academicJournals

Vol. 5(9), pp. 362-369, September, 2013
DOI: 10.5897/JPHE12.092
ISSN 2141-2316 ©2013 Academic Journals
http://www.academicjournals.org/JPHE

Journal of Public Health and Epidemiology

*Full Length Research Paper*

# Modeling the combined effects of careless susceptible and infective immigrants on the transmission dynamics of HIV/AIDS epidemics

**Mohammed I. Daabo[1], Oluwole D. Makinde[2] and Baba Seidu[3]***

[1]Wisconsin International University College, Accra, Ghana.
[2]Cape Peninsula University of Technology, South Africa.
[3]University for Development Studies, Navrongo, Ghana.

Accepted 14 August, 2013

**In this paper, a non-linear mathematical model was proposed to study the combined effect of irresponsible infectives and irresponsible susceptible immigrants on the spread of human immunodeficiency virus/acquired immune deficiency syndrome (HIV/AIDS) in a variable size population. The paper discussed biological feasibility of the model and also presents the basic reproductive number of the model. Also, the equilibrium points of the model are found, and stability of the model around the equilibria was also studied. It is realized that at the disease free equilibrium, the model is stable when $R_0 < 0$ and unstable otherwise. Also, the condition for asymptotical stability of the model near the endemic equilibrium is presented. Numerical simulations reveal that the presence of infective immigrants significantly affects the spread of the disease and that behavioral change of all classes of individuals should be considered in efforts aimed at controlling the spread of the disease.**

**Key words:** Acquired immune deficiency syndrome (AIDS) epidemic, vertical transmission, stability, infective immigrants, simulation.

## INTRODUCTION

One of the many diseases that have gained attention throughout the world today is the human immunodeficiency virus infection (HIV). It has attracted the attention of many individuals, corporate organizations and governments since its prevalence has greatly increased all over the world, especially Africa. HIV is transmitted through unsafe sexual contact with an infected individual, transfusion with contaminated blood, injection with an infected needle among others. HIV can lead to acquired immunodeficiency syndrome (AIDS) which renders the immune system defenseless to many opportunistic infections.

Mathematicians, among the groups that have taken much interest in the spread of the disease, have proposed many mathematical models that can help in the better understanding of the spread of the disease and the effects of various factors that affect the spread. Since the initial models of Anderson et al. (1986), many other models and their refinements have been proposed by mathematical modelers to study HIV/AIDS (De Arazoza and Lounes, 2002; Busenberg et al., 1995). In particular, Anderson et al., (1986) proposed a model to study the effects of some factors on patterns of AIDS. Naresh and Tripathi (2005) studied the spread of HIV infection in a

*Corresponding author. E-mail: salifubseidu@yahoo.com.

population in the presence of tuberculosis. The effect of use of condoms on the transmission of HIV/AIDS was studied by Greenhalgh et al. (2001). The effect of screening of unaware infectives on the spread of HIV infection was studied by Tripathi et al. (2007). Karrakchou et al. (2006) presented an optimal methodology for administering ant-viral medication therapies to HIV infection. Baryarama et al. (2005) presented an HIV/AIDS model with variable force of infection for the adult population. Ying-Yen and Cooke (2000) studied a model on change of behavior and treatment of core groups and its effect on the spread of HIV/AIDS and found out that change of behavior can help in the control of the spread of the disease.

It is noteworthy here that the attitude towards sex and other modes of transmission of HIV can play a major role in spreading the disease. In the world today, many people, especially in Africa and Asia, are ignorant of their HIV status due to illiteracy despite the many campaigns that encourage people to do the test. These people and those who even though are aware they are infected but behave in ways that will increase the spread of the disease can be considered careless. These careless people can play a very important role in the spread of the disease. Not much research has been done to study the effect of people with different behaviors towards HIV/AIDS on the spread of the canker (Tripathi et al., 2007; Daabo and Baba, 2012; Daabo et al., 2012).

In the present paper, we modeled the combined effect of careless susceptible and infective immigrants on the transmission dynamics of HIV/AIDS. We studied the model analytically and numerically to gain information that could be of benefit in the fight against HIV/AIDS transmission.

## METHODOLOGY

We consider a population of size N(t), which is subdivided into five classes: careful susceptibles, $S_1(t)$, careless susceptibles, $S_2(t)$, careless infectives, $I_1(t)$, careful infectives, $I_2(t)$, and full-blown AIDS patients $A(t)$ with natural mortality rate $\mu$ in all classes number of sexual partners of an infective individual, c, contact rate between a careless infectives and a careful susceptibles, $\beta_1$, contact rate between a careful infectives and a careless Susceptibles, $\beta_2$, contact rate between a careless infectives and a careless Susceptibles, $\beta_3$, contact rate between a careful infectives and a careful Susceptibles, $\beta_4$, rate of AIDS induced death, $\alpha$, immigration rate of careless Susceptibles, $\pi_1$, immigration rate of careless infectives, $\pi_2$, immigration rate of careful infectives, $\pi_3$, conversion rate of careless infectives to full blown AIDS, $\delta_1$, conversion rate of careful infectives to full blown AIDS, $\delta_2$, conversion rate of careless susceptibles to careful susceptibles, $\gamma_1$, conversion rate of careless infectives to careful infectives, $\gamma_2$, natural death rate, $\mu$, and rate of recruitment into the population, $\lambda$ 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 population under study is subdivided into five groups.
3. The HIV can only be transmitted through sexual intercourse or through infection from infected needle and blood.
4. The full-blown AIDS class is sexually inactive.
5. The rate at which careless infectives infect people with the disease is higher than that of careful infectives.
6. The possibility of careless susceptibles contracting the disease is higher than that for careful susceptibles.
7. Change of behavior is positive in the sense that careless individuals tend to become careful at varying degrees but the reverse does not occur.

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

$$\begin{aligned}
\frac{dS_1}{dt} &= (1-\pi_1-\pi_2-\pi_3)\lambda N - \frac{c(\beta_1 I_1+\beta_2 I_2)S_1}{N} + \gamma_1 S_2 - \mu S_1 \\
\frac{dS_2}{dt} &= \pi_1\lambda N - \frac{c(\beta_3 I_1+\beta_4 I_2)S_2}{N} - (\gamma_1+\mu)S_2 \\
\frac{dI_1}{dt} &= \pi_2\lambda N + \frac{c\left[(\beta_1 S_1+\beta_3 S_2)I_1+(\beta_2 S_1+\beta_4 S_2)I_2\right]}{N} - (\gamma_2+\delta_1+\mu)I_1 \\
\frac{dI_2}{dt} &= \pi_3\lambda N + \gamma_2 I_1 - (\delta_2+\mu)I_2 \\
\frac{dA}{dt} &= \delta_1 I_1 + \delta_2 I_2 - (\alpha+\mu)A
\end{aligned} \tag{1}$$

With:

$S_1(0)=S_{10}, S_2(0)=S_{20}, I_1(0)=I_{10}, I_2(0)=I_{20}, A(0)=A_0$ ,(initial conditions), $\beta_4>\beta_3>\beta_1>\beta_2$. For clarity sake, we represent $N(t), S_1(t), S_2(t)\ I_1(t), I_2$ and $A(t)$ by $N$, $S_1$, $S_2$, $I_1$, $I_2$, and $A$, respectively. By introducing $s_1=S_1/N, s_2=S_2/N, i_1=I_1/N, i_2=I_2/N$ and $a=A/N$ and still maintaining the use of the upper case letters, the (1) can be re-written as:

$$\begin{aligned}
\frac{dS_1}{dt} &= (1-\pi_1-\pi_2-\pi_3)\lambda - c(\beta_1 I_1+\beta_2 I_2)S_1 + \gamma_1 S_2 - \mu S_1 \\
\frac{dS_2}{dt} &= \pi_1\lambda - c(\beta_3 I_1+\beta_4 I_2)S_2 - (\gamma_1+\mu)S_2 \\
\frac{dI_1}{dt} &= \pi_2\lambda + c\left[(\beta_1 S_1+\beta_3 S_2)I_1+(\beta_2 S_1+\beta_4 S_2)I_2\right] - (\gamma_2+\delta_1+\mu)I_1 \\
\frac{dI_2}{dt} &= \pi_3\lambda + \gamma_2 I_1 - (\delta_2+\mu)I_2 \\
\frac{dA}{dt} &= \delta_1 I_1 + \delta_2 I_2 - (\alpha+\mu)A
\end{aligned} \tag{2}$$

**Theorem 1:** If $S_1(0), S_2(0), I_1(0), I_2(0)$ and $A(0)$ are non-negative, then so are $S_1(t), S_2(t), I_1(t), I_2(t)$ and $A(t)$ for all $t > 0$. Moreover, $\lim_{t \to \infty} SupN(t) \leq \frac{\lambda}{\mu}$, where $N = (S_1 + S_2 + I_1 + I_2 + A)$. Furthermore, if $N(0) < \frac{\lambda}{\mu}$, then $N(t) < \frac{\lambda}{\mu}, \forall\, t > 0$. In particular, the region;

$$\Gamma = \left\{ (S_1, S_2, I_1, I_2, A) \in \mathbf{R}_+^5 : S_1 + S_2 + I_1 + I_2 + A \leq \frac{\lambda}{\mu} \right\}$$

is positively invariant. This theorem implies that the dynamics of the model 1 can be sufficiently studied in $\Gamma$, in which the model can be considered mathematically and epidemiologically well-posed Hethcote (2000).

### The basic reproduction number, $\mathbf{R_0}$

The basic reproduction is one (in fact it is arguably the most widely used) of the parameters used to study the prevalence of infectious diseases in mathematical modeling. It is used because it gives information about the fate of a typical infectious individual that is introduced into a population that is entirely of susceptibles. It is defined as "the average number of new case of an infection caused by one typical infected individual, in a population consisting of susceptibles only" (Diekmann et al., 2010). It is so important because if $R_0 > 1$ it means, during its infective life-span, a typical infectious individual infects more than one susceptible leading persistence of the disease in the population. If, however, $R_0 < 1$ it means, during its infective life-span, a typical infectious individual infects less than one susceptible leading eradication of the disease in the population. For a history of the evolution of $R_0$, a recipe for its calculation is shown according to Heesterbeek (2002) and Heffernan et al. (2005). For a typical epidemiological model, $R_0$ is normally the largest eigenvalue of the next-generation matrix (Diekmann et al., 2010). With this method we obtained:

$$R_0 = \frac{c\lambda\left[\pi_1\lambda(\sigma_2 - \sigma_1) + \sigma_1(\gamma_1 + \mu)\right]}{\mu(\gamma_1 + \mu)(\gamma_2 + \delta_1 + \mu)}$$, where:

$$\sigma_1 = \beta_1 + \frac{\beta_2\gamma_2}{\delta_2 + \mu} \text{ and } \sigma_2 = \beta_3 + \frac{\beta_4\gamma_2}{\delta_2 + \mu}.$$

### *Equilibria of the model*

In the long-run, there are two scenarios of the model; either the disease is eradicated (then we have a disease-free equilibrium) or the disease remains prevalent in the system (then we have an endemic equilibrium). At the disease-free equilibrium, there are no infectives (that is, $I_1 = I_2 = A = \pi_2 = \pi_3 = 0$) and the equilibrium point is $E_0 = (S_1^0, S_2^0, 0, 0, 0)$,

Where $S_1^0 = \frac{\lambda[\gamma_1 + (1-\pi_1)\mu]}{\mu(\gamma_1 + \mu)}$ $S_2^0 = \frac{\pi_1\lambda}{\gamma_1 + \mu}$

The endemic equilibrium is of the form $E^* = (S_1^*, S_2^*, I_1^*, I_2^*, A^*)$

Where $S_1^* = \frac{\lambda(\delta_2 + \mu)\left[(1 - \pi_1 - \pi_2 - \pi_3)\left[c\beta_4\pi_3\lambda - (\delta_2 + \mu)(\gamma_1 + \mu) + c(\beta_3 + \gamma_2\beta_4)I_1^*\right] + \mu\pi_1(\delta_2 + \mu)\right]}{c\left[\beta_1(\delta_2 + \mu) + \gamma_2\beta_2\right]I_1^* + c\beta_2\pi_3\lambda + \gamma_1(\delta_2 + \mu)}$

$$S_2^* = \frac{\pi_1\lambda(\delta_2 + \mu)}{(\gamma_1 + \mu)(\delta_2 + \mu) - c\beta_4\pi_3\lambda + c\left[\beta_3(\delta_2 + \mu) + \gamma_2\beta_4\right]I_1^*},$$

$$I_2^* = \frac{\pi_3\lambda + \gamma_2 I_1^*}{\delta_2 + \mu},$$

$$A^* = \frac{\delta_2\pi_3\lambda + \left[\delta_1(\delta_2 + \mu) + \delta_2\gamma_2\right]I_1^*}{(\alpha + \mu)(\delta_2 + \mu)}$$ and $I_1^*$ is the positive non-zero real solution of the cubic equation $P(I_1^*) = \eta_3(I_1^*)^3 + \eta_1(I_1^*)^2 + \eta_1(I_1^*) + \eta_0 = 0$

Where:

$$\eta_3 = -c^2\sigma_1\sigma_2(\mu + \gamma_2 + \delta_1)$$

$$\eta_2 = c\sigma_2\sigma_1(\lambda\pi_1 + c\sigma_0) - c(\mu + \gamma_2 + \delta_1)\left[\sigma_1(c\sigma_4 + \mu + \gamma_1) + \sigma_2\mu\right]$$

$$\eta_1 = \sigma_1\gamma_1\lambda\pi_1 + \sigma_2\lambda\pi_1(c\sigma_3 + \mu) + c\sigma_1\sigma_4\lambda\pi_1 + c\sigma_0\sigma_1(c\sigma_4 + \mu + \gamma_1) + c\sigma_0\sigma_2\sigma_3 - (\mu + \gamma_2 + \delta_1)(c\sigma_3 + \mu)(c\sigma_4 + \mu + \gamma_1) + c\lambda\pi_2\left(\sigma_1(c\sigma_4 + \mu + \gamma_1) + \sigma_2\mu\right)$$

$$\eta_0 = \lambda\pi_1\left[\sigma_3\gamma_1 + \sigma_4(c\sigma_3 + \mu)\right] + (c\sigma_4 + \mu + \gamma_1)\left[\sigma_0\sigma_3 + \pi_2\lambda(c\sigma_3 + \mu)\right]$$

$$\sigma_0 = \lambda(1 - \pi_1 - \pi_2 - \pi_3), \qquad \sigma_3 = \frac{\beta_2\pi_3\lambda}{\delta_2 + \mu} \text{ and}$$

$$\sigma_4 = \frac{\beta_4\pi_3\lambda}{\delta_2 + \mu}$$

***Local stability analysis of the model***

To study the local stability of the system (2), we linearize it at the equilibrium points. The Jacobian that linearizes the model is given by:

$$\begin{bmatrix} J_{11} & \gamma_1 & -c\beta_1 S_1 & -c\beta_2 S_1 & 0 \\ 0 & J_{22} & c\beta_3 S_2 & c\beta_4 S_2 & 0 \\ c(\beta_1 I_1 + \beta_2 I_2) & c(\beta_3 I_1 + \beta_4 I_2) & J_{33} & c(\beta_2 S_1 + \beta_4 S_2) & 0 \\ 0 & 0 & \gamma_2 & -\delta_2 - \mu & 0 \\ 0 & 0 & \delta_1 & \delta_2 & -\alpha - \mu \end{bmatrix}$$

Where: $J_{11} = -c(\beta_1 I_1 + \beta_2 I_2) - \mu$, $J_{22} = c(\beta_3 I_1 + \beta_4 I_2) - \gamma_1 - \mu$, and $J_{33} = c(\beta_1 S_1 + \beta_3 S_2) - \gamma_2 - \delta_1 - \mu$

Evaluating the Jacobian matrix at the disease-free equilibrium gives:

$$\begin{bmatrix} -\mu & \gamma_1 & -\frac{c\lambda\beta_1(\mu(1-\pi_1)+\gamma_1)}{\mu(\gamma_1+\mu)} & -\frac{c\lambda\beta_2(\mu(1-\pi_1)+\gamma_1)}{\mu(\gamma_1+\mu)} & 0 \\ 0 & -\gamma_1-\mu & \frac{c\beta_3\pi_1\lambda}{\gamma_1+\mu} & \frac{c\beta_4\pi_1\lambda}{\gamma_1+\mu} & 0 \\ 0 & 0 & J_{33}(E_0) & J_{34}(E_0) & 0 \\ 0 & 0 & \gamma_2 & -\delta_2-\mu & 0 \\ 0 & 0 & \delta_1 & \delta_2 & -\alpha-\mu \end{bmatrix} \quad (3)$$

Where

$$J_{33}(0) = \frac{c\lambda(\beta_1(\mu(1-\pi_1)+\gamma_1)+\beta_3\pi_1\mu)}{\mu(\gamma_1+\mu)} - \gamma_2 - \delta_1 - \mu$$

$$J_{34}(E_0) = \frac{c\lambda(\beta_2(\mu(1-\pi_1)+\gamma_1)+\beta_4\pi_1\mu)}{\mu(\gamma_1+\mu)}$$

The characteristic equation of the Jacobian is given by:

$$f(\sigma) = (-\mu-\sigma)(-\gamma_1-\mu-\sigma)(-\alpha-\mu-\sigma)\left[(-\delta_2-\mu-\sigma)(J_{33}(E_0)-\sigma)-\gamma_2 J_{34}(E_0)\right]$$

All the first three eigenvalues of the Jacobian matrix have negative real parts. The remaining eigenvalues are solutions to the quadratic equation $\sigma^2 + A\sigma + B = 0$, where:

$$A = \frac{\mu(\gamma_1+\mu)(\gamma_2+\delta_1+2\mu+\delta_2) - c\lambda\left[\beta_1((1-\pi_1)\mu+\gamma_1)+\beta_3\pi_1\mu\right]}{\mu(\gamma_1+\mu)}$$

and

$$B = \frac{(\delta_2+\mu)\left[\mu(\gamma_1+\mu)(\gamma_2+\delta_1+\mu) - c\lambda\left[\beta_1((1-\pi_1)\mu+\gamma_1)+\beta_3\pi_1\mu\right]\right]}{\mu(\gamma_1+\mu)} - \frac{c\lambda\gamma_2\left[\beta_2((1-\pi_1)\mu+\gamma_1)+\beta_4\pi_1\mu\right]}{\mu(\gamma_1+\mu)}$$

If $R_0 < 1$, then we have:

$$c\lambda\left[\beta_1(\delta_2+\mu)+\gamma_2\beta_2\right]\left[(1-\pi_1)\mu+\gamma_1\right] + \left[\beta_3(\delta_2+\mu)+\gamma_2\beta_4\right]c\mu\pi_1\lambda < \mu(\gamma_1+\mu)(\delta_2+\mu)(\gamma_2+\delta_1+\mu)$$

$$\Rightarrow c\lambda(\delta_2+\mu)\left[\beta_1((1-\pi_1)\mu+\gamma_1)+\beta_3\pi_1\mu\right] + c\lambda\gamma_2\left[\beta_2((1-\pi_1)\mu+\gamma_1)+\beta_4\pi_1\mu\right] < \mu(\gamma_1+\mu)(\delta_2+\mu)(\gamma_2+\delta_1+\mu)$$

$$\Rightarrow (\delta_2+\mu)\left[\mu(\gamma_1+\mu)(\gamma_2+\delta_1+\mu) - c\lambda\left[\beta_1((1-\pi_1)\mu+\gamma_1)+\beta_3\pi_1\mu\right]\right] - c\lambda\gamma_2\left[\beta_2((1-\pi_1)\mu+\gamma_1)+\beta_4\pi_1\mu\right] > 0.$$

This implies that $\boldsymbol{B} > 0$ and similarly $\boldsymbol{A} > 0$. Thus, if $R_0 < 1$, then $\boldsymbol{A} > 0$ and all eigenvalues of the Jacobian matrix evaluated at the disease-free equilibrium have negative real parts, making the disease-free equilibrium locally asymptotically stable.

If $R_0 < 1$ then the model is locally asymptotically stable at the disease-free equilibrium, if $R_0 > 1$ then the disease-free equilibrium point is an unstable point and if $R_0 = 1$ then the disease-free equilibrium point is a saddle. The Jacobian matrix of the model (1.2) evaluated at the endemic equilibrium is given by:

$$\begin{bmatrix} m_{11} & m & m_{13} & m_{14} & 0 \\ 0 & m_{22} & m_{23} & m_{24} & 0 \\ m_{31} & m_{32} & m_{33} & m_{34} & 0 \\ 0 & 0 & \gamma_2 & -\delta_2-\mu & 0 \\ 0 & 0 & \delta_1 & \delta_2 & -\alpha-\mu \end{bmatrix}$$

Where:

$m_{11} = -m_{31} - \mu < 0$,

$$m_{13} = -c\beta_1 S_1^* < 0, \qquad m_{14} = -c\beta_2 S_1^* < 0,$$
$$m_{22} = m_{32} - \gamma_1 - \mu,\ m_{23} = c\beta_3 S_2^* > 0,\ m_{24} = c\beta_4 S_2^* > 0$$
$$m_{31} = c(\beta_1 I_1 + \beta_2 I_2) > 0,\ m_{32} = c(\beta_3 I_1 + \beta_4 I_2) > 0$$
$$m_{33} = -m_{13} + m_{23} - \gamma_2 - \delta_1 - \mu$$
$$m_{34} = -m_{14} + m_{24} > 0$$

The characteristic equation of the Jacobian at the endemic equilibrium is given by:

$$f(\sigma) = -\sigma^5 + a_1\sigma^4 + a_2\sigma^3 + a_3\sigma^2 + a_4\sigma + a_5$$

Where:

$$a_1 = m_{22} + m_{33} + m_{11} - 2\mu - \alpha - \delta_2$$

$$a_2 = \gamma_2 m_{34} - \mu^2 - (\alpha + \delta_2)\mu - \alpha\delta_2 + (m_{22} + m_{33} + m_{11})(2\mu + \alpha + \delta_2) - m_{11}m_{22} - m_{11}m_{33} - m_{22}m_{33} + m_{32}m_{23} + m_{31}m_{13}$$

$$a_3 = [m_{31}m_{14} + (\alpha + \mu - m_{11} - m_{22})m_{34} + m_{32}m_{24}]\gamma_2 + (m_{22} + m_{33} + m_{11})[(\alpha + \delta_2)\mu + \mu^2 + \alpha\delta_2] +$$
$$[(-m_{22} - m_{33})m_{11} - m_{22}m_{33} + m_{31}m_{13} + m_{32}m_{23}](\alpha + \delta_2 + 2\mu) + m_{31}\gamma_1 m_{23} + (m_{22}m_{33} - m_{32}m_{23})m_{11} - m_{31}m_{22}m_{13}$$

$$a_4 = [m_{32}m_{24} - m_{22}m_{34} - m_{11}m_{34} + m_{31}m_{14}][\alpha\gamma_2 + \mu] + [m_{31}\gamma_1 m_{24} + (m_{22}m_{34} - m_{32}m_{24})m_{11} - m_{31}m_{22}m_{14}]\gamma_2 +$$
$$[(-m_{22} - m_{33})m_{11} + m_{32}m_{23} - m_{22}m_{33} + m_{31}m_{13}][\alpha\mu + \mu^2 + 2\delta_2\mu] +$$
$$[m_{31}\gamma_1 m_{23} + (m_{22}m_{33} - m_{32}m_{23})m_{11} - m_{31}m_{22}m_{13}](\alpha + \delta_2 + 2\mu)$$
$$a_5 = [m_{31}\gamma_1 m_{24} + (m_{22}m_{34} - m_{32}m_{24})m_{11} - m_{31}m_{22}m_{14}](\alpha + \mu)\gamma_2 +$$
$$[m_{31}\gamma_1 m_{23} + (m_{22}m_{33} - m_{32}m_{23})m_{11} - m_{31}m_{22}m_{13}](\alpha + \mu)(\delta_2 + \mu)$$

By the Routh-Hurwitz criterion, the endemic equilibrium point is locally asymptotically stable if:

$$a_i > 0 \forall\ i \in [1, 5],\ a_1a_2a_3 > a_3^2 + a_1^2 \quad \text{and}$$
$$(a_1a_4 - a_5)(a_1a_2a_3 - a_3^2 - a_1^2a_4) > a_5(a_1a_2 - a_3)^2 + a_1a_5^2$$

## RESULTS

To observe the dynamics of the system, we numerically integrate model (2) using the fourth order Runge-Kutta method with the following parameter values:

$c = 2, \alpha = 0.01, \beta_2 = 0.1, \beta_1 = 0.2, \beta_3 = 0.3, \beta_4 = 0.4, \gamma_1 = 0.20, \gamma_2 = 0.3, \delta_1 = 0.1, \delta_2 = 0.4, \mu = 0.01$
, $\lambda = 0.1, \pi_1 = 0.1, \pi_2 = 0.20, \pi_3 = 0.30$,
$s_1 = 0.6, s_2 = 0.2, i_1 = 0.1$ $i_2 = 0.07$ and $a = 0.03$

The results of the computer simulations are graphically displayed in Figures 2 to 15 which are variations of the various groups due to variations of the indicated parameters in the legends.

## DISCUSSION

It is observed from Figures 2 to 9 that the presence of infective immigrants leads to an increase in the possibility of incidence of transmissions of the disease. This leads to a reduction in the number of susceptibles and a corresponding increase in the number of infectives. This can ultimately lead to an increase in the number of AIDS patients in the population. Thus, it is important for policymakers to consider the possibility of controlling inflow of infectives by such methods like screening. Hence, there is the need for effective immigration policies to include the services of the health sector so as to make management of the spread easier.

Also, observation of figures Figures 10 to 12 reveals that, increasing the rate at which irresponsible susceptible individuals become responsible leads to an increase in the responsible susceptibles and a reduction in the irresponsible infectives and susceptibles. The reduction in the irresponsible susceptibles in quite natural but the reduction in the infective class is attributable tothe fact that responsibility of susceptibles plays a role in the responsibility of the infectives. Hence policies aimed at behavioral change should not only target the infected class but also the susceptible class.

Further, Figures 13 to 15 reveal that efforts aimed at controlling the spread through change of behavior should not only target the infected class but also the susceptible class.

## CONCLUSION

We have in this paper proposed a mathematical model to study the combined effect of careless susceptible and infective immigrants on the spread of HIV/AIDS in a policies such as control on the number of careless immigrants into the given population could help control the spread of the disease. Also certain model parameters such as rate at which careless individuals become careful and contact rates among careful individuals and careless

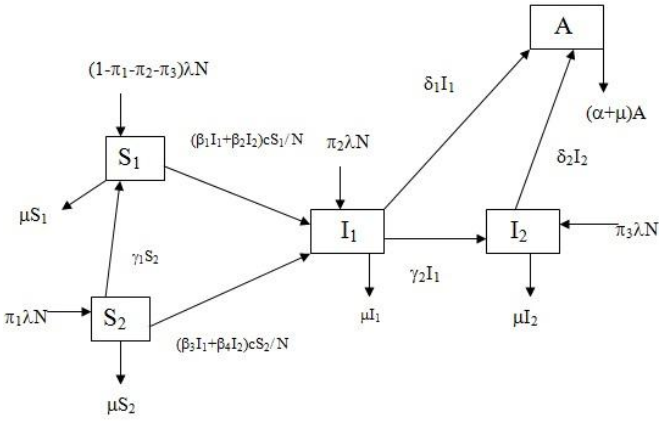


**Figure 1.** Flowchart of proposed model.

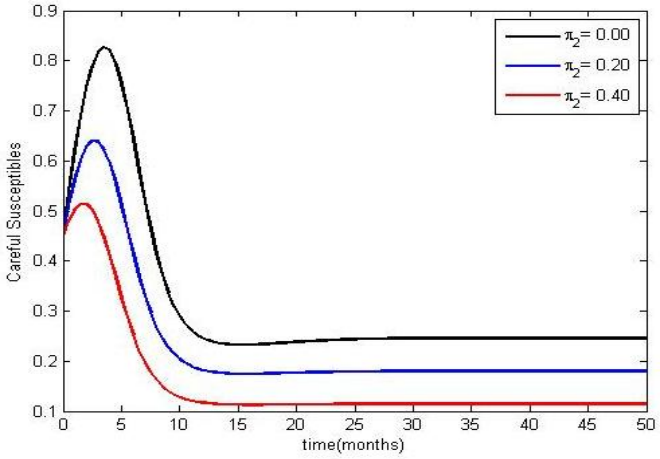


**Figure 2.** Variation of Population of Careful Susceptibles for different values of $\pi_2$

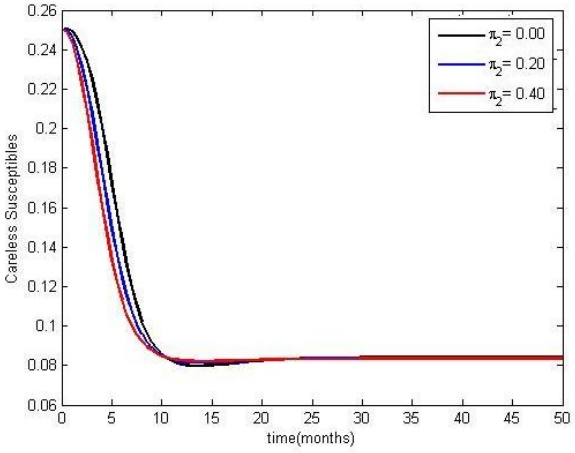


**Figure 3.** Variation of Population of Careless Susceptibles for different values of $\pi_2$

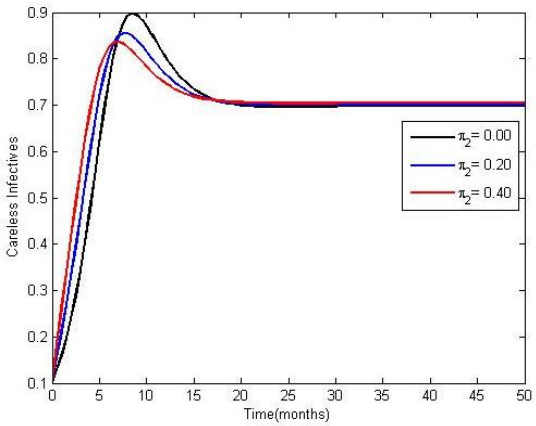


**Figure 4.** Variation of Population of Careless Infectives for different values of $\pi_2$

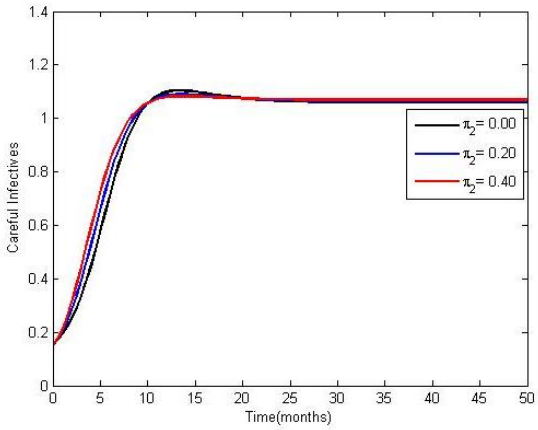


**Figure 5.** Variation of Population of Careful Infectives for different values of $\pi_2$

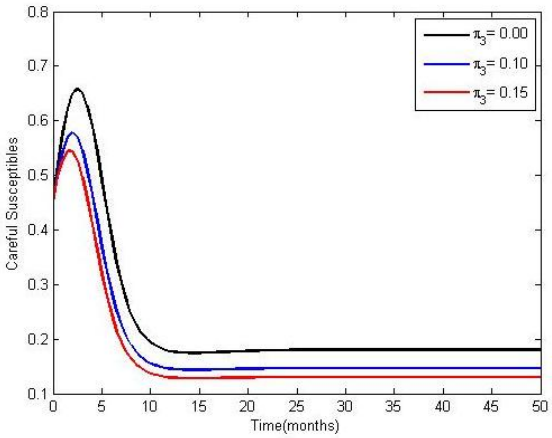


**Figure 6.** Variation of Population of Careful Susceptibles for different values of $\pi_3$

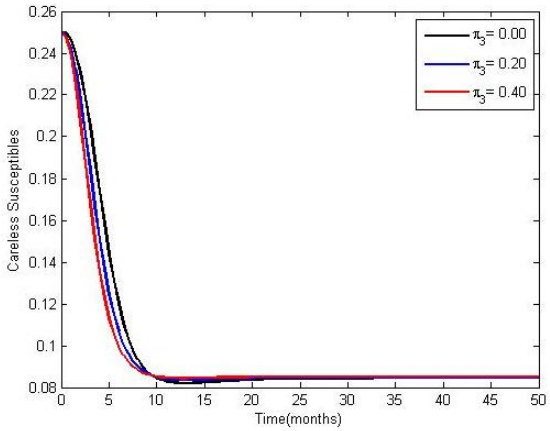


**Figure 7.** Variation of Population of Careless Susceptibles for different values of $\pi_3$

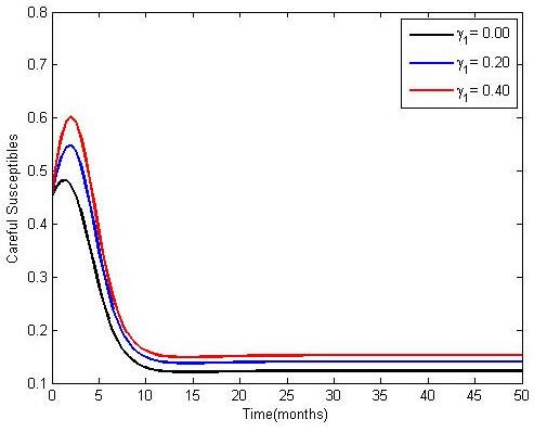


**Figure 10.** Variation of Population of Careful Susceptibles for different values of $\gamma_1$

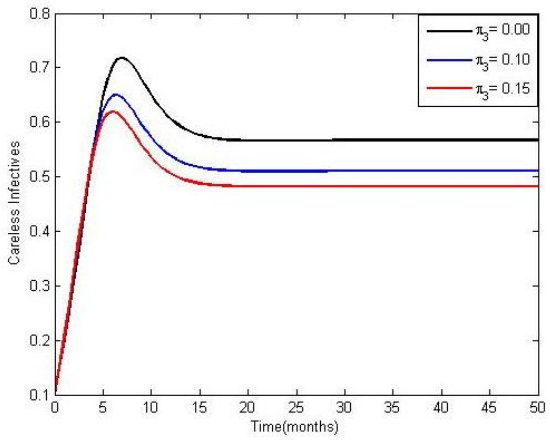


**Figure 8.** Variation of Population of Careless Infectives for different values of $\pi_3$

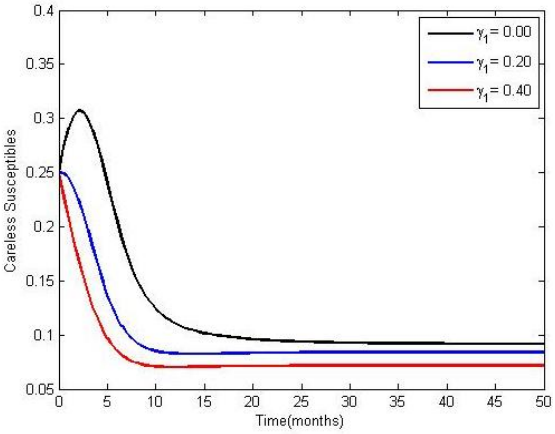


**Figure 11.** Variation of Population of Careless Susceptibles for different values of $\gamma_1$

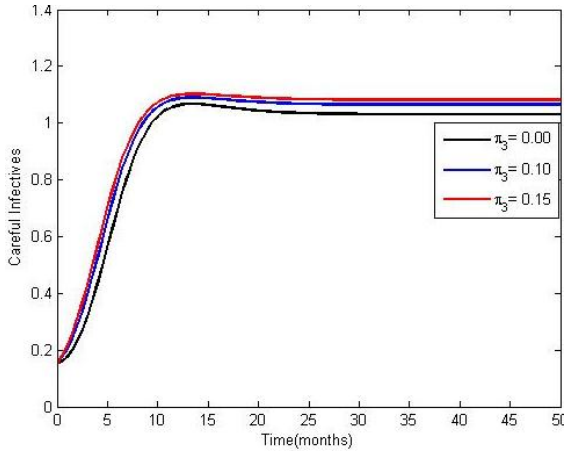


**Figure 9.** Variation of Population of Careful Infectives for different values of $\pi_3$

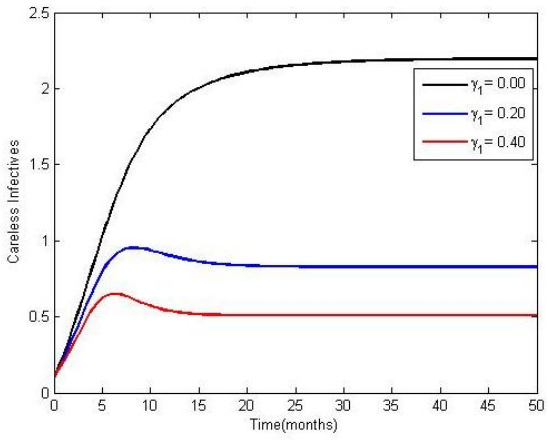


**Figure 12.** Variation of Population of Careless Infectives for different values of $\gamma_1$

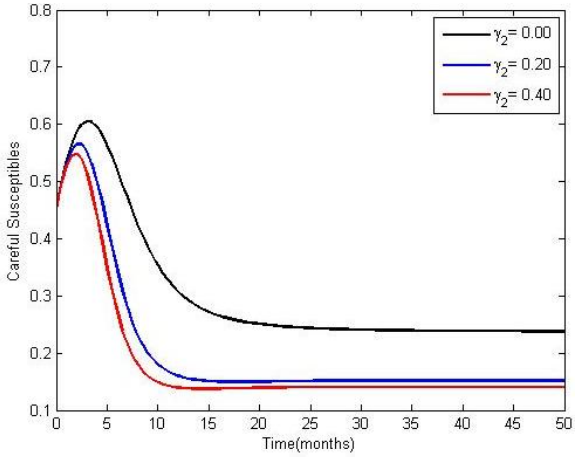


**Figure 13.** Variation of Population of Careful Susceptibles for different values of $\gamma_2$

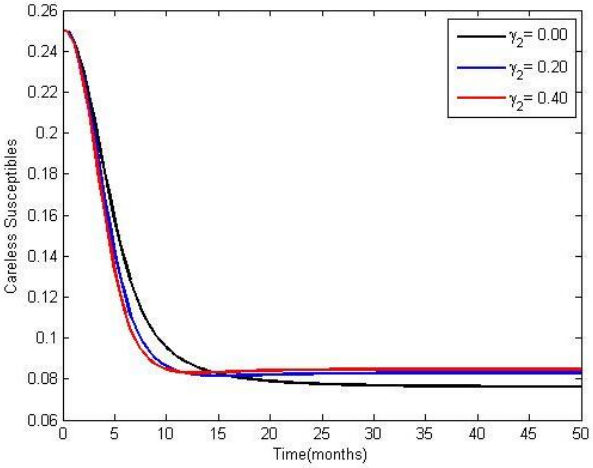


**Figure 14.** Variation of Population of Careless Susceptibles for different values of $\gamma_2$

variable size population. We also presented stability analysis of the model and performed numerical simulations of the model. It is shown 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 when $R_0 > 1$ and thus the disease remains in the population. The analysis further showed that strict immigration individuals are very important factors that play major roles in the spread of the disease. We recommend that productive campaign messages be put in place to make people careful by way of abstaining from unprotected sex, alcoholism, drugs and all other activities that are likely to influence people to make impaired judgment thereby becoming careless.

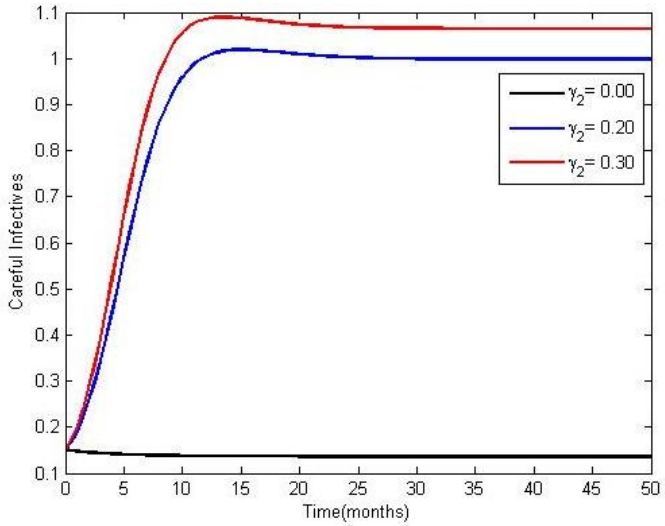


**Figure 15.** Variation of Population of Careful Infectives for different values of $\gamma_2$

**REFERENCES**

Anderson RM, Medley GF, May RM, Johnson AM (1986). A Preliminary study of the transmission dynamics of the human immunodeficiency virus (HIV) the causative agent of AIDS. IMA J. Math. Appl. med. Biol. 3(4):229-263.

Baryarama F, Mugisha JY, Luboobi LS (2005). An HIV/AIDS model with variable force of infection and its application to the epidemic in Uganda. Am. J. Appl. Sci. *2*(9):1274-1278.

Busenberg S, Cooke K, Hsieh YH (1995). A model for HIV in Asia. Math. Biosci. 128(1-2):185-210.

Daabo MI, Baba S (2012). Modelling the Effect of Irresponsible Infective Immigrants on the Transmission Dynamics of HIV/AIDS. Advan. Appl. Math. Biosci. *3*(1):31-40.

Daabo MI, Makinde OD, Baba S (2012). Modelling the spread of HIV/AIDS epidemic in the presence of irresponsible infectives. Afr. J. Biotechnol. 11(51):11287-11295.

De Arazoza H, Lounes R (2002). A nonlinear model for sexually transmitted diseases with contact tracing. IMA J. Math. Appl. Med. Biol. 9(2):221-234

Diekmann O, Heesterbeek JA, Roberts MG (2010). The construction of next-generation matrices for compartmental epidemic models. J. R. Soc. Interfac. 7(47):873-885.

Greenhalgh D, Doyle M, Lewis F (2001). A mathematical treatment of AIDS and condom use. IMA J. Math. Appl. Med. Biol. 18:225-262.

Heesterbeek JA (2002). A breif history of $R_{0}$ and a recipe for its calculation. Acta Biotheor. 50:189-204.

Heffernan JM, Smith RJ, Wahl LM (2005). Perspectives on the basic reproductive ratio. J. R. Soc. Interfac. *2*:281-293.

Hethcote HW (2000). The mathematics of infectious diseases. SIAM Rev. 42(4):599-653.

Karrakchou J, Rachik M, Gourari S (2006). Optimal control and infectiology: Application to an HIV/AIDS model. Appl. Math. Comput. 177:807-818.

Naresh R, Tripathi A (2005). Modeling and analysis of HIV-TB co-infection in a variable size population. Math. Model. Anal. 10:275-286.

Tripathi A, Naresh R, Sharma D (2007). Modelling the effect of screening of unaware infectives on the spread of HIV infection. Appl. Math. Comput. 184:1053-1068.

Ying-Yen H, Cooke K (2000). Behavior change and treatment of core groups: its effect on the spread of HIV/AIDS. IMA J. Math. Appl. Med. Biol. 17:213-241.