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Original Article

Stability and Sensitivity Analysis of HIV/AIDS Model with Saturated Incidence Rate

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Abstract

HIV/AIDS, an extremely harmful sexually transmitted disease, has had a significant impact on worldwide health, establishing itself as one of the most lethal epidemics ever recorded. In this study, a mathematical model is used to analyze how HIV/AIDS spreads and grows, taking into account high incidence rates. The model uses a set of typical differential equations to group people into different categories based on their health status, including those who are vulnerable, those who have been vaccinated, those who are asymptomatic, those who are symptomatic, and those who have AIDS. The effectiveness of the solution indicates that the model is clearly outlined and has important implications for epidemiology. By utilizing the next-generation matrix method, we calculated the basic reproduction number. In order to evaluate the model's stability, a comprehensive examination was conducted on both the local and global stability of both the disease-free and endemic equilibrium points. This analysis provides a comprehensive understanding of the model's behavior, shedding light on the conditions necessary for the disease to persist or die out. Numerical simulations focusing on these key parameters demonstrate that achieving a disease-free environment is attainable, albeit requiring targeted interventions to maintain stability. This study underscores the significance of understanding saturated incidence rates in modeling HIV/AIDS transmission dynamics. The results offer important information for policymakers and public health authorities, allowing them to create successful tactics for managing the transmission of HIV/AIDS.

Keywords: HIV/AIDS, Saturated Incidence Rate, Sensitivity Analysis, Stability Analysis.

1. Introduction

HIV/AIDS has transformed into a long-term medical condition, allowing people to coexist with the virus for extended periods as long as their immune systems are properly maintained. However, the virus gradually compromises the immune system, making it vulnerable to opportunistic diseases that leads to AIDS (Odebiyi & Oladejo, 2024).

The global statistics are alarming, with approximately 42.3 million AIDS-related deaths reported at the end of 2023, alongside 1.3 million new HIV infections and 630,000 AIDS-related deaths. The epidemic has devastated populations globally, particularly African as 65% of global incidences are from the WHO African region which has depleted the workforce, overwhelmed healthcare systems, and increased orphan numbers (Canadian Foundation for IDS Research (CANFAR), 2023; WHO, 2023).

Transmission and infection can be curbed through adequate treatment and vaccination (Adeyemi & Oluyo, 2023; Ibrahim et al., 2021; Oladejo & Oluyo, 2022; Stein & LaSalle, 1979;

Stoddart & Reyes, 2006). New drugs have improved the quality of life and life expectancy of infected individuals (Huo et al., 2016; Oladejo & Oluyo, 2022; Stoddart & Reyes, 2006). Epidemiological models utilize various infection rates to simulate disease transmission. However, researchers have explored alternative rates, including saturated and non-monotone formulations.

One such example is the saturated incidence rate, which considers the force of infection and inhibition factors. This rate is characterized by the expression $\frac{\omega SI}{(1+\gamma I)}$, where ω represents the transmission rate, S and I represent the susceptible and infected populations, and γ represents the coefficient of inhibition (Zhang et al., 2008).

Additional formulations have been proposed to capture psychological and behavioral aspects of disease transmission. These include various saturated incidence rates that incorporate parameters reflecting saturation, disease control measures, and protective behaviors.

From an epidemiological standpoint, saturation incidence marks a crucial turning point in an epidemic's trajectory, where disease prevalence stabilizes. Understanding these formulations provides valuable insights into epidemic spread. This study primarily aims to investigate strategies for reducing HIV/AIDS transmission, with a focus on examining the stability and sensitivity of HIV/AIDS model incorporating saturated incidence rates which better capture the complex dynamics of HIV transmission. Despite the considerable efforts of many researchers in developing HIV and AIDS transmission models, a significant gap remains in the existing literature. While numerous studies have explored various aspects of HIV transmission, few have incorporated the dynamics of vaccinated susceptible individuals and saturated incidence rates into their models. This study aims to address this knowledge gap by developing a novel HIV and AIDS transmission model that accounts for the complexities of vaccinated susceptible individuals and incorporates saturated incidence rates.

The organization of this study is outlined in the following manner: Section 2 provides a thorough description of the model formulation, which takes into account the model's existence and uniqueness. Meanwhile, Section 3 explores the disease-free and endemic equilibrium points, the fundamental reproduction number, and the investigation of both local and global stability. Also, sensitivity analysis is computed to identify influential model parameters while section 4 presents numerical simulations to support theoretical findings.

2. Model Formulation

This analysis introduces the susceptible and infectious epidemic model (SI). The population $N(t)$ is divided into 6 groups of people: those who are not immune, those who have been vaccinated, those who are asymptomatic, those who are symptomatic, those who are receiving treatment, and those who have AIDS. These groups are represented by $S(t), V(t), I_1(t), I_2(t), T(t),$ and$A(t)$, respectively such that $N = S + V + I_1 + I_2 + T + A$, as shown in figure 1 below:

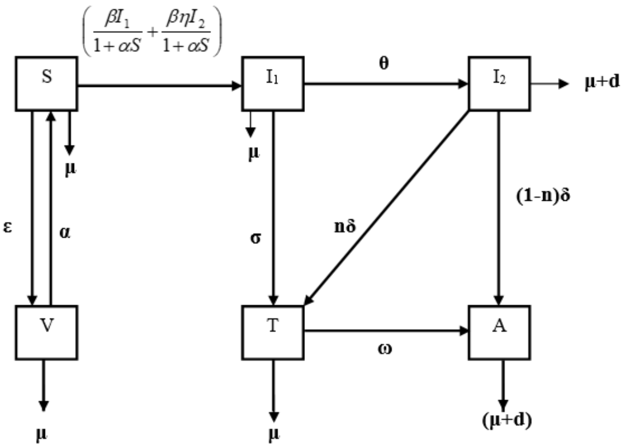

Figure 1. Transmission diagram for susceptible-infected (SI) model with saturation term to susceptible

Based on the diagram shown in figure 1, which includes the saturation factor for susceptible individuals, we have considered the following assumptions when developing our proposed model: All parameters are positive. People are recruited into the Susceptible population by birth at a rate (π). A fraction of persons placed on PrEP strategies at a rate (α) and fraction of persons placed on PrEP strategies become susceptible again at a rate (ε) due to non-adherence. Natural death rate occurs in all compartments. Asymptomatic infective population can be screened at a rate θ and progress to symptomatic compartment. Some moved from asymptomatic population to treated population at a rate (σ). Also, some progresses from symptomatic to treated population at a rate $(n\delta)$ and a fraction to AIDS class at a rate $(1-n)\delta$ respectively. A fraction of infected persons are placed on treatment with antiretroviral therapy (ARV) with viral load unsuppressed move to AIDS compartment at a rate (ω). This is due to carelessness and other factors. Also, some individuals may experience a resurgence of the virus, leading to the development of full-blown AIDS.

Given the assumptions outlined previously, the subsequent set of ordinary differential equations for the proposed model is thus established:

$$\left.\begin{aligned}
\frac{dS}{dt} &= \pi - \frac{\beta I_1 S}{1+\alpha S} - \frac{\beta\eta I_2 S}{1+\alpha S} - \mu S - \gamma S + \varepsilon V \\
\frac{dV}{dt} &= \gamma S - (\mu+\varepsilon)V \\
\frac{dI_1}{dt} &= \frac{\beta I_1 S}{1+\alpha S} + \frac{\beta\eta I_2 S}{1+\alpha S} - (\theta+\mu+\sigma)I_1 \\
\frac{dI_2}{dt} &= \theta I_1 - (\mu+\delta+d)I_2 \\
\frac{dT}{dt} &= n\delta I_2 + \sigma I_1 - (\omega+\mu)T \\
\frac{dA}{dt} &= (1-n)\delta I_2 + \omega T - (\mu+d)A
\end{aligned}\right\} \tag{1}$$

$$S(0)=S_0, V(0)=V_0, I_1(0)=I_{1_0}, I_2(0)=I_{2_0}, A(0)=A_0 \tag{2}$$

where,

$\frac{\beta I_1 S}{1+\alpha S}, \frac{\beta \eta I_2 S}{1+\alpha S}$ are the incidence rate and α is the saturation term to the susceptible.

Table 1. Model Variables

Variable	Description
$S(t)$	Susceptible population at a given time(t).
$V(t)$	Vaccinated susceptible
$I_1(t)$	Asymptomatic population at a given time
$I_2(t)$	Symptomatic population at a given time.
$T(t)$	Treated population
$A(t)$	AIDS population

Table 2. Model Variables and Parameters

Parameters/ variables	Description	Value	Source
π	Recruitment rate of Susceptible	300	Al-Sheikh (2011)
β	Transmission rate	0.0009	Odebiyi et al. (2024)
d	AIDS related death rate	0.76	Ibrahim et al. (2021)
θ	Pace at which asymptomatic population become aware of being infected after a screening process.	0.015	Al-Sheikh (2011)
μ	Natural mortality rate unrelated to AIDS	0.02	Ibrahim et al. (2021)
ε	Fraction of vaccinated that become susceptible.	0.2	Assumed
σ	Progression rate from Asymptomatic class to AIDS class.	0.2	Safiel et al. (2012)
δ	Progression rate from Symptomatic class to Treated and AID class.	0.2	Safiel et al. (2012)
η	Infectivity rate of transmission	0.3	Odebiyi et al. (2024)
n	is the fraction of Symptomatic population that moved to treated class	0.02	Assumed
γ	Fraction of persons placed on prep strategy	0.4	Oladejo & Oluyo (2022)

2.1. Positivity and boundedness of the model

In this part, we will demonstrate using model (1) that the state variables are always greater than zero, and the solutions will continue to be positive for all $t \geq 0$. Therefore, it is assumed that the parameters in the model are all positive.

Theorem 1: Let the initial conditions or values of the state variables be such that $\{(S(0) \geq 0, V(0) \geq 0, I_1(0) \geq 0, I_2(0) \geq 0, T(0) \geq 0, A(0) \geq 0) \in \Omega\}$, then the set $(S(t), I_1(t), I_2(t), T(t), A(t))$ is non-negative in Ω for all $t \geq 0$.

Proof: The positivity of the state variables is determined by examining the first equation in (1) through the approach of Adeyemi & Oluyo (2023); Keno et al. (2023); Oladejo & Oluyo (2022),

$$\frac{dS}{dt} \geq -\left(\frac{\beta I_1}{1+\alpha S} + \frac{\beta \eta I_2}{1+\alpha S} + \mu + \gamma\right) S$$

$$\frac{dS}{dt} \geq -\int\left(\frac{\beta I_1}{1+\alpha S} + \frac{\beta \eta I_2}{1+\alpha S} + \mu + \gamma\right) S$$

Using variable separable

$$\frac{dS}{S} \geq -\int \left(\frac{\beta I_1}{1+\alpha S} + \frac{\beta \eta I_2}{1+\alpha S} + \mu + \gamma \right) dt$$

$$\ln s \geq -\left(\frac{\beta I_1}{1+\alpha S} + \frac{\beta \eta I_2}{1+\alpha S} + \mu + \gamma \right) t + C$$

$$S(t) \geq e^{-\left(\frac{\beta I_1}{1+\alpha S} + \frac{\beta \eta I_2}{1+\alpha S} + \mu + \gamma \right) t} . e^{C_1}$$

$$S(t) = S_0 e^{-\left(\frac{\beta I_1}{1+\alpha S} + \frac{\beta \eta I_2}{1+\alpha S} + \mu + \gamma \right) t} .$$

$$S(0) = S_0 \Rightarrow A_1 = S_0$$

Since $S(t) \geq 0$, for all $t > 0$ provided that $S_0 \geq 0$.
Hence, $S(t) \geq 0$

For the second compartment of (1)

$$\frac{dV}{dt} \geq \gamma S - (\mu + \varepsilon) V$$

$$\frac{dV}{dt} \geq -\int (\mu + \varepsilon) V$$

Using variable separable

$$\frac{dV}{V} \geq -\int (\mu + \varepsilon) dt$$

$$V(t) \geq e^{-(\mu+\varepsilon)t} . e^{C_1}$$

$$V(t) = V_0 e^{-(\mu+\varepsilon)t} .$$

$$V(0) = V_0 \Rightarrow A_1 = V_0$$

Since $V(t) \geq 0$, for all $t > 0$ provided that $V_0 \geq 0$.
Hence, $V(t) \geq 0$

It can be demonstrated through the identical methodology for various state variables that:

$$I_1(t) \geq I_1(0) e^{-(\theta+\mu+\sigma)t} \geq 0, I_2(t) \geq I_2(0) e^{-(\mu+\gamma+d)t} \geq 0,$$

$$T(t) \geq T(0) e^{-(\sigma+\mu)t} \geq 0, A(t) \geq A(0) e^{-(\mu+d)t} \geq 0$$

This demonstrates that every solution to equation (1) is above zero for all $t \geq 0$. As a result, the HIV/AIDS transmission model outlined in equation (1) holds importance in terms of epidemiology and is well-defined numerically within a feasible region of $\Omega \geq 0$.

Theorem 2: Every solution in the region $\Omega = \left\{ \left(S(t), V(t), I_1(t), I_2(t), T(t), A(t) \in \Omega_+^6 : N(t) \leq \frac{\pi}{\mu} \right) \right\}$ is positively invariant with respect to the HIV/AIDS model (1) in the populations. The solutions for the system are contained and remain in the region Ω for all time $t \geq 0$.
Proof: After evaluating the model's equation and summing up all the derivatives with respect to time t, we arrived at

$$\frac{dN(t)}{dt} = \pi + \mu (S + V + I_1 + I_2 + T + A) - dA$$

$$\frac{dN(t)}{dt} = \pi + \mu N$$

$$N \leq \frac{\pi}{\mu} + \left(N_0 - \frac{\pi}{\mu} \right) e^{-\mu t}$$

Where N_0 is the initial size of the population
Therefore,

$$\lim_{t\to\infty} N(t) \le \frac{\pi}{\mu}$$

This outcome suggests that the HIV/AIDS model (1) has a negative solution that is not zero and is bound within the region Ω. Additionally, all solutions originating in Ω tend toward, enter, or remain in Ω. Therefore, it can be inferred that the model is effectively posed in terms of epidemiology.

3. Mathematical Analysis of the Model

3.1. Disease free equilibrium point

This refers to a situation in which the illness has been eradicated from the community, meaning there are no more infected people, no spread of the disease, and everyone in the population is vulnerable.

At the equilibrium,

$$\frac{dS}{dt} = \frac{dV}{dt} = \frac{dI_1}{dt} = \frac{dI_2}{dt} = \frac{dT}{dt} = \frac{dA}{dt} = 0 \,,$$

$$\left.\begin{aligned}
\frac{dS}{dt} &= \pi - \frac{\beta I_1 S}{1+\alpha S} - \frac{\beta\eta I_2 S}{1+\alpha S} - \mu S - \gamma S + \varepsilon V \\
\frac{dV}{dt} &= \gamma S - (\mu+\varepsilon)V \\
\frac{dI_1}{dt} &= \frac{\beta I_1 S}{1+\alpha S} + \frac{\beta\eta I_2 S}{1+\alpha S} - (\theta+\mu+\sigma)I_1 \\
\frac{dI_2}{dt} &= \theta I_1 - (\mu+\delta+d)I_2 \\
\frac{dT}{dt} &= n\delta I_2 + \sigma I_1 - (\omega+\mu)T \\
\frac{dA}{dt} &= (1-n)\delta I_2 + \omega T - (\mu+d)A
\end{aligned}\right\}$$

At the disease-free equilibrium, we set $S \neq 0, V \neq 0, I_1 = I_2 = T = A = 0$ substituting these to equation (1) and solving gives the infection – free equilibrium as

$$E^0 = (S^0, V^0, I_1^0, I_2^0, A^0) = \left(\frac{\pi(\mu+\varepsilon)}{(\mu+\gamma)(\mu+\varepsilon)-\varepsilon\gamma}, \frac{\pi\gamma}{(\mu+\gamma)(\mu+\varepsilon)-\varepsilon\gamma}, 0,0,0,0\right) \tag{2}$$

3.2. Endemic equilibrium point

The endemic equilibrium represents a point where the disease is continuously present in the population without major fluctuations. At endemic equilibrium, $S \neq 0, V \neq 0, I_1 \neq 0, I_2 \neq 0, T \neq 0, A \neq 0$. By solving equation (1) at the same time, we also have the endemic equilibrium points within the HIV model (1) known as $x^* = (S^*, V, I_1^*, I_2{}^*, T^* A^*)$ is obtained as:

$$\left\{\begin{aligned}
s^* &= \frac{K_1\pi^*}{K_1(\lambda^* + \mu + \gamma) - \varepsilon\gamma} \\
V^* &= \frac{\gamma}{K_1}\left[\frac{K_1\pi^*}{K_1(\lambda^* + \mu + \gamma) - \varepsilon\gamma}\right] \\
I_1^* &= \frac{\lambda^*}{K_2}\left[\frac{\pi^*}{((\lambda^* + \mu + \gamma)) - \varepsilon\gamma}\right] \\
I_2^* &= \frac{\theta}{K_3}\left[\frac{\lambda^*}{K_2}\frac{\pi^*}{(\lambda^* + \mu + \gamma) - \varepsilon\gamma}\right] \\
T^* &= \frac{n\delta\theta\lambda^*\pi^* + K_3\sigma\lambda^*\pi^*}{K_2K_3K_4((\lambda^* + \mu + \gamma) - \varepsilon\gamma)} \\
A^* &= \frac{K_4(1-n)\theta\lambda^*\pi^* + \omega(n\delta\theta\lambda^*\pi^* + K_3\sigma\lambda^*\pi^*)}{K_2K_3K_3K_5((\lambda^* + \mu + \gamma) - \varepsilon\gamma)}
\end{aligned}\right\} \tag{3}$$

Where,

$$K_1 = (\mu+\varepsilon), K_2 = (\theta+\mu+\sigma), K_3 = (\mu+\delta+d), K_4 = (\omega+\mu), K_5 = (\mu+d),\ \lambda^* = \left(\frac{\beta I_1}{1+\alpha S} - \frac{\beta\eta I_2}{1+\alpha S}\right)$$

3.3. Basic Reproduction Number (R_0)

The construction of the next generation matrix involves analyzing unique pathways of infection and dynamics of transmission between different compartments. Utilizing the methodology outlined by Van den Driessche & Watmough (2002),

Let M be the next generation matrix. It comprises of two parts $F$and V^{-1} where,

$$F = \left[\frac{\partial f_1(x_0)}{\partial x_j}\right], \qquad V = \left[\frac{\partial V_1(x_0)}{\partial x_j}\right]$$

The term f_i and v_i are computed as

$$F_i = \begin{bmatrix} \dfrac{\beta I_1 S}{1+\alpha S} + \dfrac{\beta\eta I_1 S}{1+\alpha S} \\ 0 \\ 0 \\ 0 \end{bmatrix}$$

$$V_i = \begin{bmatrix} -(\theta+\mu+\sigma) \\ \theta I_1 - (\mu+\delta+d)I_2 \\ n\delta I_2 + \sigma I_1 - (\omega+\mu)T \\ (1-n)\delta I_2 + \omega T - (\mu+d)A \end{bmatrix}$$

The Jacobian matrices of F_i and V_i at the disease-free equilibrium point,

$x_0 = \left(\frac{\pi(\mu+\varepsilon)}{(\mu+\gamma)(\mu+\varepsilon)-\varepsilon\gamma}, \frac{\pi\gamma}{(\mu+\gamma)(\mu+\varepsilon)-\varepsilon\gamma}, 0{,}0{,}0{,}0\right)$ are:

$$DF(x_0) = \left[\frac{\partial F_i(x_0)}{\partial x_j}\right] = \begin{bmatrix} \dfrac{\beta S_0}{1+\alpha S_0} & \dfrac{\beta\eta S_0}{1+\alpha S_0} & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix} \Rightarrow$$

$$\begin{bmatrix} \dfrac{\beta\dfrac{\pi(\mu+\varepsilon)}{(\mu+\gamma)(\mu+\varepsilon)-\varepsilon\gamma}}{1+\alpha\dfrac{\pi(\mu+\varepsilon)}{(\mu+\gamma)(\mu+\varepsilon)-\varepsilon\gamma}} & \dfrac{\beta\eta\dfrac{\pi(\mu+\varepsilon)}{(\mu+\gamma)(\mu+\varepsilon)-\varepsilon\gamma}}{1+\alpha\dfrac{\pi(\mu+\varepsilon)}{(\mu+\gamma)(\mu+\varepsilon)-\varepsilon\gamma}} & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix} \Rightarrow$$

$$\begin{bmatrix} \beta\dfrac{\pi(\mu+\varepsilon)}{(\mu+\gamma)(\mu+\varepsilon)-\varepsilon\gamma+\gamma\pi(\mu+\varepsilon)} & \beta\eta\dfrac{\pi(\mu+\varepsilon)}{(\mu+\gamma)(\mu+\varepsilon)-\varepsilon\gamma+\gamma\pi(\mu+\varepsilon)} & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix}$$

$$\Rightarrow \begin{bmatrix} \beta q & \beta\eta q & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix}$$

Where $q = \frac{\pi(\mu+\varepsilon)}{(\mu+\gamma)(\mu+\varepsilon)-\varepsilon\gamma+\gamma\pi(\mu+\varepsilon)}$,

$$V = \begin{pmatrix} (\theta+\mu+\sigma) & 0 & 0 & 0 \\ \theta & (\mu+\delta+d) & 0 & 0 \\ -\sigma & -n\delta_2 & (\omega+\mu) & 0 \\ 0 & -(1-n)\delta & -\omega & (\mu+d) \end{pmatrix}$$

$$DV(E_0) = \left[\frac{\partial V_1(E_0)}{\partial x_j}\right] = \begin{bmatrix} B_1 & 0 & 0 & 0 \\ -\theta & B_2 & 0 & 0 \\ -\sigma & -n\delta_2 & B_3 & 0 \\ 0 & -(1-n)\delta & -\omega & B_4 \end{bmatrix} \tag{5}$$

Where,

$B_1 = (\theta + \mu + \sigma)$,$B_2 = (\mu + \delta + d)$,$B_3 = (\omega + \mu)$, $B_4 = (\mu + d)$

$$V^{-1} = \begin{bmatrix} \frac{1}{B_1} & 0 & 0 & 0 \\ \frac{\theta}{B_1B_2} & \frac{1}{B_2} & 0 & 0 \\ \frac{n\theta\delta_2+\sigma B_2}{B_1B_2B_3} & \frac{n\delta_2}{B_2B_3} & \frac{1}{B_3} & 0 \\ -\frac{\delta n\theta K_3 - n\omega\theta\delta_2 - \delta\theta K_3 - \omega\sigma K_2}{B_1B_2B_3B_4} & -\frac{\delta nK_3 - n\omega\delta_2 - \delta K_3}{B_2B_3B_4} & \frac{\omega}{B_3B_4} & \frac{1}{B_4} \end{bmatrix} \tag{6}$$

$$FV^{-1} = \begin{bmatrix} \frac{\beta S_0}{B_1} + \frac{\beta\eta S_0\theta}{B_1B_2} & \frac{\beta\eta S_0\theta}{B_2} & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix} \tag{7}$$

The primary factor for reproduction, derived from the leading Eigen-value of the formula FV^{-1}, can be determined as:

$$R_0 = \frac{\beta q(\eta\theta+B_2)}{B_1B_2} \tag{8}$$

3.4. Stability analysis of the Disease-free Equilibrium

Theorem 4: If the basic reproduction number $R_0 < 1$, then the disease-free state is stable within the local area and unstable if otherwise.

Proof: We evaluate the Jacobian matrix of the model at the disease-free equilibrium

$$x_0 = \left(\frac{\pi(\mu+\varepsilon)}{(\mu+\gamma)(\mu+\varepsilon)-\varepsilon\gamma}, \frac{\pi\gamma}{(\mu+\gamma)(\mu+\varepsilon)-\varepsilon\gamma}, 0,0,0,0\right) \tag{9}$$

$$J(E_0)=\begin{bmatrix} -(\mu+\alpha) & \varepsilon & -\dfrac{\beta S_0}{1+\alpha S_0} & -\dfrac{\beta\eta S_0}{1+\alpha S_0} & 0 & 0 \\ \alpha & -(\mu+\varepsilon) & 0 & 0 & 0 & 0 \\ 0 & 0 & \dfrac{\beta S_0}{1+\alpha S_0}-B_1 & \dfrac{\beta\eta S_0}{1+\alpha S_0} & 0 & 0 \\ 0 & 0 & \theta & -B_2 & 0 & 0 \\ 0 & 0 & \sigma & n\delta & -B_3 & 0 \\ 0 & 0 & 0 & B_4 & \omega & -B_5 \end{bmatrix} \Rightarrow$$

$$J(E_0)=\begin{bmatrix} -(\mu+\alpha) & \varepsilon & -\beta q & -\beta\eta q & 0 & 0 \\ \alpha & -(\mu+\varepsilon) & 0 & 0 & 0 & 0 \\ 0 & 0 & \beta q-B_1 & \beta\eta q & 0 & 0 \\ 0 & 0 & \theta & -B_2 & 0 & 0 \\ 0 & 0 & \sigma & n\delta & -B_3 & 0 \\ 0 & 0 & 0 & K_4 & \omega & -B_5 \end{bmatrix} \qquad (10)$$

Where,
$B_1=(\theta+\mu+\sigma), B_2=(\mu+\delta+d), B_3=(\omega+\mu), B_4=(1-n)\delta, B_5=(\mu+d),$

The characteristic polynomial equation is obtained as
$(\lambda+K_5)(\lambda+K_3)(\lambda^2 A_1+A_2\lambda+A_3)(\lambda^2 P_1+P_2\lambda+P_3)=0$
where,
$A_1=1, A_2=B_1+B_2-\beta q, A_3=B_1B_2(1-R_0),\ q=\frac{\pi(\mu+\varepsilon)}{(\mu+\gamma)(\mu+\varepsilon)-\varepsilon\gamma+\gamma\pi(\mu+\varepsilon)}$ and $P_1=1, P_2=2\mu-(\alpha+\varepsilon), P_3=\mu(\mu+\alpha)-\varepsilon(\mu+2\varepsilon)$
The eigen-values $\lambda_1=-B_5, \lambda_2=-B_3$, and the remaining will be obtained from the quadratic equations $(\lambda^2A_1+A_2\lambda+A_3)=0,\quad and\quad (\lambda^2P_1+P_2\lambda+P_3)=0$
where,
$P_1=1, P_2=2\mu-(\alpha+\varepsilon), P_3=\mu(\mu+\alpha)-\varepsilon(\mu+2\varepsilon)$
$P_1>0,\ P_2>0\Rightarrow\ 2\mu>(\alpha+\varepsilon),\ P_3>0\Rightarrow\ \mu(\alpha+\mu)>\varepsilon(\mu+2\alpha)$
$A_1>0,\ A_2>0$ if $B_1+B_2>\beta q,$ and $A_3>0\Rightarrow R_0>1$

Then, by Routh Hurwitz criteria, the remaining four eigen-values are negative. Hence, the disease-free equilibrium is locally asymptotically stable.

3.5. Global asymptotic stability of Endemic Equilibrium

Theorem 5: If the basic reproduction number exceeds one, the HIV model (1) will demonstrate global asymptotic stability at the endemic equilibrium point in region D.
Proof: Consider a quadratic Lyapunov function $L: D\in R_+^6\to R_+$defined by

$$L=\frac{1}{2}\left\{(S-S^{**})+(V-V^{**})+(I_1-I_1^{**})+(I_2-I_2^{**})+(T-T^{**})(A-A^{**})\right\}^2 \qquad (11)$$

The time derivative of the Lyapunov function (11) is given by

$$\frac{dL}{dt}=\left\{(S-S^{**})+(V-V^{**})+(I_1-I_1^{**})+(I_2-I_2^{**})+(T-T^{**})+(A-A^{**})\right\}\frac{d}{dt}(S+V+I_1+I_2+T+A)$$
$$\frac{dL}{dt}=\left\{(S-S^{**})+(V-V^{**})+(I_1-I_1^{**})+(I_2-I_2^{**})+(T-T^{**})+(A-A^{**})\right\}\left\{\pi-\mu(S+V+I_1+I_2+T+A)\right\}$$

$$= -\mu\left\{\left(S - S^{**}\right) + \left(V - V^{**}\right) + \left(I_1 - I_1^{**}\right) + \left(I_2 - I_2^{**}\right) + \left(T - T^{**}\right) + \left(A - A^{**}\right)\right\}\left\{\left(S + V + I_2 + I_2 + T + A\right) - \frac{\pi}{\mu}\right\} \quad (12)$$

Since $N^{**} \leq \frac{\pi}{\mu}$, then, the following result is obtained

$$\frac{dL}{dt} \leq -\mu\left\{\left(S - S^{**}\right) + \left(V - V^{**}\right) + \left(I_1 - I_1^{**}\right) + \left(I_2 - I_2^{**}\right) + \left(T - T^{**}\right) + \left(A - A^{**}\right)\right\}\left\{\begin{array}{l}\left(S + V + I_1 + I_2 + T + A\right) - \\ \left(\begin{array}{l} S^{**} + V + I_1^{**} + I_2^{**} \\ + T^{**} + A^{**} \end{array}\right)\end{array}\right\}$$

$$= -\mu\left\{\left(S - S^{**}\right) + \left(V - V^{**}\right) + \left(I_1 - I_1^{**}\right) + \left(I_2 - I_2^{**}\right) + \left(T + T^{**}\right)\left(A - A^{**}\right)\right\} \times \left\{\begin{array}{l}\left(S - S^{**}\right) + \left(V - V^{**}\right) \\ + \left(I_1 - I_1^{**}\right) + \left(I_2 - I_2^{**}\right) \\ + \left(T - T^{**}\right) + \left(A - A^{**}\right)\end{array}\right\}$$

$$= -\mu\left\{\left(S - S^{**}\right) + \left(V - V^{**}\right) + \left(I_1 - I_1^{**}\right) + \left(I_2 - I_2^{**}\right) + \left(T - T^{**}\right) + \left(A - A^{**}\right)\right\}^2 \quad (13)$$

Since the time derivative of the continuously differentiable function G is negative semi-definite i.e.,$\frac{dL}{dt} \leq 0$, then, the function L is a Lyapunov function. Therefore, $\frac{dL}{dt} = 0$ provided that $S = S^{**}, V = V^{**}, A = A^{**}, I_1 = I_1^{**}, I_2 = I_2^{**}, T = T^{**}$ and $A = A^{**}$. Then, by LaSalle's invariance principle (Stein & LaSalle, 1979), the largest invariance set for which $\frac{dL}{dt} = 0$ is the singleton set $\{\varepsilon^{**}\}$, which implies that the endemic equilibrium point of the HIV/AIDS model with saturated incidence rate in (1) is globally asymptotically stable.

3.6. Global Asymptotic Stability of the Disease-free Equilibrium

Theorem 6: Whenever the basic reproduction number is below one, the disease-free equilibrium point of the model is stable on a global scale.

Proof: The investigation focuses on the global asymptotic stability of the HIV model by utilizing a different approach. The HIV model is condensed and rewritten in a more concise manner.

$$\frac{dX}{dt} = F(X,\ Z),$$
$$\frac{dZ}{dt} = G(X,\ Z),\ G(X,\ 0) = 0, \quad (14)$$

Where X is the uninfected class of the HIV model and Z is the infected classes of the model i.e. $X = S \in R_+^2$ and $Z = (I_1, I_2, T, A) \in R_+^4$. Also, let the disease-free equilibrium point of the HIV model be denoted by $\varepsilon_0 = (X^*, 0)$. Then, the following properties must be satisfied

H_1: For $\frac{dX}{dt} = F(X, 0)$, X^* is globally asymptotically stable

H_2: $G(X, Z) = AZ - \hat{G}(X, Z) \geq 0$,

Where $A = \partial G / \partial Z$, which is an M-matrix evaluated at $(X^*, 0)$ with non-negative off diagonal entries.

Theorem 7: The disease-free $\varepsilon_0 = (X^*, 0)$ of the HIV model (1) is globally asymptotically stable if the properties H_1 and H_2 are satisfied.

Proof: $\boldsymbol{F(X, Z)}$ and $G(X, Z)$ are obtained from the HIV model (1) as

$$S(0) = S_0, V(0) = V_0, I_1(0) = I_{1_0}, I_2(0) = I_{2_0}, A(0) = A_0$$

$$F(X, Z) = \begin{pmatrix} \pi - \left(\frac{\beta I_1 S}{1 + \alpha S} + \frac{\beta \eta I_1 S}{1 + \alpha S}\right) - \mu S - \alpha S + \varepsilon V \\ \alpha S - (\mu + \varepsilon) V \end{pmatrix} \quad (15)$$

$$G(X,Z) = \begin{pmatrix} \frac{\beta I_1 S}{1+\alpha S} - \frac{\beta\eta I_1 S}{1+\alpha S} - (\theta+\mu+\sigma)I_1 \\ \theta I_1 - (\mu+\delta+d)I_2 \\ n\delta I_2 + \sigma I_1 - (\omega+\mu)T \\ (1-n)\delta I_2 + \omega T - (\mu+d)A \end{pmatrix} \quad (16)$$

Such that,

$$F(X,\ 0) = (\pi - \mu S - \alpha S\ )$$

$$\frac{dS}{dt} = \pi - (\mu+\alpha)S \quad (17)$$

Simplifying Equation (17) gives

$$S(t) = \frac{\pi}{\mu} + \left(S(0) - \frac{\pi}{\mu}\right)\ell^{-(\mu+\alpha)t} \quad (18)$$

Also, for $F(X,0) = -(\mu+\varepsilon)V$

$$\frac{dV}{dt} + (\mu+\varepsilon)V = 0$$

Simplifying also gives

$$V(t) = V(0)e^{-(\mu+\varepsilon)t} \quad (19)$$

Irrespective of the initial sizes of the variables as $t \to \infty$, then, $S(t) \to \frac{\pi}{\mu}, V(t) \to 0$. Therefore, the DFE $(X^*,0)$ is globally asymptotically stable satisfying property H_1. Now, to establish the second property H_2, recall that

$$G(X,\ Z) = \begin{pmatrix} \frac{\beta I_1 S}{1+\alpha S} + \frac{\beta\eta I_2 S}{1+\alpha S} - (\theta+\mu+\sigma)I_1 \\ \theta I_1 - (\mu+\delta+d)I_2 \\ n\delta I_2 + \sigma I_1 - (\omega+\mu)T \\ (1-n)\delta I_2 + \omega T - (\mu+d)A \end{pmatrix} \quad (20)$$

An M-matrix whose off-diagonal entries are non-negative is obtained as

$$A = \frac{\partial G}{\partial Z} = \begin{pmatrix} \frac{\beta S^*}{1+\alpha S} - (\theta+\mu+\sigma) & \frac{\beta S^*\eta}{1+\alpha S} & 0 & 0 \\ \theta & -(\mu+\delta+d) & 0 & 0 \\ \sigma & n\delta & -(\omega+\mu) & 0 \\ 0 & (1-n)\delta & \omega & -(\mu+d) \end{pmatrix} \quad (21)$$

Where$S^* = \frac{\pi}{\mu}$. Then, from $\hat{G}(X,Z) = AZ - G(X,Z)$,

$$\hat{G}(X,\ Z) = \begin{pmatrix} \left(\frac{\beta I_1}{1+\alpha S} + \frac{\beta I_2\eta}{1+\alpha S}\right)(S^* - S) \\ 0 \\ 0 \\ 0 \end{pmatrix} \quad (22)$$

Since $0 \le S \le \frac{\pi}{\mu}$, clearly, it is obvious that $\hat{G}(X,Y) \ge 0, \hat{G}_2(X,Y) = 0, \hat{G}_3(X,Y) = 0$, $\hat{G}_4(X,Y) = 0$. Hence, property H_2 is satisfied. Therefore, the disease-free equilibrium of the HIV model (1) is globally asymptotically stable.

3.7. Sensitivity Analysis of the basic Reproduction Number

Sensitivity analysis is crucial for evaluating the stability and accuracy of mathematical models. This process helps researchers pinpoint the most influential factors driving disease transmission and assess the potential impact of interventions.

The ratio of the relative change in a variable to the relative change in a parameter is known as the normalized forward sensitivity index. If a variable can be expressed as a differentiable function of a parameter, the sensitivity index can also be determined using partial derivatives.

Adapting the methodology introduced by Chitnis et al. (2008), the normalized forward sensitivity index of a variable "b" that depends differentiable on a parameter "m" is defined as:

$$X_e^f := \frac{\partial f}{\partial e} * \frac{e}{f} \tag{23}$$

Since we have a clear equation (8) providing the formula for R_0, we can establish an analytical representation for the impact of R_0 as $X_h^g := \frac{\partial g}{\partial h} * \frac{h}{g}$ in relation to the various parameters outlined in table 3:

Table 3. Sensitivity Result

Parameters	Sensitivity Index
β	+1.0000
π	+1.0000
μ	-0.622956845
δ	-0.00048498
d	-0.00184295
ε	0.1111111112
η	0.003297911
σ	-0.3252033
θ	-0.02109233

3.8. Interpretation of Sensitivity Indices

The sensitivity indices provide valuable insights into the relationships between model parameters and outcomes. A high sensitivity index for a particular parameter indicates that small changes to that parameter can significantly impact the model's behavior. Conversely, a low sensitivity index suggests that the model is relatively insensitive to changes in that parameter. Positive sensitivity indices reveal parameters that increase the outcome, while negative indices identify parameters that decrease the outcome. By analyzing the sensitivity indices, researchers can identify key parameters driving model behavior, prioritize parameters for further investigation and also targeted interventions to optimize outcomes.

Table 2 illustrates the importance of different parameters in the baseline values, highlighting the recruitment rate (π), transmission rate (β), and infectivity rate (η) are the most sensitive parameters. When the parameters $\pi, \beta, \varepsilon,$ and η increases while other parameters remain constant, the value of R_0 also increases. More so, when the parameters $\theta, \delta, d, \alpha, \mu$ and σ increase while keeping other parameters constant, the value of R_0 also decrease. These parameters should be targeted by intervention strategies in order to have a stable and disease-free environment. For instance, $X_\beta^{R_0} = +1.0000$ means that increasing or decreasing β by 10% increases or (decreases) R_0 by 10%. Also, $X_\varepsilon^{R_0} = 0.11111112$ simply means that increasing or decreasing ε by 10%, +increases or (decreases) R_0 by 1.1111112%. More so, $X_\sigma^{R_0}$ =-0.325203252 means that increasing or (decreasing) R_0 by 10% means that increasing or (decreasing) R_0 by 3.25203252% as seen in table 2. Others can be calculated following same approach.

4. Numerical Simulations and Discussion

4.1. Numerical Simulations Analysis

A computational simulation of the model was conducted using Maple 18 Mathematical software to gain deeper insights into the dynamic transmission patterns of HIV/AIDS. This simulation facilitated an examination of how various parameters influence the basic reproduction number, providing a clearer understanding of their role in shaping the epidemic's trajectory.

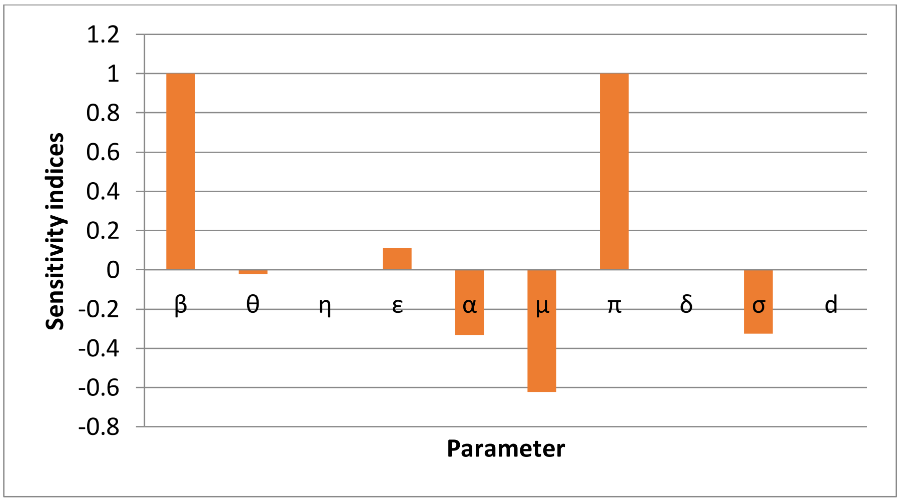

Figure 2. Graphical Representation of the Sensitivity indices of R_0

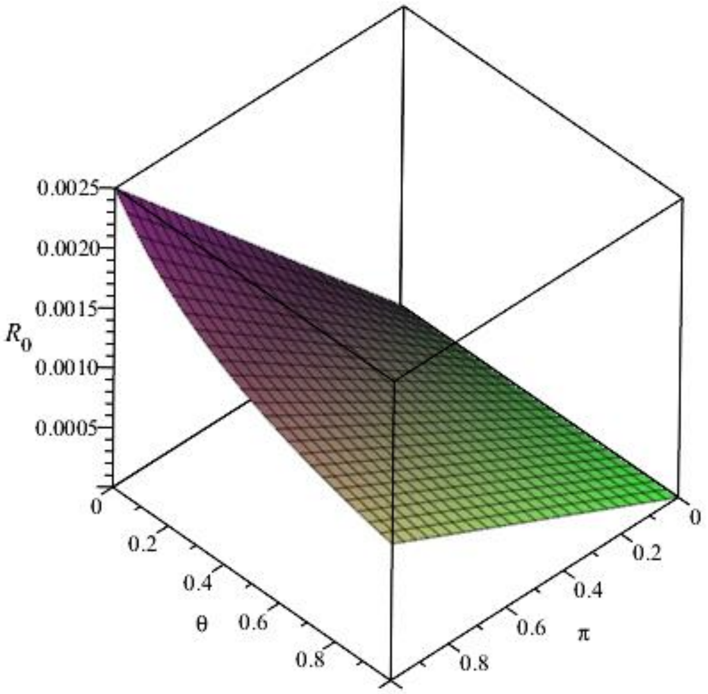

Figure 3. Sensitivity of the basic reproduction number to the parameters π and θ

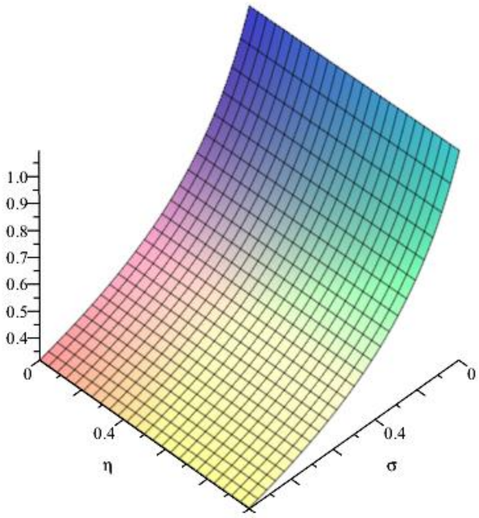

Figure 4. Sensitivity of the basic reproduction number to the parameters η and σ

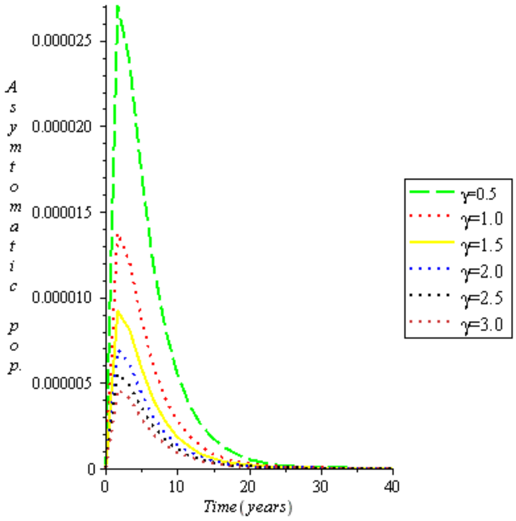

Figure 5. Plot of Fraction of Vaccinated susceptible placed on PrEP

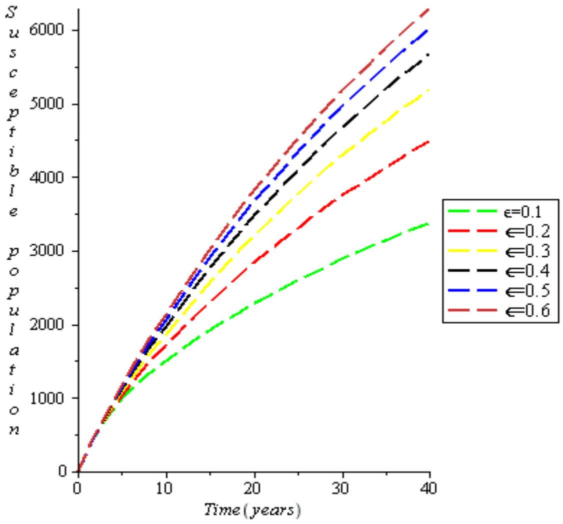

Figure 6. Plot of Fraction of Vaccinated that become susceptible

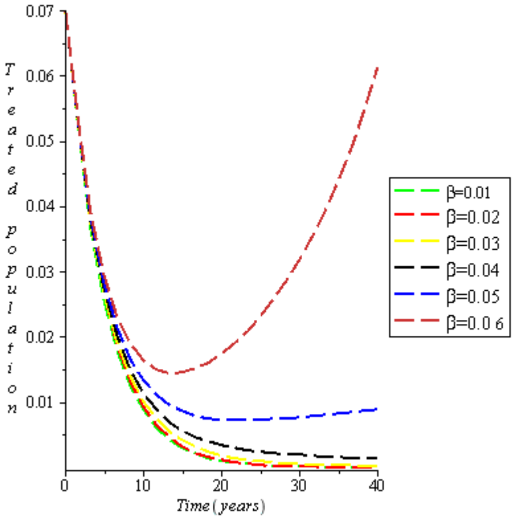

Figure 7. Graph of treated infective population against time t, showcasing various transmission values of β

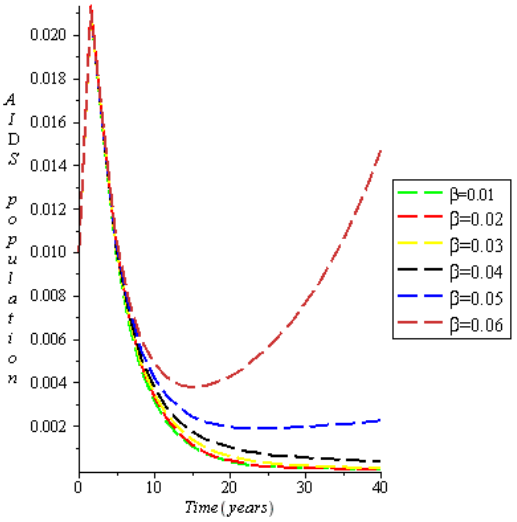

Figure 8. Graph of AIDS population against time t, showcasing various transmission values of β

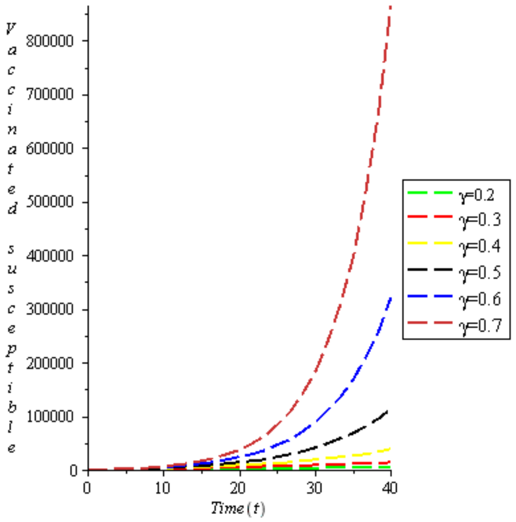

Figure 9. Plot of fraction of persons placed on prep strategies

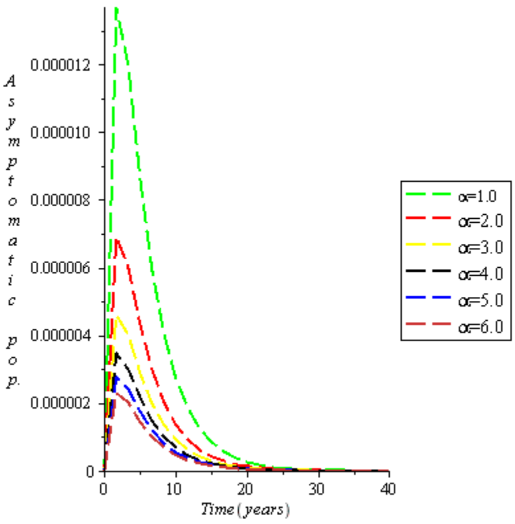

Figure 10. Variation of Asymptomatic population $I_1(t)$ against time for different values of saturation term (α)

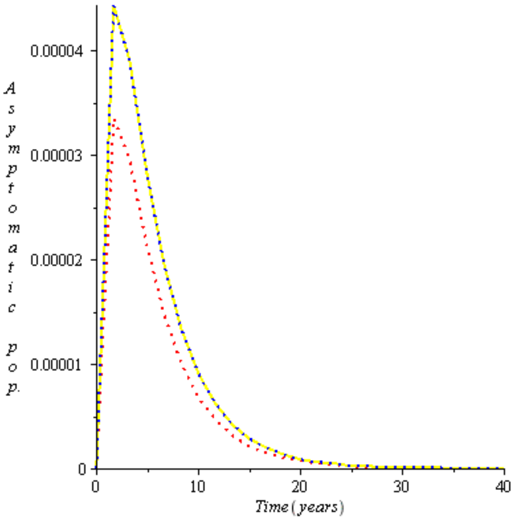

Figure 11. Global stability of Disease-free equilibrium

4.2. Discussion

Figure 2 illustrates the graphical representation of the basic reproduction shown in a bar chart. Sensitivity analysis plays a crucial role in the model. It helps to quantify the uncertainty associated with model parameters, which is essential for understanding the robustness of the model's predictions. By analyzing the sensitivity of the model to different parameters, researchers can identify the most critical parameters that drive the model's behavior. Sensitivity analysis helps to identify the key drivers of HIV/AIDS transmission, which can inform the development of targeted interventions. From figure 2, the most sensitive parameter in the analysis is the recruitment rate and transmission rate and an increase in this parameter values will certainly increase the basic reproduction number.

Figure 3 illustrates the relationship between recruitment rate of susceptible (π) and screening rate (θ) on the basic reproduction number, Notably, as the recruitment rate increases, the basic reproduction number rises. Conversely, enhancing screening efforts (θ) leads to a decrease in the basic reproduction number. Figure 4 demonstrates that as the infectivity rate (η) increases, the basic reproduction increases and also, an increase in σ, leads to decrease in the basic reproduction number. This decrease is accompanied by a slowing of the progression rate from the asymptomatic stage to AIDS (σ).

Figure 5 illustrates that a larger proportion of the population accesses Pre-Exposure Prophylaxis (PrEP) in high-risk area, therefore resulting to decrease in asymptomatic class. The decline can be attributed to the effective prevention of HIV transmission through PrEP, which is further enhanced by improved adherence to the treatment regimen. As individuals become more aware of PrEP and its benefits, they are more likely to adopt safer sexual practices, including the likelihood of transmission. Moreover, by reducing the viral load in infected individuals, PrEP decreases the risk of transmission, ultimately contributing to the decline in the asymptomatic class. This result is in perfect agreement to that of Odebiyi et al. (2024).

Figure 6 illustrates that as fraction of individuals move from vaccinated susceptible class to the susceptible class, the susceptible population increases. This shift occurs because some individuals possibly refuse to use or fail to complete their (PrEP) regimen, thereby losing their protective benefits and becoming susceptible to infection. As a result, the susceptible population grows, increasing the risk of transmission as observed in figure 6.

In Figure 7, a chart displays the infected population undergoing treatment over time t, demonstrating different levels of transmission. The findings suggest that an increase in transmission rate leads to a decrease in the number of individuals receiving treatment. This trend warrants caution, as it suggests that even among those undergoing treatment, careless interactions and ongoing high-risk behaviors can still lead to re-infection. Moreover, treatment does not guarantee a permanent cure. Treatment challenges, including incomplete adherence, drug resistance, and viral mutations, can reduce treatment efficacy, allowing the virus to persist and spread. Furthermore, persistent engagement in high-risk behaviors among treated individuals increases their likelihood of re-infection. Insufficient public health measures, such as inadequate contact tracing, testing, and education, also contribute to undiagnosed cases and perpetuate transmission. The significance of implementing thorough interventions to efficiently control and stop the transmission of infection is highlighted by these factors. This result aligns with the findings of Odebiyi & Oladejo (2024).

Figure 8 showcasing various transmission values of β. It illustrates a direct relationship between the transmission and the AIDS population, with higher transmission rates corresponding to larger AIDS Populations. This finding is consistent with Odebiyi et al. (2024). The increased spread of the virus, fueled by inadequate prevention, limited access to treatment, and persistent high-risk behaviors, contributes to the growing AIDS population. Additionally, a higher transmission rate may indicate a greater number of undiagnosed or untreated individuals, enabling the virus to spread more rapidly.

Figure 9 illustrates the proportion of individuals adopting the Pre-Exposure prophylaxis (PrEP) strategy. Notably, the vaccinated susceptible increases as more individuals opt for daily PrEP medication. This uptick can be attributed to growing awareness about the effectiveness of PrEP in preventing HIV transmission. Additionally, expanded access to PrEP services, reduced stigma around HIV prevention, and increased confidence in the medication's ability to provide protection all contribute to the rising adoption of PrEP among vaccinated susceptible individuals. The result is in agreement with Oladejo & Oluyo (2022).

Figure 10 illustrates the influence of the saturation term of the nonlinear incidence rate on the behavior of asymptomatic population. The figure demonstrates the substantial impact of the saturation term on the nonlinear incidence rate, revealing its crucial role in mitigating the spread of HIV within the population. Notably, as saturation term increases, the number of asymptomatic individuals decreases significantly. This phenomenon can be attributed to the crowding effect, where susceptible individuals exercise greater caution and adopt preventive measures, thereby reducing their likelihood of infection. Alternatively, the saturation term may also reflect the increased awareness and vigilance among susceptible individuals, leading to a decrease in asymptomatic cases. These findings align with the research conducted by Odebiyi et al. (2024) and Olaniyi (2018).

In Figure 11, different starting conditions are shown to impact the disease-free equilibrium's global stability. The main idea conveyed is that when the basic reproduction number $R_0 < 1$, the virus will not be able to persist in the community and the number of new cases will decline regardless of the initial number of infections. In simpler terms, maintaining an R_0 value below unity ensures the disease's eventual extinction, regardless of initial case numbers. As the threshold is met, the system converges toward a disease-free state, characterized by a steady decline in asymptomatic infectious individuals until they reach zero. The disease-free equilibrium suggest that PrEP is highly effective in preventing HIV transmission. Public health intervention should reinforce the importance of PrEP as a prevention strategy, particularly among high-risk individuals.

5. Conclusion

The study investigates the transmission dynamics of HIV/AIDS using a nonlinear mathematical model incorporating saturated incidence rates. The model examines the interactions between susceptible, vaccinated susceptible, asymptomatic, symptomatic, treated and AIDS populations, providing insights into the complex dynamics of HIV transmission.

Analyzing stability and sensitivity helps evaluate how important factors affect the spread of HIV/AIDS. The results reveal that achieving a disease-free environment is feasible, but requires targeted interventions to maintain stability. The sensitivity analysis identifies recruitment rate and transmission rate as crucial parameters influencing the basic reproduction number.

The research emphasizes the significance of comprehending the saturated incidence rate when modeling the dynamics of HIV/AIDS transmission. The results offer valuable information for policymakers and health officials looking to create successful approaches to prevent the spread of HIV/AIDS.

To reduce the transmission rate and protect susceptible individuals, we recommend that policymakers prioritize interventions addressing key parameters such as recruitment rate, and transmission rate. Furthermore, vaccinated susceptible and susceptible individual should stay on PrEP medication and also exercise caution and minimize unnecessary interactions with infectious individuals, thereby reducing their risk of infection and contributing to the overall decrease of the infected population. By adopting a multifaceted approach, we can effectively control the spread of HIV/AIDS and strive towards achieving a disease-free environment. The future research directions could investigate optimal control strategies for minimizing the spread of HIV and maximizing the effectiveness of treatment and develop multi-strain models to capture the spread of different HIV/AIDS strains in a population.

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