 1$, each infected individual produces, on average, more than one new infection, and the disease can spread in the population. If $R_0 < 1$, each infected individual produces, on average, less than one new infection, and the disease will die out.

3.4. Stability Analysis

3.4.1. Local Stability of the Disease-Free Equilibrium

To determine the local stability of the DFE, $E_0 = (\frac{\pi N}{\mu}, 0, 0, 0, 0)$, we analyze the eigenvalues of the Jacobian matrix of the full system (1) evaluated at E_0. The Jacobian matrix $J(E_0)$ is:

$$J(E_0) = \begin{pmatrix} -\mu & 0 & 0 & 0 & 0 \\ 0 & \frac{\lambda c \pi}{\mu} - (\beta+\mu) & 0 & 0 & 0 \\ 0 & \phi\beta & -(\gamma+\mu) & 0 & DT_{eff}\omega \\ 0 & (1-\phi)\beta & \gamma & -(\alpha+\mu+\delta) & (1-DT_{eff})\omega \\ 0 & 0 & 0 & \alpha & -(\omega+\mu) \end{pmatrix} \tag{29}$$

The eigenvalues are the diagonal entries:

$\lambda_1 = -\mu$

$\lambda_2 = \frac{\lambda c\pi}{\mu} - (\beta + \mu) = (\beta + \mu)\left(\frac{\lambda c\pi}{\mu(\beta+\mu)} - 1\right) = (\beta + \mu)(R_0 - 1)$

$\lambda_3 = -(\gamma + \mu)$

$\lambda_4 = -(\alpha + \mu + \delta)$

$\lambda_5 = -(1 + \mu)$

The DFE is locally asymptotically stable if and only if all eigenvalues have negative real parts. Clearly, $\lambda_1 = -\mu < 0$, $\lambda_3 = -(\gamma + \mu) < 0$, $\lambda_4 = -(\alpha + \mu + \delta) < 0$, and $\lambda_5 = -(1 + \mu) < 0$, since all model parameters are assumed positive. The stability condition thus depends on the sign of λ_2. We require $\lambda_2 < 0$:

$$(\beta + \mu)(R_0 - 1) < 0$$

Since $\beta + \mu > 0$, this inequality holds if and only if $R_0 - 1 < 0$, which means $R_0 < 1$. If $R_0 > 1$, then $\lambda_2 > 0$, and the DFE is unstable.

Therefore, the Disease-Free Equilibrium E_0 is locally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$. This confirms the threshold role of R_0 as defined in (28).

3.5. Sensitivity Analysis

Sensitivity analysis helps identify the model parameters that have the most significant impact on the basic reproduction number, R_0. This information is crucial for determining effective control strategies. We use the normalized forward sensitivity index, which measures the relative change in R_0 resulting from a relative change in a parameter P. The sensitivity index of R_0 with respect to a parameter P, denoted by $\Upsilon_P^{R_0}$, is defined as:

$$\Upsilon_P^{R_0} = \frac{\partial R_0}{\partial P} \times \frac{P}{R_0} \tag{30}$$

A positive index $\Upsilon_P^{R_0}$ indicates that R_0 increases as the parameter P increases, while a negative index indicates that R_0 decreases as P increases. The magnitude of the index quantifies the strength of the impact.

Let $Y = (\gamma+\mu)[(\alpha+\mu+\delta)(\omega+\mu) - \alpha((1-DT_{eff})\omega)] + (\beta + \mu)DT_{eff}\omega\gamma\alpha$ represent the denominator term. The sensitivity indices are given as follows:

For π:

$$\Upsilon_\pi^{R_0} = \frac{\partial R_0}{\partial \pi}\frac{\pi}{R_0} = 1 \tag{31}$$

For λ:

$$\Upsilon_\lambda^{R_0} = \frac{\partial R_0}{\partial \lambda}\frac{\lambda}{R_0} = 1 \tag{32}$$

For c:

$$\Upsilon_c^{R_0} = \frac{\partial R_0}{\partial c}\frac{c}{R_0} = 1 \tag{33}$$

For μ:

$$\Upsilon_\mu^{R_0} = -\frac{\mu[(\gamma + \mu)(\alpha + \mu + \delta)(2 + \mu) + DT_{eff}\alpha\gamma(1 + \beta + \mu)]}{Y} < 0 \tag{34}$$

For ϕ:

$$\Upsilon_\phi^{R_0} = -\frac{\phi(\mu^2 + \mu\beta + \mu\phi)}{\lambda\pi} < 0 \tag{35}$$

For δ:

$$\Upsilon_\delta^{R_0} = -\frac{\delta(\gamma + \mu)(1 + \mu)}{Y} < 0 \tag{36}$$

For DT_{eff}:

$$\Upsilon_{DT_{eff}}^{R_0} = -\frac{DT_{eff}\omega\alpha(\gamma(\beta + \mu) + (\gamma + \mu)\alpha)}{Y} < 0 \tag{37}$$

For α:

$$\Upsilon_\alpha^{R_0} = -\frac{\alpha((\gamma + \mu)(1 - DT_{eff})\omega + \gamma DT_{eff}\omega(\beta + \mu))}{Y} < 0 \tag{38}$$

For β:

$$\Upsilon_\beta^{R_0} = -\frac{\beta\gamma\alpha DT_{eff}\omega}{Y} < 0 \tag{39}$$

For γ:

$$\Upsilon_\gamma^{R_0} = -\frac{\gamma[(\alpha + \mu + \delta)(1 + \mu) - \alpha((1 - DT_{eff})\omega) + \alpha DT_{eff}\omega(\beta + \mu)]}{Y} < 0 \tag{40}$$

Interpretation: The sensitivity indices $\Upsilon_{\pi}^{R_0}$, $\Upsilon_{\lambda}^{R_0}$, and $\Upsilon_{c}^{R_0}$ are equal to +1. This indicates that the basic reproduction number R_0 is directly and proportionally increased by increases in the recruitment rate π, the infection rate parameter λ, and the contact rate c. Doubling any of these parameters would double R_0, highlighting their critical role in driving the epidemic.

The indices for μ (natural death rate), δ (HIV-related death rate), γ (ART abandonment rate), α (tracing initiation rate), β (treatment initiation rate), and DT_{eff} (tracing effectiveness) are negative ($\Upsilon_{\mu}^{R_0} < 0$, $\Upsilon_{\delta}^{R_0} < 0$, $\Upsilon_{\gamma}^{R_0} < 0$, $\Upsilon_{\alpha}^{R_0} < 0$, $\Upsilon_{\beta}^{R_0} < 0$, $\Upsilon_{DT_{eff}}^{R_0} < 0$). This implies that increasing any of these rates decreases the basic reproduction number R_0. For instance, increasing the effectiveness of defaulter tracing (DT_{eff}) or the rate at which individuals are traced (α) contributes to reducing the potential spread of the disease. Similarly, increasing treatment initiation (β) or reducing ART abandonment (γ) also lowers R_0. Increased death rates (μ, δ) reduce the time individuals can transmit, thus lowering R_0.

The relative magnitudes of these negative indices would determine which of these parameters offer the most effective levers for reducing R_0 below unity. The analysis suggests that interventions targeting infection rate, contact rate, tracing, treatment initiation, and retention are all theoretically beneficial for controlling the epidemic spread reflected in this particular R_0 formulation.

4. Discussion

This paper presented the formulation and mathematical analysis of a compartmental model for HIV/AIDS transmission, incorporating the dynamics of antiretroviral therapy (ART) and defaulter tracing. The analysis provides theoretical insights into the conditions governing the spread and persistence of the disease within this framework.

The confirmation of positivity and boundedness of solutions ensures that the model is epidemiologically meaningful, as population sizes remain non-negative and do not grow infinitely, confined within the feasible region Ω. This establishes the mathematical well-posedness of the system.

The analysis identified two key equilibrium states: the Disease-Free Equilibrium (DFE) and the Endemic Equilibrium (EE). The DFE, where the population is entirely free of infection, always exists. Its local stability is governed by the basic reproduction number, R_0. Our analysis, using the standard next-generation matrix method applied to the derived model equations, yielded the R_0. The condition for DFE stability is $R_0 < 1$. This result aligns with epidemiological theory, indicating that the disease cannot successfully invade the population if each primary infected individual generates, on average, less than one new infection during their initial infectious period (before treatment initiation or death). The parameters influencing this threshold are the effective contact rate (λc), the recruitment rate (π), the natural death rate (μ), and the rate of progression out of the initial infectious state (β).

Conversely, when $R_0 > 1$, the DFE becomes unstable, and the analysis indicates the existence of an Endemic Equilibrium (EE), where the infection persists within the population. The existence of the EE signifies that HIV transmission can be sustained over the long term, with the levels of infection in different compartments ($I_T^*, I_{ARV}^*, I_{NARV}^*, D_{TR}^*$) determined by the balance between new infections, treatment dynamics, defaulting, tracing, and mortality, as shown in the complex expressions derived in Section 3.2.2. While the stability of the EE was not rigorously analyzed here due to complexity, its existence when $R_0 > 1$ implies the necessity of interventions to control the disease burden.

The sensitivity analysis offers insights into potential control levers. Parameters related to transmission (λ, c) and population turnover (π) showed a positive and proportional impact on R_0, highlighting the importance of prevention measures (reducing λ, c) for controlling the epidemic threshold. Parameters related to treatment initiation (β), ART abandonment (γ), tracing initiation (α), tracing effectiveness (DT_{eff}), and mortality (μ, δ) were found to have a negative impact on the R_0. This suggests that, according to that formulation, improving treatment uptake and retention, as well as enhancing defaulter tracing efforts, could theoretically contribute to reducing R_0 and controlling the epidemic. The magnitude of these indices, if numerically evaluated, would rank the relative effectiveness of targeting each parameter.

The primary contribution of this mathematical analysis is the rigorous establishment of the model's basic properties and the threshold condition ($R_0 < 1$) for disease elimination, based on standard epidemiological methods applied to the formulated system. This theoretical foundation supports further investigation, such as numerical simulations, to quantify the impact of different tracing and retention strategies on the long-term dynamics of HIV.

5. Conclusion

In this study, we developed and analyzed a five-compartment mathematical model described by a system of ordinary differential equations to investigate the dynamics of HIV/AIDS transmission, explicitly including compartments for individuals on ART, those not on ART, and those undergoing defaulter tracing.

The mathematical analysis established the fundamental properties of the model. We proved that solutions are non-negative and bounded. We identified the Disease-Free Equilibrium (E_0) and derived the conditions for the existence of an Endemic Equilibrium (E^*). The basic reproduction number, derived using the next-generation matrix method was shown to govern the local stability of the DFE. The DFE is locally asymptotically stable if $R_0 < 1$, representing the condition for disease elimination, and unstable if $R_0 > 1$, allowing for disease persistence (existence of EE). Sensitivity analysis indicated that disease transmission-related parameters (λ, c, π) are key drivers increasing R_0, while parameters related to treatment (β, γ), tracing (α, DT_{eff}), and mortality

(μ, δ) contribute to decreasing the R_0 expression analyzed therein.

This work provides a mathematical framework for understanding the interplay between HIV transmission, treatment, and defaulter tracing. The establishment of the stability threshold R_0 offers a critical theoretical insight for public health interventions aimed at controlling and potentially eliminating HIV.

ORCID

0009-0005-1960-4896 (Sammy Maingi)
0000-0002-6741-5011 (Benjamin Kikwai)
0000-0002-5822-8687 (Mark Kimathi)

Abbreviations

AIDS	Acquired Immuno-Defficiency Syndrome
ART	Anti-Retroviral Therapy
DFE	Disease Free Equilibrium
EE	Endemic Equilibrium
HIV	Human Immuno-deficiency Virus
NGM	Next Generation Matrix
ODEs	Ordinary Differential Equations
SI	Susceptible-Infected
SIR	Susceptible-Infected-Recovered

Conflicts of Interest

The authors declare no conflicts of interest.

References

[1] Abiodun, O. E., Adebimpe, O., Ndako, J. A., Oludoun, O., Aladeitan, B. and Adeniyi, M., "Mathematical modeling of HIV-HCV co-infection model: Impact of parameters on reproduction number," *F1000Research*, vol. 11, p. 1153, 2022.

[2] Thomson, K. A., Cheti, E. O. and Reid, T., "Implementation and outcomes of an active defaulter tracing system for HIV, prevention of mother to child transmission of HIV (PMTCT), and TB patients in Kibera, Nairobi, Kenya," *Transactions of the Royal Society of Tropical Medicine and Hygiene*, vol. 105, no. 6, pp. 320–326, 2011.

[3] Estill, J., Tweya, H., Egger, M., Wandeler, G., Feldacker, C., Johnson, L. F., Blaser, N., Vizcaya, L. S., Phiri, S. and Keiser, O., "Tracing of patients lost to follow-up and HIV transmission: mathematical modeling study based on 2 large ART programs in Malawi," *JAIDS Journal of Acquired Immune Deficiency Syndromes*, vol. 65, no. 5, pp. e179–e186, 2014.

[4] Omondi, E. O., Mbogo, R. W. and Luboobi, L. S., "A mathematical model of HIV transmission between commercial sex workers and injection drug users," *Research in Mathematics*, vol. 9, no. 1, p. 2082044, 2022.

[5] Xue, L., Sun, Y., Ren, X. and Sun, W., "Modelling the transmission dynamics and optimal control strategies for HIV infection in China," *Journal of Biological Dynamics*, vol. 17, no. 1, p. 2174275, 2023.

[6] Omondi, E. O., Mbogo, R. W. and Luboobi, L. S., "Mathematical modelling of the impact of testing, treatment and control of HIV transmission in Kenya," *Cogent Mathematics & Statistics*, vol. 5, no. 1, p. 1475590, 2018.

[7] Raza, A., Ahmadian, A., Rafiq, M., Salahshour, S., Naveed, M., Ferrara, M. and Soori, A. H., "Modeling the effect of delay strategy on transmission dynamics of HIV/AIDS disease," *Advances in Difference Equations*, vol. 2020, pp. 1–13, 2020.

[8] Levy, B., Correia, H. E., Chirove, F., Ronoh, M., Abebe, A., Kgosimore, M., Chimbola, O., Machingauta, M. H., Lenhart, S. and White, K. A. J., "Modeling the effect of HIV/AIDS stigma on HIV infection dynamics in Kenya," *Bulletin of Mathematical Biology*, vol. 83, pp. 1–25, 2021.

[9] Etoori, D., Wringe, A., Renju, J., Kabudula, C. W., Gomez-Olive, F. X. and Reniers, G., "Challenges with tracing patients on antiretroviral therapy who are late for clinic appointments in rural South Africa and recommendations for future practice," *Global Health Action*, vol. 13, no. 1, p. 1755115, 2020.

[10] Chazuka, Z., Madubueze, C. E. and Mathebula, D., "Modelling and analysis of an HIV model with control strategies and cost-effectiveness," *Results in Control and Optimization*, vol. 14, p. 100355, 2024.

[11] De Angeles, K., "PMTCT care engagement as a social practice and system: insights from an mHealth intervention and routine tracing in western Kenya," PhD thesis, Karolinska Institutet, 2024.

[12] Dharmaratne, S., Sudaraka, S., Abeyagunawardena, I., Manchanayake, K., Kothalawala, M. and Gunathunga, W., "Estimation of the basic reproduction number (R0) for the novel coronavirus disease in Sri Lanka," *Virology Journal*, vol. 17, pp. 1–7, 2020.

[13] Ahmetolan, S., Bilge, A. H., Demirci, A. and Dobie, A. P., "A Susceptible–Infectious (SI) model with two infective stages and an endemic equilibrium," *Mathematics and Computers in Simulation*, vol. 194, pp. 19–35, 2022.

[14] Young, P. W., Musingila, P., Kingwara, L., Voetsch, A. C., Zielinski-Gutierrez, E., Bulterys, M., Kim, A. A., Bronson, M. A., Parekh, B. S., Dobbs, T., et al., "HIV incidence, recent HIV infection, and associated factors, Kenya, 2007–2018," *AIDS Research and Human Retroviruses*, vol. 39, no. 2, pp. 57–67, 2023.

[15] Kemnic, T. R. and Gulick, P. G., "HIV Antiretroviral Therapy," *StatPearls [Internet]*, 2024.

[16] Endebu, T., Taye, G. and Deressa, W., "Rate and predictors of loss to follow-up in HIV care in a low-resource setting: analyzing critical risk periods," *BMC Infectious Diseases*, vol. 24, no. 1, pp. 1–10, 2024.

[17] NSDCC, "KMoT Report - November 2024," National Sustainable Development Coordination Committee (NSDCC), 2024. [Online]. Available: https://analytics.nsdcc.go.ke/estimates/KMoT%20Report%20-%20November-2024.pdf. [Accessed: 2024-03-15].