Modelling the Effect of Irresponsible Infective Immigrants on the Transmission Dynamics of HIV/AIDS

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Abstract

In this paper, a non-linear mathematical model is proposed to study the effect of irresponsible infected immigrants on the spread of HIV/AIDS in a variable size population with a constant recruitment of susceptible. Stability analysis and numerical simulations on the model are presented. The results show that the basic reproductive number, $R_0$, corresponds to a disease free equilibrium, indicating that the disease is under control. The disease becomes endemic if $R_0 > 1$ and thus the disease remain in the population. The analysis further shows that strict immigration policies such as screening, and reduction in the number of immigrants in to a given population could help control the spread of the disease.

Keywords: AIDS epidemic, Vertical transmission, stability, Infective Immigrants, simulation

Introduction

In the world today, immigration poses a significant risk for disease dissemination including HIV/AIDS. The effect may be substantial in developing countries which normally do not check the complete health status of immigrants. These immigrants place their sex partners in their home countries and their destination countries at risk of the HIV/AIDS epidemic. This claim however has not been established and evaluated adequately to see the effect and exact mechanism that immigration contributes in the spread of HIV/AIDS. [13] developed a model to study the impact of migration on the spread of HIV/AIDS in South Africa using observed data. This chapter looks at a nonlinear mathematical model that has been proposed and analyzed...
Mohammed I. Daabo and Baba Seidu to study the effect of infective immigrants in the spread of HIV/AIDS in a population. Human Immunodeficiency Virus (HIV) is the agent that causes Acquired Immunodeficiency Syndrome (AIDS). HIV is transmitted through sexual contact with an infected individual, through exchange of infected blood or blood products, or to the newborn from an infected mother.

HIV infected persons may harbor the virus for many years with no clinical signs of the disease. Eventually, HIV destroys the body’s immune system, mainly by impairing a class of white blood cells whose regulatory activities are essential for immune protection. As a result, people who have AIDS are prone to lung infections, brain abscesses, and a variety of other infections caused by microorganisms that usually do not produce disease in healthy people. Those who have AIDS also are prone to cancers such as Kaposi’s sarcoma, a skin cancer rarely seen in non-HIV-infected populations[8]. HIV/AIDS is one of the most destructive diseases humankind has ever faced, with profound social, economic and public health consequences. Since the beginning of the pandemic over 25 years ago, more than 25 million people have died of AIDS-related illnesses and an estimated 33 million people are now living with HIV[7]. Sub-Saharan Africa remains the most severely affected of the pandemic with an estimated 22.5 million people with HIV, or 68% of the global total, are in Sub-Saharan Africa[3]. The pandemic has cut life expectancy significantly in many countries in sub-Saharan Africa. For example life expectancy in Botswana decreased from 65 years in 1985-1990 to 40 years in 2000-2005[21]. In addition to being a serious public health problem, HIV has far reaching consequences to all social and economic sectors and society. Mathematical models have been used extensively in research into the epidemiology of HIV/AIDS, to help improve our understanding of the main contributing factors in a given epidemic of the disease. According [10] "A mathematical model is a set of formulas or equations based on a quantitative description of a real life or physical problem and created in the hope that the behavior it predicts will resemble the real behavior on which it is based".

[17] considered a simple model on HIV transmission in India. [1] studied on the free formula milk for infants of HIV infected women. [20] looked at the variations in maternal infectivity on mother-to-child transmission of HIV.

[12] considered a structured model for heterosexual disease transmission. [9] studied the mechanism and timing of mother-to-child transmission of HIV infection. [19] also studied a mathematical analysis of HIV dynamics. [16] derived a primary sequence and secondary structures of responsive elements from HIV infective mothers and infants on vertical transmission. [2] considered an estimated timing of mother-to-child HIV transmission by using markov model. [6] studied the transmission of the human immune-deficiency virus through breast-feeding. [18] discussed a variety of diseases that transmit both horizontally and vertically, and gave a comprehensive survey of the formulation and the mathematical analysis of compartmental models that also incorporate vertical transmission. [11] studied the impact of variations in infectiousness by taking into account different levels of virus between individuals during the Chronic phase of infection. [5] studied the impact of condom use on sexual transmission of HIV/AIDS amongst a homogeneously mixing male homosexual population. [14] proposed a model for an infectious disease that
spreads in the host population through both horizontal and vertical transmission. [22] developed a mathematical model for a model community which has the structure of two classes of commercial sex workers and two classes of sexually active male customers with different levels of sexual activity. [4] developed a density dependent HIV transmission model for a Canadian population by taking into account the vertical transmission and by using simple mass action type interaction. [15] studied the spread of HIV infection in a population in the presence of tuberculosis. Research is still going on in the area of modeling. The importance of this is to partner with health experts and policy makers to see how best the spread of the disease can be reduced through medical intervention and behavioral change. This paper seeks to develop a mathematical model to study the impact of irresponsible infective immigrants on the spread of HIV/AIDS and then offers possible intervention strategies.

The model
We consider a population of size $N(t)$ at time $t$ with constant inflow of susceptibles at a rate $Q_0$. The population is subdivided into four classes; Susceptibles, $S(t)$, infectives, Irresponsible infectives $I_1(t)$, responsible infectives $I_2(t)$ and full-blown AIDS patients $A(t)$ with natural mortality rate $d$ in all classes as in figure 1.

The following assumptions are made in the development of the model.

i. The population under study is heterogeneous and varying with time

ii. The population under study is subdivided into four groups

iii. The HIV can only be transmitted through sexual intercourse or through infection from infected needle and blood.

iv. The full-blown AIDS class is sexually inactive

v. The rate at which irresponsible infectives infect people with the disease is higher than that of responsible infectives.

In view of the above assumptions, the spread of the disease is described by the following system of differential equations:

\[
\frac{dS}{dt} = Q_0 - \frac{c(\beta_1 I_1 + \beta_2 I_2)S}{N} - \mu S
\]

\[
\frac{dI_1}{dt} = \frac{c(\beta_1 I_1 + \beta_2 I_2)}{N} S + \gamma I_1 - (\theta + \delta + \mu)I_1
\]

\[
\frac{dI_2}{dt} = \theta I_1 - (\delta + \mu)I_2
\]

\[
\frac{dA}{dt} = \delta(I_1 + I_2) - (\alpha + \mu)A
\]

With initial conditions

$S(0) = S_0, I_1(0) = I_{10}, I_2(0) = I_{20}, A(0) = A_0, \beta_1 > \beta_2$
where:

\( N(t) \) = Total population size at time \( t \).

\( S(t) \) = The size of the Susceptible population at time \( t \).

\( I_1(t) \) = The size of the Irresponsible infective population at time \( t \).

\( I_2(t) \) = The size of the Responsible infective population at time \( t \).

\( A(t) \) = The size of the Full blown AIDS population at time \( t \).

\( c \) = The number of sexual partners an infective individual has.

\( \beta_1 \) = The contact rate of irresponsible infectives.

\( \beta_2 \) = The contact rate of responsible infectives.

\( \mu \) = The natural death rate (Natural mortality rate of an individual in the population).

\( \theta \) = The conversion rate of irresponsible infectives to responsible infectives.

\( \delta \) = The conversion rate of infectives to full-blown AIDS.

\( \alpha \) = The AIDS-induced mortality rate.

\( Q_0 \) = The rate of recruitment of Susceptibles into the population.

\( \gamma \) = Rate of recruitment of infective immigrants into the population.

For clarity sake, we represent \( N(t), S(t), I_1(t), I_2 \) and \( A(t) \) by \( N, S, I_1, I_2 \) and \( A \) respectively.

If the total population size is given by \( N = S + I_1 + I_2 + A \), then we have

\[
\frac{dN}{dt} = (Q_0 - \mu N - \alpha A + \gamma I_1) \quad \text{and the model (1)-(4) is re-written as follows:}
\]

\[
\frac{dN}{dt} = Q_0 - \mu N - \alpha A + \gamma I_1 
\]

\[
\frac{dI_1}{dt} = c(\beta_1 I_1 + \beta_2 I_2)(N - I_1 - I_2 - A) + \gamma I_1 - (\theta + \delta + \mu)I_1 
\]

\[
\frac{dI_2}{dt} = \theta I_1 - (\delta + \mu)I_2 
\]

\[
\frac{dA}{dt} = \delta(I_1 + I_2) - (\alpha + \mu)A 
\]

With initial conditions given by \( N(0) = N_0, I_1(0) = I_{10}, I_2(0) = I_{20}, A(0) = A_0 \), \( \beta_1 > \beta_2 \).
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**Stability Analysis of Model**

The system exhibits two types of equilibriums; disease-free and endemic equilibrium states.

**Disease-free Equilibrium**

At the disease-free equilibrium, there are no infectives and full-blown aids patients.

Hence \( I_1 = I_2 = A = 0 \) and \( N = \frac{Q_0}{\mu} \). Hence the disease-free equilibrium is \( E_0 = \left( \frac{Q_0}{\mu}, 0, 0, 0 \right) \). The Jacobian matrix corresponding to \( E_0 \) is given by

\[
M_0 = \begin{bmatrix}
-\mu & \gamma & 0 & -\alpha \\
0 & c\beta_1 - (\theta + \delta + \mu - \gamma) & c\beta_2 & 0 \\
0 & \theta & -(\delta + \mu) & 0 \\
0 & \delta & \delta & -(\alpha + \mu)
\end{bmatrix}
\]

(9)

The characteristic equation corresponding to \( M_0 \) is given by:

\[
f(\lambda) = (\mu + \lambda)(\alpha + \mu + \lambda)(\lambda^2 + \nu\lambda + \rho)
\]

Where \( \nu = (\delta + \mu) + (\theta + \delta + \mu - \gamma - c\beta_1) \) and \( \rho = (\delta + \mu)(\theta + \delta + \mu - \gamma - c\beta_1) - c\beta_2\theta \)

We note that \( E_0 \) is locally asymptotically stable when \( \nu > 0 \) and \( \rho > 0 \).

However, the condition (10) is sufficient to make \( E_0 \) locally asymptotically stable.

\[
(\delta + \mu)(\theta + \delta + \mu) > (\delta + \mu)(\gamma + c\beta_1) + c\beta_2\theta
\]

(10)

It is clear that for \( R_0 < 1 \) which corresponds to the condition (10), the disease-free equilibrium is locally asymptotically stable so that infection fades out from the population and thus the endemic equilibrium does not exist. However, for \( R_0 > 1 \) \( E_0 \) is unstable and then the infection is maintained in the population.

**Endemic Equilibrium**

At the endemic equilibrium state, the disease persists and the equilibrium point is given by \( E^* = (N^*, I_1^*, I_2^*, A^*) \), where:

\[
N^* = \frac{d(\beta_1(\delta + \mu) + \beta_2\theta)[(\theta + \delta + \mu)(\alpha + \mu + \delta)]}{(\delta + \mu)(\alpha + \mu)\xi} I_1^*
\]

\[
I_1^* = \frac{Q_0(\delta + \mu)(\alpha + \mu)\xi}{c\mu_0(\delta + \mu) + \beta_2\theta\xi[(\theta + \delta + \mu)(\alpha + \mu + \delta) + a\delta(\theta + \delta + \mu)\xi - (\delta + \mu)(\alpha + \mu)\xi\gamma]}
\]

\[
I_2^* = \frac{\theta}{\delta + \mu} I_1^* .
\]
\[ A^* = \frac{\delta(\theta + \delta + \mu)}{(\delta + \mu)(\alpha + \mu)} I_1^* \]

Where \( \xi = c\beta_1(\delta + \mu) + c\beta_2\theta + (\gamma - \theta - \delta - \mu)(\delta + \mu) \)

We note here that \( E^* \) is positive only when \( \xi > 0 \) or \( R_0 > 1 \) and
\[ \alpha\delta(\theta + \delta + \mu) - (\delta + \mu)(\alpha + \mu)\gamma > 0 \]

Where \( R_0 = \frac{(\gamma + c\beta_1)(\delta + \mu) + c\beta_2\theta}{(\theta + \delta + \mu)(\delta + \mu)} \)

**Numerical Simulation**
To observe the dynamics of the system, the model (1)-(4) is numerically integrated using the fourth order Runge-Kutta method using \( \beta_1 = 0.015, \alpha = 0.02, Q_0 = 0.40, \delta = 0.25, \beta_2 = 0.08, \theta = 0.955 \) and \( c = 10 \). With initial conditions \( s(0) = 0.65, i_1(0) = 0.20, i_2(0) = 0.10 \) and \( a(0) = 0.05 \). The results of the computer simulations are graphically displayed in figures 2 to 8. In figures 2, 3 and 4 the irresponsible infectives population, responsible infectives population and AIDS patients population are plotted against the susceptible population respectively for various initial values. It is seen from these figures that irrespective of the initial conditions chosen, the solutions curves all always tend towards the equilibrium point \( E^* \). This implies that the system (5)-(8) is globally stable about the endemic equilibrium point \( E^* \). It is also seen from figure 5 to figure 7 that the presence of infective immigrants in the system increases the total population initially. But as time goes on more susceptible individuals get infected with the disease and then develop into AIDS. They eventually die thereby reducing the total population in the long-run. In the case of figure 7 there are no recruitment of susceptibles into the population. Therefore, the total population, the infected classes and the AIDS class all initially increase but later reduce to zero. Therefore, in order to minimize the spread of the disease and prevent the total population from being wiped away as in the case of figure 7, effective immigration policies such as screening should be put in place to ensure that infected individuals who might act irresponsibly may be denied entry or are exposed so as to minimize their potential of infecing others. It is observed from figure 8 that increasing \( \delta \), the rate of conversion of infectives to full-blown AIDS patients results in an increase in the AIDS patients population in the short term. However, in the long run, increasing \( \delta \) results in a reduction of the AIDS population. That is if infectives become full-blown AIDS patients at a higher rate there will be a reduction in the infectives class and a corresponding increase in the AIDS class. However, as more infectives develop AIDS and pass out of the system(through AIDS-induced death) there will be a reduction in the AIDS class in the long-term.
Conclusion
In this paper, a non-linear mathematical model is proposed to study the effect of irresponsible infective immigrants on the spread of HIV/AIDS in a variable size population with a constant recruitment of susceptible. Stability analysis and numerical simulations on the model show that the basic reproductive number, $R_0 < 1$, corresponds to a disease free equilibrium, indicating that the disease is under control. The disease however becomes endemic if $R_0 > 1$ and thus the disease remain in the population. The analysis further show that strict immigration policies such as screening, and reduction in the number of immigrants in to a given population could help control the spread of the disease. Also certain model parameters such as contact rate and number of sexual partners of infected persons are likely to increase amongst irresponsible infectives and therefore could increase the spread of the disease. In conclusion, we recommend that productive campaign messages be put in place to make people responsible by way of abstaining from unprotected sex, alcoholism, drugs and all other activities that are likely to influence people to making impaired judgment thereby becoming irresponsible.
Figure 3: Variation of Responsible infectives population against Susceptible population

Figure 4: Variation of AIDS patients population against Susceptible population

Figure 5: Variation of population classes with Infective immigrants ($\gamma = 0, Q_0 = 2000$)

Figure 6: Variation of population classes with Infective immigrants ($\gamma = 0.95, Q_0 = 2000$)

Figure 7: Variation of population classes with Infective immigrants ($\gamma = 0.95, Q_0 = 0$)

Figure 8: Variation of Responsible Infective population for different values of $\delta$
References


