Dynamics in a reaction-diffusion epidemic model via environmental driven infection in heterogenous space

Ning Wang, Long Zhang and Zhidong Teng

College of Mathematics and System Sciences, Xinjiang University, Urumqi, Xinjiang, People’s Republic of China

ABSTRACT

In this paper, a reaction-diffusion SIR epidemic model via environmental driven infection in heterogeneous space is proposed. To reflect the prevention and control measures of disease in allusion to the susceptible in the model, the nonlinear incidence function $E_f(S)$ is applied to describe the protective measures of susceptible. In the general spatially heterogeneous case of the model, the well-posedness of solutions is obtained. The basic reproduction number $R_0$ is calculated. When $R_0 \leq 1$ the global asymptotical stability of the disease-free equilibrium is obtained, while when $R_0 > 1$ the model is uniformly persistent. Furthermore, in the spatially homogeneous case of the model, when $R_0 > 1$ the global asymptotic stability of the endemic equilibrium is obtained. Lastly, the numerical examples are enrolled to verify the open problems.

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1. Introduction

The epidemic has always been the natural enemy of human health. Especially in the past 20 years, due to the deterioration of the environment, humans are facing many epidemics caused by environmental problems, such as Acute infectious gastroenteritis by norovirus, Cholera, Hand-foot-mouth disease (see [10, 19, 39]). These epidemics are characterized by strong infectivity, rapid transmission, wide epidemic range, etc.

In recent years, due to serious damage to the ecological environment, water and food have been polluted to varying degrees (see [16, 20]). In addition, the lack of attention to the hygiene of the dining environment increases the chance of people contracting diseases from the environment (see [9]). It can be seen that environmental change is one of the important factor affecting the occurrence and spread of the epidemic. Therefore, many researchers have considered different types of environmentally driven epidemic models (see, for example [8, 12–14, 29]). Particularly, Chen et al. [8] proposed a wild animals-environment-human transmission epidemic model. Feng et al. [13] established a mathematical model of an epidemic with the environment as the transmission medium.

With the progress of science and technology, transportation is increasingly developed. Although convenient transportation can bring great convenience to people’s travel, it also makes the epidemic to spread to other areas quickly. After analysing the number of patients
with a disease in different cities, many researchers found that the number of patients was closely related to the mobility of population, and proposed different types of reaction-diffusion epidemic models on this phenomenon (see, for example [5, 23, 25, 44, 47, 48]). Especially, Yang et al. [48] studied a seasonal brucellosis SIV epidemic model with nonlocal transmissions and spatial diffusions. Bai et al. [5] considered a reaction-diffusion malaria model with seasonality and incubation period.

Take acute infectious gastroenteritis by norovirus as an example. The diffusion of the virus not only makes the virus exist in the air around the infected but also makes the virus adhere to the surface of public objects. In addition, the movement of infected people will lead to the spread of the virus in a wider range. Susceptible people are easily infected with the disease after inhaling polluted air or touching the surface of objects attached to the virus. To block the transmission of human to human, some effective measures were proposed, such as wearing masks, isolation, and treatment of the infected (see [37]). However, poor hygiene habits lead to the virus through faecal-oral transmission or contact with the secretions of infected people and other ways to infect susceptible people, such as long-term non-washing hands and sharing tableware, and so on (see [1, 4, 34]). Therefore, when the transmission route of human to human is hindered, the environment-driven infection will gradually become the only transmission route of the epidemic. Pathogens are mainly released by the infected, especially in crowded and closed environments, where the content of the pathogen is high. Furthermore, the drifting behaviour of environmental pathogens is a fact and must be considered (see, for example [22]). Similar to Acute infectious gastroenteritis by norovirus, epidemics mainly transmitted by environmental viruses, germs, and pests include Brucellosis, schistosomiasis, and so on (see [7, 27, 47]).

Based on the above discussion, we propose the following reaction-diffusion SIR epidemic model via environmental driven infection in heterogenous space:

\[
\begin{align*}
\frac{\partial}{\partial t} S(t, x) &= \nabla \cdot D_1(x) \nabla S(t, x) + A(x) - \beta(x) Ef(S) - \mu(x)S, \\
\frac{\partial}{\partial t} I(t, x) &= \nabla \cdot D_2(x) \nabla I(t, x) + \beta(x) Ef(S) - (\mu(x) + \alpha(x) + \zeta(x))I, \\
\frac{\partial}{\partial t} E(t, x) &= \nabla \cdot D_3(x) \nabla E(t, x) + \theta(x) I(1 - E) - (\xi(x) + \gamma(x))E, \\
\frac{\partial}{\partial t} R(t, x) &= \nabla \cdot D_4(x) \nabla R(t, x) + \zeta(x)I - \mu(x)R,
\end{align*}
\]

where \( t \geq 0, x \in \Omega, \Omega \subset \mathbb{R}^n \) is a bounded domain with the smooth boundary \( \partial \Omega \), \( x = (x_1, x_2, \ldots, x_n) \) and \( n \geq 1 \) is an integer. \( S(t, x), I(t, x), R(t, x) \) and \( E(t, x) \) are defined as the numbers of susceptible, infectious and removed individuals, and the concentration of environmental pathogens (virus or bacteria) at time \( t \) and spatial location \( x \), respectively. \( \nabla = \left( \frac{\partial}{\partial x_1}, \frac{\partial}{\partial x_2}, \ldots, \frac{\partial}{\partial x_n} \right) \) denotes the gradient operator. \( A(x) \) refers to the supplement rate of susceptible persons. \( \beta(x) \) denotes the infection rate of the susceptible in a polluted environment. \( \mu(x) \) denotes the natural mortality rate of the total population. \( \theta(x) \) represents the rate of infectious individuals release pathogens to the environment, and \( \theta(x) I(1 - E) \) denotes the increment of concentration of environmental pathogens per unit time. \( \gamma(x) \) denotes the artificial clearance rate of the pathogen in the environment. \( \xi(x) \) denotes the natural mortality of the pathogen in the environment. \( \alpha(x) \) denotes the induced mortality rate of the total population.
of infectious individuals. \( \zeta(x) \) refers to the isolation and therapy rate of infectious individuals. \( \mathcal{E}(S) \) denotes the protective measures of susceptible, such as home isolation, wearing protective masks, vaccination, etc. The drifting behavior of pathogens in the environment and the flow phenomenon of susceptible people, infected patients, and the removed are represented by the reaction-diffusion term in the model, where \( D_i(x) \) \((i = 1, 2, 3, 4)\) denote the spatially diffusive rate of the susceptible, infected, pathogen in the environment, and the removed, respectively.

We have noticed that epidemic models with indirect infection caused by environmental pathogens have been considered in many articles, see for example [6, 45]. In these models, we can see that environmental pathogens are measured in terms of quantity or density. However, in model (1), different from the above ways, environmental pathogens are measured by the concentration in the environment. Thus, we introduce the term \( \theta(x)I(1 - E) \) into the dynamic equation of the pathogen, where factor \((1 - E)\) is used to guarantee the inequality \(0 \leq E(t, x) \leq 1\). Since pathogens are extremely tiny as microorganisms, we think that it is more reasonable to use the concentration of environmental pathogens to describe the magnitude of pathogens than to use the number or density directly.

The main content of this paper is to study the dynamic behavior of model (1) and to explore the effects of prevention and control measures on the spread of the disease. The threshold criteria on the global stability of disease-free and endemic equilibria and the uniform persistence of solutions are established. It can be observed that the elimination and prevalence of the epidemic are determined by the basic reproduction number. The model proposed in this paper can more reasonably characterize the spread of epidemics driven by the environment and the impact of prevention and control measures on the spread of the epidemic.

This paper is organized as follows. In Section 2, the well-posedness of solutions is discussed. In Section 3, the basic reproduction number \( R_0 \) is calculated. In Section 4, the global stability of the disease-free equilibrium is obtained. In Section 5, the uniform persistence of solutions is proved. In Section 6, the global asymptotic stability of the endemic equilibrium is obtained in the homogeneous space. In Section 7, the open problems proposed in this paper are verified by the numerical simulations. Lastly, we draw a concise conclusion in Section 8.

2. Well-posedness of solutions

Since the removed \( R(t, x) \) does not appear in the first three equations of model (1), we here only need to investigate the following submodel

\[
\begin{aligned}
\frac{\partial}{\partial t} S(t, x) &= \nabla \cdot D_1(x) \nabla S(t, x) + A(x) - \beta(x) \mathcal{E}(S) - \mu(x) S, \\
\frac{\partial}{\partial t} I(t, x) &= \nabla \cdot D_2(x) \nabla I(t, x) + \beta(x) \mathcal{E}(S) - (\mu(x) + \alpha(x) + \zeta(x)) I, \\
\frac{\partial}{\partial t} E(t, x) &= \nabla \cdot D_3(x) \nabla E(t, x) + \theta(x) I(1 - E) - (\xi(x) + \gamma(x)) E.
\end{aligned}
\] (2)

In this section, we discuss the well-posedness of solutions of model (2). It is assumed that any solution \((S(t, x), I(t, x), E(t, x))\) of model (2) satisfies the following initial condition:

\[
S(0, x) = \phi_1(x), \quad I(0, x) = \phi_2(x), \quad E(0, x) = \phi_3(x), \quad x \in \Omega
\] (3)
and homogeneous Neumann boundary condition:
\[
\frac{\partial}{\partial n} S(t, x) = \frac{\partial}{\partial n} I(t, x) = \frac{\partial}{\partial n} E(t, x) = 0, \quad t \geq 0, \ x \in \partial \Omega, \quad (4)
\]
where \( \phi_i(x) (i = 1, 2, 3) \) are the nonnegative Hölder continuous bounded functions defined on \( \overline{\Omega} \) with \( 0 \leq \phi_3(x) \leq 1 \) for all \( x \in \overline{\Omega} \), and \( \frac{\partial}{\partial n} \) represents the outward normal derivative on \( \partial \Omega \). Since \( E(t, x) \) represents the concentration of environmental pathogens in model (1), it should be required to vary within the interval \([0, 1]\). Therefore, we here assume the initial function \( \phi_3(x) \in [0, 1] \) for all \( x \in \overline{\Omega} \).

For any bounded function \( q(x) \) defined on the set \( B \subset \mathbb{R}^n \), we denote \( q_o = \sup_{x \in B} q(x) \) and \( q_i = \inf_{x \in B} q(x) \).

For model (1), we always introduce the following assumptions.

\( (H_1) \) \( A(x), \beta(x), \mu(x), \alpha(x), \xi(x), \vartheta(x), \xi(x), \gamma(x) \) and \( D_i(x) \) (\( i = 1, 2, 3 \)) are bounded, continuous and positive functions for \( x \in \overline{\Omega} \).

\( (H_2) \) \( f(S) \) is nonnegative and continuously differentiable on \([0, \infty) \), \( f(0) = 0 \), and \( f'(S) \geq 0 \) for all \( S \geq 0 \).

Remark 1: There are many functions \( f(S) \) satisfying assumption \( (H_2) \). For example, \( f(S) = S \) and \( f(S) = \frac{S}{1 + \omega S} \), where \( \omega \) is a positive constant.

Denote by \( Y = C(\overline{\Omega}, R) \) the Banach space of all continuous functions \( \phi : \overline{\Omega} \to R \) with the supremum norm \( \| \phi \| = \sup_{x \in \overline{\Omega}} | \phi(x) | \). Let \( Y_+ = C(\overline{\Omega}, R_+) \) be the positive cone of \( Y \). Then \( (Y, Y_+) \) represents an ordered Banach space. Additionally, denote \( X = Y \times Y \times Y \) with the norm \( \| \phi \|_X = \max\{\| \phi_1 \|_Y, \| \phi_2 \|_Y, \| \phi_3 \|_Y \} \), where \( \phi = (\phi_1, \phi_2, \phi_3) \in X \), and \( \phi_i \in Y \) \( (i = 1, 2, 3) \). Let \( X_+ = Y_+ \times Y_+ \times Y_+ \) be the positive cone of \( X \).

Firstly, the following scalar reaction-diffusion model is considered:
\[
\begin{align*}
\frac{\partial}{\partial t} u(t, x) &= \nabla \cdot D(x) \nabla u(t, x) + \zeta(x) - \kappa(x) u(t, x), \quad x \in \Omega, \\
\frac{\partial}{\partial n} u(t, x) &= 0, \quad x \in \partial \Omega,
\end{align*}
\]
where \( D(x), \zeta(x) \) and \( \kappa(x) \) are continuous, bounded and positive functions for \( x \in \overline{\Omega} \). According to Lemma 1 in [25], we establish the following conclusion.

Lemma 2.1: Model (5) admits a unique positive equilibrium \( u_0(x) \) satisfying the equation
\[
\nabla \cdot D(x) \nabla u_0(x) + \zeta(x) - \kappa(x) u_0(x) = 0, \quad x \in \Omega
\]
with \( \frac{\partial}{\partial n} u_0(x) = 0 \) for \( x \in \partial \Omega \) which is globally asymptotically stable in \( C(\overline{\Omega}, R_+) \). In addition, if \( \zeta(x) \equiv \zeta \) and \( \kappa(x) \equiv \kappa \) are positive constants, then \( u_0(x) = \frac{\zeta}{\kappa} \).

We denote by \( T_k(t) : C(\overline{\Omega}, R) \to C(\overline{\Omega}, R) \) for \( k = 1, 2, 3 \) the \( C_0 \)-semigroup associated with \( \nabla \cdot D_k(x) \nabla - g_k(x) \) subjects to the Neumann boundary condition, where \( g_1(x) = \mu(x), g_2(x) = \mu(x) + \alpha(x) + \zeta(x) \) and \( g_3(x) = \xi(x) + \gamma(x) \), respectively.
Denote
\[
\begin{align*}
F_1(\phi)(x) &= A(x) - \beta(x)\phi_3(x)f(\phi_1(x)), \\
F_2(\phi)(x) &= \beta(x)\phi_3(x)f(\phi_1(x)), \quad x \in \Omega, \\
F_3(\phi)(x) &= \theta(x)\phi_2(x)(1 - \phi_3(x)),
\end{align*}
\]
where \( \phi = (\phi_1, \phi_2, \phi_3) \in X_+. \) We define \( u(t, \cdot , \phi) = (S(t, \cdot , \phi), I(t, \cdot , \phi), E(t, \cdot , \phi)) \) the solution of model (2) with initial value function \( \phi = (\phi_1, \phi_2, \phi_3) \in X_+ \), and then model (2) can be rewritten as the following integral equations:
\[
\begin{align*}
S(t, \cdot , \phi) &= T_1(t)\phi_1 + \int_0^t T_1(t - s)F_1(S(s, \cdot , \phi_1))ds, \\
I(t, \cdot , \phi) &= T_2(t)\phi_2 + \int_0^t T_2(t - s)F_2(I(s, \cdot , \phi_2))ds, \quad t > 0, \\
E(t, \cdot , \phi) &= T_3(t)\phi_3 + \int_0^t T_3(t - s)F_3(E(s, \cdot , \phi_3))ds.
\end{align*}
\]

On the existence and the ultimate boundedness of global solutions for model (2), the following result is established.

**Theorem 2.2:** For any initial function \( \phi = (\phi_1, \phi_2, \phi_3) \in X_+ \) with \( 0 \leq \phi_3 \leq 1 \), model (2) with initial conditions (3) has a unique nonnegative and ultimately bounded solution \( u(t, \cdot , \phi) = (S(t, \cdot , \phi), I(t, \cdot , \phi), E(t, \cdot , \phi)) \) defined on \([0, \infty) \times \Omega\). Furthermore, \( 0 \leq E(t, \cdot , \phi) \leq 1 \) for all \( t \geq 0 \) and \( x \in \Omega \).

**Proof:** A similar argument as in Lemma 1 in [30] (or see Lemma 2.1 in [24]), we easily verify that model (2) satisfies the following condition
\[
\lim_{h \to 0^+} \text{dist}(\phi + hF(\phi), X_+) = 0 \quad \text{for all} \quad \phi \in X_+,
\]
where \( F = (F_1, F_2, F_3) \). Therefore, according to Corollary 4 in [35], for any initial function \( \phi = (\phi_1, \phi_2, \phi_3) \in X_+ \) with \( 0 \leq \phi_3 \leq 1 \), model (2) has a unique nonnegative mild solution \( u(t, \cdot , \phi) = (S(t, \cdot , \phi), I(t, \cdot , \phi), E(t, \cdot , \phi)) \in X_+ \) on the interval of existence \([0, \tau_\infty)\) and \( \tau_\infty \leq \infty \). Furthermore, this solution is also a classical solution. Suppose that \( \tau_\infty < \infty \), then according to Theorem 2.4 in [35], we get \( \|u(t, \cdot , \phi)\|_X \to \infty \) as \( t \to \tau_\infty \).

From the first equation of model (2), we obtain
\[
\frac{\partial}{\partial t} S(t, \cdot , \phi) \leq \nabla \cdot D_1(x)\nabla S(t, \cdot , \phi) + A_\xi S(t, \cdot , \phi), \quad t \in [0, \tau_\infty).
\]
Using the comparison principle and Lemma 2.1, it follows that there exists a constant \( P_1 > 0 \) such that \( S(t, \cdot , \phi) \leq P_1 \) for all \( t \in [0, \tau_\infty) \) and \( x \in \Omega \).

Consider the third equation of model (2). Let \( g(E) = \theta(x)I(1 - E) - (\xi(x) + \gamma(x))E \). Since \( g(0) = \theta(x)I \geq 0 \) and \( g(1) = - (\xi(x) + \gamma(x)) < 0 \), we get that \( E = 1 \) and \( E = 0 \) are the upper and lower solutions of the third equation of model (2), respectively (see Definition 2.4.3 in [46]). Therefore, it is known from Theorem 2.4.6 in [46] that for the solution \( u(t, \cdot , \phi) = (S(t, \cdot , \phi), I(t, \cdot , \phi), E(t, \cdot , \phi)) \), as long as initial value \( 0 \leq \phi_3 \leq 1 \), then there is \( 0 \leq E(t, \cdot , \phi) \leq 1 \) for all \( t \in [0, \tau_\infty) \) and \( x \in \Omega \).
From the second equation of model (2) and assumption (H2), we obtain

$$\frac{\partial}{\partial t} I(t, \cdot, \phi) \leq \nabla \cdot D_2(x) \nabla I(t, \cdot, \phi) + \beta_i f(P_i) - (\mu_i + \alpha_i + \zeta_i) I(t, \cdot, \phi), \quad t \in [0, \tau_\infty).$$

Again using Lemma 2.1 and the comparison principle, there exists a constant $P_2 > 0$ such that $I(t, \cdot, \phi) \leq P_2$ for all $t \in [0, \tau_\infty)$ and $x \in \overline{\Omega}$. This leads to a contradiction with $\|u(t, \cdot, \phi)\|_\chi \to \infty$ as $t \to \tau_\infty$. Therefore, $\tau_\infty = \infty$, and the global existence of solution $u(t, \cdot, \phi)$ is acquired.

Now, we prove that the solution also is ultimately bounded. In fact, from Lemma 2.1 and inequality (7), we get $\limsup_{t \to \infty} S(t, \cdot, \phi) \leq \frac{\lambda_2}{\mu_i}$ uniformly for $x \in \overline{\Omega}$. This means that $S(t, \cdot, \phi)$ is ultimately bounded. For any constant $\vartheta > 0$ there is a $t_1 > 0$ such that $S(t, \cdot, \phi) < \frac{\lambda_2}{\mu_i} + \vartheta$ for all $t \geq t_1$ and $x \in \overline{\Omega}$, and then from the second equation of model (2) we have

$$\frac{\partial}{\partial t} I(t, \cdot, \phi) \leq \nabla \cdot D_2(x) \nabla I(t, \cdot, \phi) + \beta_i f \left( \frac{A_i}{\mu_i} + \vartheta \right) - (\mu_i + \alpha_i + \zeta_i) I(t, \cdot, \phi), \quad t \geq t_1.$$

Hence, the comparison theorem and Lemma 2.1 imply that

$$\limsup_{t \to \infty} I(t, \cdot, \phi) \leq \frac{\beta_i f \left( \frac{A_i}{\mu_i} + \vartheta \right)}{\mu_i + \alpha_i + \zeta_i} \text{ uniformly for } x \in \overline{\Omega}.$$

Thus, $I(t, \cdot, \phi)$ is also ultimately bounded. This completes the proof.

**Remark 2:** We denote $X^*_+ = \{ \phi = (\phi_1, \phi_2, \phi_3) \in X_+, 0 \leq \phi_3 \leq 1 \}$. Based on Theorem 3.4.8 in [21] and Theorem 2.2, we get that all nonnegative solutions $u(t, \cdot, \phi) = (S(t, \cdot, \phi), I(t, \cdot, \phi), E(t, \cdot, \phi))$ of model (2) with $\phi \in X^*_+$ generate a solution semiflow $\Lambda(t) : X^*_+ \to X^*_+$ with $\Lambda(t)\phi = u(t, \cdot, \phi)$ for all $t \geq 0$.

### 3. The basic reproduction number

In model (2), when $(I(t, x), E(t, x)) \equiv (0, 0)$ we get the following scalar reaction-diffusion equation

$$\begin{cases}
\frac{\partial}{\partial t} S(t, x) = \nabla \cdot D_1(x) \nabla S(t, x) + A(x) - \mu(x) S(t, x), & x \in \Omega, \\
\frac{\partial}{\partial n} S(t, x) = 0, & x \in \partial \Omega.
\end{cases}$$

From Lemma 2.1, it has a positive and globally asymptotically stable equilibrium $S_0(x)$. Thus, model (2) has disease-free equilibrium $W_0(x) = (S_0(x), 0, 0)$. Now, we calculate the basic reproduction number of model (2).
Linearizing model (2) at equilibrium \( W_0(x) \), we obtain the following linear system

\[
\begin{align*}
\frac{\partial}{\partial t} U_1(t, x) &= \nabla \cdot D_1(x) \nabla U_1(t, x) - \beta(x) U_3 f(S_0(x)) - \mu(x) U_1, \\
\frac{\partial}{\partial t} U_2(t, x) &= \nabla \cdot D_2(x) \nabla U_2(t, x) + \beta(x) U_3 f(S_0(x)) - (\mu(x) + \alpha(x) + \xi(x)) U_2, \\
\frac{\partial}{\partial t} U_3(t, x) &= \nabla \cdot D_3(x) \nabla U_3(t, x) + \theta(x) U_2 - (\xi(x) + \gamma(x)) U_3, \\
\frac{\partial}{\partial n} U_1(t, x) &= \nabla \cdot D_4(x) \nabla U_1(t, x) = 0, \quad x \in \partial \Omega. \\
\frac{\partial}{\partial n} U_2(t, x) &= \nabla \cdot D_5(x) \nabla U_2(t, x) = 0, \quad x \in \partial \Omega.
\end{align*}
\] (10)

We only need to investigate the following subsystem of system (10), because \( U_1(t, x) \) does not exist in the last two equations.

\[
\begin{align*}
\frac{\partial}{\partial t} U_2(t, x) &= \nabla \cdot D_2(x) \nabla U_2(t, x) + \beta(x) U_3 f(S_0(x)) - (\mu(x) + \alpha(x) + \xi(x)) U_2, \\
\frac{\partial}{\partial t} U_3(t, x) &= \nabla \cdot D_3(x) \nabla U_3(t, x) + \theta(x) U_2 - (\xi(x) + \gamma(x)) U_3, \\
\frac{\partial}{\partial n} U_2(t, x) &= \nabla \cdot D_5(x) \nabla U_2(t, x) = 0, \quad x \in \partial \Omega.
\end{align*}
\] (11)

In order to acquire the basic reproduction number of model (2), we denote

\[
F(x) = \begin{pmatrix} 0 & \beta(x) f(S_0(x)) \\ 0 & 0 \end{pmatrix},
\] (12)

\[
K(x) = \begin{pmatrix} \nabla \cdot D_2(x) \nabla - (\mu(x) + \alpha(x) + \xi(x)) & 0 \\ \theta(x) & \nabla \cdot D_3(x) \nabla - \xi(x) - \gamma(x) \end{pmatrix}.
\] (13)

That is, we have the next generation operator \( L := -F(x)K^{-1}(x) \).

Define \( T(t) : C(\overline{\Omega}, R^2) \rightarrow C(\overline{\Omega}, R^2) \) as the solution semigroup that is generated by the following equation

\[
\begin{align*}
\frac{\partial}{\partial t} M(t, x) &= K(x) M(t, x), \quad x \in \Omega, \\
\frac{\partial}{\partial n} M(t, x) &= 0, \quad x \in \partial \Omega,
\end{align*}
\]

where \( M(t, x) = (M_1(t, x), M_2(t, x))^T \). It is assumed that the disease is introduced at time \( t = 0 \) and the distribution of initial infected individuals and initial concentration of environmental pathogen is described as \( \phi(x) = (\phi_2(x), \phi_3(x))^T \). Hence, as time evolves, the distribution of new infected individuals and the concentration of environmental pathogens becomes \( F(x) T(t) \phi(x) \) at time \( t \). Thus, \( \int_0^{+\infty} F(x) T(t) \phi(x) \) denotes the total distribution of the concentration of environmental pathogens and new infected individuals.

We see that operator \( L = -F(x)K^{-1}(x) \) can be specifically defined by

\[
L(\phi)(x) = \int_0^{+\infty} F(x) T(t) \phi(x) dt = F(x) \int_0^{+\infty} T(t) \phi(x) dt.
\]

Clearly, \( L \) is a positive and continuous operator and maps the initial infection distribution \( \phi(x) \) to the distribution of the total infective individuals produced during the infection.
where, according to \[44\], we define the disease transmission basic reproduction number \( R_0 \) of model (2) by the spectral radius of \( L \). That is, \( R_0 = r(L) \).

By calculating, we obtain

\[
K^{-1}(x) = \begin{pmatrix}
(\nabla \cdot D_2(x)\nabla - a_1(x))^{-1} & 0 \\
-k_{21}(x) & (\nabla \cdot D_3(x)\nabla - a_2(x))^{-1}
\end{pmatrix},
\]

where \( k_{21}(x) = (\nabla \cdot D_3(x)\nabla - a_2(x))^{-1}\theta(x)(\nabla \cdot D_2(x)\nabla - a_1(x))^{-1} \). From the expressions of \( F(x) \) and \( K^{-1}(x) \), we obtain

\[
F(x)K^{-1}(x) = \begin{pmatrix}
-\beta(x)f(S_0(x))k_{21}(x) & \beta(x)f(S_0(x))(\nabla \cdot D_3(x)\nabla - a_2(x))^{-1}
\end{pmatrix},
\]

where \( a_1(x) = \mu(x) + \alpha(x) + \xi(x) \) and \( a_2(x) = \xi(x) + \gamma(x) \). Thus, we have

\[
R_0 = r(L) = r(\beta(x)f(S_0(x))k_{21}(x)).
\]

That is, \( R_0 \) is the spectral radius of operator \( \beta(x)f(S_0(x))k_{21}(x) \). This implies that \( R_0 \) is the principal eigenvalue of the following eigenvalue problem.

\[
\begin{cases}
\beta(x)f(S_0(x))k_{21}(x)\phi = \lambda\phi, \quad x \in \Omega, \\
\frac{\partial\phi}{\partial n}(x) = 0, \quad x \in \partial\Omega.
\end{cases}
\]

Therefore, there is a strictly positive eigenfunction \( \phi_* \) satisfying \( \int_\Omega \phi_*^2\,dx = 1 \) such that

\[
\beta(x)f(S_0(x))k_{21}(x)\phi_* = R_0\phi_*, \quad x \in \Omega
\]

and \( \frac{\partial\phi_*}{\partial n}(x) = 0 \) for \( x \in \partial\Omega \). Then, we obtain

\[
\beta(x)f(S_0(x))(\nabla \cdot D_3(x)\nabla - a_2(x))^{-1}\theta(x)\phi_* = R_0(\nabla \cdot D_2(x)\nabla - a_1(x))\phi_*, \quad x \in \Omega
\]

and \( \frac{\partial\phi_*}{\partial n}(x) = 0 \) for \( x \in \partial\Omega \). Hence, we have

\[
R_0 = -\frac{\int_\Omega \beta(x)f(S_0(x))(\nabla \cdot D_3(x)\nabla - a_2(x))^{-1}\theta(x)\phi_*^2\,dx}{\int_\Omega (D_2(x)|\nabla\phi_*|^2 + a_1(x)\phi_*^2)\,dx}, \quad \int_\Omega \phi_*^2\,dx = 1.
\]

Furthermore, we consider the equation

\[
\begin{cases}
-\beta(x)f(S_0(x))(\nabla \cdot D_3(x)\nabla - a_2(x))^{-1}\theta(x)\phi_* = \lambda^*\phi_*, \quad x \in \Omega, \\
\frac{\partial\phi_*}{\partial n}(x) = 0, \quad x \in \partial\Omega,
\end{cases}
\]

where \( \lambda^* \) is a constant. Since \( \int_\Omega \phi_*^2\,dx = 1 \), we obtain

\[
\lambda^* = -\int_\Omega \beta(x)f(S_0(x))(\nabla \cdot D_3(x)\nabla - a_2(x))^{-1}\theta(x)\phi_*^2\,dx.
\]

From (15), we further obtain

\[
\begin{cases}
-\beta(x)f(S_0(x))\theta(x)\phi_* = \lambda^*(\nabla \cdot D_3(x)\nabla - a_2(x))\phi_*, \quad x \in \Omega, \\
\frac{\partial\phi_*}{\partial n}(x) = 0, \quad x \in \partial\Omega.
\end{cases}
\]
Then, it follows that
\[
\lambda^* = \frac{\int_{\Omega} \beta(x)f(S_0(x))\theta(x)\phi^2 \, dx}{\int_{\Omega} (D_3(x)|\nabla \phi|^2 + \gamma(x)\phi^2 + \xi(x)\phi^2) \, dx}, \quad \int_{\Omega} \phi^2 \, dx = 1.
\]
Therefore, we finally have
\[
R_0 = \frac{\int_{\Omega} \beta(x)\theta(x)f(S_0(x))\phi^2 \, dx}{\int_{\Omega} (D_3(x)|\nabla \phi|^2 + a_2(x)\phi^2) \, dx \int_{\Omega} (D_2(x)|\nabla \phi|^2 + a_1(x)\phi^2) \, dx}, \quad \int_{\Omega} \phi^2 \, dx = 1.
\]
This shows that
\[
R_0 = \sup_{\phi \in H^1(\Omega), \phi \neq 0} \left\{ \frac{\int_{\Omega} \beta(x)\theta(x)f(S_0)\phi^2 \, dx}{\int_{\Omega} (D_3(x)|\nabla \phi|^2 + a_2(x)\phi^2) \, dx \int_{\Omega} (D_2(x)|\nabla \phi|^2 + a_1(x)\phi^2) \, dx} \right\}.
\]
Substituting \(U_2(t, x) = e^{\lambda t}\phi_2(x)\) and \(U_3(t, x) = e^{\lambda t}\phi_3(x)\) into model (11), we acquire the following eigenvalue problem
\[
\begin{aligned}
\lambda \phi_2(x) &= \nabla \cdot D_2(x) \nabla \phi_2(x) + \beta(x)\phi_3(x)f(S_0(x)) - (\mu(x) + \alpha(x) + \xi(x))\phi_2(x), \\
\lambda \phi_3(x) &= \nabla \cdot D_3(x) \nabla \phi_3(x) + \theta(x)\phi_2(x) - (\xi(x) + \gamma(x))\phi_3(x), \quad x \in \Omega, \\
\frac{\partial}{\partial n} \phi_2(x) &= \frac{\partial}{\partial n} \phi_3(x) = 0, \quad x \in \partial \Omega.
\end{aligned}
\]
Using Theorem 7.6.1 in [41], we acquire the following conclusion.

**Lemma 3.1:** The eigenvalue problem (17) has a principal eigenvalue \(\lambda_0 = \lambda_0(D_2, D_3, W_0(x))\) with a strictly positive eigenfunction \((\phi_2(x), \phi_3(x))\).

Additionally, from Theorem 3.1 in [44], we acquire the following conclusion.

**Lemma 3.2:**
(i) \(R_0 - 1\) has the same sign as \(\lambda_0\).
(ii) If \(R_0 < 1\), then disease-free equilibrium \(W_0(x)\) of model (2) is locally asymptotically stable.
(iii) If \(R_0 > 1\), then \(W_0(x)\) is unstable.

Consider the expression (16). First of all, it is easy to observe that when \(f(S_0)\) decreases, then \(R_0\) also decreases. This shows that the protective measures for susceptible individuals are effective and reasonable, because \(f(S_0)\) denotes the number of susceptible persons who are still exposed to the environment after taking protective measures against susceptible individuals at the early stage of the epidemic. When the protective measures are better than before, the number of \(f(S_0)\) will be less, so \(R_0\) will be further reduced. Next, if the diffusive rate \(D_2(x)\) of the infected and the diffusion rate \(D_3(x)\) of the pathogen increase, then \(R_0\) will decrease. This measure is reasonable in the early stage of the disease, because in model (2) the manner of transmission of the epidemic is mainly based on the contact between the susceptible and the environmental pathogen. When the total number of infected individuals and pathogens is very small, the epidemic prevention department could reduce the number of pathogens where the epidemic occurs by increasing the outward spread rate of
infected individuals and pathogens. Thus, the number of new patients in this area will be reduced, and the purpose of controlling the spread of the epidemic can be achieved. This also illustrates that it is beneficial to open windows and maintain the air circulation in the crowd gathering places at the early stage of the disease. However, when there are a large number of the infected in an area, if the epidemic prevention department uses the method of increasing the outward spread rate of infected individuals and pathogens, it could fail to effectively reduce the number of environmental pathogens of the local area. Moreover, this method can lead to a rapid increase in the number of pathogens in the surrounding area, even bringing the pathogen into an area where there is no pathogen invasion. This measure is likely to cause the epidemic to spread in a larger area and further expand the harm of epidemics. Additionally, we also notice that if antiviral treatment and isolated treatment are adopted for the infected, the amount of the pathogen released by the infected to the environment can be reduced. If disinfection and sterilization measures are taken to the environmental pathogen, the survival time of existing environmental pathogens can be reduced as well. The above two methods can reduce $R_0$.

To establish more comprehensive results, we will consider the spatially homogeneous model. That is, all parameters of model (2) become positive constants. Hence, in the homogeneous space, model (2) becomes the following form:

$$\begin{cases}
\frac{\partial}{\partial t} S(t, x) = D_1 \Delta S(t, x) + A - \beta E f(S) - \mu S, \\
\frac{\partial}{\partial t} I(t, x) = D_2 \Delta I(t, x) + \beta E f(S) - (\mu + \alpha + \zeta) I, \quad x \in \Omega, \\
\frac{\partial}{\partial t} E(t, x) = D_3 \Delta E(t, x) + \theta I(1 - E) - (\xi + \gamma) E,
\end{cases}$$

(18)

where $D_i$ ($i = 1, 2, 3$), $A, \beta, \mu, \alpha, \zeta, \theta, \xi$ and $\gamma$ are positive constants. Obviously, model (18) has always disease-free equilibrium $W_0 = (S_0, 0, 0)$ with $S_0 = \frac{A}{\mu}$. From eigenvalue problem (14), we can obtain that the positive eigenfunction $\phi_*$ satisfies $\int_{\Omega} \phi_*^2 dx = 1$ can be taken by $\phi_* \equiv 1$, where $|\Omega|$ denotes the volume of $\Omega$. Hence, from (16) we acquire the following conclusion for the spatially homogeneous model (18).

**Corollary 3.3:** For model (18), the basic reproduction number is

$$R_0 = \frac{\beta \theta f(S_0)}{(\xi + \gamma)(\mu + \alpha + \zeta)}.$$

**Remark 3:** The non-diffusive epidemic model corresponding to model (18) is as follows

$$\begin{cases}
\frac{d}{dt} S(t) = A - \beta E f(S) - \mu S, \\
\frac{d}{dt} I(t) = \beta E f(S) - (\mu + \alpha + \zeta) I, \quad x \in \Omega, \\
\frac{d}{dt} E(t) = \theta I(1 - E) - (\xi + \gamma) E.
\end{cases}$$

(19)

It is clear that $R_0$ given in Corollary 3.3 exactly also is the basic reproduction number of model (19). Therefore, from the perspective of the basic reproduction number, the
influence of diffusive factors on the extinction and prevalence of the epidemic is very small. Perhaps the diffusive factor can be ignored, and the reaction-diffusion equation epidemic model can be replaced by the corresponding ordinary differential equation epidemic model.

4. Stability of disease-free equilibrium

In this section, the global stability of disease-free equilibrium $W_0(x)$ of model (2) is investigated and the following conclusions are established.

**Theorem 4.1:** If $R_0 < 1$, then disease-free equilibrium $W_0(x)$ is globally asymptotically stable in $X^*_+$. 

**Proof:** For any initial function $\phi = (\phi_1, \phi_2, \phi_3) \in X^*_+$, let $u(t, x) = (S(t, x), I(t, x), E(t, x))$ be the solution of model (2) with initial conditions (3) defined on $[0, \infty) \times \Omega$, and $0 \leq E(t, x) \leq 1$ for all $t \geq 0$ and $x \in \Omega$. By the first equation of model (2) we acquire

$$\frac{\partial}{\partial t} S(t, x) \leq \nabla \cdot D_1(x) \nabla S(t, x) + A(x) - \mu(x) S(t, x).$$

Based on the comparison theorem and Lemma 2.1, we directly acquire $\lim \sup_{t \to \infty} S(t, x) \leq S_0(x)$ uniformly for $x \in \Omega$. Without loss of generality, we assume $S(t, x) \leq S_0(x)$ for all $t \geq 0$ and $x \in \Omega$. Thus, from assumption (H2) and the last two equations of model (2) we obtain the following differential inequalities

$$\begin{align*}
\frac{\partial}{\partial t} I(t, x) &\leq \nabla \cdot D_2(x) \nabla I(t, x) + \beta(x) E(t, x) (S_0) - (\mu(x) + \alpha(x) + \xi(x)) I, \\
\frac{\partial}{\partial t} E(t, x) &\leq \nabla \cdot D_3(x) \nabla E(t, x) + \theta(x) I - (\xi(x) + \gamma(x)) E, \quad t \geq 0, \quad x \in \Omega, \\
\frac{\partial}{\partial n} I(t, x) &\leq \frac{\partial}{\partial n} E(t, x) = 0, \quad t \geq 0, \quad x \in \partial \Omega.
\end{align*}$$

(20)

The corresponding comparison model is

$$\begin{align*}
\frac{\partial}{\partial t} z(t, x) &\leq \nabla \cdot D_2(x) \nabla z(t, x) + \beta(x) f(S_0) e - (\mu(x) + \alpha(x) + \xi(x)) z, \\
\frac{\partial}{\partial t} e(t, x) &\leq \nabla \cdot D_3(x) \nabla e(t, x) + \theta(x) z - (\xi(x) + \gamma(x)) e, \quad t \geq 0, \quad x \in \Omega, \\
\frac{\partial}{\partial n} z(t, x) &\leq \frac{\partial}{\partial n} e(t, x) = 0, \quad t \geq 0, \quad x \in \partial \Omega.
\end{align*}$$

(21)

If $R_0 < 1$, then $\lambda_0 < 0$. Hence, model (21) has the solution $(z(t, x), e(t, x)) = e^{\lambda_0 t}(\phi_2(x), \phi_3(x))$ with strictly positive initial value $(\phi_2, \phi_3)$ in $C(\Omega, R^+_2)$ tend to $(0, 0)$ uniformly for $x \in \Omega$ as $t \to \infty$. Choosing the constant $\sigma > 0$ such that $(I(0, x), E(0, x)) \leq \sigma (\phi_2(x), \phi_3(x))$ for all $x \in \Omega$. Since $\sigma e^{\lambda_0 t}(\phi_2(x), \phi_3(x))$ also is the solution of model (21), by the comparison principle and (20) it follows that $(I(t, x), E(t, x)) \leq \sigma e^{\lambda_0 t}(\phi_2(x), \phi_3(x))$ for all $x \in \Omega$ and $t \geq 0$. Thus, $(I(t, x), E(t, x))$ tend to $(0, 0)$ uniformly for $x \in \Omega$ as $t \to \infty$. 

Since \((I(t,x),E(t,x)) \rightarrow (0,0)\) uniformly for \(x \in \overline{\Omega}\) as \(t \rightarrow \infty\), by the first equation of model (2) we acquire the limit equation as below:

\[
\frac{\partial}{\partial t}S(t,x) = \nabla \cdot D_1(x)\nabla S(t,x) + A(x) - \mu(x)S(t,x).
\]

Clearly, by Lemma 2.1 and the theory of asymptotically autonomous semiflows (see [43]), we further acquire that \(S(t,x)\) tend to \(S_0(x)\) uniformly for \(x \in \overline{\Omega}\) as \(t \rightarrow \infty\). Thus, by Lemma 3.2, we can obtain that equilibrium \(W_0(x)\) is globally asymptotically stable. This completes the proof. □

Apart from the above method, we use the Lyapunov function theory to study the global stability of disease-free equilibrium \(W_0(x)\) of model (2) and get the following conclusion. Firstly, we introduce the following assumption.

(H₃) There exists a constant \(k > 0\) such that for any \(x \in \overline{\Omega}\),

\[
\beta(x)f(S_0(x)) \leq k \leq \frac{\mu(x) + \alpha(x) + \xi(x)}{\theta(x)}.
\]

Theorem 4.2: Assuming that (H₃) holds. Then disease-free equilibrium \(W_0(x)\) is globally asymptotically stable in \(X^*_+\).

Proof: The Lyapunov function \(H_1(t)\) is defined as follows

\[
H_1(t) = \int_{\Omega} I(t,x)dx + k \int_{\Omega} E(t,x)dx.
\]

The time derivative of \(H_1(t)\) along with any positive solution of model (2) is given by

\[
\frac{dH_1(t)}{dt} = \int_{\Omega} [\nabla \cdot D_2(x)\nabla I(t,x) + \beta(x)f(S) - (\mu(x) + \alpha(x) + \xi(x))I]dx
\]

\[
+ k \int_{\Omega} [\nabla \cdot D_3(x)\nabla E(t,x) + \theta(x)I(1 - E) - (\xi(x) + \gamma(x))E]dx.
\]

From the divergence theorem (see Theorem 3.3 in [15]), we get \(\int_{\Omega} \nabla \cdot D_2(x)\nabla I(t,x)dx = 0\) and \(\int_{\Omega} \nabla \cdot D_3(x)\nabla E(t,x)dx = 0\). Thus, we yield that

\[
\frac{dH_1(t)}{dt} = \int_{\Omega} [\beta(x)f(S) - (\mu(x) + \alpha(x) + \xi(x))I]
\]

\[
+ k\theta(x)I - k\theta(x)IE - k(\xi(x) + \gamma(x))E]dx.
\]

Using assumption (H₂), we obtain

\[
\frac{dH_1(t)}{dt} \leq \int_{\Omega} [\beta(x)f(S_0(x)) - (\mu(x) + \alpha(x) + \xi(x))I]
\]

\[
+ k\theta(x)I - k\theta(x)IE - k(\xi(x) + \gamma(x))E]dx.
\]

\[
(22)
\]

From assumption (H₃), we acquire \(k\theta(x) - (\mu(x) + \alpha(x) + \xi(x)) \leq 0\) and \(\beta(x)f(S_0(x)) - k(\xi(x) + \gamma(x)) \leq 0\) for all \(x \in \overline{\Omega}\). Therefore, from (22) we further acquire \(\frac{dH_1(t)}{dt} \leq 0\).
for all \( x \in \overline{\Omega} \). Clearly, \( \frac{dH_i(t)}{dt} = 0 \) implies that \( I(t,x)E(t,x) \equiv 0 \). If \( E(t,x) \equiv 0 \), by the third equation of model (2) we obtain \( I(t,x) \equiv 0 \). If \( I(t,x) \equiv 0 \), by the second equation of model (2) we obtain \( E(t,x) = S(t,x) \equiv 0 \). However, by the first equation of model (2) we directly have \( S(t,x) > 0 \). Hence, we acquire \( E(t,x) \equiv 0 \). Furthermore, by the first equation of model (2), we also acquire \( \limsup_{t \to \infty} S(t,x) = S_0(x) \). Thus, \( W_0(x) \) is globally asymptotically stable by the LaSalle’s invariance principle. This completes the proof. ■

As a consequence of Theorem 4.2, we acquire the following conclusion in the spatially homogeneous case.

**Corollary 4.3:** If \( R_0 \leq 1 \), then disease-free equilibrium \( W_0 = (S_0,0,0) \) of model (18) is globally asymptotically stable in \( X^*_+ \).

The proof of Corollary 4.3 is simple. In fact, when all coefficients \( \mu(x) \), \( \beta(x) \), \( \xi(x) \), \( \gamma(x) \), \( \theta(x) \), \( \alpha(x) \), \( \zeta(x) \), and \( S_0(x) \) are constants, we easily verify that the assumption \( (H_3) \) is equivalent to \( R_0 \leq 1 \).

**Remark 4:** Let \( R_0(x) = \frac{\beta(x)\theta(x)f(S_0(x))}{(\xi(x)+\gamma(x))(\mu(x)+\alpha(x)+\zeta(x))} \), we can readily observe that \( R_0(x) \) represents the basic reproduction number of the epidemic at the spatial local position \( x \in \Omega \). Assumption \( (H_3) \) shows that \( \max_{x \in \Omega} \{R_0(x)\} \leq 1 \), and also shows that the basic reproduction number of the epidemic at each local position in the area \( \Omega \) is less than or equal to 1. Theorem 4.2 displays that when assumption \( (H_3) \) holds then the epidemic would eventually become extinct in model (2). However, an interesting and important open problem is whether when \( \max_{x \in \Omega} \{R_0(x)\} \leq 1 \) the epidemic in model (2) is also extinct. If \( \max_{x \in \Omega} \{R_0(x)\} \leq 1 \), and the epidemic still be prevalent, it means that the backward bifurcation may appear, see for example [11]. The emergence of backward bifurcation shows that the epidemic will be difficult to control, so the epidemic prevention department needs to invest more resources to eliminate the epidemic.

**Remark 5:** Remark 3 and Corollary 4.3 reveal that the spatially homogeneous diffusion model (18) is identical to the corresponding non-diffusive model (19) in terms of the extinction of the epidemic. That is, when \( R_0 \leq 1 \), the epidemic is extinct in both models, and the disease-free equilibrium of both models is globally asymptotically stable.

**Remark 6:** Clearly, in the general case of model (2), the calculation of \( R_0 \) is very difficult by the spectral radius of operator \( L \) or formula (16). Therefore, if we can easily verify assumption \( (H_3) \) holds, it sufficiently implies the global asymptotic stability of disease-free equilibrium \( W_0(x) = (S_0(x),0,0) \) of model (2).

## 5. Uniform persistence

In this section, we discuss the uniform persistence of model (2). Firstly, we introduce the following conclusion.

**Lemma 5.1:** If \( R_0 > 1 \), then there is a constant \( \eta^* > 0 \) such that for any initial function \( \phi = (\phi_1, \phi_2, \phi_3) \in X^*_+ \) with \( \phi_2 \neq 0 \) and \( \phi_3 \neq 0 \), the solution \( u(t,x) = (S(t,x), I(t,x), E(t,x)) \) of
model (2) with initial value $\phi$ satisfies

$$\limsup_{t \to \infty} \|u(t, \cdot) - W_0\|_{X^+_t} \geq \eta^*.$$  

**Proof:** For any initial value $\phi \in X^+_t$ with $\phi_2 \neq 0$ and $\phi_3 \neq 0$, from the parabolic maximum principle (see [38]), we get $I(t, x) > 0$ and $E(t, x) > 0$ for all $t > 0$ and $x \in \overline{\Omega}$. By Lemma 3.2, we have $\lambda_0 > 0$. By (17), we consider the following eigenvalue problem

$$\begin{aligned}
\lambda \xi_1(x) &= \nabla \cdot D_2(x) \nabla \xi_1(x) + \beta(x) \xi_2(x) f(S_0(x) - \eta) - (\mu(x) + \alpha(x) + \zeta(x)) \xi_1(x), \\
\lambda \xi_2(x) &= \nabla \cdot D_3(x) \nabla \xi_2(x) + \theta(x) \xi_1(x)(1 - \eta) - (\xi(x) + \gamma(x)) \xi_2(x), \\
\frac{\partial}{\partial n} \xi_1(x) &= \frac{\partial}{\partial n} \xi_2(x) = 0, \quad x \in \partial \Omega.
\end{aligned}$$

(23)

Let $\lambda_0(\eta)$ be the principal eigenvalue of problem (23). There exists an enough small constant $\eta^* \in (0, 1)$, so $\lambda_0(\eta^*) > 0$, and $S_0(x) > \eta^*$ for all $x \in \overline{\Omega}$. Furthermore, the eigenvector $(\xi_1(x), \xi_2(x))$ corresponding to $\lambda_0(\eta^*)$ also is strictly positive for $x \in \overline{\Omega}$.

Supposing that the conclusion is incorrect, then there exists a $\phi = (\phi_1, \phi_2, \phi_3) \in X^+_t$ with $\phi_2 \neq 0$ and $\phi_3 \neq 0$, so $\limsup_{t \to \infty} \|u(t, \cdot) - W_0\|_{X^+_t} < \eta^*$, where $u(t, x) = (S(t, x), I(t, x), E(t, x))$ is the solution with initial condition $u(0, x) = \phi(x)$. There exists an enough large $t_2$ such that for any $t \geq t_2$ and $x \in \overline{\Omega}$,

$$0 < S_0 - \eta^* < S(t, x) < S_0 + \eta^*, \quad 0 < I(t, x) < \eta^*, \quad 0 < E(t, x) < \eta^*.$$  

Thus, according to model (2) and assumption (H$_2$) we obtain the following differential inequalities

$$\begin{aligned}
\frac{\partial}{\partial t} I(t, x) &\geq \nabla \cdot D_2(x) \nabla I(t, x) + \beta(x) E f(S_0(x) - \eta^*) - (\mu(x) + \alpha(x) + \zeta(x)) I, \\
\frac{\partial}{\partial t} E(t, x) &\geq \nabla \cdot D_3(x) \nabla E(t, x) + \theta(x) I(1 - \eta^*) - (\xi(x) + \gamma(x)) E, \\
\frac{\partial}{\partial t} y_1(t, x) &\geq \nabla \cdot D_2(x) \nabla y_1(t, x) + \beta(x) y_2 f(S_0(x) - \eta^*) - (\mu(x) + \alpha(x) + \zeta(x)) y_1, \\
\frac{\partial}{\partial t} y_2(t, x) &\geq \nabla \cdot D_3(x) \nabla y_2(t, x) + \theta(x) y_1(1 - \eta^*) - (\xi(x) + \gamma(x)) y_2,
\end{aligned}$$

(24)

Clearly, the comparison equation is

$$\begin{aligned}
\frac{\partial}{\partial t} y_1(t, x) &= \nabla \cdot D_2(x) \nabla y_1(t, x) + \beta(x) y_2 f(S_0(x) - \eta^*) - (\mu(x) + \alpha(x) + \zeta(x)) y_1, \\
\frac{\partial}{\partial t} y_2(t, x) &= \nabla \cdot D_3(x) \nabla y_2(t, x) + \theta(x) y_1(1 - \eta^*) - (\xi(x) + \gamma(x)) y_2, \\
\frac{\partial}{\partial n} y_1(t, x) &= \frac{\partial}{\partial n} y_2(t, x) = 0, \quad x \in \partial \Omega, \quad t > t_2.
\end{aligned}$$

(25)

It has the solution $(y_1(t, x), y_2(t, x)) = e^{\lambda_0(\eta^*)(t-t_2)} (\xi_1(x), \xi_2(x))$. Owing to $(I(t, x), E(t, x)) > 0$ for $x \in \overline{\Omega}$, we select a constant $\varrho > 0$ such that $(I(t_2, x), E(t_2, x)) \geq \varrho (\xi_1(x), \xi_2(x))$ for $x \in \overline{\Omega}$. Note that $\varrho (y_1(t, x), y_2(t, x))$ also is the solution of Equation (25). From the
comparison principle and (24) we acquire
\[
(I(t, x), E(t, x)) \geq \phi(y_1(t, x), y_2(t, x)), \quad x \in \overline{\Omega}, \ t > t_2.
\]
We have \(\lim_{t \to \infty} y_i(t, x) = \infty\) for \(i = 1, 2\). Since \(\lambda_0(\eta^*) > 0\), we have \(\lim_{t \to \infty} I(t, x) = \infty\) and \(\lim_{t \to \infty} E(t, x) = \infty\), which is a contradiction with the boundedness of \((I(t, x), E(t, x))\) by Theorem 2.2. This completes the proof.

From Theorem 2.2 and Remark 2, we deduce the existence of the global compact attractor of model (2) as follows.

**Corollary 5.2:** The solution semiflow \(\Lambda(t) = u(t, \cdot) : X^*_+ \to X^*_+\) of model (2) has a compact and global attractor.

Defining the set \(\Gamma_0 = \{\phi = (\phi_1, \phi_2, \phi_3) \in X^*_+ : \phi_2 \neq 0, \phi_3 \neq 0\}\). Clearly, we have \(\partial \Gamma_0 = X^*_+ \setminus \Gamma_0 = \{\phi \in X^*_+ : \phi_2 \equiv 0 \text{ or } \phi_3 \equiv 0\}\) and \(\Gamma_0\) is the invariant set of semiflow \(\Phi(t)\) for model (2). Furthermore, we define the set \(N_\beta = \{\phi \in X^*_+ : \Lambda(t)\phi \in \partial \Gamma_0, \ t \geq 0\}\). Hence, we get the following conclusion.

**Lemma 5.3:** Let \(\omega(\phi)\) be the omega limit set of solution \(\Lambda(t)\phi\) and set \(N_1 = \{W_0(x)\}\). Then we have \(\bigcup_{\phi \in N_\beta} \omega(\phi) = N_1\).

**Proof:** We have \(N_1 \subset \bigcup_{\phi \in N_\beta} \omega(\phi)\), because \(\Lambda(t)W_0(x) = W_0(x)\) for all \(t \geq 0\). Now, we demonstrate \(\bigcup_{\phi \in N_\beta} \omega(\phi) \subset N_1\). For any given \(\phi \in N_\beta\), we get \(I(t, x) = 0\) or \(E(t, x) = 0\) for all \(t \geq 0\), because \(\Lambda(t)\phi \in \partial \Gamma_0\) for all \(t \geq 0\).

If \(I(t, x) = 0\), then by the second equation of model (2), we acquire \(E(t, x) = 0\). Thus, from model (2) we further acquire the equation as follows
\[
\left\{ \begin{array}{l}
\frac{\partial}{\partial t} S(t, x) = \nabla \cdot D_1(x) \nabla S(t, x) + A(x) - \mu(x) S(t, x), \quad x \in \Omega, \\
\frac{\partial}{\partial n} S(t, x) = 0, \quad x \in \partial \Omega.
\end{array} \right.
\]
Clearly, from Lemma 2.1 we have \(\lim_{t \to \infty} S(t, x) = S_0(x)\). This implies that \(\omega(\phi) = W_0(x)\). If \(E(t, x) = 0\), then by the third equation of model (2), we get \(I(t, x) = 0\). Equally, we also get Equation (26). Thus, \(\lim_{t \to \infty} S(t, x) = S_0(x)\), showing \(\omega(\phi) = W_0(x)\).

Based on the above analysis we get \(\bigcup_{\phi \in N_\beta} \omega(\phi) \subset N_1\). Thus, we eventually acquire \(\bigcup_{\phi \in N_\beta} \omega(\phi) = N_1\). This completes the proof.

Regarding the uniform persistence of all positive solutions of model (2), we have the following conclusions.

**Theorem 5.4:** If \(R_0 > 1\), then there exists a constant \(\epsilon > 0\), such that for any initial value \(\phi = (\phi_1, \phi_2, \phi_3) \in X^*_+\) with \(\phi_2 \neq 0\) and \(\phi_3 \neq 0\), the solution \(u(t, x) = (S(t, x), I(t, x), E(t, x))\) of model (2) with initial value \(\phi\) satisfies
\[
\liminf_{t \to \infty} S(t, x) \geq \epsilon, \quad \liminf_{t \to \infty} I(t, x) \geq \epsilon, \quad \liminf_{t \to \infty} E(t, x) \geq \epsilon
\]
uniformly for \(x \in \overline{\Omega}\).
Proof: By Theorem 2.2, there exists a constant \( Y > 0 \), such that for any solution \((S(t, x), I(t, x), E(t, x))\), there is a time \( t_3 > 0 \) one has \( S(t, x) \leq Y, I(t, x) \leq Y \) and \( E(t, x) \leq Y \) for all \( x \in \Omega \) and \( t \geq t_3 \). Hence, by the first equation of model (2) we get

\[
\frac{\partial}{\partial t} S(t, x) \geq \nabla \cdot D_1(x) \nabla S(t, x) + A(x) - (\beta(x)Y + \mu(x))S(t, x), \quad t > t_3, \quad x \in \bar{\Omega}.
\]

By Lemma 2.1, we can obtain that \( S(t, x) \) has a positive lower bound, which reveals that the component \( S(t, x) \) in model (2) is uniformly persistent.

Defining a continuous function \( m : X^*_+ \rightarrow R_+ \) by

\[
m(\phi) = \min\{\min_{x \in \Omega} \phi_2(x), \min_{x \in \Omega} \phi_3(x)\}, \quad \phi \in X^*_+.
\]

Clearly, when \( m(\phi) > 0 \), we have \( \min_{x \in \Omega} \phi_2(x) > 0, \min_{x \in \Omega} \phi_3(x) > 0 \), implying \( m^{-1}(0, +\infty) \subset \Gamma_0 \). When \( \phi_2(x) \neq 0 \) and \( \phi_3(x) \neq 0 \), we acquire \( I(t, x) > 0 \) and \( E(t, x) > 0 \) for all \( t \geq 0 \) and \( x \in \bar{\Omega} \) by the parabolic maximum principle [38]. Hence, we acquire that function \( m \) has the property that if either \( m(\phi) = 0 \) and \( \phi \in \Gamma_0 \), or \( m(\phi) > 0 \), then \( m(\Lambda(t)\phi) > 0 \). Hence, \( m \) is a generalized distance function (see Theorem 3 in [42]) for semiflow \( \Lambda(t) : X^*_+ \rightarrow X^*_+ \).

By \( \bigcup_{\phi \in N_0} \omega(\phi) = N_1 \), we can readily see that when \( t \rightarrow +\infty \) any solutions on boundary \( \partial \Gamma_0 \) of model (2) tend to equilibrium \( W_0(x) \). By Lemma 5.1, we get that \( W_0(x) \) is an isolated invariant set in \( X^*_+ \), and \( W^s(W_0(x)) \cap \Gamma_0 = 0 \), where \( W^s(W_0(x)) \) marks the stable set of equilibrium \( W_0(x) \), and hence we obtain that \( W^s(W_0(x)) \cap m^{-1}(0, +\infty) = 0 \).

Furthermore, by the above arguments we can observe that no subset of \( N_1 \) forms a cycle in \( \partial \Gamma_0 \). By Corollary 5.2, solution semiflow \( \Lambda(t) : X^*_+ \rightarrow X^*_+ \) has a global compact attractor in \( X^*_+ \). By Theorem 3 in [42] and Corollary 5.2, we can obtain that there exists a constant \( \epsilon > 0 \), so \( \liminf_{t \rightarrow \infty} m(\Lambda(t)\phi) \geq \epsilon \) for all \( \phi \in \Gamma_0 \). The conclusion gives information that infected individuals and the concentration of environmental pathogens have uniform persistence. This completes the proof. \( \square \)

Corollary 5.5: When \( R_0 > 1 \), there is at least one endemic equilibrium \( W_+(x) = (S^*(x), I^*(x), E^*(x)) \) of model (2).

Remark 7: At present, we only obtain the existence of endemic equilibrium \( W^*(x) \) of model (2). Furthermore, we need to do further studies on the stability of equilibrium \( W^*(x) \). It is a pity that we don’t established any meaningful results for the general spatial heterogeneity model (2). However, for the spatially homogeneous model (18), we investigate the global stability of endemic equilibrium in the next section.

6. Stability of endemic equilibrium

We here investigate the global asymptotic stability of endemic equilibrium \( W^* \) of model (18).

Theorem 6.1: When \( R_0 > 1 \), model (18) has unique endemic equilibrium \( W^* = (S^*, I^*, E^*) \), where \( S^* = \frac{A-(\mu+\alpha + \zeta)I^*}{\mu} \), \( E^* = \frac{\beta I^*}{\xi + \gamma + \theta I^*} \) and \( I^* \in (0, \frac{A}{\mu+\alpha + \zeta}) \) is the unique
Obviously, by assumption it is clear that equilibrium $W^*$ satisfies equations

$$\begin{align*}
A - \beta E^* f(S^*) - \mu S^* &= 0, \\
\beta E^* f(S^*) - (\mu + \alpha + \zeta) I^* &= 0, \\
\theta I^* (1 - E^*) - (\xi + \gamma) E^* &= 0.
\end{align*}$$

(27)

From the first and second equations of (27), we get $\frac{A - (\mu + \alpha + \zeta) I^*}{\mu} = S^*$. By the third equation of (27), we acquire $E^* = \frac{\theta I^*}{\xi + \gamma + \theta I^*}$. By substituting them into the second equation of (27), we further acquire the equation as follows

$$H(I^*) = \frac{\beta \theta}{\xi + \gamma + \theta I^*} f\left(\frac{A - (\mu + \alpha + \zeta) I^*}{\mu}\right) - (\mu + \alpha + \zeta) = 0.$$ 

Obviously, by assumption (H2), $H(I^*)$ is decreasing for $I^* \in \left[0, \frac{A}{\mu + \alpha + \zeta}\right]$. Since $H(0) = \frac{\beta \theta f(S_0)}{\xi + \gamma} - (\mu + \alpha + \zeta)$, we have $H(0) > 0$ when $R_0 > 1$ and $H(0) \leq 0$ when $R_0 \leq 1$. Furthermore, $H\left(\frac{A}{\mu + \alpha + \zeta}\right) = -(\mu + \alpha + \zeta) < 0$. Hence, we acquire when $R_0 > 1$ equation $H(I^*) = 0$ has a unique root $I^* \in \left(0, \frac{A}{\mu + \alpha + \zeta}\right)$, and when $R_0 \leq 1$ equation $H(I^*) = 0$ has not any positive root. It can be seen from the above discussion that Theorem 6.1 is valid. This completes the proof.

Theorem 6.2: If $R_0 > 1$, then endemic equilibrium $W^* = (S^*, I^*, E^*)$ of model (18) is globally asymptotically stable in $X^*_0$.

Proof: The Lyapunov function $H_2(t)$ is defined as follows

$$H_2(t) = \int_\Omega \left[ (S - S^*) \int_{S^*}^S \frac{f(S')}{f(S)} \, dS' \right] + \left( I - I^* - I^* \ln \frac{I}{I^*} \right)$$

$$+ \frac{(\mu + \alpha + \zeta)}{\theta (1 - E^*)} \left( E - E^* - E^* \ln \frac{E}{E^*} \right) \, dx.$$ 

The time derivative of $H_2(t)$ along with any positive solution of model (2) is given by

$$\frac{dH_2(t)}{dt} = \int_\Omega \left\{ \left( 1 - \frac{f(S^*)}{f(S)} \right) [D_1 \Delta S(t,x) + A - \beta Ef(S) - \mu S] \right.$$ 

$$+ \left( 1 - \frac{I^*}{I} \right) [D_2 \Delta I(t,x) + \beta Ef(S) - (\mu + \alpha + \zeta) I]$$

$$+ \frac{(\mu + \alpha + \zeta)}{\theta (1 - E^*)} \left( 1 - \frac{E^*}{E} \right) \left( D_3 \Delta E(t,x) + \theta I(1 - E) - (\xi + \gamma) E \right) \right\} \, dx.$$
From the divergence theorem (see Theorem 3.3 in [15]), we can yield that

\[
\frac{dH_2(t)}{dt} = \int_{\Omega} \left[ A - \mu S - A^* \frac{f(S^*)}{f(S)} + \beta Ef(S^*) + \frac{f(S^*)}{f(S)} \mu S - (\mu + \alpha + \zeta)I - \beta Ef(S) \frac{I^*}{I} \right. \\
+ (\mu + \alpha + \zeta)I^* + \frac{\mu + \alpha + \zeta)I(1 - E)}{(1 - E^*)} - \frac{\xi + \gamma)(\mu + \alpha + \zeta)E}{\theta(1 - E^*)} \\
- \frac{(\mu + \alpha + \zeta)E^* I(1 - E)}{(1 - E^*)E} + \frac{(\xi + \gamma)(\mu + \alpha + \zeta)E^*}{\theta(1 - E^*)} - \frac{f(S^*)f'(S)}{f^2(S)} D_1 \| \nabla S \|^2 \\
- \frac{I^* D_2 \| \nabla I \|^2}{I^2} - \frac{(\mu + \alpha + \zeta)E^*}{\theta(1 - E^*)} D_3 \| \nabla E \|^2 \right] dx.
\]

From (27), we obtain

\[
\frac{dH_2(t)}{dt} = \int_{\Omega} \left[ (\mu + \alpha + \zeta)I^* + \mu S^* - \mu S^* - (\mu + \alpha + \zeta)I^* \frac{f(S^*)}{f(S)} - \mu S^* \frac{f(S^*)}{f(S)} \\
+ (\mu + \alpha + \zeta)I^* \frac{E}{E^*} + \frac{f(S^*)}{f(S)} \mu S^* - (\mu + \alpha + \zeta)I^* \frac{f(S^*)}{f(S^*)} E^* I \\
+ 2(\mu + \alpha + \zeta)I^* + \frac{\mu + \alpha + \zeta)I(1 - E)}{(1 - E^*)} - (\mu + \alpha + \zeta)I^* \frac{E}{E^*} \\
- \frac{(\mu + \alpha + \zeta)E^* I(1 - E)}{(1 - E^*)E} + (\mu + \alpha + \zeta)I^* \frac{E}{E^*} - (\mu + \alpha + \zeta)I^* \frac{E}{E^*} \\
- \frac{f(S^*)f'(S)}{f^2(S)} D_1 \| \nabla S \|^2 - I^* D_2 \| \nabla I \|^2 \right] dx.
\]

By calculating, we have

\[
- (\mu + \alpha + \zeta)I + \frac{(\mu + \alpha + \zeta)I(1 - E)}{(1 - E^*)} \\
- \frac{(\mu + \alpha + \zeta)E^* I(1 - E)}{(1 - E^*)E} + (\mu + \alpha + \zeta)I^* \frac{E}{E^*} \\
= - \frac{(\mu + \alpha + \zeta)(1 - E^*)E}{(1 - E^*)E} + \frac{(\mu + \alpha + \zeta)(1 - E^*)E}{(1 - E^*)E} - \frac{(\mu + \alpha + \zeta)E^* I(1 - E)}{(1 - E^*)E} \\
+ \frac{(\mu + \alpha + \zeta)(1 - E^*)E}{(1 - E^*)E} \\
= (\mu + \alpha + \zeta)I \frac{[-(1 - E^*)E + (1 - E)E - E^* (1 - E) + (1 - E^*)E]}{(1 - E^*)E} \\
= - \frac{(\mu + \alpha + \zeta)(E - E^*)^2}{(1 - E^*)E}.
\]

By \(E^* \in (0, 1)\), we obtain \(- \frac{(\mu + \alpha + \zeta)(E - E^*)^2}{(1 - E^*)E} < 0\). Thus, we further obtain

\[
\frac{dH_2(t)}{dt} = \int_{\Omega} \left[ 3(\mu + \alpha + \zeta)I^* - (\mu + \alpha + \zeta)I^* \frac{f(S^*)}{f(S)} - (\mu + \alpha + \zeta)I^* \frac{f(S^*)}{f(S^*)} E^* I \\
- (\mu + \alpha + \zeta)I^* \frac{E}{E^*} + \frac{(\mu + \alpha + \zeta)(1 - E^*)E}{(1 - E^*)E} - \frac{(\mu + \alpha + \zeta)E^* I(1 - E)}{(1 - E^*)E} \\
+ \frac{(\mu + \alpha + \zeta)(1 - E^*)E}{(1 - E^*)E} \\
= (\mu + \alpha + \zeta)I \frac{[-(1 - E^*)E + (1 - E)E - E^* (1 - E) + (1 - E^*)E]}{(1 - E^*)E} \\
= - \frac{(\mu + \alpha + \zeta)(E - E^*)^2}{(1 - E^*)E}.
\]
From assumption \((H_2)\), we obtain \(\mu(S^*-S) \left[ 1 - \frac{f(S^*)}{f(S)} \right] \leq 0\) for all \(S > 0\). Hence, by the condition of Theorem 6.2, we have \(\frac{dH_2(t)}{dt} \leq 0\). Moreover, \(\frac{dH_2(t)}{dt} = 0\) if and only if \((S, I, E) = (S^*, I^*, E^*)\). Thus, by the LaSalle’s invariable principle, it is demonstrated that endemic equilibrium \(W^* = (S^*, I^*, E^*)\) is globally asymptotically stable. This completes the proof. \(\blacksquare\)

**Remark 8:** Comparing Theorem 4.2, Corollary 4.3 with Theorem 6.2, we have a very meaningful open problem here. That is, if there exists a constant \(\tilde{k} > 0\), so for any \(x \in \overline{\Omega}\)

\[
\frac{\beta(x)f(S_0(x))}{\xi(x) + \gamma(x)} > \tilde{k} > \frac{\mu(x) + \alpha(x) + \zeta(x)}{\theta(x)},
\]

or more general condition \(\min_{x \in \overline{\Omega}} \{R_0(x)\} > 1\) is satisfied, and then whether endemic equilibrium \(W^*(x) = (S^*(x), I^*(x), E^*(x))\) of model (2) in the spatial heterogeneous case is globally asymptotically stable.

**Remark 9:** By Theorem 6.2, we find that the global stability of endemic equilibrium of diffusion model (18) in the homogeneous case is same as the global stability of the corresponding non-diffusive model (19). That is, when \(R_0 > 1\), both models have the same endemic equilibrium, and both equilibria also are globally asymptotically stable. Furthermore, in conjunction with Remark 5, we find that the global dynamics of the spatially homogeneous diffusion model (18) and the corresponding non-diffusive model (19) are almost the same, and are uniquely determined by the basic reproduction number.

### 7. Numerical examples

Due to model (2) is nonlinear and spatially heterogeneous, it is difficult to get the analytical solutions with any initial conditions. Therefore, it is necessary to solve the approximate solution of the model by using the numerical method. So far, there are many numerical methods for solving differential equations, such as numerical methods for fractional differential equations (see [2, 3]), and numerical methods for integer-order differential equations.
equations (see [31–33]). According to the different models, we can choose different numerical methods. Since model (2) is an integer-order partial differential equation, we here use the non-standard finite difference method (see [31]) to solve model (2).

We give two numerical examples to demonstrate the open problems proposed in Remarks 4 and 8. In model (2), we take $x \in \Omega = [0, 10]$, function $f(S) = S$, and the parameters

$D_1(x) = 0.8 + 0.7 \sin(2\pi x)$, $D_2(x) = 0.0012 + 0.0005 \sin(2\pi x)$, $D_3(x) = 0.0018 + 0.0005 \sin(2\pi x)$, $A(x) = 6 + 5 \sin(2\pi x)$, $\mu(x) = 0.22 + 0.05 \sin(2\pi x)$, $\xi(x) = 0.04 + 0.02 \sin(2\pi x)$, $\gamma(x) = 0.04 + 0.02 \sin(2\pi x)$ and $\zeta(x) = 0.03 + 0.02 \sin(2\pi x)$. The parameters $\beta(x)$, $\alpha(x)$ and $\theta(x)$ are chosen as free parameters.

**Example 7.1:** We select the surplus parameters $\beta(x) = 0.06 + 0.05 \sin(2\pi x)$, $\alpha(x) = 0.2 + 0.05 \sin(2\pi x)$ and $\theta(x) = 0.15 + 0.05 \sin(2\pi x)$.

By calculating we have $\max_{x \in \Omega} R_0(x) = 0.3309 < 1$. Model (2) has disease-free equilibrium $W_0(x) = (27.3, 0, 0)$. We give the initial value of solution $(S(t, x), I(t, x), E(t, x))$ as follows: $S(0, x) = 320 \exp(-10(x - 5)^2)$, $I(0, x) = 4.8 \exp(-10(x - 5)^2)$ and $E(0, x) = 0.4 \exp(-0.1(x - 5)^2)$. The numerical simulations in Figure 1 elucidate that equilibrium $W_0(x)$ is globally asymptotically stable. Thus, the open problem in Remark 4 is verified to may be right.

**Example 7.2:** We choose the surplus parameters $\beta(x) = 0.8 + 0.05 \sin(2\pi x)$, $\alpha(x) = 0.006 + 0.005 \sin(2\pi x)$ and $\theta(x) = 0.32 + 0.05 \sin(2\pi x)$.

![Figure 1](image1.png) Dynamical behaviours of $S(t, x)$ (a), $I(t, x)$ (b) and $E(t, x)$ (c). The numerical simulations indicate that the solutions finally converge to equilibrium $W_0(x)$.

![Figure 2](image2.png) Dynamical behaviours of $S(t, x)$ (a), $I(t, x)$ (b) and $E(t, x)$ (c). The numerical simulations indicate that the solutions finally converge to equilibrium $W^*(x)$. 
By calculating we have $\min_{x \in \Omega} (R_0(x)) = 1.0122 > 1$. We give the initial value of solution $(S(t, x), I(t, x), E(t, x))$ as follows: $S(0, x) = 320 \exp(-10(x - 5)^2)$, $I(0, x) = 64 \exp(-10(x - 5)^2)$ and $E(0, x) = 0.2 \exp(-0.1(x - 5)^2)$. The numerical simulations in Figure 2 elucidate that equilibrium $W^*(x)$ is globally asymptotically stable. Thus, the open problem in Remark 8 is verified to may be right.

8. Conclusion

In this paper, we propose a reaction-diffusion SIR model via environmental driven infection in heterogeneous space. In addition, the model also includes the protective measures taken by humans in response to the infectious disease. For example, the susceptible wear masks, isolation and treatment for the infected. The above methods block human to human transmission, but it also complicates the spread of the epidemic. It is clear that the nonlinear incidence rate is more suitable for the situation after the implementation of the above measures (see [17, 18, 26, 28, 36, 40]). Therefore, it is of practical significance to study the threshold dynamics of the epidemic with nonlinear incidence.

Firstly, the well-posedness of solutions is obtained. Secondly, the basic reproduction number $R_0$ is calculated. Thirdly, we establish a threshold condition for the global asymptotic stability of disease-free equilibrium $W_0(x)$ by applying the theory of asymptotically autonomous semiflows and the Lyapunov function theory, respectively. In terms of epidemiology, when $R_0 \leq 1$, the number of the infected and the content of pathogens in the environment will gradually become zero, and eventually the epidemic becomes extinct. On the contrary, when $R_0 > 1$ we get the uniform persistence of model (2) by adopting the persistence theory of dynamical systems, and model (2) has at least one endemic equilibrium. Furthermore, when $R_0 > 1$, the global asymptotic stability of endemic equilibrium $W^* = (S^*, I^*, E^*)$ is obtained in the spatially homogenous case. That is, when $R_0 > 1$ although many infected people are cured in the hospital, newly infected individuals will continue to appear so that the number of infected people and the content of pathogens in the environment remain at a high level. Therefore, the epidemic will continue to spread and pose a serious impact on human health.

However, it is a pity that we don’t acquire the global asymptotic stability of disease-free equilibrium $W_0(x)$ when $\max_{x \in \Omega} (R_0(x)) \leq 1$ and the global asymptotic stability of endemic equilibrium $W^*(x)$ when $\min_{x \in \Omega} (R_0(x)) > 1$. Therefore, we propose an open question in Remarks 4 and 8, respectively, and verify those problems by numerical examples. As for the theoretical results of the above two questions, we will study and solve them in the future.

In this paper, we think that after the implementation of the prevention and control measures of the epidemic, the transmission route of human to human has been cut off, and environment-driven infection has become the only transmission route of the epidemic. However, in the actual process of epidemic prevention and control, human to human transmission and environment-driven infection always coexist, and it is difficult to completely prevent human to human transmission of the epidemic. Therefore, in order to make the model more realistic, we propose an epidemic model with two transmission
Furthermore, if we consider the epidemic with latent period and the isolation of asymptomatic infectors and close contacts, we can establish a kind of epidemic dynamics model with reaction-diffusion and environmental pathogens infection, including susceptible class, latent class, infected class, asymptomatic infection class, isolated class, and environmental pathogens. The above model can more accurately describe the actual situation of the spread of the epidemic, so we must make a detailed theoretical analysis and numerical simulation of the model. We believe that the results will have very important practical significance.

Also, we know that since people learn about the spread of the epidemic through media reports or other channels, they will make corresponding behavioural changes based on the received information. In response to this phenomenon, some researchers have introduced the effects of human memory and learning behaviours on epidemic transmission into epidemic models (see [11]), which makes epidemic models more realistic and results of the study more meaningful. Reaction-diffusion epidemic models that take human memory and learning behaviour into account will be a very interesting research topic. We hope to investigate this in the future.

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