

Available online at http://scik.org

J. Math. Comput. Sci. 5 (2015), No. 1, 72-90

ISSN: 1927-5307

A MATHEMATICAL MODEL TO STUDY THE IMPACT OF IRRESPONSIBLE INFECTIVE IMMIGRANTS AND VERTICAL TRANSMISSION ON THE DYNAMICS OF HIV/AIDS

MADUBUEZE, E. CHINWENDU\*, NWAOKOLO, A. MARTINS AND GWERYINA, I. REUBEN

Department of Mathematics/Statistics/Computer science, University of Agriculture, P.M.B 2373, Makurdi, Nigeria Copyright © 2015 Madubueze, Nwaokolo and Gweryina. This is an open access article distributed under the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.

**Abstract:** This paper examined the combined effects of irresponsible infective immigrants and vertical transmission in a varying population. A mathematical model for the problem was proposed and transformed into proportions in order to define the prevalence of infection. Using the next-generation method, the basic reproduction number  $R_0$  was computed in terms of the parameters of the transformed model. The disease free equilibrium was obtained and found to be locally asymptotically stable when  $R_0 < 1$  and unstable for  $R_0 > 1$ . By the method of the centre manifold theory, the existence of transcritical bifurcation was investigated. The study ascertained that forward bifurcation existed if certain conditions were met. Numerical simulation of the model was carried out to assess the effect of irresponsible HIV infective immigrants and vertical transmission in the spread of HIV/AIDS disease. The result showed that screening and counselling the irresponsible HIV infective immigrants will help to reduce the spread of HIV and prevent MTCT.

**Keywords:** irresponsible infective immigrants; the basic reproduction number; backward bifurcation; vertical transmission; responsible infectives; HIV/AIDS.

2010 AMS Subject Classification: 93A30.

1.0 Introduction

Human Immunodeficiency Virus (HIV) is a single-stranded RNA virus which uses host DNA as an intermediary for its own replication. It is unable to replicate on its own and must first infect a living cell in order to replicate. HIV is a retroviral disease that was first discovered in 1981 in the United State of America [1]. The virus is the causative agent of Acquired Immunodeficiency Syndrome (AIDS).

\*Corresponding author

Received October 23, 2014

72

AIDS is a chronic, potentially life threatening condition caused by HIV by destroying the immune system of the individual leading to opportunistic infections, neurological manifestations and rare CNS malignancies. It is the sixth leading cause of death among people aged 25-44 years in the United State of America. As at the end of 2012, the World Health Organization (WHO) estimates that around 35.2 million people in the world are living with HIV. More than 60% of this global estimate resides in Sub-Sahara Africa (SSA) which has been most severely affected by the HIV/AIDS pandemic with almost 9% of its adult population living with HIV [2, 3]. HIV/AIDS is a sexually transmitted infectious disease which can also be spread through contact with infected blood, blood products and body fluids. HIV/AIDS has had profound social, economic and public health consequences in SSA. It has cut down annual growth rates in Africa by 2 to 4% per year [4].

According to [5], sexual intercourse alone accounts for over 80% of reported cases of HIV infection. The prevalence of HIV infection among irresponsible infective (e.g. truck drivers and female sex workers e.t.c) was carried out by [6]. Most of the truck drivers have wives and other sexual partners who are always at risk of HIV infection.

Vertical transmission of HIV/AIDS also called Mother-to-Child transmission (MTCT) occurs when the virus is spread from an HIV positive woman to her baby. The transmission from mother to child may occur in uterus, at the time of birth, or after birth. The risk of transmission can be as high as 90%, especially in developing countries with varying population. An estimated 220,000 exposed children are born each year. Without prevention of mother-to-child transmission (PMTCT), about 88,000 of these are infected whereas with PMTCT only 2% (4,400) are infected. Nigeria has 30% of the global burden of MTCT [7]. It is therefore imperative to consider the dynamics of vertical transmission in the spread of HIV/AIDS disease.

Several researchers have developed interest in HIV/AIDS in order to understand and explain the dynamics and spread of the disease. Thus, many mathematical models as well as methods of analysing them were proposed. The work in [8,9] studied the effect of careless susceptible, infective immigrants and irresponsible infectives in varying population, while some studied the spread of HIV/AIDS with vertical transmission [10,11,12].

In this paper, we propose a variate of the model by [8] who developed a non-linear mathematical model to study the impact of irresponsible infective immigrants on the spread of HIV/AIDS.

They established that screening and reduction in the number of immigrants into a given population could help control the spread of the disease.

The model by [8] forms the motivation for this study in which we intend to investigate the effect of irresponsible infective and vertical transmission on the dynamics of HIV/AIDS.

### 2.0 Model Formulation

Let N(t) be the total population of size at time t. The population N(t) is divided into four classes namely; susceptible S(t), irresponsible infectives,  $I_1(t)$ , responsible infectives  $I_2(t)$  and full-blown AIDS patients A(t) with natural mortality rate  $\mu$  in all classes and  $\alpha$  as the disease induced death rate in the AIDS patients class. We assumed that susceptibles are recruited at the rate bN and become infected through sexual contacts with the infectives  $I_1(t)$  and  $I_2(t)$  at rate  $\lambda_t$ , where

$$\lambda_t = \frac{c_1 \beta_1 I_1 + c_2 \beta_2 I_2}{N} \tag{1}$$

 $c_1$  represents the mean number of sexual partners of the individuals in  $I_1(t)$  and  $c_2$  for the class of individuals in  $I_2(t)$  while  $\beta_1$  and  $\beta_2$  are the respective sexual contact rates of the  $I_1(t)$  and  $I_1(t)$  classes. Note in our proposed model  $c_1 > c_2$  unlike the model in [8]. This is because irresponsible infectives are likely to have more sexual partners than responsible infectives as in the case of sex workers and truck drivers. It is assumed that the sexual contact between susceptible S(t) and irresponsible infective  $I_1(t)$  may lead to the birth of infected children with a fraction  $(1 - \varepsilon)$  while the complementary fraction  $\varepsilon$  dies during birth due to the infection. The responsible infective is conscious of infected newborns because of the counselling and awareness of HIV/AIDS disease. We assumed also direct recruitment of irresponsible infectives (immigrants) with the rate  $\gamma$ . Some irresponsible infectives progress to responsible infectives after going through counselling at the rate  $\theta$  while some  $I_1(t)$  and  $I_2(t)$  progress to AIDS class with rates  $\delta_1$  and  $\delta_2$  respectively. The full-blown AIDS class is assumed to be sexually inactive due to their illness. Where  $\varepsilon$  is fraction of newborns that are HIV free and  $\phi$  is the birth rate of newborns infected with HIV by irresponsible infectives. Irresponsible infectives are either those who may not be aware of their status or even if they are aware, they do not adhere to medication and counselling on risk reduction while responsible infectives are those who know their status and adhere to medication and counselling on risk reduction and MTCT.

Based on the above assumptions, the governing system of differential equations for the spread of the disease is given by

$$\frac{ds}{dt} = bN - \lambda_t S - \mu S \tag{2}$$

$$\frac{dI_1}{dt} = \lambda_t S + \gamma I_1 - (\theta + \delta_1 + \mu)I_1 + (1 - \varepsilon)\phi I_1 \tag{3}$$

$$\frac{dI_2}{dt} = \theta I_1 - (\delta_2 + \mu) I_2 \tag{4}$$

$$\frac{dA}{dt} = \delta_1 I_1 + \delta_2 I_2 - (\alpha + \mu) A \tag{5}$$

with initial conditions

$$S(0) = S_0$$
,  $I_1(0) = I_{10}$ ,  $I_2(0) = I_{20}$ , and  $A(0) = A_0$ 

The total population N(t) is given as

$$N(t) = S(t) + I_1(t) + I_2(t) + A(t)$$

This implies that

$$\frac{dN(t)}{dt} = \frac{dS}{dt} + \frac{dI_1}{dt} + \frac{dI_2}{dt} + \frac{dA}{dt}$$

or

$$\frac{dN}{dt} = (b - \mu)N + (\gamma + (1 - \varepsilon)\phi)I_1 - \alpha A \tag{6}$$

Now, in the above system (2) - (5), we use the following transformations:

$$s = \frac{s}{N}$$
,  $i_1 = \frac{I_1}{N}$ ,  $i_2 = \frac{I_2}{N}$ ,  $a = \frac{A}{N}$ 

to get the following normalised system:

$$\frac{ds}{dt} = b - bs - c_1 \beta_1 s i_1 - c_2 \beta_2 s i_2 - \gamma s i_1 - (1 - \varepsilon) \phi i_1 s + \alpha a s \tag{7}$$

$$\frac{di_1}{dt} = c_1 \beta_1 s i_1 + c_2 \beta_2 s i_2 - (\theta + \delta_1 + b) i_1 + (1 - \varepsilon) \phi i_1 (1 - i_1) + (\alpha \alpha + \gamma - \gamma i_1) i_1$$
 (8)

$$\frac{di_2}{dt} = \theta i_1 - (\delta_2 + b)i_2 + (\alpha a - \gamma i_1 - (1 - \varepsilon)\phi i_1)i_2 \tag{9}$$

$$\frac{da}{dt} = \delta_1 i_1 + \delta_2 i_2 - (\alpha + b)a + (\alpha a - \gamma i_1 - (1 - \varepsilon)\phi i_1)a \tag{10}$$

and

$$s(0) = s_0$$
,  $i_1(0) = i_{10}$ ,  $i_2(0) = i_{20}$  and  $a(0) = a_0$ 

## 3.0 Positivity of Solutions

For the model to be epidemiological meaningful and well posed, we need to prove that all state variables are all non-negative.

### Theorem 1.0

Let  $\Omega = \{(s, i_1, i_2, a) \in R_4^+: s + i_1 + i_2 + a = 1 \text{ and } s(0) > 0, i_1(0) \ge 0, i_2(0) \ge 0 \text{ and } a(0) \ge 0\}$ , then the solution  $\{s(t), i_1(t), i_2(t), a(t)\}$  of the equations (7)-(10) are all non-negative for all  $t \ge 0$ .

### **Proof**

Using equation (7) for  $\frac{ds}{dt}$ , we have

$$\frac{ds}{dt} \ge -(b + c_1\beta_1i_1 + c_2\beta_2i_2 + \gamma i_1 + (1 - \varepsilon)\phi i_1)s$$
 or

$$\frac{ds}{dt} \ge -\varphi s$$
, where  $\varphi = (b + c_1\beta_1i_1 + c_2\beta_2i_2 + \gamma i_1 + (1 - \varepsilon)\phi i_1)$ 

Integrating both sides and applying the initial condition  $s(0) = s_0$ , we have

$$s(t) \ge s_0 e^{-\varphi t}$$

It is clear that, as  $t \to \infty$ 

$$s(t) \ge 0$$
 since  $s(0) > 0$ 

For  $\frac{di_1}{dt}$ , we have

$$\frac{di_1}{dt} \ge -(\theta + \delta_1 + b)i_1$$

or

$$\frac{di_1}{i_1} \ge -(\theta + \delta_1 + b)dt$$

Integrating both sides, we have

$$i_1(t) \ge Ae^{-(\theta+\delta_1+b)t}$$

Applying the initial condition  $i_1(0) = i_{10}$ , we obtain

$$i_1(t) \ge i_{10}e^{-(\theta + \delta_1 + b)t}$$

and  $i_1(t) \ge 0$  as  $t \to \infty$ .

Similarly,

$$\frac{di_2}{dt} \ge -(\delta_2 + b)i_2$$

from which we obtain

$$i_2(t) \ge i_2(0)e^{-(\delta_2 + b)t}$$

and

 $i_2(t) \ge 0$  as t approaches infinity.

Furthermore,

$$\frac{da}{dt} \ge -(\alpha + b)a$$

or

$$a(t) \ge a_0 e^{-(\alpha+b)t}$$

which gives

$$a(t) \ge 0$$
 as  $t \to \infty$ .

Clearly, this proves the above result in theorem 1.0.

# 3.1 Disease - Free Equilibrium (DFE)

The disease – free equilibrium is the equilibrium where there is no disease in the population.

At equilibrium point,  $\frac{ds}{dt} = \frac{di_1}{dt} = \frac{di_2}{dt} = \frac{da}{dt} = 0$ , we have the following systems of equations to be solved for equilibrium points.

$$b - bs - c_1 \beta_1 s i_1 - c_2 \beta_2 s i_2 - \gamma s i_1 - (1 - \varepsilon) \phi i_1 s + \alpha a s = 0$$
(11)

$$c_1 \beta_1 s i_1 + c_2 \beta_2 s i_2 - (\theta + \delta_1 + \mu) i_1 + (1 - \varepsilon) \phi i_1 (1 - i_1) + (\alpha \alpha + \gamma - \gamma i_1) i_1 = 0$$
 (12)

$$\theta i_1 - (\delta_2 + b)i_2 + (\alpha a - \gamma i_1 - (1 - \varepsilon)\phi i_1)i_2 = 0$$
(13)

$$\delta_1 i_1 + \delta_2 i_2 - (\alpha + b)a + (\alpha a - \gamma i_1 - (1 - \varepsilon)\phi i_1)a = 0$$

$$\tag{14}$$

For disease – free,  $i_1 = i_2 = a = 0$ 

We have when substituted in above equations

$$b - bs = 0$$

implies s = 1 provided  $b \neq 0$ 

Thus, the disease – free equilibrium  $E_0$  is

$$E_0 = (1,0,0,0)$$

### 3.2 Stability of DFE

We shall compute the basic reproductive number  $R_0$  using the next– generation method. The basic reproductive number is a threshold quantity used to study the prevalence of an infectious

disease in epidemiological model. According to [13], the basic reproduction number  $R_0$  is the spectral radius (i.e the dominant eigenvalue) of the next generation matrix. It is given as

$$R_0 = \rho(GU^{-1})$$

where  $\rho(GU^{-1})$  is the spectral radius of the matrix  $GU^{-1}$  given as

$$GU^{-1} = \left[\frac{\partial F_i(x_0)}{\partial x_j}\right] \left[\frac{\partial V_i(x_0)}{\partial x_j}\right]^{-1}$$

 $F_i$  is rate of appearance of new infection in compartment i

 $V_i$  is the transfer of individuals in and out of compartment i by another means and  $x_0$  is the disease free equilibrium.

Using the next generation method, the system of differential equation (7) - (10) are rearrange in the order of the infected compartments first, then the uninfected compartment. We have three infected compartments namely,  $i_1$ ,  $i_2$  and a whereas s as uninfected compartment.

$$\frac{di_1}{dt} = (c_1\beta_1i_1 + c_2\beta_2i_2)s - (\theta + \delta_1 + b)i_1 + (1 - \varepsilon)\phi i_1(1 - i_1) + (\alpha a + \gamma - \gamma i_1)i_1 
\frac{di_2}{dt} = \theta i_1 - (\delta_2 + b)i_2 + (\alpha a - \gamma i_1 - (1 - \varepsilon)\phi i_1)i_2 
\frac{da}{dt} = \delta_1 i_1 + \delta_2 i_2 - (\alpha + b)a + (\alpha a - \gamma i_1 - (1 - \varepsilon)\phi i_1)a 
\frac{ds}{dt} = b - bs - c_1\beta_1 si_1 - c_2\beta_2 si_2 - \gamma si_1 - (1 - \varepsilon)\phi i_1 s + \alpha as$$

from which F, V, G, U, and  $GU^{-1}$  given as

$$F = \begin{bmatrix} c_1 \beta_1 i_1 + c_2 \beta_2 i_2 \\ 0 \\ 0 \\ 0 \end{bmatrix}, V = \begin{bmatrix} (\theta + \delta_1 + b)i_1 - (1 - \varepsilon)\phi i_1 (1 - i_1) - (\alpha a + \gamma - \gamma_i)i_1 \\ -\theta i_1 + (\delta_2 + b)i_2 - (\alpha a - \gamma i_1 - (1 - \varepsilon)\phi i_1)i_2 \\ -\delta_1 i_1 - \delta_2 i_2 + (\alpha + b)a - (\alpha a - \gamma i_1 - (1 - \varepsilon)\phi i_1)a \\ -b + bs + c_1 \beta_1 s i_1 + c_2 \beta_2 s i_2 + \gamma s i_1 + (1 - \varepsilon)\phi i_1 s - \alpha as \end{bmatrix}$$

$$G = \begin{bmatrix} \frac{\partial f_1(x_0)}{\partial i_1} & \frac{\partial f_1(x_0)}{\partial i_2} & \frac{\partial f_1(x_0)}{\partial a} \\ \frac{\partial f_2(x_0)}{\partial i_1} & \frac{\partial f_2(x_0)}{\partial i_2} & \frac{\partial f_2(x_0)}{\partial a} \\ \frac{\partial f_3(x_0)}{\partial i_1} & \frac{\partial f_3(x_0)}{\partial i_2} & \frac{\partial f_3(x_0)}{\partial a} \end{bmatrix} = \begin{bmatrix} c_1\beta_1 & c_2\beta_2 & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix}$$

$$U = \begin{bmatrix} \frac{\partial v_1(x_0)}{\partial i_1} & \frac{\partial v_1(x_0)}{\partial i_2} & \frac{\partial v_1(x_0)}{\partial a} \\ \frac{\partial v_2(x_0)}{\partial i_1} & \frac{\partial v_2(x_0)}{\partial i_2} & \frac{\partial v_2(x_0)}{\partial a} \\ \frac{\partial v_3(x_0)}{\partial i_1} & \frac{\partial v_3(x_0)}{\partial i_2} & \frac{\partial v_3(x_0)}{\partial a} \end{bmatrix} = \begin{bmatrix} (\theta + \delta_1 + b) - (1 - \varepsilon)\phi - \gamma & 0 & 0 \\ -\theta & (\delta_2 + b) & 0 \\ -\delta_1 & -\delta_2 & (\alpha + b) \end{bmatrix}$$

and

$$GU^{-1} = \begin{bmatrix} \frac{c_1\beta_1(\delta_2+b)c_2\beta_2\theta}{h(\delta_2+b)} & \frac{c_2\beta_2}{\delta_2+b} & 0\\ 0 & 0 & 0\\ 0 & 0 & 0 \end{bmatrix}$$

where

$$h = (\theta + \delta_1 + b) - (1 - \varepsilon)\phi - \gamma \tag{*}$$

We find the eigenvalue of  $GU^{-1}$  as

$$|GU^{-1} - \lambda I| = 0$$

This gives

$$\lambda_1 = \lambda_2 = 0 \text{ or } \lambda_3 = \frac{c_1 \beta_1 (\delta_2 + b) c_2 \beta_2 \theta}{h(\delta_2 + b)}$$

Thus, the spectral radius of  $GU^{-1}$  is given by

 $R_0 = max[|\lambda_1|, |\lambda_2|, |\lambda_3|]$ , this implies

$$R_0 = \frac{c_1 \beta_1 (\delta_2 + b) + c_2 \beta_2 \theta}{h(\delta_2 + b)}$$

or

$$R_0 = \frac{c_1 \beta_1(\delta_2 + b) + c_2 \beta_2 \theta}{(\theta + \delta_1 + b - (1 - \varepsilon)\phi - \gamma)(\delta_2 + b)}$$
(15)

If  $R_0 < 1$ , the infection dies out, otherwise the infection will be maintained in the population. From the equation (15), we have  $R_0$  increases as the number of sexual partners of  $i_1(t)$  and  $i_2(t)$  increase. This implies that  $R_0$  can be kept at minimum if there is a restriction on the number of sexual partners of  $i_1(t)$  and  $i_2(t)$ .

**Theorem 2:** The disease – free equilibrium of the system of ODE (5) – (8) is locally asymptotically stable if  $R_0 < 1$  and unstable if  $R_0 > 1$ .

The theorem 2 is prove using linearization method. The Jacobian matrix associated with the system (7)-(10) at the DFE  $E_0 = (1,0,0,0)$  is

$$J(E_0) = \begin{bmatrix} -b & -c_1\beta_1 - \gamma - (1-\varepsilon)\phi & -c_2\beta_2 & \alpha \\ 0 & c_1\beta_1 - h & c_2\beta_2 & 0 \\ 0 & \theta & -(\delta_2 + b) & 0 \\ 0 & \delta_1 & \delta_2 & -(\alpha + b) \end{bmatrix}$$

and the characteristics equation corresponding to  $J(E_0)$  is given by

$$\rho(\lambda) = (-b - \lambda)[(c_1\beta_1 - h - \lambda)(\delta_2 + b + \lambda)(\alpha + b + \lambda) + c_2\beta_2\theta(\alpha + b + \lambda)] = 0$$

or

$$\rho(\lambda) = (-b - \lambda)[\lambda^3 + A\lambda^2 + B\lambda + C] = 0$$

Where h is defined as in (\*) and

$$A = -[c_1\beta_1 - h - (\delta_2 + b) - (\alpha + b)]$$

$$B = -[(c_1\beta_1 - h)(\delta_2 + b) + c_2\beta_2\theta + c_1\beta_1 - h - (\delta_2 + b)(\alpha + b)]$$

$$C = -[(c_1\beta_1 - h)(\delta_2 + b)(\alpha + b) + c_2\beta_2\theta(\alpha + b)]$$

Using Routh–Hurwitz criteria,  $E_0$  is locally asymptotically stable if A > 0, B > 0, C > 0, and AB > C.

We have

$$C = -[(c_1\beta_1 - h)(\delta_2 + b)(\alpha + b) + c_2\beta_2\theta(\alpha + b)] > 0$$

This implies that

$$(c_1\beta_1-h)(\delta_2+b)(\alpha+b)+c_2\beta_2\theta(\alpha+b)<0$$

and get

$$\frac{c_1\beta_1(\delta_2+b)+c_2\beta_2\theta}{h(\delta_2+b)}<1$$

Therefore  $R_0 < 1$ . This proofs the theorem 2.

# 3.3 Existence of Backward Bifurcation

Some models can be bi-stable due to vaccination or immunity [14, 15] such that  $R_0 < 1$  is not a sufficient condition to eradicate the disease that is endemic in the population but sufficient for avoiding an epidemic caused by few infectives introduced initially in the population [16]. Some models [17, 18] being bi-stable is due to the change of stability that occurs at the bifurcation point, whereas bifurcation point (that is, a point where the leading eigenvalue of the Jacobian matrix at the DFE is zero) will occur whenever  $R_0 = 1$ . We have forward and backward bifurcation depending on the direction of the bifurcation. Bifurcation at  $R_0 = 1$  is forward when DFE is locally asymptotically stable for  $R_0 < 1$  and there is no disease while the endemic

equilibrium, is locally stable when  $R_0 > 1$ . Backward bifurcation occurs when the endemic equilibrium exists for  $R_0 < 1$  and DFE may exist when  $R_0 > 1$ . In backward bifurcation, we expect disease to invade at  $R_0 = 1$  but not in the case forward bifurcation [19]. We determine the direction of bifurcation (forward or backward) for the model (7-10) by using centre manifold theorem by [20] around the bifurcation point  $R_0 = 1$  in a neighbourhood of the DFE  $x^*$ .

Theorem 3: Centre manifold theory [20].

Consider a general system of ODEs with the parameter  $\beta$ :

$$\frac{dx}{dt} = F(x, \beta) \tag{16}$$

$$f: R \to R^n$$
 and  $f \in C^2(R^2 \times R)$ 

Where 0 is an equilibrium point for the system (7-10) for all values of the parameter  $\beta$ , that is  $f(0,\beta) \equiv 0$  for all  $\beta$  and

(1) 
$$A = D_* f(0,0) = \left[ \frac{df_i}{dx_j}(0,0) \right]$$

is the linearization point 0 with  $\beta$  evaluate at 0. Zero is a simple eigenvalue of A and all other eigenvalues of A have negative real parts.

(2) Matrix A has a non negative right eigenvector w and a left eigenvector v corresponding to the zero eigenvalue.

Let  $f_k$  be the  $k^{th}$  component of f and

$$a = \sum_{k,i,j=1}^{n} v_k w_i w_j \frac{\partial^2 f_k}{\partial x_i \partial x_j} (0,0)$$

$$b = \sum_{k,i=1}^{n} v_k w_i \frac{\partial^2 f_k}{\partial x_i \partial \beta} (0,0)$$

Then the local dynamics of the system (16) around the equilibrium point 0 is totally determined by the signs of a and b.

- i. a > 0, b > 0 when  $\beta < 0$  with  $|\beta| \ll 1$ , 0 is locally asymptotically stable and there exists a positive unstable equilibrium; when  $0 < \beta \ll 1$ , 0 is unstable and there exists a negative and locally asymptotically stable equilibrium.
- ii. a < 0, b < 0, with  $|\beta| \ll 1$ , 0 unstable; when  $0 < \beta \ll 1$ , asymptotically stable, and there exists a positive unstable equilibrium;

- iii. a > 0, b < 0, with  $|\beta| \ll 1$ , 0 unstable; and there exists a locally asymptotically stable negative equilibrium; when  $0 < \beta \ll 1$ , 0 is stable and a positive unstable equilibrium appears;
- iv. a > 0, b < 0, when  $\beta$  changes from negative equilibrium to positive, 0 changes its stability from stable to unstable, corresponding to a negative equilibrium becomes positive and locally asymptotically stable.

Particularly, if a > 0 and b > 0, then a subcritical (or backward) bifurcation occurs at  $\beta = 0$ . Applying the theorem 3 involves the following changes of variables; Let

$$s = x_1$$
,  $i_1 = x_2$ ,  $i_2 = x_3$ ,  $a = x_4$ 

with

$$x_1 + x_2 + x_3 + x_4 = 1$$
.

Let  $X = (x_1, x_2, x_3, x_4)^T$  be the vector written so that the model can be re-written in the form  $\frac{dX}{dt} = F(x)$ , where  $F = (f_1, f_2, f_3, f_4)^T$  as follows

$$\frac{dx_1}{dt} = f_1(x) = b - bx_1 - c_1\beta_1 x_1 x_2 - c_2\beta_2 x_1 x_3 - \gamma x_1 x_2 - (1 - \varepsilon)\Phi x_1 x_2 + \alpha x_1 x_4 \tag{17}$$

$$\frac{dx_2}{dt} = f_2(x) = (c_1\beta_1 x_2 + c_2\beta_2 x_3)x_1 - (\theta + \delta_1 + b)x_2 + (1 - \varepsilon)\Phi x_2(1 - x_2) + (\alpha x_4 + \gamma - \gamma x_2)x_2 \tag{18}$$

$$\frac{dx_3}{dt} = f_3(x) = \theta x_2 - (\delta_2 + b)x_3 + (\alpha x_4 - \gamma x_2 - (1 - \varepsilon)\Phi x_2)x_3 \tag{19}$$

$$\frac{dx_4}{dt} = f_4(x) = \delta_1 x_2 + \delta_2 x_3 - (\alpha + b)x_4 + (\alpha x_4 - \gamma x_2 - (1 - \varepsilon)\Phi x_2)x_4 \tag{20}$$

The Jacobian matrix of the equations (7) - (10) at the disease-free equilibrium  $J(E_0)$  is defined in previous section. Taking  $\beta_1 = \beta$  and  $\beta_2 = r\beta$ , where  $\beta$  is chose as the bifurcation parameter and the bifurcation occurs at  $R_0 = 1$ , we consider the case  $R_0 = 1$  and solve for the bifurcation parameter  $\beta$ .

We have

$$R_0 = \frac{c_1 \beta_1 (\delta_2 + b) + c_2 \beta_2 \theta}{(\theta + \delta_1 + b - (1 - \varepsilon) \phi - \gamma)(\delta_2 + b)} = 1$$

or

$$\frac{c_1\beta(\delta_2+b)+c_2r\beta\theta}{h(\delta_2+b)}=1$$

from which we obtain

$$\beta = \frac{(\delta_2 + b)h}{c_1(\delta_2 + b) + rc_2\theta}$$

where h is as defined in (\*).

The Linearized system of the transformed system (7) - (10) with  $\beta_1 = \beta$  and  $\beta_2 = r\beta$  has a simple zero eigenvalue. Hence, we analyze the dynamics of (7-10) at  $\beta_1 = \beta$  and  $\beta_2 = r\beta$  using the Centre Manifold theory. The Jacobian matrix of (7) - (10) has right eigenvector associated with the zero eigenvalue as

$$\begin{bmatrix} -b & -c_{1}\beta_{1} - \gamma - (1 - \varepsilon)\phi & -c_{2}\beta_{2} & \alpha \\ 0 & c_{1}\beta_{1} - h & c_{2}\beta_{2} & 0 \\ 0 & \theta & -(\delta_{2} + b) & 0 \\ 0 & \delta_{1} & \delta_{2} & -(\alpha + b) \end{bmatrix} \begin{bmatrix} w_{1} \\ w_{2} \\ w_{3} \\ w_{4} \end{bmatrix} = \begin{bmatrix} 0 \\ 0 \\ 0 \\ 0 \end{bmatrix}$$
(21)

where  $w = (w_1, w_2, w_3, w_4)^T$  is the right eigenvector.

Evaluating the system in (21) gives

$$w_3 = \frac{\theta}{\delta_2 + b} w_2, w_4 = \frac{m_1}{m_3} w_2, w_1 = \frac{m_1 - m_2}{b m_3} w_2$$

where

$$m_1 = \delta_1(\delta_2 + b) + \delta_2\theta$$
,  $m_3 = (\delta_2 + b)(\alpha + b)$ ,  $m_2 = (\theta + \delta_1 + b)m_3$ 

The left eigenvector of the Jacobian  $J(E_0)$  associated with the zero eigenvalue is given by  $V = (v_1, v_2, v_3, v_4)^T$ . Transposing Jacobian  $J(E_0)$  first and multiply by V, we have

$$\begin{bmatrix} -b & 0 & 0 & 0 \\ -c_1\beta_1 - \gamma - (1-\varepsilon)\varphi & c_1\beta_1 - h & \theta & \delta_1 \\ -c_2\beta_2 & c_2\beta_2 & -(\delta_2+b) & \delta_2 \\ \alpha & 0 & 0 & -(\alpha+b) \end{bmatrix} \begin{bmatrix} v_1 \\ v_2 \\ v_3 \\ v_4 \end{bmatrix} = \begin{bmatrix} 0 \\ 0 \\ 0 \\ 0 \end{bmatrix}$$

from which we get

$$v_1 = v_4 = 0, v_3 = \frac{c_2 \beta_2}{\delta_2 + b} v_2$$

Using the property w.v = 1, we obtain

$$w = \left(w_1, \frac{1}{c_2\beta_2\theta + (\delta_2 + b)^2}, \frac{\theta}{(\delta_2 + b)(c_2\beta_2\theta + (\delta_2 + b)^2)}, w_4\right) \text{ and } v = \left(0, (\delta_2 + b)^2, \frac{c_2\beta_2}{(\delta_2 + b)}, 0\right)$$

### 3.4 Computations of a and b

From the system (17-20), the associated non-zero partial derivative of F at DFE for  $v_2$ ,  $v_3$  with the expression that  $x_1 = 1 - x_2 - x_3 - x_4$  are given by

$$\begin{split} &\frac{\partial^2 f_2}{\partial x_2 \partial x_3} = -(c_1 \beta_1 + c_2 \beta_2) \ , \ \frac{\partial^2 f_2}{\partial x_2 \partial x_4} = -c_1 \beta_1 + \alpha \ , \quad \frac{\partial^2 f_2}{\partial x_2^2} = -2(c_1 \beta_1 + (1-\varepsilon)\Phi + \gamma), \ \frac{\partial^2 f_2}{\partial x_3^2} = \\ &-2c_2 \beta_2, \ \frac{\partial^2 f_2}{\partial x_3 \partial x_4} = -c_2 \beta_2 \ , \ \frac{\partial^2 f_3}{\partial x_2 \partial x_3} = -((1-\varepsilon)\Phi + \gamma) \ , \quad \frac{\partial^2 f_3}{\partial x_3 \partial x_4} = \alpha \end{split}$$

Since  $v_1 = 0$  and  $v_4 = 0$ 

It follows that

$$a = v_2 \left[ w_2 w_3 \frac{\partial^2 f_2}{\partial x_2 \partial x_3} + w_2 w_4 \frac{\partial^2 f_2}{\partial x_2 \partial x_4} + w_2^2 \frac{\partial^2 f_2}{\partial x_2^2} + w_3^2 \frac{\partial^2 f_2}{\partial x_3^2} + w_3 w_4 \frac{\partial^2 f_2}{\partial x_3 \partial x_4} \right] + v_3 \left[ w_2 w_3 \frac{\partial^2 f_3}{\partial x_2 \partial x_3} + w_3 w_4 \frac{\partial^2 f_3}{\partial x_3 \partial x_4} \right] + w_3 w_4 \frac{\partial^2 f_3}{\partial x_3 \partial x_4}$$

$$a = -v_2[2w_3^2c_1\beta_1 + w_2w_4(c_1\beta_1 - \alpha) + 2w_2^2(c_1\beta_1 + (1 - \varepsilon)\Phi + \gamma) + w_3w_4c_2\beta_2 + w_2w_3(c_1\beta_1 + c_2\beta_2)] - v_3w_3[-w_4\alpha + w_2(\gamma + (1 - \varepsilon)\Phi)]$$

For b, we have

$$b = v_2 \left[ w_1 \frac{\partial^2 f_2}{\partial x_1 \partial \beta} (0,0) + w_2 \frac{\partial^2 f_2}{\partial x_2 \partial \beta} (0,0) + w_3 \frac{\partial^2 f_2}{\partial x_2 \partial \beta} + w_4 \frac{\partial^2 f_2}{\partial x_4 \partial \beta} \right]$$

Substituting  $\beta_1 = \beta$  and  $\beta_2 = r\beta$  into F and differentiating, we have  $b = v_2[w_1c_1 + w_3c_2r]$ .

**Theorem 4:** The system (7-10) exhibits a forward bifurcation if

$$c_1\beta_1 > \alpha$$
 and  $((1-\varepsilon)\Phi + \gamma)(a+b) > (\delta_1 + \frac{\delta_2\theta}{\delta_2 + b})\alpha$ 

Otherwise, the system may show a backward bifurcation.

## 4.0 Numerical Result

To examine the dynamics of the model numerically, the system is solved using the fourth-order Runge-Kutta method with the following values for the parameters b = 0.04,  $c_1 = 5$ ,  $c_2 = 3$ ,  $\beta_1 = 0.04$ ,  $\beta_2 = 0.05$ ,  $\theta = 0.30$ ,  $\delta_1 = 0.30$ ,  $\delta_2 = 0.02$ ,  $\alpha = 0.9$ ,  $\gamma = 0.90$ ,  $\varepsilon = 0.20$ ,  $\phi = 0.03$  and initial conditions s(0) = 0.65,  $i_1 = 0.20$ ,  $i_2 = 0.10$ , a(0) = 0.05 for the period of 30 years. The results are displayed graphically in figures 2(a) - 3(d). Figures 2(a) - 2(c) show the effect of the birth rate of newborns infected by irresponsible HIV infectives in the population. The irresponsible HIV infectives decreases with increase in number of responsible HIV infectives, the birth rate of newborns infected by irresponsible infective decreases. This simply means that a proportion of irresponsible HIV infectives is becoming responsible through awareness and

counselling on risky reduction. This increase in the proportion of the responsible infectives will result in reduction of the MTCT.

Figures 3(a) - 3(d) show the effect of irresponsible HIV infective immigrants into the population. As the rate of irresponsible HIV infectives immigrants decreases, the irresponsible HIV infective decreases likewise, this leads to an increase in the proportion of responsible HIV infectives. This reduction in the rate of irresponsible HIV infective immigrants will ultimately lead to a reduction in MTCT because more people will become responsible.

Therefore, in order to reduce the spread of HIV and prevent MTCT, effective immigration policies such as screening and counselling for HIV should be prerequisites for issuance of visa so as to ensure that only responsible HIV infective immigrants are allowed entry into the population.

### 5.0 CONCLUSION

A variate of the model in [8] is proposed to incorprorate different mean numbers of sexual partners for irresponsible and responsible infectives with vertical transmission in a varying population. The model is investigated to exhibit local asymptoic stability at DFE provided  $R_0 < 1$ . The bifurcation analysis for the model is proved to be subcritical based on certain conditions. Results from numerical simulations indicate that a reduction in the number of irresponsible infective immigrants decreases the infection cases of MTCT.

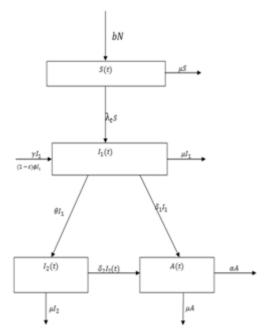


Figure 1. The flow diagram for the model

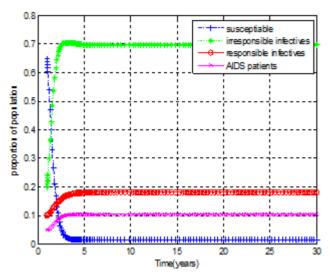


Figure 2(a). Variation of population in different classes for b=0.04 and  $\phi=1.00$ 

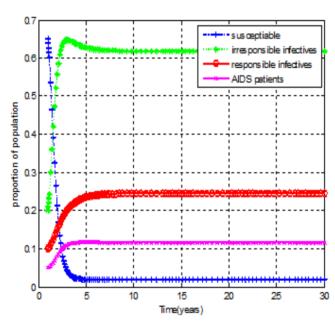


Figure 2(b). Variation of population in different classes for b=0.04 and  $\phi=0.50$ 

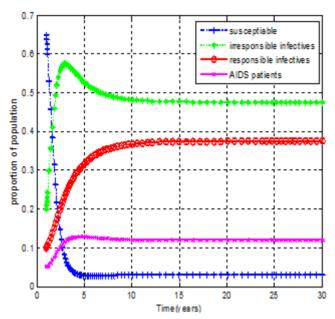


Figure 2(c).Variation of population in different classes for b=0.04 and  $\phi=0.00$ 

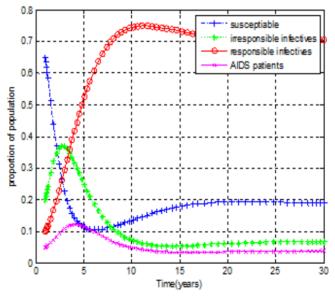


Figure 3(a).Variation of population in different classes for  $\gamma=0.00$  and  $\phi=0.03$ 

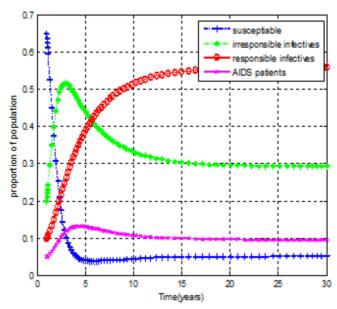


Figure 3(b).Variation of population in different classes for  $\gamma=0.60$  and  $\phi=0.03$ 

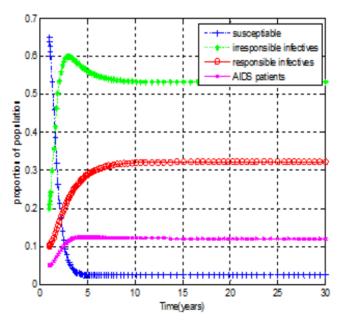


Figure 3(c).Variation of population in different classes for  $\gamma=1.00$  and  $\phi=0.03$ 

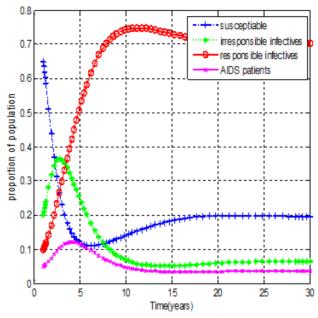


Figure 3(d). Variation of population in different classes for  $\gamma=0.00$  and  $\phi=0.00$ 

# **Conflict of Interests**

The authors declare that there is no conflict of interests.

### **REFERENCES**

- [1] CDC. Update on AIDS. United States morbidity and mortality, weekly report, 31 (1982), 507-514.
- [2] World Health Organization (WHO), Core information for the Development of Immunization policy, retrieved from http://www.who.int/vaccinesdocument/docPDF02/www557.pdf. 2002.
- [3] World Health Organization (WHO). Global Health-sector strategy for HIV/AIDS, Geneva, 32, 2003-2007.
- [4] Dixon, S., McDonald, S. and Robert, J. The impact of HIV and AIDS in Africa is economic development. BMJ, 324(2002):232-234.
- [5] Ify, M. A black woman's burden? Pharmascope 1(2004), 16.
- [6] Ogboi, S. J., Sabitu, K. Agu P.U., Ogboi, H.O. and Ayegbusi, O. Prevalence of HIV transmission in Nigerian transit town among long distance truck drivers laboratory survey, <a href="www.aacc.org/ia/transit-towns.pdf">www.aacc.org/ia/transit-towns.pdf</a>. 2001.
- [7] Ochejele, S. Overview of PMTCT of HIV Infection. Update course of the National Postgraduate Medical College of Nigeria, Makurdi. March, 18, 2011.

- [8] Daabo, M.I. and Seidu, B. Modelling the effect of irresponsible immigrants on the transmission dynamics of HIV/AIDS. Advances in Applied Math. Biosci., 3(1): 31-40,2012.
- [9] Daabo, M.I., Makinde, O.D. and Seidu, B. Modelling the combined effects of careless susceptible and infective immigrants on the transmission dynamics of HIV/AIDS epidemics. Public Health and Epidemiology, 5(2013), 362-369.
- [10] Ochoche, J.M. Modelling HIV in the presence of infected immigrants and vertical transmission: the role of incidence function. IJSTR, 2(2013), 113-133.
- [11] Basavarajaiah, D.M.B., Naraimhamurthy, K. and Maheshppa, B.L. Mathematical model approach to HIV/AIDS transmission from mother to child. IJSTR, 1(2012), 52-61.
- [12] Naresh, R., Tripathi, A. and Sharma, D. Modeling the spread of AIDS epidemic with vertical dynamics. Appl. Math. Comp., 178 (2006), 262-272.
- [13] Diekmann, O., Heesterbeek, J.A. and Metz J.A. On the definition and Computation of the basic reproduction ratio R<sub>0</sub> in models for infections diseases in heterogeneous population. *Journal of Mathematical Biology*, 28(1990), 365-382.
- [14] Garba S.M., Gumel, A.B., and Abu Bakar, M.R. Backward bifurcations in dengue transission dynamics. *Mathematical Biosciences*. 15(2008),11-25.
- [15] Chitnis, N., Cushing, J.M., and Hyman, J.M. Bifurcation analysis of a mathematical model for malaria transmission. *SIAM J. APPL. MATH.*, 67(2006), 24-45.
- [16] Piazza, N., and Hao, Wang. Bifurcation and sensitivity analysis of immunity duration in a Epidemic. *International journal of numerical analysis and modelling*, series B, 4(2013), 179-202.
- [17] Arion, J., Mccluskey, C.C., and Driessche, P. Van Den (2003). Global results for an epidemic model with vaccination that exhibits backward bifurcation. *SIAM. J. APPL. MATH.*, 64(2003), 260-276.
- [18] Huang, W., Cooke, K.L., and Carlos, C. Stability and bifurcation for a multiple-group model for the dynamics of HIV/AIDS transmission. *SIAM. J. APPL. MATH.*, 52(1992), 835-854.
- [19] Dushoff, J., Huang, W. and Carlos, C. Backwards bifurcation and catastrophe in sample models of fatal diseases. *J. Math. Bio.* 36 (1998), 227-248.
- [20] Castillo Chavez C. and Song, B. Dynamical Models of Tuberculosis and their Applications. Mathematical Biosciences and Engineering, 1(2004): 361-404.