Dynamics of HIV Transmission with Saturated Incidence and Treatment Strategy

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Abstract. This paper presents a mathematical model for dynamics of HIV transmission by considering a saturated incidence type interaction for the human to human sexual transmission. The equilibria of the model are discovered, and the basic reproduction number is calculated. The analysis shows that if the basic reproduction number is less than unity, the disease-free equilibrium is locally and globally asymptotically stable. It is proved using differential equation theory and a comparison theorem. The Lyapunov function and the LaSalle invariance principle show that the endemic equilibrium is globally asymptotically stable if the basic reproduction number is greater than unity. According to the sensitivity analysis, the effective contact rate was more sensitive to the basic reproduction rate than the treatment rate. The numerical simulations show that as the saturation incidence rate increases, the force of infection decreases. The prevalence of HIV/AIDS decreases as the saturation rate increases.

Keywords: HIV dynamics · basic reproduction number · saturated incidence · stability · treatment

1 Introduction

The development of the HIV/AIDS epidemic in the world has caused HIV/AIDS to become a global problem and is increasingly becoming a public health problem. In order to accelerate the acceleration of efforts to tackle HIV and AIDS in the world, it is very important to integrate prevention efforts with care, support, and treatment, both of which are important components and complement each other. Efforts to provide care, support, and treatment with antiretroviral therapy can be used to slow the spread of the HIV virus in the body. The prevention and control of HIV/AIDS is a public health priority due to the global pandemic and the 27 million fatalities that have occurred since the illness was discovered in 1981([1]). In order to achieve the best results, the strategic approach to combating the HIV epidemic emphasizes the need for more intensive, comprehensive, integrated, and coordinated efforts to prevent and control HIV/AIDS. To reduce the HIV/AIDS epidemic, treatment and prevention are required.

Mathematical modeling is critical in managing and controlling an epidemic of infectious diseases such as HIV/AIDS. A saturated incidence function is preferable for modeling the inhibitory effect of a change in behavior or the crowding effect of an infected

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individual. The rate at which new cases of infection emerge in a community is known as the incidence rate of a disease when researching epidemiology mathematically [2]. The infection incidence rate provides information about the frequency of new illnesses brought on by contact between people who are susceptible and those who are infected. Due to demographic changes or modifications in the behavior of the ill, there are numerous contacts between susceptible individuals and infected individuals that are saturated at high levels, hence the model uses incidence rates of saturation (see [3–6] and the references therein).

Studies [7–9] describe a model of HIV/AIDS screening of uninfected people and treatment (therapy) of those who are screened. And it was found that these actions had an effect on preventing the transmission of HIV/AIDS. This research develops a model of the HIV/AIDS epidemic with a saturated incidence rate and treatment. It is examined whether the endemic equilibrium linked to the basic reproduction number and the particular endemic equilibrium are stable. The analytical results are then validated using numerical simulations that are run after that.

The rest of the paper is organized as follows: Sect. 2 describes the HIV model's formulation, followed the model analysis in Sect. 3. It is demonstrated in this section that both the endemic equilibrium and the disease-free equilibrium are globally stable. Section 4 examines the sensitivity of the basic reproduction number. Section 5 displays the numerical simulations of the model. This section includes a numerical simulation to evaluate the effects of increasing the saturation rate. Finally, all of the results are summarized in Sect. 6.

2 Formulation of the Model

Our HIV/AIDS model includes saturated treatment and incidence rates. We consider the total population who are sexually active at time t, denoted by N(t). In addition to the total population divided into five compartments, $S(t)$ denotes susceptible or HIV negative, $I_1(t)$ denotes HIV positive or individuals in the asymptomatic stage of infection, $I_2(t)$ denotes pre-AIDS individuals, $T(t)$ denotes treated individuals, and $A(t)$ denotes AIDS patients who are not receiving ARV therapy.

The model is based on the following assumptions:

(1) It is hypothesized that susceptible people become infected by sexual interaction with asymptomatic, pre-AIDS, and treated people who have a different transmission rate. The saturated incidence rate is the pace at which an infection spreads.
(2) It is assumed that the incidence rate is saturated and that the mixing of susceptible and infectious is homogeneous.
(3) The susceptible avoid making sexual contact with AIDS patients because they are too sick to engage in sexual activity.
(4) All parameters are nonnegative.

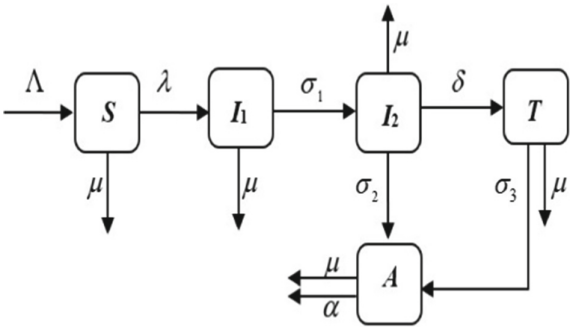


Fig. 1. Schematic diagram of the model (1).

Figure 1 depicts a flow diagram of the dynamics of each compartment. The corresponding systems of differential equations and descriptions of the parameters are given in (1) and Table 1.

$$\begin{aligned}
\frac{dS}{dt} &= \Lambda - \frac{\beta(I_1 + k_1 I_2 + k_2 T)S}{1 + \omega(I_1 + I_2 + T)} - \mu S, \\
\frac{dI_1}{dt} &= \frac{\beta(I_1 + k_1 I_2 + k_2 T)S}{1 + \omega(I_1 + I_2 + T)} - (\sigma_1 + \mu)I_1, \\
\frac{dI_2}{dt} &= \sigma_1 I_1 - (\delta + \sigma_2 + \mu)I_2, \\
\frac{dT}{dt} &= \delta I_2 - (\sigma_3 + \mu)T, \\
\frac{dA}{dt} &= \sigma_2 I_2 + \sigma_3 T - (\alpha + \mu)A,
\end{aligned} \tag{1}$$

With initial conditions of model (1) are

$$S(0) = S^0, I_1(0) = I_1^0,\ I_2(0) = I_2^0,\ T(0) = T^0,\ A(0) = A^0\,. \tag{2}$$

3 Analysis of the Model

3.1 Positivity and Boundedness

It is critical to show that the solutions of model (1) with positive initial conditions (2) are always positive.

Lemma 1. *If* $((S(t),\ I_1(t),\ I_2(t),\ T(t),\ A(t))$ *be a solution of model (1) with nonnegative initial conditions and the feasible region* Ω defined by.

$$\Omega = \left\{ (S,\ I_1, I_2, T, A) \in R_+^5 \;\middle|\; S + I_1 + I_2 + T + A \leq \frac{\Lambda}{\mu} \right\}, \tag{3}$$

then the solution of model (1) is ninnegative for all $t > 0$.

Proof. Since λ is function of I_1, I_2 and T with the initial condition are all positive, let $t_1 = \sup\{t > 0 : S > 0, I_1 > 0, I_2, T > 0, A > 0 \text{ in } [0, t]\}$.

The first equation of the model (1) is expressed as

$$\frac{dS}{dt} = \Lambda - [\lambda(I_1, I_2, T) - \mu]S. \tag{4}$$

Solving the Eq. (4) we get

$$\begin{aligned} &\frac{d}{dt}\left[S(t)\exp\left\{\mu t + \int_0^t \lambda(I_1(u), I_2(u), T(u))du\right\}\right] \\ &= \Lambda \exp\left\{\mu t + \int_0^t \lambda(I_1(u), I_2(u), T(u))du\right\}. \end{aligned} \tag{5}$$

The result is derived by integrating and rearranging both sides of (5) from $t = 0$ to $t = t_1$,

$$\begin{aligned} S(t_1) &= S(0)\exp\left\{-\left(\mu t_1 + \int_0^{t_1} \lambda(I_1(u), I_2(u), T(u))du\right)\right\} \\ &+ \exp\left\{-\left(\mu t_1 + \int_0^{t_1} \lambda(I_1(u), I_2(u), T(u))du\right)\right\} \\ &\times \Lambda \int_0^{t_1} \exp\left\{\mu x + \int_0^x \lambda(I_1(u), I_2(u), T(u))du\right\} dx \\ &> 0, \end{aligned} \tag{6}$$

The same reasoning is used to show that $I_1 > 0$, $I_2 > 0$, $T > 0$, and $A > 0$ for all $t > 0$. The proof of Lemma 1 is completed. ■

Lemma 2. *The closed region* Ω *defined above is positively invariant set for the model (1) with nonnegative initial conditions in* R_+^5.

Proof. Here $N = S + I_1 + I_2 + T + A$. The rate of change of the total population N is obtained from equation of the model (1) gives

$$\frac{dN}{dt} = \Lambda - \mu N - \alpha A \leq \Lambda - \mu N.$$

Using a comparison theorem on differential inequalities from Lakshmikantham et al. [13] it can be shown that

$$N(t) \leq N(0)e^{-\mu t} + \frac{\Lambda}{\mu}(1 - e^{-\mu t}). \tag{7}$$

This follows $N(t) \leq \Lambda/\mu$ if $N(0) \leq \Lambda/\mu$. Further, if $N(0) > \Pi/\mu$, then either the solution enters Ω or $N(t)$ reduces asymptotically to Λ/μ as $t \to \infty$. Thus, the closed region Ω is positively invariant. ■

Table 1. Parameters of the HIV model.

Parameter	Description	Values	References
Λ	Recruitment rate of susceptible	5000	Assumed
β	The effective contact rate	0.00008	Assumed
k_1	The relative infectiousness of persons in the P class	0.15	[1]
k_2	The relative infectiousness of persons in the T class	0.0016	[1]
δ	Pre-AIDS treatment rates	0.57	Assumed
σ_1	The rate at which infected patients progress to pre-AIDS status	0.57	[1]
σ_2	The rate at which pre-AIDS patients advance to AIDS patients	0.22	[1]
σ_3	The rate at which treated persons progress to AIDS patients	0.18	[1]
μ	Natural mortality rate	0.0196	[10]
α	AIDS-induced death rate	0.33	[1]
ω	Psychological or inhibiting effect measurement	4	[1]

3.2 Disease-Free Equilibrium and Basic Reproduction Number

The disease-free equilibrium of the model (1) is obtained by using $I_1 = I_2 = T = A = 0$ in the steady state conditions. It is easy to see that the disease-free equilibrium of the model (1) is given by

$$E_0 = \left(S^0,\ I_1^0,\ I_2^0,\ T^0,\ A^0\right) = \left(\frac{\Lambda}{\mu},\ 0,\ 0,\ 0,\ 0\right). \tag{8}$$

To determine the basic reproduction number of the model (1), we will use the next generation matrix approach described by van den Driessche and Watmough [11]. Let $X = (I_1,\ I_2,\ T,\ A,\ S)^T$. We transform model (1) into a matrix

$$\frac{dX}{dt} = \mathcal{F}(X) - \mathcal{V}(X) \tag{9}$$

where

$$\mathcal{F}(\mathrm{X}) = \begin{bmatrix} \lambda S \\ 0 \\ 0 \\ 0 \\ 0 \end{bmatrix},\ \mathcal{V}(X) = \begin{bmatrix} (\sigma_1 + \mu) I_1 \\ -\sigma_1 I_1 + (\delta + \sigma_2 + \mu) I_2 \\ -\delta I_2 + (\sigma_3 + \mu) T \\ -\sigma_2 I_2 - \sigma_3 T + (\alpha + \mu) A \\ -\Lambda + \lambda S + \mu S \end{bmatrix}.$$

The Jacobian matrices F and V at the disease-free equilibrium E_0 are given by

$$\begin{aligned} F &= \begin{bmatrix} \frac{\beta\Lambda}{\mu} & \frac{\beta k_1\Lambda}{\mu} & \frac{\beta k_2\Lambda}{\mu} & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix}, \\ V &= \begin{bmatrix} Q & 0 & 0 & 0 \\ -\sigma_1 & K & 0 & 0 \\ 0 & -\delta & L & 0 \\ 0 & -\sigma_2 & -\sigma_3 & M \end{bmatrix}, \end{aligned} \tag{10}$$

with

$$Q = \sigma_1 + \mu,\ K = \delta + \sigma_2 + \mu, L = \sigma_3 + \mu,\ M = \alpha + \mu.$$

The basic reproduction number of the model (1) will be the spectral radius of the matrix FV^{-1} designated and defined as (1)

$$R_0 = \frac{\beta\Lambda}{\mu Q} + \frac{\beta\Lambda k_1\sigma_1}{\mu QK} + \frac{\beta\Lambda k_2\delta\sigma_1}{\mu QKL}. \tag{11}$$

Here, the basic reproduction number shows how many new infections one HIV-positive person typically causes in a population when treatment is being used as a control.

3.3 Stability of Disease-Free Equilibrium

The following theorem both locally and globally stablely expresses the disease-free equilibrium of model (1).

Theorem 1. *In the model (1), the disease-free equilibrium,* E_0 *, is locally asymptotically stable if* $R_0 < 1$ *and unstable if* $R_0 > 1$.

Proof. The Jacobian matrix of the model (1) at the disease-free equilibrium E_0 is given by

$$J_0 = \begin{pmatrix} -\mu & -\frac{\beta\Lambda}{\mu} & -\frac{\beta\Lambda k_1}{\mu} & -\frac{\beta\Lambda k_2}{\mu} & 0 \\ 0 & -Q + \frac{\beta\Lambda}{\mu} & \frac{\beta\Lambda k_1}{\mu} & \frac{\beta\Lambda k_2}{\mu} & 0 \\ 0 & \sigma_1 & -K & 0 & 0 \\ 0 & 0 & \delta & -L & 0 \\ 0 & 0 & \sigma_2 & \sigma_3 & -M \end{pmatrix}. \tag{12}$$

The characteristic roots corresponding to matrix J_0 are $\lambda_1 = -\mu$, $\lambda_2 = -M$ and the remaining three roots of the characteristic roots of the matrix J_1

$$J_1 = \begin{pmatrix} -Q + \frac{\beta\Lambda}{\mu} & \frac{\beta\Lambda k_1}{\mu} & \frac{\beta\Lambda k_2}{\mu} \\ \sigma_1 & -K & 0 \\ 0 & \delta & -L \end{pmatrix}. \tag{13}$$

The matrix J_2 is reduced to echelon form using an elementary row operation, giving us the results shown below.

$$J_2 = \begin{pmatrix} -j_{11} & \frac{\beta\Lambda k_1}{\mu} & \frac{\beta\Lambda k_2}{\mu} \\ 0 & -j_{22} & -\frac{\beta\Lambda k_2\sigma_1}{\beta\Lambda - Q\mu} \\ 0 & \delta & -j_{33} \end{pmatrix} \tag{14}$$

where

$$j_{11} = \frac{Q\mu - \beta\Lambda}{\mu} = Q\left(1 - \frac{\beta\Lambda}{Q\mu}\right),\ j_{22} = \frac{K\left(1 - \frac{\beta\Lambda}{Q\mu} - \frac{\beta\Lambda k_1\sigma_1}{KQ\mu}\right)}{1 - \frac{\beta\Lambda}{Q\mu}},$$
$$j_{33} = \frac{L(1 - R_0)}{1 - \frac{\beta\Lambda}{Q\mu}}.$$

The matrix J_0 has five negative characteristic roots because $R_0 = \frac{\beta\Lambda}{\mu Q} + \frac{\beta\Lambda k_1\sigma_1}{\mu QK} + \frac{\beta\Lambda k_2\delta\sigma_1}{\mu QKL} < 1$ implies $1 > \frac{\beta\Lambda}{\mu Q}$, ,
$1 > \frac{\beta\Lambda}{\mu Q} + \frac{\beta\Lambda k_1\sigma_1}{\mu QK}$, and $1 > \frac{\beta\Lambda}{\mu Q} + \frac{\beta\Lambda k_1\sigma_1}{\mu QK} + \frac{\beta\Lambda k_2\delta\sigma_1}{\mu QKL}$.

Therefore, all of the Jacobi matrix's eigenvalues J_0 are negative, which implies if $R_0 < 1$, the model (1)'s disease-free equilibrium of is locally asymptotically stable. We obtain the relation $j_{33} > 0$ if $R_0 > 1$. As a result, the matrix J_0 has at least one eigenvalues with a positive real part. Thus, the disease-free equilibrium E_0 is unstable.

In the following theorem, we looked at the global stability of the disease-free equilibrium E_0 and how to create an endemic equilibrium E_1 using the Lyapunov function.

Theorem 2. *The disease-free equilibrium E_0 of the model (1) is globally asymptotically stable if $R_0 < 1$.*

Proof. We apply a comparison theorem that takes the approach from Khan et al. [12] to demonstrate the global stability of the disease-free equilibrium of E_0. In model (1), the rate of change of the variables (I_1, I_2, T, and A) or the differential equation of the infected individuals can be written as.

$$\begin{pmatrix} \dot{I}_1 \\ \dot{I}_2 \\ \dot{V} \\ \dot{T} \\ \dot{A} \end{pmatrix} = (F - V)\begin{pmatrix} I_1 \\ I_2 \\ T \\ A \end{pmatrix} - \frac{\beta(I_1 + k_1 I_2 + k_2 T)}{1 + \omega(I_1 + I_2 + T)}$$
$$\times \begin{pmatrix} \omega(I_1 + I_2 + T)S^0 + (S^0 - S) \\ 0 \\ 0 \\ 0 \end{pmatrix}, \tag{15}$$

where F and V as described in (10) and (11).

If $S \leq S^0$ for all $t \geq 0$, we have

$$\begin{pmatrix} \dot{I}_1 \\ \dot{I}_2 \\ \dot{T} \\ \dot{A} \end{pmatrix} \leq (F - V) \begin{pmatrix} I_1 \\ I_2 \\ T \\ A \end{pmatrix}. \tag{16}$$

Hence,

$$F - V = \begin{bmatrix} \frac{\beta\Lambda}{\mu} - Q & \frac{\beta k_1 \Lambda}{\mu} & \frac{\beta k_2 \Lambda}{\mu} & 0 \\ \sigma_1 & -K & 0 & 0 \\ 0 & \delta & -L & 0 \\ 0 & \sigma_2 & \sigma_3 & -M \end{bmatrix}. \tag{17}$$

There are four eigenvalues of the matrix $F - V$, one eigenvalue is $-M$ and the other eigenvalues are obtained by reducing the matrix $F - V$ to a matrix 3×3 and are obtained matrix J_1 as in (13). From the local stability results in Theorem 1, all eigenvalues of the matrix J_0 have a negative real part. Thus, model (1) is stable if $R_0 < 1$. Thus, $(I_1, I_2, T, A) \to (0, 0, 0, 0)$ as $t \to \infty$. The comparison theorem ([13]) leads to the conclusion that $(I_1, I_2, T, A) \to (0, 0, 0, 0)$ and $S \to \frac{\Lambda}{\mu}$ if $t \to \infty$. ■

3.4 Existence and Stability of the Endemic Equilibrium

Let $E_1 = \left(S^*, I_1^*, I_2^*, T^*, A^*\right)$ represents any endemic equilibrium of the model (1). Solving the equations of the model at steady states, the following components of E_1 are given:

$$\begin{aligned} S^* &= \frac{\Lambda}{\lambda^* + \mu}, \; I_1^* = \frac{\lambda^* \Lambda}{Q(\lambda^* + \mu)}, \; I_2^* = \frac{\sigma_1 I_1^*}{K}, \\ T^* &= \frac{\sigma_1 \delta I_1^*}{KL}, \; A^* = \frac{(\sigma_1 \sigma_2 L + \sigma_3 \sigma_1 \delta) I_1^*}{KLM}, \end{aligned} \tag{18}$$

where

$$\lambda^* = \frac{Q\mu}{\Lambda\omega(1 + w_1 + w_2) + Q}(R_0 - 1). \tag{19}$$

The globally asymptotic stability of the endemic equilibrium is proved below.

Theorem 3. *If $R_0 > 1$, then the endemic equilibrium E_1 of the model (1) is globally asymptotically stable.*

Proof. Let $R_0 > 1$ be such that the endemic equilibrium $E_1 = (S^*, I_1^*, I_2^*, T^*, A^*)$ exists, and construct the following Lyapunov function V as:

$$\begin{aligned} V(t) = \ln\big[&\left(S - S^*\right) + \left(I_1 - I_1^*\right) + \left(I_2 - I_2^*\right) + \left(T - T^*\right) \\ &+ \left(A - A^*\right) + 1\big]. \end{aligned} \tag{20}$$

Differentiating $V(t)$ with respect to t along the solutions of model (1), we get

$$\begin{aligned}\frac{dV}{dt} &= \frac{\partial V}{\partial S}\frac{dS}{dt} + \frac{\partial V}{\partial I_1}\frac{dI_1}{dt} + \frac{\partial V}{\partial I_2}\frac{dI_2}{dt} + \frac{\partial V}{\partial T}\frac{dT}{dt} + \frac{\partial V}{\partial A}\frac{dA}{dt} \\ &= \frac{1}{Z}\left(\frac{dS}{dt} + \frac{dI_1}{dt} + \frac{dI_2}{dt} + \frac{dT}{dt} + \frac{dA}{dt}\right) \\ &= \frac{1}{Z}\frac{dN}{dt}.\end{aligned} \tag{21}$$

where

$Z = (S - S^*) + (I_1 - I_1^*) + (I_2 - I_2^*) + (T - T^*) + (A - A^*) + 1$
$= N - N^* + 1$.

From Eq. (21), all the solutions of the model (1) satisfy $S^* + I_1^* + I_2^* + T^* + A^* = \frac{\Lambda}{\mu}$, and $\frac{dN}{dt} \leq \frac{\Lambda}{\mu}$. Thus, $V = \ln(N - N^* + 1) \geq 0$ and

$$\begin{aligned}\frac{dV}{dt} &= \frac{1}{N - N^* + 1}\frac{dN}{dt} \\ &\leq \frac{1}{N - (\Lambda/\mu) + 1}(\Lambda - \mu N) \\ &= \frac{\mu}{N - (\Lambda/\mu) + 1}\left(\frac{\Lambda}{\mu} - N\right) \\ &= -\mu\left(\frac{1}{N - (\Lambda/\mu)}\right)\left(N - \frac{\Lambda}{\mu}\right) \\ &= -\mu < 0.\end{aligned}$$

Also, $dV/dt = 0$ if and only if $S = S^*$, $I_1 = I_1^*, I_2 = I_2^*, T = T^*$, and $A = A^*$. The maximum invariant set of mode; (1) on the set $\left\{(S, I_1, I_2, T, A) \in \Omega : \frac{dV}{dt} = 0\right\}$ is the singleton $\{E_1\}$. Then by LaSalle's Invariance Principle [14], the endemic equilibrium E_1 is globally asymptotically stable in the interior of Ω for $R_0 > 1$. ■

4 Sensitivity Analysis

The basic reproduction rate (R_0) of model (1) depends on nine parameters, namely, β, Λ μ, δ, η_1, η_2, σ_1, σ_2, and σ_3. Among these parameters, we cannot control the parameters Λ and μ through routine measurements in a community. The basic reproduction rate R_0 is proportional to the initial disease transmission. This sensitivity analysis was carried out by calculating the sensitivity index of the model parameters using the approach of Chitnis *et al.* [15] and Marsudi *et al.* [16]. The sensitivity index of the basic reproduction number R_0, which depends on a parameter θ, is defined as

$$\Upsilon_\theta^{R_0} = \frac{\partial R_0}{\partial \theta}\frac{\theta}{R_0}. \tag{22}$$

Notice that if $\Upsilon_\theta^{R_0} = +k$ $(0 < k < 1)$ implies an increase (or decrease) of θ by10%, increase (or decrease) R_0 by $k \times 10\%$. On the other hand, $\Upsilon_\theta^{R_0} = -k$ implies increase (or decrease) of θ by10%, decrease (or increase) R_0 by $k \times 10\%$.

5 Numerical Simulations

In this section, we investigate the numerical behavior of the HIV/AIDS model (1) using the Runge-Kutta method with the parameter values shown in Table 1 and the initial values as follows (Marsudi *et al.* [16, 17]).

$$\begin{aligned} &S(0) = 2912403, I(0) = 2408, P(0) = 539, \\ &T(0) = 519, A(0) = 1024. \end{aligned} \tag{23}$$

The numerical simulations are conducted using MATLAB and the results are presented below.

5.1 Numerical Simulation of the Model

We present some simulations using the parameter values in Table 1 to illustrate the analytic results obtained above.

First, we choose $\beta = 0.0000015$, numerical simulation gives $R_0 = 0.7196 < 1$ and the disease-free equilibrium $E_0 = (2.551020 \times 10^5,\ 0,\ 0,\ 0,\ 0)$ is globally asymptotically stable (see Fig. 2). Figure 2 shows how, as time increases infinity, all solution trajectories converge to the disease-free equilibrium. Figure 2 depicts the proof of the disease-free equilibrium from Theorem 2 using the following initial conditions: (a) N_1(260000, 45, 30, 50, 35), (b) N_2(255000, 40, 25, 45, 40), (c) N_3 (250000, 35, 15, 30, 25), and (d) N_4 (245000, 20, 10, 15, 10), respectively.

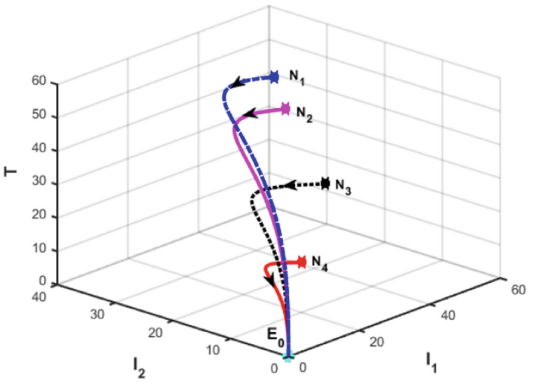
Second, we choose $\beta = 0.00008$, numerical simulation gives $R_0 = 38.251441 > 1$ and the endemic equilibrium $E_1 = (2.550267 \times 10^5,\ 2.50,\ 1.69, 5.11,\ 3.69)$ is globally asymptotically stable (see Fig. 3). Figure 3 depicts the infinite-time convergence of all solution trajectories to the endemic equilibrium. So, using the initial conditions: (a) N_1(260000, 45, 30, 50, 35), (b) N_2(255000, 40, 25, 45, 40), (c) N_3 (250000, 35, 15, 30, 25), and (d) N_4 (245000, 20, 10, 15, 10), respectively, Fig. 3 depicts the proof of the endemic equilibrium from Theorem 3.

5.2 Sensitivity Analysis of R0

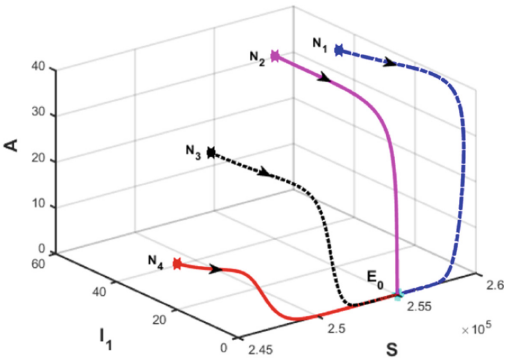
We calculated the sensitivity index with respect to each parameter, and the results are shown in Table 2. The parameters are ordered from most sensitive to least sensitive. As shown in Table 2, the effective contact rate (β) has the highest sensitivity index ($\Upsilon_{\beta}^{R_0} = 1$), which means that any increase (or decrease) of 10% in β will be followed by a increase (or decrease) of 10% in R_0.

The second highest sensitivity index is associated with the rate of progression from asymptomatic infection to pre-AIDS (σ_1). The sensitivity index of the rate of progression of infectives individuals to pre-AIDS individuals is -0.868613. In this case, shows that any increase (or decrease) of 10% in σ_1 will be followed by a decrease (or increase) of 8.68613% in R_0. The interpretation of the sensitivity index for the additional parameters is as follows: β and σ_1.

The sensitivity index of HIV/AIDS treatment rate is -0.066197, indicating that any increase (or decrease) of 10% in σ_1 will be followed by a decrease (or increase) of 0.66197% in R_0.



(a)

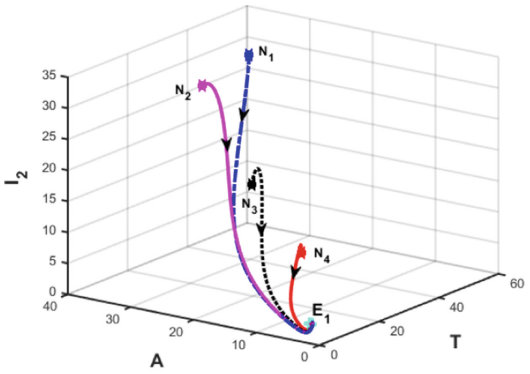


(b)

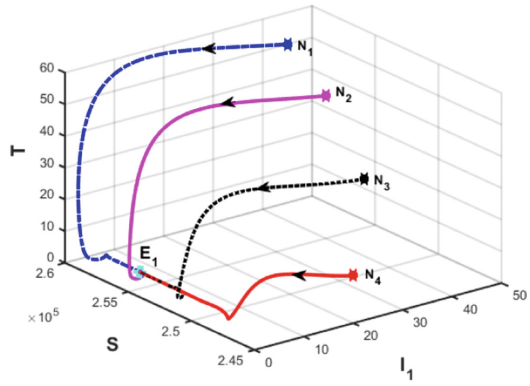
Fig. 2. Globally stable behavior of the model (1) at the endemic equilibrium E_0

5.3 Number of Active Cases for the Saturated Incidence

The psychological or inhibitory effect parameter (ω) is used to measure the saturation impact, and the basic reproduction number (R_0) does not explicitly depend on the saturation rate. Figure 4 depicts a numerical simulation of the effects of increasing the saturation rate. Figure 4(a) shows that increasing the saturation incidence rate reduces the number of active cases for the saturated incidence. The force of infection (λ) decreased as the saturation rate increased but did not reach zero, implying that the disease became endemic in the population despite the impact of the treatment intervention (Fig. 4(b)).



(a)



(b)

Fig. 3. Globally stable behavior of the model (1) at the endemic equilibrium E_1

Table 2. Sensitivity Indices of R_0

Parameter	Sensitivity indices
β	+ 1.000000
σ_1	-0.868613
η_1	+ 0.095243
δ	-0.066197
σ_2	-0.026669
η_2	+ 0.002901
σ_3	-0.002616

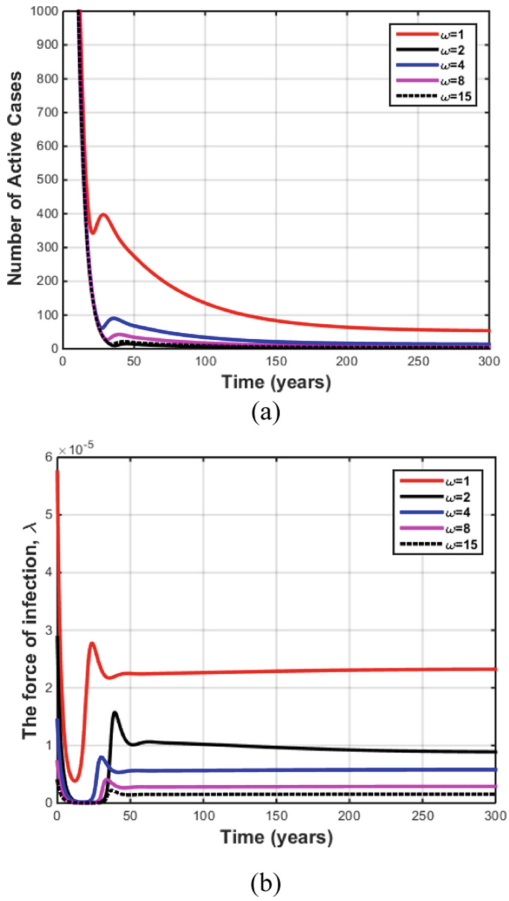


Fig. 4. Number of active cases of the disease and contact rate for the HIV/AIDS model with saturated incidence and treatment.

6 Conclusion

In this paper, we have developed and analyzed a mathematical model for dynamics of HIV transmission by considering a saturated incidence type interaction for the human to human sexual transmission including saturation incidence rates and treatment. There are five state variables and eleven parameters in the model. The model is well-posed and exists in a tractable area. We calculated the basic reproduction number, which is made up of three parts: asymptomatic infectives, pre-AIDS individuals, and treated individuals. The model has two equilibrium states. Theoretical results show that when the basic reproduction number is less than unity, the disease-free equilibrium is stable (locally and globally). If the basic reproduction number is greater than one, the endemic is asymptotically stable both locally and globally. The Lyapunov function and the LaSalle invariance principle were used to demonstrate that the disease-free equilibrium and the endemic equilibrium are globally asymptotically stable. Our analytical results are

supported by numerical simulations. The effective contact rate is the most sensitive index to the fundamental reproduction rate, followed by the rate of progression from asymptomatic infection to pre-AIDS. Finally, numerical simulations show that as the saturation rate increases, the force of infection decreases. The prevalence of HIV/AIDS decreases as the saturation rate increases.

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References

1. E. L. Kateme, J. M. Tchuenche, J. M., S. D. Hove-Musekwa, HIV/AIDS Dynamics with Three Control Strategies: The Role of Incidence Function, *ISRN Applied Mathematics*, vol. 2012, 864795, 2012. https://doi.org/10.5402/2012/864795
2. J. Zhang, Z. Ma, Global dynamics of an SEIR epidemic model with saturating contact rate, *Mathematical Biosciences*, vol. 185, no. 1, pp. 15-32, 2003. DOI: https://doi.org/10.1016/s0025-5564(03)00087-7
3. V. Capasso, G. Serio, A generalization of the Kermack-McKendrick deterministic epidemic model, *Mathematical biosciences*, vol. 42, no. 1-2, pp. 43-61, 1978. https://doi.org/10.1016/0025-5564(78)90006-8
4. Z. Jiang, J. Wei, Stability and bifurcation analysis in a delayed SIR model, *Chaos, Solitons & Fractals*, vol. 35, no. 3, pp. 609-619, 2008. https://doi.org/10.1016/j.chaos.2006.05.045
5. S. Ruan, W. Wang, Dynamical behavior of an epidemic model with a nonlinear incidence rate, *Journal of Differential Equations*, vol. 188, no. 1, pp. 135-163, 2003. https://doi.org/10.1016/S0022-0396(02)00089-X
6. O. Sharomi, C. N. Podder, A. B. Gumel, E. H. Elbasha, J. Watmough, Role of incidence function in vaccine-induced backward bifurcation in some HIV models, *Mathematical Biosciences*, vol. 210, no. 2, pp. 436-463, 2007. DOI: https://doi.org/10.1016/j.mbs.2007.05.012
7. R. Safiel, E. S. Massawe, O. D. Makinde, Modelling the effect screening and treatment on transmission of HIV/AIDS infection in a population, *American Journal of Mathematics and Statistics*, vol. 2, no. 4, pp. 75-88, 2012. DOI:https://doi.org/10.5923/J.AJMS.20120204.03
8. K. O. Okosun, O. D. Makinde, O. D., I. Takaidza, Impact of optimal control on the treatment of HIV/AIDS and screening of unaware infectives, *Applied Mathematical Modelling*, vol. 37, no. 6, pp. 3802–3820, 2013. https://doi.org/10.1016/j.apm.2012.08.004
9. H. F. Huo, L. X. Feng, Global stability for an HIV/AIDS epidemic model with different latent stages and treatment, *Applied Mathematical Modelling*, vol. 37, no. 3, pp. 1480-1489, 2013. https://doi.org/10.1016/j.apm.2012.04.013
10. Z. Mukandavire, W. Garira, C. Chiyaka, Asymtotic properties of an HIV/AIDS model with a time delay, *Journal of Mathematical Analysis and Applications,* vol. 330, no. 2, pp. 916-933, 2007. https://doi.org/10.1016/j.jmaa.2006.07.102
11. P. van den Driessche, J. Watmough, Reproduction numbers and sub-threshold endemic equilibria for compartmental models of disease transmission, *Mathematical biosciences*, vol. 180, no. 1-2, pp. 29-48, 2002. DOI: https://doi.org/10.1016/s0025-5564(02)00108-6

12. A. Khan, M. Waleed, M. Imran, Mathematical analysis of an influenza epidemic model, formulation of different controlling strategies using optimal control and estimation of basic reproduction number, *Mathematical and Computer Modelling of Dynamical Systems*, vol. 21, no. 5, pp. 432-459, 2015. DOI:https://doi.org/10.1080/13873954.2015.1016975
13. V. Lakshmikantham, S. Leela, A. A. Martynyuk, *Stability analysis of nonlinear systems,* M. Dekker, New York, 1989. DOI:https://doi.org/10.1007/978-3-319-27200-9
14. J. P. LaSalle, The stability of dynamical systems, CBMS-NSF Reg. In Conf. Ser. Appl. Math., SIAM, Philadelphia. 1976. https://doi.org/10.1137/1.9781611970432
15. N. Chitnis, J. M. Hyman, J. M. Cushing, Determining Important Parameters in the Spread of Malaria through the Sensitivity Analysis of a Mathematical Model, *Bulletin of Mathematical Biology,* vol. 70, no. 5, pp. 1272-1296, 2008. https://doi.org/10.1007/s11538-008-9299-0.
16. Marsudi, Trisilowati, A. Suryanto, and I. Darti, Global Stability and Optimal Control of an HIV/AIDS Epidemic Model with Behavioral Change and Treatment. *Engineering Letters* **29** (2), 575–591 (2021). Engineering Letters 29(2):575–591
17. Marsudi, Marjono, A. Andari, Sensitivity Analysis of Effect of Screening and HIV Therapy on the Dynamics of Spread of HIV, *Applied Mathematical Sciences*, vol. 8, no. 155, pp. 7749–7763, 2014. DOI: https://doi.org/10.12988/ams.2014.49737