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Stability Analysis of an HIV/AIDS Epidemic Model with Screening

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Abstract. We present a non-linear mathematical model which analyzes the spread and control of HIV (human immunodeficiency virus)/AIDS(acquired immunodeficiency syndrome). We divide the population into four subclasses one of them is the susceptible population S and the others are HIV infectives (HIV positives that do not know they are infected) I_1, HIV positives that know they are infected (by away of medical screening or other ways) I_2 and that of AIDS patients A. Both the disease free equilibrium and the infected equilibrium are found and their global stability is investigated. The model is analyzed by using the basic reproduction number R_0. If $R_0 < 1$, the disease free equilibrium point is globally asymptotically stable, whereas the unique positive infected equilibrium point is globally asymptotically stable when $R_0 > 1$. Also we study the effect of screening of unaware infectives on the spread of HIV disease and study the situation when aware HIV infectives take preventive measures and change their behavior so that they do not spread the

infection in the community.

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1 Introduction

AIDS stands for acquired immunodeficiency syndrome, a disease that makes it difficult for the body to fight off infectious diseases. The human immunodeficiency virus known as HIV causes AIDS by infecting and damaging the $CD4^+$ T-cells, which are a type of white blood cells in the body's immune (infection-fighting) system that is supposed to fight off invading germs.In a normal healthy individual's peripheral blood, the level of $CD4^+$ T-cells is between 800 and 1200 / mm^3 and once this number reaches 200 or below in an HIV infected patient, the person is classified as having AIDS. HIV can be transmitted through direct contact with the blood or body fluid of someone who is infected with the virus. That contact usually comes from sharing needles or by having unprotected sex with an infected person. An infant could get HIV from a mother who is infected.Although AIDS is always the result of an HIV infection, not everyone with HIV has AIDS. In fact, adults who become infected with HIV may appear healthy for years before they get sick with AIDS.

The study of HIV/AIDS transmission dynamics has been of great interest to both applied mathematicians and biologists due to its universal threat to humanity. Mathematical models have become important tools in analyzing the spread and control of HIV/AIDS as they provide short and long term prediction of HIV and AIDS incidences. Many models available in the literature represent the dynamics of the disease by systems of nonlinear differential equations. Several investigations have been conducted to study the dynamics of HIV/AIDS [1,3-5,8-18]. In particular, Srinivasa Rao [18] presented a theoretical framework for transmission of HIV/AIDS epidemic in India. It is pointed out that the screening of infectives has substantial effect on the spread of AIDS. Naresh et al. [17] have proposed a nonlinear model to study the effect of screening of unaware infectives on the spread of HIV/AIDS in a homogenous population with constant immigration of susceptibles. They have shown that screening of unaware infectives has the effect of reducing the spread of AIDS epidemic. Cai et al. [4] investigated an HIV model with treatment, they established the model with two infective stages and proved that the dynamics of the spread of the disease are completely determined by the basic reproduction number.

One of the most important concerns about any infectious disease is its ability to invade a population. Many epidemiological models have a disease free equilibrium (DFE) at which the population remains in the absence of disease. These models usually have a threshold parameter, known as the basic reproduction number, R_0, such that if $R_0 < 1$, then the DFE is locally asymptotically stable, and the disease cannot invade the population, but if $R_0 > 1$, then the DFE is unstable, (i.e If R_0 is 1 or greater, an epidemic is expected, and if R_0 ,less than 1, then infection is expected to die out.)

In this paper, we introduce a nonlinear model to study the effecct of screening of infectives that are not aware of their infection on the long term dynamics of the disease. The paper is organized as follows: In the next section, the model is presented and the basic reproduction number is obtained. In section 3, we investigate the stability of the DFE and the endemic equilibrium. In section 4, we analyze the model when aware HIV infectives do not spread the disease. Section 5, is dedicated for the analysis of the model without screaning. Section 6 presents a numerical simulation of the model systems followed by a conclusion in section 7

2 Mathematical model and the basic reproduction number

In deriving our model equations, we divided the population into four subclasses, the susceptible $S(t)$, the infectives that do not know they are infected $I_1(t)$, the infectives that know they are infected $I_2(t)$ (by way of medical screening or otherwise) and that of the AIDS population $A(t)$.

Taking the above considerations, the model dynamics is assumed to be governed by the following system of ordinary differential equations:

$$\begin{aligned} \frac{dS}{dt} &= Q_0 - (\beta_1 I_1 + \beta_2 I_2)S - \mu S \\ \frac{dI_1}{dt} &= (\beta_1 I_1 + \beta_2 I_2)S - (\theta + \mu + \delta)I_1 \\ \frac{dI_2}{dt} &= \theta I_1 - (\mu + \delta)I_2 \\ \frac{dA}{dt} &= \delta(I_1 + I_2) - (\mu + d)A \end{aligned} \tag{2.1}$$

where

Q_0 = constant rate of immigration of susceptibles,

$\beta_i (i = 1, 2)$ are the per capita contact rates for susceptibles individuals with (unaware , aware) infectives respectively,

μ = the natural mortality rate unrelated to AIDS,
θ = the rate of unaware infectives to become aware infectives by screening,
δ = the rate by which types of infectives develop AIDS,
d = the AIDS related death rate.

Since the variable A of system (2.1) does not appear in the first three equations, in the subsequent analysis, we only consider the subsystem:

$$\begin{aligned} \frac{dS}{dt} &= Q_0 - (\beta_1 I_1 + \beta_2 I_2)S - \mu S, \\ \frac{dI_1}{dt} &= (\beta_1 I_1 + \beta_2 I_2)S - (\theta + \mu + \delta)I_1, \\ \frac{dI_2}{dt} &= \theta I_1 - (\mu + \delta)I_2. \end{aligned} \tag{2.2}$$

It follows from system (2.2) that

$$\begin{aligned} \frac{dS}{dt} + \frac{dI_1}{dt} + \frac{dI_2}{dt} &= Q_0 - \mu(S + I_1 + I_2) - \delta(I_1 + I_2) \\ &\leq Q_0 - \mu(S + I_1 + I_2) \end{aligned}$$

Hence

$$\lim_{t\to\infty} \sup(S + I_1 + I_2) \leq \frac{Q_0}{\mu}.$$

Thus, the considered region for system (2.2) is

$$\Gamma = \{(S, I_1, I_2) : S + I_1 + I_2 \leq \frac{Q_0}{\mu}, S > 0, I_1 \geq 0, I_2 \geq 0\}.$$

The vector field points into the interior of Γ on the part of its boundry when $S + I_1 + I_2 = \frac{Q_0}{\mu}$. So ,

$$S(t) + I_1(t) + I_2(t) < \frac{Q_0}{\mu}, \text{ for } t > 0.$$

Hence, Γ is positively invariant.

Now we investigate the dynamic behavior of system (2.2) on Γ**.** First we find the basic reproduction number R_0 by the method of next generation matrix , see [19] for details.

The disease free equilibrium of system (2.2) is $E_0 = (\frac{Q_0}{\mu}, 0, 0)$

Let $X = (I_1, I_2, S)$, system (2.2) can be written as:

$$X' = \mathcal{F}(x) - \mathcal{V}(x)$$

where

$$\mathcal{F}(x) = \begin{bmatrix} (\beta_1 I_1 + \beta_2 I_2)S \\ 0 \\ 0 \end{bmatrix}, \mathcal{V}(x) = \begin{bmatrix} (\theta+\mu+\delta)I_1 \\ -\theta I_1 + (\mu+\delta)I_2 \\ -Q_0 + (\beta_1 I_1 + \beta_2 I_2)S + \mu S \end{bmatrix}.$$

The jacobian matrices of $\mathcal{F}(x)$ and $\mathcal{V}(x)$ at the disease free equilibrium point E_0, are

$$D\mathcal{F}(E_0) = \begin{bmatrix} \beta_1\frac{Q_0}{\mu} & \beta_2\frac{Q_0}{\mu} & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix} = \begin{bmatrix} F & 0 \\ 0 & 0 \end{bmatrix}, \text{ where } F = \begin{bmatrix} \beta_1\frac{Q_0}{\mu} & \beta_2\frac{Q_0}{\mu} \\ 0 & 0 \end{bmatrix}$$

$$D\mathcal{V}(E_0) = \begin{bmatrix} \theta+\mu+\delta & 0 & 0 \\ -\theta & \mu+\delta & 0 \\ \beta_1\frac{Q_0}{\mu} & \beta_2\frac{Q_0}{\mu} & \mu \end{bmatrix} = \begin{bmatrix} V & 0 \\ J_1 & J_2 \end{bmatrix}, \text{where} V = \begin{bmatrix} \theta+\mu+\delta & 0 \\ -\theta & \mu+\delta \end{bmatrix}$$

$FV^{-1} = \begin{bmatrix} \frac{\beta_1 Q_0}{\mu(\theta+\mu+\delta)} + \frac{\beta_2\theta Q_0}{\mu(\mu+\delta)(\mu+\delta+\theta)} & \frac{\beta_2 Q_0}{\mu(\mu+\delta)} \\ 0 & 0 \end{bmatrix}$ is the next generation matrix of system (2.2). It follows that the spectral radius of FV^{-1} is $\rho(FV^{-1}) = \frac{Q_0[\beta_1(\mu+\delta)+\beta_2\theta]}{\mu(\mu+\delta)(\mu+\delta+\theta)}$

Thus, the basic reproduction number of system (2.2) is

$$R_0 = \frac{Q_0[\beta_1(\mu+\delta)+\beta_2\theta]}{\mu(\mu+\delta)(\mu+\delta+\theta)}.$$

3 Equilibria and their stability

System (2.2) has the disease free equilibrium $E_0 = (\frac{Q_0}{\mu}, 0, 0)$ and the unique positive endemic equilibrium $E^*(S^*, I_1^*, I_2^*)$ where

$S^* = \frac{(\mu+\delta)(\mu+\delta+\theta)}{\beta_1(\mu+\delta)+\beta_2\theta} = \frac{Q_0/\mu}{R_0}$, $I_1^* = \frac{Q_0}{\mu+\delta+\theta}[1 - \frac{\mu}{Q_0}S^*] = \frac{Q_0}{\mu+\delta+\theta}[1 - \frac{1}{R_0}]$ and $I_2^* = \frac{\theta}{\mu+\delta}I_1^*$

We see that I_1^* is positive if $R_0 > 1$ and also $I_1^* = \frac{Q_0 - \mu S^*}{\mu+\delta+\theta}$.

So $E^*(S^*, I_1^*, I_2^*)$ is the unique positive endemic equilibrium point which exists if $R_0 > 1$.

3.1 Local stability of the equilibria

First we investigate the local stability of the disease free equilibrium E_o

Theorem 1 (local stability of E_0) *If $R_0 < 1$, the disease free equilibrium point E_0 of system (2.2) is locally asymptotically stable. If $R_0 = 1$, E_0 is locally stable. If $R_0 > 1$, E_0 is unstable.*

Proof. *: Linearizing system (2.2) around E_0 we get:*

$$J(E_0) = \begin{bmatrix} -\mu & -\beta_1\frac{Q_0}{\mu} & -\beta_2\frac{Q_0}{\mu} \\ 0 & \beta_1\frac{Q_0}{\mu} - \theta - \mu - \delta & \beta_2\frac{Q_0}{\mu} \\ 0 & \theta & -\mu - \delta \end{bmatrix}$$

We can write the characteristic equation as $(-\mu - \lambda)[\lambda^2 + a_1\lambda + a_2] = 0$ *where*

$$\begin{aligned} a_1 &= \theta + 2\mu + 2\delta - \beta_1\frac{Q_0}{\mu}, \\ a_2 &= (\mu+\delta)(\theta+\mu+\delta-\beta_1\frac{Q_0}{\mu}) - \beta_2\frac{Q_0\theta}{\mu} \\ &= (\mu+\delta)(\theta+\mu+\delta) - \frac{Q_0}{\mu}[\beta_1(\mu+\delta)+\beta_2\theta] \\ &= (\mu+\delta)(\theta+\mu+\delta)[1 - \frac{Q_0[\beta_1(\mu+\delta)+\beta_2\theta]}{\mu(\mu+\delta)(\mu+\delta+\theta)}] \\ &= (\mu+\delta)(\theta+\mu+\delta)(1-R_0). \end{aligned}$$

Clearly the first root of the characteristic equation is $\lambda_1 = -\mu < 0$.

If $R_0 < 1$, *then* $a_2 > 0$. *Also,* $\mu(\mu+\delta)(\theta+\mu+\delta) > Q_0\beta_1(\mu+\delta) + Q_0\beta_2\theta > Q_0\beta_1(\mu+\delta)$
which means that $\theta + \mu + \delta > \frac{Q_0\beta_1}{\mu}$ *and so* $a_1 > 0$.

Hence by applying Routh-Herwitz criteria , E_0 *is locally asymptotically stable.*

If $R_0 = 1$, *then* $a_2 = 0$ *and* E_0 *becomes ocally stable.*

If $R_0 > 1$, *then* $a_2 < 0$ *and* E_0 *becomes unstable.* ■

Now we investigate the local stability of the positive equilibrium E^*, by using the following lemma:

Lemma 2 *[4] Let* M *be a* 3×3 *real matrix. If tr(M), det(M) and det(*$M^{[2]}$*) are all negative, then all of the eigenvalues of* M *have negative real part.*

Before we apply this lemma we need the following definition:

Definition 1 (Second additive compound matrix) *[7] Let* $\mathbf{A} = (a_{ij})$ *be an* $n \times n$ *real matrix . The second additive compound of* $\mathbf{A}$ *is the matrix* $\mathbf{A}^{[2]} = (b_{ij})$ *defined as follows:*

$n = 2 : \mathbf{A}^{[2]} = a_{11} + a_{22}$

$$n = 3 : \mathbf{A}^{[2]} = \begin{bmatrix} a_{11}+a_{22} & a_{23} & -a_{13} \\ a_{32} & a_{11}+a_{33} & a_{12} \\ -a_{31} & a_{21} & a_{22}+a_{33} \end{bmatrix}.$$

Theorem 3 *The positive endemic equilibrium* E^* *of system (2.2) is locally asymptotically stable if* $R_0 > 1$.

Proof. Linearizing system (2.2) at the equilibrium $E^*(S^*, I_1^*, I_2^*)$ gives:

$$J(E^*) = \begin{bmatrix} -\mu - k\frac{I_1^*}{S^*} & -\beta_1 S^* & -\beta_2 S^* \\ k\frac{I_1^*}{S^*} & \beta_1 S^* - k & \beta_2 S^* \\ 0 & \theta & -\mu - \delta \end{bmatrix}$$

Using $\beta_1 I_1^* + \beta_2 I_2^* = k\frac{I_1^*}{S^*}$
The second additive compound matrix $J^{[2]}(E^*)$ is

$$J^{[2]}(E^*) = \begin{bmatrix} -\mu - k\frac{I_1^*}{S^*} + \beta_1 S^* - k & \beta_2 S^* & \beta_2 S^* \\ \theta & -\mu - k\frac{I_1^*}{S^*} - \mu - \delta & -\beta_1 S^* \\ 0 & k\frac{I_1^*}{S^*} & \beta_1 S^* - k - \mu - \delta \end{bmatrix}$$

$tr(J(E^*)) = -\mu - k\frac{I_1^*}{S^*} + \beta_1 S^* - k - \mu - \delta < 0$ since$\beta_1 S^* = \frac{\beta_1(\mu+\delta)(\mu+\delta+\theta)}{\beta_1(\mu+\delta)+\beta_2\theta} < k$
.
$det\ (J(E^*)) = (-\mu - k\frac{I_1^*}{S^*})[-(\mu+\delta)(\beta_1 S^* - k) - \beta_2\theta S^*] - k\frac{I_1^*}{S^*}[\beta_1 S^*(\mu+\delta) + \beta_2\theta S^*]$
$= -k\frac{I_1^*}{S^*}[\beta_1 S^*(\mu+\delta) + \beta_2\theta S^*] < 0$ since $(-\mu - k\frac{I_1^*}{S^*})[-(\mu+\delta)(\beta_1 S^* - k) - \beta_2\theta S^*] =$
0.

$$\begin{aligned} \det(J^{[2]}(E^*)) &= (-\mu - k\frac{I_1^*}{S^*} + \beta_1 S^* - k)[(\mu + k\frac{I_1^*}{S^*} + \mu + \delta)(\mu + \delta + k - \beta_1 S^*) \\ &\quad + \beta_1 k I_1^*] - \theta[-\beta_2 S^*(\mu + \delta + k - \beta_1 S^*) - \beta_2 k I_1^*] \end{aligned}$$

$$\begin{aligned} &= -(\mu + k\frac{I_1^*}{S^*})^2(\mu + \delta + k - \beta_1 S^*) - (\mu + k\frac{I_1^*}{S^*})(\mu + \delta)(\mu + \delta + k - \beta_1 S^*) \\ &\quad -(k - \beta_1 S^*)(\mu + k\frac{I_1^*}{S^*})(\mu + \delta + k - \beta_1 S^*) \\ &\quad -(k - \beta_1 S^*)(\mu + \delta)(\mu + \delta + k - \beta_1 S^*) - \beta_1 k I_1^*(\mu + k\frac{I_1^*}{S^*} + k - \beta_1 S^*) \\ &\quad +\beta_2\theta S^*(\mu + \delta + k - \beta_1 S^*) + \beta_2\theta k I_1^*. \end{aligned}$$

$$
\begin{aligned}
= \quad & -(\mu + k\frac{I_1^*}{S^*})^2(\mu+\delta+k-\beta_1 S^*) - (\mu + k\frac{I_1^*}{S^*})(\mu+\delta)^2 \\
& -\mu(\mu+\delta)(k-\beta_1 S^*) - k\frac{I_1^*}{S^*}(\mu+\delta)(k-\beta_1 S^*) \\
& -(k-\beta_1 S^*)(\mu + k\frac{I_1^*}{S^*})(\mu+\delta+k-\beta_1 S^*) \\
& -\beta_2\theta S^*(\mu+\delta+k-\beta_1 S^*) - \beta_1 k I_1^*(\mu + k\frac{I_1^*}{S^*} + k - \beta_1 S^*) \\
& +\beta_2\theta S^*(\mu+\delta+k-\beta_1 S^*) + \beta_2\theta k I_1^* \\
= \quad & -(\mu+\delta+k-\beta_1 S^*)[(\mu + k\frac{I_1^*}{S^*})^2 + (k-\beta_1 S^*)(\mu + k\frac{I_1^*}{S^*})] \\
& -(\mu + k\frac{I_1^*}{S^*})(\mu+\delta)^2 - \mu(\mu+\delta)(k-\beta_1 S^*) \\
& -\beta_1 k I_1^*(\mu + k\frac{I_1^*}{S^*} + k - \beta_1 S^*) \\
< \quad & 0.
\end{aligned}
$$

Hence, by lemma (2) E^* is locally asymptotically stable. ■

3.2 Global stability of equilibria

Now we investigate the global stability of E_0 when $R_0 \leq 1$.

Consider the Liapunov function

$$
\begin{aligned}
L &= [\beta_1(\mu+\delta)+\beta_2\theta]I_1 + \beta_2(\mu+\delta+\theta)I_2 \\
\frac{dL}{dt} &= [\beta_1(\mu+\delta)+\beta_2\theta]I_1' + \beta_2(\mu+\delta+\theta)I_2' \\
&= [\beta_1(\mu+\delta)+\beta_2\theta][(\beta_1 I_1+\beta_2 I_2)S - (\theta+\mu+\delta)I_1] \\
&\quad +\beta_2(\mu+\delta+\theta)[\theta I_1 - (\mu+\delta)I_2] \\
&= [\beta_1(\mu+\delta)+\beta_2\theta](\beta_1 I_1+\beta_2 I_2)S - (\mu+\delta)(\mu+\delta+\theta)(\beta_1 I_1+\beta_2 I_2) \\
&= [[\beta_1(\mu+\delta)+\beta_2\theta]S - (\mu+\delta)(\mu+\delta+\theta)](\beta_1 I_1+\beta_2 I_2) \\
&\leq [[\beta_1(\mu+\delta)+\beta_2\theta]\frac{Q_0}{\mu} - (\mu+\delta)(\mu+\delta+\theta)](\beta_1 I_1+\beta_2 I_2) \\
&= (\mu+\delta)(\mu+\delta+\theta)(R_0-1)(\beta_1 I_1+\beta_2 I_2) \\
&\leq \sigma(\mu+\delta)(\mu+\delta+\theta)(R_0-1)L \leq 0
\end{aligned}
$$

when $R_0 \leq 1$, where $\sigma = \max\{\frac{\beta_1}{\beta_1(\mu+\delta)+\beta_2\theta}, \frac{\beta_2}{\beta_2(\mu+\delta+\theta)}\}$

Define $G = \{(S, I_1, I_2) \in \Gamma : L' = 0\}$, $E_0 = (\frac{Q_0}{\mu}, 0, 0)$ is the maximal compact invariant set in G, the Lasalle Invariance principle theorems shows the global stability of E_0.

Now, we can derive the following theorem:

Theorem 4 *If* $R_0 \leq 1$, *then* E_0 *is globally asymptotically stable in* Γ. *If* $R_0 > 1$ *then* E_0 *is unstable.*

Now we investigate the global stability of E^* by showing that system (2.2) has no periodic solutions, homoclinic loops and oriented phase polygons inside the invariant region, we refer the reader to [2].

Let $\Gamma^* = \{(S, I_1, I_2) \in \Gamma : S + \frac{\mu+\delta}{\mu} I_1 + \frac{\mu+\delta}{\mu} I_2 = \frac{Q_0}{\mu}\}$. It is easy to prove that $\Gamma^* \subset \Gamma$, Γ^* is positively invariant and $E^* \in \Gamma^*$

We will show that system (2.2) has no periodic solutions, homoclinic loops and oriented phase polygons inside the invariant region by using Theorem (4.1) in [2] which is stated as follows:

Theorem 5 *Let* $g = (S, I_1, I_2) = \{g_1(S, I_1, I_2), g_2(S, I_1, I_2), g_3(S, I_1, I_2)\}$ *be a vector field which is piecewise smooth on* Γ^*, *and which satisfies the conditions:* $g.f = 0$, *and* $(\operatorname{curl} g).n < 0$ *in the interior of* Γ^* ,*where* n *is the normal vector to* Γ^* *and* $f = (f_1, f_2, f_3)$ *is a lipschitz field in the interior of* Γ^* *and*

$$\operatorname{curl} g = \det \begin{pmatrix} \overrightarrow{i} & \overrightarrow{j} & \overrightarrow{k} \\ \frac{\partial}{\partial S} & \frac{\partial}{\partial I_1} & \frac{\partial}{\partial I_2} \\ g_1 & g_2 & g_3 \end{pmatrix} = (\frac{\partial g_3}{\partial I_1} - \frac{\partial g_2}{\partial I_2}, \frac{\partial g_1}{\partial I_2} - \frac{\partial g_3}{\partial S}, \frac{\partial g_2}{\partial S} - \frac{\partial g_1}{\partial I_1}).$$

Then the differential equation system $S' = f_1, I_1' = f_2, I_2' = f_3$ *has no periodic solutions, homoclinic loops and oriented phase polygons inside* Γ^*.

Thus, we can state the following theorem.

Theorem 6 *:The system (2.2) has no periodic solutions, homoclinic loops and oriented phase polygons inside the invariant region* Γ^*.

Proof. Let f_1, f_2 *and* f_3 denote the right hand side of system (2.2), respectively.

Now use

$$S + \frac{\mu + \delta}{\mu} I_1 + \frac{\mu + \delta}{\mu} I_2 = \frac{Q_0}{\mu}$$

to rewrite f_1, f_2, f_3 in the equivalent forms:

$$\begin{aligned}
f_1(S,I_1) &= Q_0 - [\beta_1 I_1 + \beta_2(\frac{Q_0}{\mu} - S - \frac{\mu+\delta}{\mu}I_1)\frac{\mu}{\mu+\delta}]S - \mu S,\\
f_1(S,I_2) &= Q_0 - [\beta_1(\frac{Q_0}{\mu} - S - \frac{\mu+\delta}{\mu}I_2)\frac{\mu}{\mu+\delta} + \beta_2 I_2]S - \mu S,\\
f_2(S,I_1) &= [\beta_1 I_1 + \beta_2(\frac{Q_0}{\mu} - S - \frac{\mu+\delta}{\mu}I_1)\frac{\mu}{\mu+\delta}]S - (\mu+\delta+\theta)I_1,\\
f_2(I_1,I_2) &= (\beta_1 I_1 + \beta_2 I_2)(\frac{Q_0}{\mu} - \frac{\mu+\delta}{\mu}I_1 - \frac{\mu+\delta}{\mu}I_2) - (\mu+\delta+\theta)I_1,\\
f_3(S,I_2) &= \theta(\frac{Q_0}{\mu} - S - \frac{\mu+\delta}{\mu}I_2)\frac{\mu}{\mu+\delta} - (\mu+\delta)I_2,\\
f_3(I_1,I_2) &= \theta I_1 - (\mu+\delta)I_2.
\end{aligned}$$

Let $g = (g_1, g_2, g_3)$ be a vector field such that:

$$g_1 = \frac{f_3(S,I_2)}{SI_2} - \frac{f_2(S,I_1)}{SI_1} = \frac{\theta(Q_0-\mu S)}{SI_2(\mu+\delta)} - \beta_1 - \frac{\beta_2(Q_0-\mu S)}{I_1(\mu+\delta)} + \beta_2.$$

$$g_2 = \frac{f_1(S,I_1)}{SI_1} - \frac{f_3(I_1,I_2)}{I_1I_2} = \frac{Q_0}{SI_1} - \beta_1 + \beta_2 - \frac{\theta}{I_2} + \frac{\delta}{I_1} - \frac{\beta_2(Q_0-\mu S)}{I_1(\mu+\delta)}.$$

$$\begin{aligned}
g_3 &= \frac{f_2(I_1,I_2)}{I_1I_2} - \frac{f_1(S,I_2)}{SI_2} = \frac{\beta_1[Q_0-(\mu+\delta)I_1]}{\mu I_2} + \frac{\beta_2[Q_0-(\mu+\delta)I_2]}{\mu I_1}\\
&\quad -\frac{\mu+\delta}{\mu}(\beta_1+\beta_2) - \frac{\delta+\theta}{I_2} - \frac{Q_0}{SI_2} + \frac{\beta_1(Q_0-\mu S)}{I_2(\mu+\delta)} - \beta_1 + \beta_2.
\end{aligned}$$

Since the alternate forms of f_1, f_2 and f_3 are equivalent in Γ^*, then
$g.f = (\frac{I_2'}{SI_2} - \frac{I_1'}{SI_1})S' + (\frac{S'}{SI_1} - \frac{I_2'}{I_1I_2})I_1' + (\frac{I_1'}{I_1I_2} - \frac{S'}{SI_2})I_2' = 0$.
So, $g.f = 0$, on Γ^* .
using the normal vector $n = (\frac{\mu}{Q_0}, \frac{\mu+\delta}{Q_0}, \frac{\mu+\delta}{Q_0})$, to Γ^*, we can see that

$$(\operatorname{curl} g).n = -\frac{\beta_1\delta}{Q_0I_2} - \frac{\beta_2}{I_1^2} - \frac{\theta}{SI_2^2} - \frac{\mu+\delta}{S^2I_2} - \frac{\mu+\delta}{S^2I_1} - \frac{\beta_2\delta}{Q_0I_1} < 0$$

Thus, system (2.2) has no periodic solutions, homoclinic loops and oriented phase polygons inside the invariant region Γ^*. ■

Theorem 7 *:If $R_0 > 1$, then the infected equilibrium point E^* is globally asymptotically stable.*

Proof. *:We know that if $R_0 > 1$ in Γ, then E_0 is unstable. Also Γ^* is a positively invariant subset of Γ and the ω limit set of each solution of (2.2) is a single point in Γ^* since there is no periodic solutions, homoclinic loops and oriented phase polygons inside Γ^*. Therefore E^* is globally asymptotically stable.* ■

4 ODE model when aware HIV infectives do not spread the infection ($\beta_2 = 0$)

When aware HIV infectives do not spread the infection in the community and take preventive measuures, then $\beta_2 = 0$. Thus, the infection is spread only by unaware infectives. In this case we have the following ODE system:

$$\begin{aligned} \frac{dS}{dt} &= Q_0 - \beta_1 I_1 S - \mu S \qquad (4.1)\\ \frac{dI_1}{dt} &= \beta_1 I_1 S - (\theta + \mu + \delta) I_1 \\ \frac{dI_2}{dt} &= \theta I_1 - (\mu + \delta) I_2 \\ \frac{dA}{dt} &= \delta(I_1 + I_2) - (\mu + d) A \end{aligned}$$

Since the variable A of above system does not appear in the first three equations, in the subsequent analysis, we only consider the subsystem:

$$\begin{aligned} \frac{dS}{dt} &= Q_0 - \beta_1 I_1 S - \mu S \qquad (4.2)\\ \frac{dI_1}{dt} &= \beta_1 I_1 S - (\theta + \mu + \delta) I_1 \\ \frac{dI_2}{dt} &= \theta I_1 - (\mu + \delta) I_2 \end{aligned}$$

We also have two equilibrium points $E_0 = (\frac{Q_0}{\mu}, 0, 0)$ and E^*.

By a way similar to the previous section, we calculate the basic reproduction number R_{01}. Let $X_1 = (I_1, I_2, S)$, system (4.2) can be written as:

$$X_1' = \mathcal{F}_1(x) - \mathcal{V}_1(x)$$

The jacobian matrices of $\mathcal{F}_1(x)$ and $\mathcal{V}_1(x)$ at E_o are

$$D\mathcal{F}_1(E_0) = \begin{bmatrix} \beta_1 \frac{Q_0}{\mu} & 0 & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix} = \begin{bmatrix} F_1 & 0 \\ 0 & 0 \end{bmatrix}, \text{ where } F_1 = \begin{bmatrix} \beta_1 \frac{Q_0}{\mu} & 0 \\ 0 & 0 \end{bmatrix}$$

$$D\mathcal{V}_1(E_0) = \begin{bmatrix} \theta + \mu + \delta & 0 & 0 \\ -\theta & \mu + \delta & 0 \\ \beta_1 \frac{Q_0}{\mu} & 0 & \mu \end{bmatrix} = \begin{bmatrix} V_1 & 0 \\ J_3 & \mu \end{bmatrix}, \text{ where } V_1 = \begin{bmatrix} \theta + \mu + \delta & 0 \\ -\theta & \mu + \delta \end{bmatrix}$$

$$F_1 V_1^{-1} = \begin{bmatrix} \frac{\beta_1 Q_0}{\mu(\theta+\mu+\delta)} & 0 \\ 0 & 0 \end{bmatrix} \quad R_{01} = \frac{\beta_1 Q_0}{\mu(\mu+\delta+\theta)}.$$ We note that $R_0 \to R_{01}$ when $\beta_2 = 0$.

The considered region Γ for system (4.2) is the same as in system (2.2).

The positive equilibrium $E^*(S^*, I_1^*, I_2^*)$ is given by the following
$S^* = \frac{\mu+\delta+\theta}{\beta_1} = \frac{Q_o/\mu}{R_{01}}$, $I_1^* = \frac{Q_0-\mu S^*}{\mu+\delta+\theta} = \frac{Q_0}{\mu+\delta+\theta}(1-\frac{1}{R_{01}})$ and $I_2^* = \frac{\theta}{\mu+\delta} I_1^*$
$E^*(S^*, I_1^*, I_2^*)$ exists if $R_{01} > 1$.

We investigate the local and global stability of the disease free equilibrium E_0 and the endemic equilibrium E^* by using the same methods in section 3.

Theorem 8 *: If $R_{01} < 1$, the disease free equilibrium point E_0 of system (4.2) is locally asymptotically stable. If $R_{01} = 1$, E_0 is locally stable and if $R_{01} > 1$, then E_0 is unstable.*

Proof. *Linearizing system (3.2) at the equilibrium $E_0(\frac{Q_0}{\mu}, 0, 0)$ gives the characteristic equation $(-\mu-\lambda)[\lambda^2 + a_1\lambda + a_2] = 0$*

where

$$\begin{aligned} a_1 &= \theta + 2\mu + 2\delta - \beta_1 \frac{Q_0}{\mu} \\ a_2 &= (\mu+\delta)(\theta+\mu+\delta-\beta_1\frac{Q_0}{\mu}) \end{aligned}$$

If $R_{01} < 1$, then $a_1, a_2 > 0$. So, E_0 is locally asymptotically stable.
If $R_{01} = 1$, then $a_2 = 0$. So, E_0 is locally stable.
If $R_{01} > 1$, then $a_2 < 0$.So, E_0 is unstable. ■

Theorem 9 *The positive endemic equilibrium E^* of system (4.2) is locally asymptotically stable if $R_{01} > 1$.*

Proof. *Linearizing system (3.2) at the equilibrium $E^*(S^*, I_1^*, I_2^*)$ gives:*

$$J(E^*) = \begin{bmatrix} -\mu-\beta_1 I_1^* & -\beta_1 S^* & 0 \\ \beta_1 I_1^* & 0 & 0 \\ 0 & \theta & -\mu-\delta \end{bmatrix}$$

$tr(J(E^*)) = -2\mu - \delta - \beta_1 I_1^* < 0$
$\det(J(E^*)) = -\beta_1^2 I_1^* S^*(\mu+\delta) < 0$
The second additive compound matrix $J^{[2]}(E^*)$ is

$$J^{[2]}(E^*) = \begin{bmatrix} -\mu-\beta_1 I_1^* & 0 & 0 \\ \theta & -2\mu-\delta-\beta_1 I_1^* & -\beta_1 S^* \\ 0 & \beta_1 I_1^* & -\mu-\delta \end{bmatrix}$$

$\det(J^{[2]}(E^*)) = (-\mu-\beta_1 I_1^*)[(2\mu+\delta+\beta_1 I_1^*)(\mu+\delta) + \beta_1^2 I_1^* S^*] < 0$
Hence, E^* is locally asymptotically stable. ■

Theorem 10 *If $R_{01} \leq 1$, E_0 is globally asymptotically stable in Γ. if $R_{01} > 1$, then E_0 is unstable.*

Proof. Consider the Liapunov function. $L = \beta_1(\mu+\delta)I_1$. Then follow the same steps in the proof of theorem 4. ■

We investigate the global stability of E^* by showing that this system has no periodic solutions, homoclinic loops and oriented phase polygons inside the invariant region Γ^*. So we have the following theorem

Theorem 11 *If $R_{01} > 1$, then the infected equilibrium point E^* is globally asymptotically stable.*

5 ODE model without screening ($\theta = 0$)

In this section we consider the situation without screening of unaware infectives ($\theta = 0$). In this case we have the following ODE system:

$$\begin{aligned} \frac{dS}{dt} &= Q_0 - \beta_1 I_1 S - \mu S \\ \frac{dI_1}{dt} &= \beta_1 I_1 S - (\mu + \delta) I_1 \\ \frac{dA}{dt} &= \delta(I_1 + I_2) - (\mu + d) A \end{aligned} \tag{5.1}$$

As before we consider the subsystem:

$$\begin{aligned} \frac{dS}{dt} &= Q_0 - \beta_1 I_1 S - \mu S \\ \frac{dI_1}{dt} &= \beta_1 I_1 S - (\mu + \delta) I_1 \end{aligned} \tag{5.2}$$

We also have the disease-free equilibrium point $E_0 = (\frac{Q_0}{\mu}, 0)$ and the unique positive endemic equilibrium point $E^*(S^*,\ I_1^*)$.

The basic reproduction number here is given by $R_{02} = \frac{\beta_1 Q_0}{\mu(\mu+\delta)}$. Note that $R_{02} \longrightarrow R_0$ when $\theta = 0$

The considered region for system (5.2) is:

$$\Gamma = \{(S, I_1) : S + I_1 \leq \frac{Q_0}{\mu}, S > 0, I_1 \geq 0\}.$$

The positve equilibrium point $E^*(S^*,\ I_1^*)$ is given by: $S^* = \frac{\mu+\delta}{\beta_1} = \frac{Q_o/\mu}{R_{o2}}$, $I_1^* = \frac{Q_0 - \mu S^*}{\mu+\delta} = \frac{Q_0}{\mu+\delta}(1 - \frac{1}{R_{02}})$.

$E^*(S^*,\ I_1^*)$ existx if $R_{02} > 1$.

The local and global stability of the positive equilibrium E_0 and the endemic equilibrium E^* are stated in the following theorems whose proofs are similar to what we did in section 3.

Theorem 12 *If $R_{02} < 1$, the disease free equilibrium point E_0 of system (5.2) is locally asymptotically stable. If $R_{02} = 1$, E_0 is locally stable and if $R_{02} > 1$, then E_0 is unstable.*

Theorem 13 *If $R_{02} > 1$, then the positive endemic equilibrium point E^* is locally asymptotically stable.*

By using the same Liapunov function in theorem 10, we can prove the following theorem

Theorem 14 *If $R_{02} \leq 1$, E_0 is globally asymptotically stable in Γ . If $R_{02} > 1$ then E_0 is unstable.*

We investigate the global stability of the positive equilibrium E^* by showing that this system has no periodic solutions inside the invariant region Γ using Dulac's criterion [6].

Theorem 15 *If $R_{02} > 1$, then the infected positive equilibrium point E^* is globally asymptotically stable.*

Proof. *To prove this we use Dulac's criterion with $B(S, I_1) = \frac{1}{SI_1}$*

$$\begin{aligned} Bf_1 &= \frac{1}{SI_1}(Q_0 - \beta_1 I_1 S - \mu S) \\ &= \frac{Q_0}{SI_1} - \beta_1 - \frac{\mu}{I_1} \end{aligned}$$

and

$$\begin{aligned} Bf_2 &= \frac{1}{SI_1}(\beta_1 I_1 S - (\mu + \delta) I_1) \\ &= \beta_1 - (\mu + \delta)\frac{1}{S} \end{aligned}$$

Then

$$\frac{\partial(Bf_1)}{\partial S} + \frac{\partial(Bf_2)}{\partial I_1} = -\frac{Q_0}{S^2 I_1} \neq 0$$

Thus, there exist no periodic solutions in Γ.Hence, E^* is globally asymptotically stable. ■

6 Numerical simulations

To see the dynamical behavior of system (2.1) , we solve the system by Runge-Kutta method using the parameters; $Q_0 = 2000$, $\mu = 0.02$, $\delta = 0.1$, $\theta = 0.015$, $d = 1$, $\beta_1 = 0.0009$, $\beta_2 = 0.00027$. with different initial values:

1. $S(0) = 1100$, $I_1(0) = 16000$, $I_2(0) = 1540$, $A(0) = 1360$,
2. $S(0) = 1180$, $I_1(0) = 12850$, $I_2(0) = 1100$, $A(0) = 870$,
3. $S(0) = 2000$, $I_1(0) = 5400$, $I_2(0) = 850$, $A(0) = 500$,
4. $S(0) = 1200$, $I_1(0) = 10000$, $I_2(0) = 500$, $A(0) = 300$.

In the first two figures 1and 2, we use different initial values in four cases to display the unaware and aware population plotted against the total population. We see from these figures that for any initial value, the solution curves tend to the equilibrium E^* where $R_0 = 6917 > 1$. Hence, system (2.1) is globally a symptotically stable about E^* for the above set of parameters.

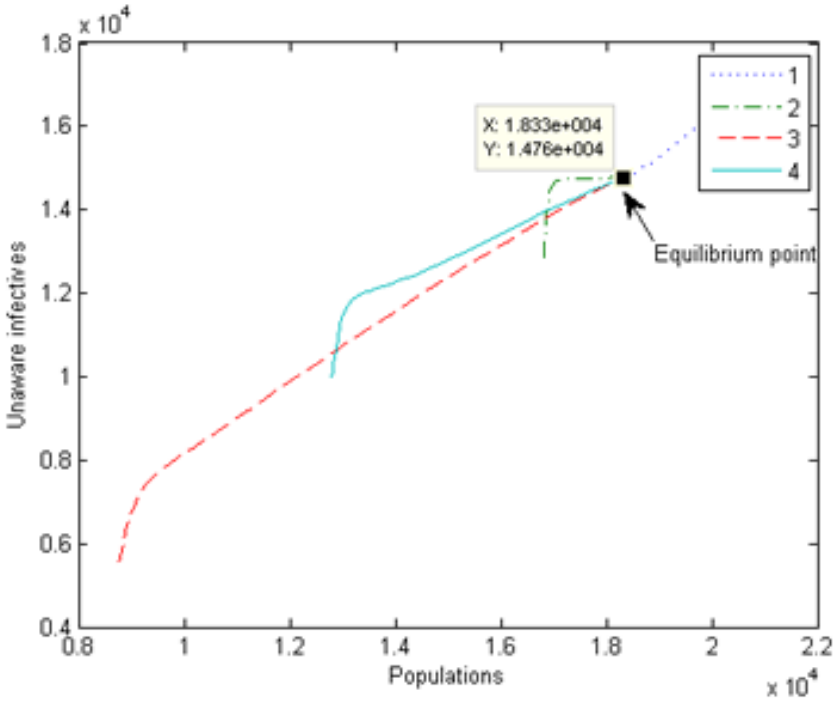

Figure 1: Variation of unaware HIV infected population against total population

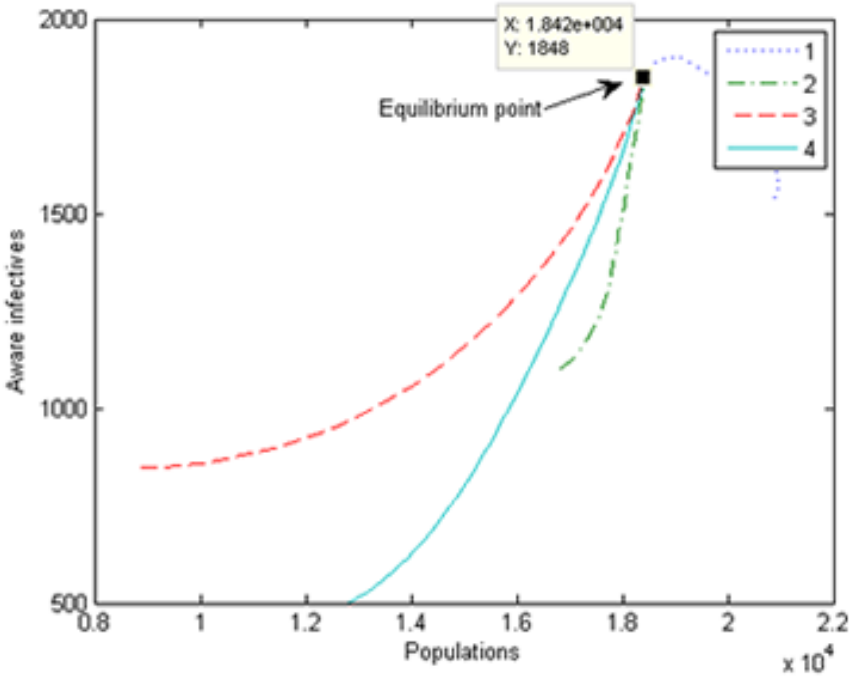

Figure 2: Variation of aware HIV infected population against total population

In the following figures 3-5, the variation of unaware and aware HIV infected population and that of AIDS patients for different rates of screening is shown by using the parameters. $Q_0 = 3000$, $\mu = 0.04$, $\delta = 0.3$, $\theta = 0.02$, $d = 1$, $\beta_1 = 0.0009$, $\beta_2 = 0.00027$. with initial values $S(0) = 15300$, $I_1(0) = 5400$, $I_2(0) = 4500$, $A(0) = 1800$. It is seen that as θ decreases to zero aware infectives will decreases to reach zero also, where as the unaware infected population will be increasing (i.e I_1 will grow), so, they will continue to spread the disease and increase the AIDS patients population.

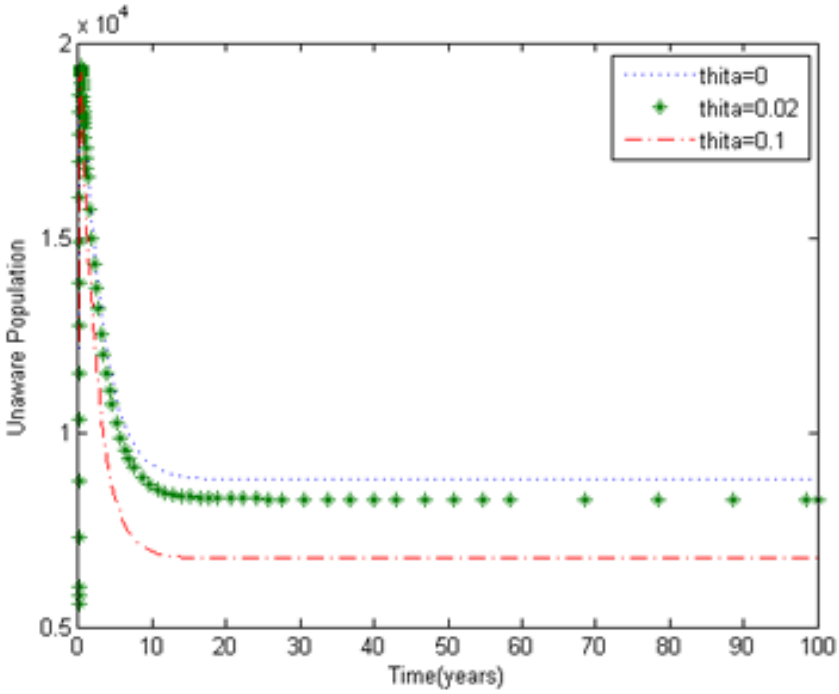

Figure 3: Variation of unaware HIV infected population for different values of θ

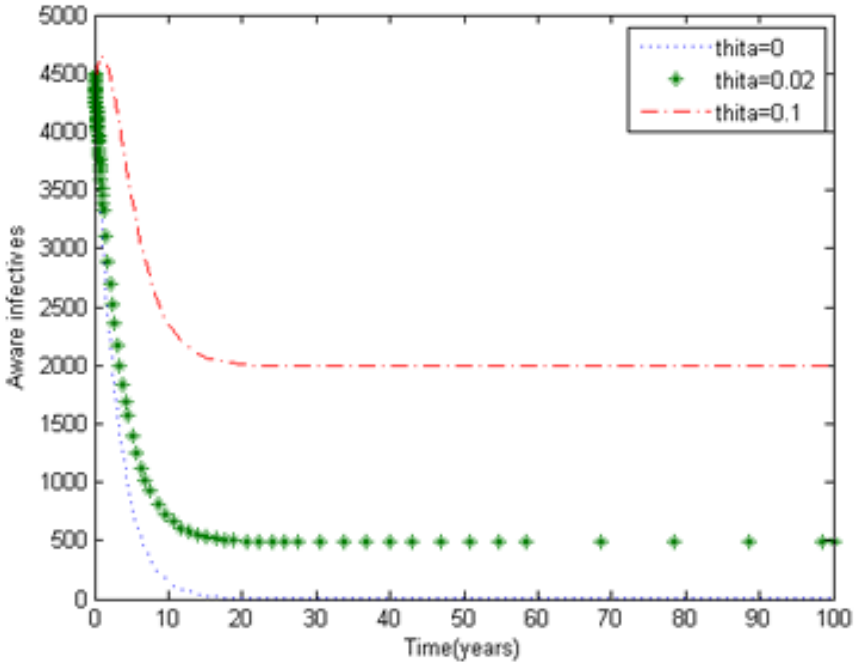

Figure 4: Variation of aware HIV infected population for different values of θ

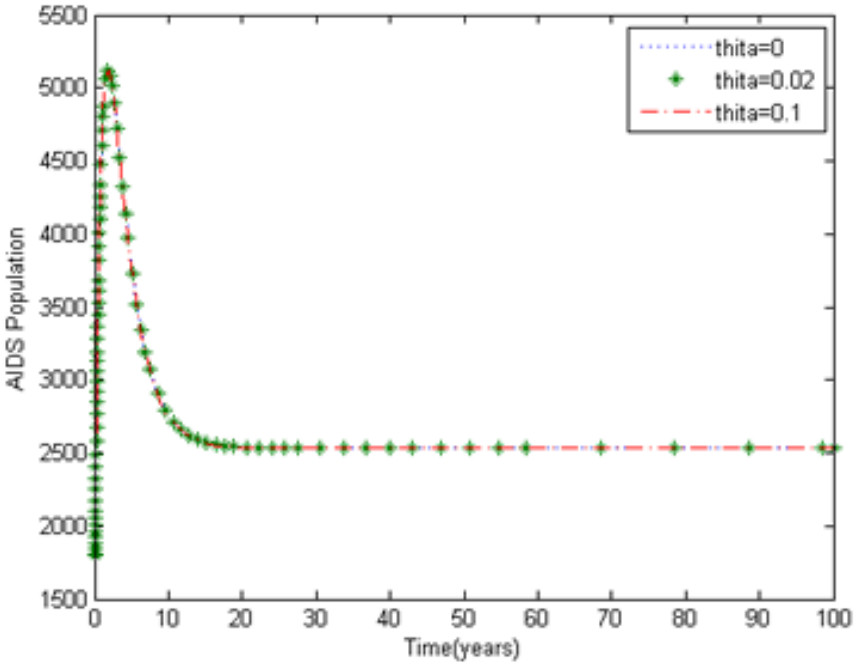

Figure 5: Variation of AIDS population for different values of θ

Under the same parameters and initial conditions, we see that Figures 6 and 7 show the role of contact rate β_2 of aware HIV infectives. When aware HIV infectives do not take preventive measures, the unaware infective population increases which leads to increase the AIDS patients. When aware HIV infectives take preventive measures (i.e $\beta_2 = 0$), the number of unaware infectives decreases leading to AIDS population decline.

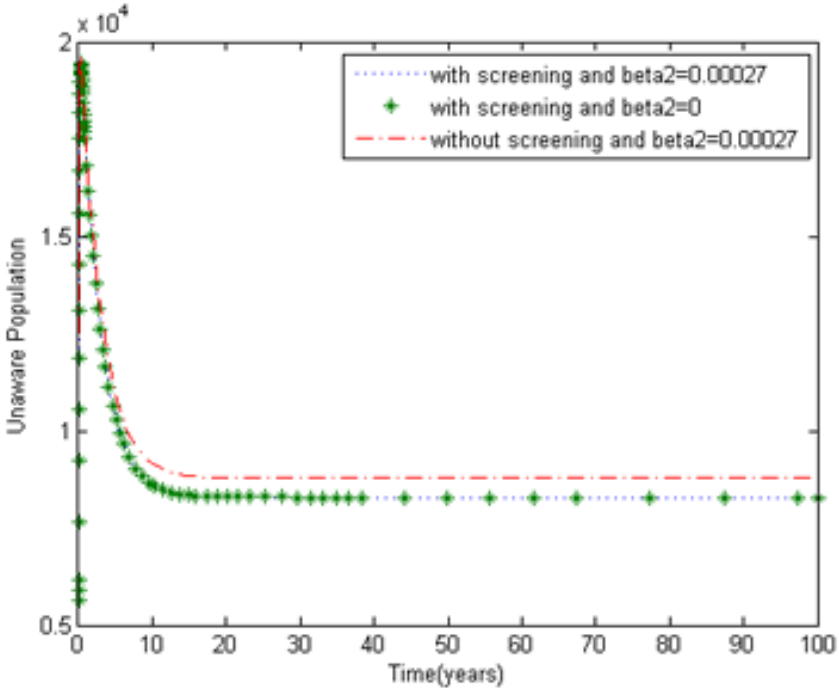

Figure 6: Variation of unaware HIV infected population for different values of β_2

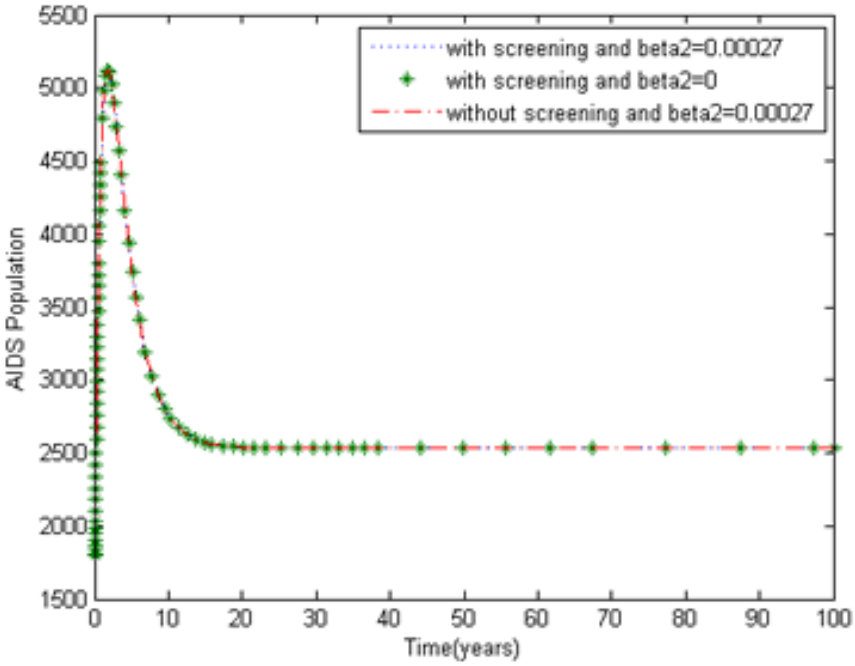

Figure 7: Variation of AIDS population for different values of β_2

7 Conclusions and Recommandations

In this paper, a non-linear mathematical model was formulated. Sufficient conditions have been given ensuring local and global stability of the disease free equilibrium point and the unique positive endemic equilibrium point. The disease-free equilibrium (E_0) is shown to be locally asymptotically stable when the associated epidemic threshold known as the basic reproduction number (R_0) for the model is less than unity. Liapunov function is used to show the global stability of E_0 when R_0 is less than unity. The positive infected equilibrium (E^*) is shown to be locally asymptotically stable when R_0 is greater than unity. By showing that this model has no periodic solutions, homoclinic loops and oriented phase polygons inside the invariant region Γ^* we proved the global asymptotically stable of E^*. The model has also been analyzed to study the effect of screening of unaware infectives on the spread of HIV disease. It is found that the spread of the disease will be controlled with increase in the rate of detection by screening, which means that the endemicity of the infection increases in the absence of screening and consequently the AIDS population increases continuously. The impact on the dynamics of HIV/AIDS is also analyzed when aware HIV infectives take preventive measures with their contact in the community.

At the end, we want to recommend as a result of our study that the most effective way to lower the incidence rate is by enforcing screening on indivisuals who are most likely to get infected like drug adects on a regular basis and to educate the population by making them aware of the consequences of preventive measures against the infection. If the population presents a positive attitude towards preventive procedures, the spread of disease can be controlled even for relatively small screening rates. Therefore, education programs must reach the community at all social levels to increase the awareness about the disease and protection techniques in the community.

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