Modeling HIV In The Presence Of Infected Immigrants And Vertical Transmission: The Role Of Incidence Function

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Abstract: We formulated three mathematical models for the transmission dynamics of HIV in the presence of infected immigrants and vertical transmission using a deterministic approach. Three forms of incidences popularly used in epidemiology were considered: The mass action, Standard and non-linear/saturated incidences. A Basic Reproduction number was derived for each model and used to prove its local stability. All the models were found to be globally asymptotically stable at the disease-free equilibrium. The mass action incidence and the non – linear incidence yielded similar analytical results e.g their Basic Reproduction numbers are identical and greater than the Basic Reproduction number of the Standard incidence model. Further, only the numerical simulation of the standard incidence model was biologically meaningful and we concluded that for sexually transmitted diseases the standard incidence is most appropriate.

Keywords: Basic Reproduction number, Epidemiology, HIV, Incidence function, Infected immigrants, Stability, Vertical transmission

1 BACKGROUND

Infectious diseases have accompanied mankind throughout history with each generation recording its own form of dreaded infectious disease and some of the diseases spanning through centuries and millennia. Smallpox started from around 6,000 BC, the last case was recorded in Somalia in 1977 [1],[2],[3]. Immediately after the eradication of smallpox, before the world could heave a sigh of relief, HIV/AIDS, another deadly infectious disease, was introduced in 1981 [4],[5]. The virus is mostly transmitted through sexual intercourse and it attacks the CD4 helper cells and microphages. If a single *virion* gain entrance into a host, an immune cell may produce over a thousand copies of the *virion* before the cell is destroyed. The virus is short-lived but prolific, with maximum daily turnover rates measured in the billions of *virions* [6]. When infected, the disease progresses through a latent period which lasts from months to years depending on the individual. After the latent period, the virus begins to attack the CD4 helper cells thereby compromising the immune system of the individual. Without adequate treatment the CD4 cells and the lymphatic tissues are destroyed. The disease then progress into the AIDS stage when the body is no longer able to fight off infections. Opportunistic infections then set in and the individual dies. AIDS has led to the mortality of millions. In 2011 2.8 million people died of the disease in china alone [7]. Expectedly, like most infectious diseases, sub-Saharan Africa bears the greatest burden of HIV/AIDS harboring about 70% of all HIV infections despite accommodating only 13% of the world's population [4].

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Nigeria, the most populous nation in sub-Haran Africa (with a population of >160 million) accounts for 10% of global HIV burden [8]. HIV is a major contributing factor to the declining life expectancy rate in the country. In 1991 the average life expectancy is 54 years while in 2010; the figure has fallen to 48 years. The disease was first reported in Nigeria in 1986 thus establishing the presence of the epidemic in the country. In 2012 Nigeria with an estimated 3.5 million people living with the disease overtook India to become the country with the second highest burden of the disease globally, next only to South Africa. The number of persons requiring ART stands at about 1.5 million in 2011 [9], however only 30% of these people have access to it. Mathematical modeling of infectious diseases can be traced to Bernoulli 1760 in his investigation into smallpox. Ross followed in 1911 with the first Vector-Host model of malaria. However most epidemiological models we have today are built on the works of Kermack and McKendrick (see [1] and the references therein). The works of Kermack and McKendrick showed that there is an epidemic threshold – a density of susceptible – that must be exceeded in order for an epidemic outbreak to occur. Mathematical modeling of HIV/AIDS was first proposed by Anderson and others [10], and later followed by [11],[12]. Due to worldwide pandemic, availability of data and socio – economic importance of the disease, HIV/AIDS became a popular candidate for modelers leading to a wide variety of HIV models being developed, analyzed and solved numerically and analytically (see [13],[14],[15],[16],[17],[19],[20]). Control strategies such as condom use [21], counseling [22], Treatment [23], [24] and vaccination [25] were investigated by several authors. Models were also built to investigate the transmission dynamics of HIV/AIDS in the presence of multi-strains of the virus [7],[26] and other authors investigated the existence of optimal control [27],[28] mostly using Pontryagin's Principle [29]. Due to immuno – compromisation, HIV/AIDS has led to the complication of other diseases in the host and mathematical models has equally been built to study the dynamics of HIV co-infection with diseases like tuberculosis [30], Malaria [31], Schistosomiasis [32] among others. The above mentioned models are mostly deterministic consisting of a disease – free equilibrium and at least one endemic equilibrium.

Attention is usually drawn to the basic reproduction number (R_0) which is the average number of secondary infections produce by a single infective when introduced into a susceptible population [33],[34]. This is because of its public health implication, more so, the stability of the models depends on the basic reproduction number. For infectious disease models, the disease – free equilibrium is locally asymptotically stable when $R_0 < 1$ and unstable when $R_0 > 1$.

1.1 Incidence Function

Disease incidence refers to the infection rate of susceptible through their contact with infectives [35], it is the number of new cases per unit time or the rate at which new waves of the disease appear. Let βN denote the number of adequate contact required for transmission of the disease where *N* is the total number of individuals in the considered region or community, let *S* and *I* represent Susceptibles and Infectives respectively. Then the incidence which determine the rate of new infection is given by $\frac{\beta NSI}{N} = \beta SI$. This form of incidence is called the mass action incidence or density dependent incidence since it assume that the pattern of daily encounter is dependent on the size of the community which implies that the contact rate is an increasing function of the population. The mass action incidence typically assumes that given enough time in the absence of control, the disease can spread to take over the entire community since the number of contact made by an infective per unit time is unlimited. It is therefore appropriate to use this form of incidence when the total number of persons in the community is not too large. In reality, diseases does not spread to take over an entire community since not all susceptible will make contact with an infective. More so, some diseases like STD's are transmitted independent of population density. In this case the number of contacts per infective per unit time is constant. Let β denote this constant and let $\frac{I}{N}$ be the infectious fraction. Then the number of new cases per unit time will be given as $\frac{\beta IS}{N}$. This form of incidence is known as the standard incidence or density – independent incidence. This type is incidence is more appropriate in modeling STD's [1], [36] in particular and human diseases in general as strongly suggested by data [12],[37]. As the number of infectives increase, the number of contact with susceptibles will not always linearly increase as well. Eventually a saturation level will be reached. A number of authors have suggested that disease incidence is a non-linear process (see [38] and the references therein). Models that incorporate behavioral change in a bid to prevent unboundedness of contact rate makes use of the non-linear incidence function. The saturation factor may arise as a result of epidemic control, crowding of infectives, response of susceptible to disease severity or intervention measures to protect susceptibles. Several types of non-linear incidence function have been formulated and studied e.g $\frac{\beta SI}{1+\alpha_1 S+\alpha_2 I}$, $\frac{\beta SI}{1+\alpha S}$, $\frac{\beta SI}{1+\alpha I}$, $\frac{\beta SI}{c+S}$, or more generally $f(I) = \frac{\beta S I^l}{1+\alpha I^h}$ where β, l, h are positive constants (see [38],[39],[40],[41] and the references therein). This form of incidence is usually referred to as the saturated or non – linear incidence. Cai *et al* [41] used the non – linear incidence to model HIV/AIDS epidemic while Mukandevire *et al* [42] used the Standard incidence to investigate the global analysis of an HIV/AIDS epidemic model and Al – Sheikh *et al* [43] used the mass action incidence also for HIV/AIDS epidemic model. In all cases the existence and stability of both disease – free and endemic equilibria was investigated. In this paper we formulated mathematical models for HIV/AIDS epidemic using the three incidences i.e, the standard, mass action and non – linear incidences while considering the possibility of vertical transmission (mother – child) and infective immigrants.

2.0 THE STANDARD INCIDENCE MODEL

2.1 Model Formulation

The total population denoted by *N* is divided into four mutually exclusive classes namely, Susceptibles S, Infectives I, Infectives on HAART H and AIDS A such that

$$N = S + I + H + A.$$

Susceptibles are recruited at the rate π and become infected with the disease after sexual contact at the rate λ, where

$$\lambda = \frac{c_1\beta_1 I + c_2\beta_2 H}{N}$$

c_1 is the average number of sexual partners of individuals in the *I* class while c_2 is the average number of sexual partners of individuals in the *H* class. β_1 is the effective contact required for HIV transmission in the *I* class while β_2 is similarly defined in the *H* class. We assumed that individuals in the AIDS class do not transmit the disease due to their ill health obvious symptoms of the disease, more so, they are stigmatized in places like Nigeria. Newly infected are generated at the rate λS. We incorporated the direct recruitment of newly infected (immigration) at the rate m_1. The model assumes that a fraction of newborns are infected during birth and are directly recruited at the $b(1-\varphi)$ while the complementing fraction $b\varphi$ are born free hence they appear in the susceptible class. Newly infected progress to treatment at the rate α and to AIDS class at the rate θ_1. Infectives on HAART *H,* are generated from the *I* class and directly through immigration at the m_2, they progress to AIDS at the rate θ_2. Individuals in the AIDS class are generated from the *I* and *H* classes and die a disease induced death at the rate δ. Individuals in all classes die at a natural death rate μ. From the model formulation and the flow diagram above we now present the model equation.

$$\left.\begin{aligned}
\frac{dS}{dt} &= \pi + \varphi b I^j - (\lambda + \mu)S \\
\frac{dI}{dt} &= \lambda S + m_1 I + b(1-\varphi)I^j - (\alpha + \theta_1 + \mu)I \\
\frac{dH}{dt} &= \alpha I + m_2 H - (\theta_2 + \mu)H \\
\frac{dA}{dt} &= \theta_1 I + \theta_2 H - (\delta + \mu)A
\end{aligned}\right\} 1$$

where $I^j = I + rH$

Table 1: State Variables and description

STATE VARIABLE	DESCRIPTION
S	Susceptibles
I	Infectives (Newly infected)
H	Infectives on treatment
A	AIDS

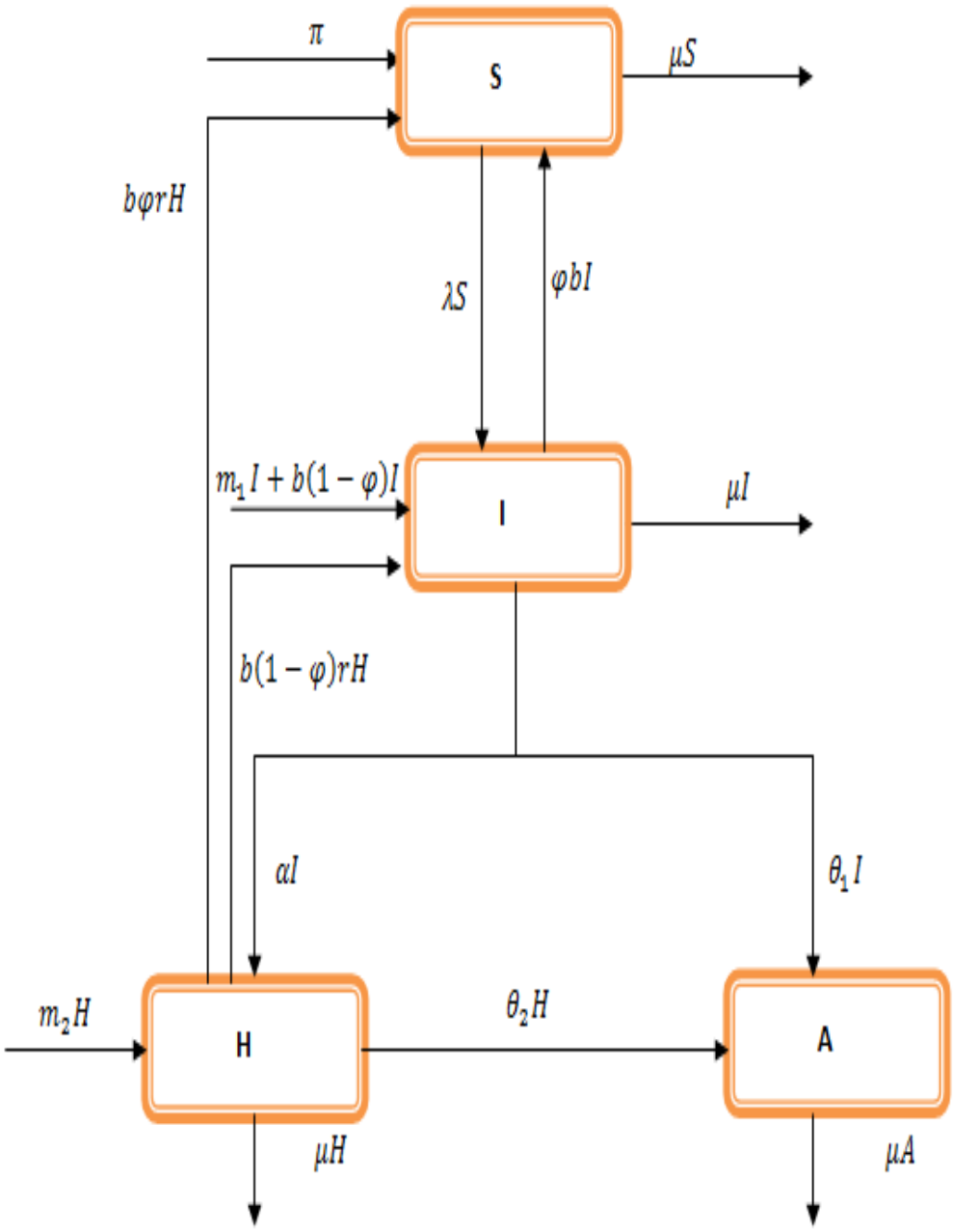

Fig. 1. Flow diagram of the standard incidence model.

Table 2: Parameters and description

PARAMETER	DESCRIPTION
π	Recruitment rate into the susceptible population
φ	Fraction of newborns that are HIV free
b	Birthrate of Infectives and Infectives on HAART
$m_i, i = 1,2$	Recruitment rate of Infectives into compartment
δ	Disease induced deathrate in the AIDS compartment
$\theta_i, i = 1,2$	Rate of progress into AIDS compartment
$\beta_i, i = 1,2$	Effective contact required for transmission
$c_i, i = 1,2$	Average number of sexual partners per unit time
α	Rate of progress from compartment *I* to *H*
μ	*Per capita* natural mortality rate
r	Modification parameter

Since the state variable A does not appear in the first three equations of the system (1) our analysis of the model shall be based on the system (2) below.

$$\left.\begin{aligned}\frac{dS}{dt} &= \pi + \varphi b I^j - (\lambda + \mu)S \\ \frac{dI}{dt} &= \lambda S + m_1 I + b(1-\varphi)I^j - (\alpha + \theta_1 + \mu)I \\ \frac{dH}{dt} &= \alpha I + m_2 H - (\theta_2 + \mu)H\end{aligned}\right\} 2$$

2.2 Positivity of Solutions

Since the model monitors human population we need to show that all the state variables remain non-negative for all times.

Theorem 1

Let $\Gamma = \{(S,I,H)\ \epsilon\ \mathbb{R}^3_+ : S(0) > 0, I(0) > 0\}$ *then the solutions of* $\{S(t), I(t), H(t)\}$ *of the system (2) are positive for all t ≥ 0.*

Proof:

Taking the first equation, we have

$$\frac{dS}{dt} = \pi + \varphi b I^j - (\lambda + \mu)S$$

$$\frac{dS}{dt} \geq -(\lambda + \mu)S$$

$$\frac{dS}{s} \geq -(\lambda + \mu)dt$$

$$\int \frac{dS}{S} \geq \int -(\lambda + \mu)dt$$

$$S(t) \geq S(0)e^{-(\lambda dt + \mu dt)}$$

$$\geq 0$$

From the second equation we have

$$\frac{dI}{dt} = \lambda S + m_1 I + b(1-\varphi)I^j - (\alpha + \theta_1 + \mu)I$$

$$\frac{dI}{dt} \geq -(\alpha + \theta_1 + \mu)I$$

$$\frac{dI}{I} \geq -(\alpha + \theta_1 + \mu)dt$$

$$\int \frac{dI}{I} \geq \int -(\alpha + \theta_1 + \mu)dt$$

$$I(t) \geq I(0)e^{-(\alpha+\theta_1+\mu)dt} \geq 0$$

Similarly, it can be shown that the third equation of the system (2) is positive.

2.3 Invariant Region

Theorem 2

The system (2) has solutions which are contained in the feasible region Ω

Proof

Let $(S,I,H)\epsilon\mathbb{R}^3_+$ be any solution of the system with non negative initial conditions then Adding the equations of the system (2), we have

$$\frac{dS}{dt} + \frac{dI}{dt} + \frac{dH}{dt} = \pi + m_2 H + m_1 I + bI^j - (\theta_1 + \delta_1)I - (\theta_2 + \delta_2)H - \mu(S + I + H) \leq \frac{\pi}{\mu}$$

Hence

$$\lim_{t\to\infty} sup\ (S + I + H) \leq \frac{\pi}{\mu}$$

Thus the considered region for the system (2) is

$$\Omega = \{(S,I,H) : (S + I + H) \leq \tfrac{\pi}{\mu}, S > 0, I \geq 0, H \geq 0\}$$

The vector field points to the interior of Ω on the part of the boundary when $(S + I + H) = \frac{\pi}{\mu}$ for $t > 0$ and Ω is positively invariant. The model system (2) has a disease free equilibrium given by $E_0 = \left(\frac{\pi}{\mu}, 0, 0\right)$.

2.4 Basic Reproduction Number

The Basic reproduction number (R_0) is defined as the average number of new cases of an infection caused by one typical infected individual in a population consisting of susceptibles only [44]. It is arguably the most important quantity in infectious disease epidemiology as it provides insights into the disease dynamics and can suggest relevant control strategies. Synonymous to R_0 in demography is the lifetime reproductive success of a typical member of the specie. We now compute the basic reproduction number using the next – generation approach (see [33,44]). Let F_i be the rate of appearance of new infection in compartment and V_i be the transfer of

individuals out of compartment by any other means, let E_0 be the disease – free equilibrium, then R_0 is the spectral radius of

$$\left[\frac{\partial F_i(E_0)}{\partial x_j}\right]\left[\frac{\partial V_i(E_0)}{\partial x_j}\right]^{-1}$$

$$F_i = \begin{bmatrix}F_1\\F_2\end{bmatrix} = \begin{bmatrix}\lambda S + b(1-\varphi)I^j\\0\end{bmatrix} = \begin{bmatrix}\left(\frac{c_1\beta_1 I + c_2\beta_2 H}{N}\right)S + b(1-\varphi)I^j\\0\end{bmatrix}$$

$$\left[\frac{\partial F_i(E_0)}{\partial x_j}\right] = \begin{bmatrix}\frac{\partial F_1}{\partial I}(E_0) & \frac{\partial F_1}{\partial H}(E_0)\\ \frac{\partial F_2}{\partial I}(E_0) & \frac{\partial F_2}{\partial H}(E_0)\end{bmatrix} = \begin{bmatrix}c_1\beta_1 + (1-\varphi) & c_2\beta_2 + (1-\varphi)\\ 0 & 0\end{bmatrix}$$

$$V_i = \begin{bmatrix}V_1\\V_2\end{bmatrix} = \begin{bmatrix}(\alpha+\theta_1+\mu)I - m_1 I\\(\theta_2+\mu)H - \alpha I - m_2 H\end{bmatrix}$$

$$\left[\frac{\partial V_i(E_0)}{\partial x_j}\right] = \begin{bmatrix}\frac{\partial V_1}{\partial I}(E_0) & \frac{\partial V_1}{\partial H}(E_0)\\ \frac{\partial V_2}{\partial I}(E_0) & \frac{\partial V_2}{\partial H}(E_0)\end{bmatrix} = \begin{bmatrix}(\alpha+\theta_1+\mu) - m_1 & 0\\ -\alpha & (\theta_2+\mu) - m_2\end{bmatrix}$$

$$V^{-1} = \begin{bmatrix}\frac{1}{(\alpha+\theta_1+\mu)-m_1} & 0\\ \frac{\alpha}{((\alpha+\theta_1+\mu)-m_1)((\theta_2+\mu)-m_2)} & \frac{1}{(\theta_2+\mu)-m_2}\end{bmatrix}$$

$$FV^{-1} = \begin{bmatrix}\frac{c_1\beta_1 + b(1-\varphi)}{(\alpha+\theta_1+\mu)-m_1} + \frac{(c_2\beta_2 + b(1-\varphi)r)\alpha}{((\alpha+\theta_1+\mu)-m_1)((\theta_2+\mu)-m_2)} & \frac{(c_2\beta_2+(1-\varphi)r)}{(\theta_2+\mu)-m_2}\\ 0 & 0\end{bmatrix}$$

$$R_0 = \frac{(\theta_2+\mu-m_2)\left(c_1\beta_1 + b(1-\varphi)\right) + (c_2\beta_2 + b(1-\varphi)r)\alpha}{(\alpha+\theta_1+\mu-m_1)(\theta_2+\mu-m_2)}$$

2.5 Local stability of the Disease – free equilibrium

Theorem 3

The disease – free equilibrium is locally asymptotically stable whenever $R_0 < 1$ We shall use the linearization approach to proof the local stability of the Disease – free equilibrium (DFE). The Jacobian matrix associated with the system (2) is:

$$J = \begin{bmatrix}-(\lambda+\mu) & \varphi b - c_1\beta_1\frac{S}{N} & \varphi br - c_2\beta_2\frac{S}{N}\\ \lambda & c_1\beta_1\frac{S}{N} + b(1-\varphi) - (\alpha+\theta_1+\mu-m_1) & c_2\beta_2\frac{S}{N} + br(1-\varphi)\\ 0 & \alpha & -(\theta_2+\mu-m_2)\end{bmatrix}$$

At the DFE which is given by $E_0 = \left(\frac{\pi}{\mu}, 0, 0\right)$, when *I* = *H* = 0 ⇒$\lambda = 0$, we have

$$J(E_0) = \begin{bmatrix}-\mu & \varphi b - c_1\beta_1 & \varphi br - c_2\beta_2\\ 0 & c_1\beta_1 + b(1-\varphi) - (\alpha+\theta_1+\mu-m_1) & c_2\beta_2 + br(1-\varphi)\\ 0 & \alpha & -(\theta_2+\mu-m_2)\end{bmatrix}$$

Clearly μ is an eigenvalue. The other two eigenvalues are determine from the characteristic equation of

$$J_1 = \begin{vmatrix}c_1\beta_1 + b(1-\varphi) - (\alpha+\theta_1+\mu-m_1) & c_2\beta_2 + br(1-\varphi)\\ \alpha & -(\theta_2+\mu-m_2)\end{vmatrix}$$

For the system (2) to be stable, the two conditions below suffices

i) $Tra(J_1) < 0$ and
ii) *Det* $(J_1) > 0$.

Tra $(J_1) = c_1\beta_1 + b(1-\varphi) - (\alpha+\theta_1+\mu-m_1) - (\theta_2+\mu-m_2)$

Tra$(J_1) < 0$ gives

$$c_1\beta_1 + b(1-\varphi) < (\alpha+\theta_1+\mu-m_1) + (\theta_2+\mu-m_2)$$

Or $\frac{c_1\beta_1+b(1-\varphi)}{(\alpha+\theta_1+\mu-m_1)+(\theta_2+\mu-m_2)} < 1$ (3)

$$Det\ (J_1) = \big(c_1\beta_1 + b(1-\varphi) - (\alpha + \theta_1 + \mu - m_1)\big)\big(-(\theta_2 + \mu - m_2)\big) - \big(c_2\beta_2 + br(1-\varphi)\big)\alpha$$

$Det(J_1) > 0$ gives

$$-\big[\big(c_1\beta_1 + b(1-\varphi)\big)(\theta_2 + \mu - m_2)\big] - \big(c_2\beta_2 + br(1-\varphi)\big)\alpha > -(\alpha + \theta_1 + \mu - m_1)(\theta_2 + \mu - m_2)$$

Or $R_0 < 1$

Note that $R_0 < 1$ implies that the inequality (3) also holds and thus we have proved theorem 3.

2.6 Global Stability of the Disease Free Equilibrium

The global asymptotic stability of the disease free state of the model is investigated using the approach by Castillo-Chavev *et al* [45]. First the model is rewritten as

$$\frac{dW}{dt} = P(W,Z),$$

$$\frac{dZ}{dt} = G(W,Z), G(W,0) = 0$$

where $W = (S) and\ Z = (I,H)$, with the components of $W \in \mathbb{R}$ denoting the uninfected population and the component of $Z \in \mathbb{R}^2$ denoting the infected population. $E_0 = (W^*, 0)$ represent the disease-free equilibrium of the system.

The conditions for global stability are

i. $\frac{dW}{dt} = P(W,0), W^*$ is globally asymptotically stable (GAS)
ii. $G(W,Z) = NZ - \hat{G}(W,Z), \hat{G}(W,Z) \geq 0$ for $(W,Z) \in \Gamma$

where $N = D_z G(W^*, 0)$ is an M-matrix (the off diagonal elements of N are non-negative) and Γ is the region where the model makes biological sense. If the system (2) satisfies the two conditions above then according to Castillo-Chavez *et al.* [45], the following theorem holds.

Theorem 4

The equilibrium point $E_0 = (W^*, 0)$ of system (2) is globally asymptotically stable provided $R_0 < 1$ (locally asymptotically stable) and that the conditions (i) and (ii) are satisfied. To apply theorem (4) on the system (2) we now proceed thus:

$$W = S(t),\ Z = (I,H), F(W,0) = (\pi - \mu S(0))$$

$$N = D_z G(W^*, 0) = \begin{pmatrix} c_1\beta_1 + b(1-\varphi) - (\alpha + \theta_1 + \mu - m_1) & c_2\beta_2 + br(1-\varphi) \\ \alpha & -(\theta_2 + \mu - m_2) \end{pmatrix}$$

and for $\hat{G}(W,Z) = NZ - G(W,Z)$, we have

$$NZ = \begin{pmatrix} c_1\beta_1 + b(1-\varphi) - (\alpha + \theta_1 + \mu - m_1) & c_2\beta_2 + br(1-\varphi) \\ \alpha & -(\theta_2 + \mu - m_2) \end{pmatrix}\begin{pmatrix} I \\ H \end{pmatrix}$$

$$G(W,Z) = \begin{pmatrix} \lambda S + m_1 I + b(1-\varphi)I^j - (\alpha + \theta_1 + \mu)I \\ \alpha I + m_2 H - (\theta_2 + \mu)H \end{pmatrix}$$

$$\hat{G}(W,Z) = \begin{pmatrix} (c_1\beta_1 I + c_2\beta_2 H)\left(1 - \frac{S}{N}\right) \\ 0 \end{pmatrix}$$

Note that $S \leq N$, we therefore have $\hat{G}(W,Z) \geq 0$ hence the DFE given by $E_0 = \left(\frac{\pi}{\mu}, 0, 0\right)$ is globally asymptotically stable. The public health implication of the above result is that if the control strategy for HIV/AIDS can force the reproduction number to less than unity then HIV/AIDS can be eliminated from the community.

2.7 Existence of endemic equilibrium

In the presence of HIV/AIDS, that is, $I(t) \neq 0, H(t) \neq 0$, the model system (2) has an equilibrium point, called the endemic equilibrium point , denoted by E_1 and given by:

$$E_1 = (S^*, I^*, H^*) \neq 0$$

E_1 is the steady state endemic equilibrium point where the disease persist in the population. For the existence and uniqueness of E_1 its coordinate has to satisfy the following conditions.

$$0 < S^*, 0 < I^*, 0 < H^*$$

At the endemic equilibrium, we set the left hand of the system (2) to zero from which we obtain the homogenous system of differential equations (4)

$$\left.\begin{aligned} 0 &= \pi + \varphi b I^j - (\lambda + \mu)S \\ 0 &= \lambda S + m_1 I + b(1-\varphi)I^j - (\alpha + \theta_1 + \mu)I \\ 0 &= \alpha I + m_2 H - (\theta_2 + \mu)H \end{aligned}\right\} 4$$

We now solve the system (4) for the endemic equilibrium point. From the first equation of (4) we obtained

$$S^* = \frac{(\pi + \varphi b I^* + \varphi br)N^*}{c_1\beta_1 I^* + c_2\beta_2 H^* + \mu N^*} \qquad (5)$$

From the second equation of (4) we obtained

$$I^* = \frac{\left(c_2\beta_2\frac{S^*}{N^*} + br(1-\varphi)\right)H^*}{A - \left(c_1\beta_1\frac{S^*}{N^*} + b(1-\varphi)\right)} \qquad (6)$$

where $A = (\alpha + \theta_1 + \mu - m_1)$.

From the third equation of (4) we obtained

$$H^* = \frac{\alpha I^*}{B} \qquad (7)$$

where $B = (\theta_2 + \mu - m_2)$.

Putting (7) into (6) we have $I^* = \frac{\left(c_2\beta_2\frac{S^*}{N^*} + br(1-\varphi)\right)\alpha I^*}{\left(A-\left(c_1\beta_1\frac{S^*}{N^*}+b(1-\varphi)\right)\right)B}$ or $1 = \frac{\left(c_2\beta_2\frac{S^*}{N^*} + br(1-\varphi)\right)\alpha}{\left(A-\left(c_1\beta_1\frac{S^*}{N^*}+b(1-\varphi)\right)\right)B}$

From which we obtained

$$S^* = \frac{(AB + Bk_1 - \alpha k_1 r)N^*}{\alpha c_2\beta_2 + Bc_1\beta_1} \qquad (8)$$

where $k_1 = b(1-\varphi)$.

Substituting S^* in (8) into (5)we have

$$\frac{(AB + Bk_1 - \alpha k_1 r)N^*}{\alpha c_2\beta_2 + Bc_1\beta_1} = \frac{(\pi + \varphi bI^* + \varphi br)N^*}{c_1\beta_1 I^* + c_2\beta_2 H^* + \mu N^*}$$

After some algebraic manipulation we obtained

$$I^* = \frac{\pi(\alpha c_2\beta_2 + Bc_1\beta_1) + \mu N^*\alpha k_1 r - \mu N^*(AB + Bk_1)}{\left(c_1\beta_1 + \frac{\alpha}{B}c_2\beta_2\right)(AB + Bk_1 - \alpha r k_1) - \left(\varphi b + \varphi br\frac{\alpha}{B}\right)(\alpha c_2\beta_2 + Bc_1\beta_1)} \qquad (9)$$

H^* can be gotten from substituting (9) into (7).

2.8 Existence of backward or forward Bifurcation

The bifurcation analysis is performed at the disease-free equilibrium by using Centre Manifold Theorem as presented in Castillo-Chavez and Song [46].

Theorem 5 (Castillo-Chavez and Song, 2004)
Consider the following system of ordinary differential equations with parameter ø such that

$\frac{dx}{dt} = f(x, ø), f: \mathbb{R}^n \times \mathbb{R} \to \mathbb{R}$ *and* $f \in \mathbb{C}^2(\mathbb{R}^n \times \mathbb{R})$.

Without loss of generality, it is assumed that zero is an equilibrium point of the system for all parameters (that is $f(0, ø) \equiv 0 \; for \; all \; ø$*) and*

1. $A = D_x f(0,0) = \left(\frac{\partial f_i}{\partial x_j}(0,0)\right)$ *is the linearization matrix of the system around the equilibrium 0 with ø evaluated at 0. Zero is a simple eigenvalue of A and 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*

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

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

Then the local dynamics of the system around the equilibrium point 0 is totally determine by the signs of a and b

i. $a > 0, d > 0$. *When* $ø < 0$ *with* $|ø| \ll 1$,*0 is locally asymptotically stable and there exists a positive unstable equilibrium; when* $0 < ø \ll 1$,*0 is unstable and there exist a negative, locally asymptotically stable equilibrium.*
ii. $a < 0, d < 0$. *When* $ø < 0$ *with* $|ø| \ll 1$,*0 is unstable; when* $0 < ø \ll 1$, *0 is locally asymptotically stable equilibrium, and there exist a positive unstable equilibrium.*
iii. $a > 0, d < 0$. *When* $ø < 0$ *with* $|ø| \ll 1$, *0 is unstable, and there exists a locally asymptotically stable negative equilibrium; when* $0 < ø \ll 1$, *0 is stable and a positive unstable equilibrium appears.*
iv. $a < 0, d > 0$. *when ø changes from negative to positive, 0 changes its stability from stable to unstable. Correspondingly a negative unstable equilibrium becomes positive and locally asymptotically stable.*

Particularly if $a > 0 \; and \; d > 0$, *then a subcrtical (or backward) bifurcation occurs at* $ø = 0$. To apply the theorem, we make the following change of variables

$S = x_1, I = x_2$ and $H = x_3$. We use the vector notation $X = (x_1, x_2, x_3)^T$ so the system (2) can be written in the form $\frac{dx}{dt} = F = (f_1, f_2, f_3)^T$, such that:

$$\lambda = \frac{c_1\beta_1 x_2 + c_2\beta_2 x_3}{\sum_{i=1}^{3} x_i}$$

and we have

$$\left.\begin{aligned} x_1' &= f_1 = \pi + \varphi bI^j - (\lambda + \mu)S \\ x_2' &= f_2 = \lambda S + m_1 I + b(1-\varphi)I^j - (\alpha + \theta_1 + \mu)I \\ x_3' &= f_3 = \alpha I + m_2 H - (\theta_2 + \mu)H \end{aligned}\right\} 10$$

The jacobian matrix of the system (10) at the disease-free equilibrium is equal to $J(E_0)$ as previously defined in section 2.4.

Taking β_1 as the bifurcation parameter and considering the case $R_0 = 1$ and solving for β_1, we have

$$1 = \frac{(\theta_2+\delta_2+\mu-m_2)(c_1\beta_1+(1-\varphi))+(c_2\beta_2+b(1-\varphi)r)\alpha}{(\alpha+\theta_1+\mu-m_1)(\theta_2+\mu-m_2)}$$

From which we obtained

$$\beta_1 = \frac{(\alpha+\theta_1+\mu-m_1)(\theta_2+\mu-m_2)-(c_2\beta_2+b(1-\varphi)r)\alpha-b(\theta_2+\mu-m_2)}{(\theta_2+\mu-m_2)c_1}$$

Let

$$\beta^* = \beta_1 = \frac{(\alpha+\theta_1+\mu-m_1)(\theta_2+\mu-m_2)-(c_2\beta_2+b(1-\varphi)r)\alpha-b(\theta_2+\mu-m_2)}{(\theta_2+\mu-m_2)c_1}$$

The linearized system of the transoformed system (10) with $\beta^* = \beta_1$ has a simple zero eigenvalue, hence the centre manifold theory can be used to analyse the dynamics of (10) near $\beta^* = \beta_1$ The right eigenvector $w = (w_1, w_2, w_3)$, can be calculated from the following

$$J(E_{01}) = \begin{bmatrix} -\mu & \varphi b - c_1\beta^* & \varphi br - c_2\beta_2 \\ 0 & c_1\beta^* + b(1-\varphi) - (\alpha+\theta_1+\mu-m_1) & c_2\beta_2 + br(1-\varphi) \\ 0 & \alpha & -(\theta_2+\mu-m_2) \end{bmatrix} \begin{bmatrix} w_1 \\ w_2 \\ w_3 \end{bmatrix}$$

$$-\mu w_1 + (\varphi b - c_1\beta^*)w_2 + (\varphi br - c_2\beta_2)w_3 = 0$$

$$\big(c_1\beta^* + b(1-\varphi) - (\alpha+\theta_1+\mu-m_1)\big)w_2 + \big(c_2\beta_2 + br(1-\varphi)\big)w_3 = 0$$

$$\alpha w_2 - (\theta_2+\mu-m_2)w_3 = 0$$

Solving for the right eigenvector we have

$$w_1 = \frac{(\varphi b - c_1\beta^*)w_2 + (\varphi br - c_2\beta_2)w_3}{\mu}$$

$$w_2 = w_2 > 0$$

$$w_3 = \frac{\alpha w_2}{(\theta_2+\delta_2+\mu-m_2)}$$

We now transpose $J(E_{01})$ and solve for the left eigenvector$v = (v_1, v_2, v_3)$, we have

$$J(E_{02}) = \begin{bmatrix} -\mu & 0 & 0 \\ \varphi b - c_1\beta^* & c_1\beta^* + b(1-\varphi) - (\alpha+\theta_1+\mu-m_1) & \alpha \\ \varphi br - c_2\beta_2 & c_2\beta_2 + br(1-\varphi) & -(\theta_2+\mu-m_2) \end{bmatrix} \begin{bmatrix} v_1 \\ v_2 \\ v_3 \end{bmatrix}$$

$$v_1 = 0$$

$$\big(c_1\beta^* + b(1-\varphi) - (\alpha+\theta_1+\mu-m_1)\big)v_2 + \alpha v_3 = 0$$

$$\big(c_2\beta_2 + br(1-\varphi)\big)v_2 - (\theta_2+\mu-m_2)v_3 = 0$$

$$v_2 = v_2 > 0$$

$$v_3 = \frac{\big(c_2\beta_2 + br(1-\varphi)\big)v_2}{(\theta_2+\delta_2+\mu-m_2)}$$

Computations of *a* and *d*

From the model system (10) the associated non varnishing partial derivatives of *F* at the disease – free equilibrium are given by

$$\frac{\partial^2 f_2}{\partial x_1 x_3} = c_1\beta^*$$

$$\frac{\partial^2 f_2}{\partial x_1 x_2} = c_2\beta_2$$

From the centre manifold theorem we have

$$a = \sum_{k,i,j=1}^{3} v_k w_i w_j \frac{\partial^2 f_k}{\partial x_i \partial x_j}(E_0),$$

$$= v_2 w_1 w_2 \frac{\partial^2 f_2}{\partial x_1 x_2}(E_0) + v_2 w_1 w_3 \frac{\partial^2 f_2}{\partial x_1 x_3}(E_0)$$

$$= v_2 w_2 \left[\frac{(\varphi b - c_1\beta^*)w_2 + (\varphi br - c_2\beta_2)w_3}{\mu}\right] + v_2 \left[\frac{(\varphi b - c_1\beta^*)w_2 + (\varphi br - c_2\beta_2)w_3}{\mu}\right] \frac{\alpha w_2}{(\theta_2 + \mu - m_2)}$$

$$= \frac{v_2 w_2}{\mu}\left((\varphi b - c_1\beta^*)w_2 + (\varphi br - c_2\beta_2)w_3\right)\left[1 + \frac{\alpha}{(\theta_2 + \mu - m_2)}\right]$$

d is associated with the following non – varnishing partial derivatives of *F*

$$\frac{\partial^2 f_2}{\partial x_2 \partial \beta^*} = c_1$$

we now have

$$d = \sum_{k,i,=1}^{3} v_k w_i \frac{\partial^2 f_k}{\partial x_i \partial \beta^*}(E_0)$$

$$v_2 w_2 \frac{\partial^2 f_k}{\partial x_2 \partial \beta^*} = c_1 v_2 w_2$$

Note that $v_2, w_2, w_3 > 0$, we therefore have $d > 0$ which is expected for most epidemiological models. The positivity of *a* is completely determined by $\varphi br > c_2\beta_2$ and $\varphi b > c_1\beta^*$ or $(\varphi b w_2 + \varphi br w_3) > (c_1\beta^* w_2 + c_2\beta_2 w_3)$. We have therefore proved the following theorem.

Theorem 6

The model system (2) exhibits a subcritical (Backward) bifurcation if

i. $\varphi br > c_2\beta_2$ *and* $\varphi b > c_1\beta^*$, *or*

ii. $(\varphi b w_2 + \varphi br w_3) > (c_1\beta^* w_2 + c_2\beta_2 w_3)$

Other wise the system exhibits a forward bifurcation

3.0 THE MASS ACTION MODEL

The model with mass action incidence is presented below

$$\left.\begin{aligned}
\frac{dS}{dt} &= \pi + \varphi b I^j - (c_1\beta_1 I + c_2\beta_2 H + \mu)S \\
\frac{dI}{dt} &= (c_1\beta_1 I + c_2\beta_2 H)S + m_1 I + b(1-\varphi)I^j - (\alpha + \theta_1 + \mu)I \\
\frac{dH}{dt} &= \alpha I + m_2 H - (\theta_2 + \mu)H \\
\frac{dA}{dt} &= \theta_1 I + \theta_2 H - (\delta + \mu)A
\end{aligned}\right\} 11$$

Where $I^j = I + rH$ as previously defined.

Once more the variable *A* does not appear in the first three equations of (11) so our analysis shall be performed on the system (12)

$$\left.\begin{aligned}
\frac{dS}{dt} &= \pi + \varphi b I^j - (c_1\beta_1 I + c_2\beta_2 H + \mu)S \\
\frac{dI}{dt} &= (c_1\beta_1 I + c_2\beta_2 H)S + m_1 I + b(1-\varphi)I^j - (\alpha + \theta_1 + \mu)I \\
\frac{dH}{dt} &= \alpha I + m_2 H - (\theta_2 + \mu)H
\end{aligned}\right\} 12$$

Following the approach in section 2.2 it is easy to show that the system (12) is uniformly bounded in a proper subset

$$\Omega = \{(S, I, H): (S + I + H) \leq \tfrac{\pi}{\mu}, S > 0, I \geq 0, H \geq 0\}$$

The vector field points to the interior of Ω on the part of the boundary when $(S + I + H) = \frac{\pi}{\mu}$ for $t > 0$ and Ω is positively invariant. The system (12) has a disease – free equilibrium when $I = H = 0$ and $S = \frac{\pi}{\mu}$ given by $E_{01} = (\frac{\pi}{\mu}, 0, 0)$

3.1 Basic Reproduction Number of the model with mass action incidence

Following the approach in section 2.3 we have

$$\begin{bmatrix} F_1 \\ F_2 \end{bmatrix} = \begin{bmatrix} (c_1\beta_1 I + c_2\beta_2 H)S + b(1-\varphi)I^j \\ 0 \end{bmatrix}$$

By linearizing around the DFE, we have

$$F = \begin{bmatrix} c_1\beta_1\frac{\pi}{\mu} + b(1-\varphi) & c_2\beta_2\frac{\pi}{\mu} + br(1-\varphi) \\ 0 & 0 \end{bmatrix}$$

Also

$$\begin{bmatrix} V_1 \\ V_2 \end{bmatrix} = \begin{bmatrix} -m_1 I + (\alpha + \theta_1 + \mu)I \\ -\alpha I - m_2 H + (\theta_2 + \mu)H \end{bmatrix}$$

At the disease free equilibrium we have

$$V = \begin{bmatrix} (\alpha + \theta_1 + \mu - m_1) & 0 \\ -\alpha & (\theta_2 + \mu - m_2) \end{bmatrix}$$

$$V^{-1} = \begin{bmatrix} \frac{1}{(\alpha + \theta_1 + \mu - m_1)} & 0 \\ \frac{\alpha}{(\alpha + \theta_1 + \mu - m_1)(\theta_2 + \mu - m_2)} & \frac{1}{(\theta_2 + \mu - m_2)} \end{bmatrix}$$

$$FV^{-1} = \begin{bmatrix} \frac{c_1\beta_1\frac{\pi}{\mu} + b(1-\varphi)}{(\alpha + \theta_1 + \mu - m_1)} + \frac{(c_2\beta_2\frac{\pi}{\mu} + br(1-\varphi))\alpha}{(\alpha + \theta_1 + \mu - m_1)(\theta_2 + \mu - m_2)} & \frac{c_2\beta_2\frac{\pi}{\mu} + br(1-\varphi)}{(\theta_2 + \mu - m_2)} \\ 0 & 0 \end{bmatrix}$$

The Basic Reproduction number of the model with mass action incidence denoted by R_{01} is the spectral radius of FV^{-1}.

$$R_{01} = \frac{\left((\theta_2 + \delta_2 + \mu - m_2)\left(c_1\beta_1\pi + \mu b(1-\varphi)\right) + \alpha\left(c_2\beta_2\pi + \mu br(1-\varphi)\right)\right)}{\mu(\alpha + \theta_1 + \mu - m_1)(\theta_2 + \mu - m_2)}$$

3.2 local stability of the disease – free equilibrium

The Jacobian matrix associated with the system (12) at the DFE is given by

$$J(E_{01}) = \begin{bmatrix} -\mu & \varphi b - c_1\beta_1\frac{\pi}{\mu} & \varphi br - c_2\beta_2\frac{\pi}{\mu} \\ 0 & c_1\beta_1\frac{\pi}{\mu} + b(1-\varphi) - (\alpha + \theta_1 + \mu - m_1) & c_2\beta_2\frac{\pi}{\mu} + br(1-\varphi) \\ 0 & \alpha & -(\theta_2 + \mu - m_2) \end{bmatrix}$$

Clearly $-\mu < 0$ is an eigenvalue, the other two eigenvalues are negative if $Tra\ (J_{11}) < 0$ and $Det\ (J_{11}) > 0$ where

$$J_{11} = \begin{vmatrix} c_1\beta_1\frac{\pi}{\mu} + b(1-\varphi) - (\alpha + \theta_1 + \mu - m_1) & c_2\beta_2\frac{\pi}{\mu} + br(1-\varphi) \\ \alpha & -(\theta_2 + \mu - m_2) \end{vmatrix}$$

$$Tra\ (J_{11}) = c_1\beta_1\frac{\pi}{\mu} + b(1-\varphi) - (\alpha + \theta_1 + \mu - m_1) - (\theta_2 + \mu - m_2)$$

$Tra\ (J_{11}) < 0$ gives

$$c_1\beta_1\tfrac{\pi}{\mu} + b(1-\varphi) < (\alpha + \theta_1 + \delta_1 + \mu - m_1) + (\theta_2 + \delta_2 + \mu - m_2)$$

Or

$$\frac{c_1\beta_1\pi + \mu b(1-\varphi)}{\mu[(\alpha + \theta_1 + \mu - m_1) + (\theta_2 + \mu - m_2)]} < 1 \qquad (13)$$

$$Det\ (J_{11}) = -(\theta_2 + \mu - m_2)c_1\beta_1\frac{\pi}{\mu} - (\theta_2 + \mu - m_2)b(1-\varphi) + (\alpha + \theta_1 + \mu - m_1)(\theta_2 + \mu - m_2) - \alpha\left[c_2\beta_2\frac{\pi}{\mu} + br(1-\varphi)\right]$$

$Det\ (J_{11}) > 0$ gives

$$(\theta_2 + \delta_2 + \mu - m_2)c_1\beta_1\frac{\pi}{\mu} + (\theta_2 + \mu - m_2)b(1-\varphi) - (\alpha + \theta_1 + \mu - m_1)(\theta_2 + \mu - m_2) + \alpha\left[c_2\beta_2\frac{\pi}{\mu} + br(1-\varphi)\right] < 0$$

Or

$$R_{01} < 1$$

Note that $R_{01} < 1$ implies that the inequality (13) also hold since

$$R_{01} > \frac{c_1\beta_1\pi + \mu b(1-\varphi)}{\mu[(\alpha+\theta_1+\mu-m_1)+(\theta_2+\mu-m_2)]}$$

we have therefore proved the following theorem

Theorem 7
The model system (12) with mass action incidence is locally asymptotically whenever $R_{01} < 1$

3.3 Global stability of the DFE of the model with mass action incidence

Theorem 8
The disease – free equilibrium of the model with mass action incidence given by $E_{01} = (\frac{\pi}{\mu}, 0, 0)$ *is locally asymptotically stable whenever* $R_{01} < 1$.

Proof:
We shall use the standard comparison theorem (see [47,48,49]) for the proof of theorem 8 All solutions starting in Ω remain in Ω and all other solution approach Ω. Thus it may be assumed

$0 \le S(t) \le S_0$ for all $t \ge 0$ where $S_0 = \frac{\pi}{\mu}$.

Consequently, the equations of the infectious compartments of the system (12) can be expressed in the following differential inequality

$$\begin{pmatrix} \frac{dI(t)}{dt} \\ \frac{dH(t)}{dt} \end{pmatrix} \le (F-V)\begin{pmatrix} I(t) \\ H(t) \end{pmatrix}$$

where *F* and *V* are the matrices previously defined.

According to Driessche and Watmough ,2002 [33] and Castillo-Chavez *et al*, 2002 [45] all eigenvalues of the matrix $F-V$ have negative real parts. It follows that the linearized differential inequality system (12) is stable whenever $R_{01} < 1$. Consequently, $\big(I(t), H(t)\big) \to (0,0)$ as $t \to \infty$. It follows, by comparison theorem, that $\big(I(t), H(t)\big) \to (0,0)$. Substituting $I = H = 0$ in the first equation of the model system (12) gives $S(t) \to S_0$ as $t \to \infty$. Thus, $\big(S(t), I(t), H(t)\big) \to \big(\frac{\pi}{\mu}, 0, 0\big)$ for $R_{01} < 1$, so that E_{01} is globally asymptotically stable in Ω if $R_{01} < 1$. The public health implication of the above result is that if the control strategy for HIV/AIDS can force the reproduction number to less than unity then HIV/AIDS can be eliminated from the community.

3.4 Existence of Endemic Equilibrium of the Model with Mass Action Incidence

In the presence of HIV/AIDS, that is, $I(t) \neq 0, H(t) \neq 0$, the model system (2) has an equilibrium point, called the endemic equilibrium point , denoted by E_1 and given by:

$E_{11} = (S^+, I^+, H^+) \neq 0$

E_{11} is the steady state endemic equilibrium point where the disease persist in the population. For the existence and uniqueness of E_{11} its coordinate has to satisfy the following conditions.

$0 < S^*, 0 < I^*, 0 < H^*$

At steady state, we equate the left hand side of (12) to zero and solve for the state variables as follows. From the third and second equation of we obtained

$$H^+ = \frac{\alpha I^+}{B} \text{ and } S^+ = \frac{\left(A - k_1 - k_1 r\frac{\alpha}{B}\right)}{c_1\beta_1 + c_2\beta_2\frac{\alpha}{B}}, \text{respectively}$$

where

H^+, I^+ and S^+ denote the state variables at the endemic equilibrium and

$$A = (\alpha + \theta_1 + \mu - m_1), \quad B = (\theta_2 + \mu - m_2), \quad k_1 = b(1-\varphi).$$

From the first equation of (12) at the steady state we have

$$\pi + \varphi b I^+ + \varphi b r\frac{\alpha}{B}I^+ - \left[c_1\beta_1 I^+ + c_2\beta_2\frac{\alpha}{B}I^+ + \mu\right]S^+ = 0$$

Or

$$\pi + \varphi b I^+ + \varphi b r\tfrac{\alpha}{B}I^+ - \left[c_1\beta_1 I^+ + c_2\beta_2\tfrac{\alpha}{B}I^+ + \mu\right]\left[\tfrac{A-k_1-k_1r\frac{\alpha}{B}}{c_1\beta_1+c_2\beta_2\frac{\alpha}{B}}\right] = 0$$

$$\Rightarrow \left[\varphi b + \varphi b r\frac{\alpha}{B}\right]I^+ - \left[c_1\beta_1 + c_2\beta_2\frac{\alpha}{B}\right]\left[\frac{A-k_1-k_1r\frac{\alpha}{B}}{c_1\beta_1+c_2\beta_2\frac{\alpha}{B}}\right]I^+ = \mu\left[\frac{A-k_1-k_1r\frac{\alpha}{B}}{c_1\beta_1+c_2\beta_2\frac{\alpha}{B}}\right] - \pi$$

Hence $I^+ = \dfrac{\mu\left[\frac{A-k_1-k_1r\frac{\alpha}{B}}{c_1\beta_1+c_2\beta_2\frac{\alpha}{B}}\right]-\pi}{\left[\varphi b+\varphi b r\frac{\alpha}{B}\right]-\left[c_1\beta_1+c_2\beta_2\frac{\alpha}{B}\right]\left[\frac{A-k_1-k_1r\frac{\alpha}{B}}{c_1\beta_1+c_2\beta_2\frac{\alpha}{B}}\right]}$ (14)

For the existence of the endemic equilibrium I^+ have to be greater than zero. This is true in (14) if $\mu\left[\frac{A-k_1-k_1r\frac{\alpha}{B}}{c_1\beta_1+c_2\beta_2\frac{\alpha}{B}}\right] > \pi$

Or $\mu(A - k_1 - k_1 r\frac{\alpha}{B}) > (c_1\beta_1 + c_2\beta_2\frac{\alpha}{B})\pi$

$$\Rightarrow \mu AB > B[\pi c_1\beta_1 + \mu k_1] + \alpha[c_2\beta_2 + \mu k_1 r] \quad (15)$$

Or $\frac{1}{R_{01}} > 1$

or $R_{01} < 1$

From (15) we can also have

$$-\mu AB < -(B[\pi c_1\beta_1 + \mu k_1] + \alpha[c_2\beta_2 + \mu k_1 r])$$

Dividing through by $-\mu AB$ gives

$1 < R_{01}$ or $R_{01} > 1$

4. THE MODEL WITH NON-LINEAR INCIDENCE

We now present the model with non-linear incidence as follows

$$\left.\begin{aligned}
\frac{dS}{dt} &= \pi + \varphi b I^j - \left(\frac{c_1\beta_1 I}{1+a_1 I^2} + \frac{c_2\beta_2 H}{1+a_2 H^2} + \mu\right) S \\
\frac{dI}{dt} &= \left(\frac{c_1\beta_1 I}{1+a_1 I^2} + \frac{c_2\beta_2 H}{1+a_2 H^2}\right) S + m_1 I + b(1-\varphi) I^j - (\alpha + \theta_1 + \mu) I \\
\frac{dH}{dt} &= \alpha I + m_2 H - (\theta_2 + \mu) H \\
\frac{dA}{dt} &= \theta_1 I + \theta_2 H - (\delta_3 + \mu) A
\end{aligned}\right\} 16$$

where the variables and parameters retain the same meanings as in previous sections. Similar to previous sections, since the variable *A* does not appear in the first three equation we shall be considering the only the equations in (17) for our analysis

$$\left.\begin{aligned}
\frac{dS}{dt} &= \pi + \varphi b I^j - \left(\frac{c_1\beta_1 I}{1+a_1 I^2} + \frac{c_2\beta_2 H}{1+a_2 H^2} + \mu\right) S \\
\frac{dI}{dt} &= \left(\frac{c_1\beta_1 I}{1+a_1 I^2} + \frac{c_2\beta_2 H}{1+a_2 H^2}\right) S + m_1 I + b(1-\varphi) I^j - (\alpha + \theta_1 + \mu) I \\
\frac{dH}{dt} &= \alpha I + m_2 H - (\theta_2 + \mu) H
\end{aligned}\right\} 17$$

We shall consider the model in the region $\Omega = \{(S, I, H): (S + I + H) \leq \frac{\pi}{\mu}, S > 0, I \geq 0, H \geq 0\}$ (see previous sections)

We note that the analytical results of the model with non – linear incidence is somewhat similar to that of the mass action model. For example in determining the basic reproduction number using same approach in section 3.1 we note that

$$F = \begin{bmatrix} c_1\beta_1 \frac{\pi}{\mu} + b(1-\varphi) & c_2\beta_2 \frac{\pi}{\mu} + br(1-\varphi) \\ 0 & 0 \end{bmatrix}$$

and

$$V = \begin{bmatrix} (\alpha + \theta_1 + \mu - m_1) & 0 \\ -\alpha & (\theta_2 + \mu - m_2) \end{bmatrix}$$

therefore the basic reproduction number of the non – linear incidence model, R_{02} is equal to the basic reproduction number of the non – linear incidence model R_{01} that is,

$$R_{02} = R_{01} = \frac{\left((\theta_2 + \mu - m_2)(c_1\beta_1\pi + \mu b(1-\varphi)) + \alpha(c_2\beta_2\pi + \mu br(1-\varphi))\right)}{\mu(\alpha + \theta_1 + \mu - m_1)(\theta_2 + \mu - m_2)}$$

The non linear incidence model has a disease free equilibrium state $E_{02} = (\frac{\pi}{\mu}, 0, 0)$ The Jacobian matix associated with the non – linear incidence model at the DFE is

$$J(E_{02}) = \begin{bmatrix} -\mu & \varphi b - c_1\beta_1 \frac{\pi}{\mu} & \varphi br - c_2\beta_2 \frac{\pi}{\mu} \\ 0 & c_1\beta_1 \frac{\pi}{\mu} + b(1-\varphi) - (\alpha + \theta_1 + \mu - m_1) & c_2\beta_2 \frac{\pi}{\mu} + br(1-\varphi) \\ 0 & \alpha & -(\theta_2 + \mu - m_2) \end{bmatrix}$$

Which is the same as the jacobian matrix associated the mass action model at its DFE, we therefore state theorem 9 whose prove follows the same approach as in theorem 7

Theorem 9

The model with non linear incidence is locally asymptotically whenever $R_{02} < 1$

4.1 Global Stability of the Disease Free Equilibrium of the model with non – linear incidence

Theorem 10

The disease – free equilibrium of the system (17) is globally asymptotically stable provided $R_{02} < 1$ *and the conditions in (18) are satisfied.*

Proof:

The global asymptotic stability of the disease free state of the model is investigated using the approach by Castillo-Chavev *et al* [45]. First the model is rewritten as

$$\frac{dW}{dt} = P(W, Z),$$

$$\frac{dZ}{dt} = G(W, Z), G(W, 0) = 0$$

where $W = (S) and\ Z = (I, H)$, with the components of $W \in \mathbb{R}$ denoting the uninfected population and the component of $Z \in \mathbb{R}^2$ denoting the infected population. $E_{02} = (W^*, 0)$ represent the disease-free equilibrium of the system. The conditions for global stability are

$$\left.\begin{array}{ll} (i) & \frac{dW}{dt} = P(W, 0), W^* \text{ is globally asymptotically stable} \\ (ii) & G(W, Z) = NZ - \hat{\mathrm{G}}(W, Z), \hat{\mathrm{G}}(W, Z) \geq 0 \text{for } (W, Z) \in \Omega \end{array}\right\} 18$$

where $N = D_z G(W^*, 0)$ is an M-matrix (the off diagonal elements of N are non-negative) and Ω is the region where the model makes biological sense. If the system (17) satisfies the two conditions above then according to Castillo-Chavez *et al.* [45], the following theorem holds. Following same approach in section 2.4, we shall apply theorem (4). To apply theorem (4) on the system (17) we now proceed thus:

$$W = S(t),\ Z = (I, H), F(W, 0) = (\pi - \mu S(0))$$

$$N = D_z G(W^*, 0) = \begin{pmatrix} c_1\beta_1 + b(1-\varphi) - (\alpha + \theta_1 + \mu - m_1) & c_2\beta_2 + br(1-\varphi) \\ \alpha & -(\theta_2 + +\mu - m_2) \end{pmatrix}$$

and for $\hat{\mathrm{G}}(W, Z) = NZ - G(W, Z)$, we have

$$NZ = \begin{pmatrix} c_1\beta_1 + b(1-\varphi) - (\alpha + \theta_1 + \mu - m_1) & c_2\beta_2 + br(1-\varphi) \\ \alpha & -(\theta_2 + \mu - m_2) \end{pmatrix} \begin{pmatrix} I \\ H \end{pmatrix}$$

$$G(W, Z) = \begin{pmatrix} \lambda S + m_1 I + b(1-\varphi) I^j - (\alpha + \theta_1 + \mu) I \\ \alpha I + m_2 H - (\theta_2 + \mu) H \end{pmatrix}$$

$$\hat{\mathrm{G}}(W, Z) = \begin{pmatrix} c_1\beta_1 I\left(1 - \frac{S}{1 + a_1 I^2}\right) + c_2\beta_2 H\left(1 - \frac{S}{1 + a_2 H^2}\right) \\ 0 \end{pmatrix}$$

Note that $\hat{\mathrm{G}}(W, Z) \geq 0$ or the positivity $\hat{\mathrm{G}}(W, Z)$ is certain only when $S < 1 + a_1 I^2$ and $S < 1 + a_2 H^2$ which may not be true therefore the system (17) may not be globally asymptotically stable since one of the conditions of (18) is violated. This completes the prove of theorem 10. The implication is that the disease free equilibrium may co-exist with the endemic equilibrium when $R_{02} < 1$ hence the tradition method of reducing the reproduction number below unity to control the disease is still necessary but no longer sufficient to control the disease.

4.2 Existence of endemic equilibrium for the model with non – linear incidence

Let

$$\lambda^{**} = \left(\frac{c_1\beta_1 I^{**}}{1 + a_1 I^{**2}} + \frac{c_2\beta_2 H^{**}}{1 + a_2 H^{**2}}\right) \qquad (19)$$

Then at the steady state we have

$$\left.\begin{array}{l} 0 = \pi + \varphi b I^{**j} - (\lambda^{**} + \mu) S^{**} \\ 0 = \lambda^{**} S^{**} + m_1 I^{**} + b(1-\varphi) I^{**j} - (\alpha + \theta_1 + \mu) I^{**} \\ 0 = \alpha I^{**} + m_2 H^{**} - (\theta_2 + \mu) H^{**} \end{array}\right\} 20$$

Where S^{**}, I^{**} and H^{**} represent the state variables at the endemic equilibrium and

$$I^{**j} = I^{**} + rH^{**}$$

We now solve for S^{**}, I^{**} and H^{**} in terms of λ^{**} From the first equation of (20) we have

$$S^{**} = \frac{\pi + \varphi b I^{**} + \varphi b r H^{**}}{\lambda^{**} + \mu} \qquad (21)$$

From the second equation of (20) we have

$$I^{**} = \frac{\lambda^{**} S^{**} + k_1 r H^{**}}{A - k_1} \qquad (22)$$

And from the third equation of (20) we have

$$H^{**} = \frac{\alpha I^{**}}{B} \qquad (23)$$

Where $A, B\ and\ k_1$ retain their previous meanings Putting (23) in (22) and solving for I^{**} we obtained

$$I^{**} = \frac{B\lambda^{**} S^{**}}{B(A - k_1) - k_1 r\alpha} \qquad (24)$$

and putting (24) in (23) we obtained

$$H^{**} = \frac{\alpha\lambda^{**} S^{**}}{B(A - k_1) - k_1 r\alpha} \qquad (25)$$

Putting (25) and (24) into (21) and solving for S^{**} we have

$$S^{**} = \frac{\pi[B(A - k_1) - k_1 r\alpha]}{(\lambda^{**} + \mu)[B(A - k_1) - k_1 r\alpha] - \varphi b\lambda^{**}(B + r\alpha)} \qquad (26)$$

Putting (26) in (25) and (24) we obtained

$$I^{**} = \frac{B\lambda^{**}\pi[B(A-k_1)-k_1r\alpha]}{[B(A-k_1)-k_1r\alpha][(\lambda^{**}+\mu)[B(A-k_1)-k_1r\alpha]-\varphi b\lambda^{**}(B+r\alpha)]} \quad (27)$$

$$\text{and} \quad H^{**} = \frac{\alpha\lambda^{**}\pi[B(A-k_1)-k_1r\alpha]}{[B(A-k_1)-k_1r\alpha][(\lambda^{**}+\mu)[B(A-k_1)-k_1r\alpha]-\varphi b\lambda^{**}(B+r\alpha)]} \quad (28)$$

respectively.

In terms of λ^{**}, which is the force of infection, S^{**}, I^{**} and H^{**} are given by (26), (27) and (28) respectively. λ^{**} can be gotten by substituting (27) and (28) into (19) and solving for λ^{**} which leads to a rigorous algebra.

5. NUMERICAL ANALYSIS

We now present numerical simulations for the standard incidence model using reasonable parameter values. All simulations are performed using Matlab. It is worthy to note that although carefully chosen our parameter values are theoretical and may not be biologically realistic.

Table 3: Nominal parameter values

PARAMETERS	Nominal value
π	100 000
φ	0.7
B	0.03
r	0.99
m_1	0.06
m_2	0.03
θ_1	[0,1]
θ_2	[0,1]
β_1	0.2
β_2	0.08
c_1	4
c_2	2
α	0.3
μ	0.2

Fig. 2 and 3 show the total number of infectives and total number of infectives on HAART (i.e. I and H) plotted against the total susceptible population. The figures were obtained from the following set of initial conditions

1. $S(0) = 10000000,\ I(0) = 40000,\quad H(0) = 1000$
2. $S(0) = 9500000,\quad I(0) = 35000,\ H(0) = 800$
3. $S(0) = 900000,\ I(0) = 30000,\ H(0) = 700$
4. $S(0) = 700000,\ I(0) = 15000,\ H(0) = 500$

The result is the dynamic behaviour of the endemic equilibrium $E_1 = (S^*, I^*, H^*)$

where $S^* = 600\,000,\ I^* = 400\,000,\ H^* = 1\,250\,000$

Since given any initial condition, the solutions of the system (2) approaches E_1 we therefore conclude that the system (2) is globally stable about the endemic equilibrium point E_1.

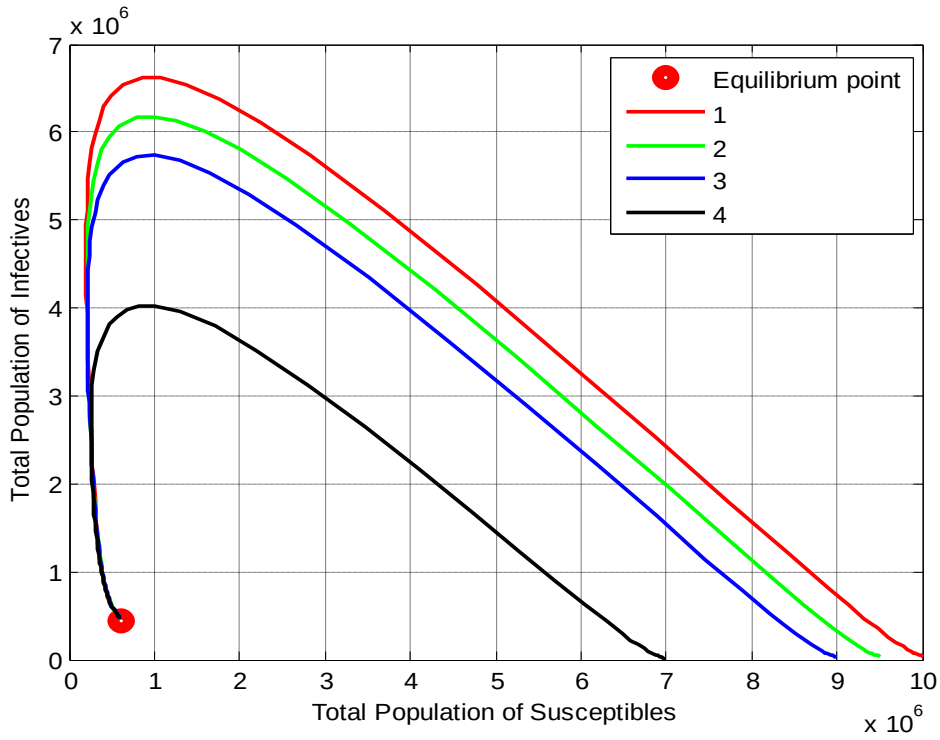

Fig. 2. Phase portrait : Infectives Vs. Susceptibles.

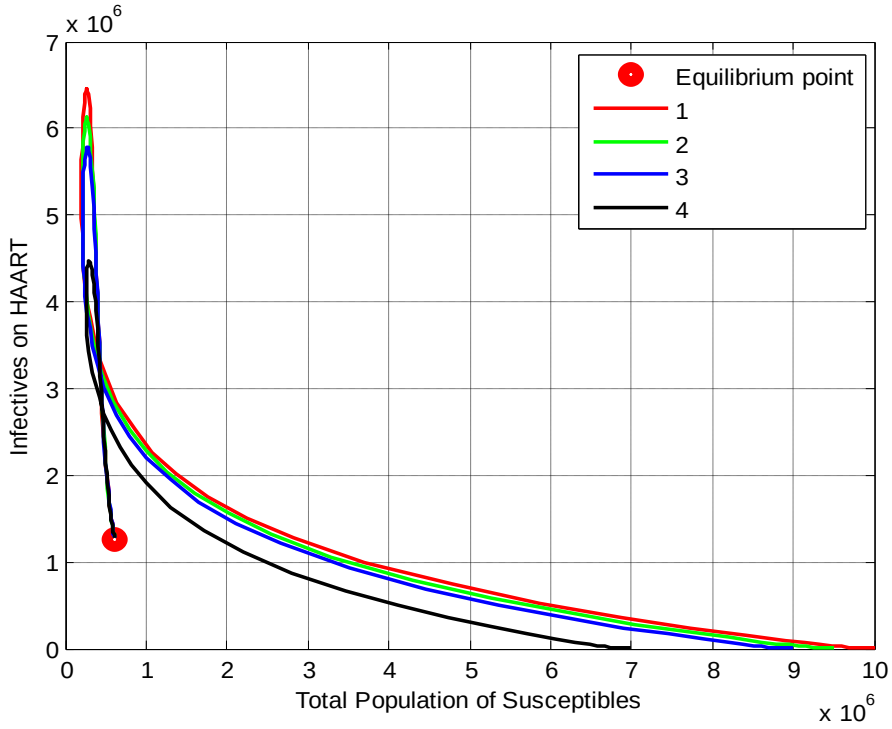

Fig. 3. Phase portrait: Infectives on HAART Vs. Susceptibles

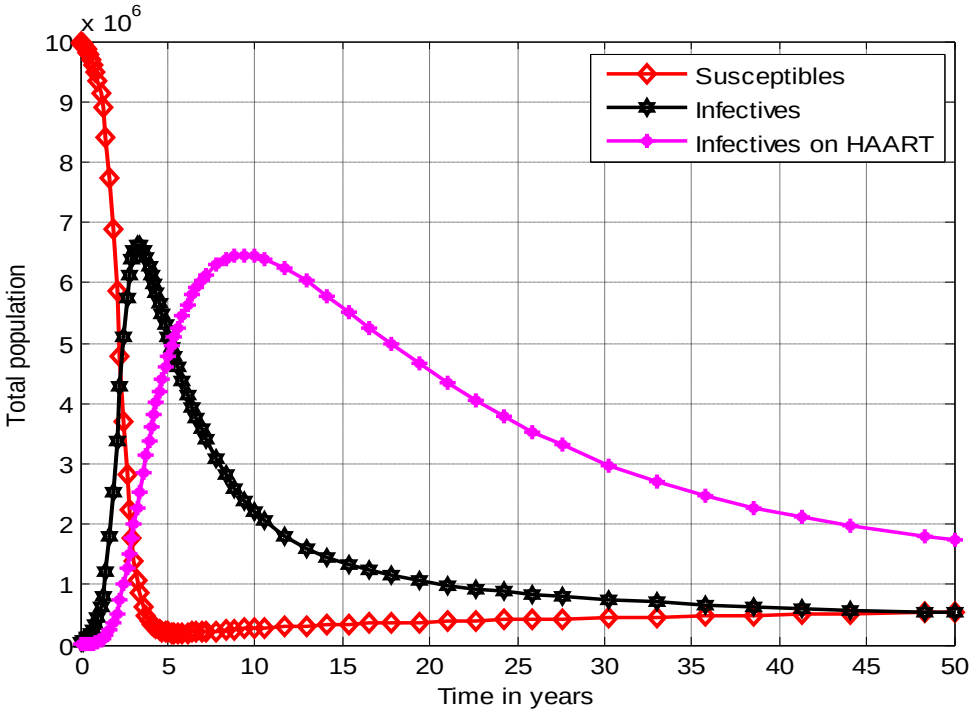

Fig. 4. Time series graph of the standard incidence model.

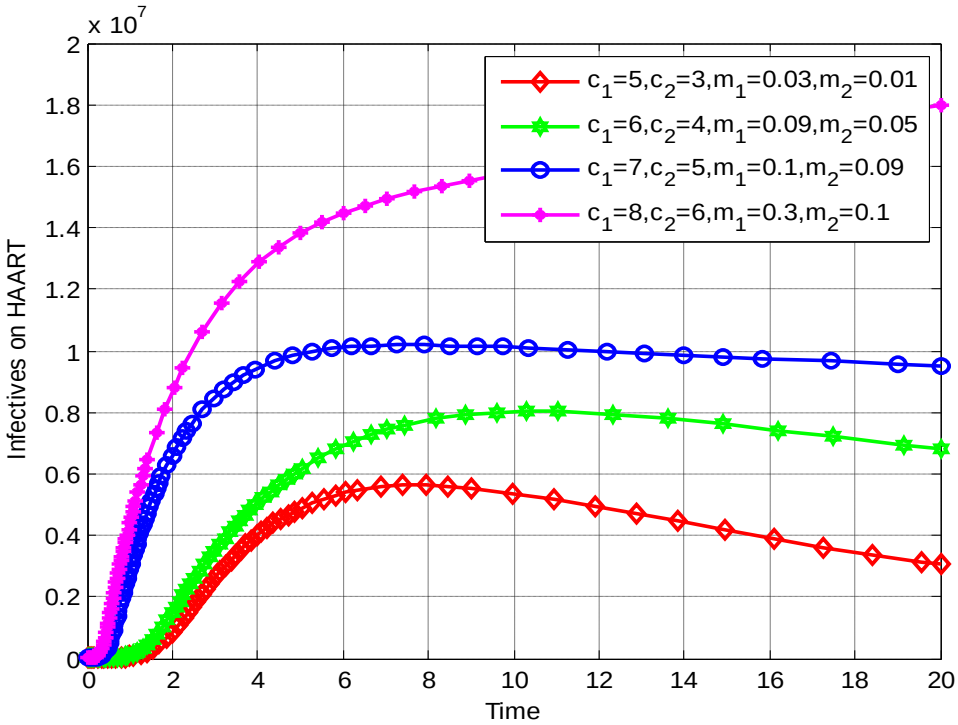

Fig. 5. Variation of parameters

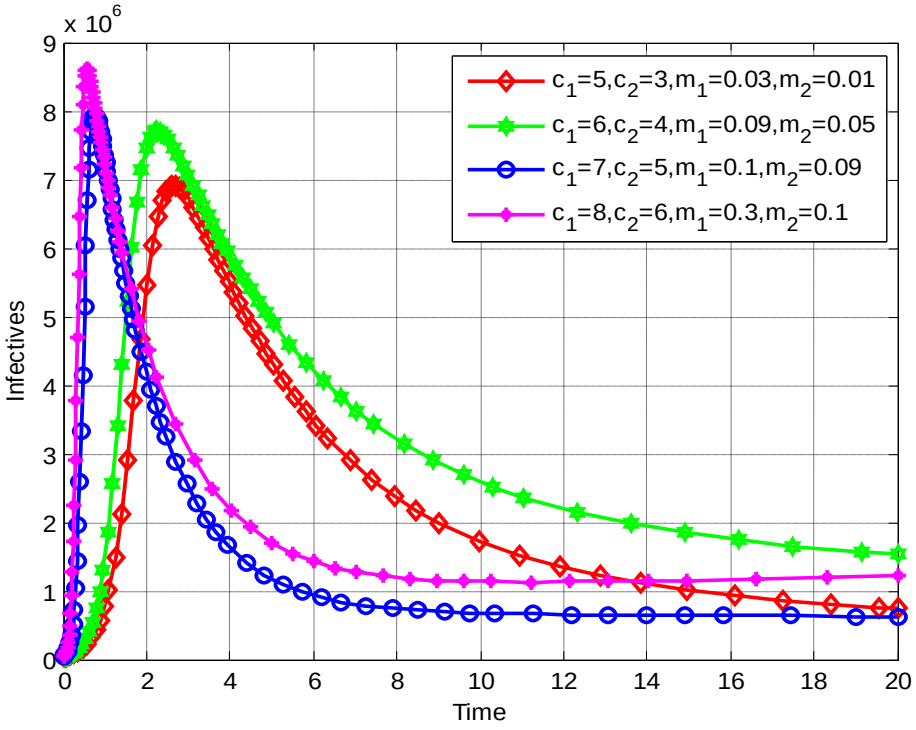

Fig. 6. Variation of parameters

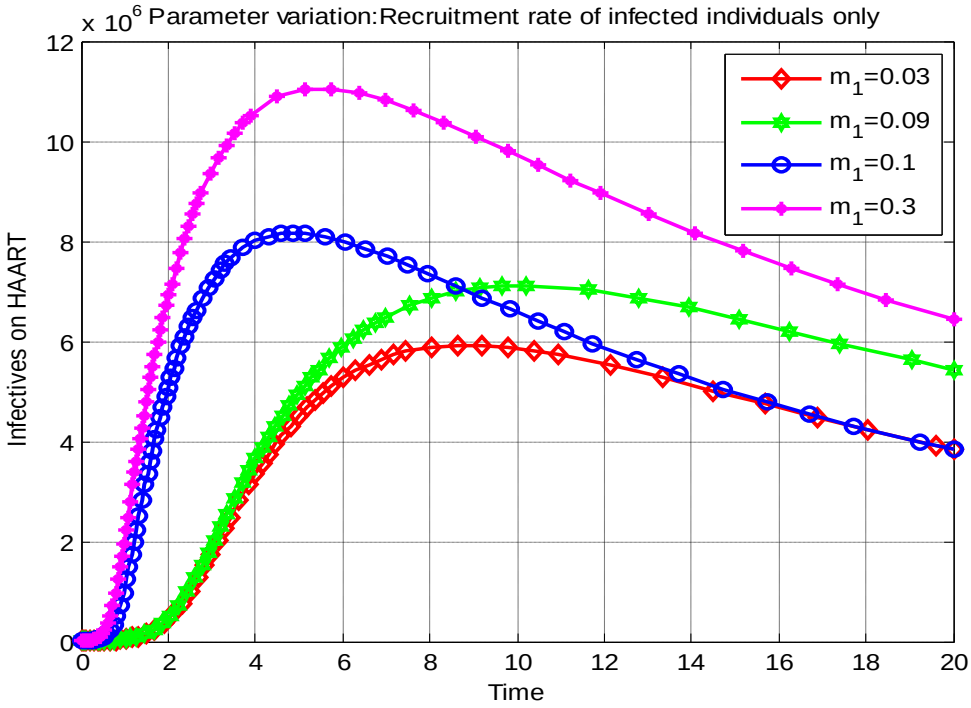

Fig. 7. Variation of parameter

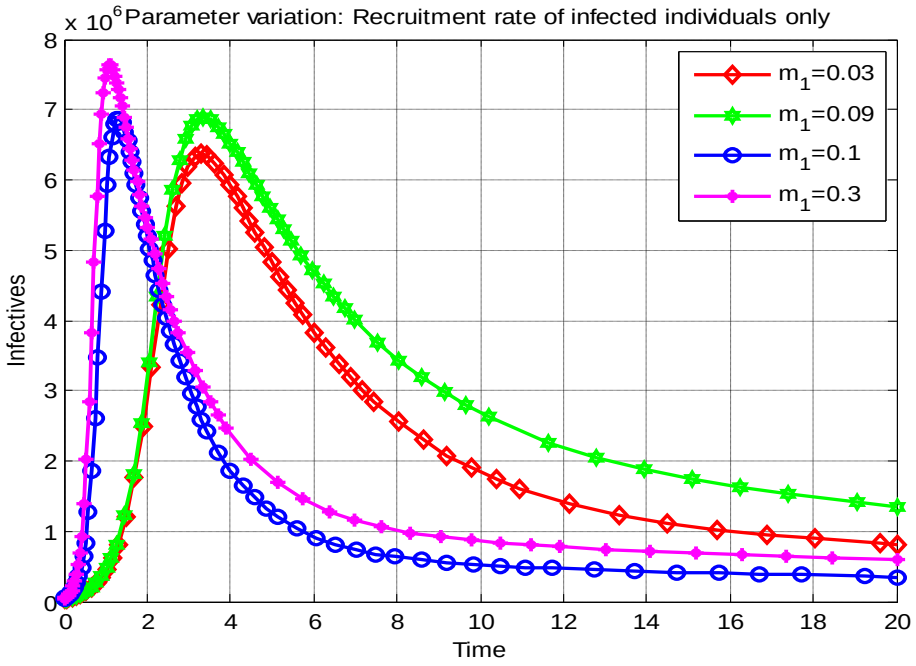

Fig. 8. Variation of parameter

Fig. 4. is a time series graph of the standard incidence model. From the graph the disease persists in the population and was never completely wiped out. Fig. 5 and 6 are simulations with varying parameters, particularly the number of sexual partners and recruitment rate of infectives. The combined effect of these two parameters increases the disease transmission. Fig. 7 and 8 varies the recruitment rate of infectives alone while keeping other parameters constant. Increasing this parameter also increases disease prevalence but effects are less as compared to combining it with increase in number of sexual partners. Attempt was made to use the same parameter values in table 3 in simulating the mass action and the saturated incidence models but the results obtained were not biologically realistic and hence not presented here.

6. CONCLUDING REMARKS

We modeled HIV/AIDS in the presence of infected immigrants and vertical transmission using three different incidence functions. Because of its simplicity, the mass action incidence was the least cumbersome to deal with analytically although it yielded results that are similar to that of the saturated incidence. For example the basic reproduction number of the mass action incidence and that saturated incidence are the same which translates to their stability conditions being the same. Further the numerical simulations of these two incidences using our parameter values yielded no biologically realistic results. Numerical simulations performed on the standard incidence model shows that the model system is globally asymptotically stable about the endemic equilibrium point. A finding of this work (which is expected) is that the basic reproduction number of the mass action model (and the saturated incidence model) is greater than that of the standard incidence model which implies that the average number of secondary transmission generated by a single infective in the period of infectiousness is greater in the mass action (and saturated incidence) model as compared to the standard incidence model. This alone might have made all the difference in the numerical simulation leading to the mass action incidence model producing biologically unrealistic result e.g a time series simulation of the mass action model shows that disease transmission reached its peak at time 0 (see appendix) which is not practical for a sexually transmitted disease. We thus uphold the claim by ([36] and references therein) that the standard incidence also called the frequency – dependent incidence is the appropriate incidence function that should be used in the modeling of STD's.

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Appendix

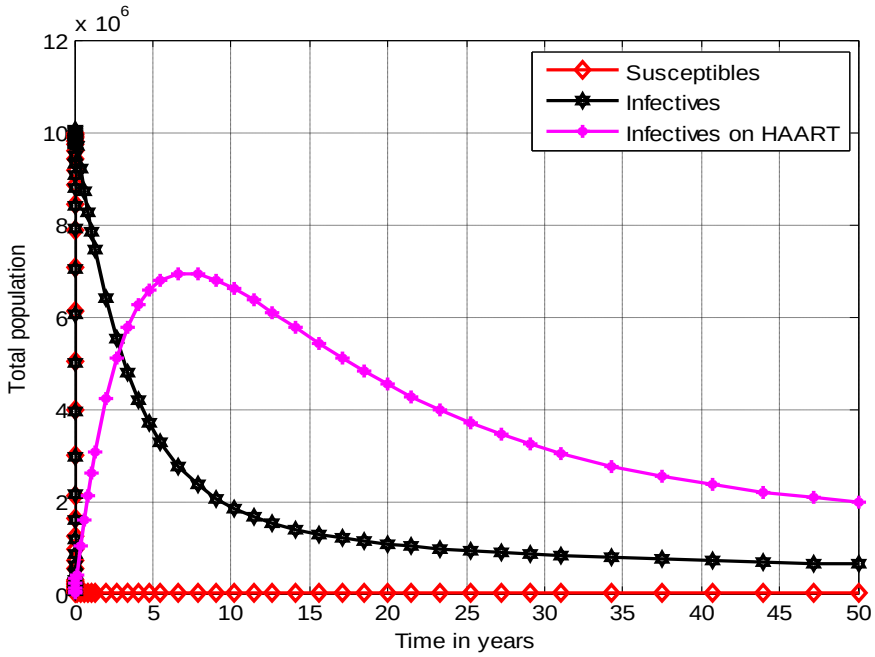

Fig.9. Time series of the mass action model