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Global stability for age-infection-structured human immunodeficiency virus model with heterogeneous transmission

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ABSTRACT

In this paper, we analyze the global asymptotic behaviors of a mathematical susceptible-infected(SI) age-infection-structured human immunodeficiency virus(HIV) model with heterogeneous transmission. Mathematical analysis shows that the local and global dynamics are completely determined by the basic reproductive number $\mathscr{R}_0$. If $\mathscr{R}_0 < 1$, disease-free equilibrium is globally asymptotically stable. If $\mathscr{R}_0 > 1$, it shows that disease-free equilibrium is unstable and the unique endemic equilibrium is globally asymptotically stable. The proofs of global stability utilize Lyapunov functions. Besides, the numerical simulations are illustrated to support these theoretical results and sensitivity analysis of each parameter for $\mathscr{R}_0$ is performed by the method of partial rank correlation coefficient(PRCC).

1. Introduction

Acquired Immunodeficiency Syndrome (AIDS) is a malignant infectious disease caused by HIV with high mortality rate. AIDS has become a serious public health problem worldwide and sexual transmission is the main transmission mode. 39 million people in the world were living with HIV in 2022, this seriously affect healthy development of society, with stronger challenges to health care issues (World health organization). Therefore, it is necessary to further study AIDS transmission from mathematical theory to provide theoretical guidance for medical and health decision-making. Epidemiological and behavioral factors are crucial to the dynamics of HIV model. In recent years, many scholars have been keen to use mathematical methods to establish and analyze HIV models. The truth is many of the issues have been extensively studied based on classical texts and documentation. Nowak and May (Nowak & May 2000), Perelson and Nelson (Perelson & Nelson, 1999) gave a basic model of infection that included a variety of mathematical expressions (Wang et al., 2015).

In (Frioui et al., 2020), the aim was to investigate a general infection model of age, in which infectivity was age-dependent from the time of infection, and where some quarantined individuals could be returned to the infectious class after some time.

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The HIV model taking into account the dependence of HIV/AIDS progression on infection age and chronological age was developed. The results of numerical simulations were used to demonstrate the impact of an impulsive treatment strategy on HIV/AIDS dynamics in (Liu et al., 2008). In (Huang et al., 2012), it investigated the basic population age structure model of HIV infection process and determined the dynamic properties without (or with) medical treatment by using the direct Lyapunov method and the corresponding Lyapunov functions. Using integrated semigroup and Lyapunov functions was used to show that the unique endemic equilibrium was globally stable in (Magal et al., 2010). In (Wu & Zhao, 2020), a multidimensional model was developed to study HIV prevalence in various high-risk groups, including men who have sex with men, foreign residents, female sex workers and injecting drug users. In (Thomas et al., 2015), an alternative method that deconstructed the larger system into smaller subsystems and captured the interactions between the smaller systems as external forces using an approximate model was presented. In (Chen et al., 2018), a generalized model of SIS-type diseases including age-dependent infections; birth and death in a heterogeneous network was to analyze the transmission mechanism and dynamic behaviour of infectious diseases in a realistic way. The proposal of a new age and spatially structured model which includes both ages of infection, spatial transmission and highly active treatment aimed to analyze the global dynamics of HIV/AIDS and study the prevalence of transmission among men who have sex with men as a group in (Wu & Zhao, 2021). In (Chekroun & Kuniya, 2020), the global asymptotic behavior of the SIR epidemic model with age-structured infection and diffusion in a general n-dimensional restricted spatial domain with a homogeneous Dirichlet boundary condition was concerned. The basic reproduction number was derived in an epidemic model in which infected individuals were initially asymptomatic and structured according to time since infection in (Barril et al., 2021).

In (Sun, 2010), using ordinary differential equation systems was to study multi-group SIR models with nonlinear incidence. But many diseases show differential infectivity concerning the age of infection as the disease progresses. Differential infectivity was important for studying different infectious pathologies during disease transmission (Chen et al., 2018). Analyzing linear systems at equilibria and checking the eigenvalues of characteristic equations, local stability of age structure models usually part of formulated as first-order partial differential equations could be demonstrated. However, the analysis of global stability becomes a complicated mathematical problem due to the complexity of the models. To obtain global stability, a common approach has been to construct a Lyapunov function (Wang et al., 2015).

Age structure models are usually a system of partial differential equations and their mathematical analysis is particularly complex. Although the dynamical analysis is hard, by constructing Lyapunov functions (Kuniya et al., 2016; Magal et al., 2010; Shen & Xiao, 2016; Zhang & Xu, 2016), global stability was investigated for several types of age structure models (Zhang & Guo, 2018). In (Shen & Xiao, 2016), the epidemiological system of SVEIR in different groups according to age at vaccination and age at infection was investigated using a graph-theoretic approach with Lyapunov functional.

To our knowledge, some researchers such as Martcheva (Martcheva, 1999; Martcheva & Milner, 1999; Wang et al., 2020) and Inaba (Inaba, 1993, 2000) had studied some properties of models with age-structured two-sex populations. Some researchers studied global dynamics of infection-age-structured HIV model (Shen et al., 2015, 2019; Wang et al., 2016). Some researchers had studied HIV transmission by building models of infectious disease compartments, most of the work focused on the traditional trend of HIV prevalence in high-risk populations. However, there were few conclusions corresponding to global characteristics of HIV model with age-infection-structured describing complex processes of interconnected infection ages with heterogeneous transmission. In this paper, a goal is to illustrate the disease dynamics relying on an age-dependent factor. Furthermore, the dynamic behaviour entirely depends on the basic reproduction number.

This paper is structured as follows. In Section 2, the age-infection-structured HIV model with heterogeneous transmission is introduced. In Section 3, we analyze the existence of equilibria and obtain the basic reproductive number. In Section 4, local stabilities of disease-free equilibrium and endemic equilibrium are showed. In Section 5, we present the global stabilities of disease-free equilibrium and endemic equilibrium using Lyapunov functions, respectively. In Section 6, numerical simulations and sensitivity analysis are showed. In Section 7, a summary is stated to conclude this work.

2. Model formulation

In HIV model, considering the different impacts for each gender, the total population can be divided into four compartments: susceptible female, infectious female, susceptible male and infectious male. Let $S_f(t)$, $I_f(t)$, $S_m(t)$ and $I_m(t)$ denote the total numbers of susceptible female, infectious female, susceptible male and infectious male population at time t, respectively. And let $N(t)$ denote the total numbers of all compartments.

Because the infection rate of infected people varies with the time of infection (Martcheva, 2015), the infectious compartment is further classified according to the age of infection. Let a denote time-since-infection of female individuals, and τ denote time-since-infection of male individuals. Let $i_f(0, t)$ and $i_m(0, t)$ respectively denote the numbers of female and male population who become infected at time t. And $i_f(a, t)$ and $i_m(\tau, t)$ respectively denote the density of female population with infection-age a and male population with infection-age τ at time t. The total infected female population in the infectious compartment is

$$I_f(t) = \int_0^\infty i_f(a,t)da,$$

and the whole infected male population is

$$I_m(t) = \int_0^\infty i_m(\tau, t)d\tau.$$

Assuming the lifespan of all the individuals is exponentially distributed (Martcheva, 2015), so each individual leaves the system due to a constant μ as natural death rate. Moreover, individuals can leave infected population with the disease-death rate α and the transition rate γ from infection class to AIDS class. Thus, equation for the number of individuals leaving infectious female compartment in time Δt is

$$i_f(a+\Delta t, t+\Delta t)\Delta a - i_f(a,t)\Delta a = -(\mu+\alpha+\gamma)\Delta t i_f(a,t)\Delta a. \tag{2.1}$$

If the partial derivatives of i_f exists and continuous, we can divide both sides of equation (2.1) by $\Delta a \Delta t$ and take the limit as $\Delta t \to 0$. Then we could obtain equation for infectious female individuals

$$\frac{\partial i_f}{\partial a}(a,t) + \frac{\partial i_f}{\partial t}(a,t) = -(\mu+\alpha+\gamma)i_f(a,t),$$

defined on domain $\{(a, t)$: $a \geq 0, t \geq 0\}$.

Similarly, equation for infectious male individuals is

$$\frac{\partial i_m}{\partial \tau}(\tau,t) + \frac{\partial i_m}{\partial t}(\tau,t) = -(\mu+\alpha+\gamma)i_m(\tau,t),$$

defined on domain $\{(\tau, t)$: $\tau \geq 0, t \geq 0\}$.

Next, to derive equations for newly infected population, we let $\beta_f(a)$ denote the incidence of infection susceptible men infected by women of infection-age a and $\beta_m(\tau)$ denote incidence of infection susceptible women infected by men of infection-age τ. Thus, all newly infected female individuals are

$$i_f(0,t) = S_f(t)\int_0^\infty \beta_m(\tau)i_m(\tau,t)d\tau,$$

and all newly infected male individuals are

$$i_m(0,t) = S_m(t)\int_0^\infty \beta_f(a)i_f(a,t)da.$$

Assuming recruitment rate Λ_f into female population and recruitment rate Λ_m into male population is to derive formulas giving the transmission process of susceptible population. So equations of susceptible female and male individuals become

$$\dot{S}_f(t) = \Lambda_f - S_f(t)\int_0^\infty \beta_m(\tau)i_m(\tau,t)d\tau - \mu S_f(t),$$

$$\dot{S}_m(t) = \Lambda_m - S_m(t)\int_0^\infty \beta_f(a)i_f(a,t)da - \mu S_m(t),$$

respectively. The flow chart is displayed in Fig. 1.

Equations for the susceptible female, infectious female, susceptible male and infectious male population define a system, which we shall consider

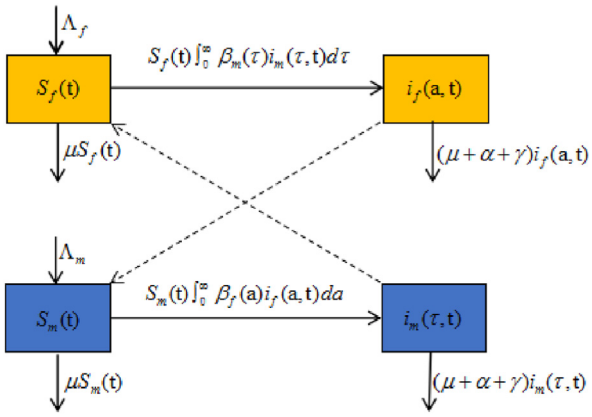

Fig. 1. Flow chart of age-infection-structured HIV model.

$$\begin{aligned}
\dot{S}_f(t) &= \Lambda_f - S_f(t)\int_0^\infty \beta_m(\tau)i_m(\tau,t)d\tau - \mu S_f(t),\\
\frac{\partial i_f}{\partial a}(a,t) &+ \frac{\partial i_f}{\partial t}(a,t) = -(\mu+\alpha+\gamma)i_f(a,t),\\
\dot{S}_m(t) &= \Lambda_m - S_m(t)\int_0^\infty \beta_f(a)i_f(a,t)da - \mu S_m(t),\\
\frac{\partial i_m}{\partial \tau}(\tau,t) &+ \frac{\partial i_m}{\partial t}(\tau,t) = -(\mu+\alpha+\gamma)i_m(\tau,t),\\
i_f(0,t) &= S_f(t)\int_0^\infty \beta_m(\tau)i_m(\tau,t)d\tau,\\
i_m(0,t) &= S_m(t)\int_0^\infty \beta_f(a)i_f(a,t)da,
\end{aligned} \tag{2.2}$$

with initial conditions

$$\begin{aligned}
S_f(0) &= S_{f0},\\
i_f(a,0) &= i_{f0}(a),\\
S_m(0) &= S_{m0},\\
i_m(\tau,0) &= i_{m0}(\tau).
\end{aligned} \tag{2.3}$$

For convenience, we let $\delta = \mu+\alpha+\gamma$, $\frac{\partial i_f}{\partial a}+\frac{\partial i_f}{\partial t} = i_{fa}+i_{ft}$, $\frac{\partial i_m}{\partial \tau}+\frac{\partial i_m}{\partial t} = i_{m\tau}+i_{mt}$ and for each nonnegative and integrable initial conditions (2.3), we could know that system (2.2) has a unique nonnegative solution by Theorem 2.2 in (Soufiane & Touaoula, 2016).

Lemma 2.1. *The solutions of system* (2.2) *with the initial conditions* (2.3) *are bounded.*

Proof Integrating with respects to a and τ the partial differential equations in system (2.2), respectively. And assuming $\lim_{a\to\infty} i_f(a,t) = 0$ and $\lim_{\tau\to\infty} i_m(\tau,t) = 0$, we can obtain

$$\begin{aligned}
\dot{I}_f(t) &= i_f(0,t) - \delta I_f(t) = S_f(t)\int_0^\infty \beta_m(\tau)i_m(\tau,t)d\tau - \delta I_f(t),\\
\dot{I}_m(t) &= i_m(0,t) - \delta I_m(t) = S_m(t)\int_0^\infty \beta_f(a)i_f(a,t)da - \delta I_m(t).
\end{aligned} \tag{2.4}$$

Adding equation (2.4) to equations for $\dot{S}_f(t)$ and $\dot{S}_m(t)$ from system (2.2), we have

$$\dot{N}(t) = \dot{S}_f(t) + \dot{I}_f(t) + \dot{S}_m(t) + \dot{I}_m(t) \le \Lambda_f + \Lambda_m - \mu N(t).$$

Thus, the numbers of total population $N(t)$ satisfies

$$N(t) \leq \max\left(N_0, \frac{\Lambda_f + \Lambda_m}{\mu}\right),$$

where $N_0 = S_{f0} + \int_0^\infty i_{f0}(a)da + S_{m0} + \int_0^\infty i_{m0}(\tau)d\tau$.

A pivotal quantity associated with the survival of infectious female individuals is $\pi(a)$ and that associated with the survival of infectious male individuals is $\pi(\tau)$. Then, if $\hat{I}_f$ female individuals become infected at a given time, after a time units, the number still infectious is $\hat{I}_f\pi(a)$. The number changes for a short period of time Δa after infection by those left the system

$$\hat{I}_f\pi(a+\Delta a) - \hat{I}_f\pi(a) = -\delta\hat{I}_f\pi(a)\Delta a,$$

and $\pi(a)$ satisfies

$$\dot{\pi}(a) = -\delta\pi(a),$$

whose solution with assuming that $\pi(0) = 1$ is

$$\pi(a) = e^{-\delta a}.$$

similarly, $\pi(\tau)$ satisfies

$$\pi(\tau) = e^{-\delta\tau}.$$

3. Equilibria and basic reproduction number

We now look for time-independent solutions (S_f, $i_f(a)$, S_m, $i_m(\tau)$) as equilibria of system (2.2). We have

$$\begin{aligned}
\Lambda_f - S_f\int_0^\infty \beta_m(\tau)i_m(\tau)d\tau - \mu S_f &= 0,\\
i_{fa}(a) &= -\delta i_f(a),\\
\Lambda_m - S_m\int_0^\infty \beta_f(a)i_f(a)da - \mu S_m &= 0,\\
i_{m\tau}(\tau) &= -\delta i_m(\tau),\\
i_f(0) &= S_f\int_0^\infty \beta_m(\tau)i_m(\tau)d\tau,\\
i_m(0) &= S_m\int_0^\infty \beta_f(a)i_f(a)da.
\end{aligned} \tag{3.1}$$

System (3.1) consists of two first-order ordinary differential equations with solutions dependent initial conditions and two algebraic equations. It is clear that solution $\mathscr{E}^0 = (\frac{\Lambda_f}{\mu}, 0, \frac{\Lambda_m}{\mu}, 0)$ is a always existent disease-free equilibrium in which the age distribution after infection is zero. Then, a nontrivial solution $\mathscr{E}^* = (S_f^*, i_f^*(a), S_m^*, i_m^*(\tau))$ will give an endemic equilibrium. We first solve differential equations in system (3.1) whose solutions are

$$i_f^*(a) = i_f^*(0)\pi(a), \quad i_m^*(\tau) = i_m^*(0)\pi(\tau).$$

These are not explicit solutions, since $i_f^*(0)$ depends on $i_f^*(a)$ and $i_m^*(0)$ depends on $i_m^*(\tau)$.

And the female and male total infectious population are

$$I_f^* = i_f^*(0)\int_0^\infty \pi(a)da, \quad I_m^* = i_m^*(0)\int_0^\infty \pi(\tau)d\tau,$$

respectively.

Then we let $i_f^*(0) = i_m^*(0)$ and we can get equations of endemic equilibrium for susceptible population

$$S_f^* = \frac{1}{\int_0^\infty \beta_m(\tau)\pi(\tau)d\tau}, \quad S_m^* = \frac{1}{\int_0^\infty \beta_f(a)\pi(a)da}.$$

We use the first and third equations in system (3.1), which become

$$\Lambda_f - i_f^*(0) - \mu S_f^* = 0, \quad \Lambda_m - i_m^*(0) - \mu S_m^* = 0.$$

Next, we can obtain

$$i_f^*(0) = \Lambda_f\left(1 - \frac{1}{\mathscr{R}_{0f}}\right), \quad i_m^*(0) = \Lambda_m\left(1 - \frac{1}{\mathscr{R}_{0m}}\right).$$

In addition, we have

$$i_f^*(0) = \frac{\Lambda_m - \mu S_m^*}{S_m^*\int_0^\infty \beta_f(a)\pi(a)da} = \frac{\Lambda_m\left(1 - \frac{1}{\mathscr{R}_{0m}}\right)}{S_m^*\int_0^\infty \beta_f(a)\pi(a)da},$$

$$i_m^*(0) = \frac{\Lambda_f - \mu S_f^*}{S_f^*\int_0^\infty \beta_m(\tau)\pi(\tau)d\tau} = \frac{\Lambda_f\left(1 - \frac{1}{\mathscr{R}_{0f}}\right)}{S_f^*\int_0^\infty \beta_m(\tau)\pi(\tau)d\tau},$$

where

$$\mathscr{R}_{0f} = \frac{\Lambda_f}{\mu S_f^*} = \frac{\Lambda_f}{\mu}\int_0^\infty \beta_m(\tau)\pi(\tau)d\tau,$$

$$\mathscr{R}_{0m} = \frac{\Lambda_m}{\mu S_m^*} = \frac{\Lambda_m}{\mu}\int_0^\infty \beta_f(a)\pi(a)da.$$

Thus, we define $\mathscr{R}_0$ as

$$\mathscr{R}_0 = \max\left(\mathscr{R}_{0f}, \mathscr{R}_{0m}\right) = \max\left(\frac{\Lambda_f}{\mu}\int_0^\infty \beta_m(\tau)\pi(\tau)d\tau, \frac{\Lambda_m}{\mu}\int_0^\infty \beta_f(a)\pi(a)da\right).$$

We can claim that the disease dies out if $\mathscr{R}_0 < 1$ and the unique endemic equilibrium exists when $\mathscr{R}_0 > 1$ in system (2.2).

4. Local stabilities of equilibria

Linearizing system (2.2) is to analyze the local stabilities of equilibria. We let $S_f(t) = S_f + x_f(t)$, $i_f(a, t) = i_f(a) + y_f(a, t)$, $S_m(t) = S_m + x_m(t)$ and $i_m(\tau, t) = i_m(\tau) + y_m(\tau, t)$, where $x_f(t)$, $y_f(a, t)$, $x_m(t)$ and $y_m(\tau, t)$ are the perturbations, and (S_f, $i_f(a)$, S_m, $i_m(\tau)$) denotes a generic equilibrium. Then we can obtain

$$\begin{aligned}
(S_f + x_f(t))' &= \Lambda_f - (S_f + x_f(t))\int_0^\infty \beta_m(\tau)(i_m(\tau) + y_m(\tau, t))d\tau - \mu(S_f + x_f(t)),\\
(i_f(a) + y_f(a,t))_a + (i_f(a) + y_f(a,t))_t &= -\delta(i_f(a) + y_f(a,t)),\\
(S_m + x_m(t))' &= \Lambda_m - (S_m + x_m(t))\int_0^\infty \beta_f(a)(i_f(a) + y_f(a,t))da - \mu(S_m + x_m(t)),\\
(i_m(\tau) + y_m(\tau,t))_\tau + (i_m(\tau) + y_m(\tau,t))_t &= -\delta(i_m(\tau) + y_m(\tau,t)),\\
i_f(0) + y_f(0,t) &= (S_f + x_f(t))\int_0^\infty \beta_m(\tau)(i_m(\tau) + y_m(\tau,t))d\tau,\\
i_m(0) + y_m(0,t) &= (S_m + x_m(t))\int_0^\infty \beta_f(a)(i_f(a) + y_f(a,t))da.
\end{aligned} \tag{4.1}$$

Using these formulas, we get

$$
\begin{aligned}
x_f'(t) &= \Lambda_f - S_f \int_0^\infty \beta_m(\tau) i_m(\tau) d\tau - S_f \int_0^\infty \beta_m(\tau) y_m(\tau,t) d\tau - x_f(t) \int_0^\infty \beta_m(\tau) i_m(\tau) d\tau \\
&\quad - x_f(t) \int_0^\infty \beta_m(\tau) y_m(\tau,t) d\tau - \mu S_f - \mu x_f(t), \\
i_{fa}(a) + y_{fa}(a,t) + y_{ft}(a,t) &= -\delta i_f(a) - \delta y_f(a,t), \\
x_m'(t) &= \Lambda_m - S_m \int_0^\infty \beta_f(a) i_f(a) da - S_m \int_0^\infty \beta_f(a) y_f(a,t) da - x_m(t) \int_0^\infty \beta_f(a) i_f(a) da \\
&\quad - x_m(t) \int_0^\infty \beta_f(a) y_f(a,t) da - \mu S_m - \mu x_m(t), \\
i_{m\tau}(\tau) + y_{m\tau}(\tau,t) + y_{mt}(\tau,t) &= -\delta i_m(\tau) - \delta y_m(\tau,t), \\
i_f(0) + y_f(0,t) &= S_f \int_0^\infty \beta_m(\tau) i_m(\tau) d\tau + S_f \int_0^\infty \beta_m(\tau) y_m(\tau,t) d\tau \\
&\quad + x_f(t) \int_0^\infty \beta_m(\tau) i_m(\tau) d\tau + x_f(t) \int_0^\infty \beta_m(\tau) y_m(\tau,t) d\tau, \\
i_m(0) + y_m(0,t) &= S_m \int_0^\infty \beta_f(a) i_f(a) da + S_m \int_0^\infty \beta_f(a) y_f(a,t) da \\
&\quad + x_m(t) \int_0^\infty \beta_f(a) i_f(a) da + x_m(t) \int_0^\infty \beta_f(a) y_f(a,t) da.
\end{aligned}
\tag{4.2}
$$

Using two techniques is to further simplify system (4.2). First, this approach simplifies equations for equilibria in system (3.1) to

$$
\begin{aligned}
x_f'(t) &= -S_f \int_0^\infty \beta_m(\tau) y_m(\tau,t) d\tau - x_f(t) \int_0^\infty \beta_m(\tau) i_m(\tau) d\tau \\
&\quad - x_f(t) \int_0^\infty \beta_m(\tau) y_m(\tau,t) d\tau \ - \mu x_f(t), \\
y_{fa}(a,t) + y_{ft}(a,t) &= -\delta y_f(a,t), \\
x_m'(t) &= -S_m \int_0^\infty \beta_f(a) y_f(a,t) da - x_m(t) \int_0^\infty \beta_f(a) i_f(a) da \\
&\quad - x_m(t) \int_0^\infty \beta_f(a) y_f(a,t) da - \mu x_m(t), \\
y_{m\tau}(\tau,t) + y_{mt}(\tau,t) &= -\delta y_m(\tau,t), \\
y_f(0,t) &= S_f \int_0^\infty \beta_m(\tau) y_m(\tau,t) d\tau + x_f(t) \int_0^\infty \beta_m(\tau) i_m(\tau) d\tau \\
&\quad + x_f(t) \int_0^\infty \beta_m(\tau) y_m(\tau,t) d\tau, \\
y_m(0,t) &= S_m \int_0^\infty \beta_f(a) y_f(a,t) da + x_m(t) \int_0^\infty \beta_f(a) i_f(a) da \\
&\quad + x_m(t) \int_0^\infty \beta_f(a) y_f(a,t) da.
\end{aligned}
\tag{4.3}
$$

Note that after this transformation, nonlinear system (4.3) contains only perturbation terms. Assuming that the perturbations are small, another technique for simplifying system (4.3) is to neglect much smaller quadratic terms. The linear system in terms of perturbations is

$$\begin{aligned}
x_f'(t) &= -S_f \int_0^\infty \beta_m(\tau) y_m(\tau,t) d\tau - x_f(t) \int_0^\infty \beta_m(\tau) i_m(\tau) d\tau - \mu x_f(t),\\
y_{fa}(a,t) + y_{ft}(a,t) &= -\delta y_f(a,t),\\
x_m'(t) &= -S_m \int_0^\infty \beta_f(a) y_f(a,t) da - x_m(t) \int_0^\infty \beta_f(a) i_f(a) da - \mu x_m(t),\\
y_{m\tau}(\tau,t) + y_{mt}(\tau,t) &= -\delta y_m(\tau,t),\\
y_f(0,t) &= S_f \int_0^\infty \beta_m(\tau) y_m(\tau,t) d\tau + x_f(t) \int_0^\infty \beta_m(\tau) i_m(\tau) d\tau,\\
y_m(0,t) &= S_m \int_0^\infty \beta_f(a) y_f(a,t) da + x_m(t) \int_0^\infty \beta_f(a) i_f(a) da.
\end{aligned} \tag{4.4}$$

Linear system (4.4) for $x_f(t)$, $y_f(a,t)$, $x_m(t)$ and $y_m(\tau,t)$ has exponential solutions like linear ordinary differential equations. Thus, it is possible to look for solutions $x_f(t) = x_f e^{\lambda t}$, $y_f(a,t) = y_f(a) e^{\lambda t}$, $x_m(t) = x_m e^{\lambda t}$ and $y_m(\tau,t) = y_m(\tau) e^{\lambda t}$, where x_f, $y_f(a)$, x_m, $y_m(\tau)$ and λ have to be determined that x_f, $y_f(a)$, x_m and $y_m(\tau)$ are not all zero. System for x_f, $y_f(a)$, x_m, $y_m(\tau)$ and λ (the bars have been omitted) is

$$\begin{aligned}
\lambda x_f &= -S_f \int_0^\infty \beta_m(\tau) y_m(\tau) d\tau - x_f \int_0^\infty \beta_m(\tau) i_m(\tau) d\tau - \mu x_f,\\
y_{fa}(a) + \lambda y_f(a) &= -\delta y_f(a),\\
\lambda x_m &= -S_m \int_0^\infty \beta_f(a) y_f(a) da - x_m \int_0^\infty \beta_f(a) i_f(a) da - \mu x_m,\\
y_{m\tau}(\tau) + \lambda y_m(\tau) &= -\delta y_m(\tau),\\
y_f(0) &= S_f \int_0^\infty \beta_m(\tau) y_m(\tau) d\tau + x_f \int_0^\infty \beta_m(\tau) i_m(\tau) d\tau,\\
y_m(0) &= S_m \int_0^\infty \beta_f(a) y_f(a) da + x_m \int_0^\infty \beta_f(a) i_f(a) da.
\end{aligned} \tag{4.5}$$

To obtain an equation for λ, we will eliminate x_f, $y_f(a)$, x_m and $y_m(\tau)$ by considering disease-free equilibrium and endemic equilibrium.

4.1. Local stability of disease-free equilibrium

Theorem 4.1. *If $\mathscr{R}_0 < 1$, disease-free equilibrium $\mathscr{E}^0$ is locally asymptotically stable. If $\mathscr{R}_0 > 1$, disease-free equilibrium $\mathscr{E}^0$ is unstable.*

Proof System (4.5) simplifies to the following system in disease-free equilibrium $\mathscr{E}^0$

$$\begin{aligned}
\lambda x_f &= -S_f^0 \int_0^\infty \beta_m(\tau) y_m(\tau) d\tau - \mu x_f,\\
y_{fa}(a) + \lambda y_f(a) &= -\delta y_f(a),\\
\lambda x_m &= -S_m^0 \int_0^\infty \beta_f(a) y_f(a) da - \mu x_m,\\
y_{m\tau}(\tau) + \lambda y_m(\tau) &= -\delta y_m(\tau),\\
y_f(0) &= S_f^0 \int_0^\infty \beta_m(\tau) y_m(\tau) d\tau,\\
y_m(0) &= S_m^0 \int_0^\infty \beta_f(a) y_f(a) da.
\end{aligned} \tag{4.6}$$

It is easy to see that the equations for $y_f(a)$ and $y_m(\tau)$ are independent of x_f and x_m, respectively. Solving the differential equations in system (4.6), we have

$$y_f(a) = y_f(0) e^{-\lambda a} \pi(a), \quad y_m(\tau) = y_m(0) e^{-\lambda \tau} \pi(\tau). \tag{4.7}$$

Substituting solutions (4.7) into boundary conditions of system (4.6), we obtain

$$y_f(0) = S_f^0 \int_0^\infty \beta_m(\tau) y_m(0) e^{-\lambda\tau} \pi(\tau) d\tau, \tag{4.8}$$

$$y_m(0) = S_m^0 \int_0^\infty \beta_f(a) y_f(0) e^{-\lambda a} \pi(a) da. \tag{4.9}$$

By (4.7) and $y_f(a,t) = y_f(a)e^{\lambda t}$, we could know that $y_f(0) \neq 0$. Similarly, $y_m(0) \neq 0$. Multiplying by equations (4.8) and (4.9), then canceling $y_f(0)y_m(0)$, we get characteristic equation

$$S_f^0 S_m^0 \int_0^\infty \beta_m(\tau) e^{-\lambda\tau} \pi(\tau) d\tau \int_0^\infty \beta_f(a) e^{-\lambda a} \pi(a) da = 1. \tag{4.10}$$

Equation (4.10) is a transcendental formula that can have many solutions. To prove that all solutions λ of equation (4.10) have negative real parts, the stability of disease-free equilibrium can be showed. Next, we define

$$\mathscr{G}(\lambda) = S_f^0 S_m^0 \int_0^\infty \beta_m(\tau) e^{-\lambda\tau} \pi(\tau) d\tau \int_0^\infty \beta_f(a) e^{-\lambda a} \pi(a) da.$$

If $\mathscr{R}_0 < 1$, then for all $\lambda = c + bi$ with $c \geq 0$, we have

$$\begin{aligned} |\mathscr{G}(\lambda)| &\leq S_f^0 S_m^0 \int_0^\infty \beta_m(\tau) |e^{-\lambda\tau}| \pi(\tau) d\tau \int_0^\infty \beta_f(a) |e^{-\lambda a}| \pi(a) da \\ &\leq S_f^0 S_m^0 \int_0^\infty \beta_m(\tau) e^{-c\tau} \pi(\tau) d\tau \int_0^\infty \beta_f(a) e^{-ca} \pi(a) da \leq \mathscr{R}_0 < 1. \end{aligned}$$

We conclude that when λ is a non-negative real part, it fails to satisfy $\mathscr{G}(\lambda) = 1$. So disease-free equilibrium is locally asymptotically stable with $\mathscr{R}_0 < 1$. If, alternatively, $\mathscr{R}_0 > 1$, endemic equilibrium $\mathscr{E}^*$ exists, system (2.2) could go to $\mathscr{E}^*$. Consequently, disease-free equilibrium is unstable in this case.

4.2. Local stability of endemic equilibrium

Now we turn to consider system (4.5) with endemic equilibrium to obtain its stability.

Theorem 4.2. *If $\mathscr{R}_0 > 1$, endemic equilibrium $\mathscr{E}^*$ is locally asymptotically stable.*

Proof From system (4.5) and equation (4.7), we obtain

$$\begin{aligned} \lambda x_f &= -S_f^* y_m(0) \int_0^\infty \beta_m(\tau) e^{-\lambda\tau} \pi(\tau) d\tau - x_f \int_0^\infty \beta_m(\tau) i_m^*(\tau) d\tau - \mu x_f, \\ \lambda x_m &= -S_m^* y_f(0) \int_0^\infty \beta_f(a) e^{-\lambda a} \pi(a) da - x_m \int_0^\infty \beta_f(a) i_f^*(a) da - \mu x_m, \\ y_f(0) &= S_f^* y_m(0) \int_0^\infty \beta_m(\tau) e^{-\lambda\tau} \pi(\tau) d\tau + x_f \int_0^\infty \beta_m(\tau) i_m^*(\tau) d\tau, \\ y_m(0) &= S_m^* y_f(0) \int_0^\infty \beta_f(a) e^{-\lambda a} \pi(a) da + x_m \int_0^\infty \beta_f(a) i_f^*(a) da. \end{aligned} \tag{4.11}$$

We notice that $\int_0^\infty \beta_m(\tau) i_m^*(\tau) d\tau$ and $\int_0^\infty \beta_f(a) i_f^*(a) da$ are positive numbers and we denote

$$B_f = \int_0^\infty \beta_m(\tau) i_m^*(\tau) d\tau, \quad B_m = \int_0^\infty \beta_f(a) i_f^*(a) da.$$

Requiring that the determinant be zero to solve system (4.11) to find a nontrivial solution

$$\begin{vmatrix} \lambda+\mu+B_f & 0 & 0 & S_f^*\int_0^\infty \beta_m(\tau)e^{-\lambda\tau}\pi(\tau)d\tau \\ -B_f & 1 & 0 & -S_f^*\int_0^\infty \beta_m(\tau)e^{-\lambda\tau}\pi(\tau)d\tau \\ 0 & S_m^*\int_0^\infty \beta_f(a)e^{-\lambda a}\pi(a)da & \lambda+\mu+B_m & 0 \\ 0 & -S_m^*\int_0^\infty \beta_f(a)e^{-\lambda a}\pi(a)da & -B_m & 1 \end{vmatrix} = 0.$$

Then we obtain

$$\begin{vmatrix} \lambda+\mu & 1 & 0 & 0 \\ -B_f & 1 & 0 & -S_f^*\int_0^\infty \beta_m(\tau)e^{-\lambda\tau}\pi(\tau)d\tau \\ 0 & 0 & \lambda+\mu & 1 \\ 0 & -S_m^*\int_0^\infty \beta_f(a)e^{-\lambda a}\pi(a)da & -B_m & 1 \end{vmatrix} = 0.$$

Expanding the determinant, we have

$$(\lambda+\mu)^2\left(1-S_f^*S_m^*\int_0^\infty \beta_m(\tau)e^{-\lambda\tau}\pi(\tau)d\tau\int_0^\infty \beta_f(a)e^{-\lambda a}\pi(a)da\right)+(\lambda+\mu)(B_f+B_m)+B_fB_m=0.$$

We shall know that the characteristic equation of endemic equilibrium is

$$1+\frac{B_f+B_m}{\lambda+\mu}+\frac{B_fB_m}{(\lambda+\mu)^2}=S_f^*S_m^*\int_0^\infty \beta_m(\tau)e^{-\lambda\tau}\pi(\tau)d\tau\int_0^\infty \beta_f(a)e^{-\lambda a}\pi(a)da. \tag{4.12}$$

We now show that equation (4.12) cannot have solutions λ with positive real part. Let $\lambda = c_1 + b_1 i$ with $c_1 \geq 0$, we get

$$\left|1+\frac{B_f+B_m}{\lambda+\mu}+\frac{B_fB_m}{(\lambda+\mu)^2}\right|>1.$$

On the other hand, for $c_1 \geq 0$ we have

$$\begin{aligned} &\left|S_f^*S_m^*\int_0^\infty \beta_m(\tau)e^{-\lambda\tau}\pi(\tau)d\tau\int_0^\infty \beta_f(a)e^{-\lambda a}\pi(a)da\right| \\ \leq\ & S_f^*S_m^*\int_0^\infty \beta_m(\tau)e^{|-\lambda\tau|}\pi(\tau)d\tau\int_0^\infty \beta_f(a)e^{|-\lambda a|}\pi(a)da \\ \leq\ & S_f^*S_m^*\int_0^\infty \beta_m(\tau)\pi(\tau)d\tau\int_0^\infty \beta_f(a)\pi(a)da=1. \end{aligned}$$

It indicates that the left-hand side is still strictly greater than 1, while the right-hand side is less than 1 for non-negative real part λ. Therefore, such λ does not satisfy the characteristic equation (4.12). We come to this conclusion: endemic equilibrium is locally asymptotically stable.

5. Global stabilities of equilibria

In this section, we use Lyapunov functions to demonstrate global stabilities of equilibria. First, solving the second equation along the characteristic curve $t - a =$ constant in system (2.2), we can get

$$i_f(a,t)=\begin{cases} i_{f0}(a-t)e^{-\delta t}, & a\geq t, \\ i_f(0,t-a)e^{-\delta a}, & a<t. \end{cases} \tag{5.1}$$

Similarly, we can get

$$i_m(\tau,t)=\begin{cases} i_{m0}(\tau-t)e^{-\delta t}, & \tau\geq t,\\ i_m(0,t-\tau)e^{-\delta\tau}, & \tau<t. \end{cases} \tag{5.2}$$

Then we know the function $h(z)=z-1-\ln z$, $z\in R_+$ has the global minimum at $z=1$ and $h(1)=0$.

5.1. Global stability of disease-free equilibrium

Theorem 5.1. *If $\mathscr{R}_0<1$, disease-free equilibrium $\mathscr{E}^0$ is globally asymptotically stable.*

Proof First, we define positive functions as

$$\xi_f(a)=\int_a^\infty S_m^0\beta_f(\theta)e^{-\int_a^\theta \delta d\sigma}d\theta,\quad \xi_m(\tau)=\int_\tau^\infty S_f^0\beta_m(l)e^{-\int_\tau^l \delta d\sigma}dl.$$

And we can know that

$$\xi_f(0)=\int_0^\infty S_m^0\beta_f(\theta)e^{-\int_0^\theta \delta d\sigma}d\theta=\mathscr{R}_{0m},$$

$$\xi_m(0)=\int_0^\infty S_f^0\beta_m(l)e^{-\int_0^l \delta d\sigma}dl=\mathscr{R}_{0f}.$$

The derivatives of $\xi_f(a)$ and $\xi_m(\tau)$ satisfy

$$\dot{\xi}_f(a)=-S_m^0\beta_f(a),\quad \dot{\xi}_m(\tau)=-S_f^0\beta_m(\tau).$$

Then we define a function $V(t)$

$$V(t)=V_{fs}(t)+V_{fi}(t)+V_{ms}(t)+V_{mi}(t),$$

let

$$V_{fs}(t)=S_f(t)-S_f^0-S_f^0\ln\frac{S_f(t)}{S_f^0},\quad V_{fi}(t)=\int_0^\infty \xi_f(a)i_f(a,t)da,$$

$$V_{ms}(t)=S_m(t)-S_m^0-S_m^0\ln\frac{S_m(t)}{S_m^0}\quad\text{and}\quad V_{mi}(t)=\int_0^\infty \xi_m(\tau)i_m(\tau,t)d\tau.$$

We could know that function $V(t)$ is nonnegatively defined for disease-free equilibrium $\mathscr{E}^0$, which has a global minimum. By (5.1), we have

$$V_{fi}(t)=\int_0^t \xi_f(t-r)i_f(0,r)e^{-\delta(t-r)}dr+\int_0^\infty \xi_f(t+r)i_{f0}(r)e^{-\delta t}dr.$$

And by (5.2), we obtain

$$V_{mi}(t)=\int_0^t \xi_m(t-r)i_m(0,r)e^{-\delta(t-r)}dr+\int_0^\infty \xi_m(t+r)i_{m0}(r)e^{-\delta t}dr.$$

Calculating the time derivatives of $V_{fs}(t)$, $V_{fi}(t)$, $V_{ms}(t)$ and $V_{mi}(t)$ along with system (2.2), respectively. We have

$$\dot{V}_{fs}(t) = -\frac{\mu(S_f(t) - S_f^0)^2}{S_f(t)} - i_f(0,t) + S_f^0 \int_0^\infty \beta_m(\tau) i_m(\tau,t) d\tau,$$

$$\dot{V}_{fi}(t) = \mathscr{R}_{0m} i_f(0,t) - S_m^0 \int_0^\infty \beta_f(a) i_f(a,t) da,$$

$$\dot{V}_{ms}(t) = -\frac{\mu(S_m(t) - S_m^0)^2}{S_m(t)} - i_m(0,t) + S_m^0 \int_0^\infty \beta_f(a) i_f(a,t) da,$$

and

$$\dot{V}_{mi}(t) = \mathscr{R}_{0f} i_m(0,t) - S_f^0 \int_0^\infty \beta_m(\tau) i_m(\tau,t) d\tau.$$

Thus, the time derivative of $V(t)$ is

$$\dot{V}(t) = -\frac{\mu(S_f(t) - S_f^0)^2}{S_f(t)} + (\mathscr{R}_{0m} - 1) i_f(0,t) - \frac{\mu(S_m(t) - S_m^0)^2}{S_m(t)} + (\mathscr{R}_{0f} - 1) i_m(0,t).$$

$\mathscr{R}_0 < 1$ ensures that $\mathscr{R}_{0f} < 1$ and $\mathscr{R}_{0m} < 1$ hold. Then $\dot{V}(t) \le 0$ for all $S_f(t)$, $i_f(0, t)$, $S_m(t)$ and $i_m(0, t) \ge 0$ with $\dot{V}(t) = 0$ only at $S_f(t) = S_f^0$, $S_m(t) = S_m^0$ and $i_f(0, t) = i_m(0, t) = 0$. Hence, it follows from the LaSalle invariance principle (Salle, 1976) that disease-free equilibrium $\mathscr{E}^0$ is globally asymptotically stable.

5.2. Global stability of endemic equilibrium

Theorem 5.2. *If $\mathscr{R}_0 > 1$, endemic equilibrium $\mathscr{E}^*$ is globally asymptotically stable.*

Proof First, we define positive functions as

$$\varphi_f(a) = \int_a^\infty S_m^* \beta_f(\theta) e^{-\int_a^\theta \delta d\sigma} d\theta, \quad \varphi_m(\tau) = \int_\tau^\infty S_f^* \beta_m(l) e^{-\int_\tau^l \delta d\sigma} dl.$$

And we can know that

$$\varphi_f(0) = \int_0^\infty S_m^* \beta_f(\theta) e^{-\int_0^\theta \delta d\sigma} d\theta = 1,$$

$$\varphi_m(0) = \int_0^\infty S_f^* \beta_m(l) e^{-\int_0^l \delta d\sigma} dl = 1.$$

The derivatives of $\varphi_f(a)$ and $\varphi_m(\tau)$ satisfy

$$\dot{\varphi}_f(a) = -S_m^* \beta_f(a), \quad \dot{\varphi}_m(\tau) = -S_f^* \beta_m(\tau).$$

Then we define a function $L(t)$ as $L(t) = L_{fs}(t) + L_{fi}(t) + L_{ms}(t) + L_{mi}(t)$, let

$$L_{fs}(t) = S_f(t) - S_f^* - S_f^* \ln \frac{S_f(t)}{S_f^*},$$

$$L_{fi}(t) = \int_0^\infty \varphi_f(a)\left(i_f(a,t) - i_f^*(a) - i_f^*(a)\ln\frac{i_f(a,t)}{i_f^*(a)}\right)da,$$

$$L_{ms}(t) = S_m(t) - S_m^* - S_m^* \ln \frac{S_m(t)}{S_m^*},$$

and

$$L_{mi}(t) = \int_0^\infty \varphi_m(\tau)\left(i_m(\tau,t) - i_m^*(\tau) - i_m^*(\tau)\ln\frac{i_m(\tau,t)}{i_m^*(\tau)}\right)d\tau.$$

We could know that function $L(t)$ is nonnegatively defined for endemic equilibrium $\mathscr{E}^*$, which has a global minimum. By (5.1), we obtain

$$\begin{aligned} L_{fi}(t) \quad &= \int_0^t \varphi_f(t-r)\left(i_f(0,r)e^{-\delta(t-r)} - i_f^*(t-r) - i_f^*(t-r)\ln\frac{i_f(0,r)e^{-\delta(t-r)}}{i_f^*(t-r)}\right)dr \\ &+ \int_0^\infty \varphi_f(t+r)\left(i_{f0}(r)e^{-\delta t} - i_f^*(t+r) - i_f^*(t+r)\ln\frac{i_{f0}(r)e^{-\delta t}}{i_f^*(t+r)}\right)dr. \end{aligned}$$

And by (5.2), we get

$$\begin{aligned} L_{mi}(t) \quad &= \int_0^t \varphi_m(t-r)\left(i_m(0,r)e^{-\delta(t-r)} - i_m^*(t-r) - i_m^*(t-r)\ln\frac{i_m(0,r)e^{-\delta(t-r)}}{i_m^*(t-r)}\right)dr \\ &+ \int_0^\infty \varphi_m(t+r)\left(i_{m0}(r)e^{-\delta t} - i_m^*(t+r) - i_m^*(t+r)\ln\frac{i_{m0}(r)e^{-\delta t}}{i_m^*(t+r)}\right)dr. \end{aligned}$$

Calculating the time derivatives of $L_{fs}(t)$, $L_{fi}(t)$, $L_{ms}(t)$ and $L_{mi}(t)$ along with system (2.2), respectively. We have

$$\dot{L}_{fs}(t) = -\frac{\mu(S_f(t) - S_f^*)^2}{S_f(t)} - \frac{S_f^*}{S_f(t)}i_f^*(0) + \frac{S_f^*}{S_f(t)}i_f(0,t) + i_f^*(0) - i_f(0,t),$$

$$\dot{L}_{fi}(t) = i_f(0,t) - i_f^*(0) - i_f^*(0)\ln\frac{i_f(0,t)}{i_f^*(0)} - \int_0^\infty S_m^*\beta_f(a)\left(i_f(a,t) - i_f^*(a) - i_f^*(a)\ln\frac{i_f(a,t)}{i_f^*(a)}\right)da,$$

$$\dot{L}_{ms}(t) = -\frac{\mu(S_m(t) - S_m^*)^2}{S_m(t)} - \frac{S_m^*}{S_m(t)}i_m^*(0) + \frac{S_m^*}{S_m(t)}i_m(0,t) + i_m^*(0) - i_m(0,t),$$

and

$$\dot{L}_{mi}(t) = i_m(0,t) - i_m^*(0) - i_m^*(0)\ln\frac{i_m(0,t)}{i_m^*(0)} - \int_0^\infty S_f^*\beta_m(\tau)\left(i_m(\tau,t) - i_m^*(\tau) - i_m^*(\tau)\ln\frac{i_m(\tau,t)}{i_m^*(\tau)}\right)d\tau.$$

Thus, the time derivative of $L(t)$ is

$$\begin{aligned} \dot{L}(t) = \quad &-\frac{\mu(S_f(t) - S_f^*)^2}{S_f(t)} + \int_0^\infty S_f^*\beta_m(\tau)i_m^*(\tau)\left(1 + \ln\frac{i_m(\tau,t)}{i_m^*(\tau)} - \frac{S_f^*}{S_f(t)} - \ln\frac{i_f(0,t)}{i_f^*(0)}\right)d\tau \\ &-\frac{\mu(S_m(t) - S_m^*)^2}{S_m(t)} + \int_0^\infty S_m^*\beta_f(a)i_f^*(a)\left(1 + \ln\frac{i_f(a,t)}{i_f^*(a)} - \frac{S_m^*}{S_m(t)} - \ln\frac{i_m(0,t)}{i_m^*(0)}\right)da. \end{aligned}$$

By

$$\int_0^\infty S_f^*\beta_m(\tau)i_m^*(\tau)\left(1 - \frac{S_f(t)i_m(\tau,t)i_f^*(0)}{S_f^*i_m^*(\tau)i_f(0,t)}\right)d\tau = 0,$$

$$\int_0^\infty S_m^*\beta_f(a)i_f^*(a)\left(1-\frac{S_m(t)i_f(a,t)i_m^*(0)}{S_m^*i_f^*(a)i_m(0,t)}\right)da=0,$$

we have

$$\begin{aligned}\dot{L}(t)=&-\frac{\mu(S_f(t)-S_f^*)^2}{S_f(t)}-\frac{\mu(S_m(t)-S_m^*)^2}{S_m(t)}\\&+\int_0^\infty S_f^*\beta_m(\tau)i_m^*(\tau)\left(1+\ln\frac{S_f^*}{S_f(t)}-\frac{S_f^*}{S_f(t)}+1+\ln\frac{S_f(t)i_m(\tau,t)i_f^*(0)}{S_f^*i_m^*(\tau)i_f(0,t)}-\frac{S_f(t)i_m(\tau,t)i_f^*(0)}{S_f^*i_m^*(\tau)i_f(0,t)}\right)d\tau\\&+\int_0^\infty S_m^*\beta_f(a)i_f^*(a)\left(1+\ln\frac{S_m^*}{S_m(t)}-\frac{S_m^*}{S_m(t)}+1+\ln\frac{S_m(t)i_f(a,t)i_m^*(0)}{S_m^*i_f^*(a)i_m(0,t)}-\frac{S_m(t)i_f(a,t)i_m^*(0)}{S_m^*i_f^*(a)i_m(0,t)}\right)da.\end{aligned}$$

We can know that $L(t)$ has nonpositive derivative. Furthermore, the equality $\dot{L}(t)=0$ holds if and only if $S_f(t)=S_f^*$, $i_f(a,t)=i_f^*(a)$, $i_f(0,t)=i_f^*(0)$, $S_m(t)=S_m^*$, $i_m(\tau,t)=i_m^*(\tau)$ and $i_m(0,t)=i_m^*(0)$. Hence, it follows from the LaSalle invariance principle (Salle, 1976) that $\mathscr{E}^*$ is globally asymptotically stable.

6. Numerical simulations and sensitivity analysis of parameters for $\mathscr{R}_0$

In this section, we illustrate the disease dynamics of age-infection-structured HIV model with heterogeneous transmission in system (2.2) by performing numerical simulations. In the numerical simulation process, time and infection age are taken as units of years. Following (Mukandavire et al., 2009), we fix the coefficients: $\mu=0.013$ and $\alpha=0.01$. To further evaluate the impact of each parameter(γ, μ, α, Λ_f, Λ_m, $\beta_f(a)$ and $\beta_m(\tau)$) on $\mathscr{R}_0$, sensitivity analysis of parameters is performed by the method of partial rank correlation coefficient. Next, we take four special cases of the basic reproduction number to study how $\mathscr{R}_0$ affect the density and number of infectious female and male individuals.

Case 1. $\mathscr{R}_{0f}<1$ and $\mathscr{R}_{0m}<1$.

The infection rate function $\beta_f(a)$ is

$$\beta_f(a)=\begin{cases}0.0275{*}10^{-5.08}, & a<15,\\ 0.0275{*}10^{-5.08}+0.0489(a-15)e^{-0.98(a-30)^2}{*}10^{-5.08}, & 15\le a<45,\\ 0.0489{*}10^{-5.08}, & a\ge 45.\end{cases}$$

and $\beta_m(\tau)$ is

$$\beta_m(\tau)=\begin{cases}0.03{*}10^{-5.08}, & \tau<15,\\ 0.03{*}10^{-5.08}+0.05(\tau-15)e^{-0.9(\tau-30)^2}{*}10^{-5.08}, & 15\le \tau<45,\\ 0.05{*}10^{-5.08}, & \tau\ge 45.\end{cases}$$

The infection rate functions chart is displayed in Fig. 2.

Fig. 3(a) and (f) show the three-dimensional diagrams with respect to infection-age and time of infectious female and infectious male, respectively. From Fig. 3(b) and (c)(3(g) and 3(h)), we can see that the density and number of infectious female(male) are all gradually falling to zero. From Fig. 3(d) and (e)(3(i) and 3(j)), we can see that the density and number of infectious female(male) are quickly rising to the peak first and then slowly falling to zero.

Fig. 4 shows that Λ_f, Λ_m, $\beta_f(a)$ and $\beta_m(\tau)$ are positive influence, and μ, α, γ are negative influence on $\mathscr{R}_0$. Among these parameters, γ, μ and α are more negative impacts on $\mathscr{R}_0$. Thus, reducing Λ_m and $\beta_m(\tau)$ to more effectively control AIDS spread.

Case 2. $\mathscr{R}_{0f}<1$ and $\mathscr{R}_{0m}>1$.

The infection rate function $\beta_f(a)$ is

$$\beta_f(a)=\begin{cases}0.23{*}10^{-5}, & a<15,\\ 0.23{*}10^{-5}+0.348(a-15)e^{-0.98(a-30)^2}{*}10^{-5}, & 15\le a<45,\\ 0.348{*}10^{-5}, & a\ge 45.\end{cases}$$

and $\beta_m(\tau)$ is

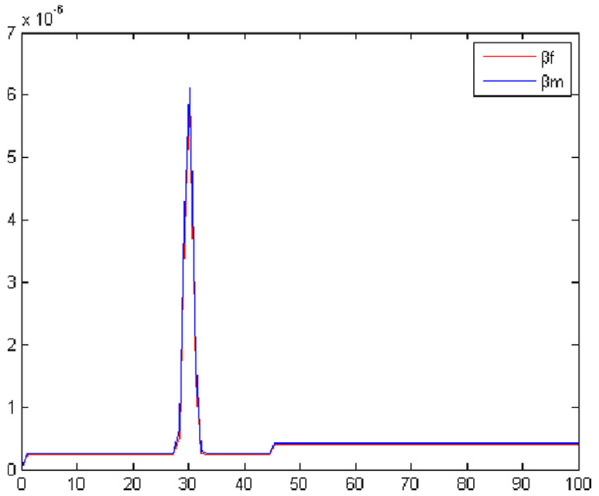

Fig. 2. The infection rate functions.

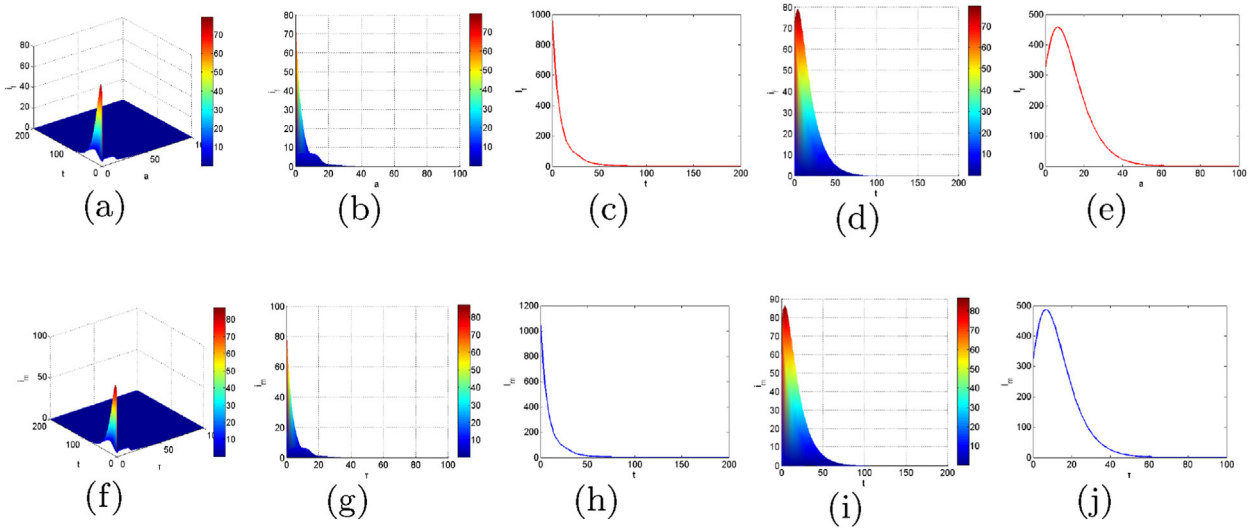

Fig. 3. $\mathscr{R}_{0f} = 0.9014$ and $\mathscr{R}_{0m} = 0.9104$, where $\gamma = 0.125$, $\Lambda_f = 6000$, $\Lambda_m = 6600$, $S_f(0) = 12675$, $S_m(0) = 13314$, $i_f(a, 0) = 43(a + 3)e^{-0.2(a+3)}$, $i_m(\tau, 0) = 47(\tau + 3)e^{-0.2(\tau+3)}$. (a) $i_f(a, t)$ changes with a and t. (b) $i_f(a, t)$ changes with a. (c) $I_f(t)$ changes with t. (d) $i_f(a, t)$ changes with t. (e) $I_f(a)$ changes with a. (f) $i_m(\tau, t)$ changes with τ and t. (g) $i_m(\tau, t)$ changes with τ. (h) $I_m(t)$ changes with t. (i) $i_m(\tau, t)$ changes with t. (j) $I_m(\tau)$ changes with τ.

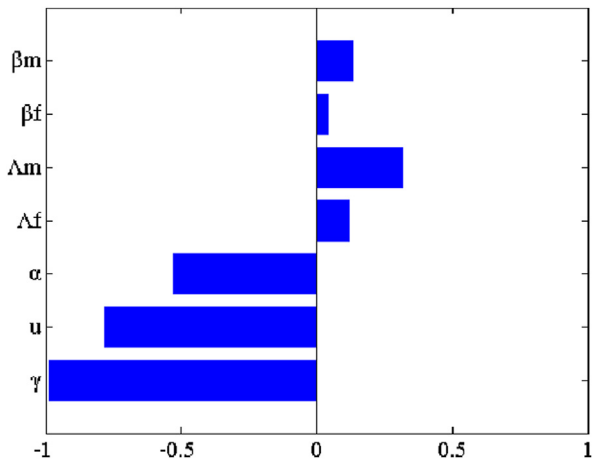

Fig. 4. Tornado plot of PRCCs in regard to $\mathscr{R}_0$.

$$\beta_m(\tau) = \begin{cases} 0.015*10^{-5}, & \tau < 15, \\ 0.015*10^{-5} + 0.029(\tau - 15)e^{-0.9(\tau-30)^2}*10^{-5}, & 15 \le \tau < 45, \\ 0.029*10^{-5}, & \tau \ge 45. \end{cases}$$

The infection rate functions chart is displayed in Fig. 5.

Fig. 6(a) and (f) show the three-dimensional diagrams with respect to infection-age and time of infectious female and infectious male, respectively. From Fig. 6(b) and (c), we can see that the density and number of infectious female are firstly decreasing, quickly rising to the peak and then slowly falling to steady state. From 6(g) and 6(h), we can see that the density and number of infectious male are slowly rising to the peak and then gradually falling to steady state. From Fig. 6(d) and (e)(6(i) and 6(j)), we can see that the density and number of infectious female(male) are gradually decreasing.

Fig. 7 shows that Λ_f, Λ_m, $\beta_f(a)$ and $\beta_m(\tau)$ are positive influence, and μ, α, γ are negative influence on $\mathscr{R}_0$. Among these parameters, γ, μ and α are more negative impacts on $\mathscr{R}_0$. Thus, reducing Λ_m and $\beta_f(a)$ to more effectively control AIDS spread.

Case 3. $\mathscr{R}_{0f} > 1$ and $\mathscr{R}_{0m} < 1$.

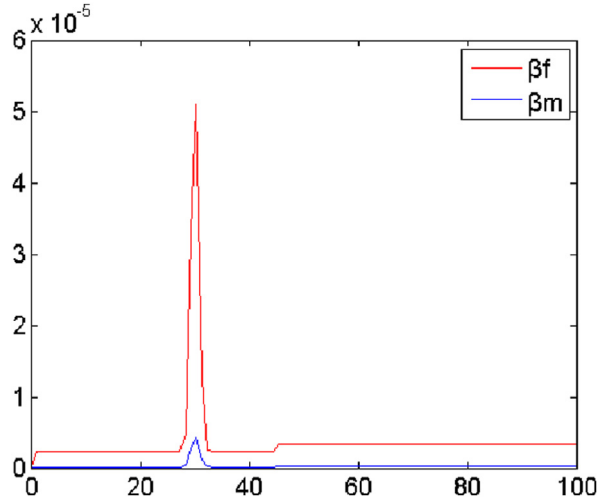

Fig. 5. The infection rate functions.

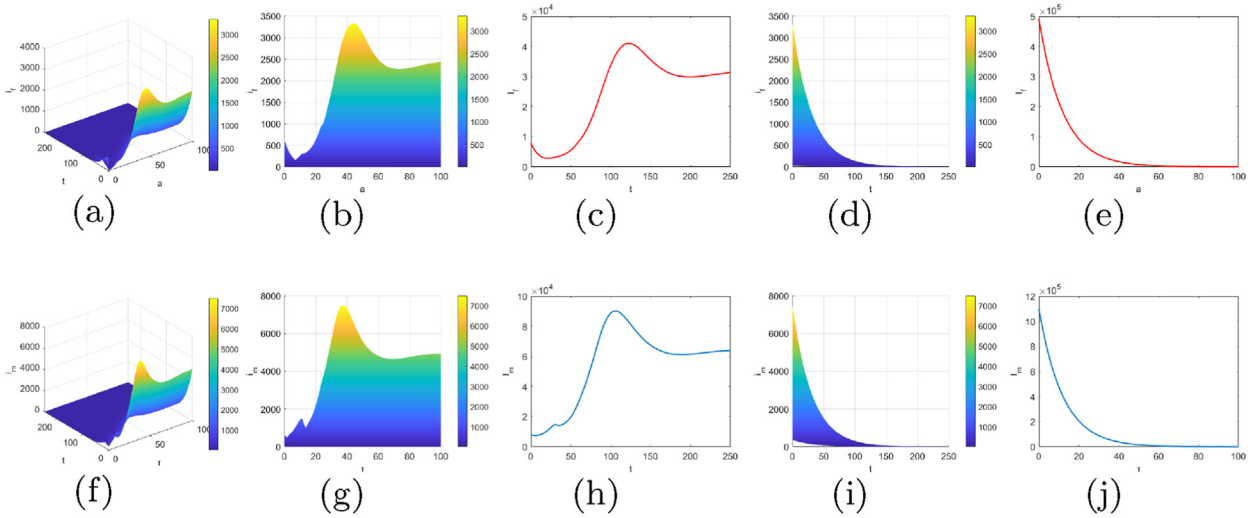

Fig. 6. $\mathscr{R}_{0f} = 0.9814$ and $\mathscr{R}_{0m} = 15.8968$, where $\gamma = 0.0601$, $\Lambda_f = 4920$, $\Lambda_m = 5600$, $S_f(0) = 12675$, $S_m(0) = 13314$, $i_f(a, 0) = 345(a + 3)e^{-0.2(a+3)}$, $i_m(\tau, 0) = 360(\tau + 3)e^{-0.2(\tau+3)}$. (a) $i_f(a, t)$ changes with a and t. (b) $i_f(a, t)$ changes with a. (c) $I_f(t)$ changes with t. (d) $i_f(a, t)$ changes with t. (e) $I_f(a)$ changes with a. (f) $i_m(\tau, t)$ changes with τ and t. (g) $i_m(\tau, t)$ changes with τ. (h) $I_m(t)$ changes with t. (i) $i_m(\tau, t)$ changes with t. (j) $I_m(\tau)$ changes with τ.

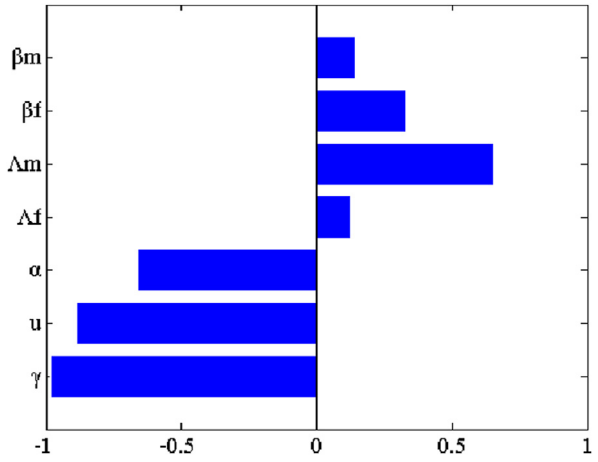

Fig. 7. Tornado plot of PRCCs in regard to $\mathscr{R}_0$.

The infection rate function $\beta_f(a)$ is

$$\beta_f(a) = \begin{cases} 0.03*10^{-5.32}, & a<15, \\ 0.03*10^{-5.32} + 0.048(a-15)e^{-0.98(a-30)^2}*10^{-5.32}, & 15 \le a<45, \\ 0.048*10^{-5.32}, & a \ge 45. \end{cases}$$

and $\beta_m(\tau)$ is

$$\beta_m(\tau) = \begin{cases} 0.25*10^{-5}, & \tau<15, \\ 0.25*10^{-5} + 0.49(\tau-15)e^{-0.9(\tau-30)^2}*10^{-5}, & 15 \le \tau<45, \\ 0.49*10^{-5}, & \tau \ge 45. \end{cases}$$

The infection rate functions chart is displayed in Fig. 8.

Fig. 9(a) and (f) show the three-dimensional diagrams with respect to infection-age and time of infectious female and infectious male, respectively. From Fig. 9(b) and (c), we can see that the density and number of infectious female are slowly rising to the peak and then gradually falling to steady state. From 9(g) and 9(h), we can see that the density and number of infectious male are firstly decreasing, quickly rising to the peak and then slowly falling to steady state. From Fig. 9(d) and (e)(9(i) and 9(j)), we can see that the density and number of infectious female(male) are gradually decreasing.

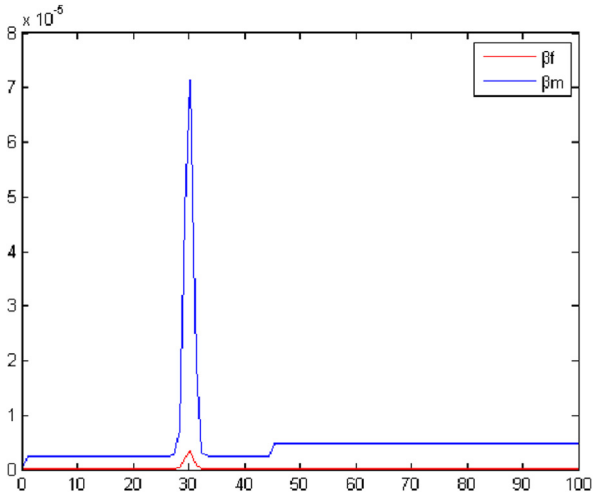

Fig. 8. The infection rate functions.

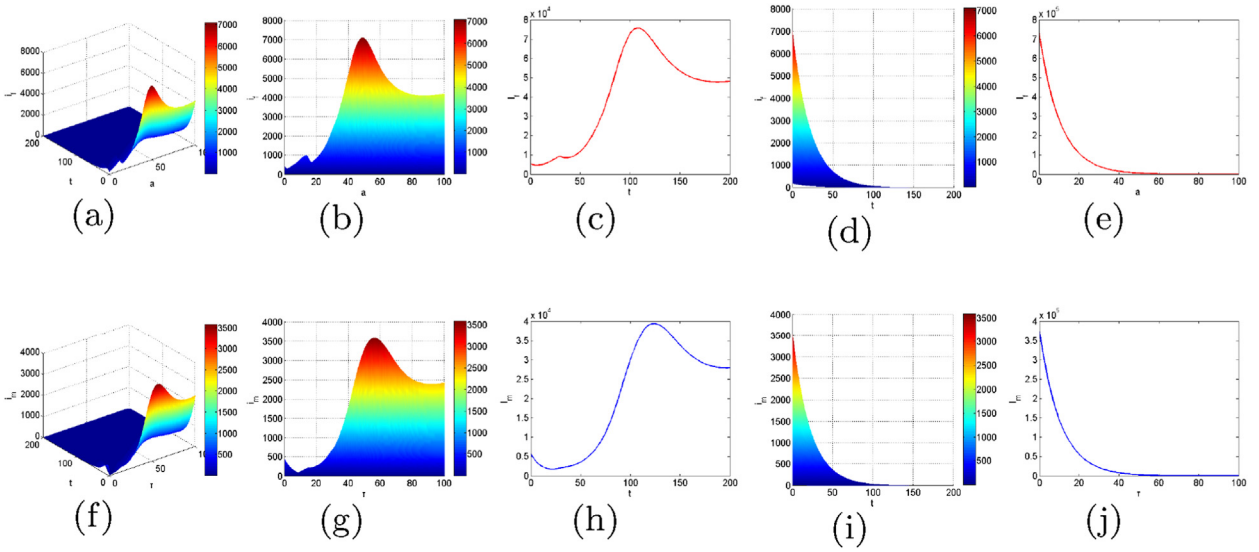

Fig. 9. $\mathscr{R}_{0f} = 13.6832$ and $\mathscr{R}_{0m} = 0.9990$, where $\gamma = 0.0715$, $\Lambda_f = 4920$, $\Lambda_m = 6600$, $S_f(0) = 7675$, $S_m(0) = 8314$, $i_f(a, 0) = 245(a + 3)e^{-0.2(a+3)}$, $i_m(\tau, 0) = 260(\tau + 3)e^{-0.2(\tau+3)}$. (a) $i_f(a, t)$ changes with a and t. (b) $i_f(a, t)$ changes with a. (c) $I_f(t)$ changes with t. (d) $i_f(a, t)$ changes with t. (e) $I_f(a)$ changes with a. (f) $i_m(\tau, t)$ changes with τ and t. (g) $i_m(\tau, t)$ changes with τ. (h) $I_m(t)$ changes with t. (i) $i_m(\tau, t)$ changes with t. (j) $I_m(\tau)$ changes with τ.

Fig. 10 shows that Λ_f, Λ_m, $\beta_f(a)$ and $\beta_m(\tau)$ are positive influence, and μ, α, γ are negative influence on $\mathscr{R}_0$. Among these parameters, γ, μ and α are more negative impacts on $\mathscr{R}_0$. Thus, reducing Λ_f, Λ_m and $\beta_m(\tau)$ to more effectively control AIDS spread.

Case 4. $\mathscr{R}_{0f} > 1$ and $\mathscr{R}_{0m} > 1$.

The infection rate function $\beta_f(a)$ is

$$\beta_f(a) = \begin{cases} 0.26*10^{-5}, & a < 15, \\ 0.26*10^{-5} + 0.68(a-15)e^{-0.98(a-30)^2}*10^{-5}, & 15 \le a < 45, \\ 0.68*10^{-5}, & a \ge 45. \end{cases}$$

and $\beta_m(\tau)$ is

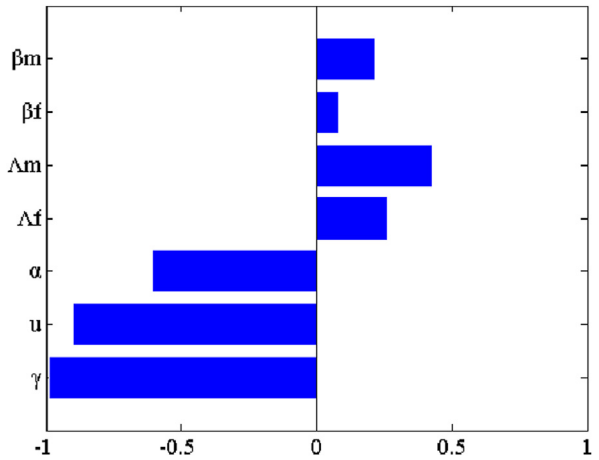

Fig. 10. Tornado plot of PRCCs in regard to $\mathscr{R}_0$.

$$\beta_m(\tau) = \begin{cases} 0.35*10^{-5}, & \tau < 15, \\ 0.35*10^{-5} + 0.79(\tau - 15)e^{-0.9(\tau-30)^2}*10^{-5}, & 15 \le \tau < 45, \\ 0.79*10^{-5}, & \tau \ge 45. \end{cases}$$

The infection rate functions chart is displayed in Fig. 11.

Fig. 12(a) and (f) show the three-dimensional diagrams with respect to infection-age and time of infectious female and infectious male, respectively. From Fig. 12(b) and (c)(9(g) and 9(h)), we can see that the density and number of infectious female(male) are firstly decreasing, quickly rising to the peak and then slowly falling to steady state. From Fig. 12(d) and (e)(12(i) and 12(j)), we can see that the density and number of infectious female(male) are gradually decreasing.

Fig. 13 shows that Λ_f, Λ_m, $\beta_f(a)$ and $\beta_m(\tau)$ are positive influence, and μ, α, γ are negative influence on $\mathscr{R}_0$. Among these parameters, γ, μ and α are more negative impacts on $\mathscr{R}_0$. Thus, reducing Λ_f and Λ_m to more effectively control AIDS spread.

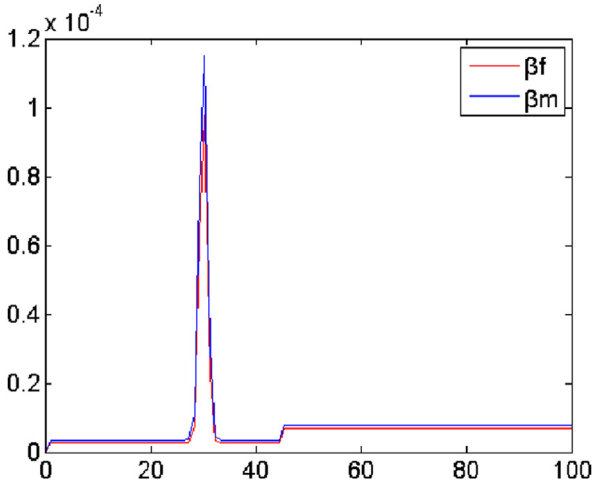

Fig. 11. The infection rate functions.

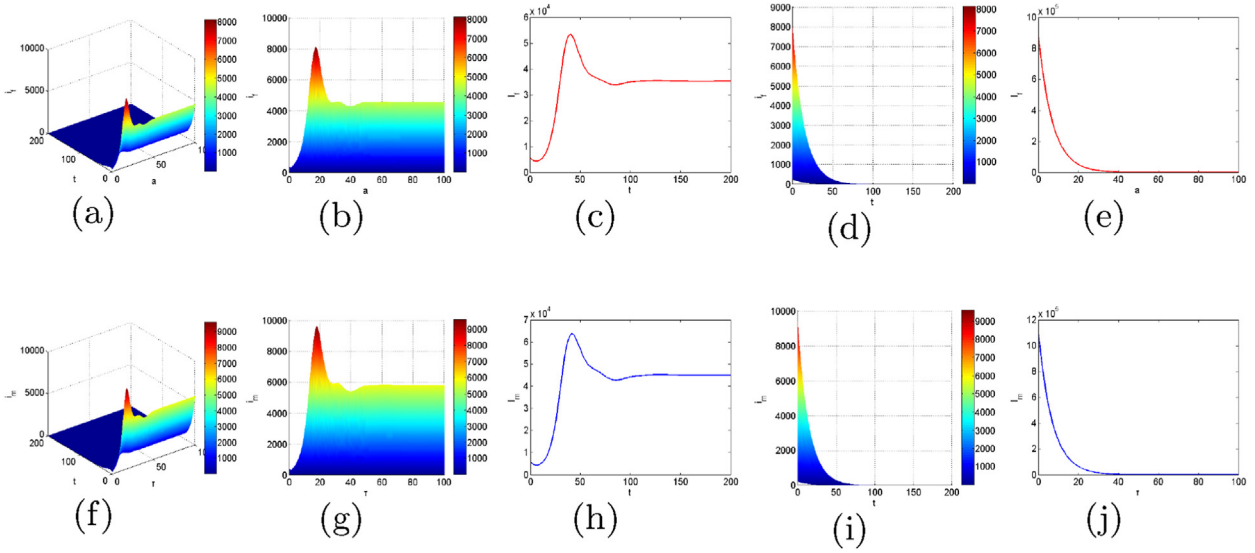

Fig. 12. $\mathscr{R}_{0f} = 10.6332$ and $\mathscr{R}_{0m} = 10.7094$, where $\gamma = 0.125$, $\Lambda_f = 4920$, $\Lambda_m = 6600$, $S_f(0) = 7675$, $S_m(0) = 8314$, $i_f(a, 0) = 245(a + 3)e^{-0.2(a+3)}$, $i_m(\tau, 0) = 260(\tau + 3)e^{-0.2(\tau+3)}$. (a) $i_f(a, t)$ changes with a and t. (b) $i_f(a, t)$ changes with a. (c) $I_f(t)$ changes with t. (d) $i_f(a, t)$ changes with t. (e) $I_f(a)$ changes with a. (f) $i_m(\tau, t)$ changes with τ and t. (g) $i_m(\tau, t)$ changes with τ. (h) $I_m(t)$ changes with t. (i) $i_m(\tau, t)$ changes with t. (j) $I_m(\tau)$ changes with τ.

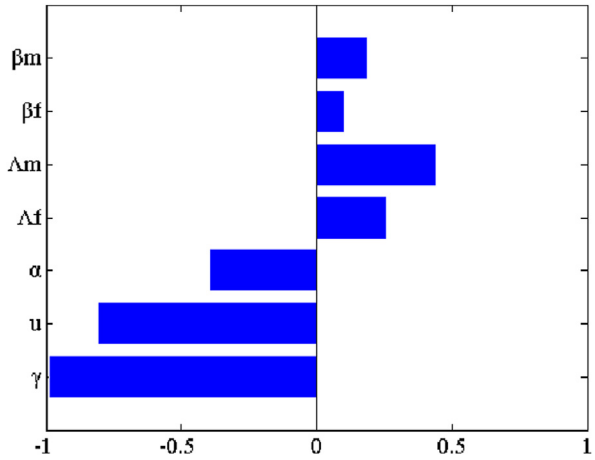

Fig. 13. Tornado plot of PRCCs in regard to $\mathscr{R}_0$.

7. Conclusions and discussion

In this paper, we mainly focus on the derivation and analysis of an age-infection-structured HIV model with heterogeneous transmission. Under our assumptions, based on the characteristics of system (2.2), we obtain it is bounded. And the local and global dynamics are shown to be completely determined by the basic reproduction number. The disease dies out if $\mathscr{R}_0$ is less than one, otherwise the disease exists. Local stabilities of disease-free equilibrium and endemic equilibrium can be proved by linearizing systems at their equilibria. Meanwhile, we construct two Lyapunov functions to show that the global stabilities of equilibria. Finally, we take four special cases of the basic reproduction number to study how $\mathscr{R}_0$ affect the density and number of infectious female and male individuals to support theoretical results and sensitivity analysis is performed by the method of partial rank correlation coefficient to observe the significance of each parameter for $\mathscr{R}_0$. By sensitivity analysis of parameters, we could know that γ, μ and α are more negative impacts on $\mathscr{R}_0$ and we could reduce recruitment rate into population and infection incidence to more effectively control AIDS spread.

The establishment of HIV dynamic model by considering age-infection and heterogeneous transmission is helpful to understand HIV transmission process. It improves the application value of HIV model in practice and provides clearer guidance for taking preventive measures. However, this paper does not consider HIV other transmission modes such as bisexual and vertical transmission. System (2.2) also applies only to sexually transmitted diseases and it is not universal. Furthermore, we will consider other influencing factors such as the different mortalities between female and male individuals, chronological age and bisexuals transmission which help to more accurately analyze HIV transmission dynamics and apply to other sexually transmitted diseases.

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CRediT authorship contribution statement

Juping Zhang: Funding acquisition, Investigation, Methodology, Project administration, Writing – original draft, Writing – review & editing. **Linlin Wang:** Methodology, Software, Writing – original draft, Writing – review & editing. **Zhen Jin:** Investigation, Methodology, Project administration, Writing – review & editing.

Declaration of competing interest

The authors have declared no conflict of interest.

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