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MODELING AND DYNAMICS OF HIV TRANSMISSION AMONG HIGH-RISK GROUPS IN GUANGZHOU CITY, CHINA*

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Abstract In this paper, a multicompartmental model is formulated to study how HIV is transmitted among different HIV high-risk groups, including MSM (men who have sex with men), FRs (foreigner residents), FSWs (female sex workers), and IDUs (injection drug users). The explicit expression for the basic reproduction number is obtained via the next generation matrix approach. We show that the disease free equilibrium is locally as well as globally asymptotically stable (the disease goes to extinction) when the basic reproduction number is less than unity, and the disease is always present when the basic reproduction number is larger than unity. As an illustration of our theoretical results, we conduct numerical simulations. We also conduct a case study where model parameters are estimated from the demographic and epidemiological data from Guangzhou. Using the parameter estimates, we predict the HIV/AIDS trend for each high-risk group. Furthermore, our study suggests that reducing the transmission routes of the disease and increasing condom use will be useful for control of HIV transmission.

Keywords Basic reproduction number, HIV high-risk groups, permanence, sensitivity analysis.

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1. Introduction

Human Immunodeficiency Virus (HIV) is a sexually transmitted disease (STD) that causes acquired immunodeficiency syndrome (AIDS). AIDS causes gradual failure of the immune system, leaving victims vulnerable to a variety of infections. Since the discovery of HIV-1 in the early 1980s, the disease has spread in successive waves to most regions around the globe. In the world, about 36.9 million people are infected with HIV, and an estimated 1.1 million people died due to AIDS in 2017 [44]. It is one of the top ten infectious diseases and a leading cause of death in mainland China, as reported by the China Center for Disease Control and Prevention (China, CDC). The cumulative total number of reported HIV infection was 89,067 as of December 2004 [20], a figure that increased to 758, 610 as of December 2017 [28].

On the one hand, the main factors by which HIV/AIDS has been spreading in China include needle-sharing among intravenous drug users and unsafe sexual

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activities in the past decade. By 2002, HIV was present amongst IDUs in all Chinese inland provinces. It is believed that IDUs may have been the core source for all later sub-epidemics in China [42]. However, in recent years, the largest number of HIV/AIDS cases have been among MSM, followed by FSWs and IDUs [12]. The new HIV/AIDs infected cases among MSM and IDUs are respective 34,358 (25.5% of new total HIV/AIDS cases) and 4,296 (3.2% of new total HIV/AIDS cases) in 2017, China [28]. Hence, the Chinese government has strengthened its intervention and control efforts to MSM group, developed national working policies and guidelines on HIV/AIDS prevention and control among MSM, and convened national technical workshops on comprehensive HIV/AIDS prevention interventions among MSM.

On the other hand, statistics in recent years indicate that there are three main reasons for the continued surge in the number of foreigners living with HIV in China: the removal of restrictions on the entry of AIDS patients by the Chinese government. Unlike other parts of China, Guangzhou benefits from the reform and opening policy and has a close linkage with the rest of the world. Guangzhou ranks the third highest in the nation for the number of FRs, increasing from 19,000 in 2008 to 49,798 in 2017 [43]. The number of HIV infection cases among FRs increased significantly [17] after the Chinese government canceled entry requirements for restrictions on AIDS and HIV infected foreigners in 2010 [4, 45]. Abroad HIV/AIDS cases found in Guangzhou are mainly young and middle-aged people, and sexual behavior is the primary route of HIV infection in these FRs HIV/AIDS cases [1]. Above all, we conclude that FRs play a crucial role in HIV transmission in Guangzhou. Based on these data and comparisons, HIV high-risk groups (groups of people with high-risk to infect with HIV) in Guangzhou include FRs, MSM, FSWs, and IDUs.

In recent time, mathematical models have been applied to the study of the spread of HIV/AIDS in the high-risk group. Hethcote etc [18] (1992) first formulated a model for MSM, studied biosynthetic properties and estimated parameters and incidences for MSM in San Francisco. Tan etc [22] (1993) proposed a stochastic model to present HIV/AIDS epidemic and infection in MSM. Tan etc [33] (1995) considered MSM groups, studied the effects of the randomness of risk factors on the HIV epidemic. Soon afterward, Tan etc [34] (1996) formulated a specific model for the HIV epidemic and the effects of age and race on MSM groups. In addition, a state-space model of the HIV epidemic in MSM was established in [35]. Zindoga etc [51] (2009) established a model with MSM (homosexuals and bisexuals) HIV infection. The model in this paper considered the emigration influence on the spread of HIV in MSM. Xu etc [46] (2011) presented a model concerning sexual transmission of HIV/AIDS among FSWs and MSM in Jiangsu province, China. Sun etc [29] (2013) proposed a mathematical model for HIV/AIDS epidemics among MSM in China that consider antiviral therapy. Zhang etc [52] (2016) developed a mathematical model on the transmission dynamics of HIV among MSM, FSWs, and IDUs groups. Li etc [23] (2019) established a mathematical model to study the persistence of HIV-1 spreading in MSM population in China. However the existing mathematical models only considered HIV transmission among the above three groups. How HIV transmission among high-risk groups when FRs group is presented remains unclear and is the subject of this study.

Based on the simplified flow chart of potential bridges for HIV infection to MSM, Guangzhou, China (Figure 1 in [20]), we give a more realistic and reasonable transmission flow diagram in Figure 1. The purpose of this study is to extend the existing mathematical models by including FRs group in HIV high-risk groups.

Moreover, we consider the interconnection by sexual behaviors among these groups. Hence, the total population will be divided into four groups: (1) MSM, (2) FRs, (3) FSWs, and (4) IDUs. Then sensitivity analysis of the basic reproduction numbers will be carried out in terms of the model parameters, from which effective control measures will be discussed for the spread of HIV among HIV high-risk groups in Guangzhou, China.

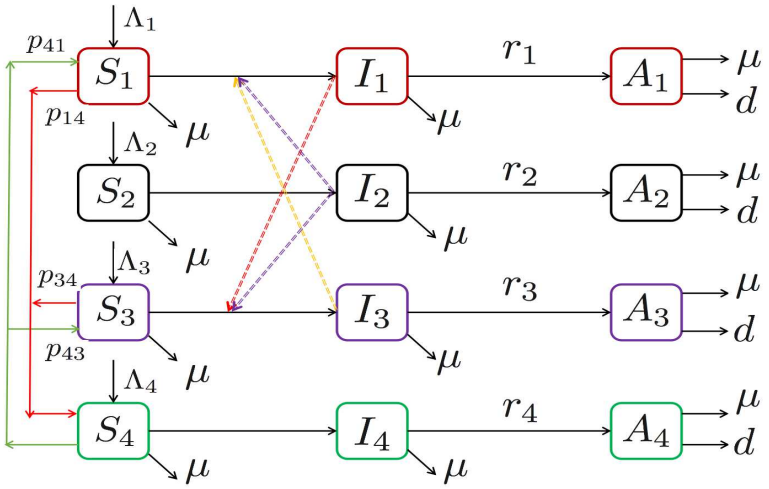


Figure 1. The flow diagram of model (2.1), the incidence rate from I_1 to S_3 is $\frac{\alpha_{13}I_1}{N_3}$ (red imaginary line), the incidence rate from I_3 to S_1 is $\frac{\alpha_{31}I_3}{N_1}$ (orange imaginary line), the incidence rates from I_2 to S_1, S_3 are $\frac{\alpha_{21}I_2}{N_1}$ and $\frac{\alpha_{23}I_2}{N_3}$ (purple imaginary line). Red solid lines show the moving rate from S_1, S_3 to S_4 and green solid lines show the moving rate from S_4 to other susceptible groups.

The paper is structured as follows. The model is formulated in Section 2. Theoretic analyses of model (2.1) (the basic reproduction ratio and the global asymptotical stability of disease-free equilibrium) are presented in Section 3 and Section 4. In Section 5, we prove the permanence of the disease. Numerical simulations to extend found analytical results are provided in Section 6. In Section 7, a case study is applied to Guangzhou, China. Conclusion and discussion round up the paper in Section 8.

2. Model development

The model sub-divides the total population at time t into the following groups: susceptible groups $S_i(t)$, infected groups $I_i(t)$, AIDS groups (the people in this group refer to first instance of AIDS) $A_i(t)$ $(i = 1,\ 2,\ 3,\ 4)$, where 1,2,3, and 4 represent MSM, FRs, FSWs, and IDUs groups, respectively. In order to construct the corresponding model, we make the following assumptions:

(1) The susceptible among MSM, FSWs, and IDUs are classified by their behaviors. That is, if a susceptible individual drugs with others by sharing needles at time t, then he/she belongs to IDUs group $S_4(t)$;

(2) Since bisexuality is difficult to distinguish among MSM, we assume that MSM group includes bisexuality in our model, which means that there is sexual transmission between MSM and FSWs;

(3) AIDS cases in the MSM and FSWs are assumed to be sexually inactive, and so do not influence the dynamics of the virus thus are not distinguished;

(4) We do not consider sexual transmission of the disease between IDUs and other three groups, and just consider the infection of FRs to other groups in our model. Since MSM, IDUs, and FSWs in this paper respective refer to the domestic men who have sex with men, injection drug users, and female sex workers, we ignore the movement between FRs and IDUs.

Flow diagram of HIV infected among high-risk groups is demonstrated in Figure 1. The model is expressed through the following system of ordinary differential equations:

$$\begin{cases} S_1'(t) =\Lambda_1 + p_{41}S_4 - p_{14}S_1 - (\alpha_{11}I_1 + \alpha_{21}I_2 + \alpha_{31}I_3)\dfrac{S_1}{N_1} - \mu S_1, \\ I_1'(t) =(\alpha_{11}I_1 + \alpha_{21}I_2 + \alpha_{31}I_3)\dfrac{S_1}{N_1} - r_1I_1 - \mu I_1, \\ A_1'(t) =r_1I_1 - \mu A_1 - \delta A_1, \\ S_2'(t) =\Lambda_2 - \dfrac{\alpha_{22}(I_2 + k_2A_2)S_2}{N_2} - \mu S_2, \\ I_2'(t) =\dfrac{\alpha_{22}(I_2 + k_2A_2)S_2}{N_2} - r_2I_2 - \mu I_2, \\ A_2'(t) =r_2I_2 - \mu A_2 - \delta A_2, \\ S_3'(t) =\Lambda_3 + p_{43}S_4 - p_{34}S_3 - (\alpha_{13}I_1 + \alpha_{23}I_2)\dfrac{S_3}{N_3} - \mu S_3, \\ I_3'(t) =(\alpha_{13}I_1 + \alpha_{23}I_2)\dfrac{S_3}{N_3} - r_3I_3 - \mu I_3, \\ A_3'(t) =r_3I_3 - \mu A_3 - \delta A_3, \\ S_4'(t) =\Lambda_4 + p_{34}S_3 + p_{14}S_1 - (p_{41} + p_{43})S_4 - \dfrac{\alpha_{44}(I_4 + k_4A_4)S_4}{N_4} - \mu S_4, \\ I_4'(t) =\dfrac{\alpha_{44}(I_4 + k_4A_4)S_4}{N_4} - r_4I_4 - \mu I_4, \\ A_4'(t) =r_4I_4 - \mu A_4 - \delta A_4, \end{cases} \tag{2.1}$$

where $N_i = S_i + I_i + A_i$ $(i = 1,\ 2,\ 3,\ 4)$ represents the total number of the ith group. Some parameters are given by the similar form to [52] as follows

$$\alpha_{11} = (1 - d_{11})c_{11},\ \alpha_{21} = (1 - d_{21})c_{21},\ \alpha_{13} = (1 - d_{13})c_{13},$$
$$\alpha_{22} = (1 - d_{22})c_{22},\ \alpha_{23} = (1 - d_{23})c_{23},\ \alpha_{44} = (1 - d_{44})c_{44}, \alpha_{31} = (1 - d_{31})c_{31},$$

where $d_{11}, d_{13}, d_{21}, d_{22}, d_{23}, d_{31}, d_{44}, c_{11}, c_{13}, c_{21}, c_{22}, c_{23}, c_{31}, c_{44}$,
and r_i, Λ_i $(i = 1,\ 2,\ 3,\ 4)$ are described in Table 8 detailedly, μ and δ represent natural death rate and AIDS related death rate, respectively.

3. The basic reproduction number R_0

The objective of this section is to calculate the basic reproduction number of system (2.1). To this purpose, we first denote the disease free equilibrium E_0=(S_1^0, 0, 0, S_2^0, 0, 0, S_3^0, 0, 0, S_4^0, 0, 0), then substituting E_0 into system (2.1), yields

$$\begin{cases} \Lambda_1 + p_{41}S_4^0 - p_{14}S_1^0 - \mu S_1^0 = 0, \\ \Lambda_3 + p_{43}S_4^0 - p_{34}S_3^0 - \mu S_3^0 = 0, \\ \Lambda_2 - \mu S_2^0 = 0, \\ \Lambda_4 + p_{14}S_1^0 + p_{34}S_3^0 - (p_{41} + p_{43})S_4^0 = 0. \end{cases}$$

It follows that

$$S_1^0 = \frac{\Lambda_1 + p_{41}S_4^0}{\mu + p_{14}},\ S_3^0 = \frac{\Lambda_3 + p_{43}S_4^0}{\mu + p_{34}},\ S_2^0 = \frac{\Lambda_2}{\mu},\ S_4^0 = \frac{\Lambda_4 + \frac{p_{14}\Lambda_1}{\mu+p_{14}} + \frac{p_{34}\Lambda_3}{\mu+p_{34}}}{\mu + \frac{p_{41}\mu}{\mu+p_{14}} + \frac{p_{43}\mu}{\mu+p_{34}}}.$$

For convenience, we denote $\bar{E}_0$=($I_1(t)$, $I_2(t)$, $I_3(t)$, $I_4(t)$, $A_1(t)$, $A_2(t)$, $A_3(t)$, $A_4(t)$). According to the concepts of the next generation matrix [5,53], $\mathscr{F}$ and $\mathscr{V}$ are given as follows

$$\mathscr{F} = \begin{pmatrix} (\alpha_{11}I_1 + \alpha_{21}I_2 + \alpha_{31}I_3)\frac{S_1}{N_1} \\ \alpha_{22}(I_2 + k_2A_2)\frac{S_2}{N_2} \\ (\alpha_{13}I_1 + \alpha_{23}I_2)\frac{S_3}{N_3} \\ \alpha_{44}(I_4 + k_4A_4)\frac{S_4}{N_4} \\ 0 \\ 0 \\ 0 \\ 0 \end{pmatrix}, \mathscr{V} = \begin{pmatrix} (r_1 + \mu)I_1 \\ (r_2 + \mu)I_2 \\ (r_3 + \mu)I_3 \\ (r_4 + \mu)I_4 \\ (\mu + \delta)A_1 - r_1I_1 \\ (\mu + \delta)A_2 - r_2I_2 \\ (\mu + \delta)A_3 - r_3I_3 \\ (\mu + \delta)A_4 - r_4I_4 \end{pmatrix}.$$

Then the matrices $\mathbf{F}$ (related to new infections) and $\mathbf{V}$ (related to transfers between classes) are given by

$$\mathbf{F} = \begin{pmatrix} F_1 & F_2 \\ 0 & 0 \end{pmatrix}, \mathbf{V} = \begin{pmatrix} B & 0 \\ C & U \end{pmatrix},$$

where

$$F_1 = \begin{pmatrix} \alpha_{11} & \alpha_{21} & \alpha_{31} & 0 \\ 0 & \alpha_{22} & 0 & 0 \\ \alpha_{13} & \alpha_{23} & 0 & 0 \\ 0 & 0 & 0 & \alpha_{44}, \end{pmatrix}, F_2 = \begin{pmatrix} 0 & 0 & 0 & 0 \\ 0 & \alpha_{22}k_2 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & \alpha_{44}k_4 \end{pmatrix},$$

$U = diag\{\mu + \delta, \mu + \delta, \mu + \delta, \mu + \delta\}$, $B = diag\{r_1 + \mu, r_2 + \mu, r_3 + \mu, r_4 + \mu\}$ and $C = diag\{-r_1, -r_2, -r_3, -r_4\}$.

Hence, the next generation matrix is

$$FV^{-1} = \begin{pmatrix} F_1B^{-1} - F_2U^{-1}CB^{-1} & F_2U^{-1} \\ 0 & 0 \end{pmatrix},$$

its eigenvalues satisfy the following characteristic equation

$$(\lambda - \alpha_{22}f_2)(\lambda - \alpha_{44}f_4)\left(\lambda^2 - \alpha_{11}f_1\lambda - \alpha_{13}\alpha_{31}f_1f_3\right) = 0, \tag{3.1}$$

where $f_1 = \frac{1}{r_1+\mu}$, $f_3 = \frac{1}{r_3+\mu}$, $f_2 = \frac{(\mu+\delta+r_2k_2)}{(r_2+\mu)(\mu+\delta)}$, $f_4 = \frac{(\mu+\delta+r_4k_4)}{(r_4+\mu)(\mu+\delta)}$.

Thus, we obtain that the basic reproduction number R_0 as follows

$$R_0 = R_0 = \max\left\{R_0^1, R_0^2, R_0^3\right\},$$

where R_0^1, R_0^2 respective represent the basic reproduction number of PRA and IDUs, R_0^3 represents the basic reproduction number of MSM and FSWs, and they can be expressed as follows

$$R_0^1 = \alpha_{22}f_2,\ R_0^2 = \alpha_{44}f_4,\ R_0^3 = \frac{1}{2}\left(\alpha_{11}f_1 + \sqrt{(\alpha_{11}f_1)^2 + 4\alpha_{13}\alpha_{31}f_1f_3}\right).$$

For convenience analysis later, we denote

$$R_0^* = R_0^M + R_0^{MF},\ R_0^M = \alpha_{11}f_1,\ R_0^{MF} = \alpha_{13}\alpha_{31}f_1f_3.$$

Then we obtain that

$$(R_0^3)^2 - R_0^MR_0^3 - R_0^{MF} = 0. \tag{3.2}$$

Next, we present the relationship between R_0^3, R_0^* and 1, respectively. Define $y(x) = x^2 - R_0^Mx - R_0^{MF}$. It follows from (3.2) that $y(R_0^3) = 0$ and $y(1) = 1 - R_0^*$. One obtains the following equivalence relations:

$$R_0^3 > 1 \Leftrightarrow R_0^* > 1,\ R_0^3 = 1 \Leftrightarrow R_0^* = 1,\ R_0^3 < 1 \Leftrightarrow R_0^* < 1.$$

4. Global stability of E_0

The main focus of this section is to analyze the local and global behavior of the disease-free equilibrium E_0 of system (2.1). First, we state and prove the result about local asymptotic stability for the disease-free equilibrium E_0.

The 12×12 Jacobian matrix $J(E_0)$ can be presented as follows

$$J(E_0) = \begin{pmatrix} K_{4\times4} & * \\ 0 & Q_{8\times8} \end{pmatrix}, \tag{4.1}$$

where

$$K_{4\times4} = \begin{pmatrix} -(p_{14}+\mu) & 0 & 0 & p_{41} \\ 0 & -\mu & 0 & 0 \\ 0 & 0 & -(p_{34}+\mu) & p_{43} \\ p_{14} & 0 & p_{34} & -(p_{41}+p_{43}+\mu) \end{pmatrix}, \tag{4.2}$$

$$Q_{8\times 8} = \begin{pmatrix} H_{2\times 2} & 0_{2\times 4} & 0_{2\times 2} \\ * & M_{4\times 4} & 0_{4\times 2} \\ * & * & G_{2\times 2} \end{pmatrix}, \tag{4.3}$$

where

$$H_{2\times 2} = \begin{pmatrix} \alpha_{22} - (r_2 + \mu) & \alpha_{22}k_2 \\ r_2 & -(\mu + \delta) \end{pmatrix}, G_{2\times 2} = \begin{pmatrix} \alpha_{44} - (r_4 + \mu) & \alpha_{44}k_4 \\ r_4 & -(\mu + \delta) \end{pmatrix}, \tag{4.4}$$

$M_{4\times 4}$ is expressed as follows:

$$\begin{pmatrix} \alpha_{11} - (r_1 + \mu) & 0 & \alpha_{31} & 0 \\ r_1 & -(\mu + \delta) & 0 & 0 \\ \alpha_{13} & 0 & -(r_3 + \mu) & 0 \\ 0 & 0 & r_3 & -(\mu + \delta) \end{pmatrix}. \tag{4.5}$$

The eigenvalues of matrix (4.1) are determined by those of matrices (4.2), (4.4), and (4.5), it suffices to prove that all eigenvalues of the above matrices must have negative real parts when $R_0 = \max\left\{R_0^1,\ R_0^2,\ R_0^3\right\} < 1$. To achieve this goal, we have to show the following Lemma

Lemma 4.1. *The following claims hold for* (4.1)–(4.5)
(1) *All the eigenvalues of* $K_{4\times 4}$ *have negative real parts;*
(2) *If* $R_0^1 < 1$, *then all the eigenvalues of* $H_{2\times 2}$ *have negative real parts. Moreover, the matrix* $C_{2\times 2}$ *has at least one eigenvalue with positive real part if* $R_0^1 > 1$*;*
(3) *If* $R_0^2 < 1$, *then all the eigenvalues of* $F_{2\times 2}$ *have negative real parts. Moreover, the matrix* $G_{2\times 2}$ *has at least one eigenvalue with positive real part if* $R_0^2 > 1$*;*
(4) *If* $R_0^* < 1$, *then all the eigenvalues of* $M_{4\times 4}$ *have negative real parts. Moreover, the matrix* $E_{4\times 4}$ *has at least one eigenvalue with positive real part if* $R_0^* > 1$.

Proof. Part (1) follows directly from $K_{4\times 4}$ is real column diagonal dominant matrix and all the diagonal entries are negative.

To prove part (2), the characteristic equation of $H_{2\times 2}$ is expressed as $\lambda^2 - a_1\lambda - a_2 = 0$, where $a_1 = \alpha_{22} - (r_2 + \mu) - (\mu + \delta)$, $a_2 = (\alpha_{22} - \mu - r_2)(\mu + \delta) + r_2k_2\alpha_{22}$. In fact, the inequalities $\alpha_{22} < r_2 + \mu$ and $(\alpha_{22} - r_2 - \mu)(\mu + \delta) + \alpha_{22}k_2r_2 < 0$ hold when $R_0^1 < 1$. Then we have $a_1 < 0, a_2 < 0$, which implies all of eigenvalues of $H_{2\times 2}$ have negative real parts. On the contrary, the matrix $H_{2\times 2}$ has at least one eigenvalue with positive real part as $R_0^1 > 1$, which proves part (2).

For part (3), the proof of this result is quite similar to that given earlier for part (2) and so is omitted.

Finally, we have to show that part (4). For this purpose, the characteristic equation of $M_{4\times 4}$ is given by

$$(\lambda + \mu + \delta)^2(\lambda^2 - a_3\lambda - a_4) = 0, \tag{4.6}$$

where $a_3 = \alpha_{11} - (r_1 + \mu) - (\mu + r_3)$, $a_4 = (\alpha_{11} - \mu - r_1)(\mu + r_3) + \alpha_{13}\alpha_{31}$. As $R_0^3 < 1$, we claim that each root of (4.6) has negative real part. To achieve this goal, it suffices

to show $a_3 < 0$, $a_4 < 0$. In fact, when $R_0^* < 1$, it implies that $a_3 < 0$. For a_4, after simple calculation we can obtain $a_4 = (\mu+r_3)(\mu+r_1)\left(\frac{\alpha_{11}}{\mu+r_1} + \frac{\alpha_{13}\alpha_{31}}{(\mu+r_3)(\mu+r_1)} - 1\right) = (\mu + r_3)(\mu + r_1)(R_0^* - 1)$, which implies $a_4 < 0$ when $R_0^* < 1$ (i.e. $R_0^3 < 1$). Therefore, all eigenvalues of (4.6) have negative real parts. If $R_0^* > 1$ i.e.,$R_0^3 > 1$ then $a_3 > 0$, which implies (4.6) has at least one eigenvalue with positive real part and part (4) follows. the proof of Lemma 4.1 is complete. □

Using these analysis together with Lemma 4.1 we obtain that the following theorem.

Theorem 4.1. *The disease-free equilibrium E_0 is locally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$.*

To complete the proof of the global stability of E_0, we first show the following Theorem.

Theorem 4.2. *For the solution $(S_1, I_1, A_1, S_2, I_2, A_2, S_3, I_3, A_3, S_4, I_4, A_4)$ of system (2.1) with nonnegative initial values, then there exists a constant $P > 0$ such that $S_i(t) \le P$, $I_i(t) \le P$, $A_i(t) \le P$ $(i = 1, 2, 3, 4)$ for large enough t.*

Proof. Let $\mathscr{L}(t) = \sum_{i=1}^{4} N_i(t)$, then $\mathscr{L}'(t)|_{(2.1)} \le \sum_{i=1}^{4} \Lambda_i - \mu\mathscr{L}(t)$. According to the standard comparison principle, there exists $t_1 > 0$ such that for any $\epsilon > 0$, $\mathscr{L}(t)|_{(2.1)} \le \frac{(\Lambda_1+\Lambda_2+\Lambda_3+\Lambda_4)}{\mu} + \epsilon := P$ as $t \geqslant t_1$. Then we have $S_i(t) \le P$, $I_i(t) \le P$, $A_i(t) \le P$ $(i = 1, 2, 3, 4)$ for $t \geqslant t_1$, which completes the proof. □

Theorem 4.3. *The disease-free equilibrium E_0 is globally attractive when $R_0 < 1$.*

Proof. We divide our proof in three steps. As to continuous and bounded function $l(t)$, we define

$$l^\infty := \limsup_{t\to\infty} l(t),\ l_\infty := \liminf_{t\to\infty} l(t).$$

Based on Theorem 4.2, we obtain that any solution of system (2.1), $I_i(t), A_i(t)$, $(i = 1, 2, 3, 4)$ satisfies

$$0 \leqslant I_{i\infty} \leqslant I_i^\infty < \infty,\ 0 \leqslant A_{i\infty} \leqslant A_i^\infty < \infty. \tag{4.7}$$

By the fluctuation lemma (Lemma 4.2 in [16]), we know there exists a sequence $\{t_n\}$, with $t_n \to \infty$ $(n \to \infty)$, such that

$$\begin{aligned} &I_i(t_n) \to I_i^\infty,\ I_i' \to 0 \text{ as } n \to \infty,\\ &A_i(t_n) \to A_i^\infty,\ A_i' \to 0 \text{ as } n \to \infty. \end{aligned} \tag{4.8}$$

Substituting the sequence $\{t_n\}$ into the infected compartments of system (2.1) and taking superior limit for both sides of those equations, we obtain that

$$I_1^\infty(r_1 + \mu) \leqslant (\alpha_{11}I_1^\infty + \alpha_{21}I_2^\infty + \alpha_{31}I_3^\infty),\ A_1^\infty(\delta + \mu) \leqslant r_1 I_1^\infty, \tag{4.9}$$

$$I_2^\infty(r_2 + \mu) \leqslant \alpha_{22}(I_2^\infty + k_2 A_2^\infty),\ A_2^\infty(\delta + \mu) \leqslant r_2 I_2^\infty, \tag{4.10}$$

$$I_3^\infty(r_3 + \mu) \leqslant (\alpha_{13}I_1^\infty + \alpha_{23}I_2^\infty),\ A_3^\infty(\mu + \delta) \leqslant r_3 I_3^\infty, \tag{4.11}$$

$$I_4^\infty(r_4 + \mu) \leqslant \alpha_{44}(I_4^\infty + k_4 A_4^\infty),\ A_4^\infty(\delta + \mu) \leqslant r_4 I_4^\infty. \tag{4.12}$$

It follows from the inequality (4.10) that $I_2^\infty \leqslant \left(\frac{\alpha_{22}}{r_2+\mu} + \frac{k_2 r_2 \alpha_{22}}{(r_2+\mu)(\delta+\mu)}\right) I_2^\infty = R_0^1 I_2^\infty$. When $R_0^1 < 1$, we have the inequality (and contradiction) $I_2^\infty \leqslant I_2^\infty$, unless $I_2^\infty = 0$,

that is to say that $\lim_{t\to\infty} I_2(t) = 0$. Thus, we obtain $\lim_{t\to\infty} A_2(t) = 0$ from the inequality (4.10). The same proof still goes for $\lim_{t\to\infty} A_4(t) = \lim_{t\to\infty} I_4(t) = 0$ when $R_0^2 < 1$.

The next thing to do this proof is show that if $R_0^* < 1$, then $I_j(t) \to 0,\ A_j(t) \to 0,\ j = 1, 3$. We assume $I_1^\infty > 0$, then it follows from inequalities (4.9), (4.11) that

$$\frac{1}{I_1^\infty} \geqslant \frac{r_1 + \mu}{\alpha_{11} I_1^\infty + \alpha_{31} I_3^\infty} \geqslant \frac{1}{(\alpha_{11} f_1 + \alpha_{31}\alpha_{13} f_1 f_3) I_1^\infty} = \frac{1}{R_0^* I_1^\infty}. \tag{4.13}$$

The inequality (4.13) holds for $R_0^* > 1$, which contradicts with $R_0^* < 1$. Hence, $I_1^\infty = 0$, it follows from inequalities (4.9)-(4.12) that $I_3^\infty = 0, A_1^\infty = 0, A_3^\infty = 0$. From the inequality (4.7), we obtain that $\lim_{t\to\infty} I_j(t) = 0,\ \lim_{t\to\infty} A_j(t) = 0,\ j = 1, 3$.

Finally, let $S(t)$=(S_1, S_2, S_3, S_4), it may be concluded that the limit system of (2.1) is $S'(t) = K_{4\times4} S(t)$. It is obvious that the unique positive equilibrium (S_1^0, S_2^0, S_3^0, S_4^0) of the system $S'(t) = K_{4\times4} S(t)$ is globally asymptotically stable, that is, $\lim\limits_{t\to\infty} S_i(t) = S_i^0$ $(i = 1, 2, 3, 4)$. From the comparison principle, we get that the disease-free equilibrium of system (2.1) is global attractivity. This complete the proof of Theorem 4.3. □

Above all, we can summarize what we have proved in Lemma 4.1 and Theorem 4.3 as the following Theorem.

Theorem 4.4. *If $R_0 < 1$, then the disease-free equilibrium E_0 is globally asymptotically stable.*

5. Permanence of the disease

In this section, to show permanence of the disease of system (2.1), we give the following Theroem.

Theorem 5.1. *For the solution $(S_1, I_1, A_1, S_2, I_2, A_2, S_3, I_3, A_3, S_4, I_4, A_4)$ of system (2.1) with initial conditions $S_i(0) \geqslant 0,\ I_i(0) \geqslant 0,\ A_i(0) \geqslant 0$ ($i = 1, 2, 3, 4$) and $I_1(0) + I_2(0) + I_3(0) + I_4(0) > 0$. If $R_0 > 1$, then there exists an $\eta > 0$ such that $\liminf\limits_{t\to\infty}(I_1(t) + I_2(t) + I_3(t) + I_4(t)) > \eta$.*

Proof. By the similar arguments as those in the paper [36]. We first introduce some notations which will be used throughout the proof process.

$$\mathbf{Z}(t) = (S_1(t), I_1(t), A_1(t), S_2(t), I_2(t), A_2(t), S_3(t)v, I_3(t), A_3(t), S_4(t), I_4(t), A_4(t)),$$
$$X = \left\{\mathbf{Z}(t) \in \mathbb{R}^{12} : S_i(t) \geqslant 0, I_i(t) \geqslant 0, A_i(t) \geqslant 0, i = 1, 2, 3, 4\right\},$$
$$X_1 = \left\{\mathbf{Z}(t) \in X : I_1(t) + I_2(t) + I_3(t) + I_3(t) > 0\right\},\ X_2 = X \setminus X_1.$$

We will show that system (2.1) is uniformly strong persistent with respect to (X_1, X_2).

It is obvious that X is positively invariant with respect to system (2.1). If $S_i \geqslant 0, I_i \geqslant 0, A_i \geqslant 0$ $(i = 1, 2, 3, 4)$, then $S_i(t) > 0, I_i(t) \geqslant 0$ and $A_i(t) \geqslant 0$ for all $t \geqslant 0$. In fact, the inequality $S_4' \geqslant \Lambda_4 - (p_{41} + p_{43} + \mu)S_4$ implies $S_4(t) > 0$ for all $t > 0$. Similarly, we can show that $S_i(t) > 0$ $(i = 2, 3, 4)$. Let $a = \max\{\mu + r_i\}$ $(i = 1, 2, 3, 4)$. Since $(I_1'(t) + I_2'(t) + I_3'(t) + I_4'(t)) \geqslant -a(I_1(t) + I_2(t) +$

$I_3(t)+I_4(t))$ and $I_1(0)+I_2(0)+I_3(0)+I_4(0)>0$, it follows that $I_1(t)+I_2(t)+I_3(t)+I_4(t)>0$, which implies X_1 is also positive invariant. Furthermore, by Theorem 4.2, there exists a compact set $\mathbb{B}$ in which all solutions of system (2.1) initiated in X will enter and remain forever after, the compactness condition $(C_{4.2})$ in [36] is easily verified for this set $\mathbb{B}$. Denote $M_\partial=\{\mathbf{Z}(0):\mathbf{Z}(t)\in X_2, t\geqslant 0\}$, $N_\partial=\{(S_1,0,0,S_2,0,0,S_3,0,0,S_4,0,0):S_i(t)\geqslant 0,\ i=1,\ 2,\ 3,\ 4\}$. Now we show that

$$M_\partial=N_\partial. \tag{5.1}$$

Suppose that $\mathbf{Z}(0)\in M_\partial$. It suffices to show $A_i(t)=0$ for any $t\geqslant 0$ $(i=1,2,3,4)$, if it is not true, then exists a $t_0>0$ such that one of $A_i(t_0)>0$ $(i=1,2,3,4)$. Without loss of generality, we may assume $A_4(t_0)>0$ then $I_4'(t_0)=\alpha_{44}k_4S_4(t_0)A_4(t_0)/N_4(t_0)>0$, it follows that there exists a small enough $\varrho>0$ such that $I_4(t)>0$ for all $t\in(t_0,t_0+\varrho)$. Thus, $\mathbf{Z}(t)\in X_1$ in $(t_0,t_0+\varrho)$, this leads to contradiction. This proves (5.1).

Denote $\Omega=\bigcup\limits_{\mathbf{Z}(0)\in M_\partial}\Big(\omega(\mathbf{Z}(0)\Big)$, where $\omega\left(\mathbf{Z}(0)\right)$ is the omega limit set of solution to (2.1) starting in $\mathbf{Z}(0)$, restricting (2.1) on M_∂ gives

$$\begin{cases} S_1'(t) =\Lambda_1+p_{41}S_4(t)-p_{14}S_1(t)-\mu S_1(t),\\ S_2'(t) =\Lambda_2-\mu S_2(t),\\ S_3'(t) =\Lambda_3+p_{43}S_4(t)-p_{34}S_3(t)-\mu S_3(t),\\ S_4'(t) =\Lambda_4+p_{14}S_1(t)+p_{34}S_3(t)-(p_{43}+p_{41})S_4(t)-\mu S_4(t). \end{cases} \tag{5.2}$$

System (5.2) has a unique equilibrium $(S_1^0,\ S_2^0,\ S_3^0,\ S_4^0)$. Thus the disease-free equilibrium E_0 is the unique equilibrium of system (2.1) in M_∂, it is easy to see that E^0 is locally asymptotically stable, which implies that E^0 is globally asymptotically stable (since system (5.2) is a linear system). Therefore, we have $\Omega=\{E_0\}$, and E_0 is covering of Ω, which is isolated and is acyclic (since there exists no solution in M_∂ which links E_0 to itself). Finally, the proof will be done if we show E_0 is a week repeller for X_1 i.e.

$$\limsup_{t\to\infty} dist(\Phi(t),E_0)>0, \tag{5.3}$$

where $\Phi(t)=(S_1(t),I_1(t),A_1(t),S_2(t),I_2(t),A_2(t),S_3(t),I_3(t),A_3(t),S_4(t),I_4(t),A_4(t))$ is an arbitrarily solution of system (2.1) with initial values in X_1.

By the proof of Lemma 3.5 in [36], (5.3) is equal to $W^s(E_0)\cap X_1=\emptyset$, where $W^s(E_0)$ is the stable manifold of E_0, contradiction method is applied to prove it.

Suppose $W^s(E_0)\cap X_1\neq\emptyset$. It implies that there exists a solution $(S_i(t),\ I_i(t),\ A_i(t))$ $(i=1,2,3,4)$ of system (2.1) in X_1 satisfies

$$S_i(t)\to S_i^0, I_i(t)\to 0, A_i(t)\to 0 \text{ as } t\to\infty. \tag{5.4}$$

Since $R_0^1=\frac{\alpha_{22}(\mu+\delta+r_2k_2)}{(\mu+r_2)(\mu+\delta)}>1$, it follows that $\frac{\alpha_{22}k_2}{\mu+\delta}>\frac{\mu+r_2-\alpha_{22}}{r_2}$, we can choose constants $\varsigma>0,\sigma_1>0,\sigma_2>0$ such that

$$\frac{\alpha_{22}k_2}{\mu+\delta}\frac{S_2^0-\varsigma}{S_2^0+3\varsigma}>\frac{\sigma_2}{\sigma_1}>\frac{\mu+r_2}{r_2}-\frac{\alpha_{22}}{r_2}\frac{S_2^0-\varsigma}{S_2^0+3\varsigma}.$$

By (5.4), there exists $T > 0$ such that for all $t \geqslant T$, the following inequality holds

$$S_2^0 - \varsigma < S_2 < S_2^0 + \varsigma, 0 \leq I_2(t) < \varsigma, 0 < A_2(t) < \varsigma.$$

Let $M(t) = \sigma_1 I_2(t) + \sigma_2 A_2(t)$, then we have

$$\begin{aligned} M'(t) =& \sigma_1 \left(\frac{\alpha_{22} S_2}{S_2 + I_2 + A_2} (I_2 + k_2 A_2) - (\mu + r_2) I_2 + \sigma_2 r_2 I_2 - (\mu + \delta) A_2 \right) \\ \geqslant& \sigma_1 \left(\frac{\alpha_{22} S_2^0 - \varsigma}{S_2^0 + 3\varsigma} (I_2 + k_2 A_2) - (\mu + r_2) I_2 + \sigma_2 r_2 I_2 - (\mu + \delta) A_2 \right) \\ \geqslant& \left(\sigma_1 \alpha_{22} \frac{S_2^0 - \varsigma}{S_2^0 + 3\varsigma} + \sigma_2 r_2 - \sigma_1 (\mu + r_2) \right) I_2 + \left(\sigma_1 k_2 \alpha_{22} \frac{S_2^0 - \varsigma}{S_2^0 + 3\varsigma} - \sigma_2 (\mu + \delta) \right) A_2 \\ \geqslant& \sigma M(t), \end{aligned}$$

where $\sigma = \min\{\bar{\sigma}_1,\ \bar{\sigma}_2\} > 0$, $\bar{\sigma}_1 = \frac{\alpha_{22}(S_2^0 - \varsigma)}{S_2^0 + 3\varsigma} + \frac{\sigma_2 r_2}{\sigma_1} - \mu - r_2$, $\bar{\sigma}_2 = \frac{\sigma_1 \alpha_{22} k_2 (S_2^0 - \varsigma)}{(S_2^0 + 3\varsigma)\sigma_2} - \mu - \delta$. Hence $M(t) \to \infty$ as $t \to \infty$, which contradicts to the boundedness of $M(t)$. The same conclusion can be drawn for $R_0^2 > 1$.

When $R_0^* > 1$, we can chose constants $\eta > 0$, $\bar{\theta}_1, \bar{\theta}_2, \bar{\theta}_3 > 0$ such that

$$\begin{aligned} &\frac{\bar{\theta}_3}{\bar{\theta}_1} \frac{\alpha_{13}(S_3^0 - \eta)}{S_3^0 + 3\eta} \geqslant -\frac{\alpha_{11}(S_1^0 - \eta)}{S_1^0 + 3\eta} + \mu + r_1, \frac{\bar{\theta}_3}{\bar{\theta}_1} \leq \frac{\alpha_{31}(S_1^0 - \eta)}{(S_1^0 + 3\eta)(\mu + r_3)}, \\ &\frac{\bar{\theta}_1}{\bar{\theta}_2} \frac{\alpha_{21}(S_1^0 - \eta)}{S_1^0 + 3\eta} + \frac{\bar{\theta}_3}{\bar{\theta}_2} \frac{\alpha_{23}(S_3^0 - \eta)}{S_3^0 + 3\eta} \geqslant -\frac{\alpha_{22}(S_2^0 - \eta)}{S_2^0 + 3\eta} + \mu + r_2. \end{aligned}$$

For $\eta > 0$, there exists a $T > 0$ such that for all $t \geqslant T$ the following inequality holds

$$S_i^0 - \eta < S_i(t) < S_i^0 + \eta, 0 \leq I_i(t) < \eta, 0 < A_i(t) < \eta \ (i = 1, 2, 3).$$

Let $H(t) = \bar{\theta}_1 I_1(t) + \bar{\theta}_2 I_2(t) + \bar{\theta}_3 I_3(t)$. From system (2.1), we obtain that

$$\begin{aligned} H'(t) =& \bar{\theta}_2 \left(\alpha_{22} (I_2 + k_2 A_2) \frac{S_2}{N_2} - (\mu + r_2) I_2 \right) + \bar{\theta}_3 \left((\alpha_{13} I_1 + \alpha_{23} I_2) \frac{S_3}{N_3} - (r_3 + \mu) I_3 \right) \\ &+ \bar{\theta}_1 \left((\alpha_{11} I_1 + \alpha_{21} I_2 + \alpha_{31} I_3) \frac{S_1}{N_1} - (\mu + r_1) I_1 \right) \\ \geqslant& \left(\bar{\theta}_1 \frac{\alpha_{11}(S_1^0 - \eta)}{S_1^0 + 3\eta} + \bar{\theta}_3 \frac{\alpha_{13}(S_3^0 - \eta)}{S_3^0 + 3\eta} - (\mu + r_1) \bar{\theta}_1 \right) I_1 \\ &+ \left(\bar{\theta}_1 \frac{\alpha_{21}(S_1^0 - \eta)}{S_1^0 + 3\eta} + \bar{\theta}_2 \frac{\alpha_{22}(S_2^0 - \eta)}{S_2^0 + 3\eta} + \bar{\theta}_3 \frac{\alpha_{23}(S_3^0 - \eta)}{S_3^0 + 3\eta} - (\mu + r_2) \bar{\theta}_2 \right) I_2 \\ &+ \left(\bar{\theta}_1 \frac{\alpha_{31}(S_1^0 - \eta)}{S_0^1 + 3\eta} - \bar{\theta}_3 (r_3 + \mu) \right) I_3 + \frac{\bar{\theta}_2 k_2 A_2 (S_2^0 - \eta)}{S_2^0 + 3\eta} \geqslant \theta H(t), \end{aligned}$$

for all $t > T$, where $\theta = \min\left\{\tilde{\theta}_1,\ \tilde{\theta}_2,\ \tilde{\theta}_3,\right\} > 0$, $\tilde{\theta}_1 = \frac{\alpha_{11}(S_1^0 - \eta)}{S_1^0 + 3\eta} + \bar{\theta}_3 \frac{\alpha_{13}(S_3^0 - \eta)}{\bar{\theta}_1 (S_3^0 + 3\eta)} - (\mu + r_1)$, $\tilde{\theta}_2 = \bar{\theta}_1 \frac{\alpha_{21}(S_1^0 - \eta)}{\bar{\theta}_2 (S_1^0 + 3\eta)} + \frac{\alpha_{22}(S_2^0 - \eta)}{S_2^0 + 3\eta} + \frac{\bar{\theta}_3}{\bar{\theta}_2} \frac{\alpha_{23}(S_3^0 - \eta)}{S_3^0 + 3\eta} - (\mu + r_2)$, $\tilde{\theta}_3 = \frac{\bar{\theta}_1}{\bar{\theta}_3} \frac{\alpha_{31}(S_1^0 - \eta)}{S_0^1 + 3\eta} - (r_3 + \mu)$. Hence $H(t) \to \infty$ as $t \to \infty$, which contradicts to the boundedness of $H(t)$. We complete the proof of Theorem 5.1. □

From Theorem 4.4 together with Theorem 5.1, we can claim that the basic reproduction number R_0 is a threshold parameter, which determines the outcome of disease, namely if $R_0 < 1$, the disease-free equilibrium E_0 is globally asymptotically stable (the disease dies out). While $R_0 > 1$, the disease will permanence.

6. Numerical simulations

In this section, we implement numerical simulation to confirm and extend the analytic results, and illustrate the effect of the basic reproduction numbers on the disease dynamics. For this pattern, we choose $r_i = 1.25$ and other parameters are the same as Table 8. The disease-free equilibrium E_0 is a unique stable state when $R_0 < 1$(theoretical result is obtained in Theorem 4.4), which is shown in Figure 2. The simulations are shown in Figure 3 extend our analytical result presented in Theorem 5.1. While we do not have results on the existence and stability of endemic equilibrium of the system (2.1), our numerical studies in Figure 3 indicate that endemic equilibrium may exist. It means that HIV/AIDS will never be eliminated in Guangzhou, China.

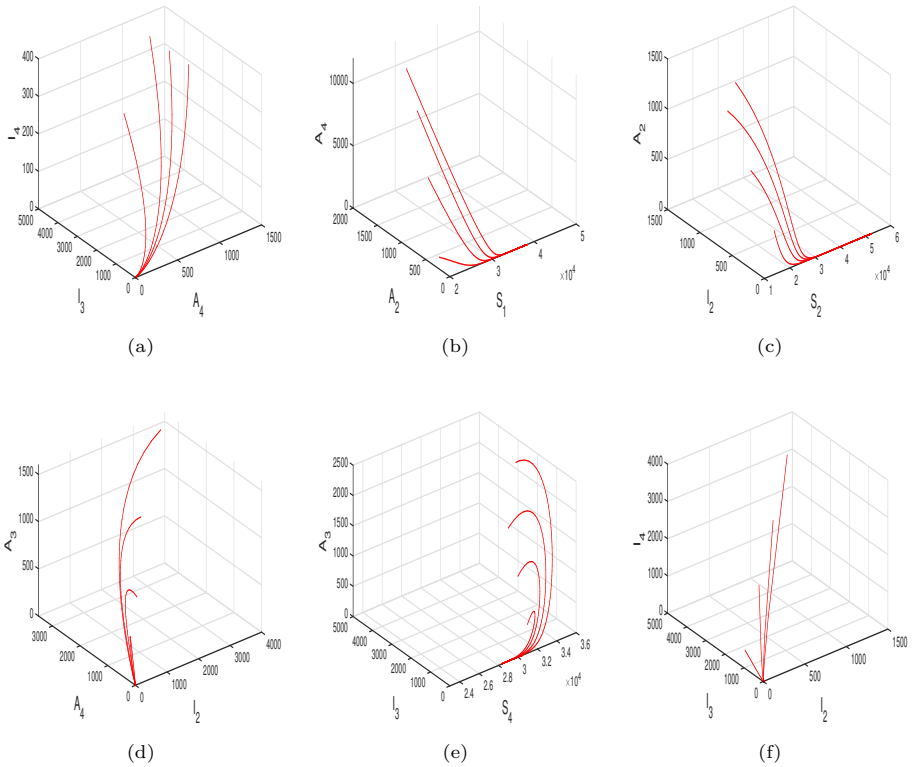


Figure 2. The stable disease-free equilibrium E_0 of system (2.1) with $r_i = 1.25$, other parameters are the same as Table 8, $R_0 < 1$.

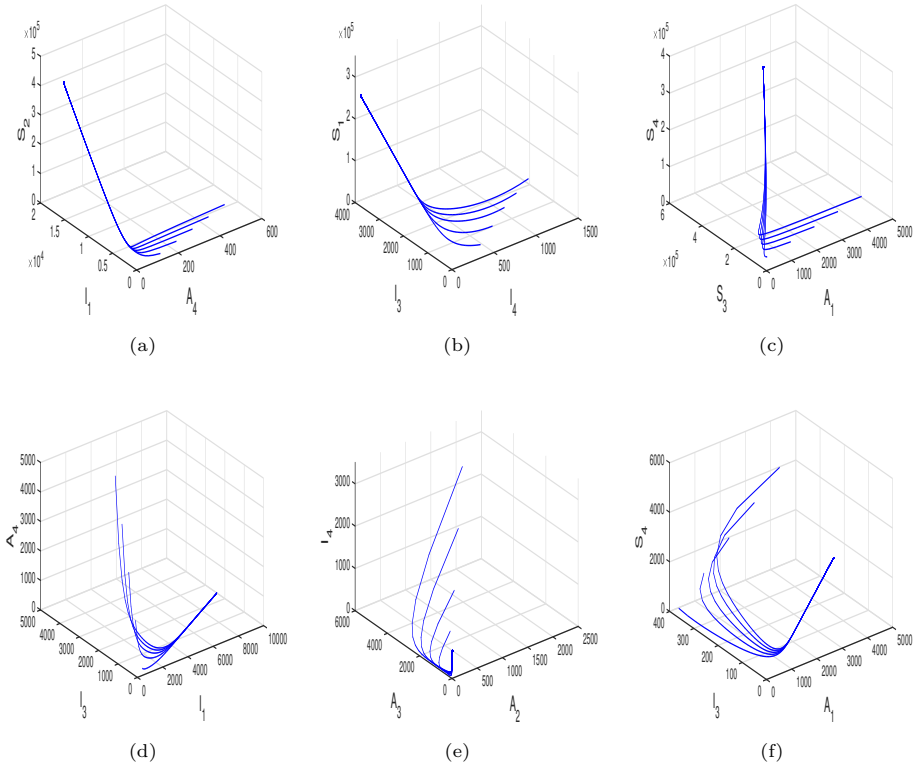


Figure 3. The existence of endemic equilibrium and persistence of system, parameters are shown in Table 8, $R_0^1 > 1$, $R_0^2 > 1$, and $R_0^3 > 1$.

7. A case study

In this section, we use system (2.1) to predict the potential HIV transmission among HIV high risk groups in Guangzhou, China. Firstly, we collect and settle the data from Guangzhou. Secondly, we estimate the parameters of system (2.1) by the data fitting. Thirdly, we give some predictions for the HIV transmission among HIV high risk groups in Guangzhou. Last but not least, we study the effect of control measure with different interventions (condom use, etc.). We obtain the following data of HIV/AIDS cases in Guangzhou from some papers and websites (see Table 1-6):

Table 1. The route of HIV/AIDS infections in Panyu district from 2005 to 2009 (%) [50].

Year	Drug injection	Heterosexual	Homosexual	Other routes
2005	80.95	16.67	0	2.38
2006	63.64	26.45	0	9.92
2007	48.94	44.68	4.26	0
2008	46.90	48.97	2.07	1.43
2009	32.31	57.69	8.46	0
Total	56.41	36.28	2.48	3.17

Table 2. The route of HIV/AIDS infections in Tianhe district up to 2010 (%) [37].

Year	Drug injection	Heterosexual	Homosexual	Other routes
2006	11	9	-	-
2007	5	25	2	5
2008	6	25	20	3
2009	3	31	20	3
2010	7	24	20	8
Total	54	121	41	6
Proportion	16.2%	36.3%	25.2%	-

Table 3. Yearly alteration of the route of HIV/AIDS transmission in Baiyun district, Guangzhou from 2001 to 2007 [49].

Year	2001	2002	2003	2004	2005	2006	2007	Total
Sexual transmission	7	6	5	7	20	48	48	141
Drug injection	36	21	16	77	227	166	99	642

Table 4. Number of new reported HIV/AIDS cases among MSM in Guangzhou ($2004-2010$) [54].

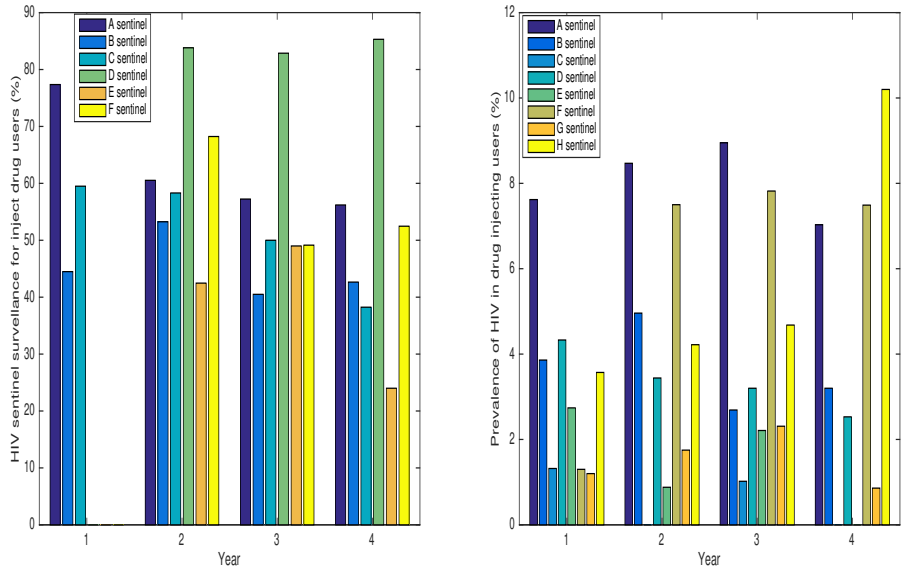
Year	Total cases	MSM cases	Proportion (%)
2004	868	2	0.2
2005	1052	1	0.1
2006	1606	11	0.7
2007	1390	24	1.7
2008	1584	117	7.4
2009	1449	189	11.1
2010	1372	179	13.1

Table 5. Prevalence of HIV among MSM in Guangzhou ($2003-2006, 2008-2010$) [54].

Year	Sample	Cases	Infectious rate(%)	95% CI
2004	201	0	0.0	0.0-1.5
2005	270	2	0.7	0.1-2.4
2006	423	7	1.3	0.3-2.7
2008	379	17	5.2	2.1-8.4
2009	400	17	4.3	2.6-6.6
2010	405	30	7.4	5.1-10.3

Table 6. The number of new reported HIV/AIDS cases among Foreigners from 2008 to 2015 in Guangzhou.

Year	Total cases	Male case	Proportion	Source
2008	85	44	51.76%	[55]
2009	92	48	51.76%	[55]
2010	-	50	-	[55]
2011	-	50	-	[55]
2012	-	57	-	[55]
2013	71	61	-	[56]
2015	114	66	57.9%	[17]



(a) (b)

Figure 4. (a) The proportion of injecting drugs among drug users from 2005 to 2008 in Guangzhou (%) [19], (b) The prevalence of HIV in drug users reported by 6 sentinel points from 2005 to 2008 in Guangzhou (%) [19].

Table 7. HIV/AIDS reported cases in Guangzhou from 2011 to 2017

Year	Reported cases	Drug injecting	Sexual transmission		Source
			Homosexual	Heterosexual	
2011	1599	20.5%	77.7%		[10]
			30.7%	69.3%	
2012	1202	17%	80%		[2]
			50%	50%	
2013	1286	9%	90%		[38]
			40.2%	59.8%	
2014	1528	5%	94.5%		[14]
			43%	51.5%	
2015	1800	3%	90%		[7]
			50%	50%	
2016	1977	3%	93.4%		[9, 39, 59]
			52.6%	47.4%	
2017	1894	3.5%	95%		[24]
			54.2%	45.8%	

7.1. Estimation of model parameters and initial values.

Here, we present a brief description of some parameters not already mentioned. The natural human death rate μ, is measured as the reciprocal of the average lifespan of human in Guangzhou. The value is selected based on data from the Guangzhou

Statistics Bureau [15]. The parameters, $1/r_1$, $1/r_2$, $1/r_3$, and $1/r_4$, whose give the respective average time HIV virus carriers convert to AIDS patients in the four HIV infected human groups. They are estimated from paper [6] to be in the range of 5-12 years. Mean value of HIV-related death of 0.7114 years^{-1} has also been cited [57]. The key parameters, condom use rates d_{1i}, d_{3i} and d_{44} are obtained from literature [19, 21, 25]. Those paper all focus on statistic and Mate analysis based on the real demographic and epidemiological data in Guangzhou city, hence our parameter values are reasonable and credible. Similarly, the infected ratio of different groups c_{1i}, c_{2i}, c_{3i}, and c_{44} are estimated on the basis of the published medical research articles [3, 40, 54, 55, 58], respectively. Table 8 lists all parameters, their definitions, and their ranges. The simulation of system (7.1) shown in Figure 5 and the references used to determine the parameter values.

Table 8. The parameters and numerical values

Parameters	Description	Range	Value ($year^{-1}$)	Source
Λ_1	Recruitment of S_1	-	2510	Fit
Λ_2	Recruitment of S_2	-	2930	Fit
Λ_3	Recruitment of S_3	-	2980	Fit
Λ_4	Recruitment of S_4	-	1140	Fit
μ	Natural death rate	-	0.0063	[15]
$\frac{1}{r_i}$	Average incubation period	[5-12] yr	8 yr	[6]
p_{14}	Moving rates from S_1 to S_4	-	0.0234	Fit
p_{34}	Moving rates from S_3 to S_4	-	0.011	Fit
p_{41}	Moving rates from S_4 to S_1	-	0.014	Fit
p_{43}	Moving rates from S_4 to S_1	-	0.0132	Fit
d_{11}, d_{13}	Condom using ratio for MSM	$[30.0 - 53.81](\%)$	53%	[21]
d_{2j} $(j = 1, 2, 3)$	Condom using ratio for FRs	-	50.0%	[27]
d_{31}	Condom using ratio for FSWs	$[17.06 - 47.5](\%)$	20.8%	[25]
d_{44}	Needle sharing rate for IDUs	$[22.91 - 51.11](\%)$	29.66%	[19]
c_{11}	Infected ratio among MSM	$[2.1 - 28.3](\%)$	0.24	[3, 54]
c_{13}	Infected ratio of MSM to FSWs	$[2.1 - 18.3](\%)$	0.18	[3, 54]
c_{21}	Infected ratio of FRs to MSM	[1.3-7.4](%)	4.7%	[55]
c_{22}	Infected ratio among FRs	[1.3-7.4](%)	5.3%	[55]
c_{23}	Infected ratio of FRs to FSWs	[1.3-7.4](%)	2.6%	[55]
c_{31}	Infected ratio of FSWs	$[0.2 - 10.3](\%)$	0.0964	[58]
c_{44}	Infected ratio of IDUs	$[1.5 - 6.73](\%)$	0.019	[40]
k_2, k_4	Infectivity of AIDS patient	0.005-0.02	0.01	[57]
δ	AIDS-related death rate	-	0.7114	[57]

From Table 4, there is sharp rise of MSM infected HIV/AIDS in 2008, when 117 new cases are seen, meanwhile , the total number of HIV/AIDS cases is 1584 in Table 7. Besides, the number of HIV/AIDS among foreigner is 85 in 2008, Guangzhou in Table 6. The initial time $t = 0$ is chosen in the year 2008. From

the statistical data of four district, Guangzhou in 2008 (see Table 1-6, Figure 4), we estimate among those HIV/AIDS cases, 32.31% are infected via injecting drug use, 57.69% are through heterosexual transmission. Based on the statistical data in the literatures [11, 50], we obtain the ratio of men to women is almost 3.5:1, and the ratio of HIV case is almost 75% of HIV/AIDS cases. Then we estimate five compartments populations and HIV/AIDS cases in 2008 ($t = 0$), respectively.

MSM compartment: From Table 4, we obtain initial value $I_1(0) + A_1(0) = 117$. $N_1(0) = 22,957$, this number is 27,900 and 35,000 in 2010, 2014, respectively [13, 43]. Hence, $N_1(0) = 22,957, S_1(0) = 22,957 - 117 = 22,840, A_1(0) = 117 \times 0.25 = 29, I_H(0) = 88$.

FRs compartment: The total FRs population $N_2(0) = 19,000$, this number is 28,000, 31,000, 36,000, 37,000, 47,000, 49,800, and 58,000 in 2010, 2011, 2013, 2014, 2015, 2017, respectively [13, 43]. From Table 6 and [55], we have $I_2(0) + A_2(0) = 85, I_2(0) = 54 \times 0.75 = 64, A_2(0) = 21, S_2(0) = 11,970 - 85 = 11,885$.

FSWs compartment: The total population $N_3(0) = 28000$ [58] (2008 data). Hence, $A_3(0) + I_3(0) = 1584 \times 57.69\%/4.5 = 203, A_3(0) = 203 \times 0.25 = 51, I_3(0) = 152, S_3(0) = 28000 - 203 = 27,7977$.

IDUs compartment: The total population $N_4(0) = 50,000 \times 47.56\% = 23,780$ [40] (2008 data), the ratio of needles sharing is 29.6% [40] (2008 data), the total data of the IDUs populations are 24,500, 28,600, 29,267, 30,800, 31,600, 34,000, 34,800, 36,000 from 2010 to 2017 [8, 47]. Thus, $I_4(0) + A_4(0) = 32.31\% \times 1584 = 512, I_4(0) = 512 \times 0.75 = 384, A_4(0) = 512 \times 0.25 = 128, S_4(0) = 23,780 - 512 = 23,268$.

To get the estimates of p_{14}, p_{34}, p_{41}, p_{43}, and Λ_i $(i = 1, 2, 3, 4)$, we drop out the terms involving I_i, A_i and $S_i \cong N_i$ (HIV/AIDS cases account for a very small proportion of the high-risk population). Then we obtain the following system from system (2.1)

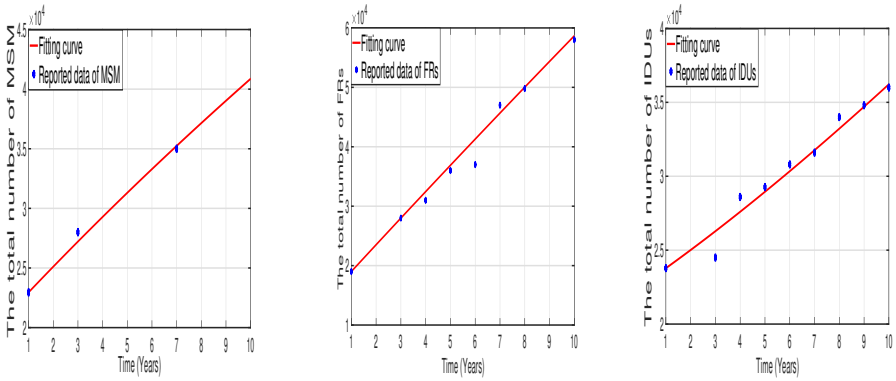
$$\begin{cases} N_1'(t) \cong \Lambda_1 + p_{41}N_4 - P_{14}N_1 - \mu N_1, \\ N_2'(t) \cong \Lambda_2 - \mu N_2, \\ N_3'(t) \cong \Lambda_3 + p_{43}N_4 - p_{34}N_3 - \mu N_3, \\ N_4'(t) \cong \Lambda_4 + p_{14}N_1 + p_{34}N_3 - (p_{41} + p_{43})N_4 - \mu N_4, \end{cases} \tag{7.1}$$

We use system (7.1) to fit the total numbers corresponding to the IDUs, MSM and FRs data, respectively (see Fig. 5), where $\mu = 0.0063$ (see Table 8).

Then we obtain the recruitment Λ_i and the moving rates p_{14}, p_{34}, p_{41}, p_{43} as follows

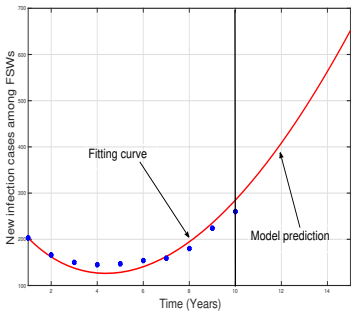
$$\Lambda_1 = 2510, \Lambda_2 = 2930, \Lambda_3 = 2980, \Lambda_4 = 1140,$$
$$p_{14} = 0.0234, p_{34} = 0.011, p_{41} = 0.014, p_{43} = 0.0132.$$

Now we choose the fitting values of model (2.1) in Table 8, and obtain $R_0^2 = 0.847 < 1$, $R_0^1 = 1.135 > 1$ and $R_0^3 = 1.607 > 1$. In general, numerical estimation results show in Figure 5 and Figure 6 indicate that our model provides a relatively good match with the annual new reported infected HIV/AIDS data of MSM, FRs, FSWs, IDUs HIV/AIDS cases in Guangzhou from 2008 to 2017, respectively (the statistical data in Table 4, 7, 6). Moreover, we use our model to predict the tend of four high-risk groups HIV/AIDS infected cases in Guangzhou, it can be seen that the HIV/AIDS infected cases of MSM, FRs and FSWs will increase in the next few

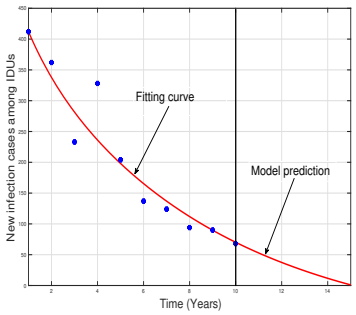


(a) (b) (c)

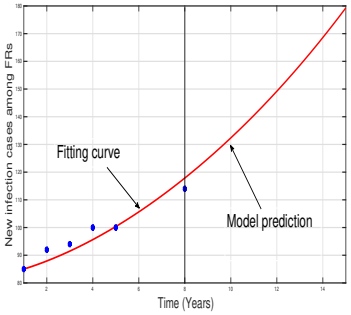
Figure 5. The fitting curves of the total population of MSM, FRs and IDUs. Other parameters are the same as Table 8.



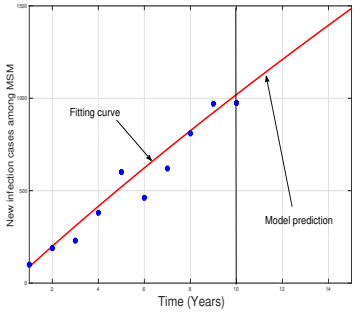
(a) The newly HIV/AIDS cases of FSWs.



(b) The newly HIV/AIDS cases of IDUs.



(c) The newly HIV/AIDS cases of FRs.



(d) The newly HIV/AIDS cases of MSM .

Figure 6. The fitting curves of new HIV/AIDS infected FSWs, IDUs, FRs and MSM in Guangzhou from 2008 to 2017.

years. Specifically, the HIV/AIDS infected populations of MSM increase rapidly. However, the HIV/AIDS infected populations of injecting drug users will decrease sharply. These results show that the sexual transmission is the main route among HIV high-risk groups, whose are in line with literature [12].

7.2. Sensitivity analysis

In order to analyze the effects of the parameter values on the basic reproduction number of system (2.1), we perform sensitivity analysis by Latin square sampling and partial rank correlation coefficient (PRCC) methods [26]. In the absence of available data on the distribution functions, we choose a uniform distribution for all input parameters with the minimum and maximum values are shown in Table 8 and tested for significant PRCCs for all parameters of R_0^3 (PRCCs is given in Table 9). Figure 7 shows PRCCs values of the parameters against the basic reproduction number, which indicates that the parameters c_{11}, d_{11} and r_1 of the model (2.1) have significant impact on the basic reproduction number. These results indicate that decreasing the transmission rate of sexual or increasing the condom use proportion are two effective measures to reduce the basic reproduction number, that is, the development of highly condom use is a critical control measure. So, studying the effects of changing these two parameters on the basic reproduction number is of significant applied value.

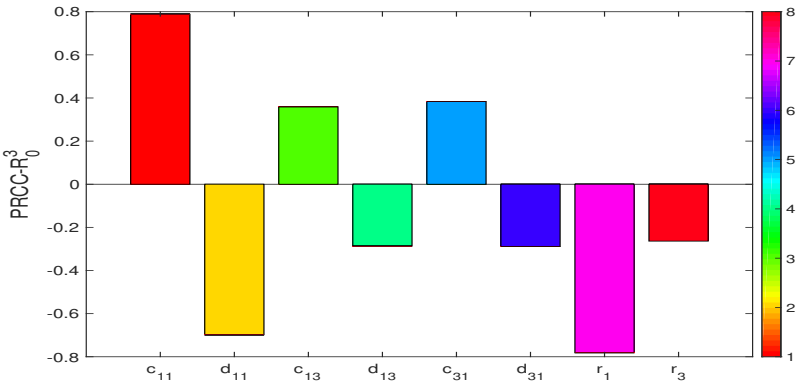


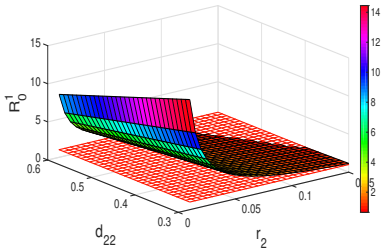
Figure 7. The PRCC of R_0^3

Table 9. PRCC for the basic reproduction ratio R_0^3.

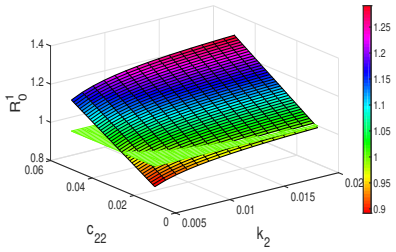
Parameters	Distribution of parameters	PRCC	p value
c_{11}	U(0.021,0.183)	0.7886	0
d_{11}	U(0.3706,0.475)	-0.6982	0.2599
c_{13}	U(0.042,0.213)	0.3596	0.6556
d_{13}	U(0.3,0.53)	-0.2867	0.5138
c_{31}	U(0.042,0.213)	0.3839	0.5505
d_{31}	U(0.3,0.53)	-0.2891	0.1108
r_1	U(0.083,0.2)	-0.7818	0
r_3	U(0.083,0.2)	-0.2638	0.4335

We carry out some sensitivity analysis to investigate the influence of parameters $r_2,\ d_{22},\ c_{22},\ k_2$ on R_0^1 and $r_4,\ d_{44},\ c_{44},\ k_4$ on R_0^2, the parameters values are the same as the above case besides r_2, d_{22} in Figure 8 (a). Similarly, in Figure 8 (b), it is shown that R_0^1 and R_0^2 are increasing functions of c_{22} and c_{44}, respectively. R_0^1 and R_0^2 are decreasing functions of d_{22} and d_{44}, respectively. Furthermore, from Figure 8 (c), (d) we can see that R_0^2 nearly less than 1, it means that the population of infected HIV among IDUs will decrease and eliminate ultimately, which implies that injecting drugs is not main route of HIV transmission any more. In Figure 9, it can be seen that R_0^3 almost more than 1, this result reveals that HIV infected cases will increase sharply among MSM (homosexual/bisexual) through sexual transmission and this trends will not be improved unless some control measures are taken.

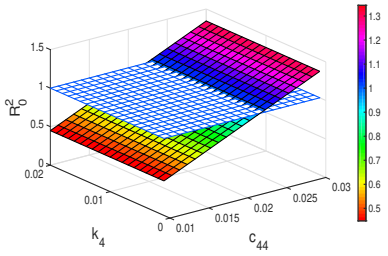
We are also interested in the impact of the recruitment $\Lambda_i\ (i = 1, 2, 3, 4)$ on the total HIV/AIDS cases. For this purpose, we examine the variation of $I_T(t) + A_T(t)$ with parameters Λ_i in Figure 10, here $I_T(t) = I_1(t) + I_2(t) + I_3(t) + I_4(t),\ A_T(t) = A_1(t) + A_2(t) + A_3(t) + A_4(t)$ and $\Lambda_i\ (i = 1, 2, 3, 4)$ being all varied in the interval [0, 4000]. From Figure 10 (a), it is revealed that $I_T(t) + A_T(t)$ is an increasing function with respect to Λ_1 and the total HIV/AIDS cases will be decrease dramatically when Λ_1 decreases. Furthermore, it follows from Figure 10 $(b), (c)$ that both Λ_2 and Λ_3 affect the number of HIV/AIDS cases in a positive manner, but to lesser extent than Λ_1. Moreover, it can be seen that Λ_4 is barely influenced on the trends of the total HIV/AIDS cases in Figure 10 (d). These results indicated that the leading cause of the increase in HIV/AIDS cases is the increase in the number of MSM, followed by FSWs, FRs. From a practical point of view, we claim that the intervention of the high-risk groups is a very effective measure to control the spread of HIV disease.



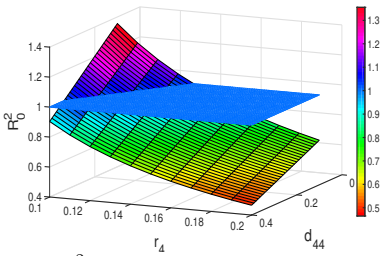
(a) R_0^1 trends with respect to $d_{22},\ r_2$.



(b) R_0^1 trends with respect to $c_{22},\ k_2$.

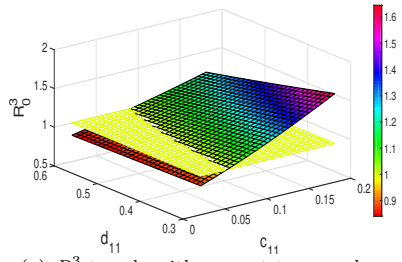


(c) R_0^2 trends with respect to $k_4,\ c_{44}$.

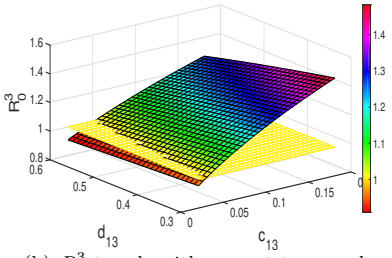


(d) R_0^2 trends with respect to $r_4,\ d_{44}$.

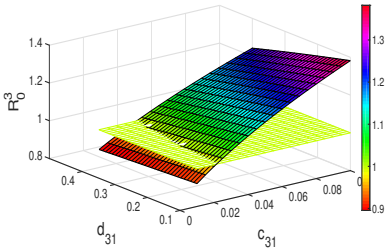
Figure 8. (a), (b): R_0^1 trends in regard to some parameters of model (2.1), (c), (d): R_0^2 trends in regard to some parameters of model (2.1), other parameters are the same as Table 8.



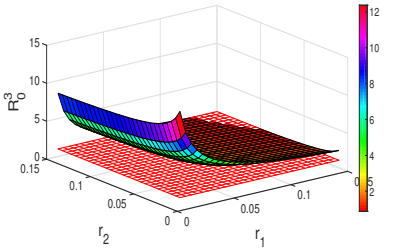
(a) R_0^3 trends with respect to c_{11}, d_{11}.



(b) R_0^3 trends with respect to c_{13}, d_{13}.

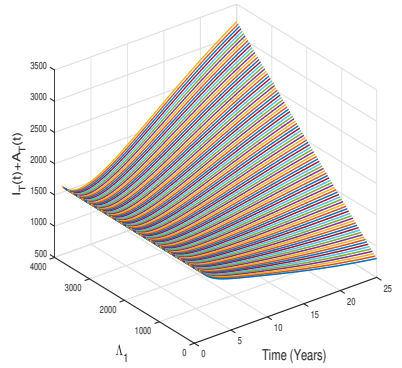


(c) R_0^3 trends with respect to c_{31}, d_{31}.

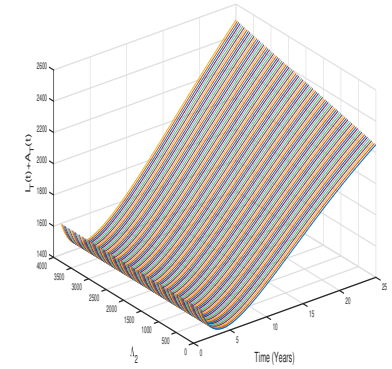


(d) R_0^3 trends with respect to r_1, r_3.

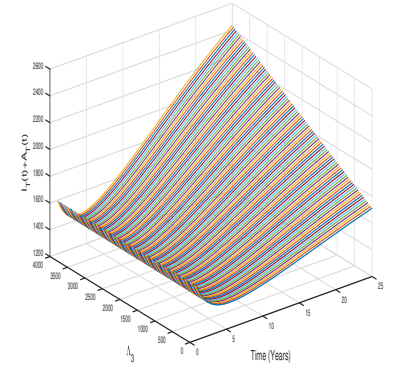
Figure 9. R_0^3 trends in regard to some parameters of model (2.1), the other parameters are the same as Table 8.



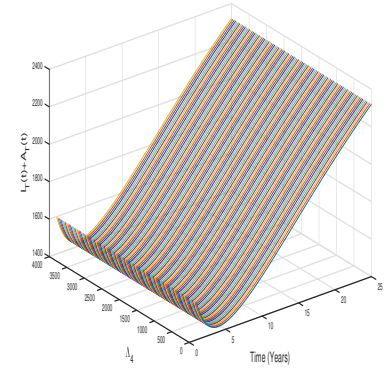
(a) $I_T(t) + A_T(t)$ trends with respect to Λ_1.



(b) $I_T(t) + A_T(t)$ trends with respect to Λ_2.



(c) $I_T(t) + A_T(t)$ trends with respect to Λ_3.



(d) $I_T(t) + A_T(t)$ trends with respect to Λ_4.

Figure 10. The value of $I_T(t) + A_T(t)$ in time and with respect to the recruitments of different compartments Λ_i $(i = 1, 2, 3, 4)$ being all varied in the interval [0, 4000], other parameters are the same as Table 8.

From the numerical fitting and prediction results in Figure 6, it can be observed that the HIV/AIDS cases among MSM, FSWs, and PRs increased while the HIV/AIDS cases among IDUs decreased year by year. By analyzing the impact of the recruitment of four high-risk groups on the total cases (see Figure 10), we found that the total HIV/AIDS cases is most affected by MSM, followed by FSWs, FRs, and IDUs. Based on the above numerical results, we conclude that the order of significance of the four high-risk groups in HIV transmission is MSM, FSWs, FRs, IDUs. These facts indicated that MSM, FSWs, and FRs should be the key targets for HIV control to prevent the spread of the disease from high-risk groups to the general population.

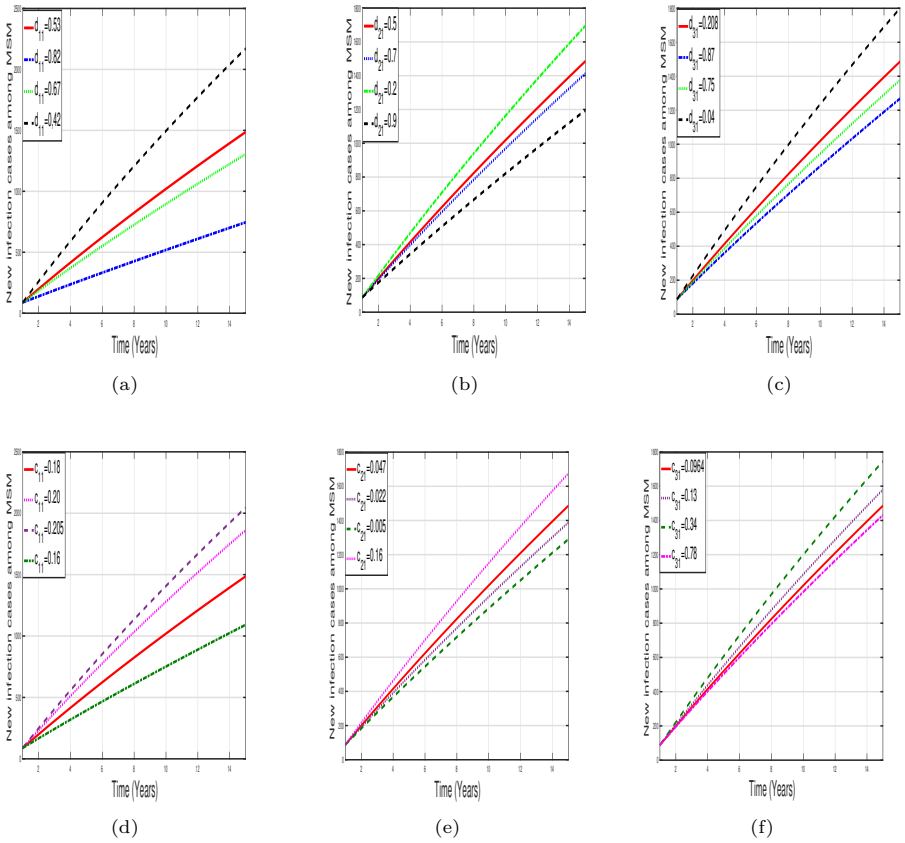


Figure 11. Different parameters influence on HIV infected among MSM. Other parameters are the same as Table 8.

7.3. Intervention and control

Simulation results show that decreasing infection rate and increasing condom use can slow down the spread of HIV transmission (see in Figure 7) and the increase in MSM is primary factor of the increase in HIV/AIDS cases among high-risk group (see in Figure 10). Hence, we now analyze the infection rate and condom use influence HIV transmission among MSM, whose always have more risky sexual

behavior and more likely to transmit HIV virus than other groups. In Figure 11 $(a)-(c)$, it can be seen that increasing condom use when MSM have sex with other groups can reduce HIV transmission. In Figure 11 $(d)-(f)$, it is shown that decreasing infection rate between MSM and other groups can achieve the same goals. Hence, to control the disease among MSM group availably, the intervention measure for whole high-risk population behaviors must be taken.

8. Conclusion and Discussion

It is known that HIV high-risk groups play an important role in the spread of HIV epidemic. Hence, compartment models for HIV transmission among high-risk groups (IDUs, FSWs, MSM) have been discussed by many researchers, but these models mainly considered a subset of high risk groups. Yang formulated only two groups (FSWs and senior male clients) [48]. Sun analyzed an HIV/AIDS epidemic only among men who have sex with men [30]. Zhang and Zhou established one model including IDUs, FSWs, MSM, Senior male clients [52]. Although four high-risk groups are considered, the interconnection by sexual behaviors among these groups is not considered. Compared to existing compartment models on HIV infection [20, 30–32, 48], the proposed model here studies potential bridges for HIV transmission among high risk groups. Specifically, the foreigner residents are considered.

Our main concerns are studying the potential bridge for HIV transmission among HIV high-risk groups (MSM, SFWs, IDUs, FRs) and their HIV/AIDS epidemic tendency. We have studied the qualitative behavior of the model. It has been shown that the dynamics of this model are simple i.e., the disease-free equilibrium is globally asymptotically stable when the basic reproduction number $R_0 < 1$, and the infection equilibrium will permanence when $R_0 > 1$. Furthermore, numerical analyses of our model based on data in Guangzhou show the following facts: (1) Foreigner residents play an important role in HIV transmission, and pose a big challenge for HIV control. (the HIV/AIDS cases among PRs in Guangzhou increased rapidly after 2008, see Table 6 and Figure 6 (c)), (2) Homosexual transmission among MSM has replaced injecting drugs as the main route of HIV transmission, which is in lines with reports [2,7,14,38], (3) The leading causes of HIV/AIDS are MSM, FSWs, and PRs and these high-risk groups should be the key targets for HIV control to prevent the spread of the disease to the general population. Sensitivity analyses of the basic reproduction number on various model parameters indicate that it is important, indeed crucial, to increase the condom use rate for the HIV high-risk groups to control the transmission of HIV in Guangzhou, China. Moreover, reduction in any one or more routes of disease transmission will be useful, although reduction in transmission of the high-risk groups will be more effective than other intervention measures.

From a practical point of view, the model established in our paper can be used to comprehend the transmission behaviors of the disease among HIV high-risk groups in Guangzhou and to predict the disease trends in a few years, which can help Guangzhou's and China's departments of health to implement preventive interventions in the HIV high-risk groups.

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