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Modeling the Influence of Treatment Accessibility and Treatment Compliance on the Dynamics of HIV/AIDS

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Abstract The invention of highly active antiretroviral treatment (HAART) revolutionalized the treatment of HIV and brought hope to millions of individuals living with the virus. However, the eradication of HIV has proved difficult owing to many factors including accessibility and treatment compliance, particularly among many individuals in low income countries. Thus, we developed a model in terms of a system of nonlinear ordinary differential equations to assess the influence of inaccessibility of treatment and noncompliance with treatment guidelines by the HIV infectives who are aware of their status on the spread of HIV. The model was studied qualitatively and quantitatively using the theory of reproductive ratio and the software Maple respectively. The results of the analysis showed that early detection, treatment accessibility and treatment compliance by the majority of the infectives who know their status are crucial to the minimization of HIV incidence and prevalence.

Keywords Isolation, incubation period, asymptomatic, symptomatic, equilibria

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1. Introduction

The emergence of human immunodeficiency virus (HIV), the causative agent of the acquired immunodeficiency syndrome (AIDS), was announced by the Center for Disease Control and Prevention (CDC) at the start of the 1980s [1]. It was hoped that vaccines would be created to halt the spread of the disease or possibly eliminate it as soon as possible. But more than four decades later, no strategy has been effective to halt or eliminate the virus.

The transmission potential of HIV is affected by viral load - the quantity of virus in the blood. At present, highly active antiretroviral therapy (HAART) is the best method to suppress viral load as it reduces the chance of HIV spread by 96% [2]. However, the application of the drugs does not cure HIV permanently, but it does lower virus multiplicity and, as a result, AIDS related morbidity and mortality.

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Theoretically, HAART is thought to have three major effects: (i) a decreased risk of secondary infection [3, 4], (ii) a decreased infectivity per contact [5, 6], and (iii) an individual effect, such as an increased life span [7] that indicates a decreased risk of both AIDS and death from AIDS [8, 9].

A number of issues shape dynamics of HIV. For example, some HIV-positive individuals who are aware of the risks and effects of their infection may not always have access to therapy because it is difficult for them to get it or because the cost is too expensive for them to bear. This situation neglects this group of infectious agents and frequently causes a sharp increase in the rate of disease transmission in the community. The scenario may be related to the fact that a significant number of HIV-positive individuals, despite being aware of their status and in need of treatment, do not have access to HAART or receive it effectively in low and middle income countries (LMICs) (particularly in sub-Saharan Africa, where the pandemic is most severe).

Up till 2019, AIDS accounted for 32.7 million deaths worldwide, and 75.7 million people became infected with HIV, based on the information from the United Nations Program on HIV/AIDS [10]. Worldwide, there were 38 million individuals infected with HIV as of the end of 2019. In addition, 1.7 million individuals became newly infected with the virus during that year, and over 690,000 people died of AIDS-related ailments. HIV/AIDS is one of the major obstacles to development; it is not merely a health problem. It has a significant impact in LMICs, where 95% of all HIV/AIDS patients reside [11].

Mathematical models remain a crucial tool in epidemiology for comprehending the transmission of infectious diseases and the effects of intervention initiatives. The literature and growth of mathematical epidemiology is well documented [12,13], also see for various models [14–19]. Basically, mathematics plays a crucial part in the prevention of virus spread by enabling decision-makers to predict the effects of certain intervention strategies or to develop more effective techniques following the insights from mathematics. Based on the importance of mathematical models in disease dynamics, several HIV/AIDS mathematical models have been developed with one of the earliest attributable to Anderson et al. [20] where two mathematical models were presented: the first model captured HIV transmission in two populations of susceptible and infectious individuals; the second model considered three populations where there is one susceptible population and two infectious populations with one of infectious populations being AIDS group. Motivated by Anderson et al. [20], several studies have been conducted over the years on various aspects of HIV/AIDS dynamics e.g. transmission [21,22], diagnosis [23,24], treatments [25,26] and optimal control [27–29].

In their contributions to the study of HIV/AIDS, Udoo and Ashezua [30] developed a mathematical model to analyze the contribution to the spread of HIV/AIDS of the HIV positive individuals who are in need of treatment but cannot receive it due to inaccessibility and poor delivery, particularly in resource-poor nations. The researchers considered five compartments - susceptible $S(t)$, infected individuals who are ignorant of their HIV infection $I_1(t)$, infected individuals who are not ignorant of their HIV infection $I_2(t)$, infected individuals who are not ignorant of their HIV infection but cannot access treatment $I_3(t)$ and infected individuals who are not ignorant of their HIV infection and are receiving treatment termed recovered class $R(t)$. The authors discovered that the inability to access treatment has the potential to increase and sustain the endemicity of HIV/AIDS epidemic.

The analysis in [30] leaves some gaps. First, while there are numerous evidences to support inaccessibility and inadequate distribution of HAART in LMICs, accessibility to HAART is also a source of concern to people living with HIV (PLHIV) in resource-rich nations. For instance, only 25% of PLHIV in the US have suppressed viral load [31]. Also, as of 2011, 65% of PLHIV were infectious as a result of unsuppressed viral load in Canada [32]. Therefore, a model that limits HAART poor coverage to only resource-poor nations does not really capture the reality on ground. Second, the use of the term "recovered class $R(t)$" in the HIV/AIDS study is inappropriate as it can be misinterpreted as HIV eradication. Experts have advocated for HIV persistence management and warned against the use of any terms that may connote eradication in HIV/AIDS analysis as the disease has not met the eradicability criteria of the World Health Organization (WHO) and the International Task Force for Disease Eradication (ITFDE) [33–35].

Aside the influence of treatment and treatment accessibility on the dynamics of infectious diseases, many scholars, for example [36,37], have created and researched mathematical models illustrating the impact of awareness programs in halting the development of epidemics because they cause changes in people's behaviors and attitudes toward infection and also make it easier to take precautions that can lower the likelihood of contracting the disease. [12] proposed an SIS epidemic model to study sexually transmitted infections (STIs) with considerations that all individuals in the population are aware of the danger of the disease but only few decides to restrain by reducing their interactions with the infected individuals and looking for instant treatments. Also, [38] constructed a model to analyze the impacts of awareness strategies on the transmission of flu. They discovered that awareness programs could minimize flu but flu eradication would prove difficult with the existence of immigration. Furthermore, the general implications of awareness campaigns on the propagation of infectious diseases have also been studied by some researchers [39,40].

Recently, Ayele et al. [27] designed a model to study the roles of awareness in HIV/AIDS transmission in Ethiopia. They considered two sets of susceptible and infectious classes. Individuals who are conscious of the risks of HIV/AIDS and those who are unconscious of it are termed aware susceptibles and unaware susceptibles respectively. Also, infectives who are unaware of their disease status and those who are aware of it are termed undiagnosed infecteds and diagnosed infecteds respectively. A separate compartment is also incorporated for infected individuals who are receiving treatment and those who have developed AIDS. The authors studied the model mathematically and discovered that an upsurge in the population of unaware susceptibles and undiagnosed infecteds is the main challenge to HIV/AIDS minimization in Ethiopia. They advocated for increase in media campaigns, screenings and treatments to overcome HIV/AIDS scourge in Ethiopia.

HAART accessiblity is one of the main challenges to HIV/AIDS control. Sirega et al. [41] and UNAIDS [42] corroborate the claim of Udoo and Ashezua [30] that a good number of PLHIV do not have access to treatment. It was reported in [41] that 24% of PLHIV in Indonesia receive HAART while only 30% of PLHIV in the entire LMICs have access to HAART [42]. Nevertheless, the place of treatment in the control of HIV cannot be overemphasized and many studies have expounded benefits of HAART development in the revolution of HIV control and restoration of hopes to PLHIV [43–45]. However, while it is one thing to have access to HAART, it is another thing to adhere to HAART guidelines. Beacroft and Hallett [46] have identified poor adherence to HAART guidelines as a major obstacle to HIV con-

trol response. It was reported in [47] that many PLHIV especially young people in South Africa (aged 15-24) do not adhere to HAART. Therefore, there is need to focus on the implications of treatment accessibility and treatment compliance on the dynamics of HIV/AIDS to properly guide the policy makers better approaches to combat HIV/AIDS pandemic. Although HIV/AIDS pandemic has been a subject of intense mathematical and non-mathematical studies, studies on the overall consequences of non compliance with the treatment procedures on the dynamics of HIV/AIDS are relatively new in the literature. Specifically, while Udoo and Ashezua [30] and Ayele et al. [27] studied HIV/AIDS models with aware infectives who are on HAART, their models exclude the effects of aware infectives who do not adhere to treatment procedures.

The present study therefore considers a population wide HIV transmission and examines the consequences of inaccessibility of HAART and non compliance with HAART guidelines on the spread of HIV/AIDS. The rest of the paper is organized as follows. In Section 2, the model and its properties are presented. In Section 3, the qualitative analysis is presented. The model is studied numerically in Section 4 and the discussion of results is offered. In Section 5, we present the conclusion.

2. Materials and methods

The model considers a low income society $N(t)$, a population that is sexually active and partitioned into compartments: $S(t)$ (individuals who can contract HIV), $I(t)$ (HIV positive individuals who have not been aware of their status), $I_1(t)$ (HIV positive individuals who have been aware of their status and are accessing treatment and complying with treatment guidelines), $I_2(t)$ (HIV positive individuals who have been aware of their status and are accessing treatment but not complying with treatment guidelines), $I_3(t)$ (HIV positive individuals who have been aware of their status but cannot access treatment), and $A(t)$ (HIV positive individuals who have developed AIDS). Hence,

$$N(t) = S(t) + I(t) + I_1(t) + I_2(t) + I_3(t) + A(t). \tag{2.1}$$

Because of its pandemic nature and the fact that HIV is associated with vertical transmission, new entrants into the population (either by birth or immigration) may share the features of individuals in $S(t)$, $I(t)$, $I_1(t)$, $I_2(t)$ or $I_3(t)$. Therefore, new recruitment is assumed to occur at rate θ with $(1-b_1-b_2-b_3-b_4)\theta$, $b_1\theta$, $b_2\theta$, $b_3\theta$ and $b_4\theta$ recruited into $S(t)$, $I(t)$, $I_1(t)$, $I_2(t)$ and $I_3(t)$ respectively. Susceptible population may contract HIV if they come into contact with individuals in $I_1(t)$, $I_2(t)$ or $I_3(t)$ with a force of infection λ defined as

$$\lambda = \beta(I + (1-\phi_1)I_1 + (1-\phi_2)I_2 + (1-\phi_3)I_3), \tag{2.2}$$

where β is the contact rate that is sufficient to result in infection and ϕ_1, ϕ_2 and ϕ_3 are the HIV reduction factors due to treatments either with or without compliance for individuals in $I_1(t)$ and $I_2(t)$ and due to just awareness of one's health status for individuals in $I_3(t)$. Since human behavior is not predictable, allowances are made for flexibility and changes in human behaviors for individuals in $I_1(t), I_2(t)$ and $I_3(t)$ in terms of ϕ_1, ϕ_2, and ϕ_3 respectively. There is tendency for individuals who have been accessing treatment and complying with the treatment guidlines to relax. On the other hand, individuals who have been accessing the treatment

but not complying with the treatment procedures may turn over a new leaf. Also, individuals who do not have access to treatment may begin to access it. These possible scenarios can account for fluctuations in the values of ϕ_1, ϕ_2 and ϕ_3 but were not considered in the previous work [27, 30].

Although individuals in $I_2(t)$ and $I_3(t)$ may exercise caution/restraint because they are aware of their status, they may spread the virus intentionally for reasons that are best known to them. On the other hand, individuals in $I(t)$ spread the virus unintentionally because they are unaware of their status. Therefore, individuals in $S(t)$ are at higher risk of HIV infection from individuals in $I(t)$.

A fraction τ of HIV positive individuals who have not been aware of their status become aware at a rate α and then move to $I_1(t)$, $I_2(t)$ and $I_3(t)$ with proportion $(1-\tau_1-\tau_2)\alpha$, $\tau_1\alpha$ and $\tau_2\alpha$ respectively. HIV infection graduates to AIDS at the final stage so individuals in $I(t), I_1(t), I_2(t)$ and $I_3(t)$ develop AIDS at rates $\vartheta_1, \vartheta_2, \vartheta_3$ and ϑ_4 respectively with $\vartheta_2 < \vartheta_3 < \vartheta_1 < \vartheta_4$. Natural mortality occurs at rate μ in all compartments while in addition to μ, death due to AIDS occurs at rate δ only in $A(t)$ compartment. Transmission diagram of the infection is illustrated in Figure 1.

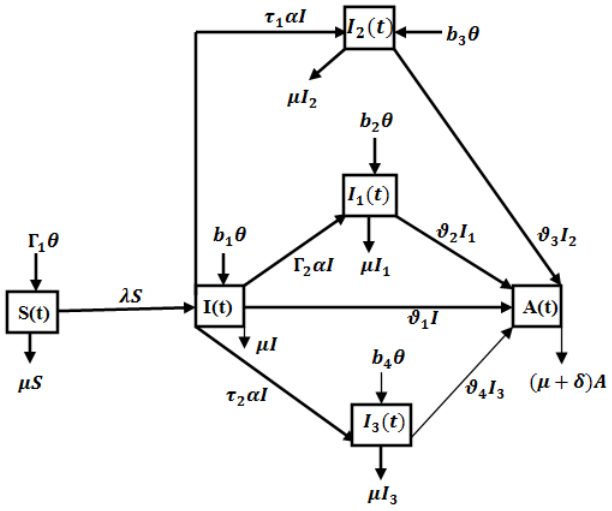


Figure 1. Transfer diagram of the model

In Figure 1, we have $\Gamma_1 = 1 - b_1 - b_2 - b_3 - b_4, \Gamma_2 = 1 - \tau_1 - \tau_2$ and $\lambda = \beta(I + (1-\phi_1)I_1 + (1-\phi_2)I_2 + (1-\phi_3)I_3)$. Given the formulation and Figure 1, we

come about the following system of ODEs

$$\begin{aligned}
\dot{S} &= (1-b_1-b_2-b_3-b_4)\theta - \beta(I+(1-\phi_1)I_1+(1-\phi_2)I_2 \\
&\quad +(1-\phi_3)I_3)S - \mu S, \\
\dot{I} &= b_1\theta + \beta(I+(1-\phi_1)I_1+(1-\phi_2)I_2+(1-\phi_3)I_3)S - (\mu+\alpha+\vartheta_1)I, \\
\dot{I}_1 &= b_2\theta + (1-\tau_1-\tau_2)\alpha I - (\mu+\vartheta_2)I_1, \\
\dot{I}_2 &= b_3\theta + \tau_1\alpha I - (\mu+\vartheta_3)I_2, \\
\dot{I}_3 &= b_4\theta + \tau_2\alpha I - (\mu+\vartheta_4)I_3, \\
\dot{A} &= \vartheta_1 I + \vartheta_2 I_1 + \vartheta_3 I_2 + \vartheta_4 I_3 - (\mu+\delta)A,
\end{aligned} \tag{2.3}$$

with nonnegative initial conditions

$$S(0) > 0, I(0) \geq 0, I_1(0) \geq 0, I_2(0) \geq 0, I_3(0) \geq 0, A(0) \geq 0. \tag{2.4}$$

Variables and parameters descriptions are stated below in Table 1 and Table 2 for ease of reference.

Table 1. Nomenclatures for the model variables

Model Variables	**Descriptions**
S	Susceptible population at time t
I	Infectious population at time t who are unaware of their HIV positive status
I_1	Infectious population at time t who are aware of their HIV positive status, have access to HAART and adhering to HAART procedures
I_2	Infectious population at time t who are aware of their HIV positive status, have access to HAART but do not adhere to HAART procedures
I_3	Infectious population at time t who are aware of their HIV positive status but do not have access to HAART

Table 2. Nomenclatures for the model parameters

Model Parameters	**Nomenclatures**
b_1, b_2, b_3, b_4	fraction of humans recruited into I, I_1, I_2 and I_3 classes respectively
θ	recruitment rate by birth or immigration
β	effective contact rate
ϕ_1, ϕ_2, ϕ_3	HIV reduction factors for individuals in $I_1(t)$, $I_2(t)$ and $I_3(t)$
τ_1, τ_2	fraction of HIV individuals who join I_2 and I_3 after awareness of their status
α	rate at which HIV individuals become aware of thier status
$\vartheta_1, \vartheta_2, \vartheta_3, \vartheta_4$	rate at which $I(t), I_1(t), I_2(t)$ and $I_3(t)$ develop AIDS
μ	mortality rate unrelated to HIV
δ	mortality rate due to HIV

The analysis can be restricted to the first five equations in (2.3) without changing the dynamics of the model since AIDS compartment does not contribute to other compartments [64–66]. The basic features of epidemic models would be verified for the HIV model and the sixth equation in (2.3) would be excluded from the study.

It is sufficient to restrict the study to the first five equations in (2.3) since the AIDS individuals do not influence disease dynamics. Hence, the system (2.3) is reduced to system (2.5) below

$$\begin{aligned}
\dot{S} &= (1-b_1-b_2-b_3-b_4)\theta - \beta(I+(1-\phi_1)I_1+(1-\phi_2)I_2 \\
&\quad +(1-\phi_3)I_3)S - \mu S, \\
\dot{I} &= b_1\theta + \beta(I+(1-\phi_1)I_1+(1-\phi_2)I_2+(1-\phi_3)I_3)S \\
&\quad -(\mu+\alpha+\vartheta_1)I, \\
\dot{I}_1 &= b_2\theta + (1-\tau_1-\tau_2)\alpha I - (\mu+\vartheta_2)I_1, \\
\dot{I}_2 &= b_3\theta + \tau_1\alpha I - (\mu+\vartheta_3)I_2, \\
\dot{I}_3 &= b_4\theta + \tau_2\alpha I - (\mu+\vartheta_4)I_3.
\end{aligned} \tag{2.5}$$

First, the well-posedness of the system (2.5) is examined and to achieve this, we verify whether the model is biologically and mathematically well defined.

Lemma 2.1. *Consider the initial conditions of the model in* (2.4)*. Then, the model solutions* $S(t), I(t), I_1(t), I_2(t)$ *and* $I_3(t)$ *are positive for all* $t > 0$.

Proof. Suppose

$$\hat{t} = sup\left\{t > 0 : S(t), I(t), I_1(t), I_2(t), I_3(t) > 0 \in [0,t]\right\}.$$

Then, $\hat{t} > 0$, and from the first equation in (2.5), it follows that

$$\dot{S} = \Gamma_1 - (\lambda+\mu)S.$$

Therefore,

$$\begin{aligned}
&\frac{d}{dt}\left[S(t)\exp\left\{\mu t + \int_0^t \lambda(s)ds\right\}\right] = \Gamma_1 \exp\left[\mu t + \int_0^t \lambda(s)ds\right] \\
\Longrightarrow\quad & S(t_1)\exp\left\{\mu t_1 + \int_0^{t_1}\lambda(s)ds\right\} - S(0) \\
&\qquad = \int_0^{t_1}\Gamma_1 \exp\left[\mu y + \int_0^y \lambda(s)ds\right]dy.
\end{aligned} \tag{2.6}$$

Hence,

$$\begin{aligned}
S(t_1) =& S(0)\exp\left\{-\mu t_1 - \int_0^{t_1}\lambda(s)ds\right\} + \exp\left\{-\mu t_1 - \int_0^{t_1}\lambda(s)ds\right\} \\
&\times \int_0^{t_1}\Gamma_1 \exp\left[\mu y + \int_0^y \lambda(s)ds\right]dy > 0.
\end{aligned} \tag{2.7}$$

Therefore, $S(t) > 0, \forall t > 0$. Similarly, we can establish that $I(t) > 0, I_1(t) > 0, I_2(t) > 0$ and $I_3(t) > 0$ which completes the proof for positivity of the model solutions. □

Also, the boundedness property can be verified for the HIV/AIDS model described in system (2.5).

Lemma 2.2. *Suppose the model's feasible region is defined as*

$$\Omega = \left\{S(t), I(t), I_1(t), I_2(t), I_3(t) \in \mathbb{R}_+^5 | 0 \le N \le \frac{\theta}{\mu}\right\}.$$

Then, Ω *is positively invariant and attracting for all* $t > 0$.

Proof. Summing up the model's equation in (2.5), we have

$$\begin{aligned} N'(t) &= \theta - \mu N - \vartheta_1 I - \vartheta_2 I_1 - \vartheta_3 I_2 - \vartheta_4 I_3 \\ &\leq \theta - \mu N. \end{aligned} \tag{2.8}$$

Hence, $N'(t) \leq 0$ if $N(t) \geq \dfrac{\theta}{\mu}$, since the RHS is bounded above by $\theta - \mu N$. Integrating (2.8) then,

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

In particular, $N(t) \leq \dfrac{\theta}{\mu}$ if $N(0) \leq \dfrac{\theta}{\mu}$. Also, if $N(0) > \dfrac{\theta}{\mu}$, then $N(t)$ enters Ω or approaches it asymptomatically. Therefore, the region Ω is positively invariant and attracting under the flow induced by the model. Thus, in Ω, the model is epidemiologically well-posed. Hence, the model can be studied in Ω. □

3. Qualitative analysis

The system (2.5) shall be studied analytically to obtain some basic results.

3.1. Disease-free equilibria (DFE)

Assuming $\mathcal{E}_\circ$ represents the DFE such that $\mathcal{E}_\circ = (S^\circ, I^\circ, I_1^\circ, I_2^\circ, I_3^\circ)$. Then, solving for the model variables when the RHS of the system (5) is zero, we obtain

$$\mathcal{E}_\circ = \left(\frac{\theta}{\mu}, 0, 0, 0, 0\right). \tag{3.1}$$

3.2. Effective reproduction ratio, $\mathcal{R}_e$

The effective reproductive ratio ($\mathcal{R}_e$) measures the average number of secondary infection which an infected individual is able to produce throughout his lifetime in an entirely susceptible population where intervention strategies against the infection are on ground [48]. The $\mathcal{R}_e$ is essential in epidemiological study to check whether the disease clears out or persists within the population. The effective reproductive ratio ($\mathcal{R}_e$) has the characteristics that when it is less than one (i.e., $\mathcal{R}_e < 1$), the population of infected individuals diminishes and the disease tends to eradication but when it is greater than one (i.e., $\mathcal{R}_e > 1$), the population of infected individuals rises and the disease escalates in the population. To derive $\mathcal{R}_e$, the next generational matrix (NGM) approach is usually used [49]. The dominant eigen value of the NGM represents the $\mathcal{R}_e$ [50]. Given the system (2.5), $\mathcal{R}_e$ is calculated from the infectious

compartments thus:

$$\begin{aligned}\mathcal{F} &= \begin{pmatrix} \beta(I+(1-\phi_1)I+(1-\phi_2)I_2+(1-\phi_3)I_3)S \\ 0 \\ 0 \\ 0 \end{pmatrix}, \\ \mathcal{V} &= \begin{pmatrix} (\mu+\alpha+\vartheta_1)I \\ (\mu+\vartheta_2)I_1-(1-\tau_1-\tau_2)\alpha I \\ (\mu+\vartheta_3)I_2-\tau_1\alpha I \\ (\mu+\vartheta_4)I_3-\tau_2\alpha I \end{pmatrix}. \end{aligned} \tag{3.2}$$

We determine F and V at DFE so that

$$\begin{aligned} F &= \begin{pmatrix} \beta S^\circ & \beta(1-\phi_1)S^\circ & \beta(1-\phi_2)S^\circ & \beta(1-\phi_3)S^\circ \\ 0 & 0 & 0 & \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix}, \\ V &= \begin{pmatrix} (\mu+\alpha+\vartheta_1) & 0 & 0 & 0 \\ -(1-\tau_1-\tau_2)\alpha & (\mu+\vartheta_2) & 0 & 0 \\ -\tau_1\alpha & 0 & (\mu+\vartheta_3) & 0 \\ -\tau_2\alpha & 0 & 0 & (\mu+\vartheta_4) \end{pmatrix}. \end{aligned} \tag{3.3}$$

The inverse of V gives

$$V^{-1}\begin{pmatrix} \dfrac{1}{(\mu+\alpha+\vartheta_1)} & 0 & 0 & 0 \\ \dfrac{(1-\tau_1-\tau_2)\alpha}{(\mu+\alpha+\vartheta_1)(\mu+\vartheta_2)} & \dfrac{1}{(\mu+\vartheta_2)} & 0 & 0 \\ \dfrac{\tau_1\alpha}{(\mu+\alpha+\vartheta_1)(\mu+\vartheta_3)} & 0 & \dfrac{1}{(\mu+\vartheta_3)} & 0 \\ \dfrac{\tau_2\alpha}{(\mu+\alpha+\vartheta_1)(\mu+\vartheta_4)} & 0 & 0 & \dfrac{1}{(\mu+\vartheta_4)} \end{pmatrix}.$$

$\mathcal{R}_e$ is the spectral radius of FV^{-1} and is computed as

$$\begin{aligned} \mathcal{R}_e = {} & \frac{\beta S^\circ}{(\mu+\alpha+\vartheta_1)} + \frac{\beta(1-\phi_1)S^\circ(1-\tau_1-\tau_2)\alpha}{(\mu+\alpha+\vartheta_1)(\mu+\vartheta_2)} + \\ & \frac{\beta(1-\phi_2)S^\circ\tau_1\alpha}{(\mu+\alpha+\vartheta_1)(\mu+\vartheta_3)} + \frac{\beta(1-\phi_3)S^\circ\tau_2\alpha}{(\mu+\alpha+\vartheta_1)(\mu+\vartheta_4)}. \end{aligned} \tag{3.4}$$

$\mathcal{R}_e$ is the threshold parameter for HIV minimization or escalation (under some suitable conditions, such as the absence of backward bifurcation [51]). Generally, if $\mathcal{R}_e > 1$, then a single HIV positive individual can spread the virus to more than

one individual, thus escalating the virus in the population. On the other hand, if $\mathcal{R}_e < 1$, then an infected individual does not spread the virus to a single person; hence the virus is minimized in the population. In (3.4), the significance of HAART is revealed in terms of the parameters ϕ_1, ϕ_2 and ϕ_3. If these parameters are absent (i.e., $\phi_1 = \phi_2 = \phi_3 = 0$,) then $\mathcal{R}_e > 1$ is more feasible. But, if $\phi_1 \to 1, \phi_2 \to 1$ and $\phi_3 \to 1$, then $\mathcal{R}_e < 1$ is likely to be achieved because HIV transmission from I_1, I_2 and I_3 becomes zero under the condition. ϕ_1 is likely to tend to one if HIV positive individuals who are aware of their status and who are accessing HAART and complying with HAART guidelines do not relax. ϕ_2 is also likely to tend to one if HIV positive individuals who are aware of their status and accessing HAART but who are not complying with HAART guidelines turn over a new leaf. Lastly, ϕ_3 is likely to tend to one if HAART becomes accessible to all HIV positive individuals who are aware of their status irrespective of their locations or economic status.

3.3. Disease-endemic equilibrium (DEE)

Disease-endemic equilibrium occurs when the virus invades the population. Under this condition $S^*, I^*, I_1^*, I_2^*, I_3^* > 0$. Reducing the RHS of (2.5) to zero then

$$\mathcal{E}^* = (S^*, I^*, I_1^*, I_2^*, I_3^*), \tag{3.5}$$

where in terms of I^*,

$$\begin{aligned}
S^* &= \frac{m_1\Gamma_1}{\beta\{m_1I^* + m_2[b_2\theta + \Gamma_2\alpha I^*] + m_3[b_3\theta + \tau_1\alpha I^*] + m_4[b_4\theta + \tau_2\alpha I^*]\} + \mu m_1},\\
I_1^* &= \frac{b_2\theta + \Gamma_2\alpha I^*}{\mu + \vartheta_2},\\
I_2^* &= \frac{b_3\theta + \tau_1\alpha I^*}{\mu + \vartheta_3},\\
I_3^* &= \frac{b_4\theta + \tau_2\alpha I^*}{\mu + \vartheta_4}
\end{aligned} \tag{3.6}$$

and

$$c_0 I^{*2} + c_1 I^* + c_2 = 0, \tag{3.7}$$

with
$c_0 = -\beta m_1(\mu + \alpha + \vartheta_1)[m_1 + m_2\Gamma_2\alpha + m_3\tau_1\alpha + m_4\tau_2\alpha]$,
$c_1 = b_1\theta m_1\beta(m_1 + m_2\Gamma_2\alpha + m_3\tau_1\alpha + m_4\tau_2\alpha) + m_1\beta\Gamma_1(m_1 + m_2\Gamma_2\alpha + m_3\tau_1\alpha + m_4\tau_2\alpha)$
$- m_1\beta\theta(\mu + \alpha + \vartheta_1)[m_2b_2 + m_3b_3 + m_4b_4] - m_1^2(\mu + \alpha + \vartheta_1)\mu$,
$c_2 = m_1b_1\theta^2\beta(m_2b_2 + m_3b_3 + m_4b_4) + m_1\beta\theta\Gamma_1(m_2b_2 + m_3b_3 + m_4b_4) + m_1^2b_1\theta\mu$,
$m_1 = (\mu + \vartheta_2)(\mu + \vartheta_3)(\mu + \vartheta_4), m_2 = (1 - \phi_1)(\mu + \vartheta_3)(\mu + \vartheta_4)$,
$m_3 = (1 - \phi_2)(\mu + \vartheta_2)(\mu + \vartheta_4), m_4 = (1 - \phi_3)(\mu + \vartheta_2)(\mu + \vartheta_3)$
$\Gamma_1 = (1 - b_1 - b_2 - b_3 - b_4), \Gamma_2 = (1 - \tau_1 - \tau_2)$.
Since $c_0 < 0$ and $c_2 > 0$, then the product of the roots $c_0c_2 < 0$. Therefore, one of the roots in (3.7) is necessarily be positive which stands for I^*. Hence, $S^* > 0$, $I^* > 0$, $I_1^* > 0$, $I_2^* > 0$ and $I_3^* > 0$ which establishes the existence and uniqueness of the disease-endemic equilibrium $\mathcal{E}^*$.

3.4. Local and global stability of DFE, $\mathcal{E}_\circ$

The stability of $\mathcal{E}_\circ$, both local and global, depends on $\mathcal{R}_e$. $\mathcal{E}_\circ$ is stable locally and globally if $\mathcal{R}_e < 1$ but it is unstable if $\mathcal{R}_e > 1$. To verify the existence of local stability for $\mathcal{E}_\circ$, we compute the Jacobian matrix of the system (2.5) around the DFE, $\mathcal{E}_\circ$ and the result is stated in (3.8)

$$J(\mathcal{E}_\circ) = \begin{pmatrix} -\mu & -\beta S^\circ & -\beta(1-\phi_1)S^\circ & -\beta(1-\phi_2)S^\circ & -\beta(1-\phi_3)S^\circ \\ 0 & \beta S^\circ - (\mu+\alpha+\vartheta_1) & \beta(1-\phi_1)S^\circ & \beta(1-\phi_2)S^\circ & \beta(1-\phi_3)S^\circ \\ 0 & (1-\tau_1-\tau_2)\alpha & -(\mu+\vartheta_2) & 0 & 0 \\ 0 & \tau_1\alpha & 0 & -(\mu+\vartheta_3) & 0 \\ 0 & \tau_2\alpha & 0 & 0 & -(\mu+\vartheta_4) \end{pmatrix}. \tag{3.8}$$

In (3.8), $\lambda_1 = -\mu$ and the remaining solutions of $J(\mathcal{E}_\circ)$ are contained in B given as

$$B = \begin{pmatrix} \beta S^\circ - (\mu+\alpha+\vartheta_1) & \beta(1-\phi_1)S^\circ & \beta(1-\phi_2)S^\circ & \beta(1-\phi_3)S^\circ \\ (1-\tau_1-\tau_2)\alpha & -(\mu+\vartheta_2) & 0 & 0 \\ \tau_1\alpha & 0 & -(\mu+\vartheta_3) & 0 \\ \tau_2\alpha & 0 & 0 & -(\mu+\vartheta_4) \end{pmatrix}. \tag{3.9}$$

Following Gershgorin's circle theorem [52], the following hold for matrix B

$$\begin{aligned} R_1 &: \ (\mu+\alpha+\vartheta_1) > \beta S^\circ + \beta(1-\phi_1)S^\circ + \beta(1-\phi_2)S^\circ + \beta(1-\phi_3)S^\circ, \\ R_2 &: \ (\mu+\vartheta_2) > (1-\tau_1-\tau_2)\alpha, \\ R_3 &: \ (\mu+\vartheta_3) > \tau_1\alpha, \\ R_4 &: \ (\mu+\vartheta_4) > \tau_2\alpha, \end{aligned}$$

which give

$$\begin{aligned} R_1 &: \ 1 > \frac{\beta S^\circ}{(\mu+\alpha+\vartheta_1)} + \frac{\beta(1-\phi_1)S^\circ}{(\mu+\alpha+\vartheta_1)} + \frac{\beta(1-\phi_2)S^\circ}{(\mu+\alpha+\vartheta_1)} + \frac{\beta(1-\phi_3)S^\circ}{(\mu+\alpha+\vartheta_1)}, \\ R_2 &: \ 1 > \frac{(1-\tau_1-\tau_2)\alpha}{(\mu+\vartheta_2)}, \\ R_3 &: \ 1 > \frac{\tau_1\alpha}{(\mu+\vartheta_3)}, \\ R_4 &: \ 1 > \frac{\tau_2\alpha}{(\mu+\vartheta_4)}. \end{aligned} \tag{3.10}$$

Combining R_1, R_2, R_3 and R_4 then

$$\begin{aligned} 1 > \ & \frac{\beta S^\circ}{(\mu+\alpha+\vartheta_1)} + \frac{\beta(1-\phi_1)S^\circ(1-\tau_1-\tau_2)\alpha}{(\mu+\alpha+\vartheta_1)(\mu+\vartheta_2)} \\ & + \frac{\beta(1-\phi_2)S^\circ\tau_1\alpha}{(\mu+\alpha+\vartheta_1)(\mu+\vartheta_3)} + \frac{\beta(1-\phi_3)S^\circ\tau_2\alpha}{(\mu+\alpha+\vartheta_1)(\mu+\vartheta_4)}. \end{aligned} \tag{3.11}$$

In view of (3.4), (3.11) becomes $1 > \mathcal{R}_e$.

Hence, $\mathcal{R}_e < 1$ and the DFE is locally asymptotically stable for $\mathcal{R}_e < 1$. To establish the global stability of DFE that guarantees total minimization of HIV regardless of the initial sizes of the susceptible and infectious populations, we construct a Lyapunov function $\mathcal{U}(t)$ as in [53] as follows

$$\mathcal{U}(t) = A_1 I + A_2 I_1 + A_3 I_2 + A_4 I_3,$$

with time derivative

$$\dot{\mathcal{U}}(t) = A_1 \dot{I} + A_2 \dot{I}_1 + A_3 \dot{I}_2 + A_4 \dot{I}_3, \tag{3.12}$$

where $A_1, \ldots, A_4$ are nonnegative constants whose values do not alter the positivity or negativity of $\dot{\mathcal{U}}(t)$. We substitute for $\dot{I}, \dot{I}_1, \dot{I}_2$ and $\dot{I}_3$ first in (3.12) and after a few algebraic manipulations with $I_1 = I_2 + I_3 = A_1 = A_2 = A_3 = A_4 = 1$ and neglecting some terms, we obtain

$$\begin{aligned}\dot{\mathcal{U}}(t) \leq \Bigg[& \beta S^\circ + (1-\tau_1-\tau_2)\alpha \frac{\beta(1-\phi_1)S^\circ}{(\mu+\vartheta_2)} \\ & + \tau_1\alpha \frac{\beta(1-\phi_2)S^\circ}{(\mu+\vartheta_3)} + \tau_2\alpha \frac{\beta(1-\phi_3)S^\circ}{(\mu+\vartheta_4)} - (\mu+\alpha+\vartheta_1) \Bigg] I\end{aligned}$$

$\Longrightarrow$

$$\begin{aligned}\dot{\mathcal{U}}(t) \leq & (\mu+\alpha+\vartheta_1)\left[\frac{\beta S^\circ}{(\mu+\alpha+\vartheta_1)} + \frac{\beta(1-\phi_1)S^\circ(1-\tau_1-\tau_2)\alpha}{(\mu+\alpha+\vartheta_1)(\mu+\vartheta_2)} \right. \\ & + \frac{\beta(1-\phi_2)S^\circ\tau_1\alpha}{(\mu+\alpha+\vartheta_1)(\mu+\vartheta_3)} \qquad \left. + \frac{\beta(1-\phi_3)S^\circ\tau_2\alpha}{(\mu+\alpha+\vartheta_1)(\mu+\vartheta_4)} - 1 \right] I.\end{aligned}$$

Hence,

$$\dot{\mathcal{U}}(t) \leq (\mu+\alpha+\vartheta_1)[\mathcal{R}_e - 1]I, \tag{3.13}$$

where I signifies the existence of HIV in the population. The inequality (3.13) indicates that $\dot{\mathcal{U}}(t) < 0$ if $\mathcal{R}_e < 1$. Again $\dot{\mathcal{U}}(t) = 0$ at DFE $\{\mathcal{E}_\circ\}$ where $I = 0$. Consequently, $(I, I_1, I_2, I_3) \to (0,0,0,0)$ as $t \to \infty$ and evaluating the model (2.5) at $I = I_1 = I_2 = I_3 = 0$ gives $S \to S^\circ$ as $t \to \infty$ for $\mathcal{R}_e < 1$, and hence following [54, Theorem 2.3.1], the DFE $\{\mathcal{E}_\circ\}$ is GAS if $\mathcal{R}_e < 1$.

3.5. Local and global stability of disease-endemic equilibrium, $\mathcal{E}^*$

As in DFE, $\mathcal{E}_\circ$, the stability of $\mathcal{E}^*$ depends on $\mathcal{R}_e$. However, unlike in $\mathcal{E}_\circ$, $\mathcal{E}^*$ is stable locally and globally if $\mathcal{R}_e > 1$ while it is unstable if $\mathcal{R}_e < 1$. To examine the local stability of $\mathcal{E}^*$, we linearize (2.5) about $\mathcal{E}^*$ and the result obtained is in (3.14)

$$J(\mathcal{E}^*) = \begin{pmatrix} -(\mu+d_1) & -d_2 & -d_3 & -d_4 & -d_5 \\ d_1 & d_2 - d_6 & d_3 & d_4 & d_5 \\ 0 & \Gamma_2 & -d_7 & 0 & 0 \\ 0 & \tau_1\alpha & 0 & -d_8 & 0 \\ 0 & \tau_2\alpha & 0 & 0 & -d_9 \end{pmatrix}, \tag{3.14}$$

where $d_1 = \beta(I^* + (1-\phi_1)I_1^* + (1-\phi_2)I_2^* + (1-\phi_3)I_3^*), d_2 = \beta S^*, d_3 = \beta(1-\phi_1)S^*, d_4 = \beta(1-\phi_2)S^*, d_5 = \beta(1-\phi_3)S^*, d_6 = (\mu+\alpha+\vartheta_1), \Gamma_2 = (1-\tau_1-\tau_2)\alpha, d_7 = (\mu+\vartheta_2), d_8 = (\mu+\vartheta_3), d_9 = (\mu+\vartheta_4)$.

The characteristic equation of (3.14), using software maple, is

$$p_0\lambda_*^5 + p_1\lambda_*^4 + p_2\lambda_*^3 + p_3\lambda_*^2 + p_4\lambda_* + p_5 = 0, \tag{3.15}$$

where
$p_0 = 1$,
$p_1 = [d_9 + d_8 + d_7 + d_6 + d_1 + \mu - d_2]$,
$p_2 = [d_1(d_6 + d_7 + d_8 + d_9) - d_2(d_7 + d_8 + d_9 + \mu) - d_3\Gamma_2 - \tau\alpha(d_4 + d_5) + d_6(d_7 + d_8 + d_9 + \mu) + (d_8 + d_9 + \mu) + d_8(d_9 + \mu) + d_9\mu]$,
$p_3 = [d_1d_6(d_7 + d_8 + d_9) + d_1d_7(d_8 + d_9) + d_1d_8d_9 - d_2d_7(d_8 + d_9 + \mu) - d_2d_8(d_9 + \mu) - d_2d_9\mu - \Gamma_2d_3(d_8 + d_9 + \mu) - d_4\tau\alpha(d_7 + d_9 + \mu) - d_5\tau\alpha(d_7 + d_8 + \mu) + d_6d_7(d_8 + d_9 + \mu) + d_6d_8(d_9 + \mu) + d_6d_9\mu + d_7d_8(d_9 + \mu) + d_7d_9\mu + d_8d_9\mu]$,
$p_4 = [-d_5d_7\tau\alpha(d_8 + \mu) - d_5d_8\tau\alpha\mu + d_1d_6d_9(d_7 + d_8) + d_1d_8d_9(d_7 + d_2) - d_2d_9\mu(d_7 + d_8) - d_3d_9\Gamma_2(d_8 + \mu) - d_4d_9\tau\alpha(d_7 + \mu) + d_7d_6d_9(d_8 + \mu) + d_8d_9\mu(d_6 + d_7) - d_4d_7\tau\alpha\mu + d_1d_6d_7d_8 - d_8\mu(d_2d_7 + d_3\Gamma_2 - d_6d_7)]$,
$p_5 = d_7d_9d_8(d_1d_6 - d_2\mu) + d_6d_7d_9d_8\mu - d_7\tau\alpha\mu(d_5d_7 + d_4d_9) - d_3d_9d_8\mu\Gamma_2$.

Following Routh-Hurwitz criteria [55], the roots of (3.15) are all negative and $\mathcal{E}^*$ is locally asymptotically stable if the following inequalities are true

$$p_1p_2p_3 > p_3^2 + p_1^2p_4, \quad (p_1p_4 - p_5)(p_1p_2p_3 - p_3^2 - p_1^2p_4) > p_5(p_1p_2 - p_3)^2 + p_1p_5^2.$$

For the global attractiveness of $\mathcal{E}^*$, we formulate a Lyapunov function $\mathcal{V}$ as in [56] as follows

$$\mathcal{V} = (x_1, \cdots, x_5) = \sum_{i-1}^{5} \frac{c_i}{2}(x_i - x_i^*)^2, \tag{3.16}$$

where $x_i = (S, I, I_1, I_2, I_3)$ and $x_i^* = \mathcal{E}^* = (S^*, I^*, I_1^*, I_2^*, I_3^*)$.

Choosing $c_i = 1$ then,

$$\mathcal{V}(S, I, I_1, I_2, I_3) = \frac{1}{2}[(S - S^*) + (I - I^*) + (I_1 - I_1^*) + (I_2 - I_2^*) + (I_3 - I_3^*)]^2. \tag{3.17}$$

Therefore,

$$\frac{dV}{dt} = [(S-S^*)+(I-I^*)+(I_1-I_1^*)+(I_2-I_2^*)+(I_3-I_3^*)]\frac{d}{dt}(S+I+I_1+I_2+I_3). \tag{3.18}$$

In (3.18),

$$\frac{d}{dt}(S + I + I_1 + I_2 + I_3) = \frac{d}{dt}N(t) \tag{3.19}$$

and from (2.8),

$$\frac{d}{dt}N(t) \leq \theta - \mu N. \tag{3.20}$$

Notice that

$$S + I + I_1 + I_2 + I_3 \leq \frac{\theta}{\mu} \tag{3.21}$$

implies that

$$S^* + I^* + I_1^* + I_2^* + I_3^* \leq \frac{\theta}{\mu}, \tag{3.22}$$

since the population before the outbreak is always greater than or equal to the population during or after the outbreak. Now, we take $\frac{d}{dt}N(t) = \theta - \mu N$ in (3.20) and $S^* + I^* + I_1^* + I_2^* + I_3^* = \frac{\theta}{\mu}$ in (3.22) and substitute the results in (3.18) to obtain

$$\frac{dV}{dt} = \left(N - \frac{\theta}{\mu}\right)(\theta - \mu N), \tag{3.23}$$

which simplifies to

$$\frac{dV}{dt} = -\frac{1}{\mu}(\theta - \mu N)^2. \tag{3.24}$$

In (3.24), $\frac{dV}{dt} < 0$ but zero if $S = S^*, I = I^*, I_1 = I_1^*, I_2 = I_2^*$ and $I_3 = I_3^*$. Further, all the solutions of (5) approaches $\mathcal{E}^*$ as $t \to \infty$ (see [57]); hence the largest compact invariant set in $\left\{S(t), I(t), I_1(t), I_2(t), I_3(t) \in \Omega : \frac{dV}{dt} < 0\right\}$ is the singleton set $\mathcal{E}^*$. Therefore, following LaSalle's invariant principle [58], the disease endemic equilibrium $\mathcal{E}^*$ is GAS in Ω whenever $\mathcal{R}_e > 1$.

3.6. Bifurcation analysis

It is claimed in subsection 3.2 that the effective reproduction ratio $\mathcal{R}_e$ is the threshold parameter for HIV minimization or escalation (under some suitable conditions, such as the absence of backward bifurcation ([51, 55]). If $\mathcal{R}_e < 1$ and backward bifurcation does not exist, then the strategies that are incorporated in the model are sufficient to put HIV transmission under perfect control in the population. On the other hand, if $\mathcal{R}_e < 1$ and backward bifurcation exists, then the strategies that are incorporated in the model do not subdue the HIV spread in the population. Hence, there is a need for additional efforts to bring the disease under control. For this reason, the analysis of bifurcation plays an important role in determining disease permanence or eradication when $\mathcal{R}_e < 1$ in mathematical epidemiology. If backward bifurcation does not exist when $\mathcal{R}_e < 1$, then policy makers can hold on to the strategies that achieve the condition to overcome the spread of HIV in the population. But, if $\mathcal{R}_e < 1$ and backward bifurcation exists, then policy makers had to put in more effort if the virus escalation is to be championed in the population. We shall investigate the existence of backward bifurcation for our model using the popular center manifold theory due to [59] employed in [51,55] to verify whether the controls incorporated in the model are sufficient to bring HIV transmission under control in the population or not. To verify the existence of backward bifurcation, we choose β as the bifurcation parameter and it changes to β^* at the bifurcation point $\mathcal{R}_e = 1$. Also, the model variables S, I, I_1, I_2, and I_3 are denoted by x_1, x_2, x_3, x_4

and x_5 respectively so that the system (2.5) is rewritten as

$$\begin{aligned}
\dot{x_1} =&(1-b_1-b_2-b_3-b_4)\theta-\beta^*(x_2+(1-\phi_1)x_3+(1-\phi_2)x_4\\
&+(1-\phi_3)x_5)x_1-\mu x_1,\\
\dot{x_2} =&b_1\theta+\beta^*(x_2+(1-\phi_1)x_3+(1-\phi_2)x_4+(1-\phi_3)x_5)x_1\\
&-(\mu+\alpha+\vartheta_1)x_2,\\
\dot{x_3} =&b_2\theta+(1-\tau_1-\tau_2)\alpha x_2-(\mu+\vartheta_2)x_3,\\
\dot{x_4} =&b_3\theta+\tau_1\alpha x_2-(\mu+\vartheta_3)x_4,\\
\dot{x_5} =&b_4\theta+\tau_2\alpha x_2-(\mu+\vartheta_4)x_5.
\end{aligned} \tag{3.25}$$

The Jacobian matrix of (3.25) at DFE $\mathcal{E}_\circ$ with $\beta=\beta^*$ is calculated in (3.26)

$$J(\mathcal{E}_0)|_{\beta=\beta^*} = \begin{pmatrix}
-\mu & -\beta^*S^\circ & -\beta^*(1-\phi_1)S^\circ & -\beta^*(1-\phi_2)S^\circ & -\beta^*(1-\phi_3)S^\circ\\
0 & \beta^*S^\circ-(\mu+\alpha+\vartheta_1) & \beta^*(1-\phi_1)S^\circ & \beta^*(1-\phi_2)S^\circ & \beta^*(1-\phi_3)S^\circ\\
0 & (1-\tau_1-\tau_2)\alpha & -(\mu+\vartheta_2) & 0 & 0\\
0 & \tau_1\alpha & 0 & -(\mu+\vartheta_3) & 0\\
0 & \tau_2\alpha & 0 & 0 & -(\mu+\vartheta_4)
\end{pmatrix}. \tag{3.26}$$

The right eigenvectors of $J(\mathcal{E}_0)|_{\beta=\beta^*}$ represented by $\mathbf{w}=(w_1,w_2,w_3,w_4,w_5)^T$ is then computed as

$$\begin{aligned}
w_1 =& -\frac{\beta^*S^\circ}{\mu}\left[\frac{(\mu+\vartheta_4)}{\tau_2\alpha}+\frac{(1-\phi_1)(1-\tau_1-\tau_2)(\mu+\vartheta_4)}{\tau_2(\mu+\vartheta_2)}\right.\\
&\left.+\frac{(1-\phi_2)\tau_1(\mu+\vartheta_4)}{\tau_2(\mu+\vartheta_3)}+(1-\phi_3)\right]w_5<0,
\end{aligned} \tag{3.27}$$

$$w_2=\frac{(\mu+\vartheta_4)}{\tau_2\alpha}w_5>0, \tag{3.28}$$

$$w_3=\frac{(1-\tau_1-\tau_2)(\mu+\vartheta_4)}{\tau_2(\mu+\vartheta_2)}w_5>0, \tag{3.29}$$

$$w_4=\frac{\tau_1(\mu+\vartheta_4)}{\tau_2(\mu+\vartheta_3)}w_5>0 \tag{3.30}$$

$$w_5=w_5>0. \tag{3.31}$$

Again, the left eigen vector $\mathbf{v}=(v_1,v_2,v_3,v_4,v_5)^T$ for $J(\mathcal{E}_0)|_{\beta=\beta^*}$ which meets the condition $\mathbf{v}.\mathbf{w}=\mathbf{1}$ is $v_1=v_3=v_4=v_5=0$ but $v_2=v_2>0$. The bifurcation coefficients, a and b with b always positive, are defined in [59, Theorem 4.1]. The definitions and computations of a and b for the present model are as follows

$$\begin{aligned}
a=&\sum_{k,i,j=1}^{5}v_kw_iw_j\frac{\partial^2f_k}{\partial x_i\partial x_j}(0,0),\\
=&2v_2w_1w_2\beta^*.
\end{aligned}$$

In view of (3.27) and (3.28),

$$a = -2v_2w_1w_2\beta^*. \tag{3.32}$$

Also,

$$\begin{aligned} b &= \sum_{k,i=1}^{5} v_k w_i \frac{\partial^2 f_k}{\partial x_i \partial \beta^*}(0,0), \\ &= v_2w_2[1 + (1-\phi_1) + (1-\phi_2) + (1-\phi_2)]S^\circ > 0. \end{aligned} \tag{3.33}$$

Since $a < 0$ and based on Theorem 4.1 in [59], the model does not undergo backward bifurcation. The implication is that HIV spread could be greatly reduced if the proposed interventions are observed with utmost seriousness. Policy makers in HIV "hot regions" particularly in sub-Saharan Africa can overcome the problem of the virus if appropriate measures are put in place to ensure that: (i) HIV positive individuals who are aware of their status and who are accessing HAART and complying with HAART guidelines do not relax; (ii) HIV positive individuals who are aware of their status and accessing HAART but who are not complying with HAART guidelines turn over a new leaf; and (iii) HAART becomes accessible to every HIV positive individuals who are aware of their status irrespective of their locations or economic status.

3.7. Sensitivity analysis

A good number of factors that have been developed theoretically in (3.4) affect HIV transmission in the population. These factors also govern the occurence of HIV escalation $\mathcal{R}_e > 1$ and HIV minimization $\mathcal{R}_e < 1$. The relative impacts of these factors to HIV spread and minimization are computed theoretically in (3.34) employing the approach in [53, 55].

$$\begin{aligned} \beta &: \frac{\beta S^\circ}{(\mu+\alpha+\vartheta_1)\mathcal{R}_e}\left\{1 + \frac{(1-\phi_1)(1-\tau_1-\tau_2)\alpha}{(\mu+\vartheta_2)} + \frac{(1-\phi_2)\tau_1\alpha}{(\mu+\vartheta_3)}\right. \\ &\left. + \frac{(1-\phi_3)\tau_2\alpha}{(\mu+\alpha+\vartheta_1)(\mu+\vartheta_4)}\right\}, \\ \phi_1 &: -\frac{\beta S^\circ(1-\tau_1-\tau_2)\alpha}{(\mu+\alpha+\vartheta_1)(\mu+\vartheta_2)} \times \frac{\phi_1}{\mathcal{R}_e}, \\ \phi_2 &: -\frac{\beta S^\circ\tau_1\alpha}{(\mu+\alpha+\vartheta_1)(\mu+\vartheta_3)} \times \frac{\phi_2}{\mathcal{R}_e}, \\ \phi_3 &: -\frac{\beta S^\circ\tau_2\alpha}{(\mu+\alpha+\vartheta_1)(\mu+\vartheta_4)} \times \frac{\phi_3}{\mathcal{R}_e}. \end{aligned} \tag{3.34}$$

The results in (3.34) provide a simple guide to the policy makers about the ways to follow to minimize HIV transmission in the society. It is shown that parameters β could increase the spread of the virus because it is positively related to $\mathcal{R}_e$. There is a need to focus the parameter with the implementation of appropriate policies. On the other hand, parameters ϕ_1, ϕ_2 and ϕ_3 could reduce HIV spread because they are negatively related to $\mathcal{R}_e$. Therefore, the three interventions should be prirotized in HIV control.

4. Simulation and discussion

Numerical simulations are performed by employing parameter values in Table 3. The values for the parameters are either assumed or estimated or taken from the related literature. We focus on a low income society and take estimates for some of our parameters from the published works that are related to sub-Saharan Africa (SSA) where the virus is most ravaging. For instance, the estimation for μ is outlined in [60]. The life expectancy for SSA in 2019 was 61.63 years so $\mu = \frac{1}{61.63} \approx 0.016$ [60]. Also, based on the estimate in [61], people living with AIDS have a life expectancy of 3 years so $\delta = \frac{1}{3} \approx 0.3333$. The units for the parameters are measured per year.

Table 3. Parameter values employed for simulations

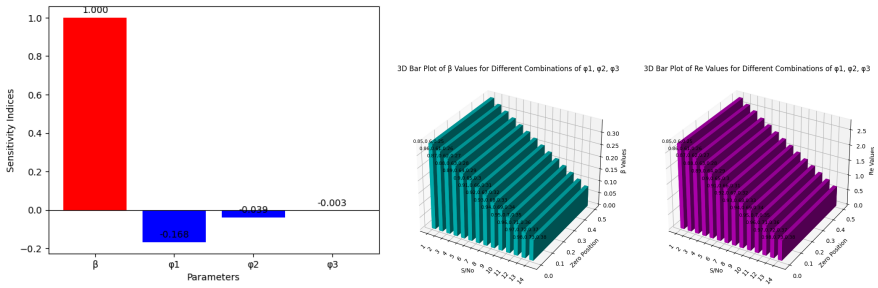
Parameters	Values	Source
b_1, b_2, b_3, b_4	0.35, 0.25, 0.17, 0.1	Assumed
μ	0.016	[60]
β	0.3425	[62]
ϕ_1, ϕ_2, ϕ_3	0.85, 0.6, 0.25	Assumed
τ_1, τ_2	0.35, 0.1	Assumed
α	0.01	Assumed
$\vartheta_1, \vartheta_2, \vartheta_3, \vartheta_4$	0.095, 0.01, 0.035, 0.065	Assumed
θ	0.015	Assumed
δ	0.3333	[61]

Given Table 3, the indices of sensitivity derived analytically in (3.34) are computed in Table 4.

Table 4. Indices of sensitivity for key parameters in relation to $\mathcal{R}_e$

Parameters	Signs	Sensitivity indices
β	+	1
ϕ_1	-	0.168
ϕ_2	-	0.039
ϕ_3	-	0.003

In Table 4, the effective contact with the HIV positive individuals (β) has a direct relationship with the transmission parameter $\mathcal{R}_e$ to the extent that a 10% change in (β) activates a corresponding 10% change in $\mathcal{R}_e$. It is therefore evident that every effort to prevent HIV transfer from HIV positive individuals to the susceptibles should be promoted. Some of these efforts are in terms of ϕ_1, ϕ_2 and ϕ_3 whose indices of sensitivity are stated in Table 4. A 100% change in ϕ_1, ϕ_2 and ϕ_3 could change $\mathcal{R}_e$ by about 20%, 4% and 1% respectively. It is evident, based on the indices of sensitivity for ϕ_1, ϕ_2 and ϕ_3, that awareness of disease status could limit HIV spread and reduce the burden of the disease in sub-Saharan Africa. Better still, these efforts would be more effective as the magnitude of ϕ_1, ϕ_2 and ϕ_3 tend to increase if there is total accessibility of HAART to everyone living with HIV and



(a) (b)

Figure 2. (a) Sensitivity indices of contact rate β and the reduction factors ϕ_1, ϕ_2 and ϕ_3. (b) Changes in the contact rate β and reproduction ratio $\mathcal{R}_e$ due to changes in the reduction factors ϕ_1, ϕ_2 and ϕ_3.

at the same time, total compliance with HAART procedures by the HAART users.

With Table 3, another important epidemiological parameter, the herd immunity, can also be evaluated to determine the coverage threshold of ϕ_1, ϕ_2 and ϕ_3 that is necessary to stem the spread of HIV in the society. Herd immunity, in disease modeling, is the fraction of the population that must be immuned to an infection through vaccination to curb the spread of the infection [63]. The herd immunity, following [63], is computed using the formula

$$H_1 = 1 - \frac{1}{\mathcal{R}_e}. \tag{4.1}$$

For the model, $\mathcal{R}_e$ is evaluated as 2.76 (approx.). Hence, the herd immunity H_1 for the system is 64% (approx.). The result obtained for H_1 shows that HAART accessibility to all that are aware of their HIV positive status and adherence to HAART procedures by these people must exceed 64% to overcome HIV transmission in the society. In the model, HAART accessibility and the level of adherence to the HAART procedures are quantified in terms of ϕ_1, ϕ_2 and ϕ_3. The herd immunity is achieved in the population when the quantities ϕ_1, ϕ_2 and ϕ_3 are combined so that $\mathcal{R}_e < 1$. We therefore investigate the combinations of ϕ_1, ϕ_2 and ϕ_3 that reduces the transmission parameter (β) to the points where $\mathcal{R}_e$ is below unity using the values in Table 3 as the base. The result of the analysis is in Table 5.

Table 5. Combinations of (ϕ_1, ϕ_2, ϕ_3) and the corresponding effects on β and $\mathcal{R}_e$

S/No.	Changes in ϕ_1	Changes in ϕ_2	Changes in ϕ_3	Effect on β	Effect on $\mathcal{R}_e$
1.	0.85	0.6	0.25	0.3425	2.76
2.	0.86	0.61	0.26	0.3225	2.60
3.	0.87	0.62	0.27	0.3025	2.45
4.	0.88	0.63	0.28	0.2825	2.27
5.	0.89	0.64	0.29	0.2625	2.11
6.	0.90	0.65	0.30	0.2425	1.94
7.	0.91	0.66	0.31	0.2225	1.78
8.	0.92	0.67	0.32	0.2025	1.62
9.	0.93	0.68	0.33	0.1825	1.46
10.	0.94	0.69	0.34	0.1625	1.30
11.	0.95	0.70	0.35	0.1425	1.14
12.	0.96	0.71	0.36	0.1225	0.98
13.	0.97	0.72	0.37	0.1025	0.82
14.	0.98	0.73	0.38	0.0825	0.66

In Table 5, the herd immunity is attained in S/No. 12 where ϕ_1 is almost 100%, ϕ_2 exceeds 70% and ϕ_3 is about 40%. This shows that to overcome the burden of HIV in endemic regions, the accessibility and the adherence level of HAART must be very high for individuals who have the knowledge of their HIV positive status. Now, with parameter values in Table 3 and given that $S(0) = 24,365, I(0) = 500, I_1(0) = 90, I_2(0) = 35$ and $I_3(0) = 10$, graphs are plotted for the model in Figures 3, 4, 5, 6 and 7 to visualize the effects of HAART accessibility and compliance with HAART procedures on the dynamics of HIV in the society. The initial conditions used to plot the graphs are proportional to the total population $N(0)$ as in Ayele et al. [27]. Figure 3 shows the effect of changes in the transmission rate on the dynamics of HIV. Figure 4 indicates the effect of changes in the rate of awareness on the dynamics of the disease. Figure 5 shows the effects of changes in the proportion of aware infectives who have no access to HAART on the dynamics of HIV. Figure 6 reveals the effect of low accessibility and low compliance with HAART procedure on the dynamics of HIV while Figure 7 shows what happens when the accessibility and the compliance rates with the HAART procedure are optimum.

As indicated in Table 4, the importance of transmission parameter β is further revealed in Figure 3. A continuous increase in transmission rate β produces a tremendous decline in the population of susceptible individuals (Figure 3(a)) which in turns instigates a rise in the population of the infectives (Figures 3(b),(c),(d),(e)). As shown in Figure 3(b), the population of HIV unaware infectives tended to be most affected where HIV transmission is not adequately checked.

As the awareness of the dangers and modes of transmission of HIV spreads and increases, the population of the unaware HIV infectives drops as indicated in Figure 4(b). It is evident from Figure 4(b) that if awareness of HIV is maintained at a high level for a long period of time, then the population of unaware HIV infectives could be drastically reduced. The increase in the population of the susceptible individuals in Figure 4(a) is not related to the awareness parameter but to influx into the population. This influx brings about an increase in the population of aware infectives who adhere to HAART guidelines and those who do not adhere to it as well as those who do not have access to HAART as indicated in Figures 4(c),(d),(e) respectively.

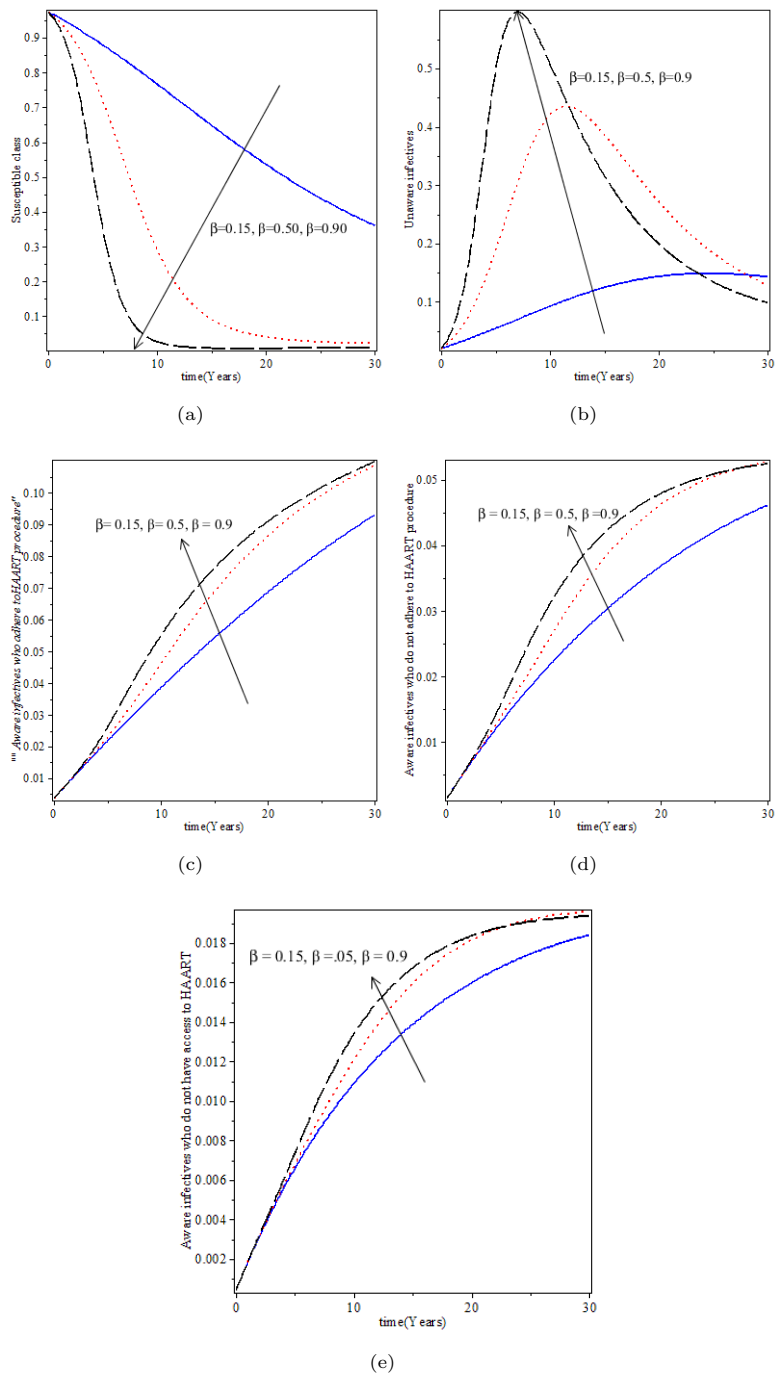


Figure 3. (a) Effect of increase in β on the population of the susceptibles. (b) Effect of increase in β on the population of unaware infective. (c) Effect of increase in β on the population of aware infective who do not default HAART. (d) Effect of increase in β on the population of aware infective who do not have access to HAART. (e) Effect of increase in β on the population of aware infective who do not have access to HAART.

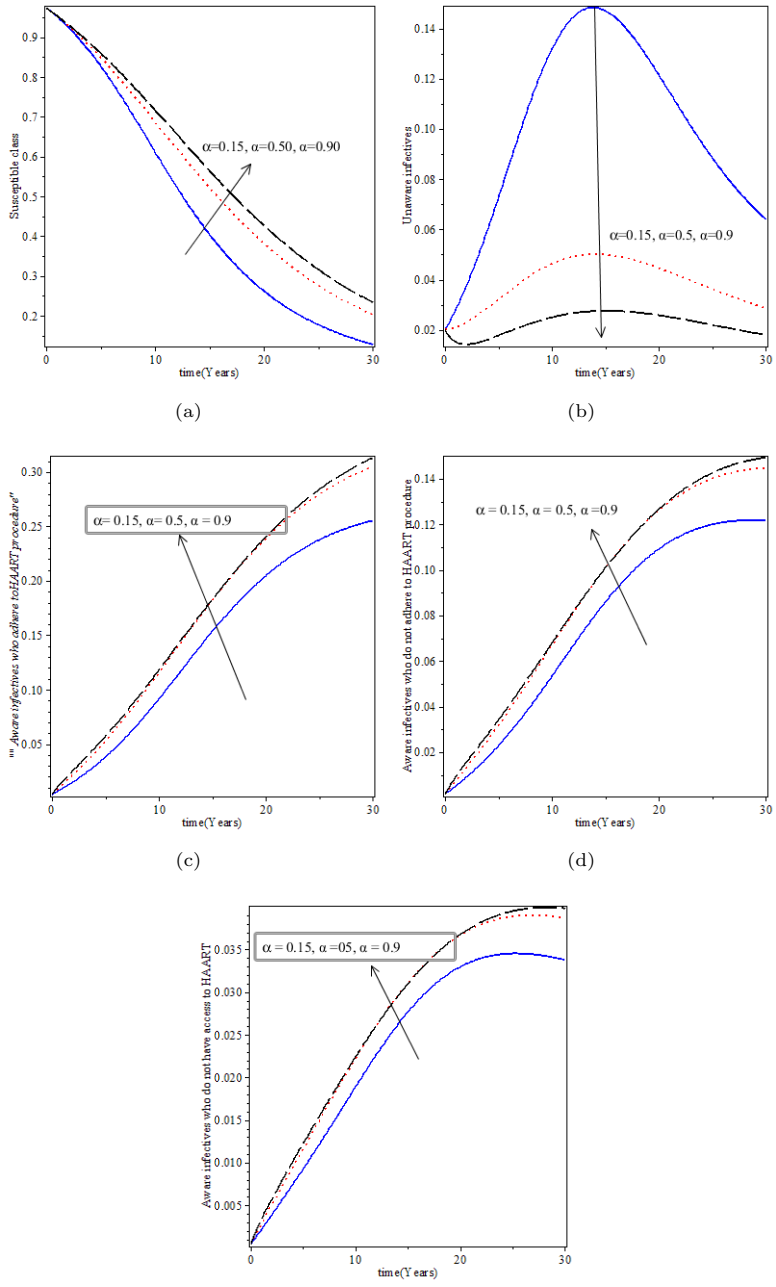


Figure 4. (a) Effect of increase in α on the population of the susceptibles. (b) Effect of increase in α on the population of unaware infective (c) Effect of increase in α on the population of aware infective who do not default HAART. (d) Effect of increase in α on the population of aware infective who do default HAART. (e) Simulation showing the effects of changes in the rate of awareness on the dynamics of HIV.

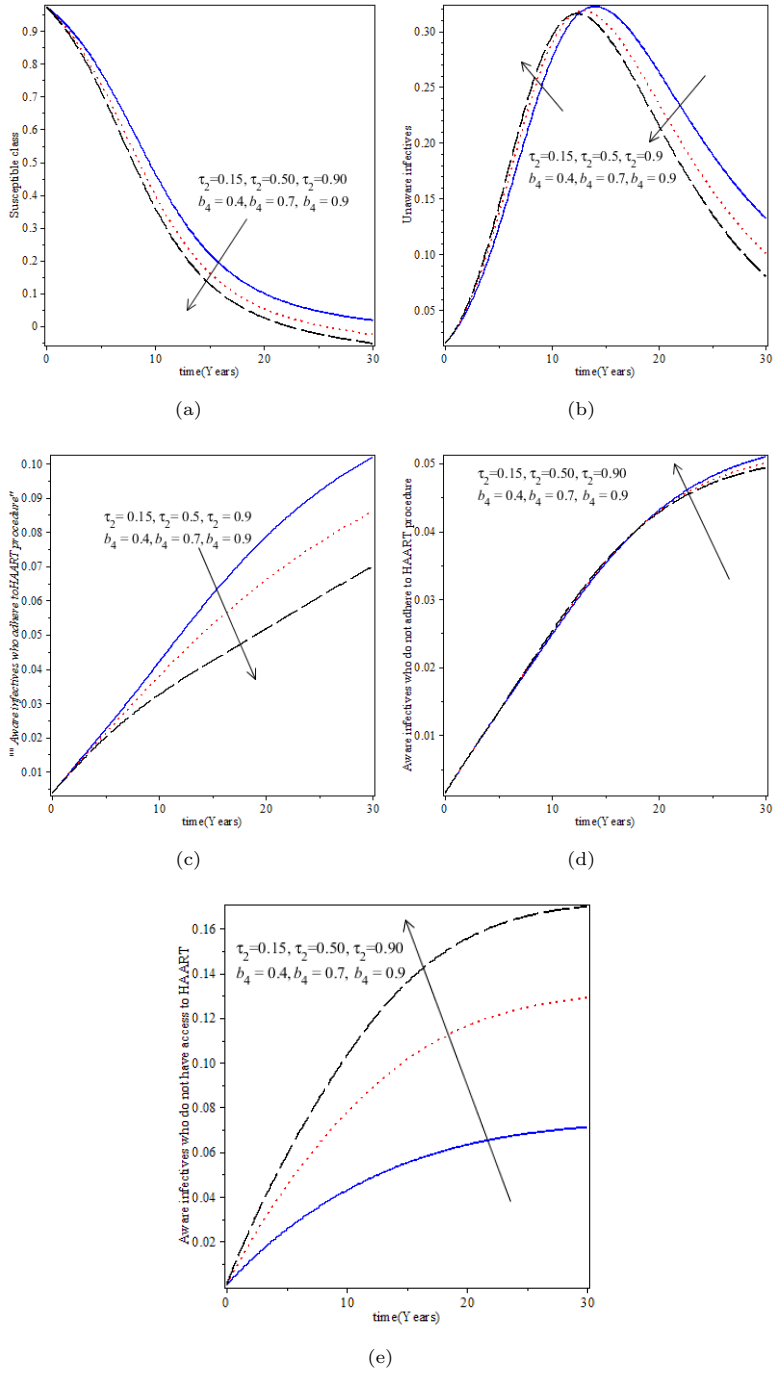


Figure 5. (a) Effect of increase in τ_2 on the population of the susceptibles. (b) Effect of increase in τ_2 on the population of unaware infective. (c) Effect of increase in τ_2 on the population of aware infective who do not default HAART. (d) Effect of increase in τ_2 on the population of aware infective who do default HAART. (e) Effect of increase in τ_2 on the population of aware infective who do not have access to HAART.

If a group of aware HIV infectives are at the disadvantage in accessing HAART either as a result of their location, social or economic status, then there is a tendency for more and more of those individuals to be recruited particularly through immigration (b_4) so that the proportion of the individuals τ_2 rises in the population. An increase in the proportion of aware infectives who have no access to HAART τ_2 has major effects on the populations of the susceptibles and unaware infectives as indicated in Figures 5(a),(b) respectively. In Figure 5(a), the population of susceptible individuals falls continuously. The fall insigates the rising part of Figure 5(b) which indicates an increase in the population of unaware infectives. When the unaware infectives become aware of their HIV positive status, the curve in Figure 5(b) begins to fall.

The population of the susceptible individuals tends to fall quickly and move to zero when many HIV positive individuals do not have access to HAART. The situation is compounded while only a few of them who have access to HAART comply with the guidelines of the medicine. In Figure 6(a), the population of the susceptibles falls and moves to zero after ten years due to poor accessibility of HAART and poor compliance with HAART procedure. This falls instigates an increase in all the populations of the infectives in Figures 6(b),(c),(d),(e).

When the HAART accessibility and the compliance rates with the HAART procedure are optimum, the population of susceptible individuals does not deplete but rather expands and there is limitation to HIV propagation. This is illustrated in Figure 7. With high rate of accessibility of HAART and high adherence rate to HAART procedure, the susceptability curves shift upward (Figure 7(a)). An upward shift of the curves in Figure 7(a) implies that few people contract HIV. Although there is an increase in the populations of all infectives as shown in Figures 7(b),(c),(d),(e), the increases are at a decreasing rate because the curves shift downward. Looking at the figures, Figure 3 through to Figure 7, one can deduce that herd immunity is attained in Figure 6 because it is the only figure where all the curves for the infective classes shift downward.

5. Conclusion

This work proposes and analyzes a mathematical model to examine how HIV transmission is impacted by accessibility of HAART and adherence to recommended practices. The set of solutions for which the model is both biologically and technically meaningful is determined using differential equation theory. Besides, there was also proof of the boundedness of the model solutions. Two potential solutions, the disease endemic equilibrium $\mathcal{E}^*$, and the disease-free equilibrium $\mathcal{E}_\circ$, were identified via model analysis. Additionally, we derived a threshold parameter $\mathcal{R}_e$, which is the model's effective reproductive ratio. It is shown that the DFE is asymptotically stable both locally and globally when $\mathcal{R}_e < 1$, and that the endemic equilibrium occurs and is stable both locally and globally when $\mathcal{R}_e > 1$. The derivation of the threshold parameter $\mathcal{R}_e$ has some health ramifications since it suggests that limiting the parameter below unity could be essential to stop the HIV epidemic from spreading provided that the model does not undergo backward bifurcation. Using the center manifold theory, we carried out bifurcation analysis and discovered that the model does not undergo backward bifurcation.

To confirm the analytical findings, we have also run numerical simulations. It has been demonstrated that a decrease in the population of the susceptibles results

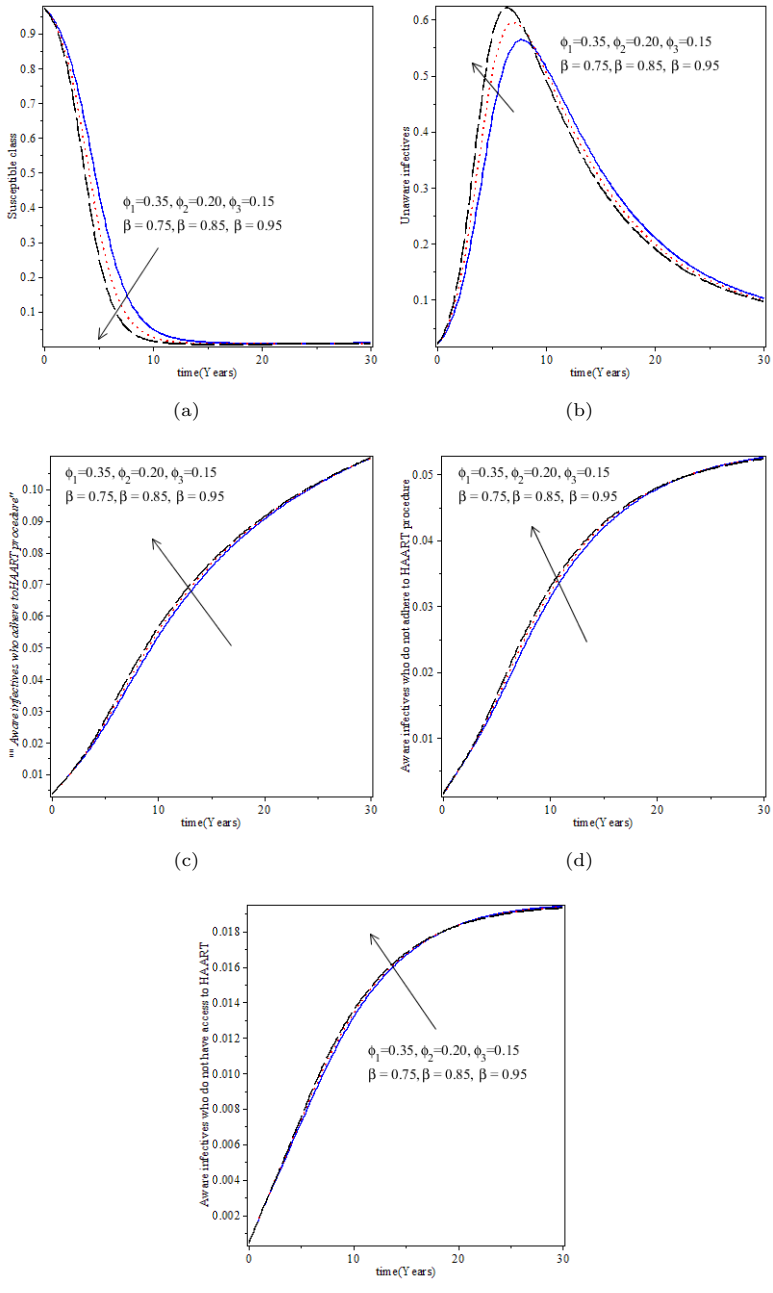


Figure 6. (a) Effect of low ϕ_1, ϕ_2 and ϕ_3 on the population of the susceptibles. (b) Effect of low ϕ_1, ϕ_2 and ϕ_3 on the population of unaware infective. (c) Effect of low ϕ_1, ϕ_2 and ϕ_3 on the population of aware infective who do not default HAART. (d) Effect of low ϕ_1, ϕ_2 and ϕ_3 on the population of aware infective who do default HAART. (e) Effect of low ϕ_1, ϕ_2 and ϕ_3 on the population of aware infective who do not have access to HAART.

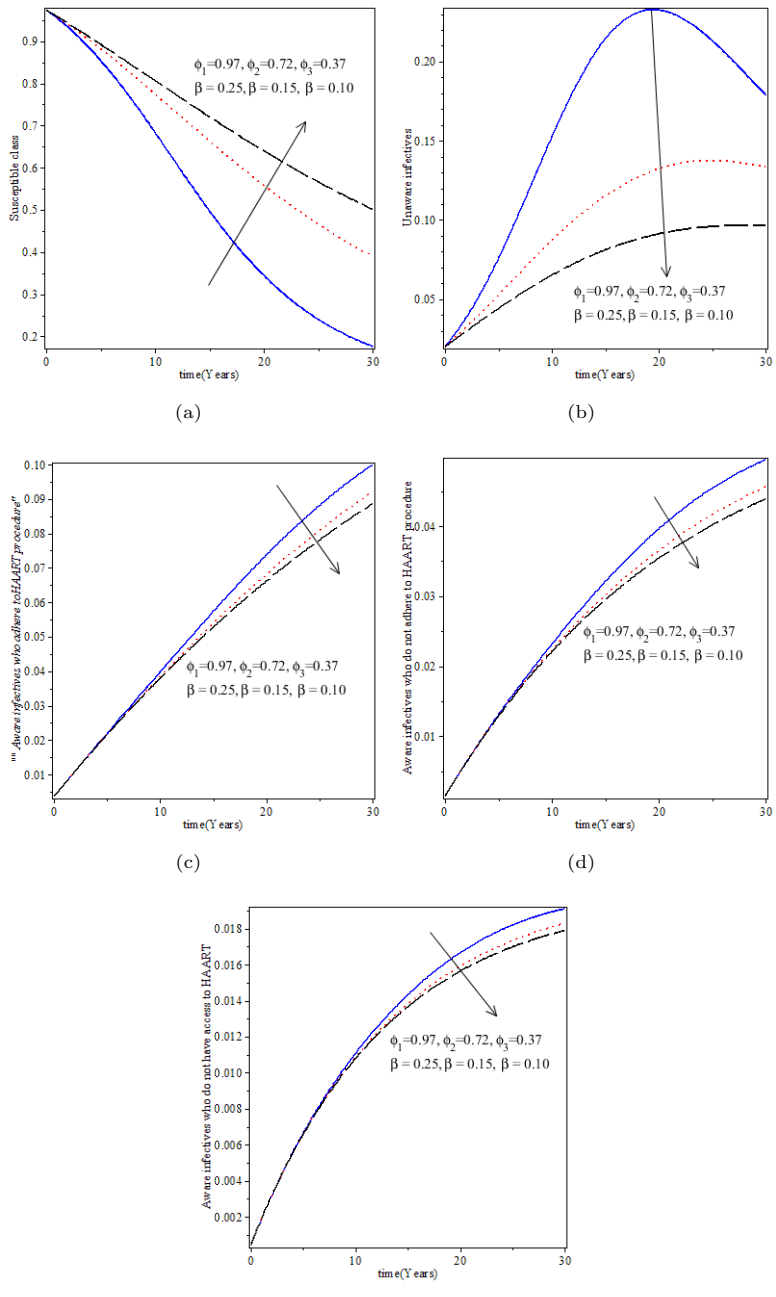


Figure 7. (a) Effect of high ϕ_1, ϕ_2 and ϕ_3 on the population of the susceptibles. (b) Effect of high ϕ_1, ϕ_2 and ϕ_3 on the population of unaware infective. (c) Effect of high ϕ_1, ϕ_2 and ϕ_3 on the population of aware infective who do not default HAART. (d) Effect of high ϕ_1, ϕ_2 and ϕ_3 on the population of aware infective who do default HAART. (e) Effect of high ϕ_1, ϕ_2 and ϕ_3 on the population of aware infective who do not have access to HAART.

from an increase in the effective contact rate between HIV infectives and susceptibles, and vice versa. The effective contact rate could also be aggravated and the population of the susceptibles be impacted significantly if the proportion of the HIV infectives who do not have access to HAART increases and if there is poor adherence to recommended practices of HAART. We have also demonstrated that when people are sufficiently aware of the risks and HIV transmission pathways, the disease burden on society declines as the number of people who are susceptible increases. This, however, is only possible when the effective rate of HIV infection among susceptible people is sufficiently lowered. Therefore, based on our model analysis, both the numerical and theoretical results attribute the persistence of the rise in HIV prevalence in the population to HIV-positive individuals that are not able to access HAART and to HIV-positive individuals that are able to access HAART but do not adhere to the recommended practices of HAART. The sensitivity and herd immunity assessments shown in Table 4 and Table 5 are supported by the graphs that were produced from our numerical simulations. Therefore, access to HAART should be increased in order to slow HIV spread in endemic areas, especially in sub-Saharan Africa. Also, awareness programs on the dangers and modes of spread of HIV should be prioritized in addition to improved testing of the new arrivals into the society to prevent upsurge in the population of unaware HIV infectives. Furthermore, while it is one thing to have access to HAART, it is another thing to adhere to the recommended practices of the medicine. Therefore, individuals who do not adhere to HAART procedure may be made to face punishment under the law to promote strict adherence to the recommended practices of HAART.

Lastly, since its appearance in 1981, HIV/AIDS and the opportunistic infections linked to the disease have been a threat to public health worldwide. This study's primary objective is to investigate how treatment compliance and accessibility affect the spread of HIV/AIDS. The model formulated is not country-specific and can be used in a variety of contexts, particularly in ones where a very high proportion of people lack access to HAART and where there is a lack of compliance with HAART's suggested procedures. In light of this, the study is set in this context and may be the first of its type to evaluate the effects of noncompliance with HAART guidelines. It is crucial to remember that being aware of one's HIV status on its own is insufficient. When utilized in conjunction with therapy and when treatment recommendations are rigorously followed, the benefits of being aware of one's HIV positive status are significantly greater. But if treatment is lacking and the recommended practices of the treatments are not adhered to then the HIV/AIDS escalation would be difficult to overcome in the population.

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