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A model for HIV transmission with two interacting high-risk groups

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ABSTRACT

We formulate a model of HIV transmission which keeps track of two interacting high-risk groups, namely female sex workers (FSW) and male injecting drug users (IDU), along with a third "bridge" group of male drug-free clients (DFC). To determine the global asymptotic behaviour of the model, we first consider the dynamics of an n-group SIR model featuring abstract, unspecified and possibly nonlinear forces of infection utilising the graph theoretic approach of Li and Shuai. It is determined that the basic reproduction number R_0, computed via the next generation method, is a threshold parameter for the stability of the disease-free and the endemic equilibrium. Global stability results for the model with two interacting high-risk groups are then obtained via suitable particularisations. We obtained partial reproduction numbers for each disease transmission route in the model, via which and our analytical results we are able to establish that if the goal of an intervention measure is to eradicate, significant reduction in transmission between FSW and IDU is needed, in addition to reduction in other routes of transmission. On the other hand, if the aim is to mitigate the disease spread, reduction in any one or more routes of disease transmission will be useful, albeit reduction in transmission between the two high-risk groups will be more impactful than others.

1. Introduction

HIV/AIDS model integrating high-risk groups has been a subject of significant interest. In particular, model with a high-risk group of female sex workers (FSW) and non-high-risk group of young unmarried males has been used to explain the rapid spread of HIV/AIDS in Thailand in the early 90s [1]. Another general model which incorporates treatment and behaviour change for HIV-infected FSW and a bridge population of young un-partnered males was proposed and analysed in [2,3]. A structured community model with two classes of direct (high activity) and indirect (low activity) FSW and two classes of sexually active

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male clients (highly active and less active) has been formulated in [3,4]. However, none of the articles above involve male injecting drug users (IDU) since there is little evidence of significant interaction between the FSW and IDU in Thailand at that time.

To provide real world background motivation for modelling interacting high-risk groups, we consider the HIV/AIDS epidemic in southwest China and how its high-risk groups became overlapped in time. The first HIV outbreak in China, recorded in 1989 in Yunnan Province, was confined to IDUs [5]. It has been estimated that 44% of the sex workers in southern China had unprotected commercial sex with their clients [6]. Peer disapproval of condom use and high intimate relationships with sex workers were determined to be barriers to consistent condom use by clients of sex workers [7], along with the fact that some FSW are often willing to engage in unprotected sex if their clients pay extra. Despite a significant decrease during 2000–2011, southwest China still bore the greatest HIV disease burden for FSW [8]. There is a high risk of HIV transmission from FSW to long-term partners or possibly to newborns through mother-to-child vertical transmission. Moreover, the male clients, once infected with HIV through buying sex, could in turn spread HIV to their partners or wives as a bridge population, since they may transmit HIV from a high risk group (FSW) to the general population [9].

Injecting drug use is another key factor in spreading HIV to the general population. A 2010 self-administered, standard behavioural surveillance survey of 12,622 FSW recruited from Guangxi indicates 2.6% non-injecting drug users and 0.5% IDU [10]. In November 2002, a community-based survey targeting HIV-seronegative IDUs was conducted in Xichang County of Sichuan Province, China. Over the following 36-month follow-up period observation, the study showed that the average HIV incidence rate was 2.3% [11,12]. In a study in Guizhou Province in 2000, nearly 30% of all IDUs were women, and a considerable number of them had engaged in commercial sex [13]. A 2004 study reported around 21% of female IDUs surveyed in Yunnan Province reported selling sex for money or drugs in the previous month [14], while 60% of female IDUs in Sichuan Province in 2003 reported selling sex for money or drugs and $<30\%$ of them reported consistent condom use with customers [15]. Surveys in Yunnan Province in early 2000's reported that HIV prevalence among female IDUs was significantly higher than HIV prevalence among male IDUs [16,17].

A 2005 study, motivated by the HIV epidemic in Yunnan province, China in 1989 which has progressed to a concentrated epidemic, compares the level of HIV risk behaviours of needle/apparatus sharing among male IDUs and unsafe commercial sex between FSW and male clients [18] and examines the effects of risk factors for HIV infection among these two groups. Prevalence rates as high as 74.5% were reported among IDUs, those reported among FSW being as high as 10% [19]. Therefore, unlike in other parts of China, there is evidence of significant interaction between the high-risk groups of FSW and IDUs, which provides motivation for our study.

More recently, a model which classifies the at-risk population into IDUs (who do not engage in commercial sex) and drug-free individuals who engage in commercial sex (sex workers and their clients) was proposed in [20]. However, in this model, the equations for the infected subpopulations decouple near the disease-free equilibrium which grossly simplifies the analysis and subsequently its biological significance. In this work, we will formulate a model with two interacting high-risk groups of FSW and IDU with which to carry out analysis.

2. The model

In this paper, we formulate an HIV transmission model with two interacting high-risk groups of FSW and IDU under the following assumptions.

1. Two HIV transmission routes are considered: needle/apparatus sharing between male IDU and commercial sex between FSW and sexually active male clients.

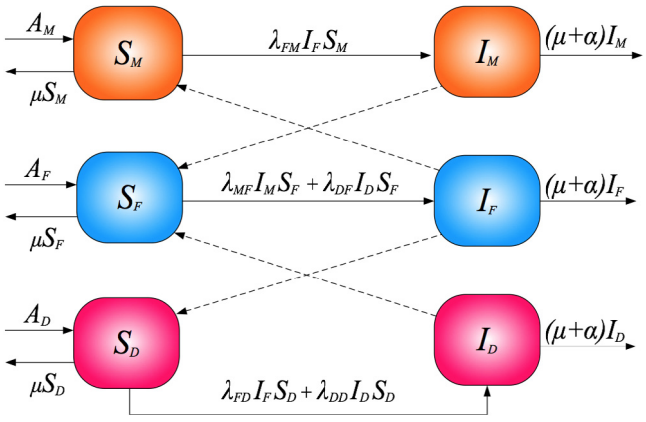

Fig. 1. Model flow diagram: solid arrow denotes disease progression, broken arrow denotes disease transmission.

2. The total population is classified into three compartments according to the disease status in the body: the susceptible individuals S; the infected individuals I before progression to AIDS; and the (removed) AIDS patients R. We only incorporate compartments S and I into the model and ignore compartment R since the AIDS patients do not have contacts (sexual or needle/apparatus sharing) with any other population groups
3. The population is assumed to mix homogeneously.
4. Incidence is mass action.
5. All newly recruited individuals to the compartments of male drug-free clients (male DFC), FSW, male IDU are respectively assumed susceptible.

These assumptions lead to the dynamic flow diagram for HIV transmission pictured in Fig. 1, the definitions of the corresponding parameters being listed in Table 1.

We formulate the following system of ordinary differential equations for the SIR model, noting that we do not give the equation for the removed class R since we assume AIDS patients do not play a role in disease transmission.

$$\begin{aligned}
\frac{dS_M}{dt} &= A_M - \lambda_{FM}I_FS_M - \mu S_M\\
\frac{dI_M}{dt} &= \lambda_{FM}I_FS_M - (\mu+\alpha)I_M\\
\frac{dS_F}{dt} &= A_F - \lambda_{MF}I_MS_F - \lambda_{DF}I_DS_F - \mu S_F\\
\frac{dI_F}{dt} &= \lambda_{MF}I_MS_F + \lambda_{DF}I_DS_F - (\mu+\alpha)I_F\\
\frac{dS_D}{dt} &= A_D - \lambda_{FD}I_FS_D - \lambda_{DD}I_DS_D - \mu S_D\\
\frac{dI_D}{dt} &= \lambda_{FD}I_FS_D + \lambda_{DD}I_DS_D - (\mu+\alpha)I_D.
\end{aligned} \tag{2.1}$$

3. The multigroup SIR model

In what follows, we shall attempt to analyse the global behaviour of a n-group version of (2.1) with nonlinear forces of infection, of which (2.1) is a special case. Let us consider the multigroup SIR

Table 1
Definition of various parameters.

Parameter	Biological meaning
A_M	Constant recruitment into the group of susceptible male DFC
A_F	Constant recruitment into the group of susceptible FSW
A_D	Constant recruitment into the group of susceptible male IDU
λ_{XY}	HIV transmission coefficient from I_X to S_Y
μ	Natural death rate
α	Rate of AIDS development

model

$$\begin{cases} \dfrac{dS_i}{dt} = A_i - d_i S_i - \displaystyle\sum_{j=1}^{n} \beta_{ij} S_i g_{ij}(I_j) \\ \dfrac{dI_i}{dt} = \displaystyle\sum_{j=1}^{n} \beta_{ij} S_i g_{ij}(I_j) - (\mu_i + \alpha_i) I_i \end{cases}, \quad 1 \le i \le n. \tag{3.1}$$

In what follows, we shall employ the following assumptions upon the functions g_{ij}, $1 \le i, j \le n$.

(i) g_{ij} are locally Lipschitz continuous function on $[0, \infty)$ satisfying $g_{ij}(0) = 0$.
(ii) $0 \le \lim_{I_j \to 0+} \frac{g_{ij}(I_j)}{I_j} = C_{ij} < \infty$.
(iii) $g_{ij}(I_j) \le C_{ij} I_j$ for all $I_j \ge 0$.
(iv) The matrix $(\beta_{ij} C_{ij})_{\substack{1 \le i \le n \\ 1 \le j \le n}}$ is irreducible.

In particular, note that we do not require all g_{ij}'s to be nonidentically zero, and some of them may be null (although not too many, due to the assumption (iv)). This feature is essential for our concrete model (2.1), since we do not account for homosexual transmission, which leads to the corresponding g_{ij}'s being null. Also, from (ii), it is seen that $C_{ij} = g'_{ij}(0)$. Further, note that (ii) and (iii) are satisfied if $g_{ij}(I_j) = C_{ij} I_j$ or $g_{ij}(I_j) = \frac{C_{ij} I_j}{1 + D_{ij} I_j}$, $C_{ij}, D_{ij} \ge 0$, that is, by functions which either directly appear in the formulations of our concrete model (2.1), or may be used to account for the effects of behavioural changes. The notion of an irreducible matrix will be detailed in Section 3.2.

3.1. The basic reproduction number

The dynamics of (3.1) will be discussed on the feasible domain

$$\Gamma = \left\{ (S_1, I_1, \ldots, S_n, I_n);\ S_i, I_i \ge 0,\ S_i \le \frac{A_i}{d_i},\ S_i + I_i \le \frac{A_i}{d_i^*} \right\},$$

where

$$d_i^* = \min(d_i, \mu_i + \alpha_i).$$

It is easy to see that the system (3.1) has a disease-free equilibrium $\mathbf{E}_0$,

$$\mathbf{E}_0 = (S_1^0, 0, S_2^0, \ldots, S_n^0, 0), \quad S_i^0 = \frac{A_i}{d_i}, \quad 1 \le i \le n.$$

In order to use the next generation matrix approach formulated in van den Driessche and Watmough [21], we define

$$\mathcal{M}^0 = \left(\frac{\beta_{ij} S_i^0 C_{ij}}{\mu_j + \alpha_j} \right)_{\substack{1 \le i \le n \\ 1 \le j \le n}} = F V^{-1}, \quad \widetilde{\mathcal{M}}^0 = \left(\frac{\beta_{ij} S_i^0 C_{ij}}{\mu_i + \alpha_i} \right)_{\substack{1 \le i \le n \\ 1 \le j \le n}} = V^{-1} F,$$

where

$$F = \left(\beta_{ij} S_i^0 C_{ij}\right)_{\substack{1\le i\le n\\ 1\le j\le n}}, \quad V = \operatorname{diag}(\mu_i + \alpha_i)_{1\le i\le n}.$$

Subsequently, we may define the basic reproduction number of the model (3.1) as $\mathcal{R}_0 = \rho(\mathcal{M}^0)$, the spectral radius of the matrix $\mathcal{M}^0$. However, since $\rho(FV^{-1}) = \rho(V^{-1}F)$, it also follows that $\mathcal{R}_0 \doteq \rho(\widetilde{\mathcal{M}}^0)$.

3.2. A summation lemma

We now introduce several matrix theory notions and notations, together with a summation lemma which will prove useful when evaluating the derivative of a Lyapunov functional along the solutions of (3.1), in the process of discussing the stability of the endemic equilibrium. Our approach is motivated by the argument employed in Li and Shuai [22], Section 7.

Let $U = (u_{kj})$, $V = (v_{kj})$ be $n \times n$ matrices. We shall write $U \le V$ if $u_{kj} \le v_{kj}$ for all $1 \le j, k \le n$ and $U < V$ if $U \le V$ and $U \ne V$. If $O_n \le U$, we shall say that U is nonnegative.

Given a nonnegative $n \times n$ matrix $A = (a_{kj})$, the directed graph $G(A)$ associated with A has vertices $1, 2, \ldots, n$, with a directed arc (k, j) starting from vertex k to vertex j if and only if $a_{kj} \ne 0$. The directed graph $G(A)$ is then said to be strongly connected if any two distinct vertices can be joined by an oriented path. Under these circumstances, the matrix A is irreducible if and only if the associated directed graph $G(A)$ is strongly connected. Equivalently, a $n \times n$ matrix A, $n \ge 2$, not necessarily nonnegative, is irreducible if for any proper subset M of $\{1, 2, \ldots, n\}$, there are $i \in M$ and $j \in \{1, 2, \ldots, n\} \setminus M$ such that $a_{ij} \ne 0$.

For a nonnegative $n \times n$ matrix $A = (a_{kj})$, $n \ge 2$, let

$$L = \begin{pmatrix} \sum\limits_{l\ne 1} a_{1l} & -a_{2l} & \ldots & -a_{n1} \\ -a_{12} & \sum\limits_{l\ne 1} a_{2l} & \ldots & -a_{n2} \\ \vdots & \vdots & & \vdots \\ -a_{1n} & -a_{2n} & \ldots & \sum\limits_{l\ne n} a_{nl} \end{pmatrix}$$

be the Laplacian matrix of the directed graph $G(A)$ associated with A and let L_{kj} be the cofactor of the (k, j) entry of L. Let also $c_i = L_{ii}$. The following result then holds as a consequence of Kirchoff's matrix tree theorem (see Guo et al. [23], Appendix 1, for further details).

Lemma 3.1. *Let c_k, $1 \le k \le n$ be defined as above. Then*

$$\sum_{k=1}^{n}\sum_{j=1}^{n} c_k a_{kj} H_k(x_k) = \sum_{k=1}^{n}\sum_{j=1}^{n} c_k a_{kj} H_j(x_j),$$

where $\{H_k(x_k)\}_{k=1}^n$ is an arbitrary family of functions.

3.3. The stability of the disease-free equilibrium

Let us also define

$$\mathbf{S} = (S_1, S_2, \ldots, S_n)^T, \quad \mathbf{S}^0 = (S_1^0, S_2^0, \ldots, S_n^0)^T, \quad \mathbf{I} = (I_1, I_2, \ldots, I_n)^T,$$
$$\mathbf{0} = (0, 0, \ldots, 0)^T, \quad \widetilde{\mathcal{M}}(\mathbf{S}) = \left(\frac{\beta_{ij} S C_{ij}}{\mu_i + \alpha_i}\right)_{\substack{1\le i\le n\\ 1\le j\le n}}.$$

Theorem 3.1.

(a) *If $\mathcal{R}_0 \leq 1$, then the disease-free equilibrium $\mathbf{E}_0$ is the unique equilibrium of (3.1) and it is globally asymptotically stable in Γ.*

(b) *If $\mathcal{R}_0 > 1$, then the disease-free equilibrium $\mathbf{E}_0$ is unstable.*

Proof. (a) It follows from conditions (ii) and (iii) that $O_n \leq \widetilde{\mathcal{M}}(\mathbf{S}) \leq \widetilde{\mathcal{M}}^0$ on Γ. Also, if $\mathbf{S} \neq \mathbf{S}^0$, it follows that $O_n < \widetilde{\mathcal{M}}(\mathbf{S}) < \widetilde{\mathcal{M}}^0$, and from Corollaries 2.1.5 and 2.1.10 of Berman and Plemmons [24] one finds that $\rho(\widetilde{\mathcal{M}}) < \rho(\widetilde{\mathcal{M}}^0)$. Therefore, if $\mathcal{R}_0 \leq 1$, then $\rho(\widetilde{\mathcal{M}}(\mathbf{S})) < 1$, which excludes the existence of any equilibrium other than $\mathbf{E}_0$, the trivial one.

We shall now consider the stability of $\mathbf{E}_0$ in Γ. Since $\widetilde{\mathcal{M}}^0$ is nonnegative and irreducible, it has a strictly positive left eigenvector $(\omega_1, \omega_2, \ldots, \omega_n)$ corresponding to the eigenvalue $\rho(\widetilde{\mathcal{M}}^0)$, that is

$$(\omega_1, \omega_2, \ldots, \omega_n)\widetilde{\mathcal{M}}^0 = \rho(\widetilde{\mathcal{M}}^0)(\omega_1, \omega_2, \ldots, \omega_n).$$

We thereby construct the following Lyapunov functional

$$W_1(t) = \sum_{i=1}^{n} \frac{\omega_i}{\mu_i + \alpha_i} I_i.$$

The derivative of W_1 along the solutions of (3.1) is then given by

$$\begin{aligned}
\frac{dW_1(t)}{dt} &= \sum_{i=1}^{n} \frac{\omega_i}{\mu_i + \alpha_i} \left(\sum_{j=1}^{n} \beta_{ij} S_i g_{ij}(I_j) - (\mu_i + \alpha_i) I_i \right) \\
&\leq \sum_{i=1}^{n} \omega_i \left(\sum_{j=1}^{n} \frac{\beta_{ij} S_i^0 C_{ij}}{\mu_i + \alpha_i} I_j \right) - \sum_{i=1}^{n} \omega_i I_i \\
&\leq (\omega_1, \omega_2, \ldots, \omega_n)[\widetilde{\mathcal{M}}^0 \mathbf{I} - \mathbf{I}] \\
&= (\rho(\widetilde{\mathcal{M}}^0) - 1)(\omega_1, \omega_2, \ldots, \omega_n)\mathbf{I} \\
&\leq 0.
\end{aligned}$$

If $\mathcal{R}_0 < 1$, then $\frac{dW_1(t)}{dt} = 0$ if and only if $\mathbf{I} = \mathbf{0}$. If $\mathcal{R}_0 = 1$, then necessarily

$$\sum_{j=1}^{n} \frac{\beta_{ij} S_i C_{ij}}{\mu_i + \alpha_i} I_j = I_i, \quad 1 \leq i \leq n,$$

that is,

$$\widetilde{\mathcal{M}}(\mathbf{S})\mathbf{I} = \mathbf{I}.$$

As above, this implies that $\mathbf{S} = \mathbf{S}^0$. Since $\{\mathbf{E}_0\}$ is the largest invariant set in

$$\left\{ (S_1, I_1, \ldots, S_n, I_n) \in \Gamma; \frac{dW_1(t)}{dt} = 0 \right\},$$

we obtain by applying the Lyapunov–LaSalle invariance principle that $\mathbf{E}_0$ is globally asymptotically stable.

(b) If $\mathcal{R}_0 > 1$ and $\mathbf{I} \neq \mathbf{0}$, we then have

$$(\omega_1, \omega_2, \ldots, \omega_n)[\mathcal{M}^0 \mathbf{I} - \mathbf{I}] = (\rho(\mathcal{M}^0) - 1)(\omega_1, \omega_2, \ldots, \omega_n)\mathbf{I} > 0.$$

By a continuity argument, one obtains that $\frac{dW_1(t)}{dt} > 0$ in a vicinity of the disease-free equilibrium $\mathbf{E}_0$, which implies that $\mathbf{E}_0$ is unstable. □

3.4. The existence and stability of the endemic equilibrium

Let us now suppose that $\mathcal{R}_0 > 1$. Using Theorem 8.2.4 of Kuang [25] together with an argument similar to the one employed in the proof of Proposition 3.3 of Li et al. [26], one may prove that the system (3.1) is uniformly persistent.

As seen from Corollary 2.8.8 in Bhatia and Szegö [27] or Theorem D.3 in Smith and Waltman [28], the uniform persistence of the system (3.1) and the uniform boundedness of its solutions in Γ ensure the existence (but not the uniqueness) of a rest point, that is, of an endemic equilibrium $\mathbf{E}^*$,

$$\mathbf{E}^* = (S_1^*, I_1^*, S_2^*, I_2^*, \ldots, S_n^*, I_n^*).$$

Consequently, the following equilibrium relations involving $\mathbf{E}^*$ are satisfied

$$A_i = d_i S_i^* + \sum_{j=1}^{n} \beta_{ij} S_i^* g_{ij}(I_j^*) \tag{3.2}$$

$$\sum_{j=1}^{n} \beta_{ij} S_i^* g_{ij}(I_j^*) = (\mu_i + \alpha_i) I_i^*, \quad 1 \le i \le n. \tag{3.3}$$

We may now focus on proving the global stability of $\mathbf{E}^*$. In particular, this will also imply its uniqueness as a byproduct.

Theorem 3.2. *If $\mathcal{R}_0 > 1$ and*

$$\big(g_{ij}(I_j) - g_{ij}(I_j^*)\big)\left(\frac{g_{ij}(I_j)}{I_j} - \frac{g_{ij}(I_j^*)}{I_j^*}\right) \le 0, \quad 1 \le i, j \le n, \tag{3.4}$$

with equality if and only if $I_j = I_j^*$ for any nonzero g_{ij}, then the endemic equilibrium $\mathbf{E}^*$ is globally asymptotically stable in the interior of Γ.

Proof. Let us construct a Lyapunov functional W_2 by

$$W_2(t) = \sum_{i=1}^{n} c_i V_i(t), \tag{3.5}$$

with

$$V_i = \left(S_i - S_i^* - S_i^* \ln \frac{S_i}{S_i^*}\right) + \left(I_i - I_i^* - I_i^* \ln \frac{I_i}{I_i^*}\right),$$

the choice of the coefficients c_i, $1 \le i \le n$ being deferred until later in the proof. In other words, the Lyapunov functional W_2 is a linear combination of Lyapunov functionals V_i involving only the ith group, the linking between groups being made, as we shall seen later, by the coefficients c_i, $1 \le i \le n$.

Using the first equilibrium relation (3.2), we observe that

$$\begin{aligned}
\frac{dV_i(t)}{dt} &= \left(1 - \frac{S_i^*}{S_i}\right)\left[A_i - d_i S_i - \sum_{j=1}^{n} \beta_{ij} S_i g_{ij}(I_j)\right] \\
&\quad + \left(1 - \frac{I_i^*}{I_i}\right)\left[\sum_{j=1}^{n} \beta_{ij} S_i g_{ij}(I_j) - (\mu_i + \alpha_i) I_i\right] \\
&= \left(1 - \frac{S_i^*}{S_i}\right)\left[d_i S_i^* + \sum_{j=1}^{n} \beta_{ij} S_i^* g_{ij}(I_j^*) - d_i S_i - \sum_{j=1}^{n} \beta_{ij} S_i g_{ij}(I_j)\right]
\end{aligned}$$

$$
\begin{aligned}
&+\left(1-\frac{I_i^*}{I_i}\right)\left[\sum_{j=1}^{n}\beta_{ij}S_i g_{ij}(I_j)-(\mu_i+\alpha_i)I_i\right]\\
&=\left(1-\frac{S_i^*}{S_i}\right)d_i(S_i^*-S_i)+\sum_{j=1}^{n}\beta_{ij}S_i^* g_{ij}(I_j^*)\left(1-\frac{S_i^*}{S_i}\right)+\sum_{j=1}^{n}\beta_{ij}S_i^* g_{ij}(I_j)\\
&\quad-\sum_{j=1}^{n}\beta_{ij}S_i g_{ij}(I_j)\frac{I_i^*}{I_i}-(\mu_i+\alpha_i)I_i+(\mu_i+\alpha_i)I_i^*.
\end{aligned}
$$

By employing the second equilibrium relation (3.3), we deduce that

$$
\begin{aligned}
\frac{dV_i(t)}{dt}&=\left(1-\frac{S_i^*}{S_i}\right)d_i(S_i^*-S_i)\\
&\quad+\sum_{j=1}^{n,*}\beta_{ij}S_i^* g_{ij}(I_j^*)\left(2-\frac{S_i^*}{S_i}+\frac{g_{ij}(I_j)}{g_{ij}(I_j^*)}-\frac{S_i}{S_i^*}\frac{g_{ij}(I_j)}{g_{ij}(I_j^*)}\frac{I_i^*}{I_i}-\frac{I_i}{I_i^*}\right),
\end{aligned}
$$

where the starred sum is taken only for those j between 1 and n for which g_{ij} is not null. Let us define

$$
F:(0,\infty)\to(-\infty,\infty),\quad F(x)=1-x+\ln x.
$$

and observe that

$$
F(x)\le 0,\quad \text{for all } x\in(0,\infty), \tag{3.6}
$$

with equality if and only if $x=1$. It is seen that

$$
\begin{aligned}
&2-\frac{S_i^*}{S_i}+\frac{g_{ij}(I_j)}{g_{ij}(I_j^*)}-\frac{S_i}{S_i^*}\frac{g_{ij}(I_j)}{g_{ij}(I_j^*)}\frac{I_i^*}{I_i}-\frac{I_i}{I_i^*}\\
&=\left(-\frac{I_i}{I_i^*}+\ln\frac{I_i}{I_i^*}+\frac{I_j}{I_j^*}-\ln\frac{I_j}{I_j^*}\right)+\left(1-\frac{S_i^*}{S_i}+\ln\frac{S_i^*}{S_i}\right)\\
&\quad+\left(1-\frac{S_i}{S_i^*}\frac{g_{ij}(I_j)}{g_{ij}(I_j^*)}\frac{I_i^*}{I_i}+\ln\frac{S_i}{S_i^*}\frac{g_{ij}(I_j)}{g_{ij}(I_j^*)}\frac{I_i^*}{I_i}\right)+\left(1-\frac{I_j}{I_j^*}\frac{g_{ij}(I_j^*)}{g_{ij}(I_j)}+\ln\frac{I_j}{I_j^*}\frac{g_{ij}(I_j^*)}{g_{ij}(I_j)}\right)\\
&\quad-1-\frac{I_j}{I_j^*}+\frac{I_j}{I_j^*}\frac{g_{ij}(I_j^*)}{g_{ij}(I_j)}+\frac{g_{ij}(I_j)}{g_{ij}(I_j^*)}\\
&=\left[F\left(\frac{I_i}{I_i^*}\right)-F\left(\frac{I_j}{I_j^*}\right)\right]+F\left(\frac{S_i^*}{S_i}\right)+F\left(\frac{S_i}{S_i^*}\frac{g_{ij}(I_j)}{g_{ij}(I_j^*)}\frac{I_i^*}{I_i}\right)+F\left(\frac{I_j}{I_j^*}\frac{g_{ij}(I_j^*)}{g_{ij}(I_j)}\right)\\
&\quad+\frac{I_j}{g_{ij}(I_j)g_{ij}(I_j^*)}\left(g_{ij}(I_j)-g_{ij}(I_j^*)\right)\left(\frac{g_{ij}(I_j)}{I_j}-\frac{g_{ij}(I_j^*)}{I_j^*}\right).
\end{aligned}
$$

Due to (3.4) and (3.6), one sees that

$$
\frac{dV_i}{dt}\le\left(1-\frac{S_i^*}{S_i}\right)d_i(S_i^*-S_i)+\sum_{j=1}^{n,*}\beta_{ij}S_i^* g_{ij}(I_j^*)\left[F\left(\frac{I_i}{I_i^*}\right)-F\left(\frac{I_j}{I_j^*}\right)\right]
$$

and consequently, accounting for the null g_{ij}'s as well,

$$
\frac{dV_i}{dt}\le\sum_{j=1}^{n}\beta_{ij}S_i^* g_{ij}(I_j^*)\left[F\left(\frac{I_i}{I_i^*}\right)-F\left(\frac{I_j}{I_j^*}\right)\right].
$$

It follows that

$$\frac{dW_2(t)}{dt} \leq \sum_{i=1}^{n} c_i \left\{ \sum_{j=1}^{n} \beta_{ij} S_i^* g_{ij}(I_j^*) \left[F\left(\frac{I_i}{I_i^*}\right) - F\left(\frac{I_j}{I_j^*}\right) \right] \right\}.$$

Let us now denote

$$A = \left(\beta_{ij} S_i^* g_{ij}(I_j^*)\right)_{\substack{1\leq i\leq n \\ 1\leq j\leq n}}$$

and let $c_i = L_{ii}$, the cofactor of the (i,i)-entry of the Laplacian matrix of the directed graph $G(A)$ associated with A, as described in Section 3.2. Then

$$\sum_{i=1}^{n}\sum_{j=1}^{n} c_i a_{ij} F\left(\frac{I_i}{I_i^*}\right) = \sum_{i=1}^{n}\sum_{j=1}^{n} c_i a_{ij} F\left(\frac{I_j}{I_j^*}\right), \tag{3.7}$$

by Lemma 3.1. Consequently, $\frac{dW_2(t)}{dt} \leq 0$ for all $t \geq 0$. If $\frac{dW_2(t)}{dt} = 0$, then necessarily $S_i = S_i^*$ for all $1 \leq i \leq n$. Also, for a given i, if g_{ij} is nonzero, then $I_j = I_j^*$, and it follows from assumption (iv) that $I_j = I_j^*$ for all $1 \leq j \leq n$ and the equality $\frac{dW_2(t)}{dt} = 0$ holds only at the endemic equilibrium $\mathbf{E}^*$. By Lyapunov–LaSalle principle, $\mathbf{E}^*$ is globally asymptotically stable in the interior of Γ, fact which ensures its uniqueness as an endemic equilibrium and completes the proof. □

Remark 3.1. The fact that condition (3.4) is expressed in terms of the components of the endemic equilibrium $\mathbf{E}^*$, which are not explicitly known, makes it difficult to verify via a post hoc analysis. This condition is, however, a priori satisfied for a large class of functions which are suitable to represent forces of infection, which includes strictly increasing functions which are concave down.

4. Applications

We now use the previously established abstract framework to discuss the global dynamics of model (2.1). Under our settings, $n = 3$, since our model considers of three groups. For notational convenience, let us associate the subscript 1 to the male DFC group, the subscript 2 to the FSW group and the subscript 3 to the male IDU group. Furthermore, assume that the natural death rates and rates of AIDS development, respectively, are as follows.

$$d_1 = d_2 = d_3 = \mu, \quad \alpha_1 = \alpha_2 = \alpha_3 = \alpha.$$

The corresponding functions and parameters defining the process of disease transmission take the following forms

$$\begin{array}{lll} g_{11}(I_1) = 0, & g_{12}(I_2) = I_2, & g_{13}(I_3) = 0, \\ g_{21}(I_1) = I_1, & g_{22}(I_2) = 0, & g_{23}(I_3) = I_3, \\ g_{31}(I_1) = 0, & g_{32}(I_2) = I_2, & g_{33}(I_3) = I_3 \end{array}$$

$$\begin{array}{lll} \beta_{11} = 0, & \beta_{12} = \lambda_{FM}, & \beta_{13} = 0, \\ \beta_{21} = \lambda_{MF}, & \beta_{22} = 0, & \beta_{23} = \lambda_{DF}, \\ \beta_{31} = 0, & \beta_{32} = \lambda_{FD}, & \beta_{33} = \lambda_{DD}. \end{array}$$

Note that conditions (i), (ii) and (iii) are verified for these choices of g_{ij}, condition (3.4) being also verified the nonzero g_{ij}, with

$$\begin{array}{lll} C_{11} = 0, & C_{12} = 1, & C_{13} = 0, \\ C_{21} = 1, & C_{22} = 0, & C_{23} = 1, \\ C_{33} = 0, & C_{32} = 1, & C_{33} = 1, \end{array}$$

which implies that the matrix $(\beta_{ij}C_{ij})$ has the following expression

$$(\beta_{ij}C_{ij}) = \begin{pmatrix} 0 & \lambda_{FM} & 0 \\ \lambda_{MF} & 0 & \lambda_{DF} \\ 0 & \lambda_{FD} & \lambda_{DD} \end{pmatrix},$$

being consequently irreducible. It then follows that condition (iv) is verified as well. One sees that

$$\mathcal{M}^0 = \begin{pmatrix} 0 & \frac{\lambda_{FM}A_M}{\mu(\mu+\alpha)} & 0 \\ \frac{\lambda_{MF}A_F}{\mu(\mu+\alpha)} & 0 & \frac{\lambda_{DF}A_F}{\mu(\mu+\alpha)} \\ 0 & \frac{\lambda_{FD}A_D}{\mu(\mu+\alpha)} & \frac{\lambda_{DD}A_D}{\mu(\mu+\alpha)} \end{pmatrix}, \quad \mathcal{R}_0 = \rho(\mathcal{M}^0).$$

The global dynamics of model (2.1) can be summarised in the following result.

Theorem 4.1.

(a) *If $\mathcal{R}_0 \leq 1$, then the disease-free equilibrium $\mathbf{E}_0$ of (2.1) is the unique equilibrium and it is globally asymptotically stable in Γ.*

(b) *If $\mathcal{R}_0 > 1$, then the disease-free equilibrium $\mathbf{E}_0$ of (2.1) is unstable. There is a unique endemic equilibrium $\mathbf{E}^*$ of (2.1) and this equilibrium is globally asymptotically stable in the interior of Γ.*

Let us now find further more explicit sufficient conditions for the stability of equilibria. To this purpose, let us observe that the characteristic equation of $\mathcal{M}^0$ is given by

$$\lambda^3 + a_2\lambda^2 + a_1\lambda + a_0 = 0, \tag{4.1}$$

where

$$\begin{aligned} a_2 &= -\frac{\lambda_{DD}A_D}{\mu(\mu+\alpha)} \\ a_1 &= -\frac{\lambda_{FM}\lambda_{MF}A_MA_F + \lambda_{FD}\lambda_{DF}A_DA_F}{\mu^2(\mu+\alpha)^2} \\ a_0 &= \frac{\lambda_{FM}\lambda_{MF}\lambda_{DD}A_MA_FA_D}{\mu^3(\mu+\alpha)^3}. \end{aligned} \tag{4.2}$$

Define

$$Q := \frac{3a_1 - a_2^2}{9}, \quad R := \frac{9a_1a_2 - 27a_0 - 2a_2^3}{54}$$

and let $D = Q^3 + R^2$ be discriminant of the cubic equation (4.1). Since the sign of D depends on the concrete values of a_0, a_1, a_2, so does the nature of the roots of (4.1).

Since $a_0 > 0$, one of the three roots is a negative real number. If $D < 0$, the remaining two roots are complex and conjugate, while if $D \geq 0$, then all three roots are real. It seems complicated, though, to find the one which has maximal modulus and give an explicit expression of $\mathcal{R}_0$. To ensure that all of them have modulus < 1, we shall employ Jury conditions, which in this case read as

$$\begin{aligned} a_0 &< 1 \\ 1 + a_2 + a_1 + a_0 &> 0 \\ 1 - a_2 + a_1 - a_0 &> 0 \\ 1 - a_0^2 &> |a_0a_2 - a_1|. \end{aligned} \tag{4.3}$$

We now analyse separately several subsystems of (2.1). The (M-F) subsystem reads as

$$\begin{aligned}\frac{dS_M}{dt} &= A_M - \lambda_{FM} I_F S_M - \mu S_M\\ \frac{dI_M}{dt} &= \lambda_{FM} I_F S_M - (\mu+\alpha) I_M\\ \frac{dS_F}{dt} &= A_F - \lambda_{MF} I_M S_F - \mu S_F\\ \frac{dI_F}{dt} &= \lambda_{MF} I_M S_F - (\mu+\alpha) I_F\end{aligned} \tag{4.4}$$

and has basic reproduction number

$$\mathcal{R}_{MF} = \frac{\sqrt{\lambda_{FM}\lambda_{MF}A_M A_F}}{\mu(\mu+\alpha)}.$$

Similarly, the (F-D) subsystem reads as

$$\begin{aligned}\frac{dS_F}{dt} &= A_F - \lambda_{DF} I_D S_F - \mu S_F\\ \frac{dI_F}{dt} &= \lambda_{DF} I_D S_F - (\mu+\alpha) I_F\\ \frac{dS_D}{dt} &= A_D - \lambda_{FD} I_F S_D - \lambda_{DD} I_D S_D - \mu S_D\\ \frac{dI_D}{dt} &= \lambda_{FD} I_F S_D + \lambda_{DD} I_D S_D - (\mu+\alpha) I_D\end{aligned} \tag{4.5}$$

and has basic reproduction number

$$\mathcal{R}_{FD} = \frac{\lambda_{DD}A_D + \sqrt{(\lambda_{DD}A_D)^2 + 4\lambda_{FD}\lambda_{DF}A_D A_F}}{2\mu(\mu+\alpha)}.$$

The (M-D) subsystem decouples, reading as

$$\begin{aligned}\frac{dS_M}{dt} &= A_M - \mu S_M\\ \frac{dI_M}{dt} &= -(\mu+\alpha) I_M\\ \frac{dS_D}{dt} &= A_D - \lambda_{DD} I_D S_D - \mu S_D\\ \frac{dI_D}{dt} &= \lambda_{DD} I_D S_D - (\mu+\alpha) I_D,\end{aligned} \tag{4.6}$$

and having the basic reproduction number

$$\mathcal{R}_D = \frac{\lambda_{DD}A_D}{\mu(\mu+\alpha)}.$$

Let us denote

$$P = \frac{\lambda_{FD}\lambda_{DF}A_D A_F}{\mu^2(\mu+\alpha)^2}$$

and observe that, in fact,

$$\mathcal{R}_{FD} = \frac{\mathcal{R}_D + \sqrt{\mathcal{R}_D^2 + 4P}}{2}, \quad \mathcal{R}_D \le \mathcal{R}_{FD}. \tag{4.7}$$

Using the "partial" reproduction numbers $\mathcal{R}_{FD}, \mathcal{R}_D$, and $\mathcal{R}_{MF}$, one may then formulate the following stability result.

Theorem 4.2.

(a) *If* $\mathcal{R}_{MF} < 1$, $\mathcal{R}_D < 1$ *and*

$$(1 - \mathcal{R}_D)(1 - \mathcal{R}_{MF}^2) > P, \tag{4.8}$$

then the disease-free equilibrium $\mathbf{E}_0$ *of* (2.1) *is globally asymptotically stable in the interior of* Γ.

(b) *If* $\mathcal{R}_{MF} > 1$ *and* $\mathcal{R}_D > 1$, *then the unique endemic equilibrium* $\mathbf{E}^*$ *of* (2.1) *is globally asymptotically stable in the interior of* Γ.

Proof. (a) We shall verify Jury's conditions. It is seen that

$$a_0 = \mathcal{R}_{MF}^2 \mathcal{R}_D < 1.$$

Also,

$$\begin{aligned} 1 + a_2 + a_1 + a_0 &= 1 - \mathcal{R}_D - \mathcal{R}_{MF}^2 - P + \mathcal{R}_{MF}^2 \mathcal{R}_D \\ &= (1 - \mathcal{R}_D)(1 - \mathcal{R}_{MF}^2) - P > 0, \\ 1 - a_2 + a_1 - a_0 &= 1 - \mathcal{R}_D + \mathcal{R}_{MF}^2 - P - \mathcal{R}_{MF}^2 \mathcal{R}_D \\ &= (1 + \mathcal{R}_D)(1 - \mathcal{R}_{MF}^2) - P > 0. \end{aligned}$$

Further

$$\begin{aligned} |a_0 a_2 - a_1| &= |P + \mathcal{R}_{MF}^2 (1 - \mathcal{R}_D^2)| \\ &= P + \mathcal{R}_{MF}^2 (1 - \mathcal{R}_D^2) \end{aligned}$$

and

$$1 - a_0^2 = 1 - \mathcal{R}_{MF}^4 \mathcal{R}_D^2.$$

It now remains to prove that

$$1 - \mathcal{R}_{MF}^4 \mathcal{R}_D^2 > P + \mathcal{R}_{MF}^2 (1 - \mathcal{R}_D^2).$$

However, this is equivalent to

$$P < (1 + \mathcal{R}_{MF}^2 \mathcal{R}_D^2)(1 - \mathcal{R}_{MF}^2),$$

which follows from (4.8).

(b) Let $\lambda_1, \lambda_2, \lambda_3$ be the characteristic roots of $\mathcal{M}^0$. Since

$$|\lambda_1 \lambda_2 \lambda_3| = |-\mathcal{R}_{MF}^2 \mathcal{R}_D| > 1,$$

it follows that $\mathcal{R}_0 = \rho(\mathcal{M}^0) > 1$. □

Remark 4.1. Note also that, by (4.7), $\mathcal{R}_{FD} < 1 \Leftrightarrow \sqrt{\mathcal{R}_D^2 + 4P} < 2 - \mathcal{R}_D$. If $\mathcal{R}_D < 1$, one further has $\mathcal{R}_{FD} < 1 \Leftrightarrow P < 1 - \mathcal{R}_D$. It follows that, in the conditions given in the first part of the theorem ($\mathcal{R}_{MF} < 1$, $\mathcal{R}_D < 1$), condition (4.8) is strictly stronger than $\mathcal{R}_{FD} < 1$.

Remark 4.2. Our model (2.1) describes, in fact, a particular scenario of the general system (3.1) with $n = 3$ by considering groups of male DFC, FSW, male IDU and illustrating the applicability of Theorem 3.2. In doing so, it perhaps relies on an oversimplification of the routes of HIV transmission. In particular, the model (2.1) assumes that males in the "bridge" group can only be infected through commercial sex and needle/apparatus sharing. It is natural for (2.1) to incorporate other risk groups, such as men who have sex with men (MSM) and bisexual men, and address their roles in HIV transmission.

This would subsequently lead to a higher dimensional model, associated with another scenario of the general system (3.1) with $n \geq 4$, for which a result parallel to Theorem 4.1 could still be formulated, provided that assumptions (i)-(iv) and condition (3.4) are still met. However, the analysis of the model (2.1) via its subsystems, as done in Theorem 4.2, relies on the lower dimensionality and on the particular form of the matrix $\mathcal{M}^0$ and would not hold in a simple form for higher dimensional models unless a high degree of "separation" between the subsystems of (2.1) is assumed.

5. Discussion

In this paper, we propose and investigate the three-group HIV propagation model (2.1) with two interacting high-risk groups: FSW and male IDU, along with a non-high-risk group, male DFC of FSW. The existence and global stability of equilibria are studied in the more comprehensive framework of a general n-group model (3.1) with separable incidences, of which our HIV propagation model (2.1) is a special case.

By using Lyapunov functionals, constructed using the graph theoretic approach of Li and Shuai [22], we determine that the basic reproduction number $\mathcal{R}_0$ is a threshold parameter for the dynamics of (3.1), in the sense that if $\mathcal{R}_0 < 1$, then the disease-free equilibrium $\mathbf{E}_0$ is globally asymptotically stable; while if $\mathcal{R}_0 > 1$, then the global stability properties are transferred to the unique endemic equilibrium $\mathbf{E}^*$ (Theorem 4.1). More explicit sufficient conditions for the global stability of equilibria of the model (2.1) are derived in terms of the partial reproduction numbers $\mathcal{R}_{MF}, \mathcal{R}_{FD}$, and $\mathcal{R}_D$, where $\mathcal{R}_{MF}$ is the basic reproduction number due to infection between FSW and their male drug-free clients, $\mathcal{R}_{FD}$ is the basic reproduction number due to infection between FSW and IDU, and $\mathcal{R}_D$ is the basic reproduction number due to infection among male IDU (Theorem 4.2).

Based on Theorem 4.2 and Remark 4.1, we know that having all 3 partial reproduction numbers being less than 1 does not guarantee stability of DFE. Furthermore, we note that Condition in Theorem 4.2(a) is sharp. That is, for DFE to be globally stable, $\mathcal{R}_{FD}$, the reproduction number for interaction between FSW and IDU, must be more than just less than unity. This result highlights the relative importance of targeting intervention to significantly reduce disease transmission between the 2 high risk groups, especially if the goal of intervention is the eventual eradication of epidemic.

However, Condition in Theorem 4.2(b) is not sharp. That is, it is conceivably for the endemic equilibrium $\mathbf{E}^*$ to be globally stable even if one or more of the partial reproduction numbers are less than unity. In other words, significant outbreak in one high risk group alone could be sufficient to drive up the epidemic in the region. This result again highlights the importance of targeting intervention to reduce disease transmission of all groups, even if the aim of the intervention is merely to mitigate the epidemic.

In conclusion, if the goal of intervention policy is to eradicate the disease, significantly reduction in transmission between the two high-risk groups, FSW and IDU, is needed, in addition to reduction in all other routes of transmission. On the other hand, if the aim of the intervention policy is merely to mitigate the disease, reduction in any route of disease transmission will be useful, albeit reduction in transmissions between the two high-risk groups will be more impactful and hence more cost-effective than others. Similar result on targeting the highest risk groups for most cost-effective intervention was also found in Hsieh and Wang [3].

The current model can be extended in many aspects. Similarly to the direct and indirect classification of FSW in Thailand in the 1990's [2–4], recent studies on FSW in China have focused on classification of different tiers of venues (e.g., [29]). High-tier venues include hotels, night clubs, star-ranked hotels with spas/saunas, karaoke clubs, dance halls, and pubs; by middle-tier venues are massage parlours, beauty salons, spas/saunas, leisure centres, and tea houses; while examples of low-tier venues are small hairdressing salons, street walkers, restaurants, temporary sublets, foot massage, unranked hostels, small hotels and small pubs. It has been established that high and middle-tier FSW have a significantly lower risk of HIV infection than lower-tier FSW [29]. Therefore, it could be meaningful to assign different contact or disease transmission rates for FSW depending on their venues of work. One may also consider steady partners of FSW as a separate bridge group, and the male customers into two groups by their levels of frequency to use commercial sex, on the lines of [4], since the n-group model (3.1) makes it possible to add further population groups to our three-group model (2.1), as long as separable incidences are employed.

Different incidence terms may also be employed for the different transmission modes of the model, which would represent another meaningful extension of the current results, in view of the approach of Li and Shuai [22] towards the construction of Lyapunov functionals employed here covers only the situation in which all incidences used in the model are separable incidences. One could opt for frequency-dependent incidence due to the higher level of prevalence often observed among high-risk groups, such as FSW and IDU, when compared with the general population. It follows that one would expect sexual transmission through commercial sex and needle/apparatus sharing to be more likely dependent on how frequently the contact are made with susceptible persons, rather than how many susceptible persons there are. However, the use of standard incidence terms to characterise some of the transmission modes in our model would render the framework constructed in Section 3 inapplicable, and provides new challenge for future work.

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