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MATHEMATICAL MODEL OF THE SPREAD OF HIV/AIDS CONSIDERING THE LEVEL OF IMMUNITY

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ABSTRACT

The immune system, crucial for defending the body against infections, is a primary target of HIV, compromising its ability to resist illnesses that may progress to AIDS. This study develops a mathematical model incorporating the immune response to simulate HIV/AIDS transmission dynamics. The model analysis includes the determination of equilibrium points, the basic reproduction number R_0, and bifurcation behavior. Two equilibrium points are identified: the disease-free and endemic equilibria. The disease-free equilibrium is asymptotically stable when $R_0 < 1$, while the endemic equilibrium is stable when $R_0 > 1$, indicating persistent transmission. A forward bifurcation occurs at $R_0 = 1$, which biologically implies that reducing R_0 below one is critical for eliminating the disease. Numerical simulations using actual data yield an estimated $R_0 = 4.1565$ with a Mean Absolute Percentage Error (MAPE) of 4.5583%, indicating good agreement between the model and data. Although the model assumes homogeneous mixing and constant parameters, it provides meaningful insights into HIV/AIDS transmission and offers a quantitative basis for evaluating control strategies.

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1. INTRODUCTION

Microorganisms that cause diseases, including bacteria, fungi, viruses, and parasites, are known as sexually transmitted infections. These infections are constantly spreading and have a substantial effect on the life expectancy of humans [1],[2]. The fatal illness known as HIV targets and compromises the human immune system, making it a serious global health concern. If left unchecked, this virus would spread and eventually reach the stage known as acquired immune deficiency syndrome (AIDS), which is marked by recurrent symptom development [3],[4],[5],[6]. Globally, HIV has affected 38.4 million people, and more than two-thirds of infected people live in Africa [7]. HIV diagnoses in Indonesia have been rising annually; in 2019, 50,282 people were infected, the highest number in the previous ten years. Meanwhile, 12,214 people were infected with AIDS in 2013, the greatest number to date. One of the Indonesian provinces with the highest rate of HIV/AIDS cases is East Java [8].

In many scientific domains, mathematical modeling plays a crucial role in making predictions about the future and comprehending complicated processes. In biology, the behavior of disease propagation and population expansion is studied using mathematical models. A study is conducted by professionals to understand the behavior of a virus in the health sector, to produce efficient treatments and discover a cure. A variety of diseases can pose a threat to human health [9]. Experts have examined the spread of illnesses like COVID-19 [10],[11],[12], malaria [13],[14],[15],[16] , HIV/AIDS [17], Dengue [18], tuberculosis [19], [20], and other infectious diseases.

One useful tool supporting this research is mathematical modeling, which helps to understand the HIV/AIDS virus. Researchers have recently constructed and modeled the dynamics of the HIV/AIDS virus's transmission as well as its control. Zamzami et al. [21] established the infected class: early-stage infection and advanced-stage or pre-AIDS. Marsudi et al. [22]established strategies to suppress the spread of the virus by educating people on using condoms and providing antiretroviral therapy. Meanwhile, Leleury et al. [17] modified the SIA mathematical model by considering the transmission rate of individuals infected with AIDS and its impact. In a different study, Abraham and Tandiangnga [23] created a mathematical model to simulate how antiretroviral therapy works by varying the amount of one type of antiretroviral drug (ARV) and a combination of three types of ARV to suppress the growth of the virus in the body. Faisah et al. [24] built a mathematical model by classifying the level of symptoms in people living with HIV.

In contrast to previous studies that model HIV/AIDS dynamics without explicitly accounting for the role of host immunity, our research introduces an immunity-aware compartmental model that differentiates individuals based on immune strength. This distinction is crucial because immunity has a significant influence on the likelihood of disease progression. In particular, we look at how HIV-positive people with weak immune systems are more likely to develop AIDS if they don't get treatment. The novelty of this study lies in incorporating immunity stratification into the transmission model and analyzing its impact on disease dynamics through equilibrium and bifurcation analysis. By doing so, we aim to provide a more biologically realistic representation of HIV/AIDS progression and identify critical thresholds for effective intervention. The objective of this study is to analyze the stability of the disease-free and endemic equilibria under different transmission rates, and to explore how immunity levels and treatment influence the long-term dynamics of HIV/AIDS within a population.

2. RESEARCH METHODS

The method employed in this research is a literature study involving the exploration and examination of journals and references related to the mathematical model in the spread of HIV/AIDS considering the immunity system. The steps conducted are as follows:

1. Exploring journals and references related to mathematical models in the spread of HIV/AIDS considering immunity system.
2. Determining the assumptions to be used for modifying the model
3. Formulating a mathematical model of HIV/AIDS transmission considering the immunity system based on predefined assumptions.
4. Identifying disease-free and endemic equilibrium points.
5. Determining parameters that influence the classes of the HIV/AIDS population.

6. Constructing the Next Generation matrix, defined as a matrix FV^{-1} to obtain the basic reproduction number with the dominant eigenvalue of the matrix FV^{-1}.
7. Identify the stability analysis of equilibrium points.
8. Determining the values of each parameter through model fitting using the least squares method.
9. Perform bifurcation analysis to examine the behavior of the system near the threshold $R_0 = 1$.
10. Conducting simulations using the 4th-order Runge-Kutta method.
11. Interpretation and conclusions.

3. RESULTS AND DISCUSSION

3.1 Model Formulation

The stages of HIV infection that lead to AIDS begin with the window period, which is the interval between HIV exposure and the ability to obtain reliable test results. The HIV stage with weak immunity comes next, then the HIV stage with strong immunity, and finally the AIDS stage. The population is split into five subpopulations in order to create a mathematical model: susceptible (S), HIV-positive people with strong immunity (I_1), HIV-positive people with weak immunity (I_2), AIDS-positive people (A), and people receiving treatment (T). Table 1 describes model parameters.

Table 1. Description of Model Parameters

Parameter	Description
Λ	The rate at which vulnerable subpopulations are recruited
β	The rate at which HIV-positive individuals spread the virus to vulnerable individuals
ω_1	The percentage of HIV-positive people with strong immunity who transfer to HIV-positive people with weak immunity
ω_2	The percentage of HIV-positive people with weakened immunity who transfer to AIDS-positive people
α	The percentage of infected people with strong immunity who receive treatment
ρ	The percentage of infected people with low immunity who receive treatment
m	The proportion of AIDS patients receiving treatment
d	Death rate from AIDS
μ	Rate of natural death

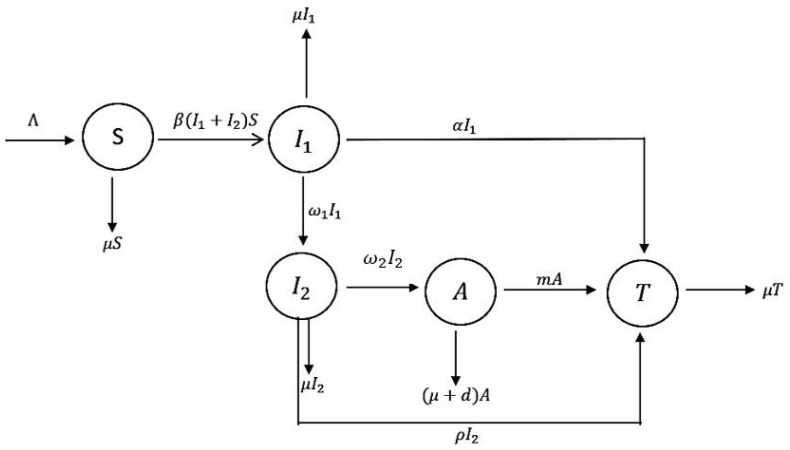


Figure 1. HIV/AIDS transmission diagram

The HIV/AIDS model is constructed using a compartmental framework, as depicted in Fig. 1. The movement of individuals among these compartments mirrors the biological realities of disease transmission and progression. New individuals are introduced to the population at a recruitment rate Λ, joining the susceptible class S, while all individuals face natural mortality at a rate μ. Susceptible individuals become infected through interactions with infected individuals, I_1 and I_2 at a rate of $\beta(I_1 + I_2)S$ subsequently entering the I_1 compartment, which represents HIV-positive individuals with strong immunity. Over time, individuals in I_1 can experience immune degradation and move to I_2 at rate ω_1, or initiate treatment at rate

α. Individuals I_2 may either progress to AIDS (A) at rate ω_2, begin treatment at rate ρ, or die naturally. Individuals in the AIDS compartment A may receive treatment at rate m, but also face an increased disease-induced death rate d, in addition to natural death. Treated individuals T leave the system through natural death at rate μ. Each transition and interaction is translated into an appropriate term in a system of differential equations that mathematically describes the dynamics of the HIV/AIDS epidemic in a population and is expressed as follows:

$$\begin{aligned}
\frac{dS}{dt} &= \Lambda - \beta S(I_1 + I_2) - \mu S \\
\frac{dI_1}{dt} &= \beta S(I_1 + I_2) - \omega_1 I_1 - \alpha I_1 - \mu I_1 \\
\frac{dI_2}{dt} &= \omega_1 I_1 - \omega_2 I_2 - \rho I_2 - \mu I_2 \\
\frac{dA}{dt} &= \omega_2 I_2 - mA - (\mu + d)A \\
\frac{dT}{dt} &= mA + \alpha I_1 + \rho I_2 - \mu T.
\end{aligned} \tag{1}$$

3.2 Boundedness and Positivity solutions

One of the crucial aspects of epidemiological models is that the solution is bounded and positive. We establish the next two theorems in support of this.

Theorem 1. *If the initial conditions are nonnegative, then the solution* $(S(t), I_1(t), I_2(t), A(t), T(t))$ *of system Eq. (1) will remain nonnegative for all* $t \geq 0$.

Proof. From the first equation in Eq. (1),

$$\frac{dS}{dt} = \Lambda - \beta S(I_1 + I_2) - \mu S$$

$$\frac{dS}{S} = X_S(S, I_1, I_2)dt, \qquad \text{where } X_S(S, I_1, I_2) = \frac{\Lambda}{S} - \beta(I_1 + I_2) - \mu$$

By integrating the above expression and given the initial conditions $t = 0$ and $S(0)$, we obtain

$$S(t) = S(0)e^{\int X_S(S, I_1, I_2)dt} \geq 0,$$

for all t using the same method obtained the solution

$$I_1(t) = I_1(0)e^{\int X_{I_1}(S, I_1, I_2)dt} \geq 0$$

$$I_2(t) = I_2(0)e^{\int X_{I_2}(I_1, I_2)dt} \geq 0$$

$$A(t) = A(0)e^{\int X_A(I_2, A)dt} \geq 0$$

$$T(t) = T(0)e^{\int X_T(I_1, I_2, A, T)dt} \geq 0$$

Thus, all solutions $(S(t), I_1(t), I_2(t), A(t), T(t))$ of Eq. (1) is nonnegative.

Theorem 2. *If the initial conditions are nonnegative, then the set* $\Omega = \left\{(S, I_1, I_2, A, T) \in \mathbb{R}^5_+ : S + I_1 + I_2, +A + T \leq \frac{\Lambda}{\mu}\right\}$ *is positively invariant with respect to the dynamical system defined by Eq. (1).*

Proof. Based on Eq. (1), we obtain $\frac{dN}{dt} = \Lambda - \mu N - dA \leq \Lambda - \mu N$. By integrating the above inequality, using the initial condition we get

$$N(t) \leq N_0 e^{-\mu t} + \frac{\Lambda}{\mu}.$$

Thus, if the initial condition taken is in the set of Ω, then the solution remains in Ω for $t \to \infty$. Therefore Ω is a positive invariant.

3.3 Stability Equilibrium Points and Bifurcation

The disease-free or disease-free equilibrium of Eq. (1) is obtained as

$$E_0 = (S^0, I_1^0, I_2^0, A^0, T^0) = \left(\frac{\Lambda}{\mu}, 0,0,0,0\right).$$

The basic reproduction number, which represents the ratio of the number of secondary cases (newly infected individuals) that result from one primary case (infected individual), is first determined in order to assess the stability of E_0. The next-generation matrix is used to obtain the basic reproduction number, as explained in [25] and [16]. The Jacobian matrices F and V in E_0 as follows

$$F = \begin{bmatrix} \frac{\partial \mathcal{F}_1}{\partial I_1} & \frac{\partial \mathcal{F}_1}{\partial I_2} \\ \frac{\partial \mathcal{F}_2}{\partial I_1} & \frac{\partial \mathcal{F}_2}{\partial I_2} \end{bmatrix} = \begin{bmatrix} \beta S^0 & \beta S^0 \\ 0 & 0 \end{bmatrix}, \qquad V = \begin{bmatrix} \frac{\partial \mathcal{V}_1}{\partial I_1} & \frac{\partial \mathcal{V}_1}{\partial I_2} \\ \frac{\partial \mathcal{V}_2}{\partial I_1} & \frac{\partial \mathcal{V}_2}{\partial I_2} \end{bmatrix} = \begin{bmatrix} (\omega_1 + \alpha + \mu) & 0 \\ -\omega_1 & (\omega_2 + \rho + \mu) \end{bmatrix}$$

Hence, the spectral radius of FV^{-1} is

$$R_0 = \frac{\beta\Lambda(\omega_2 + \rho + \mu) + \omega_1)}{\mu(\omega_1 + \alpha + \mu)(\omega_2 + \rho + \mu)}.$$

Thus, the local stability of the disease-free equilibrium point is established in the following theorem.

Theorem 3. *The disease-free equilibrium point E_0 is locally asymptotically stable if $R_0 < 1$.*

Proof. The Jacobian matrix at the equilibrium point E_0 is

$$J(E_0) = \begin{bmatrix} -\mu & -\beta\left(\frac{\Lambda}{\mu}\right) & -\beta\left(\frac{\Lambda}{\mu}\right) & 0 & 0 \\ 0 & \beta\left(\frac{\Lambda}{\mu}\right) - (\omega_1 + \alpha + \mu) & \beta\left(\frac{\Lambda}{\mu}\right) & 0 & 0 \\ 0 & \omega_1 & -(\omega_2 + \rho + \mu) & 0 & 0 \\ 0 & 0 & \omega_2 & -(m + \mu + d) & 0 \\ 0 & \alpha & \rho & m & -\mu \end{bmatrix}$$

The equation characteristic matrix $J(E_0)$ can be obtained by solving equations $|\lambda I - J(E_0)| = 0$, i.e

$$(\lambda + \mu)\left(\lambda - \beta\left(\frac{\Lambda}{\mu}\right) + (\omega_1 + \alpha + \mu)\right)(\lambda - A)(\lambda + m + \mu + d)(\lambda + \mu) = 0$$

From the characteristic equation, the eigenvalues are obtained

$$\lambda_1 = -\mu < 0,\ \lambda_2 = \beta\left(\frac{\Lambda}{\mu}\right) - (\omega_1 + \alpha + \mu) < 0\ ,\ \lambda_3 = \frac{-\beta\left(\frac{\Lambda}{\mu}\right)\omega_1}{\beta\left(\frac{\Lambda}{\mu}\right) - (\omega_1 + \alpha + \mu)} - (\omega_2 + \rho + \mu) < 0$$

$$\lambda_4 = -(m + (\mu + d) < 0,\ \lambda_5 = -\mu < 0.$$

From the results, the disease-free equilibrium point is asymptotically stable because all eigenvalues are negative when $R_0 < 1$, and unstable otherwise.

Furthermore, after performing the necessary calculations, the endemic equilibrium point $E_1 = (S^*, I_1^*, I_2^*, A^*, T^*)$, is derived and exists whenever the basic reproduction number $R_0 > 1$, where:

$$S^* = \frac{(\omega_1 + \alpha + \mu)(\omega_2 + \rho + \mu)}{\beta(\omega_2 + \rho + \mu + \omega_1)},$$

$$I_1^* = \frac{\Lambda(R_0 - 1)}{(\omega_1 + \alpha + \mu)R_0},$$

$$I_2^* = \frac{\omega_1\Lambda(R_0 - 1)}{(\omega_1 + \alpha + \mu)(\omega_2 + \rho + \mu)R_0},$$

$$A^* = \frac{\omega_1\omega_2\Lambda(R_0-1)}{(\omega_1+\alpha+\mu)(\omega_2+\rho+\mu)(m+\mu+d)R_0},$$

$$T^* = \frac{\Lambda(R_0-1)[m\omega_1\omega_2+\alpha(m+\mu+d)(\omega_2+\rho+\mu)+\rho\omega_1(m+\mu+d)]}{\mu(\omega_1+\alpha+\mu)(\omega_2+\rho+\mu)(m+\mu+d)R_0}.$$

The local stability of the equilibrium point E_1 is presented in the following theorem.

Theorem 4. *The endemic equilibrium point E_1 is locally asymptotically stable if $R_0 > 1$.*

Proof. The Jacobian matrix at the equilibrium point E_1 is

$$J(E_1) = \begin{bmatrix} K & -\frac{(\omega_1+\alpha+\mu)(\omega_2+\rho+\mu)}{(\omega_2+\rho+\mu+\omega_1)} & -\frac{(\omega_1+\alpha+\mu)(\omega_2+\rho+\mu)}{(\omega_2+\rho+\mu+\omega_1)} & 0 & 0 \\ L & \frac{-\omega_1(\omega_1+\alpha+\mu)}{(\omega_2+\rho+\mu+\omega_1)} & \frac{(\omega_1+\alpha+\mu)(\omega_2+\rho+\mu)}{(\omega_2+\rho+\mu+\omega_1)} & 0 & 0 \\ 0 & \omega_1 & -(\omega_2+\rho+\mu) & 0 & 0 \\ 0 & 0 & \omega_2 & -(m+\mu+d) & 0 \\ 0 & \alpha & \rho & m & -\mu \end{bmatrix}$$

where

$$L = \frac{-\beta\Lambda(R_0-1)(\omega_2+\rho+\mu)+\omega_1)-\mu(\omega_1+\alpha+\mu)(\omega_2+\rho+\mu)R_0}{(\omega_1+\alpha+\mu)(\omega_2+\rho+\mu)R_0}$$

$$M = \frac{\beta\Lambda(R_0-1)(\omega_2+\rho+\mu+\omega_1)}{(\omega_1+\alpha+\mu)(\omega_2+\rho+\mu)R_0}$$

Then we get

$$J(E_1) = \begin{bmatrix} L & -\frac{(\omega_1+\alpha+\mu)(\omega_2+\rho+\mu)}{(\omega_2+\rho+\mu+\omega_1)} & -\frac{(\omega_1+\alpha+\mu)(\omega_2+\rho+\mu)}{(\omega_2+\rho+\mu+\omega_1)} & 0 & 0 \\ 0 & N & N & 0 & 0 \\ 0 & 0 & \frac{\omega_1 O}{N}-(\omega_2+\rho+\mu) & 0 & 0 \\ 0 & 0 & 0 & -(m+\mu+d) & 0 \\ 0 & \alpha & 0 & 0 & -\mu \end{bmatrix}$$

where

$$N = \frac{M}{L}\left(\frac{(\omega_1+\alpha+\mu)(\omega_2+\rho+\mu)}{(\omega_2+\rho+\mu+\omega_1)}\right)-\frac{\omega_1(\omega_1+\alpha+\mu)}{(\omega_2+\rho+\mu+\omega_1)}$$

$$O = \frac{M}{L}\left(\frac{(\omega_1+\alpha+\mu)(\omega_2+\rho+\mu)}{(\omega_2+\rho+\mu+\omega_1)}\right)+\frac{(\omega_1+\alpha+\mu)(\omega_2+\rho+\mu)}{(\omega_2+\rho+\mu+\omega_1)}$$

The equation characteristic matrix $J(E_1)$ can be obtained by solving equations $|\lambda I - J(E_1)| = 0$, i.e

$$(\lambda-L)(\lambda-N)\left(\lambda-\left(\frac{\omega_1 O}{N}-(\omega_2+\rho+\mu)\right)\right)(\lambda+m+\mu+d)(\lambda+\mu)=0$$

From the characteristic equation, the eigenvalues are obtained

$$\lambda_1 = L < 0,\ \lambda_2 = N < 0,\ \lambda_3 = \frac{\omega_1 O}{N}-(\omega_2+\rho+\mu) < 0,\ \lambda_4 = -(m+\mu+d) < 0,\ and\ \lambda_5 = -\mu < 0.$$

It is known that the eigenvalues ($\lambda_1, \lambda_2, \lambda_3, \lambda_4, and\ \lambda_5$) are negative across all their real parts. Based on the characteristic roots (eigenvalues), it can be deduced that the equilibrium point E_1 is locally asymptotically stable.

3.4 Bifurcation

When passing through a bifurcation point, such as R_0, changes in parameter values can affect the stability of the equilibrium point. These changes can be observed with a bifurcation analysis of the epidemic

model. The center manifold theorem in [26] utilized to determine the proper kind of bifurcation for this epidemic model.

Theorem 5. *The dynamical system defined by Eq. (1) undergoes forward bifurcation when* $R_0 = 1$.

Proof. From Eq. (1), we obtain

$$\begin{aligned} f_1 &= \Lambda - \beta x_1(x_2 + x_3) - \mu x_1 \\ f_2 &= \beta x_1(x_2 + x_3) - \omega_1 x_2 - \alpha x_2 - \mu x_2 \\ f_3 &= \omega_1 x_2 - \omega_2 x_3 - \rho x_3 - \mu x_3 \\ f_4 &= \omega_2 x_3 - m x_4 - (\mu + d) x_4 \\ f_5 &= m x_4 + \alpha x_2 + \rho x_3 - \mu x_5 \end{aligned} \tag{2}$$

where $x_1 = S, x_2 = I_1$, $x_3 = I_2, x_4 = A$, and $x_5 = T$. Consider the case when $R_0 = 1$, which is the bifurcation point. Suppose, futher that $\beta = \beta^*$ is choosen as bifurcation parameter. Solving for β from $R_0 = 1$ gives $\beta^* = \frac{\mu(\omega_1+\alpha+\mu)(\omega_2+\rho+\mu)}{\Lambda(\omega_2+\rho+\mu+\omega_1)}$, and the Jacobian denoted by J_{β^*} is given by

$$J_{\beta^*} = \begin{bmatrix} -\mu & -\beta^*\left(\frac{\Lambda}{\mu}\right) & -\beta^*\left(\frac{\Lambda}{\mu}\right) & 0 & 0 \\ 0 & \beta^*\left(\frac{\Lambda}{\mu}\right) - (\omega_1 + \alpha + \mu) & \beta^*\left(\frac{\Lambda}{\mu}\right) & 0 & 0 \\ 0 & \omega_1 & -(\omega_2 + \rho + \mu) & 0 & 0 \\ 0 & 0 & \omega_2 & -(m + \mu + d) & 0 \\ 0 & \alpha & \rho & m & -\mu \end{bmatrix}$$

has a right eigenvector $\mathbf{w} = \left(-\frac{\beta^*\Lambda}{\mu^2}\left(\frac{(\omega_2+\rho+\mu+\omega_1)}{\omega_1}\right), \frac{(\omega_2+\rho+\mu)}{\omega_1}, 1, \frac{\omega_2}{(m+\mu+d)}, \frac{\alpha(\omega_2+\rho+\mu)(m+\mu+d)+\rho\omega_1(m+\mu+d)+m\omega_1\omega_2}{\mu\omega_1(m+\mu+d)}\right)^T$

and left eigenvector $\mathbf{v} = \left(0, \frac{\mu\omega_1(\omega_2+\rho+\mu)}{\mu(\omega_2+\rho+\mu)^2+\beta^*\Lambda\omega_1}, \frac{\beta^*\Lambda\omega_1}{\mu(\omega_2+\rho+\mu)^2+\beta^*\Lambda\omega_1}, 0, 0\right)$, such that $\mathbf{v}.\mathbf{w} = \mathbf{1}$. To determine the direction of bifurcation, we compute and determine the sign of the two bifurcation coefficients a dan b. At the DFE, the bifurcation coefficient a is given by

$$a = \sum_{k,i,j}^{5} v_k w_i w_j \frac{\partial^2 f_k}{\partial x_i \partial x_j}([E_0 \quad \beta^*]^T)$$

$$= -\frac{\beta^*\Lambda^2(\omega_2 + \rho + \mu + \omega_1)(\omega_2 + \rho + \mu)(\omega_2 + \rho + \mu + \omega_1)}{\mu\omega_1(\mu(\omega_2 + \rho + \mu)^2) + \beta^*\Lambda\omega_1)}$$

The second bifurcation coefficient b is given by

$$b = \sum_{k,i}^{5} v_k w_i \frac{\partial^2 f_k}{\partial x_i \partial \beta}([E_0 \quad \beta^*]^T)$$

$$= \frac{\Lambda(\omega_2 + \rho + \mu)(\omega_2 + \rho + \mu + \omega_1)}{\mu(\omega_2 + \rho + \mu)^2 + \beta^*\Lambda\omega_1}.$$

Because a is negative and b is positive, by Theorem 4 in [27], this shows that a forward bifurcation occurs.

3.5 Global Stability of the Equilibrium Points

According to the results above, whenever $R_0 > 1$ a small number of infectious people introduced into a population that is fully susceptible will cause the disease to continue to exist in the community.

Theorem 6. *The equilibrium point* $E_0 = (S^0, I_1^0, I_2^0, A^0, T^0)$ *is globally asymptotically stable if* $R_0 \leq 1$.

Proof. To show the DFE's global stability, we used the Lyapunov function approach, as described in **[28]**. Let $V: \mathbb{R}^5 \to \mathbb{R}$ be the Lyapunov function

$$V(S,I_1,I_2,A,T)=\left(S-S^0-S^0\ln\frac{S}{S^0}\right)+k_1I_1+k_2I_2+k_3A+k_4T \tag{3}$$

with k_1,k_2,k_3,k_4 positive constants. Then we have

$$\begin{aligned}\frac{\partial V}{\partial t}=&\left(1-\frac{S^0}{S}\right)(\Lambda-\beta S(I_1+I_2)-\mu S)+k_1(\beta S(I_1+I_2)-\omega_1I_1-\alpha I_1-\mu I_1)\\&+k_2(\omega_1I_1-\omega_2I_2-\rho I_2-\mu I_2)+k_3(\omega_2I_2-mA-(\mu+d)A)\\&+k_4(mA+\alpha I_1+\rho I_2-\mu T)\end{aligned}$$

At the disease-free equilibrium point E_0 we obtain

$$\begin{aligned}\frac{\partial V}{\partial t}=&\left[\left(k_1\left(\frac{\beta\Lambda}{\mu}-(\omega_1+\alpha+\mu)\right)+k_2\omega_1+k_4\alpha\right)I_1+\left(k_1\frac{\beta\Lambda}{\mu}-k_2(\omega_2+\rho+\mu)+k_3\omega_2+k_4\rho\right)I_2\right.\\&\left.+(k_3(m+\mu+d)+k_4\,m)A-k_4\mu T\right]\end{aligned}$$

Choose $k_1=(\omega_2+\rho+\mu)-\omega_2-\rho$; $k_2=\frac{\beta\Lambda}{\mu}(1-\omega_2-\rho)$; $k_3=\frac{\beta\Lambda}{\mu}(1-(\omega_2+\rho+\mu)-\rho)$; $k_4=\frac{\beta\Lambda}{\mu}(1-(\omega_2+\rho+\mu)-\omega_2)$, thus

$$\begin{aligned}\frac{\partial V}{\partial t}=&\left(\frac{\beta\Lambda}{\mu}\big((\omega_2+\rho+\mu)+\omega_1\big)-(\omega_1+\alpha+\mu)(\omega_2+\rho+\mu)\right.\\&\left.-\frac{\beta\Lambda}{\mu}\big((1+(\omega_2+\rho+\mu)+\omega_2)\alpha+\rho+\omega_1(1-\omega_2+\rho)\big)-(\omega_1+\alpha+\mu)(\omega_1+\rho)\right)I_1\\&+\left(\frac{\beta\Lambda}{\mu}\big(m+(\mu+d)\big)(1-(\omega_2+\rho+\mu)-\rho)+m(1-(\omega_2+\rho+\mu)-\omega_2\right)\\&-(\beta\Lambda(1-(\omega_2+\rho+\mu)-\omega_2)T\end{aligned}$$

$$\begin{aligned}\Rightarrow\frac{\partial V}{\partial t}=&\left((\omega_1+\alpha+\mu)(\omega_2+\rho+\mu)(R_0-1)\right.\\&\left.-\frac{\beta\Lambda}{\mu}\big((1+(\omega_2+\rho+\mu)+\omega_2)\alpha+\rho+\omega_1(1-\omega_2+\rho)\big)-(\omega_1+\alpha+\mu)(\omega_1+\rho)\right)I_1\\&+\left(\frac{\beta\Lambda}{\mu}\big(m+(\mu+d)\big)(1-(\omega_2+\rho+\mu)-\rho)+m(1-(\omega_2+\rho+\mu)-\omega_2\right)\\&-(\beta\Lambda(1-(\omega_2+\rho+\mu)-\omega_2)T\end{aligned}$$

Therefore $\frac{\partial V}{\partial t}\le 0$ if $R_0<1$ and $\frac{\partial V}{\partial t}=0$ if and only if $I_1=0,T=0$. Further, one sees that $(S,I_2,A)\to\left(\frac{\Lambda}{\mu},0,0\right)$ as $t\to\infty$. Based on Lyapunov function, E_0 is globally asympttotically stable in Ω if $R_0\le 1$.

The global stability of endemic equilibrium point E_1 stated in the Theorem 7 below.

Theorem 7. *The endemic equilibrium point $E_1=(S^*,I_1^*,I_2^*,A^*,T^*)$ is globally asymptotically stable if $R_0>1$.*

Proof. Let the Lyapunov function $V:\mathbb{R}^5\to\mathbb{R}$ is defined by

$$\begin{aligned}V(S,I_1,I_2,A,T)=&\left(S-S^*-S^*\ln\frac{S}{S^*}\right)+B\left(I_1-I_1^*-I_1^*\ln\frac{I_1}{I_1^*}\right)+C\left(I_2-I_2^*-I_2^*\ln\frac{I_2}{I_2^*}\right)\\&+D\left(A-A^*-A^*\ln\frac{A}{A^*}\right)+E\left(T-T^*-T^*\ln\frac{T}{T^*}\right)\end{aligned}$$

The Lyapuov derivative given by

$$\frac{\partial V}{\partial t}=\left[\left(1-\frac{S^*}{S}\right)+B\left(1-\frac{I_1^*}{I_1}\right)+C\left(1-\frac{I_2^*}{I_2}\right)+D\left(1-\frac{A^*}{A}\right)+E\left(1-\frac{T^*}{T}\right)\right]\frac{\partial(S+I_1+I_2+A+T)}{\partial t}$$

At the endemic equilibrium point $E_1 = (S^*, I_1^*, I_2^*, A^*, T^*)$ we obtain from Eq. (1) $\frac{dN}{dt} = \Lambda - \mu N - dA$ and $\Lambda = \mu(S^* + I_1^* + I_2^* + A^* + T^*) + dA^*$. Then,

$$\frac{dV}{dt} = \left[\left(1 - \frac{S^*}{S}\right) + B\left(1 - \frac{I_1^*}{I_1}\right) + C\left(1 - \frac{I_2^*}{I_2}\right) + D\left(1 - \frac{A^*}{A}\right) + E\left(1 - \frac{T^*}{T}\right)\right][\mu(S^* + I_1^* + I_2^* + A^* + T^*) + dA^* - \mu(S + I_1 + I_2 + A + T) - dA]$$

$$\Rightarrow \frac{dV}{dt} = -\mu(S - S^*)\left[\left(1 - \frac{S^*}{S}\right) + B\left(1 - \frac{I_1^*}{I_1}\right) + C\left(1 - \frac{I_2^*}{I_2}\right) + D\left(1 - \frac{A^*}{A}\right) + E\left(1 - \frac{T^*}{T}\right)\right]$$

$$-\mu(I_1 - I_1{}^*)\left[\left(1 - \frac{S^*}{S}\right) + B\left(1 - \frac{I_1^*}{I_1}\right) + C\left(1 - \frac{I_2^*}{I_2}\right) + D\left(1 - \frac{A^*}{A}\right) + E\left(1 - \frac{T^*}{T}\right)\right]$$

$$-\mu(I_2 - I_2{}^*)\left[\left(1 - \frac{S^*}{S}\right) + B\left(1 - \frac{I_1^*}{I_1}\right) + C\left(1 - \frac{I_2^*}{I_2}\right) + D\left(1 - \frac{A^*}{A}\right) + E\left(1 - \frac{T^*}{T}\right)\right]$$

$$-\mu(A - A^*)\left[\left(1 - \frac{S^*}{S}\right) + B\left(1 - \frac{I_1^*}{I_1}\right) + C\left(1 - \frac{I_2^*}{I_2}\right) + D\left(1 - \frac{A^*}{A}\right) + E\left(1 - \frac{T^*}{T}\right)\right]$$

$$-\mu(T - T^*)\left[\left(1 - \frac{S^*}{S}\right) + B\left(1 - \frac{I_1^*}{I_1}\right) + C\left(1 - \frac{I_2^*}{I_2}\right) + D\left(1 - \frac{A^*}{A}\right) + E\left(1 - \frac{T^*}{T}\right)\right]$$

$$-d(A - A^*)\left[\left(1 - \frac{S^*}{S}\right) + B\left(1 - \frac{I_1^*}{I_1}\right) + C\left(1 - \frac{I_2^*}{I_2}\right) + D\left(1 - \frac{A^*}{A}\right) + E\left(1 - \frac{T^*}{T}\right)\right].$$

Hence, we conclude that $\frac{dV}{dt} \leq 0$, and $\frac{dV}{dt} = 0$ if and only if $S = S^*, I_1 = I_1^*, I_2 = I_2^*, A = A^*, T = T^*$. Thus, we conclude that the endemic equilibrium point, $E_1 = (S^*, I_1^*, I_2^*, A^*, T^*)$ is globally asymptotically stable.

3.6 Model Fitting of East Java's HIV/AIDS Data

The reported number of people living with HIV/AIDS in East Java Province from 2009 to 2021, as documented by the Ministry of Health of the Republic of Indonesia, will be used to validate the system of differential equations presented in Eq. (1) in this section. By minimizing the SSE quadratic objective function, we estimate parameter values using the least squares method

$$SSE = \sum_{j=1}^{n} \left(Y_j - I(t_j)\right)^2$$

where $Y_j, I(t_j), n$ represents the cumulative actual HIV/AIDS infected case, the model solution for jth observation, and the total number of available data, respectively. We estimate the initial values of each parameter within a reasonable range in the following ways for the purpose of data fitting:

1. Based on the average life expectancy of the population of East Java from 2009 to 2021 is 70.5 years, so μ = 1/70.5=0.01418.
2. Statistics Indonesia, known as Badan Pusat Statistik (BPS) shows the population of East Java in 2009 reached 37,271,775. So the recruitment rate $\Lambda = \mu \times N = 528{,}678$.
3. The death rate due to AIDS from 2009 to 2021 has decreased, from 6.12% to 0.59%. Therefore, the value for parameter d ranges between [0.005,0.07].
4. Individuals who are newly infected with HIV still have strong immunity. With time, the immunity of the HIV-infected individual will weaken. The weakening of the body's immunity lasts about 2-10 years, so $\omega_1 = [0.1, 0.5]$.
5. Individuals with weakened immunity have a high chance of contracting AIDS because they have lost the ability to fight viruses that enter the body. The time span until individuals contract AIDS is about 10-15 years, so $\omega_2 = [0.06, 0.1]$.
6. Based on the HIV/AIDS report of the Kementerian Kesehatan Republik Indonesia from 2013 to 2022, it shows that around 31% - 41.5% of HIV patients received treatment, where in 2013 = 31%, 2014 = 34%, 2015 = 33%, 2016 = 33.5%, 2017 = 32.6%, 2018 = 33.145%, 2019 = 34%, 2020 = 40%, 2021 = 40%, and 2020 = 41%. So, referring to the data, the value of $\alpha = [0.3, 0.5]$.

7. Since there is no available real data to determine the values of the parameters m, ρ and β, we assume m and ρ to lie within the range $[0.1, 1]$ and $\beta \in [10^{-8}, 0.001]$ based on relevant literature [1], [29], [30] and [31].

By using the Least Squares method, we simulate all parameters and initial values model in Eq. (1). All estimated values for each parameter can be seen in Table 2. The results of fitting model Eq. (1) are as shown in Fig. 2.

Table 2. Values of Each Parameter

Parameter	Value
Λ	493212
β	1.9403×10^{-8}
ω_1	0.24053
ω_2	0.15845
α	0.088402
ρ	0.069664
m	0.14067
d	0.022414
μ	0.096956

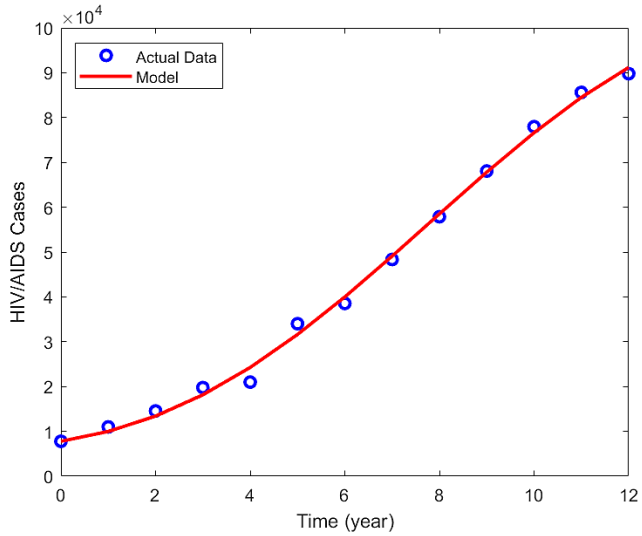


Figure 2. Simulation of HIV/AIDS Data with the Model

Fig. 2 depicts the model's fit to real-world data. The mean absolute percentage error (MAPE) score is 4.5583, indicating that the estimation findings are accurate [32].

3.7 Numerical Solution

In this section, a numerical simulation is performed to analyze the dynamics of the model built using the parameter values listed in Table 2. The purpose of this simulation is to evaluate the behavior of the system under various conditions of the basic reproduction number (R_0), both when $R_0 < 1$ and $R_0 > 1$. Additionally, variations are made to the key parameters to assess the model's sensitivity. This simulation is also intended to identify the presence of bifurcation phenomena.

3.7.1 Population Dynamics for $R_0 < 1$.

The disease-free equilibrium point $E_0 = (S^0, I_1^0, I_2^0, A^0, T^0) = \left(\frac{\Lambda}{\mu}, 0,0,0,0\right) = (50\,869\,675,0,0,0,0)$ is shown in Fig. 3 for $R_0 = 0.4032$. The susceptible individual experiences a decrease at the beginning of time due to disease transmission by infected individuals, then increases until it is constant towards its equilibrium point. Each of the model's solution curves converges to zero at the disease-free equilibrium point. This further demonstrates the asymptotic stability of the equilibrium point E_0.

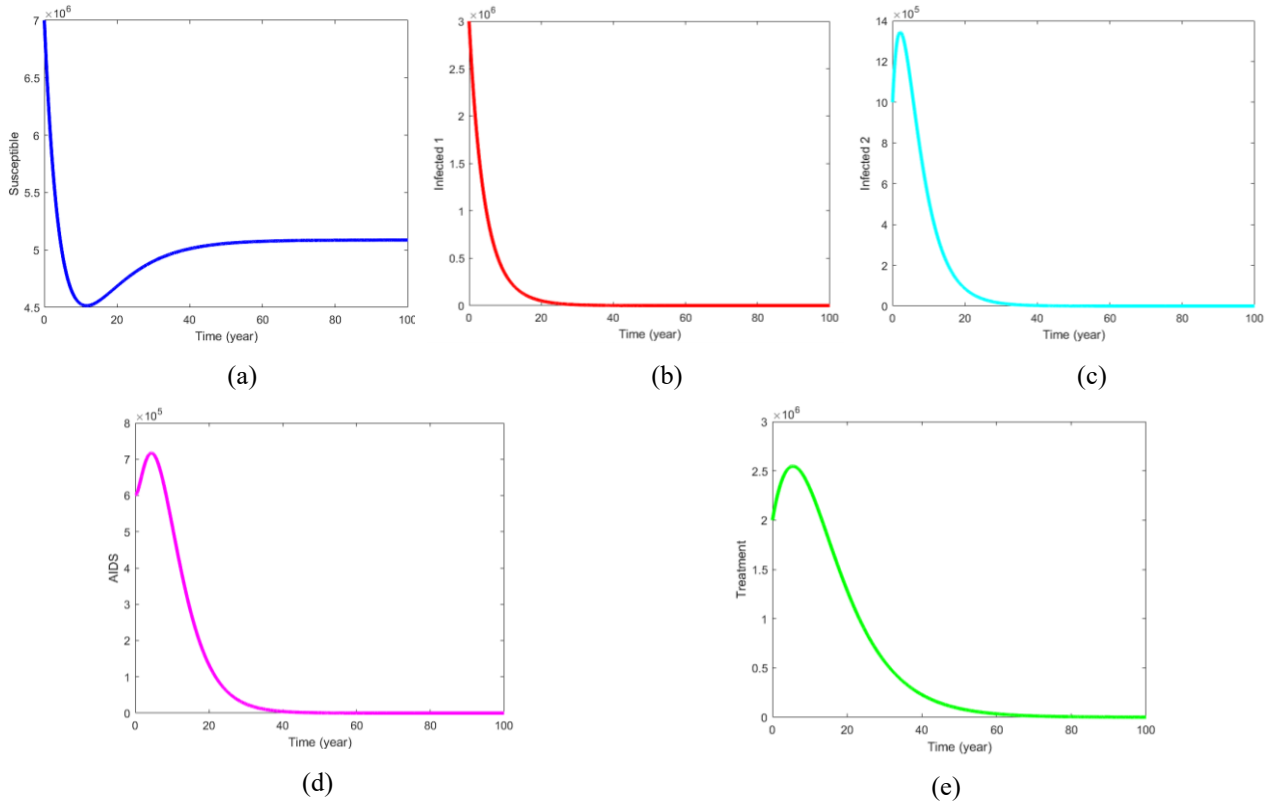


Figure 3. Numerical Results when $R_0 < 1$ of the Sub-Populations (a) Susceptible, $S(t)$, (b) HIV-Positive People with Strong Immunity, $I_1(t)$, (c) HIV-Positive People with Weak Immunity, $I_2(t)$, (d) AIDS-Positive People, A(t), and (e) People Receiving Treatment, T(t).

The graphs shown in Fig. 3 presents the dynamics of each subpopulation when $R_0 < 1$, indicating a scenario where the infection is expected to die out over time. In subfigure (a), the susceptible population $S(t)$ initially decreases due to new infections, but then gradually increases and stabilizes as the disease is brought under control, reflecting successful prevention and reduced transmission. Subfigures (b), (c), and (d) show that the populations of infected individuals with strong immunity $I_1(t)$, weak immunity $I_2(t)$, and AIDS $A(t)$ all exhibit a rapid decline toward zero, indicating that the disease is not sustained in the population. Subfigure (e) similarly demonstrates that the number of individuals receiving treatment $T(t)$ also declines over time, consistent with the elimination of new cases. Biologically, these trends confirm that when the basic reproduction number is below one, HIV/AIDS transmission cannot be maintained, and all infected-related compartments eventually vanish, leading the system toward the disease-free equilibrium. This supports the asymptotic stability of the equilibrium point and highlights the importance of keeping R_0 below one to eliminate the disease from the community.

3.7.2 Population Dynamics for $\mathbf{R_0 > 1}$.

The solution of HIV/AIDS model (1) converges to the endemic equilibrium point $E_1 = (S^*, I_1^*, I_2^*, A^*, T^*) = (1\,223\,863.26,\ 879\,457.92,\ 650\,739.9,\ 396\,514.9,\ 18\,447\,206.23)$ for $R_0 = 4.1565$ with $\beta = 2 \times 10^{-7}$. Each population's model solution reaches the endemic equilibrium point after sixty years. This further demonstrates the asymptotic stability of the equilibrium point E_1. This indicates that, from a biological perspective, the disease will persist in the population and continue to spread.

The graphs shown in Fig. 4 displays the model's numerical results when the basic reproduction number $R_0 = 4.1565 > 1$, indicating an endemic scenario where HIV/AIDS persists in the population. In subfigure (a), the susceptible population S initially decreases sharply due to rapid transmission, then stabilizes at a lower level, suggesting a significant portion of the population becomes infected and the number of susceptible individuals declines. Subfigures (b) and (c) show that the populations of HIV-positive individuals with strong immunity $I_1(t)$ and weak immunity $I_2(t)$ experience a rapid initial rise, then gradually decline and settle at non-zero values, indicating a sustained presence of infection with a dynamic balance between progression and treatment. In subfigure (d), the number of AIDS patients $A(t)$ increases rapidly, peaks around year 10, and then declines toward an endemic level, implying that some individuals transition into AIDS but do not

dominate the population over time. Subfigure (e) illustrates that the treatment population $T(t)$ also peaks early and stabilizes later, reflecting the ongoing need for treatment in managing the epidemic. Biologically, these results demonstrate the persistence of HIV/AIDS across all subgroups when $R_0 > 1$, affirming that the disease reaches a stable endemic state, where control strategies must focus on reducing transmission and improving long-term care.

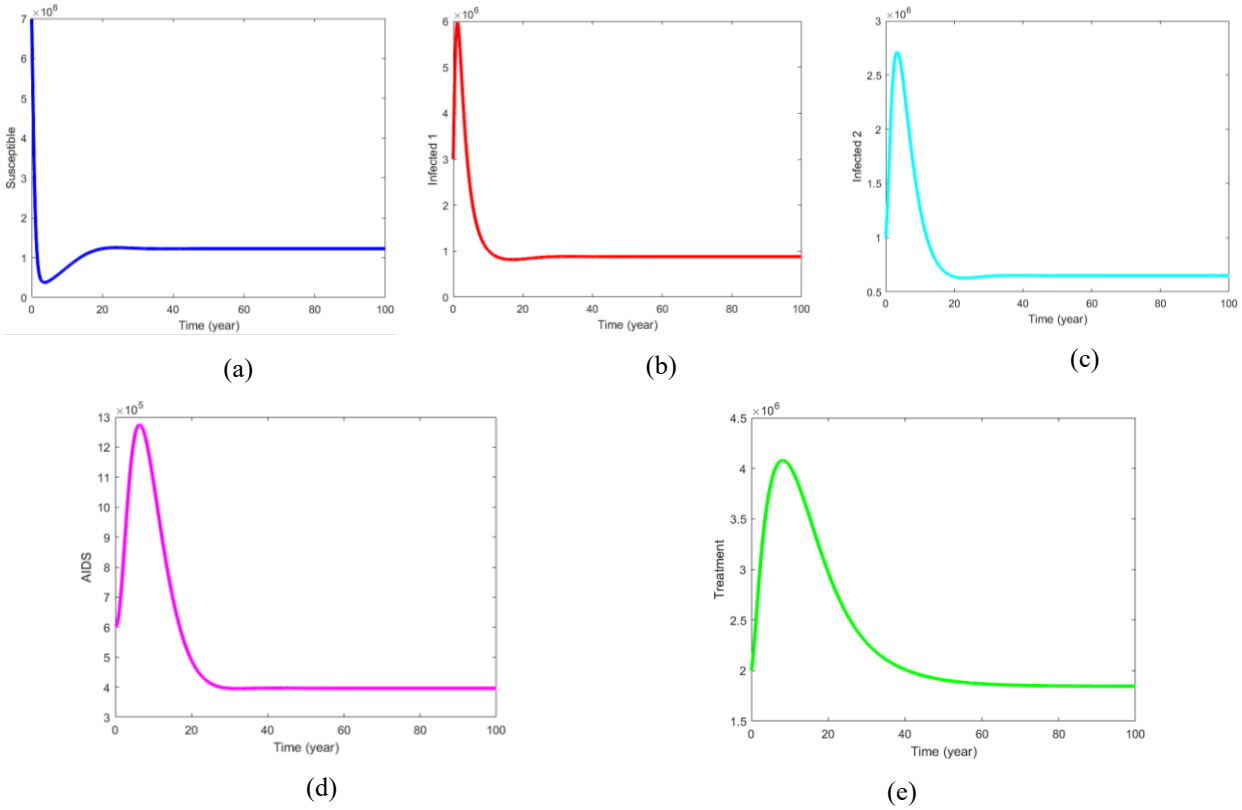
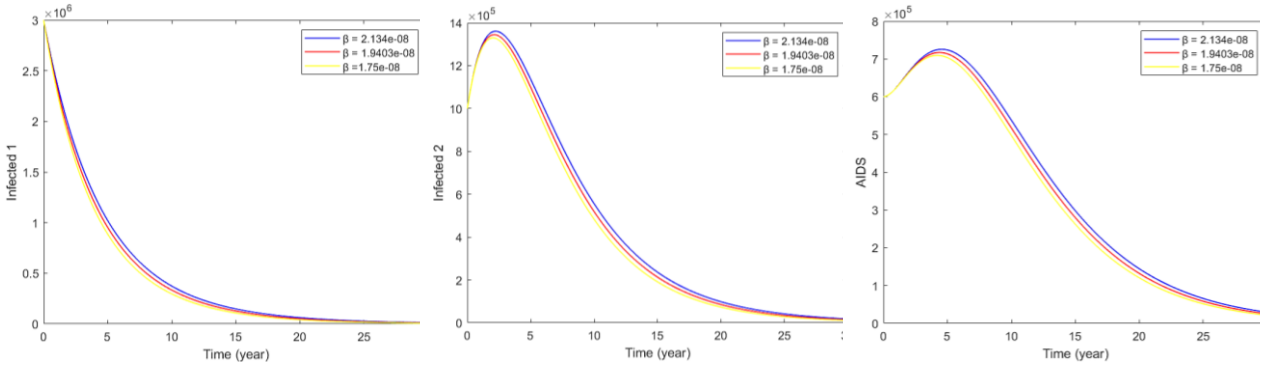


Figure 4. Numerical Results when $R_0 > 1$ of the Sub-Populations (a) Susceptible,$S(t)$, (b) HIV-Positive People with Strong Immunity, $I_1(t)$, (c) HIV-Positive People with Weak Immunity, $I_2\ (t)$, (d) AIDS-Positive People, $A(t)$, and (e) People Receiving Treatment, $T(t)$.

3.7.3 Parameter Variation

In this section, variations of the parameter β are conducted to analyze the effect of the HIV transmission rate on the dynamics of disease spread within the population. Fig. 5 shows that reducing the rate at which HIV disease is transmitted to vulnerable individuals, β, also lowers the reproduction rate, suggesting a decrease in the overall spread of HIV/AIDS in the population. According to biology, the HIV can be eliminated from the population when $\beta < 1.9403 \times 10^{-8}$ or when the basic reproduction number, $R_0 < 1$.

The graphs shown in Fig. 5 illustrates the effect of varying the transmission rate β on the dynamics of three subpopulations over 30 years. Subfigure (a) shows the population of HIV-positive individuals with strong immunity $I_1(t)$. All curves demonstrate a consistent decline, indicating that over time, individuals in this category tend to progress to weaker immune states or AIDS. A higher β value slightly delays this decline, suggesting that stronger transmission prolongs the presence of newly infected individuals with initially strong immunity. Subfigure (b) presents the dynamics of HIV-positive individuals with weak immunity $I_2(t)$. This population rises initially, peaks around year 5, and then decreases. The peak is more pronounced and occurs earlier with higher β, indicating that increased transmission accelerates the transition from strong to weak immunity among infected individuals. In subfigure (c), the AIDS population $A(t)$ also increases during the first few years and then slowly decreases over time. Higher β values lead to a higher peak of AIDS cases, suggesting that increased transmission leads to faster and greater progression to the AIDS stage. These trends demonstrate that although all populations eventually decline due to recovery or mortality, higher transmission rates result in more severe early-stage outbreaks and a larger burden on the population in the short to medium term.



(a) (b) (c)

Figure 5. Effect of Changing β on the Subpopulations (a) HIV-Positive People with Strong Immunity, $I_1(t)$, (b) HIV-Positive People with Weak Immunity, $I_2\ (t)$, and (c) AIDS-Positive People, $A(t)$.

3.7.4 Bifurcation

Bifurcation analysis is used to see changes in the stability of the model caused by changes in parameter values when passing through a bifurcation point, such as R_0. In determining the appropriate type of bifurcation for this epidemic model, the bifurcation theorem in [27] is used for Eq. (1).

$$\begin{aligned} f_1 &= \Lambda - \beta x_1(x_2 + x_3) - \mu x_1 \\ f_2 &= \beta x_1(x_2 + x_3) - \omega_1 x_2 - \alpha x_2 - \mu x_2 \\ f_3 &= \omega_1 x_2 - \omega_2 x_3 - \rho x_3 - \mu x_3 \\ f_4 &= \omega_2 x_3 - m x_4 - (\mu + d) x_4 \\ f_5 &= m x_4 + \alpha x_2 + \rho x_3 - \mu x_5 \end{aligned} \tag{4}$$

where $x_1 = S, x_2 = I_1,\ x_3 = I_2, x_4 = A,$ and $x_5 = T$. Consider the case when $R_0 = 1$, which is the bifurcation point. Suppose, futher that $\beta = \beta^*$ is choosen as bifurcation parameter. Solving for β from $R_0 = 1$ gives $\beta^* = \frac{\mu(\omega_1+\alpha+\mu)(\omega_2+\rho+\mu)}{\Lambda(\omega_2+\rho+\mu+\omega_1)}$, and the Jacobian denoted by J_{β^*} is given by

$$J_{\beta^*} = \begin{bmatrix} -\mu & -\beta^*\left(\frac{\Lambda}{\mu}\right) & -\beta^*\left(\frac{\Lambda}{\mu}\right) & 0 & 0 \\ 0 & \beta^*\left(\frac{\Lambda}{\mu}\right) - (\omega_1 + \alpha + \mu) & \beta^*\left(\frac{\Lambda}{\mu}\right) & 0 & 0 \\ 0 & \omega_1 & -(\omega_2 + \rho + \mu) & 0 & 0 \\ 0 & 0 & \omega_2 & -(m + \mu + d) & 0 \\ 0 & \alpha & \rho & m & -\mu \end{bmatrix}$$

has a right eigenvector $\mathbf{w} =$

$$\left[-\frac{\beta^*\Lambda}{\mu^2}\left(\frac{(\omega_2 + \rho + \mu + \omega_1)}{\omega_1}\right) \quad \frac{(\omega_2 + \rho + \mu)}{\omega_1} \quad 1 \quad \frac{\omega_2}{(m + \mu + d)} \quad \frac{\alpha(\omega_2 + \rho + \mu)(m + \mu + d) + \rho\omega_1(m + \mu + d) + m\omega_1\omega_2}{\mu\omega_1(m + \mu + d)}\right]$$

and left eigenvector $\mathbf{v} = \left[0 \quad \frac{\mu\omega_1(\omega_2+\rho+\mu)}{\mu(\omega_2+\rho+\mu)^2+\beta^*\Lambda\omega_1} \quad \frac{\beta^*\Lambda\omega_1}{\mu(\omega_2+\rho+\mu)^2+\beta^*\Lambda\omega_1} \quad 0 \quad 0\right]$. To determine the direction of bifurcation, we compute and determine the sign of the two bifurcation coefficient $\boldsymbol{c}$ dan $\boldsymbol{d}$. At the DFE, the bifurcation coefficient $\boldsymbol{c}$ is given by

$$\boldsymbol{c} = \sum_{k,i,j}^{5} \boldsymbol{v_k w_i w_j} \frac{\partial^2 f_k}{\partial x_i \partial x_j}([E_0 \quad \beta^*]^T)$$

$$= -\frac{\beta^*\Lambda^2(\omega_2 + \rho + \mu + \omega_1)(\omega_2 + \rho + \mu)(\omega_2 + \rho + \mu + \omega_1)}{\mu\omega_1(\mu(\omega_2 + \rho + \mu)^2 + \beta^*\Lambda\omega_1)}$$

The second bifurcation coeficient $\boldsymbol{d}$ is given by

$$\boldsymbol{d} = \sum_{k,i}^{5} \boldsymbol{v_k w_i} \frac{\partial^2 f_k}{\partial x_i \partial \beta}([E_0 \quad \beta^*]^T)$$

$$= \frac{\Lambda(\omega_2 + \rho + \mu)(\omega_2 + \rho + \mu + \omega_1)}{\mu(\omega_2 + \rho + \mu)^2 + \beta^* \Lambda \omega_1}$$

Because $\boldsymbol{c}$ is negative and $\boldsymbol{d}$ is positive, this shows that a forward bifurcation occurs.

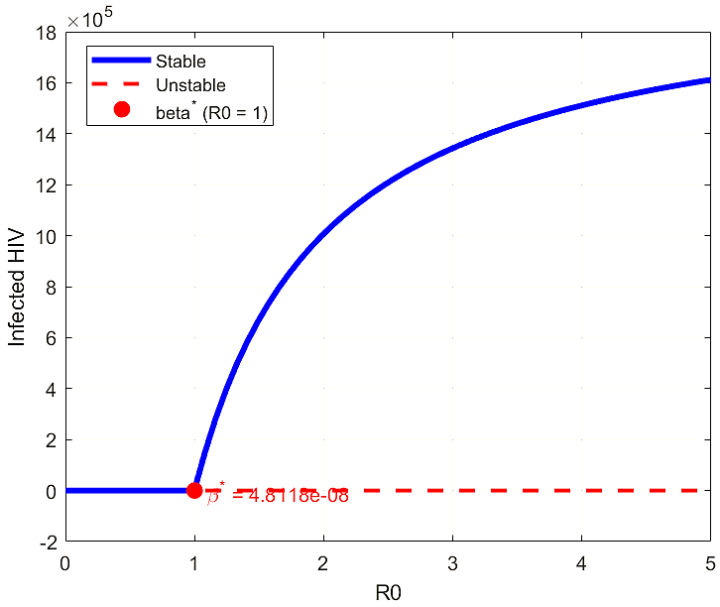


Figure 6. The Occurrence of Forward Bifurcation at $R_0 = 1$

The graph in Fig. 6 shows that with the set of parameter values on Table 2, forward bifurcation occur whenever $R_0 = 1$ which corresponding to $\beta = 4.81186 \times 10^{-8}$. Biologically, this indicates that when the transmission rate exceeds this threshold value, the infection can invade the population and become endemic. Conversely, suppose the transmission rate is kept below this critical value. In that case, the disease will eventually die out, highlighting the importance of reducing transmission, such as through behavioral change, treatment, or preventive measures to control the spread of HIV/AIDS.

4. CONCLUSION

We have examined a mathematical model for the immune transmission of HIV/AIDS in this article. The model's relevance in mathematics and biology is underscored by its boundedness and positivity, ensuring biological feasibility. The analysis involved determining the equilibrium points, assessing their stability, estimating parameters, and conducting bifurcation analysis. Using the data presented in Table 2, we carried out numerical simulations of the full model to observe the dynamic behavior of HIV/AIDS transmission. Our findings indicate that the disease-free equilibrium is asymptotically stable when the basic reproduction number is less than one, suggesting that the disease may eventually be eradicated from the population. Conversely, when the reproduction number exceeds one, the disease persists, and the population approaches an endemic state. These findings highlight the importance of lowering the basic reproduction number below one to eradicate the disease. Increasing treatment rates, enhancing immune responses through therapy or vaccination, and reducing transmission through medical or behavioral preventive measures are some possible intervention strategies that can be derived from the model. To improve the predictive accuracy of the model, future research could focus on improving parameter estimation using real-time epidemiological data. To provide a more comprehensive framework for understanding and managing HIV/AIDS, the model could also be expanded to include more complex elements such as age structure, spatial heterogeneity, treatment delays, or drug resistance dynamics.

Author Contributions

Anisa Sukma Linarta: Conceptualization, methodology, Writing-Original Draft, Formal Analysis, Software, Validation. Yudi Ari Adi: Conceptualization, Data Curation, Resources, Validation, Software, Visualization, Writing-Review and Editing. All authors discussed the results and contributed to the final manuscript.

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Declarations

The authors declare no competing interests.

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