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Mathematical Modeling of HIV Transmission Dynamics: Incorporating Treatment and Removal as Control Strategies

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ABSTRACT

This study develops and analyzes a modified mathematical model to examine the transmission dynamics of HIV, emphasizing treatment and removal as control strategies. Extending the susceptible-infectious (SI) model by Odebiyu et al. (2024), the developed framework incorporates additional compartments for exposed, asymptomatic, symptomatic, treated and removed individuals, thereby offering a more comprehensive representation of HIV progression. The analytical investigation establishes the existence, uniqueness, positivity and boundedness of the model's solutions, ensuring their biological relevance. Both the disease-free equilibrium and endemic equilibrium points are derived, while the basic reproduction number R_0 is computed using the next-generation matrix approach. The results reveal that effective treatment and timely removal of infected individuals significantly reduce R_0 and can halt disease persistence if $R_0 < 1$. The findings highlight the importance of treatment accessibility, removal of infectious individuals through recovery or death and sustained screening programs in curtailing HIV spread. The model offers valuable insights to policymakers and public health stakeholders, particularly in high-prevalence regions, where integrated strategies remain essential for controlling and eventually eliminating HIV.

Keywords: HIV Transmission; Mathematical Modeling; Basic Reproduction Number (R_0); Treatment and Removal Strategies; Epidemiological Modeling; Disease Control and Prevention.

1. Introduction

The HIV/AIDS epidemic continues to pose a major global health challenge, affecting millions worldwide, with sub-Saharan Africa carrying the highest burden (WHO, 2021). Mathematical modeling has been instrumental in understanding HIV transmission dynamics and evaluating the impact of interventions. Initially, traditional models focused on the basic interactions among susceptible, infected and removed individuals. However, the advent of effective treatment and prevention measures such as Highly Active Antiretroviral Therapy (HAART), Pre-Exposure Prophylaxis (PrEP) and other antiretroviral drugs (ARVs) has shifted the focus toward incorporating these strategies into models as active control mechanisms. These treatments significantly reduce viral loads, prevent transmission and alter disease progression, particularly in regions with accessible healthcare (Johnson et al., 2020; Abdulla et al., 2020). Recent studies have shown that early and widespread treatment helps curb new infections and modifies the dynamics between infected and susceptible individuals, though access limitations in developing regions still hinder progress (Fitzgerald et al., 2022; Chun et al., 2021).

Beyond capturing biological processes, mathematical models serve as vital tools for simulating transmission, evaluating "what-if" scenarios and informing effective public health policies (Khademi et al., 2018). These models assist in assessing the outcomes of various interventions such as condom use, mass treatment and education campaigns, even though they are constrained by assumptions and data uncertainties (NCDC, 2022). In Nigeria, the HIV epidemic followed continental trends, with high infection rates during the 1980s and 1990s driven by stigma, rapid urbanization and limited treatment access (Huo & Chen, 2015). The introduction of major initiatives like PEPFAR and the Global Fund in the 2000s expanded testing and antiretroviral therapy, leading to reduced incidence and mortality by the 2010s (CDC, 2023). However, persistent challenges such as cultural stigma and inequitable healthcare access continue to emphasize the need for data-driven modeling to strengthen HIV control and prevention strategies.

The spread of infectious diseases, particularly those with varying degrees of symptoms such as HIV, poses a significant challenge to public health systems globally. In their 2024 study, Odebiyu et al. formulated a susceptible-infectious (SI) model in which the infectious compartment was subdivided into asymptomatic and symptomatic populations. Their findings revealed that increasing the rate of detection through screening could help control the spread of the disease. While their model provides valuable insights into the role of detection and treatment in curbing transmission, it does not comprehensively address the dynamics of HIV transmission when screening are actively incorporated as control measures. In this model, this research was modified the model due to Odebiyu et al. (2024) by incorporate the exposed class asymptotic and symptotic classes and use treatment and removal as a dynamic control strategies.

Screening and early diagnosis are foundational steps in managing HIV. Voluntary counseling and testing, guided by the WHO's five Cs-consent, confidentiality, counseling, correct results and connection to care are vital for initiating effective treatment (Granich et al., 2019; Gurmu et al., 2021). When combined with ART, early diagnosis significantly reduces community viral load, thus limiting transmission. Nevertheless, various challenges hinder the success of these strategies, such as poor access to healthcare, stigma, social discrimination and inadequate linkage to care (Arachchige, 2021). To maximize effectiveness, test-and-treat must be part of a broader prevention strategy that includes PrEP, condom distribution, education and harm reduction (Ayele et al., 2021). Recent models have expanded in scope to include demographic characteristics, treatment stages, disease progression and co-infection dynamics. Granich et al. (2019) and Nthiiri et al. (2015) presented compartmental models dividing populations into susceptible, infected and treated groups, capturing HIV transmission and ART impact. Further, Feldman et al. (2017), Khademi et al. (2018) and Nsuami & Witbooi (2018) highlighted bacterial co-infections, PrEP efficacy and combined interventions. Together, these studies underscore the multifactorial nature of HIV dynamics and affirm the value of mathematical modeling in crafting comprehensive, evidence-based public health strategies.

Ayele et al. (2021) utilized mathematical modeling to tailor HIV/AIDS control strategies to the Ethiopian context, emphasizing the value of context-specific interventions in improving public health outcomes. Similarly, Gurmu et al. (2021) introduced a model incorporating optimal control strategies, highlighting the critical balance between prevention and treatment in minimizing HIV transmission. Arachchige (2021) proposed an innovative immunotherapeutic approach using CAR-NK cells to potentially eradicate HIV, signaling a shift beyond conventional ART towards curative treatments. Chukwu et al. (2022) examined HIV/AIDS-listeriosis co-infection dynamics, underscoring the importance of integrated management strategies for co-infections. Zhao et al. (2022) contributed to the field by applying fractional differential equations to model HIV dynamics, offering refined tools for capturing complex disease behavior. Chazuka et al. (2024) assessed the cost-effectiveness of various HIV control strategies, providing a financial framework for sustainable policymaking. Finally, Odebiyu et al. (2024) developed an SI model with asymptomatic and symptomatic compartments, concluding that enhancing detection rates through screening significantly curtails disease spread.

1.1. Study Objectives

The objectives are:

1) To establish the theorem of existence and uniqueness solution of the model and provide it prove,
2) To establish the theorem of positivity and boundedness of the solution and provide it prove,
3) To obtain the disease free and endemic equilibrium points of the model, and
4) To obtain the basic reproductive number.

2. Development of the Model

Odebiyu et al. (2024) formulate a mathematical modelling on assessing the impact of screening on HIV/AIDS transmission dynamics. Certain assumptions were incorporated, parameters and variables were meticulously defined and the chapter introduces the model equation and methodology employed in the study. The existing model by Odebiyu et al. (2024) was formulated with four class of susceptible, symptomatic infectious, asymptomatic compartmental and AIDS population.

2.1. Modified Model

This section presents a formulation of the mathematical modeling approach for understanding the transmission and spread of HIV/AIDS. The new model was modified due to Odebiyu et al. (2024) by incorporate the exposed class, asymptotic and asymptotical classes and use treatment and removal as a dynamic control strategy.

2.2. Variables and Parameters of the Modified Model

Table 2.1. Variables and their Meaning of the Modified Model

Variables	**Meaning**
$S(t)$	Susceptible population at a time *t*
$E(t)$	Exposed population at a time *t*
$I_A(t)$	Asymptomatically infected population at a time *t*
$I_S(t)$	Symptomatic infected population at a time *t*
$A(t)$	Population of people with AIDS at a time *t*
$T(t)$	Treated individuals at a time *t*
$R(t)$	Removal individuals at a time *t*

Table 2.2. Parameters and their Meaning of the Modified Model

Parameter	**Meaning**
Λ	The recruitment rate
μ	Natural mortality rate
β	Contact rate between I_A, I_S and susceptible population
D	Death due to sickness
B	Rate of transfer from susceptible class to exposed class

θ	Rate of movement from exposed class of asymptomatically infected classes
$1-\theta$	Probability of people moved from exposed class of symptomatically infected classes
π	transfer rate from asymptomatically infected class
γ	Rate of movement from symptomatically infected classes to AIDS compartment
η	Rate of transfer of individuals from exposed class to symptomatic infected individuals class
σ	Rate of movement from asymptomatic infected individuals class to AIDS class
τ	Movement rate from AIDS compartment to Treatment class
λ	Propagation rate from symptomatically infected class to removed class
ω	Movement rate from treatment class to removed class
α	Movement rate from symptomatically infected class to treatment compartment

2.3. Assumptions for the Modified Model

The following assumptions were made for the modified model:

1) There is natural death in all the compartments,

2) If susceptible population come in contact into infected, there is exposure,

3) Individuals can be infected asymptomatically or symptomatically,

4) Asymptomatically and symptomatically infected individuals can be moved to AIDS compartment when the sickness worsens,

5) It is possible for individuals to be removed or removed when treated.

2.4. Formulation of the Modified Model

The modified mathematical modelling of HIV/AID is divided into seven compartments and each compartment has it its variables and parameters. The susceptible compartment signify $S(t)$ increase by the recruitment rate Λ and decreases by $\beta b(I_A + I_S)S$ and μS to form a differential equation of the for

$$\frac{dS}{dt} = \Lambda - \beta b(I_A + I_S)S - \mu S \qquad (2.1)$$

The exposed compartment denote by $E(t)$. The compartment increase by $\beta b(I_A + I_S)S$ and decreases by $(1+\mu)E$. The differential equation is given by

$$\frac{dE}{dt} = \beta b(I_A + I_S)S - (1+\mu)E \qquad (2.2)$$

The Asymptomatically infected population denotes $I_A(t)$. It increase by θE and decreases by $\pi I_A, \sigma I_A$ and $(\mu + d)I_A$ based on the increase and decrease, the differential equation is

$$\frac{dI_A}{dt} = \theta E - \pi I_A - \sigma I_A - (\mu + d)I_A \qquad (2.3)$$

The Symptomatically infected compartment $I_S(t)$ increases by $(1-\theta)E$ and πI_A , it also decreases by $\gamma I_S, \alpha I_S, \lambda I_S$ and $(\mu+d)I_S$ with the equation

$$\frac{dI_S}{dt} = (1-\theta)E + \pi I_A - \gamma I_S - \alpha I_S - \lambda I_S - (\mu+d)I_S \qquad (2.4)$$

The AIDS compartment denotes $A(t)$, increases by γI_S and αI_S, decreases by ηA and $(\mu+d)A$. Its differential equation is of the form

$$\frac{dA}{dt} = \gamma I_S + \sigma I_A - \eta A - (\mu+d)A \qquad (2.5)$$

The treated compartment $T(t)$ increases by $\alpha I_S, \eta A$ and decreases by $\omega T, (\mu+d)T$ to form a differential equation

$$\frac{dT}{dt} = \alpha I_S + \eta A - \omega T - (\mu+d)T \qquad (2.6)$$

The Removed compartment signify $R(t)$ increases by λI_S , ωT and decrease by μR. The differential equation is given by

$$\frac{dR}{dt} = \lambda I_S + \omega T - \mu R \qquad (2.7)$$

Based on the model assumptions and formulation, the schematic diagram of the modified model are presented as

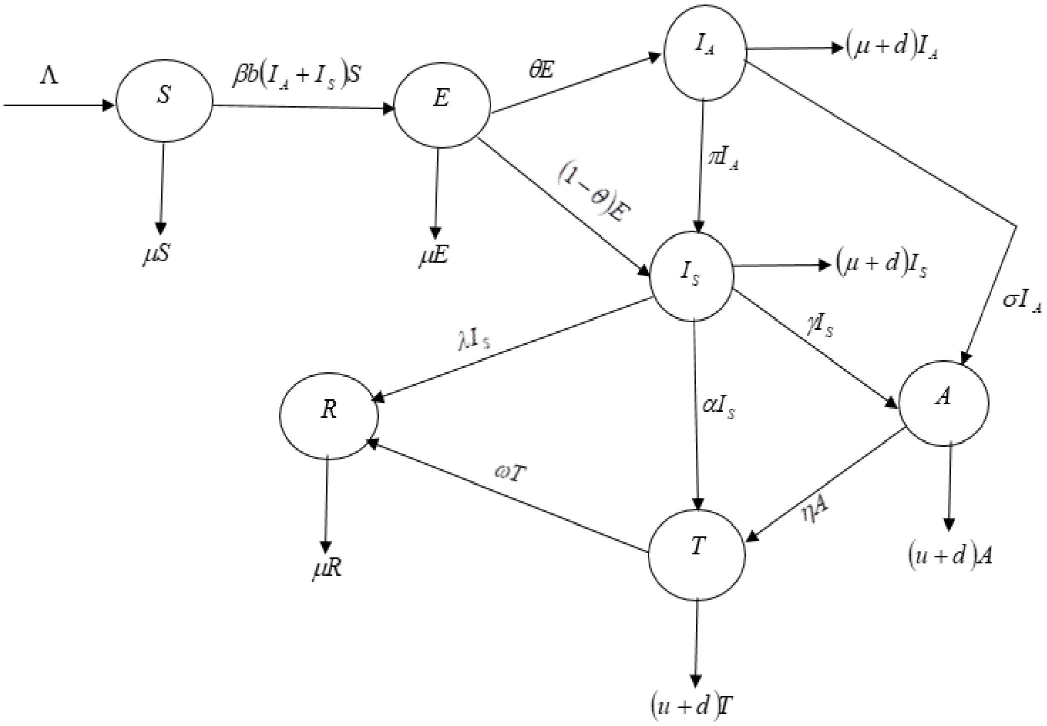

Figure 2.1. Schematic diagram of modified model

The model equations were derived from figure 2.1 and presented in equation (2.8) as

$$\left.\begin{aligned}
\frac{dS}{dt} &= \Lambda - \beta b\left(I_A + I_S\right)S - \mu S\\
\frac{dE}{dt} &= \beta b\left(I_A + I_S\right)S - \left(1+\mu\right)E\\
\frac{dI_A}{dt} &= \theta E - \pi I_A - \sigma I_A - \left(\mu + d\right)I_A\\
\frac{dI_S}{dt} &= \left(1-\theta\right)E + \pi I_A - \gamma I_S - \alpha I_S - \lambda I_S - \left(\mu + d\right)I_S\\
\frac{dA}{dt} &= \gamma I_S + \sigma I_A - \eta A - \left(\mu + d\right)A\\
\frac{dT}{dt} &= \alpha I_S + \eta A - \omega T - \left(\mu + d\right)T\\
\frac{dR}{dt} &= \lambda I_S + \omega T - \mu R
\end{aligned}\right\} \qquad (2.8)$$

3. HIV/AIDS Model Analysis

Model analysis encompasses a range of methods used to explore and understand the behavior and properties of mathematical or computational models, such as those describing the dynamics of HIV/AIDS. It is a fundamental tool across fields like engineering, physics, biology and economics, where models are used to represent complex systems. Key components of this analysis include assessing the Existence and Uniqueness of the Solution, positivity of solutions, identifying disease-free and endemic equilibrium states and determining the basic reproduction number.

3.1. Existence and Uniqueness of the Solution

If $f\left(t, y\right)$ has continuous partial derivative $\frac{\partial f_i}{\partial y_i}$ for $i = 1, 2, \cdots, n$ on a bounded convex domain R, then it satisfies a Lipchitz condition in R, $\left\| f\left(t, y\right) - f\left(t, y_{n-1}\right)\right\| \le k\left\| y_n - y_{n-1}\right\|, i = 1, 2, 3, \cdots$

The existence and uniqueness of the solution is explained using the theorem 3.1.

Theorem 3.1

There exist a domain D in which the solution set $\left\{S, E, I_A, I_S, A, T, R\right\}$ is contained and unique.

Proof:

Given that the solution set with positive initial condition

$\left\{S\left(0\right) = S_0, E\left(0\right) = E_0, I_A\left(0\right) = I_{A0}, I_S\left(0\right) = I_{S0}, A\left(0\right) = A_0, T\left(0\right) = T_0, R\left(0\right) = R_0\right\}$, we let

$\left\{S, E, I_A, I_S, A, T, R\right\} = S\left(t\right), E\left(t\right), I_A\left(t\right), I_S\left(t\right), A\left(t\right), T\left(t\right), R\left(t\right)$. The derivative $\frac{dm}{dt}$ along solution of the system (2.1) is obtained by

$$\frac{dm}{dt}=\frac{dm}{dS}\frac{dS}{dt}+\frac{dm}{dE}\frac{dE}{dt}+\frac{dm}{dI_A}\frac{dI_A}{dt}+\frac{dm}{dI_S}\frac{dI_S}{dt}+\frac{dm}{dA}\frac{dA}{dt}+\frac{dm}{dT}\frac{dT}{dt}+\frac{dm}{dR}\frac{dR}{dt} \qquad (3.1)$$

It follows that

$$\frac{dm}{dt}\le \Lambda-\mu m \qquad (3.2)$$

Solving the differential inequalities, we have

$$\frac{dm}{dt}\le \frac{\Lambda}{\mu}\left(1-e^{-\mu t}\right)+m\left(S_0, E_0, I_{A0}, I_{S0}, A_0, T_0, R_0\right)e^{-\mu t} \qquad (3.3)$$

Hence, taking the limit as $t\to\infty$ gives $\psi=\frac{\Lambda}{\mu}$.

Thus all the solution are contained in the modified model exists and is given by

$$D=\left\{S_0, E_0, I_{A0}, I_{S0}, A_0, T_0, R_0 \in \Re_+^7 : N\le\frac{\Lambda}{\mu}\right\} \qquad (3.4)$$

This ends the prove.

3.2. Positivity and Boundedness of Solution

To establish the positivity of the model's solution, it is necessary to verify that all population compartments are non-negative. Ensuring non-negative values is essential, as negative population sizes are not physically interpretable. This property was demonstrated by providing the proof to theorem 3.2.

Theorem 3.2

Let the initial solution set be $\{S_0\ge 0, E_0\ge 0, I_{A0}\ge 0, I_{S0}\ge 0, A_0\ge 0, T_0\ge 0, R_0\ge 0\}\in R_+^7$

Then the solution set $\{S(t), E(t), I_A(t), I_S(t), A(t), T(t), R(t)\}$ is positive for all $t>0$.

Proof

$$\frac{dS}{dt}=\Lambda-\beta b\left(I_A+I_S\right)S-\mu S$$

i.e $\frac{dS}{dt}=\Lambda-\left[\beta b\left(I_A+I_S\right)-\mu\right]S \qquad (3.5)$

Since we are considering only the negative terms susceptible population S, then

$$\frac{dS}{dt}\ge-\left[\beta b\left(I_A+I_S\right)-\mu\right]S \qquad (3.6)$$

This results to

$$\frac{dS}{dt} \geq -(\lambda + \mu)S \qquad (3.7)$$

Where

$$\lambda = \beta b(I_A + I_S) \qquad (3.8)$$

Solving for (3.7) by separating the variables, we have

$$\frac{dS}{S} \geq -(\lambda + \mu)dt \qquad (3.9)$$

Integrating (3.9) we have

$$\ln(S) \geq -\int(\lambda + \mu)dt \qquad (3.10)$$

i.e $\ln(S) \geq -(\lambda + \mu)t + C$ (3.11)

Taking the exponential of (3.11)

$$S(t) \geq e^{-(\lambda+\mu)t+C} \qquad (3.12)$$

i.e $S(t) \geq e^{-(\lambda+\mu)t} + e^{C}$ (3.13)

$$S(t) \geq Ke^{-(\lambda-\mu)t} \qquad (3.14)$$

Where

i.e $K = e^{C}$ (3.15)

Applying the initial conditions at i.e $t = 0$, equation (3.14) becomes

$$S(t) \geq Ke^{-(\lambda+\mu)0} \qquad (3.16)$$

$$S(0) \geq K \qquad (3.17)$$

Substituting (3.15) into (3.12), we have

$$S(t) \geq S(0)e^{-(\lambda+\mu)t} > 0 \qquad (3.18)$$

For all $t \geq 0$

In a similar way, we can text the positivity of the remaining variables.

3.3. Disease Free Equilibrium (DEF) of the Model

The Disease-Free Equilibrium (DFE) refers to a condition where the entire population (2.1) is uninfected, with all compartments associated with infection set to zero. To find this state, the derivatives of the infected variables are equated to zero under the assumption of no disease presence. This means that $E = I_A = I_S = A = T = R = 0$.

Theorem 3.3

A disease-free equilibrium state of the model (2.1) exist at a point

$$E_0 = \left(S^0, E^0, {I_A}^0, {I_S}^0, A^0, T^0, R^0\right) = \left(\frac{\Lambda}{\mu}, 0, 0, 0, 0, 0, 0\right).$$

Proof:

Let $(S, E, I_A, I_S, A, T, R) = S^0, E^0, {I_A}^0, {I_S}^0, A^0, T^0, R^0$ be at equilibrium state.

From

$$\frac{dS}{dt} = \Lambda - \beta b(I_A + I_S)S - \mu S = 0$$
$$\Lambda - \beta b(I_A + I_S)S - \mu S = 0$$
$$\Lambda - \mu S = 0$$
$$\Lambda = \mu S$$
$$S^0 = \frac{\Lambda}{\mu}$$

From

$$\frac{dE}{dt} = \beta b(I_A + I_S)S - (1+\mu)E = 0$$
$$\beta b(I_A + I_S)S - (1+\mu)E = 0$$
$$-(1+\mu)E = 0$$
$$E^0 = \frac{0}{(1+\mu)} = 0$$

From

$$\frac{dI_A}{dt} = \theta E - \pi I_A - \sigma I_A - (\mu + d)I_A = 0$$
$$\theta E - (\pi + \sigma + \mu + d)I_A = 0$$
$$-(\pi + \sigma + \mu + d)I_A = 0$$
$${I_A}^0 = \frac{0}{(\pi + \sigma + \mu + d)} = 0$$

From

$$\frac{dI_S}{dt} = (1-\theta)E + \pi I_A - \gamma I_S - \alpha I_S - \lambda I_S - (\mu + d)I_S = 0$$
$$(1-\theta)E + \pi I_A - \gamma I_S - \alpha I_S - \lambda I_S - (\mu + d)I_S = 0$$
$$-(\gamma - \alpha - \lambda - \mu + d)I_S = 0$$
$${I_S}^0 = \frac{0}{(\gamma - \alpha - \lambda - \mu + d)} = 0$$

Similarly, $A = T = R = 0$.

$$\therefore \left(S^0, E^0, I_A{}^0, I_S{}^0, A^0, T^0, R^0\right) = \left(\frac{\Lambda}{\mu}, 0, 0, 0, 0, 0, 0\right)$$

3.4. Endemic Equilibrium Point of the Model E^*

The endemic equilibrium point of the model (2.1) is the point where

$S \neq 0, E \neq 0, I_A \neq 0, I_S \neq 0, A \neq 0, T \neq 0, R \neq 0$.

Theorem 3.4

The HIV presence/Endemic equilibrium point is

$$E^* = \left(S^*, E^*, I_A{}^*, I_S{}^*, A^*, T^*, R^*\right)$$

Where

$$S^* = \frac{\Lambda}{\beta b\left(I_A{}^* + I_S{}^*\right) + \mu}$$

$$E^* = \frac{\beta b\left(I_A{}^* + I_S{}^*\right)S}{(1+\mu)}$$

$$I_A{}^* = \frac{\theta E^*}{\pi + \sigma + \mu + d}$$

$$I_S{}^* = \frac{(1-\theta)E^* + \pi I_A{}^*}{\gamma + \alpha + \lambda + \mu + d}$$

$$A^* = \frac{\gamma I_S{}^* + \alpha I_S{}^*}{\eta - (\mu + d)}$$

$$T^* = \frac{\alpha I_S{}^* + \eta A^*}{\omega + \mu + d}$$

$$R^* = \frac{\lambda I_S{}^* + \omega T^*}{\mu}$$

3.5. Basic Reproduction Number (R_0)

The Basic Reproduction Number, often denoted as R_0, is a key epidemiological metric that indicates the average number of secondary infections caused by one infected individual in a fully susceptible population. This number depends on factors such as contact rate, transmission probability and the infectious period.

To find the basic reproduction number, R_0, for the model equation (2.1), we use the next-generation matrix approach, which involves linearizing the model around the Disease-Free Equilibrium (DFE). The infected compartments are $E(t), I_S(t), A(t), T(t)$ with the relevant equations

$$\left.\begin{aligned}
\frac{dE}{dt} &= \beta b(I_A + I_S)S - (1+\mu)E \\
\frac{dI_S}{dt} &= (1-\theta)E + \pi I_A - \gamma I_S - \alpha I_S - \lambda I_S - (\mu + d)I_S \\
\frac{dA}{dt} &= \gamma I_S + \sigma I_A - \eta A - (\mu + d)A \\
\frac{dT}{dt} &= \alpha I_S + \eta A - \omega T - (\mu + d)T
\end{aligned}\right\} \qquad (3.19)$$

At the DFE, the susceptible population S are at their equilibrium values

$$S = \frac{\pi}{\mu}$$

The infected Matrix (F)

$$F = \begin{pmatrix} \beta b(I_A + I_S)S \\ 0 \\ 0 \\ 0 \end{pmatrix} = \begin{bmatrix} 0 & \beta bS & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix}$$

and Transition Matrix (V)

$$V = \begin{pmatrix} (1+\mu)E \\ \gamma I_S + \alpha I_S + \lambda I_S + \mu I_S + dI_S - (1-\theta)E - \pi I_A \\ \eta A + \mu A + dA - \gamma I_S - \sigma I_A \\ \omega T + \mu T + dT - \alpha I_S - \eta A \end{pmatrix} = \begin{bmatrix} 1+\mu & 0 & 0 & 0 \\ -1-\theta & \gamma + \alpha + \lambda + \mu + d & -\pi & 0 \\ 0 & -\gamma & \eta + \mu + d & 0 \\ 0 & -\alpha & -\eta & \omega + \mu + d \end{bmatrix}$$

The next-generation matrix G of the F (new infection terms) and V (transition terms) is given by:

$$G = F \cdot V^{-1}$$

To calculate G we first need to find the inverse of V as

$$V^{-1} = \begin{bmatrix} \dfrac{1}{1+\mu} & 0 & 0 & 0 \\ \dfrac{(-1+\theta)b}{(-ba+\gamma\pi)(1+\mu)} & -\dfrac{b}{-ba+\gamma\pi} & \dfrac{-\pi}{-ba+\gamma\pi} & 0 \\ \dfrac{\gamma(-1+\theta)}{(-ba+\gamma\pi)(1+\mu)} & -\dfrac{\gamma}{-ba+\gamma\pi} & -\dfrac{a}{-ba+\gamma\pi} & 0 \\ \dfrac{(-1+\theta)(\alpha b+\gamma\eta)}{(-ba+\gamma\pi)(1+\mu)c} & -\dfrac{\alpha b+\gamma\eta}{(-ba+\gamma\pi)c} & -\dfrac{\eta a+\alpha\pi}{(-ba+\gamma\pi)c} & \dfrac{1}{c} \end{bmatrix}$$

The basic reproduction number R_0 is the spectral radius (dominant eigenvalue) of the next-generation matrix G.

$G = F \cdot V^{-1}$ as

$$G = \begin{bmatrix} \frac{\beta bS\,(-1+\theta)\,b}{(-b\,a+\gamma\pi)\,(1+\mu)} & -\frac{\beta bS\,b}{-b\,a+\gamma\pi} & -\frac{\beta bS\,\pi}{-b\,a+\gamma\pi} & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix}$$

$\psi = G - zI = 0$ as

$$\psi = \begin{bmatrix} \frac{\beta bS\,(-1+\theta)\,b}{(-b\,a+\gamma\pi)\,(1+\mu)} - z & -\frac{\beta bS\,b}{-b\,a+\gamma\pi} & -\frac{\beta bS\,\pi}{-b\,a+\gamma\pi} & 0 \\ 0 & -z & 0 & 0 \\ 0 & 0 & -z & 0 \\ 0 & 0 & 0 & -z \end{bmatrix}$$

The determinant is given by

$$-\left(\frac{\beta bS\,(-1+\theta)\,b}{(-b\,a+\gamma\pi)\,(1+\mu)} - z\right) z^3$$

Solving for z, we have

$$z_1 = 0,\ z_2 = 0,\ z_3 = 0,\ z_4 = \frac{\beta bS(-1+\theta)b}{(-ba+\gamma\pi)(1+\mu)}$$

From the structure, the eigenvalues are the entries:

$$R_0 = \frac{\beta bS(-1+\theta)b}{(-ba+\gamma\pi)(1+\mu)}$$

4. Discussion of Results

A comprehensive analysis of the Modified HIV Model (equation 2.1), emphasizing theoretical proofs and numerical illustrations to validate the model's behavior. The analytical exploration begins with the examination of the existence and uniqueness of the solution, where Theorem 4.1 confirms that the model possesses a unique solution within a defined domain. This foundational result ensures that the model is well-posed and reliable for further analytical and numerical investigation. The proof, based on differential inequalities and bounded growth of solutions, validates that solutions remain within a biologically meaningful region.

The positivity and boundedness of the solutions were established next, a critical step in epidemiological modeling. It confirms that each compartment (e.g., susceptible, exposed, infected, etc.) maintains non-negative values throughout the simulation. Negative populations have no physical meaning, so proving positivity via separation of variables and integration ensures biological relevance. The exponential nature of the solution form shows how population levels evolve over time while remaining within realistic limits. The model further guarantees that the spread of HIV and related interventions can be accurately studied without risk of computational anomalies such as negative or infinite values.

In evaluating the Disease-Free Equilibrium (DFE), the system is linearized under the condition that no infection is present in the population. This theoretical state helps to analyze the stability of the model when no HIV exists. From this, the Endemic Equilibrium (EE) is derived, where the disease persists at a steady state. This equilibrium point is critical in determining whether HIV will die out or become permanently established. These equilibrium analyses form the basis for calculating the basic reproduction number R_0.

The basic reproduction number R_0 is derived using the next-generation matrix method, which captures how new infections propagate from existing ones. This method involves computing Jacobian matrices of new infection and transition terms and taking the spectral radius of their product.

5. Summary and Conclusion

This study developed and analyzed a modified mathematical model for HIV transmission dynamics, incorporating treatment and removal as active control strategies. Building upon the foundational work of Odebiyu et al. (2024), the model introduced additional compartments for exposed individuals, asymptomatic and symptomatic infections, treatment and removed classes. The primary aim was to offer a more comprehensive depiction of HIV progression and control dynamics.

The research began by establishing the existence and uniqueness of the solutions of the model, positivity of the model's solutions, ensuring all population compartments (susceptible, exposed, infected, treated, etc.) remained non-negative over time. The Disease-Free Equilibrium (DFE) was then derived, indicating a steady state where HIV is absent from the population and endemic equilibrium point. The local stability of this equilibrium was shown to depend on the basic reproduction number, which was computed using the Next Generation Matrix method.

The results of this study affirm the effectiveness of incorporating treatment and removal as control mechanisms in HIV modeling. Through mathematical formulation and analysis, the modified model provides insights into how early detection, treatment adherence and removing individuals from the infectious pool through recovery or death can reduce the spread of HIV.

6. Suggestions for Further Studies

To enhance and refine the findings of this study, future research could consider the following directions:

1) Extending the model to include co-infections such as HIV-TB or HIV-syphilis dynamics.

2) Incorporating stochastic elements to capture uncertainties in transmission rates and treatment response.

3) Calibrating the model with real-world data from high-prevalence areas to improve the predictive power.

4) Exploring cost-effectiveness analyses to determine optimal allocation of limited healthcare resources.

5) Modeling the impact of emerging technologies such as long-acting injectable and HIV vaccines when they become available.

Declarations

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Competing Interests Statement

The authors declare that they have no competing interests related to this work.

Consent for publication

The authors declare that they consented to the publication of this study.

Authors' contributions

All the authors took part in literature review, analysis, and manuscript writing equally.

Availability of data and materials

Supplementary information is available from the authors upon reasonable request.

Institutional Review Board Statement

Not applicable for this study.

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Not applicable for this study.

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