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Full Length Research Paper

Modelling the spread of HIV/AIDS epidemic in the presence of irresponsible infectives

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In this study, a non-linear mathematical model was proposed and analyzed to study the effect of irresponsible infectives in the spread of human immunodeficiency virus (HIV)/acquired immunodeficiency syndrome (AIDS) in a variable size population. The population was divided into four subclasses, of susceptibles (HIV negatives who can contract the disease), irresponsible infectives (people who are infected with the virus but do not know or live irresponsible life styles) , responsible infectives (HIV positives who know they are infected and are careful) and full-blown AIDS patients. Susceptibles were assumed to be infected through sexual contact with infectives and all infectives develop AIDS at a constant rate. Stability analysis and numerical simulations of the resulting model are presented. The model analysis shows that the disease-free equilibrium is always locally asymptotically stable and in such a case the basic reproductive number $R_0<1$ and the endemic equilibrium does not exist. The disease is thus eliminated from the system. If $R_0>1$, the endemic equilibrium exists and the disease remains in the system. It is shown that the endemicity of the disease is reduced when irresponsible infectives become responsible.

Key words: Vertical transmission, stability, simulation, irresponsible infectives.

INTRODUCTION

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 (Chin and Lwanga, 1991). 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 (International AIDS Society, 2009). 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 (WHO, 2007). 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 to 1990 to 40 years in 2000-2005 (Sharomi, 2006). In addition to being a serious public health problem, HIV has far reaching consequences to all social and economic sectors and society. It exacerbates poverty,

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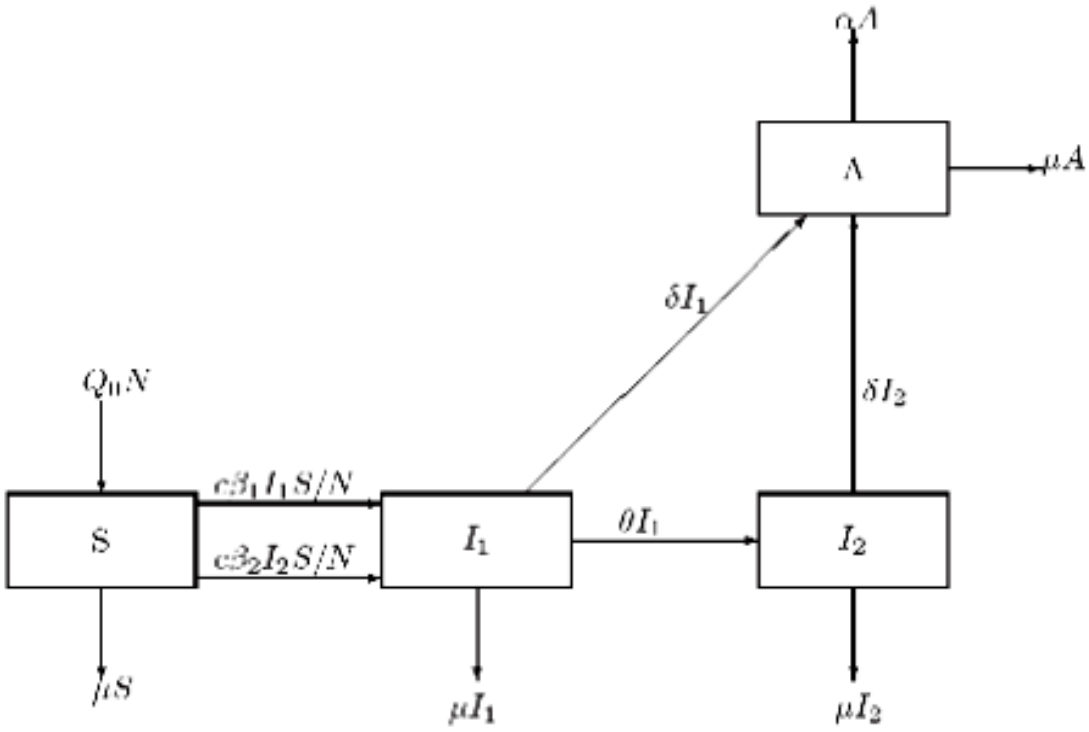

Figure 1. Proposed flow chart for irresponsible infectives model.

reduces educational opportunities, devastates the work force, creates large numbers of orphans and exerts tremendous pressure on the limited health and social services (Sharomi, 2006). For example, HIV/AIDS has cut annual growth rates in Africa by 2 to 4% per year (Dixon et al., 2002). Mathematical models of the transmission dynamics of HIV play an important role in our better understanding of epidemiological patterns for disease control as they provide short and long term predication of HIV/AIDS incidence. Anderson et al. (1986) and Anderson (1988) initial work on modeling saw various refinements being made into modeling frameworks and over the years specific issues have been looked at by researchers. Makinde (2009) studied the transmission dynamics of infectious diseases with waning immunity using the non-purtubative approach. Makinde (2007) again looked at the Adomian decomposition approach to a SIR epidemic model with constant vaccination strategy. Alexander et al. (2006) also studied the effect of the booster vaccination on disease epidemiology. Flessa (1999) developed a model on decision support for malaria control programmes. Mogadas and Gumel (2003) proposed a mathematical model to study childhood diseases with non-permanent immunity. Misra and Mishra (2009) considered the effect of booster vaccination on the transmission dynamics of diseases that spread by droplet infection. Agraj et al. (2006) studied the spread of AIDS epidemic with vertical transmission by considering a non-linear mathematical model. Arazoza and Lounes (2002) considered a non-linear model on sexually transmitted diseases with contact tracing.

This paper seeks to develop a mathematical model to study the impact of irresponsible infectives on the spread of HIV/AIDS infection and then offer possible intervention strategies. The research will also develop a theoretical framework that would predict the possible intervention strategies to prevent the spread of HIV/AIDS infection resulting from infective immigrants. It will provide a numerical solution for non-linear systems of differential equations resulting from the modeling of the impact of irresponsible infectives and infective immigrants on the spread of HIV/AIDS. The aim was to offer both short and long term strategies to control the spread of HIV/AIDS.

The model

We consider a population of size N(t) at time t with constant inflow of suscptibles 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.

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

2. The HIV disease can only be transmitted through sexual intercourse or through infection from an infected needle and blood.
3. The full-blown AIDS group is sexually inactive.
4. 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:

$$\frac{\mathrm{d}S}{\mathrm{d}t} = Q_0 N - \frac{c(\beta_1 I_1 + \beta_2 I_2)S}{N} - \mu S \quad (1)$$

$$\frac{dI_1}{dt} = \frac{c(\beta_1 I_1 + \beta_2 I_2)S}{N} - (\delta + \theta + \mu)I_1 \quad (2)$$

$$\frac{dI_2}{dt} = \theta I_1 - (\delta + \mu)I_2 \quad (3)$$

$$\frac{dA}{dt} = \delta(I_1 + I_2) - (\alpha + \mu)A \quad (4)$$

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

Where, N(t) is the total population size at time t; S(t) is the the size of the Susceptible population at time t; $I_1(t)$ is the the size of the Irresponsible infective population at time t; $I_2(t)$ is the the size of the Responsible infective population at time t; A(t) is the the size of the Full blown AIDS population at time t; c is the the number of sexual partners an infective individual has; β_1 is the the contact rate of irresponsible infectives; β_2 is the the contact rate of responsible infectives; μ is the the natural death rate (Natural mortality rate of an individual in the population); θ is the the conversion rate of irresponsible infectives to responsible infectives; δ is the the conversion rate of infectives to full-blown AIDS; α is the the AIDS-induced mortality rate and Q_0 is the the rate of recruitment of Susceptibles into the population.

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

STABILITY ANALYSIS OF MODEL

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

Disease-free equilibrium

In order to solve the system of equations, we need to normalize the model by defining new variables $s = \frac{S}{N}$, $i_1 = \frac{I_1}{N}$, $i_2 = \frac{I_2}{N}$ and $a = \frac{A}{N}$.

With these new variables, the model becomes:

$$\frac{ds}{dt} = Q_0 - c(\beta_1 i_1 + \beta_2 i_2)s - Q_0 s + \alpha a s \quad (5)$$

$$\frac{di_1}{dt} = c(\beta_1 i_1 + \beta_2 i_2)s - (Q_0 + \delta + \theta)i_1 + \alpha a i_1 \quad (6)$$

$$\frac{di_2}{dt} = \theta i_1 - (Q_0 + \delta)i_2 + \alpha a i_2 \quad (7)$$

$$\frac{da}{dt} = \delta(i_1 + i_2) - (Q_0 + \alpha)a + \alpha a^2 \quad (8)$$

With $s(0) = s_0$, $i_1(0) = i_{10}$, $i_2(0) = i_{20}$, $a(0) = a_0$ and $s + i_1 + i_2 + a \le 1$.

The Jacobian that appropriately linearizes the governing equations of the model is given by

$$J(s,i_1,i_2,a) = \begin{bmatrix} \eta_1 & -c\beta_1 s & -c\beta_2 s & \alpha s \\ c(\beta_1 i_1 + \beta_2 i_2) & \eta_2 & c\beta_2 s & 0 \\ 0 & \theta & \eta_3 & \alpha i_2 \\ 0 & \delta & \delta & \eta_4 \end{bmatrix} \quad (9)$$

where, η_1 is the $-Q_0 - c(\beta_1 i_1 + \beta_2 i_2) + \alpha a$, $\eta_2 = c\beta_1 s - Q_0 - \delta - \theta + \alpha a$, $\eta_3 = -Q_0 - \delta + \alpha a$ and $\eta_4 = -Q_0 - \alpha + 2\alpha a$

We study the system in the closed set $\Gamma = \{(s, i_1, i_2, a) \in R_+^4 | s + i_1 + i_2 + a \le 1\}$.

The system will have either of two long-term behaviour; disease-free equilibrium and the endemic equilibrium. The disease-free equilibrium is attained when there are no infectives and no full-blown AIDS patients. Thus, the disease-free equilibrium will be given by $E_0 = (1,0,0,0)$ When the disease is completely eliminated from the system, the solutions approaches the disease free equilibrium of the form of E_0.

The Jacobian evaluated at E_0 is given by

$$J(E_0) = \begin{bmatrix} -Q_0 & -c\beta_1 & -c\beta_2 & \alpha \\ 0 & c\beta_1 - Q_0 - \delta - \theta & c\beta_2 & 0 \\ 0 & \theta & -Q_0 - \delta & 0 \\ 0 & \delta & \delta & -Q_0 - \alpha \end{bmatrix} \quad (10)$$

The eigenvalue of this jaccobian is the solution of the function:

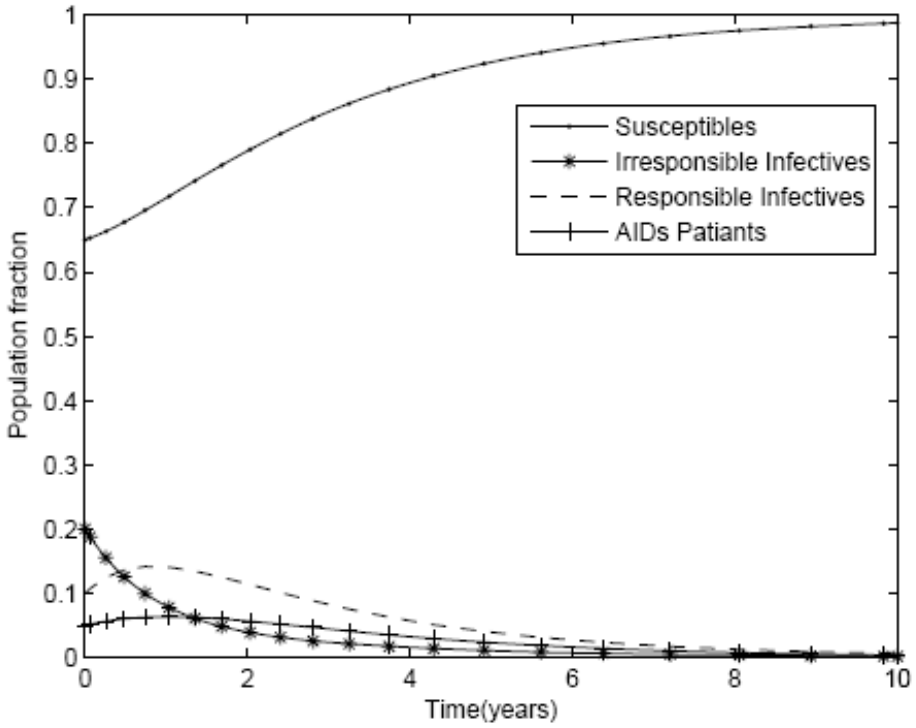

Figure 2. Variation of population in different classes for c = 10, θ = 0.95.

$$f(\lambda) = (Q_0 + \lambda)(Q_0 + \alpha + \lambda)(\lambda^2 + \nu\lambda + \rho) = 0$$

where $\nu = c\beta_1 + 2Q_0 + 2\delta + \theta$ and $\rho = (-c\beta_1 + Q_0 + \delta + \theta)(Q_0 + \delta) - c\beta_2\theta$

The disease-free equilibrium is locally asymptotically stable if v>0 and $\rho > 0$. However, $\rho > 0$ or correspondingly $R_0 = \frac{c\beta_1(Q_0+\delta) + c\beta_2\theta}{(Q_0+\delta+\theta)(Q_0+\delta)} < 1$ is sufficient for E_0 to be locally asymptotically stable.

Endemic equilibrium

Note that $N=S+I1+I_2+A$. Then the model (1) to (4) is given as:

$$\frac{dN}{dt} = (Q_0 - \mu)N - \alpha A \quad (11)$$

$$\frac{dI_1}{dt} = \frac{c(\beta_1 I_1 + \beta_2 I_2)(N - I_1 - I_2 - A)}{N} - (\delta + \theta + \mu)I_1 \quad (12)$$

$$\frac{dI_2}{dt} = \theta I_1 - (\delta + \mu)I_2 \quad (13)$$

$$\frac{dA}{dt} = \delta(I_1 + I_2) - (\alpha + \mu)A \quad (14)$$

Then the endemic equilibrium $E^*=(N^*, I^*_1, I^*_2, A)$ is given given by:

$$N^* = \frac{\alpha\delta(\delta + \mu + \theta)}{\theta(\alpha + \mu)(Q_0 - \mu)} I_2^*$$

$$I_1^* = \frac{(\delta + \mu)}{\theta} I_2^*$$

$$A^* = \frac{\delta(\delta + \mu + \theta)}{\theta(\alpha + \mu)} I_2^*$$

NUMERICAL SIMULATION

To observe the dynamics of the system, the model (5)-(8) is numerically integrated using the fourth order Runge-Kutta method using β_2=0.015,α=0.5, μ=0.02, Q_0=0.40, δ=0.25, β_1=0.08, θ=0.955 and c=10, with initial conditions $s(0)$=0.65, $i_1(0)$=0.20, $i2(0)$=0.10 and $a(0)$=0.05

The results of the computer simulations is graphically displayed in Figures 2 to 9. It is observed from Figures 2 to 5 that increasing θ, the conversion rate of irresponsible infectives to responsible infectives reduces both the

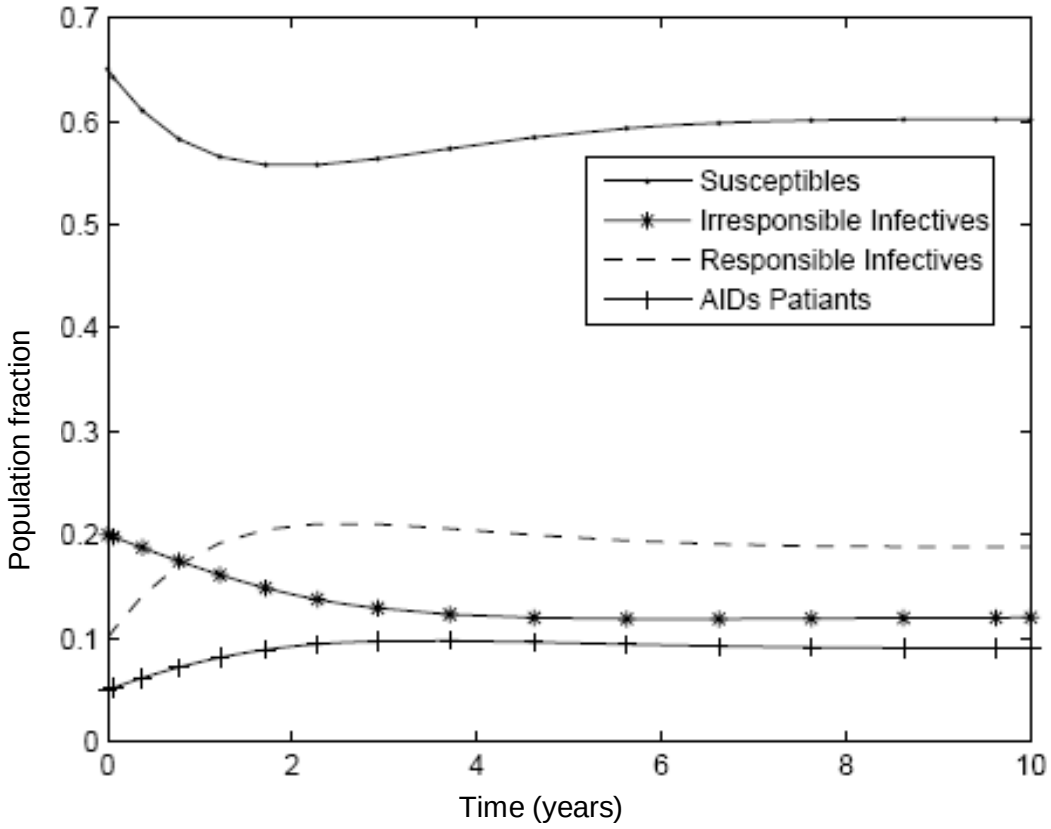

Figure 3. Variation of population in different classes for c = 25, θ= 0.95.

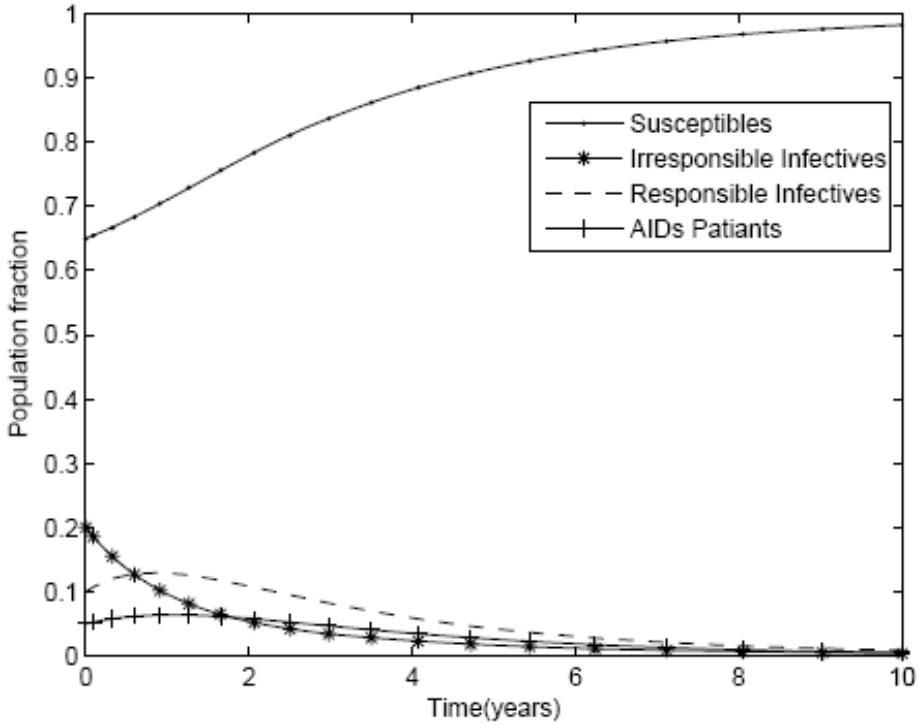

Figure 4. Variation of population in different classes for c = 10, θ =0.75.

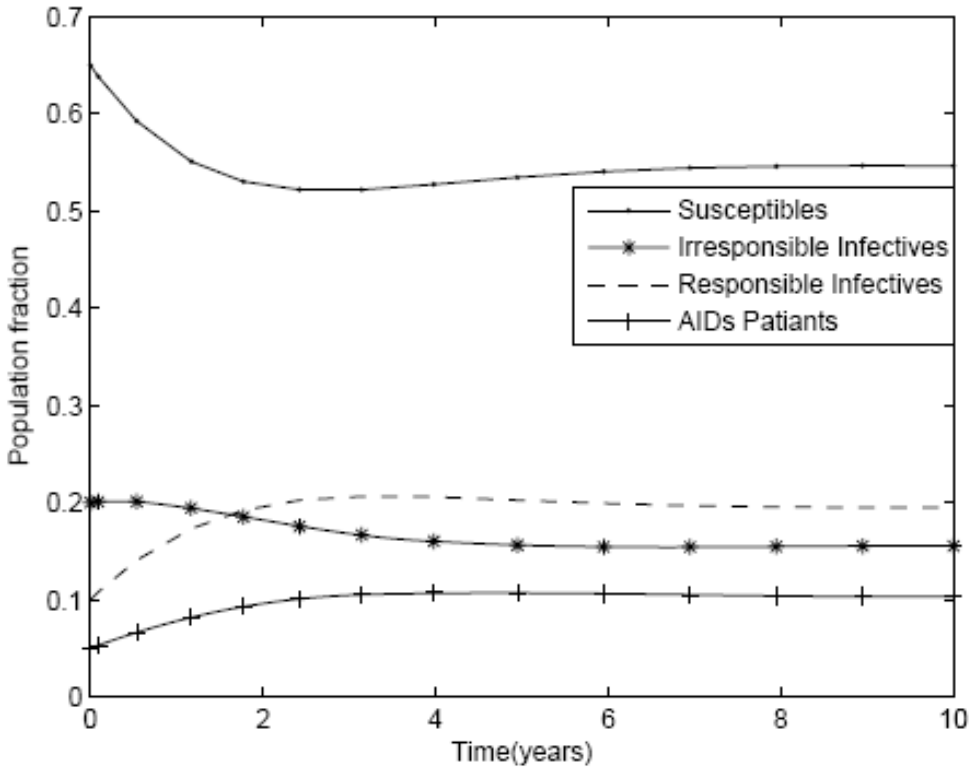

Figure 5. Variation of population in different classes for c = 25, θ = 0.75.

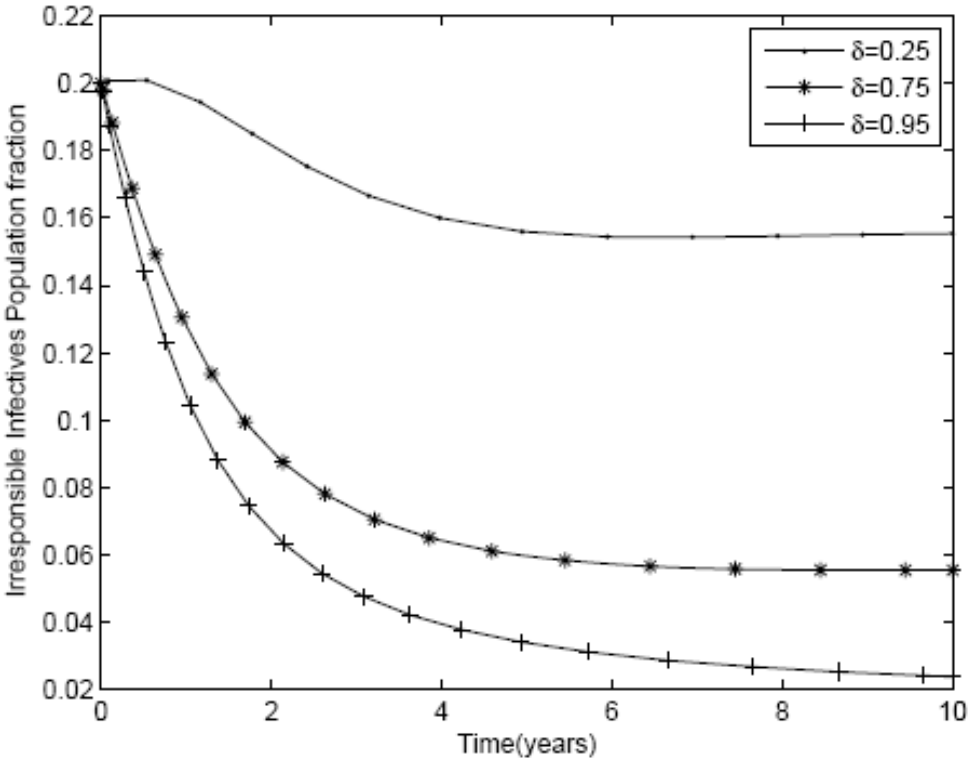

Figure 6. Variation of Irresponsible Infective population for different values of δ.

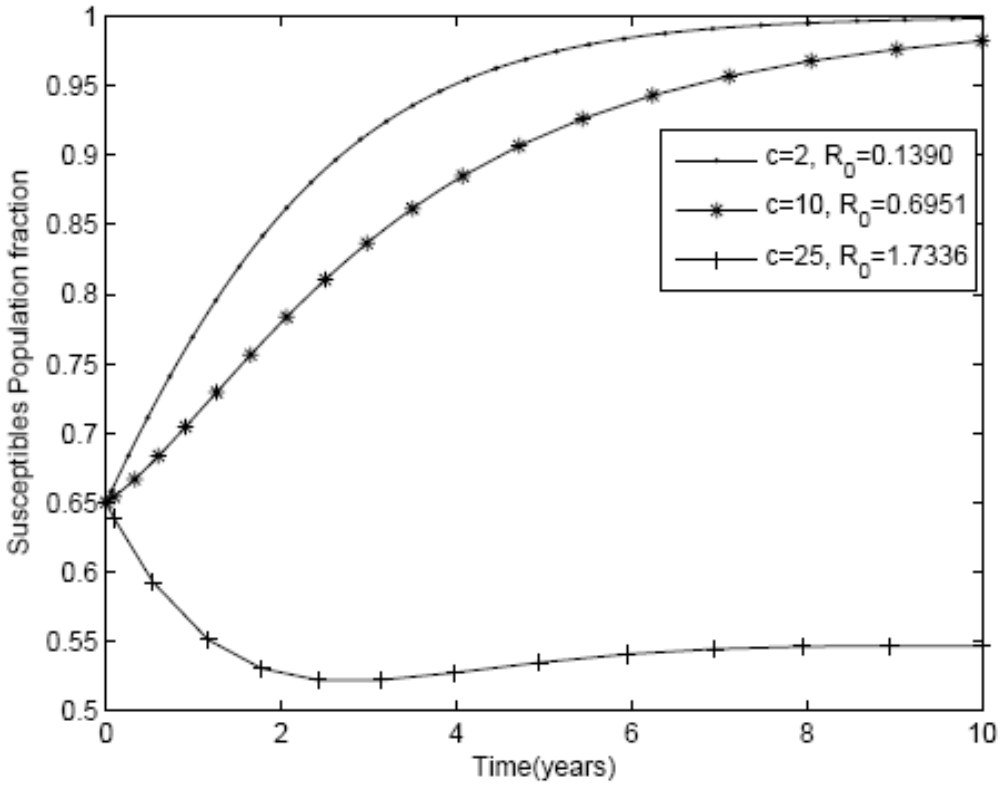

Figure 7. Variation of susceptible population for different values of c.

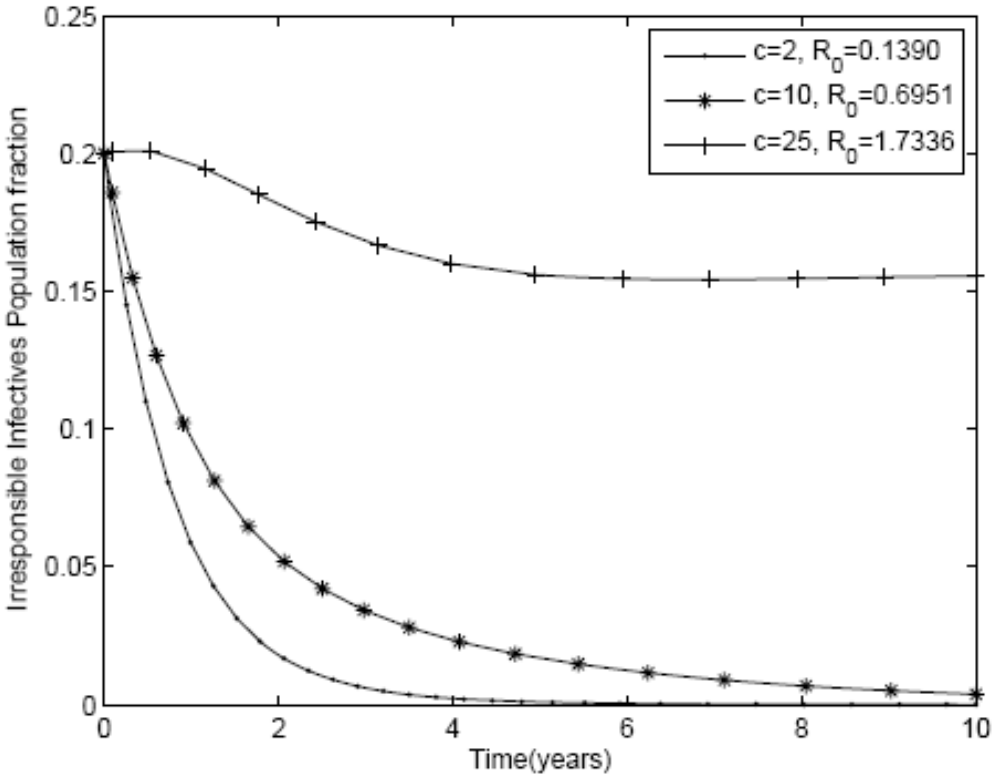

Figure 8. Variation of irresponsible Infective population for different values of c.

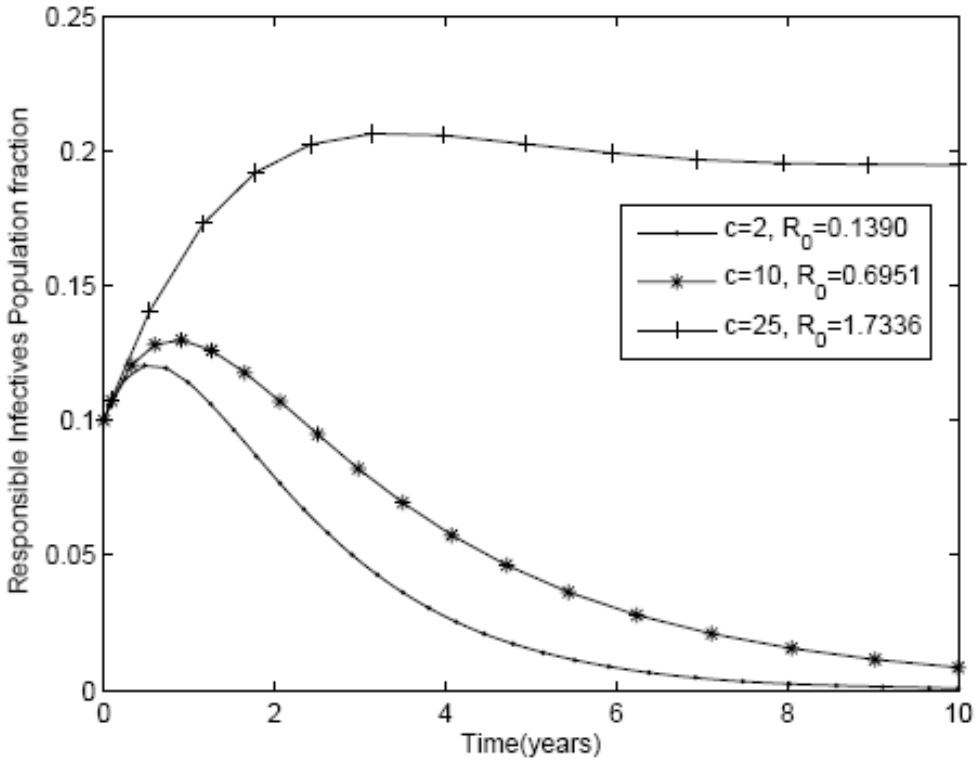

Figure 9. Variation of Responsible Infective population for different values of c.

irresponsible infectives population and AIDS patients population and increases the responsible infectives population. That is, increasing the rate at which irresponsible infectives convert to responsible infectives results in more of the infectives becoming responsible and thus not spreading the disease. This goes to reduce the number of people contracting the disease. Thus, to help check the spread of the disease, policies that can lead to an increase in θ such as mass education, mass screening and the development of strategies that could lead to sexual inactivity of infectives should be considered.

Also, we observed from Figure 6 that increasing δ, the rate at which infectives convert to full-blown AIDS patients reduces the irresponsible infectives populations. Thus, if infectives could be made to quickly develop AIDS, the spread of the disease could be checked and brought under control.

Again, it can be seen from Figures 7 to 9 that increasing c, the number of sexual partners results in a decrease in the susceptible population and an accompanying increase in the infective populations. That is, if the number of sexual partners increases, there will be the tendency for more people to get infected with the virus. This will lead to a reduction in the susceptible population with a concomitant increase in the infective and AIDS populations.

CONCLUSION

In this paper, a non-linear mathematical model is proposed to study the spread of HIV/AIDS in the presence of irresponsible infectives in a variable size population with constant recruitment of susceptibles and infectives. By analyzing the model analytically, a threshold quantity, R_0 is established. It is found that this threshold value determines the endemicity or otherwise of the disease. The model has two (2) equilibria namely; the disease-free equilibrium and the endemic equilibrium. It is found that the disease-free equilibrium is locally asymptotically stable if $R_0<1$, corresponding to the disappearance of the disease from the system. Also, $R_0>1$ shows that the system is unstable and the spread of the disease is maintained in the population.

The computer simulation of the model further shows that a reduction in the number of sexual partners and the contact rate of infectives will greatly reduce the spread of the disease. This can be achieved throughmass education on drug abuse, alcoholism and screening, which will normally make infectives behave irresponsibly.

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