

Research Article

HIV/AIDS Model with Early Detection and Treatment

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A classical epidemiological framework is used to qualitatively assess the impact of early detection and treatment on the dynamics of HIV/AIDS. Within this theoretical framework, two classes of infected populations: those infected but unaware of their serological status and those who are aware of their disease status, are considered. In this context, we formulate and analyze a deterministic model for the transmission dynamics of HIV/AIDS and assess the potential population-level impact of early detection in curtailing the epidemic. A critical threshold parameter for which case detection will have a positive impact is derived. Model parameters sensitivity analysis indicates that the number of partners is the most sensitive (in increasing the average number of secondary transmission) parameter. However, the case detection coverage is the main drivers in reducing the initial disease transmission. Numerical simulations of the model are provided to support the analytical results. Early detection and treatment alone are insufficient to eliminate the disease, and other control strategies are to be explored.

1. Introduction

HIV/AIDS has killed more than 25 million people globally since its emergence in 1981, making it one of the most destructive epidemics in recorded history. The disease continues to inflict a significant morbidity, mortality, and social-economic and public health burden. For the estimated 33.3 million people living with HIV after nearly 30 years into a very complex epidemic, the gains are real but still fragile, even as the number of annual AIDS-related deaths worldwide has steadily decreased from the peak of 2.1 million in 2004 to an estimated 1.8 million in 2009 [1].

Various preventative and therapeutic measures have been embarked upon, aiming at combating one of the greatest pandemics in modern times [2, 3]. In sub-Saharan Africa, many infected individuals are unaware of their disease status. Recent randomized control trials have found that treating HIV-positive individuals with antiretroviral drugs reduces the risk

of them transmitting the disease to their heterosexual partners by more than 90% [4]. As the current treatment therapy has been proven beyond reasonable doubt to reduce transmission, it is imperative to identify those who are infected and put them on treatment when eligible. It is therefore desirable to encourage voluntary testing that will increase case detection, thereby reducing the number of secondary infections of individuals receiving treatment. There is no explicit mathematical account of the potential population level impact of case detection when treatment is available known to us. Thus, a dynamical system model is formulated in order to assess the trade-off/population-level impact between treatment and early detection of HIV positive. The results are sensitive to parameter values, and for this reason, a deterministic sensitivity analysis is carried out.

The rest of this work is organized as follows. The basic model formulation and its analysis are provided in Section 2. The extended model incorporating case detection and treatment is described and analyzed in Section 3. The model simulation using heuristic parameter values for the purpose of illustration follows in Section 4.

2. Model Formulation and Analysis

We begin by formulating a deterministic sex-structured basic HIV/AIDS model (i.e., without interventions). Individuals are identified as male and female only in connection with features peculiar to their sex. The male to female infectivity rate is greater than the female to male [5]. We also assume that the mixing between individuals is homogeneous; individuals may become HIV-infected only through sexual contacts with HIV infected individuals. Those in the final disease stage are considered too ill to remain sexually active. We ignore important HIV transmission path such as intravenous drug injections, vertical transmission, breast feeding, blood transfusion, and needle sharing. It is also assumed that there is no recruitment of HIV positive. The total heterosexual population is divided into male and female subpopulations with the following epidemiological subgroups (the classification is based on individuals disease status): susceptible male and female (S_m, S_f), infected male and female (I_m, I_f), and symptomatic individuals in the final disease (AIDS) stage (A_m, A_f).

New recruits enter the heterosexually active population at constant rates Λ_m and Λ_f for male and female, respectively (all recruits into the population are assumed susceptible). Male and female susceptible acquire infection at time-dependent rates λ_m and λ_f and become infectious. Infectious individuals exhibit AIDS clinical defining symptoms at rates h and z for male and female, respectively. In the absence of the disease, individuals in the population die of natural death at the rate μ . The disease-induced mortality rate is d for both individuals in the infectious and AIDS classes. A full description of the model variables and parameters used in the model is described in Tables 2 and 3, respectively.

Based on our model description and assumptions, we establish the following equations. We note that the red and dash arrows, respectively, in Figures 1 and 2 are the feedback branches which indicate how the male and female subpopulations are coupled (via the force of infection)

$$\begin{array}{ll}
 \text{Male} & \text{Female} \\
 \frac{dS_m}{dt} = \Lambda_m - (\mu + \lambda_m)S_m, & \frac{dS_f}{dt} = \Lambda_f - (\mu + \lambda_f)S_f, \\
 \frac{dI_m}{dt} = \lambda_m S_m - (\mu + d + h)I_m, & \frac{dI_f}{dt} = \lambda_f S_f - (\mu + d + z)I_f, \\
 \frac{dA_m}{dt} = hI_m - (\mu + d)A_m, & \frac{dA_f}{dt} = zI_f - (\mu + d)A_f.
 \end{array} \tag{2.1}$$

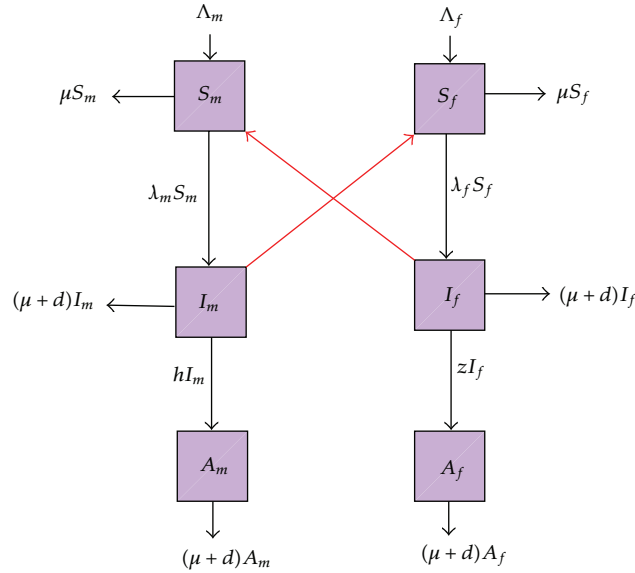


Figure 1: The basic HIV/AIDS model flowchart.

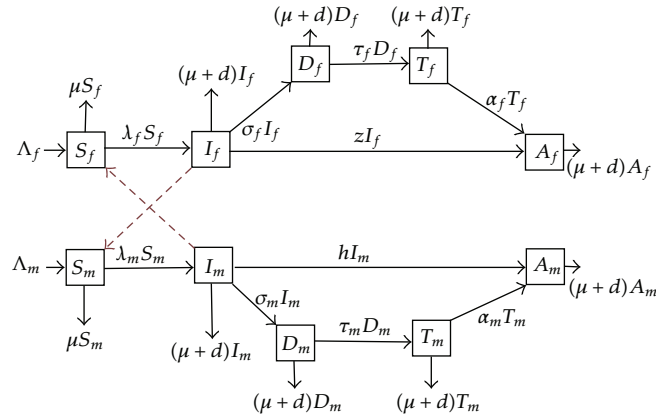


Figure 2: Flow diagram of the HIV/AIDS model with interventions.

The forces of infection for male and female are, respectively, given by

$$\lambda_m = \eta_f \beta_m \frac{I_f}{N_f}, \quad \lambda_f = \eta_m \beta_f \frac{I_m}{N_m}, \quad (2.2)$$

where β_m, β_f and η_m, η_f are, respectively, the probabilities of acquiring HIV and the average number of male and female sexual partners, respectively. The basic model (2.1) with nonnegative initial conditions is epidemiologically meaningful and mathematically well

posed. Thus, system (2.1) is dissipative (i.e., all feasible solutions are uniformly bounded [6, 7]). System (2.1) has a disease-free equilibrium (DFE) given by

$$E_0 = (S_{0m}, I_{0m}, A_{0m}, S_{0f}, I_{0f}, A_{0f}) = \left(\frac{\Lambda_m}{\mu}, 0, 0, \frac{\Lambda_f}{\mu}, 0, 0 \right). \quad (2.3)$$

Using the next-generation operator method [8], the basic model reproduction number R_0 of model (2.1), defined as the number of secondary infections caused by a typical infected individual introduced into the entire susceptible population during his entire period of infectiousness [9], is given by

$$R_0 = \sqrt{\frac{\beta_f \eta_m}{(\mu + d + h)} \frac{\beta_m \eta_f}{(\mu + d + z)}}. \quad (2.4)$$

The expression of R_0 is a geometric mean of the average number of secondary male infections produced by one female, and the average number of secondary female infections produced by one male. From Theorem 2 of van den Driessche and Watmough [8], the following result holds.

Lemma 2.1. *The DFE E_0 of system (2.1) is locally asymptotically stable if $R_0 < 1$, and unstable if $R_0 > 1$.*

If $R_0 < 1$, then on average an infected individual produces less than one new infection over its infectious period, and the epidemic cannot grow. That is, a small influx of infected individuals would not generate large outbreaks if $R_0 < 1$. Conversely, if $R_0 > 1$, then each infected individual produces on average more than one new infection, and the disease can invade the population. However, in order for disease elimination to be independent of the initial sizes of the subpopulations of the model when $R_0 < 1$, global stability of E_0 is required.

Lemma 2.2. *The E_0 is globally asymptotically stable if $R_0 < 1$, and unstable otherwise.*

Proof. The proof is based on a comparison theorem [10]. The rate of change of the variables representing the infected components of the system (2.1) can be written as

$$\begin{pmatrix} \frac{dI_m}{dt} \\ \frac{dA_m}{dt} \\ \frac{dI_f}{dt} \\ \frac{dA_f}{dt} \end{pmatrix} = (F - V) \begin{pmatrix} I_m \\ A_m \\ I_f \\ A_f \end{pmatrix} - \begin{pmatrix} \lambda_m S_m \\ 0 \\ \lambda_f S_f \\ 0 \end{pmatrix}, \quad (2.5)$$

where the matrices F and V are given, respectively, by

$$F = \begin{pmatrix} 0 & 0 & \frac{\beta_m \eta_f \Lambda_m}{\mu N_f} & 0 \\ 0 & 0 & 0 & 0 \\ \frac{\beta_f \eta_m \Lambda_f}{\mu N_m} & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix}, \quad V = \begin{pmatrix} \mu + d + h & 0 & 0 & 0 \\ -h & \mu + d & 0 & 0 \\ 0 & 0 & \mu + d + z & 0 \\ 0 & 0 & -z & \mu + d \end{pmatrix}. \quad (2.6)$$

Thus,

$$\begin{pmatrix} \frac{dI_m}{dt} \\ \frac{dA_m}{dt} \\ \frac{dI_f}{dt} \\ \frac{dA_f}{dt} \end{pmatrix} \leq (F - V) \begin{pmatrix} I_m \\ A_m \\ I_f \\ A_f \end{pmatrix}. \quad (2.7)$$

Using the fact that the eigenvalues of the matrix $F - V$ all have negative real parts, it follows that the linearized differential inequality above is stable whenever $R_0 < 1$. Consequently, $(I_m, A_m, I_f, A_f) \rightarrow (0, 0, 0, 0)$ as $t \rightarrow \infty$. By a comparison Theorem [10], $(I_m, A_m, I_f, A_f) \rightarrow (0, 0, 0, 0)$ as $t \rightarrow \infty$. Thus, $(S_m, I_m, A_m, S_f, I_f, A_f) \rightarrow (\Lambda_m/\mu, 0, 0, \Lambda_f/\mu, 0, 0)$ as $t \rightarrow \infty$ for $R_0 < 1$, and hence, the DFE is globally asymptotically stable if $R_0 < 1$. \square

The above result indicates that HIV could be eliminated from the community if the threshold quantity R_0 can be brought to (and maintained at) a value less than unity. The endemic equilibrium (EE) of (2.1) is given by

$$E^* = (S_m^*, I_m^*, A_m^*, S_f^*, I_f^*, A_f^*), \quad (2.8)$$

where

$$\begin{aligned} S_m^* &= \frac{\Lambda_m}{(\lambda_m^* + \mu)}, & I_m^* &= \frac{\Lambda_m \lambda_m^*}{(\lambda_m^* + \mu)(\mu + d + h)}, & A_m^* &= \frac{h \Lambda_m \lambda_m^*}{(\lambda_m^* + \mu)(\mu + d + h)(\mu + d)}, \\ S_f^* &= \frac{\Lambda_f}{(\lambda_f^* + \mu)}, & I_f^* &= \frac{\Lambda_f \lambda_f^*}{(\lambda_f^* + \mu)(\mu + d + z)}, & A_f^* &= \frac{z \Lambda_f \lambda_f^*}{(\lambda_f^* + \mu)(\mu + d + z)(\mu + d)}, \end{aligned} \quad (2.9)$$

where $\lambda_m^* = \eta_f \beta_m (I_f^*/N_f^*)$ and $\lambda_f^* = \eta_m \beta_f (I_m^*/N_m^*)$. Solving for λ_m^* using the values of I_m^* , I_f^* in (2.9) and the value of λ_f^* , after some lengthy algebraic manipulations, the endemic equilibrium of HIV/AIDS basic model satisfies the following linear equation:

$$A\lambda_m^* - B = 0, \quad (2.10)$$

where

$$\begin{aligned} A &= \mu N_m N_f (\mu + d + h) (\mu + d + z) + N_f \eta_m \beta_f \Lambda_m (\mu + d + z), \\ B &= \mu^2 N_m N_f (\mu + d + h) (\mu + d + z) \left[1 - \frac{\eta_f \beta_m \Lambda_f \eta_m \beta_f \Lambda_m}{\mu^2 N_m N_f (\mu + d + h) (\mu + d + z)} \right] \\ &= \mu^2 N_m N_f (\mu + d + h) (\mu + d + z) [1 - R_0^2], \end{aligned} \quad (2.11)$$

$A > 0$ while $B < 0$ provided $R_0 > 1$, and consequently, the linear system $A\lambda_m^* - B = 0$ has a unique positive solution $\lambda_m^* = B/A$, whenever $R_0 > 1$. The components of the endemic equilibrium E^* are then determined by substituting $\lambda_m^* = B/A$ into (2.9). Noting that $R_0 < 1$ implies $B > 0$; thus, for $R_0 < 1$, the force of infection λ_m^* at steady state is negative (and biologically meaningless). Hence, the model has no endemic equilibrium in this case. Thus, we have established the following result.

Lemma 2.3. *The HIV/AIDS model (2.1) has a unique positive EE E^* whenever $R_0 > 1$ and none otherwise.*

The uniqueness of E^* and the global stability of the DFE imply that the model does not exhibit the phenomenon of backward or subcritical bifurcation where a locally stable EE coexists with a stable DFE when the reproduction number is less than unity. Because the model parameters are taken from different sources, a deterministic sensitivity analysis is carried out using the approach in [11]. Sensitivity indices (of the reproduction number) which measure initial disease transmission allow us to estimate the relative change in a state variable when a parameter changes. The sensitivity indices of R_0 to the parameters for the HIV/AIDS model are given in Table 1. The negative parameters simply means that an increase in that parameter leads to a decrease in the reproductive number. For instance, a 10% increase of the number of sexual partners will lead to a 5% increase of the value of R_0 (initial disease transmission threshold).

3. Analysis of the Model with Interventions

The basic model is extended to include screening (detected classes, D_m, D_f) and treatment classes (T_m, T_f). It is assumed that the number of contacts made by susceptible individuals under treatment is less than or equal to the number of contacts made with an untreated infective due to behavioral change. This is captured via the parameters r_m and r_f which are both less than or equal to unity ($0 \leq r_m, r_f \leq 1$). Also, treatment reduces infectivity, accounted herein by the parameters $p_m, p_f < 1$. The model compartments and flow are depicted in Figure 2, while the additional variables and parameters for the extended model are described in Tables 2 and 3, respectively.

Table 1: Numerical values of sensitivity indices of R_0 .

Parameter symbol	Sensitivity index
d	-0.6935
$\Lambda_m, \Lambda_f, \eta_m, \eta_f, \beta_m, \beta_f$	+0.5000
h	-0.1302
z	-0.1302

Table 2: Model variables and their description.

Symbol	Description
$S_m(t)$	Number of susceptible males at time t
$I_m(t)$	Number of infectious males at time t
$A_m(t)$	Number of fully symptomatic AIDS males at time t
$S_f(t)$	Number of susceptible females at time t
$I_f(t)$	Number of infectious females at time t
$A_f(t)$	Number of fully symptomatic AIDS females at time t
$D_m(t)$	The number of detected (screened) males at time t
$T_m(t)$	The number of treated males at time t
$D_f(t)$	The number of detected (screened) females at time t
$T_f(t)$	The number of treated females at time t

With the above assumptions and terminology, the model is given by the following system of nonlinear equations:

$$\begin{array}{ll}
 \text{Male} & \text{Female} \\
 \frac{dS_m}{dt} = \Lambda_m - (\lambda_m + \mu)S_m, & \frac{dS_f}{dt} = \Lambda_f - (\lambda_f + \mu)S_f, \\
 \frac{dI_m}{dt} = \lambda_m S_m - (\mu + d + h + \sigma_m)I_m, & \frac{dI_f}{dt} = \lambda_f S_f - (\mu + d + z + \sigma_f)I_f, \\
 \frac{dD_m}{dt} = \sigma_m I_m - (\mu + d + \tau_m)D_m, & \frac{dD_f}{dt} = \sigma_f I_f - (\mu + d + \tau_f)D_f, \\
 \frac{dT_m}{dt} = \tau_m D_m - (\mu + d + \alpha_m)T_m, & \frac{dT_f}{dt} = \tau_f D_f - (\mu + d + \alpha_f)T_f, \\
 \frac{dA_m}{dt} = \alpha_m T_m + h I_m - (\mu + d)A_m, & \frac{dA_f}{dt} = \alpha_f T_f + z I_f - (\mu + d)A_f.
 \end{array} \tag{3.1}$$

The force of infections of male and female is, respectively, given by

$$\lambda_m = \eta_f \beta_m \frac{I_f + r_f D_f + p_f T_f}{N_f}, \quad \lambda_f = \eta_m \beta_f \frac{I_m + r_m D_m + p_m T_m}{N_m}. \tag{3.2}$$

The DFE of model system (3.1) denoted by E_{00} is given by

$$E_{00} = \left(\frac{\Lambda_m}{\mu}, 0, 0, 0, 0, 0, \frac{\Lambda_f}{\mu}, 0, 0, 0, 0, 0 \right). \tag{3.3}$$

Table 3: Parameter definitions and their values.

Parameter	Definition	Value (yr) ⁻¹	Source
μ	Natural mortality rate	0.0222	[1]
d	Disease-induced death rate	0.333	[12]
β_m	Male effective contact rate	0.025	Assumed
β_f	Female effective contact rate	0.015	Assumed
η_m	Average number of male partners per female	3	BMC
η_f	Average number of female partners per male	5	BMC
τ_m	Treatment rate of detected male	$0 < \tau_m < 1$	Assumed
τ_f	Treatment rate of detected female	$0 < \tau_f < 1$	Assumed
h	Progression rate of infected females to AIDS	0.125	[13]
z	Progression rate of infected males to AIDS	0.125	[13]
α_m	Progression rate of males under treatment to AIDS	0.004	Assumed
α_f	Progression rate of females under treatment to AIDS	0.004	Assumed
p_m	Reduction in transmission of HIV from treated males	0.02	BMC
p_f	Reduction in transmission of HIV from treated females	0.02	BMC
r_m	Reduction in transmission of HIV from detected male	0.004	Assumed
r_f	Reduction in transmission of HIV from detected female	0.0025	Assumed
σ_m	Rate at which infected males are detected	0.512	BMC
σ_f	Rate at which infected females are detected	0.675	BMC
Λ_m	Net flow rate into the susceptible male class	100,000	STH
Λ_f	Net flow rate into the susceptible female class	100,000	STH

Using the next-generation matrix operator [8], the reproduction number of (3.1) is

$$R_T = \sqrt{R_{mT} R_{fT}}, \quad (3.4)$$

where

$$R_{mT} = \frac{\beta_f \eta_m}{\mu + d + h + \sigma_m} \left(1 + \frac{r_m \sigma_m}{\mu + d + \tau_m} + \frac{p_m \tau_m \sigma_m}{(\mu + d + \tau_m)(\mu + d + \alpha_m)} \right),$$

$$R_{fT} = \frac{\beta_m \eta_f}{\mu + d + z + \sigma_f} \left(1 + \frac{r_f \sigma_f}{\mu + d + \tau_f} + \frac{p_f \tau_f \sigma_f}{(\mu + d + \tau_f)(\mu + d + \alpha_f)} \right). \quad (3.5)$$

Thus, using Theorem 2 of van den Driessche and Watmough [8], the following result holds.

Lemma 3.1. *The DFE E_{00} of model system (3.1) is locally asymptotically stable if $R_T < 1$, and unstable if $R_T > 1$.*

R_{mT} and R_{fT} are the reproduction numbers for males and females, respectively (i.e., R_{mT} represents the number of females infected by a single male during his entire period of infectiousness in a population where treatment is available). If the interventions are dropped,

that is, $\sigma_m = 0$, $\sigma_f = 0$, and $\tau_m = 0$, $\tau_f = 0$, then the effective reproduction number R_T reduces to the basic reproduction number R_0 . The endemic equilibrium of (3.1) denoted by E^{**} is

$$E^{**} = (S_m^{**}, I_m^{**}, D_m^{**}, T_m^{**}, A_m^{**}, S_f^{**}, I_f^{**}, D_f^{**}, T_f^{**}, A_f^{**}), \quad (3.6)$$

$$\begin{aligned} S_m^{**} &= \frac{\Lambda_m}{\mu + AI_f^{**}}, & I_m^{**} &= \frac{A\Lambda_f[AB\Lambda_m\Lambda_f - \mu^2(\mu + d + z + \sigma_f)(\mu + d + h + \sigma_m)]}{(\mu + d + h + \sigma_m)(\mu AB\Lambda_m + A^2B\Lambda_m\Lambda_f)}, \\ S_f^{**} &= \frac{\Lambda_f}{\mu + BI_m^{**}}, & I_f^{**} &= \frac{AB\Lambda_m\Lambda_f - \mu^2(\mu + d + z + \sigma_f)(\mu + d + h + \sigma_m)}{\mu(\mu + d + z + \sigma_f)(\mu + d + h + \sigma_m)A + AB\Lambda_m}, \\ D_m^{**} &= \frac{\sigma_m}{\mu + d + \tau_m} I_m^{**}, & D_f^{**} &= \frac{\sigma_f}{\mu + d + \tau_f} I_f^{**}, \\ T_m^{**} &= \frac{\sigma_m \tau_m}{(\mu + d + \tau_m)(\mu + d + \alpha_m)} I_m^{**}, & A_m^{**} &= \frac{\alpha_m \sigma_m \tau_m + h(\mu + d + \tau_m)(\mu + d + \alpha_m)}{(\mu + d)(\mu + d + \tau_m)(\mu + d + \alpha_m)} I_m^{**}, \\ T_f^{**} &= \frac{\sigma_f \tau_f}{(\mu + d + \tau_f)(\mu + d + \alpha_f)} I_f^{**}, & A_f^{**} &= \frac{\alpha_f \sigma_f \tau_f + z(\mu + d + \tau_f)(\mu + d + \alpha_f)}{(\mu + d)(\mu + d + \tau_f)(\mu + d + \alpha_f)} I_f^{**}, \end{aligned} \quad (3.7)$$

where

$$\begin{aligned} A &= \frac{\beta_m \eta_f}{N_f} \left(1 + \frac{r_f \sigma_f}{\mu + d + \tau_f} + \frac{p_f \tau_f \sigma_f}{(\mu + d + \tau_f)(\mu + d + \alpha_f)} \right), \\ B &= \frac{\beta_f \eta_m}{N_m} \left(1 + \frac{r_m \sigma_m}{\mu + d + \tau_m} + \frac{p_m \tau_m \sigma_m}{(\mu + d + \tau_m)(\mu + d + \alpha_m)} \right). \end{aligned} \quad (3.8)$$

The endemic equilibrium exists provided $I_m^{**} > 0$ and $I_f^{**} > 0$. Consider I_f^{**} given in (3.7), then

$$\begin{aligned} I_f^{**} &= \frac{AB\Lambda_m\Lambda_f - \mu^2(\mu + d + z + \sigma_f)(\mu + d + h + \sigma_m)}{\mu(\mu + d + z + \sigma_f)(\mu + d + h + \sigma_m)A + AB\Lambda_m} \\ &= \frac{AB\Lambda_m\Lambda_f - \mu^2(\mu + d + z + \sigma_f)(\mu + d + h + \sigma_m)}{\mu(\mu + d + z + \sigma_f)(\mu + d + h + \sigma_m)A + AB\Lambda_m} \\ &= \left[\frac{AB\Lambda_m\Lambda_f}{\mu^2(\mu + d + z + \sigma_f)(\mu + d + h + \sigma_m)} - 1 \right]. \end{aligned} \quad (3.9)$$

Substituting A and B in (3.9), after some rearrangement, we obtain

$$I_f^{**} = R_T^2 - 1. \quad (3.10)$$

It is therefore evident that $I_f^{**} > 0$ provided $R_T > 1$. A similar expression can be derived for I_m^{**} . Thus, we have established the following result.

Lemma 3.2. *The model system (3.1) has a unique positive EE whenever $R_T > 1$ and none otherwise.*

Table 4: Sensitivity indices of R_T .

Parameter	Sensitivity index
$\Lambda_m, \Lambda_f, \eta_m, \eta_f, \beta_m, \beta_f$	+0.5000
σ_f	-0.3206
σ_m	-0.2808
h	-0.0194
z	-0.0072
r_f	+0.0023
r_m	+0.0015
τ_m	-0.0003
τ_f	-0.0002
p_m	+0.0002
p_f	+0.0001

We analytically investigate the impact of case detection on HIV/AIDS dynamics. By partially differentiating R_{mT} with respect to the case detection rate σ_m , we obtain

$$\frac{\partial R_{mT}}{\partial \sigma_m} = \frac{\beta_f \eta_m}{(\mu + d + h + \sigma_m)^2} (\Delta - 1), \quad (3.11)$$

where $\Delta = ((\mu + d + h) / (\mu + d + h + \tau_m))(r_m + p_m \tau_m / (\mu + d + \alpha_m))$. For $\Delta < 1$, (3.11) is negative and consequently, early detection will always have a positive impact on the dynamics of HIV/AIDS. If $\Delta = 1$, then case detection has no impact (this case will only arise if treatment of those eligible does not follow). From epidemiological and demographical standpoint, the threshold parameter $\Delta \leq 1$ is to be expected.

The sensitivity indices of R_T are given in Table 4. Next, we numerically investigate the impact of the number of partners, case detection, and treatment on the disease dynamics. Tanzania started HIV/AIDS care and treatment in October 2004, and the target for the first year was to cover 44,000 patients. About 96 care and treatment providing facilities were selected to initiate the services, which included four referral hospitals; Muhimbili, Kilimanjaro Christian Medical Centre (KCMC), Bugando Medical Centre (BMC), and Mbeya Medical Centre. Some of the parameter values (see Table 3) are provided courtesy of the regional medical officer estimated based on data from the Bugando Medical Centre (BMC) and the Sekou Toure Hospital (STH) both located in Mwanza City in northern Tanzania. Others are taken from the literature, and the remaining ones are assumed within realistic range for the purpose of illustration.

Multiple partnerships increase the risk factor of acquiring HIV. When the number of partners is small over a long time period, the rate of infection is minimal. In this case, the disease tends to die down (Figure 3(a)). The disease will persist when multiple and concurrent partnerships are frequent in the community, in which case infections are on the increase over time (Figure 3(b)).

Since it is assumed that all detected HIV-positive individuals are treated if eligible, the shapes of the time trends of detected (Figure 4(a)) and treated individuals (Figure 4(b)) are similar. The slight difference is due to the rate of developing full blown AIDS from the treated class.

Figure 5 depicts the effect of increasing early detection and treatment on the reproduction numbers R_0 and R_T .

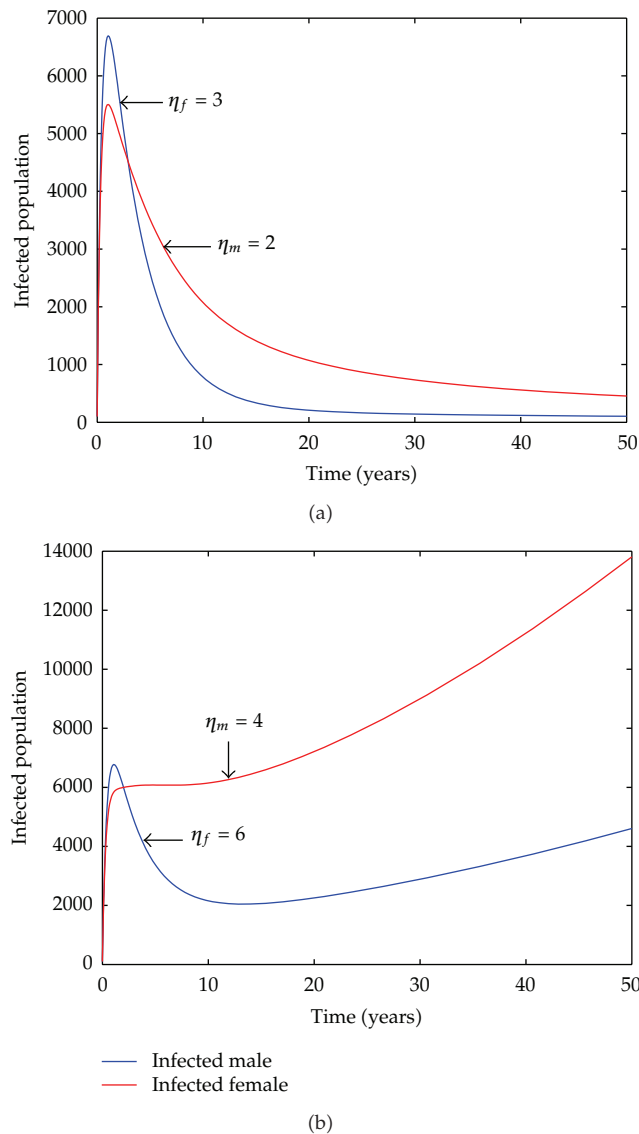


Figure 3: Effect of the number of partners on the dynamics of HIV-positive (a) low sexual activity: $\eta_m = 2$ and $\eta_f = 3$, (b) high sexual activity: $\eta_m = 4$ and $\eta_f = 6$.

4. Conclusion

The dynamic and determinants of the HIV epidemics are multiple and are shaped by the sexual patterns which are related to social, cultural, and economic factors: for example, promiscuity, low and inconsistent condom use, intergenerational sex, concurrent sexual partners, and various opportunistic infections. Screening is a barometer for achieving success in the fight against the epidemic. It is against this background that a simple deterministic HIV/AIDS model which accounts for case detection and antiretroviral therapy was formulated and analyzed. Conditions for the global stability which rules out any possibility for

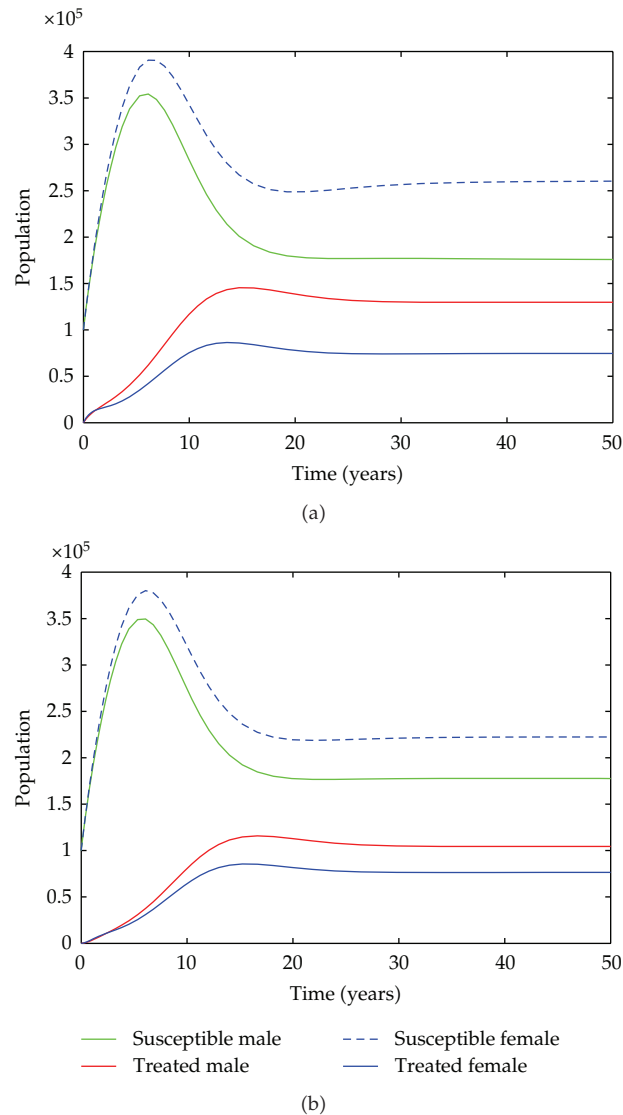


Figure 4: Time series of susceptible, detected, and treated individuals.

the model to exhibit the phenomenon of backward bifurcation were provided. Therefore, the classical requirement of the reproduction number to be less than unity might be sufficient for disease elimination. However, HIV/AIDS is inherently a multifaceted disease with poverty-drug use-behavioral/attitudinal change and gender inequality are some of the social factors that need to be addressed. Sensitivity results point to the case detection rate as a driving factor in stemming the tide of the epidemic. Thus, increasing voluntary and or mass screening will always have a positive impact on HIV/AIDS control in reducing the disease burden.

Numerical simulations clearly show that early detection and treatment alone are insufficient to eliminate the disease (Figures 4 and 5). Other control strategies such as condom and microbicides used are to be explored. The study is not exhaustive and can be extended in various ways by incorporating a potential (imperfect) vaccine, withdrawal from sexual

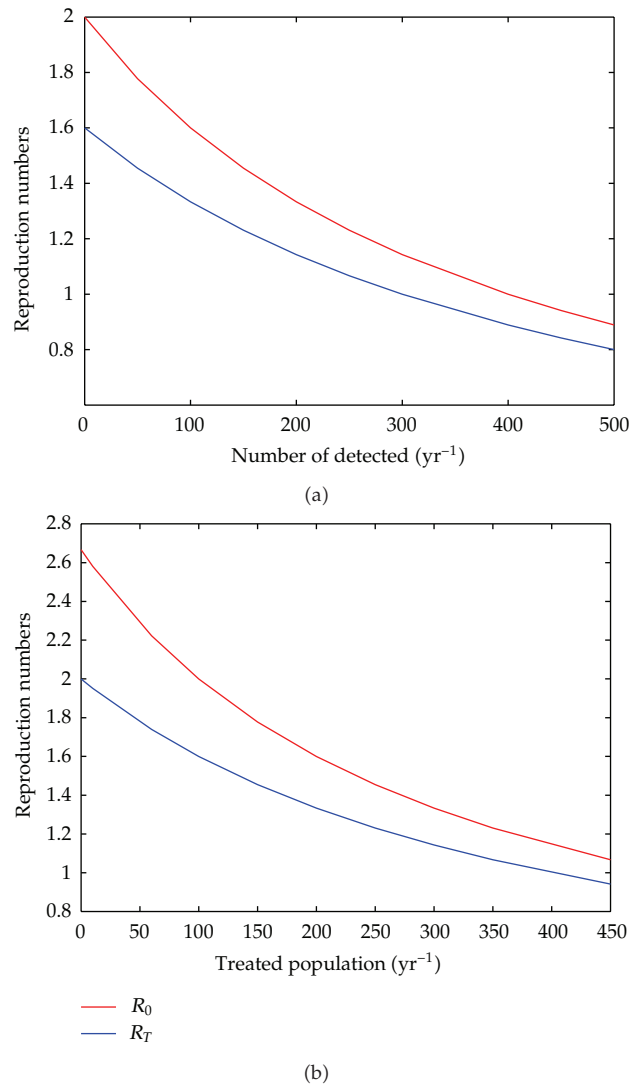


Figure 5: Graphical representation of R_0 as detection and treatment rates are varied.

activity of a fraction of individuals in the AIDS defining stage, and development of drug resistance and superinfection (with different virus strains).

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