RESEARCH PAPER

Mathematical dynamics for HIV infections with public awareness and viral load detectability

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Abstract

In this paper, we develop a nonlinear deterministic model that incorporates public awareness and treatment to describe the dynamics of HIV/AIDS in an infected population with detectable and undetectable viral load. The model undergoes backward bifurcation in which a stable disease-free equilibrium coexists with a stable endemic equilibrium. Numerical simulations carried out show the behavior of the state variables and the impact of public awareness in controlling the spread of HIV. The results show that public awareness will help in curtailing the spread of HIV infection, and when treatment is applied to infected individuals with detectable viral load can easily suppress their virus to become undetectable so that they cannot transmit HIV through sexual intercourse.

Keywords: HIV; viral load detectability; parameter calibration; bifurcation analysis; confidence interval
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1 Introduction

Human Immunodeficiency Virus (HIV) is a virus affecting the cells of the immune system ($CD4^+$ T) making the body vulnerable to other infectious diseases [1, 2]. $CD4^+$ T cells are orchestrators, regulators, and direct effectors of antiviral immunity [1]. If HIV is not treated, it leads to a severe

stage of HIV infection called Acquired Immunodeficiency Syndrome (AIDS) [3]. The virus is transmitted via direct contact with different kinds of body fluids such as blood, vaginal fluids, rectal fluids, semen, and breast milk of the infected individual or through mother to her child during the pregnancy period (i.e, vertical transmission) [3, 4]. HIV still remains one of the most severe global public health threats [5, 6]. Since its emergence, more than 79.3 million people became infected with HIV/AIDS, among which 36.3 million people have died from AIDS-related illness [5]. About 37.7 million people were living with HIV by 2020 [5]. As of 2020, out of the total HIV-infected individuals, about 20.6 million people (55%) with HIV were in Eastern and Southern Africa, 4.7 million people (13%) in Western and Central Africa, 5.7 million people (15%) in Asia and the Pacific region and 2.2 million people (6%) in Western and Central Europe and North America [5]. The majority of people infected with HIV are from low and middle-income countries.

As part of global commitment to decrease the transmission of HIV infection, the number of people accessing Antiretroviral Therapy (ART) has increased significantly from 7.8 million in 2010 to 27.5 million in 2020. New infection declined by 30% from 2.1 million in 2010 to 1.5 million people, and 84% of people living with HIV are aware of their status, while the remaining 16% needs access to be diagnosis and HIV test [5]. Before the introduction of ART, a minority of the people infected with HIV maintained normal $CD4+$ cell counted healthy range of $(450–1650 \, cells/ml)$ and remained symptoms-free without treatment for several years and did not progress to AIDS stages [7]. Some of these individuals have low or non-detectable viral load and are referred to as non-progressors, classified as long-term non-progressors and controllers due to their resistance to viral replication in the absence of ART. Controllers are sub-divided into elite controllers (EC) with HIV RNA less than 50 $copies/ml$ and Viremic Controllers (VCs) with $50 − 2000 \, copies/ml$ [7, 8]. HIV treatments aim at reducing the viral load until the virus is no longer detectable.

It was reported that taking a full dose of ART could effectively suppress the viral load (i.e., the amount of HIV in a person's blood) of infected individuals [4]. If the viral load is lower than 200 $copies/ml$ in blood, it is unlikely to be detected using a blood antibody test. In this scenario, an infected individual cannot transmit HIV through sexual intercourse [9]. Up till now, there is no cure for HIV infection. Still, the ART helps to suppress viral replication within the patient's body and allows immune system recovery to strengthen and regain the ability to fight new infection [4]. WHO endorsed ART regardless of $CD4+$ cell count to all people with HIV in 2016. Also, ART should be offered simultaneously with diagnosis among individuals that are ready for treatment. In June 2021, 187 countries adopted the first recommendation, and 82 low- and middle-income countries implemented the second policy [4].

Many models have been formulated to study the dynamics of HIV/AIDS infection with different control strategies incorporated in the model. The study by [10] showed that public health awareness on risk behaviours could help in decreasing the persistence of HIV/AIDS. A mathematical model for assessing the impact of condom usage, ART and voluntary testing in decreasing the spread of HIV was proposed by [6]. The study revealed that the hope of controlling HIV transmission using the intervention listed was highly remarkable. Furthermore, a study by [11] highlighted that the rate of vertical transmission which leads to an increase in the pre-AIDS and AIDS population is proportional to the infective population. The spread of the diseases can be reduced significantly by controlling the vertical population. A study by Bhunu et al. [12] suggested that even in the absence of ART, effective guidance and testing have tremendous effects on the prevention and control of the epidemic. [13] analyzed HIV/AIDS dynamics for a situation when only HIV-infected individuals who did not develop AIDS symptoms and are not under ART transmit the HIV virus. Recently, nonlinear fractional order models are also used to describe HIV/AIDS transmission dynamics. [14] proposed a dynamical fractional first-order HIV-1 with

Caputo derivative that studies the infection between cancer cells, healthy CD4$^+$ T lymphocytes and virus-infected CD4$^+$ T lymphocytes. The result revealed that fractional order derivatives have a significant effect on the dynamics process. The fractional order model of viral kinetics for primary infection of HIV-1 with immune control and treatment was analyzed by [15]. A nonlinear fractional-order HIV epidemic model solved via the $L1$ scheme was proposed by [16]. The result showed that the homotopy analysis method applied has effectiveness and strength in solving a compartmental model. Naik et al. [17] proposed a nonlinear fractional order model for HIV transmission dynamics with optimal control. The study recommended that to decrease the spread of HIV infections there is a need for personal precaution and periodic monitoring by researchers and medical professionals.

For further references to the related studies mentioned above, one can visit [6, 10, 17–21]. In this research work, we have formulated a new mathematical model of HIV/AIDS dynamics considering infected individuals with detectable viral load and infected individuals with undetectable viral load.

The significance of this research is to assess the impact of viral load detectability on HIV/AIDS transmission when some of the infected individuals with low viral load (undetectable viral load) can not transmit HIV sexually, and also assess the impact of public education and treatment on uninfected and infected population, respectively.

The paper is organized as follows: the model is developed and analyzed in Sections 2 and 3, respectively. Basic reproduction number, equilibria, and their stabilities and bifurcation analysis are given in 4. Model fitting, parameter estimation, and sensitivity analysis are conducted and presented in Section 5. Numerical simulation of the model is presented in Section 6. Discussion of the results and conclusion are provided in Section 7.

2 Model description

A nonlinear mathematical model is developed to study the transmission dynamics of HIV/AIDS to assess the impact of public awareness and treatment on the overall dynamics. The total population at time t, denoted by $N(t)$ is divided into the following disjoint compartments: uneducated susceptible $S_u(t)$ (individuals that are unaware on how to prevent HIV infection), educated susceptible $S_e(t)$ (individuals that are aware on how to prevent HIV infection), newly infected individuals $I_1(t)$, infected with detectable viral load $I_2(t)$ (infected individuals with higher viral load (> 200 copies/ml) that can be detected using a blood test and they can transmit HIV through sexual intercourse), infected with undetectable viral load $I_3(t)$ (infected individuals with a low level of HIV virus in their blood (< 200 copies/ml) that can not be detected using blood test and they cannot transmit the disease through sexual intercourse), infected individuals receiving treatment $I_t(t)$, AIDS patients(infected individuals with higher viral load and developed certain opportunistic infections). The risk behaviours that can lead to HIV/AIDS infection include unprotected sex, sharing of drugs and injection needles, lack or absence of blood tests for couples before getting married, or lack of condom usage during sex. We considered sexual transmission as the only mode of transmission, as such I_3 are considered non-infectious since they can not transmit HIV through sex, and assumed AIDS patients to be sexually inactive because their immune system is weak and unable to fight infections which cause several opportunistic diseases to them.

Recruitment of new individuals into the susceptible population occurs at a rate π (which are assumed to be sexually active and susceptible to HIV infection). p is the fraction of recruited individuals that are educated. Uneducated susceptible individuals become educated through public awareness campaigns on HIV infection at a rate τ. Susceptible uneducated and educated individuals become infected when in contact with the infected individuals at a rate λ and $\alpha\lambda$, respectively. It is assumed that susceptible educated are avoiding risk behaviour which reduces

their rate of HIV infection by α, with $0 < \alpha < 1$. Newly infected individuals become either infected with detectable viral load at a rate $\epsilon\theta$ or infected controllers which are undetectable at a rate $(1-\epsilon)\theta$ where θ is the progression rate from the newly infected compartment, while ϵ is the fraction of newly infected with detectable viral load. Infected individuals with detectable viral load move to treatment at a rate ϕ while some progress to AIDS at the rate ρ. Infected individuals under treatment when taking a full dose of ART their viral load will be undetectable through a blood test and assumed to move into the infected undetectable viral load class at the rate γ. Infected with the undetectable viral load when their viral load becomes detected through blood test moves to infected detectable class at the rate ω.

AIDS patients move to treatment class at a rate σ. Natural death of individuals occurs at a rate μ. δ_1 and δ_2 are the disease mortality rates of infected individuals at AIDS and treatment compartment, respectively, where $\delta_2 < \delta_1$ (we assume that individuals under treatment die at a rate less than AIDS patients that refused to go for treatment). Thus, we have $N(t) = S_u(t) + S_e(t) + I_1(t) + I_2(t) + I_3(t) + I_t(t) + A(t)$.
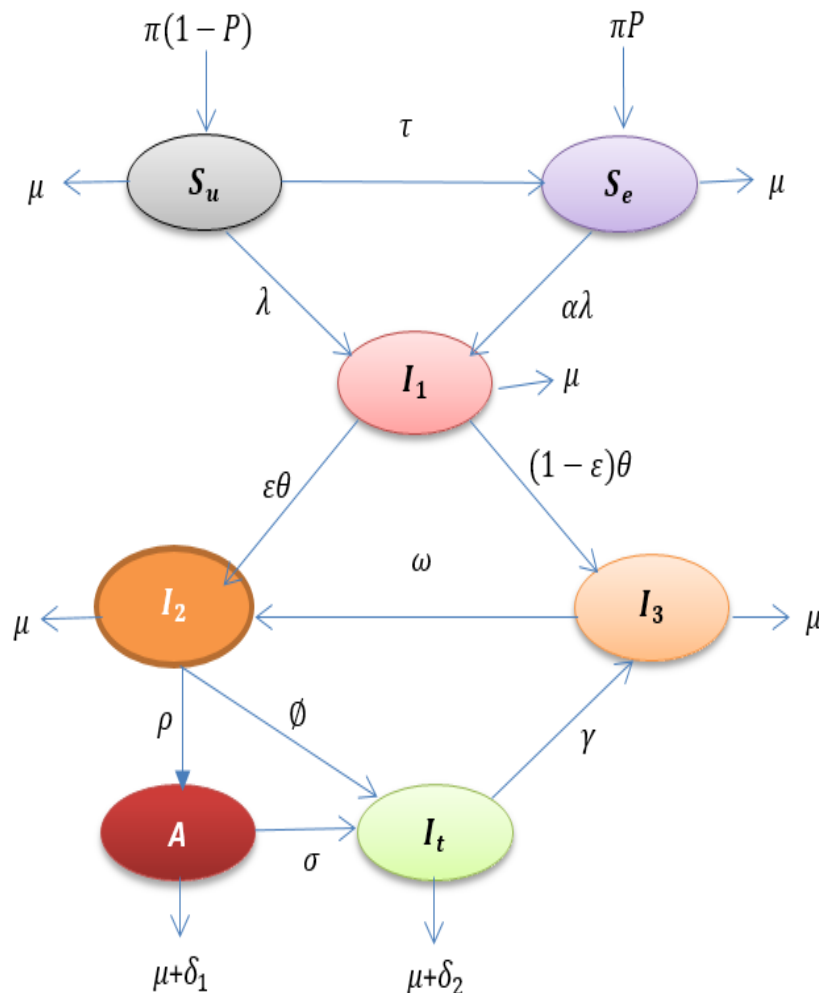

Figure 1. Schematic diagram of model (1). Solid arrows indicate transitions and expressions next to arrows show the *per capita* flow rate between compartments.

By considering the explanations of the model parameters and compartments, we obtain the

following system:

$$\frac{dS_u}{dt} = \pi(1-p) - (\mu + \tau + \lambda)S_u,$$

$$\frac{dS_e}{dt} = \pi p + \tau S_u - (\mu + \alpha\lambda)S_e,$$

$$\frac{dI_1}{dt} = \lambda(S_u + \alpha S_e) - (\mu + \theta)I_1,$$

$$\frac{dI_2}{dt} = \epsilon\theta I_1 + \omega I_3 - (\mu + \rho + \phi)I_2, \qquad (1)$$

$$\frac{dI_3}{dt} = (1-\epsilon)\theta I_1 + \gamma I_t - (\mu + \omega)I_3,$$

$$\frac{dI_t}{dt} = \phi I_2 + \sigma A - (\mu + \gamma + \delta_2)I_t,$$

$$\frac{dA}{dt} = \rho I_2 - (\mu + \sigma + \delta_1)A,$$

where, the force of infection for the transmission of HIV in this model is given by, $\lambda = \beta(\frac{\eta_1 I_1 + \eta_2 I_2 + I_t}{N})$ and β is the effective contact rate that may result in HIV/AIDS infection, η_1, and η_2 ($\eta_1 > \eta_2$) denote an increase in infectiousness for newly infected individuals and infected individuals with higher viral load, respectively.

The description of the variables and parameters of the model are shown in Table 1.

Table 1. Interpretation and definitions of the state variables and parameters used in model (1).

Variable	Description
N	Total human population
S_u	Uneducated susceptible individuals
S_e	Educated susceptible individuals
I_1	Newly infected individuals
I_2	Infected individuals with detectable viral load
I_3	Infected individuals with undetectable viral load
I_t	Infected individuals under treatment
A	AIDS patients
λ	Force of infection
Parameter	
π	Recruitment rate of susceptible individuals
μ	Natural mortality rate
δ_1, δ_2	Death rate due to disease
p	Proportion of π that are educated
τ	Rate at which uneducated susceptible become educated about HIV infection
β	Effective contact rate
α	Parameter for decrease of infectiousness in S_e
θ	Rate of movement from infectious class
ϵ	Fraction of the rate of movement from infectious compartment
ϕ	Movement rate of infected with detectable viral load to treatment
ρ	Progression rate to AIDS compartment
γ	Rate at which treated individuals become undetectable viral load
ω	Rate at which infected individuals with undetectable viral load become detectable
σ	rate of movement of AIDS patients to treatment class at a rate
η_1, η_2	Infectiousness factor for newly infected and individuals with higher viral load

3 Analysis of the model

Boundedness and positivity of solutions

The model deals with the human population, each of its parameters, and the state variables are non-negative for all $t \geq 0$. We can now prove that each of the state variables of model (1) are non-negative for all $t \geq 0$.

Theorem 1 *System* (1) *defines a dynamical system in the closed set, given by,*

$$\Omega = \left\{ (S_u(t), S_e(t), I_1(t), I_2(t), I_3(t), I_t(t), A(t)) \, \epsilon \, R_+^7 : N \leq \frac{\pi}{\mu} \right\}.$$

Proof We are to show that R_+^7 is positively invariant, that is all solution of system (1) initiated in Ω do not leave Ω see (Theorem 2.1.5) of [22]. Let $S_u(0) > 0, S_e(0) > 0, I_1(0) > 0, I_2(0) > 0, I_3(0) > 0, I_t(0) > 0$, and $A(0)) > 0$. Suppose $S_u(0)$ and $S_e(0)$ are not positive, then there exists a time $\tilde{t} > 0$, such that $S_u(t) > 0$ and $S_e(t) > 0$ for $t \in [0, \tilde{t})$ and $S_u(\tilde{t}) = S_e(\tilde{t}) = 0$.

From the third, fourth, and fifth equation of model (1), we obtain,

$$\begin{aligned}
\frac{dI_1(t)}{dt} &\geq -(\theta + \mu)I_1(t) \quad \text{for } t \in [0, \tilde{t}), \\
\frac{dI_2(t)}{dt} &\geq -(\rho + \Phi + \mu)I_2(t) \quad \text{for } t \in [0, \tilde{t}), \\
\frac{dI_3(t)}{dt} &\geq -(\mu + \omega)I_3(t) \quad \text{for } t \in [0, \tilde{t}), \\
\frac{dI_t(t)}{dt} &\geq -(\gamma + \mu + \delta_2)I_t(t) \quad \text{for } t \in [0, \tilde{t}), \\
\frac{dA(t)}{dt} &\geq -(\mu + \sigma + \delta_1)A(t) \quad \text{for } t \in [0, \tilde{t}).
\end{aligned} \tag{2}$$

It follows that $I_1(0) > 0, I_2(0) > 0, I_3(0) > 0, I_t(0) > 0$ and $A(0) > 0$ for $t \in [0, \tilde{t})$. Thus, from the first and second equations of the system, we have

$$\frac{dS_u(t)}{dt} \geq -(\tau + \mu + \lambda)S_u(t) \quad \text{for } t \in [0, \tilde{t}),$$

$$\frac{dS_e(t)}{dt} \geq -(\mu + \alpha\lambda)S_e(t) \quad \text{for } t \in [0, \tilde{t}).$$

One can clearly see that, $S_u(0) > 0$ and $S_e(0) > 0$ which contradict our assumption of $S_u(\tilde{t}) = S_e(\tilde{t}) = 0$. Hence $S_u(t)$ and $S_e(t)$ are positive. Similarly, the positivity of the remaining state variable of the model can be seen from subsystem of (1) excluding the first and second equation written in matrix form as follows:

$$\frac{dX(t)}{dt} = \mathcal{M}Y(t) + B(t), \tag{3}$$

with

$$Y(t) = \begin{pmatrix} I_1, & I_2, & I_3, & I_t, & A \end{pmatrix}^T,$$

$$\mathcal{M} = \begin{pmatrix} \eta_1 K - K_1 & \eta_2 K & 0 & K & 0 \\ \epsilon\theta & -K_2 & \omega & 0 & 0 \\ K_6\theta & 0 & -K_3 & \gamma & 0 \\ 0 & \Phi & 0 & -K_4 & \sigma \\ 0 & \rho & 0 & 0 & -K_5 \end{pmatrix}, \tag{4}$$

$$B(t) = \begin{pmatrix} 0 & 0 & 0 & 0 & 0 \end{pmatrix}^T,$$

where, $K = \beta \frac{S_u + \alpha S_e}{N}$, $K_1 = \theta + \mu$, $K_2 = \rho + \Phi + \mu$, $K_3 = \omega + \mu$, $K_4 = \gamma + \mu + \delta_2$, $K_5 = \sigma + \mu + \delta_1$ and $K_6 = (1 - \epsilon)$. Clearly, $\mathcal{M}$ is a Metzler matrix for the fact that both $S_u(t)$ and $S_e(t)$ are non-negative. Which shows subsystem (3) is a monotone system [23]. Thus, $\mathbb{R}_+^5$ is invariant under the flow of subsystem (3). Therefore, $\mathbb{R}_+^7$ is positively invariant under the flow of system (1). ∎

System (1) has a disease-free equilibrium which is determined by setting its right-hand sides to zero.

$$\epsilon^0 = (S_u^0, S_e^0, I_1^0, I_2^0, I_3^0, I_t, A) = \left(\frac{\pi(1-P)}{\tau + \mu}, \frac{\pi(\tau + \mu p)}{\mu(\tau + \mu)}, 0, 0, 0, 0, 0 \right).$$

4 Basic reproduction number

The basic reproduction number (denoted by $R_0 = \rho(FV^{-1})$, where ρ is the spectral radius of the next generation matrix, (FV^{-1}) of model (1) is the number of new infections produced by HIV infected individuals with a detectable viral load when interacted with the fully susceptible population in the absence of awareness and treatment. It is determined using the next generation matrix approach [24] to establish the stability of the equilibrium. The matrix F represents the new infection terms and V for the remaining transition terms are respectively given by, R_0

$$F = \begin{bmatrix} \frac{\eta_1\beta\left(S_u^0 + \alpha S_e^0\right)}{N^0} & \frac{\eta_2\beta\left(S_u^0 + \alpha S_e^0\right)}{N^0} & 0 & \frac{\beta\left(S_u^0 + \alpha S_e^0\right)}{N^0} & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{bmatrix}, \tag{5}$$

$$V = \begin{bmatrix} K_1 & 0 & 0 & 0 & 0 \\ -\epsilon\theta & K_2 & -\omega & 0 & 0 \\ -K_6\theta & 0 & K_3 & -\gamma & 0 \\ 0 & -\phi & 0 & K_4 & -\sigma \\ 0 & -\rho & 0 & 0 & K_5 \end{bmatrix}. \tag{6}$$

The basic reproduction number R_0 is obtained as,

$$R_0 = \frac{\beta \left(\left(K_4 K_6 \eta_2 \theta + \phi \left(-\gamma \eta_1 + K_6 \theta \right) \right) \omega + \left(\left(\varepsilon \theta \eta_2 + \eta_1 K_2 \right) K_4 + \phi \varepsilon \theta \right) K_3 \right) K_5 \left(\mu(1-p) + \alpha(\tau + \mu p) \right)}{k_1 \left(\left(-\gamma \omega \phi + K_2 K_3 K_4 \right) K_5 - \gamma \omega \rho \sigma \right) (\tau + \mu)}$$

$$+ \frac{\beta \rho \sigma \left(\left(-\gamma \eta_1 + K_6 \theta \right) \omega + K_3 \varepsilon \theta \right) \left(\mu(1-p) + \alpha(\tau + \mu p) \right)}{k_1 \left(\left(-\gamma \omega \phi + K_2 K_3 K_4 \right) K_5 - \gamma \omega \rho \sigma \right) (\tau + \mu)},$$

where $K_1 = \theta + \mu, K_2 = \rho + \phi + \mu, K_3 = \omega + \mu, K_4 = \gamma + \mu + \delta_2, K_5 = \sigma + \mu + \delta_1, K_6 = (1-\epsilon)$ and are all positives.

Theorem 2 *The disease-free equilibrium (DFE) ϵ^0, of model* (1)*, is locally-asymptotically stable (LAS) in Ω if $R_0 < 1$, and unstable if $R_0 > 1$.*

Global stability of disease-free equilibrium

Theorem 3 *The disease-free equilibrium (DFE) ϵ^0, of model* (1) *is globally-asymptotically stable (GAS) in Ω if $R_0 < 1$, and unstable if $R_0 > 1$.*

Proof To prove the GAS of DFE, the two axioms $[G_1]$ and $[G_2]$ must be satisfied for $R_0 < 1$ [25]. We re-write system (1) in the form:

$$\begin{aligned} \frac{dY_1}{dt} &= F(Y_1, Y_2), \\ \frac{dY_2}{dt} &= G(Y_1, Y_2) : G(Y_1, 0) = 0, \end{aligned} \tag{7}$$

where $Y_1 = (S_u^0, S_e^0)$ and $Y_2 = (I_1^0, I_2^0, I_3^0, I_t^0, A^0)$ with the elements of $Y_1 \in R_+^2$ representing the uninfected population and the elements of $Y_2 \in R_+^5$ representing the infected population. The DFE is now denoted as $E^0 = (Y_1^*, 0)$, where $Y_1^* = (N^0, 0)$. Now for the first condition, that is GAS of Y_1^*, gives,

$$\frac{dY_1}{dt} = F(Y_1, 0) = \left[\begin{array}{c} \pi(1-P) - (\mu + \tau)S_u^0 \\ \pi p + \tau S_u^0 - \mu S_e^0 \end{array} \right].$$

Solving the linear differential equations gives,

$$S_u^0(t) = \frac{\pi(1-p)}{(\mu + \tau)} - \frac{\pi(1-p)}{(\mu + \tau)} e^{-(\mu+\tau)t} + S_u^0(0)e^{-(\mu+\tau)t},$$

$$S_e^0(t) = \frac{\pi p + \tau S_u^0}{\mu} - \frac{\pi p + \tau S_u^0}{\mu} e^{-\mu t} + S_e^0(0)e^{-\mu t}.$$

Now, it is easy to show that $S_u^0(t) + S_e^0(t) \to N^0(t)$ as $t \to \infty$ regardless of the value of $S_u^0(t)$ and $S_e^0(t)$. Thus, $Y_1^* = (N^0, 0)$ is globally asymptotically stable. Furthermore, for the second condition,

that is $\tilde{G}(Y_1, Y_2) = BY_2 - \tilde{G}(Y_1, Y_2)$ gives:

$$
B = \begin{pmatrix}
-(\mu + \theta) + \frac{\beta\eta_1 S_u^0}{N^0} + \frac{\alpha\beta\eta_1 S_e^0}{N^0} & \frac{\beta\eta_2 S_u^0}{N^0} + \frac{\beta\eta_2 S_e^0}{N^0} & 0 & \frac{\beta S_u^0 + \alpha\beta S_e^0}{N^0} & 0 \\
\epsilon\theta & -(\mu + \rho + \phi) & \omega & 0 & 0 \\
(1 - \epsilon)\theta & 0 & -(\mu + \omega) & \gamma & 0 \\
0 & \phi & 0 & -(\mu + \delta_2 + \gamma) & \sigma \\
0 & \rho & 0 & 0 & -(\mu + \delta_1 + \sigma)
\end{pmatrix}.
$$

$$(8)$$

This is clearly Metziller matrix

$$
\tilde{G}(Y_1, Y_2) = \begin{pmatrix}
\lambda S_u^0 + \alpha\lambda S_e^0 - (\mu + \theta) I_1^0 \\
\epsilon\theta I_1^0 + \omega I_3^0 - (\mu + \rho + \phi) I_2^0 \\
(1 - \epsilon)\theta I_1^0 + \gamma I_t^0 - (\mu + \omega) I_3^0 \\
\phi I_2^0 + \sigma A^0 - (\mu + \gamma + \delta_2) I_t^0 \\
\rho I_2^0 - (\mu + \sigma + \delta_1) A^0
\end{pmatrix}.
$$

$$(9)$$

Then,

$$
\tilde{G}(Y_1, Y_2) = BY_2 - \tilde{G}(Y_1, Y_2) = \begin{bmatrix} 0 \\ 0 \\ 0 \\ 0 \\ 0 \end{bmatrix}.
$$

Thus we have

$$
\tilde{G}(Y_1, Y_2) = \begin{bmatrix} 0 & 0 & 0 & 0 & 0 \end{bmatrix}^T,
$$

It is clear that $\tilde{G}(Y_1, Y_2) = 0$. ■

Endemic equilibrium point

When HIV persists in the population, at least one of the infectious compartments of model (1) is not empty. As such, model (1) has endemic equilibrium which is obtained by setting the vector field of the model (1) to zero. Defined the endemic equilibrium state as;

$$
\epsilon^{**} = (S_u^{**}, S_e^{**}, I_1^{**}, I_2^{**}, I_3^{**}, I_t^{**}, A^{**}),
$$

where,

$$
\begin{aligned}
S_u^{**} &= \frac{\pi (1 - p)}{\lambda^{**} + \mu + \tau}, \\
S_e^{**} &= \frac{\pi (P\lambda^{**} + P\mu + P\tau + \tau)}{\alpha \lambda^{**2} + \alpha \lambda^{**} \mu + \alpha \lambda^{**} \tau + \lambda^{**} \mu + \mu^2 + \mu \tau}, \\
I_1^{**} &= \frac{\lambda^{**} \pi (P\alpha \lambda^{**} + P\alpha \mu + P\alpha \tau + \alpha \lambda^{**} + \alpha \tau + \mu)}{K_1 \left(\alpha \lambda^{**2} + \alpha \lambda^{**} \mu + \alpha \lambda^{**} \tau + \lambda^{**} \mu + \mu^2 + \mu \tau\right)},
\end{aligned}
$$

$$(10)$$

$$I_2^{**} = -\frac{(\epsilon K_3 + \omega K_6)\,\lambda^{**}\,(((\lambda^{**}+\tau)\,\alpha + \mu)\,\pi\,(1-p) + P\pi\,\alpha\,(\lambda^{**}+\mu+\tau))\,\theta\,K_5 K_4}{(\lambda^{**}+\mu+\tau)\,(\alpha\,\lambda^{**}+\mu)\,K_1\,(\gamma\,(\phi\,K_5+\rho\,\sigma)\,\omega - K_2 K_3 K_4 K_5)},$$

$$I_3^{**} = -\frac{\lambda^{**}\,(((\lambda^{**}+\tau)\,\alpha + \mu)\,\pi\,(1-p) + P\pi\,\alpha\,(\lambda^{**}+\mu+\tau))\,\theta\,((\epsilon\,\gamma\,\phi + K_2 K_4 K_6)\,K_5 + \gamma\,\rho\,\epsilon\,\sigma)}{(\lambda^{**}+\mu+\tau)\,(\alpha\,\lambda^{**}+\mu)\,K_1\,((\gamma\,\omega\,\phi - K_2 K_3 K_4)\,K_5 + \gamma\,\omega\,\rho\,\sigma)},$$

$$I_t^{**} = -\frac{(\epsilon K_3 + \omega K_6)\,\lambda^{**}\,(((\lambda^{**}+\tau)\,\alpha + \mu)\,\pi\,(1-p) + P\pi\,\alpha\,(\lambda^{**}+\mu+\tau))\,(\phi\,K_5 + \rho\,\sigma)\,\theta}{((\gamma\,\omega\,\phi - K_2 K_3 K_4)\,K_5 + \gamma\,\omega\,\rho\,\sigma)\,K_1\,(\alpha\,\lambda^{**}+\mu)\,(\lambda^{**}+\mu+\tau)},$$

$$A^{**} = -\frac{(\epsilon K_3 + \omega K_6)\,\lambda^{**}\,(((\lambda^{**}+\tau)\,\alpha + \mu)\,\pi\,(1-p) + P\pi\,\alpha\,(\lambda^{**}+\mu+\tau))\,\theta\,\rho\,K_4}{(\lambda^{**}+\mu+\tau)\,(\alpha\,\lambda^{**}+\mu)\,K_1\,(\gamma\,(\phi\,K_5+\rho\,\sigma)\,\omega - K_2 K_3 K_4 K_5)}.$$

Existence of Endemic Equilibrium

Descartes rule of signs is applied in determining the existence of EEP.

Descartes rule of sign

Let $P(x)$ be a polynomial of degree n such that $n \geq 2$ with real coefficients. The number of positive roots or zeros of P is equal to the number of changes of sign of $P(x)$ or less by an even number. The force of infection at equilibrium of 1 is given by;

$$\lambda^{**} = \beta\left(\frac{\eta_1 I_1^{**} + \eta_2 I_2^{**} + I_t^{**}}{N^{**}}\right), \tag{11}$$

where,

$$N^{**} = S_u^{**} + S_1^{**} + I_2^{**} + I_3^{**} + I_t^{**} + A^{**}.$$

Substituting Eq. (10) into Eq. (11) gives $\lambda^{**} = 0$ and the quadratic equation in terms of λ^{**} stated as follows;

$$C_1 \lambda^{**^2} + C_2 \lambda^{**} + C_3 = 0, \tag{12}$$

where,

$$\begin{aligned}
C_1 &= \alpha[(\phi+\rho)\,(\mu+\delta_2)\,\omega K_5 + (\mu+\delta_1)\,\rho\gamma\omega + (K_2+\omega)\,\mu K_4 K_5 \\
&\quad + \theta\,(\epsilon K_3 + \omega K_6)\,(K_4 K_5 + \phi K_5 + \rho\sigma + \rho K_4)] + \alpha\,[(\epsilon\gamma\phi + K_2 K_4 K_6)\,\theta K_5 + \gamma\rho\sigma\epsilon\theta], \\
C_2 &= [(\phi+\rho)\,(\mu+\delta_2)\,\omega K_5 + (\mu+\delta_1)\,\rho\gamma\omega + (K_2+\omega)\,\mu K_4 K_5][K_1(\alpha(1-p)+p) + \alpha\,(\mu+\tau p) + \mu\,(1-p)] \\
&\quad + (\epsilon K_3 + \omega K_6)\,\theta\,(K_4 K_5 + \phi K_5 + \rho\sigma + \rho K_4) + (\epsilon\gamma\phi + K_2 K_4 K_6)\,\theta K_5 + \gamma\rho\sigma\epsilon\theta \\
&\quad - \alpha\beta\,[\eta_1\,((\phi+\rho)\,(\mu+\delta_2)\,\omega K_5 + (\mu+\delta_1)\,\rho\gamma\omega + (K_2+\omega)\,\mu K_4 K_5)] \\
&\quad + (\epsilon K_3 + \omega K_6)\,\theta\,(\eta_2 K_4 K_5 + \phi K_5 + \rho\sigma), \\
C_3 &= K_1\,(\mu+\tau)\,[(\phi+\rho)\,(\mu+\delta_2)\,\omega K_5 + (\mu+\delta_1)\,\rho\gamma\omega + (K_2+\omega)\,\mu K_4 K_5]\,(1-R_0).
\end{aligned}$$

The following results from the quadratic equation (12) are validated using the theorem below.

Theorem 4 *The endemic equilibrium (EE) of model (1) has a unique positive equilibrium whenever $R_0 > 1$.*

- (a) If $C_2 > 0$ and
  (i) $C_3 \geq 0$, model (1) has no positive equilibrium
  (ii) $C_3 < 0$, model (1) has a unique positive equilibrium
- (b) If $C_2 < 0$ and $C_3 > 0$ and
  (i) $C_2^2 - 4C_1 C_3 > 0$ model (1) has two positive equilibria

(ii) $C_2^2 - 4C_1C_3 = 0$ model (1) has a unique positive equilibrium

(iii) $C_2^2 - 4C_1C_3 < 0$ model (1) has no positive equilibrium

- (c) If $C_2 < 0$ and $C_3 \leqslant 0$, model (1) has a unique positive equilibrium.

Clearly, C_1 is always a positive real number, since all the parameters of the model are positive. The following cases are considered.

- case 1:if $C_2 > 0$ and $C_3 \geq 0 \Longleftrightarrow R_0 \leqslant 1$
  The quadratic equation (12) has no positive real root, which implies the model has no positive equilibrium.
- Vase 2: if $C_2 > 0$ and $C_3 < 0 \Longleftrightarrow R_0 > 1$
  The quadratic equation (12) has one positive real root, which implies the model has a unique positive equilibrium.
- Case 3: if $C_2 < 0$ and $C \leqslant 0 \Longleftrightarrow R_0 \leqslant 1$
  The quadratic equation (12) has one positive real root, which implies the model has a unique positive equilibrium.
- Case 4: if $C_2 < 0$ and $C_3 > 0 \Longleftrightarrow R_0 < 1$
  Quadratic equation (12) has either two, one or no positive real root depending on $C_2^2 - 4C_1C_3$, which implies the model has either two positive equilibria, unique positive equilibrium or no positive equilibrium.

From Case (4) when $R_0 < 1$, we have two equilibria exist. This implies the occurrence of backward bifurcation in model (1).

Bifurcation analysis

The HIV/AIDS dynamics model (1) exhibits backward (Subcritical) bifurcation near $R_o = 1$, that is the coexistence of disease-free equilibrium and endemic equilibrium when $R_0 < 1$. The epidemiological consequence of backward bifurcation is that $R_0 < 1$ will not guarantee the condition for disease control.

Centre manifold theorem stated in [25] is applied to model (1) for bifurcation analysis, to analyze the stability near disease-free equilibrium at $R_0 = 1$.

Let $\beta = \beta^{**}$ be the bifurcation parameter and $R_0 = 1$,
which implies;

$$\beta^{**} = \frac{k_1((-\gamma\,\omega\,\phi + K_2K_3K_4)K_5 - \gamma\,\omega\,\rho\,\sigma)(\tau + \mu)}{(((K_4K_6\eta_2\theta + \phi\,(-\gamma\,\eta_1 + K_6\theta))\omega + ((\varepsilon\theta\,\eta_2 + \eta_1K_2)K_4 + \phi\,\varepsilon\theta)K_3)K_5 + \rho\,\sigma((-\gamma\,\eta_1 + K_6\theta)\omega + K_3\varepsilon\theta))(\mu(1-p) + \alpha(\tau + \mu p))}.$$

The Theorem is applied by making the change of variables, let,
$S_u = x_1, S_e = x_2, I_1 = x_3, I_2 = x_4, I_3 = x_5, I_t = x_6$ and $A = x_7$ such that, $N = x_1 + x_2 + x_3 + x_4 + x_5 + x_6 + x_7$.

Therefore; the equation of model (1) can be written in the form:

$$\frac{dX}{dt} = (f_1, f_2, f_3, f_4, f_5, f_6, f_7)^T,$$

such that

$$\frac{dX_1}{dt} = f_1 = \pi(1-p) - (\mu+\tau)x_1 - \frac{\beta^{**}x_1(\eta_1 x_3 + \eta_2 x_4 + x_6)}{N},$$

$$\frac{dX_2}{dt} = f_2 = \pi p + \tau x_1 - \mu x_2 + \frac{\alpha\beta^{**}x_2(\eta_1 x_3 + \eta_2 x_4 + x_6)}{N},$$

$$\frac{dX_3}{dt} = f_3 = \frac{\beta^{**}x_1(\eta_1 x_3 + \eta_2 x_4 + x_6)}{N} + \frac{\alpha\beta^{**}x_2(\eta_1 x_3 + \eta_2 x_4 + x_6)}{N} - (\mu+\theta)x_3,$$

$$\frac{dX_4}{dt} = f_4 = \epsilon\theta x_3 + \omega x_5 - (\mu+\rho+\phi)x_4, \qquad (13)$$

$$\frac{dX_5}{dt} = f_5 = (1-\epsilon)\theta x_3 + \gamma x_6 - (\mu+\omega)x_5,$$

$$\frac{dX_6}{dt} = f_6 = \phi x_4 + \sigma x_7 - (\mu+\gamma+\delta_2)x_6,$$

$$\frac{dX_7}{dt} = f_7 = \rho x_4 - (\mu+\sigma+\delta_1)x_7.$$

Now, the Jacobian matrix of system (13) at disease-free equilibrium ϵ^0 is given by,

$$J(\epsilon^0) = \begin{bmatrix} -D_1 & 0 & -\eta_1 D_2 & -\eta_2 D_2 & 0 & D_2 & 0 \\ \tau & -\eta_1 & -\eta_1 D_3 & -\eta_2 D_3 & 0 & -D_3 & 0 \\ 0 & 0 & \eta_1(D_2+D_3)-K_3 & \eta_2(D_2+D_3) & 0 & (D_2+D_3) & 0 \\ 0 & 0 & \epsilon\theta & -K_2 & \omega & 0 & 0 \\ 0 & 0 & K_6\theta & 0 & -K_3 & \gamma & 0 \\ 0 & 0 & 0 & \phi & 0 & -K_4 & \sigma \\ 0 & 0 & 0 & \rho & 0 & 0 & -K_5 \end{bmatrix}, \qquad (14)$$

where, $D_1 = (\mu+\tau), D_2 = \frac{\beta^{**}\mu(1-p)}{\mu+\tau}, D_3 = \frac{\alpha\beta^{**}(\tau+\mu p)}{\mu+\tau}, K_1 = (\theta+\mu), K_2 = (\rho+\phi+\mu),$
$K_3 = (\omega+\mu), K_4 = (\gamma+\mu+\delta_2), K_5 = (\sigma+\mu+\delta_1)$ and $K_6 = (1-\epsilon)$.
The linearized system (14) with $\beta = \beta^{**}$ has a zero eigenvalues. Now, let $V = [v_1, v_2, v_3, v_4, v_5, v_6, v_7]$ and $W = [w_1, w_2, w_3, w_4, w_5, w_6, w_7]^T$ be the corresponding left and right eigenvectors associated with the simple zero eigenvalues of the Jacobian Matrix of system (14), respectively.
Solving for the left eigenvectors W we have,

$$w_1 = \frac{\rho\mu K_4 w_2 + \eta_1\rho D_3 K_4 w_3 + D_3(\eta_2 K_4 K_5 + \rho\sigma + \phi K_5)w_7}{\rho K_4} > 0,$$

$$w_2 = w_2 > 0, w_3 = w_3 > 0, w_4 = \frac{K_5 w_7}{\rho} > 0,$$

$$w_5 = \frac{\rho\theta K_4 K_6 w_3 + \gamma(\rho\sigma + \phi K_5)w_7}{\rho K_3 K_4} > 0,$$

$$w_6 = \frac{(\rho\sigma + \phi K_5)w_7}{\rho K_4} > 0, w_7 = w_7 > 0.$$

Similarly, solving for the right eigenvectors V we have,

$$v_1 = v_2 = 0, v_3 = \frac{K_4 K_5 v_7 - \rho \gamma v_5}{\rho(D_2 + D_3)} > 0,$$

$$v_4 = \frac{K_3 v_5}{\omega} < 0, v_5 = v_5 < 0, v_6 = \frac{K_5 v_7}{\sigma} > 0, v_7 = v_7 > 0.$$

We used [25] as stated by [10] to find the direction of the bifurcation by computing a and b with,

$$a = \sum_{k,i,j=1}^{7} v_k w_i w_j \frac{\partial^2 f_k}{\partial x_i \partial x_j}(0,0), \quad b = \sum_{k,i=1}^{7} v_k w_i \frac{\partial^2 f_k}{\partial x_i \partial \beta^{**}}(0,0).$$

Now, computing the partial derivatives of system (13) which are non-zero. Since $v_1 = v_2 = 0$, and the second partial derivative of f_4, f_5, f_6 and f_7 are zeros, we only consider for $k = 3$ that is;

$$\frac{dX_3}{dt} = f_3 = \frac{\beta^{**} x_1 (\eta_1 x_3 + \eta_2 x_4 + x_6)}{N} + \frac{\alpha \beta^{**} x_2 (\eta_1 x_3 + \eta_2 x_4 + x_6)}{N} - (\mu + \theta) x_3.$$

We get;

$$\frac{\partial^2 f_3}{\partial x_1 \partial x_3} = \frac{\beta^{**} \eta_1}{N}, \frac{\partial^2 f_3}{\partial x_1 \partial x_4} = \frac{\beta^{**} \eta_2}{N}, \frac{\partial^2 f_3}{\partial x_1 \partial x_6} = \frac{\beta^{**}}{N},$$

$$\frac{\partial^2 f_3}{\partial x_2 \partial x_3} = \frac{\alpha \beta^{**} \eta_1}{N}, \frac{\partial^2 f_3}{\partial x_2 \partial x_4} = \frac{\alpha \beta^{**} \eta_2}{N}, \frac{\partial^2 f_3}{\partial x_2 \partial x_6} = \frac{\alpha \beta^{**}}{N}.$$

Therefore;

$$a = v_3 \sum_{i,j=3}^{7} w_i w_j \frac{\partial^2 f_3}{\partial x_i \partial x_j}(0,0) = \beta^{**} v_3 (w_1 + \alpha w_2)(\eta_1 w_3 + \eta_2 w_4 + w_6) > 0.$$

Similarly;

$$b = v_3 \sum_{i=3}^{7} w_i \frac{\partial^2 f_3}{\partial x_i \partial \beta^{**}}(0,0) = \frac{v_3 (\eta_1 w_3 + \eta_2 w_4 + w_6)(\mu(1-p) + \alpha(\tau + \mu p))}{\mu + \tau} > 0.$$

Thus, we have $a > 0$ and $b > 0$. The following theorem holds:

Theorem 5 *When $\beta^{**} < 0$ the system is locally asymptotically stable and there exists a positive unstable equilibrium, while if $\beta^{**} > 0$ is unstable and there exists a negative and locally asymptotically stable equilibrium. Hence the requirement of having $R_0 < 1$ will not suffice the condition for the control of HIV/AIDS.*

Global stability of endemic equilibrium

Theorem 6 *The endemic equilibrium (EE), ϵ^{**} of model (1) is globally asymptotically stable (GAS) if $R_0 > 1$ and unstable if $R_0 < 1$.*

Proof We construct a Lyapunov function

$$V = \left(S_u - S_u^{**} - S_u^{**}ln\left(\frac{S_u}{S_u^{**}}\right)\right) + \left(S_e - S_e^{**} - S_e^{**}ln\left(\frac{S_e}{S_e^{**}}\right)\right) + \left(I_1 - I_1^{**} - I_1^{**}ln\left(\frac{I_1}{I_1^{**}}\right)\right)$$

$$+ B_1\left(I_2 - I_2^{**} - I_2^{**}ln\left(\frac{I_2}{I_2^{**}}\right)\right) + B_2\left(I_3 - I_3^{**} - I_3^{**}ln\left(\frac{I_3}{I_3^{**}}\right)\right) + B_3\left(I_t - I_t^{**} - I_t^{**}ln\left(\frac{I_t}{I_t^{**}}\right)\right)$$

$$+ B_4\left(A - A^{**} - A^{**}ln\left(\frac{A}{A^{**}}\right)\right),$$

(15)

where B_1, B_2, B_3 and B_4 are non negative constant. Differentiating V, we get

$$V' = \left(1 - \frac{S_u^{**}}{S_u}\right)S_u' + \left(1 - \frac{S_e^{**}}{S_e}\right)S_e' + \left(1 - \frac{I_1^{**}}{I_1}\right)I_1' + B_1\left(1 - \frac{I_2^{**}}{I_2}\right)I_2'$$

$$+ B_2\left(1 - \frac{I_3^{**}}{I_3}\right)I_3' + B_3\left(1 - \frac{I_t^{**}}{I_t}\right)I_t' + B_4\left(1 - \frac{A^{**}}{A}\right)A'.$$

(16)

Substituting (1) that is $S_u', S_e', I_1', I_2', I_3, I_t'$ and A' in to (16) we have,

$$V' = \pi(1-p) - \lambda S_u - \mu S_u - \frac{S_u^{**}}{S_u}(\pi(1-p) - \lambda S_u - \mu S_u) + \pi p - \alpha\lambda S_e - \mu S_e$$

$$- \frac{S_e^{**}}{S_e}(\pi p - \alpha\lambda S_e - \mu S_e) + \lambda S_u + \alpha\lambda S_e - (\mu+\theta)I_1 - \frac{I_1^{**}}{I_1}(\lambda S_u + \alpha\lambda S_e - (\mu+\theta)I_1)$$

$$+ B_1(\omega I_3 - (\mu+\rho)I_2) - B_1\frac{I_2^{**}}{I_2}(\omega I_3 - (\mu+\rho)I_2) + B_2(\gamma I_t - (\mu+\omega)I_3)$$

$$- B_2\frac{I_3^{**}}{I_3}(\gamma I_t - (\mu+\omega)I_3) + B_3(\sigma A - (\mu+\gamma)I_t) - B_3\frac{I_t^{**}}{I_t}(\sigma A - (\mu+\gamma)I_t)$$

$$+ B_4(\rho I_2 - (\mu+\sigma)A) - B_4\frac{A^{**}}{A}(\rho I_2 - (\mu+\sigma)A).$$

Setting the coefficient of I_2, I_3, I_t, and A to zero, the positive constant is determined as, $B_1 = (\mu+\omega), B_2 = \omega, B_3 = \frac{(\mu+\omega)(\mu+\rho)(\mu+\sigma)}{\rho\sigma}, B_4 = \frac{(\mu+\omega)(\mu+\rho)}{\rho}$. Now setting $\delta_1 = \delta_2 = 0$ in system (1), $N \to \frac{\pi}{\mu}$ as $t \to \infty$

Let $\tilde{\beta} = \frac{\mu\beta}{\pi}$,

$$V' = \pi(1-p) - \lambda S_u - \mu S_u - \frac{S_u^{**}}{S_u}(\pi(1-p) - \lambda S_u - \mu S_u) + \pi p - \alpha\lambda S_e - \mu S_e$$

$$- \frac{S_e^{**}}{S_e}(\pi p - \alpha\lambda S_e - \mu S_e) + \lambda S_u + \alpha\lambda S_e - (\mu+\theta)I_1 - \frac{I_1^{**}}{I_1}(\lambda S_u + \alpha\lambda S_e - (\mu+\theta)I_1)$$

$$+ (\mu+\omega)(\omega I_3 - (\mu+\rho)I_2) - (\mu+\omega)\frac{I_2^{**}}{I_2}(\omega I_3 - (\mu+\rho)I_2)$$

$$+ \omega(\gamma I_t - (\mu+\omega)I_3) - \omega\frac{I_3^{**}}{I_3}(\gamma I_t - (\mu+\omega)I_3)$$

$$+ \frac{(\mu+\omega)(\mu+\rho)(\mu+\sigma)}{\rho\sigma}(\sigma A - (\mu+\gamma)I_t) - \frac{(\mu+\omega)(\mu+\rho)(\mu+\sigma)}{\rho\sigma}\frac{I_t^{**}}{I_t}(\sigma A - (\mu+\gamma)I_t)$$

$$+ \frac{(\mu+\omega)(\mu+\rho)}{\rho}(\rho I_2 - (\mu+\sigma)A) - \frac{(\mu+\omega)(\mu+\rho)}{\rho}\frac{A^{**}}{A}(\rho I_2 - (\mu+\sigma)A),$$

which can be shown from model (1) that at steady state,

$$\pi(1-p) = \lambda S_u^{**} + \mu S_u^{**}, \pi p = \alpha \lambda S_e^{**} + \mu S_e^{**}, (\mu + \theta) I_1^{**} = \lambda S_u^{**} + \alpha \lambda S_e^{**},$$
$$\omega I_3^{**} = (\mu + \rho) I_2^{**}, \gamma I_t^{**} = (\mu + \omega) I_3^{**}, \sigma A^{**} = (\mu + \gamma) I_t^{**}, \rho I_2^{**} = (\mu + \sigma) A^{**}.$$

Using the above relations, we have,

$$
\begin{aligned}
V' \leqslant & \left(2 - \frac{S_u}{S_u^{**}} - \frac{S_u^{**}}{S_u}\right) + \mu S_e^{**} \left(2 - \frac{S_e}{S_e^{**}} - \frac{S_e^{**}}{S_e}\right) + \lambda S_u^{**} \left(3 - \frac{S_u^{**}}{S_u} - \frac{I_1}{I_1^{**}} - \frac{I_1^{**}}{I_1}\right) \\
& + \alpha \lambda S_e^{**} \left(3 - \frac{S_e^{**}}{S_e} - \frac{I_1}{I_1^{**}} - \frac{I_1^{**}}{I_1}\right) + (\mu + \omega)(\mu + \rho) I_2^{**} \left(3 - \frac{A^{**}}{A} - \frac{I_2^{**} I_3}{I_2 I_3^{**}} - \frac{I_t I_3^{**}}{I_t^{**} I_3}\right) \\
& + \frac{(\mu + \omega)(\mu + \rho)(\mu + \sigma) A^{**}}{\rho} \left(2 - \frac{I_t}{I_t^{**}} - \frac{I_t^{**}}{I_t}\right).
\end{aligned}
$$

From the fact that the arithmetic mean surpasses the geometric mean, the following, inequalities hold:

$$\left(2 - \frac{S_u}{S_u^{**}} - \frac{S_u^{**}}{S_u}\right) \leqslant 0, \left(2 - \frac{S_e}{S_e^{**}} - \frac{S_e^{**}}{S_e}\right) \leqslant 0, \left(3 - \frac{S_u^{**}}{S_u} - \frac{I_1}{I_1^{**}} - \frac{I_1^{**}}{I_1}\right) \leqslant 0,$$

$$\left(3 - \frac{S_e^{**}}{S_e} - \frac{I_1}{I_1^{**}} - \frac{I_1^{**}}{I_1}\right) \leqslant 0, \left(3 - \frac{A^{**}}{A} - \frac{I_2^{**} I_3}{I_2 I_3^{**}} - \frac{I_t I_3^{**}}{I_t^{**} I_3}\right) \leqslant 0, \left(2 - \frac{I_t}{I_t^{**}} - \frac{I_t^{**}}{I_t}\right) \leqslant 0.$$

Thus, we have that, $V' \leqslant 0$ for $R_0 > 1$. The equality condition $V' = 0$ will strictly hold only when $S_e = S_e^{**}, I_1 = I_1^{**}, I_2 = I_2^{**}, I_3 = I_3^{**}, I_t = I_t^{**}$ and $A = A^{**}$. Thus the endemic equilibrium ϵ^* is the only invariant set of model (1). Therefore, by applying the Lasalle invariance principle [26] the result follows. Therefore, the endemic equilibrium (EE) of model (1) is globally asymptotically stable (GAS). ■

5 Model fitting and parameter calibration

After proposing an epidemiological model in terms of a nonlinear system of ordinary differential equations, it is of utmost importance to calibrate and estimate the suitable values of the biological parameters for the model to be of some use. It can be made possible only when one reaches authentic information about the actual data set for the epidemic over a certain period. This approach also helps one validate the proposed model for the disease under analysis. Several methods exist for calibrating and estimating such parameters, including the single shooting method, Gauss-Newton method, Nelder-Mead method, least squares, Monte Carlo sampling, and the local smoothing approach. Among the existing ones, the practice of least-squares is the most frequently used statistical approach for parameters' calibration in a nonlinear system of ordinary differential equations. The method minimizes discrepancies between actual data and the values predicted from the model's simulations for the infected class. The available data mainly presents the individuals infected with the disease. With the help of the least-squares method, the suitable estimated values of the parameters accompanying other essential information, including standard error, t-statistic, p-value, and the confidence interval, are computed in Table 2 wherein all p-values are < 0.05 with 95% confidence interval for each parameter. It may also be noted that the approximate value for the basic reproduction number is $R_0 = 3.85142$ while using the calibrated parameters given above and those estimated with the nonlinear least-squares method as shown in

Table 2. The most crucial statistical information, including minimum value, first quartile, second quartile, average, third quartile, maximum value, and standard deviation is collected in Table 3 for both real HIV cases and those predicted from the simulations of model (1). The values are in perfect agreement with each other for both cases. It is further ascertained in Figure 2 wherein the curve from the simulation of the newly infected cases (solid line) approaches the real HIV cases (solid dots) very well and thus in good alignment with the surveillance data. The residual plots of varying nature in Figure 3 are shown, with the x-axis being the period (1987-2014) and the y-axis for the residual values. These residuals seem to be equally and randomly spaced around the horizontal axis, making an ideal residual plot. Finally, a box and whisker plot is created in Figure 4 to obtain the additional detail for the analysis carried out in this section. The five statistics from the plot seem to have a good agreement, with the predicted one having an outlier.

Table 2. Values of fitted biological parameters including some important statistical measures obtained via least-squares non-linear curve fitting technique.

Par	Estimate	Standard error	t-statistic	p-value	Confidence interval
β	6.08348×10^{-1}	1.93409×10^{-2}	3.14541×10^{1}	1.26038×10^{-21}	$(5.68515 \times 10^{-1}, 6.48182 \times 10^{-1})$
ρ	7.14263	1.77252×10^{-1}	4.02965×10^{1}	2.8928×10^{-24}	$(6.77758, 7.50769)$
τ	5.51178×10^{-2}	6.80934×10^{-3}	8.09445	1.89584×10^{-8}	$(4.10937 \times 10^{-2}, 6.91419 \times 10^{-2})$

Table 3. Summary statistics for the real data, and the predicted data points obtained under simulations of model (1) for the newly infected individuals with HIV (I_1).

Data	Min.	1st Qu.	Median	Mean	3rd Qu.	Max.	SD
Real cases	6.1e+01	3.4750e+02	8.4600e+02	1.5013e+03	2.4720e+03	4.9460e+03	1.4877e+03
Predicted	6.1e+01	4.4027e+02	9.6770e+02	1.5474e+03	2.3224e+03	5.1929e+03	1.4394e+03

Table 4. Baseline values and ranges for parameters of model (1).

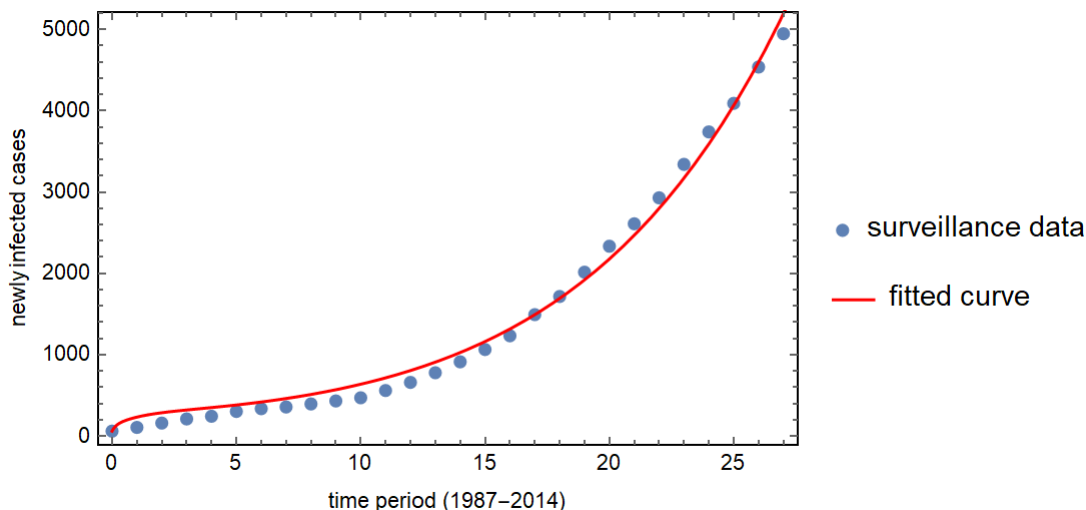| Parameters	Baseline (Range)	Units	Sources
N	$12,976,600$	Persons	Estimated
π	$969,907$	Year^{-1}	Estimated
μ	1.438×10^{-2}	Year^{-1}	[12]
δ_1	0.8	Year^{-1}	Estimated by [12]
δ_2	0.6	Year^{-1}	Estimated by [10]
p	0.02	Year^{-1}	fitted
τ	5.51178×10^{-2}	Year^{-1}	fitted
β	6.08348×10^{-1}	Year^{-1}	fitted
α	0.002	Year^{-1}	Estimated by [10]
θ	0.005	Year^{-1}	Estimated by [27]
ϵ	0.04	Year^{-1}	Estimated by [27]
ϕ	0.02	Year^{-1}	fitted
ρ	7.14263	Year^{-1}	fitted
γ	0.8	Year^{-1}	fitted
ω	0.93	Year^{-1}	fitted
η_1	0.6	Year^{-1}	fitted
η_2	0.5	Year^{-1}	fitted
σ	0.004	Year^{-1}	fitted

Figure 2. The best curve fitting for the real HIV cases [13] and the compartment of the newly infected cases from the proposed model given in model (1)
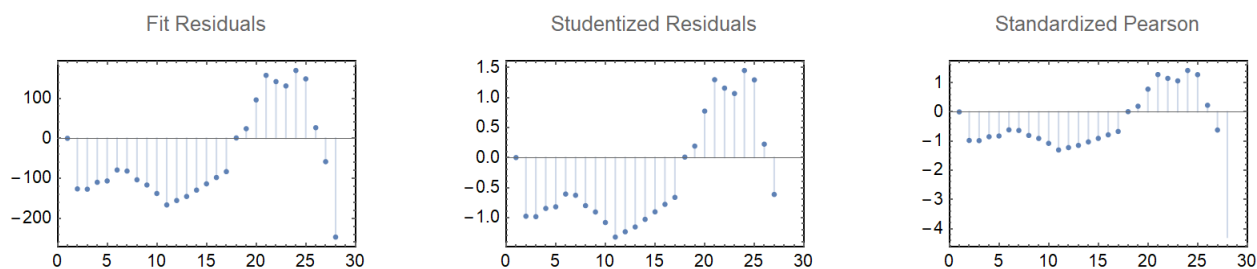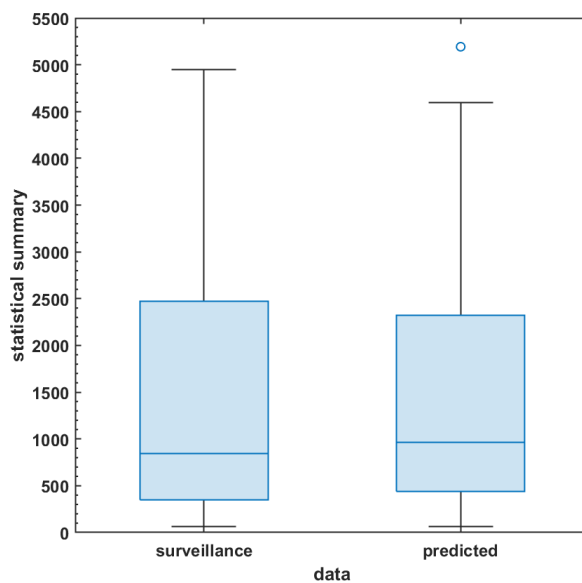

Figure 3. The residuals

Figure 4. The BoxWhisker chart for each real surveillance HIV data value and those predicted from the proposed model (1)

Sensitivity analysis

In this section, we use the forward sensitivity index method to analyze the proposed HIV model in relation to the reproduction number R_0 with respect to the biological parameters used in the model. The method used to describe the sign of each parameter to determine the most sensitive parameters used in the model, those parameters with the negative sign are regarded as the most sensitive for decreasing the value of R_0 while parameters with positive values are sensitive for the increase of R_0 [28, 29]. The normalized local sensitivity index of R_0 with respect to the parameters is given by,

$$\chi_C^{R_0} = \frac{C}{R_0} \times \frac{\partial R_0}{\partial C}. \tag{17}$$

The indices for R_0 with respect to parameters are obtained as shown in Table 5.

Table 5. Forward normalized sensitivity indices

Parameters	Elasticity indices	Values of the elasticity index
θ	$\chi_\theta^{R_0}$	-0.2532
σ	$\chi_\sigma^{R_0}$	-0.01304
ω	$\chi_\omega^{R_0}$	0.0317
β	$\chi_\beta^{R_0}$	1.0000
δ_2	$\chi_{\delta_2}^{R_0}$	0.4789
α	$\chi_\alpha^{R_0}$	0.0096
τ	$\chi_\tau^{R_0}$	-0.0600
ρ	$\chi_{\eta_2}^{R_0}$	0.070
ϕ	$\chi_\phi^{R_0}$	-0.1493

Figure 5. Bar chart pictorial representation

The most sensitive epidemiological parameters that effectively determined the control of the spread of HIV infection are obtained and represented using a bar chart given in the forward normalized sensitivity indices Table 5.

6 Numerical scenarios and discussion

The transmission dynamics of the governing model may be efficiently investigated by utilizing numerical simulations using state variables of interest. This section looks at several forms of time-series graphs using the parameters determined by the nonlinear minimum-squares fitting approach. The transmission dynamics of the model have been simulated by using state variables and the parameters in Table 4. The behaviour of the state variables and pattern of movement from one compartment to another are examined.

(a)   **(b)**
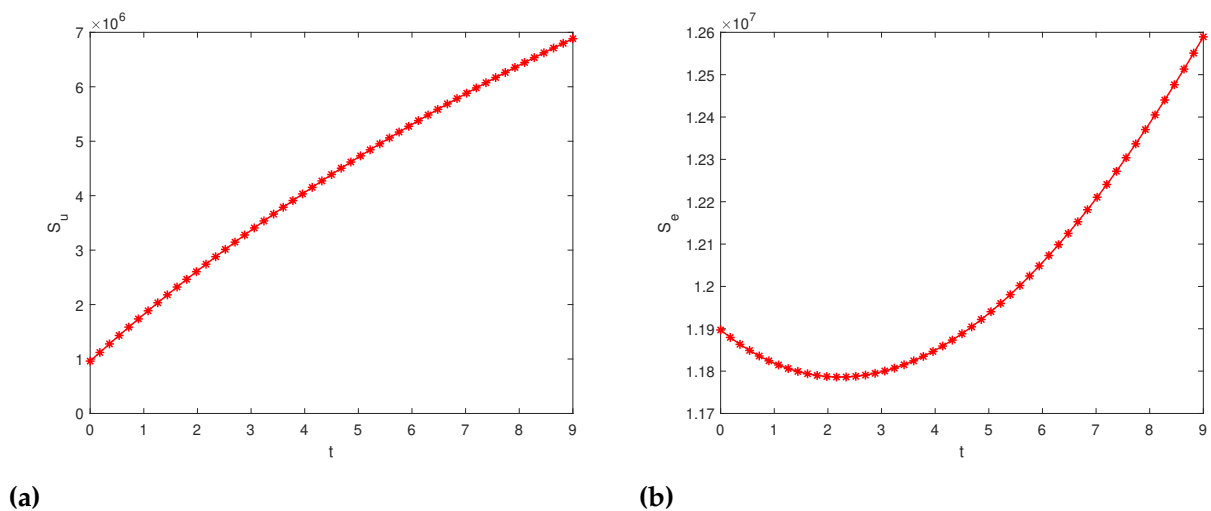
Figure 6. Behavior of the state variables (a) Susceptible Uneducated S_u, (b) Susceptible Educated S_e

(a)   **(b)**
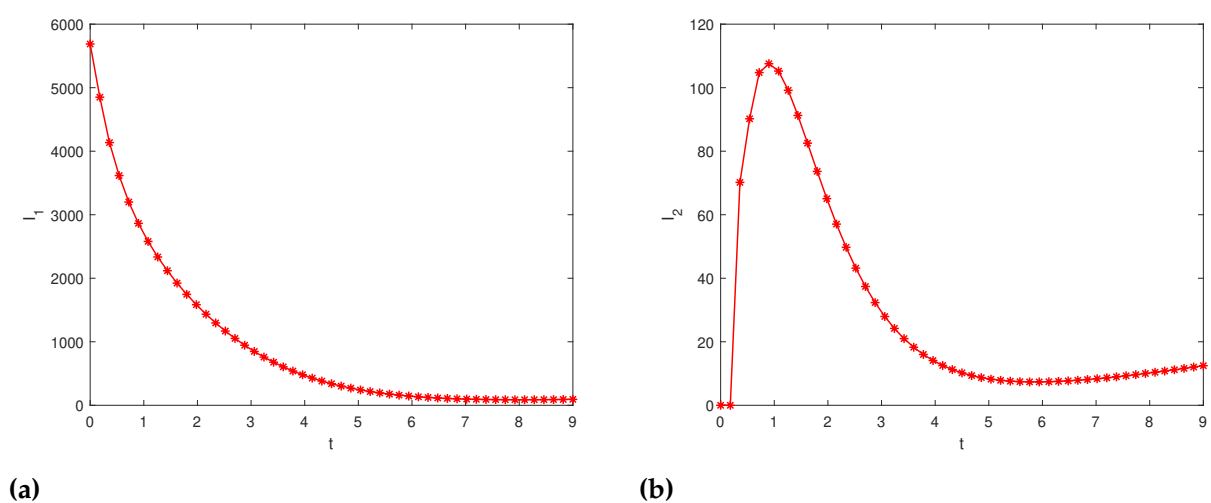
Figure 7. Behavior of the state variables (a) Newly infected individuals I_1, (b) Infected individuals with detectable viral load I_2

Figure 6(a) shows how the number of uneducated susceptible changes with time. The numbers

rise initially due to the recruitment of individuals and start decreasing due to the movement of individuals from the compartment to susceptible educated or newly infected compartment after being infected. Figure 6(b) shows how the number of susceptible educated rises with respect to time due to the movement of individuals from the uneducated compartment. Figure 7(a) shows how the newly infected individuals decrease persistently due to their movement into either infected individuals with undetectable viral load or infected individuals with detectable viral load. Figure 7(b) shows how the infected with detectable viral load raises due to the movement of newly infected individuals into the compartment, while decreases due to the movement of individuals into treatment or AIDS compartment.

(a)　　　　　　　　　　　　　　　　　　　　　**(b)**
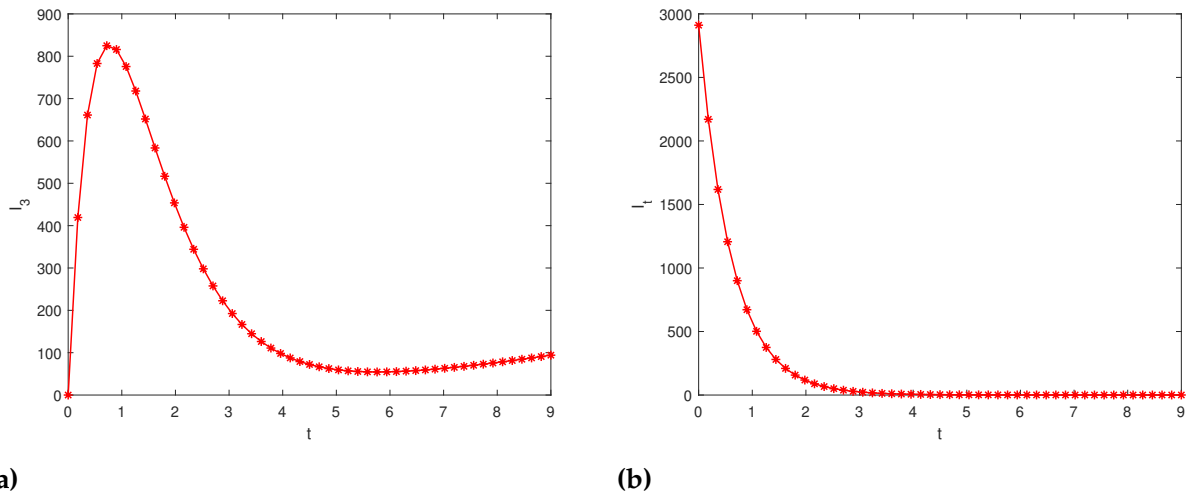
Figure 8. Behavior of the state variables (a) Infected individuals with undetectable viral load I_3, (b) Infected individuals under treatment I_t
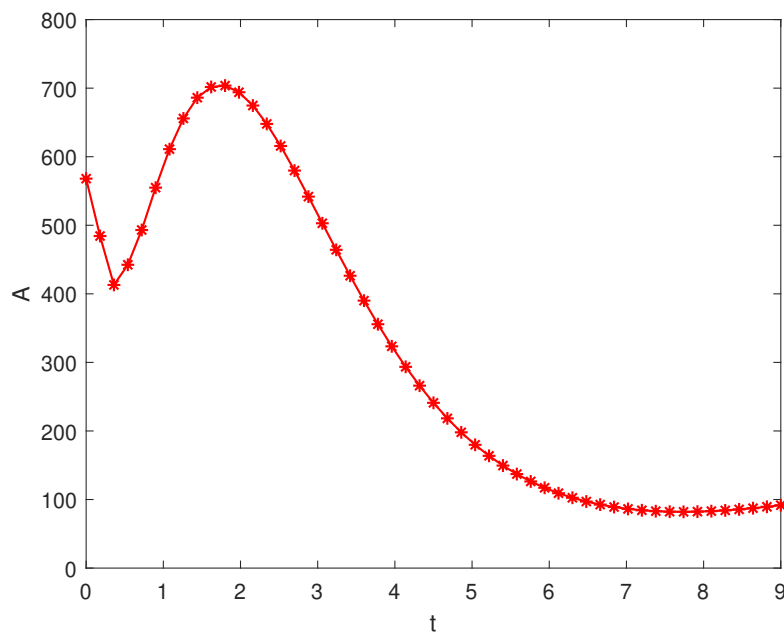
Figure 9. Behavior of the state variables AIDS patients A
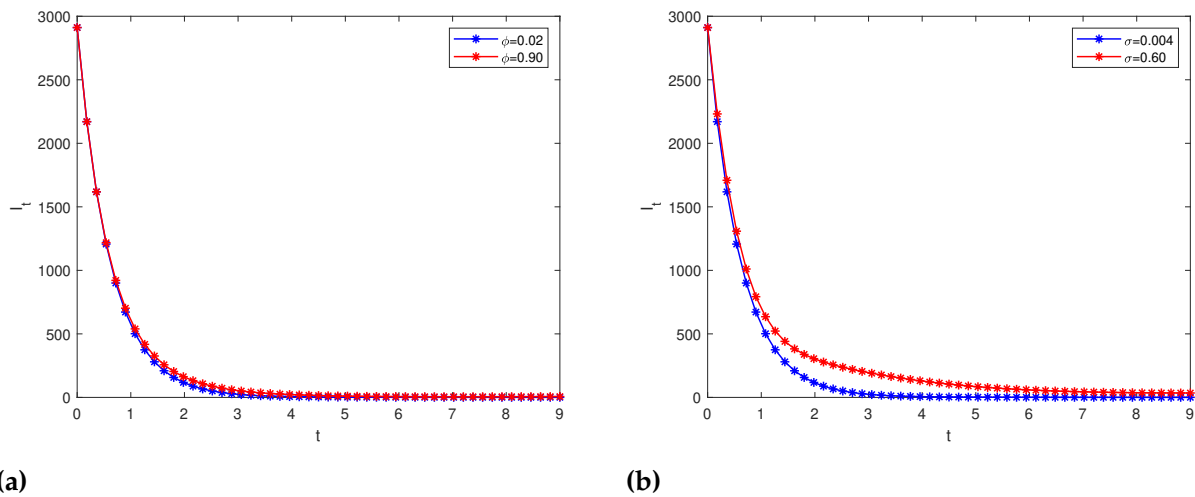
Figure 10. Patterns of I_t: (A) with different values of movement rate of infected with detectable viral load to treatment ϕ, (B) with different values of rate of movement of AIDS patients to treatment class at a rate σ

Figure 8(a) shows how infected individuals with undetectable viral load raises due to the movement of newly infected individuals into the compartment, while decreases after their viral load becomes detectable and moves into infected individuals with detectable viral load compartment. Figure 8(b) shows how infected individuals under treatment move out of the compartment to infected individuals with undetectable viral load after their viral load is suppressed.
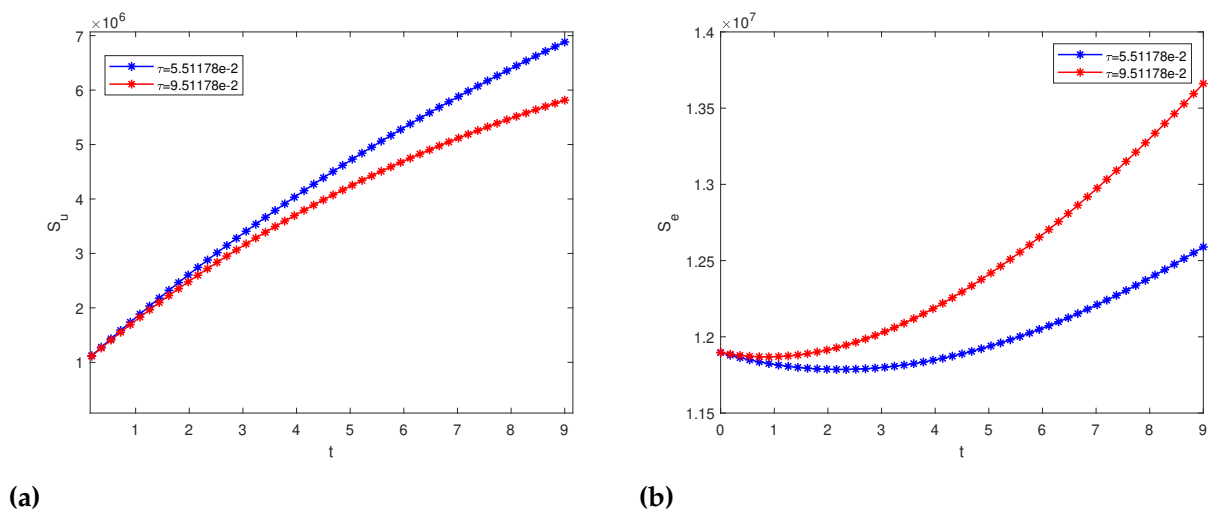
Figure 11. (a) Patterns of S_u with different values of rate at which uneducated susceptible become educated about HIV infection τ, (b) Patterns of S_e with different values of rate of movement of AIDS patients to treatment class at a rate τ

Figure 9 shows how AIDS patients decrease initially, possibly due to death, and later increase due to movement of infected individuals with detectable viral load, and decreases due to movement into treatment and death-related illness. Figures 10(a) and 10(b) show the pattern of infected individuals under treatment with different values of progression rate from undetectable viral load to treatment ϕ and progression rate from AIDS into treatment σ respectively. Despite the change in values of ϕ and σ, the graphs follow the exact pattern of I_t as in Figure 7(b). Change in values of ϕ and σ will be effective on the number of infected individuals with undetectable viral load and

AIDS-infected individuals, respectively. Figures 11(a) and 11(b) show the pattern of uneducated and educated susceptible individuals with different levels of education campaigns. The plots clearly show the impact of education parameter τ. As τ increases, the number of uneducated individuals decreases while educated individuals increase.

7 Summary and conclusion

In this paper, we have developed a nonlinear deterministic model that incorporates public awareness and treatment for the transmission dynamics of HIV/AIDS in an infected population with detectable and undetectable viral load. The analysis of the model reveals that the disease-free equilibrium is globally asymptotically stable whenever the associated reproduction number $R_0 < 1$ and unstable when $R_0 > 1$. Contrarily, the endemic equilibrium is globally asymptotically stable when the associated reproduction number is $R_0 > 1$ and unstable when $R_0 < 1$. Furthermore, the model undergoes the phenomenon of backward bifurcation in which a stable disease-free equilibrium coexists with a stable endemic equilibrium. The epidemiological implication of backward bifurcation is $R_0 < 1$ is a necessary but not sufficient condition for HIV control even when the classical requirement is satisfied, however the backward bifurcation analysis shows that when the bifurcation parameter $\beta^{**} < 0$ the system is locally asymptotically stable and there exists a positive unstable equilibrium, while if $\beta^{**} > 0$ is unstable and there exists a negative and locally asymptotically stable equilibrium. Hence, the requirement of having $R_0 < 1$ will not suffice the condition for the control of HIV/AIDS. The biological parameters of the model are fitted using the least square method with p-values < 0.05 and 95% confidence interval as shown in Table 2. The model was fitted with real HIV data cases on the newly infected compartment as shown in Figure 2. The most sensitive parameters for the control of the spread of HIV are identified using the forward sensitivity index method as shown in Figure 5, the most sensitive parameters that increase R_0 are β, ρ and ω, respectively. In addition, the numerical simulations carried out show the behavior of the state variables as shown in Figures 6,7,8, and 9. Similarly, Figure 11 shows the impact of public awareness. Finally, the results show that public awareness will help in curtailing the spread of HIV infection, and when treatment is applied to infected individuals with detectable viral load can easily suppress their virus to become undetectable so that they cannot transmit HIV through sexual intercourse. Future research should extend public awareness to infected individuals. In addition to this, a fractional order differential equation system can be used to describe HIV/AIDS transmission dynamics incorporating viral load detectability as the order has an effect on the dynamics and an optimal control problem can be applied to determine the optimal strategies for HIV eradication.

Declarations

Ethical approval

The authors state that this research complies with ethical standards. This research does not involve either human participants or animals.

Consent for publication

Not applicable.

Conflicts of interest

The authors declare that they have no conflict of interest.

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