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Global stability of a diffusive Leishmaniasis model with direct and indirect infection rate

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Abstract

Visceral leishmaniasis (VL), or black fever (kala-azar), is a fatal parasitic disease that infects a hosts internal organs. According to the World Health Organization (WHO), leishmaniasis is a major public health problem that has been neglected, and the control measures in various seriously infected areas have not been successful. Therefore, in order to analyze and control the spread of leishmaniasis, we establish a diffusive model with direct and indirect infection rate. Firstly, we prove the uniform bounds of solutions of the system, and analyze the sensitivity of the parameters. Secondly, sufficient conditions for the existence of the disease-free equilibrium and the endemic equilibrium are given, respectively. In addition, the stability of the model is studied in local and global sense by using the Routh Hurwitz criterion and Lyapunov theory, we prove that the disease-free equilibrium is globally asymptotically stable when the basic reproduction number $R_0 \leqslant 1$ and the endemic equilibrium is globally asymptotically stable when the basic reproduction number $R_0 > 1$. Finally, the theoretical results of the diffusive Leishmaniasis model with direct and indirect infection rate are verified by simulation. The results show that direct or indirect infection rates may affect the prevalence of the disease.

Keywords: Leishmaniasis disease, direct and indirect infection rate, diffusive model, globally asymptotically stable, Lyapunov functions.

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1. Introduction

Leishmaniasis is the world's second largest vector borne disease parasitic on humans, especially caused by protozoan parasites belonging to the genus Leishmania. Visceral leishmaniasis (VL), post kala azar cutaneous leishmaniasis (PKDL), cutaneous leishmaniasis (CL) and cutaneous leishmaniasis involve mucosal lesions, also known as mucocutaneous leishmaniasis (MCL), which are four different clinical manifestations of the disease, the main symptoms are weight loss, anemia, hepatosplenomegaly and irregular fever. Contrary to well-known NTDS such as: endemic treponematoses, dracunculiasis, and trypanosomiasis, if patients do not receive treatment of kala azar, more than 95% of cases may be fatal. However, even after treatment, VL infection can still become post kala azar cutaneous leishmaniasis (PKDL) ([31]). It has resulted in the death of 20,000 to 40,000 people worldwide ([40]), with 200,000 to

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400,000 new cases every year ([18]). India, East Africa and Brazil are mainly affected by it. Nearly 5000 to 9000 cases of visceral leishmaniasis occur every year, but only 25% to 45% of cases are reported to the World Health Organization (WHO) ([9]). Considering its transmission and death, it is considered as one of the major parasitic diseases.

In order to control and prevent leishmaniasis, we can diminish the morbidity and mortality of leishmaniasis through early diagnosis and effective and timely treatment. It also helps to diminish the transmission rate and observe the spread of diseases. Treatment of leishmaniasis, especially visceral leishmaniasis. Vector control helps to reduce the spread of diseases by reducing the number of sandflies. To this end, we can effectively reduce the spread of leishmaniasis through pesticide spraying, personal protection, community adaptation education and mobilization, etc. ([2, 7, 12, 32, 37, 39]).

Epidemiology plays a significant role in different disciplines like medical, engineering, chemistry, physics, economics and other disciplines. A diseases model is investigated with the help of well-known branches of mathematics like spatio-temporal, stochas-tic, fractional, and fractal fractional ([1, 29, 30, 33–35]). In 1996, Dye introduced the ordinary differential equation (ODE) model to describe the prevalence of VL, but this model only considers susceptible, latent, infectious, and recovering populations ([14]). Courtney et al. ([10]) improved Dye's model, taking the dog population as an infection source, and determined that the human infection rate was significantly affected by the size of the dog population. In 2013, Ribas et al. ([36]) proposed a mathematical model for the optimal control of zoonotic VL. Agyingi et al. ([3]) proposed a model considering the transmission dynamics of leishmaniasis. ELmojtaba et al. ([15]) conducted a mathematical analysis on the dynamics of Sudan, the results show that human treatment helps in disease control, and its synergy with vector control will more likely result in the elimination of the disease. Almeida et al. ([11]) proposed a mathematical model of the immune systems response in CL. In 2017, Boukhalfa et al. ([8]) proposed a mathematical model to describe the dynamics of visceral leishmaniasis in the dog population, and this study identifies the key parameters that play a key role on the disease dynamics, and thereby contributing in the design of effective control strategies. Coffeng et al. ([9]) investigated the detection, control and impact of visceral leishmaniasis VL in the Indian subcontinent. In 2018, Mubayi et al. ([28]) studied the epidemiological comparison of different cutaneous leishmaniasis outbreaks in Madrid and Tolima regions by estimating the number of reproduction. Ghosh et al. ([16]) studied the epidemiology of post kala azar cutaneous leishmaniasis (PKDL) in different regions of India in 2021, and proposed that PKDL caused many social problems. In 2023, Wang et al. ([41]) investigated the effects of spatial heterogeneity and temporal periodicity on disease transmission, a nonlocal periodic reaction-diffusion equation model is developed, explain that periodic delays can reduce the risk of disease transmission under certain conditions. Different from the nonlinear incidence rate of other articles ([17, 19–21, 27, 42, 44, 45, 47]), we not only consider spatial diffusion, but also introduce the nonlinear infection rate bI^p $(0 < p < 1)$ between infected individuals and infected vectors, therefore, the indirect infection rate of susceptible and infected individuals can be expressed as αbS^qI^p, where α indicates the contact rate between susceptible population and infection vector. This is related to the chemotaxis of mammals in spatial movement, chemotaxis ([22, 23]), the process by which organisms (or cells) migrate according to an external chemical gradient, in biological populations, orderly flocks of birds and fish occur by changing the movement of individuals relative to their neighbors. In infectious diseases, there are also chemotaxis phenomena among susceptible individuals, infected vectors and infected individuals. On the one hand, susceptible individuals may indirectly become latent individuals in poultry by contacting with infectious vectors. On the other hand, susceptible individuals may also become latent individuals by direct contact with infected individuals in the spatial movement of the population. At the same time, compared with the indirect infection, the direct infection rate will be higher than the indirect infection rate, so we use βS^qI to represent the direct infection rate of susceptible population and infected population, where β represents the probability of direct contact between susceptible populations and infected populations. Based on the above analysis, we will propose a diffusive Leishmaniasis model with direct and indirect infection rate.

The organizational structure of this paper is as follows. In Section 2, we establish a diffusive Leishmaniasis model with direct and indirect infection rate, introduce the flow chart (Figure 1), and give

parameter explanations in Table 1. In Section 3, we prove the uniform bounds of solutions of the system, and investigate the sensitivity of the parameters. In Section 4, sufficient conditions for the existence of the disease-free equilibrium and the endemic equilibrium are given, respectively. In addition, the local stability analysis of the disease-free equilibrium and the endemic equilibrium are obtained, respectively. In Section 5, by constructing various Lyapunov functions, the global stability analysis of the disease-free equilibrium point and the endemic equilibrium are given, respectively. Some numerical simulations and examples are presented in Section 6. Lastly, we end this paper with some conclusions and significance in Section 7.

2. Formulation of model

In biology, spatial diffusion plays an important role in describing the spread of infectious diseases. In order to design effective prevention and control measures, it is indispensable to study the spatial diffusive behavior of individuals. The spatial diffusive model is widely used in the research of spatial transmission of infectious diseases ([24, 26, 43, 46]). Therefore, we will propose a diffusive leishmaniasis model in this section. On the other hand, in order to better describe the disease transmission between susceptible and infected individuals, the nonlinear incidence form I^pS^q is used in certain situation, where $0 < p \leqslant 1$ and q are positive constants. Several mathematical models with this incidence rate have been proposed to investigate the dynamic of epidemic disease ([6, 20]). Different from the nonlinear incidence of the above articles, we will also consider the direct infection rate and indirect infection rate between susceptible individuals and infected individuals, which are represented by βS^qI and αbS^qI^p, respectively. So we proposed a diffusive Leishmaniasis model with direct and indirect infection rate as follows

$$\begin{cases} \frac{\partial S}{\partial t} - d_S\Delta S = \Lambda - \alpha bS^qI^p - \beta S^qI - \delta S, & x \in \Omega,\ t \geqslant 0,\\ \frac{\partial L}{\partial t} - d_L\Delta L = \alpha bS^qI^p + \beta S^qI - (\sigma+\delta)L, & x \in \Omega,\ t \geqslant 0,\\ \frac{\partial I}{\partial t} - d_I\Delta I = \sigma L - (\delta+\mu+r)I, & x \in \Omega,\ t \geqslant 0,\\ \frac{\partial R}{\partial t} - d_R\Delta R = rI - \delta R, & x \in \Omega,\ t \geqslant 0,\\ \frac{\partial S(t,x)}{\partial n} = \frac{\partial L(t,x)}{\partial n} = \frac{\partial I(t,x)}{\partial n} = \frac{\partial R(t,x)}{\partial n} = 0, & x \in \partial\Omega,\ t \geqslant 0,\\ S(0,x) = S_0(x) \geqslant 0,\ L(0,x) = L_0(x) \geqslant 0, & x \in \Omega,\\ I(0,x) = I_0(x) \geqslant 0,\ R(0,x) = R_0(x) \geqslant 0, & x \in \Omega, \end{cases} \tag{2.1}$$

where Ω is a bounded smooth domain of R^N $(N \geqslant 1)$, the whole population is divided into four compartments named as: susceptible populations, latent populations (infected but not infectious), infected and recovered populations, denoted by $S(x,t)$,$L(x,t)$,$I(x,t)$ and $R(x,t)$, respectively as shown in Figure 1. The parameters $\Lambda, \delta, \sigma, \beta, \alpha, p, b, q, r$ and μ are positive constants, and Λ is the birth rate of the susceptible individuals, δ represented as mortality rate of people, σ signifies the transmission rate of latent populations to infected populations and then again enter into infected class, β represents the probability of direct contact between susceptible populations and infected populations, α represents the probability of contact between susceptible individuals and infection vector, b is the infection parameter of infected individuals to the vector, r signifies natural recovery rate of infected populations, μ represents the death rate of infected populations due to leishmaniasis. The nonlinear infection rate βS^qI denotes the direct transmission of diseases between susceptible and infected individuals, and the nonlinear infection rate αbS^qI^p denotes the indirect infection transmission between susceptible and infected individuals. The positive constants d_S, d_L, d_I, and d_R are the diffusive rates of susceptible individuals, latent populations (infected but not infectious), infectious, and recovered populations, respectively. The parameters and physical relevance is summarized in Table 1. Homogeneous Neumann boundary conditions imply that there is no population flow across the boundary $\partial\Omega$. For any given continuous and nonnegative initial data $\big(S_0(x), L_0(x), I_0(x), R_0(x)\big)$, system (2.1) admits a classical solution $\big(S(x,t), L(x,t), I(x,t), R(x,t)\big)$ by the standard existence theory of parabolic equations. We define the basic reproduction number:

$$R_0 = \left(\frac{\Lambda}{\delta}\right)^q \frac{(\beta+\alpha b)\sigma}{(\sigma+\delta)(\delta+\mu+r)}.$$

By direct calculation it can be concluded that (2.1) has disease-free equilibrium (DFE) which is the only non-negative constant equilibrium if $R_0 \leqslant 1$. We use (S_0, L_0, I_0, R_0) to denote the DFE. The system (2.1) has a constant endemic equilibrium (EE) if $R_0 > 1$, which is unique. We use (S_*, L_*, I_*, R_*) to denote the constant EE if it exists. The goal of this paper is to prove the global asymptotic stability of DFE and EE by constructing various forms of Lyapunov functions.

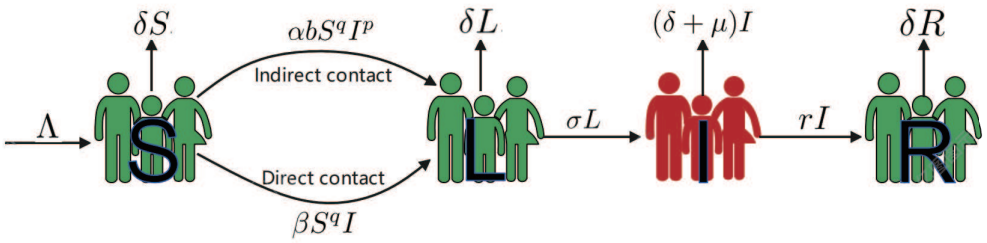


Figure 1: System dynamic diagram of VL transmission model.

Table 1: Biological description of the parameters.

Parameter	Environmental Interpretation	Value
(S_0, L_0, I_0, R_0)	Initial concentrations	$(\sin \pi x, \sin \pi x, \sin \pi x, \sin \pi x)$
Λ	Represented the birth rate of the susceptible individuals	1
δ	Represented as mortality rate of people	0.5
σ	Signifies the transmission rate of latent individuals to infectious populations and then again enter into infected class	0.4
β	Represents the probability of direct contact between susceptible populations and infected populations	0.5(DFE) or 0.9(EE)
α	Represents the probability of contact between susceptible individuals and infection vector	0.5
b	Represents is the infection multiple of infected individuals to the vector	0.4
r	Signifies natural recovery rate of infected individuals	0.5
μ	Represents the death rate of people due to leishmaniasis	0.5
d_S	Represents diffusive rates of susceptible individuals	1
d_L	Represents diffusive rates of latent populations(infected but not infectious)	1
d_I	Represents diffusive rates of infectious populations	1
d_R	Represents diffusive rates of recovered populations	1
q	Represents a positive constants	-
p	Represents a positive constants	-

3. Analysis of model

3.1. Uniform bounds of solutions of system (2.1)

Proposition 3.1. According to the initial data, there is a positive number C_1 such that the solution (S, L, I, R) of (2.1) satisfies

$$\|S(.,t)\|_{l^\infty(\Omega)} + \|L(.,t)\|_{l^\infty(\Omega)} + \|I(.,t)\|_{l^\infty(\Omega)} + \|R(.,t)\|_{l^\infty(\Omega)} \leqslant C_1, \qquad \forall t \geqslant 0.$$

In addition, there is a positive number C_2 independent of the initial data such that

$$\|S(.,t)\|_{l^\infty(\Omega)} + \|L(.,t)\|_{l^\infty(\Omega)} + \|I(.,t)\|_{l^\infty(\Omega)} + \|R(.,t)\|_{l^\infty(\Omega)} \leqslant C_2, \quad \forall t \geqslant T, \tag{3.1}$$

for some large time $T > 0$.

Proof. Let

$$V(t) = \int_\Omega \big[S(x,t) + L(x,t) + I(x,t) + R(x,t)\big]\,dx, \quad \forall t \geqslant 0.$$

According to (2.1), by calculation we have

$$\frac{dV(t)}{dt} = \int_\Omega \big[\Lambda - \delta S - \delta L - (\delta+\mu)I - \delta R\big]\,dx \leqslant \int_\Omega \Lambda dx - \delta V(t).$$

Thus

$$\int_\Omega [S(x,t)+L(x,t)+I(x,t)+R(x,t)]dx \leqslant e^{-\delta t}\int_\Omega [S_0(x)+L_0(x)+I_0(x)+R_0(x)]dx + \frac{|\Omega|\Lambda}{\delta}(1-e^{-\delta t}), \ \forall t \geqslant 0.$$

In view of [13, Lemma 2.1] and the positiveness of S, L, I, and R, we obtain

$$\|S(.,t)\|_{l^\infty(\Omega)} + \|L(.,t)\|_{l^\infty(\Omega)} + \|I(.,t)\|_{l^\infty(\Omega)} + \|R(.,t)\|_{l^\infty(\Omega)} \leqslant C_1, \ \ \forall t \geqslant 0,$$

and

$$\limsup_{t\to\infty} \int_\Omega \big[S(x,t) + L(x,t) + I(x,t) + R(x,t)\big]\,dx \leqslant \frac{|\Omega|\Lambda}{\delta}.$$

Combined with [13, Lemma 2.1], we can get that (3.1) holds.

3.2. Analysis of sensitivity of parameters

The parameters used in this model play a significant role in the dynamic process of disease. In this section, we analyze the sensitivity of parameters as follows

$$\xi_{n_\sigma} = \frac{\frac{\partial R_0}{R_0}}{\frac{\partial \sigma}{\sigma}} = \frac{\sigma}{R_0}\times\frac{\partial R_0}{\partial \sigma} = \frac{\delta}{\sigma+\delta} > 0, \qquad \xi_{n_\beta} = \frac{\frac{\partial R_0}{R_0}}{\frac{\partial \beta}{\beta}} = \frac{\beta}{R_0}\times\frac{\partial R_0}{\partial \beta} = \frac{\beta}{\beta+\alpha b} > 0,$$

$$\xi_{n_\Lambda} = \frac{\frac{\partial R_0}{R_0}}{\frac{\partial \Lambda}{\Lambda}} = \frac{\Lambda}{R_0}\times\frac{\partial R_0}{\partial \Lambda} = q > 0, \qquad \xi_{n_\delta} = \frac{\frac{\partial R_0}{R_0}}{\frac{\partial \delta}{\delta}} = \frac{\delta}{R_0}\times\frac{\partial R_0}{\partial \delta} = -q - \frac{\delta}{\delta+\mu+r} < 0,$$

$$\xi_{n_\mu} = \frac{\frac{\partial R_0}{R_0}}{\frac{\partial \mu}{\mu}} = \frac{\mu}{R_0}\times\frac{\partial R_0}{\partial \mu} = \frac{-\mu}{\delta+\mu+r} < 0, \qquad \xi_{n_r} = \frac{\frac{\partial R_0}{R_0}}{\frac{\partial r}{r}} = \frac{r}{R_0}\times\frac{\partial R_0}{\partial r} = \frac{-r}{\delta+\mu+r} < 0,$$

$$\xi_{n_\alpha} = \frac{\frac{\partial R_0}{R_0}}{\frac{\partial p}{p}} = \frac{\alpha}{R_0}\times\frac{\partial R_0}{\partial \alpha} = \frac{\alpha b}{\delta+\mu+r} > 0, \qquad \xi_{n_b} = \frac{\frac{\partial R_0}{R_0}}{\frac{\partial b}{b}} = \frac{b}{R_0}\times\frac{\partial R_0}{\partial b} = \frac{\alpha b}{\delta+\mu+r} > 0.$$

The above results conclude that $\sigma, \beta, \Lambda, \alpha, b$ are sensitive, and the rest are insensitive. □

4. Local stability analysis

In this section, we will use the following recognized results to study the local stability of the spatial diffusive model of leishmaniasis at the equilibrium point of the model.

We first study the existence and uniqueness of nonnegative equilibria, which fulfills

$$\Lambda - \alpha b S^q I^p - \beta S^q I - \delta S = 0, \quad \alpha b S^q I^p + \beta S^q I - (\sigma+\delta)L = 0, \quad \sigma L - (\delta+\mu+r)I = 0, \quad rI - \delta R = 0.$$

Obviously, system (2.1) always has a disease-free equilibrium:

$$E_0 = (S_0, L_0, I_0, R_0) = (\frac{\Lambda}{\delta}, 0, 0, 0).$$

Next, we need to determine the existence of endemic equilibrium (EE). It is clear that the EE of (2.1) satisfies:

$$\begin{cases} rI = \delta R, \ \sigma L = (\delta + \mu + r)I, \ \ \alpha b S^q I^p + \beta S^q I = (\sigma + \delta)L, \\ \Lambda = \alpha b S^q I^p + \beta S^q I + \delta S = \delta S + (\sigma + \delta)L. \end{cases}$$

So, we have $R = \frac{rI}{\delta}$, $I = \frac{\sigma}{(\delta+\mu+r)}L$, and $S = (\frac{(\sigma+\delta)(\delta+\mu+r)^p L}{\alpha b \sigma^p L^p + (\beta\sigma)(\delta+\mu+r)^{p-1}L})^{\frac{1}{q}}$. Therefore, L satisfies

$$\Lambda - (\sigma + \delta)L - \delta(\frac{(\sigma + \delta)(\delta + \mu + r)^p L}{\alpha b \sigma^p L^p + (\beta\sigma)(\delta + \mu + r)^{p-1}L})^{\frac{1}{q}} = 0.$$

Let us take function as $F(L) = \Lambda - (\sigma + \delta)L - \delta(\frac{(\sigma+\delta)(\delta+\mu+r)^p L}{\alpha b \sigma^p L^p + (\beta\sigma)(\delta+\mu+r)^{p-1}L})^{\frac{1}{q}} = 0$, $F'(L)$ can be obtained by taking the derivative, which is

$$F'(L) = -(\sigma + \delta) - \frac{\delta}{q}(\frac{(\sigma + \delta)(\delta + \mu + r)^p L}{\alpha b \sigma^p L^p + (\beta\sigma)(\delta + \mu + r)^{p-1}L})^{\frac{1}{q}-1}[\frac{\alpha b \sigma^p L^p (\sigma + \delta)(\delta + \mu + r)^p (1 - p)}{(\alpha b \sigma^p L^p + (\beta\sigma)(\delta + \mu + r)^{p-1}L)^2}].$$

Obviously, when $0 < p < 1$ or $p = 1$, $F'(L) < 0$. That is, when $0 < p < 1$, $F(L)$ is monotonically decreasing in $[0, +\infty)$ with $F(0) = \Lambda > 0$ and $\lim\limits_{L \to +\infty} F(L) < 0$, then there exists a unique positive constant L_* such that $F(L_*) = 0$. To sum up, when $0 < p < 1$ or $p = 1$ is satisfied, there will be a unique positive equilibrium point E_*. For the case $0 < p < 1$, the unique endemic equilibrium (S_*, L_*, I_*, R_*) can be explicitly expressed as

$$(S_*,\ L_*,\ I_*,\ R_*) = (\ (\frac{(\sigma + \delta)(\delta + \mu + r)^p}{\alpha b \sigma^p {L_*}^{p-1} + (\beta\sigma)(\delta + \mu + r)^{p-1}})^{\frac{1}{q}},\ L_*, \frac{\sigma}{\delta + \mu + r}L_*, \frac{r\sigma}{\delta(\delta + \mu + r)}L_*).$$

For the case $p = 1$, it is evident that (2.1) has a unique EE

$$\begin{aligned}(S_*,\ L_*,\ I_*,\ R_*) = (\ &(\frac{(\sigma + \delta)(\delta + \mu + r)}{(\alpha b + \beta)\sigma})^{\frac{1}{q}},\ \frac{\Lambda[(\alpha b + \beta)\sigma]^{\frac{1}{q}} - \delta[(\sigma + \delta)(\delta + \mu + r)]^{\frac{1}{q}}}{[(\alpha b + \beta)\sigma]^{\frac{1}{q}}(\sigma + \delta)}, \\ &\frac{\sigma}{\delta + \mu + r}(\frac{\Lambda[(\alpha b + \beta)\sigma]^{\frac{1}{q}} - \delta[(\sigma + \delta)(\delta + \mu + r)]^{\frac{1}{q}}}{[(\alpha b + \beta)\sigma]^{\frac{1}{q}}(\sigma + \delta)}), \\ &\frac{r\sigma}{\delta(\delta + \mu + r)}(\frac{\Lambda[(\alpha b + \beta)\sigma]^{\frac{1}{q}} - \delta[(\sigma + \delta)(\delta + \mu + r)]^{\frac{1}{q}}}{[(\alpha b + \beta)\sigma]^{\frac{1}{q}}(\sigma + \delta)})),\end{aligned}$$

the sufficient and necessary condition is $\Lambda[(\alpha b + \beta)\sigma]^{\frac{1}{q}} - \delta[(\sigma + \delta)(\delta + \mu + r)]^{\frac{1}{q}} > 0$, i.e., $R_0 > 1$. On the other hand, when the coefficients satisfy $\Lambda[(\alpha b + \beta)\sigma]^{\frac{1}{q}} - \delta[(\sigma + \delta)(\delta + \mu + r)]^{\frac{1}{q}} \leqslant 0$, i.e., $R_0 \leqslant 1$, system (2.1) has a unique equilibrium, which is the DFE, $E_0 = (S_0, L_0, I_0, R_0) = (\frac{\Lambda}{\delta}, 0, 0, 0)$.

Before developing our argument, let us set up the following notations.

(i) $0 = \mu_0 < \mu_1 < \mu_2 < \cdots < \mu_i \to +\infty$ are the eigenvalues of $-\Delta$ on Ω under homogeneous Neumann boundary condition.

(ii) $E(\mu_i)$ is the space of eigenfunctions corresponding to μ_i for $i = 0, 1, 2, \ldots$.

(iii) $X_{ij} := \{c \cdot \phi_{ij} | c \in R^4\}$, where ϕ_{ij} are orthonormal basis of $E(\mu_i)$ for $j = 1, 2, \ldots, dimE(\mu_i)$.

(iv) $X := \{E = (S, L, I, R) \in C^1(\overline{\Omega}) | \frac{\partial S}{\partial n} = \frac{\partial L}{\partial n} = \frac{\partial I}{\partial n} = \frac{\partial R}{\partial n} = 0, x \in \partial\Omega\}$, and so $X = \bigoplus_{i=1}^{\infty} X_i$, where $X_i = \bigoplus_{j=1}^{dimE(\mu_i)} X_{ij}$.

We next investigate the local stability of DFE and EE in Theorems 4.1 and 4.2, respectively.

Theorem 4.1. *The disease-free equilibrium* $E_0 = (S_0, L_0, I_0, R_0) = (\frac{\Lambda}{\delta}, 0, 0, 0)$ *is locally asymptotical stable (LAS) if* $R_0 \leqslant 1$.

Proof. Now, we discuss the local stability of the disease-free equilibrium (DFE) state $E_0 = (\frac{\Lambda}{\delta}, 0, 0, 0)$ of system (2.1). Let $G = \mathrm{diag}(d_S, d_L, d_I, d_R)$, $H = G\Delta + F_E(E_0) = G\Delta + (a_{ij})_{4\times 4}$, where

$$F_E(E_0) = \begin{pmatrix} -\delta & 0 & -\beta(\frac{\Lambda}{\delta})^q & 0 \\ 0 & -(\sigma+\delta) & \beta(\frac{\Lambda}{\delta})^q & 0 \\ 0 & \sigma & -(\delta+\mu+r) & 0 \\ 0 & 0 & r & -\delta \end{pmatrix}.$$

The linearization of (2.1) at the disease-free equilibrium $E_0 = (\frac{\Lambda}{\delta}, 0, 0, 0)$ can be expressed by $E_t = HE$, and for each $i \geqslant 1$, X_i is invariant under the operator H, so λ is the eigenvalue of H in X_i if and only if it is the eigenvalue of matrix $-\mu_i G + F_E(E_0)$. Consider the characteristic equation

$$\varphi_i(\lambda) := |\lambda I + \mu_i G - F_E(E_0)| = (\lambda + \mu_i d_S + \delta)(\lambda + \mu_i d_R + \delta)(\lambda^2 + a_i\lambda + b_i), \tag{4.1}$$

where

$$a_i = \mu_i d_L + \mu_i d_I + (\sigma+\delta) + (\delta+\mu+r),$$
$$b_i = {\mu_i}^2 d_L d_I + \mu_i d_L(\delta+\mu+r) + \mu_i d_I(\sigma+\delta) + (\sigma+\delta)(\delta+\mu+r) - \beta\sigma\left(\frac{\Lambda}{\delta}\right)^q.$$

Obviously, (4.1) has two roots $\lambda_1 = -d_S\mu_i - \delta$ and $\lambda_2 = -d_R\mu_i - \delta$. Notice if $R_0 \leqslant 1$, we can obtain $(\beta + \alpha b)\sigma(\frac{\Lambda}{\delta})^q \leqslant (\sigma+\delta)(\delta+\mu+r)$ and $\beta\sigma(\frac{\Lambda}{\delta})^q < (\sigma+\delta)(\delta+\mu+r)$, then $a_i > 0$ and $b_i > 0$. Thus, it follows from Routh-Hurwitz criterion that any root of the following equation

$$g(\lambda) = \lambda^2 + a_i\lambda + b_i = 0$$

has negative real part for $R_0 \leqslant 1$. Therefore, DFE is locally asymptotically stable. □

Theorem 4.2. *The endemic equilibrium* EE *is locally asymptotical stable (LAS) if* $R_0 > 1$.

Proof. When $R_0 > 1$, system (2.1) has a unique positive endemic equilibrium (EE). Now, we discuss the local stability of the endemic equilibrium (EE) state of system (2.1), the endemic equilibrium (EE) is as follows

$$(S_*,\ L_*,\ I_*,\ R_*) = \left(\left(\frac{(\sigma+\delta)(\delta+\mu+r)^p}{\alpha b\sigma^p {L_*}^{p-1} + (\beta\sigma)(\delta+\mu+r)^{p-1}}\right)^{\frac{1}{q}},\ L_*,\ \frac{\sigma}{\delta+\mu+r}L_*,\ \frac{r\sigma}{\delta(\delta+\mu+r)}L_*\right).$$

Let $G = \mathrm{diag}(d_S, d_L, d_I, d_R)$, $H = G\Delta + F_E(E_*) = G\Delta + (a_{ij})_{4\times 4}$, where

$$F_E(E_*) = \begin{pmatrix} -q\alpha b{S_*}^{q-1}I_*^p - q\beta{S_*}^{q-1} - \delta & 0 & -p\alpha b{S_*}^q I_*^{p-1} - \beta{S_*}^q & 0 \\ q\alpha b{S_*}^{q-1}I_*^p + q\beta{S_*}^{q-1} & -(\sigma+\delta) & p\alpha b{S_*}^q I_*^{p-1} + \beta{S_*}^q & 0 \\ 0 & \sigma & -(\delta+\mu+r) & 0 \\ 0 & 0 & r & -\delta \end{pmatrix}.$$

The linearization of (2.1) at the endemic equilibrium $E_* = (S_*, L_*, I_*, R_*)$ can be expressed by $E_t = HE$, and for each $i \geqslant 1$, X_i is invariant under the operator H, so λ is the eigenvalue of H in X_i if and only if it is the eigenvalue of matrix $-\mu_i G + F_E(E_*)$. Consider the characteristic equation

$$\varphi_i(\lambda) := \left|\lambda I + \mu_i G - F_E(E_*)\right| = (\lambda + d_R\mu_i + \delta)(\lambda^3 + A_i\lambda^2 + B_i\lambda + C_i) = 0, \tag{4.2}$$

for the convenience of calculation, we also make the following marks

$$m = q\alpha b{S_*}^{q-1}I_*^p + q\beta{S_*}^{q-1} + \delta > 0, \quad n = p\alpha b{S_*}^q I_*^{p-1} > 0,$$

where

$$\begin{aligned}
A_i &= \mu_i(d_S + d_L + d_I) + m + (\sigma+\delta) + \delta + (\delta+\mu+r),\\
B_i &= {\mu_i}^2(d_S d_L + d_S d_I + d_L d_I) + \mu_i[d_S(\sigma+2\delta+\mu+r) + d_L(m+\delta+\mu+r) + d_I(m+\sigma+\delta)]\\
&\quad + m(\sigma+2\delta+\mu+r) + \delta(\delta+\mu+r) + (((\beta+\alpha b)\sigma+1)^2 S_*^q - (\sigma+\delta)^2(\delta+\mu+r)^2,\\
C_i &= {\mu_i}^3 d_S d_L d_I + {\mu_i}^2[d_S d_L(\delta+\mu+r) + d_S d_I(\sigma+\delta) + d_L d_I m]\\
&\quad + \mu_i\{d_S[\delta(\delta+\mu+r) + (\delta+\mu+r+n+\beta S_*^q)\sigma] + d_L m(\delta+\mu+r) + d_I m(\sigma+\delta)\}\\
&\quad + m\delta(\delta+\mu+r) + (((\beta+\alpha b)\sigma+1)^2 S_*^q - (\sigma+\delta)^2(\delta+\mu+r)^2.
\end{aligned}$$

By direct calculation, we can obtain

$$\begin{aligned}
A_iB_i - C_i &= {\mu_i}^3({d_S}^2 d_L + {d_S}^2 d_I + d_S {d_L}^2 + d_S {d_I}^2 + {d_L}^2 d_I + d_L {d_I}^2 + 2 d_S d_L d_I)\\
&\quad + {\mu_i}^2[{d_S}^2(\sigma+2\delta+\mu+r) + (d_S d_L + d_S d_I)(2m+2\sigma+2\mu+2r+5\delta)]\\
&\quad + {\mu_i}^2 d_L d_I(m+\sigma+2\mu+2r+4\delta)\\
&\quad + \mu_i\{{d_L}^2(m+\delta+\mu+r) + {d_I}^2(m+\sigma+\delta) + d_S[(\sigma+2\delta+\mu+r)(m+1)]\}\\
&\quad + \mu_i d_L[(m+\delta+\mu+r)(\delta+1) + m + (((\beta+\alpha b)\sigma)^2 S_*^q - (\sigma+\delta)^2(\delta+\mu+r)^2]\\
&\quad + \mu_i d_I[(m+\delta)(\delta+\mu+r) + (\delta+\mu+r+n+\beta S_*^q)\sigma]\\
&\quad + m[(\sigma+\delta)^2 + (2\delta+\mu+r)^2] + (\sigma+\delta)(\delta+\mu+r)(\sigma+2\delta+m)\\
&\quad + (((\beta+\alpha b)\sigma)^2 S_*^q - (\sigma+\delta)^2(\delta+\mu+r)^2 + (m+n+\beta S_*^q)\sigma(2\delta+\mu+r).
\end{aligned}$$

Obviously, equation (4.2) has a root $\lambda_1 = -(d_R\mu_i + \delta)$. Notice $S_*^q = (\frac{\Lambda}{\delta})^q \frac{1}{R_0}$, when $[(\beta+\alpha b)\sigma]^2 S_*^q - (\sigma+\delta)^2(\delta+\mu+r)^2 > 0$, that is, when $(\frac{\Lambda}{\delta})^q \frac{(\beta+\alpha b)\sigma}{(\alpha+\delta)(\delta+\mu+r)} = R_0 > 1$ we can get $A_i > 0$, $B_i > 0$, $C_i > 0$ and $A_iB_i - C_i > 0$. Thus, it follows from Routh-Hurwitz criterion that any root of the following equation

$$g(\lambda) = \lambda^3 + A_i\lambda^2 + B_i\lambda + C_i = 0$$

has negative real part. Therefore, if $R_0 > 1$, EE is locally asymptotically stable. □

5. Global stability analysis

Theorem 5.1. *Assume that* $p = 1$, *the disease-free equilibrium* $E_0 = (S_0, L_0, I_0, R_0) = (\frac{\Lambda}{\delta}, 0, 0, 0)$ *is globally asymptotical stable (GAS) if* $R_0 \leqslant 1$.

Proof. For the disease-free equilibrium of (2.1), there are two cases. We define two different Lyapunov functions.

Case 1: $p = q = 1$, we define

$$V_1(t) = \int_\Omega [S - S_0 \ln S + L + \tfrac{\sigma+\delta}{\sigma} I + \tfrac{\beta S_0}{r} R]dx.$$

For all $t > 0$, by some straightforward computations, it follows from $R_0 \leqslant 1$ that

$$\begin{aligned}
\frac{dV_1(t)}{dt} &= -\int_\Omega d_S|\nabla S|^2(\frac{S_0}{S^2})dx + \int_\Omega \{(1-\frac{S_0}{S})[\Lambda - (\alpha b+\beta)SI - \delta S] + (\alpha b+\beta)SI - (\sigma+\delta)L\\
&\quad + \frac{\sigma+\delta}{\sigma}[\sigma L - (\delta+\mu+r)I] + \frac{\beta S_0}{r}(rI - \delta R)\}dx\\
&= -\int_\Omega d_S|\nabla S|^2(\frac{S_0}{S^2})dx + \int_\Omega \{\Lambda(2 - \frac{S_0}{S} - \frac{S}{S_0}) + [(\alpha b+\beta)(\frac{\Lambda}{\delta}) - \frac{(\sigma+\delta)(\delta+\mu+r)}{\sigma}]I\}dx.
\end{aligned}$$

From the relationship between geometric mean and arithmetic mean, we can obtain $\Lambda(2 - \frac{S_0}{S} - \frac{S}{S_0}) \leqslant 0$. Further we obtain $\frac{dV_1(t)}{dt} \leqslant 0$ and $\frac{dV_1(t)}{dt} = 0$ if and only if $(S_0, L_0, I_0, R_0) = (\frac{\Lambda}{\delta}, 0, 0, 0)$. Therefore, we can get that DFE is globally asymptotically stable when $p = q = 1$.

Case 2: $p = 1,\ q \neq 1$, we define

$$V_2(t) = \int_\Omega \{S[1 - \frac{1}{1-q}(\frac{S_0}{S})^q] + L + \frac{\sigma+\delta}{\sigma} I + \frac{\beta S_0}{r} R\} dx.$$

Since $R_0 \leqslant 1$ again, by direct computation, we have

$$\begin{aligned}\frac{dV_2(t)}{dt} &= -\int_\Omega q d_S |\nabla S|^2 (\frac{S_0{}^q}{S^{q+1}}) dx + \int_\Omega \{[1 - (\frac{S_0}{S})^q][\Lambda - (\alpha b + \beta) S^q I - \delta S] + (\alpha b + \beta) S^q I - (\sigma + \delta) L \\ &\quad + \frac{\sigma+\delta}{\sigma}[\sigma L - (\delta + \mu + r) I] + \frac{\beta S_0}{r}(rI - \delta R)\} dx \\ &= -\int_\Omega q d_S |\nabla S|^2 (\frac{S_0{}^q}{S^{q+1}}) dx + \int_\Omega \{\Lambda(1 - \frac{S}{S_0})[1 - (\frac{S_0}{S})^q] + [(\alpha b + \beta)(\frac{\Lambda}{\delta})^q - \frac{(\sigma+\delta)(\delta+\mu+r)}{\sigma}] I\} dx.\end{aligned}$$

From the relationship between geometric mean and arithmetic mean, we can obtain $\Lambda(1 - \frac{S}{S_0})[1 - (\frac{S_0}{S})^q] \leqslant 0$. Further we obtain $\frac{dV_2(t)}{dt} \leqslant 0$ and $\frac{dV_2(t)}{dt} = 0$ if and only if $(S_0, L_0, I_0, R_0) = (\frac{\Lambda}{\delta}, 0, 0, 0)$. Therefore, we can get that DFE is globally asymptotically stable when $p = 1,\ q \neq 1$.

To sum up, the above four situations are analyzed. Note that $\frac{dV_i(t)}{dt} = 0\ (i = 1, 2)$ if and only if $E_0 = (S_0, L_0, I_0, R_0) = (\frac{\Lambda}{\delta}, 0, 0, 0)$. Thus, $V_i(t)\ (i = 1, 2)$ are Lyapunov functions of (2.1) for four cases, respectively. By some standard arguments, it is evident that

$$(S(x,t), L(x,t), I(x,t), R(x,t)) \to (\frac{\Lambda}{\delta}, 0, 0, 0) \quad \text{in } [L^2(\Omega)]^4, \text{ as } t \to +\infty.$$

From the uniform boundedness (3.2) in Proposition 3.1, the parabolic L^p-theory, sobolev embedding theorem and a standard compactness argument guarantee that there exist a positive constant C and $T_0 > 0$ such that

$$\|S(.,t)\|_{C^2(\overline{\Omega})} + \|L(.,t)\|_{C^2(\overline{\Omega})} + \|I(.,t)\|_{C^2(\overline{\Omega})} + \|R(.,t)\|_{C^2(\overline{\Omega})} \leqslant C, \qquad \forall t \geqslant T_0.$$

Therefore, the Sobolev embedding theorem allows one to assert

$$(S(x,t), L(x,t), I(x,t), R(x,t)) \to (\frac{\Lambda}{\delta}, 0, 0, 0) \quad \text{in } [L^2(\Omega)]^4, \text{ as } t \to +\infty.$$

This implies that the DFE, $E_0 = (S_0, L_0, I_0, R_0) = (\frac{\Lambda}{\delta}, 0, 0, 0)$ attracts all solutions of (2.1). □

Theorem 5.2. *Assume that* $0 < p < 1$ *or* $p = 1$ *and* $R_0 > 1$. *Then the* EE *is globally asymptotically stable.*

Proof. For the endemic equilibrium of (2.1), there are four cases. We define four different Lyapunov functions.

Case 1: $p = q = 1$, we define

$$V_3(t) = \int_\Omega [S - S_* \ln S + L - L_* \ln L + \frac{\sigma+\delta}{\sigma}(I - I_* \ln I) + \frac{\beta S_*}{r}(R - R_* \ln R)] dx.$$

For all $t > 0$, a direct calculation yields

$$\begin{aligned}\frac{dV_3(t)}{dt} &= -[\int_\Omega d_S |\nabla S|^2 (\frac{S_*}{S^2}) dx + \int_\Omega d_L |\nabla L|^2 (\frac{L_*}{L^2}) dx + \frac{\sigma+\delta}{\sigma} \int_\Omega d_I |\nabla I|^2 (\frac{I_*}{I^2}) dx + \frac{\beta S_*}{r} \int_\Omega d_R |\nabla R|^2 (\frac{R_*}{R^2}) dx] \\ &\quad + \int_\Omega \{(1 - \frac{S_*}{S})[\Lambda - (\alpha b + \beta) SI - \delta S] + (1 - \frac{L_*}{L})[(\alpha b + \beta) SI - (\sigma + \delta) L] \\ &\quad + \frac{\sigma+\delta}{\sigma}(1 - \frac{I_*}{I})[\sigma L - (\delta + \mu + r) I] + \frac{\beta S_*}{r}(1 - \frac{R_*}{R})(rI - \delta R)\} dx \\ &= -[\int_\Omega d_S |\nabla S|^2 (\frac{S_*}{S^2}) dx + \int_\Omega d_L |\nabla L|^2 (\frac{L_*}{L^2}) dx + \frac{\sigma+\delta}{\sigma} \int_\Omega d_I |\nabla I|^2 (\frac{I_*}{I^2}) dx + \frac{\beta S_0}{r} \int_\Omega d_R |\nabla R|^2 (\frac{R_*}{R^2}) dx]\end{aligned}$$

$$+\int_\Omega\{\delta S_*(2-\frac{S_*}{S}-\frac{S}{S_*})+\alpha bS_*I_*(3-\frac{S_*}{S}-\frac{LI_*}{L_*I}-\frac{SIL_*}{S_*I_*L})$$
$$+\beta S_*I_*(4-\frac{S_*}{S}-\frac{LI_*}{L_*I}-\frac{IR_*}{I_*R}-\frac{SRL_*}{S_*R_*L})\}dx,$$

where we used the fact $\Lambda=(\alpha b+\beta)S_*I_*+\delta S_*$, $(\alpha b+\beta)S_*I_*=(\sigma+\delta)L_*$, $\sigma L_*=(\delta+\mu+r)I_*$, $rI_*=\delta R_*$. From the relationship between geometric mean and arithmetic mean, we can get $\delta S_*(2-\frac{S_*}{S}-\frac{S}{S_*})\leqslant 0$, $\alpha bS_*I_*(3-\frac{S_*}{S}-\frac{LI_*}{L_*I}-\frac{SIL_*}{S_*I_*L})\leqslant 0$, $\beta S_*I_*(4-\frac{S_*}{S}-\frac{LI_*}{L_*I}-\frac{IR_*}{I_*R}-\frac{SRL_*}{S_*R_*L})\leqslant 0$, and $\delta S_*(2-\frac{S_*}{S}-\frac{S}{S_*})=0$, $\alpha bS_*I_*(3-\frac{S_*}{S}-\frac{LI_*}{L_*I}-\frac{SIL_*}{S_*I_*L})=0$, $\beta S_*I_*(4-\frac{S_*}{S}-\frac{LI_*}{L_*I}-\frac{IR_*}{I_*R}-\frac{SRL_*}{S_*R_*L})=0$ if and only if $(S,L,I,R)=(S_*,L_*,I_*,R_*)$, i.e., $\frac{dV_3(t)}{dt}\leqslant 0$ and $\frac{dV_3(t)}{dt}=0$ if and only if $(S,L,I,R)=(S_*,L_*,I_*,R_*)$. According to the limit theory and Theorem 4.2, we can get that EE is globally asymptotically stable when $p=q=1$.

Case 2: $p=1$, $q\neq 1$, we define

$$V_4(t)=\int_\Omega\{S[1-\tfrac{1}{1-q}(\tfrac{S_*}{S})^q]+L-L_*\ln L+\tfrac{\sigma+\delta}{\sigma}(I-I_*\ln I)+\tfrac{\beta S_*^q}{r}(R-R_*\ln R)\}dx.$$

Through some elementary calculations, noting that $\Lambda=(\alpha b+\beta)S_*^qI_*+\delta S_*$, $(\alpha b+\beta)S_*^qI_*=(\sigma+\delta)L_*$, $\sigma L_*=(\delta+\mu+r)I_*$, $rI_*=\delta R_*$, we have

$$\begin{aligned}\frac{dV_4(t)}{dt}&=-[\int_\Omega qd_S|\nabla S|^2(\frac{S_*^q}{S^{q+1}})dx+\int_\Omega d_L|\nabla L|^2(\frac{L_*}{L^2})dx+\frac{\sigma+\delta}{\sigma}\int_\Omega d_I|\nabla I|^2(\frac{I_*}{I^2})dx+\frac{\beta S_*^q}{r}\int_\Omega d_R|\nabla R|^2(\frac{R_*}{R^2})dx]\\&+\int_\Omega\{[1-(\frac{S_*}{S})^q][\Lambda-(\alpha b+\beta)S^qI-\delta S]+(1-\frac{L_*}{L})[(\alpha b+\beta)S^qI-(\sigma+\delta)L]\\&+\frac{\sigma+\delta}{\sigma}(1-\frac{I_*}{I})[\sigma L-(\delta+\mu+r)I]+\frac{\beta S_*^q}{r}(1-\frac{R_*}{R})(rI-\delta R)\}dx\\&=-[\int_\Omega qd_S|\nabla S|^2(\frac{S_*^q}{S^{q+1}})dx+\int_\Omega d_L|\nabla L|^2(\frac{L_*}{L^2})dx+\frac{\sigma+\delta}{\sigma}\int_\Omega d_I|\nabla I|^2(\frac{I_*}{I^2})dx+\frac{\beta S_*^q}{r}\int_\Omega d_R|\nabla R|^2(\frac{R_*}{R^2})dx]\\&+\int_\Omega\{\delta S_*(1-\frac{S}{S_*})[1-(\frac{S_*}{S})^q]+\alpha bS_*^qI_*(3-\frac{S_*^q}{S^q}-\frac{LI_*}{L_*I}-\frac{S^qIL_*}{S_*^qI_*L})\\&+\beta S_*^qI_*(4-\frac{S_*^q}{S^q}-\frac{LI_*}{L_*I}-\frac{IR_*}{I_*R}-\frac{S^qRL_*}{S_*^qR_*L})\}dx,\end{aligned}$$

where we used the fact $\Lambda=(\alpha b+\beta)S_*^qI_*+\delta S_*$, $(\alpha b+\beta)S_*^qI_*=(\sigma+\delta)L_*$, $\sigma L_*=(\delta+\mu+r)I_*$, $rI_*=\delta R_*$. From the relationship between geometric mean and arithmetic mean, we can get $\delta S_*(1-\frac{S}{S_*})[1-(\frac{S_*}{S})^q]\leqslant 0$, $\alpha bS_*^qI_*(3-\frac{S_*^q}{S^q}-\frac{LI_*}{L_*I}-\frac{S^qIL_*}{S_*^qI_*L})\leqslant 0$, $\beta S_*^qI_*(4-\frac{S_*^q}{S^q}-\frac{LI_*}{L_*I}-\frac{IR_*}{I_*R}-\frac{S^qRL_*}{S_*^qR_*L})\leqslant 0$, and $\delta S_*(1-\frac{S}{S_*})[1-(\frac{S_*}{S})^q]=0$, $\alpha bS_*^qI_*(3-\frac{S_*^q}{S^q}-\frac{LI_*}{L_*I}-\frac{S^qIL_*}{S_*^qI_*L})=0$, $\beta S_*^qI_*(4-\frac{S_*^q}{S^q}-\frac{LI_*}{L_*I}-\frac{IR_*}{I_*R}-\frac{S^qRL_*}{S_*^qR_*L})=0$, if and only if $(S,L,I,R)=(S_*,L_*,I_*,R_*)$, i.e., $\frac{dV_4(t)}{dt}\leqslant 0$ and $\frac{dV_4(t)}{dt}=0$ if and only if $(S,L,I,R)=(S_*,L_*,I_*,R_*)$. According to the limit theory and Theorem 4.2, we can get that EE is globally asymptotically stable when $p=1$, $q\neq 1$.

Case 3: $p\neq 1$, $q=1$, we define

$$V_5(t)=\int_\Omega\{S-S_*\ln S+L-L_*\ln L+\tfrac{\alpha bS_*I_*^{p-1}}{\delta+\mu+r}I[1-\tfrac{1}{1-p}(\tfrac{I_*}{I})^p]+\tfrac{\beta S_*}{r}(R-R_*\ln R)\}dx.$$

In view of $\Lambda=(\alpha bI_*^p+\beta I_*)S_*+\delta S_*$, $(\alpha bI_*^p+\beta I_*)S_*=(\sigma+\delta)L_*$, $\sigma L_*=(\delta+\mu+r)I_*$, $rI_*=\delta R_*$, we have

$$\begin{aligned}\frac{dV_5(t)}{dt}&=-[\int_\Omega d_S|\nabla S|^2(\frac{S_*}{S^2})dx+\int_\Omega d_L|\nabla L|^2(\frac{L_*}{L^2})dx+\frac{\alpha bS_*^qI_*^{p-1}}{\delta+\mu+r}\int_\Omega pd_I|\nabla I|^2(\frac{I_*^p}{I^{p+1}})dx\\&+\frac{\beta S_*}{r}\int_\Omega d_R|\nabla R|^2(\frac{R_*}{R^2})dx]\\&+\int_\Omega\{(1-\frac{S_*}{S})[\Lambda-(\alpha bI^p+\beta I)S-\delta S]+(1-\frac{L_*}{L})[(\alpha bI^p+\beta I)S-(\sigma+\delta)L]\end{aligned}$$

$$
\begin{aligned}
&+\frac{\alpha b S_* I_*^{p-1}}{\delta+\mu+r}[1-(\frac{I_*}{I})^p][\sigma L-(\delta+\mu+r)I]+\frac{\beta S_*}{r}(1-\frac{R_*}{R})(rI-\delta R)\}dx\\
&=-[\int_\Omega d_S|\nabla S|^2(\frac{S_*}{S^2})dx+\int_\Omega d_L|\nabla L|^2(\frac{L_*}{L^2})dx+\frac{\alpha b S_* I_*^{p-1}}{\delta+\mu+r}\int_\Omega p d_I|\nabla I|^2(\frac{I_*^p}{I^{p+1}})dx\\
&+\frac{\beta S_*}{r}\int_\Omega d_R|\nabla R|^2(\frac{R_*}{R^2})dx]\\
&+\int_\Omega\{\delta S_*(2-\frac{S_*}{S}-\frac{S}{S_*})+\alpha b S_* I_*{}^p[3-\frac{S_*}{S}-\frac{LI_*^p}{L_*I^p}-\frac{SI^pL_*}{S_*I_*^pL}+(\frac{I^p}{I_*^p}-1)(1-\frac{I^{1-p}}{I_*^{1-p}})]\\
&+\beta S_* I_*(3-\frac{S_*}{S}-\frac{RI_*}{R_*I}-\frac{SIR_*}{S_*I_*R})\}dx.
\end{aligned}
$$

From the relationship between geometric mean and arithmetic mean, we can get $\delta S_*(2-\frac{S_*}{S}-\frac{S}{S_*})\leqslant 0$, $\alpha b S_* I_*{}^p[3-\frac{S_*}{S}-\frac{LI_*^p}{L_*I^p}-\frac{SI^pL_*}{S_*I_*^pL}+(\frac{I^p}{I_*^p}-1)(1-\frac{I^{1-p}}{I_*^{1-p}})]\leqslant 0$, $\beta S_* I_*(3-\frac{S_*}{S}-\frac{RI_*}{R_*I}-\frac{SIR_*}{S_*I_*R})\leqslant 0$, and $\delta S_*(2-\frac{S_*}{S}-\frac{S}{S_*})=0$, $\alpha b S_* I_*^p[3-\frac{S_*}{S}-\frac{LI_*^p}{L_*I^p}-\frac{SI^pL_*}{S_*I_*^pL}+(\frac{I^p}{I_*^p}-1)(1-\frac{I^{1-p}}{I_*^{1-p}})]=0$, $\beta S_* I_*(3-\frac{S_*}{S}-\frac{RI_*}{R_*I}-\frac{SIR_*}{S_*I_*R})=0$, if and only if $(S,L,I,R)=(S_*,L_*,I_*,R_*)$, i.e., $\frac{dV_5(t)}{dt}\leqslant 0$ and $\frac{dV_5(t)}{dt}=0$ if and only if $(S,L,I,R)=(S_*,L_*,I_*,R_*)$. According to the limit theory and Theorem 4.2, we can get that EE is globally asymptotically stable when $p\neq 1$, $q=1$.

Case 4: $p\neq 1$, $q\neq 1$, we define

$$
V_6(t)=\int_\Omega\{S[1-\frac{1}{1-q}(\frac{S_*}{S})^q]+L-L_*\ln L+\frac{\alpha b S_*^q I_*^{p-1}}{\delta+\mu+r}I[1-\frac{1}{1-p}(\frac{I_*}{I})^p]+\frac{\beta S_*^q}{r}(R-R_*\ln R)\}dx.
$$

As $\Lambda=(\alpha b S_*^q I_*^p+\beta S_* I_*)+\delta S_*$, $(\alpha b S_*^q I_*^p+\beta S_* I_*)=(\sigma+\delta)L_*$, $\sigma L_*=(\delta+\mu+r)I_*$, $rI_*=\delta R_*$, we can check that

$$
\begin{aligned}
\frac{dV_8(t)}{dt}&=-[\int_\Omega q d_S|\nabla S|^2(\frac{S_*^q}{S^{q+1}})dx+\int_\Omega d_L|\nabla L|^2(\frac{L_*}{L^2})dx+\frac{\alpha b S_*^q I_*^{p-1}}{\delta+\mu+r}\int_\Omega p d_I|\nabla I|^2(\frac{I_*^p}{I^{p+1}})dx\\
&+\frac{\beta S_*^q}{r}\int_\Omega d_R|\nabla R|^2(\frac{R_*}{R^2})dx]\\
&+\int_\Omega\{(1-\frac{S_*}{S})[\Lambda-(\alpha b I^p+\beta I)S^q-\delta S]+(1-\frac{L_*}{L})[(\alpha b I^p+\beta I)S^q-(\sigma+\delta)L]\\
&+\frac{\alpha b S_*^q I_*^{p-1}}{\delta+\mu+r}[1-(\frac{I_*}{I})^p][\sigma L-(\delta+\mu+r)I]+\frac{\beta S_*^q}{r}(1-\frac{R_*}{R})(rI-\delta R)\}dx\\
&=-[\int_\Omega q d_S|\nabla S|^2(\frac{S_*^q}{S^{q+1}})dx+\int_\Omega d_L|\nabla L|^2(\frac{L_*}{L^2})dx+\frac{\alpha b S_*^q I_*^{p-1}}{\delta+\mu+r}\int_\Omega p d_I|\nabla I|^2(\frac{I_*^p}{I^{p+1}})dx\\
&+\frac{\beta S_*^q}{r}\int_\Omega d_R|\nabla R|^2(\frac{R_*}{R^2})dx]\\
&+\int_\Omega\{\delta S_*(1-\frac{S}{S_*})[1-(\frac{S_*}{S})^q]+\alpha b S_*^q I_*^p[3-\frac{S_*^q}{S^q}-\frac{LI_*^p}{L_*I^p}-\frac{S^qI^pL_*}{S_*^qI_*^pL}+(\frac{I^p}{I_*^p}-1)(1-\frac{I^{1-p}}{I_*^{1-p}})]\\
&+\beta S_*^q I_*(3-\frac{S_*^q}{S^q}-\frac{RI_*}{R_*I}-\frac{S^qIR_*}{S_*^qI_*R})\}dx.
\end{aligned}
$$

From the relationship between geometric mean and arithmetic mean, we can get $\delta S_*(1-\frac{S}{S_*})[1-(\frac{S_*}{S})^q]\leqslant 0$, $\alpha b S_*^q I_*^p[3-\frac{S_*^q}{S^q}-\frac{LI_*^p}{L_*I^p}-\frac{S^qI^pL_*}{S_*^qI_*^pL}+(\frac{I^p}{I_*^p}-1)(1-\frac{I^{1-p}}{I_*^{1-p}})]\leqslant 0$, $\beta S_*^q I_*(3-\frac{S_*^q}{S^q}-\frac{RI_*}{R_*I}-\frac{S^qIR_*}{S_*^qI_*R})\leqslant 0$, and $\delta S_*(1-\frac{S}{S_*})[1-(\frac{S_*}{S})^q]=0$, $\alpha b S_*^q I_*^p[3-\frac{S_*^q}{S^q}-\frac{LI_*^p}{L_*I^p}-\frac{S^qI^pL_*}{S_*^qI_*^pL}+(\frac{I^p}{I_*^p}-1)(1-\frac{I^{1-p}}{I_*^{1-p}})]=0$, $\beta S_*^q I_*(3-\frac{S_*^q}{S^q}-\frac{RI_*}{R_*I}-\frac{S^qIR_*}{S_*^qI_*R})=0$, if and only if $(S,L,I,R)=(S_*,L_*,I_*,R_*)$, i.e., $\frac{dV_6(t)}{dt}\leqslant 0$ and $\frac{dV_6(t)}{dt}=0$ if and only if $(S,L,I,R)=$

(S_*, L_*, I_*, R_*). According to the limit theory and Theorem 4.2, we can get that EE is globally asymptotically stable when $p \neq 1,\ q \neq 1$.

As a result, $V_i(t)$ $(i = 3,4,5,6)$ are Lyapunov functionals for (2.1), namely, $V_i(t) \leqslant 0$ for $i = 3,4,5,6$, $\forall t > 0$ along all trajectories except at (S_*, L_*, I_*, R_*) where $V_i(t) = 0$ for $i = 3,4,5,6$, $\forall t > 0$. By a similar argument as in the proof of Theorem 5.1, we can get

$$(S(x,t), L(x,t), I(x,t), R(x,t)) \to (S_*, L_*, I_*, R_*) \quad \text{in} \quad [L^\infty(\Omega)]^4, \ \text{as} \ t \to +\infty.$$

Thus, the EE (S_*, L_*, I_*, R_*) is globally attractive. □

Remark 5.3.

(i) For the case $p = 1$, we can follow the theory in [44] to derive an explicit expression for the basic reproduction number $R_0 = \left(\frac{\Lambda}{\delta}\right)^q \frac{(\beta+\alpha b)\sigma}{(\sigma+\delta)(\delta+\mu+r)}$. Therefore, from Theorems 5.1 and 5.2, the threshold dynamics in terms of the basic reproduction number is established: the DFE is globally asymptotically stable if $R_0 \leqslant 1$, and the EE is globally asymptotically stable if $R_0 > 1$.

(ii) For the case $p \neq 1$, from the discussion in Section 5, it is clear that the system (2.1) admits a unique EE. It follows from Theorem 5.2 that the unique EE is globally attractive, which indicates that the infectious disease always exists in this case.

(iii) For the case $0 < p < 1$, due to the difficulty in judging the global stability of DFE, it is hoped that this difficulty can be overcome in the future to make the article more complete.

6. Numerical simulation

Here we show numerical simulations regarding our model to illustrate and support the theoretical results of the previous sections. From Section 4, we can see that R_0 is the threshold parameter of disease persistence in the relevant population. Next, we will give some simulations to show that DFE is globally asymptotically stable when $R_0 \leqslant 1$, and EE is globally asymptotically stable when $R_0 > 1$.

In order to show that the DFE is globally asymptotically stable if the basic reproduction number is less than 1, we provide an example and set a set of parameters as follows.

$$\Lambda = 1,\ \delta = 0.5,\ \sigma = 0.4,\ \mu = 0.5,\ \beta = 0.5,\ b = 0.4,\ \alpha = 0.5,\ q = 1,\ p = 1,\ r = 0.5. \tag{6.1}$$

Setting the initial value of this model as $S(0,x) = L(0,x) = I(0,x) = R(0,x) = \sin \pi x$, and $d_S = d_L = d_I = d_R = 1$, then we obtain $R_0 = 0.860 < 1$. It implies that $(S_0, L_0, I_0, R_0) = (2,0,0,0)$ is globally asymptotically stable by Theorem 5.1, where VL will extinct and Figure 2 confirms this. In order to further observe the influence of q and p on the stability of DFE, on the premise of keeping other parameters unchanged, we change $q = 1$ to $q = 5$, as shown in Figure 3. On the other hand, we only change $p = 1$ i0 (6.1) to $p = 0.8$, and other parameters remain unchanged, as shown in Figure 4. In order to show that the EE is globally asymptotically stable if the basic reproduction number is greater than 1, we provide an example and set a set of parameters as follows.

$$\Lambda = 1,\ \delta = 0.5,\ \sigma = 0.4,\ \mu = 0.5,\ \beta = 0.9,\ b = 0.4,\ \alpha = 0.5,\ q = 1,\ p = 1,\ r = 0.5. \tag{6.2}$$

Setting the initial value of this model as $S(0,x) = L(0,x) = I(0,x) = R(0,x) = \sin \pi x$, and $d_S = d_L = d_I = d_R = 1$, then we obtain $R_0 = 1.019 > 1$. It implies that $(S_*, L_*, I_*, R_*) = (1.250, 0.4167, 0.1111, 0.1111)$ is globally asymptotically stable by Theorem 5.2. This indicates that VL will continue to spread in the world and Figure 5 confirms this.

In order to further observe the influence of q and p on the stability of EE, on the premise of keeping other parameters unchanged, we change $q = 1$ to $q = 5$, as shown in Figure 6. On the other hand, we only change $p = 1$ in (6.2) to $p = 0.8$, and other parameters remain unchanged, as shown in Figure 7.

To explore the impact of α and b on the stability of DFE, let's keep the parameter data in Figure 2 unchanged, change $\alpha = 0.5$ to $\alpha = 0.8$, and change $b = 0.4$ to $b = 0.8$, as shown in Figure 8.

By comparing Figure 2 with Figure 3, we can see that under $S(0,x) = L(0,x) = I(0,x) = R(0,x) = \sin \pi x$, $\Lambda = 1, \delta = 0.5, \sigma = 0.4, \mu = 0.5, \beta = 0.5, b = 0.4, \alpha = 0.5, q = 1, p = 1, r = 0.5$, and $d_S = d_L = d_I = d_R = 1$, we change $q = 1$ to $q = 5$, the values of q will not affect the stability of the disease-free equilibrium (DFE) of the system (2.1).

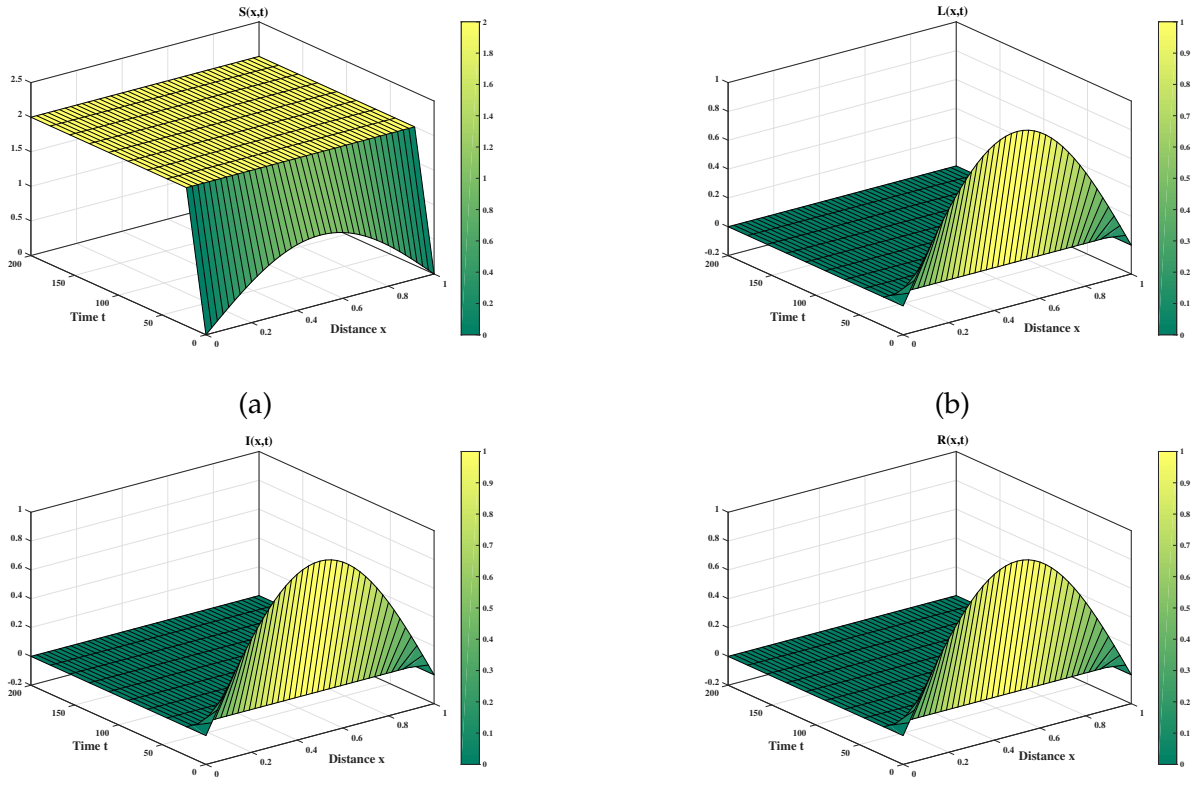
By comparing Figure 2 with Figure 4, we can see that under $S(0,x) = L(0,x) = I(0,x) = R(0,x) = \sin \pi x$, $\Lambda = 1, \delta = 0.5, \sigma = 0.4, \mu = 0.5, \beta = 0.5, b = 0.4, \alpha = 0.5, q = 1, p = 1, r = 0.5$, and $d_S = d_L = d_I = d_R = 1$, we change $p = 1$ to $p = 0.8$, the values of p will not affect the stability of the disease-free equilibrium (DFE) of the system (2.1).

By comparing Figures 5 and Figure 6 with Figure 7, we can see that under $S(0,x) = L(0,x) = I(0,x) = R(0,x) = \sin \pi x$, $\Lambda = 1$, $\delta = 0.5$, $\sigma = 0.4$, $\mu = 0.5$, $\beta = 0.9$, $b = 0.4$, $\alpha = 0.5$, $q = 1$, $p = 1$, $r = 0.5$, and $d_S = d_L = d_I = d_R = 1$, changing the values of q and p will slightly affect the position of the endemic equilibrium (EE) of the system (2.1), but it will not affect the stability of EE.

By comparing Figure 2 with Figure 5, we can see that when other parameters are the same, VL will extinct if $\beta = 0.5$, and VL will exist in the world if $\beta = 0.9$. From this we can get the value of β may have some influence on the propagation of VL.

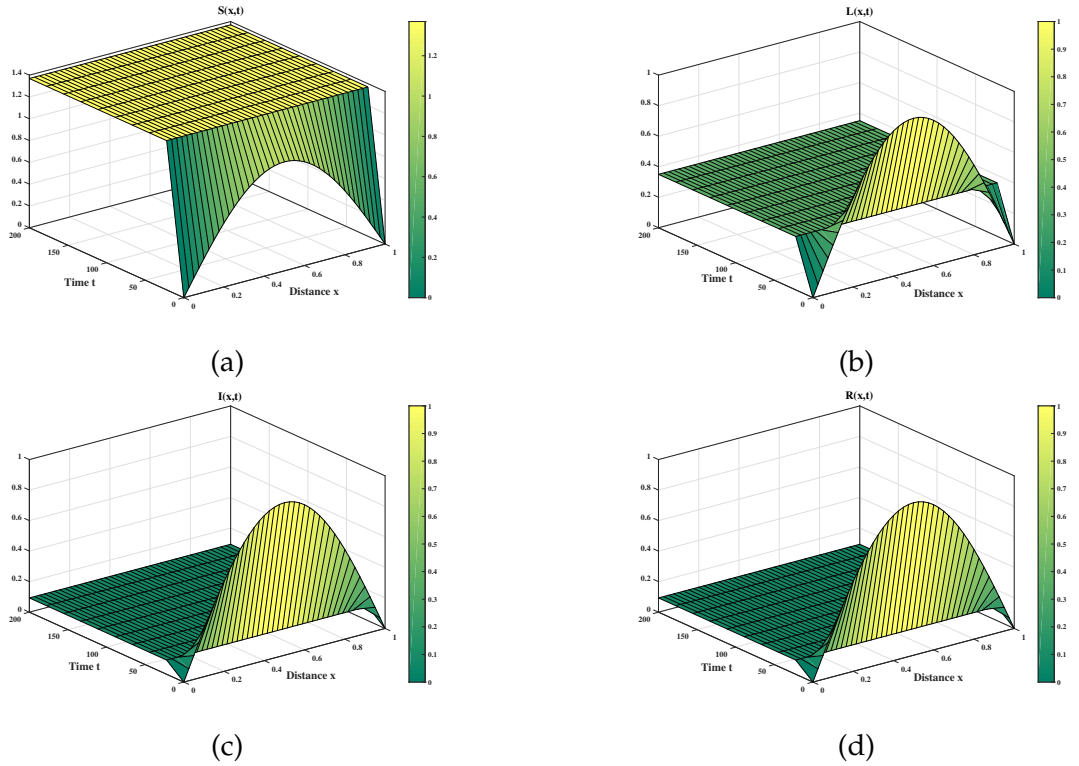
By comparing Figure 2 with Figure 8, we can see that when other parameters are the same, if $\alpha = 0.5$ and $b = 0.4$, VL will extinct, if $\alpha = 0.8$ and $b = 0.8$, VL will exist in the world. From this we can get the value of α and b may have some influence on the propagation of VL.

To sum up the analysis of the above examples, direct or indirect contact between susceptible individuals and infected individuals can also affect the spread of the disease, so we can further reduce the risk of infection through appropriate isolation measures.



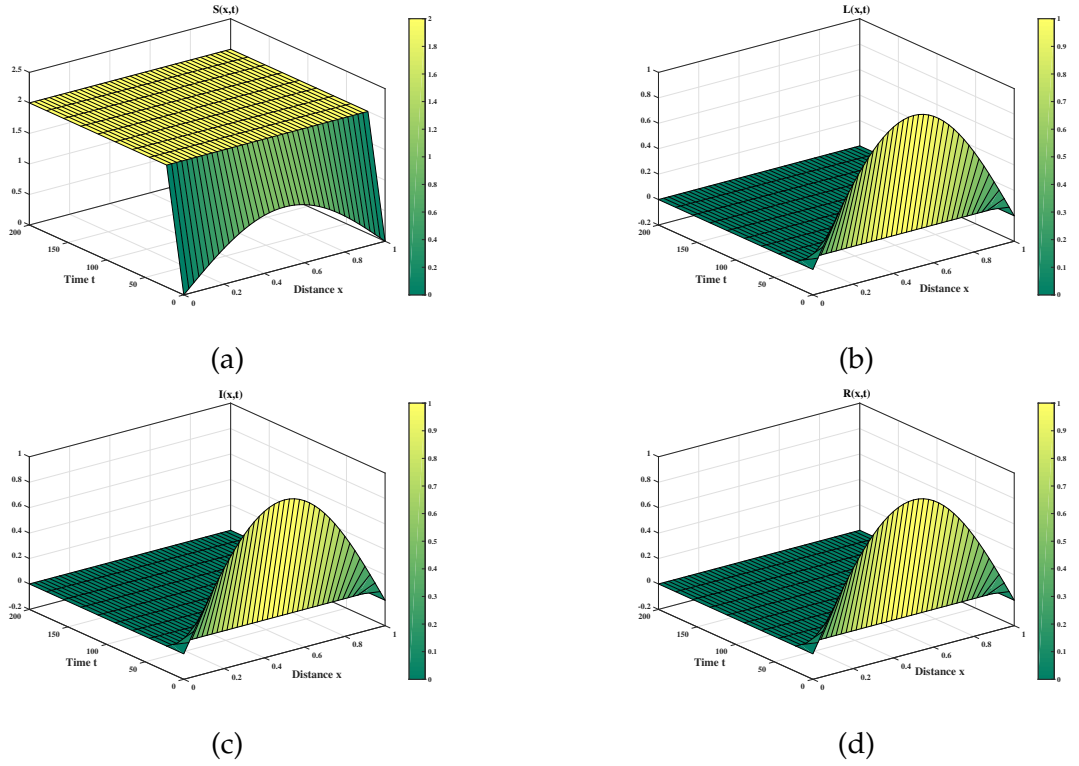
(a) (b) (c) (d)

Figure 2: Dynamics behavior of system (2.1) with $S(0,x) = L(0,x) = I(0,x) = R(0,x) = \sin \pi x$; $d_S = d_I = d_C = d_R = 1$; $\Lambda = 1, \delta = 0.5, \sigma = 0.4, \mu = 0.5, \beta = 0.5, b = 0.4, \alpha = 0.5, q = 1, p = 1, r = 0.5$.



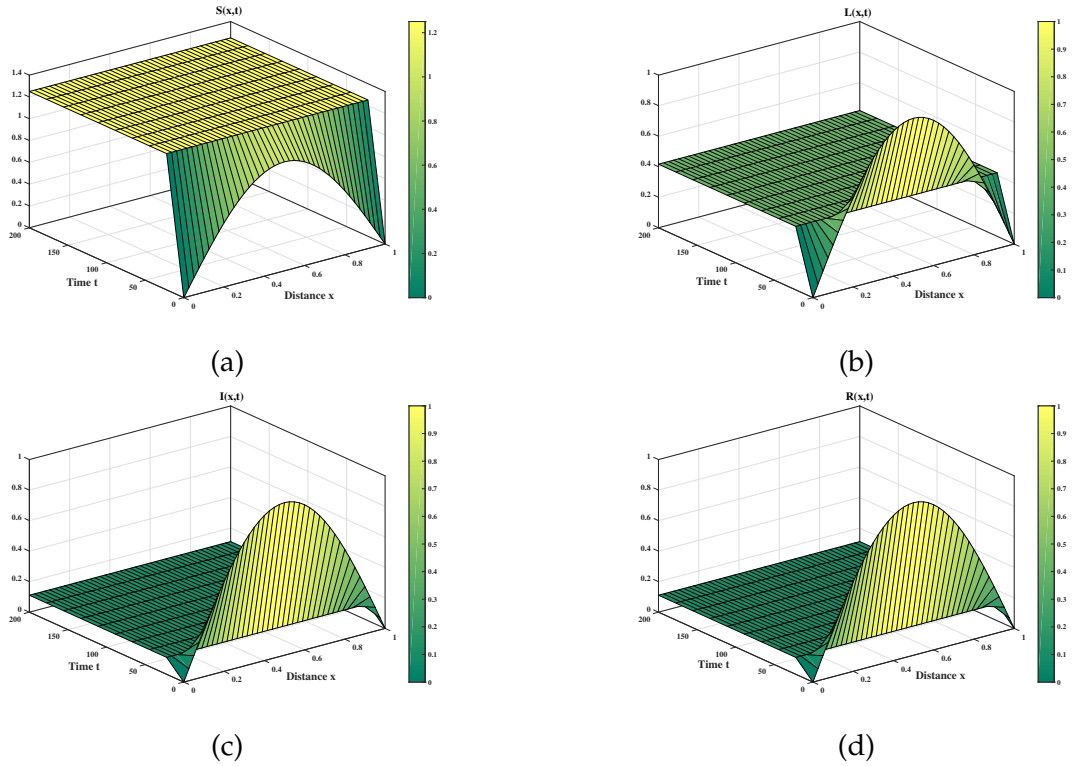
(a) (b) (c) (d)

Figure 3: Dynamics behavior of system (2.1) with $S(0,x) = L(0,x) = I(0,x) = R(0,x) = \sin \pi x$; $d_S = d_I = d_C = d_R = 1$; $\Lambda = 1, \delta = 0.5, \sigma = 0.4, \mu = 0.5, \beta = 0.5, b = 0.4, \alpha = 0.5, q = 5, p = 1, r = 0.5$.



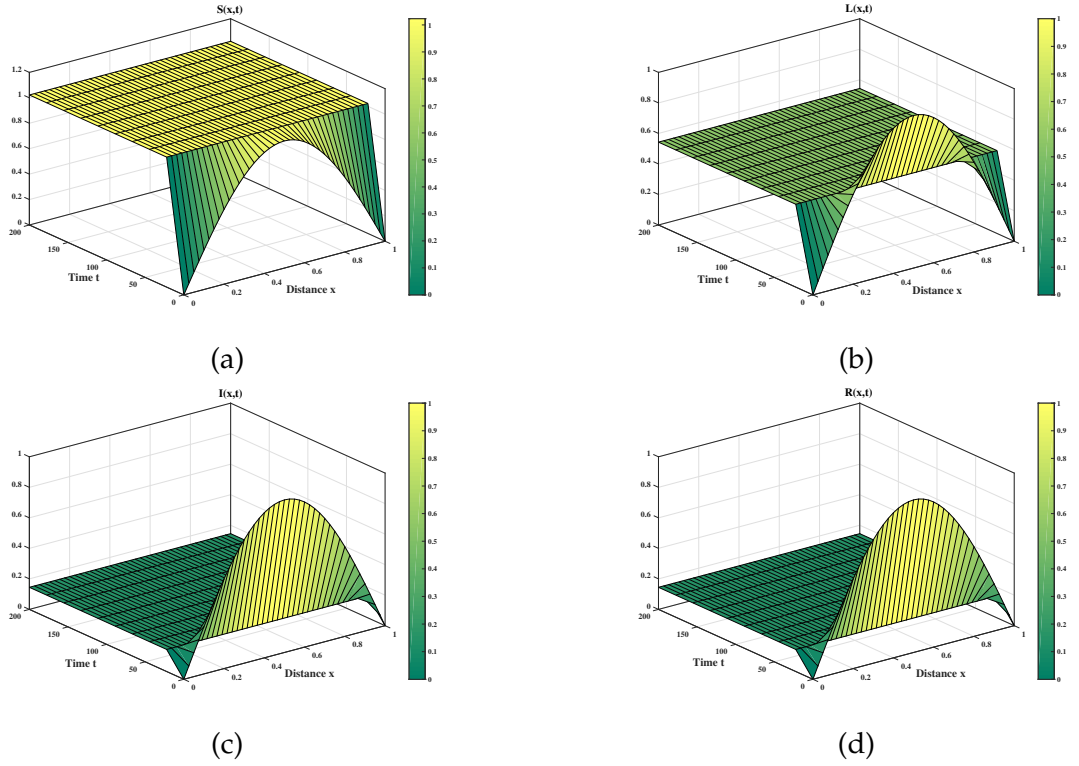
(a) (b) (c) (d)

Figure 4: Dynamics behavior of system (2.1) with $S(0,x) = L(0,x) = I(0,x) = R(0,x) = \sin \pi x$; $d_S = d_I = d_C = d_R = 1$; $\Lambda = 1, \delta = 0.5, \sigma = 0.4, \mu = 0.5, \beta = 0.5, b = 0.4, \alpha = 0.5, q = 1, p = 0.8, r = 0.5$.



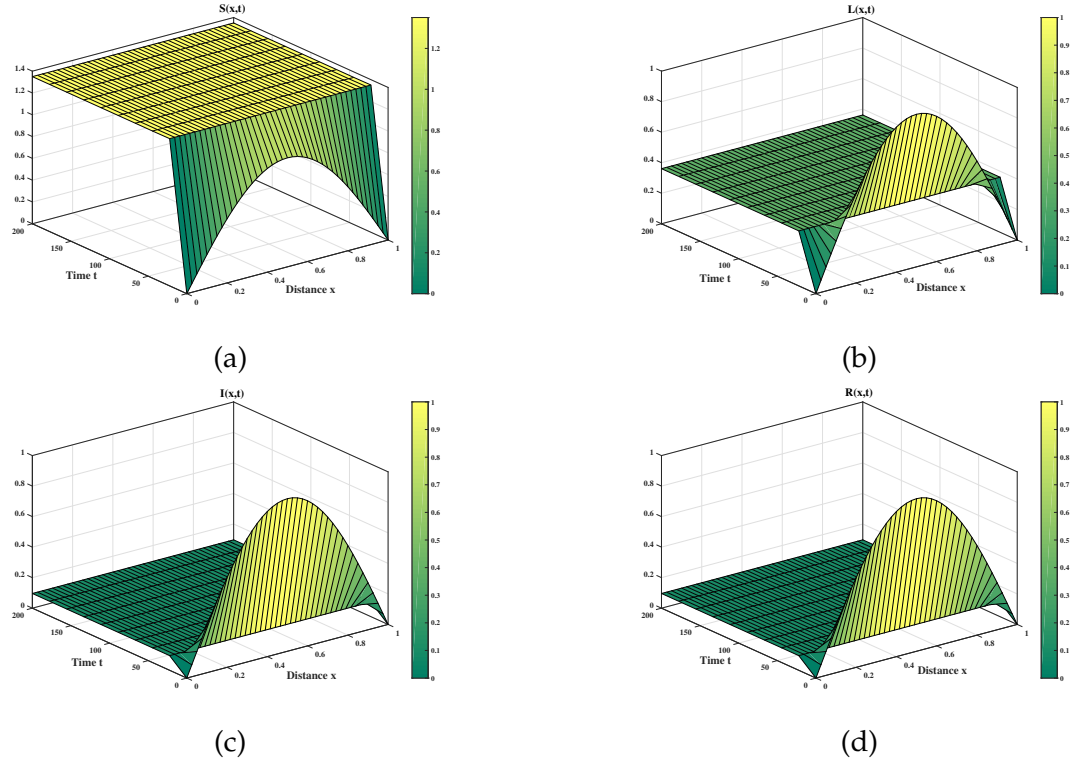
(a) (b) (c) (d)

Figure 5: Dynamics behavior of system (2.1) with $S(0,x) = L(0,x) = I(0,x) = R(0,x) = \sin \pi x$; $d_S = d_I = d_C = d_R = 1$; $\Lambda = 1$, $\delta = 0.5$, $\sigma = 0.4$, $\mu = 0.5$, $\beta = 0.9$, $b = 0.4$, $\alpha = 0.5$, $q = 1$, $p = 1$, $r = 0.5$.



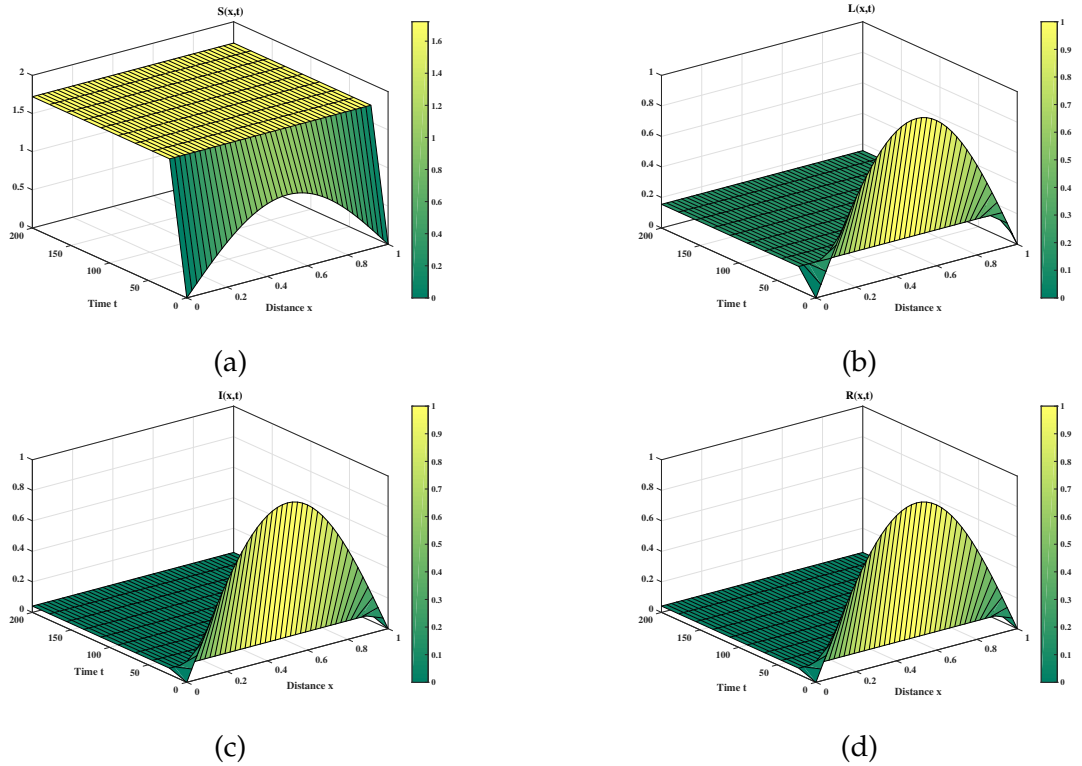
(a) (b) (c) (d)

Figure 6: Dynamics behavior of system (2.1) with $S(0,x) = L(0,x) = I(0,x) = R(0,x) = \sin \pi x$; $d_S = d_I = d_C = d_R = 1$; $\Lambda = 1$, $\delta = 0.5$, $\sigma = 0.4$, $\mu = 0.5$, $\beta = 0.9$, $b = 0.4$, $\alpha = 0.5$, $q = 5$, $p = 1$, $r = 0.5$.



(a) (b) (c) (d)

Figure 7: Dynamics behavior of system (2.1) with $S(0,x) = L(0,x) = I(0,x) = R(0,x) = \sin \pi x$; $d_S = d_I = d_C = d_R = 1$; $\Lambda = 1$, $\delta = 0.5$, $\sigma = 0.4$, $\mu = 0.5$, $\beta = 0.9$, $b = 0.4$, $\alpha = 0.5$, $q = 1$, $p = 0.8$, $r = 0.5$.



(a) (b) (c) (d)

Figure 8: Dynamics behavior of system (2.1) with $S(0,x) = L(0,x) = I(0,x) = R(0,x) = \sin \pi x$; $d_S = d_I = d_C = d_R = 1$; $\Lambda = 1$, $\delta = 0.5$, $\sigma = 0.4$, $\mu = 0.5$, $\beta = 0.5$, $b = 0.8$, $\alpha = 0.8$, $q = 1$, $p = 1$, $r = 0.5$.

7. Conclusions and discussions

In this paper, we study a class of leishmaniasis infection epidemic model with direct and indirect infection rate and spatial diffusion. Firstly, we prove the boundedness of the solution of the system, and analyze the sensitivity of the parameters. Secondly, sufficient conditions for the existence of the disease-free equilibrium and the endemic equilibrium are given, respectively. In addition, the local stability analysis of the disease-free equilibrium and the endemic equilibrium are obtained, respectively. Thirdly, by constructing various Lyapunov functions, we prove that when the reproduction number $R_0 \leqslant 1$, the disease-free equilibrium is globally asymptotically stable and the virus will be eliminated. When the reproduction number $R_0 > 1$, the endemic equilibrium is globally asymptotically stable, the infection will continue, and the number of infected people will eventually tend to a constant value. Finally, we give some numerical simulations to confirm the theoretical analysis. The results show that the virus could be eliminated by controlling the incidence rate and making the reproduction number $R_0 \leqslant 1$. Theorem 4.1, Theorem 4.2, Theorem 5.1 and Theorem 5.2 show that the diffusive rates of individuals have no impact on the basic reproduction number and the stability of the equilibria, but the diffusive rates of individuals makes the spatial density of infected individuals tend to be uniform over time. From the perspective of numerical simulation, we can also draw the conclusion that the direct or indirect contact between susceptible people and infected people may affect the spread of disease. We can draw inspiration from it, and we can also develop some appropriate isolation strategies to further reduce the risk of disease transmission.

On the other hand, most epidemic models rely on a hypothesis called the law of mass action or bilinear incidence rate SI. It suggests that the incidence rate is proportional to susceptibility to S and infection I. By extension, Severo [38] assumed that the number of new infections is represented by the term $S^q I^p$, which is commonly known as the nonlinear incidence rate. It can be observed that when $p = q = 1$, the incidence rate becomes the bilinear incidence rate SI. As is shown in [25], there is a much wider range of dynamical behaviors of the nonlinear incidence rate than the bilinear incidence rate. Different from the simple nonlinear incidence, we introduce direct and indirect infection rates to describe the spread of leishmaniasis, and it is found that direct or indirect contact may affect the prevalence of the disease. It is worth noting that, in order to capture the influence of spatial heterogeneity of environment and individual motion on the persistence and extinction of disease, Allen et al. [4] studied the asymptotic distribution of the steady-state solution of a class of SIS infectious disease reaction-diffusion model. Allen et al. [5] then proposed a spatial SIS (susceptible-infected-susceptible) reaction-diffusion model to study the existence, uniqueness, especially asymptotic behavior of susceptible individuals when the diffusive rate tends to zero in the case of producing so-called low-risk subhabitats. Therefore, it is necessary to analyze the impact of the spread of susceptible and infected populations on the asymptotic behavior of epidemic equilibrium in other heterogeneous environments, which will be considered in our future work.

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