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Original articles

Analysis of a degenerated reaction–diffusion cholera model with spatial heterogeneity and stabilized total humans[☆]

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Abstract

In this paper, we perform a complete analysis on a degenerated reaction–diffusion cholera model with stabilizing total humans and non-mobility of cholera bacteria in a spatially heterogeneous bounded domain. The existence of a global attractor is established through introducing the Kuratowski measure of non-compactness. The basic reproduction number and its equivalent characterizations have been used to analyze the threshold-type results, where the persistence and extinction of cholera can also be characterized by the dispersal rate of infected humans. In the homogeneous case, the global attractivity of the unique positive equilibrium is achieved by the Lyapunov function. Moreover, we compare the basic reproduction numbers for the models without and with considering the mobility of cholera bacteria. Our results suggest that: the basic reproduction numbers attain different values as the dispersal rates of infected humans and cholera approach to infinity, while they attain the same value as the dispersal rates of infected humans and cholera approach to zero. Numerical simulations support our analytical results and discuss the impact of the dispersal rate of infected humans on the transmission of cholera.

Keywords: Spatial heterogeneity; Degenerated reaction–diffusion model; Threshold dynamics; Lyapunov function; Basic reproduction number

1. Introduction

Cholera, caused by *Vibrio cholerae*, belongs to an acute diarrheal disease, involving the interactions between human hosts, pathogens, and environments. When studying the transmission dynamics of cholera, many nonlinear differential equations are formulated with two transmission routes, including direct person-to-person route and indirect *Vibrio cholerae*-to-person route (see, for example, [18,27,31,32,34]). During the past years, many contributions have been made to study the impacts of the mobility of humans and environmental heterogeneity on the spread of disease, see a series of work on reaction–diffusion SIS models with/without advection, but are not limited

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to [1,4,5,11,21,22,29,35,37,38]. The environmental heterogeneity and individual motility have also been accepted as central roles towards understanding the spatial spreading of cholera [27,30–32,34]. In general, the habitat of humans is assumed to be an isolated spatially bounded and heterogeneous domain $\Omega \subset \mathbb{R}^n$ with smooth boundary $\partial\Omega$. If there are no specific requirements, we still denote that the humans and *Vibrio cholerae* are subject to the spatial-variable x and time-variable t, respectively. The following degenerated reaction–diffusion cholera model was studied in [32]:

$$\begin{cases} \dfrac{\partial \bar{S}}{\partial t} = d_S \Delta \bar{S} + \lambda(x) - \alpha(x)\bar{S}\bar{I} - \beta(x)\bar{S}\bar{P} - a(x)\bar{S}, \\ \dfrac{\partial \bar{I}}{\partial t} = d_I \Delta \bar{I} + \alpha(x)\bar{S}\bar{I} + \beta(x)\bar{S}\bar{P} - b(x)\bar{I}, \\ \dfrac{\partial \bar{P}}{\partial t} = c(x)\bar{I} - m(x)\bar{P}, \end{cases} \tag{1.1}$$

in $(x, t) \in \Omega \times (0, \infty)$ with boundary and initial conditions,

$$\begin{cases} \dfrac{\partial \bar{S}}{\partial \nu} = \dfrac{\partial \bar{I}}{\partial \nu} = 0, \ x \in \partial\Omega, \ t > 0, \\ (\bar{S}(x,0), \bar{I}(x,0), \bar{P}(x,0)) = (\bar{S}^0(x), \bar{I}^0(x), \bar{P}^0(x)), \ x \in \Omega. \end{cases} \tag{1.2}$$

Here $\bar{S} := \bar{S}(x,t)$, $\bar{I} := \bar{I}(x,t)$, and $\bar{P} := \bar{P}(x,t)$ are the densities of susceptible humans, infected humans, and *Vibrio cholerae* in the water source, respectively; $d_S > 0$ and $d_I > 0$, respectively, represent the dispersal rates of susceptible and infected humans; $\lambda(x)$ is Hölder continuous and positive function, representing the recruitment rate of susceptible humans; $a(x)$ and $m(x)$ are Hölder continuous and positive functions, standing for the rates of death of susceptible individuals and *Vibrio cholerae*, respectively; the positive function $b(x)$ is Hölder continuous, standing for the rate of removal from infected individuals; $c(x)$ is Hölder continuous and positive function, measuring the shedding rate of *Vibrio cholerae* from infected humans; the positive functions $\alpha(x)$ and $\beta(x)$ are Hölder continuous, measuring the rates of disease transmission for direct transmission route and indirect transmission route, respectively. The boundary condition, $\frac{\partial \bar{S}}{\partial \nu} = \frac{\partial \bar{I}}{\partial \nu} = 0$, means that no population flux crosses the boundary $\partial\Omega$, where $\frac{\partial}{\partial \nu}$ is the derivative along the outward normal ν. $\bar{S}^0(x)$, $\bar{I}^0(x)$, and $\bar{P}^0(x)$ are nonnegative and continuous functions.

Since the lack of diffusion term in the $\bar{P}$-equation of (1.1), the compactness of solution semiflow is established by confirming the κ-contraction condition through the Kuratowski measure of noncompactness. Nonnegative steady state solutions of (1.1) are composed of two types: disease-free steady state (DFSS) and positive steady state (PSS). The former is $(\tilde{S}(x), \tilde{I}(x), \tilde{P}(x)) \in C^2(\bar{\Omega}) \times C^2(\bar{\Omega}) \times C^2(\bar{\Omega})$ with $\tilde{I}(x) = \tilde{P}(x) = 0$ on $\bar{\Omega}$ while the latter is the one that $\tilde{S}(x), \tilde{I}(x), \tilde{P}(x) > 0$ on $\bar{\Omega}$. By the general results of [6,35], the basic reproduction number (BRN) is defined as the spectral radius of next generation operator (NGO), and written as a variational characterization. It was also found that BRN is monotonically decreasing in d_I, and attains the maximum of local basic reproduction number (LBRN) as $d_I \to 0$ and the certain value as $d_I \to \infty$. In this sense, the threshold-type results of (1.1) in terms of the BRN can also be characterized by d_I. The asymptotic profiles of PSS as $d_S \to 0$ and $d_I \to 0$, and the relation between the BRN and LBRN were also established in [32]. It should be noted that cholera can be eliminated through restricting d_S below a certain value, while restricting d_I below a certain value, the infected humans shall concentrate in some locations of the domain, which agrees with the circumstances described in [38].

In system (1.1), the terms $\bar{S}\bar{I}$ and $\bar{S}\bar{P}$ usually called as mass action transmission terms [10]. Other classical infection mechanisms are the standard incidence transmission term [9] and saturation incidence transmission term [7,31]. It is also interesting to note that the BRN for the model adopting mass action depends on the population size, while the BRN for the model adopting standard incidence mechanism does not depend on the population size [17]. There are plenty of works on reaction–diffusion cholera model related to model (1.1). In [3], the authors explored the diffusive cholera model with indirect transmission route and heterogeneous mixed population. In [28,39,40], the authors established the threshold-type results of cholera models with space-dependent parameter functions. The existence of traveling waves for diffusive cholera models was investigated in [41,42]. With a reaction–convection–diffusion system in a periodic environment, the spatiotemporal threshold dynamics of cholera was established in [36].

Inspired by the above-mentioned works, we shall revisit the model (1.1) with the following assumptions:

- *The total humans stabilize at $H(x)$, $x \in \Omega$.* Assume that the all populations $\bar{H}(x,t) = \bar{S} + \bar{I}$ remain confined to Ω for all time, perform an unbiased random walk and satisfy $\lim_{t\to\infty} \bar{H}(\cdot,t) = H(\cdot)$, that is, total humans stabilize at $H(x)$ without being altered by the cholera epidemic, see, for example, [2,13];
- *Only the mobility of humans is allowed.* To make things simple, we assume that the *Vibrio cholerae* is fixed in a water environment in Ω [32,33].

The main model of this paper takes the following form:

$$\begin{cases} \dfrac{\partial \bar{I}}{\partial t} = d_I \Delta \bar{I} + \alpha(x)(H(x) - \bar{I})\bar{I} + \beta(x)(H(x) - \bar{I})\bar{P} - b(x)\bar{I}, & (x,t) \in \Omega \times (0,\infty), \\ \dfrac{\partial \bar{P}}{\partial t} = c(x)\bar{I} - m(x)\bar{P}, & (x,t) \in \Omega \times (0,\infty), \\ \bar{I}(x,0) = \bar{I}^0(x) \ge 0, \ \ \bar{P}(x,0) = \bar{P}^0(x) \ge 0, & x \in \bar{\Omega}, \end{cases} \tag{1.3}$$

with boundary condition

$$\frac{\partial \bar{I}}{\partial \nu} = 0, \quad (x,t) \in \partial\Omega \times (0,\infty). \tag{1.4}$$

For model (1.3)–(1.4), establishing the well-posedness becomes a necessary job, and in Section 2, we will explore this topic. Section 3 shows threshold dynamics of model (1.3)–(1.4) consisting of the following subsections: Section 3.1 is devoted to studying BRN for model (1.3) and its properties; Section 3.2 provides the eigenvalue problem (EP) associated with (1.3) to obtain the relation between its principal eigenvalue (PE) and BRN; Sufficient condition for cholera eradication is provided in Section 3.3; Section 3.4 studies the existence and uniqueness of PSS to (1.3); Section 3.5 explores the extinction of cholera with LBRN. Section 3.6 illustrates the global attractivity of PSS when all parameters are constants. Based on (1.3), Section 4 investigates the threshold dynamics of model including distinct diffusion rates of humans and *Vibrio cholerae* to analyze the impact of diffusion rates on the transmission of cholera. Finally, the conclusion and discussion are presented.

2. Well-posedness of the problem

We make the following assumptions on each parameter in (1.3):

- $d_I > 0$.
- $H(\cdot), \alpha(\cdot), \beta(\cdot), b(\cdot), c(\cdot)$ and $m(\cdot)$ are strictly positive and Hölder continuous on $\bar{\Omega}$.

Moreover, we use the following symbols:

- Throughout of the paper, we shall write

$$\mathfrak{g}^* := \max_{x\in\bar{\Omega}}\{\mathfrak{g}(x)\} \text{ and } \mathfrak{g}_* := \min_{x\in\bar{\Omega}}\{\mathfrak{g}(x)\},$$

  where $\mathfrak{g} \in \{H, \alpha, \beta, b, c, m\}$. Moreover, for any $G \in L^1(\Omega)$, we shall write the average of G over Ω as $G^\sharp$, that is,

$$G^\sharp := \frac{\int_\Omega G(x)dx}{|\Omega|}.$$

- Let $\mathbb{X} := C(\bar{\Omega}, \mathbb{R}^2)$ be the Banach space of continuous functions, equipped with norm $\|\phi\|_{\mathbb{X}} := \max(\|\phi_1\|_\infty, \|\phi_2\|_\infty)$, where $\phi = (\phi_1, \phi_2)^{\mathrm{T}} \in \mathbb{X}$, T denotes the transpose of a vector and $\|\psi\|_\infty := \sup_{x\in\Omega} |\psi(x)|$ for any $\psi \in C(\bar{\Omega}, \mathbb{R})$. Let $\mathbb{X}^+ := C(\bar{\Omega}, \mathbb{R}^2_+)$ be the positive cone of $\mathbb{X}$.
- Denote by Γ the Green function of operator $d_I\Delta - b(\cdot)$ with (1.4).
- Let $\{T_1(t)\}_{t\ge0}$ and $\{T_2(t)\}_{t\ge0}$ be the strongly continuous semigroups of bounded linear operators on $C(\bar{\Omega}, \mathbb{R})$ to itself, generated by $d_I\Delta - b(\cdot)$ and $-m(\cdot)$ with suitable domains, respectively. That is, for $\varphi \in C(\bar{\Omega}, \mathbb{R}_+)$, $t \ge 0$ and $x \in \Omega$,

$$(T_1(t)\varphi)(x) = \int_\Omega \Gamma(t,x,y)\varphi(y)dy \quad \text{and} \quad (T_2(t)\varphi)(x) = e^{-m(x)t}\varphi(x).$$

- Let $\mathbb{X}_H \subset \mathbb{X}$ be defined by

$$\mathbb{X}_H := \left\{ \phi = \begin{pmatrix} \phi_1 \\ \phi_2 \end{pmatrix} \in \mathbb{X}^+ : 0 \le \phi_1 \le H(x) \text{ for all } x \in \bar{\Omega} \right\}.$$

Our first result concerns the local solution $u := (\bar{I}, \bar{P})^{\mathrm{T}}$ of (1.3) on $\mathbb{X}_H$.

Lemma 2.1. *For any $\phi \in \mathbb{X}_H$, there exists a $\tau_m = \tau_m(\phi) > 0$ such that (1.3) with (1.4) has a unique positive noncontinuable mild solution $u(\cdot, t) = u(\cdot, t; \phi)$ on $[0, \tau_m)$ with $u(\cdot, 0; \phi) = \phi$. Moreover, $u(\cdot, t; \phi) \in \mathbb{X}_H$ is a classical solution on $[0, \tau_m)$.*

Proof. For $\phi = (\phi_1, \phi_2)^{\mathrm{T}} \in \mathbb{X}$ and $t \ge 0$, let $T(t)\phi := (T_1(t)\phi_1, T_2(t)\phi_2)^{\mathrm{T}}$. We then see that $\{T(t)\}_{t\ge 0}$ is a strongly continuous semigroup of bounded linear operators (see also [20, Section 1.2]) on $\mathbb{X}$ to itself. The mild solution $u = (\bar{I}, \bar{P})^{\mathrm{T}}$ to problem (1.3) with (1.4) should satisfy

$$u(t) = T(t)\phi + \int_0^t T(t-s)F(u(\cdot, s))ds, \quad u(0) = \phi = \begin{pmatrix} \phi_1 \\ \phi_2 \end{pmatrix} = \begin{pmatrix} \bar{I}^0 \\ \bar{P}^0 \end{pmatrix} \in \mathbb{X}_H,$$

where $F : \mathbb{X}_H \to \mathbb{X}$ is defined by

$$F(\phi) := \begin{pmatrix} F_1(\phi) \\ F_2(\phi) \end{pmatrix} = \begin{pmatrix} \alpha(\cdot)(H(\cdot) - \phi_1)\phi_1 + \beta(\cdot)(H(\cdot) - \phi_1)\phi_2 \\ c(\cdot)\phi_1 \end{pmatrix}, \quad \phi = \begin{pmatrix} \phi_1 \\ \phi_2 \end{pmatrix} \in \mathbb{X}_H.$$

Note that F is Lipschitz. For each $\phi \in \mathbb{X}_H$ and $\ell \ge 0$, we have

$$\begin{aligned} \phi + \ell F(\phi) &= \begin{pmatrix} \phi_1 + \ell[\alpha(\cdot)(H(\cdot) - \phi_1)\phi_1 + \beta(\cdot)(H(\cdot) - \phi_1)\phi_2] \\ \phi_2 + \ell c(\cdot)\phi_1 \end{pmatrix} \\ &\ge \begin{pmatrix} \phi_1[1 - \ell(\alpha^*\phi_1 + \beta^*\phi_2)] \\ \phi_2 \end{pmatrix} \end{aligned}$$

and

$$H(\cdot) - (\phi_1 + \ell F_1(\phi)) = (H(\cdot) - \phi_1)[1 - \ell(\alpha(\cdot)\phi_1 + \beta(\cdot)\phi_2)].$$

Hence, it follows that

$$\lim_{\ell \to 0^+} \frac{1}{\ell} \mathrm{dist}(\phi + \ell F(\phi), \mathbb{X}_H) = 0 \quad \text{for all } \phi \in \mathbb{X}_H.$$

By [23, Theorem 3.1 in Chapter 7], (1.3) with (1.4) has a unique positive solution $u(\cdot, t; \phi)$ on $[0, \tau_m)$, where $0 < \tau_m \le \infty$. This proves Lemma 2.1. □

We next prove that the solution $u(\cdot, t; \phi)$ of (1.3) with (1.4) exists globally and there exists a connected global attractor.

Lemma 2.2. *For $\phi \in \mathbb{X}_H$ and $u(\cdot, 0; \phi) = \phi$, we then have:*

(i) The solution $u(\cdot, t; \phi)$ of (1.3) with (1.4) exists globally. Furthermore, $u(\cdot, t; \phi)$ is ultimately uniformly bounded.
(ii) The semiflow $\Psi(t) : \mathbb{X}_H \to \mathbb{X}_H$ $(t \ge 0)$ induced by the solution of (1.3)–(1.4), admits a connected global attractor on $\mathbb{X}_H$.

Proof. Let $N_0 := \|H(\cdot)\|_\infty > 0$. By Lemma 2.1, $\bar{I}(\cdot, t) \le N_0$ for all $(x, t) \in \Omega \times [0, \tau_m)$. Due to the comparison principle, we obtain that $\bar{P} \le P_1$ on $\bar{\Omega} \times [0, \tau_m)$, where P_1 is the solution of

$$\begin{cases} \dfrac{\partial P_1}{\partial t} = c^* N_0 - m_* P_1, & (x, t) \in \Omega \times (0, \tau_m), \\ P_1(x, 0) = \bar{P}^0(x), & x \in \bar{\Omega}. \end{cases} \tag{2.1}$$

Solving this problem yields

$$P_1(x, t) = \left[\bar{P}^0(x) - \frac{c^* N_0}{m_*} \right] e^{-m_* t} + \frac{c^* N_0}{m_*} \quad \text{for all } (x, t) \in \bar{\Omega} \times [0, \tau_m). \tag{2.2}$$

Hence, we have

$$\lim_{t\to\tau_m-0}\|u(\cdot,t)\|_{\mathbb{X}} \le \max\left(N_0,\ \|\bar{P}^0\|_\infty + \frac{c^*N_0}{m_*}\right) < \infty.$$

By [23, Theorem 3.1 (c) in Chapter 7], we see that $\tau_m = \tau_m(\phi) = \infty$ for all $\phi \in \mathbb{X}_H$. That is, the solution u exists globally. Moreover, by (2.2), we have

$$\lim_{t\to\infty}\|u(\cdot,t)\|_{\mathbb{X}} \le \max\left(N_0, \frac{c^*N_0}{m_*}\right) < \infty, \tag{2.3}$$

which implies that u is ultimately uniformly bounded. This proves (i).

By [23, Theorem 3.1 (d) in Chapter 7], the semiflow $\Psi(t) : \mathbb{X}_H \to \mathbb{X}_H$ $(t \ge 0)$ is induced by the solution u of (1.3)–(1.4): $\Psi(t)\phi = u(\cdot, t; \phi)$ with $u(\cdot, 0; \phi) = \phi \in \mathbb{X}_H$. Note that $\Psi(t)$ lacks the compactness due to the absence of the diffusion term in $\bar{P}$-equation of (1.3). We shall apply [8, Lemma 2.3.4] to show the asymptotic smoothness of $\Psi(t)$. To this end, we introduce the Kuratowski measure of non-compactness, that is, for any bounded $\mathbb{B} \subset \mathbb{X}_H$,

$$\kappa(\mathbb{B}) := \inf\{r : \mathbb{B} \text{ has a finite cover of diameter } < r\}. \tag{2.4}$$

In view of [38, Lemma 2.6], we first decompose $\Psi(t)$ as $\Psi(t) = \Psi_1(t) + \Psi_2(t)$, where

$$\Psi_1(t)\phi = \begin{pmatrix} \bar{I}(\cdot,t,\phi) \\ \int_0^t e^{-m(\cdot)(t-s)}c(\cdot)\bar{I}(\cdot,s,\phi)\mathrm{d}s \end{pmatrix} \quad \text{and} \quad \Psi_2(t)\phi = \begin{pmatrix} 0 \\ e^{-m(\cdot)t}\phi_2 \end{pmatrix}.$$

With the help of [38, Lemma 2.5], $\Psi_1(t)\mathbb{B}$ is precompact, and then $\kappa(\Psi_1(t)\mathbb{B}) = 0$. Moreover,

$$\|\Psi_2(t)\|_{\mathbb{X}} = \sup_{\phi\in\mathbb{X}} \frac{\|\Psi_2(t)\phi\|_{\mathbb{X}}}{\|\phi\|_{\mathbb{X}}} \le \sup_{\phi\in\mathbb{X}_H} \frac{\|\left(0, e^{-m_*t}\phi_2\right)^{\mathrm{T}}\|_{\mathbb{X}}}{\|\phi\|_{\mathbb{X}}} \le e^{-m_*t}, \quad t > 0,$$

and hence

$$\kappa(\Psi(t)\mathbb{B}) \le \kappa(\Psi_1(t)\mathbb{B}) + \kappa(\Psi_2(t)\mathbb{B}) \le \|\Psi_2(t)\|\kappa(\mathbb{B}) \le e^{-m_*t}\kappa(\mathbb{B}), \quad t > 0.$$

Therefore, $\Psi(t)$ satisfies the κ-contraction condition on $\mathbb{X}_H$ for all $t > 0$. Thus, by [8, Lemma 2.3.4], $\Psi(t)$ is asymptotically smooth. Moreover, by (2.3), $\Psi(t)$ is point dissipative. Hence, the existence of a connected global attractor in $\mathbb{X}_H$ is a consequence of [8, Theorem 2.4.6]. This proves (ii). □

3. Threshold dynamics

3.1. Basic reproduction number

In what follows, for an operator $\mathcal{L}$, denote $\sigma(\mathcal{L})$ the spectrum of $\mathcal{L}$ and denote

$$r(\mathcal{L}) = \sup\{|\lambda| : \lambda \in \sigma(\mathcal{L})\} \quad \text{and} \quad s(\mathcal{L}) = \sup\{\mathrm{Re}\lambda : \lambda \in \sigma(\mathcal{L})\}$$

the spectral radius and the spectral bound of $\mathcal{L}$, respectively.

Clearly, $Q_0 = (0, 0)^{\mathrm{T}} \in \mathbb{X}_H$ is the DFSS of (1.3) with (1.4). Let $\mathcal{A} := \mathcal{B} + \mathcal{F}$, where

$$\mathcal{B} = \begin{pmatrix} d_I\Delta - b(\cdot) & 0 \\ c(\cdot) & -m(\cdot) \end{pmatrix} \quad \text{and} \quad \mathcal{F} = \begin{pmatrix} \alpha(\cdot)H(\cdot) & \beta(\cdot)H(\cdot) \\ 0 & 0 \end{pmatrix}.$$

Linearizing (1.3) around Q_0 gives the following cooperative system:

$$\begin{cases} \begin{pmatrix} \frac{\partial \bar{I}}{\partial t} \\ \frac{\partial \bar{P}}{\partial t} \end{pmatrix} = \mathcal{A}\begin{pmatrix} \bar{I} \\ \bar{P} \end{pmatrix}, & (x,t) \in \Omega \times (0,\infty), \\ \dfrac{\partial \bar{I}}{\partial \nu} = 0, & (x,t) \in \partial\Omega \times (0,\infty). \end{cases} \tag{3.1}$$

Let $\Phi(t)$ (resp. $\bar{\Phi}(t)$) be the semigroup generated by $\mathcal{A}$ (resp. $\mathcal{B}$). Note that $\mathcal{B}$ can be decomposed as $\mathrm{diag}(d_I\Delta, 0) - V$, where

$$V = \begin{pmatrix} b(\cdot) & 0 \\ -c(\cdot) & m(\cdot) \end{pmatrix}.$$

Thanks to the fact that $\mathcal{B}$ and $-V$ are cooperative, one knows that $\Phi(t)$ and $\bar{\Phi}(t)$ are positive semigroups. With the aid of [26, Theorem 3.12], we see that both $\mathcal{A}$ and $\mathcal{B}$ are resolvent-positive. Moreover, since $s(\mathcal{B}) < 0$, we can define the following positive operator on $\mathbb{X}$,

$$L(\phi)(x) := \mathcal{F}(-\mathcal{B})^{-1}\phi(x) = \int_0^\infty \mathcal{F}(x)\bar{\Phi}(t)\phi(x)dt = \mathcal{F}(x)\int_0^\infty \bar{\Phi}(t)\phi(x)dt, \quad \phi \in \mathbb{X},$$

which is called the NGO. Following the classical definition (see, e.g., [26,35]), the BRN for model (1.3) is defined by

$$\Re_0^{(1.3)} := r(L). \tag{3.2}$$

The following result comes from [26, Theorem 3.5].

Lemma 3.1. $\Re_0^{(1.3)} - 1$ *has the same sign as* $s(\mathcal{A})$.

Furthermore, we study the relation between $\Re_0^{(1.3)}$ and the PE of the EP related to (1.3)–(1.4).

Lemma 3.2. *We have the following statements:*

(i) Let $\bar{\lambda}_0$ *be the PE of the EP*

$$\begin{cases} d_I\Delta\psi - b(\cdot)\psi + \bar{\lambda}\left(\alpha(\cdot)H(\cdot) + \dfrac{c(\cdot)\beta(\cdot)H(\cdot)}{m(\cdot)}\right)\psi = 0, & x \in \Omega, \\ \dfrac{\partial\psi}{\partial\nu} = 0, & x \in \partial\Omega, \\ \psi \in C^2(\Omega, \mathbb{R}^n) \cap C^1(\overline{\Omega}, \mathbb{R}^n). \end{cases} \tag{3.3}$$

Then, $\Re_0^{(1.3)} = 1/\bar{\lambda}_0$.

(ii) Let $\bar{\eta}_0$ *be the PE of the EP*

$$\begin{cases} d_I\Delta\phi - b(\cdot)\phi + \left(\alpha(\cdot)H(\cdot) + \dfrac{c(\cdot)\beta(\cdot)H(\cdot)}{m(\cdot)}\right)\phi = \bar{\eta}\phi, & x \in \Omega, \\ \dfrac{\partial\phi}{\partial\nu} = 0, & x \in \partial\Omega, \\ \phi \in C^2(\Omega, \mathbb{R}^n) \cap C^1(\overline{\Omega}, \mathbb{R}^n). \end{cases} \tag{3.4}$$

Then, $\Re_0^{(1.3)} - 1$ *has the same sign as* $\bar{\eta}_0$.

Proof. We first prove (i). As in [35, Theorem 3.3], we rewrite $\mathcal{F}$ and V as

$$\mathcal{F} = \begin{pmatrix} F_{11} & F_{12} \\ F_{21} & F_{22} \end{pmatrix} \text{ and } V = \begin{pmatrix} V_{11} & V_{12} \\ V_{21} & V_{22} \end{pmatrix},$$

where

$$F_{11} := \alpha(\cdot)H(\cdot),\ F_{12} := \beta(\cdot)H(\cdot),\ F_{21} := 0,\ F_{22} := 0,$$
$$V_{11} := b(\cdot),\ V_{12} := 0,\ V_{21} := -c(\cdot),\ V_{22} := m(\cdot).$$

Then, by [35, Theorem 3.2 and Theorem 3.3 (ii)], we have $\Re_0^{(1.3)} = r(-\mathcal{F}\mathcal{B}^{-1}) = r(-\mathcal{B}^{-1}\mathcal{F}) = r(-\mathcal{B}_1^{-1}\mathcal{F}_2)$ due to $F_{21} = 0$ and $F_{22} = 0$, where

$$\mathcal{B}_1 := d_I\Delta - (V_{11} - V_{12}V_{22}^{-1}V_{21}) = d_I\Delta - b(\cdot)$$

and

$$\mathcal{F}_2 := F_{11} - F_{12}V_{22}^{-1}V_{21} = \alpha(\cdot)H(\cdot) + \frac{c(\cdot)\beta(\cdot)H(\cdot)}{m(\cdot)}.$$

It follows that

$$(-\mathcal{B}_1^{-1}\mathcal{F}_2)\psi = -[d_I\Delta - b(\cdot)]^{-1}\left(\alpha(\cdot)H(\cdot) + \frac{c(\cdot)\beta(\cdot)H(\cdot)}{m(\cdot)}\right)\psi,$$

where $\psi \in C^2(\Omega, \mathbb{R}^n) \cap C^1(\bar{\Omega}, \mathbb{R}^n)$. As $\Re_0^{(1.3)} = r(-\mathcal{B}_1^{-1}\mathcal{F}_2)$, we have

$$-[d_I \Delta - b(\cdot)]^{-1}\left(\alpha(\cdot)H(\cdot) + \frac{c(\cdot)\beta(\cdot)H(\cdot)}{m(\cdot)}\right)\psi = \Re_0^{(1.3)}\psi.$$

That is,

$$d_I \Delta\psi - b(\cdot)\psi + \left(\alpha(\cdot)H(\cdot) + \frac{c(\cdot)\beta(\cdot)H(\cdot)}{m(\cdot)}\right)\frac{1}{\Re_0^{(1.3)}}\psi = 0. \tag{3.5}$$

This proves (i).

We next prove (ii). Obviously, the EP (3.4) admits a least eigenvalue $\bar{\eta}_0$ with a positive eigenfunction. The assertion can be then proved just as in the proof of [1, Lemma 2.3(d)]. This proves (ii). □

Remark 3.1 (*see also [1, Lemma 2.3]*)**.** By variational formula and Lemma 3.2, we have

$$\Re_0^{(1.3)} = \frac{1}{\bar{\lambda}_0} = \sup_{\psi \in H^1(\Omega),\ \psi \neq 0} \left\{ \frac{\int_\Omega \left(\alpha(\cdot)H(\cdot) + \frac{c(\cdot)\beta(\cdot)H(\cdot)}{m(\cdot)}\right)\psi^2 \mathrm{d}x}{\int_\Omega \left(d_I |\nabla\psi|^2 + b(\cdot)\psi^2\right)\mathrm{d}x} \right\}. \tag{3.6}$$

Similar to [1, Theorem 2] (see also [38]), together with (3.6), we have the following result on the relation between $\Re_0^{(1.3)}$ and d_I.

Theorem 3.1. *Let $\Re_0^{(1.3)}$ be defined in (3.6), we then have*

(i) $\Re_0^{(1.3)}$ is decreasing in d_I,

$$\lim_{d_I \to 0} \Re_0^{(1.3)} = \max_{x\in\Omega} \left\{ \frac{\alpha(\cdot)H(\cdot)m(\cdot) + c(\cdot)\beta(\cdot)H(\cdot)}{m(\cdot)b(\cdot)} \right\}$$

and

$$\lim_{d_I \to \infty} \Re_0^{(1.3)} = \frac{\int_\Omega \left(\alpha(\cdot)H(\cdot) + \frac{c(\cdot)\beta(\cdot)H(\cdot)}{m(\cdot)}\right)\mathrm{d}x}{\int_\Omega b(\cdot)\mathrm{d}x}.$$

(ii) If $\int_\Omega \left(\alpha(\cdot)H(\cdot) + \frac{c(\cdot)\beta(\cdot)H(\cdot)}{m(\cdot)}\right)\mathrm{d}x > \int_\Omega b(\cdot)\mathrm{d}x$, then $\Re_0^{(1.3)} > 1$ for all $d_I > 0$.

(iii) If $\int_\Omega \left(\alpha(\cdot)H(\cdot) + \frac{c(\cdot)\beta(\cdot)H(\cdot)}{m(\cdot)}\right)\mathrm{d}x < \int_\Omega b(\cdot)\mathrm{d}x$, at the same time, $\alpha(\cdot)H(\cdot)m(\cdot) + c(\cdot)\beta(\cdot)H(\cdot) > m(\cdot)b(\cdot)$ holds for some $x \in \Omega$, we then obtain that there exists a $\bar{d}_I$ such that $\Re_0^{(1.3)} > 1$ when $d_I < \bar{d}_I$, and $\Re_0^{(1.3)} < 1$ when $d_I > \bar{d}_I$.

3.2. Principal eigenvalue

In this subsection, we pay our attention to the following EP,

$$\begin{cases} \lambda \begin{pmatrix} \psi_1 \\ \psi_2 \end{pmatrix} = \mathcal{A}\begin{pmatrix} \psi_1 \\ \psi_2 \end{pmatrix}, & x \in \Omega, \\ \frac{\partial \psi_1}{\partial \nu} = 0, & x \in \partial\Omega, \\ \psi_1 \in C^2(\Omega, \mathbb{R}) \cap C^1(\bar{\Omega}, \mathbb{R}),\ \ \psi_2 \in C(\bar{\Omega}, \mathbb{R}). \end{cases} \tag{3.7}$$

Lemma 3.3. *If $\Re_0^{(1.3)} \geq 1$, then $s(\mathcal{A})$ is the PE of (3.7).*

Proof. According to the similar technique in (ii) of Lemma 2.2 and (3.1), $\Phi(t)$, which is the semigroup generated by $\mathcal{A}$, satisfies κ-contraction condition on $\mathbb{X}_H$. Hence, the essential growth bound and the essential spectral radius of $\Phi(t)$, written by $\omega_{ess}(\Phi)$ and $r_e(\Phi(t))$, respectively, fulfill

$$\omega_{ess}(\Phi) \leq -m_* \ \text{ and } \ r_e(\Phi(t)) \leq e^{-m_* t} < 1 \ \text{ for all } t > 0.$$

Here $\omega_{ess}(\Phi) := \lim_{t\to\infty} \frac{\ln\alpha(\Phi(t))}{t}$ and $\alpha(\mathcal{L})$ is the measure of non-compactness of bounded linear operator $\mathcal{L}$ on $\mathbb{X}_H$ defined by

$$\alpha(\mathcal{L}) := \inf\{\varepsilon > 0 : \kappa(\mathcal{L}\mathbb{B}) \le \varepsilon\kappa(\mathbb{B}),\ \mathbb{B} \subset \mathbb{X}_H \text{ is bounded}\},$$

where $\kappa(\cdot)$ denotes the Kuratowski measure of non-compactness defined by (2.4). By Lemma 3.1, we know that $s(\mathcal{A}) \ge 0$ when $\Re_0^{(1.3)} \ge 1$. Hence, $r(\Phi(t)) = e^{s(\mathcal{A})t} \ge 1$ for all $t > 0$, which tells us that $r_e(\Phi(t)) < r(\Phi(t))$ for all $t > 0$. By the generalized Krein–Rutman Theorem [19], we see that there exists a positive non-zero $\psi^0 = (\psi_1^0, \psi_2^0)^{\mathrm{T}} \in \mathbb{X}_H$ such that

$$\Phi(t)\psi^0 = r(\Phi(t))\psi^0 = e^{s(\mathcal{A})t}\psi^0, \quad t > 0.$$

The assertion holds by differentiating both sides of this equation. This proves Lemma 3.3. □

Lemma 3.4. *Suppose $m(x) \equiv m$. Then $s(\mathcal{A})$ is the PE of (3.7).*

Proof. Let $L_\lambda = d_I\Delta + \alpha(\cdot)H(\cdot) + \beta(\cdot)c(\cdot)H(\cdot)/(\lambda + m) - b(\cdot)$, $\lambda > -m$ with Neumann boundary condition. We note that $s(L_\lambda)$ is decreasing with respect to λ.

Denote $C_1 := \min_{x\in\bar{\Omega}}\{\alpha(\cdot)H(\cdot)\}$ and $C_2 := \min_{x\in\bar{\Omega}}\{\beta(\cdot)c(\cdot)H(\cdot)\}$. It is easily seen that the scalar equation

$$\begin{cases} \eta\varphi = d_I\Delta\varphi - b(\cdot)\varphi, & x \in \Omega, \\ \dfrac{\partial\varphi}{\partial\nu} = 0, & x \in \partial\Omega, \end{cases}$$

admits a PE, written by $\hat{\eta}$, with eigenfunction $\varphi^0 \gg 0$. Denote by $\hat{\lambda} = \frac{1}{2}[(\hat{\eta} - m + C_1) + \sqrt{(m + C_1 + \hat{\eta})^2 + 4C_2}]$ the larger solution of

$$\lambda^2 + (m - C_1 - \hat{\eta})\lambda - (C_2 + m(C_1 + \hat{\eta})) = 0.$$

Hence, $\hat{\lambda} > -m$. Obviously,

$$L_{\hat{\lambda}}\varphi^0 = d_I\Delta\varphi^0 + \alpha(\cdot)H(\cdot)\varphi^0 + \frac{\beta(\cdot)c(\cdot)H(\cdot)}{\hat{\lambda} + m}\varphi^0 - b(\cdot)\varphi^0 \ge (\hat{\eta} + C_1 + \frac{C_2}{\hat{\lambda} + m})\varphi^0 = \hat{\lambda}\varphi^0.$$

An application of [29, Lemma 2.6] and [35, Theorem 2.3] gives the assertion. This proves Lemma 3.4. □

3.3. Cholera extinction

We now prove the global asymptotic stability of the DFSS for $\Re_0^{(1.3)} < 1$.

Theorem 3.2. *If $\Re_0^{(1.3)} < 1$, then the DFSS $Q_0 = (0, 0)^{\mathrm{T}}$ is globally asymptotically stable in $\mathbb{X}_H$.*

Proof. The local stability of Q_0 directly follows from [35, Theorem 3.1]. We next study the global attractivity of Q_0. By the comparison principle [16], we obtain $(\bar{I}, \bar{P}) \le (\hat{I}, \hat{P})$ on $\bar{\Omega} \times [0, \infty)$, where $(\hat{I}, \hat{P})$ fulfills

$$\begin{cases} \begin{pmatrix} \frac{\partial\hat{I}}{\partial t} \\ \frac{\partial\hat{P}}{\partial t} \end{pmatrix} = \mathcal{A}\begin{pmatrix} \hat{I} \\ \hat{P} \end{pmatrix}, & (x, t) \in \Omega \times (0, \infty), \\ \hat{I}(x, 0) = \bar{I}^0(x), \quad \hat{P}(x, 0) = \bar{P}^0(x), & x \in \bar{\Omega}, \\ \dfrac{\partial\hat{I}}{\partial\nu} = 0, & (x, t) \in \partial\Omega \times (0, \infty). \end{cases} \tag{3.8}$$

Let $\omega_0 = \omega_0(\Phi)$ be the growth bound of Φ such that, for some $M \ge 1$,

$$\|\Phi(t)\|_{\mathrm{op}} \le Me^{\omega_0 t} \text{ for all } t \ge 0,$$

where $\|\cdot\|_{\mathrm{op}}$ denotes the operator norm. Note that $\omega_0 = \max\{s(\mathcal{A}), \omega_{ess}(\Phi)\}$. From Lemma 3.1, $s(\mathcal{A}) < 0$. Moreover, as shown in the proof of Lemma 3.3, $\omega_{ess}(\Phi) \le -m_* < 0$. Hence, we have $\omega_0 < 0$, which tells us that $(\hat{I}, \hat{P}) \to (0, 0)$ uniformly on $x \in \bar{\Omega}$ as $t \to \infty$. Accordingly, $(\bar{I}, \bar{P}) \to (0, 0)$ as $t \to \infty$ uniformly on $x \in \bar{\Omega}$. This completes the proof of Theorem 3.2. □

3.4. Existence and uniqueness of PSS

The next theorem states the uniform persistence of system (1.3) and the existence of PSS for $\Re_0^{(1.3)} > 1$.

Theorem 3.3. *If $\Re_0^{(1.3)} > 1$, then there exists a $\varsigma > 0$ such that the solution $(\bar{I}, \bar{P})^{\mathrm{T}}$ of (1.3)–(1.4) with any positive non-zero initial value $(\bar{I}^0, \bar{P}^0)^{\mathrm{T}} \in \mathbb{X}_H$ fulfills*

$$\min\left(\liminf_{t\to\infty} \bar{I}(x,t),\ \liminf_{t\to\infty} \bar{P}(x,t)\right) \geq \varsigma \quad \text{for all } x \in \bar{\Omega}. \tag{3.9}$$

Furthermore, the problem (1.3)–(1.4) has at least one PSS, denoted by $\bar{Q}^{ss} = (\bar{I}^{ss}, \bar{P}^{ss})^{\mathrm{T}} \in \mathbb{X}_H$.

Proof. Set

$$\mathbb{X}_{H0} := \left\{\phi = \begin{pmatrix} \phi_1 \\ \phi_2 \end{pmatrix} \in \mathbb{X}_H :\ \phi_1(\cdot) \not\equiv 0 \ \text{ and } \ \phi_2(\cdot) \not\equiv 0 \right\},$$

and

$$\partial\mathbb{X}_{H0} := \mathbb{X}_H \setminus \mathbb{X}_{H0} = \left\{\phi = \begin{pmatrix} \phi_1 \\ \phi_2 \end{pmatrix} \in \mathbb{X}_H :\ \phi_1(\cdot) \equiv 0 \ \text{ and/or } \ \phi_2(\cdot) \equiv 0 \right\}.$$

Then $\mathbb{X}_H = \mathbb{X}_{H0} \cup \partial\mathbb{X}_{H0}$. Let $M_\partial := \{\phi \in \partial\mathbb{X}_{H0} : \Psi(t)\phi \in \partial\mathbb{X}_{H0}$ for all $t \geq 0\}$. Clearly, $\Psi(t)\mathbb{X}_{H0} \subseteq \mathbb{X}_{H0}$ for all $t \geq 0$. In fact, suppose that $(\bar{I}^0, \bar{P}^0)^{\mathrm{T}} \in \mathbb{X}_{H0}$. By $\partial\bar{I}/\partial t \geq d_I \Delta \bar{I} - b(x)\bar{I}$, one knows that $\bar{I}$ is an upper solution of

$$\begin{cases} \dfrac{\partial \check{I}}{\partial t} = d_I \Delta \check{I} - b(\cdot)\check{I}, & (x,t) \in \Omega \times (0,\infty), \\ \dfrac{\partial \check{I}}{\partial \nu} = 0, & (x,t) \in \partial\Omega \times (0,\infty), \\ \check{I}(\cdot,0) = \bar{I}(\cdot,0) = \bar{I}^0, & x \in \bar{\Omega}. \end{cases} \tag{3.10}$$

By appealing to the maximum principle and $\bar{I}^0 \not\equiv 0$, $\check{I}(x,t) > 0$ for all $(x,t) \in \bar{\Omega} \times (0,\infty)$. Further from the comparison principle, $\bar{I} \geq \check{I} > 0$ for all $(x,t) \in \bar{\Omega} \times (0,\infty)$. On the other hand, the $\bar{P}$-equation of (1.3) fulfills

$$\bar{P} = e^{-m(\cdot)t}\bar{P}^0(\cdot) + \int_0^t e^{-m(\cdot)(t-s)} c(\cdot)\bar{I}(\cdot,s)\mathrm{d}s, \tag{3.11}$$

which tells us that $\bar{P} > 0$ for all $(x,t) \in \bar{\Omega} \times (0,\infty)$. Therefore, $\bar{I}(\cdot,t) \neq 0$ and $\bar{P}(\cdot,t) \neq 0$ for all $t > 0$, which implies that $\Psi(t)\mathbb{X}_{H0} \subseteq \mathbb{X}_{H0}$ for all $t \geq 0$.

Let $\omega(\phi)$ be the ω-limit set for orbit $\{\Psi(t)\phi : t \geq 0\}$, $\phi \in \mathbb{X}_{H0}$ defined by $\omega(\phi) := \cap_{t\geq 0}\overline{\cup_{s\geq t}\Psi(s)\phi}$. As for $\phi \in M_\partial$, we know that $\bar{I} \equiv 0$ and/or $\bar{P} \equiv 0$. If $\bar{I} \equiv 0$, then we have from the first equation in (1.3) that $0 \equiv \beta(x)H(x)\bar{P}$, and thus, $\bar{P} \equiv 0$. On the other hand, if $\bar{P} \equiv 0$, then we have from the second equation in (1.3) that $0 \equiv c(x)\bar{I}$, and thus, $\bar{I} \equiv 0$. This yields $\cup_{\phi\in M_\partial}\omega(\phi) = \{Q_0\}$. We then see that $\{Q_0\}$ is an isolated and compact invariant set for Ψ restricted in M_∂.

By $\Re_0^{(1.3)} > 1$ and Lemma 3.3, one knows that $\lambda^0 > 0$, where λ^0 is the PE of (3.7). Therefore, there is a sufficiently small $\epsilon_1 > 0$ such that $\lambda^0_{\epsilon_1} > 0$, where $\lambda^0_{\epsilon_1}$ is the PE of

$$\begin{cases} \lambda \begin{pmatrix} \psi_1 \\ \psi_2 \end{pmatrix} = \mathcal{A}_{\epsilon_1}\begin{pmatrix} \psi_1 \\ \psi_2 \end{pmatrix}, & x \in \Omega, \\ \dfrac{\partial \psi_1}{\partial \nu} = 0, & x \in \partial\Omega, \\ \psi_1 \in C^2(\Omega,\mathbb{R}) \cap C^1(\bar{\Omega},\mathbb{R}),\ \ \psi_2 \in C(\bar{\Omega},\mathbb{R}), & \end{cases} \tag{3.12}$$

where

$$\mathcal{A}_{\epsilon_1} = \begin{pmatrix} d_I\Delta - b(\cdot) + \alpha(\cdot)(H(\cdot)-\epsilon_1) & \beta(\cdot)(H(\cdot)-\epsilon_1) \\ c(\cdot) & -m(\cdot) \end{pmatrix}.$$

In the sequel, we are going to show that $\limsup_{t\to\infty}\|\Psi(t)\phi - Q_0\|_{\mathbb{X}} \geq \epsilon_1$ for all $\phi \in \mathbb{X}_{H0}$, that is, $\{Q_0\}$ is a uniform weak repeller. We proceed indirectly and suppose that $\limsup_{t\to\infty}\|\Psi(t)\phi - Q_0\|_{\mathbb{X}} < \epsilon_1$ for the fixed $\epsilon_1 > 0$. Thus, there exists a $t_3 > 0$ such that $0 < \bar{I}(x,t) < \epsilon_1$ and $0 < \bar{P}(x,t) < \epsilon_1$ for all $x \in \Omega$ and $t \geq t_3$. Further we have $H(x) - \bar{I}(x,t) > H(x) - \epsilon_1$ for all $x \in \Omega$ and $t \geq t_3$. Let $(\phi_1^{\epsilon_1}(\cdot), \phi_2^{\epsilon_1}(\cdot))$ be the positive eigenvector corresponding to $\lambda^0_{\epsilon_1}$ in (3.12). Hence, there exists a number $C^* > 0$ such that $C^*(\phi_1^{\epsilon_1}(\cdot), \phi_2^{\epsilon_1}(\cdot)) \leq (\bar{I}(\cdot, t_3; \phi), \bar{P}(\cdot, t_3; \phi))$. Consequently, $(\bar{I}, \bar{P})$ is the upper solution of

$$\begin{cases} \begin{pmatrix} \frac{\partial \check{I}}{\partial t} \\ \frac{\partial \check{P}}{\partial t} \end{pmatrix} = \mathcal{A}_{\epsilon_1} \begin{pmatrix} \check{I} \\ \check{P} \end{pmatrix}, & (x,t) \in \Omega \times (t_3, \infty), \\ \dfrac{\partial \check{I}}{\partial \nu} = 0, & (x,t) \in \partial\Omega \times (t_3, \infty), \\ (\check{I}(\cdot, t_3), \check{P}(\cdot, t_3)) = C^*(\phi_1^{\epsilon_1}(\cdot), \phi_2^{\epsilon_1}(\cdot)), & x \in \bar{\Omega}. \end{cases} \tag{3.13}$$

Note that

$$(\check{I}, \check{P}) = C^* e^{\lambda^0_{\epsilon_1}(t-t_3)}(\phi_1^{\epsilon_1}(x), \phi_2^{\epsilon_1}(x))$$

is the unique solution to (3.13). An application of the comparison principle obtains $(\bar{I}, \bar{P}) \geq (\check{I}, \check{P})$. Consequently, $\bar{I} \to \infty$ and $\bar{P} \to \infty$ as $t \to \infty$, a contradiction against Lemma 2.2, and so $\{Q_0\}$ is indeed a uniform weak repeller.

To apply [24, Theorem 3], we define a continuous function $\rho : \mathbb{X}_H \to \mathbb{R}_+$ by

$$\rho(\phi) = \min\{\min_{x\in\bar{\Omega}} \phi_1(x), \min_{x\in\bar{\Omega}} \phi_2(x)\}, \quad \phi = \begin{pmatrix} \phi_1 \\ \phi_2 \end{pmatrix} \in \mathbb{X}_H.$$

Note that $\rho(\Psi(t)\phi) > 0$ for all $t > 0$ either if $\rho(\phi) > 0$ or if $\rho(\phi) = 0$ and $\phi \in \mathbb{X}_{H0}$. That is, ρ is a generalized distance function for Ψ. The above discussions imply that $\cup_{\phi\in\partial\mathbb{X}_{H0}} \omega(\phi) = \{Q_0\}$ and $W^s(Q_0) \cap \rho^{-1}(0, \infty) = \emptyset$, where $W^s(Q_0)$ is the stable set of Q_0 defined by

$$W^s(Q_0) := \left\{\phi \in \mathbb{X}_H : \lim_{t\to\infty} \|\Psi(t)\phi - Q_0\|_{\mathbb{X}} = 0\right\}.$$

Moreover, $\{Q_0\}$ is an isolated invariant set in $\mathbb{X}_H$ and no subset of $\{Q_0\}$ forms a cycle in $\partial\mathbb{X}_{H0}$. Therefore, by [24, Theorem 3], we see that there exists a $\varsigma > 0$ such that

$$\min_{\phi\in\mathbb{L}} \rho(\phi) > \varsigma,$$

where $\mathbb{L} \subset \mathbb{X}_H \setminus \{Q_0\}$ is an any compact chain transitive set. This implies that (4.12) holds for any non-zero initial value $(\bar{I}^0, \bar{P}^0)^{\mathrm{T}} \in \mathbb{X}_H$.

Combining with [15, Theorem 4.7], we conclude that (1.3) has at least one PSS $\bar{Q}^{ss}$ in $\mathbb{X}_{H0}$. This proves Theorem 3.3. □

Note that Theorem 3.3 only ensures the existence of PSS of (1.3). We are now in a position to explore the uniqueness of PSS. Here the PSS is the positive solution of the following elliptic problem:

$$\begin{cases} d_I \Delta I + \alpha(\cdot)(H(\cdot) - I)I + \beta(\cdot)(H(\cdot) - I)P - b(\cdot)I = 0, & x \in \Omega, \\ c(\cdot)I - m(\cdot)P = 0, & x \in \Omega, \\ \dfrac{\partial I}{\partial \nu} = 0, & x \in \partial\Omega. \end{cases} \tag{3.14}$$

By using $P = c(\cdot)I/m(\cdot)$, and canceling it in the first equation of (3.14), one can get

$$\begin{cases} d_I \Delta I + \left(\alpha(\cdot) + \dfrac{\beta(\cdot)c(\cdot)}{m(\cdot)}\right)\left(H(\cdot) - I\right)I - b(\cdot)I = 0, & x \in \Omega, \\ \dfrac{\partial I}{\partial \nu} = 0, & x \in \partial\Omega. \end{cases} \tag{3.15}$$

In what follows, we will focus on (3.15) instead of (3.14). We shall revisit the EP (3.4). The following Lemma will be used in the forthcoming discussions, which comes from [1].

Lemma 3.5. *For $h \in L^\infty(\Omega)$ and $d > 0$, let $\lambda_0(d, h)$ be the PE of*

$$\begin{cases} d\Delta\phi + h(\cdot)\phi = \lambda\phi, & x \in \Omega, \\ \dfrac{\partial \phi}{\partial \nu} = 0, & x \in \partial\Omega, \\ \phi \in C^2(\Omega, \mathbb{R}^n) \cap C^1(\bar{\Omega}, \mathbb{R}^n). \end{cases} \tag{3.16}$$

Then,

(i) $\lambda_0(d,h)=\sup\left\{\int_\Omega(h(\cdot)\phi^2-d|\nabla\phi|^2)\mathrm{d}x : \phi\in H^1(\Omega) \text{ with } \int_\Omega\phi^2\mathrm{d}x=1\right\}$*, which depends continuously on d and h;*

(ii) $\lambda_0(d,h)$ *is increasing in h, i.e., if* $h_1\geq h_2$ *in* Ω*, then* $\lambda_0(d,h_1)\geq\lambda_0(d,h_2)$ *with equality holds iff* $h_1=h_2$*, a.e. in* Ω*;*

(iii) $\lambda_0(d,h)$ *is decreasing in d and fulfills*

$$\lim_{d\to 0}\lambda_0(d,h)=\max\{h(\cdot):\ x\in\bar{\Omega}\} \text{ and } \lim_{d\to\infty}\lambda_0(d,h)=h^\sharp,$$

where $h^\sharp=\int_\Omega h(x)\mathrm{d}x/|\Omega|$.

In the sequel, we rewrite the PE of (3.4) as

$$\Upsilon_0=\bar{\eta}_0\left(d_I,\left(\alpha(\cdot)+\frac{\beta(\cdot)c(\cdot)}{m(\cdot)}\right)H(\cdot)-b(\cdot)\right). \tag{3.17}$$

According to Lemma 3.2, we then directly have: If $\Re_0^{(1.3)}<1$ then $\Upsilon_0<0$; If $\Re_0^{(1.3)}>1$ then $\Upsilon_0>0$.

Theorem 3.4. *Consider the elliptic problem (3.15). If* $\Upsilon_0<0$*, then (3.15) has no positive solution, while (3.15) admits a unique positive solution if* $\Upsilon_0>0$*.*

Proof. We proceed with the proof of the first assertion indirectly and suppose that (3.15) has a positive solution I when $\Upsilon_0<0$. We multiply both sides of (3.15) by I, and then integrate it over Ω, obtaining that

$$-d_I\int_\Omega|\nabla I|^2\mathrm{d}x+\int_\Omega\left(\left(\alpha(\cdot)+\frac{\beta(\cdot)c(\cdot)}{m(\cdot)}\right)(H(\cdot)-I)-b(\cdot)\right)I^2\mathrm{d}x=0,$$

which implies

$$-d_I\int_\Omega|\nabla I|^2\mathrm{d}x+\int_\Omega\left(\left(\alpha(\cdot)+\frac{\beta(\cdot)c(\cdot)}{m(\cdot)}\right)H(\cdot)-b(\cdot)\right)I^2\mathrm{d}x>0. \tag{3.18}$$

By the variational formula and (3.18), we know

$$\Upsilon_0\geq\left(-d_I\int_\Omega|\nabla I|^2\mathrm{d}x+\int_\Omega\left(\left(\alpha(\cdot)+\frac{\beta(\cdot)c(\cdot)}{m(\cdot)}\right)H(\cdot)-b(\cdot)\right)I^2\mathrm{d}x\right)\Big/\int_\Omega I^2\mathrm{d}x>0,$$

a contradiction. Therefore, (3.15) has no positive solution if $\Upsilon_0\leq 0$.

We next prove the second assertion. Denote by $\tilde{\phi}>0$ the eigenfunction of Υ_0. Denote

$$\mathbb{F}(I)=d_I\Delta I+\left(\left(\alpha(\cdot)+\frac{\beta(\cdot)c(\cdot)}{m(\cdot)}\right)(H(\cdot)-I)-b(\cdot)\right)I.$$

Let $I_1=\epsilon_2\tilde{\phi}$ with $\epsilon_2>0$. Due to $\Upsilon_0>0$, one knows that

$$\begin{aligned}\mathbb{F}(I_1)&=\epsilon_2 d_I\Delta\tilde{\phi}+\left(\left(\alpha(\cdot)+\frac{\beta(\cdot)c(\cdot)}{m(\cdot)}\right)\left(H(\cdot)-\epsilon_2\tilde{\phi}\right)-b(\cdot)\right)\epsilon_2\tilde{\phi}\\&=\epsilon_2\left(\Upsilon_0-\left(\alpha(\cdot)+\frac{\beta(\cdot)c(\cdot)}{m(\cdot)}\right)\epsilon_2\tilde{\phi}\right)\tilde{\phi}>0,\end{aligned}$$

if ϵ_2 is small, which tells us that I_1 is the lower solution of (3.15). Let $I_2=\bar{M}>0$ being sufficiently large. Directly checking gives $\mathbb{F}(I_2)<0$, which implies I_2 is an upper solution of (3.15). Consequently, (3.15) has at least one solution in $[I_1,I_2]$ by the method of upper/lower solutions, which is positive.

If (3.15) has two positive solutions, denoted by I' and I''. Based on the above discussions, by choosing small enough ϵ_2 and large enough $\bar{M}$, we directly have $I',I''\in[I_1,I_2]$. Again from the method of upper/lower solutions, we know that in $[I_1,I_2]$, there are a minimal solution and a maximal solution, denoted by I_m and I_M, respectively, satisfying $I_m\leq I',I''\leq I_M$. Here I_m and I_M are the positive solutions of (3.15). We now multiply both sides of (3.15) with $I=I_m$ by I_M, and $I=I_M$ by I_m respectively, and apply subtraction on the two resulting equations, obtaining that $0=\int_\Omega\left(\alpha(x)+\frac{\beta(x)c(x)}{m(x)}\right)I_mI_M\left(I_M-I_m\right)\mathrm{d}x$. It tells us that $I_M=I_m$ as $I_M\geq I_m$. Therefore, $I'=I''$, i.e., the existence and uniqueness of PSS are ensured when $\Upsilon_0>0$ (resp. $\Re_0^{(1.3)}>1$, from Lemma 3.2). This proves Theorem 3.4. □

3.5. The local basic reproduction number

When there is no diffusion term in (1.3), that is,

$$\begin{cases} \dfrac{\partial \bar{I}}{\partial t} = \alpha(x)(H(x) - \bar{I})\bar{I} + \beta(x)(H(x) - \bar{I})\bar{P} - b(x)\bar{I}, \\ \dfrac{\partial \bar{P}}{\partial t} = c(x)\bar{I} - m(x)\bar{P}, \\ \bar{I}(x,0) = \bar{I}^0(x) \geq 0, \ \ \bar{P}(x,0) = \bar{P}^0(x) \geq 0, \end{cases} \tag{3.19}$$

for $(x,t) \in \Omega \times (0,\infty)$ with boundary condition (1.4), system (1.3) is a system at a specific location $x \in \Omega$. By applying the method of the next generation matrix, the BRN of (3.19) is termed as LBRN, denoted by

$$\Re_0^L(x) = \frac{\alpha(x)H(x)m(x) + \beta(x)H(x)c(x)}{b(x)m(x)}.$$

We next explore the relation between Υ_0 and $\Re_0^L(x)$. Thanks to (i) of Lemma 3.5 and (3.17), we know that

$$\begin{aligned} \Upsilon_0 &= \bar{\eta}_0\left(d_I, \left(\alpha(\cdot) + \frac{\beta(\cdot)c(\cdot)}{m(\cdot)}\right)H(\cdot) - b(\cdot)\right) \\ &= \sup\left\{\int_\Omega \left(b(\cdot)(\Re_0^L(\cdot) - 1)\phi^2 - d_I|\nabla\phi|^2\right)\mathrm{d}x : \phi \in H^1(\Omega) \text{ with } \int_\Omega \phi^2 \mathrm{d}x = 1\right\}. \end{aligned}$$

Obviously, if $\Re_0^L(x) < 1$ for $x \in \Omega$, then $\Upsilon_0 < 0$, i.e., the cholera will be eradicated if $\Re_0^L(x) < 1$. Here, location $x \in \Omega$ with $\Re_0^L(x) < 1$ is termed as the non-favorite site for cholera.

If $\Re_0^L(x) > 1$ for some $x \in \Omega$ (where x is called the favorite site for cholera), we then cannot determine the sign of Υ_0 directly. Note that Υ_0 is decreasing in d_I, and by (iii) of Lemma 3.5, we have

$$\begin{aligned} \lim_{d_I \to \infty} \bar{\eta}_0\left(d_I, \left(\alpha(\cdot) + \frac{\beta(\cdot)c(\cdot)}{m(\cdot)}\right)H(\cdot) - b(\cdot)\right) &= \left(\left(\alpha(\cdot) + \frac{\beta(\cdot)c(\cdot)}{m(\cdot)}\right)H(\cdot) - b(\cdot)\right)^\sharp \\ &= \frac{1}{|\Omega|}\int_\Omega (b(\cdot)(\Re_0^L(\cdot) - 1))\mathrm{d}x. \end{aligned}$$

Note that $\left(\left(\alpha(\cdot) + \frac{\beta(\cdot)c(\cdot)}{m(\cdot)}\right)H(\cdot) - b(\cdot)\right)^\sharp < 0$ can be estimated as

$$\int_\Omega \left(\alpha(\cdot) + \frac{\beta(\cdot)c(\cdot)}{m(\cdot)}\right)H(\cdot)\mathrm{d}x < \int_\Omega b(\cdot)\mathrm{d}x.$$

Here we call Ω as non-favorite domain for the cholera. This, together with the assertion (ii) and (iii) of Theorem 3.1, reflects that the cholera may be vanishing or spreading depending on d_I if $\Re_0^L(x) > 1$. Specifically, if $\Re_0^L(x) > 1$ but with non-favorite domain, that is, $\left(\left(\beta_1(\cdot) + \frac{\beta_2(\cdot)c(\cdot)}{m(\cdot)}\right)H(\cdot) - b(\cdot)\right)^\sharp < 0$, we can still eradicate cholera epidemic in the domain.

3.6. Global stability of PPS: a homogeneous case

In the homogeneous case that parameter functions of (1.3) are replaced with positive constants, model (1.3) will become

$$\begin{cases} \dfrac{\partial \bar{I}}{\partial t} = d_I\Delta\bar{I} + \alpha(H - \bar{I})\bar{I} + \beta(H - \bar{I})\bar{P} - b\bar{I} := d_I\Delta\bar{I} + f_1(\bar{I}, \bar{P}), & (x,t) \in \Omega \times (0,\infty), \\ \dfrac{\partial \bar{P}}{\partial t} = c\bar{I} - m\bar{P} := f_2(\bar{I}, \bar{P}), & (x,t) \in \Omega \times (0,\infty), \end{cases} \tag{3.20}$$

with (1.4).

Noticing that Theorems 3.3 and 3.4 still hold for model (3.20). The BRN of (3.20) can be calculated as

$$\Re_0^c = \frac{1}{\lambda_0} = \frac{m\alpha H + c\beta H}{bm}. \tag{3.21}$$

Denote by $Q^c = (I^c, P^c)^{\mathrm{T}} = \left(\frac{H}{\Re_0^c}(\Re_0^c - 1), \frac{cH}{m\Re_0^c}(\Re_0^c - 1)\right)^{\mathrm{T}}$ the constant equilibrium when $\Re_0^c > 1$. We next study the global stability of Q^c by the Lyapunov function.

Theorem 3.5. *Let $\Re_0^c$ be defined by (3.21). If $\Re_0^c > 1$, then Q^c is globally attractive.*

Proof. We proceed directly and define

$$\mathbb{L}_c(\bar{I}, \bar{P})(t) := \int_\Omega L_c(x,t)\mathrm{d}x,$$

where

$$L_c(x,t) = I^c g\left(\frac{\bar{I}}{I^c}\right) + \frac{\beta(H - I^c)P^c}{m} g\left(\frac{\bar{P}}{P^c}\right),$$

and $g(\hbar) = \hbar - 1 - \ln\hbar \geq 0$, $\hbar > 0$, and $g(\hbar) = 0$ iff $\hbar = 1$.

Directly calculating the derivative of $\mathbb{L}_c(t)$ gives

$$\frac{d\mathbb{L}_c(t)}{dt} = \int_\Omega \left(1 - \frac{I^c}{\bar{I}}\right) d_I \Delta I \mathrm{d}x + \int_\Omega \left[\left(1 - \frac{I^c}{\bar{I}}\right) f_1(\bar{I}, \bar{P}) + \frac{\beta(H - I^c)}{m}\left(1 - \frac{P^c}{\bar{P}}\right) f_2(\bar{I}, \bar{P})\right]\mathrm{d}x.$$

Since

$$\int_\Omega \left(1 - \frac{I^c}{\bar{I}}\right) d_I \Delta \bar{I} \mathrm{d}x = -\int_\Omega d_I \frac{I^c}{\bar{I}^2} |\nabla \bar{I}^2| \mathrm{d}x \leq 0,$$

it then directly gives

$$\begin{aligned}
\frac{d\mathbb{L}_c(t)}{dt} &\leq \int_\Omega \left[\left(1 - \frac{I^c}{\bar{I}}\right) f_1(\bar{I}, \bar{P}) + \frac{\beta(H - I^c)}{m}\left(1 - \frac{P^c}{\bar{P}}\right) f_2(\bar{I}, \bar{P})\right]\mathrm{d}x \\
&= -\int_\Omega \frac{\alpha\bar{I} + \beta\bar{P}}{\bar{I}}(\bar{I} - I^c)^2 \mathrm{d}x + \int_\Omega \beta(H - I^c)P^c\left(2 - \frac{I^c P}{\bar{I}P^c} - \frac{\bar{I}P^c}{I^c\bar{P}}\right)\mathrm{d}x \\
&\leq -\int_\Omega \beta(H - I^c)P^c\left[g\left(\frac{I^c\bar{P}}{\bar{I}P^c}\right) + g\left(\frac{\bar{I}P^c}{I^c\bar{P}}\right)\right]\mathrm{d}x \\
&\leq 0.
\end{aligned}$$

We omit the details here, since it is similar to [25, Section 9.9]. This proves Theorem 3.5. □

4. Threshold dynamics of cholera model with the dispersal rates of humans and Vibrio cholerae

Based on (1.3), we consider the following cholera model:

$$\begin{cases}
\frac{\partial \bar{I}}{\partial t} = d_I \Delta \bar{I} + \alpha(\cdot)(H(\cdot) - \bar{I})\bar{I} + \beta(\cdot)(H(\cdot) - \bar{I})\bar{P} - b(\cdot)\bar{I}, & x \in \Omega,\ t > 0, \\
\frac{\partial \bar{P}}{\partial t} = d_P \Delta \bar{P} + c(\cdot)\bar{I} - m(\cdot)\bar{P}, & x \in \Omega,\ t > 0, \\
\frac{\partial \bar{I}}{\partial \nu} = \frac{\partial \bar{P}}{\partial \nu} = 0, & x \in \partial\Omega,\ t > 0, \\
(\bar{I}(x,0), \bar{P}(x,0)) = (\bar{I}^0(x), \bar{P}^0(x)), & x \in \Omega,
\end{cases} \tag{4.1}$$

where $d_P > 0$ represents the mobility of *Vibrio cholerae* in the water environment. The standard theory for parabolic equations, a semigroup approach and the proof of Lemma 2.1, guarantee that (4.1) admits a unique classical solution $(\bar{I}, \bar{P})^{\mathrm{T}} \in \mathbb{X}_H$. Similar to Lemma 2.2, we can conclude that the ultimate boundedness of solution. Further from the Hopf boundary lemma and strong maximum principle, we have $\bar{I} > 0$ and $\bar{P} > 0$ for all $(x,t) \in \bar{\Omega} \times (0, \infty)$.

4.1. Basic reproduction number of (4.1)

Generally speaking, the spread and extinction of cholera according to reproduction numbers provide important implications to study the complicated impacts of environmental heterogeneity on disease transmission. For the method used here, we refer to [6,12,14,26,35] and references therein.

Denote the DFSS and PSS of system (4.1) by $Q_0 = (0, 0)^{\mathrm{T}}$ and $\tilde{Q}^{ss} = (\tilde{I}^{ss}, \tilde{P}^{ss})^{\mathrm{T}}$ if they exist, respectively. Similar to (3.2), we define the BRN of (4.1) as

$$\Re_0^{(4.1)} := r(\tilde{L}) = \sup\{|\lambda|,\ \lambda \in \sigma(\tilde{L})\}, \tag{4.2}$$

where

$$\tilde{L}(\phi)(x) = \int_0^\infty \tilde{\mathcal{F}}(x)\hat{\Phi}(t)\phi(x)dt = \tilde{\mathcal{F}}(x)\int_0^\infty \hat{\Phi}(t)\phi(x)dt, \quad \phi \in \mathbb{X}_H,\ x \in \bar{\Omega},$$

with

$$\tilde{\mathcal{F}} = \begin{pmatrix} \alpha(\cdot)H(\cdot) & \beta(\cdot)H(\cdot) \\ 0 & 0 \end{pmatrix} \tag{4.3}$$

and $\hat{\Phi}(t)$ being the semigroup generated by

$$\tilde{\mathcal{B}} = \begin{pmatrix} d_I\Delta - b(\cdot) & 0 \\ c(\cdot) & d_P\Delta - m(\cdot) \end{pmatrix}. \tag{4.4}$$

Due to $\Re_0^{(4.1)}$ is difficult to visualize, we are now in a position to seek the equivalent characterization of $\Re_0^{(4.1)}$. Unlike in Lemma 3.2, where the associated EP consists of only one equation, here we allow $\Re_0^{(4.1)}$ being related to the PE of EP consisting of two equations.

The following results demonstrate the relation between $\Re_0^{(4.1)}$ and a PE of the associated EP. The first result comes from [26, Theorem 3.5].

Lemma 4.1. *Let $\tilde{\lambda}_0$ be the PE of*

$$\begin{cases} \tilde{\lambda}\psi = (\tilde{\mathcal{F}} + \tilde{\mathcal{B}})\psi, \quad \psi = \begin{pmatrix} \psi_1 \\ \psi_2 \end{pmatrix}, & x \in \Omega, \\ \dfrac{\partial \psi_1}{\partial \nu} = \dfrac{\partial \psi_2}{\partial \nu} = 0, & x \in \partial\Omega, \\ \psi_1, \psi_2 \in C^2(\Omega, \mathbb{R}^n) \cap C^1(\bar{\Omega}, \mathbb{R}^n). \end{cases} \tag{4.5}$$

Then, $\Re_0^{(4.1)} - 1$ has the same sign as $\tilde{\lambda}_0 = s(\tilde{\mathcal{F}} + \tilde{\mathcal{B}})$, where $\tilde{\mathcal{F}}$ and $\tilde{\mathcal{B}}$ be defined in (4.3) and (4.4)*, respectively.*

Lemma 4.2. *Let $\Re_0^{(4.1)}$ be defined in (4.2), $D = diag(d_I, d_P)$, and $\tilde{V} = \begin{pmatrix} b(x) & 0 \\ -c(x) & m(x) \end{pmatrix}$. Consider the following EP:*

$$\begin{cases} -D\Delta\varphi + \tilde{V}\varphi = \kappa\tilde{\mathcal{F}}\varphi, \quad \varphi = \begin{pmatrix} \varphi_1 \\ \varphi_2 \end{pmatrix}, & x \in \Omega, \\ \dfrac{\partial \varphi_1}{\partial \nu} = \dfrac{\partial \varphi_2}{\partial \nu} = 0, & x \in \partial\Omega, \\ \varphi_1, \varphi_2 \in C^2(\Omega, \mathbb{R}^n) \cap C^1(\bar{\Omega}, \mathbb{R}^n). \end{cases} \tag{4.6}$$

Then we have:

(i) EP (4.6) admits a unique PE, $\kappa_0 > 0$.
(ii) $\Re_0^{(4.1)} = 1/\kappa_0$.

Proof. We first prove (i). Let (κ, φ) be an eigenvalue pair of problem (4.6) with $\varphi = (\varphi_1, \varphi_2)^{\mathrm{T}}$, that is,

$$\begin{cases} -d_I\Delta\varphi_1 + b(\cdot)\varphi_1 = \kappa\alpha(\cdot)H(\cdot)\varphi_1 + \kappa\beta(\cdot)H(\cdot)\varphi_2, & x \in \Omega, \\ -d_P\Delta\varphi_2 + m(\cdot)\varphi_2 = c(\cdot)\varphi_1, & x \in \Omega. \end{cases}$$

Let $\{\tilde{T}_i(t)\}_{t\geq 0}, i = 1, 2 : \mathbb{X} \to \mathbb{X}$ be the C_0 semigroups generated by the operator $\tilde{A}_i$, where $\tilde{A}_1 := d_I\Delta - b(\cdot)$ and $\tilde{A}_2 := d_P\Delta - m(\cdot)$ subject to (4.1), respectively. Due to [26, Theorem 3.12], we get that for $\varphi \in \mathbb{X}$,

$$(\kappa\mathbb{I}_d - \tilde{A}_i)^{-1}\varphi = \int_0^\infty e^{-\kappa t}\tilde{T}_i(t)\varphi dt \ \ \text{for all } \kappa > s(\tilde{A}_i),\ i = 1, 2, \tag{4.7}$$

where $\mathbb{I}_d$ denotes the identity operator since

$$s(\tilde{A}_1) \le -\min_{x\in\bar{\Omega}}\{b(x)\} < 0 \quad \text{and} \quad s(\tilde{A}_2) \le -\min_{x\in\bar{\Omega}}\{m(x)\} < 0.$$

By letting $\kappa = 0$ in (4.7), one can get

$$-\tilde{A}_1^{-1}\varphi = \int_0^\infty \tilde{T}_1(t)\varphi dt \quad \text{and} \quad -\tilde{A}_2^{-1}\varphi = \int_0^\infty \tilde{T}_2(t)\varphi dt \quad \text{for all } \varphi \in \mathbb{X}.$$

Notice that $-\tilde{A}_1^{-1}$ and $-\tilde{A}_2^{-1}$ are compact and strongly positive operators. We rewrite system (4.6) as

$$\begin{cases} -\tilde{A}_1\varphi_1 = \kappa\alpha(\cdot)H(\cdot)\varphi_1 + \kappa\beta(\cdot)H(\cdot)\varphi_2, & x \in \Omega, \\ -\tilde{A}_2\varphi_2 = c(\cdot)\varphi_1, & x \in \Omega, \end{cases}$$

which implies that $\varphi_2 = -\tilde{A}_2^{-1}c(\cdot)\varphi_1$ and (κ, φ_1) satisfies

$$\frac{1}{\kappa}\varphi_1 = -\tilde{A}_1^{-1}\alpha(\cdot)H(\cdot)\varphi_1 + \tilde{A}_1^{-1}\beta(\cdot)H(\cdot)\tilde{A}_2^{-1}c(\cdot)\varphi_1. \tag{4.8}$$

Thanks to $\beta_1(\cdot), H(\cdot), \beta_2(\cdot), c(\cdot) > 0$ for all $x \in \Omega$, we directly confirm that

$$-\tilde{A}_1^{-1}\alpha(\cdot)H(\cdot) \text{ and } \tilde{A}_1^{-1}\beta(\cdot)H(\cdot)\tilde{A}_2^{-1}c(\cdot)$$

are strongly positive and compact on $C(\Omega, \mathbb{R})$. An application of the Krein–Rutman Theorem ensures that EP (4.8) has a unique PE $\kappa_0 > 0$, with an eigenfunction $\tilde{\varphi}_1 > 0$ in $C(\Omega, \mathbb{R}^n)$. By letting $\tilde{\varphi}_2 = -\tilde{A}_2^{-1}c(\cdot)\tilde{\varphi}_1$, we obtain that $\tilde{\varphi}_2 > 0$. This proves (i).

The assertion (ii) is directly obtained according to [35, Theorem 3.2]. This proves Lemma 4.2. □

Note that systems (1.1) and (4.1) share the same LBRN. Inspired by a recent work [14], we next study the relation between $\Re_0^{(4.1)}$ and $\Re_0^L(x)$, $x \in \Omega$. To this end, we rewrite $\Re_0^L(x)$ as

$$\Re_0^L(x) = \Re_1^1(x) + \Re_1^2(x)\Re_2(x), \tag{4.9}$$

where

$$\Re_1^1(x) = \frac{\alpha(x)H(x)}{b(x)}, \ \Re_1^2(x) = \frac{\beta(x)H(x)}{b(x)}, \ \text{and } \Re_2(x) = \frac{c(x)}{m(x)}.$$

Here, $\Re_1^1(x)$, $\Re_1^2(x)$, and $\Re_2(x)$ are multiplication operators on $C(\Omega)$ owning the biological meanings: for $x \in \Omega$, $\Re_1^1(x)$ (resp. $\Re_1^2(x)$) represents the impact of one infected human (resp. *Vibrio cholerae*) on the susceptible humans, while $\Re_2(x)$ represents the impact of one infected human on the Vibrio cholerae.

Following the line of [14,26,35], the $\Re_0^{(4.1)}$ is defined by

$$\Re_0^{(4.1)} = r(-\tilde{\mathcal{F}}\tilde{\mathcal{B}}^{-1}), \tag{4.10}$$

where $\tilde{\mathcal{B}}$ and $\tilde{\mathcal{F}}$ can be found in (4.3) and (4.4), respectively. According to the approach developed in [14, Theorem 3.1], we directly have

$$\Re_0^{(4.1)} = r(L_1(\Re_1^1(\cdot) + \Re_1^2(\cdot)L_2\Re_2(\cdot))), \tag{4.11}$$

where

$$L_1 = (b(\cdot) - d_I\Delta)^{-1}b(\cdot) \quad \text{and} \quad L_2 = (m(\cdot) - d_P\Delta)^{-1}m(\cdot)$$

are strongly positive compact linear operators on $C(\Omega)$.

The main result of this subsection on $\Re_0^{(4.1)}$ with $(d_I,\ d_P) \to (\infty,\ \infty)$ is described below, which can be viewed as a direct consequence of [14, Theorem 3.6 and Remark 4.8]. We omit the proof here.

Lemma 4.3. *Let $\Re_0^{(4.1)}$ be defined in (4.11). The following statements are valid:*

(i) $\lim_{d_I\to\infty}\lim_{d_P\to\infty} \Re_0^{(4.1)} = \lim_{d_P\to\infty}\lim_{d_I\to\infty} \Re_0^{(4.1)} = \frac{(\alpha H)^\sharp m^\sharp + (\beta H)^\sharp c^\sharp}{b^\sharp m^\sharp}$.

(ii) $\lim_{(d_I,\ d_P)\to(\infty,\ \infty)} \Re_0^{(4.1)} = \frac{(\alpha H)^\sharp m^\sharp + (\beta H)^\sharp c^\sharp}{b^\sharp m^\sharp}$.

The main result of this subsection on $\Re_0^{(4.1)}$ with $(d_I,\ d_P) \to (0, 0)$ is described below, which can be viewed as a direct consequence of [14, Theorem 4.10 and Theorem 4.11]. We omit the proof here.

Lemma 4.4. *Let $\Re_0^{(4.1)}$ and $\Re_0^L(x)$ are defined in (4.9)* and *(4.11), respectively. We obtain*

(i) $\lim_{d_I\to 0}\lim_{d_P\to 0}\ \Re_0^{(4.1)} = \lim_{d_P\to 0}\lim_{d_I\to 0}\ \Re_0^{(4.1)} = \max_{x\in\bar{\Omega}}\{\Re_0^L(x)\}$.

(ii) $\lim_{(d_I,\ d_P)\to(0,0)}\ \Re_0^{(4.1)} = \max_{x\in\bar{\Omega}}\{\Re_0^L(x)\}$.

4.2. Threshold dynamics of (4.1)

The following result demonstrates that cholera vanishing and persistence depends on the sign of $\Re_0^{(4.1)} - 1$.

Theorem 4.1. *Let $\Re_0^{(4.1)}$ be defined by (4.2).*

(i) If $\Re_0^{(4.1)} < 1$, then the DFSS Q_0 is asymptotically stable, and further, it is globally asymptotically stable in $\mathbb{X}_H$.

(ii) If $\Re_0^{(4.1)} > 1$, then there exists a $\varsigma > 0$ such that the solution $(\bar{I}, \bar{P})^{\mathrm{T}}$ of (4.1) with any positive non-zero initial value $(\bar{I}^0(x), \bar{P}^0(x))^{\mathrm{T}} \in \mathbb{X}_H$ fulfills

$$\min\left(\liminf_{t\to\infty}\bar{I}(x,t), \liminf_{t\to\infty}\bar{P}(x,t)\right) \geq \varsigma \quad \text{for all } x \in \bar{\Omega}. \tag{4.12}$$

Furthermore, (4.1) has at least one PSS, denoted by $\tilde{Q}^{ss} = (\tilde{I}^{ss}, \tilde{P}^{ss})^{\mathrm{T}}$.

Proof. We first prove (i). The local stability of DFSS Q_0 follows from [35, Theorem 3.1]. We next proceed the global attractivity of Q_0 directly by the comparison principle. By Lemma 4.1, we have $\tilde{\lambda}_0 < 0$ since $\Re_0^{(4.1)} < 1$. We then have

$$\begin{cases} \dfrac{\partial \bar{I}}{\partial t} \leq d_I \Delta \bar{I} + H(\cdot)(\alpha(\cdot)\bar{I} + \beta(\cdot)\bar{P}) - b(\cdot)\bar{I}, & x \in \Omega,\ t > 0, \\ \dfrac{\partial \bar{P}}{\partial t} \leq d_P \Delta \bar{P} + c(\cdot)\bar{I} - m(\cdot)\bar{P}, & x \in \Omega,\ t > 0, \\ \dfrac{\partial \bar{I}}{\partial \nu} = \dfrac{\partial \bar{P}}{\partial \nu} = 0, & x \in \partial\Omega,\ t > 0. \end{cases}$$

Let $(\bar{\psi}_1, \bar{\psi}_2)^{\mathrm{T}}$ be the positive eigenfunction of $\tilde{\lambda}_0$. Choosing sufficiently large $\bar{M} > 0$ such that $(\bar{I}^0, \bar{P}^0)^{\mathrm{T}} \leq \bar{M}(\bar{\psi}_1, \bar{\psi}_2)^{\mathrm{T}}$. This, together with the comparison principle, gives

$$\begin{pmatrix} \bar{I} \\ \bar{P} \end{pmatrix} \leq \bar{M} \begin{pmatrix} \bar{\phi}_1 \\ \bar{\phi}_2 \end{pmatrix} e^{\lambda_0 t}, \quad t \geq 0.$$

Therefore, $\lim_{t\to\infty}(\bar{I}, \bar{P}) = (0, 0)$, uniformly on $x \in \Omega$. This completes the proof of (i).

The assertion (ii) can be proved analogously as Theorem 3.3. This proves (ii). $\square$

5. Numerical simulation

In this section, we give numerical examples that support our analytical results.

5.1. Spatially one-dimensional case

For model (1.3), we fix the following parameters.

$$\begin{cases} n = 1,\ \Omega = (0, 1),\ H(x) = b(x) = m(x) = 1, \\ \alpha(x) = 0.6\left(1 + \dfrac{1}{2}\sin\dfrac{\pi x}{2}\right),\ \beta(x) = 0.5\left(1 + \dfrac{1}{2}\cos\dfrac{\pi x}{2}\right),\ c(x) = 0.2\left(1 + \dfrac{1}{2}\sin\dfrac{\pi x}{2}\right), \\ \bar{I}^0(x) = 0.01,\ \bar{P}(x) = 0,\ x \in \Omega, \end{cases} \tag{5.1}$$

where n is the dimension of the domain Ω. Note that these values are chosen for a technical reason and there is no specific biological meaning. For these parameters, we obtain

$$\lim_{d_I\to 0}\Re_0^{(1.3)} \approx 1.0578 > 1 \quad \text{and} \quad \lim_{d_I\to\infty}\Re_0^{(1.3)} \approx 0.9641 < 1.$$

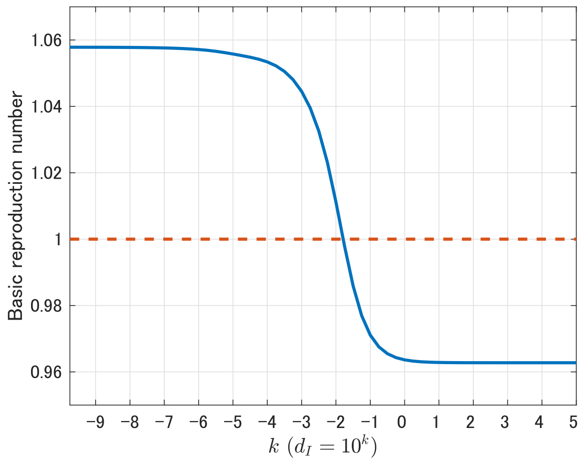
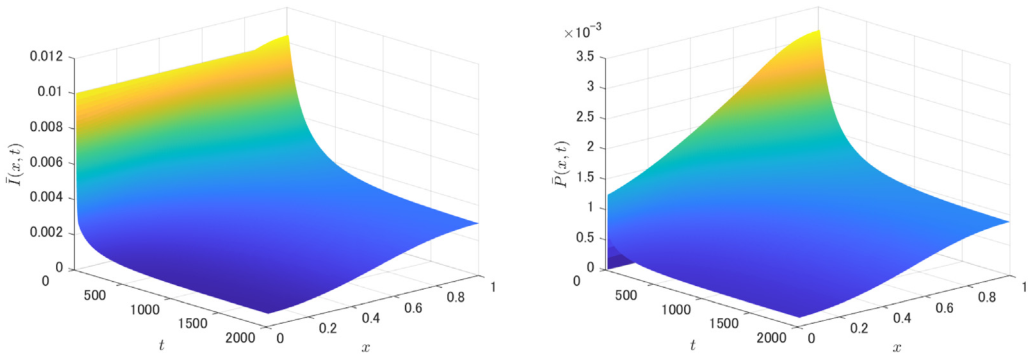


Fig. 1. Basic reproduction number $\Re_0^{(1.3)}$ versus $d_I = 10^k$ $(-10 \le k \le 5)$ for parameters (5.1).



(a) (b)

Fig. 2. Time evolution of (a) $\bar{I}$ and (b) $\bar{P}$ of model (1.3) for parameters (5.1) and $d_I = 0.015 < \bar{d}_I \approx 0.0178$ ($\Re_0^{(1.3)} \approx 1.0018 > 1$).

Hence, by Theorem 3.1 (iii), there exists a critical value $\bar{d}_I > 0$ such that $\Re_0^{(1.3)} > 1$ for $d_I < \bar{d}_I$ and $\Re_0^{(1.3)} < 1$ for $d_I > \bar{d}_I$. In other words, the diffusion-induced stability change can occur. $\Re_0^{(1.3)}$ can be approximated by discretizing the EP (3.3), and $\Re_0^{(1.3)}$ for d_I is plotted in Fig. 1, where $d_I = 10^k$, $-10 \le k \le 5$. From Fig. 1, we can verify that $\Re_0^{(1.3)}$ is monotonically decreasing with respect to d_I, and $\bar{d}_I \approx 0.0178$.

For $d_I = 0.015 < \bar{d}_I$, we have $\Re_0^{(1.3)} \approx 1.0018 > 1$. Hence, by Theorems 3.3 and 3.4, the system is uniformly persistent and there exists the unique PSS. In fact, in Fig. 2, the solution converges to PSS as time evolves.

On the other hand, for $d_I = 0.02 > \bar{d}_I$, we have $\Re_0^{(1.3)} \approx 0.9953 < 1$. Hence, by Theorem 3.2, DFSS is globally asymptotically stable. In fact, in Fig. 3, the solution converges to DFSS $Q_0 = (0, 0)$ as time evolves.

5.2. Spatially two-dimensional case

Next, for model (1.3), we fix the following parameters.

$$\begin{cases} n = 2, \ \ \Omega = (0,4) \times (0,1), \ \ \alpha(x) = 0.5, \ \ \beta(x) = 1, \ \ c(x) = 0.01, \ \ m(x) = 1, \\ H(x) = \dfrac{(1 - 0.9\cos \pi x_1)(1 - 0.9 \cos 2\pi x_2)}{4}, \quad b(x) = (1 + 0.9\cos \pi x_1)(1 + 0.9\cos 2\pi x_2), \\ \bar{I}^0(x) = 0.01 H(x), \ \ \bar{P}(x) = 0, \quad x = \begin{pmatrix} x_1 \\ x_2 \end{pmatrix} \in \Omega. \end{cases} \tag{5.2}$$

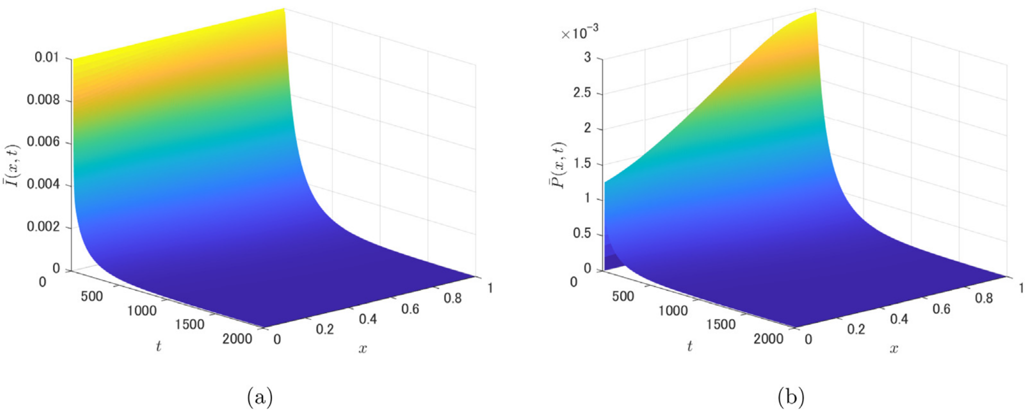


Fig. 3. Time evolution of (a) $\bar{I}$ and (b) $\bar{P}$ of model (1.3) for parameters (5.1) and $d_I = 0.02 > \bar{d}_I \approx 0.0178$ ($\Re_0^{(1.3)} \approx 0.9953 < 1$).

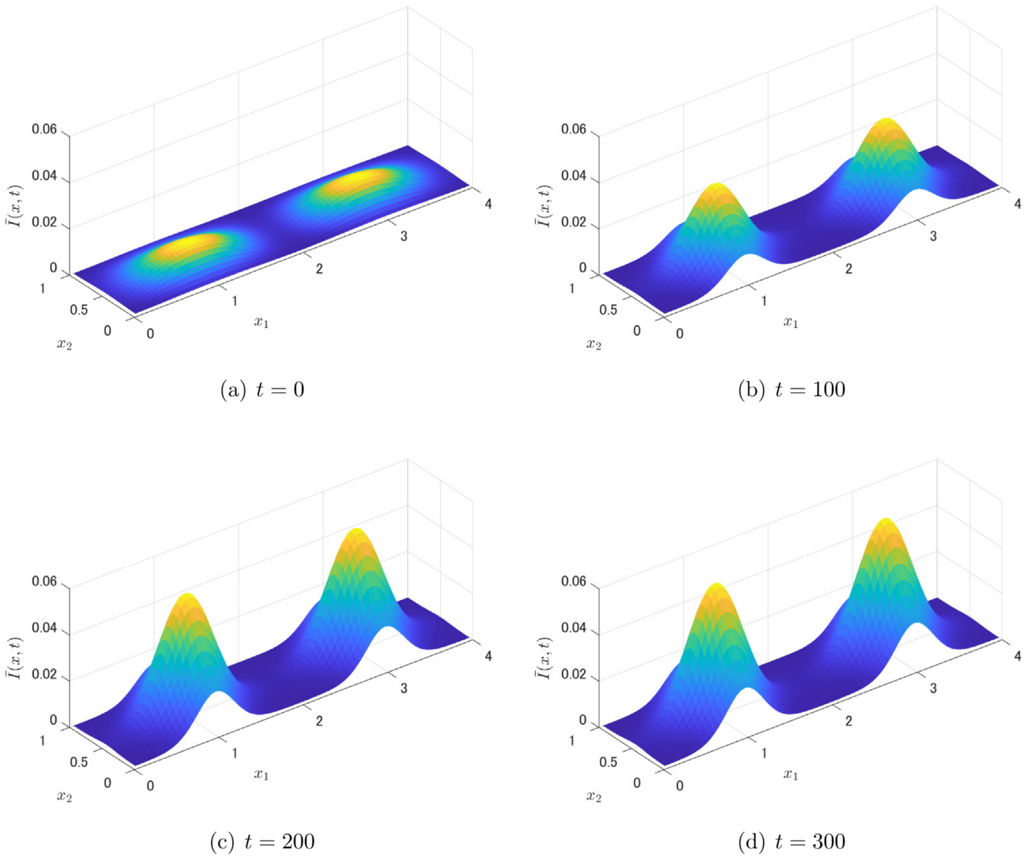


Fig. 4. Time evolution of $\bar{I}$ of model (1.3) for parameters (5.2) and $d_I = 0.014$ ($\Re_0^{(1.3)} \approx 1.0573 > 1$).

Note that, as in the previous subsection, these values are chosen for a technical reason and there is no specific biological meaning. To compute $\Re_0^{(1.3)}$, we apply "solvepdeeig" command in the Partial Differential Equations Toolbox of MATLAB to the EP (3.3).

For $d_I = 0.014$, we obtain $\Re_0^{(1.3)} \approx 1.0573 > 1$. In this case, by Theorems 3.3 and 3.4, we see that the system is uniformly persistent and there exists the unique PSS. In fact, Fig. 4 shows that $\bar{I}$ converts to a positive distribution as time evolves.

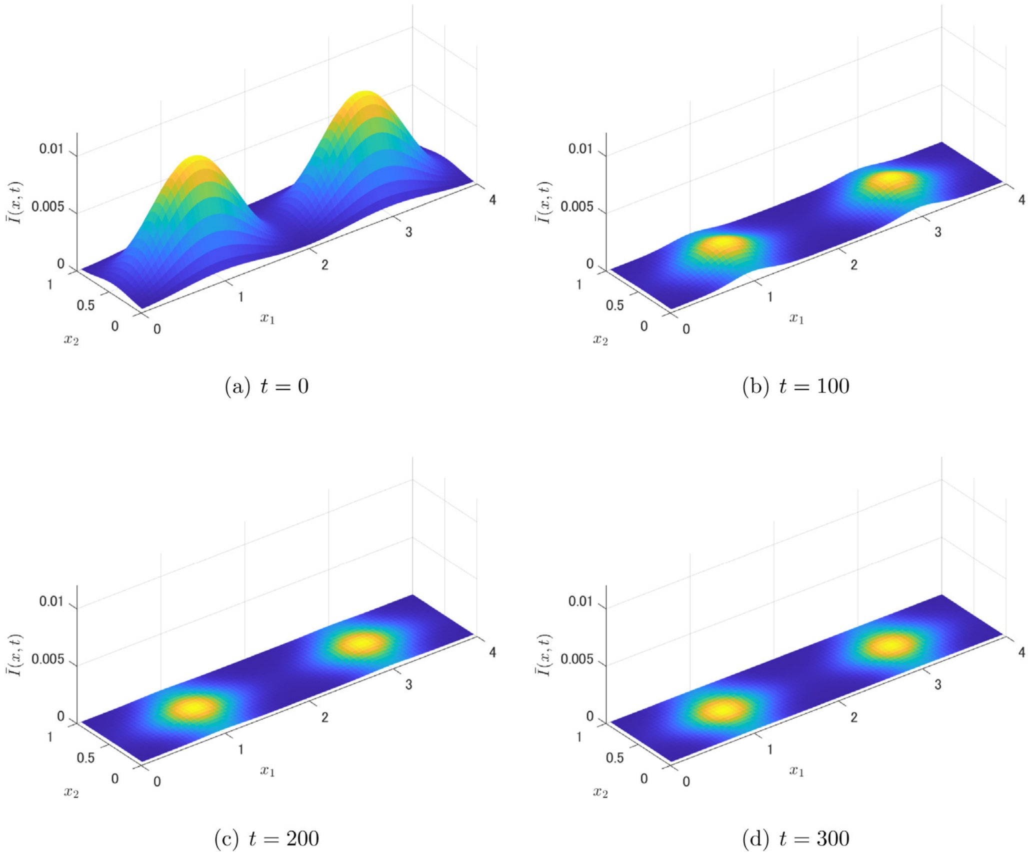


Fig. 5. Time evolution of $\bar{I}$ of model (1.3) for parameters (5.2) and $d_I = 0.017$ ($\Re_0^{(1.3)} \approx 0.9423 < 1$).

On the other hand, for $d_I = 0.017$, we obtain $\Re_0^{(1.3)} \approx 0.9423 < 1$. In this case, by Theorem 3.2, the DFSS is globally asymptotically stable. In fact, Fig. 5 shows that $\bar{I}$ converges to zero as time evolves.

6. Conclusion and discussion

This paper performs a complete analysis of a degenerated reaction–diffusion cholera model with two disease transmission routes. The model is formulated in a spatially bounded domain, allowing spatial heterogeneity for the model parameters. We assume that the total humans stabilize at $H(x)$ for all $x \in \Omega$ performing an unbiased random walk and the *Vibrio cholerae* live in contaminated water without diffusion. With the aim to explore what is the impact of the mobility of the humans and *Vibrio cholerae* on cholera epidemics, we also study the model with *Vibrio cholerae* diffusion in contaminated water to reveal the differences in the aspect of threshold dynamics governed by BRN.

Firstly, the well-posedness of the model (1.3) is established (see Lemma 2.1). Combining with the comparison principle and [23, Theorem 3.1 in Chapter 7], we further confirm that the unique global solution is ultimately bounded (see (i) of Lemma 2.2). We remark that the solution semiflow of (1.3), $\Psi(t)$, is not compact since the $\bar{P}$-equation of (1.3) is lack of diffusion term. For this reason, we verify the asymptotic compactness of $\Psi(t)$ by considering the κ-contraction condition, and so the existence of a connected global attractor is ensured (see (ii) of Lemma 2.2).

We follow the method in [35] to introduce BRN $\Re_0^{(1.3)}$ of (1.3) as the spectral radius of the NGO (see (3.2)). $\Re_0^{(1.3)}$ is also characterized in Lemmas 3.1, 3.2, 3.3, and Lemma 3.4. In particular, according to the variational formula of $\Re_0^{(1.3)}$ (see Theorem 3.1), we can demonstrate the effect of d_I on $\Re_0^{(1.3)}$, that is, (i) the infection risk can be reduced by increasing d_I; (ii) $\Re_0^{(1.3)}$ approaches to the maximum value of $\Re_0^L(x)$ when d_I tends to zero, while

$\Re_0^{(1.3)}$ approaches to the ratio of $\int_\Omega \alpha H + \frac{c\beta H}{m} \mathrm{d}x$ to $\int_\Omega b \mathrm{d}x$ when d_I tends to infinity; (iii) if $\int_\Omega \left(\alpha H + \frac{c\beta H}{m}\right) \mathrm{d}x$ is greater than $\int_\Omega b \mathrm{d}x$, then the habitat is regarded as a high infection risk domain; (iv) if $\int_\Omega \left(\alpha H + \frac{c\beta H}{m}\right) \mathrm{d}x$ is smaller than $\int_\Omega b \mathrm{d}x$, there exists $\bar{d}_I > 0$ such that the habitat is regarded as a low infection risk domain when $d_I > \bar{d}_I$.

Further, Theorems 3.2 and 3.3 demonstrate that $\Re_0^{(1.3)}$ is a biologically meaningful threshold value that can predict the cholera vanishing and persistence. Additionally, with the help of Lemma 3.5, we introduce another threshold parameter Υ_0 (equivalent to $\bar{\eta}_0$ as in (ii) of Lemma 3.2) to study the uniqueness of PSS of associated elliptic problem, which also reflects the threshold dynamics of model (1.3) (see Theorem 3.4). We also emphasize this result by defining the LBRN $\Re_0^L(x)$ (see Section 3.5), that is, cholera completely extincts in the case that $\Re_0^L(x) < 1$, while it is still possible to eradicate cholera in the case that $\Re_0^L(x) > 1$. In the homogeneous case, the global attractivity of PSS for system (3.20) is confirmed by the Lyapunov function (see Theorem 3.5).

Taking into account the mobility of cholera bacteria in contaminated water, we formulate model (4.1) based on (1.3), the infected humans and cholera bacteria dispersal with distinct rates. The relations between $\Re_0^{(4.1)}$ and the PE of associated EP are also addressed (see Lemmas 4.1 and 4.2). Since model (4.1) has multiple infective compartments, $\Re_0^{(4.1)}$ is difficult to depict. We turn our attention to the relation between $\Re_0^{(4.1)}$ and $\Re_0^L(x)$ for model (4.1). Following the line of [14, Theorem 3.1], $\Re_0^{(4.1)}$ can be rewritten as (4.11). An application of [14, Theorem 3.6 and Remark 4.8] and [14, Theorem 4.10 and Theorem 4.11] gives the asymptotic behaviors of $\Re_0^{(4.1)}$ when $(d_I, d_P) \to (\infty, \infty)$ and $(d_I, d_P) \to (0, 0)$. Specifically,

(i) if $(d_I, d_P) \to (\infty, \infty)$, $\Re_0^{(4.1)}$ approaches to the ratio of $(\alpha H)^\sharp m^\sharp + (\beta H)^\sharp c^\sharp$ to $b^\sharp m^\sharp$ (see Lemma 4.3), which is essentially different to that in (i) of Theorem 3.1;

(ii) if $(d_I, d_P) \to (0, 0)$, $\Re_0^{(4.1)}$ approaches to the maximum of $\Re_0^L(x)$ (see Lemma 4.4). This result is consistent with that in (i) of Theorem 3.1.

Further, the threshold-type results of (4.1) relying on $\Re_0^{(4.1)}$ are also addressed by appealing to the comparison principle and Theorem 3.3, see Theorem 4.1. Finally, numerical examples are given to reinforce the analytical results.

Biologically, our results suggest that the BRN plays the essential role in determining whether the cholera epidemic will be eradicated or not, and the mobility of infected humans and *Vibrio cholerae* has a large effect on the size of the BRN. The application of our model for more epidemiological considerations using real data would be an important future work to combat against the practical problem of the cholera epidemic.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

References

[1] L.J.S. Allen, B.M. Bolker, Y. Lou, A.L. Nevai, Asymptotic profiles of the steady states for an SIS epidemic reaction–diffusion model, Discrete Contin. Dyn. Syst. 21 (2008) 1–20.

[2] Z. Bai, R. Peng, X.-Q. Zhao, A reaction–diffusion malaria model with seasonality and incubation period, J. Math. Biol. 77 (2018) 201–228.

[3] F. Capone, V. De Cataldis, R. De Luca, Influence of diffusion on the stability of equilibria in a reaction–diffusion system modeling cholera dynamic, J. Math. Biol. 71 (2015) 1107–1131.

[4] R. Cui, K.-Y. Lam, Y. Lou, Dynamics and asymptotic profiles of steady states of an epidemic model in advective environments, J. Differential Equations 263 (2017) 2343–2373.

[5] R. Cui, Y. Lou, A spatial SIS model in advective heterogeneous environments, J. Differential Equations 261 (2016) 3305–3343.

[6] O. Diekmann, J.A.P. Heesterbeek, J.A.J. Metz, On the definition and the computation of the basic reproduction ratio R_0 in models for infectious diseases in heterogeneous populations, J. Math. Biol. 28 (4) (1990) 365–382.

[7] M.C. Eisenberg, Z. Shuai, J.H. Tien, P. van den Driessche, A cholera model in a patchy environment with water and human movement, Math. Biosci. 246 (2013) 105–112.

[8] J.K. Hale, Asymptotic Behavior of Dissipative Systems, American Mathematical Society, Providence, 1988.

[9] M.C.M. de Jong, O. Diekmann, H. Heesterbeek, How Does Transmission of Infection Depend on Population Size in: Epidemic Models: Their Structure and Relation to Data, Cambridge University Press, 1995, pp. 84–94.

[10] W.O. Kermack, A.G. McKendrick, Contributions to the mathematical theory of epidemics-I, Proc. R. Soc. Lond. Ser. A Math. Phys. Eng. Sci. 115 (1927) 700–721.
[11] H. Li, R. Peng, F. Wang, Varying total population enhances disease persistence: Qualitative analysis on a diffusive SIS epidemic model, J. Differential Equations 262 (2009) 885–913.
[12] X. Liang, L. Zhang, X.-Q. Zhao, Basic reproduction ratios for periodic abstract functional differential equations (with application to a spatial model for lyme disease), J. Dynam. Differential Equations 31 (2017) 1247–1278.
[13] Y. Lou, X.-Q. Zhao, A reaction–diffusion malaria model with incubation period in the vector population, J. Math. Biol. 62 (2011) 543–568.
[14] P. Magal, G. Webb, Y. Wu, On the basic reproduction number of reaction–diffusion epidemic models, SIAM J. Appl. Math. 79 (1) (2019) 284–304.
[15] P. Magal, X.-Q. Zhao, Global attractors and steady states for uniformly persistent dynamical systems, SIAM. J. Math. Anal. 37 (2005) 251–275.
[16] R.H. Martin, H.L. Smith, Abstract functional differential equtions and reaction–diffusion systems, Trans. Amer. Math. Soc. 321 (1990) 1–44.
[17] H. McCallum, N. Barlow, J. Hone, How should pathogen transmission be modelled, Trends Ecol. Evol. 16 (2001) 295–300.
[18] Z. Mukandavire, S. Liao, J. Wang, H. Gaff, D.L. Smith, J.G. Morris, Estimating the reproductive numbers for the 2008–2009 cholera outbreaks in Zimbabwe, Proc. Nat. Acad. Sci. USA 108 (2011) 8767–8772.
[19] R.D. Nussbaum, Eigenvectors of nonlinear positive operator and the linear krein–rutman theorem, in: E. Fadell, G. Fournier (Eds.), Fixed Point Theory, in: Lecture Notes in Mathe-matics, vol. 886, Springer, New York/Berlin, 1981, pp. 309–331.
[20] A. Pazy, Semigroups of Linear Operators and Application to Partial Differential Equations, Springer-Verlag, New York, 1983.
[21] R. Peng, Asymptotic profiles of the positive steady state for an SIS epidemic reaction–diffusion model. Part I, J. Differential Equations 247 (2009) 1096–1119.
[22] R. Peng, F. Yi, Asymptotic profile of the positive steady state for an SIS epidemic reaction–diffusion model: Effects of epidemic risk and population movement, Physica D 259 (2013) 8–25.
[23] H.L. Smith, Monotone Dynamical Systems: An Introduction to the Theory of Competitive and Cooperative Systems, American Mathematical Soc., 1995.
[24] H.L. Smith, X.-Q. Zhao, Robust persistence for semidynamical systems, Nonlinear Anal. 47 (2001) 6169–6179.
[25] P. Song, Y. Lou, Y. Xiao, A spatial SEIRS reaction–diffusion model in heterogeneous environment, J. Differential Equations 267 (2019) 5084–5114.
[26] H.R. Thieme, Spectral bound and reproduction number for intinite-dimensional population structure and time heterogeneity, SIAM J. Appl. Math. 70 (2009) 188–211.
[27] X. Wang, D. Gao, J. Wang, Influence of human behavior on cholera dynamics, Math. Biosci. 267 (2015) 41–52.
[28] X. Wang, D. Posny, J. Wang, A reaction-convection–diffusion model for cholera spatial dynamics, Disc. Contin. Dyn. Syst. Ser. B 21 (2016) 2785–2809.
[29] F.-B. Wang, J. Shi, X. Zou, Dynamics of a host-pathogen system on a bounded spatial domain, Commun. Pure Appl. Anal. 14 (6) (2015) 2535–2560.
[30] X. Wang, J. Wang, Analysis of cholera epidemics with bacterial growth and spatial movement, J. Biol. Dyn. 9 (1) (2015) 233–261.
[31] X. Wang, F.-B. Wang, Impact of bacterial hyperinfectivity on cholera epidemics in a spatially heterogeneous environment, J. Math. Anal. Appl. 480 (2019) 123407.
[32] J. Wang, J. Wang, Analysis of a reaction–diffusion cholera model with distinct dispersal rates in the human population, J. Dynam. Differential Equations 33 (2021) 549–575.
[33] J. Wang, X. Wu, Dynamics and profiles of a diffusive cholera model with bacterial hyperinfectivity and distinct dispersal rates, J. Dynam. Differential Equations http://dx.doi.org/10.1007/s10884-021-09975-3.
[34] J. Wang, F. Xie, T. Kuniya, Analysis of a reaction–diffusion cholera epidemic model in a spatially heterogeneous environment, Commun. Nonlinear Sci. Numer. Simul. 80 (2020) 104951.
[35] W. Wang, X.-Q. Zhao, Basic reproduction number for reaction–diffusion epidemic models, SIAM J. Appl. Dyn. Syst. 11 (2012) 1652–1673.
[36] X. Wang, X.-Q. Zhao, J. Wang, A cholera epidemic model in a spatiotemporally heterogeneous environemnt, J. Math. Anal. Appl. 468 (2018) 893–912.
[37] Y. Wu, X. Zou, Asymptotic profiles of steady states for a diffusive SIS epidemic model with mass action infection mechanism, J. Differential Equations 261 (2016) 4424–4447.
[38] Y. Wu, X. Zou, Dynamics and profiles of a diffusive host-pathogen system with distinct dispersal rates, J. Differential Equations 264 (2018) 4989–5024.
[39] K. Yamazaki, X. Wang, Global well-posedness and asymptotic behavior of solutions to a reaction-convection–diffusion cholera epidemic model, Discrete Contin. Dyn. Syst. Ser. B 21 (2016) 1297–1316.
[40] K. Yamazaki, X. Wang, Global stability and uniform persistence of the reaction-convection–diffusion cholera epidemic model, Math. Biosci. Eng. 14 (2) (2017) 559–579.
[41] L. Zhang, Z. Wang, Y. Zhang, Dynamics of a reaction–diffusion waterborne pathogen model with direct and indirect transmission, Comput. Math. Appl. 72 (2016) 202–215.
[42] X. Zhang, Y. Zhang, Spatial dynamics of a reaction–diffusion cholera model with spatial heterogeneity, Disc. Contin. Dyn. Syst. Ser. B 23 (2018) 2625–2640.