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Modelling impact of public awareness and behaviour changes with treatment on HIV/AIDS dynamics in resource-poor settings

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Abstract

HIV/AIDS prevalence continues to be on the increase in populations around the globe, especially in resource-poor nations with limited resources and inadequate public health services. A simple nonlinear *SIR* -type mathematical model that incorporates the use of HIV public awareness as a control in the presence of treatment of HIV infectives is proposed to theoretically assess the contributions of behaviour changes among the aware HIV susceptible population. The awareness programme causes a change in the behaviour of aware HIV susceptible individuals resulting into three susceptible classes. Qualitative analyses of the model including those of the basic properties are presented. The basic reproduction number of the model is determined and stability of equilibria analysed. The disease-free equilibrium point of the model is shown to be both locally and globally asymptotically stable when its corresponding basic reproduction number is less than unity. The Centre Manifold theory is used to show that the endemic equilibrium point is locally asymptotically stable when its corresponding reproduction number is greater than unity. Using a Lyapunov function of the Goh-Volterra type we also proved that the endemic equilibrium point is globally asymptotically stable when its corresponding reproduction number is greater than unity. Finally, a numerical study of the model was carried out to validate the results of our qualitative analyses.

Keywords: mathematical model; HIV/AIDS; HIV awareness programme; behaviour change; bifurcation

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1 Introduction

Since coming to international recognition in 1981 [6] the pandemic of Human Immunodeficiency Virus (HIV) infection, the cause of the Acquired Immunodeficiency Syndrome (AIDS), has infected more than 76 million people and claimed more than 35 million lives according to recent estimates by The Joint United Nations Programme on HIV/AIDS (UNAIDS) 2017 updates [15] and ranks among the most devastating scourges of the 21st century. Globally, HIV/AIDS has grown from a handful of reported cases in 1981 to a global pandemic that has affected virtually every country in the world. Although prevalence of HIV infection has dropped significantly in recent years, there were about 36.7 million people worldwide living with HIV in 2016 [15]. For example, based on current UNAIDS estimates, approximately 1.8 million people became infected with HIV and about 1 million died from AIDS-related causes in 2016 [15]. Eastern and Southern Africa are the worst hit; hosting about 19.4 million people living with HIV in 2016 [15]. As of June 2017, only 20.9 million people living with HIV were accessing ART. HIV/AIDS has destroyed the health of individuals and the well being of families and communities. It also threatens the economies of entire nations, especially in the developing countries. In fact, HIV/AIDS is a serious growing public health problem worldwide. The cause is known and the principal routes of transmission understood, but the resources for treating HIV infected individuals and combating the spread of the virus are limited. There are many factors such as lack of awareness and education by susceptible individuals, unsafe sex and low condoms usage, injecting drug use, wide-spread stigma, gender inequality, migration and mobility etc, which also affect the spread of HIV infection.

Although there has been a considerable success in medically managing HIV infection (through antiretroviral therapy (ART)) which improves the length and quality of life of people living with HIV, there is no well-documented case of anyone being truly 'cured' of HIV infection [9]. So far, important success in HIV prevention have been achieved with the following proven strategies: (1) HIV testing and counselling; (2) awareness/education and behaviour changes/modification; (3) mass-media campaigns; (4) condoms (male and female); (5) screening of blood supplies; (6) treatment and prevention of drug and alcohol abuse; (7) clean needles and syringes (that is, "needle exchange" programmmes); (8) antiretroviral therapy (ART) for interruption of HIV transmission from mother to child; (9) ART for post-exposure prophylaxis; and (10) medically supervised adult male circumcision [9]. Combination therapy with multiple antiretroviral drugs (ARVs) (which, however, do not cure patients of HIV infection) has resulted in dramatic reductions in HIV prevalence and AIDS-related illnesses and deaths wherever the medications have been available and

appropriately used in rich and poor countries alike [13, 24]. However, ARVs are still yet not available and readily accessible particularly in resource poor nations which suffer the vast majority of the HIV burden globally. For instance, only about 20.9 million people out of a global total of about 36.7 million people living with HIV in 2016 were assessing ART as at June, 2016 [14]. Thus in other to reduce the staggering number of new HIV infections in these countries, prevention programmes such as HIV public health awareness campaigns must be dramatically scaled up and in some cases refined and improved.

HIV/AIDS public awareness is an important tool or strategy against the acquisition and the spread of HIV as it exposes the populace to the risks and consequences of being HIV positive and encouraging behaviour changes in individuals. Moreover, it also helps individuals to take necessary precautions to reduce their chances of being infected. These behavioural changes also help governments and non-governmental health care providers to control the spread of the HIV/AIDS diseases.

Several authors have proposed and studied mathematical models of HIV/AIDS in other to monitor and explore the impact of intervention programmes that are being implemented. Similarly, numerous mathematical models have been developed to examine the role of various modes of HIV transmission, sexual transmission of HIV incorporating effect of awareness and treatment, effect of screening of HIV infectives [3, 10, 18, 22]. For example, [20] proposed and studied the effects of awareness programmes through media on disease dynamics in a variable population with immigration. [8] proposed and studied the role of media and treatment on an SIR model. The analyses of their model showed that if treatment is not available, then the infection is high even if awareness is present. Recently, [21] have studied a slow-fast dynamics with the effect of awareness program in disease outbreak.

There are several ways HIV public awareness campaigns can be targeted to effectively combat the burden of HIV disease in a community. This study adapts the strategy of targeting the unaware susceptible populations in a community where treatment of HIV infected individuals is present. It is important to note that realistic human behavioural changes relating to the spread of diseases and the efficacy of interventions remains an important factor in epidemic disease outbreaks as this help in sustaining control programmes and prospects for elimination. Also, modifications of behaviour are likely to occur as perception of risk shifts along with transmission. Thus, incorporating variation in behaviour in epidemiological models over the course of interventions is essential if models are to reliably predict the impact of interventions. In this study, therefore, we are investigating the impacts of HIV public awareness on unaware HIV susceptible individuals in a population as a control to manage the acquisition and the spread of HIV/AIDS in resource-poor communities where treatment of HIV infected individuals is present. We adapt the model from [19]. [19] which is an optimal control model, has three susceptible classes depending on

behaviour changes and is having three different transmission rates and with time-varying education campaign levels. The present model also has three susceptible classes depending on behaviour changes due to HIV awareness and is also having two different transmission rates but without time-varying awareness campaign levels, when treatment of HIV infectives is present. Thus, in this study, we propose a simple SIR-type network mathematical model of HIV/AIDS dynamics to study the impact of HIV awareness campaigns on the dynamics of HIV/AIDS targeting the unaware susceptible populations in the presence of ART in a community. Here, by "awareness", we mean unaware HIV susceptible individuals who have received proper or effective HIV public health education/counselling against risky sexual behaviours that may result in HIV infection. It is assumed in this study that the HIV public awareness campaigns against the disease may encourage the aware HIV susceptibles to isolate themselves from infected individuals and thus form separate class(es).

The study is arranged as follows. In the next section, we formulate the mathematical model and discuss its basic properties and stability analysis. In section 3, we establish the local and global asymptotic stability of equilibria. Numerical study and discussions are in section 4, and conclusion is in the final section.

2 Model Formulation

We develop a deterministic sex-structured mathematical model of Susceptible, Infected and Recovered - an SIR-type model that incorporates HIV awareness as the intervention strategy that helps to change the sexual behaviours of some individuals in the susceptible class. Here, by "awareness", we mean unaware HIV susceptible individuals who have received proper or effective HIV public health education/counselling against risky sexual behaviours that may result in HIV infection. This change in behaviour leads to subdividing the susceptibles into three subclasses, namely unaware HIV susceptible individuals S, aware HIV susceptible individuals who modify their sexual behaviours S_1, and aware HIV susceptible individuals who remain faithful to their uninfected sexual partners for life S_2. Individuals only enter the general unaware HIV susceptible class S.

The recruitment into the S class is at a rate Λ and the natural death rate for all subclasses is μ. As a result of awareness (through HIV counselling and testing) a proportion of the susceptibles leave the general unaware susceptibles class S at the rate ρ $(0<\rho<1)$ out of which a fraction ϕ $(0<\phi<1)$ reduce their sexual behaviours sufficiently by remaining faithful to their uninfected sexual partners for the rest of their lives (and are thus literally "immune" to HIV infection by sexual contact) and so move to the S_2 subclass while the complementary $(1-\phi)$ reduce their sexual activities

and so move to the S_1 subclass. The expected reduction in risky sexual behaviours by the S_1 class as a result of awareness is accounted for by the parameter $0 < \tau < 1$. It is pertinent to note here that, the inclusion of the S_2 subclass is very significant because an appreciable number of people have now changed their sexual behaviours sufficiently due to the awareness of the widespread nature of the HIV in society, the monumental deaths resulting from the disease, increasing knowledge of the agony and psychological trauma experienced by the infected individuals, and better enlightenment due to intense HIV/AIDS educational campaigns [25]. The significance of the S_2 subclass is that it emphasizes the importance of prevention for a disease, such as HIV, that has no cure. Increasing the members of this subclass is one of the keys to controlling the spread of the disease. Since it is assumed in this study that the S_2 subclass is not involved in extra-marital activities and hence transmission, the infection with HIV is with S and S_1 subclasses only, with effective infection contact rates β_1 and β_2, respectively, due to their interaction with the Infected class I. Thus the disease spreads due to the direct contact between them S and S_1 subclasses with the infected class (I). Individuals in the I class, with a diseased-induced death rate α, receive treatment with ARVs at a rate δ and hence move to the Removed class R of individuals who are assumed in this study not to be involved in transmission as a result of efficacy of ART. The R class has an additional disease-induced death rate of υ.

Thus, the total population of size $N(t)$, at time t, is given by

$$N(t) = S(t) + S_1(t) + S_2(t) + I(t) + R(t). \tag{1}$$

Based on the fact that the infectious period of HIV is very long $(\geq 10 years)$, we regard the population size as varying and not staying constant. A schematic diagram for the model is shown in Figure 1.

From the model assumptions and flow diagram (Fig.1), the model takes the form of the following deterministic system of nonlinear ordinary differential equations that describes the interaction of HIV public awareness with unaware HIV susceptibles as:

$$\left.\begin{aligned} S' &= \Lambda - \lambda S - \rho S - \mu S \\ S_1' &= (1-\phi)\rho S - \tau\lambda_1 S_1 - \mu S_1 \\ S_2' &= \phi\rho S - \mu S_2 \\ I' &= \lambda S + \tau\lambda_1 S_1 - (\delta + \alpha + \mu) I \\ R' &= \delta I - (\upsilon + \mu) R \end{aligned}\right\}, \tag{2}$$

with initial conditions $S(0) > 0, S_1(0) > 0, S_2(0) > 0, I(0) \geq 0, R(0) \geq 0$. The rate of change of the total populations is obtained by adding the equations of the model system Eq. (2) to give

$$N' = \Lambda - \mu N - \alpha I - \upsilon R. \tag{3}$$

In (2), the forces of infection for the S and S_1 subclasses are λ and λ_1 respectively, where $\lambda = \beta_1 I$ and $\lambda_1 = \beta_2 I$.

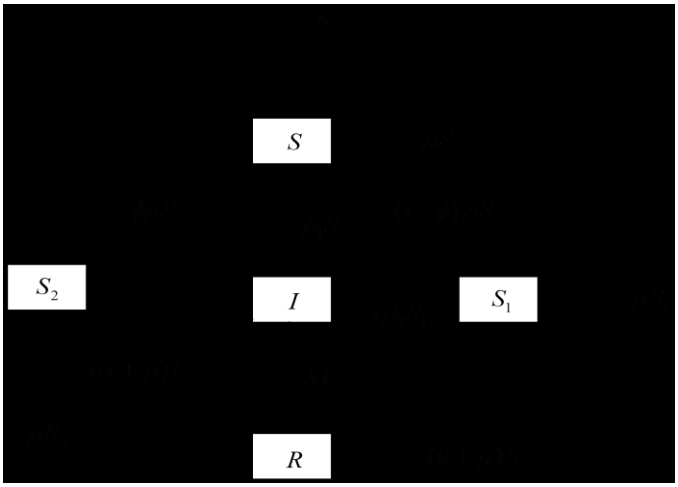


Figure 1. A flow diagram of the model system Eq. (2).

Since the model system Eq. (2) monitors human populations and HIV public awareness with treatment, it is assumed that all the state variables are non-negative for all time $t \geq 0$. All parameters in the model are assumed to be non-negative and one can show that the solutions of the system are non-negative, given non-negative initial conditions.

2.1 Basic Properties of the Model

2.1.1 Invariant Region

Theorem 2.1 *The region* $\Sigma \subset$ *is positively-invariant for the basic model system Eq. (2) with non-negative initial conditions in*

Proof. From Eq. (3), we have that

$$N' = \Lambda - \mu N - \alpha I - \upsilon R.$$

$$N' \leq \Lambda - \mu N. \qquad (4)$$

Solving the differential inequality equation (4), we find that

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

In particular, $N(t) \leq \frac{\Lambda}{\mu}$ if $N(0) \leq \frac{\Lambda}{\mu}$. Thus, the model system Eq. (2) will be analyzed in the following feasible region, $\Sigma \subset$ with

$$\Sigma = \left\{ \left(S(t), S_1(t), S_2(t), I(t), R(t) \right) \in \quad \leq \frac{\Lambda}{\mu} \right\}, \tag{5}$$

and is positively- invariant and attracting for the model system Eq.(2). This completes the proof. □

2.1.2 Positivity of Solutions

Theorem 2.2 ***T****he variables of the model system Eq. (2) are non-negative for all time. In other words, solutions of the model system Eq. (2) with positive initial data will remain positive for all time* $t \geq 0$.

Proof. Let $t_1 = \sup\{t > 0 : S > 0, S_1 > 0, S_2 > 0, I > 0, R > 0 \in [0, t]\}$. Thus $t_1 > 0$. It follows from the first equation of the model system Eq. (2) that

$$\begin{aligned} \frac{dS}{dt} &= \Lambda - \lambda(t)S(t) - \rho S(t) - \mu S(t) \\ &= \Lambda - (\lambda(t) + \rho(t) + \mu)S(t), \text{ where } \lambda(t) = \beta_1 I. \end{aligned} \tag{6}$$

This is the same as

$$\frac{dS}{dt} + (\lambda(t) + \rho(t) + \mu)S(t) = \Lambda,$$

and this implies that

$$\frac{d}{dt}\left[S(t) \exp\left\{ dt + \int_0^t (\lambda(\tau) + \rho(\tau)) d\tau \right\} \right] = \Lambda \exp\left\{ dt + \int_0^t (\lambda(\tau) + \rho(\tau)) d\tau \right\}. \tag{7}$$

Hence,

$$S(t_1) \exp\left\{ dt_1 + \int_0^{t_1} (\lambda(\tau) + \rho(\tau)) d\tau \right\} - S(0) = \int_0^{t_1} \Lambda \exp\left\{ dy + \int_0^y (\lambda(\in) + \rho(\in)) d \in \right\} dy. \tag{8}$$

Hence,

$$\begin{aligned} S(t_1) &= S(0) \exp\left\{ -(dt_1 + \int_0^{t_1} (\lambda(\tau) + \rho(\tau)) d\tau \right\} \\ &\quad + \left[\exp\left\{ -(dt_1 + \int_0^{t_1} (\lambda(\tau) + \rho(\tau)) d\tau \right\} \right] \\ &\quad \times \int_0^{t_1} \Lambda \exp\left\{ dy + \int_0^y (\lambda(\in) + \rho(\in)) d \in \right\} \\ &\geq 0. \end{aligned} \tag{9}$$

Similarly, it can be shown that $S_1 > 0, S_2 > 0, I \geq 0$ and $R \geq 0,$ for all time $t \geq 0.$ This completes the proof. □

The above Theorem is important because it guaranties that the model variables are continuously biologically meaningful, since population size cannot be negative. Thus, for any starting non-negative initial conditions, the trajectory lies in Σ. Therefore, the system is both mathematically and epidemiologically well-posed [11, 16]. Thus, it is sufficient to consider the dynamics of the flow generated by the model system Eq. (2) in Σ.

3 Equilibrium points and Stability Analysis

The model system Eq. (2) has two non-negative equilibriums: *Disease-free equilibrium (DFE) point* and *Endemic equilibrium (EE) point.*

3.1 DFE and Basic Reproduction Number, R_0.

The model system Eq. (2) has a DFE, obtained by setting the right-hand sides of the equations in the model system Eq. (2) to zero, and is given by

$$\mathrm{E}_0 = \left(S^0, S_1^0, S_2^0, I^0, R^0\right) = \left(\frac{\Lambda}{\rho+\mu}, \frac{(1-\phi)\rho\Lambda}{\mu(\rho+\mu)}, \frac{\phi\rho\Lambda}{\mu(\rho+\mu)}, 0, 0\right). \tag{10}$$

The local stability of E_0 can be established using the next-generation operator method on the model system Eq. (2). We take, I, as our infected compartment, then using the notation in [23], the Jacobian matrices F and V for the new infection terms and the remaining transfer terms are respectively given by

$$F = [\beta_1 S^0 + \tau\beta_2 S_1^0], \text{ and } V = [\delta + \alpha + \mu].$$

Taking the inverse of V, we have

$$V^{-1} = \left[\frac{1}{\delta+\alpha+\mu}\right], \text{ so that } FV^{-1} = \left[\frac{\beta_1 S^0 + \varepsilon\beta_2 S_1^0}{\delta+\alpha+\mu}\right].$$

It follows that the basic reproduction number of the model system Eq. (2), denoted by R_0, is given by

$$R_0 = \rho(FV^{-1}) = \frac{\beta_1 S^0 + \tau\beta_2 S_1^0}{(\delta+\alpha+\mu)} = \frac{\beta_1\Lambda}{(\rho+\mu)(\delta+\alpha+\mu)} + \frac{\tau\beta_2(1-\phi)\rho\Lambda}{\mu(\rho+\mu)(\delta+\alpha+\mu)},$$

where ρ is the spectral radius, i.e. the dominant eigen-value of $\left|FV^{-1} - \lambda I\right| = 0.$ Furthermore, using Theorem 2 in [23], we obtained the following result.

Theorem 3.1 *The disease-free equilibrium of the model system Eq. (2), given by* E_0*, is locally asymptotically stable (LAS) if* $R_0 < 1$ *, and unstable if* $R_0 > 1$.

Note that R_0 can also be written as

$$R_0 = \frac{\beta_1 \Lambda}{(\rho+\mu)(\delta+\alpha+\mu)} + \frac{(1-\phi)\rho\tau\beta_2\Lambda}{\mu(\rho+\mu)(\delta+\alpha+\mu)},$$

$$= R_s + R_{s_1}, \tag{11}$$

where,

$$R_s = \frac{\beta_1 \Lambda}{(\rho+\mu)(\delta+\alpha+\mu)}, \quad R_{s_1} = \frac{(1-\phi)\rho\tau\beta_2\Lambda}{\mu(\rho+\mu)(\delta+\alpha+\mu)}.$$

Here, R_s represents the contribution of unaware HIV susceptible individuals (S) to secondary infections, while R_{s_1} is the contribution of aware susceptible individuals (S_1) to secondary infections.

The threshold parameter R_0 measures the average number of new HIV infections generated by a single HIV infected individual throughout his/her infectious period in a completely susceptible population [1, 7, 11, 23] in which a proportion of aware susceptibles (S_2) are not involved in the dynamics of the disease in the presence of ART. Thus, Theorem 3 implies that the infection can be eliminated from the population if the initial sizes of the sub-populations are in the basin of attraction of the DFE E_0.

To ensure that disease elimination is independent of the initial sizes of sub-populations, it is necessary to show that the DFE E_0 is globally asymptotically stable (GAS) if $R_0 < 1$. This is explored below.

3.2 Global Stability of DFE, E_0

To prove global stability of the DFE E_0 of the model system Eq. (2), we apply the approach by [5], by rewriting the model system Eq. (2) as

$$\frac{dX}{dt} = F(X, Y),$$

$$\frac{dY}{dt} = G(X, Y), \quad G(X, 0) = 0, \tag{12}$$

where $X \in \mathbb{R}$ $_1, S_2)$ denotes the number of uninfected individuals (susceptible) and $Y \in \mathbb{R}$) denotes the number of infected individuals. The DFE of the model system Eq. (2) is now denoted as $\mathrm{E}_0 = (X^0, 0), \; X^0 = (S^0, S_1^0, S_2^0)$.

The conditions for global stability of E_0 are given by

HI $\frac{dX}{dt} = F(X^0, 0), \quad X^0$ is globally asymptotically stable (GAS).

HII $G(X,Y) = NY - G(X,Y),\ G(X,Y) \geq 0$ for $(X,Y) \in \Sigma,$ (13)

where $N = D_Y G(X^0, 0)$ is an M-matrix (whose diagonal elements are non-negative) and Σ is the region where the model system Eq. (2) makes biological and epidemiological sense.If the model system (12) satisfies the conditions (HI) and (HII) in (13) then, according to [5], the following theorem holds:

Theorem 3.2 *The DFE* $E_0 = (X^0, 0)$ *of model system (5) is GAS provided* $R_0 < 1$ *and that conditions (HI) and (HII) are satisfied.*

Proof: To apply Theorem 4 on the model system (12), we proceed thus.

From the model systems Eq. (2) and (12), we have

$$F(X,0) = \begin{pmatrix} \Lambda - (\rho + \mu)S \\ (1-\phi)\rho S - \mu S_1 \\ \phi\rho S - \mu S_2 \end{pmatrix},$$

$$G(X,Y) = NY - G(X,Y),$$

where,

$$N = D_Y G(X^0, 0) = \begin{pmatrix} \beta_1 S^0 + \tau\beta_2 S_1^0 - (\delta + \alpha + \mu) \\ \delta \end{pmatrix},$$

and, from system (12), and the conditions (HI) and (HII) in (13), we obtain the following result:

$$\begin{aligned} G(X,Y) &= NY - G(X,Y) \\ &= \begin{bmatrix} \beta_1 S^0 + \tau\beta_2 S_1^0 - (\delta + \alpha + \mu) \\ \delta \end{bmatrix} I - \begin{bmatrix} \beta_1 IS + \tau\beta_2 IS_1 - (\delta + \alpha + \mu)I \\ \delta I - (\nu + \mu)R \end{bmatrix} \\ &= \begin{bmatrix} \beta_1 I(S^0 - S) + \tau\beta_2 I(S_1^0 - S_1) \\ (\nu + \mu)R \end{bmatrix}. \end{aligned}$$

Since $S^0 \geq S$ and $S_1^0 \geq S_1$, it is clear that $G(X,Y) \geq 0$. Therefore, conditions (HI) and (HII) of Theorem 4 are satisfied. Hence, the DFE of model system Eq. (2), given by E_0, is globally asymptotically stable (GAS) when $R_0 < 1$. This completes the proof. □

The implication of the above result to public health is that if the control strategy for HIV/AIDS can force the reproduction number to below the value of one (unity), then HIV/AIDS can be eliminated from the community/population.

3.3 Existence and Stability of Endemic Equilibrium Point (EEP) E_e

In this section, the possible existence and stability of endemic (positive) equilibrium point of the model system Eq. (2) (i.e. equilibrium where at least one of the infected components of the model is non-zero) will be explored.

3.3.1 Existence of the (EE) E_e

When $R_0 > 1$ HIV infection can establish itself or persist in the population. Otherwise the infection will be eliminated. If $R_0 > 1$, system Eq. (2) has a unique endemic equilibrium E_e given by $E_e = (S^*, S_1^*, S_2^*, I^*, R^*)$, where

$$
\begin{aligned}
S^* &= \frac{\Lambda}{\beta_1 I^* + (\rho + \mu)}, \\
S_1^* &= \frac{(1-\phi)\rho S^*}{\tau\beta_2 I^* + \mu} = \frac{(1-\phi)\rho\Lambda}{(\tau\beta_2 I^* + \mu)[\beta_1 I^* + (\rho + \mu)]}, \\
S_2^* &= \frac{\phi\rho S^*}{\mu} = \frac{\phi\rho\Lambda}{\mu[\beta_1 I^* + (\rho + \mu)]}, \\
(\delta + \alpha + \mu) I^* &= \beta_1 I^* S^* + \tau\beta_2 I^* S_1^* = \frac{\Lambda\beta_1 I^*}{\beta_1 I^* + (\rho + \mu)} + \frac{(1-\phi)\rho\Lambda\tau\beta_2 I^*}{(\tau\beta_2 I^* + \mu)[\beta_1 I^* + (\rho + \mu)]}, \\
R^* &= \frac{\delta I^*}{(\nu + \mu)}.
\end{aligned}
\tag{14}
$$

From the fourth equation of system (14), $I^* = 0$ corresponds to the DFE, E_0 (whose stability has already been established) and the other nonzero root is obtained from (15):

$$
\frac{\Lambda\beta_1 I^*}{\beta_1 I^* + (\rho + \mu)} + \frac{(1-\phi)\rho\Lambda\tau\beta_2 I^*}{(\tau\beta_2 I^* + \mu)[\beta_1 I^* + (\rho + \mu)]} - (\delta + \alpha + \mu) = 0. \tag{15}
$$

For backward bifurcation to occur, multiple nonzero (endemic) equilibria must exist. Now, simplifying equation (15) we obtain the following quadratic equation in I^*, given by

$$
A_1 I^{*2} + A_2 I^* + A_3 = 0, \tag{16}
$$

so that the quadratic equation (16) can be analyzed for the possibility of multiple equilibria, where

$$
A_1 = \beta_1\tau\beta_2(\delta + \alpha + \mu) > 0,
$$

$$
A_2 = (\delta + \alpha + \mu)[\tau\beta_2(\rho + \mu) + \beta_1\mu] - \beta_1\tau\beta_2\Lambda
$$

$$= \tau\beta_2(\rho+\mu)(\delta+\alpha+\mu)\left[1-\frac{\beta_1\Lambda}{(\rho+\mu)(\delta+\alpha+\mu)}\right]+\beta_1\mu(\delta+\alpha+\mu),$$

$$A_3 = \mu(\rho+\mu)(\delta+\alpha+\mu)-\beta_1\mu\Lambda-\tau\beta_2(1-\phi)\rho\Lambda \quad (17)$$

$$= (1-R_0)\mu(\rho+\mu)(\delta+\alpha+\mu).$$

For the endemic equilibria to exist, the solutions of (16) must be real and positive. We note that

$$A_1 \geq 0, A_3 < 0 \Leftrightarrow R_0 > 1;$$

$$A_3 \geq 0 \Leftrightarrow R_0 \leq 1. \quad (18)$$

Equation (16) is a quadratic equation with respect to I^* since $A_1 > 0$. Let the discriminant be $\Delta = A_2^2 - 4A_1A_3$. If $A_2 \geq 0$, then (16) has no positive solution. Also if $\Delta < 0$, then (16) has no real solution. So we see that if $A_2 < 0$ and $\Delta \geq 0$, then (16) has two positive solutions. Solving for $\Delta = 0$ in terms of R_0, we get $R_0 = R_0^c$, where

$$R_0^c = 1-\frac{A_2^2}{4\beta_1\tau\beta_2\mu(\rho+\mu)(\delta+\alpha+\mu)^2}. \quad (19)$$

We can clearly note the following equivalent relations:

$$\Delta < 0 \Leftrightarrow R_0 < R_0^c, \ \Delta = 0 \Leftrightarrow R_0 = R_0^c,$$

$$\Delta > 0 \Leftrightarrow R_0 > R_0^c. \quad (20)$$

We thus have the following results on existence of the endemic equilibrium.

Theorem 3.3 *The HIV/AIDS model system Eq. (2) has*

(i) a unique endemic equilibrium whenever $R_0 > 1$;

(ii) a unique endemic equilibrium whenever $R_0 = 1$ *and* $A_2 < 0$;

(iii) a unique endemic equilibrium of multiplicity 2 when $R_0 = R_0^c$ *and* $A_2 < 0$;

(iv) two endemic equilibria $E_1(S_1, S_{11}, S_{21}, I_1, R_1)$ *and* $E_2(S_2, S_{12}, S_{22}, I_2, R_2)$, *when* $R_0^c < R_0 < 1$ *and*

$A_2 < 0$, *where*

$$I_1 = \frac{-A_2+\sqrt{\Delta}}{2\tau\beta_1\beta_2(\delta+\alpha+\mu)} \text{ and } I_2 = \frac{-A_2-\sqrt{\Delta}}{2\tau\beta_1\beta_2(\delta+\alpha+\mu)}.$$

Thus, setting $S_1 = \frac{\Lambda}{\beta_1 I_1+(\rho+\mu)}$ *and* $S_2 = \frac{\Lambda}{\beta_1 I_2+(\rho+\mu)}$;

$$S_{11} = \frac{(1-\phi)\rho\Lambda}{(\tau\beta_2 I_1+\mu)[\beta_1 I_1+(\rho+\mu)]} \text{ and } S_{12} = \frac{(1-\phi)\rho\Lambda}{(\tau\beta_2 I_2+\mu)[\beta_1 I_2+(\rho+\mu)]};$$

$$S_{21} = \frac{\phi\rho\Lambda}{\mu[\beta_1 I_1+(\rho+\mu)]} \text{ and } S_{22} = \frac{\phi\rho\Lambda}{\mu[\beta_1 I_2+(\rho+\mu)]};$$

$$R_1 = \frac{\delta I_1}{(\nu+\mu)} \text{ and } R_2 = \frac{\delta I_2}{(\nu+\mu)};$$

then $E_i = (S_i, S_{1i}, S_{2i}, I_i, R_i),\ i = 1,2$ *are endemic equilibria of the HIV/AIDS model system Eq. (2).*

(v) has no endemic equilibria whenever $R_0 < R_0^c$ *and* $A_2 > 0$ *or whenever* $R_0 \le 1$ *and* $A_2 > 0$.

From Theorem 5, we note that there is a unique EE when $R_0 > 1$ which approaches zero as $R_0 \to 1_+$ and there cannot be an EE if $R_0 < 1$. In this case it is possible to have a backward bifurcation at $R_0 = 1$. However, if $A_2 < 0$, system Eq. (2) has a unique EE when $R_0 > 1$ and has two different endemic equilibria when $R_0^c < R_0 < 1$, and system Eq. (2) has no EE when $0 < R_0 < R_0^c$. Hence system Eq.(2) has a backward bifurcation at $R_0 = 1$ from the DFE to two endemic equilibria. To conclude, we have the following result.

Theorem 3.4 *The HIV/AIDS model system Eq. (2) has a backward bifurcation at* $R_0 = 1$ *if and only if* $A_2 < 0$.

The public health implication of backward bifurcation is that the classical requirement of having the basic reproduction number less than unity, although necessary, is not sufficient for disease control.

3.3.2 Local Stability of the EE E_e

Using the standard linearization of the model system Eq. (2) to determine the local asymptotic stability of an EE point is labourious to tract mathematically. We now employ Theorem 4.1 in [4], to establish the local asymptotic stability of the EE. We make the following change of variables in order to apply the Centre Manifold Theory:

$$S = x_1,\ S_1 = x_2,\ S_2 = x_3,\ I = x_4,\ R = x_5. \tag{21}$$

We now use the vector $X = (x_1, x_2, x_3, x_4, x_5)^T$. Then model system Eq. (2) can be written in the form $\frac{dX}{dt} = F$, where $F = (f_1, f_2, f_3, f_4, f_5)^T$, such that

$$\begin{aligned}
\frac{dx_1}{dt} &= f_1 = \Lambda - \beta_1 x_4 x_1 - \rho x_1 \mu x_1 \\
\frac{dx_2}{dt} &= f_2 = (1-\phi)\rho x_1 - \tau\beta_2 x_4 x_2 - \mu x_2 \\
\frac{dx_3}{dt} &= f_3 = \phi\rho x_1 - \mu x_3
\end{aligned} \tag{22}$$

$$\frac{dx_4}{dt} = f_4 = \beta_1 x_4 x_1 + \tau\beta_2 x_4 x_2 - (\delta + \alpha + \mu)x_4$$
$$\frac{dx_5}{dt} = f_5 = \delta x_4 - (\nu + \mu)x_5$$

The Jacobian matrix of the model system Eq. (2) at E_0 is given by

$$J_{(E_0)} = \begin{bmatrix} -(\rho+\mu) & 0 & 0 & -\frac{\beta_1\Lambda}{(\rho+\mu)} & 0 \\ (1-\phi)\rho & -\mu & 0 & -\frac{\tau\beta_2(1-\phi)\rho\Lambda}{\mu(\rho+\mu)} & 0 \\ \phi\rho & 0 & -\mu & 0 & 0 \\ 0 & 0 & 0 & \frac{\beta_1\Lambda}{(\rho+\mu)} + \frac{\tau\beta_2(1-\phi)\rho\Lambda}{\mu(\rho+\mu)} - (\delta+\alpha+\mu) & 0 \\ 0 & 0 & 0 & \delta & -(\nu+\mu) \end{bmatrix}, \tag{23}$$

from which it can be shown that the reproduction number is

$$R_0 = \left(\frac{\beta_1\Lambda}{(\rho+\mu)} + \frac{\tau\beta_2(1-\phi)\rho\Lambda}{\mu(\rho+\mu)}\right)\left(\frac{1}{\delta+\alpha+\mu}\right).$$

If β_1 is taken as a bifurcation point and if we consider $R_0 = 1$ and solve for β_1 gives

$$\beta_1 = \beta^* = \frac{\mu(\rho+\mu)(\delta+\alpha+\mu) - \tau\beta_2(1-\phi)\rho\Lambda}{\mu\Lambda}. \tag{24}$$

It can simply be observed that the Jacobian matrix $J_{(E_0)}$ of the linearized system of the transformed Eq. (22) with $\beta_1 = \beta^*$ possesses a simple zero eigenvalue and the rest of the eigenvalues have negative real parts. Therefore, the Centre Manifold Theory in [4] is appropriate to analyse the dynamics of the system Eq. (22) near $\beta_1 = \beta^*$. For the case where $R_0 = 1$, it can be shown that the Jacobian matrix $J_{(E_0)}$ at $\beta_1 = \beta^*$ has a right-eigenvector (corresponding to the zero eigenvalue) expressed as $U = (u_1, u_2, u_3, u_4, u_5)^T$ where

$$u_1 = \frac{r_1}{(\rho+\mu)}u_4, u_2 = \frac{(1-\phi)\rho u_1 + r_2 u_4}{\mu}, u_3 = \frac{\phi\rho}{\mu}u_1 = \frac{r_1\phi\rho}{\mu(\rho+\mu)}u_4,$$
$$u_5 = \frac{\delta}{(\nu+\mu)}u_4, u_4 = u_4 > 0. \tag{25}$$

The left-eigenvector of $J_{(E_0)}$ associated with the zero eigenvalue at $\beta_1 = \beta^*$ is given by $V = (v_1, v_2, v_3, v_4, v_5)^T$, where

$$v_2 = v_3 = v_5 = 0, v_1 = -\frac{r_3}{r_1}v_4, v_4 = v_4 > 0. \tag{26}$$

In order to establish the condition for the existence of backward bifurcations, we use Theorem 7 proven in [4].

Theorem 3.5 *(See Theorem 4.1 in [4]) Consider the following general system of ordinary differential equations with a parameter* ϕ:

$$\frac{dx}{dt} = f(x,\phi), f:\Re^n \times \Re \to \Re^n \text{ and } f \in C^2(\Re^n \times \Re). \tag{27}$$

Without loss of generality, it is assumed that 0 *is an equilibrium for system (27) for all values of the parameter* ϕ; *that is,* $f(0,\phi) = 0$ *for all* ϕ. *Assume*

A1: $A = D_x f(0,0) = \left(\frac{\partial f_i}{\partial x_j}(0,0) \right)$ *is the linearization of system (27) around the equilibrium* 0 *with* ϕ *evaluated at* 0. *Zero is a simple eigenvalue of* A *and other eigenvalues of* A *have negative real parts.*

A2: *Matrix* A *has a right eigenvector* w *and a left eigenvector* v *corresponding to the zero eigenvalue. Let* f_k *be the* k^{th} *component of* f *and*

$$a = \sum_{k,i,j=1}^{n} v_k w_i w_j \frac{\partial^2 f_k}{\partial x_i \partial x_j}(0,0),$$

$$b = \sum_{k,i=1}^{n} v_k w_i \frac{\partial^2 f_k}{\partial x_i \partial \phi}(0,0). \tag{28}$$

The local dynamics of (27) around 0 *are governed by* a *and* b *in the following manner:*

i. $a > 0, b > 0$, *when* $\phi < 0$ *with* $|\phi| << 1, 0$ *is locally asymptotically stable, and there exists a positive unstable equilibrium; when* $0 < \phi << 1, 0$ *is unstable and there exists a negative and locally asymptotically stable equilibrium;*

ii. $a < 0, b < 0$. *When* $\phi < 0$ *with given by* $|\phi| << 1, 0$ *is unstable; when* $0 < \phi << 1, 0$ *is locally asymptotically stable, and there exists a positive unstable equilibrium;*

iii. $a > 0, b < 0$. *When* $\phi < 0$ *with* $|\phi| << 1, 0$ *is unstable, and there exists a locally asymptotically stable negative equilibrium; when* $0 < \phi << 1, 0$ *is stable, and a positive unstable equilibrium appears;*

iv. $a < 0, b > 0$. *When* ϕ *changes from negative to positive,* 0 *changes its stability from stable to unstable. Correspondingly, a negative equilibrium becomes positive and locally asymptotically stable.*

Using Theorem 4.1 in [4] as reproduced in Theorem 7, we determine the signs of a and b, where

$$a = \sum_{h,i,j=1}^{5} v_h u_i u_j \frac{\partial^2 f_h}{\partial x_i \partial x_j}(E_0, \beta^*), \quad b = \sum_{h,i=1}^{5} v_h u_i \frac{\partial^2 f_h}{\partial x_i \partial \beta^*}(E_0, \beta^*).$$

Then for system Eq. (22), the non-zero partial derivatives of F associated with a at the DFE are given by

$$\frac{\partial^2 f_1}{\partial x_1 \partial x_4} = \frac{\partial^2 f_1}{\partial x_4 \partial x_1} = -\beta^*, \frac{\partial^2 f_2}{\partial x_2 \partial x_4} = \frac{\partial^2 f_2}{\partial x_4 \partial x_2} = -\tau\beta_2, \frac{\partial^2 f_4}{\partial x_1 \partial x_4} = \frac{\partial^2 f_4}{\partial x_4 \partial x_1} = \beta^*,$$
$$\frac{\partial^2 f_4}{\partial x_2 \partial x_4} = \frac{\partial^2 f_4}{\partial x_4 \partial x_2} = \tau\beta_2. \tag{29}$$

Taking into account of Eq. (25) and (26), it follows from the expressions in (29) that

$$\begin{aligned} a &= v_1 u_1 u_4 \frac{\partial^2 f_1}{\partial x_1 \partial x_4} + v_1 u_4 u_1 \frac{\partial^2 f_1}{\partial x_4 \partial x_1} + v_2 u_2 u_4 \frac{\partial^2 f_2}{\partial x_2 \partial x_4} + v_2 u_4 u_2 \frac{\partial^2 f_2}{\partial x_4 \partial x_2} + v_4 u_1 u_4 \frac{\partial^2 f_4}{\partial x_1 \partial x_4} + v_4 u_4 u_1 \frac{\partial^2 f_4}{\partial x_4 \partial x_1} \\ &\quad + v_4 u_2 u_4 \frac{\partial^2 f_4}{\partial x_2 \partial x_4} + v_4 u_4 u_2 \frac{\partial^2 f_4}{\partial x_4 \partial x_2} \\ &= v_1 u_1 u_4 (-\beta^*) + v_1 u_4 u_1 (-\beta^*) + v_2 u_2 u_4 (-\tau\beta_2) + v_2 u_4 u_2 (-\tau\beta_2) + v_4 u_1 u_4 (\beta^*) + v_4 u_4 u_1 (\beta^*) \\ &\quad + v_4 u_2 u_4 (\tau\beta_2) + v_4 u_4 u_2 (\tau\beta_2) \\ &= -2v_1 u_1 u_4 \beta^* - 2v_2 u_2 u_4 \tau\beta_2 + 2v_4 u_1 u_4 \beta^* + 2v_4 u_2 u_4 \tau\beta_2 \\ &= -2v_1 u_1 u_4 \beta^* + 2v_4 u_1 u_4 \beta^* + 2v_4 u_2 u_4 \tau\beta_2 \\ &= -2\frac{\left[\beta^* \Lambda^2 \mu^2 + \tau^2 \beta_2^2 (1-\phi)\rho\Lambda(\rho+\mu)^2 + \beta^*(\delta+\alpha+\mu)(\rho+\mu)^2 \mu^2 - \beta^{*2}\Lambda(\rho+\mu)\mu^2\right]}{\mu(\rho+\mu)^3}. \end{aligned} \tag{30}$$

For the sign of b, it is associated with the following non-vanishing partial derivatives of F:

$$\frac{\partial^2 f_1}{\partial x_4 \partial \beta^*} = -\frac{\Lambda}{\rho+\mu}, \frac{\partial^2 f_4}{\partial x_4 \partial \beta^*} = \frac{\Lambda}{\rho+\mu}. \tag{31}$$

And taking into account Eq. (25) and (26), it follows from the expressions of (31) that

$$\begin{aligned} b &= v_1 u_4 \frac{\partial^2 f_1}{\partial x_4 \partial \beta^*} + v_4 u_4 \frac{\partial^2 f_4}{\partial x_4 \partial \beta^*} \\ &= v_1 u_4 \left(-\frac{\Lambda}{\rho+\mu}\right) + v_4 u_4 \left(\frac{\Lambda}{\rho+\mu}\right) \\ &= \frac{(\delta+\alpha+\mu)(\rho+\mu)\mu - \tau\beta_2(1-\phi)\rho\Lambda}{\mu(\rho+\mu)\beta^*}. \end{aligned} \tag{32}$$

Obviously, from the calculations in Eq. (30) and (32), $a < 0$ and $b > 0$, and hence, according to Theorem 7 item (iv), the following result is established.

Theorem 3.6 *The endemic equilibrium E_e of the HIV/AIDS model system Eq. (2) is locally asymptotically stable for $R_e > 1$, but close to 1.*

3.3.3 Global Stability of the EEP E_e

***Theorem* 3.7** *If $R_0 > 1$, then there exist an endemic equilibrium E_e and it is globally asymptotically stable (GAS).*

Proof: Given that $R_0 > 1$, then the existence of the endemic equilibrium is guaranteed. Considering the following Lyapunov function candidate $V(S, S_1, S_2, I, R)$, given as

$$V = \frac{1}{2}\left(S - S^*\right)^2 + \frac{1}{2}\left(S_1 - S_1^*\right)^2 + \frac{1}{2}\left(S_2 - S_2^*\right)^2 + \in\left(I - I^* - I^* \ln\left(\frac{I}{I^*}\right)\right) + \frac{1}{2}\left(R - R^*\right)^2, \tag{33}$$

(where $\in$ is a positive constant to be chosen suitably)

Now differentiating with respect to time along the solutions path of the model system Eq. (2), we get

$$V' = \left(S - S^*\right)S' + \left(S_1 - S_1^*\right)S_1' + \left(S_2 - S_2^*\right)S_2' + \frac{\in}{I}\left(I - I^*\right)I' + \left(R - R^*\right)R'. \tag{34}$$

Substituting the model system Eq. (2) into the relation equation (34) gives

$$V' = \left(S - S^*\right)\left(\Lambda - \beta_1 IS - \rho S - \mu S\right) + \left(S_1 - S_1^*\right)\left((1-\phi)\rho S - \tau\beta_2 IS_1 - \mu S_1\right) + \left(S_2 - S_2^*\right)\left(\phi\rho S - \mu S_2\right)$$

$$+ \frac{\in}{I}\left(I - I^*\right)\left(\beta_1 IS + \tau\beta_2 IS_1 - (\delta + \mu)I\right) + \left(R - R^*\right)\left(\delta I - (\nu + \mu)R\right). \tag{35}$$

We recall that at the endemic equilibrium point E_e, we have

$$\begin{aligned}
\Lambda &= \beta_1 I^* S^* + (\rho + \mu)S^*, \\
(1-\phi)\rho &= \tau\beta_2 I^* \frac{S_1^*}{S^*} + \mu\frac{S_1^*}{S^*}, \\
\phi\rho &= \mu\frac{S_2^*}{S^*}, \\
(\delta + \alpha + \mu) &= \beta_1 S^* + \tau\beta_2 S_1^*, \\
\delta &= (\nu + \mu)\frac{R^*}{I^*}.
\end{aligned} \tag{36}$$

Using the equilibrium conditions equation (36), equation (35) becomes

$$V' = \left(S - S^*\right)\left(\beta_1 I^* S^* + (\rho + \mu)S^* - \beta_1 IS - (\rho + \mu)S\right)$$
$$+ \left(S_1 - S_1^*\right)\left(\tau\beta_2 I^* S_1^* \frac{S}{S^*} + \mu S_1^* \frac{S}{S^*} - \tau\beta_2 IS_1 - \mu S_1\right)$$

$$+\left(S_2-S_2^*\right)\left(\mu S_2^*\frac{S}{S^*}-\mu S_2\right)+\frac{\in}{I}\left(I-I^*\right)\left(\beta_1 IS+\tau\beta_2 IS_1-(\beta_1 S^*+\tau\beta_2 S_1^*)I\right)$$

$$+\left(R-R^*\right)\left[(\nu+\mu)R^*\frac{I}{I^*}-(\nu+\mu)R\right]$$

$$=\left(S-S^*\right)\left[\beta_1(S^*I^*-SI)+(\rho+\mu)(S^*-S)\right]+(S_1-S_1^*)\left[\tau\beta_2(S_1^*I^*-S_1I)+\mu(S_1^*-S_1)\right]$$

$$+(S_2-S_2^*)\left[\mu(S_2^*-S_2)\right]+\in(I-I^*)\left[\beta_1(S-S^*)+\tau\beta_2(S_1-S_1^*)\right]-(\nu+\mu)(R-R^*)^2$$

$$(Since\ S\le S^*, I\le I^*)$$

$$=-(\rho+\mu)(S-S^*)^2-\mu(S_1-S_1^*)^2-\mu(S_2-S_2^*)^2-(\nu+\mu)(R-R^*)^2+\beta_1(S-S^*)(S^*I^*-SI)$$

$$+\tau\beta_2(S_1-S_1^*)(S_1^*I^*-S_1I)+\in\beta_1(I-I^*)(S-S^*)+\in\tau\beta_2(I-I^*)(S_1-S_1^*)$$

$$=-(\rho+\mu)(S-S^*)^2-\mu(S_1-S_1^*)^2-\mu(S_2-S_2^*)^2-(\nu+\mu)(R-R^*)^2$$

$$+\beta_1(S-S^*)\left[S^*(I^*-I)-I(S-S^*)\right]+\tau\beta_2(S_1-S_1^*)\left[S_1^*(I^*-I)-I(S_1-S_1^*)\right]$$

$$+\in\beta_1(I-I^*)(S-S^*)+\in\tau\beta_2(I-I^*)(S_1-S_1^*)$$

$$=-(\rho+\mu)(S-S^*)^2-\mu(S_1-S_1^*)^2-\mu(S_2-S_2^*)^2-(\nu+\mu)(R-R^*)^2$$

$$-\beta_1 I(S-S^*)^2-\beta_1 S^*(S-S^*)(I-I^*)-\tau\beta_2 I(S_1-S_1^*)^2-\tau\beta_2 S_1(S_1-S_1^*)(I-I^*)$$

$$+\in\beta_1(I-I^*)(S-S^*)+\in\tau\beta_2(I-I^*)(S_1-S_1^*)$$

$$=-(\rho+\mu+\beta_1 I)(S-S^*)^2-(\mu+\tau\beta_2 I)(S_1-S_1^*)^2-\mu(S_2-S_2^*)^2-(\nu+\mu)(R-R^*)^2$$

$$+\beta_1(\in-S^*)(S-S^*)(I-I^*)+\tau\beta_2(\in-S_1^*)(S_1-S_1^*)(I-I^*)$$

$$\le 0. \qquad setting\ \in=S^*, \in=S_1^*$$

It is important to note that $V'=0$ holds only at E_e. By LaSalle's invariant principle [17], every solution to the system Eq. (2), with positive initial conditions in Σ, approaches E_e as $t\to\infty$ if $R_0>1$. Hence, the endemic equilibrium E_e is globally asymptotically stable (GAS) in Σ if $R_0>1$. This ends the proof. □

4 Numerical Simulation of Analytical Results and Discussions

To study the behaviour of the model system Eq. (2) numerically, a fourth order Runge-Kutta scheme is used. A hypothetical population of about one million sexually active individuals was considered. Table 1 summarizes all the parameters used in the simulations. That is, to illustrate some of the theoretical results arrived at in this paper, simulations of the model system Eq. (2) using various initial conditions were carried out using parameter values in Table 1. The HIV/AIDS transmission model system Eq. (2) that incorporates HIV public awareness was studied to investigate the impacts of behaviour changes by the aware HIV susceptible individuals when treatment of

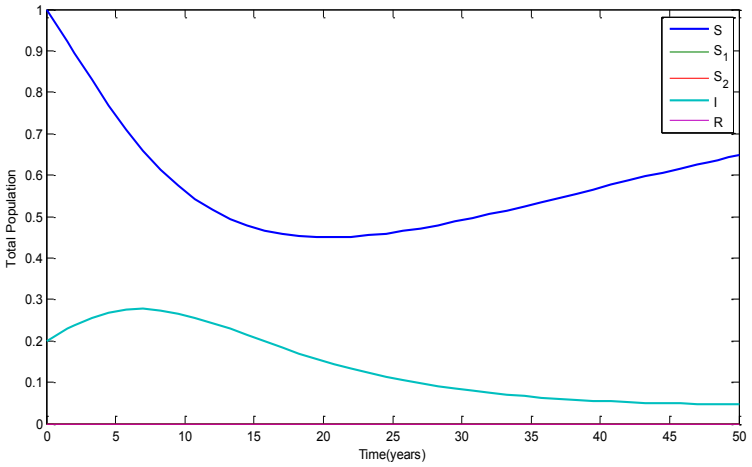
HIV infected individuals is present, using parameter values in Table 1 and the following initial conditions:

$$S(0)=1, S_1(0)=0, S_2(0)=0, I(0)=0.2, \text{ and } R(0)=0,$$

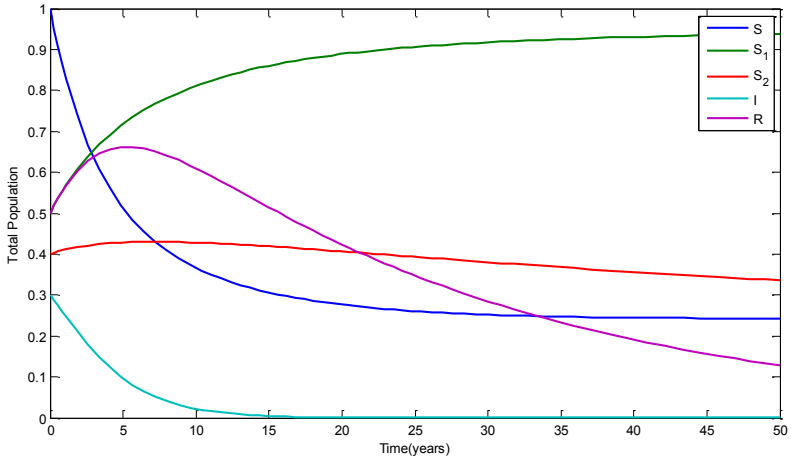
except when otherwise stated. Here $S(0), S_1(0), S_2(0), I(0)$ and $R(0)$, as well as the corresponding states in the figures, are in millions of individuals.

Parameter	Parameter description	Approx. value/year	Source
Λ	Recruitment rate per unit time	0.029	[2]
μ	Natural death rate per unit time	0.02	[2]
β_1	Incidence rate of unaware susceptibles per unit time	$0 \le \beta_1 \le 1$	[12]
β_2	Incidence rate of aware susceptibles per unit time	$0 \le \beta_2 \le 1$	[12]
ρ	Rate of susceptibles knowing their HIV status	$0 < \rho < 1$	[2]
ϕ	Proportion of aware susceptibles who remain faithful to their uninfected sexual partners for life	$0 < \phi < 1$	[2]
α	Disease-induced death rate of HIV infectives	0.18	[12]
δ	Treatment rate of HIV infectives	$0 < \delta < 1$	Assumed
ν	Disease-induced death rate of treated HIV infectives	0.02	Assumed
τ	Modification parameter	$0 \le \tau \le 1$	Assumed

Table 1. Model Parameters and their interpretations



1(a)

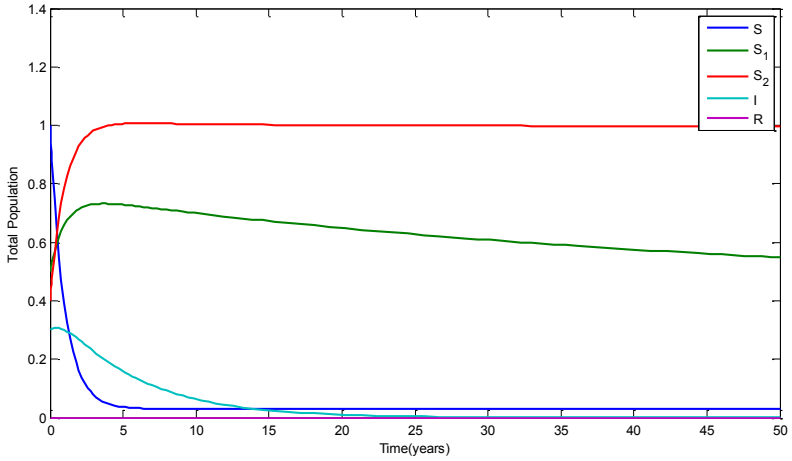


1(b)

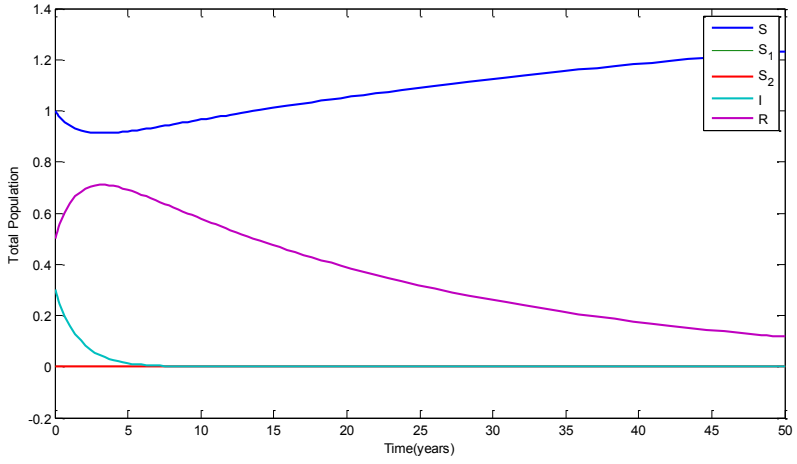
Figure 1. Impact of (a) absence of HIV public awareness and treatment of HIV infectives (i.e., $\rho = 0, \delta = 0$), and (b) presence of very low HIV public awareness with some behaviour change and treatment of HIV infectives (i.e., $\rho = 0.1, \phi = 0.2, \delta = 0.3$).

In Fig. 1(a), it is shown that when there is neither an HIV public awareness programme (targeting unaware HIV susceptibles) nor a treatment programme (for HIV infectives) available, then the rate of infection increases and gets saturated at high level and finally decreases. However, in Fig. 1(b), the impact of HIV public awareness programme implementation rate $\rho = 0.1$ (with a fraction $\phi = 0.2$ of aware HIV susceptibles remaining faithful to their uninfected sexual partners for life) and

treatment rate $\delta = 0.3$ (of HIV infectives) on the dynamics of the disease is shown simultaneously. It is observed that, even at low level implementation rates of awareness and treatment, there is a decrease in the infected population and it settles down to zero level in about 17 years. This shows that disease eradication could be possible under the given conditions. This indicates that HIV awareness (which leads to sufficient behaviour changes) and treatment programmes are important for HIV control.



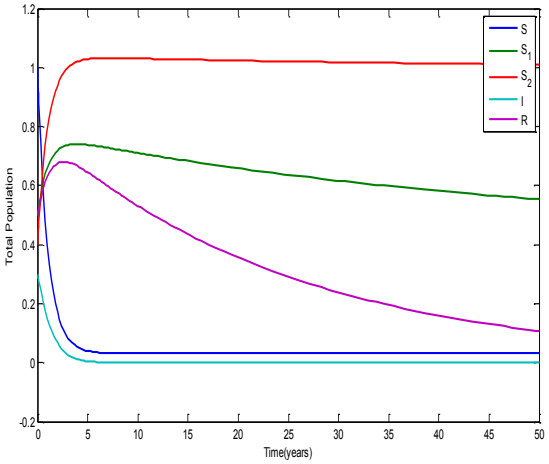
2(a)



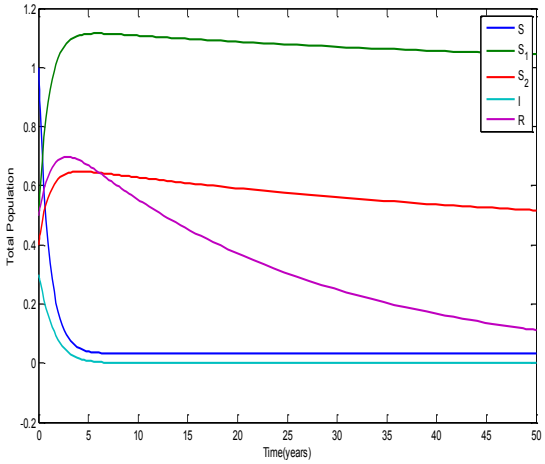
2(b)

Figure 2. Impacts of (a) of a very high HIV public awareness with sufficient behaviour changes in the absence of treatment of HIV infectives (i.e., $\rho = 0.9, \phi = 0.7, \delta = 0$), and (b) absence of HIV public awareness in the presence of very high treatment rate of HIV infectives (i.e., $\rho = 0, \phi = 0, \delta = 0.7$).

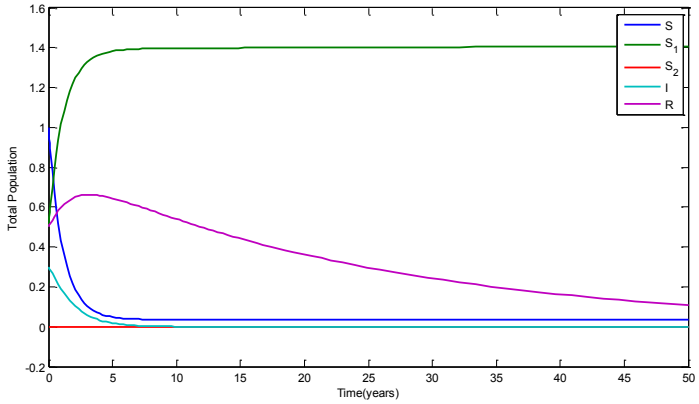
Fig. 2 represent the impacts of a very high rate ($\rho = 0.9$) of HIV awareness by unaware HIV susceptible individuals with a very high fraction ($\phi = 0.7$) of them modifying their sexual behaviours sufficiently by remaining faithful to their uninfected sexual partners for life in the absence of treatment for HIV infected individuals (Fig. 2(a)). Here, it is observed that there is a tremendous decline in the number of unaware HIV susceptible individuals (S) as a result of the awareness programme accompanied by a significant decrease in the number of HIV infectives (I) (which led to disease eradication in about 26 years). On the other hand, Fig. 2(b) illustrates the impacts of a very high treatment rate ($\delta = 0.7$) for HIV infectives in the absence of an HIV awareness programme for the unaware HIV susceptible individuals (S). Here, it is noticed an initial slow decline followed by a subsequent rise in the number of unaware HIV susceptible individuals (S) (as a result of absence of an HIV awareness programme), a significant decline in the number of HIV infectives (I) (which reduces the pool of HIV infected individuals) resulting into disease eradication in about 8 years.



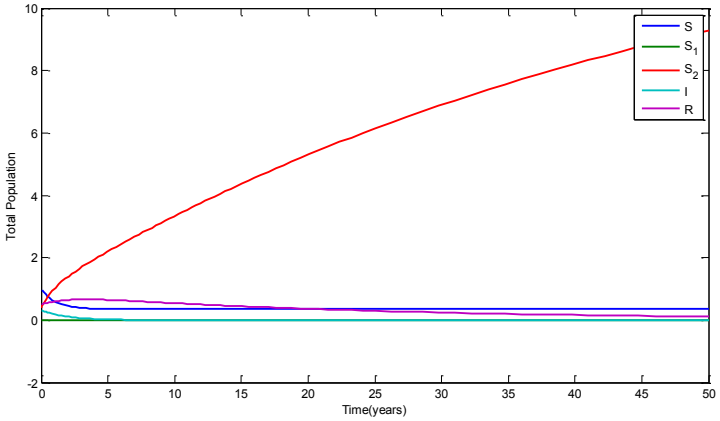
(3a)



(3b)



3(c)



3(d)

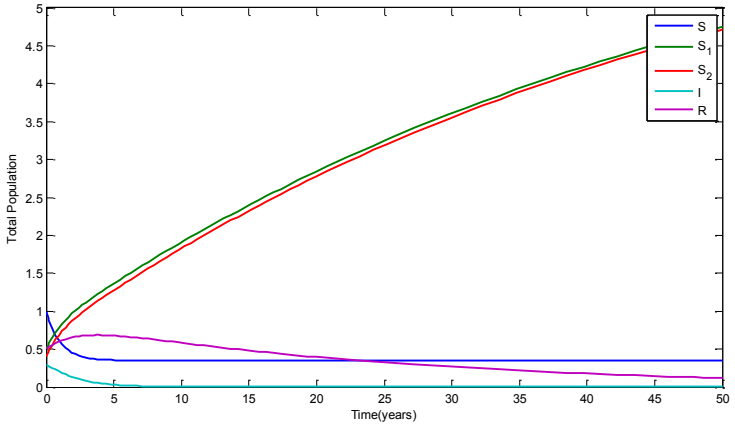
Figure 3. Impacts of (a) of very high effective rates of HIV public awareness ($\rho = 0.9, \phi = 0.7$) by unaware HIV susceptibles with sufficient behaviour changes and treatment ($\delta = 0.7$) of HIV infectives, (b) same as in 3(a) but with sufficient behaviour changes rate reduced from $\phi = 0.7$ to $\phi = 0.3$, (c) same as in 3(a) but with sufficient behaviour changes rate reduced from $\phi = 0.7$ to $\phi = 0$, and (d) same as in 3(a) but with sufficient behaviour changes rate increased from $\phi = 0.7$ to $\phi = 1$.

The simulation results illustrated by Figures 2(a) and (b), respectively, shows that HIV awareness campaigns or treatment of HIV infected individuals, as a single strategy alone, is not sufficient for the timely control and eradication of HIV/AIDS. This is because treatment of HIV infectives only significantly boosts the immunity of HIV infectives thus prolonging their life-span but does not cure them of the disease or prevent its transmission. This shows that the simultaneous implementation of

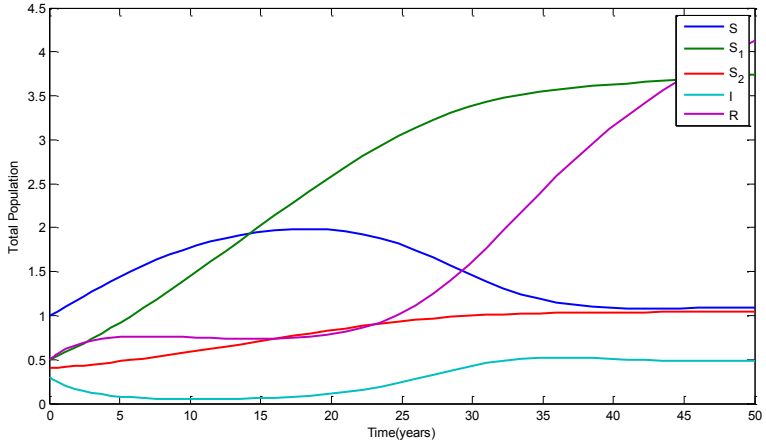
HIV public awareness targeting the unaware susceptibles (that results into sufficient behaviour modifications) with treatment of HIV infectives in a population will significantly reduce the contraction and spread of the HIV disease in the population /community.

In Fig. 3(a), the impacts of very high effective rates of HIV public awareness ($\rho = 0.9, \phi = 0.7$) by unaware HIV susceptible individuals S, with sufficient behaviour modifications, in the presence of very high treatment rate ($\delta = 0.7$) of HIV infected individuals is displayed. It is observed that the combined impacts of these two elaborate strategies leads to disease eradication in about 5 years. However, when all the parameter values in Fig. 3(a) are maintained but the sufficient behaviour modification rate ϕ was reduced from $\phi = 0.7$ to $\phi = 0.3$, it is observed, in Fig. 3(b) that it will take a longer period (of about 6 years) for disease eradication to occur. Similarly, maintaining all the parameter values in Fig. 3(a) but further reducing the sufficient behaviour modification rate ϕ from $\phi = 0.7$ to $\phi = 0$, it is noticed in Fig. 3(c) that, it will take a much longer period (of about 10 years) for disease eradication to occur. On the other hand, when all the aware susceptible individuals sufficiently modify their sexual behaviours and decide to remain faithful to their uninfected sexual partners for life (that is, $\phi = 1$), it is observed, as depicted in Fig. 3(d) that, it will take a very short period (of about 3 years) for disease eradication to occur.

In varying the recruitment (or birth) rate into the unaware HIV susceptible population S from $\Lambda = 0.029$ to $\Lambda = 0.29$ (a tenfold increase), it is observed in Fig. 4(a), by comparing with the simulation result displayed in Fig. 3(a) (where $\Lambda = 0.029$) that there is an astronomical increase in the populations of the S_1, S_2 subclasses, respectively, with tremendous decreases in the numbers of unaware HIV susceptible individuals in the S class and the I class, which resulted in disease eradication (in about 7 years); but this takes a longer period as compared with the result of Fig. 3(a) (which took about 5 years). Similarly, maintaining all the parameter values in Fig. 4(a) but reducing the HIV public awareness rate of unaware susceptible individuals from $\rho = 0.9$ to $\rho = 0.1$ and the behaviour modification rate of aware HIV susceptibles from $\phi = 0.7$ to $\phi = 0.3$, one can observe in Figure 4(b) the fluctuations over time in the plots of each of the numbers of individuals in the S, S_1, S_2, I and R classes, respectively, which however, did not lead to the desired result of eradicating the disease in finite time. Thus, the results of the simulations displayed in Fig. 4(a) and (b) shows that it is also very important to control the recruitment rate into the unaware susceptible population as well as encouraging sufficient behaviour changes among aware HIV susceptibles so as to effectively control and eradicate the HIV/AIDS epidemic from the population/community.



4(a)



4(b)

Figure 4. Impacts of (a) varying the recruitment rate into the unaware susceptible population from $\Lambda = 0.029$ to $\Lambda = 0.29$ (a tenfold increase) while maintaining all the other parameter values in Fig. 3(a), (b) maintaining all the parameter values in Fig. 4(a) but reducing the HIV awareness rate from $\rho = 0.9$ to $\rho = 0.1$ and the sufficient behaviour modification rate from $\phi = 0.7$ to $\phi = 0.3$.

5 Conclusion

In this paper, a mathematical model to study the impacts of HIV public awareness programme targeting the unaware HIV susceptibles on HIV/AIDS dynamics when treatment of HIV infectives is in place is introduced. The awareness programme results in dividing the HIV susceptible population into three classes of unaware, aware, and aware susceptibles who sufficiently modify their sexual behaviours by

remaining faithful to their uninfected sexual partners for life. The local and global dynamics of this model have been studied. It has been shown that there exist only two equilibrium points: the disease-free equilibrium E_0, that is total elimination of disease (as $I = 0$) and the endemic E_e, that is the disease will persist. The DFE is locally asymptotically stable for reproduction number $R_0 < 1$ and the endemic equilibrium exists for $R_0 > 1$ and is globally stable under the conditions stated in Theorem 6. Numerical simulations to validate the analytical results have also been carried out. It has been shown that if awareness programmes are not available in the population then the infection is very high and if awareness programmes is introduced, the infection decreases and this can further be reduced by treatment of HIV infectives. It has also been shown that when there is enough HIV awareness among unaware susceptibles with sufficient behaviour modifications and enough treatment is available for HIV infectives, then the disease can be eradicated completely. But if either awareness or treatment is lacking then the disease cannot be eradicated.

Finally, this work has shown that the prospect of effectively controlling the spread of HIV/AIDS in resource-poor settings is very bright. Preventive measures through the use of control strategies should concentrate on awareness programmes and treatment. Also, the awareness programmes should be sustained over a long period of time (especially in places with high endemic cases of HIV/AIDS) so as to effectively maintain and sustain behaviour changes among aware susceptible individuals and thus control and eradicate the HIV disease from the population in finite time.

Conflict of interest: The author declares that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

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