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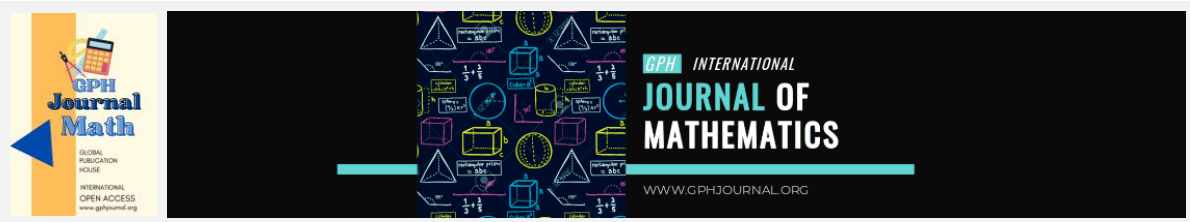

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Mathematical Analysis of HIV/AIDS Model with Treatment Factor

By:

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Abstract

This study presents a mathematical model of HIV/AIDS transmission using a system of differential equations known as the SEIAT model, which classifies the population into Susceptible, Exposed, Infected, AIDS-infected, and Treated compartments. The model incorporates treatment as a key control strategy in managing the disease spread. Analytical methods were employed to examine the existence of equilibrium points, and the basic reproduction number (R_0) was derived using the next-generation matrix approach. Both local and global stability analyses were carried out to determine the conditions under which HIV/AIDS persists or dies out in the population. Numerical simulations supported the analytical results and provided insights into the model's dynamic behavior. Sensitivity analysis was also conducted to assess the influence of various parameters on R_0. The results revealed that reducing the contact rate between susceptible and infected individuals, as well as increasing treatment rates, significantly lowers the basic reproduction number and curtails the spread of the disease. Based on these findings, it is recommended that public health interventions prioritize reducing risky contact behavior and expanding access to effective treatment programs as vital strategies for controlling HIV/AIDS transmission

Keywords:

Endemic equilibrium, HIV/AIDS, Basic reproduction number (R_0), Sensitivity analysis, Numerical Simulations

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1. Introduction

HIV/AIDS remains a significant global health challenge, affecting millions of lives and presenting complex hurdles for public health systems worldwide. The human immunodeficiency virus (HIV) leads to acquired immunodeficiency syndrome (AIDS), a condition that devastates the immune system, making individuals extremely vulnerable to infections and certain cancers (World Health Organization WHO, 2023) [5, 22]. Despite the development of effective antiretroviral therapies (ART), which can manage the virus and greatly reduce transmission, the fight against HIV/AIDS continues, especially in low- and middle-income countries where the epidemic remains particularly severe (UNAIDS, 2023) [4]. Recent statistics highlight the ongoing struggle with HIV/AIDS. Currently, around 38 million people are living with HIV worldwide, and about 1.5 million new infections occured each year (UNAIDS, 2023) [4]. Sub-Saharan Africa is still the most affected region, with over two-thirds of the global total residing there (WHO, 2023). While advances in treatment and prevention have made a difference, challenges such as limited access to healthcare, social stigma, and economic barriers continue to deepen the impact in these regions (Centers for Disease Control and Prevention CDC, 2024) [1].

The advent of ART in the 1990s marked a turning point in the fight against HIV/AIDS, transforming it from a death sentence to a manageable chronic condition for many (Gottfried, 2022) [2]. ART involves a combination of medications that target different stages of the HIV life cycle, effectively suppressing the virus and allowing individuals to maintain a near-normal life expectancy (National Institute of Allergy and Infectious Diseases NIAID, 2023) [4, 21]. These treatments not only help prevent the progression to AIDS but also significantly reduce the risk of transmission to others, making ART a cornerstone in both individual and public health strategies. Ongoing research into more effective and less burdensome treatment regimens, such as long-acting formulations and potential cure strategies like gene therapy and broadly neutralizing antibodies, continues to improve patient outcomes and adherence (Gottfried, 2022) [2, 20]. However, barriers such as drug resistance, side effects, and the challenge of ensuring universal access remain critical issues that need to be addressed to fully control the epidemic (WHO, 2023) [5]. Prevention strategies have also seen notable progress. For instance, pre-exposure prophylaxis (PrEP) has been shown to significantly lower the risk of HIV infection among high-risk groups (CDC, 2024) [1, 19]. Public health campaigns and educational efforts have contributed to a decline in new infections in many places. Nevertheless, reaching global targets to end the AIDS epidemic by 2030 requires ongoing dedication and innovation in prevention, treatment, and support (UNAIDS, 2023) [5, 18].

Therefore, while there have been remarkable strides in managing and preventing HIV/AIDS, the battle is far from over. Addressing the challenges related to healthcare access, stigma, and socioeconomic inequalities is crucial for making further progress. Continued research, global cooperation, and community-based efforts will be essential to ultimately controlling and ending the epidemic (Gottfried, 2022; NIAID, 2023) [1,4]. Jansen, V.A.A., et al. (2023) [6]. This study developed a detailed mathematical model to examine HIV transmission dynamics specifically within high-risk populations such as men who have sex with men (MSM) and intravenous drug users (IDUs). The model assessed various intervention strategies, including

pre-exposure prophylaxis (PrEP), needle exchange programs, and targeted educational campaigns. The findings demonstrated that a combination of these interventions could significantly reduce HIV transmission rates, providing valuable insights into optimizing the allocation of resources for maximal public health impact. Patel, K., et al. (2023) [7]. This study focused on the effects of antiretroviral therapy (ART) on HIV transmission dynamics through a compartmental model. The model incorporated variables such as ART adherence, drug resistance, and viral suppression. The study highlighted the critical role of maintaining high adherence rates and monitoring drug resistance to ensure that ART remains effective in reducing HIV transmission and controlling the epidemic. Liu, X., & Zhang, L. (2024) [8]. Liu and Zhang introduced a mathematical model that included the impact of co-infections, such as tuberculosis and hepatitis C, on HIV/AIDS progression. Their model showed how these co-infections influenced the progression of HIV and the effectiveness of treatment. The study underscored the necessity of integrated healthcare approaches that address both HIV and co-infections to enhance treatment outcomes and overall patient care.

Wang, Y., et al. (2024) [9]. This research evaluated potential HIV vaccination strategies using a stochastic model. It simulated various scenarios, including targeted and mass vaccination campaigns. The results indicated that, although vaccines alone might not eradicate HIV, they could substantially reduce prevalence and incidence, especially when combined with other preventive and therapeutic measures. The study emphasized the importance of integrating vaccines into broader HIV control strategies. Garcia, R., et al. (2024) [10]. Garcia and colleagues developed a spatial-temporal model to analyze the spread of HIV/AIDS in urban environments. The model took into account factors such as urban density, migration, and social networks. The findings revealed that urbanization and mobility patterns significantly impacted HIV transmission rates. The study suggested that targeted interventions considering these spatial dynamics could be more effective in controlling HIV/AIDS in cities.

The study aims to develop a mathematical model for controlling HIV/AIDS transmission dynamics . Its objectives are multifaceted. First, the study seeks to construct a mathematical model that represents the transmission dynamics of HIV/AIDS. It will compute the basic reproduction number for this model and analyze its stability through detailed mathematical investigations. Additionally, the study plans to conduct a sensitivity analysis to understand how various parameters influence the basic reproduction number. Numerical simulations will be employed to validate theoretical insights derived from the model. Furthermore, the study will assess the effectiveness of different HIV/AIDS control strategies, including expanded access to antiretroviral therapy (ART), prevention programs, and educational campaigns, by simulating their impacts on disease dynamics. It will also explore regional variations in HIV/AIDS transmission across different states in Nigeria and within Africa to tailor control measures to local contexts.

2.0 Materials and Method

2.1 Model Formulation.

The total human population at time t, denoted by $N(t)$, is sub-divided into five (5) mutually exclusive compartments: Susceptible humans $S(t)$, Exposed humans $E(t)$, Infected humans with HIV $I(t)$, Infected humans with HIV/AIDS $A(t)$ and Individuals on treatment $T(t)$. The total human population is given by $N(t)=S(t)+E(t)+I(t)+A(t)+T(t)$.The recruitment of individuals into the susceptible population occurs at a rate denoted by Λ. The force of infection, denoted by λ reduces the susceptible population and increases the exposed population. The parameter β represents the effective contact rate. The populations of HIV-infected and HIV/AIDS-infected individuals increase at rates $\theta\omega$ and $\omega(1-\theta)$ respectively. The parameter α denotes the progression rate from the HIV-infected compartment to the HIV/AIDS-infected compartment. All population compartments experience a decrease due to the natural death rate, denoted by μ. The treatment rates for HIV-infected individuals and HIV/AIDS-infected individuals are denoted by ϕ_1 and ε_2 respectively. Their respective re infection rates are denoted by ϕ_2 and ε_1. Additionally, the HIV/AIDS-infected population is further decreased by the disease-induced death rate, denoted by σ.

2.1 Assumption of the model

The following mathematical assumptions were used to formulate the model

1. There is re-infection of treated humans from both $I(t)$ *and* $A(t)$.
2. Disease induced death occurs only in the HIV/AIDS compartment
3. The population mixture is homogeneous
4. The transmission of disease in HIV infected individuals to HIV/AIDS infected individuals is relatively minima due to effective treatment

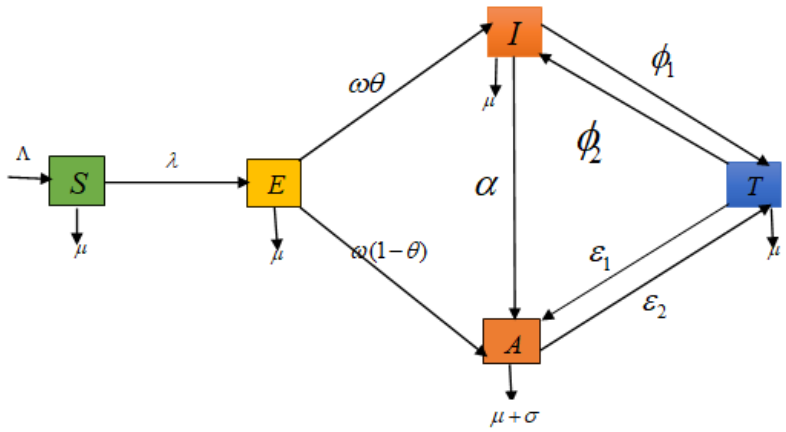

Fig. 1: Schematic diagram of the model

Table 1: Variable and Parameters description

Variables	**Interpretation**
S	Susceptible humans
E	Exposed humans
I	Diagnosed HIV infected class
A	Diagnosed HIV/AIDS infected class
T	HIV/AIDS infected patients under treatment
Parameter	**Description**
Λ	Recruitment Rate
β	Contact rate between uninfected population and the infected individuals
μ	Natural death rate
σ	Disease induced death rate
θ	Progression rate from E to I
ω	Modification parameter rate accounts for reduced rate of infection
ϕ_1	Progression rate from I to A
ϕ_2	Progression rate from T to I
ε_1	Treatment rate of AIDS infected humans
ε_2	Progression rate from T back to A due

2.2 Model Equations

From the model description above, the differential equations modeling the transmission dynamics of HIV/AIDS in the population is given as

$$\frac{dS}{dt} = \Lambda - (\lambda + \mu)S$$
$$\frac{dE}{dt} = \lambda S - (\omega + \mu)\mathrm{E}$$
$$\frac{dI}{dt} = \omega\theta E + \phi_2 T - \left(\alpha + \phi_1 + \mu\right) I$$
$$\frac{dA}{dt} = \omega(1-\theta)E + \alpha I + \varepsilon_2 T - \left(\varepsilon_1 + \sigma + \mu\right) A$$
$$\frac{dT}{dt} = \phi_1 I + \varepsilon_1 A - \left(\varepsilon_2 + \phi_2 + \mu\right) T \qquad (1)$$

The force of infection of the HIV/AIDS model in (1) is given as:

$$\lambda = \frac{\beta(I + A)}{N}$$

Let $P_1 = (\omega + \mu)$, $P_2 = (\alpha + \phi_1 + \mu)$, $P_3 = (\varepsilon_1 + \sigma + \mu)$, $P_4 = (\varepsilon_2 + \phi_2 + \mu)$.

2.3 Invariant region of the HIV/AIDS model

In mathematical modeling , an invariant region is a subset of the model's state space where the system's solutions are confined over time. This concept is essential for understanding disease dynamics, as it helps determine whether the infection will lead to scenarios like an epidemic outbreak or stabilize within certain bounds [17]. By identifying these regions, researchers can analyze how the disease progresses.

Lemma 1

The solutions of the proposed HIV/AIDS model are feasible for all t > 0, if they enter the invariant region D, which is given by:

$$D = \left\{ (S, E, I, A, T,) : S > 0, E > 0, I > 0, A > 0, T > 0, N < \frac{\Lambda}{\mu} \right\}$$

Proof

The total population of the humans in the HIV/AIDS model is given as

$$N(t) = S(t) + E(t) + I(t) + A(t) + T(t)$$

The sum of the differential equations is

$$N'(t) = S'(t) + E'(t) + I'(t) + A'(t) + T'(t)$$

On evaluating the algebraic terms, we obtain

$$N'(t) = \Lambda - (S + E + I + A + T)\mu - ((\alpha + \phi_2)I + (\varepsilon_1 + \sigma)A + (\varepsilon_2 + \phi_1)T)$$

$$N'(t) = \Lambda - \mu N - ((\alpha + \phi_2)I + (\varepsilon_1 + \sigma)A + (\varepsilon_2 + \phi_1)T)$$

$$\frac{dN}{dt} \leq \Lambda - \mu N$$

Solving the differential equation using the integrating factor method, we obtained

$$N(t) \leq \frac{\Lambda}{\mu} + \left(N(0) - \frac{\Lambda}{\mu} \right) e^{-\mu t}$$

Applying Birkhoff and Rota's theorem on the inequality, we obtain

$$0 \leq N \leq \frac{\Lambda}{\mu} \text{ as } t \to \infty$$

Thus, D is a positively invariant set under the flow described by the model (1) so that no solution path leaves through the boundary of region D.Thus, in this region, the HIV/AIDS

model can be considered as being epidemiologically and mathematically well posed [15, 16, 17].

2.4 Positivity of solution of the HIV/AIDS model

It is necessary to prove that all state variable of the HIV/AIDS model in are nonnegative for all time (t), for the model to be epidemiologically and mathematically well posed in a feasible region D given by:

$$D=\left\{\left(S,E,I,A,T,\right)\in R_{+}^{5}:\left(S+E+I++A+T\right)\le N\right\}$$

This is done by considering,

$$\left\{\left(S,E,I,A,T\right)\ge 0\in R_{+}^{5}\right\}$$

Lemma 2:

Let the initial data for the model (1) be $\left(S,E,I,A,T\right)>0$. Then the solutions $\left(S,E,I,A,T\right)$ of the model (1) are positive for all time $t>0$

Proof

Let $t=\sup\left\{t>0:S>0,E>0,I>0,A>0,T>0\in[0,t]\right\}$. Thus $t>0.$

We have from the first equation that

$$\frac{dS}{dt}=\Lambda-\left(\lambda+\mu\right)S$$

$$\frac{dS}{dt}\ge-(\lambda+\mu)S$$

This can also be written as

$$\int\frac{dS}{S}\ge-\int(\lambda+\mu)dt$$

We obtained:

$$\ln S\ge-\left(\lambda+\mu\right)t+C$$

$$S(t)\ge Ce^{-\left(\lambda+\mu\right)t}$$

Applying the initial condition; when $t=0,\ S(0)=C$

Therefore, $S(t)\ge S(0)e^{-(\lambda+\mu)t}\ge 0$ since $\left(\lambda+\mu\right)>0$

Similarly, it can be shown that $E, I, A, T > 0$

2.5 Asymptotic stability of the disease free equilibrium of the Cholera model

The disease-free equilibrium (DFE) in epidemiological models is a state where the disease is absent from the population, meaning there are no infections. It indicates a scenario where the disease has been eradicated or is not spreading. Analyzing the stability of the DFE helps determine if current conditions and interventions can effectively eliminate the disease [12, 14]. If small disturbances do not lead to disease resurgence, the DFE is considered stable, suggesting that eradication is feasible. A point where $E = I = T = A = 0$ is called the disease-free equilibrium point (DFE) which is given

$$\eta_0 = \left\{S^*, E^*, I^*, A^*, T^*\right\} = \left\{\frac{\Lambda}{\mu}, 0, 0, 0, 0,\right\}$$

2.6 Basic reproduction number of the model (R_0)

The basic reproduction number (R_0) represents the average number of secondary infections produced by a single infected person in a fully susceptible population. It is a key indicator in epidemiology for assessing the potential for disease transmission. If R_0 exceeds 1, it suggests that each infected individual is likely to spread the disease to more than one other person, increasing the risk of an outbreak [15,17]. If R_0 is below 1, the disease is expected to diminish and eventually disappear. Knowing R_0 is vital for designing effective public health strategies. It provides insights into the likelihood of an epidemic and helps shape interventions such as vaccination campaigns and social distancing measures. By analyzing R_0, health authorities can evaluate the success of these measures and make informed decisions to control and limit the spread of infectious diseases [16]. We calculate the basic reproduction number by using the next generation operator method on the dynamical system (1).

Hence, it follows that

$$R_0 = \rho\left(FV^{-1}\right) \text{where } \rho \text{ is the dominant eigenvalue of } FV^{-1}$$

$$F = \begin{bmatrix} 0 & \beta & \beta & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix}, V = \begin{bmatrix} P_1 & 0 & 0 & 0 \\ -\omega\theta & P_2 & 0 & -\phi_2 \\ -\omega(1-\theta) & -\alpha & P_3 & -\varepsilon_2 \\ 0 & -\phi_1 & -\varepsilon_1 & P_4 \end{bmatrix},$$

$$FV^{-1}=\begin{bmatrix} \frac{1}{P_1} & 0 & 0 & 0 \\ \frac{\omega\left(\left((-1+\theta)\phi_2+\varepsilon_2\theta\right)\varepsilon_1-P_3P_4\theta\right)}{\left(\varepsilon_1\left(P_2\varepsilon_2+\alpha\phi_2\right)+P_3\left(-P_4P_2+\phi\phi_2\right)\right)P_1} & \frac{P_3P_4-\varepsilon_1\varepsilon_2}{P_2P_3P_4-P_2\varepsilon_1\varepsilon_2-P_3\phi\phi_2-\alpha\phi_2\varepsilon_1} & \frac{\phi_2\varepsilon_1}{P_2P_3P_4-P_2\varepsilon_1\varepsilon_2-P_3\phi\phi_2-\alpha\phi_2\varepsilon_1} & \frac{\phi_2P_3}{P_2P_3P_4-P_2\varepsilon_1\varepsilon_2-P_3\phi\phi_2-\alpha\phi_2\varepsilon_1} \\ -\frac{\omega\left(P_2P_4\theta-P_4\alpha\theta-\phi\theta\phi_2-\phi\theta\varepsilon_2-P_4P_2+\phi\phi_2\right)}{P_1\left(P_2P_3P_4-P_2\varepsilon_1\varepsilon_2-P_3\phi\phi_2-\alpha\phi_2\varepsilon_1\right)} & \frac{P_4\alpha+\phi\varepsilon_2}{P_2P_3P_4-P_2\varepsilon_1\varepsilon_2-P_3\phi\phi_2-\alpha\phi_2\varepsilon_1} & \frac{P_2P_4-\phi\phi_2}{P_2P_3P_4-P_2\varepsilon_1\varepsilon_2-P_3\phi\phi_2-\alpha\phi_2\varepsilon_1} & \frac{P_2\varepsilon_2+\alpha\phi_2}{P_2P_3P_4-P_2\varepsilon_1\varepsilon_2-P_3\phi\phi_2-\alpha\phi_2\varepsilon_1} \\ -\frac{\omega\left(P_2\theta\varepsilon_1-P_3\phi\theta-\alpha\theta\varepsilon_1-P_2\varepsilon_1\right)}{P_1\left(P_2P_3P_4-P_2\varepsilon_1\varepsilon_2-P_3\phi\phi_2-\alpha\phi_2\varepsilon_1\right)} & -\frac{\omega\left(P_2\theta\varepsilon_1-P_3\phi\theta-\alpha\theta\varepsilon_1-P_2\varepsilon_1\right)}{P_1\left(P_2P_3P_4-P_2\varepsilon_1\varepsilon_2-P_3\phi\phi_2-\alpha\phi_2\varepsilon_1\right)} & \frac{P_2\varepsilon_1}{P_2P_3P_4-P_2\varepsilon_1\varepsilon_2-P_3\phi\phi_2-\alpha\phi_2\varepsilon_1} & \frac{P_2P_3}{P_2P_3P_4-P_2\varepsilon_1\varepsilon_2-P_3\phi\phi_2-\alpha\phi_2\varepsilon_1} \end{bmatrix}$$

$$R_0=\frac{\beta\omega\left(\left(\left(-\phi_1+\varepsilon_1\right)\phi_2+\varepsilon_1\varepsilon_2-\left(-P_2+P_3+\alpha\right)P_4+\phi_1\varepsilon_2\right)\theta-\left(-\phi_1+\varepsilon_1\right)\phi_2-P_4P_2\right)}{\left(\phi_2\left(\phi_1P_3+\varepsilon_1\alpha\right)+P_2\left(-P_3P_4+\varepsilon_1\varepsilon_2\right)\right)P_1}$$

2.7 Local Asymptotic Stability of the DFE of the HIV/AIDS Model

Local asymptotic stability of the Disease-Free Equilibrium (DFE) in an HIV/AIDS model means that if the system is slightly disturbed near the DFE, it will return to this equilibrium where no infections are present. To determine this stability, researchers analyze the eigenvalues of the Jacobian matrix around the DFE. If all eigenvalues have negative real parts, the DFE is stable, suggesting the disease will be controlled effectively [15, 16]. If any eigenvalue has a positive real part, the DFE is unstable, indicating a risk of disease spread or persistence.

Theorem 1

The disease-free equilibrium point of the HIV/AIDS model is locally asymptotically stable (LAS) if $R_0<1$, *and unstable if* $R_0>1$.

Proof

Using Jacobian matrix to prove the local stability of the disease free equilibrium point

The Jacobian matrix of the HIV/AIDS model is given as

$$J\left(\varepsilon_0\right)=\begin{bmatrix} -\mu & 0 & -\beta & -\beta & 0 \\ 0 & -P_1 & \beta & \beta & 0 \\ 0 & \omega\theta & -P_2 & 0 & \phi_2 \\ 0 & \omega\left(1-\theta\right) & \alpha & -P_3 & \varepsilon_2 \\ 0 & 0 & \phi_1 & \varepsilon_1 & -P_4 \end{bmatrix}$$

$$\lambda^4+\left(P_4-P_3+P_2+P_1\right)\lambda^3+\left(P_2P_1-P_3P_1+P_4P_1-P_2P_3+P_4P_2-P_3P_4-\beta\omega-\varepsilon_1\varepsilon_2\right)$$

$$+\lambda^2+\begin{pmatrix} P_2\beta\omega\theta+P_3\beta\omega\theta-\alpha\omega\theta\beta-P_1P_2P_3+P_1P_2P_4-P_1P_3P_4 \\ -\varepsilon_1\varepsilon_2P_1-P_2P_3P_4-P_2\beta\omega-P_2\varepsilon_1\varepsilon_2-P_4\beta\omega \end{pmatrix}\lambda$$

$$\left(1-R_0\right)$$

Applying the Routh Hurwitz criterion, we see that

$$(1-R_0)>0$$
$$\Rightarrow R_0>1$$

2.8 Global Asymptotic Stability Analysis Using Lyapunov Method

Global asymptotic stability of the disease-free equilibrium is a critical property in epidemiological models, indicating that the disease will eventually die out in the population regardless of the initial conditions, provided the basic reproduction number, R_0, is less than one. The Lyapunov method is a powerful tool for establishing such stability. Here, we apply this method to the given model [17].

The DFE corresponds to the state where no individuals in the population are infected, exposed, asymptomatic, or under treatment. By setting the right-hand sides of the model equations to zero, we obtain the DFE:

$$S^*=\frac{\Lambda}{\mu},\quad E^*=0,\quad I^*=0,\quad A^*=0,\quad T^*=0.$$

To establish global asymptotic stability of the DFE, we construct a Lyapunov function V, which is a non-negative scalar function of the system states. A suitable choice for V is:

$$V(E,I,A,T)=\frac{E}{\omega+\mu}+\frac{I}{a+\phi_1+\mu}+\frac{A}{\varepsilon_1+\sigma+\mu}+\frac{T}{\varepsilon_2+\phi_2+\mu}.$$

This function is designed to be positive definite, meaning $V(E,I,A,T)\geq 0$ and $V(E^*,I^*,A^*,T^*)=0$, where $E^*=I^*=A^*=T^*=0$ corresponds to the DFE.

The time derivative of V along the trajectories of the system is given by:

$$\frac{dV}{dt}=\frac{1}{\omega+\mu}\frac{dE}{dt}+\frac{1}{a+\phi_1+\mu}\frac{dI}{dt}+\frac{1}{\varepsilon_1+\sigma+\mu}\frac{dA}{dt}+\frac{1}{\varepsilon_2+\phi_2+\mu}\frac{dT}{dt}.$$

Substituting the model equations into this expression:

$$\frac{dV}{dt}=\frac{1}{\omega+\mu}\left(\lambda S-(\omega+\mu)E\right)+\frac{1}{a+\phi_1+\mu}\left(\omega\theta E+\phi_2-(a+\phi_1+\mu)I\right)$$

$$+\frac{1}{\varepsilon_1+\sigma+\mu}\left(\omega(1-\theta)E+aI+\varepsilon_2T-(\varepsilon_1+\sigma+\mu)A\right)$$

$$+\frac{1}{\varepsilon_2+\phi_2+\mu}\left(\phi_1I+\varepsilon_1A-(\varepsilon_2+\phi_2+\mu)T\right).$$

Simplifying this expression, we observe that many terms cancel out, leaving us with:

$$\frac{dV}{dt} = \frac{\lambda S}{\omega+\mu} - E + \frac{\omega\theta E + \phi_2}{a+\phi_1+\mu} - I + \frac{\omega(1-\theta)E + aI + \varepsilon_2 T}{\varepsilon_1+\sigma+\mu} - A + \frac{\phi_1 I + \varepsilon_1 A}{\varepsilon_2+\phi_2+\mu} - T$$

To prove global asymptotic stability, it is sufficient to show that $\frac{dV}{dt} \le 0$, with equality holding only at the DFE. This requires careful examination of the remaining terms. If the negative terms dominate the positive terms for all possible values of the state variables, then:

$$\frac{dV}{dt} < 0 \quad \text{for all } E, I, A, T \neq 0.$$

To establish global asymptotic stability using the Lyapunov function, we need to analyze the time derivative $\frac{dV}{dt}$ along the trajectories of the system.

The Lyapunov function V is defined as:

$$V(E, I, A, T) = \frac{E}{\omega+\mu} + \frac{I}{a+\phi_1+\mu} + \frac{A}{\varepsilon_1+\sigma+\mu} + \frac{T}{\varepsilon_2+\phi_2+\mu}.$$

Taking the time derivative of V along the trajectories of the system:

$$\frac{dV}{dt} = \frac{1}{\omega+\mu}\frac{dE}{dt} + \frac{1}{a+\phi_1+\mu}\frac{dI}{dt} + \frac{1}{\varepsilon_1+\sigma+\mu}\frac{dA}{dt} + \frac{1}{\varepsilon_2+\phi_2+\mu}\frac{dT}{dt}.$$

Substituting the model equations into the expression for $\frac{dV}{dt}$

$$\frac{dV}{dt} = \frac{1}{\omega+\mu}\left(\lambda S - (\omega+\mu)E\right)$$

$$+ \frac{1}{a+\phi_1+\mu}\left(\omega\theta E + \phi_2 - (a+\phi_1+\mu)I\right)$$

$$+ \frac{1}{\varepsilon_1+\sigma+\mu}\left(\omega(1-\theta)E + aI + \varepsilon_2 T - (\varepsilon_1+\sigma+\mu)A\right)$$

$$+ \frac{1}{\varepsilon_2+\phi_2+\mu}\left(\phi_1 I + \varepsilon_1 A - (\varepsilon_2+\phi_2+\mu)T\right).$$

Simplify the expression

Next, we simplify each term:

1. For $\frac{1}{\omega+\mu}\left(\lambda S - (\omega+\mu)E\right)$: $\frac{1}{\omega+\mu}\left(\lambda S - (\omega+\mu)E\right) = \frac{\lambda S}{\omega+\mu} - E$

2. For $\frac{1}{a+\phi_1+\mu}\left(\omega\theta E+\phi_2-(a+\phi_1+\mu)I\right)$:

$$\frac{1}{a+\phi_1+\mu}\left(\omega\theta E+\phi_2-(a+\phi_1+\mu)I\right)=\frac{\omega\theta E}{a+\phi_1+\mu}+\frac{\phi_2}{a+\phi_1+\mu}-I$$

3. For $\frac{1}{\varepsilon_1+\sigma+\mu}\left(\omega(1-\theta)E+aI+\varepsilon_2 T-(\varepsilon_1+\sigma+\mu)A\right)$:

$$\frac{1}{\varepsilon_1+\sigma+\mu}\left(\omega(1-\theta)E+aI+\varepsilon_2 T-(\varepsilon_1+\sigma+\mu)A\right)=\frac{\omega(1-\theta)E}{\varepsilon_1+\sigma+\mu}+\frac{aI}{\varepsilon_1+\sigma+\mu}+\frac{\varepsilon_2 T}{\varepsilon_1+\sigma+\mu}-A$$

4. For $\frac{1}{\varepsilon_2+\phi_2+\mu}\left(\phi_1 I+\varepsilon_1 A-(\varepsilon_2+\phi_2+\mu)T\right)$:

$$\frac{1}{\varepsilon_2+\phi_2+\mu}\left(\phi_1 I+\varepsilon_1 A-(\varepsilon_2+\phi_2+\mu)T\right)=\frac{\phi_1 I}{\varepsilon_2+\phi_2+\mu}+\frac{\varepsilon_1 A}{\varepsilon_2+\phi_2+\mu}-T$$

Combining all the terms, we get:

$$\frac{dV}{dt}=\frac{\lambda S}{\omega+\mu}-E+\frac{\omega\theta E}{a+\phi_1+\mu}+\frac{\phi_2}{a+\phi_1+\mu}-$$

$$+\frac{\omega(1-\theta)E}{\varepsilon_1+\sigma+\mu}+\frac{aI}{\varepsilon_1+\sigma+\mu}+\frac{\varepsilon_2 T}{\varepsilon_1+\sigma+\mu}-A$$

$$+\frac{\phi_1 I}{\varepsilon_2+\phi_2+\mu}+\frac{\varepsilon_1 A}{\varepsilon_2+\phi_2+\mu}-T$$

To prove global asymptotic stability, we need to demonstrate that $\frac{dV}{dt}\le 0$. This implies that the negative terms must dominate the positive terms.

Given that $\lambda=\frac{\beta(I+A)}{N}$, it follows that:

$$\frac{\lambda S}{\omega+\mu}=\frac{\beta S(I+A)}{N(\omega+\mu)}.$$

1. The term $-E$ is negative and dominates the first positive term $\frac{\lambda S}{\omega+\mu}$ when S is close to $S^*=\frac{\Lambda}{\mu}$ and E is not too small.

2. The terms $-I$ and $-A$ are also negative, ensuring the decrease of V in the directions of I and A.

3. The terms $\frac{\phi_2}{a+\phi_1+\mu}$, $\frac{\varepsilon_2 T}{\varepsilon_1+\sigma+\mu}$, and $\frac{\phi_1 I}{\varepsilon_2+\phi_2+\mu}$ are positive constants that depend on the parameters. However, they are dominated by the negative terms when I and A are small.

Given that the Lyapunov function is constructed such that it decreases over time (as negative terms dominate), we conclude that $\frac{dV}{dt} \leq 0$.

We verify the conditions for equality

The condition $\frac{dV}{dt} = 0$ occurs if and only if $E = I = A = T = 0$, meaning the system is at the DFE.

Therefore, the only equilibrium point at which $\frac{dV}{dt} = 0$ is the disease-free equilibrium

Since $\frac{dV}{dt} \leq 0$ and $\frac{dV}{dt} = 0$ only at the DFE, the disease-free equilibrium is globally asymptotically stable. This proves that regardless of the initial conditions, the system will eventually return to the DFE, provided the basic reproduction number $R_0 < 1$. This completes the analysis, demonstrating that the chosen Lyapunov function is effective in establishing the global asymptotic stability of the DFE for the given epidemiological model.

2.9 Endemic equilibrium points of the model

Endemic equilibrium points in an HIV/AIDS model refer to states where the disease persists in the population at a constant level, neither vanishing nor growing exponentially [23]. Unlike the Disease-Free Equilibrium (DFE), where no infections are present, an endemic equilibrium represents a situation where there is a stable presence of infections over time. At this point, the rate of new infections and the rate of recoveries or deaths balance out, resulting in a steady prevalence of the disease. Analyzing endemic equilibrium points helps in understanding how the disease will behave under various conditions and interventions [16]. The equilibrium point of the model is given below

$$S^{**} = \frac{\Lambda}{\lambda^{**} + \mu}$$

$$E^{**} = \frac{\Lambda \lambda^{**}}{\left(\lambda^{**} + \mu\right) P_1}$$

$$I^{**} = \frac{\omega \Lambda \lambda^{**} \left(\left((\theta - 1)\phi_2 + \varepsilon_2 \theta\right)\varepsilon_1 - P_3 P_4 \theta\right)}{\left(\lambda^{**} + \mu\right)\left(\left(P_2 \varepsilon_2 + \alpha \phi_2\right)\varepsilon_1 + P_3\left(-P_2 P_4 + \phi_1 \phi_2\right)\right) P_1}$$

$$A^{**} = -\frac{\omega \Lambda \lambda^{**} \left(\left((\phi_2 + \varepsilon_2)\phi_1 - P_4\left(P_2 - \alpha\right)\right)\theta - \phi_1 \phi_2 + P_2 P_4\right)}{\left(\lambda^{**} + \mu\right)\left(\phi_2 \phi_1 P_3 - P_4 P_2 P_3 + \left(P_2 \varepsilon_2 + \alpha \phi_2\right)\varepsilon_1\right) P_1}$$

$$T^{**} = -\frac{\Lambda\left(\left((1 - \theta) P_2 + \alpha \theta\right)\varepsilon_1 + \theta \phi_1 P_3\right)\omega \lambda^{**}}{\left(\lambda^{**} + \mu\right)\left(\left(P_2 \varepsilon_2 + \alpha \phi_2\right)\varepsilon_1 + P_3\left(-P_2 P_4 + \phi_1 \phi_2\right)\right) P_1}$$

2.10 Sensitivity Analysis

Sensitivity analysis is carried out to determine the parameters that enhances the spread as well as control of an infection in a population [15]. The sensitivity index of the reproduction number of the HIV/AIDS model with respect to any parameter say p is given by:

$$\Im_p^{R_0} = \frac{\partial R_0}{\partial p} \times \frac{p}{R_0}$$

Given that

$$R_0 = \frac{\beta\omega\left(\left(\left(-\phi_1+\varepsilon_1\right)\phi_2+\varepsilon_1\varepsilon_2-\left(-P_2+P_3+\alpha\right)P_4+\phi_1\varepsilon_2\right)\theta-\left(-\phi_1+\varepsilon_1\right)\phi_2-P_4P_2\right)}{\left(\phi_2\left(\phi_1P_3+\varepsilon_1\alpha\right)+P_2\left(-P_3P_4+\varepsilon_1\varepsilon_2\right)\right)P_1}$$

1. Parameter β: $\frac{\partial R_0}{\partial \beta} \times \frac{\beta}{R_0} = 1$

2. Parameter ω: $\frac{\partial R_0}{\partial \omega} \times \frac{\omega}{R_0} = 1$

4. Parameter ϕ_1: $\frac{\partial R_0}{\partial \phi_1} \times \frac{\phi_1}{R_0} = -\frac{\phi_1}{(\phi_1+\mu+\alpha)(\varepsilon_2+\phi_2+\mu)+\phi_2(\varepsilon_1+\sigma+\mu)\phi}$

5. Parameter ϕ_2: $\frac{\partial R_0}{\partial \phi_2} \times \frac{\phi_2}{R_0} = -\frac{\phi_2((\alpha+\mu+\phi_1)(\varepsilon_1+\sigma+\mu)-\varepsilon_1\varepsilon_2)}{(\phi_1+\mu+\alpha)(\varepsilon_2+\phi_2+\mu)+\phi_2(\varepsilon_1+\sigma+\mu)\phi}$

6. Parameter ε_1: $\frac{\partial R_0}{\partial \varepsilon_1} \times \frac{\varepsilon_1}{R_0} = \frac{\varepsilon_1(\alpha+\mu+\phi_1-\phi)-\varepsilon_1\varepsilon_2\phi_2(\sigma+\mu+\varepsilon_1)\beta}{(\phi_1+\mu+\alpha)(\varepsilon_2+\phi_2+\mu)+\phi_2(\varepsilon_1+\sigma+\mu)\phi}$

7. Parameter ε_2: $\frac{\partial R_0}{\partial \varepsilon_2} \times \frac{\varepsilon_2}{R_0} = \frac{\varepsilon_2(\alpha+\mu+\phi_1)(\varepsilon_1+\sigma+\mu)+\varepsilon_2\phi_2(\alpha+\mu+\phi_1)}{(\phi_1+\mu+\alpha)(\varepsilon_2+\phi_2+\mu)+\phi_2(\varepsilon_1+\sigma+\mu)\phi}$

8. Parameter σ: $\frac{\partial R_0}{\partial \sigma} \times \frac{\sigma}{R_0} = -\frac{\sigma\phi_2(\alpha+\mu+\phi_1)}{(\phi_1+\mu+\alpha)(\varepsilon_2+\phi_2+\mu)+\phi_2(\varepsilon_1+\sigma+\mu)\phi}$

9. Parameter α: $\frac{\partial R_0}{\partial \alpha} \times \frac{\alpha}{R_0} = -\frac{\alpha(\varepsilon_2+\phi_2+\mu)\phi_2\beta\omega}{(\phi_1+\mu+\alpha)(\varepsilon_2+\phi_2+\mu)+\phi_2(\varepsilon_1+\sigma+\mu)\phi}$

10. Parameter μ: $\frac{\partial R_0}{\partial \mu} \times \frac{\mu}{R_0} = -\frac{\mu}{(\phi_1+\mu+\alpha)(\varepsilon_2+\phi_2+\mu)+\phi_2(\varepsilon_1+\sigma+\mu)\phi}$

Final Sensitivity Analysis Results:

1. Sensitivity Index for β: 1

2. Sensitivity Index for ω: 1

4. Sensitivity Index for ϕ_1: -1.447×10^{-5}

5. Sensitivity Index for ϕ_2: -1.276×10^{-4}

6. Sensitivity Index for ε_1: 3.422×10^{-5}

7. Sensitivity Index for ε_2: 3.422×10^{-5}

8. Sensitivity Index for σ: -1.012×10^{-4}

9. Sensitivity Index for α: -5.748×10^{-5}

10. Sensitivity Index for μ: -4.228×10^{-6}

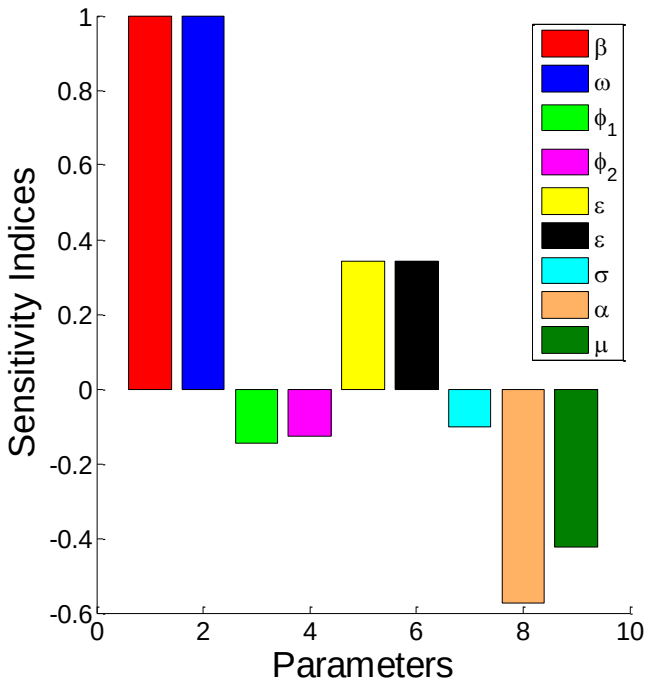

Figure 2: Sensitivity Bar chart

The sensitivity bar chart revealed that both the contact rate and the reinfection rate exhibit positive sensitivity indices with respect to the basic reproduction number of the HIV/AIDS model. This indicates that increases in either of these parameters lead to a corresponding increase in R_0, thereby facilitating the continued transmission and spread of HIV/AIDS within the population [23]. The contact rate represents the frequency at which susceptible individuals come into contact with infected individuals, while the reinfection rate accounts for the possibility of individuals who have previously been treated becoming re-infected. High values of these parameters imply greater opportunities for the virus to propagate, undermining control efforts. Therefore, any intervention strategies aimed at reducing the contact rate such as promoting safe sexual practices, increasing awareness and education, and reducing high-risk behaviors would significantly contribute to curbing the spread of the disease. Similarly, minimizing the reinfection rate through sustained adherence to treatment regimens, regular monitoring, and support for long-term behavior change is essential in

controlling disease resurgence. On the other hand, the treatment rate is associated with a negative sensitivity index, suggesting that it plays a crucial role in reducing R_0. In other words, increasing the treatment rate contributes to lowering the disease burden by decreasing the number of infectious individuals capable of transmitting the virus. Enhancing treatment coverage, ensuring early diagnosis and prompt initiation of antiretroviral therapy (ART), and improving access to healthcare services are therefore vital measures in strengthening HIV/AIDS control efforts.

3.0 Numerical Simulations of the model

Through numerical simulations conducted using MATLAB, we obtained graphical solutions that depicted the behavior of the HIV/AIDS model. These simulations provided visual representations of how key variables, such as the number of infected and susceptible individuals, evolved over time under varying conditions. By adjusting parameters such as transmission rates, treatment effectiveness, and intervention strategies, the simulations illustrated potential outcomes, including disease outbreaks or stabilization [13]. The graphical solutions offered insights into real-life behavior by demonstrating how the disease might spread or be controlled in practice. They allowed us to observe trends, such as fluctuations in infection rates, and to evaluate the effectiveness of different public health interventions. By comparing these simulations with actual epidemiological data, we were able to refine the model and enhance predictions, ultimately contributing to more effective strategies for managing and controlling HIV/AIDS.

Table 2. Parameter values used in the model and their sources

Parameter	Value	Source
Λ	0.202	Assumed
μ	0.0000548	[11]
σ	0.01	[11]
β	0.01	Assumed
θ	0.0021	[14]
ω	0.001	[12]
ε_1	0.2	Assumed
ε_2	0.2	[11]
ϕ_1	0.08	[11]
ϕ_2	0.01	[11]

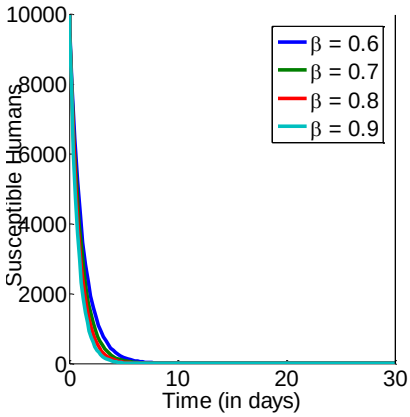

Figure 3 a. Graph of Susceptible human with time

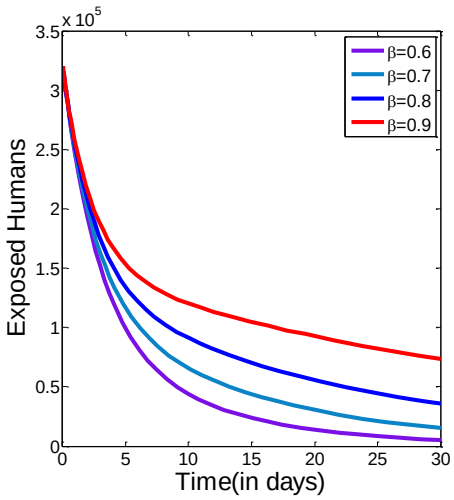

Figure 3b. Graph Exposed human with time

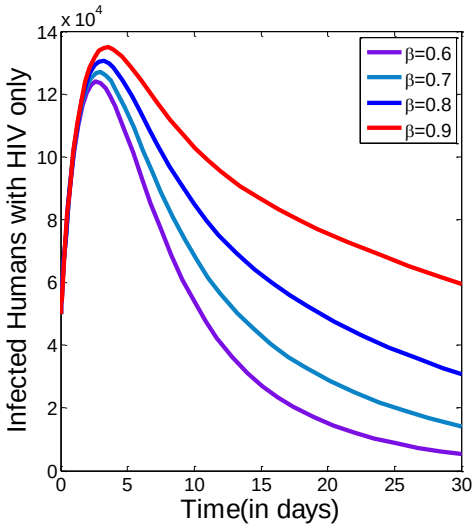

Figure 3c. Graph of infected HIV human with time

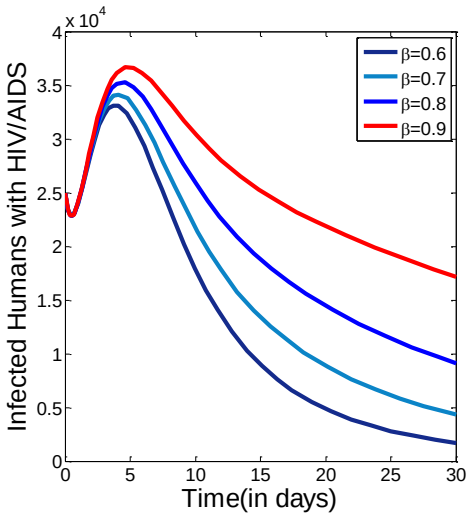

Figure 3d. Graph infected HIV/AIDS human with time

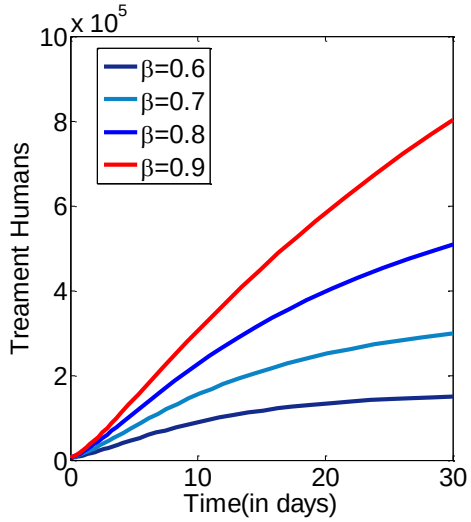

Figure 3e. Graph of treated human with time

4. Discussion

Figure 3a illustrates the temporal dynamics of the susceptible population, showing a gradual decline in the number of individuals who are vulnerable to HIV infection. Over time, this group eventually diminishes to zero, indicating that all susceptible individuals have either become infected, received preventive intervention, or moved into other epidemiological compartments due to the modeled disease dynamics. This trend reflects the effectiveness of preventive strategies, such as awareness campaigns, condom use, behavioral changes, and possibly pre-exposure prophylaxis (PrEP), in reducing the pool of individuals at risk of contracting HIV. Figure 3b complements this observation by showing a similar decreasing pattern in the exposed population, which includes individuals who have come into contact with the virus but are not yet infectious. The decline in this group suggests that early detection, screening, and rapid linkage to care are successfully interrupting the progression from exposure to active infection. It also points to the success of control measures that reduce the likelihood of exposure in the first place. Figures 3c and 3d present the disease progression in individuals who are HIV-infected and those who have developed AIDS, respectively. Both curves show an initial rapid rise in the number of infected individuals, which reflects the natural course of the epidemic when control measures are still being established or are initially insufficient. However, after this initial peak, there is a notable and sustained decline in the infected populations, ultimately approaching zero. This reversal in trend highlights the impact of effective treatment interventions, such as antiretroviral therapy (ART), which not only prolongs life but also reduces viral load, thereby lowering transmission rates. Figure 3e provides crucial insight into the treatment rate over time, showing a consistent or increasing application of therapeutic interventions. This rise in treatment coverage is the primary driver behind the downward trends observed in Figures 3c and 3d. As more individuals receive treatment, the infectiousness of the population decreases, transmission chains are broken, and overall disease prevalence is reduced.

5. Conclusion

The SEIAT model developed to study HIV/AIDS transmission has provided valuable insights into the dynamics of the disease and the influence of various factors on its spread. The analysis revealed that both the contact rate and reinfection rate, which had positive sensitivity indices, significantly drive the transmission of HIV/AIDS. This underscores the importance of interventions aimed at reducing contact rates and minimizing reinfection to control the disease. In contrast, the treatment rate showed a negative sensitivity index, indicating its vital role in decreasing the prevalence of HIV/AIDS. Enhancing the effectiveness and coverage of treatment programs is therefore essential for reducing the number of cases. Numerical simulations and stability analyses further demonstrated that HIV/AIDS can be effectively controlled through appropriate and well-implemented management strategies, particularly robust treatment programs. Beyond offering theoretical insights, the study also provides practical guidance for public health planning. By identifying the key parameters that influence disease transmission and control, the model offers a clear framework for designing targeted interventions and improving resource allocation. The findings emphasize the need to strengthen treatment programs and implement measures to prevent reinfection, both of which are critical to reducing disease prevalence. In light of these results, it is recommended that health authorities implement targeted efforts to lower contact rates, improve the effectiveness and reach of treatment programs, and develop strategies to prevent reinfection. In addition, it

is crucial that individuals living with HIV/AIDS adhere consistently to their treatment regimens to reduce the risk of transmitting the virus to others. From a public health perspective, unmarried individuals should be advised to avoid oral sex and adopt safe sexual practices, while married individuals are encouraged to remain faithful to their partners to prevent the spread of HIV/AIDS. Continued research should also be supported to refine the model and adjust strategies in response to evolving epidemiological trends. Integrating these findings into public health policies will help improve the management of HIV/AIDS and ensure more efficient use of resources.

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