Modelling the Effect of Screening and Treatment on Transmission of HIV/AIDS Infection in a Population

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Abstract
This paper examines the effect of screening and treatment on the transmission of HIV/AIDS infection in a population. A nonlinear mathematical model for the problem is proposed and analysed qualitatively using the stability theory of the differential equations. The effective reproduction number of the normalised model system (3) was obtained by using the next generation operator method. The results show that the disease free equilibrium is locally stable by using Routh Hurwitz criteria at threshold parameter less than unity and unstable at threshold parameter greater than unity. Globally, the disease free equilibrium is not stable due existence of forward bifurcation at threshold parameter equal to unity. Using Lyapunov method, endemic equilibrium is globally stable under certain conditions. However, analysis shows that, screening of unaware HIV infectives and treatment of screened HIV infectives have the effect of reducing the transmission of the disease. Numerical simulation of the model is implemented to investigate the sensitivity of certain key parameters on the voluntary screening and treatment of the screened infectives and full blown AIDS victims.

Keywords
HIV/AIDS, Screening Of HIV, HIV Treatment With ARV, Equilibrium; Stability, Reproduction Number, Sensitivity Index

1. Introduction

The acquired immunodeficiency syndrome (AIDS) emerged in 1981 and has become an alarming sexuality transmitted disease throughout the world. HIV has shown a high degree of prevalence in populations all over the world [1]. It is common to young and adults. Individuals aged 15years and above are the most susceptible group of acquiring infection[2]. This is because they are sexually active and thus capable of reproduction. Routine screening of unaware infectives have now become an integral part of programs in low and middle income countries. People can get HIV tests at a health clinic, at special HIV voluntary counselling and testing (VCT) sites (UNAIDS, 2002). Screening for HIV facilitates immune system monitoring and early management of side infections and sexually transmitted infections (STI’s) which can greatly improve long term health. It also facilitates referral to social and peer support. HIV positive test may help in changing behaviours that may help in transmitting infections. It also enables infectives to be aware and immediately take advantage of antiretroviral therapy (ART) that will help to manage infections and delay the onset of HIV symptoms. Improvement to access to HIV testing and counselling and antiretroviral therapy could significantly reduce infection rates ([3],[4]). Estimates developed through epidemiological modelling suggest that HIV related mortality can be reduced by 20% between 2010 and 2015 if the guidelines for early treatment are broadly implemented The link between infectious diseases and screening must be understood in relation to infectives on the spread of HIV infections. From the initial models of[5, 6, 7], various refinements have been added into modelling frameworks, and specific issues have been addressed by[8, 9, 10, 11].

So far few studies have been developed to analyse mathematically the impact of the screening of unaware infectives on the spread of HIV infections in a homogeneous population. However, none of these studies had considered the aspect of screening and treatment on the transmission of HIV/AIDS infections. It is well known that treatment of screened infectives may also play a major role in the transmission dynamics of the disease in a homogeneous population. [11] presented a theoretical framework for transmission of HIV/AIDS with screening of unaware infectives.[12] established and analyzed a mathematical model of the effect of screening the HIV infection in a homogeneous population with infective immigrants. However, in all the above studies, none of them incorporated the treatment of screened infectives and full blown AIDS patients. In this paper, it is therefore intended to analyze a
model which will incorporate screening and treatment on the transmission of HIV/AIDS infection in a population. This is an extension of the work by[5] and the work by[12], by including the aspect of treatment of screened infectives in a homogeneous population in order to put more knowledge on the long and short term behaviour of the dynamics of the disease transmission and predict whether the disease will disappear (curbing down) or will persist. Thus we study and analyze a non linear mathematical model of the effect of screening and treatment on transmission of HIV/AIDS infection in a population. The model incorporates the assumption that all three infectives (Unaware infectives, Screened infectives and Treated infectives) move to full blown AIDS at different rates.

2. Model Formulation

A non linear mathematical model is proposed and analysed to study the effect of screening and treatment on transmission of HIV/AIDS infection in a population. The proposed model subdivides the population of interest into five sub population compartments depending on the HIV status of individuals. In modelling the dynamics, the population is divided into five subclasses: Susceptibles \( S(t) \), Unaware infectives \( I_1(t) \), Screened infectives \( I_2(t) \), Treated class \( T(t) \) and Full blown AIDS class \( A(t) \). The susceptibles are individuals that have not contracted the HIV infection (HIV negative); the unaware infectives are individuals that have contracted the HIV infection but are not aware of their infection; the screened infectives are individuals that have contracted the HIV infection and are known to be infected after being detected by a medical screening method, treated class (not cured but undergoing treatment) are individuals who use ARV (anti retrovirus) therapy after being screened and become screened infectives or develop full blown AIDS.

In formulating the model, the following assumptions are taken into consideration:

(i) The rate of transmission is direct proportional to the susceptibles population and also to the ratio between the members of infected population to the total population.

(ii) Only screened infectives and full blown AIDS can be treated with ARV therapy (i.e. can move to treated class) at different rates \( \gamma_1 \) and \( \gamma_2 \) respectively.

(iii) Unaware infectives, screened infectives and treated class will move to full blown AIDS at different rates \( \delta_1, \delta_2 \) and \( \sigma \) respectively where \( \sigma < \delta_2 < \delta_1 \).

(iv) Unaware infectives can only move to screened class and full blown AIDS and unaware infective can be screened at a rate \( \theta \).

(v) Unaware infectives, screened infectives and treated class can infect susceptibles class at different rates \( \beta_1, \beta_2 \) and \( \beta_3 \) respectively where \( \beta_3 < \beta_2 < \beta_1 \).

Following mixing of susceptibles, unaware infectives, screened infectives, treated class and full blown AIDS patients, susceptibles acquire HIV infection with the force of infection \( \frac{c_{1}I_1 + c_{2}I_2 + c_{3}A}{N} \). The population considered to be homogeneously mixed and each susceptible individual has equal chance of acquiring HIV infection when comes into contact with infectious individual. The population under study comprises of persons with at least 15 years, so in this case it is assumed that there is no vertical transmission and all recruitment are natural birth without disease, population is variable and all parameters are positive. Taking into account the above considerations, we then have the following schematic flow diagram:

The model is thus governed by the following system of non linear ordinary differential equations:

\[
\begin{align*}
\frac{dS}{dt} &= \lambda N - \frac{c_{1}I_{1}S}{N} - \frac{c_{2}I_{2}S}{N} - \frac{c_{3}A}{N} - \mu S \\
\frac{dI_{1}}{dt} &= \frac{c_{1}I_{1}S}{N} - \frac{c_{2}I_{2}S}{N} - \frac{c_{3}A}{N} - (\theta + \delta_{1} + \mu)I_{1} \\
\frac{dI_{2}}{dt} &= \theta I_{1} - (\gamma_{1} + \delta_{2} + \mu)I_{2} \\
\frac{dA}{dt} &= \gamma_{1}I_{2} + \gamma_{2}A - (\sigma + \mu)A \\
\end{align*}
\]

where

\[
\begin{align*}
S(0) &> 0, \quad I_{1}(0) \geq 0, \quad I_{2}(0) \geq 0, \quad T(0) \geq 0, \quad A(0) \geq 0
\end{align*}
\]

The total population at time \( t \) is given by,

\[
N(t) = S(t) + I_{1}(t) + I_{2}(t) + T(t) + A(t)
\]

This gives

\[
\frac{dN}{dt} = (\lambda - \mu)N - \alpha A
\]

We note that in the absence of the disease, the total population size \( N \) is stationary for \( \lambda = \mu \), declines for \( \lambda < \mu \) and grows exponentially for \( \lambda > \mu \). So we shall assume that mortality rate \( \mu \), will be a function of state variables. For convenience, we analyse our model in terms of proportions of quantities instead of actual populations. This can be done by scaling the population of each class by the total populations. We make the transformation \( s = \frac{S}{N} \),

\[
i_1 = \frac{I_1}{N}, \quad i_2 = \frac{I_2}{N}, \quad h = \frac{T}{N}, \quad a = \frac{A}{N}
\]

in the classes \( S, \ I_1, \ I_2, \ T, \) and \( A \) respectively.
differentiating the fractions with respect to time it is
easier to verify that \( s, i_1, i_2, h, \) and \( a \) satisfy the
following system of non-linear differential equations:

\[
\begin{align*}
\frac{ds}{dt} &= \lambda - c_1 \beta_1 s - c_2 \beta_2 s - c_3 \beta_3 h s - (\lambda - \alpha a) s \\
\frac{di_1}{dt} &= c_1 \beta_1 i s + c_2 \beta_2 i s + c_3 \beta_3 h s - (\theta + \delta_1 + \lambda - \alpha a) i_1 \\
\frac{di_2}{dt} &= \theta i_1 - (\gamma_1 + \delta_2 + \lambda - \alpha a) i_2 \\
\frac{dh}{dt} &= \gamma_1 i_2 + \gamma_2 a - (\sigma + \lambda - \alpha a) h \\
\frac{da}{dt} &= \delta_1 i_1 + \delta_2 i_2 + \sigma h - (\gamma_2 + \alpha + \lambda - \alpha a) a
\end{align*}
\]

where

\[
\begin{align*}
s + i_1 + i_2 + h + a &= 1 \quad s(t) > 0, \quad i_1(t) \geq 0, \quad i_2(t) \geq 0, \quad h(t) \geq 0, \quad a(t) \geq 0, \quad \forall t \geq 0
\end{align*}
\]

Therefore the normalised model system of equations (3) is both mathematically and epidemiologically well posed.

3. Model Analysis

The normalized model system of equations (3) will be analysed qualitatively to get insight into its dynamical features which will give a better understanding of the effects of screening and treatment on the transmission of HIV/AIDS infection in a population. Threshold which governs elimination or persistence of HIV/AIDS will be determined and studied.

3.1. Disease Free Equilibrium (DFE)

The disease free equilibrium denoted by \( E_0 \) of the normalised model system (3) was obtained as

\[
E_0 = (s, 0, 0, 0, 0) = (1, 0, 0, 0, 0)
\]

3.2. The Effective reproduction number \( R_e \)

The effective reproduction number, \( R_e \) of the normalised model system (3) was obtained by using the next generation operator method and is given by

\[
R_e = \frac{c_1 \beta_1 (\gamma_1 + \delta_2 + \lambda) (\sigma + \lambda) + c_2 \beta_2 \theta (\sigma + \lambda) + c_3 \beta_3 \theta \lambda}{(\theta + \delta_1 + \lambda)(\gamma_1 + \delta_2 + \lambda) (\sigma + \lambda)}
\]

3.3. Numerical Sensitivity Analysis
In determining how best to reduce human mortality and morbidity due to AIDS, the sensitivity indices of the effective reproductive number ' \( R_e \)' to the parameters in the model was calculated using approach of [13]. These indices tell us how crucial each parameter is to disease transmission and prevalence and discover parameters that have a high impact on \( R_e \) and should be targeted by intervention strategies. The normalized forward sensitivity index of a variable to a parameter is a ratio of the relative change in the variable to the relative change in the parameter. When a variable is a differentiable function of the parameter, the sensitivity index may be alternatively defined using partial derivatives.

### Table 1. Numerical values of sensitivity indices of \( R_e \)

| Parameter Symbol | Sensitivity Index   |
|------------------|---------------------|
| 1                | \( \beta_1 \) and \( c_1 \) | +0.9283775048 |
| 2                | \( \gamma_1 \)      | -0.6433613445 |
| 3                | \( \lambda \)       | -0.4368749246 |
| 4                | \( \theta \)        | -0.3899159664 |
| 5                | \( \delta_1 \)      | -0.1538461538 |
| 6                | \( \beta_2 \) and \( c_2 \) | +0.04318034906 |
| 7                | \( \beta_3 \) and \( c_3 \) | +0.02844214609 |
| 8                | \( \delta_2 \)      | -0.0000477483009 |
| 9                | \( \sigma \)        | -0.00005677075068 |

**Definition 1:** The normalized forward sensitivity index of a variable ' \( p \) ' that depends differentiable on a parameter ' \( q \) ' is defined as:

\[
X^p_q = \frac{\delta R}{\delta q} \times \frac{q}{R} \tag{6}
\]

As we have an explicit formula for \( R_e \) in equation (6), we derive an analytical expression for the sensitivity of \( R_e \) as \( X^p_q = \frac{\delta R}{\delta q} \times \frac{q}{R_e} \) to each of parameters involved in \( R_e \).

For example the sensitivity indices of \( R_e \) with respect to \( \beta_1 \) and \( \theta \) are given by:

\[
X^p_q = \frac{\delta R}{\delta \beta_1} \times \frac{\beta_1}{R_e} = +0.9283775048 \\
X^p_q = \frac{\delta R}{\delta \theta} \times \frac{\theta}{R_e} = -0.3899159664
\]

respectively. Other indices \( X^p_{\beta_1}, X^p_{\beta_2}, X^p_{\beta_3}, X^p_{\delta_1}, X^p_{\delta_2}, X^p_{\gamma_1}, X^p_{\gamma_2}, X^p_{\sigma}, X^p_{\lambda}, X^p_{c_1}, X^p_{c_2}, X^p_{c_3}, X^p_{\theta}, X^p_{\delta_1}, X^p_{\delta_2}, X^p_{\gamma_1}, X^p_{\gamma_2}, X^p_{\sigma}, X^p_{\lambda}, X^p_{c_1}, X^p_{c_2}, X^p_{c_3}, \) are obtained following the same method and tabulated as follows:

The parameters are ordered from most sensitive to the least.

#### 3.3.1. Interpretation of Sensitivity Indices

From table 1 above, generally it shows that when the parameters \( \beta_1, \beta_2, \beta_3, c_1, c_2, c_3 \) increase while the other parameters remain constant the value of \( R_e \) increases implying that they increase the endemicity of the disease as they have positive indices. When the parameters \( \theta, \gamma_1, \delta_1, \sigma, \lambda \) increase while keeping other parameters constant, the value of \( R_e \) decreases, implying that they decrease the endemicity of the disease as they have negative indices.

The most sensitive parameter is the contact rate of unaware HIV infective \( \beta_1 \), with susceptibles followed by the rate \( \gamma_1 \) at which screened HIV infective are treated, followed by recruitment rate \( \lambda \) and the rate at which unaware HIV infectives \( \theta \) are screened. Other sensitive parameters are the rate at which unaware infectives \( \delta_1 \) develop full blown AIDS followed by the contact rate \( \beta_2 \) of screened HIV infective with susceptibles, the contact rate \( \beta_3 \) of treated infective with susceptibles and the rate \( \delta_2 \) at which screened HIV infectives develop full blown AIDS respectively. The least sensitive parameter is the rate \( \sigma \) at which treated infectives develop full blown AIDS.

#### 3.4. Local Stability of Disease-free Equilibrium

To determine the local stability of disease free equilibrium, the variational matrix \( M_0 \) of the normalised model system (3) corresponding to disease free \( E_0 \) is obtained as

\[
M_0 = \begin{pmatrix}
-\lambda & -c_1\beta_1 & -c_2\beta_2 & -c_3\beta_3 & \alpha \\
0 & r_1 & c_2\beta_2 & c_3\beta_3 & 0 \\
0 & 0 & -\gamma_1 - \delta_2 - \lambda & 0 & 0 \\
0 & 0 & \gamma_1 & -\sigma - \lambda & \gamma_2 \\
0 & \delta_1 & \delta_2 & \sigma & -\gamma - \alpha - \lambda
\end{pmatrix}
\tag{7}
\]

Where \( r_1 = c_1\beta_1 - (\theta + \delta_1 + \lambda) \)

The characteristic equation corresponding to \( M_0 \) is

\[
f(\eta) = (\eta + \lambda)(\eta + \gamma_2 + \alpha + \lambda)(\eta^3 + a_1\eta^2 + a_2\eta + a_3) = 0 \tag{8}
\]

where

\[
a_1 = \theta + \delta_1 + \delta_2 + \gamma_1 + \sigma + 3\lambda - c_1\beta_1
\]
\[ a_2 = \left( \gamma_1 + \delta_2 + \lambda \right) \left( \sigma + \lambda \right) + \left( \theta + \delta_1 + \lambda \right) \left( \gamma_1 + \delta_2 + \sigma + 2 \lambda \right) \]
\[ -c_1 \beta_1 \left( \gamma_1 + \delta_2 + \sigma + 2 \lambda \right) + c_2 \beta_2 \theta \left( \sigma + \lambda \right) \]
\[ a_3 = \left( \theta + \delta_1 + \lambda \right) \left( \gamma_1 + \delta_2 + \lambda \right) \left( \sigma + \lambda \right) \]
\[ -c_1 \beta_1 \left( \gamma_1 + \delta_2 + \lambda \right) \left( \sigma + \lambda \right) + c_2 \beta_2 \theta \left( \sigma + \lambda \right) + c_3 \beta_3 \theta \gamma_1 \]

Thus according to Routh Hurwitz Criteria, \( E_0 \) is locally asymptotically stable when

\[ a_1 > 0, \quad a_2 > 0, \quad a_3 > 0 \quad \text{and} \quad a_4 a_2 > a_3 \]
\[ c_1 \beta_1 \left( \gamma_1 + \delta_2 + \lambda \right) \left( \sigma + \lambda \right) + c_2 \beta_2 \theta \left( \sigma + \lambda \right) \]
\[ + c_3 \beta_3 \theta \gamma_1 < \left( \theta + \delta_1 + \lambda \right) \left( \gamma_1 + \delta_2 + \lambda \right) \left( \sigma + \lambda \right) \]

(9)

The condition (8) is sufficient to satisfy all the equations

\[ a_1 > 0, \quad a_2 > 0, \quad a_3 > 0 \quad \text{and} \quad a_4 a_2 > a_3 \]

(10)

So it is clear that for \( R_c < 1 \) which corresponds to (9), the disease free equilibrium \( E_0 \) is locally asymptotically stable so that the infection does not persist in the population and under this condition the endemic equilibrium \( E^* \) does not exist. It is unstable for \( R_c > 1 \) and then \( E^* \) exists and the infection is maintained in the population.

This situation can also be realised easily when we try to assess the contribution of \( i'_1, i'_2 \) and \( h \) in terms of \( \beta_1, \beta_2 \) and \( \beta_3 \) respectively from equation (5) above.

Let

\[ R_{e1} = \frac{c_1 \beta_1}{\theta + \delta_1 + \lambda} \]

(11)

\[ R_{e2} = \frac{c_2 \beta_2 \theta}{\left( \theta + \delta_1 + \lambda \right) \left( \gamma_1 + \delta_2 + \lambda \right)} \]

(12)

\[ R_{eh} = \frac{c_3 \beta_3 \theta \gamma_1}{\left( \theta + \delta_1 + \lambda \right) \left( \gamma_1 + \delta_2 + \lambda \right) \left( \sigma + \lambda \right)} \]

(13)

where

\[ R_c = R_{e1} + R_{e2} + R_{eh} \]

(14)

From the equations (11)-(13) above, it is clear that

\[ R_{e1} > R_{e2} > R_{eh} \]

which implies that unaware infectives \( i'_1 \) are the ones contributing the most on the transmission of the HIV/AIDS infection followed by screened infectives \( i'_2 \) which keeps the disease endemic (i.e. \( R_c > 1 \)) in the population via \( c_1 \beta_1 \) and \( c_2 \beta_2 \) respectively compared to treated individuals \( h \) via \( c_3 \beta_3 \). In the absence of infection, the population size approaches the steady state \( n = 1 \) for normalised model system (3) and \( \frac{\lambda N}{\mu} \) for the original model system (1). However when the disease remain endemic the disease induced deaths reduce the equilibrium population size from \( n = 1 \) to \( s^* \).

3.5. Endemic Equilibrium (EEP) and Local Stability

The endemic equilibrium of the normalised model system (3) is given by \( E^* \left(s^* (t), i'_1 (t), i'_2 (t), h^* (t), a^* (t) \right) \)

which exists for \( R_c > 1 \). \( s^* (t), i'_1 (t), h^* (t) \) and \( a^* (t) \) are expressed in terms of \( i'_1 (t) \) which satisfies the following relations:

\[ s^* = \frac{\lambda - \left( \theta + \delta_1 + \lambda - aa^* \right) i'_1}{\lambda - aa^*} \]

(15)

\[ i'_2 = \frac{\theta}{\gamma_1 + \delta_2 + \lambda - aa^*} i'_1 \]

\[ h^* = \frac{\theta \theta K_1 + \gamma_2 \left( \gamma_1 + \delta_2 + \lambda - aa^* \right) \left( \sigma + \lambda - aa^* \right) + \delta \theta \left( \sigma + \lambda - aa^* \right) + \sigma \gamma_1 i'_1}{\left( \gamma_1 + \delta_2 + \lambda - aa^* \right) K_1} \]

\[ a^* = \frac{\sigma \left( \delta \left( \gamma_1 + \delta_2 + \lambda - aa^* \right) + \delta \theta \left( \sigma + \lambda - aa^* \right) + \sigma \gamma_1 \right)}{\left( \gamma_1 + \delta_2 + \lambda - aa^* \right) K_1} \]

and \( i'_1 (t) \) is the solution of the following quadratic polynomial

\[ Gi'^2 + Hi'^* + K = 0 \]

(16)

where

\[ K = 0, \quad G = \left( \theta + \delta_1 + \lambda - aa^* \right) \left( \gamma_1 + \delta_2 + \lambda \right) \left( \sigma + \lambda \right) R_0 - aa^* K_2 \]

\[ H = \left( \theta + \delta_1 + \lambda - aa^* \right) \left( \gamma_1 + \delta_2 + \lambda - aa^* \right) \left( \sigma + \lambda - aa^* \right) K_1 \]

\[ -\lambda \left( \theta + \delta_1 + \lambda \right) \left( \gamma_1 + \delta_2 + \lambda \right) \left( \sigma + \lambda \right) R_0 - aa^* K_2 \]

\[ K_1 = \left( \gamma_1 + \delta_2 + \lambda - aa^* \right) \left( \sigma + \lambda - aa^* \right) - \gamma_2 \sigma, \]

\[ K_2 = c_1 \beta_1 \left( \gamma_1 + \delta_2 + \lambda + 2 \lambda - aa^* \right) + c_2 \beta_2, \]

\[ K_3 = c_3 \beta_3 \theta \gamma_1 \left( \gamma_1 + \delta_2 + \lambda - aa^* \right) \left( \sigma + \lambda - aa^* \right) + \delta \theta \left( \sigma + \lambda - aa^* \right) + \sigma \gamma_1 \]

From equation (16) it follows that

\[ i'^* = 0 \]

which corresponds to the disease free equilibrium already being discussed.

\[ \frac{\lambda \left( \theta + \delta_1 + \lambda \right) \left( \gamma_1 + \delta_2 + \lambda \right) \left( \sigma + \lambda \right) R_0 - aa^* K_2}{G} \]

\[ i'_1 = \frac{- \left( \theta + \delta_1 + \lambda - aa^* \right) \left( \gamma_1 + \delta_2 + \lambda - aa^* \right) \left( \sigma + \lambda - aa^* \right) K_1}{\left( \theta + \delta_1 + \lambda \right) \left( \gamma_1 + \delta_2 + \lambda \right) \left( \sigma + \lambda \right) R_0 - aa^* K_2 + K_1 \}

(17)

corresponding to unique endemic equilibrium \( E^* \) when \( R_c > 1, \ H < 0 \) and \( G > 0 \).

From this result we state the following theorem which will be proved by using bifurcation diagram and Centre Manifold theorem.

**Theorem 3.1.** The unique endemic equilibrium \( E^* \) exists
if \( G > 0, H < 0 \) and \( R_e > 1 \), and is locally stable if \( R_e > 1 \) and unstable if \( R_e < 1 \).

### 3.5.1. Determination of Forward or Backward Bifurcation

From equation (16), it follows that there is no backward bifurcation since the value of \( K = 0 \), hence no multiple equilibria. Therefore existence of unique endemic equilibrium which is locally stable for \( R_e > 1 \) and unstable if \( R_e < 1 \) was explored by a forward bifurcation diagram obtained when a graph of proportion of unaware infective \( I_1 \) against reproduction number \( R_e \) was sketched as shown below:

![Figure 2](image-url)

**Figure 2.** The figure of proportion of unaware infective \( I_1 \) versus effective reproduction number \( R_e \)

- Figure 2 exhibit a forward bifurcation when:
  - \( \alpha = 0.5 \), \( \beta_1 = 0.86 \), \( \beta_2 = 0.05 \), \( \beta_3 = 0.01 \),
  - \( \delta_1 = 0.02 \), \( \delta_2 = 0.01 \), \( \sigma = 0.001 \), \( \gamma_1 = 0.99 \), \( \gamma_2 = 0.1 \), \( \theta = 0.6 \), \( c_1 = 3 \), \( c_2 = 2 \), \( c_3 = 1 \), \( a^* = 0.0002 \).

In figure 2 above, the two equilibrium points exchange stabilities depending on the value of \( R_e \). A transcritical/forward bifurcation in the equilibrium points occur at \( R_e = 1 \). If \( R_e < 1 \), no biologically meaningful endemic equilibrium solution exists and the disease free equilibrium is the only local attractor. But if \( R_e > 1 \), the endemic equilibrium exists and it is the only local attractor while the disease free equilibrium is a saddle point. Thus there is a forward bifurcation because in the neighbourhood of the bifurcation point, the endemic disease prevalence (proportion of unaware infectives \( I_1^* \)) is an increasing function of \( R_e \) as shown in equation (17).

The local asymptotic stability of endemic equilibrium is analyzed by using the Centre Manifold theory\(^{[14]}\) and shows that it is stable under certain conditions. The normalised model system (3) shows that it will exhibit a backward bifurcation which occurs at \( R_e = 1 \) under certain condition otherwise it will exhibit a forward bifurcation at \( R_e = 1 \) as shown in figure 2 and is locally stable.

### 3.6. Global Stability of Endemic Equilibrium

**Theorem 3.3** If \( R_e > 1 \) the endemic equilibrium \( E^* \) of the normalised model system (3) is globally asymptotically stable.

**Proof** To establish the global stability of endemic equilibrium \( E^* \) we construct the following positive Lyapunov function \( V \) as follows:

\[
V(s, i_1, i_2, h, a) = \left(s - s^* \log \frac{s}{s^*}\right) + \left(\frac{i_1 - i_1^*}{i_1^*}\right) + \left(\frac{i_2 - i_2^*}{i_2^*}\right) - \left(h - h^* \log \frac{h}{h^*}\right) + \left(a - a^* \log \frac{a}{a^*}\right)
\]

(18)

By direct calculation of the derivative of \( V \) along the solutions of (3) we have

\[
\frac{dV}{dt} = F - Y
\]

(20)

where

\[
F = \left\{c_1\beta_1 i_1 + c_2\beta_2 i_2 + c_3\beta_3 h + \lambda + aa\right\}\left(s - s^*\right)^2 \left(i_1 - i_1^*\right)^2 \left(i_2 - i_2^*\right)^2 + \frac{c_1\beta_3 s + aa}{i_1 i_2^*} + \frac{c_2\beta_3 h^* + c_3\beta_3 h^*}{h} + \frac{c_2\beta_3 i_2^* s + c_2\beta_2 i_2^* s + c_3\beta_3 h^*}{h} + \frac{c_3\beta_3 h^*}{a} + \frac{c_2\beta_3 h^* s + c_2\beta_2 h^* + c_3\beta_3 h s}{a} + \frac{c_2\beta_3 h^* s + c_2\beta_2 h^* + c_3\beta_3 h s}{a} + \frac{c_2\beta_3 h^* s + c_2\beta_2 h^* + c_3\beta_3 h s}{a} + \frac{c_2\beta_3 h^* s + c_2\beta_2 h^* + c_3\beta_3 h s}{a}
\]

\[
Y = \left\{h - h^* \log \frac{h}{h^*}\right\} + \left(a - a^* \log \frac{a}{a^*}\right)\left(i_1^* + i_2^* + \frac{h}{h^*} + \frac{a}{a^*}\right)
\]
and

\[ Y = \frac{c_i \beta_i i_s + c_j \beta_j j_s + c_k \beta_k k_s + \alpha a} {s} + \frac{\sigma + \alpha a} {h} \left( (\beta_1 + \lambda) + (\beta_2 + \lambda) \right) \left( i_1 - i_s \right) \]

\[ + \frac{\gamma_1 + \beta_2 + \lambda + \alpha a} {i_2} \left( i_2 - i_2 \right) \]

\[ + \frac{\gamma_2 + \alpha + \alpha a} {a} \left( a - a \right) \]

\[ + \frac{c_3 \beta_3 i_s s + c_2 \beta_2 i_s s + c_3 \beta_3 h s + c_3 \beta_3 h s} {s} \]

\[ + \frac{\theta i_1} {i_2} \]

\[ + \frac{\gamma_1 + \gamma_2 a} {h} \]

\[ + \frac{\delta_1 i_1 + \delta_2 i_2 + \sigma h} {a} \]

Thus from equation (20), if \( F < Y \) then \( \frac{dV}{dt} < 0 \). Also it follows that

\[ \frac{dV}{dt} = 0 \] if and only if \( s = s^* \), \( i_1 = i_s \), \( i_2 = i_s \), \( h = h^* \), \( a = a^* \).

Therefore the largest compact invariant set in \( \left( s^*, i_1^*, i_2^*, h^*, a^* \right) \in \Omega : \frac{dV}{dt} = 0 \) is the singleton \( \{ E^* \} \).

\[ E^* \] is the endemic equilibrium of the normalised model system (3). By LaSalles’s invariant principle, then it implies that \( E^* \) is globally asymptotically stable in \( \Omega \) if \( F < Y \).

3.7. Model with Screening and Treatment When Treated HIV Infectives do not Transmit the Infection in a Population \( \left( \gamma_1 \neq 0, \gamma_2 \neq 0, \beta_3 = 0 \right) \)

In this case, we consider the situation where the treated HIV infectives take the preventive measures and change their behaviour so as not to transmit the infection by sexual interaction in the community \( \left( \beta_3 = 0 \right) \). Thus the infection is transmitted by those infectives who are not aware of their infection and those who are screened only (not using ARV). The basic reproduction number in this case is given by

\[ R_{e1} = \frac{c_1 \beta_1 \left( \gamma_1 + \delta_2 + \lambda \right) + c_2 \beta_2 \theta \left( \sigma + \lambda \right)} {\left( \theta + \delta_1 + \lambda \right) \left( \gamma_1 + \delta_2 + \lambda \right) \left( \sigma + \lambda \right)} \]

and we note that \( R_{e1} < R_e \). We also note that \( R_e \to R_{e1} \) as \( \beta_2 \to 0 \). Therefore we can conclude that the endemicity of the infection is reduced when the treated HIV infectives present a positive attitude towards preventive measures and do not take part in the transmission of the disease.

3.8. Model with Screening and Treatment When Both Screened and Treated HIV Infectives Respectively do not Transmit the Infection in a Population \( \left( \theta \neq 0, \beta_2 = 0, \gamma_1 \neq 0, \gamma_2 \neq 0, \beta_3 = 0 \right) \)

In this case, we consider the situation where both the screened and treated HIV infectives respectively take the preventive measures and change their behaviours so as not to transmit the infection by sexual interaction in the community \( \left( \beta_2 = 0, \beta_3 = 0 \right) \). Thus the infection is transmitted only by those infectives who are not aware of their infections. The basic reproduction number in this case is given by

\[ R_{e2} = \frac{c_2 \beta_2 \left( \gamma_1 + \delta_2 + \lambda \right) + c_2 \beta_2 \theta \left( \sigma + \lambda \right)} {\left( \theta + \delta_1 + \lambda \right) \left( \sigma + \lambda \right)} \]

and we note that \( R_{e2} < R_{e1} < R_e \) and \( R_e \to R_{e2} \) as \( \beta_2 \to 0 \) and \( \beta_1 \to 0 \). Therefore we can conclude that the endemicity of the infection is reduced when screened and treated HIV infectives both reflect a positive attitude towards preventive measures and do not take part in the transmission of the disease.

3.9. Model in the Absence of Treatment (Screening only, \( \gamma_1 = 0 \) and \( \gamma_2 = 0 \))

Here we consider the situation where there is no ARV treatment for the screened HIV infectives \( \left( \gamma_1 = 0 \right) \) and AIDS population who are not treated \( \left( \gamma_2 = 0 \right) \). The normalized model system (3) for this case is reduced to the group of proportion of susceptibles, unaware infectives, screened infectives and AIDS class population. As there is no treatment, i.e. \( \gamma_1 \to 0 \) and \( \gamma_2 \to 0 \) then \( h \to 0 \) we obtain the basic reproduction number

\[ R_{e3} = \frac{c_1 \beta_1 \left( \delta_2 + \lambda \right) + c_2 \beta_2 \theta \left( \sigma + \lambda \right)} {\left( \theta + \delta_1 + \lambda \right) \left( \delta_2 + \lambda \right)} \]

We note that \( R_e < R_{e3} \) and \( R_e \to R_{e3} \) as \( \gamma_1 \to 0 \) and \( \gamma_2 \to 0 \). Therefore we can conclude that the endemicity of the infection in this case increases in the absence of treatment with ARV for the screened infectives and AIDS population.

3.10. Model in the Absence of Screening \( \left( \theta = 0 \right) \)

Here we consider the situation without screening of unaware infectives. In this case the normalised model system (3) is reduced to the group of proportion of susceptibles,
unaware infectives and AIDS class population. As there is no screening of unaware infectives, then \( \theta = 0 \). This implies that \( i_2 \to 0 \) and \( h \to 0 \) in which case we obtain the basic reproduction number as

\[
R_{e4} = \frac{c_i \beta_i}{\delta_1 + \lambda}.
\]

We note that \( R_e < R_{e3} < R_{e4} \) and \( R_{e3} \to R_{e4} \) as \( \theta \to 0 \). Therefore we can conclude that the endemicity of the infection in this case increases in the absence of detection of unaware infectives by screening.

By analysing the four epidemiological situations discussed above, it may be concluded that in the presence of screening and treatment, if both the screened and treated HIV infectives decide to take preventive measures and do not transmit the infection, the disease will tend to the endemic state if both the screening and treatment rates are small. If the annual screening and treatment rates are very high (say 99% of the initial population), the disease may tend to disease free equilibrium point. However if screened and treated HIV infectives also contribute to the transmission of the disease, then even if both screening and treatment rates are very high, the disease is set up among the population as the system continues towards asymptotically stable endemic equilibrium point. Analysis also shows that the endemicity of the disease is reduced by screening of unaware HIV infectives and treatment of the screened HIV infectives in the population.

4. Numerical Simulations

![Numerical Simulations](image-url)
In order to illustrate the analytical results of the study, numerical simulations of the normalised model system (3) are carried out using the set of estimated parameter values below:

\[ \beta_1 = 0.86, \quad \beta_2 = 0.15, \quad \beta_3 = 0.1, \quad \alpha = 1, \quad \lambda = 0.5, \]
\[ \delta_1 = 0.2, \quad \delta_2 = 0.01, \quad \sigma = 0.001, \quad \gamma_1 = 0.99, \quad \gamma_2 = 0.1, \]
\[ c_1 = 3, \quad c_2 = 2, \quad c_3 = 1 \quad (21) \]

However these parameters may (or may not) be biologically feasible.

Figures 4.1 below show the proportion of HIV infective populations (unaware, screened and treated) and proportion of AIDS infectives all plotted against the proportion of susceptible population. This shows the dynamic behaviour of the endemic equilibrium of the normalised model system (3) using the parameter values in equation (21) for different initial starting values in four cases as shown below.

1. \( s(0) = 0.5, \quad i_1(0) = 0.3, \quad i_2(0) = 0.12, \quad h(0) = 0.07, \quad a(0) = 0.01 \)
2. \( s(0) = 0.6, \quad i_1(0) = 0.1, \quad i_2(0) = 0.08, \quad h(0) = 0.03, \quad a(0) = 0.02 \)
3. \( s(0) = 0.75, \quad i_1(0) = 0.45, \quad i_2(0) = 0.25, \quad h(0) = 0.1, \quad a(0) = 0.05 \)
4. \( s(0) = 0.63, \quad i_1(0) = 0.5, \quad i_2(0) = 0.126, \quad h(0) = 0.008, \quad a(0) = 0.002 \)

The equilibrium points of the endemic equilibrium \( E^* \) was obtained as

\[ s^* = 0.4607, \quad i_1^* = 0.2173, \quad i_2^* = 0.0878, \quad h^* = 0.1762, \quad a^* = 0.0180 \]

It is seen from these figures that for any starting initial value, the solution curves tend to the equilibrium \( E^* \). Therefore we conclude that the normalised model system (3) is globally stable about this endemic equilibrium point \( E^* \) for the estimated parameters above.

Figure 4.2 below show the variation of the proportion of total population in all classes

It is seen that the proportion of susceptible population decreases with time and then reaches its equilibrium position. This is due to treatment with ARV. Therefore infection becomes less endemic in the population. Initially proportion of unaware infectives increases but due to the increase in screening and treatment, the rates \( \theta \) and \( \gamma_1 \) respectively, decreases then reaches its equilibrium position. This ultimately leads to the decrease of the proportion of AIDS infectives.

In figures 4.3-4.4, the variation of proportions of susceptible, unaware, screened and treated HIV infective populations and proportion of AIDS patient population for different rates of screening is shown.
It is seen that as the screening rate increases, the proportion of susceptible population increases (Figure 4.3). From Figure 4.4 (i), it is seen that the unaware HIV infectives become aware of their infection which results in the decrease of the proportion of unaware infectives population and increase in the proportions of screened and treated HIV infectives (Figure 4.4 (ii) and (iii)) respectively. This ultimately leads to decrease of the proportion of AIDS infected population as shown in Figure 4.4 (iv) above. Thus as the screening rate becomes zero, the unaware infectives will continue maintaining sexual relationships in the community leading to the increase in the proportion of AIDS infective population.

Figures 4.5 - 4.6 show the variation of proportions of susceptibles, unaware, screened and treated HIV infectives and proportion of AIDS patients population for different rates of treatment.
It is seen that as the treatment rate increases, the proportion of susceptible population increases (Figure 4.5 (i)). The screened HIV infectives decided to use ARV treatment which results in the decrease of the proportion of screened infective population (Figure 4.5 (iii)) leading to the increase in the proportion of the treated HIV infective population (Figure 4.5 (iv)). This ultimately leads to decrease the proportion of unaware infective population (Figure 4.5 (ii)) leading to decrease the proportion of AIDS patient’s population (Figure 4.6). Thus as the treatment rate becomes zero, the proportion of unaware and screened HIV infectives population increases (Figure 4.5 (ii) and (iii)) respectively leading to the increase in proportion of AIDS patients population (Figure 4.6).

Figures 4.7 shows the role of contact rates $\beta_2$ and $\beta_3$ of the screened and treated HIV infectives respectively.
Figure 4.7(iv). Variation of proportion of AIDS patients for different values of $\beta_2$ and $\beta_3$ respectively.

Figure 4.8(i). Variation of proportion of susceptibles for different values of $\delta_1$, $\delta_2$ and $\sigma$.

Figure 4.8(ii). Variation of proportion of unaware infectives for different values of $\delta_1$, $\delta_2$ and $\sigma$.

Figure 4.8(iii). Variation of proportion of screened infectives for different values of $\delta_1$, $\delta_2$ and $\sigma$.

Figure 4.8(iv). Variation of proportion of treated infectives for different values of $\delta_1$, $\delta_2$ and $\sigma$.

Figure 4.9. Variation of proportion of AIDS patients population for different values of $\delta_1$, $\delta_2$ and $\sigma$. 
It can be observed that as screened and treated HIV infectives choose to participate in sexual interactions without exposing themselves, the proportion of unaware HIV infective population increases which in turn increases the proportion of AIDS patients population. When screened and treated HIV infectives do not take part in sexual interactions, means that $\beta_2 = 0$ and $\beta_3 = 0$ respectively and the proportion of unaware HIV infectives decreases leading to the decrease of proportion of AIDS patients population. From these figures, it is also clear that in the absence of screening of unaware HIV infectives and treatment of screened HIV infectives, the higher proportion of unaware and screened HIV infectives respectively leads to increase of the proportion of AIDS patients population. Thus to keep the transmission of the epidemic under control, the screened and treated HIV infectives individuals should be counselled to either abstain from sexual interaction or use preventive measures like condoms to stop transmitting HIV infection.

Figures 4.8 - 4.9 show the variations of proportions of susceptibles, HIV infective classes and AIDS patients population with time for different values of movement rates $\delta_1, \delta_2$ and $\sigma$ of unaware, screened and treated infectives respectively.

It is seen that as the movement rates from infective classes increases, proportions of all infective classes populations decreases (Figures 4.8 (ii), (iii) and (iv)) which in turn increases the proportion of AIDS patients population (Figure 4.9) and also increases the proportion of susceptible population (Figure 4.8 (i)).

Figures 4.10 show the variation of proportion of AIDS patients population for different values of disease induced death rate $\alpha$ and recruitment rate $\lambda$ of susceptibles respectively.

It is observed from the figures that as $\alpha$ increases the proportion of AIDS patients population decreases and it is also observed that as the recruitment rate increases, the proportion of AIDS patient population increases.

5. Discussion and Conclusions

A non linear mathematical model has been proposed and analyzed to study the effect of screening of unaware HIV infectives and treatment of screened HIV infectives on the transmission of HIV/AIDS infection in a population. The disease free and endemic equilibria were obtained and their stabilities investigated. The model showed that the disease free equilibrium is locally stable by using Routh Hurwitz criteria at threshold parameter less than unity and unstable at threshold parameter greater than unity, but globally the disease free equilibrium is not stable due existence of forward bifurcation at threshold parameter equal to unity. Also the model analysis showed the existence of unique endemic equilibrium that is locally stable under certain conditions when the threshold parameter exceeds unity due to existence of forward bifurcation at threshold parameter equal to unity. Using Lyapunov method, endemic equilibrium is globally stable under certain conditions. A sensitivity analysis shows that $\beta_1$ is the most sensitive parameter on $R_0$ and the least is $\sigma$. A numerical study of the model was carried out to see the effect of key parameters on the transmission of HIV/AIDS infections. The analysis shows that the screening of unaware HIV infectives and treatment of screened HIV infectives have the effect of reducing the transmission of the disease. It is observed that when the screened infectives and treated infectives do not participate in the transmission of the infection, the AIDS population is significantly reduced in comparison to the case where there is no screening and treatment. It is found that the disease becomes more endemic in the absence of screening and treatment and consequently the AIDS population increase. This has a consequence of people not knowing that they are infected and still practice sexual relationships without taking precautions. Screening makes people aware of their infection and reduce their viral load by using ARV
treatment and consequently AIDS patients population decreases. This is due to the fact that on being aware of their infection and using ARV, people either abstain to interact sexually or use preventive measures like using condoms and by changing their behaviour which results in the decline of AIDS epidemic.

Based on the results of the study, we conclude that the most effective way to reduce the transmission of HIV and AIDS epidemic infection is to educate people to go for voluntary HIV screening and become aware of their HIV status and if an individual finds himself/herself HIV positive, then he/she has to be willing to undergo ARV treatment and therapy so as to reduce the viral load hence prolonging life. Furthermore people should be educated to be aware of the consequences of practising unsafe sex and other preventive measures against the infection. If the population shows positive attitude towards voluntary screening, treatment with ARV and preventive procedures, then the transmission of the disease can be controlled. Therefore the national HIV/AIDS control programs for all developing countries should increase the education programs on the importance of voluntary HIV screening, ARV treatment and preventive procedures to the community at all social classes especially lower classes and high risk groups so that the transmission of the disease can be controlled. Finally, more HIV/AIDS centres for voluntary screening and ARV treatment should be established across each country to ensure that more people have access to the facilities hence reducing the transmission of HIV and therefore reduce the AIDS epidemic.

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