Applied Mathematics 2014, 4(3): 86-96
DOI: 10.5923/j.am.20140403.03

Nonlinear Dynamics and Chaos in HIV/AIDS Epidemic Model with Treatment

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Abstract A nonlinear dynamical system and qualitatively analysis of HIV/AIDS epidemic model with treatment is investigated. The model allows for some infected individuals to move from the symptomatic phase to the asymptomatic phase by all sorts of treatment methods. Mathematical analyses establish that the global dynamics of the spread of the HIV infectious disease are completely determined by the basic reproduction number R_0. If $R_0 \leq 1$, the disease free equilibrium is globally stable, where as the unique infected equilibrium is globally asymptotically stable if $R_0 \leq 1$. Finally, numerical simulations are performed to illustrate the analytical results.

Keywords Nonlinear Dynamics, HIV\AIDS, Epidemics, Treatment

1. Introduction

HIV/AIDS is one of the most deadly diseases humankind has ever faced, with profound social, economic and public health consequences. It has gradually over the decades become a global pandemic with Ghana not an exception. The number of people living with HIV rose from around 8 million in 1990 to 34 million by the end of 2011(USAID, 2012). The increasing trends of HIV pose a significant public health concern. Although there have been several attempts to curb the spread of HIV, the continual spread of the disease has persisted and there has been reported cases worldwide.

Mathematical models have been extensively used over the years in researching into the epidemiology of HIV/AIDS, to help improve our understanding of major contributing factors in a given epidemic (Naresh *et al*., 2006).

(Lima *et al.* 2008) developed a mathematical model to analyse the potential impact of scaling up highly active antiretroviral therapy (HAART) as a strategy to decrease HIV load at the population level on the spread of HIV. Results indicated that a higher HAART coverage consistently leads to decrease in the number of individuals testing newly positive for HIV.

Other researchers have sought to study the effects of various factors that can affect the transmission of the disease. In particular, (Anderson and May, 1998) developed a HIV transmission dynamics model using difference equations in the deterministic case and state transition probabilities in the stochastic case that represents the progression from HIV+ status to AIDS where the population is divided into categories of progressive infectious stages. Patterns of HIV have been studied extensively for over half a century. (Simwa *et al*. 2003) formulated a deterministic mathematical model for HIV epidemic transmission through heterosexual contact and vertically from an infected mother to her unborn child with three stages of disease progression among infected patients using two systems of ordinary differential equations.

With regard to the spread of disease, it has been established that the disease becomes more endemic due to immigration therefore the focus on infective immigrants is inevitable and comes in to ensure that the endemicity of the disease is practically reduced (Issa *et al.* 2011).

In terms of HIV treatment, (Montaner *et al.* 2006) established from their study on universal HIV testing, the use of antiretroviral (ARVs) for prevention of mother-to-child transmissions (PMTCT) and through post-exposure prophylaxis for sexual assaults and needle-stick injuries. In this paper, we seek to develop a nonlinear deterministic system to study the dynamics of the HIV disease at four compartments of the populations with treatment.

2. Model Formulation

In order to derive the model equations, the total population (N) is assumed to be constant and categorised into four compartments namely susceptible $(S(t))$, infective $(I(t))$, treated class $(T(t))$ and AIDS class $(A(t))$. The detailed transition between these four compartments is depicted in Figure 1. There is an inflow of newly recruited to the susceptible population at a rate α. Moreover, with the introduction of infectives and homogeneous mixing in the population, an individual become infected at rate β. It is

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Published online at http://journal.sapub.org/am

assumed that some people are not aware of their HIV status and thus do not seek medical attention. The treated class represents people who seek medical attention. Treatment is the process of offering the HIV positive individual with a life prolonging drug/medicine known as antiretroviral (ARV) medicine or antiretroviral treatment (ART). ART drugs are the main types of treatment for HIV/AIDS. New recruits into the treated class occur at a rate λ. Again, people who are ignorant of their HIV status in the infective class are recruited into the AIDS class at rate γ. Natural death at the various compartments occurs at a rate μ.

With the assumptions given and the illustrations in Figure. 1, the systems of initial value nonlinear differential equation for the SITA model are formulated as follows:

$$\frac{dS}{dt} = \alpha N - \frac{\beta SI}{N} - \mu S \tag{1}$$

$$\frac{dI}{dt} = \frac{\beta SI}{N} - (\gamma + \lambda + \mu) I \tag{2}$$

$$\frac{dI}{dt} = \lambda I - (\sigma + \mu) T \tag{3}$$

$$\frac{dA}{dt} = \gamma I + \sigma T - (\delta + \mu) A \tag{4}$$

Where

$S(0) = S_0,\ I(0) = I_0, T(0) = T_0,\ A(0) = A_0 \quad \forall\ t \geq 0$.

The total population, N is given by the formula: $N = S + I + T + A$

From equation (1)-(4)

$$\frac{dN}{dt} = \alpha N - \mu N - \delta A \tag{5}$$

In order to express the systems of equations in equations (1)-(4) as a fraction of the total population, and since the state variable A does not appear in the first three equations of system (1)-(4), we use the following substitutions: $s = \frac{S}{N}, i = \frac{I}{N}, z = \frac{T}{N}$

Hence resulting systems of equations shall be based on

$$\frac{ds}{dt} = \alpha - \beta si - \mu s \tag{6}$$

$$\frac{di}{dt} = \beta si - (\gamma + \lambda + \mu) i \tag{7}$$

$$\frac{dz}{dt} = \lambda i - (\sigma + \mu) z \tag{8}$$

3. Basic Properties of the Model

3.1. Positivity of the Solutions

Since the model monitors human population we need to show that all the state variables remain non-negative for all times.

Theorem 1: Let $\Omega = \{(s,i,z) \in \mathbb{R}^3_+ : s(0) > 0, i(0) > 0,\}$ then the solutions of $\{s(t), i(t), z(t)\}$ of the system (6)-(8) are positive for all $t \geq 0$.

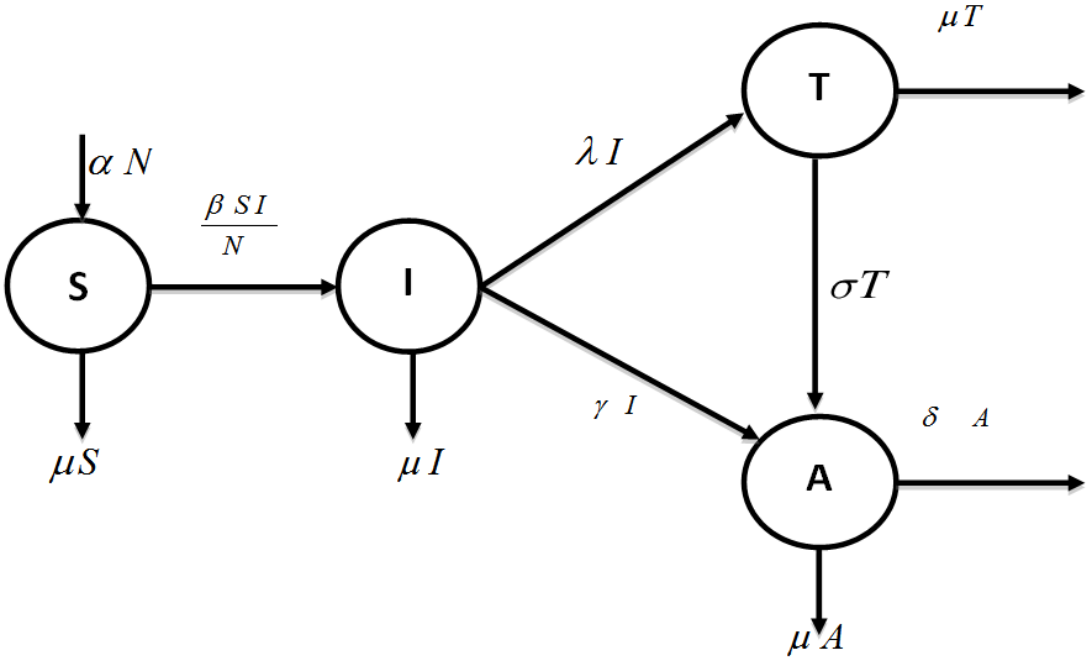


Figure 1. Schematics of the Susceptible-Infective-Treated-Aids (SITA) model

Proof:

Taking the first equation, we have

$$\frac{ds}{dt} = \alpha - \beta si - \mu s$$
$$\frac{ds}{dt} \geq -\mu s$$
$$\frac{ds}{s} \geq -\mu dt$$
$$\int \frac{ds}{s} \geq \int -\mu\, dt$$
$$s(t) \geq s(0)e^{-\mu dt} \geq 0$$

From the second equation we have

$$\frac{di}{dt} = \beta si - (\gamma + \lambda + \mu)i$$
$$\frac{di}{dt} \geq -(\gamma + \lambda + \mu)i$$
$$\frac{ds}{i} \geq -(\gamma + \lambda + \mu)dt$$
$$\int \frac{di}{i} \geq \int -(\gamma + \lambda + \mu)dt$$
$$i(t) \geq i(0)e^{-(\gamma+\lambda+\mu)dt} \geq 0$$

Finally, from the third equation, we have

$$\frac{dz}{dt} = \lambda i - (\sigma + \mu)i$$
$$\frac{dz}{dt} \geq -(\sigma + \mu)i$$
$$\frac{dz}{i} \geq -(\sigma + \mu)dt$$
$$\int \frac{dz}{z} \geq \int -(\sigma + \mu)dt$$
$$z(t) \geq z(0)e^{-(\sigma+\mu)dt} \geq 0$$

3.2. Invariant Region

The system (6)–(8) has solutions, which are contained, in the feasible region Γ.

Proof

Let $(s,i,z) \in \mathbb{R}^3_+$ be any solution of the system with non negative initial conditions then Adding the equations of the system (6)-(8), we have

$$\frac{ds}{st} + \frac{di}{dt} + \frac{dz}{dt} = \alpha - \gamma i - \sigma z - \mu(s + i + z) \leq \frac{\alpha}{\mu}$$

Hence

$$\limsup_{t\to\infty}(s + i + z) \leq \frac{\alpha}{\mu}$$

Thus the considered region for the system (6)-(8) is

$$\Gamma = \left\{(s,i,z) : (s + i + z) \leq \frac{\alpha}{\mu}, s \geq 0, i \geq 0, z \geq 0\right\}$$

The vector field points to the interior of Γ on the part of the boundary when $(s + i + z) = \frac{\alpha}{\mu}$ for and is positively invariant.

4. Equilibria

Lemma 4.1. The disease-free equilibrium of system (1)-(4) is given by

$$E_0 = (s,i,z) = \left(\frac{\alpha}{\mu}, 0, 0\right)$$

and the endemic equilibrium by

$$E_1 = (s^*, i^*, z^*),$$

where

$$s^* = \frac{\gamma + \lambda + \mu}{\beta}$$
$$i^* = -\frac{-\alpha\beta + \mu\gamma + \mu\lambda + \mu^2}{\beta(\gamma + \lambda + \mu)}$$
$$z^* = -\frac{\lambda(-\alpha\beta + \mu\gamma + \mu\lambda + \mu^2)}{\beta(\sigma\gamma + \sigma\lambda + \sigma\mu + \mu\gamma + \mu\lambda + \mu^2)}$$

With the natural mortality rate, μ considered constant throughout the model, the duration spent in the infectious class is given by $\frac{1}{(\gamma + \lambda)}$.

4.1. Basic Reproductive Ratio (R_0)

One of the fundamental questions of mathematical epidemiology is to find threshold conditions that determine whether an infectious disease will spread in a susceptible population when the disease is introduced into the population. It is defined as the average number of susceptible who can be infected by a typical infective in a population in which everybody is considered as susceptible (Diekmann *et. al.*, 1990). If the basic reproductive ratio is found to be greater than one, the disease will spread throughout the entire

population and also if it is less than one the disease eventually die off. Thus, the basic reproductive ratio determines the direction of the disease.

Although there have been several theories proposed by various researchers in the estimation of the basic reproductive ratio, we use the Next Generation Matrix approach (van der Driessche *et. al*. 2002, Diekmann *et. al*. 2000). It is given mathematically as

$$R_0 = \rho\left(FV^{-1}\right)$$

where ρ is defined as the spectral radius of the Next Generation Matrix $\left(FV^{-1}\right)$, F is the rate of appearance of new infections in compartment i and V is the transfer of individuals out of compartment i by all other means.

Given the DFE, R_0 is calculated as the largest eigenvalue (spectral radius) of the matrix of partial derivatives:

$$\left[\frac{\partial F_i\left(E_0\right)}{\partial x_j}\right]\left[\frac{\partial V_i\left(E_0\right)}{\partial x_j}\right]$$

where

$$\left[\frac{\partial F_i\left(E_0\right)}{\partial x_j}\right] = \begin{bmatrix} \frac{\partial F_1}{\partial i}\left(E_0\right) & \frac{\partial F_1}{\partial z}\left(E_0\right) \\ \frac{\partial F_2}{\partial i}\left(E_0\right) & \frac{\partial F_2}{\partial z}\left(E_0\right) \end{bmatrix} = \begin{bmatrix} \frac{\alpha\beta}{\mu} & 0 \\ 0 & 0 \end{bmatrix} \quad (9)$$

and

$$\left[\frac{\partial V_i\left(E_0\right)}{\partial x_j}\right] = \begin{bmatrix} \frac{\partial V_1}{\partial i}\left(E_0\right) & \frac{\partial V_1}{\partial z}\left(E_0\right) \\ \frac{\partial V_2}{\partial i}\left(E_0\right) & \frac{\partial V_2}{\partial z}\left(E_0\right) \end{bmatrix}:$$

$$= \begin{bmatrix} \left(\gamma+\lambda+\mu\right) & 0 \\ -\lambda & \left(\sigma+\mu\right) \end{bmatrix} \quad (10)$$

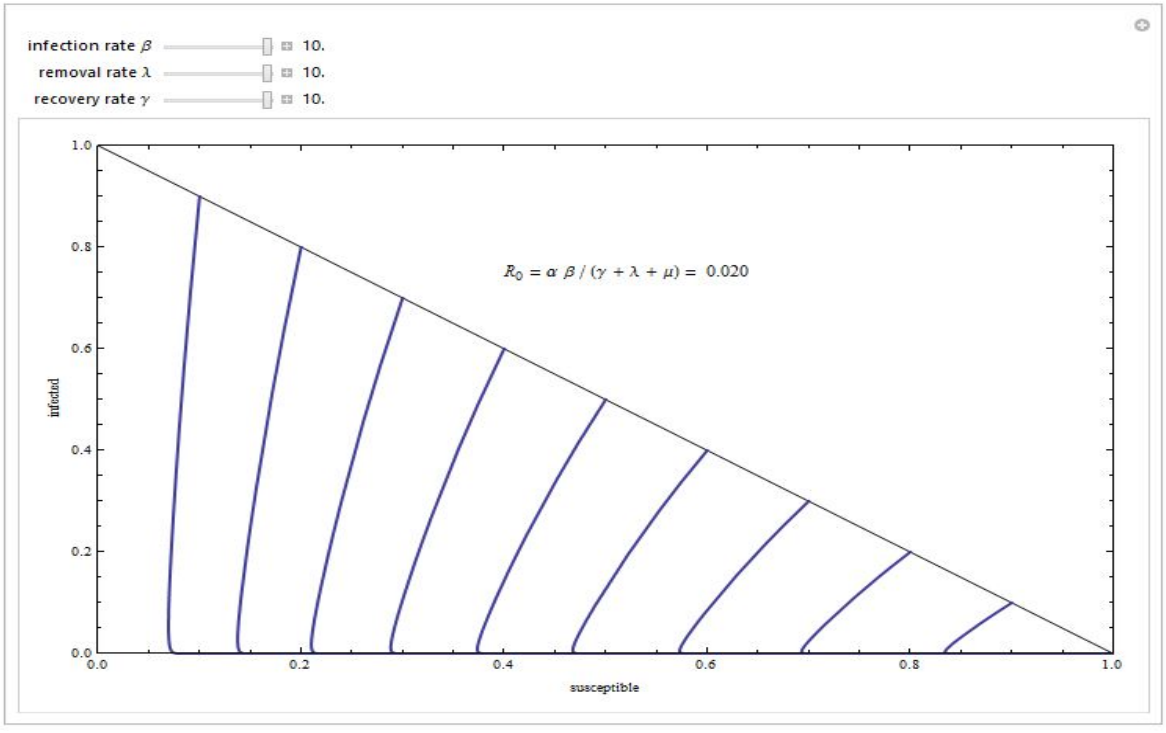
$$V^{-1} = \begin{bmatrix} \frac{1}{\gamma+\lambda+\mu} & 0 \\ \frac{\lambda}{\left(\sigma+\mu\right)\left(\gamma+\lambda+\mu\right)} & \frac{1}{\sigma+\mu} \end{bmatrix}$$

Therefore,

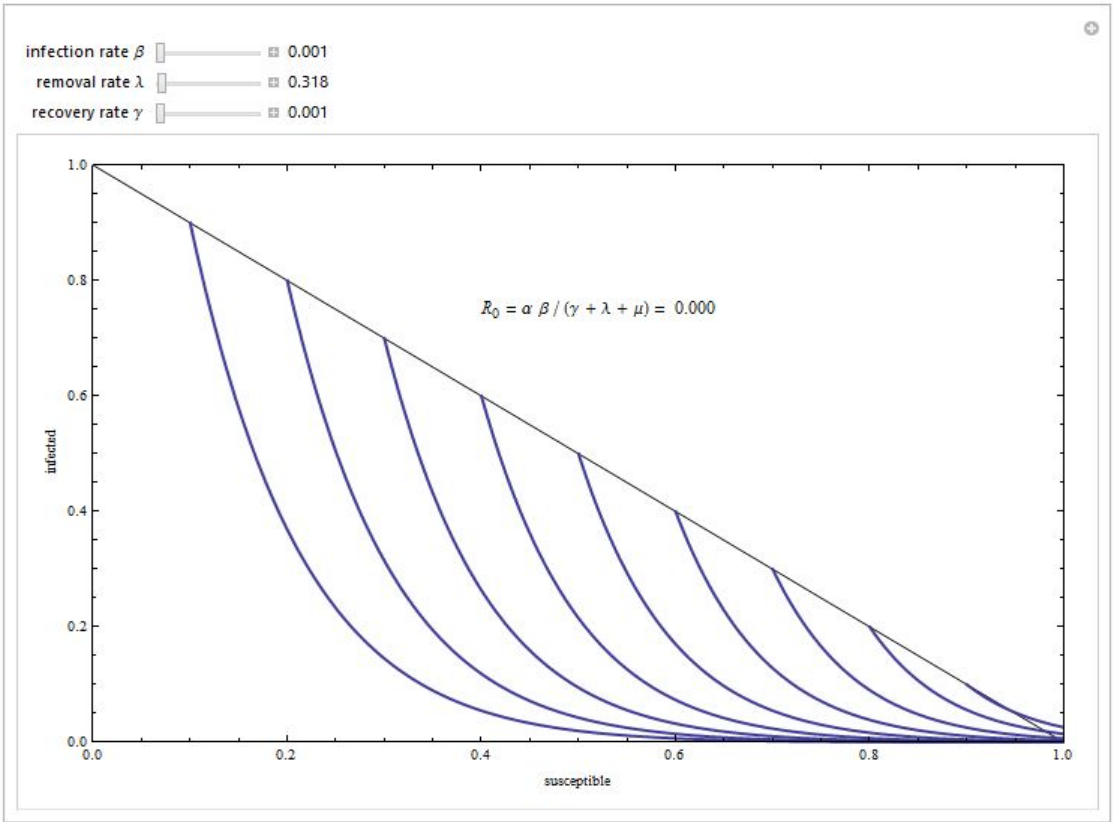
$$FV^{-1} = \begin{bmatrix} \frac{\alpha\beta}{\mu\left(\gamma+\lambda+\mu\right)} & 0 \\ 0 & 0 \end{bmatrix}$$

The spectral radius of the next generation matrix is

$$R_0 = \frac{\alpha\beta}{\mu\left(\gamma+\lambda+\mu\right)} \quad (11)$$



(a)



(b)

Figure 2. Phase plane portrait for the classic HIV endemic model with treatment rate σ = 0.01

4.2. Local Stability of the Disease-Free Equilibrium (DFE)

Lemma 4.2: The disease-free equilibrium is locally asymptotically stable whenever $R_0 < 1$. We shall use the linearization approach to proof the local stability of the disease-free equilibrium (DFE). The Jacobian matrix associated with the system (6)-(8) is:

$$J = \begin{bmatrix} -\beta i & -\beta s & 0 \\ \beta i & \beta s - \gamma - \lambda - \mu & 0 \\ 0 & \lambda & -\sigma - \mu \end{bmatrix}$$

At the DFE, which is given by $E_0 = \left(\frac{\alpha}{\mu}, 0, 0\right)$, we have

$$J(E_0) = \begin{bmatrix} -\mu & -\frac{\alpha\beta}{\mu} & 0 \\ 0 & \frac{\alpha\beta}{\mu} - \gamma - \lambda - \mu & 0 \\ 0 & \lambda & -\sigma - \mu \end{bmatrix}$$

Clearly the eigenvalues at the DFE are given by:

$$T_1 = -\mu,\ T_2 = -\frac{-\alpha\beta + \gamma\mu + \lambda\mu + \mu^2}{\mu},\ T_3 = -\sigma - \mu$$

For the positive parameters $\alpha, \beta, \sigma, \gamma, \lambda$ and μ, it can be seen that eigenvalues of the DFE are all negative and hence the DFE is stable.

Since $T_2 < 0$, we have

$$\begin{aligned} & -\frac{-\alpha\beta + \gamma\mu + \lambda\mu + \mu^2}{\mu} < 0 \\ & \Rightarrow \alpha\beta - \gamma\mu - \lambda\mu - \mu^2 < 0 \\ & \Rightarrow \alpha\beta < \gamma\mu + \lambda\mu + \mu^2 \\ & \Rightarrow \frac{\alpha\beta}{\mu(\gamma + \lambda + \mu)} < 1 \end{aligned} \tag{12}$$

Note that $R_0 < 1$ implies that the inequality (12) also holds and thus we have proved Lemma 4.2.

4.3. Global Stability of the Disease-Free Equilibrium (DFE)

Lemma 4.3: If $R_0 \leq 1$ then the disease-free equilibrium $E_0 = \left(\frac{\alpha}{\mu}, 0, 0\right)$ is globally asymptotically stable in $\mathbb{R}^3_+$.

Proof: Given that $R_0 \le 1$, then there exist only the disease free equilibrium $E_0 = (s_0, i_0, z_0) = \left(\frac{\alpha}{\mu}, 0, 0\right)$. Considering that Lyapunov function candidate $L(s,i,z): \mathbb{R}^3 \to \mathbb{R}^+$ defined as

$$L(s,i,z) = \omega i \qquad \omega \ge 0$$

Differentiating $L(s,i,z)$ with respect to time yields

$$L' = \omega i'$$

Substituting the system (6)-(8), we have

$$\begin{aligned}
\dot{L} &= \omega(\beta s i - \gamma i - \lambda i - \mu i) \\
&= \omega(\beta s - (\gamma + \lambda + \mu)) i \\
&\le \omega\left(\frac{\alpha\beta}{\mu} - (\gamma + \lambda + \mu)\right) i \qquad \text{since } s \le s_0 = \frac{\alpha}{\mu} \\
&= \omega(\gamma + \lambda + \mu)\left(\frac{\alpha\beta}{\mu(\gamma + \lambda + \mu)} - 1\right) i \qquad \left(\text{using } \omega = \frac{1}{\gamma + \lambda + \mu}\right) \\
&= (R_0 - 1) i \\
&\le 0
\end{aligned}$$

It is important to note that, $\dot{L} = 0$ only when $i = 0$. However, substituting $i = 0$ into the equations for $\dot{s}$ and $\dot{z}$ in (6)-(8) shows that $s \to \frac{\alpha}{\mu}$ and $z \to 0$ as $t \to \infty$. Therefore, the maximum invariant set in $\left\{(s,i,z) \in \Gamma \middle| \dot{L} \le 0\right\}$ is the singleton set $\{E_0\}$. Hence, the global stability of $\boldsymbol{E_0}$ when $R_0 \le 1$ follows from LaSalle's invariance principle (Lasalle, 1976 and Tewa *et. al*. 2009).

4.4. Local Stability of Endemic Equilibrium (EE)

Lemma 4.4: The endemic equilibrium is locally asymptotically stable if $R_0 > 1$.

Proof. The Jacobian equilibrium is locally asymptotically stable if $R_0 > 1$.

$$J(E_1) = \begin{pmatrix} -R_0 & -\gamma - \lambda - \mu & 0 \\ R_0 + \mu & 0 & 0 \\ 0 & \lambda & -\sigma - \mu \end{pmatrix}$$

The eigenvalues of $J(E_0)$ are

$$T_1 = -(\sigma + \mu), \quad T_{2,3} = \frac{-R_0 \pm \sqrt{R_0^2 - 4(R_0 + \mu)(\gamma + \lambda + \mu)}}{2}$$

Hence, if $R_0 > 1$, then $T_2 < 0$ and $T_3 < 0$.

4.5. Global Stability of the Endemic Equilibrium (EE)

Lemma 4.5: *For* $\boldsymbol{R_0 > 1}$*, system (6)-(8) is globally asymptotically stable, if* $\boldsymbol{s = s^*}$, $\boldsymbol{i = i^*}$, $z = z^*$, *and* $X < Y$, *and unstable* $R_0 \le 1$.

Proof. Using the constructed Lyapunov function by (Cai, L. and Li, Z., 2010), the global stability of the endemic equilibrium is proved. By defining the Lyapunov function as follows.

$$V\left(s^*,i^*,z^*\right)=\left(s-s^*-s^*\log\frac{s^*}{s}\right)+\left(i-i^*-i^*\log\frac{i^*}{i}\right)+\left(z-z^*-z^*\log\frac{z^*}{z}\right)$$

By direct calculating the derivative of V along the solution of system (6)-(8) we have;

$$\frac{dV}{dt}=\left(\frac{s-s^*}{s}\right)\frac{ds}{dt}+\left(\frac{i-i^*}{i}\right)\frac{di}{dt}+\left(\frac{z-z^*}{z}\right)\frac{dz}{dt}$$

$$\frac{dV}{dt}=\left(\frac{s-s^*}{s}\right)\left(\alpha-\beta si-\mu s\right)+\left(\frac{i-i^*}{i}\right)\left(\beta si-\left(\gamma+\lambda+\mu\right)i\right)+\left(\frac{z-z^*}{z}\right)\left(\lambda i-\left(\sigma+\mu\right)z\right)$$

It implies that

$$\frac{dV}{dt}=\left(\frac{s-s^*}{s}\right)\left(\alpha-\beta\left(s-s^*\right)\left(i-i^*\right)-\mu\left(s-s^*\right)\right)+\left(\frac{i-i^*}{i}\right)\left(\beta\left(s-s^*\right)\left(i-i^*\right)-\left(\gamma+\lambda+\mu\right)\left(i-i^*\right)\right)$$

$$+\left(\frac{z-z^*}{z}\right)\left(\lambda\left(i-i^*\right)-\left(\sigma+\mu\right)\left(z-z^*\right)\right)$$

$$\frac{dV}{dt}=\alpha\left(\frac{s-s^*}{s}\right)-\beta\left(\frac{\left(s-s^*\right)^2}{s}\right)\left(i-i^*\right)-\mu\left(\frac{\left(s-s^*\right)^2}{s}\right)+\beta\left(s-s^*\right)\left(\frac{\left(i-i^*\right)^2}{i}\right)-\left(\frac{\left(i-i^*\right)^2}{i}\right)\left(\gamma+\lambda+\mu\right)$$

$$+\lambda\left(\frac{z-z^*}{z}\right)\left(i-i^*\right)-\left(\sigma+\mu\right)\left(\frac{\left(z-z^*\right)^2}{z}\right)$$

$$\frac{dV}{dt}=\alpha\left(\frac{s-s^*}{s}\right)-\left(\frac{\left(s-s^*\right)^2}{s}\right)\left(\beta\left(i-i^*\right)+\mu\right)+\left(\frac{\left(i-i^*\right)^2}{i}\right)\left(\beta\left(s-s^*\right)-\left(\gamma+\lambda+\mu\right)\right)$$

$$-\left(\sigma+\mu\right)\left(\frac{\left(z-z^*\right)^2}{z}\right)+\lambda\left(\frac{z-z^*}{z}\right)\left(i-i^*\right)$$

$$\frac{dV}{dt}=\alpha-\alpha\frac{s^*}{s}-\left(\frac{\left(s-s^*\right)^2}{s}\right)\beta i+\left(\frac{\left(s-s^*\right)^2}{s}\right)\left(\beta i^*-\mu\right)+\left(\frac{\left(i-i^*\right)^2}{i}\right)\beta s-\left(\frac{\left(i-i^*\right)^2}{i}\right)\left(\beta s^*+\left(\gamma+\lambda+\mu\right)\right)$$

$$-\left(\sigma+\mu\right)\left(\frac{\left(z-z^*\right)^2}{z}\right)+\lambda i-\lambda i^*-\frac{\lambda z^* i}{z}+\frac{\lambda z^* i^*}{z} \tag{13}$$

Rearranging the positive and negative terms in (13) leads to

$$\frac{dV}{dt}=X-Y \tag{14}$$

Where

$$X = \alpha + \left(\frac{\left(s-s^*\right)^2}{s}\right)\left(\beta i^* + \mu\right) + \left(\frac{\left(i-i^*\right)^2}{i}\right)\beta s + \lambda i + \frac{\lambda z^* i^*}{z}$$

$$Y = \alpha\frac{s^*}{s} + \left(\frac{\left(s-s^*\right)^2}{s}\right)\beta i + \left(\frac{\left(i-i^*\right)^2}{i}\right)\left(\beta s^* + \left(\gamma+\lambda+\mu\right)\right) + \left(\sigma+\mu\right)\left(\frac{\left(z-z^*\right)^2}{z}\right) + \lambda i^* + \frac{\lambda z^* i}{z}$$

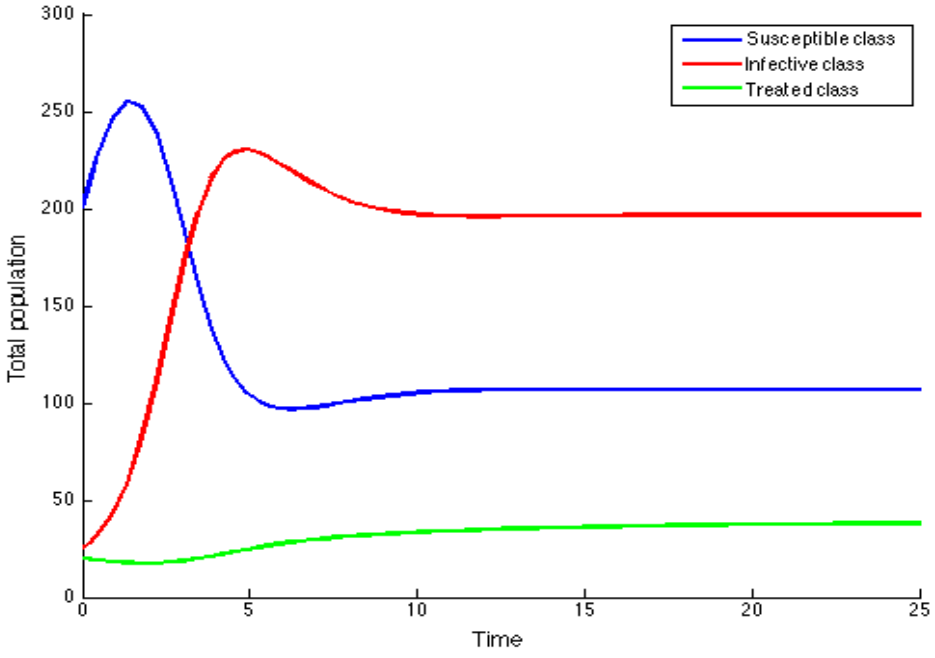


Figure 3. Population dynamics of the HIV/AIDS epidemic model

Hence if $X < Y$ then we obtain $\frac{dV}{dt} \leq 0$. Noting that $\frac{dV}{dt} = 0$ if and only if $s = s^*$, $i = i^*$, $z = z^*$, therefore the largest compact invariant set $\left\{\left(s^*, i^*, z^*\right) \in \Gamma : \frac{dV}{dt} = 0\right\}$ is the singleton $\left\{E^*\right\}$, where E^* is the endemic equilibrium. Hence by the LaSalle's invariant principle, it implies that E^* is globally asymptotically stable in Γ if $X < Y$.

5. Numerical Analysis

We now present numerical simulations for the nonlinear and chaos HIV model using parameter values in Table 1. Some values assigned to the parameters have been derived from epidemiological literature and WHO database while other parameters have been allowed to vary within the possible intervals. All simulations are performed using Matlab and Mathematica. It is worthy to note that although carefully chosen our parameter values are theoretical and may not be biologically realistic.

Table 1. Parameter values used for the HIV/AIDS epidemic model

Variables / Parameters	Description	Values
s	Susceptible Class	200
i	Infective Class	25
z	Treated Class	20
α	Recruitment rate	120
β	Contact rate	0.005
γ	Rate at which infected individuals develop full blown AIDS	0.36
λ	Rate at which infected individuals move to the treated class	0.3
σ	treatment rate	0.01
μ	Natural mortality rate	0143

The results show a sharp decrease in the number of susceptible class corresponding to an increase in the infective class during the initial stages of the epidemic before

settling to a steady state solution (disease-free or endemic equilibrium).

Figure (4a and 4b) below, illustrates the invariance properties of the model. Precisely, for varying initial conditions the model solutions either converges to the disease-free or the endemic state. It can be observed from these figures that for any initial starting point, the solution curves tend to the endemic equilibrium point E_1. The phase portrait in figure (4a) indicates that the trajectories for any initial populations result in a situation where there are no infective individuals, that is, the disease-free equilibrium. From figure (4b) the phase portrait indicates that for any starting initial value, the solution curves tend to the equilibrium E_1. Hence, we infer that the system (6)-(8) is globally stable about the endemic equilibrium point E_1 for the set of parameters chosen.

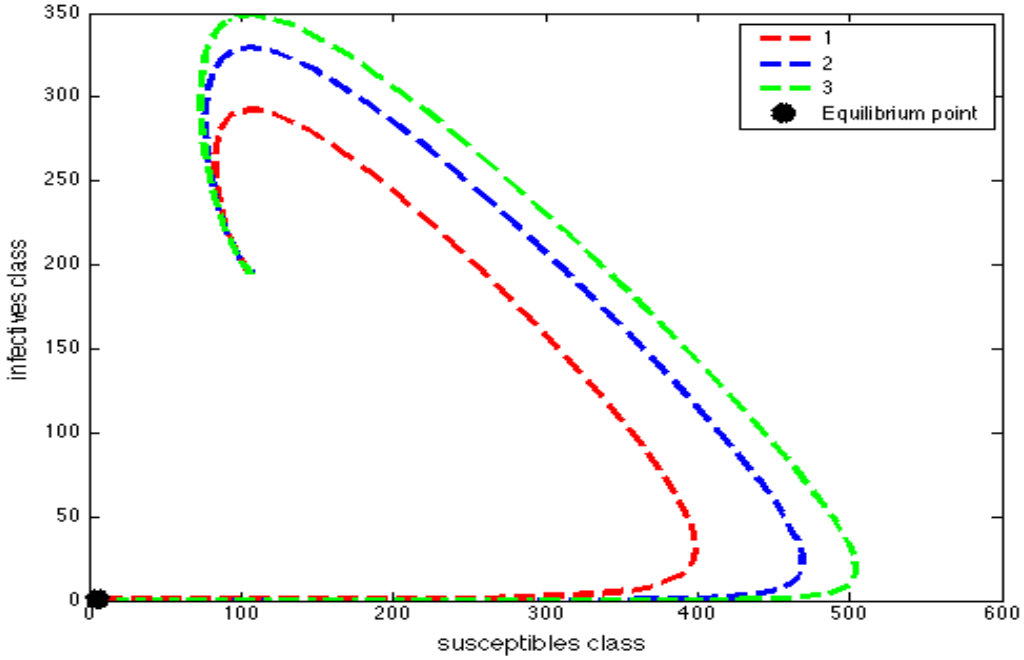


Figure 4a. Phase portrait of the dynamics of susceptibles class and the infective class

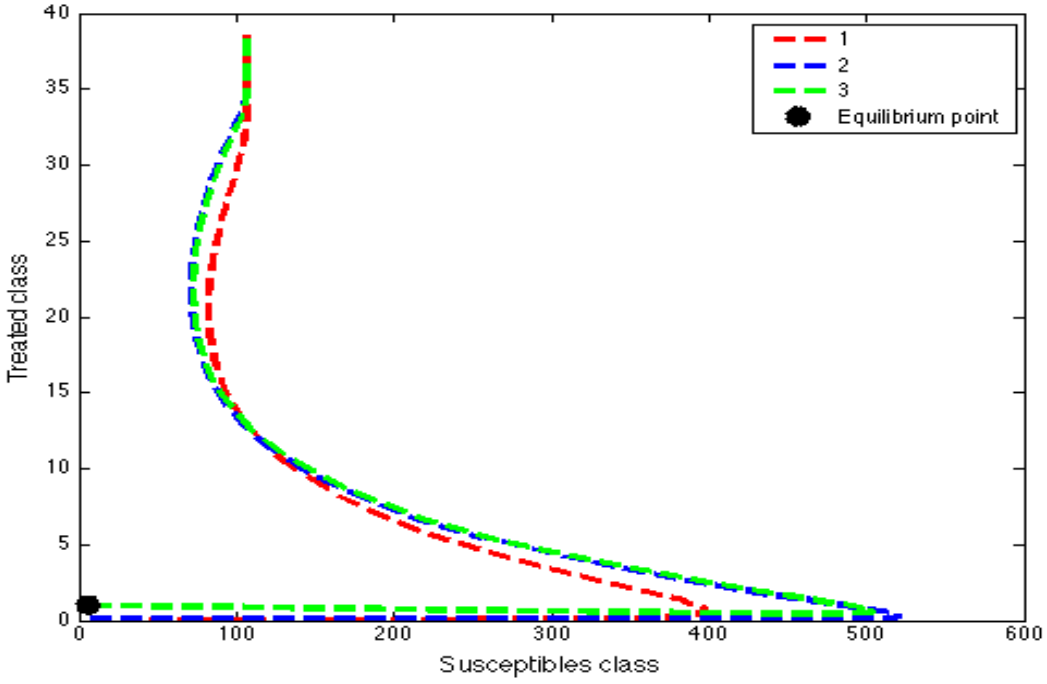


Figure 4b. Phase portrait of the dynamics of susceptibles class and the treated class

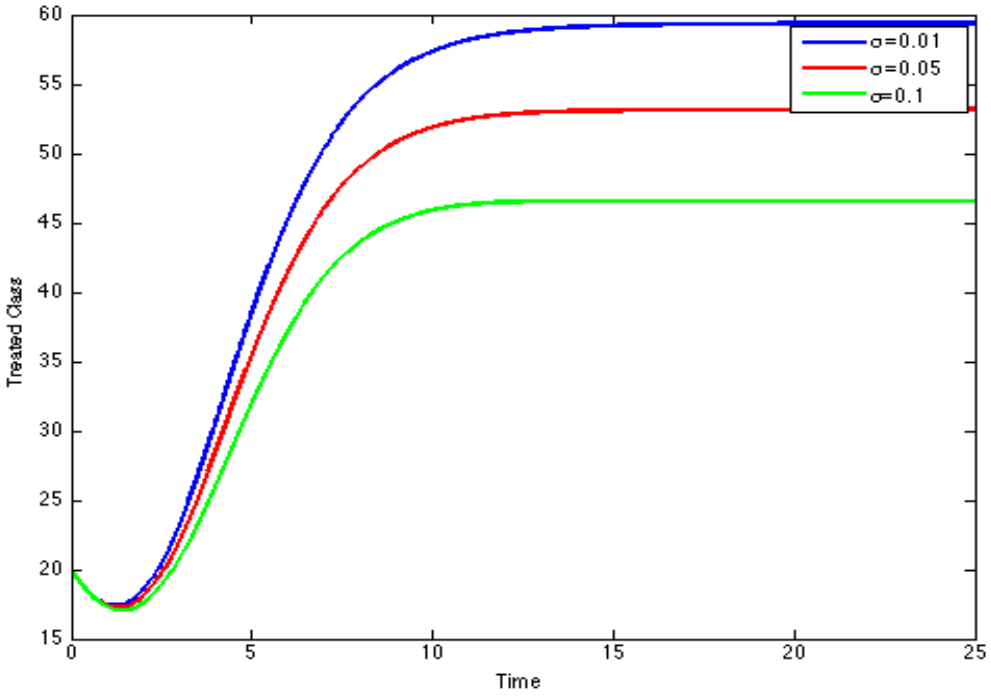


Figure 5. Disease prevalence in treated class as the rate of treatment increases.

To investigate the effect of treatment on the dynamical behaviour of HIV/AIDS infection we simulate the model over different values of the treatment rate $\sigma(0.01, 0.05, 0.1)$. These values depict the result in figure (5) and show that, increasing treatment rate has the effect of reducing the number of secondary cases and subsequently reduce the HIV/AIDS epidemic. The results further show that increasing the treatment rate decreases the severity of the epidemic as seen by gradual decrease in the peaks and time lags between peaks, as σ increases.

6. Conclusions

In this paper, an SITA epidemic HIV/AIDS model with a nonlinear dynamics and chaos is designed and analysed. The model consisted of nonlinear ordinary differential equations for a population with variable size structure and studied the effect of treatment dynamics of HIV/AIDS transmission. Some of the theoretical and epidemiological findings of the study are as follows.

(1) The dynamics behavior of the nonlinear chaos HIV/AIDS treatment model (6)-(8) such as the basic reproduction number R_0 were derived and it was shown that the disease can be eradicated if the basic reproduction is less or equal to unity.

(2) The model (6)-(8) has a locally stable disease-free equilibrium whenever the associated reproduction number is less than unity.

(3) The DFE of the model (6)-(8) is shown to be globally asymptotically stable when $R_0 < 1$.

(4) The endemic equilibrium of the reduced model (6)-(8), is shown to be globally asymptotically stable, when $R_0 > 1$.

To explain that treatment may result in the disease persisting or in the disease dying out, depending on parameter value, we simulated the model over different values of the treatment rate $\sigma(0.01, 0.05, 0.1)$. The results shows that increasing the treatment rate decreases the severity of the epidemic as seen by gradual decrease in the peaks and time lags between peaks, as σ increases.

We conclude that treatment as an intervention strategy can help to contain the HIV/AIDS epidemic but can lead to evolution of drug resistance, which can reverse the benefits of treatment. Although, treatment may lead to evolution of drug resistance, it helps to reduce the proportion of vertically infected and prolongs the lives of all infected individuals.

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