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MODELING THE EPIDEMIOLOGY AND THE TRANSMISSION DYNAMICS OF HIV/AIDS IN A HETEROSEXUAL POPULATION: BASIC MODELS AND ANALYSIS OF LOCAL STABILITY

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Abstract

Heterosexual dynamical models for HIV transmission in Nigeria are studied with the population divided into Susceptible, Infectious and AIDS compartments each for the male and female subpopulations. We assume that the AIDS compartment does not participate in sexual activities that could further cause infections among the susceptibles. The disease free and the endemic equilibria of the systems are studied analytically. The asymptotic behaviour of the endemic conditions of the systems is studied

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numerically. It is observed that both the disease free and the endemic equilibria for the systems are Locally Asymptotically Stable (LAS). Given uniform parameters (except for the global parameters), infections among females are observed to be relatively higher than infections among males.

1. Introduction

Many mathematical models that provide understanding of the epidemiology and the transmission dynamics of HIV/AIDS in Sub-Saharan Africa have been published [1-12]. Mathematical models have been used to study various aspects of HIV/AIDS epidemic: the epidemiology, the control, effect of some parameters and scenarios, interactions with some opportunistic infections, etc [1-17]. Unfortunately, only a few studies have addressed some specific areas of the epidemic in Nigeria [1, 5, 23].

Judging by the volume of mathematical models that have been suggested for the course of HIV/AIDS, it is surprising to note that this research area has not received due interest and attempt in Nigeria. In this paper, we present comprehensive transmission models for the transmission of HIV/AIDS in Nigeria. The models take into consideration the heterosexual sex which characterizes sexual networks in the Nigerian population. Results of a detailed exploration of different models and threshold parameters are also presented.

Although it is noted that some models addressing dynamics of HIV in heterosexual population in Sub-Saharan Africa have been published [4, 9, 10, 12-14, 18, 19], this work presents a comprehensive HIV transmission models suggested for HIV infection in a heterosexual population with some assumptions focusing on the understanding of local dynamics in Nigeria.

Specifically, Mukandavire and Garira [12] proposed a sex-structured model for heterosexual transmission of HIV/AIDS in which the population is divided into three subgroups of susceptible, infectives and AIDS. Dividing the subgroups into high-risk and low-risk sexual activity groups, movement of individuals from high to low-risk group due to the effect of public health education campaign was studied. It was discovered that although the presence of sex workers enlarges and fuels the epidemics among heterosexuals, public health educational campaign among the high-risk heterosexual population can help slow or eradicate the epidemic. Sani et al. [19] applied some stochastic models to the spread of HIV in a heterosexual population under the assumption of constant and varying population sizes. Using numerical experiments, the dynamic behaviour of the models and their

approximations were explored extensively. In addition, Garnett et al. [10] presented analysis of a mathematical model of the spread and demographic impact of HIV in heterosexual communities in developing countries. The behaviour of the model and sensitivity analyses were conducted numerically to assess the significance of different assumptions and parameters. Also, simulated pattern of HIV spread across the two sexes and various age groups were compared with observed patterns in Uganda [10]. It was found that the pattern of mixing between age, sexual activity classes and the assumptions have major influence on the prediction pattern of HIV spread and demographic impact of AIDS.

Furthermore, using a deterministic mathematical model, Renton et al. [14] used survey derived estimates of sexual behaviours in a heterosexual London population to estimate the effect on an HIV epidemic of different levels of STD prevalence in such a population. It was found that it is likely that epidemics of heterosexually transmitted HIV infection in developed countries have been by the relative success of effort to control STD. In the developing countries, simple STD care is likely to be a highly cost effective strategy to prevent HIV transmission.

2. Model Description

We formulated mathematical models to assess the effect of commercial sex workers in the transmission of HIV infection in Nigeria. In order to remove ambiguities from our models, we make some assumptions that are primarily applicable to the HIV infections in Nigeria as follows:

- HIV transmission is majorly through heterosexual sex and every other route through which infection could occur are regarded as minor routes.
- It is not possible for infection to occur between individuals of the same sex; we assume that there are no homosexuals.
- The population is divided in the following major groups: the populations of men (married and unmarried) who are sexually active and women (married or unmarried) who are sexually active. We further assume that each of these population consists of susceptible, infected and the AIDS compartments.
- For simplicity and based on our local understanding of the dynamics of HIV/AIDS in Nigeria, we assume that the individuals in the AIDS group do not contribute to the transmission of HIV since sexual intercourse is the

mode of transmission and it is difficult for any reasonable person to have sex with a person who is obviously AIDS.

We denote the population of the sexually active men at time t as $m(t)$ and the sexually active women as $fe(t)$ (this group shall hereafter be referred to as "female"). In addition, $m_i(t)$, $(i = 0, 1, 2)$ represent the number of men who are susceptible to HIV, those already infected but with CD4 cell count greater than 200mg/l and the AIDS patients, respectively. The same compartments for the female are represented by $fe_i(t)$, $(i = 0, 1, 2)$.

Furthermore, the average probability of (male-to-female) HIV transmission per sexual partner for male is β_m and the average probability of a female-to-male transmission is $\beta_f \cdot \mu_A$ and ν denotes the rate of AIDS associated death and progression to AIDS (by HIV infected individual), respectively, while ξ_i, $(i = m, f)$ is the rate of infection through none main route (such as through Injected Drug Use-IDU, homosexuality, blood transfusion due to accidents and other causes, etc) among the ith population/risk group, where $i = m$ denotes among men and $i = f$ denotes among female. Similarly, θ_i, $(i = m, f)$ and θ_{Ii}, $(i = m, f)$ are the rates of immigration into the ith population group of the susceptible and the infectious, respectively. μ_i, $(i = m, f)$ are the death rates (due to causes other than AIDS) among men and the female while K_i, $(i = m, f)$ are the rates of partner changes for the male and female population group.

The initial models, where $m(t) = m_0(t) + m_1(t) + m_2(t)$ and $fe(t) = fe_0(t) + fe_1(t) + fe_2(t)$, are given as follows:

The rate of change in the population of susceptible men $m_0(t)$ at time t is:

$$\dot{m_0}(t) = \theta_m - \xi_m m_0(t) - k_m \beta_f m_0(t) \frac{fe_1(t)}{fe(t)} - \mu_m m_0(t), \tag{2.1}$$

where all the parameters are as defined above.

The rate of change in the population of infected men $m_1(t)$ at time t is:

$$\dot{m_1}(t) = \theta_{\text{Im}} + \xi_m m_0(t) + k_m \beta_f m_0(t) \frac{fe_1(t)}{fe(t)} - \mu_m m_1(t) - \nu m_1(t), \tag{2.2}$$

where all the parameters are as defined earlier.

The rate of change in the population of men who are AIDS patients, $m_2(t)$ at time t is:

$$\dot{m_2}(t) = \nu m_1(t) - \mu_m m_2(t) - \mu_A m_2(t), \tag{2.3}$$

where all the parameters and variable are defined earlier.

Putting equations (2.1) to (2.3) together and letting $\lambda_f = k_m \beta_f \frac{fe_1(t)}{fe(t)}$, we have:

$$\left.\begin{aligned} \dot{m_0}(t) &= \theta_m - \xi_m m_0(t) - \lambda_f m_0(t) - \mu_m m_0(t) \\ \dot{m_1}(t) &= \theta_{\mathrm{Im}} + \xi_m m_0(t) + \lambda_f m_0(t) - \mu_m m_1(t) - \nu m_1(t) \\ \dot{m_2}(t) &= \nu m_1(t) - \mu_m m_2(t) - \mu_A m_2(t) \\ &\text{where} \\ \lambda_f &= k_m \beta_f \frac{fe_1(t)}{fe(t)} \end{aligned}\right\}, \tag{2.4}$$

λ_f is the force of infection for female-male HIV transmission.

Furthermore, for ease of explanation and analysis, further simplification of the model in equation (2.4) gives:

$$\left.\begin{aligned} \dot{m_0}(t) &= \theta_m - (\xi_m + \lambda_f + \mu_m) m_0(t) \\ \dot{m_1}(t) &= \theta_{\mathrm{Im}} + (\xi_m + \lambda_f) m_0(t) - (\mu_m + \nu) m_1(t) \\ \dot{m_2}(t) &= \nu m_1(t) - (\mu_m + \mu_A) m_2(t) \end{aligned}\right\}. \tag{2.5}$$

Similarly, following the same procedures and formulations, we also have the models for the female as follows:

$$\left.\begin{aligned} \dot{fe_0}(t) &= \theta_f - \lambda_m fe_0(t) - \xi_f fe_0(t) - \mu_f fe_0(t) \\ \dot{fe_1}(t) &= \theta_{If} + \lambda_m fe_0(t) + \xi_f fe_0(t) - \mu_f fe_1(t) - \nu fe_1(t) \\ \dot{fe_2}(t) &= \nu fe_1(t) - \mu_f fe_2(t) - \mu_A fe_2(t) \\ \lambda_m &= k_f \beta_m \frac{m_1(t)}{m(t)} \end{aligned}\right\}, \tag{2.6}$$

where λ_m is the force of infection for male-female HIV transmission and all the variables and parameters are as defined earlier.

Further simplification yields the equivalent model:

$$\left.\begin{aligned} fe_0^{\bullet}(t) &= \theta_f - (\xi_f + \lambda_m + \mu_f)\, fe_0(t) \\ fe_1^{\bullet}(t) &= \theta_{If} + (\lambda_m + \xi_f)\, fe_0(t) - (\mu_f + \nu)\, fe_1(t) \\ fe_2^{\bullet}(t) &= \nu fe_1(t) - (\mu_f + \mu_A)\, fe_2(t) \end{aligned}\right\}. \tag{2.7}$$

3. The Basic Reproductive Numbers

The basic reproductive number, R_0 is the average number of secondary infection in a wholly susceptible population resulting from the introduction of a typical infectious individual during the time span of the infection [15, 28]. In this study, R_0 is summarized by the behavioural characteristics of the host and the biological characteristics of the HIV pathogen in the host. In particular, R_0 measures the number of new infections generated by the introduction of a single high-risk HIV infectious individual in a community [12] and it is the probability of HIV transmission per unit time multiplied by the duration of the infectiousness. In equations (2.5) and (2.7), the parameters describing the biological characteristics of the pathogen are β_f, μ_m and ν for equation (2.5) and β_m, μ_f and ν for equation (2.7) while the parameters describing the behavioural characteristics of the host are K_m and K_f for equations (2.5) and (2.7), respectively. However, the parameters decaying the cohort of the infectious group are μ_m and ν for equation (2.5) and μ_f and ν for equation (2.7). Therefore, the basic reproductive numbers, R_{0m} for equation (2.5) (for introducing a single infectious female among totally susceptible men) and R_{0f} for equation (2.7) (for introducing a single infectious male among totally susceptible female) are:

$$R_{0m} = K_m \beta_f \left(\frac{1}{\nu + \mu_m}\right) \tag{3.1}$$

and

$$R_{0f} = K_f \beta_m \left(\frac{1}{\nu + \mu_f}\right), \tag{3.2}$$

where all the parameters have been explained earlier.

In summary, R_0 is defined mathematically as the probability of transmission per unit time multiplied by the duration of infection [15] and if $R_0 < 1$, then the epidemic can be eradicated or eventually disappears from the population because, on average, each infected person cannot ensure transmission of the infectious agent to a susceptible one; this results in new waves of infection being of lesser amplitude than preceding ones and finally, to disease elimination [20]. On the other hand, if $R_0 \geq 1$, the disease remains endemic and in particular, for $R_0 > 1$, the epidemic builds up [15, 20].

4. Stability Analysis of the Equilibria

In this section, we study all feasible equilibrium admitted by system (2.5). Notice that system (2.5) describes the dynamics of HIV transmission among heterosexual men in Nigeria. The stability analysis of the trivial equilibrium state, $\varepsilon_{0m} = (0, 0, 0)$ (which describes a situation where there are neither susceptible nor infection) is excluded from this study as it is epidemiologically meaningless and has no information to contribute to this study.

4.1. Stability analysis of the disease-free equilibrium, ε_{1m}

The disease-free equilibrium obtained by setting the derivatives in (2.5) to zero is

$$\varepsilon_{1m} = \left(\frac{(\theta_m + \theta_{\mathrm{Im}})}{\mu_m}, 0, 0 \right). \tag{4.1}$$

Evaluating the Jacobian of the system (2.5) at ε_{1m} gives

$$J_{1m} = \begin{bmatrix} -(\xi_m + \lambda_f + \mu_m) & 0 & 0 \\ (\xi_m + \lambda_f) & -(\mu_m + \nu) & 0 \\ 0 & \nu & -(\mu_m + \mu_A) \end{bmatrix}. \tag{4.2}$$

Using properties of matrix algebra, the eigenvalue of J_{1m} can be written as

$$(J_{1m} - \lambda I) K = 0, \tag{4.3}$$

where I is the multiplicative identity and the polynomial

$$P(\lambda) = | J_{1m} - \lambda I | \tag{4.4}$$

is the characteristic polynomial of the 3×3 matrix J_{1m}. Thus, the calculation of the eigenvalue of a 3×3 matrix reduces to finding the root of the polynomial of degree 3.

Accordingly, the eigenvalues of the matrix J_{1m} are the roots of the polynomial

$$P(\lambda) = -(\xi_m + \lambda_f + \mu_m)\begin{vmatrix} -(\mu_m + \nu) & 0 \\ \nu & -(\mu_m + \mu_m) \end{vmatrix}$$

$$= \lambda^3 + \lambda^2 K_1 + \lambda K_2 + K_3 = 0, \tag{4.5}$$

where

$$K_1 = (\xi_m + \lambda_f + \mu_m) + (\mu_m + \nu) + (\mu_m + \mu_A),$$

$$K_1 = K_3 = (\xi_m + \lambda_f + \mu_m) + (\mu_m + \nu) + (\mu_m + \mu_A).$$

Theorem 4.1. *The disease free equilibrium state* ε_{1m} *of the system* (2.5) *is locally asymptotically stable.*

Proof. The variational matrix associated with the disease free equilibrium is given by J_{1m} in equation (4.2) above and the corresponding characteristic polynomial is given in equation (4.5). It is clear that $K_1 > 0$, $K_2 > 0$, $K_3 > 0$ and $K_1 K_2 - K_3 > 0$. Thus, by the Routh-Hurwitz criteria, ε_{1m} is locally asymptotically stable (LAS) [12, 19, 24-27].

In addition, it is easy to see from equation (4.5) that the eigenvalues of J_{1m} and hence the roots of the polynomial $P(\lambda)$ are $\lambda_1 = -(\xi_m + \lambda_f + \mu_m)$, $\lambda_2 = -(\mu_m + \nu)$ and $\lambda_3 = -(\mu_m + \mu_A)$ which are all negative. Therefore, the system (2.5) is locally asymptotically stable [12, 19, 24-27].

Lemma 4.1. *The system* (2.5) *is unstable* [*stable*] *if the sum of the diagonal elements, i.e., trace of the variational matrix,* J_{1m} *is positive* [*negative*].

Proof. The eigenvalues of J_{1m} are $\lambda_1 = -(\xi_m + \lambda_f + \mu_m)$, $\lambda_2 = -(\mu_m + \nu)$ and $\lambda_3 = -(\mu_m + \mu_A)$. Notice that these are the diagonal elements of the matrix J_{1m}. Also, notice that

$$\sum_{i=1}^{n} \lambda_i = trace(J_{1m}) < 0. \tag{4.6}$$

If $\sum_{i=1}^{n}\lambda_i > 0$, then at least one of the λ_i, $i = 1, 2, 3$ is positive (or has a positive real part) and the system (2.5) would be unstable.

4.2. Stability analysis of the endemic equilibrium, ε_{2m}

For $R_{0m} > 1$, the endemic equilibrium ε_{2m} exists and it can be shown that ε_{2m} given in terms of R_{0m} is

$$\varepsilon_{2m} = (m_0^*(t),\ m_1^*(t),\ m_2^*(t)), \tag{4.7}$$

where

$$m_0^*(t) = \frac{\theta_m R_{0m}}{K_m\beta_f - R_{0m}(\nu - \xi_m - \lambda_f)},$$

$$m_1^*(t) = \theta_{\mathrm{Im}} - \frac{\theta_m R_{0m}(\xi_m + \lambda_f)}{K_m\beta_f - R_{0m}(\nu - \xi_m - \lambda_f)}$$

and

$$m_2^*(t) = \frac{R_{0m}\nu(\theta_{\mathrm{Im}}(K_m\beta_f - R_{0m}(\nu - \xi_m - \lambda_f)) - R_{0m}\theta_m(\xi_m + \lambda_f))}{K_m\beta_f(\xi_m + \lambda_f)(K_m\beta_f - R_{0m}(\nu - \xi_m - \lambda_f))}.$$

Theorem 4.2. *Let* R_{0m}, *the reproductive number, be as defined in equation* (3.1). *Then the endemic equilibrium,* ε_{2m} *is locally asymptotically stable if* $R_{0m} > 1\ (\beta_f > 0)$ *and does not exist if* $R_{0m} < 1$.

Proof. Given that $R_{0m} > 1$, the variational matrix for the endemic equilibrium ε_{2m} is given in terms of R_{0m} as

$$J_{2m} = \begin{pmatrix} -\alpha_1\left(1 - \dfrac{R_{0m}(\nu - \xi_m - \lambda_f)}{K_m\beta_f}\right) & 0 & 0 \\ (\xi_m + \lambda_f) & -\alpha_2 & 0 \\ 0 & \alpha_3\left(1 - \dfrac{R_{0m}\mu_m}{K_m\beta_f}\right) & -\alpha_4\left(1 - \dfrac{R_{0m}(\nu + \mu_A)}{K_m\beta_f}\right) \end{pmatrix}, \tag{4.8}$$

where $\alpha_1 = \alpha_2 = \alpha_3 = \alpha_4 = K_m\beta_f / R_{0m}$.

It can be shown that the eigenvalues of J_{2m} are

$$\lambda_1 = -\left(\frac{K_m\beta_f - R_{0m}(\nu - \xi_m - \lambda_f)}{R_{0m}}\right),\ \lambda_2 = -\left(\frac{K_m\beta_f}{R_{0m}}\right)$$

and

$$\lambda_3 = -\left(\frac{K_m\beta_f - R_{0m}(\nu - \mu_A)}{R_{0m}}\right).$$

Clearly, these eigenvalues show that the endemic steady state is locally asymptotically stable [12, 19, 24-27] (if $\beta_f > 0$) since all the eigenvalues of the variational matrix are negative.

Furthermore, if $R_{0m} < 1$, then by the definition of R_{0m} (the basic reproduction number), the infection can be eradicated or eventually dies out from the population because, on average, each infected person cannot ensure transmission of the infection to a susceptible one and hence, the endemic equilibrium state does not exist.

5. Analysis of the Model for the Dynamics of HIV among Female

The model for HIV transmission in the female population in Nigeria differs from that of the male population only in terms of parameter values and state space (models) variables; the structure and the dynamics of the models follow the same pattern because of the similarity in sexual activities (they are both heterosexuals) (see systems (2.5) and (2.7) for details).

Consequently, the state of equilibria and the variational matrices for the disease free equilibrium and the endemic equilibrium (when $R_{0f} > 1$) and their stability analyses are summarized in the theorems below but note that the analysis of the trivial equilibrium $\varepsilon_{0f} = (0, 0, 0)$ is not included in this analysis because of the reasons given in Section 4 above.

Theorem 5.1. *The disease-free equilibrium,* ε_{1f} *for the dynamics of HIV transmission in the female population*; *system* (2.7) *exist if* $\mu_f \neq 0$ *and it is locally asymptotically stable*.

Proof. The disease-free equilibrium state for the system (2.7) derived as in equation (4.1) is given as

$$\varepsilon_{1m} = \left(\frac{(\theta_f + \theta_{If})}{\mu_f}, 0, 0 \right). \tag{5.1}$$

It is obvious to see that ε_{1f} exists only if $\mu_f \neq 0$ and it is not equal to ε_{0f} iff $(\theta_f + \theta_{If}) \neq 0$. Evaluating the Jacobian of the system (2.7) at ε_{1f} gives

$$J_{1f} = \begin{bmatrix} -(\xi_f + \lambda_m + \mu_f) & 0 & 0 \\ (\xi_f + \lambda_m) & -(\mu_f + \nu) & 0 \\ 0 & \nu & -(\mu_f + \mu_A) \end{bmatrix}. \tag{5.2}$$

It follows from equation (4.5), Theorem 4.1 and Lemma 4.1 that the eigenvalues of J_{1f} are $\lambda_{1f} = -(\xi_f + \lambda_m + \mu_f)$, $\lambda_{2f} = -(\mu_f + \nu)$ and $\lambda_{3f} = -(\mu_f + \mu_A)$ which are all negative. Therefore, the system (2.7) is locally asymptotically stable [12, 19, 24-27].

Theorem 5.2. *The endemic equilibrium,* ε_{2f} *of the system* (2.7) *is locally asymptotically stable if* $R_{0f} > 1\,(\beta_m > 0)$, *and does not exist for* $R_{0f} < 1$.

Proof. Following from equation (4.7), we derived the expression for the endemic equilibrium state in terms of R_{0f} as

$$\varepsilon_{2f} = (fe_0^*(t),\ fe_1^*(t),\ fe_2^*(t)), \tag{5.3}$$

where

$$fe_0^*(t) = \frac{\theta_f R_{0f}}{K_f \beta_m - R_{0f}(\nu - \xi_f - \lambda_m)},$$

$$fe_1^*(t) = \theta_{If} - \frac{\theta_f R_{0f}(\xi_f + \lambda_m)}{K_f \beta_m - R_{0m}(\nu - \xi_f - \lambda_m)}$$

and

$$m_2^*(t) = \frac{R_{0f}\nu(\theta_{If}(K_f\beta_m - R_{0f}(\nu - \xi_f - \lambda_m)) - R_{0f}\theta_f(\xi_f + \lambda_m))}{K_f\beta_m(\xi_f + \lambda_m)(K_f\beta_m - R_{0f}(\nu - \xi_f - \lambda_m))}.$$

Expressing the variational matrix for the endemic equilibrium, ε_{2f} in terms of R_{0f} yields

$$J_{2f} = \begin{pmatrix} -\psi_1\left(1-\dfrac{R_{0f}(\nu-\xi_f-\lambda_m)}{K_f\beta_m}\right) & 0 & 0 \\ (\xi_f+\lambda_m) & -\psi_4 & 0 \\ 0 & \psi_3\left(1-\dfrac{R_{0f}\mu_f}{K_f\beta_{fm}}\right) & -\psi_4\left(1-\dfrac{R_{0f}(\nu+\mu_A)}{K_f\beta_m}\right) \end{pmatrix}, \tag{5.4}$$

where $\psi_1 = \psi_2 = \psi_3 = \psi_4 = K_f\beta_m / R_{0f}$.

Accordingly, the eigenvalues of J_{2f} are

$$\lambda_1 = -\left(\frac{K_f\beta_m - R_{0f}(\nu-\xi_f-\lambda_m)}{R_{0f}}\right), \quad \lambda_2 = -\left(\frac{K_f\beta_m}{R_{0f}}\right)$$

and

$$\lambda_3 = -\left(\frac{K_f\beta_m - R_{0f}(\nu-\mu_A)}{R_{0f}}\right).$$

Clearly, these eigenvalues show that the endemic steady state for the transmission of HIV in the female population represented by system (2.7) is locally asymptotically stable [12, 19, 24-27] since all the eigenvalues of the variational matrix, J_{2f} are negative.

If $R_{0f} < 1$, then by the definition of R_{0f}, the disease dies out and under this condition, the endemic equilibrium ε_{2f} does not exist but when $R_{0f} > 1$, the infection is maintained in the population and hence, the existence of the endemic equilibrium ε_{2f}.

6. Numerical Experiments and Discussion

In this section, we illustrate the behaviour of the male and female population models and their deterministic approximations via a number of numerical experiments. We give numerical simulation of the endemic equilibrium conditions of the models (2.5) and (2.7) using MATLAB® R2007a.

Apart from the other (local) parameters explained in the various experiments, the following parameters are the same in each experiment and are therefore regarded as global parameters: the transmission probabilities for male and female; $\beta_m = 0.1637$ and $\beta_f = 0.0497$, respectively, [12], the natural death rate for male and female; $\mu_m = 1/48$ and $\mu_f = 1/50$ (which corresponds to the life expectancies in Nigeria - 48 years for men and 50 years for women [19, 21, 22]), the death rate due to AIDS is $\mu_A = 1/20$ (which means a life expectancy for AIDS people of only 20 years [19]) and the rate of progression from HIV infection to AIDS is $\nu = 1/12$ (corresponding to incubation period of 12 years before an infected person develops AIDS).

6.1. Exploring the dynamics for the population of males

We study the dynamics of HIV infection in a small heterosexual population of men and women in Nigeria. For the sake of the experiments, we let $m_0(t) = 10000$ and $fe_0(t) = 100000$ susceptibles and studied the level of infection with the introduction of a single infected male into the population of this completely susceptible heterosexuals. Focusing on the behavioral parameters in the model, namely: the number of partner change per year, K_m, the rate of infections through none major route, ξ_m (this defines the level of involvement in other risky behaviours other than heterosexual sex) and the number of infected immigrants (particularly infected female), θ_{If}, we studied the behaviour of the system as time increases.

Figure 1 shows the endemic infection, $I^*(t)$ and the susceptibles, $S^*(t)$ among the male population at time t while Figures 2 to 4 show the variation in the level of infection among men due to changes in the number of infected female immigrants, the rate of infection through none major routes and the number of partner change in a year, respectively. It was observed in Figure 1 that within the first ten years after the introduction of the single infected male, the epidemic rises sharply but declined thereafter only to rise slowly again (after thirty years into the epidemic) and then remains endemic in the population. An approximate estimate of the endemic infection was 6,000 people.

In Figure 2, we observed that within 30 years after the introduction of the infective, the level of infection remains the same irrespective of the number of

infected female immigrants. However, a significant difference was observed in the level of infection as the epidemic grows older with a possibility of asymptotic convergence while in Figure 3, variation in the rate of infections through none major routes did not produce much difference in the level of infection. Figure 4 shows that the higher the number of sexual partners change, the higher the level of infection, but the levels of infection, when number of partner changes is greater or equal to 7, are very similar.

6.2. Exploring the dynamics for the population of females

Similarly, the results of the experiments for the dynamics of HIV infection among the female population were done using $fe_0(t) = 10000$ and $m_0(t) = 100000$ susceptible with the introduction of a single infected female into the population of this completely susceptible heterosexuals is presented in Figures 5 to 9.

In Figure 5, we see the behaviour of the endemic infection and the susceptibles among the female population at time *t*. It was observed that within the first ten years after the introduction of the single infected female, the epidemic rises sharply, declined thereafter to remain endemic in the population. An approximate estimate of the endemic infection was 7,000 people.

In Figures 6 to 8, the results for the variation in the level of infection among men due to changes in the number of infected female immigrants, the rate of infection through none major routes and the number of partners change in a year, respectively, are presented. In Figure 6, it was observed that within 30 years after the introduction of the infectious female, the level of infection remains the same irrespective of the number of infected male immigrants into the population. However, a significant difference was observed in the level of infection as the epidemic grows older with a possibility of an asymptotic convergence. The levels of infection due to variation in the rate of infections through none major routes are similar for the various rates of infection (Figure 7) while we observed in Figure 4 that higher number of sexual partner change increases the level of infection. However, the levels of infection for changes in the number of partners greater or equal to 5 are similar.

6.3. Comparison of HIV infections among male and female populations

The results of the systems (2.5) and (2.7) were compared when every other parameter values (except the global parameters listed in Section 6) and variables are

taken to be the same. In Figures 9 to 12, we compared the level of the endemic infection among male and female populations. For equal number of partners change, the endemic infections among females were higher particularly for partners change less than 5 while for the number of partners greater or equal to 5, the levels of infection in the two populations are asymptotically the same (Figure 9). The levels of infection remained higher among females for all rates of infections through none major routes considered (Figure 10).

In addition, it was also observed in Figures 11 and 12 that whether the population was closed to immigrants or not, infections among females were higher than infections among males.

7. Conclusions

In this paper, transmission dynamical models are proposed and analyzed to study the transmission of HIV/AIDS in a heterosexual community of adults in a community like Nigeria. The models allow for constant recruitment into the susceptible and the infectious compartment. Recruitment into the infectious compartment is possible under the assumption that many infected people (in Nigeria) get diagnosed only when AIDS has set into their conditions and even if they get to know of their status early, they are very likely to migrate to areas where people are not conversant with their HIV status.

We found that the threshold parameters R_{0m} and R_{0f} determine whether the infection will persist in the populations or not. If $R_{0m} > 1$, then the endemic equilibrium point, ε_{2m} exists and the infection remains endemic in the male population while if $R_{0f} > 1$, then the endemic equilibrium point, ε_{2f} exists and the infection is endemic in the female population. Also, if $R_{0m} > 1$ and $R_{0f} > 1$, then the endemic equilibria, ε_{2m} and ε_{2f} for the systems (2.5) and (2.7), respectively, are Locally and Asymptotically Stable (LAS).

From the numerical analysis, it was observed that when a single infected person is introduced into the population of either sex, the epidemic is likely to rise sharply at the unset and then stabilizes asymptotically as time increases. It was also observed that although we cannot doubt the existence of infections with HIV from sources

other than heterosexual sex in Nigeria, the models show that the transmission dynamics of HIV/AIDS in Nigeria is more sensitive to HIV transmissions through heterosexual sex than any other route. Comparative studies of the level of infections among male and female populations show that women, rather than men, stand higher risk of HIV infections in Nigeria.

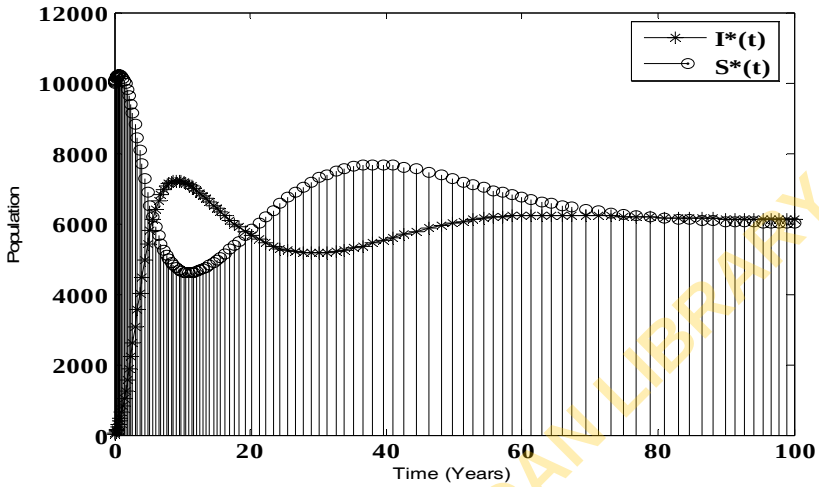

Figure 1. The endemic level of infection and the susceptible men.

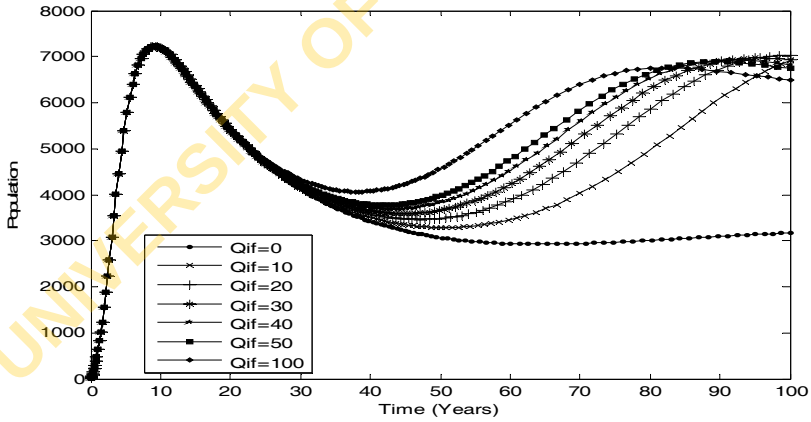

Figure 2. Variation of infected men due to variation in number of infectious female immigrants.

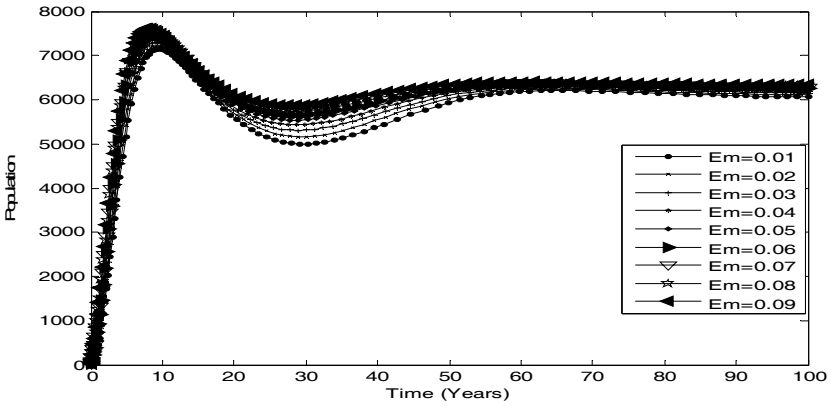

Figure 3. Variation of infected men due to variation in the rate of infection from none major routes.

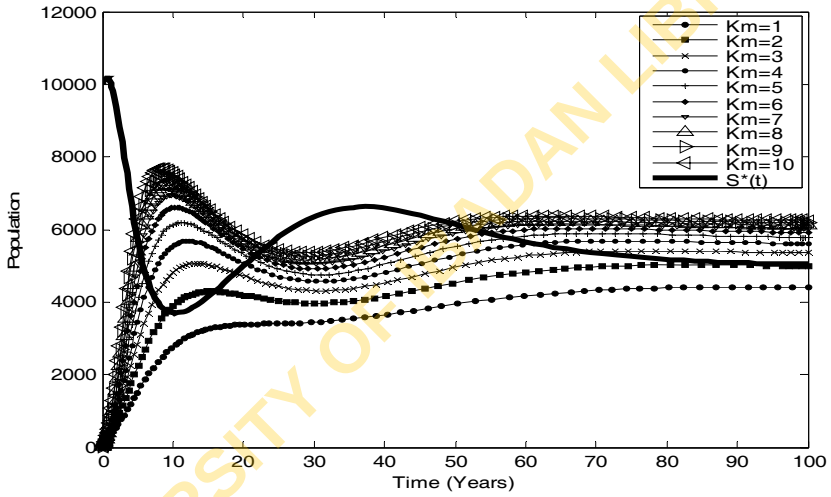

Figure 4. Variation of infected men due to variation in the number of sexual partners per year.

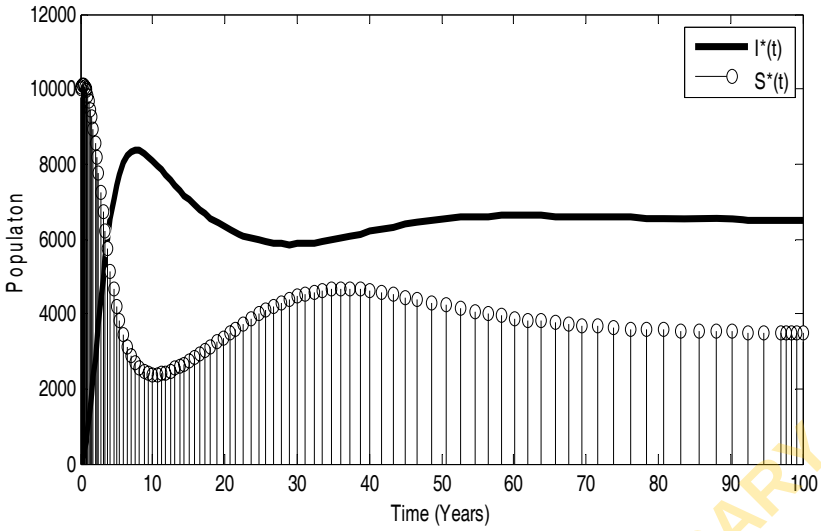

Figure 5. The endemic infectious and the susceptible women.

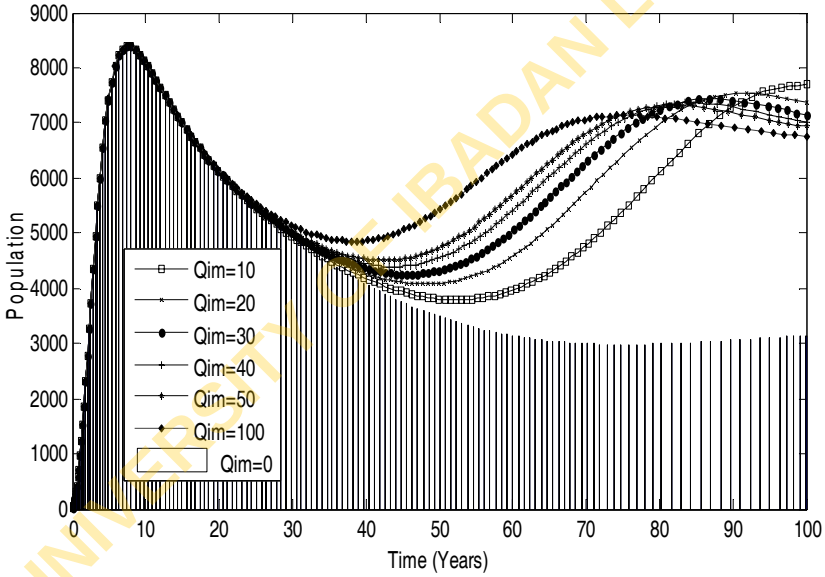

Figure 6. Variation of infected women due to variation in number of infectious male immigrants.

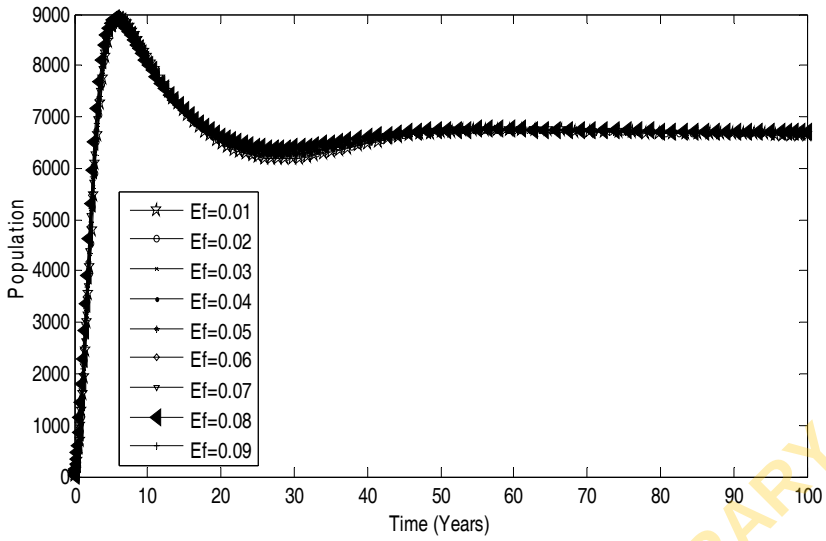

Figure 7. Variation of infected women due to variation in the rate of infection from none major routes.

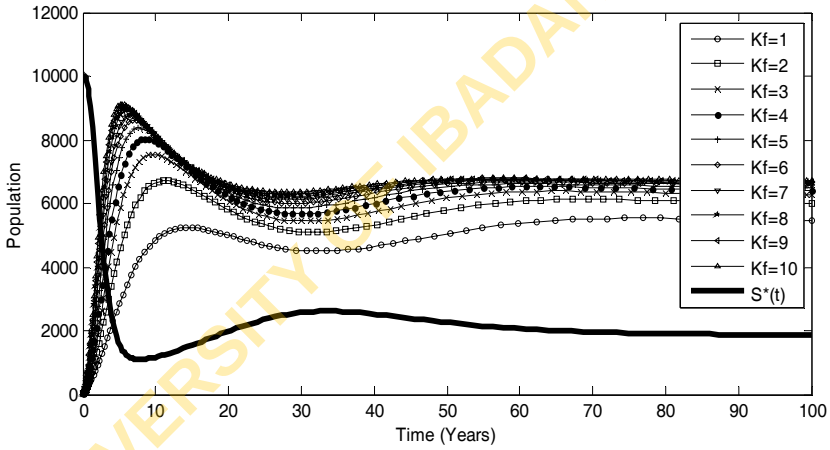

Figure 8. Variation of infected men due to variation in the number of sexual partners per year.

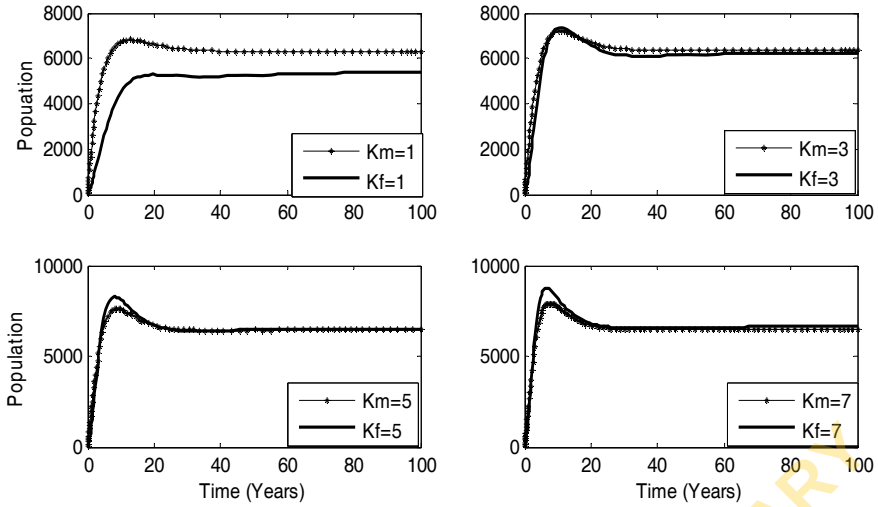

Figure 9. Variation of infected men and women due to variation in the number of sexual partners per year (solid lines for infection among female).

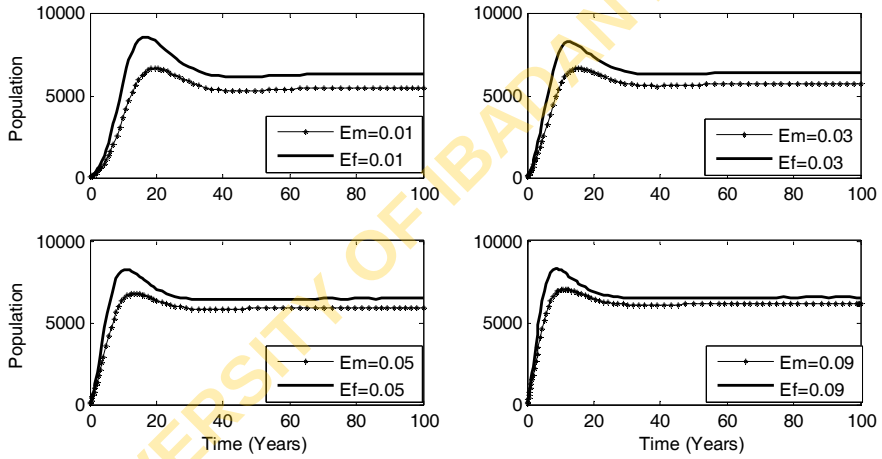

Figure 10. Variation of infected men and women due to variation in the rate of infection from none major routes (solid lines for infection among female).

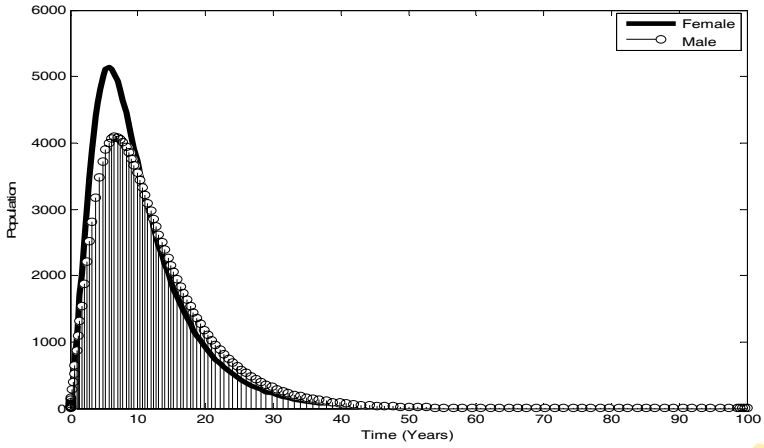

Figure 11. Infection among men and women in a closed community.

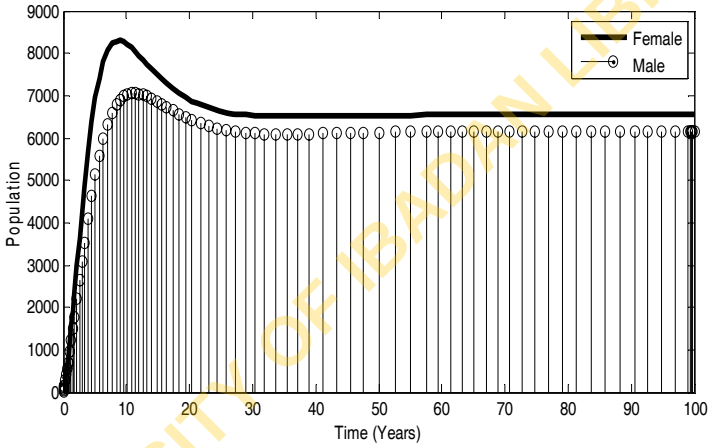

Figure 12. Infection among men and women in a non-closed community.

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