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Research article

Role of media coverage in a SVEIR-I epidemic model with nonlinear incidence and spatial heterogeneous environment

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Abstract: In this paper, we propose a SVEIR-I epidemic model with media coverage in a spatially heterogeneous environment, and study the role of media coverage in the spread of diseases in a spatially heterogeneous environment. In a spatially heterogeneous environment, we first set up the well-posedness of the model. Then, we define the basic reproduction number R_0 of the model and establish the global dynamic threshold criteria: when $R_0 < 1$, disease-free steady state is globally asymptotically stable, while when $R_0 > 1$, the model is uniformly persistent. In addition, the existence and uniqueness of the equilibrium state of endemic diseases were obtained when $R_0 > 1$ in homogeneous space and heterogeneous diffusion environment. Further, by constructing appropriate Lyapunov functions, the global asymptotic stability of disease-free and positive steady states was established. Finally, through numerical simulations, it is shown that spatial heterogeneity can increase the risk of disease transmission, and can even change the threshold for disease transmission; media coverage can make people more widely understand disease information, and then reduce the effective contact rate to control the spread of disease.

Keywords: SVEIR-I epidemic model; nonlinear incidence; media coverage; global stability; spatial heterogeneous environment

1. Introduction

In epidemiological theory, environmental heterogeneity has been regarded as an indispensable factor in the transmission of infectious diseases. Due to altitude, temperature, humidity, latitude, climate, life factors and other factors, the spread of epidemics in different environments varies greatly. In the process of COVID-19 transmission, due to differences in population density, medical resources and climatic conditions in different regions, the outbreak degree of different regions is also different. For example, during the first outbreak, the epidemic spread faster and more widely in southern and large parts of eastern China, because these areas were densely populated, developed rapidly, and also easy to

move people, resulting in a concentrated performance at the peak of the outbreak. In addition, Smith et al. [1] observed temporary fluctuations in mosquito populations affected by environmental variables such as rainfall, humidity and temperature. These spatio-temporal heterogeneity have a significant impact on the mode of transmission of mosquito-borne diseases. Wang et al. [2] found that cholera transmission is influenced by spatial variations and seasonal fluctuations, so disease control measures should pay more attention to the spread process. Cai et al. [3] showed that combinations of spatial heterogeneity tended to enhance the persistence of influenza disease in the model, in other words, influenza infection risk would be very important if spatial heterogeneity was taken into account. Luo et al. [4] observed that dengue is a climate-sensitive disease, and that climatic factors, such as temperature and rainfall, promote dengue transmission through potential changes in vector mosquito density and human behavior, leading to increased exposure to dengue, especially in areas where temperatures favor dengue and other vector-borne diseases. Therefore, in order to study the effects of heterogeneous environments on disease transmission, more and more reaction-diffusion epidemic models have been developed [4–12].

As we all know, clinical results show that for certain diseases, including hepatitis B (HBV) [13], hepatitis C (HCV) [14], most human tuberculosis [15], herpes virus and other infectious diseases, since the recovered person still carries the pathogen, the recovered person may appear with the reactivation of the infection when the pathogen lurking in the tissue reproduces to a certain extent. For example, herpes simplex virus type 2 (HSV-2) is usually transmitted through close physical or sexual contact and can cause genital herpes [16]. The main incidence of genital herpes is due to its frequent recurrence rate. In one study [17], 89% of HSV-2 patients had at least one recurrence during follow-up, with a mean monthly recurrence rate of 34%, 38% of patients relapsed at least six times in the first year, and 20% relapsed more than 10 times. They concluded that almost all initially symptomatic patients with HSV-2 infection had a relapse of symptoms, more than 35% of such patients relapse frequently. Recurrence rates were particularly high in patients with prolonged onset of first infection, whether or not they received acyclovir antiviral chemotherapy. In fact, relapse has been extensively studied as an important feature of human or animal diseases, see [18–23]. Tudor [24] was the first to incorporate recurrence into mathematical models, building a bilinear compartment model of morbidity and constant population size (later called the SIRI model). The results show that the basic reproduction number is a threshold parameter for the stability of the system.

On the one hand, people's perception of disease risk is influenced by information widely disseminated by the media. In the early stages of an outbreak, due to lack of medical diagnosis and vaccination, effective media coverage can reduce infection peaks [25]. In order to characterize the impact of media coverage on disease transmission and control, more and more mathematical models have been proposed. For example, Cui et al. [26] proposed an infectious disease model with Logistic growth under media coverage, with $\beta(I) = \mu e^{-mI}$ as the effective contact rate and m as the influence parameter of media coverage on the effective contact rate. In [27, 28], the model adopted $\beta_1 - \beta_2 \frac{I}{m+I}$ as the effective contact rate to reflect the reduced amount of contact rate due to media coverage. In [29], the model uses $\beta_i(I) = a_i - b_i m(I)$ as the effective contact rate, the results suggest that changes in human behavior in response to media coverage can reduce outbreaks and reduce the speed of disease transmission. In [30], the author assumes that the media reports of infectious function of $f(I) := (\beta - \frac{\beta_1 I}{m+I})I$, and established an age-structured epidemic model for a class of incomplete vaccination. In [31], to model the impact of sanitation and awareness on infectious disease

transmission, the authors selected $\beta(M) = (\beta - \beta_1 \frac{k_1 M}{p+k_1 M})$ as the effective contact rate of susceptible individuals. They showed through model analysis that health and advocacy programs have the ability to lower epidemic thresholds and thus control the spread of infection.

However, these models based on ordinary differential equations ignore the important factor of spatial heterogeneity. Undoubtedly, it would be more practical to consider the impact of media coverage on disease transmission in heterogeneous environments, but there has been little research in this area. In a recent study, Song et al. [32] studied a class of diffuse epidemic systems with spatial heterogeneity and lagging effects of media influence, where the media influence function is $\beta(x)e^{-m(x)I(x,t-r)}$. Inspired by [27, 28], we suggest that, as the number of infected people increases, corresponding interventions reported by the media can provide profound psychological cues to susceptible individuals to help them reduce their contact with infected individuals. Therefore, the contact rate between susceptible and infected individuals should be assumed to be a monotonically decreasing function. Motivated by the above works, in this paper, we use the general smooth function $\beta(x) - \beta_1(x)f(I)$ as the effective contact rate under the influence of media coverage in a heterogeneous environment, where $f(I)$ is the media coverage function with saturated psychological effect. Consider that "asymptomatic" individuals can still spread the infection even if they do not show any symptoms (for example, a particular but critical feature of the recent COVID-19 epidemic is that a large proportion of the population infected with SARS-Cov-2 virus originates from asymptomatic individuals [33, 34].) The main reason is that asymptomatic cases are often unrecognized during the incubation period and therefore have more contacts than symptomatic cases. Therefore, this paper incorporates the transmission of exposed persons to susceptible populations into the mathematical epidemic model. Additionally, since the latent has no obvious symptoms during the incubation period, the effective contact rate is not affected by media reports.

On the other hand, we also know that vaccination is an important means of preventing infection and relapse of these diseases [35–37]. Hence, the effect of spatial factors on relapse and vaccination should be taken into account. Inspired by the above literature, in this paper, we study a SVEIR-I epidemic response-diffusion model that incorporates the impact of media coverage on disease transmission and considers the spread of asymptomatic infected individuals.

$$\begin{cases} \dfrac{\partial S(t,x)}{\partial t} = \nabla \cdot (D_1(x)\nabla S) + \Lambda(x) - [\beta(x) - \beta_1(x)f(I)]SI - \beta_2(x)SE - p(x)S - \mu(x)S, \\ \dfrac{\partial V(t,x)}{\partial t} = \nabla \cdot (D_2(x)\nabla V) + p(x)S - \sigma(x)[\beta(x) - \beta_1(x)f(I)]VI - \sigma(x)\beta_2(x)VE - \mu(x)V, \\ \dfrac{\partial E(t,x)}{\partial t} = \nabla \cdot (D_3(x)\nabla E) + \{[\beta(x) - \beta_1(x)f(I)]I + \beta_2(x)E\}(S + \sigma(x)V) - \alpha(x)E - \mu(x)E, \\ \dfrac{\partial I(t,x)}{\partial t} = \nabla \cdot (D_4(x)\nabla I) + \alpha(x)E + \rho(x)R - \mu(x)I - \eta(x)I - \delta(x)I, \\ \dfrac{\partial R(t,x)}{\partial t} = \nabla \cdot (D_5(x)\nabla R) + \delta(x)I - \mu(x)R - \rho(x)R, \\ (S(0,x), V(0,x), E(0,x), I(0,x), R(0,x)) = (\phi_1(x), \phi_2(x), \phi_3(x), \phi_4(x), \phi_5(x)), \ x \in \Omega, \end{cases} \tag{1.1}$$

with the homogeneous Neumann boundary condition(**NBC**)

$$\frac{\partial S(t,x)}{\partial n} = \frac{\partial V(t,x)}{\partial n} = \frac{\partial E(t,x)}{\partial n} = \frac{\partial I(t,x)}{\partial n} = \frac{\partial R(t,x)}{\partial n} = 0, \ x \in \partial\Omega, \ t \geq 0,$$

where $\Omega \subset R^n$ is a bounded domain with the smooth boundary $\partial\Omega$. $\frac{\partial}{\partial n}$ denotes the outward normal derivative on $\partial\Omega$. $S(t,x), V(t,x), E(t,x), I(t,x)$ and $R(t,x)$ represent the population density of susceptible individuals, vaccinated individuals, exposed individuals, infected individuals and recovered individuals at location x and time t, respectively. $D_i(x)(i = 1,2,3,4,5)$ respectively represent the diffusion rate of corresponding individuals at position x. We assume that $\Lambda(x)$, $D_i(x)$, $\mu(x)$, $\beta(x)$, $\beta_i(x)(i = 1,2)$, $p(x)$, $\sigma(x)$, $\delta(x)$, $\alpha(x)$, $\rho(x)$ and $\eta(x)$ are positive, continuous and bounded on $\bar{\Omega}$. The meanings of other parameters in the Table 1.

Table 1. The meaning of parameters in model (1.1).

Symbol	Meaning
$\mu(x)$	Natural mortality rate at location x.
$p(x)$	Vaccination rate at position x.
$\rho(x)$	Relapse rate at position x.
$\delta(x)$	Per-capita recovery (treatment) rate at position x.
$\Lambda(x)$	The recruitment rate of S at position x.
$\eta(x)$	Disease-related death rate at position x.
$\alpha(x)$	The conversion rate from exposed to infected individuals.
$\beta(x)$	The infection rate of S affected by I at position x.
$\beta_2(x)$	The infection rate of S affected by E at position x.
$\beta_1(x)$	The maximum reduced contact rate of S at position x affected by media coverage.
$\sigma(x)$	The fraction of V being infected and entering E at position x.

This paper is structured as follows. In Section 2, we first introduce some spaces, give some symbols and make some assumptions. In Section 3, we present and prove some basic results on the existence and ultimate boundedness of global solutions. In Section 4, we define the basic reproduction number R_0 for model (1.1), and give the relationship between the principal eigenvalue and the basic reproduction number R_0. In Section 5, the dynamics with R_0 as the threshold are established, that is, when $R_0 < 1$, disease-free steady state is globally asymptotically stable, and when $R_0 > 1$, the system is uniformly persistent. In Section 6, we consider the global dynamics of a spatially homogeneous model. In Section 7, some numerical examples are given to illustrate our results and show the impact of media coverage and spatial heterogeneity on disease transmission. A brief conclusion is given in Section 8.

2. Preliminaries

Throughout this paper, we first introduce the following assumptions.

(A_1) $f(I)$ is continuously differentiable on $[0,+\infty)$, and it satisfies $f'(I) > 0$, $f(0) = 0$ and $\lim_{I\to\infty} f(I) = 1$.

(A_2) The limitations of media coverage indicate $\beta(x) > \beta_1(x)$ for all $t > 0$ and $x \in \bar{\Omega}$.

(A_3) The incidence of disease is a strictly monotonically increasing function of I, so $\frac{\partial\{[\beta-\beta_1 f(I)]SI\}}{\partial I} > 0$.

Let $X := C(\bar{\Omega}, R^5)$, $Y := C(\bar{\Omega}, R^3)$ and $Z := C(\bar{\Omega}, R)$ are Banach spaces with the supremum norm $\|\cdot\|$ of continuous functions. Furthermore, $X_+ := C(\bar{\Omega}, R^5_+)$, $Y_+ := C(\bar{\Omega}, R^3_+)$ and $Z_+ := C(\bar{\Omega}, R_+)$ are the positive cones corresponding to X, Y and Z, respectively. Then, (X, X_+), (Y, Y_+) and (Z, Z_+) are ordered

Banach spaces. For any function $\aleph(x)$ defined on some nonempty set U, we denote

$$\aleph^* := \max_{x\in U} \aleph(x), \quad \aleph_* := \min_{x\in U} \aleph(x).$$

Let $A_i(t)(i = 1, 2, 3, 4, 5) : C(\bar{\Omega}, R) \to C(\bar{\Omega}, R)$ are the C_0 semigroup associated with $\nabla\cdot(D_i(x)\nabla)-\pi_i(x)$ subjects to **NBC**, where $\pi_1 = \mu(x) + p(x)$, $\pi_2 = \mu(x)$, $\pi_3 = \mu(x) + \alpha(x)$, $\pi_4 = \mu(x) + \eta(x) + \delta(x)$ and $\pi_5 = \mu(x) + \rho(x)$. Then, we have

$$(A_i(t)\phi)(x) = \int_\Omega G_i(t, x, y)\phi(y)dy, \quad t > 0, \quad \phi \in C(\bar{\Omega}, R), \tag{2.1}$$

where $G_i(t, x, y)$ is the Green function with $\nabla \cdot (D_i(x)\nabla) - \pi_i(x)(i = 1, 2, 3, 4, 5)$ subjects to **NBC**. Furthermore, based on ([38], Corollary 7.2.3), we know that $A_i(t)$ is compact and strongly positive for all $t > 0$. Thus, there exist constants $M_i > 0$ such that $\|A_i(t)\| \le M_i e^{\alpha_i t}$ for all $t \ge 0$, where $\alpha_i < 0$ are the principal eigenvalue of $\nabla \cdot (D_i(x)\nabla) - \pi_i(x)$ under **NBC**. For any initial value function $\phi = (\phi_1, \phi_2, \phi_3, \phi_4, \phi_5) \in X_+$, we denote $u(t, \cdot, \phi) = (S(t, \cdot, \phi), V(t, \cdot, \phi), E(t, \cdot, \phi), I(t, \cdot, \phi), R(t, \cdot, \phi))^T$ be the solution of model (1.1). Let

$$\begin{cases} F_1(\phi)(x) =& \Lambda(x) - [\beta(x) - \beta_1(x)f(\phi_4(x))]\phi_1(x)\phi_4(x) - \beta_2(x)\phi_1(x)\phi_3(x), \\ F_2(\phi)(x) =& p\phi_1(x) - \sigma(x)[\beta(x) - \beta_1(x)f(\phi_4(x))]\phi_2(x)\phi_4(x) - \sigma(x)\beta_2(x)\phi_2(x)\phi_3(x), \\ F_3(\phi)(x) =& [\beta(x) - \beta_1(x)f(\phi_4(x))]\phi_1(x)\phi_4(x) + \beta_2(x)\phi_1(x)\phi_3(x) \\ & + \sigma(x)[\beta(x) - \beta_1(x)f(\phi_4(x))]\phi_2(x)\phi_4(x) + \sigma(x)\beta_2(x)\phi_2(x)\phi_3(x), \\ F_4(\phi)(x) =& \alpha(x)\phi_3(x) + \rho(x)\phi_5(x), \ F_5(\phi)(x) = \delta(x)\phi_4(x), \end{cases}$$

then model (1.1) can be written in the following form:

$$\begin{cases} u(t, \cdot, \phi) = \mathcal{A}(t)\phi + \int_0^t \mathcal{A}(t - s)\mathcal{F}(u(s, \cdot, \phi))ds, \\ u(0) = \phi, \end{cases} \tag{2.2}$$

where $\mathcal{A}(t) = diag(A_1(t), A_2(t), A_3(t), A_4(t), A_5(t))$ and $\mathcal{F}(\phi)(x) = diag(F_1(\phi)(x), F_2(\phi)(x), F_3(\phi)(x), F_4(\phi)(x), F_5(\phi)(x))^T$.

3. The well-posedness of the model

In this section, we give the existence of global solutions and the ultimate boundedness of system (1.1).

Lemma 1. *For any $\phi = (\phi_1, \phi_2, \phi_3, \phi_4, \phi_5) \in X_+$, model (1.1) has a unique non-negative solution $u(t, \cdot, \phi) = (S(t, \cdot, \phi), V(t, \cdot, \phi), E(t, \cdot, \phi), I(t, \cdot, \phi), R(t, \cdot, \phi)) \in X_+$ on $[0, \tau_\infty)$ and $\tau_\infty \le \infty$. Moreover, this solution is a classical solution.*

Proof. Similar to the proof of in ([10], Lemma 1), it is sufficient to show the following subtangential conditions holds

$$\lim_{h\to 0^+} \text{dist}(\phi + h\mathcal{F}(\phi), X_+) = 0, \quad \phi \in X_+.$$

We can prove that for any $\phi \in X_+$ and a sufficiently small $h \geq 0$,

$$\begin{aligned}
&\phi(x) + h\mathcal{F}(\phi)(x)\\
&= \begin{pmatrix}
\phi_1(x) + h\{\Lambda(x) - [\beta(x) - \beta_1(x)f(\phi_4(x))]\phi_1(x)\phi_4(x) - \beta_2(x)\phi_1(x)\phi_3(x)\}\\
\phi_2(x) + h\{p(x)\phi_1(x) - \sigma(x)[\beta(x) - \beta_1(x)f(\phi_4(x))]\phi_2(x)\phi_4(x) - \sigma(x)\beta_2(x)\phi_2(x)\phi_3(x)\}\\
\phi_3(x) + h\{[(\beta(x) - \beta_1(x)f(\phi_4(x)))\phi_4(x) + \beta_2(x)\phi_3(x)][\phi_1(x) + \sigma(x)\phi_2(x)]\}\\
\phi_4(x) + h[\alpha(x)\psi_3(x) + \rho(x)\phi_5(x)]\\
\phi_5(x) + h\delta(x)\phi_4(x)
\end{pmatrix}\\
&\geq \begin{pmatrix}
\phi_1(x) - h[\beta(x) - \beta_1(x)f(\phi_4(x))]\phi_1(x)\phi_4(x)\\
\phi_2(x) - h\sigma(x)[\beta(x) - \beta_1(x)f(\phi_4(x))]\phi_2(x)\phi_4(x)\\
\phi_3(x)\\
\phi_4(x)\\
\phi_5(x)
\end{pmatrix}\\
&> 0,
\end{aligned}$$

which indicates that $\phi + h\mathcal{F}(\phi) \in X_+$. That completes the proof. □

To continue our research, first, consider the following system,

$$\begin{cases}
\dfrac{\partial \omega(t,x)}{\partial t} = \nabla \cdot (d(x)\nabla\omega(t,x)) + \alpha(x) - \beta(x)\omega(t,x),\ x \in \Omega,\ t \geq 0,\\
\dfrac{\partial \omega(t,x)}{\partial n} = 0,\ x \in \partial\Omega,\ t \geq 0,
\end{cases} \tag{3.1}$$

where $d(x)$, $\alpha(x)$ and $\beta(x)$ are positive continuous functions defined on $\bar{\Omega}$, and we have the following lemma.

Lemma 2. *[39] System* (3.1) *admits a unique positive steady state $\omega^0(x)$, which satisfies the equation*

$$\begin{cases}
\nabla \cdot (d(x)\nabla\omega^0(x)) + \alpha(x) - \beta(x)\omega^0(x) = 0,\ x \in \Omega,\\
\dfrac{\partial \omega^0(x)}{\partial n} = 0,\quad x \in \partial\Omega,
\end{cases}$$

and it is globally asymptotically stable in $C(\bar{\Omega}, R_+)$. In addition, $\omega^0(x) = \frac{a}{b}$ when $\alpha(\cdot) \equiv a$ and $\beta(\cdot) \equiv b$ are positive constants.

Theorem 1. *For any $\phi = (\phi_1, \phi_2, \phi_3, \phi_4, \phi_5) \in X_+$, model* (1.1) *has a unique classical solution $u(t, \cdot, \phi) = (S(t, \cdot, \phi), V(t, \cdot, \phi), E(t, \cdot, \phi), I(t, \cdot, \phi), R(t, \cdot, \phi)) \in X_+$ defined on $[0, \infty)$, and this solution is also ultimately bounded.*

Proof. Using a standard argument (see, e.g., ([40], Theorem 3.2)), it is only necessary to get the boundness of solution in $\bar{\Omega} \times [0, \tau_\infty)$. From the first equation of (1.1), we have

$$\frac{\partial S}{\partial t} \leq \nabla \cdot (D_1(x)\nabla S) + \Lambda^* - p_* S - \mu_* S,\ \ t \in [0, \tau_\infty),\ x \in \bar{\Omega}. \tag{3.2}$$

From the comparison principle [41] and Lemma 2, we can see that there exists a constant $M_1 > 0$ such that $S(t, x) \leq M_1$ for all $t \in [0, \tau_\infty)$ and $x \in \bar{\Omega}$. From the second equation of (1.1), we have

$$\frac{\partial V}{\partial t} \leq \nabla \cdot (D_2(x)\nabla V) + p^* M_1 - \mu_* V,\ \ t \in [0, \tau_\infty),\ x \in \bar{\Omega}. \tag{3.3}$$

Through similar analysis, there exists a constant $M_2 > 0$ such that $V(t,x) \le M_2$ for all $t \in [0,\tau_\infty)$ and $x \in \bar{\Omega}$.

Then we can get

$$\begin{cases} \dfrac{\partial E(t,x)}{\partial t} \le \nabla\cdot(D_3(x)\nabla E) + \beta^* M_1 I + \beta_2^* M_1 E + \sigma^*\beta^* M_2 I + \sigma^*\beta_2^* M_2 E \\ \qquad\qquad - (\alpha_* + \mu_*)E, \\ \dfrac{\partial I(t,x)}{\partial t} \le \nabla\cdot(D_4(x)\nabla I) + \alpha^* E + \rho^* R - \mu_* I - \eta_* I - \delta_* I, \qquad t \in [0,\tau_\infty), x \in \Omega, \\ \dfrac{\partial R(t,x)}{\partial t} \le \nabla\cdot(D_5(x)\nabla R) + \delta^* I - \mu_* R - \rho_* R, \\ \dfrac{\partial E}{\partial n} = \dfrac{\partial I}{\partial n} = \dfrac{\partial R}{\partial n} = 0, \quad x \in \partial\Omega,\ t > 0, \end{cases} \tag{3.4}$$

Consider the following comparison system:

$$\begin{cases} \dfrac{\partial v_1}{\partial t} = \nabla\cdot(D_3(x)\nabla v_1) + \beta^* M_1 v_2 + \beta_2^* M_1 v_1 + \sigma^*\beta^* M_2 v_2 + \sigma^*\beta_2^* M_2 v_1 \\ \qquad - (\alpha_* + \mu_*)v_1, \\ \dfrac{\partial v_2}{\partial t} = \nabla\cdot(D_4(x)\nabla v_2) + \alpha^* v_1 + \rho^* v_3 - \mu_* v_2 - \eta_* v_2 - \delta_* v_2, \qquad t > 0, x \in \Omega, \\ \dfrac{\partial v_3}{\partial t} = \nabla\cdot(D_5(x)\nabla v_3) + \delta^* v_2 - \mu_* v_3 - \rho_* v_3, \\ \dfrac{\partial v_1}{\partial n} = \dfrac{\partial v_2}{\partial n} = \dfrac{\partial v_3}{\partial n} = 0, \quad x \in \partial\Omega,\ t > 0. \end{cases} \tag{3.5}$$

According to the Krein-Rutman theorem [42], the eigenvalue problem associated with the system (3.5) has a strongly positive eigenfunction $\xi = (\xi_1, \xi_2, \xi_3)$ corresponding to the principal eigenvalue λ. Thus, system (3.5) admits a solution $ce^{\lambda t}\xi(x)$ for $t \ge 0$, where c is a positive constant and satisfies $c\xi = (v_1(x,0), v_2(x,0), v_3(x,0)) \ge (E(x,0), I(x,0), R(x,0))$ for all $x \in \bar{\Omega}$. Then, using the principle of comparison

$$E(t,x), I(t,x), R(t,x) \le ce^{\lambda t}\xi(x), \quad t \in [0,\tau_\infty),\ x \in \bar{\Omega}.$$

This means that there is a constant $M_3 > 0$ such that

$$E(t,x) \le M_3,\ \ I(t,x) \le M_3,\ \ R(t,x) \le M_3,\ \ t \in [0,\tau_\infty),\ x \in \bar{\Omega}.$$

Therefore, $\tau_\infty = \infty$. This shows the global existence of $u(t,\cdot,\phi)$.

We next show that the solution is ultimately bounded. According to the comparison principle, from inequality (3.2), (3.3) and Lemma 2, it follows that there exists constants of $N_1 > 0$, $N_2 > 0$ and times $t_1 > 0$, $t_2 > 0$ such that $S(t,x) \le N_1$ and $V(t,x) \le N_2$ for all $t \ge \max\{t_1, t_2\}$, $x \in \bar{\Omega}$, which means that $S(t,x)$ and $V(t,x)$ are ultimately bounded.

Let

$$P(t) = \int_\Omega (S(t,x) + V(t,x) + E(t,x) + I(t,x) + R(t,x))dx,$$

Using ([43], Theorem 3.7), we have

$$\begin{aligned}\frac{dP}{dt} =& \int_\Omega [\Lambda(x) - \mu(x)S(t,x) - \mu(x)V(t,x) - \mu(x)E(t,x) - \mu(x)I(t,x) - \eta(x)I(t,x) - \mu(x)R(t,x)]dx \\ \leq& \Lambda^*|\Omega| - \mu_* P, \quad t \in [0, \tau_\infty),\end{aligned} \tag{3.6}$$

where $|\Omega|$ is the volume of Ω. Hence, there exists a constant $N_3 > 0$ and $t_3 > 0$ such that $P(t) \leq N_3$ for all $t \geq t_3$. Consequently, we have

$$\int_\Omega E(t,x)dx \leq N_3, \ \int_\Omega I(t,x)dx \leq N_3, \ \int_\Omega R(t,x)dx \leq N_3. \tag{3.7}$$

Then, using the similar method in ([9], Theorem 1), for any $t \geq t_4 = \max\{t_1, t_2, t_3\}$, we have

$$\begin{aligned}E(t,x) =& A_3(t)E(t_4,x) + \int_{t_4}^t A_3(t-s)\Big\{[\beta(x) - \beta_1(x)f(I(s,x))]I(s,x) + \beta_2(x)E(s,x)\Big\}[S(s,x) \\ &+ \sigma(x)V(s,x)]ds \\ \leq& M_3 e^{\alpha_3(t-t_4)}\|E(t_4,x)\| + \omega_3 \int_{t_4}^t e^{-(\alpha_*+\mu_*)(t-s)}[\beta^* N_1 \int_\Omega I(s,y) + \beta_2^* N_1 \int_\Omega E(s,y) \\ &+ \sigma^*\beta^* N_2 \int_\Omega I(s,y) + \sigma^*\beta_2^* N_2 \int_\Omega E(s,y)]dyds \\ \leq& M_3 e^{\alpha_3(t-t_4)}\|E(t_4,x)\| + \frac{\omega_3 N_1 N_2 N_3(\beta^* + \beta_2^* + \sigma^*\beta^* + \sigma^*\beta_2^*)}{\alpha_* + \mu_*}.\end{aligned}$$

So, we have

$$\limsup_{t\to\infty} \|E(t,x)\|_Z \leq \frac{\omega_3 N_1 N_2 N_3(\beta^* + \beta_2^* + \sigma^*\beta^* + \sigma^*\beta_2^*)}{\alpha_* + \mu_*}.$$

This shows that $E(t,x)$ is ultimately bounded. Similarly, for $I(t,x)$ and $R(t,x)$ we get

$$\limsup_{t\to\infty} \|I(t,x)\|_Z \leq \frac{\omega_4 N_3(\alpha^* + \rho^*)}{\mu_* + \eta_* + \delta_*}, \quad \limsup_{t\to\infty} \|R(t,x)\|_Z \leq \frac{\omega_5 N_3 \delta^*}{\mu_* + \rho_*},$$

where $\omega_4 > 0$ and $\omega_5 > 0$ are constants. This indicates that the solution is ultimately bounded. □

Corollary 1. *For any $\phi \in X_+$, the solution semiflow $Q(t)\phi = u(t,\cdot,\phi) : X_+ \to X_+$ generated by model* (1.1) *has a compact global attractor.*

Proof. By Theorem 1, we know that the system (1.1) is ultimately bounded, which means that the solution semiflow $Q(t)\phi = u(t,\cdot,\phi) : X_+ \to X_+$ is point dissipative on X_+. Furthermore, from ([44], Theorem 2.6), we can see that $Q(t)$ is compact for any $t > 0$. Thus, from ([45], Theorem 1.1.3), we get that $Q(t) : X_+ \to X_+$ has a compact global attractor on X_+.

□

4. Basic reproduction number

According to Lemma 2, the system (1.1) has a unique disease-free steady state $D_0(x) = (S^0(x), V^0(x), 0, 0, 0)$, where $S^0(x)$ and $V^0(x)$ satisfies

$$\begin{cases} -\nabla \cdot (D_1(x)\nabla S^0(x)) = \Lambda(x) - p(x)S^0(x) - \mu(x)S^0(x), \\ -\nabla \cdot (D_2(x)\nabla V^0(x)) = p(x)S^0(x) - \mu(x)V^0(x), \\ \dfrac{\partial S^0(x)}{\partial n} = \dfrac{\partial V^0(x)}{\partial n} = 0, \quad x \in \partial\Omega,\ t > 0. \end{cases} \quad x \in \Omega,\ t > 0,$$

By linearizing model (1.1) at $D_0(x)$, we obtain the following linear system

$$\begin{cases} \dfrac{\partial E(t,x)}{\partial t} = \nabla \cdot (D_3(x)\nabla E) + [\beta(x)I + \beta_2(x)E][S^0(x) + \sigma(x)V^0(x)] \\ \qquad\qquad - (\alpha(x) + \mu(x))E, \\ \dfrac{\partial I(t,x)}{\partial t} = \nabla \cdot (D_4(x)\nabla I) + \alpha(x)E + \rho(x)R - \mu(x)I - \eta(x)I - \delta(x)I, \\ \dfrac{\partial R(t,x)}{\partial t} = \nabla \cdot (D_5(x)\nabla R) + \delta(x)I - \mu(x)R - \rho(x)R, \\ \dfrac{\partial E}{\partial n} = \dfrac{\partial I}{\partial n} = \dfrac{\partial R}{\partial n} = 0,\ x \in \partial\Omega,\ t > 0. \end{cases} \quad x \in \Omega,\ t > 0, \tag{4.1}$$

Let $(E(t,x), I(t,x), R(t,x)) = (e^{\lambda t}\varpi_3(x), e^{\lambda t}\varpi_4(x), e^{\lambda t}\varpi_5(x))$, then we get the following eigenvalue problem

$$\begin{cases} \lambda\varpi_3(x) = \nabla \cdot (D_3(x)\nabla\varpi_3(x)) + [\beta(x)\varpi_4(x) + \beta_2(x)\varpi_3(x)][S^0(x) + \sigma(x)V^0(x)] \\ \qquad\qquad - (\alpha(x) + \mu(x))\varpi_3(x), \\ \lambda\varpi_4(x) = \nabla \cdot (D_4(x)\nabla\varpi_4(x)) + \alpha(x)\varpi_3(x) + \rho(x)\varpi_5(x) - \mu(x)\varpi_4(x) \\ \qquad\qquad - \eta(x)\varpi_4(x) - \delta(x)\varpi_4(x), \\ \lambda\varpi_5(x) = \nabla \cdot (D_5(x)\nabla\varpi_5(x)) + \delta(x)\varpi_4(x) - \mu(x)\varpi_5(x) - \rho(x)\varpi_5(x), \\ \dfrac{\partial\varpi_3}{\partial n} = \dfrac{\partial\varpi_4}{\partial n} = \dfrac{\partial\varpi_5}{\partial n} = 0,\ x \in \partial\Omega,\ t > 0. \end{cases} \quad x \in \Omega,\ t > 0, \tag{4.2}$$

From ([38], Theorem 7.6.1), we have the following result.

Lemma 3. (4.2) *has a unique principal eigenvalue* $\lambda_0 = \lambda_0(S^0(x), V^0(x))$ *with positive eigenvector* $(\varpi_3(x), \varpi_4(x), \varpi_5(x))$.

Define

$$D = \begin{pmatrix} D_3(x) & 0 & 0 \\ 0 & D_4(x) & 0 \\ 0 & 0 & D_5(x) \end{pmatrix},\ V = \begin{pmatrix} \alpha(x) + \mu(x) & 0 & 0 \\ -\alpha(x) & \mu(x) + \eta(x) + \delta(x) & -\rho(x) \\ 0 & -\delta(x) & \mu(x) + \rho(x) \end{pmatrix},$$

$$F = \begin{pmatrix} \beta_2(x)S^0(x) + \sigma(x)\beta_2(x)V^0(x) & \beta(x)S^0(x) + \sigma(x)\beta(x)V^0(x) & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix}.$$

Then system (4.1) is

$$\frac{\partial \eth}{\partial t} = \nabla \cdot (D(x)\nabla \eth) + (F - V)\eth,$$

where $\eth = (E, I, R)^T$. Let the positive semigroup $\mathcal{P}(t) : Y_+ \to Y_+$ generated by the following system

$$\frac{\partial \eth}{\partial t} = \mathcal{B}\eth,$$

where $\mathcal{B} := D - V$. Then we know that $\mathcal{B}$ is a resolvable positive operator [46] and $\mathcal{P}(t)Y_+ \subset Y_+$ can be obtained. Moreover, we denote the spectral radius of Q as $r(Q) = \sup\{|\lambda|, \lambda \in \sigma(Q)\}$ and the spectral bound of Q as $s(Q) = \sup\{Re\lambda, \lambda \in \sigma(Q)\}$.

Denote $\psi = (\psi_3(x), \psi_4(x), \psi_5(x))$ is initial infection distribution. Based on a similar discussion by Luo et al. [10], we define the following operators

$$\mathscr{L}(\psi)(x) = \int_0^{+\infty} F(x)\mathcal{P}(t)\psi(x)dt = F(x)\int_0^{+\infty} \mathcal{P}(t)\psi(x)dt, \tag{4.3}$$

where $F(x)\int_0^{+\infty} \mathcal{P}(t)\psi(x)dt$ denotes the total distribution of new infections. Then, based on [46–49], the basic reproduction number for model (1.1) is defined by $R_0 := r(\mathscr{L})$. Further, through the argument in [46, 47], we get the following lemma.

Lemma 4. *(i) sign*$(R_0 - 1) =$ *sign*(λ_0).
(ii) If $R_0 < 1$*, then* $D_0(x)$ *of system* (1.1) *is locally asymptotically stable.*
(iii) If $R_0 > 1$*, then* $D_0(x)$ *is unstable.*

5. Threshold dynamics

5.1. Extinction of the disease

In this section, we study the global stability of the disease-free steady state $D_0(x)$ of the system (1.1), and establish the following results.

Theorem 2. *The disease-free steady state* $D_0(x)$ *of the model* (1.1) *is globally asymptotically stable in* X_+ *when* $R_0 < 1$.

Proof. According to Lemma 4, we know that $D_0(x)$ is locally asymptotically stable when $R_0 < 1$. Thus we only need to show that $D_0(x)$ is globally attractive. From the first equation of model (1.1), we get

$$\begin{cases} \dfrac{\partial S}{\partial t} \le \nabla \cdot (D_1(x)\nabla S) + \Lambda(x) - \mu(x)S, \quad x \in \Omega,\ t > 0, \\ \dfrac{\partial S}{\partial n} = 0, \quad x \in \partial\Omega,\ t > 0. \end{cases}$$

Using the comparison principle and Lemma 2, we obtain $\limsup_{t\to\infty} S(t, x) \le S^0(x)$ uniformly for $x \in \bar{\Omega}$, which means that there exist positive constants t_1 and ϵ_1 such that $S(t, x) \le S^0(x) + \epsilon_1$ for any $t \ge t_1$. Similarly, from the second equation of model (1.1), we have

$$\begin{cases} \dfrac{\partial V}{\partial t} \le \nabla \cdot (D_2(x)\nabla V) + p(x)(S^0(x) + \epsilon_1) - \mu(x)V, \quad x \in \Omega,\ t > 0, \\ \dfrac{\partial V}{\partial n} = 0, \quad x \in \partial\Omega,\ t > 0. \end{cases}$$

and then we have there exist positive constants ϵ_2 and t_2 such that $V(t,x) \le V^0(x) + \epsilon_2$ for any $t \ge t_2$ and $x \in \bar{\Omega}$. Hence, for any $t \ge t_3$ we have

$$\begin{cases} \dfrac{\partial E}{\partial t} \le \nabla \cdot (D_3(x)\nabla E) + \beta(x)(S^0(x)+\epsilon_1)I + \beta_2(x)(S^0(x)+\epsilon_1)E \\ \qquad\qquad + \sigma(x)\beta(x)(V^0(x)+\epsilon_2)I + \sigma(x)\beta_2(x)(V^0(x)+\epsilon_2)E - (\alpha(x)+\mu(x))E, \\ \dfrac{\partial I}{\partial t} \le \nabla \cdot (D_4(x)\nabla I) + \alpha(x)E + \rho(x)R - (\mu(x)+\eta(x)+\delta(x))I, \qquad x \in \Omega, \\ \dfrac{\partial R}{\partial t} \le \nabla \cdot (D_5(x)\nabla R) + \delta(x)I - (\mu(x)+\rho(x))R, \\ \dfrac{\partial E}{\partial n} = \dfrac{\partial I}{\partial n} = \dfrac{\partial R}{\partial n} = 0, \quad x \in \partial\Omega. \end{cases}$$

Let $(\mathcal{E}(t,x), \mathcal{I}(t,x), \mathcal{R}(t,x))$ be the solution of the following system

$$\begin{cases} \dfrac{\partial \mathcal{E}}{\partial t} = \nabla \cdot (D_3(x)\nabla \mathcal{E}) + \beta(x)(S^0(x)+\epsilon_1)\mathcal{I} + \beta_2(x)(S^0(x)+\epsilon_1)\mathcal{E} \\ \qquad\qquad + \sigma(x)\beta(x)(V^0(x)+\epsilon_2)\mathcal{I} + \sigma(x)\beta_2(x)(V^0(x)+\epsilon_2)\mathcal{E} - (\alpha(x)+\mu(x))\mathcal{E}, \\ \dfrac{\partial \mathcal{I}}{\partial t} = \nabla \cdot (D_4(x)\nabla \mathcal{I}) + \alpha(x)\mathcal{E} + \rho(x)\mathcal{R} - (\mu(x)+\eta(x)+\delta(x))\mathcal{I}, \qquad x \in \Omega, \\ \dfrac{\partial \mathcal{R}}{\partial t} = \nabla \cdot (D_5(x)\nabla \mathcal{R}) + \delta(x)\mathcal{I} - (\mu(x)+\rho(x))\mathcal{R}, \\ \dfrac{\partial \mathcal{E}}{\partial n} = \dfrac{\partial \mathcal{I}}{\partial n} = \dfrac{\partial \mathcal{R}}{\partial n} = 0, \quad x \in \partial\Omega. \end{cases} \tag{5.1}$$

Using comparison principle, $(E(t,x), I(t,x), R(t,x)) \le (\mathcal{E}(t,x), \mathcal{I}(t,x), \mathcal{R}(t,x))$. It follows from the Lemma 4 that $\lambda_0(S^0(x)+\epsilon_1, V^0(x)+\epsilon_2) < 0$ when $R_0 < 1$, where $\lambda_0(S^0(x)+\epsilon_1, V^0(x)+\epsilon_2) < 0$ is the principal eigenvalue of system (5.1). Denote $(a_3(x), a_4(x), a_5(x))$ be the eigenfunction corresponding to this principal eigenvalue $\lambda_0(S^0(x)+\epsilon_1, V^0(x)+\epsilon_2) < 0$, we can get the following solution for system (5.1),

$$(\mathcal{E}(t,x), \mathcal{I}(t,x), \mathcal{R}(t,x)) = (a_3(x), a_4(x), a_5(x))e^{\lambda_0(S^0(x)+\epsilon_1, V^0(x)+\epsilon_2)(t-t_3)}, t \ge t_3.$$

Therefore, $(E(t,x), I(t,x), R(t,x)) \to 0$ uniformly on $x \in \bar{\Omega}$ as $t \to \infty$. Further, we get the following limit system

$$\begin{cases} \dfrac{\partial S}{\partial t} = \nabla \cdot (D_1(x)\nabla S) + \Lambda(x) - \mu(x)S, \ x \in \Omega, \ t > 0, \\ \dfrac{\partial V}{\partial t} = \nabla \cdot (D_2(x)\nabla V) + p(x)S - \mu(x)V, \ x \in \Omega, \ t > 0, \\ \dfrac{\partial S}{\partial n} = \dfrac{\partial V}{\partial n} = 0, \ x \in \partial\Omega, \ t > 0. \end{cases}$$

Based on the theory of asymptotically autonomous semiflows ([50], Corollary 4.3) and Lemma 3, we have $S(t,x) \to S^0(x)$ and $V(t,x) \to V^0(x)$ uniformly for $x \in \bar{\Omega}$ as $t \to \infty$. That means $D_0(x)$ is globally asymptotically stable. □

5.2. Uniform persistence of the disease

In this subsection, we prove that R_0 is the threshold for disease persistence. We first give the following results.

Theorem 3. *If $R_0 > 1$, then (1.1) is uniformly persistent, that is, there is a constant $\varrho > 0$ such that for any initial value $\phi = (\phi_1, \phi_2, \phi_3, \phi_4, \phi_5) \in X_+$ with $\phi_3 \neq 0$, $\phi_4 \neq 0$ and $\phi_5 \neq 0$, $u(t, \cdot, \phi) = (S(t, \cdot, \phi), V(t, \cdot, \phi), E(t, \cdot, \phi), I(t, \cdot, \phi), R(t, \cdot, \phi))$ satisfies*

$$\liminf_{t\to\infty} S(t, \cdot, \phi) \geq \varrho, \ \liminf_{t\to\infty} V(t, \cdot, \phi) \geq \varrho \ \liminf_{t\to\infty} E(t, \cdot, \phi) \geq \varrho, \ \liminf_{t\to\infty} I(t, \cdot, \phi) \geq \varrho, \ \liminf_{t\to\infty} R(t, \cdot, \phi) \geq \varrho,$$

uniformly for $x \in \bar{\Omega}$. Moreover, model (1.1) has at least one endemic steady state $D^*(x) = (S^*(x), V^*(x), E^*(x), I^*(x), R^*(x))$.

Proof. Let

$$X_0 := \{\phi = (\phi_1, \phi_2, \phi_3, \phi_4, \phi_5) \in X_+ : \phi_3 \neq 0, \phi_4 \neq 0 \text{ and } \phi_5 \neq 0\},$$
$$\partial X_0 := X_+ \setminus X_0 = \{\phi = (\phi_1, \phi_2, \phi_3, \phi_4, \phi_5) \in X_+ : \phi_3 = 0 \text{ or } \phi_4 = 0 \text{ or } \phi_5 = 0\}.$$

and

$$M_\partial := \{\phi \in X_+ : Q(t)\phi \in \partial X_0 \ \text{ for all } \ t \geq 0\}, \quad M_1 = \{D_0(x)\}.$$

First, we give the following two claims:

Claim 1. $\cup_{\phi\in M_\partial}\omega(\phi) = M_1$ for any given $\phi \in M_\partial$ (where $\omega(\phi)$ be the omega limit set of ϕ for $Q(t)$).

Proof. Since $Q(t)D_0(x) = D_0(x)$ for all $t \geq 0$, then $M_1 \subset \cup_{\phi\in M_\partial}\omega(\phi)$. Now, we just have to prove $\cup_{\phi\in M_\partial}\omega(\phi) \subset M_1$. Since $Q(t)\phi \in \partial X_0$ for any given $\phi \in M_\partial$ and $t \geq 0$, then $E(t, \cdot, \phi) \equiv 0$ or $I(t, \cdot, \phi) \equiv 0$ or $R(t, \cdot, \phi) \equiv 0$ for all $t \geq 0$.

The first case, $E(t, x) \equiv 0$. By the third and fourth equations of the system (1.1), we yield

$$\frac{\partial I(t,x)}{\partial t} + \frac{\partial R(t,x)}{\partial t} = \nabla \cdot (D_4(x)\nabla I) + \nabla \cdot (D_5(x)\nabla R) - \mu(x)I - \eta(x)I - \mu(x)R,$$

by the parabolic maximum principle [41], we immediately obtain $I(t, x) = 0$ and $R(t, x) = 0$ from model (1.1). Thus, we yield

$$\begin{cases} \dfrac{\partial S(t,\cdot,\phi)}{\partial t} = \nabla \cdot (D_1(x)\nabla S(t, \cdot, \phi)) + \Lambda(x) - \mu(x)S(t, \cdot, \phi), \quad x \in \Omega, \ t > 0, \\ \dfrac{\partial V(t,\cdot,\phi)}{\partial t} = \nabla \cdot (D_2(x)\nabla V(t, \cdot, \phi)) + p(x)S(t, \cdot, \phi) - \mu(x)V(t, \cdot, \phi), \quad x \in \Omega, \ t > 0, \\ \dfrac{\partial S(t,\cdot,\phi)}{\partial n} = \dfrac{\partial V(t,\cdot,\phi)}{\partial n} = 0, \quad x \in \partial\Omega, \ t > 0. \end{cases} \tag{5.2}$$

According to the comparison principle and Lemma 2, $S(t, x) \to S^0(x)$ and $V(t, x) \to V^0(x)$ uniformly on $x \in \bar{\Omega}$ as $t \to \infty$ hold. This implies $\omega(\phi) = D_0(x)$.

Second case, $I(t, x) \equiv 0$. According to the fourth equation of system (1.1), we know that $\alpha(x)E(t, x) + \rho(x)R(t, x) = 0$. Due to $\alpha(x) > 0$ and $\rho(x) > 0$, we have $E(t, x) = 0$ and $R(t, x) = 0$. Thus, by asymptotically autonomous semiflow theory [50], we get $S(t, x) \to S^0(x)$ and $V(t, x) \to V^0(x)$ uniformly on $x \in \bar{\Omega}$ as $t \to \infty$. This also implies $\omega(\phi) = D_0(x)$.

The third case, $R(t,x) \equiv 0$. Similar to the first case, we get $I(t,x) = 0$ and $E(t,x) = 0$. Additionally, $S(t,x) \to S^0(x)$ and $V(t,x) \to V^0(x)$ uniformly on $x \in \bar{\Omega}$ as $t \to \infty$. This implies $\omega(\phi) = D_0(x)$. Thus, we have $\cup_{\phi \in M_\partial} \omega(\phi) \subset M_1$, and finally get $\cup_{\phi \in M_\partial} \omega(\phi) = M_1$.

□

Claim 2. If $R_0 > 1$, then there exists a constant $\delta > 0$ such that for any $\phi \in X_0$, the solution $u(t,\cdot,\phi)$ of the model (1.1) is satisfied

$$\limsup_{t\to\infty} \|u(t,\cdot,\phi) - D_0(x)\|_X \geq \delta.$$

Proof. Suppose that **Claim 2** does not hold, then there exists an enough large T_1 such that $S^0(x) - \delta < S(t,\cdot,\phi) \leq S^0(x) + \delta$, $V^0(x) - \delta < V(t,\cdot,\phi) \leq V^0(x) + \delta$, $0 \leq E(t,\cdot,\phi) < \delta$, $0 \leq I(t,\cdot,\phi) < \delta$, $0 \leq R(t,\cdot,\phi) < \delta$, for all $t \geq T_1$ and $x \in \bar{\Omega}$. From model (1.1), we have

$$\begin{cases} \dfrac{\partial E}{\partial t} \geq \nabla\cdot(D_3(x)\nabla E) + [\beta(x) - \beta_1(x)](S^0(x) - \delta)I + \beta_2(x)(S^0(x) - \delta)E \\ \qquad\qquad + \sigma(x)[\beta(x) - \beta_1(x)](V^0(x) - \delta)I + \sigma(x)\beta_2(x)(V^0(x) - \delta)E - (\alpha(x) + \mu(x))E, \\ \dfrac{\partial I}{\partial t} \geq \nabla\cdot(D_4(x)\nabla I) + \alpha(x)E + \rho(x)R - \mu(x)I - \eta(x)I - \delta(x)I, \qquad x \in \Omega, \\ \dfrac{\partial R}{\partial t} \geq \nabla\cdot(D_5(x)\nabla R) + \delta(x)I - \mu(x)R - \rho(x)R, \\ \dfrac{\partial E}{\partial n} = \dfrac{\partial I}{\partial n} = \dfrac{\partial R}{\partial n} = 0, \quad x \in \partial\Omega. \end{cases} \tag{5.3}$$

Study the following corresponding comparison system with (5.3)

$$\begin{cases} \dfrac{\partial v_3}{\partial t} = \nabla\cdot(D_3(x)\nabla v_3) + [\beta(x) - \beta_1(x)](S^0(x) - \delta)v_4 + \beta_2(x)(S^0(x) - \delta)v_3 \\ \qquad\qquad + \sigma(x)[\beta(x) - \beta_1(x)](V^0(x) - \delta)v_4 + \sigma(x)\beta_2(x)(V^0(x) - \delta)v_3 - (\alpha(x) + \mu(x))v_3, \\ \dfrac{\partial v_4}{\partial t} = \nabla\cdot(D_4(x)\nabla v_4) + \alpha(x)v_3 + \rho(x)v_5 - \mu(x)v_4 - \eta(x)v_4 - \delta(x)v_4, \qquad x \in \Omega, \\ \dfrac{\partial v_5}{\partial t} = \nabla\cdot(D_5(x)\nabla v_5) + \delta(x)v_4 - \mu(x)v_5 - \rho(x)v_5, \\ \dfrac{\partial v_3}{\partial n} = \dfrac{\partial v_4}{\partial n} = \dfrac{\partial v_5}{\partial n} = 0, \quad x \in \partial\Omega. \end{cases} \tag{5.4}$$

For any initial value $\phi \in X_0$, it is obtained by the parabolic maximum principle [41] for all $t \geq T_1$ and $x \in \bar{\Omega}$, there is $E(t,x) > 0$, $I(t,x) > 0$ and $R(t,x) > 0$.

Next, we consider the following eigenvalue problem

$$\begin{cases} \lambda\phi_3^\varrho = \nabla\cdot(D_3(x)\nabla\phi_3^\varrho) + [\beta(x) - \beta_1(x)](S^0(x) - \delta)\phi_4^\varrho + \beta_2(x)(S^0(x) - \delta)\phi_3^\varrho \\ \qquad\qquad + \sigma(x)[\beta(x) - \beta_1(x)](V^0(x) - \delta)\phi_4^\varrho + \sigma(x)\beta_2(x)(V^0(x) - \delta)\phi_3^\varrho - (\alpha(x) + \mu(x))\phi_3^\varrho, \\ \lambda\phi_4^\varrho = \nabla\cdot(D_4(x)\nabla\phi_4^\varrho) + \alpha(x)\phi_3^\varrho + \rho(x)\phi_5^\varrho - \mu(x)\phi_4^\varrho - \eta(x)\phi_4^\varrho - \delta(x)\phi_4^\varrho, \qquad x \in \Omega, \\ \lambda\phi_5^\varrho = \nabla\cdot(D_5(x)\nabla\phi_5^\varrho) + \delta(x)\phi_4^\varrho - \mu(x)\phi_5^\varrho - \rho(x)\phi_5^\varrho, \\ \dfrac{\partial\phi_3^\varrho}{\partial n} = \dfrac{\partial\phi_4^\varrho}{\partial n} = \dfrac{\partial\phi_5^\varrho}{\partial n} = 0, \quad x \in \partial\Omega. \end{cases} \tag{5.5}$$

Let $\lambda_0(\epsilon)$ be the principal eigenvalue of (5.5). There is a constant that is small enough $\epsilon_0 > 0$, such that $\lambda_0(\epsilon_0) > 0$ and $S^0(x) - \epsilon_0 > 0$, $V^0(x) - \epsilon_0 > 0$ for all $x \in \bar{\Omega}$. In addition, the eigenvalue $\lambda_0(\epsilon_0)$ corresponds to a strictly positive eigenfunction $(\phi_3^\varrho(x), \phi_4^\varrho(x), \phi_5^\varrho(x))$ for $x \in \bar{\Omega}$. Clearly, system (5.4) has a solution $(v_3(t,x), v_4(t,x), v_5(t,x)) = e^{\lambda_0(\epsilon_0)(t-T_1)}(\phi_3^\varrho(x), \phi_4^\varrho(x), \phi_5^\varrho(x))$. Then we can choose a constant $c_1 > 0$, according to the comparison principle, we get

$$(E(t,x), I(t,x), R(t,x)) > c_1(\phi_3^\varrho(x), \phi_4^\varrho(x), \phi_5^\varrho(x))e^{\lambda_0(\epsilon_0)(t-T_1)}, \quad t \geq T_1.$$

Owing to $\lambda_0(\epsilon_0) > 0$, we have $\lim_{t\to\infty} E(t,x) = +\infty$, $\lim_{t\to\infty} I(t,x) = +\infty$ and $\lim_{t\to\infty} R(t,x) = +\infty$. However, by Theorem 1, we know from the boundedness of $(E(t,x), I(t,x), R(t,x))$ that this is a contradiction, which shows **Claim 2** holds. □

Next, we prove the required persistence. By Theorem 1, the solution $u(t,\cdot,\phi)$ of model (1.1) is ultimately bounded, that is, for a constant $M > 0$ and time $T_2 > 0$, such that $u(t,\cdot,\phi) \leq M$ for all $t > T_2$ and $x \in \bar{\Omega}$. Therefore, according to the first and second equations of model (1.1), we get

$$\begin{cases} \dfrac{\partial S}{\partial t} \geq \nabla \cdot (D_1(x)\nabla S) + \Lambda_* - \beta^* S M - \beta_2^* S M - p^* S - \mu^* S, & x \in \Omega,\ t > 0, \\ \dfrac{\partial V}{\partial t} \geq \nabla \cdot (D_2(x)\nabla V) + p_* S - \sigma^*\beta^* V M - \sigma^*\beta_2^* V M - \mu^* V, & x \in \Omega,\ t > 0, \\ \dfrac{\partial S}{\partial n} = \dfrac{\partial V}{\partial n} = 0, & x \in \partial\Omega,\ t > 0, \end{cases}$$

for any $t \geq T_2$. It follows from Lemma 2 and the comparison principle [41]

$$\liminf_{t\to\infty} S(t,\cdot,\phi) \geq \frac{\Lambda_*}{(\beta^* + \beta_2^*)M + \mu^* + p^*},$$
$$\liminf_{t\to\infty} V(t,\cdot,\phi) \geq \frac{p_*\Lambda_*}{[(\beta^* + \beta_2^*)M + \mu^* + p^*][(\beta^* + \beta_2^*)\sigma^* M + \mu^*]},$$

which implies that $S(t,\cdot,\phi)$ and $V(t,\cdot,\phi)$ has a positive lower bound. Therefore, the uniform persistence of $S(t,\cdot,\phi)$ and $V(t,\cdot,\phi)$ in model (1.1) is proved.

Define a continuous function $p : X_+ \to [0, +\infty)$ by

$$p(\phi) = \min\{\min_{x\in\bar{\Omega}} \phi_3(x), \min_{x\in\bar{\Omega}} \phi_4(x), \min_{x\in\bar{\Omega}} \phi_5(x)\}.$$

Obviously, $p^{-1}(0, +\infty) \subseteq X_0$ and p satisfies the following properties: $p(\phi) = 0$ and $\phi \in X_0$ or $p(Q(t)\phi) > 0$. Based on the definition ([51], Section 3), we know that p is a generalized distance function for semiflow $Q(t) : X_+ \to X_+$. In addition, as can be seen from the discussion in **Claim 1** and **Claim 2**, all the solution of the model (1.1) tend to $D_0(x)$ on boundary ∂X_0, which implies that $D_0(x)$ is a isolated invariant set in X_+, no subset of M_1 forms a cycle in ∂X_0 and $W^s(D_0) \cap X_0 = \emptyset$, where $W^s(D_0)$ is the stable set of $D_0(x)$. Thus, $W^s(D_0(x)) \cap p^{-1}(0, \infty) = \emptyset$. By ([51], Theorem 3), there exists a constant $\varrho_2 > 0$ such that $\liminf_{t\to\infty} p(Q(t)\phi) \geq \varrho_2$ for all $\phi \in X_0$, which implies

$$\liminf_{t\to\infty} E(t,\cdot,\phi) \geq \varrho_2, \ \liminf_{t\to\infty} I(t,\cdot,\phi) \geq \varrho_2, \ \liminf_{t\to\infty} R(t,\cdot,\phi) \geq \varrho_2.$$

Define $\varrho_1 = \min\left\{\frac{\Lambda_*}{(\beta^*+\beta_2^*)M+\mu^*+p^*}, \frac{p_*\Lambda_*}{[(\beta^*+\beta_2^*)M+\mu^*+p^*][(\beta^*+\beta_2^*)\sigma^* M+\mu^*]}\right\}$ and let $\varrho = \min\{\varrho_1, \varrho_2\}$, the uniform persistence of the system (1.1) is obtained. From the above proof and Theorem 4.7 in [52], we have the following result.

Lemma 5. *When $R_0 > 1$, model* (1.1) *has at least one positive steady state $D^*(x) = (S^*(x), V^*(x), E^*(x), I^*(x), R^*(x))$.*

□

6. Global stability in homogeneous Spaces

In this section, we study the dynamic behavior of model (1.1) in homogeneous space, where all the coefficients of model (1.1) are positive constants except the diffusion coefficient.

$$\begin{cases} \dfrac{\partial S}{\partial t} = \nabla \cdot (D_1(x)\nabla S) + \Lambda - [\beta - \beta_1 f(I)]SI - \beta_2 SE - pS - \mu S, \\ \dfrac{\partial V}{\partial t} = \nabla \cdot (D_2(x)\nabla V) + pS - \sigma[\beta - \beta_1 f(I)]VI - \sigma\beta_2 VE - \mu V, \\ \dfrac{\partial E}{\partial t} = \nabla \cdot (D_3(x)\nabla E) + \{[\beta - \beta_1 f(I)]I + \beta_2 E\}(S + \sigma V) - \alpha E - \mu E, \ \ x \in \Omega, \ t \geq 0. \\ \dfrac{\partial I}{\partial t} = \nabla \cdot (D_4(x)\nabla I) + \alpha E + \rho R - \mu I - \eta I - \delta I, \\ \dfrac{\partial R}{\partial t} = \nabla \cdot (D_5(x)\nabla R) + \delta I - \mu R - \rho R, \\ \dfrac{\partial S}{\partial n} = \dfrac{\partial V}{\partial n} = \dfrac{\partial E}{\partial n} = \dfrac{\partial I}{\partial n} = \dfrac{\partial R}{\partial n} = 0, \ \ x \in \partial\Omega, \ t \geq 0. \end{cases} \tag{6.1}$$

Obviously, by Lemma 2, the system (6.1) has a disease-free equilibrium $Q^0 = (S^0, V^0, 0, 0, 0)$ with $S^0 = \frac{\Lambda}{\mu+p}$ and $V^0 = \frac{p\Lambda}{\mu(\mu+p)}$. By simple computation, one has

$$R_0 = \frac{\beta_2(\mu\Lambda + \sigma p\Lambda)}{\mu(\mu+p)(\alpha+\mu)} + \frac{\beta\alpha(\mu\Lambda + \sigma p\Lambda)(\mu+\rho)}{\mu(\mu+p)(\alpha+\mu)[(\mu+\eta+\delta)(\mu+\rho) - \rho\delta]} \triangleq R_{01} + R_{02}.$$

Remark 1. *R_{01} and R_{02} can be expressed as the contribution of exposed and infected individuals to the basic reproduction number, respectively. It can be seen that a primary case in the latent population has a rate $\frac{\beta_2(\mu\Lambda+\sigma p\Lambda)}{\mu(\mu+p)}$ of infectious contact with the susceptible population in the expected time $\frac{1}{\alpha+\mu}$, while a primary case in the infected population has a rate $\frac{\beta\alpha(\mu\Lambda+\sigma p\Lambda)(\mu+\rho)}{\mu(\mu+p)}$ of infectious contact with the susceptible population in the expected time $\frac{1}{(\alpha+\mu)[(\mu+\eta+\delta)(\mu+\rho)-\rho\delta]}$.*

Theorem 4. *When $R_0 > 1$, there exists a unique endemic equilibrium for system* (6.1).

Proof. Suppose that there exists an endemic equilibrium $Q^*(S^*, V^*, E^*, I^*, R^*)$ for system (6.1), then Q^* satisfies

$$\begin{cases} \Lambda - [\beta - \beta_1 f(I^*)]S^*I^* - \beta_2 S^*E^* - \mu S^* - pS^* = 0, \\ pS^* - \sigma[\beta - \beta_1 f(I^*)]V^*I^* - \beta_2 V^*E^* - \mu V^* = 0, \\ \{[\beta - \beta_1 f(I^*)]I^* + \beta_2 E^*\}(S^* + \sigma V^*) - \alpha E^* - \mu E^* = 0, \\ \alpha E^* + \rho R^* - \mu I^* - \eta I^* - \delta I^* = 0, \\ \delta I^* - \mu R^* - \rho R^* = 0. \end{cases} \tag{6.2}$$

By a simple computation, we have

$$S^* = \frac{\Lambda}{[\beta - \beta_1 f(I^*)]I^* + \beta_2 E^* + \mu + p}, \ E^* = \frac{[(\mu+\rho)(\mu+\eta+\delta) - \rho\delta]I^*}{\alpha(\mu+\rho)},$$
$$V^* = \frac{p\Lambda}{\{[\beta - \beta_1 f(I^*)]I^* + \beta_2 E^* + \mu + p\}\{\sigma[\beta - \beta_1 f(I^*)]I^* + \sigma\beta_2 E^* + \mu\}}.$$

Since $I^* \neq 0$, by the third equation in (6.2), we have

$$S^* + \sigma V^* = \frac{(\alpha+\mu)E^*}{[\beta - \beta_1 f(I^*)] + \beta_2 E^*} = \frac{(\alpha+\mu)[(\mu+\rho)(\mu+\eta+\delta) - \rho\delta]}{\alpha(\mu+\rho)[\beta - \beta_1 f(I^*)] + \beta_2[(\mu+\rho)(\mu+\eta+\delta) - \rho\delta]}$$

Let $C(I) = \alpha(\mu+\rho)[\beta - \beta_1 f(I)]I + \beta_2[(\mu+\rho)(\mu+\eta+\delta) - \rho\delta]I$. According to the known formula, we have

$$\begin{aligned} S^* + \sigma V^* =& \frac{\Lambda}{[\beta - \beta_1 f(I^*)]I^* + \beta_2 E^* + \mu + p}\left[1 + \frac{\sigma p}{\sigma[\beta - \beta_1 f(I^*)]I^* + \sigma\beta_2 E^* + \mu}\right] \\ =& \frac{\alpha\Lambda(\mu+\rho)[\sigma C(I^*) + \alpha(\mu+\rho)(\mu+\sigma p)]}{[C(I^*) + \alpha(\mu+p)(\mu+\rho)][\sigma C(I^*) + \alpha\mu(\mu+\rho)]}. \end{aligned}$$

Thus,

$$\frac{\alpha\Lambda(\mu+\rho)[\sigma C(I^*) + \alpha(\mu+\rho)(\mu+\sigma p)]}{[C(I^*) + \alpha(\mu+p)(\mu+\rho)][\sigma C(I^*) + \alpha\mu(\mu+\rho)]} = \frac{(\alpha+\mu)[(\mu+\rho)(\mu+\eta+\delta) - \rho\delta]}{\alpha(\mu+\rho)[\beta - \beta_1 f(I^*)] + \beta_2[(\mu+\rho)(\mu+\eta+\delta) - \rho\delta]}.$$

Define $H(I) := \psi(I) - \varphi(I) = 0$, where

$$\begin{aligned} \psi(I) &= \frac{\alpha(\mu+\rho)[\sigma C(I) + \alpha(\mu+\rho)(\mu+\sigma p)]}{[C(I) + \alpha(\mu+p)(\mu+\rho)][\sigma C(I) + \alpha\mu(\mu+\rho)]}, \\ \varphi(I) &= \frac{(\alpha+\mu)[(\mu+\rho)(\mu+\eta+\delta) - \rho\delta]}{\Lambda\{\alpha(\mu+\rho)[\beta - \beta_1 f(I)] + \beta_2[(\mu+\rho)(\mu+\eta+\delta) - \rho\delta]\}}. \end{aligned} \tag{6.3}$$

Obviously, $\varphi(I)$ is increasing for $I > 0$. Further

$$\psi'(I) = \frac{h(I)}{\{[C(I^*) + \alpha(\mu+p)(\mu+\rho)][\sigma C(I^*) + \alpha\mu(\mu+\rho)]\}^2},$$

where

$$\begin{aligned} h(I) =& \alpha(\mu+\rho)C'(I)\Big\{\sigma[C(I) + \alpha(\mu+\rho)(\mu+p)][\sigma C(I) + \alpha\mu(\mu+\rho)] \\ &- [\sigma C(I) + \alpha(\mu+\rho)(\mu+\sigma p)][\sigma C(I) + \alpha\mu(\mu+\rho)] \\ &- [\sigma C(I) + \alpha(\mu+\rho)(\mu+\sigma p)]\sigma[C(I) + \alpha(\mu+\rho)(\mu+p)]\Big\} < 0. \end{aligned}$$

Combined with (A_1), (A_2) and (A_3), we obtain that function $\psi(I)$ is decreasing for $I > 0$, and then $H(I)$ is decreasing for $I > 0$.

In addition, from (6.3) we get

$$H(0) = \frac{(\alpha+\mu)[(\mu+\rho)(\mu+\eta+\delta) - \rho\delta]}{\Lambda\{\beta\alpha(\mu+\rho) + \beta_2[(\mu+\rho)(\mu+\eta+\delta) - \rho\delta]\}}(R_0 - 1), \tag{6.4}$$

and

$$H(I) < \frac{\alpha(\mu+\rho)}{C(I)} - \frac{(\alpha+\mu)[(\mu+\rho)(\mu+\eta+\delta)-\rho\delta]}{\Lambda\{\alpha(\mu+\rho)[\beta-\beta_1 f(I)]+\beta_2[(\mu+\rho)(\mu+\eta+\delta)-\rho\delta]\}}.$$

It is easy to see that $H(0) > 0$ and $\lim_{I\to\infty} H(I) < 0$ when $R_0 > 1$. Based on the monotonicity of $H(I)$, we can get system (6.1) has unique endemic equilibrium $Q^* = (S^*, V^*, E^*, I^*, R^*)$ when $R_0 > 1$. This completes the proof.

□

Next we mainly discuss the global stability of disease-free equilibrium Q^0 and endemic equilibrium Q^*. Based on the discussion of Theorem 2, we give the following invariant domain

$$\Gamma = \{\phi = (\phi_1, \phi_2, \phi_3, \phi_4, \phi_5) \in X_+ : \phi_1(x) \le S^0, \phi_2(x) \le V^0, \quad x \in \bar{\Omega}\}.$$

Theorem 5. (*a*) *If $R_0 \le 1$, then disease-free equilibrium Q^0 is globally asymptotically stable in domain* Γ.

(*b*) *If $R_0 > 1$, then equilibrium Q^0 is unstable.*

Proof. Define

$$W(t) = \int_\Omega \Big[S^0 G\Big(\frac{S}{S^0}\Big) + V^0 G\Big(\frac{V}{V^0}\Big) + E + \frac{\alpha+\mu}{\alpha} I + \frac{(\alpha+\mu)\rho}{\alpha(\mu+\rho)} R\Big] dx,$$

where $G(x) = x - 1 - \ln x$. According to [43, Theorem 3.7], we have

$$\begin{aligned}
\frac{\mathrm{d}W(t)}{\mathrm{d}t} &= \int_\Omega \Big\{(1-\frac{S^0}{S})[\nabla\cdot(D_1(x)\nabla S) + \Lambda - [\beta-\beta_1 f(I)]SI - \beta_2 SE - (\mu+p)S] \\
&\quad + (1-\frac{V^0}{V})[\nabla\cdot(D_2(x)\nabla V) + pS - \sigma[\beta-\beta_1 f(I)]VI - \sigma\beta_2 VE - \mu V] \\
&\quad + \nabla\cdot(D_3(x)\nabla E) + \{[\beta-\beta_1 f(I)]I + \beta_2 E\}(S+\sigma V) - (\alpha+\mu)E \\
&\quad + \frac{\alpha+\mu}{\alpha}[\nabla\cdot(D_4(x)\nabla I) + \alpha E + \rho R - (\mu+\eta+\delta)I] \\
&\quad + \frac{(\alpha+\mu)\rho}{\alpha(\mu+\rho)}[\nabla\cdot(D_5(x)\nabla R) + \delta I - (\mu+\rho)R]\Big\} dx \\
&\le \int_\Omega \Big\{\mu S^0\Big(2 - \frac{S^0}{S} - \frac{S}{S^0}\Big) + \mu V^0\Big(3 - \frac{S^0}{S} - \frac{V}{V^0} - \frac{SV^0}{S^0 V}\Big) \\
&\quad + [\beta-\beta_1 f(I)]S^0 I + \beta_2 S^0 E + \sigma[\beta-\beta_1 f(I)]V^0 I + \sigma\beta_2 V^0 E - (\alpha+\mu)E \\
&\quad + \frac{\alpha+\mu}{\alpha}[\alpha E + \rho R - (\mu+\eta+\delta)I] + \frac{(\alpha+\mu)\rho}{\alpha(\mu+\rho)}[\delta I - (\mu+\rho)R]\Big\} dx \\
&\le \int_\Omega \Big[\mu S^0\Big(2 - \frac{S^0}{S} - \frac{S}{S^0}\Big) + \mu V^0\Big(3 - \frac{S^0}{S} - \frac{V}{V^0} - \frac{SV^0}{S^0 V}\Big)\Big] dx \\
&\quad + \frac{1}{\alpha\mu(\mu+p)(\mu+\rho)} \int_\Omega I\Big\{\mu(\mu+p)(\alpha+\mu)[(\mu+\eta+\delta)(\mu+\rho) - \rho\delta](R_0 - 1)\Big\} dx.
\end{aligned} \tag{6.5}$$

Therefore, $\frac{dW(t)}{dt} \le 0$ when $R_0 < 1$ and $\frac{dW(t)}{dt} = 0$ if and only if $S = S^0$, $V = V^0$ and $I(t,x) = 0$, from model (6.1), we can obtain $E(t,x) = 0$ and $R(t,x) = 0$. This means that Q^0 is the largest invariant

set in $\{(S, V, E, I, R) \in \Gamma : \frac{dW(t)}{dt} = 0\}$. By the invariance principle, we can conclude that Q^0 is globally asymptotically stable for model (6.1).

□

Theorem 6. *If $R_0 > 1$ and*

$$[g(I) - g(I^*)]\Big[\frac{g(I)}{I} - \frac{g(I^*)}{I^*}\Big] \le 0, \quad (E - E^*)\Big(\frac{E}{I} - \frac{E^*}{I^*}\Big) \le 0, \tag{6.6}$$

where $g(I) = [\beta - \beta_1 f(I)]I$, then endemic equilibrium Q^* is globally asymptotically stable.

Proof. Define

$$L(t) = \int_\Omega [W_1(t) + W_2(t) + W_3(t) + W_4(t) + W_5(t)]dx$$

where

$$W_1(t) = S - S^* - S^* \ln \frac{S}{S^*},\ W_2(t) = V - V^* - V^* \ln \frac{V}{V^*},\ W_3(t) = E - E^* - E^* \ln \frac{E}{E^*},$$
$$W_4(t) = \frac{\alpha+\mu}{\alpha}(I - I^* - I^* \ln \frac{I}{I^*}),\ W_5(t) = \frac{(\alpha+\mu)\rho}{\alpha(\mu+\rho)}(R - R^* - R^* \ln \frac{R}{R^*}).$$

Define $\beta(I) = \beta - \beta_1 f(I)$, we yield that

$$\begin{aligned}
\frac{dL(t)}{dt} &= \int_\Omega \Big\{(1 - \frac{S^*}{S})\frac{dS}{dt} + (1 - \frac{V^*}{V})\frac{dV}{dt} + (1 - \frac{E^*}{E})\frac{dE}{dt} \\
&\quad + \frac{\alpha+\mu}{\alpha}(1 - \frac{I^*}{I})\frac{dI}{dt} + \frac{(\alpha+\mu)\rho}{\alpha(\mu+\rho)}(1 - \frac{R^*}{R})\frac{dR}{dt}\Big\}dx \\
&= \int_\Omega \Big\{(1 - \frac{S^*}{S})[\nabla \cdot (D_1(x)\nabla S) + \Lambda - \beta(I)SI - \beta_2 SE - pS - \mu S] \\
&\quad + (1 - \frac{V^*}{V})[\nabla \cdot (D_2(x)\nabla V) + pS - \sigma\beta(I)VI - \sigma\beta_2 VE - \mu V] \\
&\quad + (1 - \frac{E^*}{E})[\nabla \cdot (D_3(x)\nabla E) + \beta(I)SI + \beta_2 SE + \sigma\beta(I)VI + \sigma\beta_2 VE - \alpha E - \mu E] \\
&\quad + \frac{\alpha+\mu}{\alpha}(1 - \frac{I^*}{I})[\nabla \cdot (D_4(x)\nabla I) + \alpha E + \rho R - (\mu + \eta + \delta)I] \\
&\quad + \frac{(\alpha+\mu)\rho}{\alpha(\mu+\rho)}(1 - \frac{R^*}{R})[\nabla \cdot (D_5(x)\nabla R) + \delta I - (\mu+\rho)R]\Big\}dx.
\end{aligned} \tag{6.7}$$

From [43, Theorem 3.7], we can deduce that

$$\begin{aligned}
\frac{dL(t)}{dt} &= \int_\Omega \Big\{ - S^* D_1(x)\frac{\|\nabla S\|^2}{S^2} - V^* D_2(x)\frac{\|\nabla V\|^2}{V^2} - E^* D_3(x)\frac{\|\nabla E\|^2}{E^2} \\
&\quad - \frac{\alpha+\mu}{\alpha} I^* D_4(x)\frac{\|\nabla I\|^2}{I^2} - \frac{(\alpha+\mu)\rho}{\alpha(\mu+\rho)} R^* D_5(x)\frac{\|\nabla R\|^2}{R^2} \\
&\quad + (1 - \frac{S^*}{S})[\Lambda - \beta(I)SI - \beta_2 SE - pS - \mu S] + (1 - \frac{V^*}{V})[pS - \sigma\beta(I)VI - \sigma\beta_2 VE - \mu V] \\
&\quad + (1 - \frac{E^*}{E})[\beta(I)SI + \beta_2 SE + \sigma\beta(I)VI + \sigma\beta_2 VE - \alpha E - \mu E]
\end{aligned}$$

$$
\begin{aligned}
&+(1-\frac{I^*}{I})[(\alpha+\mu)E+\frac{(\alpha+\mu)\rho}{\alpha}R-\frac{(\alpha+\mu)(\mu+\eta+\delta)}{\alpha}I]\\
&+(1-\frac{R^*}{R})[\frac{(\alpha+\mu)\rho\delta}{\alpha(\mu+\rho)}I-\frac{(\alpha+\mu)\rho}{\alpha}R]\Big\}dx\\
\leq&\int_\Omega\Big\{\mu S^*(2-\frac{S^*}{S}-\frac{S}{S^*})+\mu V^*(3-\frac{S^*}{S}-\frac{V}{V^*}-\frac{SV^*}{S^*V})\\
&+[\sigma\beta(I^*)V^*I^*+\sigma\beta_2V^*E^*](2-\frac{S^*}{S}-\frac{S}{S^*})+(1-\frac{S^*}{S})[\beta(I^*)S^*I^*+\beta_2V^*E^*]+\beta(I)S^*I\\
&+\beta_2S^*E+(\frac{S^*}{S}-\frac{SV^*}{S^*V})[\sigma\beta(I^*)V^*I^*+\sigma\beta_2V^*E^*]+\sigma\beta(I)V^*I+\sigma\beta_2V^*E-\beta(I)SI\cdot\frac{E^*}{E}\\
&-\beta_2SE^*-\sigma\beta(I)VI\cdot\frac{E^*}{E}-\sigma\beta_2VE^*+(\alpha+\mu)E^*-(\alpha+\mu)E^*\cdot\frac{I}{I^*}-(\alpha+\mu)E^*\cdot\frac{EI^*}{E^*I}\\
&+(\alpha+\mu)E^*+\frac{2(\alpha+\mu)\rho}{\alpha}R^*-\frac{(\alpha+\mu)\rho}{\alpha}R\cdot\frac{I^*}{I}-\frac{(\alpha+\mu)\rho\delta}{\alpha(\mu+\rho)}I\cdot\frac{R^*}{R}\Big\}dx\\
=&\int_\Omega\Big\{\mu S^*\Big(2-\frac{S^*}{S}-\frac{S}{S^*}\Big)+\mu V^*\Big(3-\frac{S^*}{S}-\frac{V}{V^*}-\frac{SV^*}{S^*V}\Big)\\
&+\beta(I^*)S^*I^*\Big(3-\frac{S^*}{S}+\frac{\beta(I)I}{\beta(I^*)I^*}-\frac{S\beta(I)IE^*}{S^*\beta(I^*)I^*E}-\frac{I}{I^*}-\frac{I^*E}{IE^*}\Big)\\
&+\beta_2S^*E^*\Big(3-\frac{S^*}{S}-\frac{S}{S^*}+\frac{E}{E^*}-\frac{I}{I^*}-\frac{I^*E}{IE^*}\Big)\\
&+\sigma\beta(I^*)V^*I^*\Big(4-\frac{S^*}{S}-\frac{SV^*}{S^*V}+\frac{\beta(I)I}{\beta(I^*)I^*}-\frac{V\beta(I)IE^*}{V^*\beta(I^*)I^*E}-\frac{I}{I^*}-\frac{I^*E}{IE^*}\Big)\\
&+\sigma\beta_2V^*E^*\Big(4-\frac{S^*}{S}-\frac{SV^*}{S^*V}-\frac{V}{V^*}+\frac{E}{E^*}-\frac{I}{I^*}-\frac{I^*E}{IE^*}\Big)\\
&+\frac{(\alpha+\mu)\rho\delta}{\alpha(\mu+\rho)}I^*\Big(2-\frac{I^*R}{IR^*}-\frac{IR^*}{I^*R}\Big)\Big\}dx.
\end{aligned}
$$

Let $g(I)=\beta(I)I$ and $F(x)=1-x+\ln x$. Obviously, we have $F(x)\leq 0$ for all $x>0$, and $F(x)=0$ for $x=1$, then

$$
\begin{aligned}
&3-\frac{S^*}{S}+\frac{g(I)}{g(I^*)}-\frac{Sg(I)E^*}{S^*g(I^*)E}-\frac{I}{I^*}-\frac{I^*E}{IE^*}\\
=&3-\frac{S^*}{S}+\frac{g(I)}{g(I^*)}-\frac{Sg(I)E^*}{S^*g(I^*)E}-\frac{I}{I^*}-\frac{I^*E}{IE^*}+\ln\frac{S^*}{S}+\ln\frac{Sg(I)E^*}{S^*g(I^*)E}+\ln\frac{Ig(I^*)}{I^*g(I)}+\ln\frac{I^*E}{IE^*}\\
=&F(\frac{S^*}{S})+F(\frac{Sg(I)E^*}{S^*g(I^*)E})+F(\frac{Ig(I^*)}{I^*g(I)})+F(\frac{I^*E}{IE^*})+\frac{I}{g(I)g(I^*)}[g(I)-g(I^*)]\Big[\frac{g(I)}{I}-\frac{g(I^*)}{I^*}\Big],\\
&3-\frac{S^*}{S}-\frac{S}{S^*}+\frac{E}{E^*}-\frac{I}{I^*}-\frac{I^*E}{IE^*}\\
=&F(\frac{S^*}{S})+F(\frac{S}{S^*})+F(\frac{I^*E}{IE^*})+F(\frac{IE^*}{I^*E})+\frac{I}{EE^*}(E-E^*)\Big(\frac{E}{I}-\frac{E^*}{I^*}\Big),\\
&4-\frac{S^*}{S}-\frac{SV^*}{S^*V}-\frac{V}{V^*}+\frac{E}{E^*}-\frac{I}{I^*}-\frac{I^*E}{IE^*}\\
=&F(\frac{S^*}{S})+F(\frac{SV^*}{S^*V})+F(\frac{V}{V^*})+F(\frac{I^*E}{IE^*})+F(\frac{IE^*}{I^*E})+\frac{I}{EE^*}(E-E^*)\Big(\frac{E}{I}-\frac{E^*}{I^*}\Big),
\end{aligned}
$$

$$
\begin{aligned}
&4-\frac{S^*}{S}-\frac{SV^*}{S^*V}+\frac{g(I)}{g(I^*)}-\frac{Vg(I)E^*}{V^*g(I^*)E}-\frac{I}{I^*}-\frac{I^*E}{IE^*}\\
=&F(\frac{S^*}{S})+F(\frac{SV^*}{S^*V})+F(\frac{Vg(I)E^*}{V^*g(I^*)E})+F(\frac{Ig(I^*)}{I^*g(I)})+F(\frac{I^*E}{IE^*})+\frac{I}{g(I)g(I^*)}[g(I)-g(I^*)]\Big[\frac{g(I)}{I}-\frac{g(I^*)}{I^*}\Big].
\end{aligned}
$$

Therefore, from (6.6) we further have $\frac{dL(t)}{dt} \leq 0$ for all $S > 0$, $V > 0$, $E > 0$, $I > 0$ and $R > 0$. Moreover, $\frac{dL(t)}{dt} = 0$ if and only if $S = S^*$, $V = V^*$, $E = E^*$, $I = I^*$ and $R = R^*$, which means that $\{Q^*\}$ is the largest invariant set for $\frac{dL(t)}{dt} = 0$. Using invariance principle, we obtain that Q^* of system (6.1) is globally asymptotically stable.

□

7. Numerical simulations

7.1. Simulation of threshold dynamics

For convenience, we pay our attention to $\Omega = [0, 10]$. The nonlinear term under the influence of media reports is expressed as $f(I) = \frac{q_1 I}{1+c_1 I}$ (where c_1 reflects the deviation degree of people's and relevant departments' understanding of disease information). At the same time, we take the initial value as follows:

$$
\begin{cases}
S(x,0) = 9700 \times 0.92 \times e^{-10(x-5)^2},\ V(x,0) = 9600 \times 0.94 \times e^{-10(x-5)^2},\\
E(x,0) = 4000 \times 0.04 \times e^{-10(x-5)^2},\ I(x,0) = 40 \times 0.02 \times e^{-10(x-5)^2},\ R(x,0) = 0.
\end{cases} \tag{7.1}
$$

Case 1: In order to simulate the result of Theorem 2, we take the parameters in the model (1.1) as shown in Table 2. By the numerical scheme in [47], we get the basic reproduction number $R_0 =$

Table 2. Values of all parameters in model (1.1) for Case 1.

Parameter	Value	Parameter	Value	Parameter	Value
$\Lambda(x)$	$40(1 + 0.5\sin(2\pi x))$	$c_1(x)$	0.15	$q_1(x)$	0.08
$\beta(x)$	$4 \times 10^{-4}(1 + 0.5\sin(2\pi x))$	$\sigma(x)$	0.6	$\alpha(x)$	$0.2(1 + 0.2\sin(2\pi x))$
$\beta_1(x)$	$3.6 \times 10^{-4}(1 + 0.5\sin(2\pi x))$	$\delta(x)$	$0.3(1 + 0.5\sin(2\pi x))$	$\eta(x)$	$0.2(1 + 0.2\sin(2\pi x))$
$\beta_2(x)$	$8 \times 10^{-4}(1 + 0.5\sin(2\pi x))$	$p(x)$	$0.3(1 + 0.5\sin(2\pi x))$	$\rho(x)$	$0.4(1 + 0.5\sin(2\pi x))$
$\mu(x)$	$0.1(1 + 0.5\sin(2\pi x))$	$D_1(x)$	$0.09 + 0.005\sin(2\pi x)$	$D_2(x)$	$0.08 + 0.005\sin(2\pi x)$
$D_3(x)$	$0.07 + 0.005\sin(2\pi x)$	$D_4(x)$	$0.06 + 0.005\sin(2\pi x)$	$D_5(x)$	$0.085 + 0.005\sin(2\pi x)$

$0.9484 < 1$. Moreover, we plot the time evolution of exposed individuals $E(x,t)$ (Figure 1(a)–(c)) and infected individuals $I(x,t)$ (Figure 1(d)–(f)) for model (1.1). Figure 1 shows that the density of individuals $E(x,t)$ and infected individuals $I(x,t)$ converge to zero over time, which indicates the Theorem 2.

Case 2: To support the conclusion of Theorem 3, we choose $\beta(x) = 8 \times 10^{-4} \times (1 + 0.5\sin(2\pi x))$, $\beta_2(x) = 1 \times 10^{-3} \times (1 + 0.5\sin(2\pi x))$, $\alpha(x) = 0.1 \times (1 + 0.2\sin(2\pi x))$, $\eta(x) = 0.08 \times (1 + 0.2\sin(2\pi x))$ and the other parameters remain the same as Table 2. By the numerical scheme in [47], we get the basic reproduction number $R_0 = 1.8605 > 1$. Moreover, we plot the time evolution of exposed individuals $E(x,t)$ (Figure 2(a)–(c)) and infected individuals $I(x,t)$ (Figure 2(d)–(f)) for model (1.1). Indeed, as seen in Figure 2, the densities of exposed individuals $E(x,t)$ and infected individuals $I(x,t)$ converge to a spatially heterogeneous steady state over time, which shows Theorem 3.

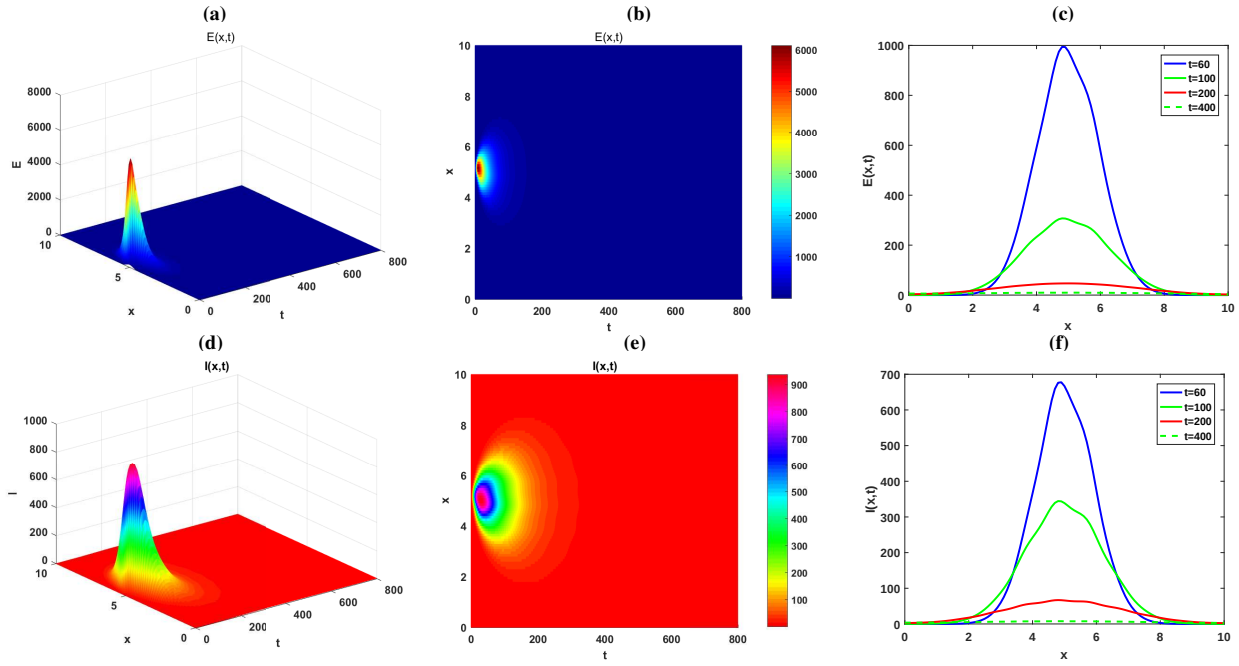

Figure 1. Time evolution of system (1.1) with initial values. (a)–(c): The evolution of the $E(x,t)$ over time; (d)–(f): The evolution of the $I(x,t)$ over time.

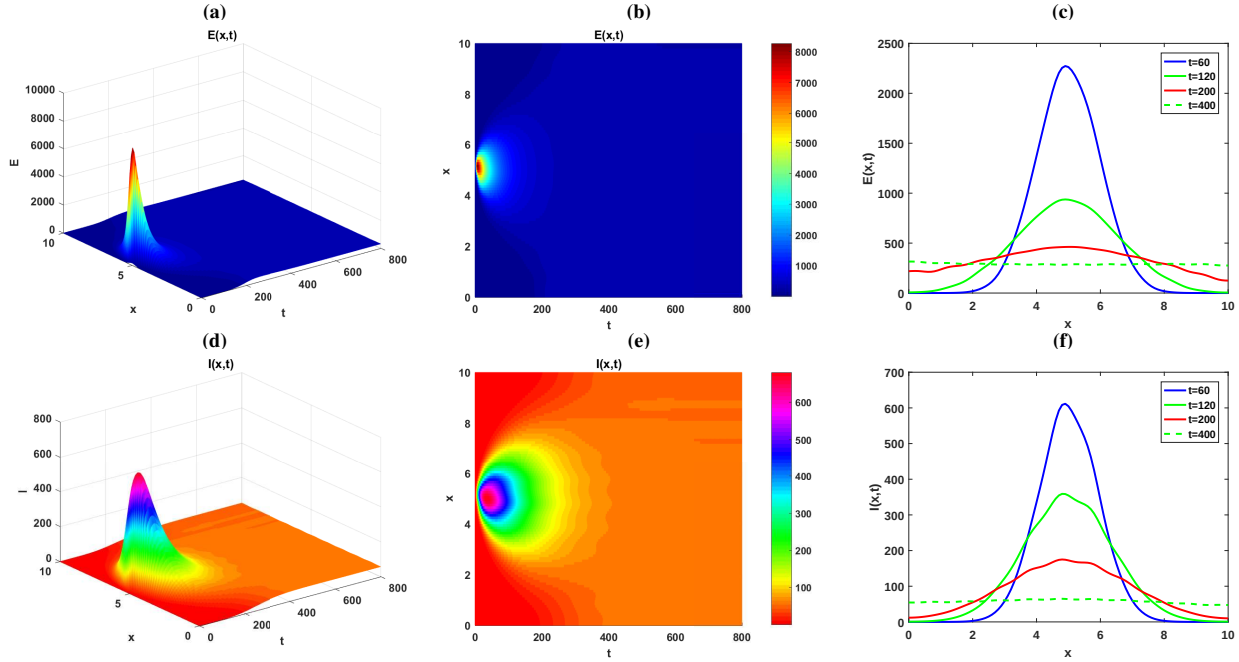

Figure 2. Time evolution of system (1.1) with initial values. (a)–(c): The evolution of the $E(x,t)$ over time; (d)–(f): The evolution of the $I(x,t)$ over time.

Case 3: To support the conclusion of Theorems 5 and 6, we first select the parameters as shown in Figure 3, and keep the diffusion coefficient as shown in Table 2. In this case, we calculate R_0 =

0.4808 < 1. It can be seen from the Figure 3 that the density of exposed individuals $E(x,t)$ and infected individuals $I(x,t)$ tends to zero over time, which indicates Theorem 5. Then we choose $\beta = 3.6\times10^{-3}$, $\beta_1 = 6\times10^{-4}$, $\beta_2 = 6\times10^{-3}$ and the other parameters remain the same as in Figure 3. In this case, we calculate $R_0 = 4.8081 > 1$. As can be seen from the Figure 4, the density of exposed individuals $E(x,t)$ and infected individuals $I(x,t)$ tends to a steady state over time, which indicates Theorem 6.

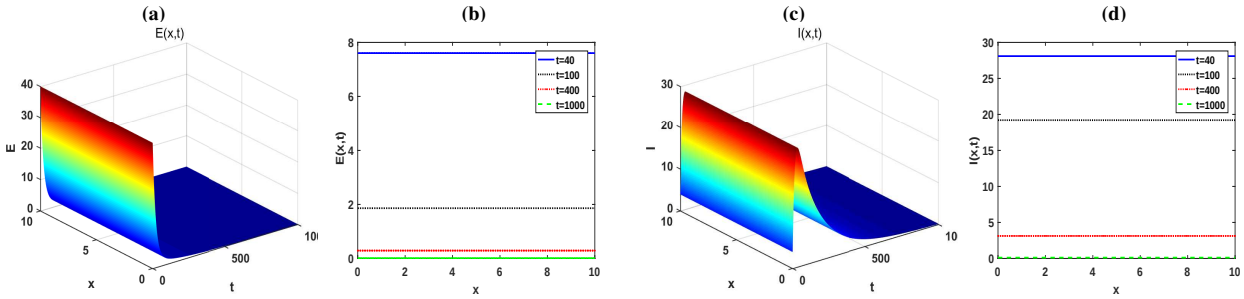

Figure 3. (a)–(d): Evolution of the model (6.1) solutions with time when $R_0 = 0.4808$. $\Lambda = 10000/(2.79\times365)$, $\mu = 0.03$, $\beta = 3.6\times10^{-4}$, $\beta_1 = 6\times10^{-5}$, $\beta_2 = 6\times10^{-4}$, $p = 0.4$, $\alpha = 0.6$, $\eta = 0.1$, $\rho = 0.3$, $\delta = 0.16$, $\sigma = 0.4$, $q_1 = 0.3$, $c_1 = 0.15$. The initial value is $(200, 120, 40, 4, 0)$.

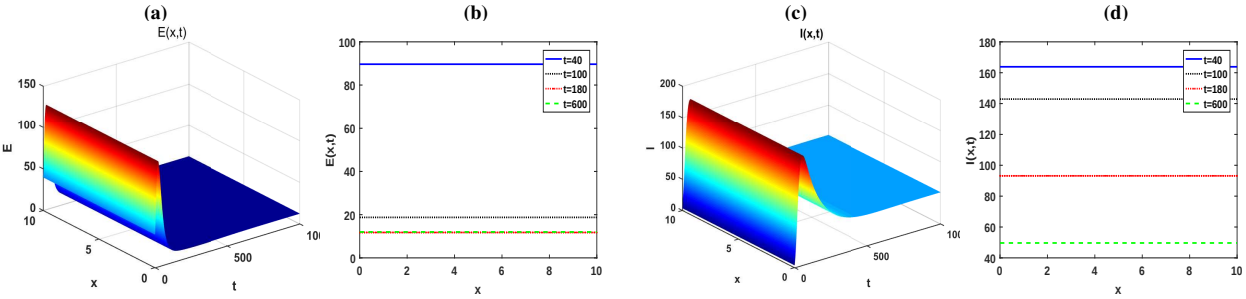

Figure 4. (a)–(d): Evolution of the model (6.1) solutions with time when $R_0 = 4.8081$. $\Lambda = 10000/(2.79\times365)$, $\mu = 0.03$, $\beta = 3.6\times10^{-3}$, $\beta_1 = 6\times10^{-4}$, $\beta_2 = 6\times10^{-3}$, $p = 0.4$, $\alpha = 0.6$, $\eta = 0.1$, $\rho = 0.3$, $\delta = 0.16$, $\sigma = 0.4$, $q_1 = 0.3$, $c_1 = 0.15$. The initial value is $(200, 120, 40, 4, 0)$.

7.2. Impact of spatial heterogeneity on disease transmission

Basic reproduction number is an important index to assess the extinction or persistence of disease, so it is necessary to study the effect of spatial heterogeneity on R_0. First, we choose $\beta(x) = 4\times10^{-4}\times(1 + c\times\cos(2\pi x))$ and $\beta_2(x) = 8.491\times10^{-4}\times(1 + 0.5\times\cos(2\pi x))$ and keep the other parameters as in Table 2 to study the influence of spatial heterogeneity on R_0, where $c \in [0, 1]$ reflects the spatial heterogeneity intensity of the environment. Specifically, when $c = 0$, the space is homogeneous, while as c increases, the spatial heterogeneity of the environment also increases. Second, we choose $\beta(x) = 4\times10^{-4}\times(1+0.5\times\cos(2\pi x))$ and $\beta_2(x) = 8.49\times10^{-4}\times(1+c\times\cos(2\pi x))$ to study the influence of spatial heterogeneity, and the other parameters are the same as in Table 2. It can be seen from Figure 5(a),(b) that with the increase of the intensity of spatial heterogeneity c, the basic reproduction

number R_0 increases. Further, we can see that R_0 is minimum in the case of spatial homogeneity of $c = 0$, and R_0 is greater than 1 when c exceeds the critical value. On the other hand, c_2 is defined as the spatial heterogeneity of the diffusion coefficient, we choose $\beta(x) = 6 \times 10^{-4} \times (1 + 0.5 \times \cos(2\pi x))$, $\beta_2(x) = 9 \times 10^{-4} \times (1 + 0.5 \times \cos(2\pi x))$ and $D_4(x) = 0.07 \times (1 + c_2 \times \cos(2\pi x))$, and other parameters are the same as in Example 2. Figure 5(c) shows that the basic reproduction number R_0 decreases as the spatial heterogeneity c_2 of the diffusion coefficient increases, and R_0 is less than 1 when c_2 passes a critical value. In other words, in the case of spatial heterogeneity, diffusion coefficient of the change can change the final state of the disease.

Further, we study the effect of heterogeneity in treatment rate $\delta(x)$, recurrence rate $\rho(x)$ and disease-related death rate $\eta(x)$ on R_0, because the difference of treatment level in different regions will affect the spread of the disease, and the recurrence of people in different regions will also affect the spread of the disease due to environmental heterogeneity. We use $c_i (i = 3, 4, 5)$ to indicate the level of treatment in the area. Take $\delta(x) = 0.3 \times (1 + c_3 \times \sin(2\pi x))$, $\rho(x) = 0.4 \times (1 + c_4 \times \sin(2\pi x))$ and $\eta(x) = 0.2 \times (1 + c_5 \times \sin(2\pi x))$, where $0 \le c_i \le 1$ $(i = 3, 4, 5)$, and keep the other parameters as in Table 2. In this case, we obtain Figure 5(d)–(f), we get that R_0 is a decreasing function of treatment intensity, and R_0 is an increasing function of relapse intensity. Therefore, a higher treatment rate and a better treatment effect (a lower recurrence rate) can reduce the transmission capacity of the disease. This means that the spread of the disease can be effectively controlled by increasing medical facilities and improving treatment. In addition, we find that the basic reproduction number R_0 is not monotonic for $\eta(x)$. For some reason, R_0 may increase with the enhancement of spatial heterogeneous $\eta(x)$.

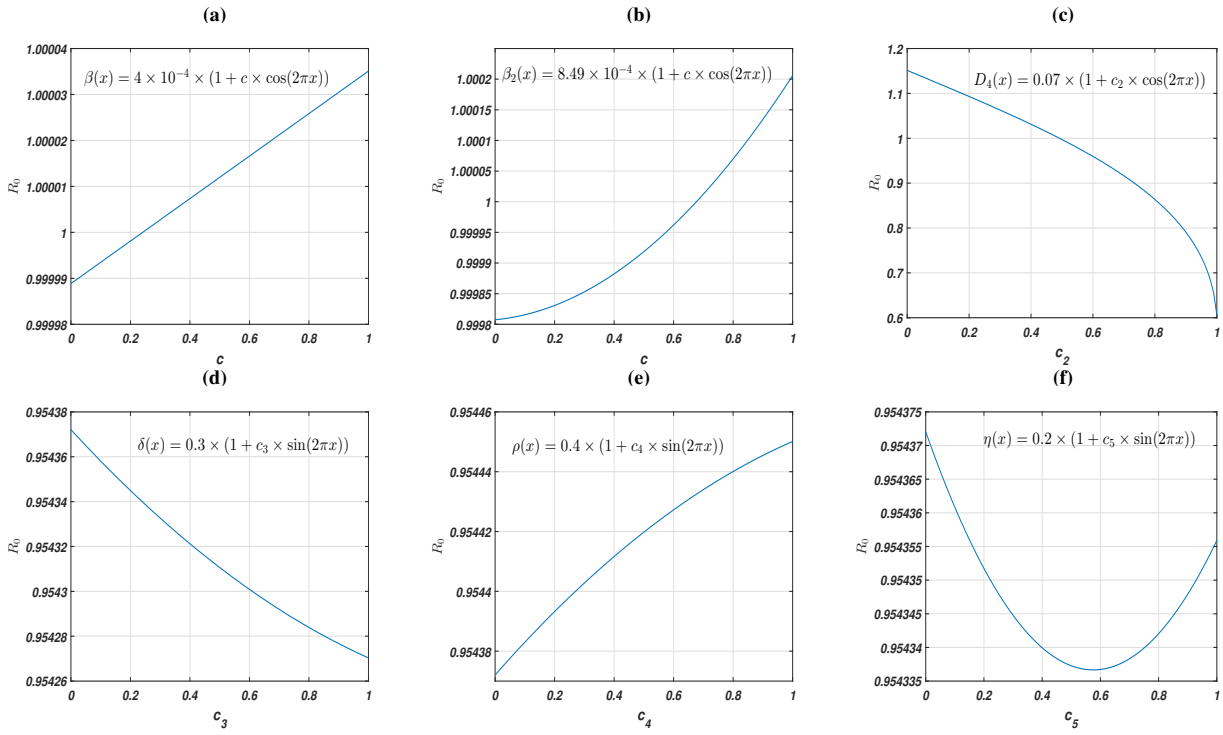

Figure 5. Relationship between R_0 and spatial heterogeneity of parameters.

7.3. *The impact of media coverage on the spread of disease*

In order to study the effect of media coverage on disease transmission, we assume that $c_1 = 0.04$ means that there are extensive media reports, and people have a full understanding of disease information and a strong awareness of prevention, $c_1 = 500$ means there is little media coverage and people are completely unaware of disease information. In the case of spatial heterogeneity, other parameters are taken to be the same as those in Table 2, and the changes of the density of infected individuals over time under different media coverage intensity are obtained when $R_0 < 1$ (see Figure 6), where Figure 6(a),(b) represented strong media coverage and Figure 6(c),(d) represented small media coverage intensity. By comparing Figure 6(b),(d), it can be seen that at the same time t and position x, the stronger the media coverage, the smaller the density of infected individuals $I(x,t)$, which means that in the case of spatial heterogeneity, increasing the intensity of media coverage can reduce the peak value of infected individuals. Similarly, when $R_0 > 1$, the density of infected individuals changes over time under different media coverage intensity (see Figure 7). By comparing Figure 7(b),(d), it can be seen that at the same time t and position x, the stronger the media coverage, the smaller the density of infected individuals $I(x,t)$. This means that in the case of spatial heterogeneity, increasing the intensity of media coverage can reduce the density of infected individuals and reduce the spread of the disease.

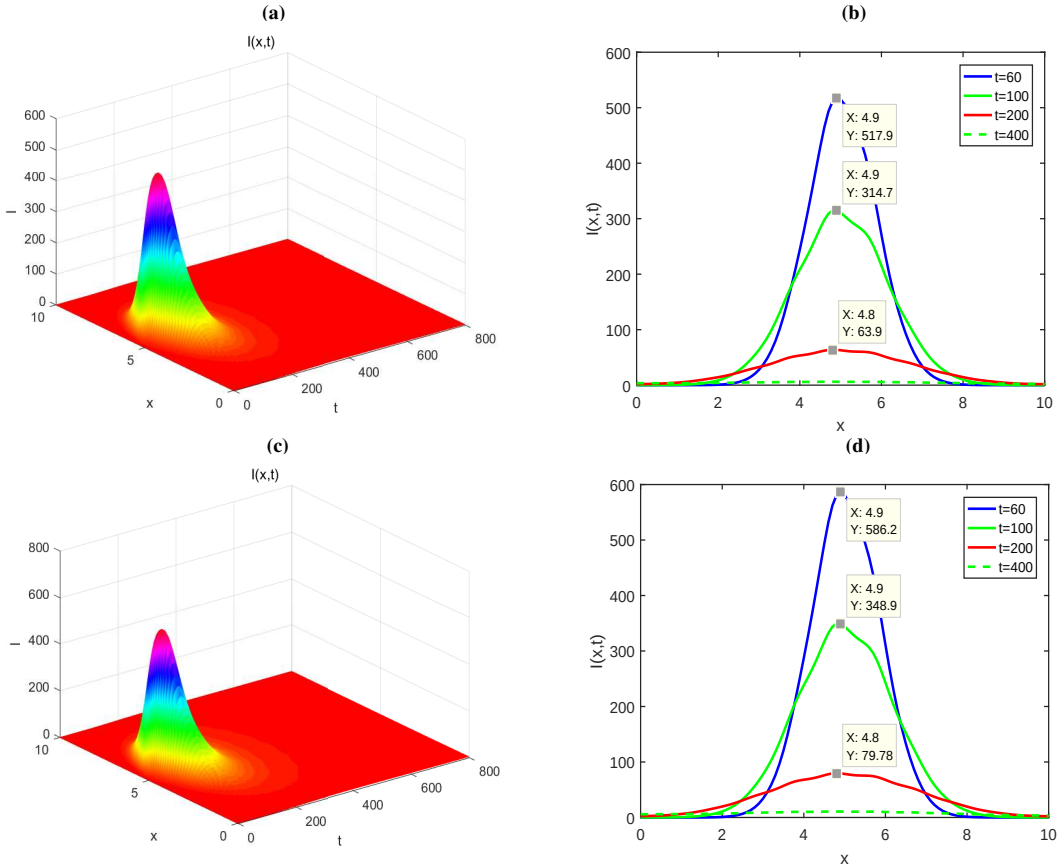

Figure 6. The effect of different levels of media coverage on the number of $I(x,t)$ when $R_0 < 1$. (a)–(b): $c_1 = 0.04$, (c)–(d): $c_1 = 500$.

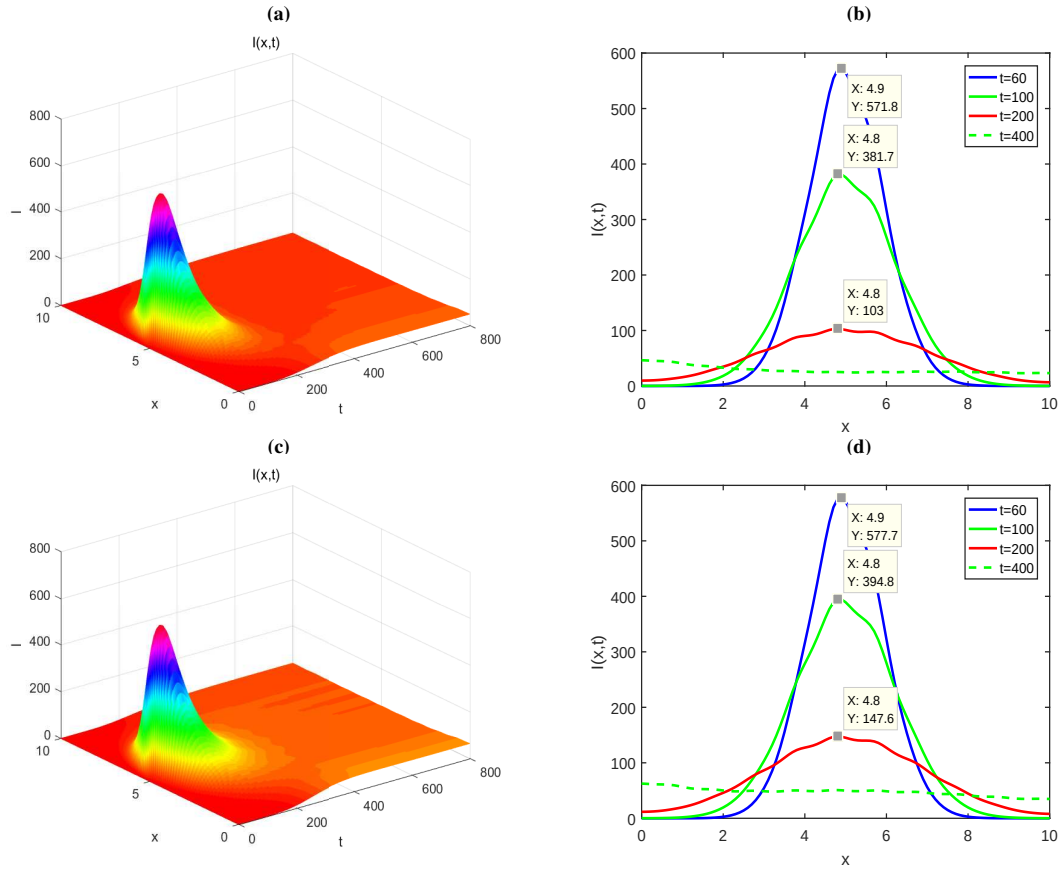

Figure 7. The effect of different levels of media coverage on the number of $I(x,t)$ when $R_0 > 1$. (a)–(b): $c_1 = 0.04$, (c)–(d): $c_1 = 500$.

In the case of spatial homogeneity but heterogeneous diffusion, using the parameters in Figures 3 and 4, we fixed the position x and obtained the curve of the density $I(x,t)$ of infected individuals changing with time t under different media reports. It can be observed from the Figure 8 that when $R_0 < 1$, the number of latent and infected people tends to zero under different media coverage degrees. When c_1 is smaller (and more widely reported in the media), the time to peak the number of infected individuals, the peak size and the time to elimination of the disease are also smaller. This means that a reduction in effective exposure to media coverage can reduce the number of infections and accelerate the disappearance of infectious diseases.

It can be observed from Figure 9 that when $R_0 > 1$, the number of latent and infected people tend to be different stable values under different media coverage levels. When c_1 is smaller, which is more widely reported in the media, the peak and final numbers of the number of latent and infected individuals are also smaller. This means that increased media coverage can control the effective exposure rate of the population and thus reduce the spread of the disease.

8. Conclusions

In this paper, we propose a reaction-diffusion SVEIR-I model with media coverage in spatially heterogeneous environment. First, we analysis the well-posedness of this model. Second, we define

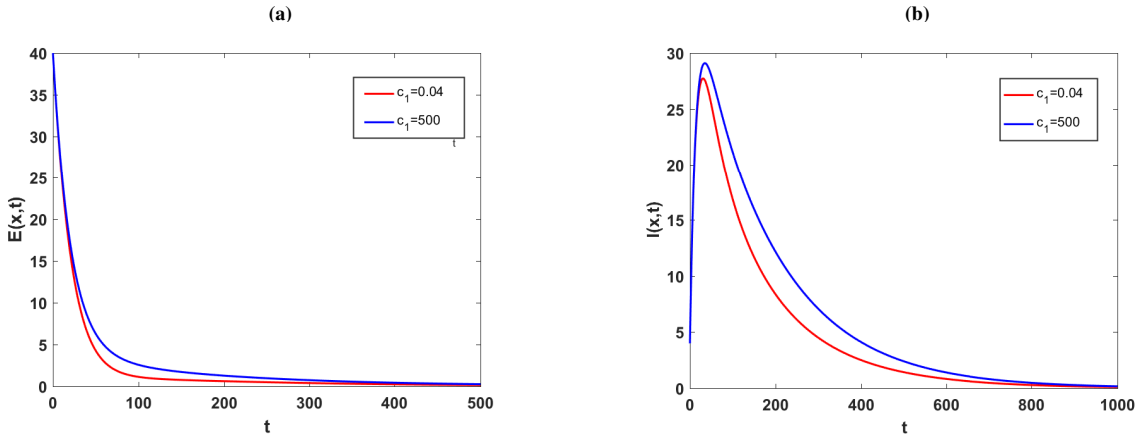

Figure 8. The effect of different levels of media coverage on the number of $E(x,t)$ and $I(x,t)$ when $R_0 < 1$.

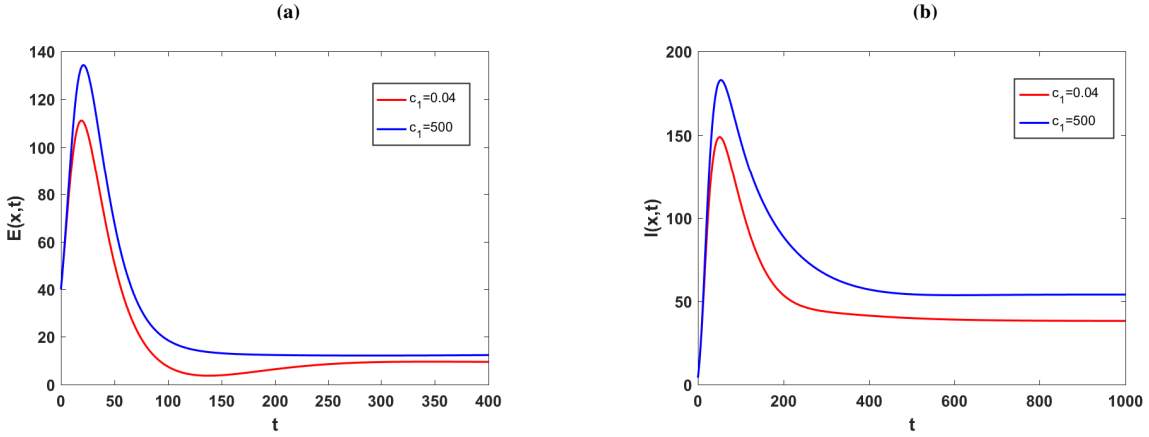

Figure 9. The effect of different levels of media coverage on the number of $E(x,t)$ and $I(x,t)$ when $R_0 > 1$.

and prove that the basic reproduction number R_0 is a threshold parameter that determines the extinction and persistence of the disease in the heterogeneous case. Then, we analyze the dynamic behavior of the model when the space is homogeneous, obtain the existence and uniqueness of the endemic equilibrium, and the global asymptotic stability of the disease-free equilibrium and the endemic equilibrium is proved by establishing an appropriate Lyapunov function. Finally, some numerical simulation examples are given to illustrate our major results.

Numerical simulation results show that spatial heterogeneity will increase the risk of disease transmission. First, we use the infection rate of spatial heterogeneity to study the impact of heterogeneity on R_0. Here, we define c as the heterogeneity strength of the spatial infection rate $\beta(x)$ and $\beta_2(x)$, and find that R_0 will increase with the increase of heterogeneity intensity c, R_0 is the minimum when $c = 0$, and when c exceeds a certain critical value, R_0 is greater than 1, the disease status would change. From a biological perspective, increasing the contact rate between people would make the disease spread faster, which causes disease break out. Second, we study the effect of diffusion coefficient on R_0 under spatial heterogeneity. Here, we define c_2 as the heterogeneity strength of the spatial diffusion coefficient $D_4(x)$. We find that R_0 will decrease with the increase of

heterogeneity intensity c_2, and R_0 reach its maximum when $c_2 = 0$. When c_2 exceed a certain critical value, R_0 is less than 1, and the threshold state of the disease would change. According to the above analysis, we conclude that the final state of the disease may be affected by spatial heterogeneity. In addition, we analyze the effect of the other parameters on R_0, and find that some measures to prevent diseases, such as strengthening the construction of regional medical facilities or improving the medical level, could reduce the spread of diseases to a certain extent. Moreover, we focus on the impact of media coverage on disease transmission, including the case of spatially heterogeneous environments and spatially homogeneous environments. The results show that in both cases, when $R_0 < 1$, strengthening media coverage can shorten the time of the peak of the number of infected individuals, the size of the peak and the time to eliminate the disease, and when $R_0 > 1$, strengthening media coverage can reduce the size of the final scale of infected individuals. Therefore, as a non-pharmaceutical measure to improve information awareness, media coverage plays a very important role in the public response and implementation of disease control.

Use of AI tools declaration

The authors declare they have not used Artificial Intelligence (AI) tools in the creation of this article.

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Conflict of interest

All authors declare no conflicts of interest in this paper.

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