Threshold dynamics of an age-structured infectious disease model with limited medical resources

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Abstract

In this paper, an age-structured infectious disease dynamical model that considers two diseases simultaneously but with limited medical resources is proposed and analysed. The asymptotic smoothness and persistence of the solution semi-flow are investigated. Then conditions for the existence of a global attractor are derived, which means that disease persists when $\Re_0 > 1$. By using a Lyapunov function, it is shown that the infection-free equilibrium is globally asymptotically stable if $\Re_0 < 1$ and the infection equilibrium is globally asymptotically stable if $\Re_0 > 1$. In the presence of limited medical resources, the results suggest that equitable distribution for the limited medical resources is significant when treating low-risk and high-risk diseases and that keeping a resource sharing coefficient at a moderate level helps to eliminate the disease.

Keywords: Age-structured model, Limited medical resources, Global stability, Lyapunov function

1. Introduction

Many infectious diseases, such as SARS [1], influenza [2], Lassa Fever [3], COVID-19 [4–8] etc., have seriously endangered human health. According to the World Health Report [9], more than 448 million people have been diagnosed with COVID-19 and 6 million people have died from it. At present,

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the number of infections is still increasing, which has aroused extensive attention on the prevention and control of infectious diseases all over the world. Therefore, different types of mathematical models have been established in order to study the pathogenesis of infectious diseases and the transmission mechanisms, so as to prevent the outbreak of diseases.

As early as 1927, Kermack and McKendrick constructed the famous SIR compartment model to study the transmission dynamics of Black Death and plague [10], where S is the susceptible class, I is the infected class, and R is the recovered and removed class. They proposed the "threshold theory" to establish if there will be a disease outbreak or not. Subsequently, many dynamical models were put forward to study different infectious diseases [11–19], emphasising that when emerging infectious diseases break out there are many different factors that affect their spread [20–25], especially medical resources and triaging pressure. In the initial stages of outbreaks, a hospital will have sufficient medical resources to treat patients infected with several infectious diseases. However, with the rapid spread of high-risk diseases, the patients then pose a threat to public health. Meanwhile, because of limited medical resources (such as doctors, beds, drugs, equipment etc.), medical staff will face the pressure of triaging, i.e., choosing between treating patients with high or low risk diseases, which restricts the prevention and control of infectious diseases to a certain extent.

In order to study the impact of limited medical resources on the spread of infectious diseases, Wang proposed an SIR model with limited treatment resources [20], the results of which showed that insufficient treatment capacity would lead to backward bifurcation and a bistable local equilibrium was also observed due to limited resources. Al-sheikh discussed a class of SEIR epidemic model with limited treatment resources, and analyzed the existence and stability of infection-free and infection equilibria [21]. To describe the impact of medical resources (hospital beds) on infectious diseases, Shan and Zhu established a SIR model with standard incidence and a nonlinear recovery rate [22]. It was found that maintaining enough beds is very important for controlling infectious diseases. A threshold strategy was also proposed to study the effects of limited medical resources and triaging pressure, i.e., if the number of high-risk infectious diseases is lower than a threshold level, then medical resources are not limited and there is no triaging pressure, but above the threshold doctors had to treat only high-risk infectious diseases due to limited resources [23–26]. In particular, Qin and Tang proposed a series of non-smooth infectious disease models with triaging pressure and pointed out that in the presence of limited medical resources triaging pressure contributed to the prevention of emerging infectious diseases [23]. In addition, they further concluded that reducing the infection threshold, treating patients promptly or increasing the maximum cure rate were helpful for controlling emerging infectious diseases [24].

However, although the above studies investigated the epidemic dynamics of emerging infectious diseases from a different point of view, they ignored the effects of the time when a susceptible individual became infected, that is, the age of infection. In reality, a disease may have different infection, mortality and recovery rates amongst different age groups, so it is more realistic to use age-structured dynamical systems to describe the influence of infection age. Magal proposed and studied an infection age model for disease transmission, and obtained the global stability of the two equilibria [15]. Soufiane further considered a model with infection age and nonlinear incidence rate [27]. Subsequently, Chekroun noticed that the recovered individuals were re-infected and studied a new infection age model [28]. But they have not taken into account the impact of medical resources on disease transmission and treatment. In fact, the frequent occurrence of emerging infectious diseases can lead to some social problems, such as shortage of medical resources and triaging pressure. Therefore, we establish a novel age-structured model based on emerging infectious diseases, focusing on the following three topics: (1) the impacts of limited medical resources on the spread of low-risk diseases and high-risk diseases; (2) how triaging pressure affects the outbreaks of emerging infectious diseases and (3) the prevention and control of infectious diseases.

This paper is organized as follows. In Section 2, we propose an age-structured infectious disease model with limited medical resources. Preliminaries and properties of solutions are carried out in Section 3, including showing the existence of steady states and the asymptotic smoothness of the semi-flow. In Section 4, the local stability of both an infection-free equilibrium and an infection equilibrium of system (3) is studied, the uniform persistence of the solution semi-flow is also proved under certain conditions and then the global stability is explored by constructing Lyapunov functions. In section 5, numerical simulations are presented and biological implications are discussed. The paper ends with conclusions in section 6.

2. Model formulation

Once several infectious diseases appear at the same time, there will be insufficient medical resources such that medical staff will face pressure to choose treatment in the crowd. Qin and Tang developed the following dynamical system to describe the selection pressure when both SARS and influenza (flu) cases occur [23],

$$\left\{ \begin{array}{l} \frac{dS(t)}{dt} = A - \mu_S S - \beta_1 S I_1 - \beta_2 S I_2, \\ \frac{dI_1(t)}{dt} = \beta_1 S I_1 - \mu_1 I_1 - v_1 I_1 - \frac{p_1 c_1 I_1}{1+p_1 b_1 I_1 + p_2 b_2 I_2}, \\ \frac{dI_2(t)}{dt} = \beta_2 S I_2 - \mu_2 I_2 - v_2 I_2 - \frac{p_2 c_2 I_2}{1+p_1 b_1 I_1 + p_2 b_2 I_2}, \\ \frac{dR(t)}{dt} = v_1 I_1 + v_2 I_2 + \frac{p_1 c_1 I_1 + p_2 c_2 I_2}{1+p_1 b_1 I_1 + p_2 b_2 I_2} - \mu_R R, \end{array} \right. \tag{1}$$

where $S(t)$ and $R(t)$ denote the number of susceptible and recovered individuals at time t, $I_1(t)$ and $I_2(t)$ denote the number of individuals infected by SARS and flu at time t, respectively. A is the recruitment rate of susceptible individuals, μ_S and μ_R are death rates for susceptible and recovered individuals, β_i denotes the infection constant of I_i, μ_i presents the death rate of I_i, p_i is the treating probability of I_i, c_i is the maximum recovery rate of I_i, b_i shows the effects of medical resource limitation on the treatment for I_i, v_i denotes the recovery rate for I_i, i=1,2.

At the beginning of the SARS outbreaks, there were enough medical resources to treat both flu patients and SARS patients at the same time. But with the fast spreading of SARS, the limited medical resources could were inadequate to deal with the increasing number of SARS cases, and whether flu cases were treated or not depended on a threshold level of SARS patients. These considerations led to the modifications of model (1),

$$\left\{ \begin{array}{l} \frac{dS(t)}{dt} = A - \mu_S S - \beta_1 S I_1 - \beta_2 S I_2, \\ \frac{dI_1(t)}{dt} = \beta_1 S I_1 - \mu_1 I_1 - v_1 I_1 - \frac{c_1 I_1}{1+b_1 I_1 + \varepsilon b_2 I_2}, \\ \frac{dI_2(t)}{dt} = \beta_2 S I_2 - \mu_2 I_2 - v_2 I_2 - \frac{c_2 I_2}{1+b_1 I_1 + \varepsilon b_2 I_2}, \\ \frac{dR(t)}{dt} = v_1 I_1 + v_2 I_2 + \frac{c_1 I_1 + c_2 I_2}{1+b_1 I_1 + \varepsilon b_2 I_2} - \mu_R R, \end{array} \right. \tag{2}$$

with

$$\varepsilon = \left\{ \begin{array}{ll} 1, & H(z) < 0, \\ 0, & H(z) > 0, \end{array} \right.$$

where $H(z) = I_1 - I_c$, and I_c indicates the threshold level of SARS patients.

For simplicity, in each time step we treat the number of people with flu cases as a constant, i.e.,$I_2 = k$. However, because of the high risk of SARS, people who are infected with SARS need isolation and therapy once they are diagnosed. This means that the impact of medical resources on SARS did not need to be considered, i.e., $b_1 = 0$. Therefore, model (2) can be reduced to

$$\begin{cases} \frac{dS(t)}{dt} = A - \mu S - \beta_1 S I_1, \\ \frac{dI_1(t)}{dt} = \beta_1 S I_1 - v I_1 - \frac{c_1 I_1}{1+\varepsilon b_2 k}, \end{cases}$$

where $\mu = \mu_S + \beta_2 k, v = \mu_1 + v_1$. In the presence of limited medical resources, Qin and Tang suggested that triaging pressure is helpful for controlling e-merging infectious diseases [23], but they ignored the effects of infection age on the spread of the diseases. In fact, people of different ages have different infection degrees for the same infectious disease and infection ages are also different [29, 30]. Therefore, we consider the infection rate, natural recovery rate, disease-related death rate, natural death rate and maximum recovery rate as a function of age, in which age is defined as the time after infection [31]. For simplicity, parameter ε is assumed to be a constant. Based on the above modifications, we derived the following age-structured infectious diseases model,

$$\begin{cases} \frac{dS(t)}{dt} = A - \mu S(t) - \int_0^\infty \beta_1(a) S(t) I(t,a) da, \\ \frac{\partial I(t,a)}{\partial a} + \frac{\partial I(t,a)}{\partial t} = -v(a) I(t,a) - \frac{c_1(a)}{1+\varepsilon b_2 k} I(t,a), \end{cases} \tag{3}$$

with the boundary conditions

$$I(t,0) = \int_0^\infty \beta_1(a) S(t) I(t,a) da \tag{4}$$

for $t \geq 0$ and the initial conditions

$$S(0) = S_0, \quad I(0,a) = I_0(a) \tag{5}$$

for $a \geq 0$, S_0 is non-negative real number and $I_0(a)$ is non-negative essentially bounded and Lebesgue integrable function. $S(t)$ denotes the susceptible population at time t, $I(t,a)$ represents the density of the infected population at time t with age a, where a denotes the infection age.

3. Preliminaries and basic properties of solutions

3.1. Preliminaries

In this section, we provide some assumptions and present the basic properties of solutions.

For simplicity, when $a \geq 0$, denote

$$\epsilon(a) = v(a) + \frac{c_1(a)}{1+\varepsilon b_2 k}, \quad \Omega(a) = e^{-\int_0^a \epsilon(s)ds}, \quad c = \int_0^\infty \beta_1(a)\Omega(a)da,$$

with $\Omega(0) = 1$.

Assumption 3.1. *Let $L^1_+(0,\infty)$ be the space of all essentially bounded and positive functions that are Lebesgue integrable, and assume that*
(i) $A, \mu, \varepsilon, b_2, k > 0$;
(ii) $\beta_1(a), \epsilon(a) \in L^1_+(0,\infty)$ have essential upper bounds $\overline{\beta}, \overline{\epsilon}$,respectively;
(iii) $\beta_1(a)$ is defined on R^+ and is Lipschitz continuous with Lipschitzian L_β;
(iv) There exists $\mu_0 \in (0,\mu]$ such that $\epsilon(a) \geq \mu_0$ for all $a \geq 0$.

According to (ii) and (iv) of Assumption 3.1,

$$0 \leq \Omega(a) \leq e^{-\mu_0 a} \leq 1, \quad a \geq 0, \tag{6}$$

and $\Omega'(a) = -\Omega(a)\epsilon(a)$ hold for $a \geq 0$.

Now we define the function space Γ as

$$\Gamma = R^+ \times L^1_+(0,\infty),$$

which is endowed with the norm

$$\|(y_1, y_2)\|_\Gamma = |y_1| + \int_0^\infty |y_2(a)|\, da.$$

In the positive cone of the space Γ, the subsidiary initial conditions (5) is rewritten by

$$y_0 = (S_0, I(0,\cdot)) \in \Gamma. \tag{7}$$

Next, define a continuous semi-flow $\Phi : R^+ \times \Gamma \to \Gamma$ for system (3), i.e.,

$$\Phi(t, y_0) = \Phi_t(y_0) = (S(t), I(t,\cdot)), \quad t \geq 0, \quad y_0 \in \Gamma. \tag{8}$$

Then the norm of the semi-flow $\Phi_t(\cdot)$ is defined as

$$\|\Phi_t(y_0)\|_\Gamma = \|(S(t), I(t,\cdot))\| = |S(t)| + \int_0^\infty |I(t,a)|\, da.$$

Remark 3.1. *By making use of the standard theories of functional differential equations, we can prove the existence of the semi-flow $\Phi_t(\cdot)$ of (8), that is to say, system (3) with initial conditions (5) has a unique non-negative solution. Therefore, the semi-flow Φ (8) can be obtained.*

Let

$$P(t) = \int_0^\infty \beta_1(a)I(t,a)da, t \geq 0.$$

Solving the second equation of system (3) along the characteristic curve $t - a = constant$, then

$$I(t,a) = \begin{cases} I_0(a-t)\frac{\Omega(a)}{\Omega(a-t)}, & a \geq t \geq 0, \\ S(t-a)P(t-a)\Omega(a), & t > a \geq 0. \end{cases} \tag{9}$$

The state space of system (3) can be defined as follows,

$$\Delta = \left\{ S(t), I(t,\cdot) : S(t) + \int_0^\infty I(t,a)da \leq \frac{A}{\mu_0} \right\}. \tag{10}$$

Then the following proposition holds.

Proposition 3.1. *For system (3),*
(i) $\Phi(t, y_0) \in \Delta$ for any $t \geq 0$ and $y_0 \in \Delta$, i.e., the semi-flow Φ has a positively invariant Δ ;
(ii) Φ is point dissipative and any point initiating from space Γ tends to Δ.

Proof. It follows from (8) that

$$\frac{d}{dt}\|\Phi_t(y_0)\|_\Gamma = \frac{d}{dt}S(t) + \frac{d}{dt}\int_0^\infty I(t,a)da. \tag{11}$$

According to Eq.(9),

$$\int_0^\infty I(t,a)da = \int_0^t S(t-a)P(t-a)\Omega(a)da + \int_t^\infty I_0(a-t)\frac{\Omega(a)}{\Omega(a-t)}da.$$

Let $a = t - \sigma$ in the first integral and $a = t + \tau$ in the second integral, then differentiating with respect to t yields

$$\begin{aligned} \frac{d}{dt}\int_0^\infty I(t,a)da = \frac{d}{dt}\int_0^t S(\sigma)P(\sigma)\Omega(t-\sigma)d\sigma + \frac{d}{dt}\int_0^\infty I_0(\tau)\frac{\Omega(t+\tau)}{\Omega(\tau)}d\tau \\ = S(t)P(t)\Omega(0) + \int_0^t S(\sigma)P(\sigma)\frac{d}{dt}\Omega(t-\sigma)d\sigma \\ + \int_0^\infty I_0(\tau)\frac{d}{dt}\frac{\Omega(t+\tau)}{\Omega(\tau)}d\tau. \end{aligned}$$

Since $\Omega(0) = 1$ and $\Omega'(a) = -\Omega(a)\epsilon(a)$, we have

$$\frac{d}{dt}\int_0^\infty I(t,a)da = S(t)P(t) - \int_0^\infty \epsilon(a)I(t,a)da. \tag{12}$$

Adding the first equation of (3) and (12), the (iv) of Assumption 3.1 leads to

$$\begin{aligned}\frac{d}{dt}\left(S(t) + \int_0^\infty I(t,a)da\right) &= A - \mu S(t) - \int_0^\infty \epsilon(a)I(t,a)da \\ &\leq A - \mu_0\left(S(t) + \int_0^\infty I(t,a)da\right), \quad t \geq 0.\end{aligned}$$

Solving the above equation yields

$$S(t) + \int_0^\infty I(t,a)da \leq \frac{A}{\mu_0} - e^{-\mu_0 t}\left\{\frac{A}{\mu_0} - \left(S(0) + \int_0^\infty I(0,a)da\right)\right\}, \quad t \geq 0. \tag{13}$$

when $y_0 \in \Delta$, for any solutions of system (3) the following inequality holds

$$S(t) + \int_0^\infty I(t,a)da \leq \frac{A}{\mu_0}, \quad t \geq 0. \tag{14}$$

From the norm of semi-flow $\Phi_t(y_0)$ and (13), then

$$\|\Phi_t(y_0)\|_\Gamma \leq \frac{A}{\mu_0} - e^{-\mu_0 t}\left\{\frac{A}{\mu_0} - \|y_0\|_\Gamma\right\}, \quad t \geq 0.$$

Thus, for any solution of system (3), $\Phi_t(y_0) \in \Delta$ holds true for $y_0 \in \Delta$ and $t \geq 0$, which means that Δ is positive invariant.

Moreover, one has

$$\limsup_{t\to\infty} \|\Phi_t(y_0)\|_\Gamma \leq \frac{A}{\mu_0}, \quad y_0 \in \Gamma.$$

Therefore, Φ is point dissipative and Δ attracts all points initiating from space Γ. This completes the proof.

Remark 3.2. *For any constant $N \geq A/\mu_0$ and $t \geq 0$, if $y_0 \in \Gamma$ and $\|y_0\|_\Gamma \leq N$, then it follows from Assumption 3.1 and Proposition 3.1 that we have*
(i) $S(t) \leq N$, $\int_0^\infty I(t,a)da \leq N$;
(ii) $P(t) \leq \bar{\beta}N$;
(iii) $I(t,0) \leq \bar{\beta}N^2$.

3.2. The existence of steady states

This subsection deals with the existence of the steady states of system (3), it is found that the steady states of the system (3) satisfy the following equalities

$$\begin{cases} A - \mu\bar{S} - \int_0^\infty \beta_1(a)\bar{S}\bar{I}(a)da = 0, \\ \frac{d\bar{I}(a)}{da} = -\epsilon(a)\bar{I}(a), \\ \bar{I}(0) = \int_0^\infty \beta_1(a)\bar{S}\bar{I}(a)da. \end{cases} \tag{15}$$

By calculations, system (3) always has an infection-free equilibrium $E_0(S_0, 0)$, where $S_0 = A/\mu$. Next, solving (15) to get the infection equilibrium $\bar{E}(\bar{S}, \bar{I})$,

$$\bar{S} = \frac{S_0}{\Re_0}, \quad \bar{I}(a) = \frac{(\Re_0 - 1)A}{\Re_0}\Omega(a), \tag{16}$$

where

$$\Re_0 = S_0 c. \tag{17}$$

Here, c denotes the probability of becoming infected so when c is multiplied by the susceptible population S_0 this leads to $\Re_0$, which is the number of people infected by infectious people during their average period of infection.

Theorem 3.1. *For the system (3) with boundary condition (4) and initial condition (5), then the system always has a unique infection-free equilibrium $E_0(S_0, 0)$; if $\Re_0 > 1$, then the system has a unique infection equilibrium $\bar{E}(\bar{S}, \bar{I})$.*

3.3. Asymptotic smoothness of semi-flow $\Phi(t)_{t\geq 0}$

In order to investigate the global properties of the semi-flow, we must ensure the asymptotic smoothness of semi-flow $\Phi(t)_{t\geq 0}$. To this end, the following useful definition is introduced.

Definition 3.1. *The semi-flow $\Phi(t, y_0) : R^+ \times \Gamma \to \Gamma$ is called asymptotically smooth, if for any non-empty and closed bounded set $B \subset \Gamma$ for which $\Phi(t, B) \subset B$, there is a compact set $B_0 \subset B$ such that B_0 attracts B.*

Now, we decompose $\Phi : R^+ \times \Gamma \to \Gamma$ into the operators $\phi : R^+ \times \Gamma \to \Gamma$ and $\varphi : R^+ \times \Gamma \to \Gamma$,

$$\phi(t, y_0) = (0, \tilde{\phi_I}(t, \cdot)), \tag{18}$$

$$\varphi(t, y_0) = (S(t), \tilde{\varphi_I}(t, \cdot)), \tag{19}$$

where

$$\tilde{\phi_I}(t,a) = \begin{cases} 0, & t > a \geq 0; \\ I(t,a), & a \geq t \geq 0, \end{cases} \quad \textit{and} \quad \tilde{\varphi_I}(t,a) = \begin{cases} I(t,a), & t > a \geq 0; \\ 0, & a \geq t \geq 0. \end{cases} \tag{20}$$

Thereby, $\Phi(t, y_0) = \phi(t, y_0) + \varphi(t, y_0)$. From Proposition 3.13 [32], one has the following results.

Theorem 3.2. *The semi-flow $\Phi : R^+ \times \Gamma \to \Gamma$ is asymptotically smooth in Γ provided that*
(i) there exists a continuous function $f : R^+ \times R^+ \to R^+$ such that, for any $h > 0$, $\lim_{t\to\infty} f(t,h) = 0$. Further, if $y_0 \in \Delta$ with $\|y_0\|_\Gamma \leq h$, then $\|\phi(t, y_0)\|_\Gamma \leq f(t,h)$ for $t \geq 0$;
(ii) for $t \geq 0$, $\varphi(t, y_0)$ is completely continuous.

In order to prove that condition (i) of Theorem 3.2 holds true, the following lemma is needed.

Lemma 3.1. *For $h > 0$, let $f(t,h) = e^{-\mu_0 t}h$, then $\lim_{t\to\infty} f(t,h) = 0$, and for $t \geq 0$, $\|\phi(t, y_0)\|_\Gamma \leq f(t,h)$ if $y_0 \in \Delta$ with $\|y_0\|_\Gamma \leq h$.*

Proof. Obviously, $\lim_{t\to\infty} f(t,h) = 0$. Following the lead of (9) and (20),

$$\widetilde{\phi_I}(t,a) = \begin{cases} 0, & t > a \geq 0; \\ I_0(a-t)\frac{\Omega(a)}{\Omega(a-t)}, & a \geq t \geq 0. \end{cases}$$

For $\|y_0\|_\Gamma \leq h$ with $y_0 \in \Delta$, then

$$\begin{aligned} \|\phi(t, y_0)\|_\Gamma &= |0| + \int_0^\infty \left|\tilde{\phi_I}(t,a)\right| da \\ &= \int_t^\infty \left| I_0(a-t)\frac{\Omega(a)}{\Omega(a-t)} \right| da \\ &= \int_0^\infty \left| I_0(\sigma)\frac{\Omega(t+\sigma)}{\Omega(\sigma)} \right| d\sigma \\ &= \int_0^\infty \left| I_0(\sigma) e^{-\int_\sigma^{t+\sigma} \epsilon(s) da} \right| d\sigma. \end{aligned}$$

In the light of Assumption 3.1, $\epsilon(a) \geq \mu_0$ holds true. Moreover,

$$\begin{aligned} \|\phi(t, y_0)\|_\Gamma &\leq e^{-\mu_0 t} \int_0^\infty |I_0(\sigma)| d\sigma \\ &\leq e^{-\mu_0 t} \|y_0\|_\Gamma \leq e^{-\mu_0 t} h = f(t,h). \end{aligned}$$

Therefore, there is a continuous function f that satisfies $\lim_{t\to\infty} f(t,h) = 0$ and $\|\phi(t,y_0)\|_\Gamma \le f(t,h)$ for $\|y_0\|_\Gamma \le h$. This completes the proof.

Since the spaces Γ and $L^1_+(0,\infty)$ we are studying are infinite dimensional spaces. It is known that in such spaces only boundedness does not imply precompactness. Hence, to show the condition (ii) of Theorem 3.2, we apply the following lemma.

Lemma 3.2. *([33]) Let $\mathcal{C} \subset L^p(0,\infty)$ be a bounded and closed subset for $p \ge 1$. Then $\mathcal{C}$ is compact if the following four conditions are true.*
(i) $\sup_{g\in\mathcal{C}} \int_0^\infty g(a)da < \infty$;
(ii) $\lim_{h\to\infty} \int_h^\infty g(a)da = 0$ *uniformly with respect to* $g \in \mathcal{C}$;
(iii) $\lim_{u\to 0^+} \int_0^\infty |g(a+u) - g(a)|\, da = 0$ *uniformly with respect to* $g \in \mathcal{C}$;
(iv) $\lim_{u\to 0^+} \int_0^u g(a)da = 0$ *uniformly with respect to* $g \in \mathcal{C}$.

Lemma 3.3. *For $t \ge 0$, $\varphi(t,y_0)$ is completely continuous.*

Proof. Lemma 3.2 implies that $\varphi(t,B)$ is compact for any bounded and closed set $B \subset \Gamma$. It follows from Remark 3.2 that $S(t)$ remains in the compact set $[0, A/\mu_0] \subset [0,N]$. Thus, it is sufficient to prove that $\tilde{\varphi}_I(t,a)$ remains in a precompact subset of $L^1_+(0,\infty)$, which is independent of $y_0 \in \Delta$.

Due to (9) and (20), $\tilde{\varphi}_I(t,a)$ is non-negative and

$$\tilde{\varphi}_I(t,a) = \begin{cases} S(t-a)P(t-a)\Omega(a), & t > a \ge 0; \\ 0, & a \ge t \ge 0. \end{cases} \tag{21}$$

By making use of Remark 3.2 and (21), we have

$$\tilde{\varphi}_I(t,a) \le \bar{\beta} N^2 e^{-\mu_0 a}.$$

Hence, the conditions (i), (ii) and (iv) of Lemma 3.2 are proved. To verify condition (iii), assume that $u \in (0,t)$, then

$$\begin{aligned}
&\int_0^\infty |\tilde{\varphi}_I(t,a+u) - \tilde{\varphi}_I(t,a)|\, da = \int_0^\infty |I(t,a+u) - I(t,a)| da \\
&= \int_0^{t-u} |I(t,a+u) - I(t,a)| da + \int_{t-u}^t |I(t,a+u) - I(t,a)| da \\
&= \int_0^{t-u} |S(t-a-u)P(t-a-u)\Omega(a+u) - S(t-a)P(t-a)\Omega(a)| da \\
&\quad + \int_{t-u}^t |0 - S(t-a)P(t-a)\Omega(a)| da \\
&= \int_0^{t-u} |I(t-a-u,0)\Omega(a+u) - I(t-a,0)\Omega(a)| da \\
&\quad + \int_{t-u}^t |I(t-a,0)\Omega(a)| da \\
&\le \int_0^{t-u} I(t-a-u,0)\, |\Omega(a+u) - \Omega(a)| da + \int_{t-u}^t |I(t-a,0)\Omega(a)| da \\
&\quad + \int_0^{t-u} \Omega(a)\, |I(t-a-u,0) - I(t-a,0)|\, da.
\end{aligned} \tag{22}$$

For simplicity, denote

$$\Xi = \int_0^{t-u} \Omega(a)\, |I(t-a-u,0) - I(t-a,0)|\, da.$$

Because of $0 \le \Omega(a) \le e^{-\mu_0 a} \le 1$ and $\Omega(a)$ is a non-increasing function with respect to a, one has

$$\begin{aligned}
\int_0^{t-u} |\Omega(a+u) - \Omega(a)| da &= \int_0^{t-u} \Omega(a) da - \int_0^{t-u} \Omega(a+u) da \\
&= \int_0^{t-u} \Omega(a) da - \int_u^t \Omega(a) da \\
&= \int_0^{t-u} \Omega(a) da - \int_u^{t-u} \Omega(a) da - \int_{t-u}^t \Omega(a) da \\
&= \int_0^u \Omega(a) da - \int_{t-u}^t \Omega(a) da \\
&\le u.
\end{aligned}$$

From (22) and (iii) of Remark 3.2, it follows that

$$\int_0^\infty |\tilde{\varphi}_I(t, a+u) - \tilde{\varphi}_I(t, a)| da \leq \Xi + 2\bar{\beta} N^2 u. \tag{23}$$

Then Assumption 3.1 and (i) of Remark 3.2 lead to

$$\left| \frac{dS(t)}{dt} \right| \leq A + \mu N + \bar{\beta} N^2 = L_s,$$

which means that $|dS(t)/dt|$ is bounded. Therefore, $S(t)$ is Lipschitz continuous on $[0, \infty)$ and the Lipschitz constant is L_s. Thereby,

$$\Xi \leq \frac{L_s u}{\mu_0}. \tag{24}$$

Using (23) and (24), one has

$$\int_0^\infty |\tilde{\varphi}_I(t, a+u) - \tilde{\varphi}_I(t, a)| da \leq (\frac{L_s}{\mu_0} + 2\bar{\beta} N^2) u.$$

Hence, this integral converges uniformly to 0 because u tends to 0. It implies that (iii) holds true for any $y_0 \in B$, which means that $\tilde{\varphi}_I(t, a)$ remains in a precompact subset $B_{\tilde{\varphi}_I}$ of $L^1_+(0, \infty)$. This completes the proof.

From the above analysis, the following results can be obtained which are useful in the rest of the paper.

Theorem 3.3. *The semi-flow $\Phi(t)_{t\geq 0}$ has a global attractor $\mathcal{A}$ in Γ, which attracts all bounded subsets of Γ.*

4. Dynamical behaviors

This section mainly focused on the global dynamical behaviors of system (3), including local stability, uniform persistence and global stability.

4.1. Local stability analysis

In this subsection, we will study the local stabilities of the infection-free equilibrium E_0 and the infection equilibrium $\bar{E}$.

Theorem 4.1. *If $\Re_0 < 1$, then the infection-free equilibrium E_0 is locally asymptotically stable.*

Proof. We first perform the following variable substitutions,

$$x_1(t) = S(t) - S_0, \quad x_2(t,a) = I(t,a).$$

Then system (3) can be linearized at E_0, i.e.,

$$\begin{cases} \frac{dx_1(t)}{dt} = -\mu x_1(t) - S_0 \int_0^\infty \beta_1(a) x_2(t,a)da, \\ \frac{\partial x_2(t,a)}{\partial a} + \frac{\partial x_2(t,a)}{\partial t} = -\epsilon(a) x_2(t,a), \\ x_2(t,0) = S_0 \int_0^\infty \beta_1(a) x_2(t,a)da. \end{cases} \tag{25}$$

Suppose the form of the solution of system (25) is as follows,

$$x_1(t) = x_1^0 e^{\lambda t}, \quad x_2(t,a) = x_2^0(a) e^{\lambda t}, \tag{26}$$

where the expressions of x_1^0 and $x_2^0(a)$ can be derived later. Taking (26) into (25) yields

$$\begin{cases} \lambda x_1^0 = -\mu x_1^0 - S_0 \int_0^\infty \beta_1(a) x_2^0(a)da, \\ \lambda x_2^0(a) + \frac{dx_2^0(a)}{da} = -\epsilon(a) x_2^0(a), \\ x_2^0(0) = S_0 \int_0^\infty \beta_1(a) x_2^0(a)da. \end{cases} \tag{27}$$

Integrating the second equation of system (27), then

$$x_2^0(a) = x_2^0(0) e^{-\int_0^a (\epsilon(s)+\lambda)ds}. \tag{28}$$

Inserting (28) into the third equation of system (27), we get the characteristic equation at E_0,

$$S_0 \int_0^\infty \beta_1(a) e^{-\int_0^a (\epsilon(s)+\lambda)ds} da = 1. \tag{29}$$

Denote the left hand side of (29) by $F(\lambda)$, then

$$\lim_{\lambda \to +\infty} F(\lambda) = 0, \quad \lim_{\lambda \to -\infty} F(\lambda) = \infty, \quad \frac{d}{d\lambda} F(\lambda) < 0.$$

Therefore, there is a unique real λ^* such that $F(\lambda^*) = 1$ for equation (29). It is found that $F(0) = \Re_0$, if $\Re_0 < 1$, then $\lambda^* < 0$; if $\Re_0 > 1$, then $\lambda^* > 0$. Moreover, if equation (29) has a complex root $\lambda = a + ib$, assuming that $Re(\lambda) \geq 0$, then

$$|F(\lambda)| = \left| S_0 \int_0^\infty \beta_1(a) e^{-\int_0^a (\epsilon(s)+\lambda)ds} da \right| \leq \left| S_0 \int_0^\infty \beta_1(a) e^{-\int_0^a \epsilon(s)ds} da \right| = S_0 c = \Re_0,$$

which contradicts (29), meaning that the real parts of the characteristic roots of (29) are all negative if and only if $\Re_0 < 1$. Meanwhile, if $\Re_0 > 1$, then at least one real part of the characteristic root is positive. It is concluded that the infection-free equilibrium E_0 is locally asymptotically stable if $\Re_0 < 1$ and unstable if $\Re_0 > 1$. This completes the proof.

Theorem 4.2. *If $\Re_0 > 1$, then the infection equilibrium $\bar{E}$ is locally asymptotically stable.*

Proof. To prove the local stability of $\bar{E}$, the following variable substitutions are carried out,

$$y_1(t) = S(t) - \bar{S}, \quad y_2(t,a) = I(t,a) - \bar{I}(a).$$

By calculation, the linearized system (3) at $\bar{E}$ can be obtained,

$$\begin{cases} \frac{dy_1(t)}{dt} = -\mu y_1(t) - \int_0^\infty \beta_1(a) y_1(t)\bar{I}(a)da - \bar{S}\int_0^\infty \beta_1(a) y_2(t,a)da, \\ \frac{\partial y_2(t,a)}{\partial a} + \frac{\partial y_2(t,a)}{\partial t} = -\epsilon(a) y_2(t,a), \\ y_2(t,0) = \int_0^\infty \beta_1(a) y_1(t)\bar{I}(a)da + \bar{S}\int_0^\infty \beta_1(a) y_2(t,a)da. \end{cases} \tag{30}$$

Denoting the solution of system (30) by

$$y_1(t) = y_1^0 e^{\lambda t}, \quad y_2(t,a) = y_2^0(a) e^{\lambda t}, \tag{31}$$

where the expressions of y_1^0 and $y_2^0(a)$ will be provided later. Substituting (31) into (30), we get

$$\begin{cases} \lambda y_1^0 = -\mu y_1^0 - \int_0^\infty \beta_1(a) y_1^0 \bar{I}(a)da - \bar{S}\int_0^\infty \beta_1(a) y_2^0(a)da, \\ \lambda y_2^0(a) + \frac{dy_2^0(a)}{\partial t} = -\epsilon(a) y_2^0(a), \\ y_2^0(0) = \int_0^\infty \beta_1(a) y_1^0 \bar{I}(a)da + \bar{S}\int_0^\infty \beta_1(a) y_2^0(a)da. \end{cases} \tag{32}$$

It follows from the second equation of system (32) that

$$y_2^0(a) = y_2^0(0) e^{-\int_0^a (\epsilon(s)+\lambda)ds}. \tag{33}$$

Substituting (33) into the first and third equations of system (32), then the characteristic equation at $\bar{E}$ can be derived,

$$\int_0^\infty \beta_1(a)\frac{\mu - Ac}{(\lambda+\mu)c}\Omega(a)da + \bar{S}\int_0^\infty \beta_1(a) e^{-\int_0^a (\epsilon(s)+\lambda)ds} da = 1. \tag{34}$$

Denote

$$H(\lambda) = \int_0^\infty \beta_1(a) e^{-\int_0^a (\epsilon(s)+\lambda) ds} da.$$

Hence, (34) can be simplified by the following equation

$$\frac{\mu - Ac}{(\lambda + \mu)} + \bar{S}H(\lambda) = 1.$$

i.e.,

$$\frac{\lambda + Ac}{(\lambda + \mu)} = \bar{S}H(\lambda), \tag{35}$$

where $H(0) = c$ and $\bar{S} = 1/c$. Suppose that equation (35) has at least one root λ^0 such that $Re(\lambda^0) \geq 0$, then $H(\lambda^0) \leq c$. In fact, $(\lambda + Ac)/(\lambda + \mu) > 1$ holds true, which means that the two sides of equation (35) are contradictory. Therefore, $Re(\lambda) < 0$ and all roots of (34) have negative real parts. Accordingly, the infection equilibrium $\bar{E}$ of system (3) is locally asymptotically stable. This completes the proof.

4.2. Uniform persistence

In this subsection, we focus on the uniform persistence of system (3) when $\Re_0 > 1$. Let $i(t) = I(t, 0)$, (9) can be further denoted as

$$I(t,a) = \begin{cases} I_0(a-t)\frac{\Omega(a)}{\Omega(a-t)}, & a \geq t \geq 0; \\ i(t-a)\Omega(a), & t > a \geq 0. \end{cases} \tag{36}$$

Substituting (36) into the boundary condition yields

$$i(t) = \int_0^t \beta_1(a)S(t)i(t-a)\Omega(a)da + \int_t^\infty \beta_1(a)S(t)I_0(a-t)\frac{\Omega(a)}{\Omega(a-t)}. \tag{37}$$

Furthermore, the first equation of (3) can be denoted by

$$\frac{dS(t)}{dt} = A - \mu S(t) - i(t). \tag{38}$$

Hence, we have the following lemma for the weak persistence of $i(t)$.

Lemma 4.1. *The system (3) is uniformly weakly persistent if $\Re_0 > 1$, i.e., there exists a positive constant $\xi > 0$ such that*

$$\limsup_{t\to\infty} i(t) > \xi. \tag{39}$$

Proof. From (37), one has

$$i(t) \geq \int_0^t \beta_1(a)S(t)i(t-a)\Omega(a)da. \tag{40}$$

Assume that there is a sufficiently small $\xi \in (0, A)$, if $\Re_0 > 1$, then

$$\frac{A-\xi}{\mu}\int_0^\infty \beta_1(a)\Omega(a)da > 1. \tag{41}$$

Therefore, system (3) is uniformly weakly persistent. Otherwise, there exists a state with a sufficiently large constant $M > 0$ such that $i(t) \leq \xi$ for all $t \geq M$. From (38), it follows that

$$\frac{dS(t)}{dt} \geq A - \mu S(t) - \xi, \quad t \geq M,$$

which implies that $\liminf_{t\to\infty} S(t) \geq (A-\xi)/\mu$. Hence, there is a $M_0 > 0$ for any $\xi_0 > 0$ such that $S(t) \geq (A-\xi-\xi_0)/\mu$ for all $t \geq M_0$. It follows from (40) that

$$i(t) \geq \frac{A-\xi-\xi_0}{\mu}\int_0^t \beta_1(a)i(t-a)\Omega(a)da, \quad t \geq M_0. \tag{42}$$

Then Laplace transforms are performed at both sides of (42) at the same time and then it can be obtained from the convolution theorem that

$$\hat{i}(t) \geq \frac{A-\xi-\xi_0}{\mu}\int_0^\infty \beta_1(a)\Omega(a)e^{-\lambda a}da\hat{i}(t),$$

where $\hat{i}(t)$ denotes the Laplace transform of i and it is strictly positive because of (37) and Assumption 3.1. Dividing both sides of the above inequality by $\hat{i(t)}$ and letting $\lambda \to 0$ yield

$$1 \geq \frac{A-\xi-\xi_0}{\mu}\int_0^\infty \beta_1(a)\Omega(a)da.$$

Since $\xi_0 > 0$ is arbitrary, assume $\xi_0 \to 0^+$, then

$$1 \geq \frac{A-\xi}{\mu}\int_0^\infty \beta_1(a)\Omega(a)da.$$

which contradicts (41). This completes the proof.

Theorem 4.3. *The system (3) is uniformly strongly persistent provided that* $\Re_0 > 1$, *that is, there exists a positive constant* $\eta > 0$ *such that*

$$\liminf_{t\to\infty} i(t) > \eta.$$

Proof. In space Γ, let the total Φ-trajectories be a function of Θ, where Θ is defined as: $\Theta : R \to \Gamma$ with $\Theta(r) = (S(r), I(r, \cdot))(r \in R)$, and it satisfies $\Theta(t + r) = \Phi(t, \Theta(r))$. It is worth noting that

$$I(t, a) = I(t - a, 0)\Omega(a) = i(t - a)\Omega(a), \quad t > a \geq 0.$$

The norm of (36) leads to

$$\|I(t, \cdot)\| \geq \int_0^t i(t - a)\Omega(a)da.$$

From the Lebesgue-Fatou lemma [[34], Section B.2], one has

$$\liminf_{t\to\infty} \|I(t, \cdot)\| \geq \liminf_{t\to\infty} i(t) \int_0^\infty \Omega(a)da.$$

Hence, there exists a state with a positive constant η such that $\liminf_{t\to\infty} i(t) > \eta$ if $\Re_0 > 1$. This completes the proof.

It follows from the persistence of $i(t)$ and (38) that $S(t)$ is also persistent. Therefore, the following theorem is obtained.

Theorem 4.4. *If* $\Re_0 > 1$, *then the semi-flow* $\{\Phi(t)\}_{t\geq 0}$ *defined by (3) is uniformly persistent in* Γ, *which means that there is a constant* $\eta > 0$ *that satisfies*

$$\liminf_{t\to\infty} S(t) > \eta \quad \textit{and} \quad \liminf_{t\to\infty} \|I(t, \cdot)\| > \eta.$$

4.3. Global stability

In this subsection, we will prove the global stabilities of the infection-free equilibrium E_0 and the infection equilibrium $\bar{E}$. To this end, we first define

$$g(x) = x - 1 - lnx,$$

where $g(x) \geq 0$ for $x > 1$ and $g(x)$ has a global minimum at $x = 1$ and $g(1) = 0$.

Theorem 4.5. *If $\Re_0 \leq 1$, then the infection-free equilibrium E_0 is globally asymptotically stable.*

Proof. Defining a Lyapunov function $V(t) = V_1(t) + V_2(t)$ with

$$V_1(t) = S_0 g\left(\frac{S(t)}{S_0}\right) \quad \text{and} \quad V_2(t) = \int_0^\infty K(a) I(t,a) da,$$

the non-negative kernel function $K(a)$ will be given later.

Calculating the derivative of $V_1(t)$ with respect to time t along the positive solutions of (3) yields

$$\begin{aligned}\frac{dV_1(t)}{dt} &= \left(1 - \frac{S_0}{S(t)}\right)\left(A - \mu S(t) - \int_0^\infty \beta_1(a) S(t) I(t,a) da\right) \\ &= -\frac{\mu (S(t) - S_0)^2}{S(t)} - \int_0^\infty \beta_1(a) S(t) I(t,a) da + \int_0^\infty \beta_1(a) S_0 I(t,a) da.\end{aligned} \tag{43}$$

Similarly,

$$\begin{aligned}\frac{dV_2(t)}{dt} &= \int_0^\infty K(a) \frac{\partial I(t,a)}{\partial t} da \\ &= \int_0^\infty K(a)\left(-\epsilon(a) I(t,a) - \frac{\partial I(t,a)}{\partial a}\right) da \\ &= -\int_0^\infty K(a) \frac{\partial I(t,a)}{\partial a} da - \int_0^\infty K(a)\epsilon(a) I(t,a) da \\ &= -K(a) I(t,a) \left.\right|_{a=0}^{a=\infty} + \int_0^\infty I(t,a) \frac{dK(a)}{da} da - \int_0^\infty K(a)\epsilon(a) I(t,a) da \\ &= K(0) I(t,0) + \int_0^\infty \left(\left(\frac{dK(a)}{da} - K(a)\epsilon(a)\right) I(t,a)\right) da.\end{aligned} \tag{44}$$

Choosing $K(a)$ such that

$$K(a) = \int_a^\infty S_0 \beta_1(\tau) e^{-\int_a^\tau \epsilon(s) ds} d\tau,$$

then

$$\begin{aligned}\frac{dK(a)}{da} &= K(a)\epsilon(a) - S_0 \beta_1(a), \\ K(0) &= \int_0^\infty S_0 \beta_1(\tau) e^{-\int_0^\tau \epsilon(s) ds} d\tau = S_0 c = \Re_0.\end{aligned}$$

Substituting $K(a)$ into (44) gives

$$\frac{dV_2(t)}{dt} = K(0)I(t,0) - \int_0^\infty \beta_1(a)S_0 I(t,a)da.$$

From (43), (44) and boundary condition $I(t,0) = \int_0^\infty \beta_1(a)S(t)I(t,a)da$, $dV(t)/dt$ is derived as follows,

$$\begin{aligned}\frac{dV(t)}{dt} &= -\frac{\mu(S(t)-S_0)^2}{S(t)} - \int_0^\infty \beta_1(a)S(t)I(t,a)da + K(0)I(t,0)\\ &= -\frac{\mu(S(t)-S_0)^2}{S(t)} - I(t,0) + S_0 cI(t,0)\\ &= -\frac{\mu(S(t)-S_0)^2}{S(t)} + (\Re_0 - 1)I(t,0).\end{aligned}$$

Clearly, $V'(t) \le 0$ provided that $\Re_0 \le 1$ and $V'(t) = 0$ means $S(t) = S_0$. Thus, $\{V'(t) = 0\}$ holds true if and only if it is at $\{E_0\}$. Therefore, according to LaSalle's invariance principle, the infection-free equilibrium E_0 is globally asymptotically stable provided that $\Re_0 \le 1$. This completes the proof.

To show the global asymptotical stability of the infection equilibrium $\bar{E}$, we first present the following lemma for simplicity.

Lemma 4.2. *The following equation holds*

$$\int_0^\infty \beta_1(a)\bar{S}\bar{I}(a)\left(1 - \frac{S(t)\bar{I}(0)I(t,a)}{\bar{S}I(t,0)\bar{I}(a)}\right)da = 0. \tag{45}$$

Proof.

$$\begin{aligned}&\int_0^\infty \beta_1(a)\bar{S}\bar{I}(a)\left(1 - \frac{S(t)\bar{I}(0)I(t,a)}{\bar{S}I(t,0)\bar{I}(a)}\right)da\\ =&\bar{I}(0) - \frac{\bar{I}(0)}{I(t,0)}\int_0^\infty \beta_1(a)\bar{S}\bar{I}(a)\frac{S(t)}{\bar{S}}\frac{I(t,a)}{\bar{I}(a)}da\\ =&\bar{I}(0) - \frac{\bar{I}(0)}{I(t,0)}\int_0^\infty \beta_1(a)S(t)I(t,a)da\\ =&\bar{I}(0) - I(t,0)\frac{\bar{I}(0)}{I(t,0)} = 0.\end{aligned}$$

This completes the proof.

Theorem 4.6. *If $\Re_0 > 1$, then the infection equilibrium $\bar{E}$ is globally asymptotically stable.*

Proof. Consider the following Lyapunov function

$$W(t) = W_1(t) + W_2(t),$$

where

$$W_1(t) = \bar{S}g\left(\frac{S(t)}{\bar{S}}\right), \quad W_2(t) = \int_0^\infty \bar{K}(a)\bar{I}(a)g\left(\frac{I(t,a)}{\bar{I}(a)}\right)da,$$

and the non-negative kernel function $\bar{K}(a)$ will be derived later.

Calculating the derivatives of $W_i, i = 1, 2$ with respect to time t along the positive solutions of (3), respectively, then

$$\begin{aligned}
\frac{dW_1(t)}{dt} &= \left(1 - \frac{\bar{S}}{S(t)}\right)\left(A - \mu S(t) - \int_0^\infty \beta_1(a)S(t)I(t,a)da\right) \\
&= \left(1 - \frac{\bar{S}}{S(t)}\right)(A - \mu S(t) - I(t,0)) \\
&= \left(1 - \frac{\bar{S}}{S(t)}\right)(\mu\bar{S} - \mu S(t) + \bar{I}(0) - I(t,0)) \\
&= -\frac{\mu(S(t)-\bar{S})^2}{S(t)} + \bar{I}(0) - I(t,0) - \bar{I}(0)\frac{\bar{S}}{S(t)} + I(t,0)\frac{\bar{S}}{S(t)}.
\end{aligned}$$

Before introducing the derivative of $W_2(t)$, we give the following formula

$$\begin{aligned}
\frac{\partial}{\partial a}g\left(\frac{I(t,a)}{\bar{I}(a)}\right) &= \left(1 - \frac{\bar{I}(a)}{I(t,a)}\right)\frac{\partial}{\partial a}\left(\frac{I(t,a)}{\bar{I}(a)}\right) \\
&= \left(1 - \frac{\bar{I}(a)}{I(t,a)}\right)\left[\frac{1}{\bar{I}(a)}\frac{\partial I(t,a)}{\partial a} - \frac{I(t,a)}{(\bar{I}(a))^2}\frac{d\bar{I}(a)}{da}\right] \\
&= \left(\frac{I(t,a)}{\bar{I}(a)} - 1\right)\left(\frac{I_a(t,a)}{I(t,a)} + \epsilon(a)\right) \\
&= -\frac{\partial}{\partial t}g\left(\frac{I(t,a)}{\bar{I}(a)}\right).
\end{aligned} \tag{46}$$

In the light of (46), the derivative of $W_2(t)$ can be obtained,

$$\begin{aligned}\frac{dW_2(t)}{dt} &= \int_0^\infty \bar{K}(a)\bar{I}(a)\frac{\partial}{\partial t}g\left(\frac{I(t,a)}{\bar{I}(a)}\right)da \\ &= -\int_0^\infty \bar{K}(a)\bar{I}(a)\frac{\partial}{\partial a}g\left(\frac{I(t,a)}{\bar{I}(a)}\right)da \\ &= -\bar{K}(a)\bar{I}(a)g\left(\frac{I(t,a)}{\bar{I}(a)}\right)|_{a=0}^{a=\infty} + \int_0^\infty g\left(\frac{I(t,a)}{\bar{I}(a)}\right)\frac{d}{da}(\bar{K}(a)\bar{I}(a))da.\end{aligned}$$

Choose

$$\bar{K}(a) = \int_a^\infty \bar{S}\beta_1(u)e^{-\int_a^u \epsilon(s)ds}du$$

such that

$$\begin{aligned}\frac{\bar{K}(a)}{da} &= \bar{K}(a)\epsilon(a) - \bar{S}\beta_1(a), \\ \bar{K}(0) &= \int_0^\infty \bar{S}\beta_1(u)e^{-\int_0^u \epsilon(s)ds}du = \bar{S}c = 1.\end{aligned}$$

Thus,

$$\begin{aligned}\frac{dW_2(t)}{dt} =& \bar{I}(0)g\left(\frac{I(t,0)}{\bar{I}(0)}\right) - \int_0^\infty \beta_(a)\bar{S}\bar{I}(a)g\left(\frac{I(t,a)}{\bar{I}(a)}\right)da \\ =& \int_0^\infty \beta_1(a)\bar{S}\left(\bar{I}(a) - I(t,a) + \bar{I}(a)ln\frac{I(t,a)}{\bar{I}(a)}\right)da \\ &+ I(t,0) - \bar{I}(0) - \bar{I}(0)ln\frac{I(t,0)}{\bar{I}(0)}.\end{aligned}$$

Integrating $W_1'(t)$ and $W_2'(t)$, one has

$$\begin{aligned}\frac{dW(t)}{dt} =& -\frac{\mu(S(t)-\bar{S})^2}{S(t)} + \bar{I}(0) - I(t,0) - \bar{I}(0)\frac{\bar{S}}{S(t)} + I(t,0)\frac{\bar{S}}{S(t)} \\ &+ \int_0^\infty \beta_1(a)\bar{S}\left(\bar{I}(a) - I(t,a) + \bar{I}(a)ln\frac{I(t,a)}{\bar{I}(a)}\right)da \\ &+ I(t,0) - \bar{I}(0) - \bar{I}(0)ln\frac{I(t,0)}{\bar{I}(0)} \\ =& -\frac{\mu(S(t)-\bar{S})^2}{S(t)} - \bar{I}(0)\frac{\bar{S}}{S(t)} + I(t,0)\frac{\bar{S}}{S(t)} - \bar{I}(0)ln\frac{I(t,0)}{\bar{I}(0)} \\ &+ \int_0^\infty \beta_1(a)\bar{S}\left(\bar{I}(a) - I(t,a) + \bar{I}(a)ln\frac{I(t,a)}{\bar{I}(a)}\right)da.\end{aligned}$$

Denote $\bar{I}(0) = \int_0^\infty \beta_1(a)\bar{S}\bar{I}(a)da$, we have

$$\begin{aligned}
\frac{dW(t)}{dt} =& -\frac{\mu(S(t)-\bar{S})^2}{S(t)} + \int_0^\infty \beta_1(a)\bar{S}\bar{I}(a)\left(1-\frac{\bar{S}}{S(t)}+ln\frac{\bar{S}}{S(t)}\right)da \\
&+\int_0^\infty \beta_1(a)\bar{S}\bar{I}(a)\left(ln\frac{I(t,a)}{\bar{I}(a)} - ln\frac{I(t,0)}{\bar{I}(0)} - ln\frac{\bar{S}}{S(t)}\right)da \\
=& -\frac{\mu(S(t)-\bar{S})^2}{S(t)} - \int_0^\infty \beta_1(a)\bar{S}\bar{I}(a)g\left(\frac{\bar{S}}{S(t)}\right)da \\
&+\int_0^\infty \beta_1(a)\bar{S}\bar{I}(a)\left(1-\frac{S(t)\bar{I}(0)I(t,a)}{\bar{S}I(t,0)\bar{I}(a)} + ln\frac{S(t)\bar{I}(0)I(t,a)}{\bar{S}I(t,0)\bar{I}(a)}\right)da \\
&-\int_0^\infty \beta_1(a)\bar{S}\bar{I}(a)\left(1-\frac{S(t)\bar{I}(0)I(t,a)}{\bar{S}I(t,0)\bar{I}(a)}\right)da.
\end{aligned}$$

Due to Lemma 4.2,

$$\begin{aligned}
\frac{dW(t)}{dt} =& -\frac{\mu(S(t)-\bar{S})^2}{S(t)} - \int_0^\infty \beta_1(a)\bar{S}\bar{I}(a)g\left(\frac{\bar{S}}{S(t)}\right)da \\
&-\int_0^\infty \beta_1(a)\bar{S}\bar{I}(a)g\left(\frac{S(t)\bar{I}(0)I(t,a)}{\bar{S}\bar{I}(a)I(t,0)}\right)da.
\end{aligned}$$

Therefore, $W'(t) \leq 0$ and the equality holds only when $S(t) = \bar{S}$, $I(t,a) = \bar{I}(a)$. Moreover, the largest invariant subset of $\{W'(t) = 0\}$ is the singleton $\{\bar{E}\}$. Thereby, LaSalle's invariance principle indicates that the infection equilibrium $\bar{E}$ is globally asymptotically stable if $\Re_0 > 1$. This completes the proof.

5. Numerical studies and biological implications

In this section, numerical investigations are carried out to support the theoretical results and the corresponding biological significance is also addressed. To do this, we will use symbolic integrals to calculate integral equations, and use Euler method to discretize the ordinary and partial differential equations in system (3). The numerical method is based on an iterative process, and we have chosen a (relatively) simple finite difference schemes for the PDE system, which is convenient for numerical implementation while maintaining good numerical stability. In each iteration, the system is solved by forward time differences and forward age differences with initial conditions, replacing the derivatives by forward finite difference quotients, and the integral is approximated by the Riemann sum [35]. Making use of the numerical simulation method given above, the results of discretization of system (3) are obtained

$$\begin{cases} I(t+1,a+1) = \epsilon(a)I(t,a), \\ I(t+1,1) = hS(t)\sum\limits_{i=1}^{a_2} I(t,i)\beta(i), \\ S(t+1) = S(t) + (A - \mu S(t) - I(t+1,1)), \\ I_1(t+1) = h\sum\limits_{i=1}^{a_1} I(t,i), \\ I_2(t+1) = h\sum\limits_{i=a_1+1}^{a_2} I(t,i), \\ S(1) = S_0, I_1(1) = I_{00}, I_2(1) = I_0, \end{cases}$$

where h is the step size.

We first divide the infection age into two periods, one is the exposed period from 0 to a_1 (non-infectious), and the other is the infectious period from a_1 to a_2 (infectious). The total numbers in the exposed population $I_1(t)$ and the infected population $I_2(t)$ at time t are described by

$$I_1(t) = \int_0^{a_1} I(t,a)da, \quad I_2(t) = \int_{a_1}^{a_2} I(t,a)da.$$

Assume that the maximum age for the infected population is 21 days and the three age-dependent functions are taken as follows [15],

$$\beta(a) = \begin{cases} 0, & 0 \le a \le 5, \\ 0.01(1 + \frac{\sin(a-5)\pi}{20}), & a > 5, \end{cases}$$

$$v(a) = 0.004 + 0.005 + e^{-a-5} + 0.001, \quad c_1(a) = 0.6(1 - e^{-a}).$$

Since $\Re_0$ is very sensitive to the key parameters of (3) and further determines the stability of the equilibria, it is critical to conduct sensitivity analysis to show the influence of parameters on $\Re_0$. By fixing the parameter values as shown in Fig. 1, it can be found that $\Re_0$ decreases with the increasing of ε and finally tends to a stable value. It means that a large value of ε ensures that $\Re_0 < 1$, which indicates that the infectious capacity of high-risk infectious diseases will be reduced and further controlled, and then limited medical resources can be shared with low-risk infectious diseases. In Fig. 1(b) and (c), two limit cases for ε are discussed. For $\varepsilon = 0$, $\Re_0$ is a constant which is greater than 1, and values of k have no influence on $\Re_0$.

Biologically, the extent of high-risk infectious disease outbreaks in this case is such that limited medical resources are preferentially allocated to patients infected with high-risk infectious diseases. For $\varepsilon = 1$, it is observed that $\Re_0$ increases from $\Re_0 < 1$ to $\Re_0 > 1$ when k and b_2 increase. Because b_2 denotes the effect of medical resource limitation on the treatment for low-risk infectious disease and k is the number of people infected by low-risk infectious disease, it implies that when the number of people infected by high-risk infectious diseases is less than a certain threshold (i.e., $\Re_0 < 1$), the impact of medical resources on treating low-risk infectious diseases is increasing, thus some of the medical resources can be allocated to patients with low-risk diseases. As b_2 and k increase (i.e., $\Re_0 > 1$), because patients with low-risk diseases consume a part of the medical resources, the medical resources for treating high-risk diseases are reduced, resulting in outbreaks of high-risk diseases. This suggests that equitable distribution for the limited medical resources is significant when treating low-risk and high-risk diseases. In Fig. 2, three-dimensional diagrams are carried out to address the effects of key parameters ε, b_2 and k on $\Re_0$.

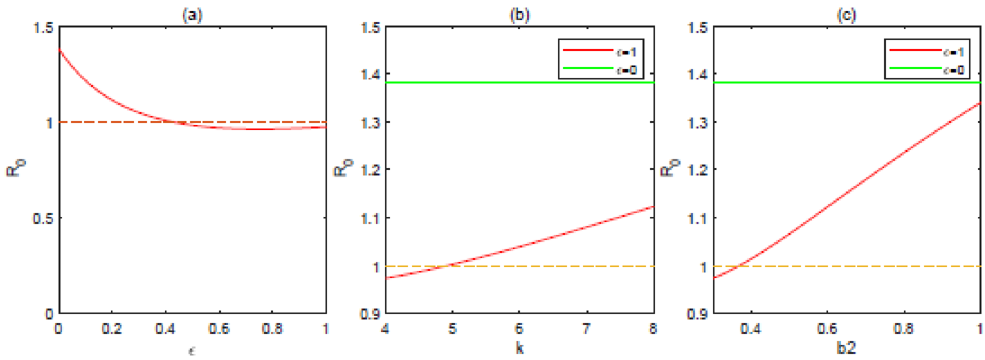

Figure 1: The plots show sensitivity of ε, k and b_2 on $\Re_0$. Parameters are $a_1 = 5, A = 0.1, \mu = 0.011, v_1 = 0.005$, and $(a) b_2 = 0.3, k = 4; (b) b_2 = 0.3; (c) k = 4$.

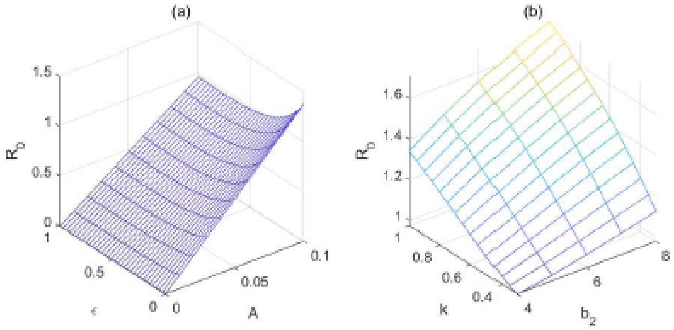

Figure 2: (a) shows sensitivity of A and ε on $\Re_0$, (b) shows sensitivity of b_2 and k on $\Re_0$. Parameters are $a_1 = 5, \mu_s = 0.007, \beta_2 = 0.001, v_1 = 0.005$, and $(a) b_2 = 0.3, k = 4; (b) A = 0.1, \varepsilon = 1$.

In addition, if we fix parameter values as shown in Fig. 2(a), let $A = 0.1$ and $\varepsilon = 1$, by calculation we have $\Re_0 \approx 0.9741 < 1$, it follows from Theorems 4.1 and Theorems 4.5 that E_0 is globally asymptotically stable (Fig. 3). It is observed that the number in the susceptible population tends to $S \approx 9.09$ and the number in the infected population tends to $I = 0$, which means that the disease will eventually die out. Let $A = 1$ and $\varepsilon = 0$, $\Re_0 \approx 13.8103 > 1$. Theorems 4.2 and Theorems 4.6 show that $\bar{E}$ is globally asymptotically stable (Fig. 4). It is noted that the number in the susceptible population tends to stable values, i.e., $\bar{S} \approx 48.98, I_1 \approx 2.306, I_2 \approx 0.7579$. While the number in the infected population is stabilized at a positive stationary distribution of age a, that is, the disease will eventually form an endemic disease.

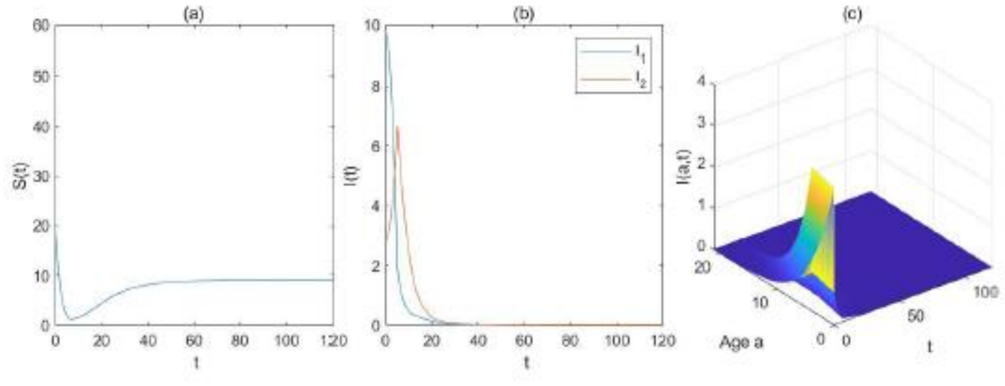

Figure 3: The plots show the dynamical behavior for $\Re_0 < 1$. Parameters are $a_1 = 5, A = 0.1, \varepsilon = 1, \mu_s = 0.007, k = 4, \beta_2 = 0.001, b_2 = 0.3, v_1 = 0.005$.

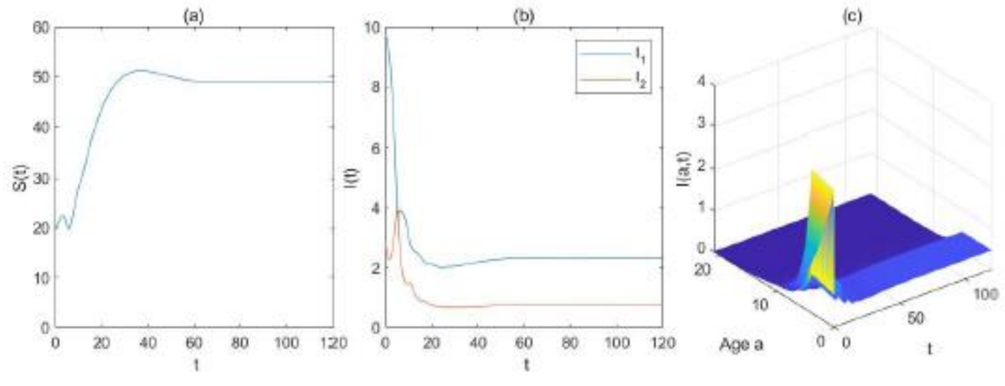

Figure 4: The plots show the dynamical behavior for $\Re_0 > 1$. Parameters are $a_1 = 5, A = 1, \varepsilon = 0.1, \mu_s = 0.007, k = 4, \beta_2 = 0.001, b_2 = 0.3, v_1 = 0.005$.

6. Conclusions and discussions

Mathematical modeling is an important tool to study the transmission dynamics of infectious diseases, which plays a vital role in controlling the spread of diseases and predicting the outcomes of prevention and control measures [12–26]. The emergence of some infectious diseases is usually accompanied by a series of social problems due to their suddenness, such as a shortage of medical resources, triaging pressure on medical staff etc. [20–25]. In this paper, we establish an age-structured infectious disease model with limited medical resources to investigate the impacts of limited medical resources and triaging pressures on the transmission dynamics of diseases.

Based on the assumptions that the infection rate, mortality rate, natural recovery rate and maximum treatment rate are age-dependent functions, the asymptotic smoothness and persistence of the solution semi-flow are investigated. Then we derive threshold conditions for the existence of the global attractor. By constructing appropriate Lyapunov functions, it is proved that the infection-free equilibrium is globally asymptotically stable if $\Re_0 < 1$. If $\Re_0 > 1$, then the infection equilibrium is globally asymptotically stable and the disease persists when $\Re_0 > 1$.

It is found that a large value of ε ensures that $\Re_0 < 1$, which reveals that the infectious capacity of the high-risk infectious diseases will be reduced and further controlled, and then limited medical resources can be shared with low-risk infectious diseases (details see Fig. 1). From Fig. 1(b) and (c), for $\varepsilon = 0$, $\Re_0 > 1$ always holds true, which implies that high-risk infectious disease outbreaks and so limited medical resources are preferentially allocated to patients infected with high-risk infectious diseases. For $\varepsilon = 1$, $\Re_0$ increases from $\Re_0 < 1$ to $\Re_0 > 1$ when k and b_2 increase, biologically, when the number of people infected by high-risk infectious diseases is less than a certain threshold (i.e., $\Re_0 < 1$), the impact of medical resources on treating low-risk infectious diseases is increasing, thus parts of the limited medical resources can be allocated to patients with low-risk diseases. Increasing b_2 and k such that $\Re_0 > 1$, owing to patients with low-risk diseases consuming a part of the medical resources, the medical resources for treating high-risk diseases are reduced, resulting in outbreaks of high-risk diseases. In reality, we should increase the value of ε and decrease the value of both b_2 and k to make sure that $\Re_0 < 1$, which also suggests that equitable distribution of the limited medical resources is reasonable and inevitable when medical staff are facing low-risk and high-risk diseases simultaneously.

Compared with previous studies [23, 27, 28], some highlights of the paper are listed as follows: (1) an age-structured infectious disease model concerning limited medical resources and triaging pressure is proposed and analyzed, which simultaneously considers deaths (including includes both the disease-related death and the natural death), self-healing, and hospital treatment affected by medical resources in people infected with high-risk diseases, and parameter ε is used to describe the allocation of medical resources; (2) conditions for the global stability of the system are derived; (3) biologically, when the number of patients infected by high-risk infectious diseases is small, medical resources are not limited. While the number of patients with high-risk infectious diseases is large, medical staff will face triaging pressure and the increase of infection age will force them to treat only patients with high-risk infectious diseases.

There is still a lot of further work to be studied. We regard the number of patients infected with low-risk infectious diseases (I_2) as a constant every year. In fact, the number of patients infected with low-risk infectious diseases varies from year to year. Therefore, if the change of I_2 is taken into account, then the dynamic behavior of the system will be more complex. It is very interesting to show how the two infectious diseases with differing risks interact, and how limited medical resources and triaging pressure affect the transmission and treatment of the two diseases. We leave this work as our future study.

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