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Mathematical Modeling of HIV/AIDS Disease Spread with Public Awareness

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ABSTRACT

The spread of HIV/AIDS remains a significant public health concern worldwide, necessitating effective strategies for disease management and prevention. Mathematical modeling offers a powerful tool for understanding disease transmission dynamics and assessing interventions' impact. This study develops a mathematical model for the spread of HIV/AIDS by the population, which is divided into seven sub-populations, namely the susceptible unaware HIV subpopulation, the susceptible aware HIV sub-population, the infected sub-population, the pre-AIDS sub-population, the ARV treatment sub-population, the AIDS sub-population, and the unlikely to be infected with HIV/AIDS sub-population. Further, the formed model is analyzed for its dynamic properties. In this mathematical model, two equilibrium points are obtained, namely the disease-free equilibrium point and the disease-endemic equilibrium point. Additionally, the model calculates the basic reproduction number (R_0). The stability analysis shows that the disease-free equilibrium point is locally asymptotically stable if $R_0 < 1$ and the disease-endemic equilibrium point is locally asymptotically stable if $R_0 > 1$. Numerical simulations of the equilibrium points are carried out to provide an overview of the analyzed results, with parameter values from several sources. Based on the sensitivity analysis, the parameters that significantly affect the spread of HIV/AIDS are the contact rate of HIV-unaware individuals with infected individuals and the transmission rate of HIV infection.

Keywords: HIV/AIDS; Public Awareness; Basic Reproduction Number; Equilibrium Point Stability

INTRODUCTION

Human Immunodeficiency Virus (HIV) is a virus that infects white blood cells, leading to the weakening of the human immune system. Untreated HIV infection can progress to a more severe stage and result in Acquired Immunodeficiency Syndrome (AIDS)[1]. According to WHO (2019), transmission of HIV/AIDS can occur through the exchange of bodily fluids from an infected person, such as blood, breast milk, semen, and vaginal fluids. It can also be transmitted from a mother to her child during pregnancy and childbirth. One cannot get infected through everyday contact like kissing, hugging,

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shaking hands, or sharing personal items. HIV/AIDS can affect anyone, with key populations including the Lesbian, Gay, Bisexual, and Transgender (LGBT) community, sex workers, people who share needles, and prison inmates being the most at risk for transmission [2].

The spread of HIV/AIDS in Indonesia was first identified in the province of Bali in 1987. By the year 2021, HIV/AIDS had spread to 498 out of 514 districts/cities across all provinces in Indonesia. Out of this total, only 474 districts/cities reported cases of HIV/AIDS [3]. Over the past eleven years, the number of HIV cases in Indonesia reached its peak in 2019, with a total of 50.282 cases. As for the highest number of AIDS cases in the past eleven years, it was recorded in 2013, with a total of 12.214 cases [4]. Adolescents are the most vulnerable age group to HIV/AIDS infection. The lack of public awareness about the disease also exacerbates the risk of HIV/AIDS transmission. According to WHO data, only 34% of adolescents can accurately demonstrate knowledge about HIV/AIDS, and only 26% of the female adolescent population and 33% of the male adolescent population are aware of how HIV/AIDS is transmitted. The low public awareness of HIV/AIDS contributes to the continued high population of individuals with HIV/AIDS and limited access to Antiretroviral (ARV) treatment [5].

It is well recognized that mathematical modeling is a crucial approach for understanding any epidemic's dynamics and further developing various control and prevention policies. Numerous studies have delved into and discussed the mathematical modeling of HIV/AIDS. In one such study [6], modeling the dynamics of HIV/AIDS infection using the Atangana-Baleanu derivative with the Susceptible, Infected, Cronis, and AIDS (*SICA*) model, which comprises the susceptible population, divided into Susceptible Unaware (S_u) and Susceptible Aware (S_a), the infected population (Infected), the HIV-Infected population under treatment (Cronis), and the AIDS population (AIDS). Subsequently, the study [7] discusses the transmission of HIV/AIDS from Mother to Child using the Susceptible, Infected, Pre-AIDS, Treatment and AIDS (*SIPTA*) model, which includes the susceptible population (Susceptible), the population of individuals infected with HIV (Infected), the population of individuals infected with HIV but not yet with AIDS (Pre-AIDS), the population of individuals receiving treatment (Treatment), and the population of individuals with AIDS (AIDS). Further in the study [8], a development is presented in the treatment model of HIV/AIDS with distinct stages of HIV infection, employing the Susceptible, HIV, Pre-AIDS, AIDS, Treatment and Remove (*SHPATR*) model. This model encompasses the susceptible population (Susceptible), the population of individuals infected with HIV (HIV), the population of individuals infected with HIV but not yet with AIDS and not yet under treatment (Pre-AIDS), the population of individuals with AIDS (AIDS), the population of individuals under treatment (Treatment), and the population of individuals who alter and maintain sexual behaviors (Removed).

Based on the description above, there hasn't been any discussion regarding mathematical models of HIV/AIDS involving public awareness. Therefore, in this study, we developed the *SIPTA* model by dividing the susceptible compartment into Susceptible Unaware of HIV (S_1)and Susceptible Aware of HIV (S_2). Furthermore, we introduced an additional compartment for individuals who are unlikely to be infected with HIV/AIDS. Subsequently, the obtained model's dynamic properties were analyzed, and numerical simulations were conducted.

METHODS

The steps in this research are as follows, starting with a literature review. This is followed by determining assumptions, variables, and parameters to construct a compartment diagram. After that, create the model equations. Subsequently, seek the equilibrium point of disease-free, the basic reproduction number, and the equilibrium point of endemic disease. Then, analyze the stability of the disease-free and endemic equilibrium points. To perform numerical simulations, parameter values obtained from various reference sources are needed, and there are several parameter values assumed by the researcher. Then, these parameter values are inputted to observe the stability curve of equilibrium points. Next, conduct model simulations and sensitivity analysis using the Maple 2021 application to identify parameter values significantly influencing the basic reproduction number. Finally, draw conclusions based on the obtained analysis results.

RESULTS AND DISCUSSION

This section will discuss the formulation of mathematical models of HIV/AIDS disease spread and analysis of its dynamic systems. The analysis begins with determining the equilibrium points of disease-free and endemic equilibrium points, the basic reproduction number, numerical simulations, and sensitivity analysis of the basic reproduction number.

Mathematical Model

The assumptions for constructing a mathematical model of HIV/AIDS spread with public awareness can be outlined as follows. (1)The model employed is the S_1S_2IPTAR model, where Susceptible Unaware (S_1) represents individuals who are susceptible and unaware of HIV, Susceptible Aware (S_2) represents susceptible individuals who are aware of HIV, Infected (I) denotes individuals infected with HIV, Pre-AIDS (P) represents individuals who are infected with HIV but have not progressed to AIDS, Treatment (T) includes individuals undergoing ARV treatment, AIDS (A) represents individuals infected with AIDS, and Removed (R) encompasses individuals who are unlikely to be infected with HIV/AIDS. (2) The population is assumed to be closed, meaning there is no movement of people out of or into the population (no migration). (3) The population is assumed to be homogeneous, meaning that each individual has an equal probability of making contact with others. (4) Recruitment rates occur in each subpopulation and enter the unaware HIV subpopulation (S_1), while natural deaths occur in every compartment. (5) There are deaths due to AIDS. (6) Susceptible unaware of HIV individuals can transition into susceptible aware of HIV individuals after receiving information about HIV, and vice versa. (7) There are three possibilities after an individual is infected with HIV: they can undergo treatment, enter the pre-AIDS stage, or progress directly to AIDS infection. (8) After the pre-AIDS period concludes, individuals in this subpopulation will enter the treatment subpopulation (ARV treatment). If the immune system remains strong, patients will stay in the treatment subpopulation (ARV treatment). However, if the immune system is weakened, they will transition into the AIDS subpopulation. (9) Individuals undergoing treatment can still be infected with AIDS due to opportunistic infections. Individuals infected with AIDS can seek treatment

to prolong their lives. (10) Individuals infected with HIV and AIDS can undergo ARV treatment. (11) For susceptible, aware HIV individuals, there is a possibility of not getting infected with HIV/AIDS because they are knowledgeable about HIV/AIDS prevention and adopt a healthy lifestyle. Next, the parameters forming the model are defined in the following Table 1.

Table 1. List of Parameters for the HIV/AIDS Disease Spread Model with Public Awareness

No.	Parameter	Description	Condition	Unit
1.	π	Recruitment rate.	$\pi > 0$	$\frac{1}{year}$
2.	θ	Rate of transition from susceptible unaware of HIV individuals to susceptible aware of HIV individuals.	$0 \leq \theta \leq 1$	$\frac{1}{year}$
3.	η	Rate of transition from susceptible aware of HIV individuals to susceptible unaware of HIV individuals.	$0 \leq \eta \leq 1$	$\frac{1}{year}$
4.	β	Contact rate between susceptible unaware of HIV individuals and infected individuals.	$0 < \beta \leq 1$	$\frac{1}{year}$
5.	δ	Rate of transmission from the infected class.	$0 \leq \delta \leq 1$	$\frac{1}{year}$
6.	α_1	Proportion of δ transitioning to the treatment class.	$0 \leq \alpha_1 \leq 1$	
7.	α_2	Proportion of δ transtitioning to the pre-AIDS class.	$0 \leq \alpha_2 \leq 1$	
8.	γ	Rate of transition from the pre-AIDS class.	$0 \leq \gamma \leq 1$	$\frac{1}{year}$
9.	m	Proportion of γ undergoing treatment.	$0 \leq m \leq 1$	
10.	σ	Rate of transition from individuals undergoing treatment to individuals infected with AIDS.	$0 \leq \sigma \leq 1$	$\frac{1}{year}$
11.	ρ	Rate of AIDS individuals undergoing treatment.	$0 \leq \rho \leq 1$	$\frac{1}{year}$
12.	μ	Rate of natural death.	$0 \leq \mu \leq 1$	$\frac{1}{year}$
13.	τ	Rate of death due to AIDS.	$0 \leq \tau \leq 1$	$\frac{1}{year}$
14.	ω	Rate of individuals who are are unlikely to be infected with HIV/AIDS infection due to adopting a healthy lifestyle.	$0 \leq \omega \leq 1$	$\frac{1}{year}$

Schematically, the process of HIV/AIDS spreading with public awareness within a population can be illustrated in a transfer diagram, as shown in Figure 1. Based on the transfer diagram model in Figure 1, the mathematical equations for the spread of HIV/AIDS with public awareness are obtained as follows:

$$\begin{aligned}
\frac{dS_1}{dt} &= \pi + \eta S_2 - \left(\mu + \theta + \frac{\beta I}{N}\right) S_1, \\
\frac{dS_2}{dt} &= \theta S_1 - (\mu + \eta + \omega) S_2, \\
\frac{dI}{dt} &= \frac{\beta S_1 I}{N} - (\mu + \delta) I, \\
\frac{dP}{dt} &= \alpha_2 \delta I - (\mu + \gamma) P, \\
\frac{dT}{dt} &= \alpha_1 \delta I + m\gamma P + \rho A - (\mu + \sigma) T, \\
\frac{dA}{dt} &= (1 - m)\gamma P + (1 - \alpha_1 - \alpha_2)\delta I + \sigma T - (\mu + \tau + \rho) A, \\
\frac{dR}{dt} &= \omega S_2 - \mu R,
\end{aligned} \tag{1}$$

with $N = S_1 + S_2 + I + P + T + A + R$. Furthermore, in the system of equations (1), the variable R does not affect the other equations, so the equation for R can temporarily be disregarded. Thus, system (1) can be written as follows:

$$\begin{aligned}
\frac{dS_1}{dt} &= \pi + \eta S_2 - \left(\mu + \theta + \frac{\beta I}{N}\right) S_1, \\
\frac{dS_2}{dt} &= \theta S_1 - (\mu + \eta + \omega) S_2, \\
\frac{dI}{dt} &= \frac{\beta S_1 I}{N} - (\mu + \delta) I, \\
\frac{dP}{dt} &= \alpha_2 \delta I - (\mu + \gamma) P, \\
\frac{dT}{dt} &= \alpha_1 \delta I + m\gamma P + \rho A - (\mu + \sigma) T, \\
\frac{dA}{dt} &= (1 - m)\gamma P + (1 - \alpha_1 - \alpha_2)\delta I + \sigma T - (\mu + \tau + \rho) A.
\end{aligned} \tag{2}$$

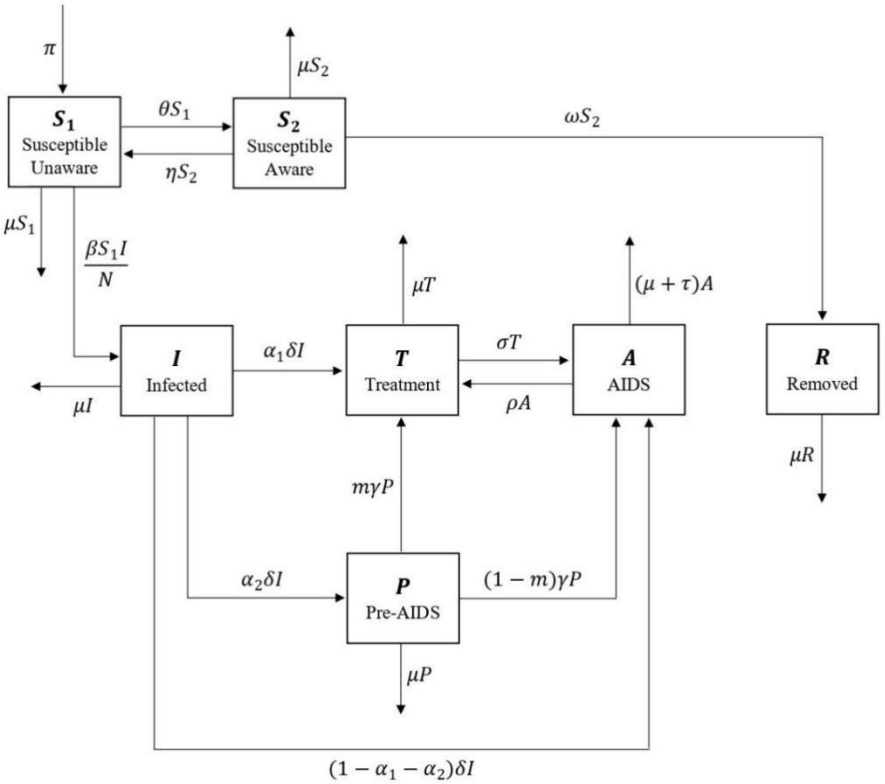

Figure 3. Transfer Diagram of HIV/AIDS Disease Spread Model with Public Awareness

Theorem 1. All solutions of the HIV/AIDS model with public awareness (1) that depend on non-negative initial values are non-negative and bounded.

Proof. First, we will prove that the solutions of system (1) are non-negative $S_1(t) \geq 0, S_2(t) \geq 0, I(t) \geq 0, P(t) \geq 0, T(t) \geq 0, A(t) \geq 0, R(t) \geq 0$.
The first and second equations of system (1) are

$$\frac{dS_1(t)}{dt} = \pi + \eta S_2(t) - \left(\mu + \theta + \frac{\beta I(t)}{N}\right) S_1(t),$$

$$\frac{dS_2(t)}{dt} = \theta S_1(t) - (\mu + \eta + \omega) S_2(t).$$

By assuming its contradiction, $S_1(t) < 0, S_2(t) < 0, I(t) < 0, P(t) < 0, T(t) < 0, A(t) < 0, R(t) < 0$. Let's assume that $S_1(t_1) = 0$ and $S_2(t_2) = 0$.
We obtain

$$\left.\frac{dS_1(t)}{dt}\right|_{t = t_1} = \pi + \eta S_2(t_1), \tag{3}$$

$$\frac{dS_2(t)}{dt}\Big|_{t=t_2} = \theta S_1(t_2). \quad (4)$$

Since the right-hand sides of equations (3) and (4) depend on t_1 and t_2, the proof is divided into two cases:

1. If $t_1 \leq t_2$, then $S_2(t_1) \geq 0$.
   We have
   $$\frac{dS_1(t)}{dt}\Big|_{t=t_1} = \pi + \eta S_2(t_1) > 0.$$
   This means that $S_1(t) > 0$ in the interval $(t_1, t_1 + \varepsilon_1)$ for any small positive constant ε_1. This leads to a contradiction.
   As a result, $S_1(t) \geq 0, \forall\, t \geq 0$, which implies $\frac{dS_2(t)}{dt}\Big|_{t=t_2} = \theta S_1(t_2) \geq 0$.
   Consequently, $S_2(t) \geq 0$ in the interval $(t_2, t_2 + \varepsilon_2)$ for any small positive constant ε_2. This also leads to a contradiction.
   Therefore, $S_2(t) \geq 0, \forall\, t \geq 0$.
2. If $t_1 > t_2$, then $S_1(t_2) > 0$.
   We have
   $$\frac{dS_2(t)}{dt}\Big|_{t=t_2} = \theta S_1(t_2) > 0.$$
   This means that $S_2(t) > 0$ in the interval $(t_2, t_2 + \varepsilon_2)$ for any small positive constant ε_2. This leads to a contradiction
   As a result, $S_2(t) \geq 0, \forall\, t \geq 0$, which implies $\frac{dS_1(t)}{dt}\Big|_{t=t_1} = \pi + \eta S_2(t_1) > 0$.
   Consequently, $S_1(t) > 0$ in the interval $(t_1, t_1 + \varepsilon_1)$ for any small positive constant ε_1. This also leads to a contradiction.
   Therefore, $S_1(t) \geq 0, \forall\, t \geq 0$. (5)

Thus, it has been proven that $S_1(t) > 0$ and $S_2(t) > 0$ for all $t \geq 0$. Other equations can be proven similarly for non-negative solutions.

Next, we will prove that the solutions of system (1) are bounded. By summing all the equations in system (1), we obtain:

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

From equation (6), we have $N(t) \leq \frac{\pi}{\mu}$.

Thus, the domain of the system (1) is $\Omega = \left\{(S_1, S_2, I, P, T, A, R) \in \mathbb{R}_+^7 : 0 < N \leq \frac{\pi}{\mu}\right\}$.

Hence, **Theorem 1** has been proven to be true. ■

Equilibrium Points

Equilibrium points for the HIV/AIDS disease spread model with public awareness in system (2) are obtained when:

$$\pi + \eta S_2 - \left(\mu + \theta + \frac{\beta I}{N}\right) S_1 = 0, \quad (7)$$

$$\theta S_1 - (\mu + \eta + \omega) S_2 = 0, \quad (8)$$

$$\frac{\beta S_1 I}{N} - (\mu + \delta) I = 0, \quad (9)$$

$$\alpha_2 \delta I - (\mu + \gamma) P = 0, \quad (10)$$

$$\alpha_1 \delta I + m\gamma P + \rho A - (\mu + \sigma) T = 0, \quad (11)$$

$$(1 - m)\gamma P + (1 - \alpha_1 - \alpha_2)\delta I + \sigma T - (\mu + \tau + \rho) A = 0. \quad (12)$$

From the above model, two equilibrium points are obtained, namely:

a. The disease-free equilibrium point is obtained when there is no disease in the population, resulting in $I = 0$. The disease-free equilibrium point is given by:

$$E_1(S_1, S_2, I, P, T, A) = \left(\frac{\pi(\mu + \eta + \omega)}{(\mu + \theta)(\mu + \eta + \omega) - \theta\eta}, \frac{\pi\theta}{(\mu + \theta)(\mu + \eta + \omega) - \theta\eta}, 0,0,0,0 \right)$$

b. The endemic equilibrium point occurs when the infected class is non-zero, signifying the presence of disease within the population. An endemic equilibrium point implies that there are always individuals affected by the disease in the population, resulting in I at the endemic equilibrium point being $I^* > 0$. The endemic equilibrium point is given by:

$$E_2(S_1, S_2, I, P, T, A) = (S_1{}^*, S_2{}^*, I^*, P^*, T^*, A^*)$$

where

$$S_1{}^* = \frac{\pi(\mu+\eta+\omega)N}{\mu N(\mu+\eta+\omega)+\theta N(\mu+\omega)+\beta I^*(\mu+\eta+\omega)}$$

$$S_2{}^* = \frac{\pi\theta N}{\mu N(\mu+\eta+\omega)+\theta N(\mu+\omega)+\beta I^*(\mu+\eta+\omega)}$$

$$I^* = \frac{\beta\pi(\mu+\eta+\omega)-\mu N(\mu+\eta+\omega)(\mu+\delta)-\theta N(\mu+\omega)(\mu+\delta)}{\beta(\mu+\eta+\omega)(\mu+\delta)}$$

$$P^* = \frac{\alpha_2\delta I^*}{(\mu+\gamma)}$$

$$T^* = \left(\frac{1}{(\mu+\sigma)(\mu+\gamma)\big((\mu+\tau+\rho)(\mu+\sigma)-\sigma\rho\big)}\right)(\mu+\gamma)\big((\mu+\tau+\rho)(\mu+\sigma)-\sigma\rho\big)\alpha_1\delta I^* + \big((\mu+\tau+\rho)(\mu+\sigma)-\sigma\rho\big)m\gamma\alpha_2\delta I^* + \rho\big((\mu+\sigma)(1-m)\gamma\alpha_2\delta I^* + (\mu+\sigma)(\mu+\gamma)(1-\alpha_1-\alpha_2)\delta I^* + \sigma(\alpha_1\delta I^*(\mu+\gamma) + m\gamma\alpha_2\delta I^*)\big)$$

$$A^* = \frac{(\mu+\sigma)(1-m)\gamma\alpha_2\delta I^*+(\mu+\sigma)(\mu+\gamma)(1-\alpha_1-\alpha_2)\delta I^*+\sigma(\alpha_1\delta I^*(\mu+\gamma)+m\gamma\alpha_2\delta I^*)}{(\mu+\gamma)\big((\mu+\tau+\rho)(\mu+\sigma)-\sigma\rho\big)}$$

Basic Reproduction Number

Next, determining the basic reproduction number (R_0) of system (2) involves finding the maximum eigenvalue obtained from the Next Generation Matrix. Next, to abbreviate the writing, let's assume $B = (\mu + \gamma)$, $C = (\mu + \sigma)$, $D = (\mu + \tau + \rho)$ $W = (\mu + \delta), X = (\mu + \eta + \omega)$, $Y = (\mu + \theta)$, and $Z = \theta\eta$. The steps for determining the basic reproduction number are as follows:

1. Taking the equations that describe the new infection cases and changes in the infection compartments from system (2), namely I, P, T, and A.
2. Linearizing the subsystem related to infections at the disease-free equilibrium point. This linear system is represented by the Jacobian Matrix (J) as follows:

$$J_{(I,P,T,A)} = \begin{bmatrix} \frac{\beta S_1}{N} - W & 0 & 0 & 0 \\ \alpha_2\delta & -B & 0 & 0 \\ \alpha_1\delta & m\gamma & -C & \rho \\ (1-\alpha_1-\alpha_2)\delta & (1-m)\gamma & \sigma & -D \end{bmatrix}.$$

3. Decomposing the Jacobian Matrix (J) into $J = F - V$, where F is the Transmission matrix and V is the Transition matrix.

$$F = \begin{bmatrix} \frac{\beta\pi X}{N(YX-Z)} & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix},$$

$$V = \begin{bmatrix} W & 0 & 0 & 0 \\ -\alpha_2\delta & B & 0 & 0 \\ -\alpha_1\delta & -m\gamma & C & -\rho \\ -(1-\alpha_1-\alpha_2)\delta & -(1-m)\gamma & -\sigma & D \end{bmatrix}.$$

4. Calculate R_0 using the formula $R_0 = \rho(FV^{-1})$.

   By solving the equation $\det(\lambda I - FV^{-1}) = 0$ or $\left(\lambda - \frac{\beta\pi X}{N(YX-Z)A}\right)\lambda^3 = 0$, we get $\lambda_{1,2,3} = 0$ and $\lambda_4 = \frac{\beta\pi X}{N(YX-Z)W}$. Since the basic reproduction number is obtained from the spectral radius or the largest eigenvalue, we obtain:

$$R_0 = \frac{\beta\pi(\mu+\eta+\omega)}{N\big((\mu+\theta)(\mu+\eta+\omega)-\theta\eta\big)(\mu+\delta)}.$$

Theorem 2. If $R_0 > 1$ then there exists an endemic equilibrium point $E_2 = (S_1{}^*, S_2{}^*, I^*, P^*, T^*, A^*)$.

Proof. To prove that every element in E_2 exists, it will be shown that $I^* > 0$ if and only if $R_0 > 1$.

Given that

$$R_0 = \frac{\beta\pi(\mu+\eta+\omega)}{N\big((\mu+\theta)(\mu+\eta+\omega)-\theta\eta\big)(\mu+\delta)} = \frac{\beta\pi(\mu+\eta+\omega)}{\mu N(\mu+\eta+\omega)(\mu+\delta)+\theta N(\mu+\omega)(\mu+\delta)}.$$

Thus, we obtain

$$\begin{aligned} I^* &= \frac{\beta\pi(\mu+\omega)-\mu N(\mu+\eta+\omega)(\mu+\delta)-\theta N(\mu+\omega)(\mu+\delta)}{\beta(\mu+\eta+\omega)(\mu+\delta)} \\ &= \frac{R_0}{\left(\frac{\beta(\mu+\eta+\omega)(\mu+\delta)}{\mu N(\mu+\eta+\omega)(\mu+\delta)+\theta N(\mu+\omega)(\mu+\delta)}\right)} - \left(\frac{\mu N(\mu+\eta+\omega)(\mu+\delta)+\theta N(\mu+\omega)(\mu+\delta)}{\beta(\mu+\eta+\omega)(\mu+\delta)}\right) \\ &= R_0\left(\frac{\mu N(\mu+\eta+\omega)(\mu+\delta)+\theta N(\mu+\omega)(\mu+\delta)}{\beta(\mu+\eta+\omega)(\mu+\delta)}\right) - \left(\frac{\mu N(\mu+\eta+\omega)(\mu+\delta)+\theta N(\mu+\omega)(\mu+\delta)}{\beta(\mu+\eta+\omega)(\mu+\delta)}\right) \\ &= \left(\frac{\mu N(\mu+\eta+\omega)(\mu+\delta)+\theta N(\mu+\omega)(\mu+\delta)}{\beta(\mu+\eta+\omega)(\mu+\delta)}\right)(R_0-1). \end{aligned}$$

Hence, it follows that $I^* > 0$ if and only if $R_0 > 1$. Therefore, **Theorem 2** has been proven to be correct. ■

Analysis of the Stability of the Disease-Free Equilibrium Point

The stability of the disease-free equilibrium point will be analyzed using eigenvalue analysis of the Jacobian matrix.

Theorem 3. If $R_0 < 1$, then the disease-free equilibrium point is locally asymptotically stable.

Proof. The stability of the disease-free equilibrium point of system (2) will be investigated. The Jacobian Matrix of system (2), obtained through the linearization of the mathematical model of HIV/AIDS disease spread, is as follows:

$$J_{(E_1)} = \begin{bmatrix} -Y & \eta & -\frac{\beta S_1}{N} & 0 & 0 & 0 \\ \theta & -X & 0 & 0 & 0 & 0 \\ 0 & 0 & \frac{\beta S_1}{N} - W & 0 & 0 & 0 \\ 0 & 0 & \alpha_2\delta & -B & 0 & 0 \\ 0 & 0 & \alpha_1\delta & m\gamma & -C & \rho \\ 0 & 0 & (1-\alpha_1-\alpha_2)\delta & H & \sigma & -D \end{bmatrix}.$$

$$\det\big(\lambda I - J_{(E_1)}\big) = 0$$

$$\Leftrightarrow \begin{vmatrix} \lambda+Y & -\eta & \frac{\beta S_1}{N} & 0 & 0 & 0 \\ -\theta & \lambda+X & 0 & 0 & 0 & 0 \\ 0 & 0 & \lambda-\left(\frac{\beta S_1}{N}-A\right) & 0 & 0 & 0 \\ 0 & 0 & -\alpha_2\delta & \lambda+B & 0 & 0 \\ 0 & 0 & -\alpha_1\delta & -m\gamma & \lambda+C & -\rho \\ 0 & 0 & -(1-\alpha_1-\alpha_2)\delta & -H & -\sigma & \lambda+D \end{vmatrix} = 0.$$

So, we get the characteristic equation of $J_{(E_1)}$ as

$$\left(\lambda-\left(\frac{\beta\pi(\mu+\eta+\omega)}{N\left((\mu+\theta)(\mu+\eta+\omega)-\theta\eta\right)}-(\mu+\delta)\right)\right)(\lambda+\mu+\gamma)PQ=0$$

with

$P=[\lambda^2+(C+D)\lambda+CD-\rho\sigma]$

$Q=[\lambda^2+(X+Y)\lambda+XY-\theta\eta]$

We obtain $\lambda_1=\frac{\beta\pi(\mu+\eta+\omega)}{N\left((\mu+\theta)(\mu+\eta+\omega)-\theta\eta\right)}-(\mu+\delta)=(\mu+\delta)(R_0-1)$ and $\lambda_2=-(\mu+\gamma)$.

Since μ and γ are positive, the real part of λ_2 is negative. Given $R_0<1$, we have $\lambda_1<0$.

The characteristic equations for the other four eigenvalues are as follows:

$P=[\lambda^2+(C+D)\lambda+CD-\rho\sigma]=0,$ (13)

we have $a_{P0}=1, a_{P1}=C+D, a_{P2}=CD-\rho\sigma$.

According to the Routh-Hurwitz criterion [9], all eigenvalues will be negative if $a_{P1}>0$ and $a_{P2}>0$ for a second-degree polynomial.

$a_{P1}=C+D=2\mu+\sigma+\tau+\rho>0,$

$a_{P2}=CD-\rho\sigma=(\mu+\tau+\rho)\mu+(\mu+\tau)\sigma>0.$

Thus, the Routh-Hurwitz criterion is satisfied with $a_{P1}>0$ and $a_{P2}>0$.

$Q=[\lambda^2+(X+Y)\lambda+XY-\theta\eta]=0,$ (14)

we have $a_{Q0}=1, a_{Q1}=X+Y, a_{Q2}=XY-\theta\eta$.

According to the Routh-Hurwitz criterion [9], all eigenvalues will be negative if $a_{Q1}>0$ and $a_{Q2}>0$ for a second-degree polynomial.

$a_{Q1}=X+Y=2\mu+\eta+\omega+\theta>0$

$a_{Q2}=XY-\theta\eta=(\mu+\eta+\omega)\mu+(\mu+\omega)\theta>0$

Hence, the Routh-Hurwitz criterion is satisfied with $a_{Q1}>0$ and $a_{Q2}>0$.

Thus, it has been proven that the disease-free equilibrium point E_1 is locally asymptotically stable. ■

Analysis of the Stability of the Endemic Equilibrium Point

The stability of the endemic equilibrium point will be analyzed using eigenvalue analysis of the Jacobian matrix.

Theorem 4. If $R_0>1$, then the endemic equilibrium point of the disease is locally asymptotically stable.

Proof. The stability of the endemic equilibrium point of the disease in system (2) will be investigated. The Jacobian Matrix of system (2), obtained through the linearization of the mathematical model of HIV/AIDS disease spread, is as follows:

$$J_{(E_2)} = \begin{bmatrix} -\left(Y + \frac{\beta I^*}{N}\right) & \eta & -\frac{\beta {S_1}^*}{N} & 0 & 0 & 0 \\ \theta & -X & 0 & 0 & 0 & 0 \\ \frac{\beta I^*}{N} & 0 & \frac{\beta {S_1}^*}{N} - A & 0 & 0 & 0 \\ 0 & 0 & \alpha_2\delta & -B & 0 & 0 \\ 0 & 0 & \alpha_1\delta & m\gamma & -C & \rho \\ 0 & 0 & G & H & \sigma & -D \end{bmatrix}.$$

$$\det\left(\lambda I - J_{(E_2)}\right) = 0,$$

$$\Leftrightarrow \begin{vmatrix} \lambda + Y + \frac{\beta I^*}{N} & -\eta & \frac{\beta {S_1}^*}{N} & 0 & 0 & 0 \\ -\theta & \lambda + X & 0 & 0 & 0 & 0 \\ -\frac{\beta I^*}{N} & 0 & \lambda - \left(\frac{\beta {S_1}^*}{N} - A\right) & 0 & 0 & 0 \\ 0 & 0 & -\alpha_2\delta & \lambda + B & 0 & 0 \\ 0 & 0 & -\alpha_1\delta & -m\gamma & \lambda + C & -\rho \\ 0 & 0 & -G & -H & -\sigma & \lambda + D \end{vmatrix} = 0.$$

So, we get the characteristic equation of $J_{(E_2)}$ as $(\lambda + \mu + \gamma)PQ = 0$, with

$$P = \left[\lambda^3 + \left(A + X + Y + \left(\frac{\beta I^*}{N}\right) - \left(\frac{\beta {S_1}^*}{N}\right)\right)\lambda^2 + \left(AX + XY + AY + \left(\frac{\beta I^*}{N}\right)A + \left(\frac{\beta I^*}{N}\right)X - \left(\frac{\beta {S_1}^*}{N}\right)X - \left(\frac{\beta {S_1}^*}{N}\right)Y - \theta\eta\right)\lambda + \left(AXY + \left(\frac{\beta I^*}{N}\right)AX + \theta\eta\left(\frac{\beta {S_1}^*}{N}\right) - \left(\frac{\beta {S_1}^*}{N}\right)XY - \theta\eta A\right)\right]$$

$$Q = [\lambda^2 + (C + D)\lambda + CD - \rho\sigma].$$

We obtain $\lambda_1 = -(\mu + \gamma)$, since μ and γ are positive, the real part of this eigenvalue is negative. The characteristic equations for the other five eigenvalues are as follows:

$$P = \left[\lambda^3 + \left(A + X + Y + \left(\frac{\beta I^*}{N}\right) - \left(\frac{\beta {S_1}^*}{N}\right)\right)\lambda^2 + \left(AX + XY + AY + \left(\frac{\beta I^*}{N}\right)A + \left(\frac{\beta I^*}{N}\right)X - \left(\frac{\beta {S_1}^*}{N}\right)X - \left(\frac{\beta {S_1}^*}{N}\right)Y - \theta\eta\right)\lambda + \left(AXY + \left(\frac{\beta I^*}{N}\right)AX + \theta\eta\left(\frac{\beta {S_1}^*}{N}\right) - \left(\frac{\beta {S_1}^*}{N}\right)XY - \theta\eta A\right)\right] = 0. \quad (15)$$

We have

$a_{P0} = 1,$

$a_{P1} = A + X + Y + \left(\frac{\beta I^*}{N}\right) - \left(\frac{\beta {S_1}^*}{N}\right),$

$a_{P2} = AX + XY + AY + \left(\frac{\beta I^*}{N}\right)A + \left(\frac{\beta I^*}{N}\right)X - \left(\frac{\beta {S_1}^*}{N}\right)X - \left(\frac{\beta {S_1}^*}{N}\right)Y - \theta\eta,$

$a_{P3} = AXY + \left(\frac{\beta I^*}{N}\right)AX + \theta\eta\left(\frac{\beta {S_1}^*}{N}\right) - \left(\frac{\beta {S_1}^*}{N}\right)XY - \theta\eta A,$

According to the Lienard-Chipart criterio [10], all eigenvalues will be negative if and only if a_{P1}, a_{P3}, and Δ_2 are positive for a third-degree polynomial.

$$a_{P1} = A + X + Y + \left(\frac{\beta I^*}{N}\right) - \left(\frac{\beta {S_1}^*}{N}\right) = 2\mu + \eta + \omega + \theta + \left(\frac{\beta I^*}{N}\right) > 0,$$

$$a_{P3} = AXY + \left(\frac{\beta I^*}{N}\right)AX + \theta\eta\left(\frac{\beta {S_1}^*}{N}\right) - \left(\frac{\beta {S_1}^*}{N}\right)XY - \theta\eta A = \left(\frac{\beta I^*}{N}\right)(\mu + \delta)(\mu + \eta + \omega) > 0,$$

$$\Delta_2 = \begin{vmatrix} a_{P1} & 1 \\ a_{P3} & a_{P2} \end{vmatrix} = a_{P1}a_{P2} - a_{P3},$$

$$\Delta_2 = (\mu + \eta + \omega)(\mu + \eta + \omega)\mu + (\mu + \eta + \omega)(\mu + \omega)\theta + \left(\frac{\beta I^*}{N}\right)(\mu + \eta + \omega)^2 + (\mu + \theta)(\mu + \eta + \omega)\mu + (\mu + \theta)(\mu + \omega)\theta + \left(\frac{\beta I^*}{N}\right)(\mu + \theta)(\mu + \delta) + \left(\frac{\beta I^*}{N}\right)(\mu + \theta)(\mu + \eta + \omega) + \left(\frac{\beta I^*}{N}\right)(\mu + \eta + \omega)\mu + \left(\frac{\beta I^*}{N}\right)(\mu + \omega)\theta + \left(\frac{\beta I^*}{N}\right)^2(\mu + \delta) + \left(\frac{\beta I^*}{N}\right)^2(\mu + \eta + \omega) > 0.$$

According to Theorem 2, $I^* > 0$ if and only if $R_0 > 1$. Thus, the Lienard-Chipart criterion is satisfied with $a_{P1} > 0$, $a_{P3} > 0$, and $\Delta_2 > 0$.

$$Q = [\lambda^2 + (C + D)\lambda + CD - \rho\sigma] = 0. \quad (16)$$

We have $a_{Q0} = 1$, $a_{Q1} = C + D$, $a_{Q2} = CD - \rho\sigma$.

According to the Routh-Hurwitz criterion [9], all eigenvalues will be negative if $a_{Q1} > 0$ and $a_{Q2} > 0$ for a second-degree polynomial.

$a_{Q1} = C + D = 2\mu + \sigma + \tau + \rho > 0$,

$a_{Q2} = CD - \rho\sigma = (\mu + \tau + \rho)\mu + (\mu + \tau)\sigma > 0$.

Hence, the Routh-Hurwitz criterion is satisfied $a_{Q1} > 0$ and $a_{Q2} > 0$.

The endemic equilibrium point E_2 is locally asymptotically stable when the Routh-Hurwitz criteria are met: a_{P1}, a_{P3}, a_{Q1}, a_{Q2} are positive, and the determinant of the Routh-Hurwitz matrix Δ_2 is positive when $R_0 > 1$.

Thus, it has been proven that the endemic equilibrium point E_2 is locally asymptotically stable. ■

Model Simulation

The simulation of the mathematical model of HIV/AIDS disease spread with public awareness is performed to observe the stability of the disease-free equilibrium point and the endemic equilibrium point using Maple 2021 software, with parameters taken from previous research related to the mathematical model of HIV/AIDS disease.

Table 2. The values of the parameters for the disease-free equilibrium point in system (2)

Parameter	Value	Unit	Reference
N	272.682.500	$individuals$	[11]
π	$3{,}71 \times 10^6$	$\frac{1}{year}$	[11]
μ	0,014	$\frac{1}{year}$	[11]
θ	0,2351	$\frac{1}{year}$	[6]
η	0,015	$\frac{1}{year}$	[12]
β	0,3465	$\frac{1}{year}$	[6]
δ	0,5	$\frac{1}{year}$	[13]
α_1	0,34		[3]
α_2	0,35		[3]
γ	0,067	$\frac{1}{year}$	[4]
m	0,4		[8]
σ	0,01	$\frac{1}{year}$	[14]
ρ	0,4	$\frac{1}{year}$	[15]
τ	0,27	$\frac{1}{year}$	[16]
ω	0,03	$\frac{1}{year}$	[8]

Based on the parameter values in Table 2, the basic reproduction number is obtained $R_0 = 0{,}048444005$. The disease-free equilibrium point is $E_1(S_1, S_2, I, P, T, A) = (19.595.538{,}21; 78.083.237{,}84; 0; 0; 0; 0)$. With initial values $S_1(0) = 93.697.530$,

$S_2(0) = 97.132.470$, $I(0) = 427.201$, $P(0) = 350.000$, $T(0) = 144.632$, $A(0) = 131.147$, and $R(0) = 80.799.520$. The simulation graph is obtained as follows:

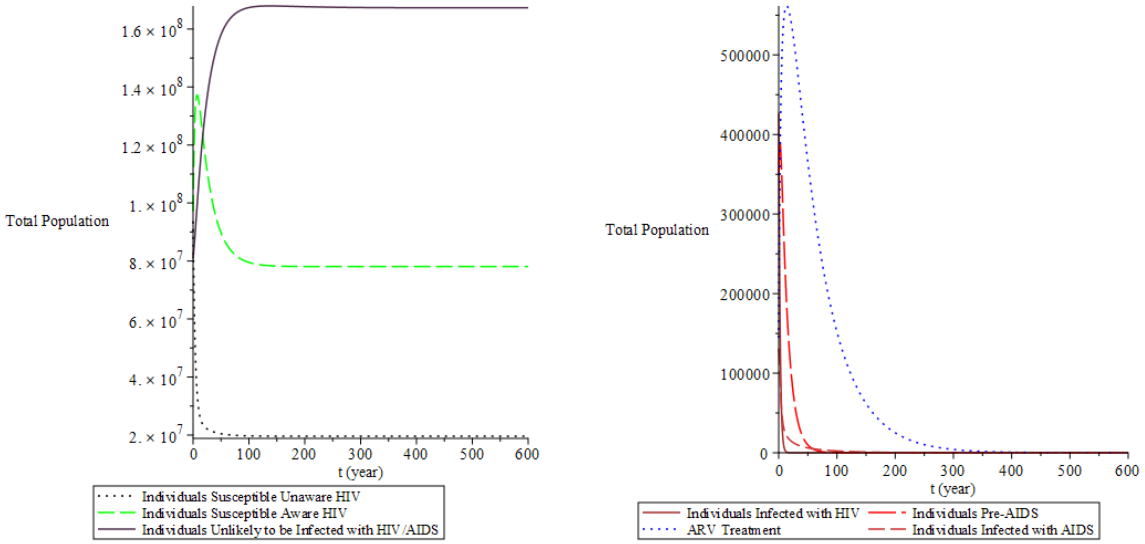

Figure 4. Numerical Simulation Toward the Disease-Free Equilibrium Point

Figure 2 shows that in the 600th year, the population in each sub-population stabilizes towards its disease-free equilibrium point. This is in line with Theorem 3, which states that the disease-free equilibrium point is asymptotically stable if $R_0 < 1$.

Next, a numerical simulation will be conducted for $R_0 > 1$. Based on Table 2, if the parameter value β is increased to 0.95, the parameter value θ is decreased to 0.03, the parameter value η is increased to 0.08, and the parameter value ω is decreased to 0.005 then the basic reproduction number is obtained as $R_0 = 1{,}272751076$. Then the value of the endemic equilibrium point of the disease is obtained as:
$E_2(S_1^*, S_2^*, I^*, P^*, T^*, A^*) =$
$\begin{pmatrix} 147.535.584{,}2 ; 44.707.752{,}79; 1.546.798{,}674; 3.341.848{,}988; 31.472.318{,}10; \\ 1.007.045{,}768 \end{pmatrix}$. With any initial values $S_1(0) = 93.697.530$, $S_2(0) = 97.132.470$, $I(0) = 427.201$, $P(0) = 350.000$, $T(0) = 144.632$, $A(0) = 131.147$, and $R(0) = 80.799.520$. The simulation graph is obtained as follows:

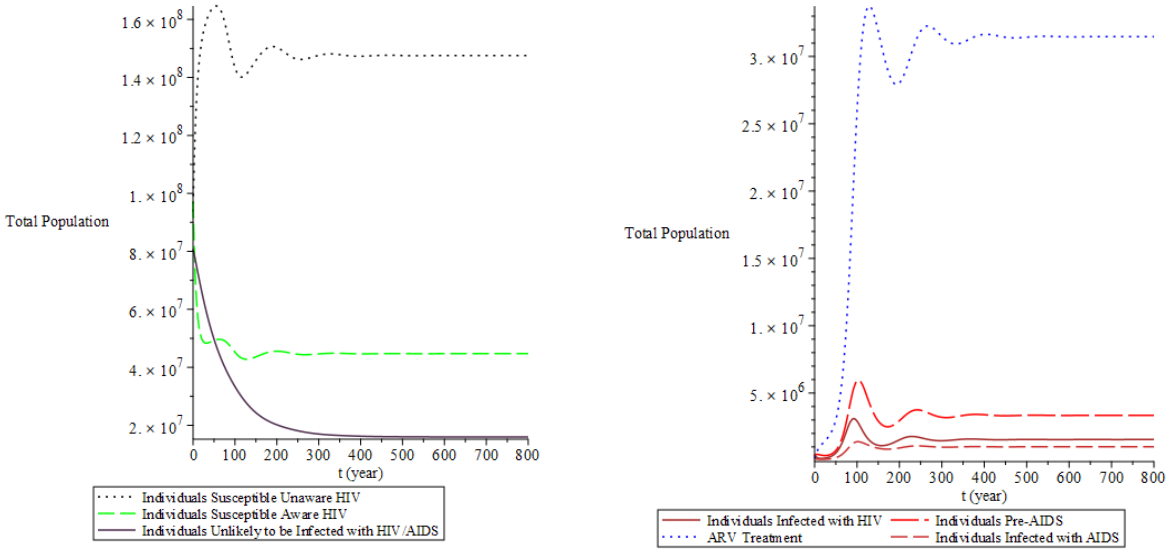

Figure 5. Numerical Simulation Toward the Endemic Equilibrium Point of the Disease

Figure 3 shows that in the 800th year, the population in each sub-population stabilizes towards its endemic equilibrium point. This is in line with Theorem 4, which states that the endemic equilibrium point is asymptotically stable if $R_0 > 1$.

Next, sensitivity analysis is conducted to identify parameters that have the most significant impact on the value of R_0. The parameter that has the greatest influence on R_0 indicates that it has a dominant effect on the spread of HIV/AIDS.

Table 3. Parameter Sensitivity Index

Parameter	Sensitivity Index
$\boldsymbol{\beta}$	$+1{,}000000000$
$\boldsymbol{\pi}$	$+0{,}9999999996$
$\boldsymbol{\delta}$	$-0{,}9727626462$
$\boldsymbol{\theta}$	$-0{,}9260545726$
$\boldsymbol{\eta}$	$+0{,}2354376032$
$\boldsymbol{\mu}$	$-0{,}1760947457$
$\boldsymbol{\omega}$	$-0{,}1605256383$

In Table 3, the sensitivity indices are arranged based on the extent of each parameter's influence on the value of R_0. Positive sensitivity index values indicate that if the parameter is increased while keeping other parameters constant, the value of R_0 will increase. Conversely, if the parameter is decreased while keeping other parameters constant, the value of R_0 will decrease. Negative sensitivity index values indicate that if the parameter is increased while keeping other parameters constant, the value of R_0 will decrease, and if the parameter is decreased while keeping other parameters constant, the value of R_0 will increase.

The sensitivity index for parameter β (the contact rate of susceptible unaware HIV individuals with infected individuals) is the most significant (positive) parameter affecting HIV/AIDS with a sensitivity index value of $+1.000000000$. This means that if the parameter β is increased (or decreased) by 10%, the value of R_0 will increase (or decrease) by 10.00000000%.

The sensitivity index for parameter δ (the rate of transmission from the infected class) is the most significant (negative) parameter affecting HIV/AIDS with a sensitivity index value of -0.9727626462. This means that if the parameter δ is increased (or decreased) by 10%, the value of R_0 will decrease (or increase) by 9.727626462%.

Next, a numerical simulation will be conducted to observe the influence of several parameters that characterize the HIV/AIDS spread model using different values.

1. Influence of HIV-Aware Population

To determine the extent of the influence of the HIV-aware population by changing the parameter value θ (the rate of transition from susceptible unaware HIV individuals to susceptible aware HIV individuals), the results are displayed in the following table:

Table 4. Influence of HIV-Aware Population

$\boldsymbol{\theta}$	$\boldsymbol{R_0}$	Infected Condition
0	0,6551318698	The disease disappears in the 47th year
0,4	0,02936822558	The disease disappears in the 40th year
0,6	0,01987581446	The disease disappears in the 30th year
1	0,01207198779	The disease disappears in the 23th year

Below is the simulation graph from Table 4:

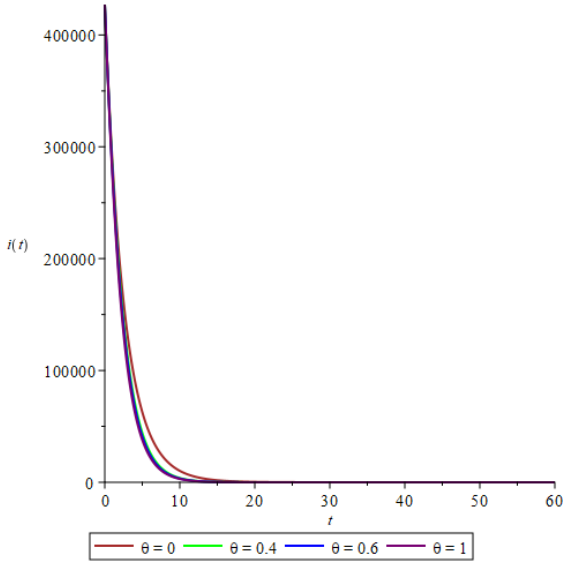

Figure 6. Simulation of point I when $\theta = 0$; $\theta = 0{,}4$; $\theta = 0{,}6$; and $\theta = 1$

Based on Table 4 and Figure 4, the influence of individual awareness parameter on HIV/AIDS disease is apparent; the greater the individual awareness, the faster HIV/AIDS disappears from the population.

2. Influence of Unaware HIV Population

To determine the extent of the influence of the unaware HIV population by changing the parameter value η (the rate of transition from susceptible aware HIV individuals to susceptible unaware HIV individuals), the results are displayed in the following table:

Table 5. Influence of Unaware HIV Population

η	R_0	Infected Condition
0	0,03681993650	The disease disappears in the 31st year
0, 4	0,2459058780	The disease disappears in the 37th year
0, 6	0,3050902327	The disease disappears in the 42nd year
1	0,3836239568	The disease disappears in the 47th year

Below is the simulation graph from Table 5:

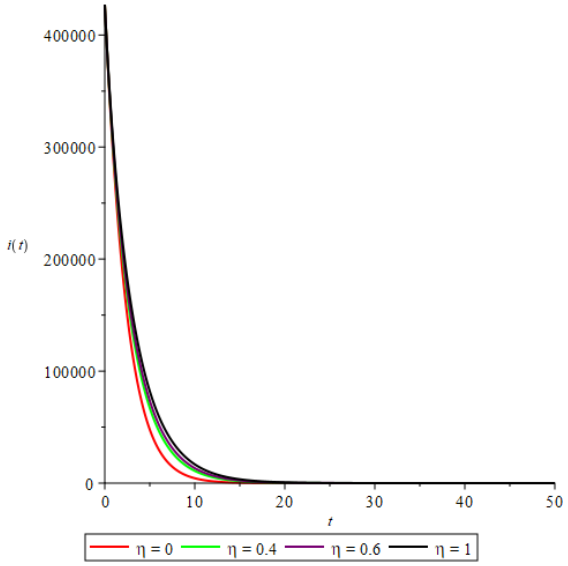

Figure 7. Simulation of point I when $\eta = 0$; $\eta = 0{,}4$; $\eta = 0{,}6$; and $\eta = 1$

Based on Table 5 and Figure 5, the influence of individual unawareness parameter on HIV/AIDS disease is apparent; the greater the individual unawareness, the slower HIV/AIDS disappears from the population.

CONCLUSIONS

Based on the constructed transfer diagram in this study, the S_1S_2IPTAR model is obtained, consisting of Susceptible Unaware (S_1), Susceptible Aware (S_2), Infected with HIV (I), Pre-AIDS (P), ARV Treatment (T), AIDS (A), and unlikely to be infected with HIV/AIDS (R). The formed model has two equilibrium points: the disease-free equilibrium point $E_1 = \left(\frac{\pi(\mu+\eta+\omega)}{(\mu+\theta)(\mu+\eta+\omega)-\theta\eta}, \frac{\pi\theta}{(\mu+\theta)(\mu+\eta+\omega)-\theta\eta}, 0,0,0,0\right)$ and the endemic equilibrium point of the disease $E_2 = ({S_1}^*, {S_2}^*, I^*, P^*, T^*, A^*)$ that exists when $R_0 > 1$. The basic reproduction number R_0 of the model is $R_0 = \frac{\beta\pi(\mu+\eta+\omega)}{N((\mu+\theta)(\mu+\eta+\omega)-\theta\eta)(\mu+\delta)}$. The disease-free equilibrium point is locally asymptotically stable if $R_0 < 1$, meaning the disease will disappear from the population. On the other hand, the endemic equilibrium point is locally asymptotically stable if $R_0 > 1$, indicating that the disease will persist in the population. From the numerical simulation results and sensitivity analysis, it is found that some significant parameters are the contact rate between susceptible individuals and infected individuals (β) and the rate of transmission from the infected class (δ). To control and ultimately eliminate HIV/AIDS from the population, several measures can be taken. These include reducing contact with infected individuals (β), lowering the rate of transmission from the infected class (δ), increasing the rate of transition from unaware susceptible to aware susceptible (θ), and decreasing the rate of transition from aware susceptible to unaware susceptible (η).

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