Effects of public health educational campaigns and the role of sex workers on the spread of HIV/AIDS among heterosexuals

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Abstract

This paper presents a sex-structured model for heterosexual transmission of HIV/AIDS in which the population is divided into three subgroups: susceptibles, infectives and AIDS cases. The subgroups are further divided into two classes, consisting of individuals involved in high-risk sexual activities and individuals involved in low-risk sexual activities. The model considers the movement of individuals from high to low sexual activity groups as a result of public health educational campaigns. Thus, in this case public health educational campaigns are resulting in the split of the population into risk groups. The equilibrium and epidemic threshold, which is known as the basic reproductive number ($R_0$), are obtained, and stability (local and global) of the disease-free equilibrium is investigated. The model is extended to incorporate sex workers, and their role in the spread of HIV/AIDS in settings with heterosexual transmission is explored. Comprehensive analytic and numerical techniques are employed in assessing the possible community benefits of public health educational campaigns in controlling HIV/AIDS. From the study, we conclude that the presence of sex workers enlarges the epidemic threshold $R_0$, thus fuels the epidemic among the heterosexuals, and that public health educational campaigns among the high-risk heterosexual population reduces $R_0$, thus can help slow or eradicate the epidemic.

Keywords: HIV/AIDS model; Public health educational campaigns; Risk groups; Sex workers; Basic reproductive number; Stability

1. Introduction

HIV/AIDS has killed more than 25 million people since it was first recognized in 1981, making it one of the most destructive epidemics in recorded history (UNAIDS/WHO, 2005). The epidemic has remained one of the leading causes of death in the world and has been destructive in Africa with Sub-Saharan Africa remaining the epidemiological locus of the epidemic. In Sub-Saharan Africa, heterosexuality including prostitution has remained the principal mode of transmission since the epidemic became visible (Mufune, 2004; UNAIDS, 2004). Overtime and due to the fact that women are inordinately affected, vertical transmissions from mother to child during childbirth are of increasing importance in transmission and this is in contrast to other areas of the globe where the principal mode of transmission includes men who have sex with other men (homosexuals) and intravenous drug users (Mufune, 2004). Sub-Saharan Africa remains hardest-hit and is currently home to 25.8 million (23.8–28.9 million) people living with HIV, almost one million more than in 2003 (UNAIDS/WHO, 2003). Two-thirds of all people living with HIV are in Sub-Saharan Africa, as are 77% of all women with HIV. An estimated 2.4 million (2.1–2.7 million) people died of HIV-related illnesses in this region in 2005, while a further 3.2 million (2.8–3.9 million) became infected with HIV (UNAIDS/WHO, 2003). In some Sub-Saharan Africa countries, declines in HIV prevalence related to changes in behaviour and prevention programmes have been observed. Among the notable new trends are the recent declines in national HIV prevalence in Kenya and Zimbabwe, urban areas of Burkina Faso and similarly in Haiti, alongside indications of significant behavioural change including increased condom

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use, fewer partners and delayed sexual debut (UNAIDS, 2006). However, the number of people living with HIV has continued to rise, due to population growth and, more recently, the life prolonging effects of antiretroviral therapy (UNAIDS, 2006).

In the continuing absence of a cure for HIV/AIDS, the use of antiretroviral drugs (ARVs) has remained the only feasible option for infected individuals. Current combinations of ARVs increase the survival time of HIV-infected individuals, but do not lead to viral eradication within infected individuals hence do not cure and over time this causes the pool of potential transmitters of infection to grow; thus, the two factors decreased infectivity and increased duration of infectiousness have opposing effects on transmission (Blower et al., 2005). Studies (Anderson et al., 1991; Velasco-Hernandez and Hsieh, 1994; Hsieh and Velasco-Hernandez, 1995; Hsu Schmitz, 2000, 2002; Baggaley et al., 2006; Mukandavire and Garira, 2006) have also shown that, treatment which prolongs the incubation period but do not reduce HIV/AIDS transmission rate may even enlarge the epidemic. Further, Velasco-Hernandez and Hsieh (1994) showed that treatment can benefit the community if accompanied by sexual behaviour changes. On the issue of vaccines, there is no approved vaccine for HIV/AIDS despite the efforts on vaccine research and it is unlikely that a highly effective vaccine will be available soon; thus, vaccination as a control strategy is not yet available for HIV/AIDS. Mclean and Blower (1993, 1995), Blower and Mclean (1994), Blower et al. (2002, 2003a, b) and Smith and Blower (2004) studied theoretical models of partially effective HIV vaccines and the potential changes in risky behaviour associated with a vaccination campaign and found that the benefits offered by an only partly effective vaccination program may be offset by rises in potentially infectious contacts unless an education campaign accompanies it. Del Valle et al. (2004) studied the effects of education in a set up with vaccination and treatment on HIV transmission in homosexuals with heterogeneity following the concepts of Mclean and Blower (1993, 1995) and Blower and Mclean (1994) that partly effective vaccines should be accompanied by educational campaigns. The joint effects of vaccines and widespread treatment with antiretroviral therapy (ART) have also been theoretically examined by Blower et al. (2003a, 2003b) and Gray et al. (2003). They concluded that widespread ART that reduces infectiousness combined with low efficacy vaccine could reduce infections to very low levels as long as behavioural reversals do not overwhelm the reductions in the risk of transmission per sexual act.

In this paper we formulate and analyse a new sex-structured HIV/AIDS model in which the population is divided into three subgroups: susceptibles, infectives and AIDS cases. The model is formulated using integro-differential equations, which are shown to be equivalent to delay differential equations with a time delay due to incubation period. Any deterministic model without a fixed delay will permit some instantaneous progression to AIDS because of the structure of ordinary differential equations, but the average progression time in these models can still be made to coincide with the statistical average time (see Mukandavire and Garira, 2006, 2007 for a similar approach). The subgroups in the population are divided into two classes, consisting of individuals involved in high-risk sexual activities and individuals involved in low-risk sexual activities. Population numbers in each class denoted as functions of time by \( S_i(t), I_i(t) \) and \( A_i(t) \) where \( i = f, m \) denote female and male populations, \( l = 1, 2 \) denote high-risk and low-risk sexual activities, respectively. HIV/AIDS models with risk groups due to sexual activities have been studied (Hethcote and Van Ark, 1980; Roberts and Dangerfield, 1990; Griffiths et al., 2000a, 2000b). We differ from these studies in that; we consider movement from high to low sexual activity groups to be a result of public health educational campaigns, thus in this case public health educational campaigns are resulting in the split of the population into risk groups. We define public health education as the counseling of individuals to have fewer sexual partners, abstain, and/or otherwise reduce risky behaviour (see Del Valle et al., 2004). Further, we extend our HIV/AIDS model to explore the role of sex workers (which is one of the oldest profession in the world, New Internationalist, 1994) in the spread of HIV/AIDS in settings with heterosexual transmission and investigating the community gains if female sex workers are educated. We define a sex worker to be an individual who sells sexual intercourse for money. Sex workers have been found to be the major source of infection in Africa due to unemployment, poverty and underdevelopment (Caldwell and Quiggin, 1989; Kribs-Zaleta et al., 2005). To the best of our knowledge, no work has been done in modelling the effect of public health educational campaigns and exploring the role of female sex workers in the context of sex-structures incorporating discrete time delays due to the long incubation period of the disease in heterosexual settings.

The paper is organised as follows: in the next section we present the model formulation and analysis (including stability and reproductive numbers), in Section 3 we extend the model to explore the role of sex workers in the spread of HIV/AIDS in settings with heterosexual transmission and overall community gains if female sex workers are educated. In Section 4 we have the summary and concluding remarks.

2. Model description

The model classifies the heterosexually active community into the following categories: high-risk susceptible (\( S_1 \)), high-risk infective (\( I_1 \)), high-risk AIDS cases (\( A_1 \)), low-risk susceptibles \( S_2 \), low-risk infectives \( I_2 \) and low-risk AIDS cases (\( A_2 \)) where \( i = f, m \) denote female and male, respectively. Individuals are referred to as male or female only in connection with features peculiar to their sex. In particular, the male to female infectivity rate is greater than that for female to male
and some HIV/AIDS intervention strategies such as condom use, circumcision and vaginal microbicides are sex dependent. Further, there are gender-based issues associated with HIV/AIDS transmission (Matlin and Spence, 2000). As transmission is mainly heterosexual, each of the categories is divided into high-risk and low-risk: those in the high-risk group represent males and females who are promiscuous and engage in high-risk sexual activity frequently with a number of different sexual partners, whereas low-risk represents those who have a steady sexual relationship. The model assumes that at any moment in time, new recruits enter the heterosexually active population at a rate $\Lambda$. A proportion $\rho$ of these individuals are assumed to be high-risk female susceptibles (categorised in the $S_f$ class) and the complementary proportion $(1 - \rho)$, are high-risk male susceptibles and move to the high-risk male susceptible class ($S_m$). Individuals in category $S_h$ acquire infection at a time dependent rate $i_h$. Category $S_{hi}$ individuals are educated and transfer to low-risk susceptible class $S_l$ at a constant rate $o_h$. Upon becoming infected with HIV, a proportion $\pi$, of not educated individuals enter class $I_h$ of high-risk infectives with the complementary proportions of educated individuals entering class $I_l$ of low-risk infectives. We assume no movement of the high-risk infectives to low-risk infectives during the course of infection for simplicity. The natural death rate is assumed to be proportional to the number in each class, $\mu > 0$. AIDS cases have an additional disease-induced mortality rate, $\nu$. The model assumes a constant emigration rate $\beta > 0$ of individuals to other countries except for the AIDS cases ($A_t$). This assumption makes the model more appropriate to developing countries where a significant proportion of the population emigrates to developed countries for better educational facilities and in search of employment. The model assumes a constant incubation period ($\tau > 0$) for susceptible individuals in class $S_h$ from the time of being infected to the development of AIDS symptoms. The probability that an infected individual remains in the incubation period time $t$ units before developing AIDS is given by a step function with value 1 for $0 \leq t < \tau$ and value zero for $t \geq \tau$. The probability that an infected individual in the incubation period time $t$ units has survived to develop AIDS is $k = e^{-(\mu+\nu)t}$. High-risk infected individuals $I_h$ progress to AIDS stage at a rate $\pi_k k_m S_h (t - \tau)$ with proportion $a_h$ entering high-risk AIDS cases and the complementary proportion entering the low-risk AIDS cases when diagnosed as having AIDS. Low-risk infected individuals $I_l$ progress to AIDS stage at a rate $(1 - \pi_k) k_m S_l (t - \tau)$, with proportions $b_i$ entering high-risk AIDS cases when diagnosed as having AIDS, with the complementary proportion entering the low-risk AIDS cases. The model assumes individuals change of behaviour on the diagnosis of AIDS. Individuals in $A_h$ move to the low-risk AIDS classes $A_l$ at constant rate $\tau_i$ due to public health educational campaigns. In this case, $i_i$ is the average per capita risk of infection of individuals infected $t$ time units. The infection rate $\lambda^n$ for $n = 0, \tau$ depends on the probability of transmission per partnership ($\beta_i$), the rate at which an individual acquires new sexual partners per unit time ($c_i$) and the proportion of infected individuals in each category ($I_h$ and $A_h$). The probability that an infected female will infect her male sexual partner is $\beta_i$ and the probability that an infected male infects his female sexual partner is $\beta_m$. The probability of HIV transmission from a person in category $I_h$ and $A_h$ to a susceptible in category $S_h$ is $\beta_i$. In reality, the risk of infectiousness of individuals in category $I_h$ and $A_h$ is different (see Elobasha and Gumel, 2006, for example). Assuming homogeneous mixing we have

$$i_h^n = \frac{\beta_i c_i (I_h(t - n) + A_h(t - n))}{N_h(t - n)},$$

$$N_h(t - n) = S_h(t - n) + I_h(t - n) + A_h(t - n),$$

$$n = 0, \tau, \ i, j = f, m \ and \ i \neq j,$$

(1)

where $N_h(t - n)$ for $n = 0, \tau$ is the total sexually active population. The model structure is shown in Fig. 1. The model equations are as follows:

$$S_{f1}(t) = \rho \Lambda - i_m^0 S_{f1}(t) - (\omega_f + \mu + \beta) S_{f1}(t),$$

$$S_{f2}(t) = \omega_f S_{f1}(t) - (\omega_f + \beta) S_{f2}(t),$$

$$I_{f1}(t) = \pi_f \int_{-\tau}^{0} i_m^0 S_{f1}(u)e^{-(\mu+\beta)(t-u)} du,$$

$$I_{f2}(t) = (1 - \pi_f) \int_{-\tau}^{0} i_m^0 S_{f1}(u)e^{-(\mu+\beta)(t-u)} du,$$

$$A_{f1}(t) = a_f \pi_f k_m^2 S_{f1}(t - \tau) + b_f (1 - \pi_f) k_m^2 S_{f1}(t - \tau) - (\omega_f + \mu + \nu) A_{f1}(t),$$

$$A_{f2}(t) = (1 - a_f) \pi_f k_m^2 S_{f1}(t - \tau) + (1 - b_f) (1 - \pi_f) k_m^2 S_{f1}(t - \tau) + \omega_f A_{f1} - (\mu + \nu) A_{f2}(t),$$

$$S_{m1}(t) = (1 - \rho) \Lambda - i_m^0 S_{m1}(t) - (\omega_m + \mu + \beta) S_{m1}(t),$$

$$S_{m2}(t) = \omega_m S_{m1}(t) - (\omega_m + \beta) S_{m2}(t),$$

$$I_{m1}(t) = \pi_m \int_{-\tau}^{0} i_m^0 S_{m1}(u)e^{-(\mu+\beta)(t-u)} du,$$

$$I_{m2}(t) = (1 - \pi_m) \int_{-\tau}^{0} i_m^0 S_{m1}(u)e^{-(\mu+\beta)(t-u)} du,$$

$$A_{m1}(t) = a_m \pi_m (1 - \rho) k_m^2 S_{m1}(t - \tau) + b_m (1 - \pi_m) k_m^2 S_{m1}(t - \tau) - (\omega_m + \mu + \nu) A_{m1}(t),$$

$$A_{m2}(t) = (1 - a_m) \pi_m (1 - \rho) k_m^2 S_{m1}(t - \tau) + (1 - b_m) (1 - \pi_m) k_m^2 S_{m1}(t - \tau) + \omega_m A_{m1} - (\mu + \nu) A_{m2}(t).$$
where males and females are constrained to obey certain balance laws when the populations form a closed network of contacts (Busenberg, 1991; Doyle et al., 1998; Doyle and Greenhalgh, 1999) that parameters describing sexual contact rates between $i$ and $j$:

$$\lambda_{ij} = \pi_{ij} \lambda_{i} (1-\pi_{ij}) \lambda_{j}$$

The initial condition for model system (2) is given as

$$I_{m1}(t) = (1 - \pi_{m1}) \int_{t-\tau}^{t} \lambda_{ij}^{0} S_{m1}(t-\tau) - (\pi_{m1} \tau + \mu + \nu) A_{m1}(t),$$

$$A_{m2}(t) = (1 - \pi_{m2}) \int_{t-\tau}^{t} \pi_{ij} \lambda_{i} \lambda_{j}^{0} S_{m1}(t-\tau) + (1 - \pi_{m2}) \lambda_{ij}^{0} S_{m1}(t-\tau) + \pi_{m1} A_{m1} - (\pi_{m2} \tau + \mu + \nu) A_{m2}(t).$$

The initial condition for model system (2) is given as

$$\begin{align*}
S_{m1}(\theta) &= \phi_{1}(\theta), \\
S_{m2}(\theta) &= \phi_{4}(\theta), \\
S_{m3}(\theta) &= \phi_{4}(\theta), \\
S_{m4}(\theta) &= \phi_{4}(\theta), \\
S_{m5}(\theta) &= \phi_{4}(\theta), \\
S_{m6}(\theta) &= \phi_{4}(\theta), \\
S_{m7}(\theta) &= \phi_{4}(\theta), \\
S_{m8}(\theta) &= \phi_{4}(\theta), \\
S_{m9}(\theta) &= \phi_{4}(\theta), \\
S_{m10}(\theta) &= \phi_{4}(\theta), \\
S_{m11}(\theta) &= \phi_{4}(\theta), \\
S_{m12}(\theta) &= \phi_{4}(\theta), \\
S_{m13}(\theta) &= \phi_{4}(\theta),
\end{align*}$$

where $\phi = (\phi_{1}, \phi_{2}, \phi_{3}, \ldots, \phi_{12})^{T} \in C$ such that $\phi_{q}(\theta) = \phi_{q}(\theta) \geq 0(\theta \in [0, \pi], q = 1, 2, 3, \ldots, 12)$, $\phi_{q}(\theta) \geq 0(\theta \in [0, \pi], q = 1, 2, 3, \ldots, 12)$, $\phi_{q}(\theta) \geq 0(\theta \in [0, \pi], q = 1, 2, 3, \ldots, 12)$. We define

$$R_{12} = \{(S_{m1}, S_{m2}, I_{m1}, I_{m2}, A_{m1}, A_{m2}) \in R_{12} | S_{m1} > 0, S_{m2} > 0, I_{m1} > 0, I_{m2} > 0, A_{m1} > 0, A_{m2} > 0\}$$

for $i = f, m$. In models for diseases transmitted by heterosexual contact, it has been established (Castillo-Chavez and Busenberg, 1991; Doyle et al., 1998; Doyle and Greenhalgh, 1999) that parameters describing sexual contact rates between males and females are constrained to obey certain balance laws when the populations form a closed network of contacts.
(the group contact constraint) given by

\[ c_m N_{m_1} = \epsilon_f N_{f_1}. \]  

(5)

In the present context, the group contact constraint is actually not satisfied in our setting, simply because \( N_{m_1} \) and \( N_{f_1} \) vary in the course of time. The modelling problem of satisfying the group contact constraint in a dynamic situation deserves careful attention. An alternative would be to follow the approach of Heesterbeek and Metz (1993) and derive a more complicated non-linear incidence term that is consistent by construction. Subsequently it needs to be checked how the results presented here are affected. Equations for \( S_{f_1}'(t), I_{f_1}(t) \) and \( A_{f_1}'(t) \) completely decouple from the equation for \( S_{i_1}'(t), I_{i_1}(t) \) and \( A_{i_2}(t) \) for \( i = f, m \) for model system (2) and reducing the model to a system of delay differential equations becomes

\[
S_{f_1}'(t) = \rho \Lambda - \alpha_{m}^{0} S_{f_1}(t) - (\omega_f + \mu + \beta) S_{f_1}(t),
\]

\[
I_{f_1}'(t) = \pi_f \int_{0}^{t} \alpha_{m}^{0} S_{f_1}(t - \tau) - (\mu + \beta) I_{f_1}(t),
\]

\[
A_{f_1}'(t) = \alpha_f \pi_f k \int_{m}^{0} S_{f_1}(t - \tau) + \beta_f (1 - \pi_f) k \int_{m}^{0} S_{f_1}(t - \tau) - (\omega_f + \mu + \nu) A_{f_1}(t),
\]

\[
S_{m_1}'(t) = (1 - \rho) \Lambda - \alpha_{m}^{0} S_{m_1}(t) - (\omega_m + \mu + \beta) S_{m_1}(t),
\]

\[
I_{m_1}'(t) = \pi_m \alpha_{m}^{0} S_{m_1}(t) - \pi_m k \int_{m}^{0} S_{m_1}(t - \tau) - (\mu + \beta) I_{m_1}(t),
\]

\[
A_{m_1}'(t) = \alpha_m \pi_m k \int_{m}^{0} S_{m_1}(t - \tau) + \beta_m (1 - \pi_m) k \int_{m}^{0} S_{m_1}(t - \tau) - (\omega_m + \mu + \nu) A_{m_1}(t).
\]

(6)

The mathematical properties for model system (6) are considered in the following sections.

2.1. Basic properties

The HIV/AIDS model system (6) describes human population and therefore it is very important to prove that all the state variables are non-negative for all time. We prove that all solutions of system (6) with positive initial data will remain positive for all \( t > 0 \).

**Theorem 1.** Let the initial data be \( S_{f_1}(s) = S_{f_1}(s) > 0, S_{m_1}(s) = S_{m_1}(s) > 0, I_{f_1}(s) = I_{f_1}(s), I_{m_1}(s) = I_{m_1}(s) > 0 \) for all \( s \in [-\infty, 0] \) with \( S_{f_1}(0) > 0, S_{m_1}(0) > 0, I_{f_1}(0) > 0, I_{m_1}(0) > 0, A_{f_1}(0) > 0, A_{m_1}(0) > 0, A_{m_1}(0) > 0 \) and \( A_{m_1}(0) > 0 \). Then solutions \( S_{f_1}(t), S_{m_1}(t), I_{f_1}(t), I_{m_1}(t), A_{f_1}(t) \) and \( A_{m_1}(t) \) of system (6) are positive for all \( t > 0 \).

**Proof.** Let \( T = \sup\{t \geq 0 : S_{f_1} > 0, I_{f_1} > 0, A_{f_1} > 0, S_{m_1} > 0, I_{m_1} > 0, A_{m_1} > 0, A_{m_1} > 0, A_{m_1} > 0 \} \). We have \( T > 0 \), and if \( T < \infty \) then one of \( S_{f_1}, I_{f_1}, A_{f_1}, S_{m_1}, I_{m_1}, A_{m_1} \) must be zero at some time.

For model system (6) we have

\[
\frac{d}{dt} \left[ S_{f_1} \exp \left[ \int_{0}^{t} \alpha_{m}^{0} du + (\omega_f + \mu + \beta) t \right] \right] = \rho \Lambda \exp \left[ \int_{0}^{t} \alpha_{m}^{0} du + (\omega_f + \mu + \beta) t \right].
\]

Hence,

\[
S_{f_1}(T) \exp \left[ \int_{0}^{T} \alpha_{m}^{0} du + (\omega_f + \mu + \beta) T \right] = S_{f_1}(0) - \rho \Lambda \exp \left[ \int_{0}^{T} \alpha_{m}^{0} du + (\omega_f + \mu + \beta) T \right] \times \int_{0}^{T} \rho \Lambda \exp \left[ \int_{0}^{u} \alpha_{m}^{0} dv + (\omega_f + \mu + \beta) v \right] du.
\]

G\n
\[
S_{f_1}(T) = S_{f_1}(0) - \rho \Lambda \exp \left[ \int_{0}^{T} \alpha_{m}^{0} du + (\omega_f + \mu + \beta) T \right] \times \int_{0}^{T} \rho \Lambda \exp \left[ \int_{0}^{u} \alpha_{m}^{0} dv + (\omega_f + \mu + \beta) v \right] du > 0.
\]

(9)

We have

\[
I_{f_1}(t) = \pi_f \int_{t-\tau}^{t} \alpha_{m}^{0} S_{f_1}(u) e^{-(\mu + \beta)(t-u)} du > 0,
\]

and is strictly positive as everything is positive in \([0, \tau]\) for small \( \tau \).
Similarly, we have
\[
\frac{d(A_f(t) e^{(\tau_f + \tau_m) t})}{dt} = \left( a_f \pi_f + b_f (1 - \pi_f) \right) \int_0^t k \lambda_m S_f(u - \tau) \, du
\]
(11)
giving
\[
A_f(T) = A_f(0) e^{-(\tau_f + \tau_m) T} + e^{-(\tau_f + \tau_m) T} \left( a_f \pi_f + b_f (1 - \pi_f) \right) \int_0^T k \lambda_m S_f(u - \tau) \, du > 0.
\]
(12)
Similarly, it can be shown that \( S_m(T) \), \( I_m(T) \) and \( A_m(T) > 0 \). Thus, we conclude that solutions of system (6) remain positive for all \( t > 0 \). This completes the proof. \( \square \)

2.2. Stability and the basic reproductive number

The disease-free equilibrium for model system (6) is given by
\[
E_0 = (S^0, S^0_m, I^0_f, I^0_m, A^0_f, A^0_m) = \left( \frac{A \rho}{(\mu + \delta + \omega_f)}, \frac{A(1 - \rho)}{(\mu + \delta + \omega_m)}, 0, 0, 0, 0 \right).
\]
The basic reproductive number \( R_0 \), defined as the expected number of secondary infections caused by an infective individual upon entering a totally susceptible population (see Diekmann et al., 1990; Anderson and May, 1991; Hethcote, 2000; van den Driessche and Watmough, 2002) for model system (6), is obtained as follows: we consider a single newly infected high-risk male entering the disease-free population at equilibrium. This individual is still present in the population and infectious at \( t < \tau \) with probability \( e^{-(\mu + \delta) t} \) and in this case infects females at a rate
\[
\pi_f \beta_m e^{S_f/S_m}.
\]
(13)
Hence, the expected number of females infected by this high-risk male is approximately
\[
R_{f1} = \int_0^\tau \frac{\pi_f \beta_m e^{S_f/S_m}}{(\mu + \delta) S_m} e^{-(\mu + \delta) t} \, dt = \frac{\pi_f \beta_m e^{S_f} S_m}{(\mu + \delta) S_m} (1 - k),
\]
(14)
and similarly the expected number of males infected by each of these females is approximately
\[
R_{m1} = \frac{\pi_m \beta_f \epsilon_m S_m}{(\mu + \delta) S_f} (1 - k).
\]
(15)
Considering a high-risk female AIDS case entering the disease-free population at equilibrium in a similar fashion we have
\[
R_{f2} = \int_0^\infty \frac{(a_f \pi_f + b_f (1 - \pi_f)) \beta_m e^{S_f/S_m}}{(\alpha_f + \mu + \nu) S_m} e^{-(\alpha_f + \mu + \nu) t} \, dt = \frac{(a_f \pi_f + b_f (1 - \pi_f)) \beta_m e^{S_f} S_m}{(\alpha_f + \mu + \nu) S_m} (1 - k),
\]
(16)
and the expected number of high-risk male susceptibles infected by each of these females is approximately
\[
R_{m2} = \frac{(a_m \pi_m + b_m (1 - \pi_m)) \beta_f \epsilon_m e^{S_m}}{(\alpha_m + \mu + \nu) S_f} (1 - k).
\]
(17)
The expected average number of secondary cases per generation produced by each infectious male is
\[
\sqrt{R_{f1} R_{m1} + R_{f2} R_{m1} + R_{f1} R_{m2} + R_{f2} R_{m2}}
\]
(18)
interpreting generation as alternating male and female cases and the expected average number of secondary cases per generation produced by each infectious female is also
\[
\sqrt{R_{f1} R_{m1} + R_{f2} R_{m1} + R_{f1} R_{m2} + R_{f2} R_{m2}}.
\]
(19)
Thus, we have the spectral radius for model system (6) as

\[ \mathcal{R}_0 = \sqrt{\mathcal{R}_1 \mathcal{R}_m + \mathcal{R}_2 \mathcal{R}_1 + \mathcal{R}_3 \mathcal{R}_m + \mathcal{R}_4 \mathcal{R}_m} \]

\[ = \left( \beta_f \beta_m c_f c_m \left( \frac{\pi_f \pi_m(1-k)^2}{(\beta + \mu)^2} + \frac{k(1-k)(\mu + v + \gamma_f)}{(\beta + \mu)} \frac{\pi_m b_f(1 - \pi_f) + a_f \pi_f}{(\mu + v + \gamma_f)} \right) \right)^{1/2}. \]  

(20)

Biologically speaking, \( \mathcal{R}_0 \) measures the number of new secondary infections generated by a single HIV-infected individual in a community. In our context \( \mathcal{R}_0 \) measures the number of new secondary infections generated by a single high-risk HIV-infected individual in a community.

2.3. Local stability at \( \mathcal{E}_0 \)

The linearised form for model system (6) may be written in matrix notation as

\[ \frac{dv}{dt} = \mathbf{M}_1 v(t) + \mathbf{M}_2 v(t - \tau), \quad \mathbf{M}_1 = (a_{ij}), \quad \mathbf{M}_2 = (b_{ij}), \]  

(21)

where \( v \) is a vector with components \( v_i \). The zero solution of model system (6) is asymptotically stable if and only if the zero solution of the linearisation (21) is asymptotically stable. If \( v(t) = e^{zt}u \) is a solution of (21), where \( u \) is a constant vector, then

\[ (\mathbf{I} - \mathbf{M}_1 - \mathbf{M}_2 e^{-zt})u = 0. \]  

(22)

Consequently, such solutions exist if and only if \( z \) is a root of the characteristic equation,

\[ \det \Delta(z) = |(\mathbf{I} - \mathbf{M}_1 - e^{-zt} \mathbf{M}_2)| = 0. \]  

(23)

The following facts are known when \( \tau > 0 \) (see Busenberg and Cooke, 1993).

(i) If all characteristic roots \( z \) have negative real parts, then all solutions of (21) tend to zero as \( t \to \infty \), and the zero solution of (6) is asymptotically stable.

(ii) The equation \( \det \Delta(z) = 0 \) always has a root. In any vertical strip \( -\infty < c_1 \leq \Re(z) \leq c_2 < \infty \), there are at most a finite number of roots. There exists \( c_2 \) such that there are no roots in the half-plane \( \Re(z) > c_2 \).

(iii) If all roots satisfy \( \Re(z) \leq 0 \), then a sufficient condition for stability of the zero solution of (21) is that all roots with zero real parts be simple roots.

(iv) The characteristic roots, which are the zeros of the entire function \( \det \Delta(z) \), depend continuously on \( \tau \) for \( \tau > 0 \). For \( \tau = 0 \), there are finitely many roots and for \( \tau > 0 \), there may be infinitely many. The infinitely many roots that arise at \( \tau = 0 \) appear with real parts at \( \infty \).

(v) It is not easy to give necessary and sufficient conditions, directly in terms of \( \mathbf{M}_1, \mathbf{M}_2 \) and \( \tau \) for asymptotic stability. It is worth noting that the natural state space for a functional differential equation is infinite-dimensional. The space that is often chosen is \( C \), the continuous functions on \([-\tau, 0]\) or on \([0, \tau]\).

Linearising model system (6) near the disease-free equilibrium (\( \mathcal{E}_0 \)) gives \( \mathbf{M}_1 \) and \( \mathbf{M}_2 \) as \( 6 \times 6 \) matrices given by

\[ \mathbf{M}_1 = \begin{pmatrix}
-\delta_1 & 0 & 0 & 0 & -\beta_m c_f \frac{S^0_f}{S^0_m} & -\beta_m c_f \frac{S^0_f}{S^0_m} \\
0 & -\delta_2 & 0 & 0 & \pi_f \beta_m c_f \frac{S^0_f}{S^0_m} & \pi_f \beta_m c_f \frac{S^0_f}{S^0_m} \\
0 & 0 & -\delta_3 & 0 & 0 & 0 \\
0 & -\beta_c m \frac{S^0_m}{S^0_f} & -\beta_f c_m \frac{S^0_m}{S^0_f} & -\delta_4 & 0 & 0 \\
0 & \pi_m \beta_c m \frac{S^0_m}{S^0_f} & \pi_m \beta_c m \frac{S^0_m}{S^0_f} & 0 & -\delta_5 & 0 \\
0 & 0 & 0 & 0 & 0 & -\delta_6
\end{pmatrix}. \]  

(24)
where \( \delta_1 = \omega_f + \mu + \beta \), \( \delta_2 = \mu + \beta \), \( \delta_3 = \omega_f + \mu + \beta \), \( \delta_4 = \omega_m + \mu + \beta \), \( \delta_5 = \beta + \mu \) and \( \delta_6 = \omega_m + \mu + \beta \).

\[
M_2 = \begin{pmatrix}
0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & -k \pi_f \beta_m c_f \frac{S_f^0}{S_m^0} & -k \pi_f \beta_m c_f \frac{S_f^0}{S_m^1} \\
0 & 0 & 0 & 0 & k D_f \beta_m c_f \frac{S_f^0}{S_m^0} & k D_f \beta_m c_f \frac{S_f^0}{S_m^1} \\
0 & 0 & 0 & 0 & 0 & 0 \\
0 & -k \pi_m \beta_f c_m \frac{S_m^0}{S_f^0} & -k \pi_m \beta_f c_m \frac{S_m^0}{S_f^1} & 0 & 0 & 0 \\
k E_f \beta_f c_m \frac{S_m^1}{S_f^0} & k E_f \beta_f c_m \frac{S_m^0}{S_f^1} & 0 & 0 & 0 & 0.
\end{pmatrix}
\]  

(25)

Here \( D_1 = (a \pi_f + b_f (1 - \pi_f)) \) and \( E_1 = (a m \pi_m + b_m (1 - \pi_m)) \). Evaluating the characteristic function (23) at \( \varepsilon_0 \) gives

\[
z = - (\alpha_f + \beta + \mu), - (\alpha_m + \beta + \mu), \text{ and solutions } z \text{ of the equation}
\]

\[
f(z) = \beta_f \beta_m c_f c_m \left( \begin{array}{c}
\frac{\pi_f \pi_m (1 - e^{- (\beta_f + \pi_f + \beta + \mu)} z) \pi_m (1 - e^{- (\beta + \pi_f + \beta + \mu)} z)}{\alpha_f (1 - e^{- (\beta_f + \pi_f + \beta + \mu)} z)} \\
\frac{\pi_f \pi_m (1 - e^{- (\beta_f + \pi_f + \beta + \mu)} z) \pi_m (1 - e^{- (\beta + \pi_f + \beta + \mu)} z) \pi_m (1 - e^{- (\beta + \pi_f + \beta + \mu)} z)}{\alpha_f (1 - e^{- (\beta_f + \pi_f + \beta + \mu)} z)} \\
\end{array} \right) \frac{1}{(\beta + \mu)^2} = 1.
\]  

(26)

If \( \tau > 0 \) and \( \Re(z) \geq 0 \), then

\[
|f(z)| \leq f(0) = R_0^2,
\]

(27)

where \( k = e^{- (\beta_f + \pi_f)} \) as defined. Then, if \( R_0 < 1, \forall z \text{ such that } \Re(z) \geq 0, |f(z)| \leq R_0^2 < 1 \), showing that there are no solutions to \( f(z) = 1 \) with positive real part. The absolute value of the left side of Eq. (26) at disease-free is less than unity and thus there can be no root of Eq. (26) with \( \Re(z) \geq 0 \) and thus \( \varepsilon_0 \) is locally asymptotically stable if \( R_0 < 1 \).

2.4. Global stability of disease-free equilibrium

We first state Lemma 1 due to Kribs-Zaleta (1999) for the proof of global stability.

Lemma 1. Given \( x \leq P_0 x + P_1 y, y \leq Q_0 x + Q_1 y \), \( P_i, Q_i, x, y, \geq 0 \), (scalars) for \( i = 0, 1 \), if \( i \) \( P_0 < 1 \), \( Q_i < 1 \) and \( ii \) \( P_0 Q_i - P_i Q_0 > (P_0 + Q_1) - 1 \), then \( x = y = 0 \).

Theorem 2. If \( R_0 < 1 \), the disease-free equilibrium is globally asymptotically stable. If \( R_0 > 1 \), this equilibrium is unstable.

Proof. We first rewrite \( I_f(t) \), \( I_m(t) \), \( A_f(t) \) and \( A_m(t) \) from system (2) as

\[
I_f(t) = \pi_f \int_0^t \beta_f c_f \frac{N_f(t-x) - I_f(t-x) - A_f(t-x) (I_m(t-x) + A_m(t-x)) e^{- (\mu + \beta) x} dx,}
\]

(28)

\[
I_m(t) = \pi_m \int_0^t \beta_f c_m \frac{N_m(t-x) - I_m(t-x) - A_m(t-x) (I_f(t-x) + A_f(t-x)) e^{- (\mu + \beta) x} dx,}
\]

(29)

\[
A_f(t) = k D_f \int_0^\infty \beta_f c_m \frac{N_f(t-x) - I_f(t-x) - A_f(t-x) (I_m(t-x) + A_m(t-x)) e^{- (\mu + \beta) x} dx,}
\]

(30)
where $D_1 = (a_f \pi_f + b_f (1 - \pi_f))$, and
\[ A_{m_1}(t) = kE_1 \int_0^\infty \beta_f c_m \frac{(N_{m_1}(t-x) - I_{m_1}(t-x) - A_{m_1}(t-x))(I_{f_1}(t-x) + A_{f_1}(t-x))}{N_{f_1}(t-x)} e^{-(\mu + \nu + 2\gamma)(x-t)} \, dx, \]
(31)

where $E_1 = (a_m p_m + b_m (1 - p_m))$, $N_{f_1}(t-x) = S_{f_1}(t-x) + I_{f_1}(t-x) + A_{f_1}(t-x)$ and $N_{m_1}(t-x) = S_{m_1}(t-x) + I_{m_1}(t-x) + A_{m_1}(t-x)$.

To show that the condition $R_0 < 1$ is sufficient for global asymptotic stability we consider the following:

\[ \limsup_{t \to \infty} I_{f_1}(t) \]
\[ = \limsup_{t \to \infty} \int_0^\infty \pi_f \beta_m c_f \frac{N_{f_1}(t-x) - I_{f_1}(t-x) - A_{f_1}(t-x)}{N_{m_1}(t-x)} \frac{m_{f_1}(t-x)}{N_{f_1}(t-x)} e^{-(\mu + \nu + 2\gamma)(x-t)} \, dx \]
\[ \leq \int_0^\infty \pi_f \beta_m c_f \limsup_{t \to \infty} \frac{N_{f_1}(t-x) - I_{f_1}(t-x) - A_{f_1}(t-x)}{N_{m_1}(t-x)} \frac{m_{f_1}(t-x)}{N_{f_1}(t-x)} \frac{m_{f_1}(t-x)}{N_{f_1}(t-x)} \frac{m_{f_1}(t-x)}{N_{f_1}(t-x)} e^{-(\mu + \nu + 2\gamma)(x-t)} \, dx \]
\[ \leq \pi_f \beta_m c_f \left(1 - e^{-(\mu + \nu + 2\gamma)(x-t)}\right) \left( \limsup_{t \to \infty} I_{m_1}(t) + \limsup_{t \to \infty} A_{m_1}(t) \right) \]
\[ \leq \frac{\pi_f \beta_m c_f (1 - k)}{\mu + \theta} \left( \limsup_{t \to \infty} \int_0^\infty \pi_m \beta_f c_m \frac{(N_{m_1}(t-x) - I_{m_1}(t-x) - A_{m_1}(t-x))(I_{f_1}(t-x) + A_{f_1}(t-x))}{N_{f_1}(t-x)} \frac{m_{f_1}(t-x)}{N_{f_1}(t-x)} e^{-(\mu + \nu + 2\gamma)(x-t)} \, dx \right) \]
\[ \leq \frac{\pi_f \beta_m c_f (1 - k)}{\mu + \theta} \left[ \pi_m \beta_f c_m (1 - k) \left( \limsup_{t \to \infty} I_{f_1}(t) + \limsup_{t \to \infty} A_{f_1}(t) \right) \right] \]
(32)

and

\[ \limsup_{t \to \infty} A_{f_1}(t) \]
\[ = \limsup_{t \to \infty} \int_0^\infty kD_1 \beta_m c_f \frac{N_{f_1}(t-x) - I_{f_1}(t-x) - A_{f_1}(t-x)}{N_{m_1}(t-x)} \frac{m_{f_1}(t-x)}{N_{f_1}(t-x)} e^{-(\mu + \nu + 2\gamma)(x-t)} \, dx \]
\[ \leq \int_0^\infty kD_1 \beta_m c_f \limsup_{t \to \infty} \frac{N_{f_1}(t-x) - I_{f_1}(t-x) - A_{f_1}(t-x)}{N_{m_1}(t-x)} \frac{m_{f_1}(t-x)}{N_{f_1}(t-x)} e^{-(\mu + \nu + 2\gamma)(x-t)} \, dx \]
\[ \leq kD_1 \beta_m c_f \limsup_{t \to \infty} \frac{N_{f_1}(t-x) - I_{f_1}(t-x) - A_{f_1}(t-x)}{N_{m_1}(t-x)} \left( \limsup_{t \to \infty} I_{m_1}(t) + \limsup_{t \to \infty} A_{m_1}(t) \right) \]
From the above analysis we obtain the following inequalities

\[
\limsup_{t \to \infty} I_{f_1}(t) \leq P \limsup_{t \to \infty} I_{f_1}(t) + P \limsup_{t \to \infty} A_{f_1}(t),
\]

\[
\limsup_{t \to \infty} A_{f_1}(t) \leq Q \limsup_{t \to \infty} I_{f_1}(t) + Q \limsup_{t \to \infty} A_{f_1}(t),
\]

(34)

here,

\[
P = \frac{\pi_{f_1} b_{f_1} c_{f_1} (1-k)}{(\mu + \beta)} \left[ \frac{\pi_m (1-k)}{(\mu + \beta)} + \frac{k e_{f_1} c_{f_1}}{(\mu + v + z_m)} \right],
\]

\[
Q = \frac{\beta_{f_1} b_{f_1} c_{f_1} k (a_f \pi_f + b_f (1-\pi_f))}{(\mu + v + z_f)} \left[ \frac{\pi_m (1-k)}{(\mu + \beta)} + \frac{k (a_m \pi_m + b_m (1-\pi_m))}{(\mu + v + z_m)} \right].
\]

Now applying Lemma 1. Let \( x = \limsup_{t \to \infty} I_{f_1}(t), y = \limsup_{t \to \infty} A_{f_1}(t) \) and \( P_0 = P_1 = P, Q_0 = Q_1 = Q \) be as in inequalities (34). The basic reproductive number

\[
R_0 = \sqrt{P + Q} > \sqrt{\max(P, Q)}.
\]

Thus, \( R_0 < 1 \Rightarrow \max(P, Q) < 1 \), which fulfills (i) of Lemma 1. Clearly, hypothesis (ii) is fulfilled for \( R_0 < 1 \). Thus, if \( R_0 < 1 \), then \( \limsup_{t \to \infty} I_{f_1}(t) = \limsup_{t \to \infty} A_{f_1}(t) = 0 \). In a similar fashion, we have the same conclusion from the analysis of \( \limsup_{t \to \infty} I_{m_1}(t) \) and \( \limsup_{t \to \infty} A_{f_1}(t) \) that if \( R_0 < 1 \), then \( \limsup_{t \to \infty} I_{m_1}(t) = \limsup_{t \to \infty} A_{m_1}(t) = 0 \). This completes the proof. \( \square \)

2.5. Endemic-equilibrium

The endemic-equilibrium for model system (6) is

\[
\mathcal{E}_e = \left( S_{e_1}^*, S_{e_m}^*, I_{e_1}^*, I_{e_m}^*, A_{e_1}^*, A_{e_m}^* \right)
\]

\[
= \left( \frac{\lambda \rho}{(\mu + \beta + \omega_f + \lambda_m^*)} (\mu + \beta + \omega_m + \lambda_f^*)) (\mu + \beta) (\mu + \beta + \omega_f + \lambda_m^*) \right),
\]

\[
\frac{(1-k)e_{f_1} e_{f_1} k}{(\beta + \mu)(\mu + \beta + \omega_m + \lambda_f^*)} \left( \beta_f (1 - \pi_f) + a_f \pi_f \right) (\beta_f + \mu + \omega_f + \lambda_m^*)
\]

\[
\frac{k (b_m (1-\pi_m) + a_m \pi_m) (1-\rho) \lambda e_{f_1}^*}{(\mu + v + \omega_f + \lambda_f^*) (\beta + \mu + \omega_f + \lambda_m^*)}
\]

(36)

where \( \lambda_f^* = \beta_{f_1} c_{f_1} (I_{f_1}^* + A_{f_1}^*) / N_{f_1}^* \). It is straightforward to establish the relationship between the quantities \( \lambda_f = \lambda_f^* = \lambda_f^* \) and \( \lambda_m = \lambda_m^* = \lambda_m^* \) at equilibrium.

\[
\lambda_f^* = \frac{\beta_{f_1} c_{f_1} e_{f_1}^* k}{(\beta + \mu)(\mu + v + \omega_f + \lambda_f^*)} (1-k)(\mu + v + \omega_f + \lambda_f^*)
\]

\[
\lambda_m^* = \frac{\beta_{f_1} c_{f_1} e_{f_1}^* k}{(\beta + \mu)(\mu + v + \omega_m + \lambda_m^*)} (1-k)(\mu + v + \omega_m + \lambda_m^*)
\]

(37)

Clearly, \( \lambda_i^* = 0 \) corresponds to the disease-free equilibrium of model system (2). The endemic equilibrium of the model corresponds to the case where \( R_0 > 1 \) for \( \mathcal{E}_0 \) to be unstable, there exists a unique non-zero solution.
(\hat{\lambda}_f^e, \hat{\lambda}_m^e) \in \Re^+ \times \Re^+$ satisfying the expressions in (37). That is, there is a unique endemic solution for $R_0 \geq 1$. We define the algebraic expressions in (37) as follows: $\hat{\lambda}_f^e = G_f(\hat{\lambda}_m^e)$ and $\hat{\lambda}_m^e = G_m(\hat{\lambda}_f^e)$, respectively, in the positive quadrant of the $(\hat{\lambda}_f^e, \hat{\lambda}_m^e)$ plane. For fixed $\hat{\lambda}_f^e > 0$, we consider the real-valued function $G_f(\hat{\lambda}_m^e)$. It can be seen that $G_f(0) \geq 0$ and $\lim_{\hat{\lambda}_m^e \to \infty} G_f(\hat{\lambda}_m^e) < \infty$.

Thus, $G_f(\hat{\lambda}_m^e)$ is a bounded function for every $\hat{\lambda}_f^e > 0$. Since, in this case, $R_0 > 1$, we show that

$$\frac{\partial G_f(\hat{\lambda}_m^e)}{\partial \hat{\lambda}_f^e} = \frac{\beta_1 \epsilon_f \epsilon_m (\mu + v + \alpha)(k(\mu + \alpha)(b(1 - \pi_f) + \alpha_f \pi_f) + \pi_f(1 - k)(\mu + v + \alpha))}{[(\mu + \alpha)(\mu + v + \alpha) + k(\mu + \alpha)\hat{\lambda}_f^e][b(1 - \pi_f) + \alpha_f \pi_f] + (1 - k)\pi_f \hat{\lambda}_f^e(\mu + v + \alpha)]^2} > 0,$$

and

$$\frac{\partial^2 G_f(\hat{\lambda}_m^e)}{\partial \hat{\lambda}_f^2} = -\frac{2\beta_1 \epsilon_f \epsilon_m (\mu + v + \alpha)[k(\mu + \alpha)(b(1 - \pi_f) + \alpha_f \pi_f) + \pi_f(1 - k)(\mu + v + \alpha)]^2}{[(\mu + \alpha)(\mu + v + \alpha) + k(\mu + \alpha)\hat{\lambda}_f^e][b(1 - \pi_f) + \alpha_f \pi_f] + (1 - k)\pi_f \hat{\lambda}_f^e(\mu + v + \alpha)]^3} < 0. \tag{38}$$

Since $\frac{\partial G_f(\hat{\lambda}_m^e)}{\partial \hat{\lambda}_f^e} > 0$ and $\frac{\partial^2 G_f(\hat{\lambda}_m^e)}{\partial \hat{\lambda}_f^2} < 0$, this implies that $G_f(\hat{\lambda}_m^e)$ is an increasing concave function which has no change in convexity. Consequently, there is a unique $\hat{\lambda}_f^e$ such that $G_f(\hat{\lambda}_m^e) = \hat{\lambda}_f^e$. Clearly, convexity of both $G_f(\hat{\lambda}_m^e)$ and $G_m(\hat{\lambda}_f^e)$ guarantees uniqueness of the endemic solution for $R_0 > 1$. We summarise this result in Theorem 3.

**Theorem 3.** Model system (2) has a unique positive endemic equilibrium $E_e$ when $R_0 > 1$.

Stability analysis of the endemic equilibria of the model system (2) is a daunting one (owing to the nature of $E_e$) and is not reported here.

## 2.6. Effects of educating the high-risk groups

As mentioned in the previous section that the reproductive number ($R_0$) is the effective number of secondary cases produced by a typical infected individual during its entire period of infectiousness in a demographically steady susceptible population. That is, $R_0$ measures the power of a disease to invade a population under conditions that facilitate maximal growth. Since 1911, control and intervention efforts have been based on the concept of the basic reproductive number, introduced by Ross (1911) and Kermack and McKendrick (1927). Therefore, in order to study whether public health educational campaigns can slow down the epidemic in a community we must investigate $R_0$. We can rewrite the reproductive number $R_0$ for model system (6) as

$$R_0 = \sqrt{R_{I_1} + R_{A_1}}, \tag{39}$$

where the partial reproductive numbers

$$R_{I_1} = \frac{\beta_1 \beta_m \epsilon_f \epsilon_m}{(\mu + \alpha)} \left( \frac{\pi_f \pi_m (1 - k)^2}{(\mu + \alpha)^2} \right)^{1/2}$$

and

$$R_{A_1} = \frac{\beta_1 \beta_m \epsilon_f \epsilon_m}{(\mu + \alpha)} \left( \frac{k(1 - k)}{(\mu + \alpha)} \right) \left( \frac{\pi_m (b(1 - \pi_f) + \alpha_f \pi_f)}{(\mu + v + \alpha_f)} \right) + \pi_f (b(1 - \pi_m) + \alpha_m \pi_m) \right) \left( \frac{1}{(\mu + v + \alpha_m)} \right) \right)^{1/2}$$

epidemiologically denote the contribution of high-risk infectives and AIDS cases to the number of secondary infections, respectively. Epidemiological evidence also supports the hypothesis that AIDS cases are capable of and do engage in risky behaviour defined in terms of inconsistent condom use or having multiple sexual partners (Lansky et al., 2000). For example, in a study of HIV-1-infected transfusion male recipients and their female sex partners, O’Brien et al. (1994) show that advanced AIDS patients are more likely to infect their partners (odd ratio 7.9) compared to recipient with no advanced immunodeficiency. The contribution of AIDS cases in HIV transmission considered in our case has also been considered by Elbasha and Gumel (2006), thereby relaxing the widely used assumption that AIDS patients do not partake in the transmission of HIV. It can be shown from Eq. (39) that HIV/AIDS can be eradicated in the community if either $R_{I_1}$ or $R_{A_1}$ exceeds the threshold values of $R_{I_1}$ or $R_{A_1}$, respectively, for $0 < (R_{I_1}, R_{A_1}) < 1$. Corresponding critical values
of $x_i, a_i, b_i$ and $b_i^*$ at which the epidemic may be eradicated can also be obtained, but are hindered here by the complicated expression for $R_0$.

We analyse the effects of educating high-risk groups in slowing the development of HIV/AIDS epidemic for the following cases:

(i) Case 1: When there is completely no education, $\pi_i = a_i = b_i = 1$ and $x_i = 0$. For this case we have

$$\lim_{\pi_i, a_i, b_i \to 1, x_i \to 0} R_0 = R_1$$

$$= \left( \beta_i \beta_m c_{ij} c_m \left( \frac{(1-k)^2}{(\mu + \nu)^2} + \frac{k(1-k)}{(\mu + \nu)} \frac{1}{(\mu + \nu)} + \frac{k}{(\mu + \nu)(\mu + \nu)} \right) \right)^{1/2},$$

where $R_1$ is the reproductive number when there is no education.

(ii) Case 2: When there is effective education, $\pi_i = a_i = b_i = 0$ and $x_i > 0$. For this case we have,

$$\lim_{\pi_i, a_i, b_i \to 0} R_0 = R_2 = 0.$$  

From case 1, we have that $R_1 > R_0$ suggesting that lack of effective educational campaigns in communities with HIV/AIDS, result in an increase in the number of secondary infections. From our numerical values in Table 2 we have $R_0 = 1.83$ and $R_1 = 2.31$, thus confirming the relationship that $R_1 > R_0$ and showing a 126% increase on $R_0$. The reproductive number $R_1$ when there is no education in the community can also be defined to be the reproductive when community is not categorised into risk levels (assuming all individuals are at high-risk). Since $R_1 > R_0$ we note that in resource-poor settings, the identification of risk groups helps in reducing the quantity of resources (e.g., condoms) required in controlling the epidemic.

From case 2, we have $R_2 < R_0 < R_1$ suggesting that with effective public health education campaigns in a community, the number of secondary infections may be reduced thus effective education can help slow or eradicate the epidemic if properly implemented in communities affected by the epidemic. We illustrate the dynamics of the infected population proportion to be 0.0014 and for males-to-females is taken to be 0.005 which are the highest values estimated by Royce et al. (1997). We define $\zeta$ to be the transmission probability per sexual contact. An estimate of $\zeta$ for females-to-males is taken to be 0.0014 and for males-to-females is taken to be 0.005 which are the highest values estimated by Royce et al. (1997). Adopting an approach by Hyman et al. (1999) for estimating the average probability ($\bar{\beta}$) of transmission per partner acquisition we have

$$\bar{\beta} = 1 - (1-\zeta)^n,$$  

where $n$ is the number of contacts with a partner. There is no known relationship between the average number of contacts per partner and the mean probability of transmission per contact (Hyman et al., 1999). Although studies on sexual behaviour are difficult, it appears that partner acquisition rates vary a great deal between communities and that HIV can spread even in populations with fairly low values of partner acquisition ($\zeta$). In the numerical simulations we take $c_i = 5$ (Hyman et al., 1999).

The numerical results in Figs. 2(a)–(c) show that the population trend for $R_1 = 2.31$ is always higher than the population trends for $R_0 = 1.83$ and $R_2 = 0$ with the trend for $R_2 = 0$ being the lowest. The results from Figs. 2(a)–(c) are in agreement with the analytic results that lack of public health educational campaigns in communities with HIV/AIDS, result in an increase in the number of secondary infections ($R_2 < R_0 < R_1$) thus increases the proportion of the infected population (as illustrated in Figs. 2(a) and (b)) and hence HIV/AIDS prevalence as shown in Fig. 2(c). The values of HIV prevalence range 20–25% in Fig. 2(c) are in the range of the prevalence for Zimbabwe which reached of maximum value of 24% [range 20–28%] in 2003 (Zimbabwe Ministry of Health and Child Welfare, 2003; WHO, 2005).
3. The role of sex workers

We extend model system (6) to explore the role of female sex workers in the spread of HIV/AIDS. Sex workers have been found to be the major source of infection in Africa due to unemployment, poverty and underdevelopment which are push factors that motivate individuals and groups of people to move in search of jobs. Migration (in search of employment), however, means that wives and husbands are separated from each other for considerable period of time. Sexual liaisons in places of destination automatically mean multiple sexual partners which in turn increase the demand for prostitution. By definition migrants in low jobs have little education and many have ineffectual knowledge of reproductive health issues and little access to information that would enable them to make decisions on sex that can save them from HIV infection. Kribs-Zaleta et al. (2005) studied HIV/AIDS damage on the Sub-Saharan Africa transportation sector, where long
distance truck drivers are at increased risk of HIV/AIDS infection due to the migratory nature of their job and their prolonged absence from home. As a result truck drivers are more likely to have sexual interactions with sex workers, who often provide them with affordable food and lodging during their journey and the spread of HIV/AIDS is exacerbated by highly sexually active lifestyles of both the truck drivers and the sex workers they visit. Sex workers in Africa have revealed that most of their clientele insist on not using condoms (Campbell, 2000), thus increasing the risk of HIV infection.

The extended model assumes that a proportion $p$ of the high-risk infected females is non-sex workers and is categorised in the $I_f$ class and the complementary proportion is sex workers and is categorised in the $I_{fp}$ class. The model assumes that there are no sex workers among the high-risk female AIDS class. It is assumed that high-risk male susceptibles are capable of sexually interacting with either a sex worker, non-sex worker or high-risk AIDS cases in the female population.

The relative risk of high-risk male susceptible acquiring HIV transmission from female sex worker is modelled by a parameter $r$. We assume $r > 1$ because of the high-risk of acquiring HIV transmission from female sex workers. Assuming homogeneous mixing we have

$$
\dot{S}_{m}^{n} = \beta_m c_f \frac{I_m(t-n) + A_m(t-n)}{N_m(t-n)},
$$

where $N_m(t-n) = S_m(t-n) + I_m(t-n) + A_m(t-n), n = 0, \tau,$ (41)

and

$$
\dot{I}_f(t) = \beta_f c_m I_f(t-n) + r I_{fp}(t-n) + A_f(t-n),
$$

where $N_f(t-n) = S_f(t-n) + I_f(t-n) + I_{fp}(t-n) + A_f(t-n), n = 0, \tau,$ (42)

The model equations take the following form:

$$
S_f(t) = \rho N - \beta_f S_f(t) - (\omega_f + \beta_f)S_f(t),
$$

$$
I_f(t) = p \beta_f S_f(t) - p \beta_f k S_f(t) - (\mu + \beta_f)I_f(t),
$$

$$
I_{fp}(t) = (1 - p) \beta_f S_f(t) - (1 - p) \beta_f k S_f(t) - (\mu + \beta_f)I_{fp}(t),
$$

$$
A_f(t) = a_f \beta_f k S_f(t) + b_f (1 - \beta_f) S_f(t) - (\omega_f + \beta_f + v_1) A_f(t),
$$

$$
S_{m}(t) = (1 - \rho) N - \beta_f S_{m}(t) - (\omega_m + \beta_f) S_{m}(t),
$$

$$
I_{m}(t) = \pi_m \beta_m S_{m}(t) - \pi_m k \beta_m S_{m}(t) - (\mu + \beta_f) I_{m}(t),
$$

$$
A_{m}(t) = a_{m} \pi_m k \beta_m S_{m}(t) + b_{m} (1 - \pi_m) k \beta_m S_{m}(t) - (\omega_m + \beta_f + v_1) A_{m}(t).
$$

(43)

The mathematical properties of model system (43) are similar to those of model system (6) with the initial conditions given as

$$
\begin{align*}
S_f(0) &= \varphi_1(0), & S_m(0) &= \varphi_2(0), & I_f(0) &= \varphi_3(0), & I_{fp}(0) &= \varphi_4(0), \\
I_{m}(0) &= \varphi_5(0), & A_f(0) &= \varphi_6(0), & A_{m}(0) &= \varphi_7(0), & \theta \in [-\tau, 0].
\end{align*}
$$

(44)

The disease-free equilibrium for model system (43) is

$$
E_0 = (S_f^0, S_m^0, I_f^0, I_{fp}^0, A_f^0, A_{m}^0) = \left( \frac{\Lambda}{(\mu + \beta + \omega_f)}, \frac{\Lambda(1 - \rho)}{(\mu + \beta + \omega_m)}, 0, 0, 0, 0 \right),
$$

and the endemic equilibrium

$$
E_\ast = (S_f^\ast, S_m^\ast, I_f^\ast, I_{fp}^\ast, A_f^\ast, A_{m}^\ast)
$$

$$
= \left( \frac{\Lambda \rho}{(\mu + \beta + \omega_f + \omega_m)}, \frac{\Lambda(1 - \rho)}{(\mu + \beta + \omega_m + \omega_f)} \right),
$$

$$
\left( 1 - k \right) \pi_f \Lambda \beta_m^\ast \left( \beta_f \right) + \left( \beta_f \right) \left( 1 - k \right) \pi_m \Lambda \beta_m^\ast \left( \beta_f \right),
$$

$$
\left( \beta_f \right) \left( 1 - k \right) \pi_m \Lambda \beta_m^\ast \left( \beta_f \right),
$$

$$
\left( \beta_f \right) \left( 1 - k \right) \pi_m \Lambda \beta_m^\ast \left( \beta_f \right),
$$

$$
\frac{k \left( b_f (1 - \beta_f) \right)}{(\mu + \beta_f + \omega_f + \omega_m)}, \frac{k \left( b_m (1 - \pi_m) \right)}{(\mu + \beta_f + \omega_f + \omega_m)}, \frac{k \left( b_m (1 - \pi_m) \right)}{(\mu + \beta_f + \omega_f + \omega_m)}.
$$

(45)
where $l^c_m = \beta_m c_m \left( \frac{I^c_{m1} + A^c_{m1}}{N^c_{m1}} \right)$ and $l^c_f = \beta_f c_f \left( \frac{I^c_{f1} + r I^c_{f1}}{N^c_{f1}} \right)$. It is straightforward to show the uniqueness of the endemic equilibrium for basic reproductive number greater than unity. The reproductive number for the model system (43) is

$$R_P = \sqrt{R_{f1}R_{m1}f(p,r) + R_{f2}R_{m1} + R_{f1}R_{m2}f(p,r) + R_{f2}R_{m2}}$$

$$= \left( \beta_f \beta_m c_f c_m \left( \frac{\pi_f \pi_m (1 - k)^2}{(\beta + \mu)^2} f(p,r) + \frac{k(1 - k)}{(\beta + \mu)} \frac{\pi_m (b_f (1 - \pi_f) + a_f \pi_f)}{(\mu + v + \alpha_f)} + \frac{\pi_f (b_m (1 - \pi_m) + a_m \pi_m)}{(\mu + v + \alpha_m)} f(p,r) \right) + \frac{k^2 (b_f (1 - \pi_f) + a_f \pi_f) (b_m (1 - \pi_m) + a_m \pi_m)}{(\mu + v + \alpha_f) (\mu + v + \alpha_m)} \right)^{1/2}$$

(46)

where $f(p,r) = p + r - pr$. The function $f(p,r)$ measures the combined efficacy of interventions targeted at sex workers and in this case, the interventions are:

(i) reducing the proportion of female sex workers, and
(ii) educating sex workers to engage in safer sex.

![Simulation results showing the dynamics of: (a) the behaviour of the function $f(p,r)$ versus $p$, (b) the behaviour of the function $f(p,r)$ versus $r$. Parameter $p$ is varied from 0 to 1 in steps of 0.1 and corresponding values of $r$ varying from 1 to 11 in steps of 1 for both cases.](image-url)
We illustrate the behaviour of function \( f \) in Figs. 3(a) and (b). The numerical illustration confirms that \( f > 1, \forall 0 < p < 1, r > 1 \). We analyse sex worker-induced reproductive number \( R_p \) for the following cases.

(i) Case 1: When there are no female sex workers in the community, \( p = 1 \Rightarrow f(1, r) = 1 \) and we have for this case \( R_p = R_0 \).

(ii) Case 2: When there are female sex workers with no effective education or with completely no education, \( 0 < p < 1, r > 1 \Rightarrow f(p, r) > 1 \). Thus, for this case we have \( R_p > R_0, \forall 0 < p < 1 \).

(iii) Case 3: When sex workers are effectively educated, \( r \leq 1 \), we have \( R_p \leq R_0, \forall 0 < p < 1 \).

From case 1, we have \( R_p = R_0 \) suggesting that the reproductive number for model systems (6) and (43) is equal for \( p = 1 \) (model system (43) equals (6) for \( p = 1 \)). For case 2, we note that the presence of female sex workers in a community fuels the epidemic \( (R_p > R_0) \) and for case 3 we note that effective education of sex workers reduces the number of secondary infections and can thus slow down the epidemic.

In Figs. 3(a) and (b), parameter \( p \) is varied from 0 to 1 in steps of 0.1 and corresponding values of \( r \) varying from 1 to 11 in steps of 1 for both cases. The plot of \( f(p, r) \) versus \( p \) and \( f(p, r) \) versus \( r \) for the concurrent increase in \( p \) and \( r \) are shown in Figs. 3(a) and (b), respectively. The results in Figs. 3(a) and (b) illustrate that for a concurrent increase in the proportion \( p \) and the risk factor \( r \) in a setting with sex workers will result in an increase in the combined efficacy of interventions \( f(p, r) \) (from \( f(0, 1) = 1 \)) to a maximum value \( f(0.5, 6) = 3.5 \) and falls to a minimum value \( f(1, 11) = 1 \). We conclude from the results in Figs. 3(a) and (b) that a reduction in the proportion of females becoming sex workers in a setting with increasing risk of acquiring HIV transmission from sex workers will result in an increase in the number of secondary infections (hence HIV prevalence) to a maximum value (attained at \( f(0.5, 6) = 3.5 \) in our case) after which it will fall to a minimum value (attained at \( f(1, 11) = 1 \) in our case).

For numerical simulations of model system (43) we use the parameter values in Tables 1 and 2 unless stated. The parameters in Tables 1 and 2 give \( R_p = 2.289 \) and a 125% increase in \( R_0 = 1.83 \) due to the presence of sex workers.

### Table 1

**Data for the HIV/AIDS model**

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Symbol</th>
<th>Value</th>
<th>Source</th>
</tr>
</thead>
<tbody>
<tr>
<td>Recruitment rate</td>
<td>( \Lambda )</td>
<td>0.029 yr(^{-1} )</td>
<td>CSOZ</td>
</tr>
<tr>
<td>Proportion of female recruits</td>
<td>( \rho )</td>
<td>0.5</td>
<td>CSOZ</td>
</tr>
<tr>
<td>Natural death rate</td>
<td>( \mu )</td>
<td>0.02 yr(^{-1} )</td>
<td>CSOZ</td>
</tr>
<tr>
<td>AIDS-related death rate</td>
<td>( \nu_1 )</td>
<td>0.333 yr(^{-1} )</td>
<td>CSOZ</td>
</tr>
<tr>
<td>Emigration rate</td>
<td>( \bar{g} )</td>
<td>0.01 yr(^{-1} )</td>
<td>CSOZ</td>
</tr>
<tr>
<td>Average incubation period</td>
<td>( \tau )</td>
<td>8 yr</td>
<td>CSOZ</td>
</tr>
<tr>
<td>Probability of transmission</td>
<td>((\beta_f, \beta_m))</td>
<td>(0.0497, 0.1637)</td>
<td>Estimate</td>
</tr>
<tr>
<td>Partner acquisition rate</td>
<td>((\gamma_s, \gamma_m))</td>
<td>(5.5) partners/year</td>
<td>Estimate</td>
</tr>
<tr>
<td>Rate of transfer to low-risk susceptible group ( (a_m = \omega_m) )</td>
<td>0.1 yr(^{-1} )</td>
<td>Estimate</td>
<td></td>
</tr>
<tr>
<td>Rate of transfer to low-risk AIDS group ( (a_m = \gamma_s) )</td>
<td>0.5 yr(^{-1} )</td>
<td>Estimate</td>
<td></td>
</tr>
<tr>
<td>Proportion of susceptibles</td>
<td>( a_m = \gamma_m )</td>
<td>0.9</td>
<td>Estimate</td>
</tr>
<tr>
<td>who remain high-risk</td>
<td>( a_m = \gamma_m )</td>
<td>0.5</td>
<td>Estimate</td>
</tr>
<tr>
<td>Infectives who remain high-risk</td>
<td>( h_m = \beta_f )</td>
<td>0.1</td>
<td>Estimate</td>
</tr>
<tr>
<td>Proportion of low-risk infectives</td>
<td>( (R_0, R_1, R_2) )</td>
<td>(1.83, 2.31, 0)</td>
<td></td>
</tr>
</tbody>
</table>

### Table 2

**Data for the HIV/AIDS model**

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Symbol</th>
<th>Value</th>
<th>Source</th>
</tr>
</thead>
<tbody>
<tr>
<td>Proportion of non-sex workers</td>
<td>( p )</td>
<td>0.8</td>
<td>Estimate</td>
</tr>
<tr>
<td>Sex workers risk-factor</td>
<td>( r )</td>
<td>5</td>
<td>Estimate</td>
</tr>
<tr>
<td>Reproductive numbers</td>
<td>( (R_0, R_p) )</td>
<td>(1.83, 2.289)</td>
<td></td>
</tr>
</tbody>
</table>
Partial derivatives of $f(p,r)$ with respect to $p$ and $r$ gives

$$\frac{\partial f}{\partial p} = 1 - r, \quad \frac{\partial f}{\partial r} = 1 - p.$$  \hfill (47)

For settings with female sex workers with a risk factor $r > 1$, we have from (47)

$$\frac{\partial f}{\partial p} < 0,$$  \hfill (48)

showing that an increase in the proportion ($p$) of non-sex workers reduces $f$ and hence reduces $R_P$; thus, overall it slows down the epidemic. Fig. 4(a) shows a decrease in the proportion of the total infected population for $p$ increasing in the direction of the arrow thus confirms the result that increase in $p$ slows down the development of HIV/AIDS epidemic.

We note from (47) that

$$\frac{\partial f}{\partial r} > 0, \quad \forall 0 < p < 1,$$  \hfill (49)

Fig. 4. Simulation results showing: (a) a decrease in the total infected population (HIV/AIDS prevalence) for $p$ increasing ($p = 0, 0.5, 1$) in the direction of the arrow with $r = 5$, (b) an increase in the total infected population for $r$ increasing ($r = 1, 5, 10$) in the direction of the arrow with $p = 0.8$. Parameter values in Table 1 are used for the numerical simulations with, $S_{1i} = 0.35, I_{1i} = 0.075, I_{f1} = 0.075, A_{1i} = 0.0, S_{m1} = 0.35, I_{m1} = 0.15$ and $A_{m1} = 0.0$. 

showing that an increase in sex workers risk factor $r$ increases $f$ and thus increases $R_p$; hence, it enlarges the epidemic. Fig. 4(b) shows an increase in the total infected proportion for $r$ increasing in the direction of the arrow thus supporting the result that an increase in sex workers risk behaviour enlarges the epidemic. We conclude from the analysis that the epidemic can be contained in settings with female sex workers if the proportion of females becoming sex workers is reduced and if female sex workers are to be educated on ways of reducing the risk of acquiring HIV infection. Numerical results in Figs. 4(a) and (b) also explain that a reduction in the risk factor of acquiring HIV infection has significant impact on HIV/AIDS prevalence than is a change in the proportion of females becoming sex workers which has noticeable impact after decades.

In general, the sex workers population in Africa is driven by economic desperation and feelings of powerlessness, with individual women negotiating better situations for themselves as their financial situations improve (Caldwell and Quiggin, 1989; Kribs-Zaleta et al., 2005). In (Campbell, 2000) several factors that impede sex workers from insisting on condom use, including clients’ refusal to do so, competition for clients, lack of a common language between sex workers and their clients, and lack of self-confidence stemming from early life experiences are cited. To empower sex workers, several studies have recommended developing peer support networks of sex workers through which information and education could be transmitted (Campbell, 2000; Gysels et al., 2002; Hall, 2003).

4. Summary and concluding remarks

In this paper we formulated and analysed a sex-structured HIV/AIDS model in which the population is divided into three subgroups: susceptibles, infectives and AIDS cases which are further divided into two classes, those involved in high-risk sexual activities and those involved in low-risk sexual activities. The movement of individuals from high to low sexual activity groups is considered to be a result of public health educational campaigns. The mathematical properties of the model which includes stability of the disease-free equilibrium and uniqueness of the endemic equilibrium were investigated. The model considers the contribution of AIDS cases in HIV transmission thereby relaxing the widely used assumption that AIDS cases do not partake in the transmission of HIV, a similar approach in Elbasha and Gumel (2006). The epidemic threshold known as the basic reproductive number, $R_0$, was obtained and split into two partial reproductive numbers $R_{P1}$ and $R_{A1}$ showing the contribution of the high-risk infectives and the AIDS cases to $R_0$ (the number of secondary infections), respectively. We extended the model to explore the role of sex workers in the spread of HIV/AIDS in settings with heterosexual transmission and overall community gains if female sex workers are educated.

The analysis of $R_0$ gives $R_{P1}$ and $R_2$ as the reproductive numbers in the case with completely no public health educational campaigns and when there are effective public health educational campaigns, respectively. We noted from the analytic approach that $R_2 < R_0 < R_{P1}$ and numerical analysis of the model also confirms the same relationship suggesting that public health educational campaigns may reduce the number of secondary infections among the high-risk population and can thus help slow down the epidemic if properly implemented in affected communities. An extension of the basic model to explore the role of sex workers results in a sex worker-induced reproductive number $R_P$, which is related to $R_0$ by a combined efficacy function $f(p, r)$, where $p$ is the proportion of high-risk infected females that are non-sex workers and $r$ is the relative risk of acquiring HIV from a sex worker. Analysis of $R_P$ for the case with no sex workers gives $R_P = R_0$, for the case with sex workers with completely no education gives $R_P > R_0$ and for the case with effective education gives $R_P < R_0$ suggesting that the presence of female sex workers in a community fuels the epidemic but educating this group may slow down the epidemic. We also note from a comprehensive analysis of the model that the epidemic can be contained in setting with female sex workers if the proportion of female becoming sex workers is reduced and if female sex workers are to be educated on ways of reducing the risk of acquiring HIV infection.

We conclude from the study that the presence of sex workers enlarges the epidemic threshold $R_0$, thus fuels the epidemic among the heterosexuals, and that public health educational campaigns among the high-risk heterosexual population reduces $R_0$, thus can help to slow or eradicate the epidemic. The most urgent public-health problem in the world today is to devise effective strategies to minimise the destruction caused by HIV/AIDS epidemic, but public-health policies may have consequences at population level. The model presented in this study did not consider the effects of therapeutic intervention strategies (such as the use of antiretroviral drugs) in the transmission dynamics of HIV/AIDS in heterosexual settings. Following the concepts of Blower et al. (2000, 2001, 2005), we will extend our work to consider the potential consequences of antiretroviral treatment and subsequent evolution of drug resistant strains elsewhere.

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