Modelling the role of awareness and screening of infectives in the transmission dynamics of HIV

Navjot Kaur†, Mini Ghosh‡, S. Bhatia§

Department of Biology, Indian Institute of Science Education and Research (IISER) Mohali, Punjab, India.
School of Advanced Sciences, VIT, Chennai, India.
School of Mathematics, Thapar University, Patiala, Punjab, India.

(Received September 4 2015, Accepted March 4 2016)

Abstract. Prevention is better than the cure, more so for incurable infectious diseases. The awareness, screening of infectives and counseling can help in devising suitable preventive policies for controlling the incurable infectious diseases. This paper proposes a nonlinear mathematical model to study the effect of awareness, screening of infectives and counseling on the spread of HIV infection in the endemic region. In this work, we determine the basic reproduction number \( R_0 \) of the model by the method of next generation matrix and it is found 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 always dies out and the disease-free equilibrium is globally stable. If \( R_0 > 1 \), the positive endemic equilibrium is locally asymptotically stable and it may be globally stable under some conditions on parameters. The effect of awareness, screening of infectives and counseling on the transmission of HIV is studied using numerical simulation and we observe that all these parameters have positive impact in reducing the transmission of HIV in the population. Our analysis show that with some suitable choice of parameters corresponding to awareness, screening and counseling, the disease can be eliminated from the population within a reasonable duration of time.

Keywords: awareness, HIV, stability theory, simulation

1 Introduction

Acquired Immune Deficiency Syndrome (AIDS) was first identified as a distinct infectious disease in the year 1981 as per the report from the US Centers for Disease Control and Prevention. Two years later the causative virus named as the Human Immunodeficiency Virus (HIV) was identified. HIV is a retrovirus that infects and kills \( CD4^+ \) T-cells in a human body. It gradually destroys the immune cells and makes the body unable to fight infections. The deterioration of the immune system results in the development of opportunistic infections that lead to AIDS\(^{[24]}\). HIV infection, due to its various modes of infection, is the main matter of concern as it can be transmitted by having sex with an infected partner/person, by sharing needles for injection drug as well by blood transfusion. HIV/AIDS is a major health problem in many countries of the world, and is considered as a pandemic (i.e. a disease outbreak which is present over a large geographical area and that is actively spreading)\(^{[22]}\). A global report presented by UNAIDS in the year 2012 estimated that 35.3 (32.2–38.8) million people are living with HIV.

It should be noticed that the HIV infected individuals undergo a long latency period (typically 5–10 years) prior to the surfacing of the disease. During this time span, the individuals remain unaware of their infected status, but remain infectious. Delayed diagnosis of HIV infection can be considered as the major contributor

---

\( ^* \) The authors thank the handling editor and anonymous referees for their valuable comments and suggestions that led to an improvement of our original manuscript. This research work was partially supported by the research grants of DST, Govt. of India, via a sponsored research project: SR/S4/MS:681/10.

\( ^\dagger \) E-mail address: navjotkaur@iisermohali.ac.in.
and an epidemiologically significant reason behind the growth of HIV. The high-risk sexual behavior is very common among HIV-positive individuals who are unaware of their infection status as compared to those who know they are HIV positive. The studies around the world show that the number of individuals who get tested for HIV infection have increased as compared to the previous year figures. However, the fear and misconceptions still prevent people from knowing their HIV status. As a result the undiagnosed infection remains a significant factor that fuels the transmission of the disease.

Although the HIV/AIDS is a pandemic and significant research of over 20 years has gone into development of effective control measures for HIV/AIDS, it remains a difficult target to invent a vaccine or drug that can cure it. Recently the use of Active Antiretroviral Drugs, in particular Highly Active Antiretroviral Therapy (HAART) remained successful in reducing its spread and AIDS related deaths among HIV infected patients. However, these life extending medicines are not extensively available in some countries which are in serious need of this therapy due to high HIV incidence and prevalence rates. Clearly, the disease is challenging as there is no effective medicine to cure HIV. Due to which the person who once get infected with this infection will remain infective for his/her life-time without any recovery. In the absence of curative treatment and due to severity of HIV infection, prevention is the only feasible way to reduce its spread.

The estimated cost of the available treatment and care of HIV/AIDS infected patients is somehow unaffordable in many of the developing countries. Public screening and education of HIV/AIDS is a powerful tool in combating the disease burden as it provides information to the individuals about causes of the disease that how it is transmitted, the precautionary measures that individuals can take to prevent the transmission of HIV, and the steps to be taken after the detection of the disease among susceptible individuals. The prevention is about creating an environment of information and guiding people to adopt careful practices that will reduce their chances of getting infected.

The grim nature of this epidemic has motivated the researchers to develop mathematical models to understand its dynamical nature and the mechanism responsible to accelerate its growth on the population level. Mathematical modeling offers an efficient mode to create intelligent information that can help to develop suitable and effective policies. Since the recognition of the AIDS, various mathematical models have been developed to understand its immunological and epidemiological dynamics. The preliminary work in this regard is done by Anderson, Anderson and May, Castillo-Chavez, May and Anderson and Schwager et al. Some researchers considered the proportional mixing of male population with different rates of contact with their new sexual partners in their mathematical model of HIV transmission. Various mathematical models to study the impact of sexual/social mixing structures are carried out and their results give emphasis on the urgency of understanding the mixing patterns to protect the future generations from the virus. The impact of core risk group (such as commercial-sex workers) is discussed by several researchers. In these studies, the researchers have suggested that the prevention through behavioral changes among high-risk groups can result in the reduction of new infections. Epidemic models for HIV transmission have also been developed to study the impact of awareness, treatment, effect of drug therapy, response to immune system and effect of co-infection.

In the authors have given emphasis on promoting counseling and testing of HIV positive patients as this may be helpful in reducing the risky sexual activities in resource limited communities. The parameters such as considerable variability of the infection rates for different sub populations at risk (e.g. homosexuals, heterosexuals, drug users, etc.); the long incubation period before the occurrence of symptoms; the variability of the infection based on evolution of the infection in an individual; etc., are discussed in the Lecture Notes in Biomathematics. Moreover, these parameters of HIV/AIDS infections imply a relevant impact on the demography of the population at risk. Tripathi et al. proposed an epidemiological model to analyze the effect of screening of unaware infectives on the transmission of HIV infection with constant immigration of susceptibles in a homogeneous population. The model analysis has shown that the screening of unaware infectives has positive impact on reducing HIV prevalence.

To the best of our knowledge, relatively few studies have been carried out to mathematically analyze the impact of screening on the spread of HIV infection. In the present study, we have considered the impact of effective screening, i.e. the screening with proper counselling which plays a major role in the transmission dynamics of any infection in a homogeneous population. Here, by effective screening we mean screening and
subsequent counseling which convinces HIV infectives not to transmit this disease to others. The effective screening acts as a powerful intervention in reducing the diagnostic delays and can help to initiate the timely treatment of the HIV infectives to suppress the viral load.

In the view of above facts, present paper is devoted to analyze a nonlinear mathematical model to study the effect of awareness, screening of infectives and counseling on the spread of HIV infection in the endemic region. This model is formulated based on the basic principles developed by Anderson and May [2, 3] and May and Anderson [27]. These principles have been widely adopted to understand the dynamic nature and impact of the epidemic in various epidemiological and social environments. The basic reproduction number $R_0$ of the model is determined by the method of next generation matrix. Mathematical analysis establishes that the global dynamics of the spread of the HIV infection is completely determined by the basic reproduction number $R_0$. The effect of awareness, screening of infectives and counseling on the transmission of HIV is demonstrated using numerical simulation and we show that all these have positive impact in reducing the transmission of HIV in the population. Furthermore, it is evident that for some suitable choice of parameters corresponding to awareness, screening and counseling this disease can be eliminated from the population.

The remaining of this paper is organized as follows: Section 1 is further divided into two subsections. The Subsection 1.1 describes the mathematical model (1) for HIV along with a discussion on the basic groundwork behind the formulation of present model. The Subsection 1.2 covers the basic properties of the model. Section 2 is also divided in two subsections, in Subsection 2.1 we derive the expression for basic reproduction number and thereafter Subsection 2.2 shows the existence and uniqueness of endemic equilibrium point. In Section 3 the local and global stability analysis of the equilibria is proved. In Section 4, some numerical simulations to support our analytical findings have been presented. Finally, in Section 5 the epidemiological significance of the observations obtained from the analysis have been discussed.

1.1 Model description

In view of the discussion in Section 1, a mathematical model has been formulated to study the dynamics of HIV with emphasis on screening and treatment of infectives. Almost 90% of HIV infection occurs due to sexual transmission and the individuals between the age of 15-24 are the most vulnerable to this infection. Hence, in this study only the adult population has been taken into account.

The whole population (and here whole population we means whole sexually active adult population) under consideration is divided into three compartments: susceptibles $S(t)$, who are not infected with HIV/AIDS, HIV infected individuals $I(t)$, who are infected with HIV and can transmit the virus, and AIDS $A(t)$, the total number of individuals who have fully developed AIDS, and thus they no longer take part in the transmission. The population of interest is homogeneously mixed, that means all susceptible individuals are equally likely to become infected through an HIV positive person in case of effective contact. The following assumptions have been made while formulating the model:

- It is assumed that population is subjected to general awareness programme about HIV and there are several health centers where screening and subsequent counseling of HIV infectives is performed.
- The infected individuals who are unaware of having disease will transmit this infection to others unknowingly as they may not take precautionary measures to reduce the transmission. So, the rate of transmission due to this group of individuals will be comparatively more than the transmission due to aware HIV infectives.
- Further, it is assumed that in case of 100% effective counseling, the screened HIV infected individuals will not take part in the transmission of HIV. Whereas the infected individuals may take part in the transmission if counseling is not proper. It should be noticed that some orthodox/convetional people who never give consideration to counselors’ advises (they actually give a deaf ear to the advisors) may also take part in the transmission of this disease. On the basis of these observations, we have taken different disease transmission rates through aware and unaware HIV infectives.

Additionally, general awareness about the transmission of this disease and possible consequences may influence individuals to take precautionary measures to protect themselves against HIV. In this way, the HIV infection through sexual transmission may lower down in this group of individuals. The discrimination and
stigma are still associated with the illness and due to this some HIV infected patients develop psychological problems and they try to infect others around them with increased rate. So in this case, the rate of transmission in this group of screened HIV infectives will be very high as compared to normal rate of transmission. The transition diagram representing the disease dynamics is shown in Fig. 2.

Assembling all the above mentioned observations and assumptions, the following standard incidence mathematical model governed by the system of ordinary differential equations is formulated as follows:

\[
\begin{align*}
\frac{dS}{dt} &= \Gamma - \frac{\beta_1 \alpha (1 - \gamma) I + \beta_2 (1 - \alpha) (1 - \gamma) I + \beta_3 (1 - \epsilon) \gamma I}{N} \{(1 - \alpha) + \eta \alpha\} S - \mu S, \\
\frac{dI}{dt} &= \frac{\beta_1 \alpha (1 - \gamma) I + \beta_2 (1 - \alpha) (1 - \gamma) I + \beta_3 (1 - \epsilon) \gamma I}{N} \{(1 - \alpha) + \eta \alpha\} S \\
&\quad - \tau_1 \delta \gamma I - \tau_2 \{(1 - \delta) \gamma + (1 - \gamma)\} I - (\mu + \nu_1) I, \\
\frac{dA}{dt} &= \tau_1 \delta \gamma I + \tau_2 \{(1 - \delta) \gamma + (1 - \gamma)\} I - (\mu + \nu_2) A, \\
S(0) &= S_0 > 0, \quad I(0) = I_0 \geq 0, \quad A(0) = A_0 \geq 0, \quad N = S(t) + I(t) + A(t).
\end{align*}
\]

The parameters used in the model have their physical interpretation. The descriptions of all parameters considered in model formulation are summarized in Tab. 1. The model (1) can be re-written as follows:

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>(\Gamma)</td>
<td>Recruitment rate into the population,</td>
</tr>
<tr>
<td>(\beta_i) ((i = 1; 2))</td>
<td>Rates of transmission of infection by unscreened infectives to susceptibles,</td>
</tr>
<tr>
<td>(\beta_3)</td>
<td>Rate of transmission of infection by screened infectives who are not listening to counsellors,</td>
</tr>
<tr>
<td>(\mu)</td>
<td>Natural death rate,</td>
</tr>
<tr>
<td>(\gamma)</td>
<td>Fraction of screening of unaware infectives,</td>
</tr>
<tr>
<td>(\alpha)</td>
<td>Fraction at which individuals are projected to general awareness programmes,</td>
</tr>
<tr>
<td>(\delta)</td>
<td>Fraction of HIV infectives that are under treatment and are identified through screening test,</td>
</tr>
<tr>
<td>(\epsilon)</td>
<td>Fraction of screened infectives not participating in the transmission of disease,</td>
</tr>
<tr>
<td>(\eta)</td>
<td>Fraction which reduces the transmission in aware susceptible individuals,</td>
</tr>
<tr>
<td>(\tau_1, \tau_2)</td>
<td>Progression rates to AIDS for treated and untreated HIV infectives, respectively,</td>
</tr>
<tr>
<td>(\nu_1, \nu_2)</td>
<td>Disease-induced mortality for HIV infection and AIDS, respectively.</td>
</tr>
</tbody>
</table>
\[
\begin{align*}
\frac{dN}{dt} &= \Gamma - \mu N - \nu_1 I - \nu_2 A \\
\frac{dI}{dt} &= k_1 \frac{I(N - I - A)}{N} - k_2 I - (\mu + \nu_1)I \\
\frac{dA}{dt} &= k_2 I - (\mu + \nu_2)A,
\end{align*}
\]

where
\[
k_1 = \{\beta_1 \alpha (1 - \gamma) + \beta_2 (1 - \alpha)(1 - \gamma) + \beta_3 (1 - \epsilon) \gamma\} \{(1 - \alpha) + \eta \alpha\},
\]
\[
k_2 = \tau_1 \delta \gamma + \tau_2 \{(1 - \delta) \gamma + (1 - \gamma)\}.
\]

### 1.2 Basic properties

Since the model under consideration monitors human populations, it is henceforth assumed that all the associated model variables and parameters are non-negative. This result is summarized in following lemma:

**Lemma 1.** For all time \( t \geq 0 \), all the solutions of the system (1) are eventually confined in the compact subset \( \mathcal{D} = \{(S, I, A) \in \mathbb{R}_+^3 : N = (S(t) + I(t) + A(t)) \leq \frac{\Gamma}{\mu}\} \); i.e. the closed set \( \mathcal{D} \) is positively invariant for the model (1).

**Proof.** We follow the following steps to show the positive invariance of \( \mathcal{D} \) i.e. all the solutions of (1) which initiate in \( \mathcal{D} \) remain in the region \( \mathcal{D} \).

The rate of change of the total population \( N \), is calculated by adding all the equations considered in model system (1), is given by

\[
\frac{dN}{dt} = \Gamma - \mu N - \nu_1 I - \nu_2 A.
\]

It follows that whenever \( N > \frac{\Gamma}{\mu} \), then \( \frac{dN}{dt} < 0 \).

Since, \( \frac{dN}{dt} \) is bounded by \( \Gamma - \mu N \), therefore the standard comparison theorem\(^{21}\) is used to show, \( N \leq \frac{\Gamma}{\mu}(1 - e^{-\mu t}) + N_0 e^{-\mu t} \), and for \( t \to \infty \), we have

\[
\lim_{t \to \infty} \sup N \leq \frac{\Gamma}{\mu}.
\]

In particular, \( N(t) < \frac{\Gamma}{\mu} \) if \( N(0) < \frac{\Gamma}{\mu} \).

Clearly, it has been proved that all the solutions of (1) which initiate in \( \mathbb{R}_+^3 \) are confined in the region \( \mathcal{D} \). Thus, \( \mathcal{D} \) is positive-invariant and attracting. Hence the system considered is biologically and mathematically well posed in the region \( \mathcal{D} \).\(^{17}\)

### 2 Basic reproduction number and existence of equilibria

#### 2.1 Basic reproduction number

The model has a disease-free equilibrium which is obtained by setting the right hand sides of the equations in the model (2) to zero, and \( I = 0 = A \), given by \( E_0 = (N_0, 0, 0) \) with \( N_0 = \frac{\Gamma}{\mu} \). The stability of this equilibrium will be investigated by using the next generation matrix \(^{15,35}\). Following the standard notation described by Van den Driessche and Watmough in \(^{35}\) on the system (2), the matrices \( F \) and \( V \), for the new infection terms and the remaining transfer terms, are, respectively, given by
\[
F = \begin{pmatrix}
0 & 0 & 0 \\
0 & k_1 & 0 \\
0 & 0 & 0
\end{pmatrix},
\]

and

\[
V = \begin{pmatrix}
\mu & \nu_1 & \nu_2 \\
0 & (\nu_1 + \mu + k_2) & 0 \\
0 & 0 & (\mu + \nu_2)
\end{pmatrix}.
\]

Reproduction number is the spectral radius or the largest eigenvalue of its next generation matrix \(FV^{-1}\) and is computed as

\[
R_0 = \rho(FV^{-1}) = \frac{k_1}{\nu_1 + k_2 + \mu}.
\]  

The threshold quantity \(R_0\) is the basic reproduction number for HIV infection. It computes the average number of new HIV infections to be generated by a single infectious in a completely susceptible population. The disease-free equilibrium, \(E_0\) of the re-written model system (2) is locally asymptotically stable whenever \(R_0 < 1\) and unstable if \(R_0 > 1\). This means that HIV might be eliminated from the population (when \(R_0 < 1\)) if the solutions of the population of system (2) are in the basin of attraction of the disease-free equilibrium \(E_0\).

2.2 The existence and uniqueness of endemic equilibrium

For the model system (2) there exist two equilibria namely Disease-free equilibrium \(E_0\) (as discussed in Subsection 2.1) and Endemic equilibrium \(E^*\). Here we will discuss the existence of unique endemic equilibrium point \(E^* = (N^*, I^*, A^*)\). The positive endemic equilibrium (steady state with \(I > 0\)) can be obtained by setting the right hand side of the equations in model (2) equal to zero, i.e.

\[
\Gamma - \mu N - \nu_1 I - \nu_2 A = 0
\]

\[
k_1 \frac{(N - I - A)}{N} - k_2 I - (\mu + \nu_1) I = 0
\]

\[
k_2 I - (\mu + \nu_2) A = 0,
\]

which gives

\[
N^* = \frac{\Gamma - \nu_1 I^* - \nu_2 A^*}{\mu},
\]

\[
A^* = \frac{k_2}{\mu + \nu_2} I^*,
\]

where

\[
I^* = \frac{\Gamma(\mu + \nu_2)(k_2 + \mu + \nu_1)(R_0 - 1)}{k_1(\mu + \nu_2) + k_2 \mu + (R_0 - 1)(k_2 + \nu_1 + \mu)((\mu + \nu_2)\nu_1 + \nu_2 k_2)}.
\]

This ensures the existence of endemic equilibrium point \(E^*\) for \(R_0 > 1\).

3 Stability analysis

In the following subsections, we shall discuss the local and global stability results of disease-free equilibrium point and endemic equilibrium point of the model (2), by using the standard linearization technique and the well-known Routh-Hurwitz criteria\(^{25}\).
3.1 Stability analysis of disease-free equilibrium

**Theorem 1.** The disease-free equilibrium \( E_0 \) of the system (2) is locally asymptotically stable when \( R_0 \leq 1 \).

**Proof.** To study the stability of the system (2) at disease-free equilibrium \( E_0 \), we have calculated Jacobian matrix at disease-free equilibrium \( E_0 \). The three eigenvalues are

\[-\mu, (R_0 - 1)(k_2 + \mu + \nu_1) \text{ and } - (\mu + \nu_2).\]

Clearly, all the eigenvalues are negative for \( R_0 < 1 \), hence the system (2) is locally asymptotically stable around its disease-free equilibrium \( E_0 \).

**Theorem 2.** For \( \frac{k_1}{\mu + \nu_1} < 1 \), disease-free equilibrium \( E_0 \) is globally asymptotically stable.

**Proof.** Consider a Lyapunov function \( \dot{V} = \dot{I} + \dot{A} \). Then

\[
\dot{V} = \frac{k_1SI}{N} - (\mu + \nu_1)I - (\mu + \nu_2)A \\
\leq [k_1 - (\mu + \nu_1)]I - (\mu + \nu_2)A \\
= (\mu + \nu_1)[\frac{k_1}{(\mu + \nu_1)} - 1] - (\mu + \nu_2)A.
\]

For \( \frac{k_1}{\mu + \nu_1} < 1 \), it is easy to obtain \( \dot{V} \leq 0 \). Hence by using LaSalle’s Invariance Principle [32], the global stability of \( E_0 \) can be established.

3.2 Stability analysis of endemic equilibrium

**Theorem 3.** The endemic-equilibrium \( E^* \) of model (2) is locally asymptotically stable whenever it exists.

**Proof.** The Jacobian matrix at \( E^* \) is given as

\[
M = \begin{pmatrix}
-\mu & -\nu_1 & -\nu_2 \\
m_{21} & m_{22} & m_{33} \\
0 & k_2 & -(\mu + \nu_2)
\end{pmatrix}
\]

where,

\[
m_{21} = \frac{\{k_1 - (k_2 + \mu + \nu_1)\}I^*}{N^*}, m_{22} = -\frac{k_1I^*}{N^*}, m_{33} = -\frac{k_1I^*}{N^*}.
\]

The characteristic equation corresponding to the matrix \( M \) at disease-free equilibrium is given as follows:

\[
\lambda^3 + a_2\lambda^2 + a_1\lambda + a_0 = 0,
\]

where

\[
a_2 = 2\mu + \nu_2 + \frac{k_1I^*}{N^*}, \\
a_1 = \left(\mu + \frac{k_1I^*}{N^*}\right)(\mu + \nu_2) + \frac{\nu_1(R_0 - 1)(k_2 + \mu + \nu_1)I^*}{N^*} + \frac{k_1I^*\mu}{N^*} + \frac{k_1k_2I^*}{N^*}, \\
a_0 = (\mu + \nu_2)\left[\frac{\nu_1(R_0 - 1)(k_2 + \mu + \nu_1)I^*}{N^*} + \frac{k_1I^*\mu}{N^*}\right] + k_2\left[\frac{\nu_2(R_0 - 1)(k_2 + \mu + \nu_1)I^*}{N^*} + \frac{k_1I^*\mu}{N^*}\right].
\]

It is clear from above that \( a_2, a_1 \) and \( a_0 \) are positive for \( R_0 > 1 \).

Here we obtain
\[ a_2a_1 - a_0 = (\mu - m_{22})(\mu + \nu_2) + (\nu_1 m_{21} - m_{22} \mu - k_{2} m_{23}) \]
\[ + (\mu - m_{22})(\mu + \nu_2)^2 + \frac{k_{2} \nu_2 (k_2 + \mu + \nu_1) I^*}{N^*}. \]

Clearly, \((a_2a_1 - a_0) > 0\) for \(R_0 > 1\). Thus the equilibrium point \(E^*\) is locally asymptotically stable whenever it exists.

**Theorem 4.** The endemic equilibrium \(E^*\) exists, then it is globally asymptotically stable, provided the following condition is satisfied:

\[
\max \left\{ \frac{2 \nu_1^2}{\mu}, \frac{k_{2} \nu_2^2}{2 \mu (\mu + \nu_2)} \right\} < \frac{\mu}{2}.
\]

**Proof.** Consider the positive definite function about endemic equilibrium \(E^*\)

\[
V = \frac{1}{2} (N - N^*)^2 + l_1 (I - I^*) - I^* \ln \frac{I}{I^*} + l_2 (A - A^*)^2,
\]

where the constant \(k_1, k_2\) and \(k_3\) are to be chosen suitably. The derivative of \(V\) along the solution of the system (2) can be written as

\[
\frac{dV}{dt} = (N - N^*) \frac{dN}{dt} + l_1 \frac{I - I^*}{I} dI + l_2 (A - A^*) \frac{dA}{dt}.
\]

Some algebraic calculation result in simplification of \(\frac{dV}{dt}\), which is given as

\[
\frac{dV}{dt} = - \mu (N - N^*)^2 - l_1 k_1 \frac{I}{N^*} (I - I^*)^2 - l_2 (\mu + \nu_2)(A - A^*)^2 - \left[ \nu_1 - \frac{k_1 (I + A)}{NN^*} \right] (I - I^*) (N - N^*) - \nu_2 (N - N^*)(A - A^*) + (l_2 k_2 - \frac{l_1 k_1}{N^*}) (I - I^*) (A - A^*).
\]

Substituting \(l_2 = \frac{l_1 k_1}{k_2 N^*}\), and thereafter rearranging the terms, the sufficient conditions for \(\frac{dV}{dt}\) to be negative definite come out as follows:

\[
\nu_2 < 2 \mu l_2 (\mu + \nu_2) \Rightarrow l_2 > \frac{\nu_2}{2 \mu (\mu + \nu_2)} \Rightarrow l_1 > \frac{k_{2} \nu_2 N^*}{2 k_1 \mu (\mu + \nu_2)},
\]

\[
\nu_1 < \frac{l_1 k_1}{2 N^*} \Rightarrow l_1 > \frac{2 \nu_1^2 N^*}{\mu k_1}, \left\{ \frac{k_1 (I + A) l_1}{NN^*} \right\}^2 < \frac{\mu l_1 k_1}{2 N^*}.
\]

On maximizing the last inequality, we obtain \(l_1 < \frac{\mu N^*}{2 k_1}\). Thus, the final condition for the negative definiteness of the derivative of the Lyapunov function \(V\) turns out to be

\[
\max \left\{ \frac{2 \nu_1^2 N^*}{\mu k_1}, \frac{k_{2} \nu_2^2 N^*}{2 k_1 \mu (\mu + \nu_2)} \right\} < l_1 < \frac{\mu N^*}{2 k_1}.
\]

Hence, \(l_1\) can be chosen arbitrarily satisfying the above inequality provided the condition stated in the theorem is satisfied. Thus, under the condition (7) the endemic equilibrium point \(E^*\) is globally stable.

### 4 Numerical simulations

The system (2) is simulated numerically for various set of parameter values using XPPAUT\(^{[16]}\). The primary purpose of the numerical simulation is to analyze the change in states of disease progression with time and to check the impact of various parameters on transmission dynamics of the virus. The stability of disease-free equilibrium point \(E_0\) is shown in Figs. 2 and 3, where the reproduction number \(R_0\) is equal to 0.5094 which is less than one and parameter values are as follows:

\[ WJMS email for contribution: submit@wjms.org.uk \]
Fig. 2: Variation of $N$ with time, showing the stability of disease-free equilibrium point $E_0$ for $R_0 = 0.5094$.

Fig. 3: Variation of $I$ and $A$ with time, showing the stability of disease-free equilibrium point $E_0$ for $R_0 = 0.5094$.

\[ \Gamma = 200, \beta_1 = 0.03, \beta_2 = 0.05, \beta_3 = 0.02, \alpha = 0.5, \eta = 0.1, \epsilon = 0.1, \]
\[ \gamma = 0.3, \nu_1 = 0.01, \mu = 0.01666, \tau_1 = 0.005, \tau_2 = 0.01, \delta = 0.4, \nu_2 = 0.05 \]

The phase portraits in N-I plane and N-A plane are shown in Figs. 4 and 5, respectively for the following set of parameter values:

\[ \Gamma = 200, \beta_1 = 0.2, \beta_2 = 0.8, \beta_3 = 0.02, \alpha = 0.5, \eta = 0.1, \epsilon = 0.1, \]
\[ \gamma = 0.3, \nu_1 = 0.01, \mu = 0.01666, \tau_1 = 0.005, \tau_2 = 0.01, \delta = 0.4, \nu_2 = 0.05 \]

For this set of parameter values, the endemic equilibrium point is stable. Here we find that the reproduction number $R_0 = 1.809817$ and the endemic equilibrium point $E^*$ is given by

\[ S^* = 8566.57, I^* = 3359.45, A^* = 473.73. \]
In Figs. 6 and 7, the effect of parameter $\alpha$ is shown. The increase in the value of $\alpha$ shows significant reduction in the equilibrium level of infective population and AIDS patient. Figs. 8 and 9 show the effect of $\gamma$ on infectives and AIDS patients.

Here again the increase in $\gamma$ decreases the equilibrium level of infectives and AIDS patients. Fig. 10 shows the combined effect of $\alpha$ and $\gamma$ where increase in these parameters causes the reduction of the basic reproduction number $R_0$ below one which forces the system towards disease-free equilibrium point. From these simulation, it is clear that general awareness and screening of HIV infectives play considerably important role in reducing the transmission of this disease.

As discussed in Subsection 1.1, sometimes screened infectives develop psychological problems which forces them to transmit this disease to the individuals who come in their contact. In this case the rate of transmission $\beta_3$ will be large as compare to $\beta_1$ and $\beta_2$ and this is an alarming situation for the society leading to increased levels of HIV and AIDS patients. On the other hand, if counseling is 100% effective then $\epsilon$ should be one and if it is not at all effective then should be equal to zero. If counseling is partially effective then $\epsilon$
Fig. 6: Effect of the fraction of individuals who are subject to general awareness $\alpha$ on the equilibrium level of $I$, where all other parameter values are same as used in plotting Fig. 3

Fig. 7: Effect of the fraction of individuals who are subject to general awareness $\alpha$ on the equilibrium level of $A$, where all other parameter values are same as used in plotting Fig. 3

will be somewhere between zero and one. The effects of these parameters on the equilibrium level of HIV and AIDS are shown in Figs. 11 and 12.

5 Conclusion

In this paper, we formulated a mathematical model to investigate the impact of screening of unaware infectives, awareness and treatment of HIV/AIDS infectives on the transmission dynamics of the disease in a homogeneous population with constant immigration of susceptibles. It is observed that with appropriate screening and treatment, lives of many individuals can be changed. In particular, the quality of life of the people living with HIV can be improved. On the population-level, these changes can be framed with coordinated efforts by public health practitioners and with the support of government. The assurance of higher fractions of screening at all clinical settings serving population at risk of HIV can increase the number of
Fig. 8: Effect of the fraction of individuals who are screened $\gamma$ on the equilibrium level of $I$, where all other parameter values are same as used in plotting Fig. 3

Fig. 9: Effect of the fraction of individuals who are screened $\gamma$ on the equilibrium level of $A$, where all other parameter values are same as used in plotting Fig. 3

cases detected and treated, and further it can reduce the harmful HIV consequences. The spread of the disease can be controlled, if the population under consideration presents constructive attitude towards prevention and control measures. The community based educational programs must reach the general public to enhance the awareness about the disease and to extend the prevention and control techniques. Hence, the demand of time is to widen and effectively implement the prevention policies. The global health challenge that has come across us is to provide an overview of the pandemic to the individuals from high risk group that include the health, demographic, social, and economic effects of AIDS and the up-to-date information in prevention, care, and treatment of the disease. To disseminate the knowledge and awareness about this infection, awareness programs/camps incorporating screening of infectives can play a prominent role.

WJMS email for contribution: submit@wjms.org.uk
Fig. 10: Combined effect of $\alpha$ and $\gamma$ on the equilibrium level of $I$ and $A$, where all other parameter values are same as used in plotting Fig. 3

Fig. 11: Effect of $\beta_3$ on the equilibrium level of $I$ and $A$, where all other parameter values are same as used in plotting Fig. 3

References


Fig. 12: Effect of $\epsilon$ on the equilibrium level of $I$ and $A$, with $\beta_3 = 0.2$, where all other parameter values are same as are used in Fig. 3.


WJMS email for contribution: submit@wjms.org.uk


