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Threshold Dynamics of a Spatially Heterogeneous SVEIAR-B Reaction-Diffusion Model under Media Intervention *

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Abstract. This paper investigates a class of spatially heterogeneous SVEIAR-B reaction-diffusion epidemic models that incorporate media intervention and environmental pathogen transmission pathways. The model characterizes a dual intervention mechanism of media reports in terms of reducing human contact rates and enhancing the intensity of environmental disinfection. By applying semigroup theory and linear operator theory, the global existence, uniqueness, and ultimate boundedness of the non-negative classical solution are established. By defining the basic reproduction number $\mathcal{R}_0$, the threshold dynamics of the system are analyzed: when $\mathcal{R}_0 < 1$, the disease-free equilibrium is globally asymptotically stable, leading to the extinction of the disease; when $\mathcal{R}_0 > 1$, the system exhibits uniform persistence. Numerical simulations are performed to verify the theoretical results. Furthermore, the impacts of parameters such as media intervention intensity and vaccination rates on $\mathcal{R}_0$ are discussed, alongside the role of spatial heterogeneity in shaping the steady-state distribution of infected individuals.

Keywords: spatial heterogeneity; SVEIAR-B model; media intervention; reaction-diffusion equations; basic reproduction number; stability analysis

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1 Introduction

Since the outbreak of the COVID-19 pandemic in 2019, the transmission and dissemination of infectious diseases have once again emerged as a focal point of global scholarly attention[1–3]. To gain a profound understanding of disease transmission mechanisms and to formulate effective prevention and control strategies, mathematical modeling has become an indispensable tool for researchers to investigate the dynamic evolution of epidemics[4]. Kermack and McKendrick pioneered the SIR compartmental model, which categorizes the population into susceptible (S), infected (I), and recovered (R) individuals[5]. Subsequently, researchers have extensively extended these classical models, with a particular focus on exploring the impact of vaccination on the evolution of epidemics. Gumel et al.[6] demonstrated that vaccination coverage and vaccine efficacy are equally critical for alleviating the disease burden. Similarly, Zou et al. [7] established SVEIR models to assess the impact of imperfect vaccination on infectious diseases such as tuberculosis and SARS.

In epidemiological research, environmental heterogeneity is recognized as a pivotal factor governing transmission patterns. Influenced by various geographical and social determinantssuch as altitude, temperature, humidity, and population densitythe severity of epidemic outbreaks exhibits significant regional variations. Typically, areas characterized by high population density and advanced transportation networks experience spread rates substantially higher than those in remote regions. To characterize such spatiotemporal non-uniformity, an increasing number of reaction-diffusion equation models have been employed to investigate the spatial dynamics of infectious diseases[8–10]. Within these models, the incidence function depends not only on the population size of each compartment but is also intrinsically linked to spatial location[11, 12].

Beyond direct person-to-person transmission, the indirect role of environmental pathogens in disease spread has garnered significant attention in recent years. Regarding diseases such as influenza, cholera, dengue fever,

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and COVID-19, pathogens can persist in the external environment for varying durations, thereby establishing indirect environment-to-human transmission pathways[13, 14]. Moreover, the presence of asymptomatic carriers, characterized by high concealment and the associated difficulty of timely detection and isolation, poses a formidable challenge to epidemic control. Concurrently, the intervention effect of media reports during the initial stages of an outbreak is non-negligible. Effective media campaigns can guide the public in practicing social distancing and wearing masks, which in turn reduces the effective contact rate[16–18]. Furthermore, driven by media-disseminated information, relevant authorities often intensify disinfection and sterilization in public areas, accelerating the clearance rate of pathogens from the environment.

However, most extant epidemic models are formulated based on ordinary differential equations (ODEs), frequently overlooking the intricate interplay between spatial heterogeneity and environmental pathogen reservoirs. While recent studies have begun to explore diffusive systems under the influence of media coverage, scant research has successfully integrated the dual intervention mechanisms of media specifically, the concurrent reduction of human contact rates and the intensification of environmental disinfection within a comprehensive spatially heterogeneous framework that simultaneously accounts for vaccination, asymptomatic infection, and environmental transmission.

Motivated by the aforementioned literature, this paper proposes and investigates a class of SVEIAR-B reaction-diffusion epidemic models within a spatially heterogeneous environment. The model comprehensively incorporates vaccination, asymptomatic infection, and an environmental pathogen compartment. Furthermore, it assumes that media coverage not only reduces the effective contact rate between susceptible individuals and infectious sources but also enhances the clearance rate of pathogens in the environment. To this end, we establish the following SVEIAR-B reaction-diffusion model

$$\left\{ \begin{array}{l} \frac{\partial S(t, x)}{\partial t} = \nabla \cdot (D_1(x) \nabla S) + \Lambda(x) - [\beta(x) - \beta_1(x)f(I)](\beta_I(x)I + \beta_A(x)A + \beta_B(x)B)S \\ \quad - (p(x) + \mu(x))S, \\ \frac{\partial V(t, x)}{\partial t} = \nabla \cdot (D_2(x) \nabla V) + p(x)S - \sigma(x)[\beta(x) - \beta_1(x)f(I)](\beta_I(x)I + \beta_A(x)A \\ \quad + \beta_B(x)B)V - \mu(x)V, \\ \frac{\partial E(t, x)}{\partial t} = \nabla \cdot (D_3(x) \nabla E) + [\beta(x) - \beta_1(x)f(I)](\beta_I(x)I + \beta_A(x)A + \beta_B(x)B)(S \\ \quad + \sigma(x)V) - (\alpha_1(x) + \alpha_2(x) + \mu(x))E, \\ \frac{\partial I(t, x)}{\partial t} = \nabla \cdot (D_4(x) \nabla I) + \alpha_1(x)E - (\delta_1(x) + \eta(x) + \mu(x))I, \\ \frac{\partial A(t, x)}{\partial t} = \nabla \cdot (D_5(x) \nabla A) + \alpha_2(x)E - (\delta_2(x) + \mu(x))A, \\ \frac{\partial R(t, x)}{\partial t} = \nabla \cdot (D_6(x) \nabla R) + \delta_1(x)I + \delta_2(x)A - \mu(x)R, \\ \frac{\partial B(t, x)}{\partial t} = \nabla \cdot (D_7(x) \nabla B) + g_1(x)I + g_2(x)A - (d_B(x) + \psi(I))B. \end{array} \right. \quad (1.1)$$

where $x \in \Omega$ and $t > 0$. The model is subject to homogeneous Neumann boundary conditions (NBC)

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

Here, $\Omega \subset \mathbb{R}^n$ is a bounded domain with a smooth boundary $\partial\Omega$, and $\frac{\partial}{\partial n}$ denotes the outward normal derivative on $\partial\Omega$. The variables $S(t, x)$, $V(t, x)$, $E(t, x)$, $I(t, x)$, $A(t, x)$, $R(t, x)$ and $B(t, x)$ denote the population densities of susceptible, vaccinated, exposed, symptomatic infected, asymptomatic infected, and recovered individuals, as well as the concentration of environmental pathogens at time t and location x , respectively. The terms $D_i(x)$ ($i = 1, 2, \dots, 7$) represent the diffusion coefficients for the corresponding populations or pathogens at location x . It is assumed that all parameters in the model are positive, continuous, and bounded functions

on $\bar{\Omega}$. The specific biological meanings of these parameters are detailed in Table 1. In our model, the media response functions $f(I)$ and $\psi(I)$ are assumed to depend solely on the density of symptomatic individuals I . This assumption reflects the epidemiological reality that public health reporting and news coverage typically prioritize confirmed clinical cases, as asymptomatic carriers often remain undetected and thus do not directly trigger public risk-avoidance behaviors or intensified disinfection measures.

Table 1: Descriptions for parameters in the model (1.1)

Parameters	Meanings
$\mu(\cdot)$	Natural death rate
$\Lambda(\cdot)$	Recruitment rate of susceptible individuals
$p(\cdot)$	Vaccination rate
$\sigma(\cdot)$	Factor of breakthrough infection for vaccinated individuals V
$\beta(\cdot)$	Baseline infection rate
$\beta_1(\cdot)$	Maximum reduction intensity of the contact rate due to media coverage
$\beta_I(\cdot)$	Transmission rate from symptomatic individuals I to susceptibles
$\beta_A(\cdot)$	Effective contact rate and transmission probability from asymptomatic individuals A to susceptible population
$d_B(\cdot)$	Natural decay rate of environmental pathogens
$\beta_B(\cdot)$	Rate of indirect infection for susceptible individuals via exposure to environmental pathogens B
$\alpha_1(\cdot)$	Progression rate from exposed to symptomatic individuals
$\alpha_2(\cdot)$	Progression rate from exposed to asymptomatic individuals
$\delta_1(\cdot)$	Recovery rate of symptomatic individuals
$\delta_2(\cdot)$	Recovery rate of asymptomatic individuals
$\eta(\cdot)$	Disease-induced death rate of symptomatic individuals
$g_1(\cdot)$	Shedding rate of pathogens into the environment by symptomatic individuals
$g_2(\cdot)$	Shedding rate of pathogens into the environment by asymptomatic individuals
$\psi(I)$	Anthropogenic environmental disinfection rate in response to media reports

2 Preliminaries

In this section, we provide the mathematical assumptions, function space definitions, and notations necessary for the analysis of model (1.1). To characterize the practical impact of media reports on disease transmission and environmental disinfection, and to satisfy both epidemiological realism and the requirements of dynamical analysis, we propose the following assumptions regarding the nonlinear functions $f(I)$ and $\psi(I)$.

(H₁) $f(I)$ and $\psi(I)$ are C^1 monotonically increasing functions on $[0, +\infty)$ such that $f(0) = 0, \psi(0) = 0, \lim_{I \rightarrow \infty} f(I) = 1$, and $\lim_{I \rightarrow \infty} \psi(I) = \psi_{\max} > 0$.

(H₂) The risk avoidance factors induced by media reports satisfy $\beta(x) > \beta_1(x)$ for all $x \in \bar{\Omega}$, ensuring that the overall effective contact rate $[\beta(x) - \beta_1(x)f(I)]$ remains positive.

(H₃) The incidence function is strictly monotonically increasing with respect to the infectious population I ; specifically, it satisfies: $\frac{\partial}{\partial I} \left\{ [\beta(x) - \beta_1(x)f(I)] \left(I + \frac{\beta_A(x)}{\beta_I(x)} A + \frac{\beta_B(x)}{\beta_I(x)} B \right) \right\} > 0$.

Let $\bar{\Omega} \subset \mathbb{R}^n$ denote a bounded domain with a smooth boundary $\partial\Omega$. Let $X := C(\bar{\Omega}, \mathbb{R}^7)$ be the Banach space of continuous functions defined on $\bar{\Omega}$, equipped with the norm $\|\phi\| = \sum_{i=1}^7 \|\phi_i\|_\infty$. The corresponding positive cone is defined as $X_+ = C(\bar{\Omega}, \mathbb{R}_+^7)$. To characterize the influence of spatial heterogeneity on the parameters, for any continuous function $\aleph(x)$ defined on $\bar{\Omega}$, we denote its maximum and minimum values by $\aleph^* := \max_{x \in \bar{\Omega}} \aleph(x)$ and $\aleph_* := \min_{x \in \bar{\Omega}} \aleph(x)$ respectively.

Define the operator $L_i \phi := \nabla \cdot (D_i(x) \nabla \phi) - \pi_i(x) \phi$. Let $\{A_i(t)\}_{t \geq 0}$ ($i = 1, \dots, 7$) be the C_0 semigroup generated by the operator L_i subject to homogeneous Neumann boundary conditions, where $\pi_1(x) = p(x) + \mu(x)$, $\pi_2(x) = \mu(x)$, $\pi_3(x) = \alpha_1(x) + \alpha_2(x) + \mu(x)$, $\pi_4(x) = \delta_1(x) + \eta(x) + \mu(x)$, $\pi_5(x) = \delta_2(x) + \mu(x)$, $\pi_6(x) = \mu(x)$ and $\pi_7(x) = d_B(x)$. It follows that

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

where $G_i(t, x, y)$ is the Green function corresponding to the operator $\nabla \cdot (D_i(x) \nabla) - \pi_i(x)$ under homogeneous Neumann boundary conditions (NBC). Furthermore, based on ([28], Corollary 7.2.3), the operator $A_i(t)$ is compact and strongly positive. Consequently, there exists a constant $M_i > 0$ such that $\|A_i(t)\| \leq M_i e^{\alpha_i t}$ for all $t \geq 0$, where $\alpha_i < 0$ is the principal eigenvalue of the operator $\nabla \cdot (D_i(x) \nabla) - \pi_i(x)$ subject to NBC. For any initial value function $\phi = (\phi_1, \phi_2, \phi_3, \phi_4, \phi_5, \phi_6, \phi_7)^T \in X_+$, let $u(t, \cdot, \phi) = (S, V, E, I, A, R, B)^T$ denote the solution of model (1.1). By defining the incidence term as $\mathcal{J}(x, \phi) = [\beta(x) - \beta_1(x)f(\phi_4)](\beta_I(x)\phi_4 + \beta_A(x)\phi_5 + \beta_B(x)\phi_7)$, we have

$$\begin{cases} F_1(\phi)(x) = \Lambda(x) - \mathcal{J}(x, \phi)\phi_1(x), \\ F_2(\phi)(x) = p(x)\phi_1(x) - \sigma(x)\mathcal{J}(x, \phi)\phi_2(x), \\ F_3(\phi)(x) = \mathcal{J}(x, \phi)(\phi_1(x) + \sigma(x)\phi_2(x)), \\ F_4(\phi)(x) = \alpha_1(x)\phi_3(x), \\ F_5(\phi)(x) = \alpha_2(x)\phi_3(x), \\ F_6(\phi)(x) = \delta_1(x)\phi_4(x) + \delta_2(x)\phi_5(x), \\ F_7(\phi)(x) = g_1(x)\phi_4(x) + g_2(x)\phi_5(x) - \psi(\phi_4(x))\phi_7(x), \end{cases} \quad (2.2)$$

Then, model (1.1) can be reformulated into the following abstract integral equation form:

$$\begin{cases} u(t, \cdot, \phi) = \mathcal{A}(t)\phi + \int_0^t \mathcal{A}(t-s)\mathcal{F}(u(s, \cdot, \phi))ds, \\ u(0) = \phi, \end{cases} \quad (2.3)$$

where $\mathcal{A}(t) = \text{diag}(A_1(t), A_2(t), A_3(t), A_4(t), A_5(t), A_6(t), A_7(t))$, and $\mathcal{F}(\phi)(x) = (F_1(\phi)(x), F_2(\phi)(x), F_3(\phi)(x), F_4(\phi)(x), F_5(\phi)(x), F_6(\phi)(x), F_7(\phi)(x))^T$.

3 The well-posedness of the model

In this section, we establish the existence of the global solution and the ultimate boundedness of the solutions for model (1.1).

Lemma 3.1. *For any initial value function $\phi = (\phi_1, \phi_2, \phi_3, \phi_4, \phi_5, \phi_6, \phi_7)^T \in X_+$, model (1.1) admits a unique non-negative solution $u(t, \cdot, \phi) = (S, V, E, I, A, R, B)^T$. This solution is defined on the interval $[0, \tau_\infty)$ with $\tau_\infty \leq \infty$. Furthermore, the solution is a classical solution.*

Proof. Similar to the proof of in ([20], Lemma 1), it suffices to verify that for all $\phi \in X_+$, the following subtangential condition is satisfied

$$\lim_{h \rightarrow 0^+} \frac{1}{h} \text{dist}(\phi + h\mathcal{F}(\phi), X_+) = 0, \quad \phi \in X_+.$$

It can be verified that for any $\phi \in X_+$ and sufficiently small $h \geq 0$,

$$\begin{aligned}
& \phi(x) + h\mathcal{F}(\phi)(x) \\
&= \begin{pmatrix} \phi_1(x) + h\{\Lambda(x) - \mathcal{J}(x, \phi)\phi_1(x)\} \\ \phi_2(x) + h\{p(x)\phi_1(x) - \sigma(x)\mathcal{J}(x, \phi)\phi_2(x)\} \\ \phi_3(x) + h\{\mathcal{J}(x, \phi)(\phi_1(x) + \sigma(x)\phi_2(x))\} \\ \phi_4(x) + h\{\alpha_1(x)\phi_3(x)\} \\ \phi_5(x) + h\{\alpha_2(x)\phi_3(x)\} \\ \phi_6(x) + h\{\delta_1(x)\phi_4(x) + \delta_2(x)\phi_5(x)\} \\ \phi_7(x) + h\{g_1(x)\phi_4(x) + g_2(x)\phi_5(x) - \psi(\phi_4(x))\phi_7(x)\} \end{pmatrix} \\
&\geq \begin{pmatrix} \phi_1(x) - h\{[\beta(x) - \beta_1(x)f(\phi_4)](\beta_I(x)\phi_4 + \beta_A(x)\phi_5 + \beta_B(x)\phi_7)\phi_1(x)\} \\ \phi_2(x) - h\{\sigma(x)[\beta(x) - \beta_1(x)f(\phi_4)](\beta_I(x)\phi_4 + \beta_A(x)\phi_5 + \beta_B(x)\phi_7)\phi_2(x)\} \\ \phi_3(x) \\ \phi_4(x) \\ \phi_5(x) \\ \phi_6(x) \\ \phi_7(x) - h[\psi(\phi_4(x))\phi_7(x)] \end{pmatrix} \\
&> 0,
\end{aligned}$$

where $\mathcal{J}(x, \phi) = [\beta(x) - \beta_1(x)f(\phi_4)](\beta_I(x)\phi_4 + \beta_A(x)\phi_5 + \beta_B(x)\phi_7)$, which indicates that $\phi + h\mathcal{F}(\phi) \in X_+$. This completes the proof. $\square$

To proceed with our analysis, we first consider the following system,

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

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

Lemma 3.2. [21] *System (3.1) admits a unique positive steady state $\omega^0(x)$ satisfying the following equations*

$$\begin{cases} \nabla \cdot (d(x)\nabla \omega^0(x)) + \alpha(x) - \beta(x)\omega^0(x) = 0, & x \in \Omega, \\ \frac{\partial \omega^0(x)}{\partial n} = 0, & x \in \partial\Omega, \end{cases} \quad (3.2)$$

and this steady state is globally asymptotically stable in $C(\bar{\Omega}, \mathbb{R}_+)$. Moreover, when $\alpha(\cdot) \equiv a$ and $\beta(\cdot) \equiv b$ are positive constants, then $\omega^0(x) = \frac{a}{b}$.

Theorem 3.3. *For any initial value function $\phi = (\phi_1, \phi_2, \phi_3, \phi_4, \phi_5, \phi_6, \phi_7)^T \in X_+$, model (1.1) admits a unique non-negative classical solution $u(t, \cdot, \phi)$, which is ultimately bounded on $\bar{\Omega}$.*

Proof. Following standard arguments (see, e.g., ([22], Theorem 3.2)), it suffices to establish the boundedness of the solution in $\bar{\Omega} \times [0, \tau_\infty)$. From the first equation of model (1.1), it follows that

$$\frac{\partial S}{\partial t} \leq \nabla \cdot (D_1(x)\nabla S) + \Lambda^* - (p_* + \mu_*)S, \quad t \in [0, \tau_\infty), x \in \bar{\Omega}, \quad (3.3)$$

By the comparison principle [23] and Lemma 2, there exists a constant $M_1 > 0$ such that $S(t, x) \leq M_1$ for all $t \in [0, \tau_\infty)$ and $x \in \bar{\Omega}$. Similarly, from the second equation of model (1.1), we obtain

$$\frac{\partial V}{\partial t} \leq \nabla \cdot (D_2(x)\nabla V) + p^*M_1 - \mu_*V, \quad t \in [0, \tau_\infty), x \in \bar{\Omega}, \quad (3.4)$$

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

$$\left\{ \begin{array}{l} \frac{\partial E(t, x)}{\partial t} \leq \nabla \cdot (D_3(x) \nabla E) + \beta^* (\beta_I^* I + \beta_A^* A + \beta_B^* B) (M_1 + \sigma^* M_2) - (\alpha_{1*} + \alpha_{2*} + \mu_*) E, \\ \frac{\partial I(t, x)}{\partial t} \leq \nabla \cdot (D_4(x) \nabla I) + \alpha_1^* E - (\delta_{1*} + \eta_* + \mu_*) I, \\ \frac{\partial A(t, x)}{\partial t} \leq \nabla \cdot (D_5(x) \nabla A) + \alpha_2^* E - (\delta_{2*} + \mu_*) A, \\ \frac{\partial R(t, x)}{\partial t} \leq \nabla \cdot (D_6(x) \nabla R) + \delta_1^* I + \delta_2^* A - \mu_* R, \\ \frac{\partial B(t, x)}{\partial t} \leq \nabla \cdot (D_7(x) \nabla B) + g_1^* I + g_2^* A - (d_{B*} + \psi_*) B, \\ \frac{\partial E}{\partial n} = \frac{\partial I}{\partial n} = \frac{\partial A}{\partial n} = \frac{\partial R}{\partial n} = \frac{\partial B}{\partial n} = 0, x \in \partial\Omega, t > 0, \end{array} \right. \quad t \in [0, \tau_\infty), x \in \Omega, \quad (3.5)$$

Consider the following linear comparison system:

$$\left\{ \begin{array}{l} \frac{\partial \nu_1}{\partial t} = \nabla \cdot (D_3(x) \nabla \nu_1) + \beta^* \beta_I^* (M_1 + \sigma^* M_2) \nu_2 + \beta^* \beta_A^* (M_1 + \sigma^* M_2) \nu_3 + \beta^* \beta_B^* (M_1 + \sigma^* M_2) \nu_5 \\ \quad - (\alpha_{1*} + \alpha_{2*} + \mu_*) \nu_1, \\ \frac{\partial \nu_2}{\partial t} = \nabla \cdot (D_4(x) \nabla \nu_2) + \alpha_1^* \nu_1 - (\delta_{1*} + \eta_* + \mu_*) \nu_2, \\ \frac{\partial \nu_3}{\partial t} = \nabla \cdot (D_5(x) \nabla \nu_3) + \alpha_2^* \nu_1 - (\delta_{2*} + \mu_*) \nu_3, \\ \frac{\partial \nu_4}{\partial t} = \nabla \cdot (D_6(x) \nabla \nu_4) + \delta_1^* \nu_2 + \delta_2^* \nu_3 - \mu_* \nu_4, \\ \frac{\partial \nu_5}{\partial t} = \nabla \cdot (D_7(x) \nabla \nu_5) + g_1^* \nu_2 + g_2^* \nu_3 - (d_{B*} + \psi_*) \nu_5, \\ \frac{\partial \nu_1}{\partial n} = \frac{\partial \nu_2}{\partial n} = \frac{\partial \nu_3}{\partial n} = \frac{\partial \nu_4}{\partial n} = \frac{\partial \nu_5}{\partial n} = 0, \quad x \in \partial\Omega, t > 0, \end{array} \right. \quad t > 0, x \in \Omega, \quad (3.6)$$

By the Krein-Rutman theorem [24], the eigenvalue problem associated with system (3.6) admits a strongly positive eigenfunction $\xi = (\xi_1, \xi_2, \xi_3, \xi_4, \xi_5)$ corresponding to the principal eigenvalue λ^* . Consequently, system (3.6) possesses a solution of the form $ce^{\lambda^* t} \xi(x)$, where c is a positive constant satisfying $c\xi \geq (E(x, 0), I(x, 0), A(x, 0), R(x, 0), B(x, 0))$ for all $x \in \bar{\Omega}$. According to the comparison principle,

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

Since the term $ce^{\lambda^* t} \xi(x)$ is bounded on any finite time interval $[0, T] \subset [0, \tau_\infty)$, it follows that the components $E(t, x), I(t, x), A(t, x), R(t, x)$ and $B(t, x)$ do not blow up in finite time. Together with the proven boundedness of $S(t, x)$ and $V(t, x)$, we conclude that the solution $u(t, \cdot, \phi)$ exists for all $t > 0$, i.e., $\tau_\infty = \infty$, ensuring the global existence of the solution to model (1.1). The uniform ultimate boundedness of these components will be further established in the subsequent section using the total population estimate.

Next, we establish the ultimate boundedness of the solutions. By the comparison principle, it follows from inequalities (3.3), (3.4) and Lemma 3.2 that there exist constants $N_1, N_2 > 0$ and time points $t_1, t_2 > 0$ such that $S(t, x) \leq N_1$ and $V(t, x) \leq N_2$ for $t \geq \max\{t_1, t_2\}$. This indicates that $S(t, x)$ and $V(t, x)$ are ultimately bounded on $\bar{\Omega}$.

Let $P(t) = \int_{\Omega} (S(t, x) + V(t, x) + E(t, x) + I(t, x) + A(t, x) + R(t, x)) dx$ denote the total population size. Utilizing the first six equations of model (1.1) and the homogeneous Neumann boundary conditions (NBC), we have

$$\begin{aligned} \frac{dP(t)}{dt} &= \int_{\Omega} [\Lambda(x) - \mu(x)(S + V + E + I + A + R) - \eta(x)I] dx \\ &\leq \Lambda^* |\Omega| - \mu_* P(t), t \in [0, \tau_x), \end{aligned} \quad (3.7)$$

where $|\Omega|$ is the measure of the domain Ω . Therefore, one can find positive constants N_3, N_4 and t_3 such that $P(t) \leq N_3$ whenever $t \geq t_3$. It follows that

$$\int_{\Omega} E(t, x) dx \leq N_3, \int_{\Omega} I(t, x) dx \leq N_3, \int_{\Omega} A(t, x) dx \leq N_3, \int_{\Omega} R(t, x) dx \leq N_3, \quad (3.8)$$

Regarding the environmental pathogens $B(t, x)$, integrating the last equation of model (1.1) over Ω and utilizing the homogeneous Neumann boundary conditions yields

$$\begin{aligned} \frac{d}{dt} \int_{\Omega} B(t, x) dx &= \int_{\Omega} [g_1(x)I + g_2(x)A - (d_B(x) + \psi(I))B] dx \\ &\leq g_1^* N_3 + g_2^* N_3 - d_{B^*} \int_{\Omega} B(t, x) dx \end{aligned}$$

This implies that $\int_{\Omega} B(t, x) dx$ is also ultimately bounded, with $\int_{\Omega} B(t, x) dx \leq N_4$. By an argument similar to that in ([25], Theorem 1), for any $t \geq t_4 = \max\{t_1, t_2, t_3\}$, we obtain

$$\begin{aligned} E(t, x) &= A_3(t)E(t_4, x) + \int_{t_4}^t A_3(t-s) \{ [\beta(x) - \beta_1(x)f(I(s, x))] (\beta_I(x)I(s, x) + \beta_A(x)A(s, x) \\ &\quad + \beta_B(x)B(s, x)) (S(s, x) + \sigma(x)V(s, x)) \} ds \\ &\leq M_3 e^{\alpha_3(t-t_4)} \|E(t_4, \cdot)\| + \omega_3 \int_{t_4}^t e^{-(\alpha_{1*} + \alpha_{2*} + \mu_*)(t-s)} \{ \beta^* (\beta_I^* N_3 + \beta_A^* N_3 + \beta_B^* N_4) (N_1 + \sigma^* N_2) \} ds \\ &\leq M_3 e^{\alpha_3(t-t_4)} \|E(t_4, \cdot)\| + \frac{\omega_3 \beta^* (N_1 + \sigma^* N_2) (\beta_I^* N_3 + \beta_A^* N_3 + \beta_B^* N_4)}{\alpha_{1*} + \alpha_{2*} + \mu_*}, \end{aligned}$$

Therefore, we have

$$\limsup_{t \rightarrow \infty} \|E(t, \cdot)\|_{\mathbb{Z}} \leq \frac{\omega_3 \beta^* (N_1 + \sigma^* N_2) (\beta_I^* N_3 + \beta_A^* N_3 + \beta_B^* N_4)}{\alpha_{1*} + \alpha_{2*} + \mu_*}.$$

This implies that $E(t, x)$ is ultimately bounded on $\bar{\Omega}$. Similarly, for $I(t, x)$ and $A(t, x)$, from the fourth and fifth equations of model (1.1), we obtain

$$\limsup_{t \rightarrow \infty} \|I(t, \cdot)\|_{\mathbb{Z}} \leq \frac{\omega_4 \alpha_1^* N_3}{\delta_{1*} + \eta_* + \mu_*}, \quad \limsup_{t \rightarrow \infty} \|A(t, \cdot)\|_{\mathbb{Z}} \leq \frac{\omega_5 \alpha_2^* N_3}{\delta_{2*} + \mu_*},$$

For the recovered population $R(t, x)$, we have

$$\limsup_{t \rightarrow \infty} \|R(t, \cdot)\|_{\mathbb{Z}} \leq \frac{\omega_6 (\delta_1^* N_3 + \delta_2^* N_3)}{\mu_*},$$

Finally, for the environmental pathogen reservoir $B(t, x)$, it follows from the last equation and the aforementioned boundedness results that

$$\limsup_{t \rightarrow \infty} \|B(t, \cdot)\|_{\mathbb{Z}} \leq \frac{\omega_7 (g_1^* N_3 + g_2^* N_3)}{d_{B^*}},$$

where $\omega_i > 0 (i = 3, \dots, 7)$ are constants. In summary, the solution $u(t, \cdot, \phi)$ of model (1.1) is ultimately bounded on $\bar{\Omega}$. This completes the proof. $\square$

Corollary 3.4. *The solution semigroup $\{Q(t)\}_{t \geq 0}$ on X_+ generated by model (1.1) possesses a compact global attractor.*

Proof. It follows from Theorem 3.3 that system (1.1) is ultimately bounded, which implies that the solution semigroup $\{Q(t)\}_{t \geq 0}$ generated by model (1.1) is point dissipative on X_+ . Furthermore, $Q(t)$ is compact for any $t > 0$ according to [26]. Therefore, according to [27], the semigroup $Q(t) : X_+ \rightarrow X_+$ possesses a compact global attractor in X_+ . This completes the proof. $\square$

4 Basic reproduction number

According to Lemma 3.2, system (1.1) admits a unique disease-free steady state $D_0(x) = (S^0(x), V^0(x), 0, 0, 0, 0, 0)$, where $S^0(x)$ and $V^0(x)$ are governed by the following system

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

By linearizing model (1.1) about the disease-free steady state $D_0(x)$, we focus on the infection-related compartments (E, I, A, B) . Since the recovered compartment $R(t, x)$ does not contribute to the generation of new infections, it is omitted from the linearized system. Let $\Phi(t, x) = (E(t, x), I(t, x), A(t, x), B(t, x))^T$, then we obtain the following linearized system,

$$\begin{cases} \frac{\partial E(t, x)}{\partial t} = \nabla \cdot (D_3(x) \nabla E) + \beta(x)[\beta_I(x)I + \beta_A(x)A + \beta_B(x)B](S^0(x) + \sigma(x)V^0(x)) \\ \quad - (\alpha_1(x) + \alpha_2(x) + \mu(x))E, \\ \frac{\partial I(t, x)}{\partial t} = \nabla \cdot (D_4(x) \nabla I) + \alpha_1(x)E - (\delta_1(x) + \eta(x) + \mu(x))I, \\ \frac{\partial A(t, x)}{\partial t} = \nabla \cdot (D_5(x) \nabla A) + \alpha_2(x)E - (\delta_2(x) + \mu(x))A, \\ \frac{\partial B(t, x)}{\partial t} = \nabla \cdot (D_7(x) \nabla B) + g_1(x)I + g_2(x)A - d_B(x)B, \\ \frac{\partial E}{\partial n} = \frac{\partial I}{\partial n} = \frac{\partial A}{\partial n} = \frac{\partial B}{\partial n} = 0, \end{cases} \quad t > 0, x \in \Omega, \quad (4.1)$$

Setting $(E, I, A, B) = (e^{\lambda t} \varpi_3(x), e^{\lambda t} \varpi_4(x), e^{\lambda t} \varpi_5(x), e^{\lambda t} \varpi_7(x))$, we obtain the following eigenvalue problem

$$\begin{cases} \lambda \varpi_3(x) = \nabla \cdot (D_3(x) \nabla \varpi_3(x)) + \beta(x)[\beta_I(x)\varpi_4(x) + \beta_A(x)\varpi_5(x) + \beta_B(x)\varpi_7(x)](S^0(x) \\ \quad + \sigma(x)V^0(x)) - (\alpha_1(x) + \alpha_2(x) + \mu(x))\varpi_3(x), \\ \lambda \varpi_4(x) = \nabla \cdot (D_4(x) \nabla \varpi_4(x)) + \alpha_1(x)\varpi_3(x) - (\delta_1(x) + \eta(x) + \mu(x))\varpi_4(x), \\ \lambda \varpi_5(x) = \nabla \cdot (D_5(x) \nabla \varpi_5(x)) + \alpha_2(x)\varpi_3(x) - (\delta_2(x) + \mu(x))\varpi_5(x), \\ \lambda \varpi_7(x) = \nabla \cdot (D_7(x) \nabla \varpi_7(x)) + g_1(x)\varpi_4(x) + g_2(x)\varpi_5(x) - d_B(x)\varpi_7(x), \\ \frac{\partial \varpi_3}{\partial n} = \frac{\partial \varpi_4}{\partial n} = \frac{\partial \varpi_5}{\partial n} = \frac{\partial \varpi_7}{\partial n} = 0, \end{cases} \quad t > 0, x \in \Omega, \quad (4.2)$$

According to ([28], Theorem 7.6.1), we have the following result

Lemma 4.1. *The eigenvalue problem (4.2) admits a unique principal eigenvalue $\lambda_0 = \lambda_0(S^0(x), V^0(x))$, and the corresponding eigenfunction $(\varpi_3(x), \varpi_4(x), \varpi_5(x), \varpi_7(x))$ is strongly positive.*

Define the following matrices

$$D(x) = \text{diag}(D_3(x), D_4(x), D_5(x), D_7(x)),$$

$$V(x) = \begin{pmatrix} \alpha_1(x) + \alpha_2(x) + \mu(x) & 0 & 0 & 0 \\ -\alpha_1(x) & \delta_1(x) + \eta(x) + \mu(x) & 0 & 0 \\ -\alpha_2(x) & 0 & \delta_2(x) + \mu(x) & 0 \\ 0 & -g_1(x) & -g_2(x) & d_B(x) \end{pmatrix},$$

$$F(x) = \begin{pmatrix} 0 & \beta(x)\beta_I(x)S(x) & \beta(x)\beta_A(x)S(x) & \beta(x)\beta_B(x)S(x) \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix},$$

where $S(x) = S^0(x) + \sigma(x)V^0(x)$. Let $Q(t)$ be the positive semigroup generated by the linear operator $\mathcal{A} := \nabla \cdot (D(x)\nabla \cdot) - V(x)$ subject to homogeneous Neumann boundary conditions. Define the next generation operator $L : C(\bar{\Omega}, \mathbb{R}^4) \rightarrow C(\bar{\Omega}, \mathbb{R}^4)$ as follows:

$$L(\psi)(x) = \int_0^{+\infty} F(x)[Q(t)\psi](x)dt,$$

Here, the function $\psi(x)$ denotes the initial distribution of infectious individuals at location x . According to the theory established by Thieme [29], the basic reproduction number R_0 for model (1.1) is defined as the spectral radius of the operator L , i.e., $R_0 := r(L)$. Combining Lemma 4.1 with the variational principle, it follows that the basic reproduction number R_0 serves as a crucial threshold for determining the evolutionary trend of the disease. Based on the construction above and the arguments in [30], we present the following lemma regarding the properties of R_0 .

Lemma 4.2. *The following properties hold regarding the basic reproduction number R_0 and the principal eigenvalue λ_0 :*

- (i) $R_0 - 1$ and λ_0 have the same sign;
- (ii) If $R_0 < 1$, the disease-free steady state $D_0(x)$ of system (1.1) is locally asymptotically stable;
- (iii) If $R_0 > 1$, the disease-free steady state $D_0(x)$ of system (1.1) is unstable.

5 Threshold dynamics

5.1 Disease extinction

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

Theorem 5.1. *If $R_0 < 1$, the disease-free steady state $D_0(x)$ of model (1.1) is globally asymptotically stable in X_+ .*

Proof. It follows from Lemma 4.2 that the disease-free steady state $D_0(x)$ is locally asymptotically stable when $R_0 < 1$. Therefore, to establish its global asymptotic stability, it suffices to show that $D_0(x)$ is globally attractive; that is, to prove that each component of the solution converges to the disease-free steady state as $t \rightarrow \infty$.

From the first equation of model (1.1), we have

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

By the comparison principle and Lemma 3.2, for any arbitrarily small $\epsilon_1 > 0$, there exists $t_1 > 0$ such that $S(t, x) \leq S^0(x) + \epsilon_1$ holds uniformly on $\bar{\Omega}$ for all $t > t_1$.

Similarly, from the second equation of model (1.1) and the estimate of S , we obtain

$$\frac{\partial V}{\partial t} \leq \nabla \cdot (D_2(x)\nabla V) + p(x)(S^0(x) + \epsilon_1) - \mu(x)V,$$

Applying the comparison principle once again, for any arbitrarily small $\epsilon_2 > 0$, there exists $t_2 > t_1$ such that $V(t, x) \leq V^0(x) + \epsilon_2$ holds uniformly on $\bar{\Omega}$ for all $t > t_2$.

Now, consider the infected compartments (E, I, A, B) . For $t > t_3 = \max\{t_1, t_2\}$, based on model (1.1) and the upper bound estimates for S and V , we obtain the following system of linear differential inequalities

$$\left\{ \begin{array}{l} \frac{\partial E}{\partial t} \leq \nabla \cdot (D_3(x) \nabla E) + \beta(x) [\beta_I(x)I + \beta_A(x)A + \beta_B(x)B] H_\epsilon(x) \\ \quad - (\alpha_1(x) + \alpha_2(x) + \mu(x)) E, \\ \frac{\partial I}{\partial t} \leq \nabla \cdot (D_4(x) \nabla I) + \alpha_1(x)E - (\delta_1(x) + \eta(x) + \mu(x))I, \\ \frac{\partial A}{\partial t} \leq \nabla \cdot (D_5(x) \nabla A) + \alpha_2(x)E - (\delta_2(x) + \mu(x))A, \\ \frac{\partial B}{\partial t} \leq \nabla \cdot (D_7(x) \nabla B) + g_1(x)I + g_2(x)A - d_B(x)B, \\ \frac{\partial E}{\partial n} = \frac{\partial I}{\partial n} = \frac{\partial A}{\partial n} = \frac{\partial B}{\partial n} = 0, x \in \partial\Omega, \end{array} \right. \quad x \in \Omega, \quad (5.1)$$

where $S_\epsilon(x) = S^0(x) + \epsilon_1$, $V_\epsilon(x) = V^0(x) + \epsilon_2$ and $H_\epsilon(x) = S_\epsilon(x) + \sigma(x)V_\epsilon(x)$. Consider the corresponding linear limiting system

$$\frac{\partial \Phi}{\partial t} = \nabla \cdot (D(x) \nabla \Phi) + (F_\epsilon - V)\Phi,$$

where $\Phi = (E, I, A, B)^T$. According to Lemma 4.2, the condition $R_0 < 1$ is equivalent to the principal eigenvalue of the unperturbed system at the disease-free steady state being negative, i.e., $\lambda_0(S^0, V^0) < 0$. Since the principal eigenvalue depends continuously on the coefficients of the operator, it follows that for sufficiently small $\epsilon_1, \epsilon_2 > 0$, the principal eigenvalue of the perturbed linear system satisfies $\lambda_0(S^0 + \epsilon_1, V^0 + \epsilon_2) < 0$.

Let $\phi_0(x)$ denote the strongly positive eigenfunction corresponding to the principal eigenvalue $\lambda_0 < 0$. By the comparison principle, there exists a constant $M > 0$ such that

$$(E(t, x), I(t, x), A(t, x), B(t, x))^T \leq M e^{\lambda_0(t-t_3)} \phi_0(x),$$

Since $\lambda_0 < 0$, the term on the right-hand side of the above inequality tends to zero as $t \rightarrow \infty$. Consequently, we have

$$\lim_{t \rightarrow \infty} (E(t, x), I(t, x), A(t, x), B(t, x)) = (0, 0, 0, 0),$$

uniformly for $x \in \bar{\Omega}$. Furthermore, considering the equation for the recovered compartment R : $\frac{\partial R}{\partial t} = \nabla \cdot (D_6(x) \nabla R) + \delta_1 I + \delta_2 A - \mu R$, it is easy to verify that $\lim_{t \rightarrow \infty} R(t, x) = 0$ as $I, A \rightarrow 0$.

As $(E, I, A, B, R) \rightarrow (0, 0, 0, 0, 0)$, model (1.1) reduces to the following asymptotically autonomous limiting system for (S, V)

$$\left\{ \begin{array}{l} \frac{\partial S}{\partial t} = \nabla \cdot (D_1(x) \nabla S) + \Lambda(x) - p(x)S - \mu(x)S, x \in \Omega, t > 0, \\ \frac{\partial V}{\partial t} = \nabla \cdot (D_2(x) \nabla V) + p(x)S - \mu(x)V, \\ \frac{\partial S}{\partial n} = \frac{\partial V}{\partial n} = 0, x \in \partial\Omega, t > 0, \end{array} \right. \quad x \in \Omega, t > 0, \quad (5.2)$$

According to Lemma 3.2, this system admits a unique positive steady state $(S^0(x), V^0(x))$, which is globally asymptotically stable. It follows from the theory of asymptotically autonomous semiflows that

$$\lim_{t \rightarrow \infty} S(t, x) = S^0(x), \quad \lim_{t \rightarrow \infty} V(t, x) = V^0(x),$$

uniformly on $\bar{\Omega}$.

Consequently, if $R_0 < 1$, the solutions of system (1.1) tend to the disease-free steady state $D_0(x)$. The proof is complete. $\square$

5.2 Uniform persistence of the disease

In this subsection, we demonstrate that R_0 serves as the threshold for disease persistence. To begin with, we present the following result.

Theorem 5.2. *If $R_0 > 1$, then system (1.1) is uniformly persistent. Specifically, there exists a constant $\varrho > 0$ such that for any initial function $\phi = (\phi_1, \phi_2, \phi_3, \phi_4, \phi_5, \phi_6, \phi_7) \in X_+$ satisfying $\phi_3 \neq 0, \phi_4 \neq 0, \phi_5 \neq 0$, and $\phi_7 \neq 0$, the corresponding solution $u(t, \cdot, \phi) = (S, V, E, I, A, R, B)$ satisfies*

$$\liminf_{t \rightarrow \infty} S(t, \cdot, \phi) \geq \varrho, \quad \liminf_{t \rightarrow \infty} V(t, \cdot, \phi) \geq \varrho, \quad \liminf_{t \rightarrow \infty} E(t, \cdot, \phi) \geq \varrho,$$

$$\liminf_{t \rightarrow \infty} I(t, \cdot, \phi) \geq \varrho, \quad \liminf_{t \rightarrow \infty} A(t, \cdot, \phi) \geq \varrho, \quad \liminf_{t \rightarrow \infty} R(t, \cdot, \phi) \geq \varrho, \quad \liminf_{t \rightarrow \infty} B(t, \cdot, \phi) \geq \varrho,$$

uniformly for $x \in \bar{\Omega}$. Furthermore, model (1.1) possesses at least one positive steady state $D^*(x) = (S^*(x), V^*(x), E^*(x), I^*(x), A^*(x), R^*(x), B^*(x))$.

Proof. To prove the uniform persistence of system (1.1), we first define the following sets:

$$X_0 := \{\phi = (\phi_1, \phi_2, \phi_3, \phi_4, \phi_5, \phi_6, \phi_7) \in X_+ : \phi_3 \neq 0, \phi_4 \neq 0, \phi_5 \neq 0, \phi_7 \neq 0\},$$

$$\partial X_0 := X_+ \setminus X_0 = \{\phi \in X_+ : \phi_3 = 0 \text{ or } \phi_4 = 0 \text{ or } \phi_5 = 0 \text{ or } \phi_7 = 0\}.$$

Obviously, X_0 is forward invariant under the solution semigroup $Q(t)$. According to Lemma 3.1, $Q(t)$ is point dissipative and possesses a global attractor. In what follows, the proof is completed through the following two claims: $\square$

Claim 5.3. $\bigcup_{\phi \in M_\partial} \omega(\phi) = \{D_0(x)\}$, where $M_\partial = \{\phi \in \partial X_0 : Q(t)\phi \in \partial X_0, t \geq 0\}$.

Proof. It is obvious that $\{D_0(x)\} \subset \bigcup_{\phi \in M_\partial} \omega(\phi)$. Thus, it suffices to show the reverse inclusion $\bigcup_{\phi \in M_\partial} \omega(\phi) \subset \{D_0(x)\}$. For any given $\phi \in M_\partial$, it follows from the definition of M_∂ that $Q(t)\phi \in \partial X_0$ for all $t \geq 0$. This implies that for all $t \geq 0$, either $E(t, \cdot, \phi) \equiv 0, I(t, \cdot, \phi) \equiv 0, A(t, \cdot, \phi) \equiv 0$ or $B(t, \cdot, \phi) \equiv 0$.

If there exists some $t_0 \geq 0$ such that $E(t_0, \cdot, \phi) \not\equiv 0$, it follows from the strong maximum principle for parabolic equations that $E(t, \cdot, \phi) > 0$ for all $t > t_0$. Similarly, due to the coupling relationships among the subsequent equations in model (1.1), one can show that $I(t, \cdot, \phi) > 0, A(t, \cdot, \phi) > 0$ and $B(t, \cdot, \phi) > 0$ for sufficiently large t . This contradicts the fact that $Q(t)\phi \in \partial X_0$. Therefore, we must have $E(t, \cdot, \phi) \equiv 0, I(t, \cdot, \phi) \equiv 0, A(t, \cdot, \phi) \equiv 0$ and $B(t, \cdot, \phi) \equiv 0$ for all $t \geq 0$. Substituting these conditions into the fourth and fifth equations of model (1.1), we obtain $\alpha_1 E - (\mu + \eta + \delta_1)I = 0$ and $\delta_1 I + \delta_2 A - \mu R = 0$. Since $E = I = A \equiv 0$ and $\mu(x) > 0$, it directly follows that $R(t, \cdot, \phi) \equiv 0$. Consequently, for $\phi \in M_\partial$, model (1.1) reduces to the system for (S, V) as shown in (5.2), subject to homogeneous Neumann boundary conditions. According to Lemma 3.2, this linear system admits a unique positive steady state $(S^0(x), V^0(x))$, which is globally asymptotically stable. This implies that $(S(t, \cdot, \phi), V(t, \cdot, \phi)) \rightarrow (S^0(x), V^0(x))$ as $t \rightarrow \infty$.

Consequently, for any $\phi \in M_\partial$, we have $\omega(\phi) = \{(S^0(x), V^0(x), 0, 0, 0, 0, 0)\} = \{D_0(x)\}$. $\square$

Claim 5.4. *If $R_0 > 1$, then there exists a constant $\lambda^\# > 0$ such that for any $\phi \in X_0$, the solution $u(t, \cdot, \phi)$ of model (1.1) satisfies*

$$\limsup_{t \rightarrow \infty} \|u(t, \cdot, \phi) - D_0(x)\|_X \geq \lambda^\#.$$

Proof. We proceed by contradiction. Suppose that Claim 5.4, is false. Then, for any arbitrarily small $\kappa > 0$, there exists a sufficiently large time $T_1 > 0$ such that for all $t \geq T_1$, the following inequalities hold uniformly for $x \in \bar{\Omega}$:

$$\begin{aligned} S^0(x) - \kappa &< S(t, x) < S^0(x) + \kappa, & V^0(x) - \kappa &< V(t, x) < V^0(x) + \kappa, \\ 0 &\leq E(t, x) < \kappa, & 0 &\leq I(t, x) < \kappa, & 0 &\leq A(t, x) < \kappa, & 0 &\leq B(t, x) < \kappa. \end{aligned}$$

According to model (1.1) and Assumption (H₁), since $f(0) = 0$, the term $\beta_1(x)f(I)$ approaches zero when I is small. Given that $\sigma(x)$ is the vaccine failure coefficient, for $t \geq T_1$, we can derive the following system of linear differential inequalities from the equations for the infected compartments (E, I, A, B) in model (1.1),

$$\left\{ \begin{array}{l} \frac{\partial E}{\partial t} \geq \nabla \cdot (D_3(x)\nabla E) + \beta_\kappa(x)S_\kappa(x) (\beta_I(x)I + \beta_A(x)A + \beta_B(x)B) \\ \quad - (\alpha_1(x) + \alpha_2(x) + \mu(x))E, \\ \frac{\partial I}{\partial t} \geq \nabla \cdot (D_4(x)\nabla I) + \alpha_1(x)E - (\delta_1(x) + \eta(x) + \mu(x))I, \\ \frac{\partial A}{\partial t} \geq \nabla \cdot (D_5(x)\nabla A) + \alpha_2(x)E - (\delta_2(x) + \mu(x))A, \\ \frac{\partial B}{\partial t} \geq \nabla \cdot (D_7(x)\nabla B) + g_1(x)I + g_2(x)A - (d_B(x) + \psi(\delta))B, \\ \frac{\partial E}{\partial n} = \frac{\partial I}{\partial n} = \frac{\partial A}{\partial n} = \frac{\partial B}{\partial n} = 0, x \in \partial\Omega, \end{array} \right. \quad x \in \Omega, \quad (5.3)$$

where $S_\kappa(x) = (S^0(x) - \kappa) + \sigma(x)(V^0(x) - \kappa)$ represents the perturbed total effective susceptible population, and $\beta_\kappa(x) = \beta(x) - \beta_1(x)f(\kappa)$ denotes the minimum infection rate under the influence of media intervention. Consider the linear eigenvalue problem associated with the aforementioned system of inequalities. Let $\lambda_0(\kappa)$ be the principal eigenvalue of the operator for this perturbed system subject to homogeneous Neumann boundary conditions. It is known that when $\kappa = 0$, this eigenvalue reduces to the principal eigenvalue λ_0 corresponding to the basic reproduction number R_0 . According to Lemma 4.2, $R_0 > 1$ is equivalent to $\lambda_0 > 0$. Since the eigenvalue $\lambda_0(\kappa)$ is continuous with respect to κ , there must exist a sufficiently small $\kappa > 0$ such that $\lambda_0(\kappa) > 0$ still holds.

Let $\Phi(x) = (\omega_3(x), \omega_4(x), \omega_5(x), \omega_7(x))^T$ be the strongly positive eigenfunction associated with $\lambda_0(\kappa)$. By the comparison principle for parabolic equations, there exists a constant $c_1 > 0$ such that for all $t \geq T_1$,

$$(E(t, x), I(t, x), A(t, x), B(t, x))^T \geq c_1 \Phi(x) e^{\lambda_0(\kappa)(t-T_1)}.$$

Since $\lambda_0(\kappa) > 0$, the term on the right-hand side grows exponentially to infinity as $t \rightarrow \infty$. That is,

$$\lim_{t \rightarrow \infty} E(t, x) = +\infty, \quad \lim_{t \rightarrow \infty} I(t, x) = +\infty, \quad \lim_{t \rightarrow \infty} A(t, x) = +\infty, \quad \lim_{t \rightarrow \infty} B(t, x) = +\infty.$$

However, according to the ultimate boundedness of the solution in Theorem 3.3, all solutions of model (1.1) are ultimately uniformly bounded on $\bar{\Omega}$. This leads to a clear contradiction. Thus, the initial assumption is false. This establishes the existence of a constant $\lambda^\# > 0$ such that $\limsup_{t \rightarrow \infty} \|u(t, \cdot, \phi) - D_0(x)\|_X \geq \lambda^\#$. $\square$

Corollary 5.5. *If $R_0 > 1$, then model (1.1) possesses at least one positive steady state $D^*(x) = (S^*(x), V^*(x), E^*(x), I^*(x), A^*(x), R^*(x), B^*(x))$.*

6 Global stability in a homogeneous spaces

In this section, we investigate the dynamical behavior of model (1.1) in a homogeneous space. In this case, all coefficients of model (1.1), except for the diffusion coefficients, are assumed to be positive constants.

$$\left\{ \begin{array}{l} \frac{\partial S}{\partial t} = \nabla \cdot (D_1(x) \nabla S) + \Lambda - [\beta - \beta_1 f(I)](\beta_I I + \beta_A A + \beta_B B)S - (p + \mu)S, \\ \frac{\partial V}{\partial t} = \nabla \cdot (D_2(x) \nabla V) + pS - \sigma[\beta - \beta_1 f(I)](\beta_I I + \beta_A A + \beta_B B)V - \mu V, \\ \frac{\partial E}{\partial t} = \nabla \cdot (D_3(x) \nabla E) + [\beta - \beta_1 f(I)](\beta_I I + \beta_A A + \beta_B B)(S + \sigma V) \\ \quad - (\alpha_1 + \alpha_2 + \mu)E, \\ \frac{\partial I}{\partial t} = \nabla \cdot (D_4(x) \nabla I) + \alpha_1 E - (\delta_1 + \eta + \mu)I, \\ \frac{\partial A}{\partial t} = \nabla \cdot (D_5(x) \nabla A) + \alpha_2 E - (\delta_2 + \mu)A, \\ \frac{\partial R}{\partial t} = \nabla \cdot (D_6(x) \nabla R) + \delta_1 I + \delta_2 A - \mu R, \\ \frac{\partial B}{\partial t} = \nabla \cdot (D_7(x) \nabla B) + g_1 I + g_2 A - (d_B + \psi(I))B, \\ \frac{\partial S}{\partial n} = \frac{\partial V}{\partial n} = \frac{\partial E}{\partial n} = \frac{\partial I}{\partial n} = \frac{\partial A}{\partial n} = \frac{\partial R}{\partial n} = \frac{\partial B}{\partial n} = 0, x \in \partial\Omega, t \geq 0. \end{array} \right. \quad x \in \Omega, t \geq 0, \quad (6.1)$$

Clearly, according to Lemma 3.2, system (6.1) admits a unique disease-free equilibrium $Q^0 = (S^0, V^0, 0, 0, 0, 0, 0)$, where $S^0 = \frac{\Lambda}{p+\mu}$ and $V^0 = \frac{p\Lambda}{\mu(p+\mu)}$. Define $S^0 + \sigma V^0 = \frac{\Lambda(\mu+\sigma p)}{\mu(p+\mu)}$. By direct calculation, the basic reproduction number of model (6.1) can be expressed as:

$$R_0 = \frac{\beta(S^0 + \sigma V^0)}{\alpha_1 + \alpha_2 + \mu} \left[\frac{\beta_I \alpha_1}{\delta_1 + \eta + \mu} + \frac{\beta_A \alpha_2}{\delta_2 + \mu} + \frac{\beta_B}{d_B} \left(\frac{g_1 \alpha_1}{\delta_1 + \eta + \mu} + \frac{g_2 \alpha_2}{\delta_2 + \mu} \right) \right].$$

Remark 6.1. The basic reproduction number R_0 can be interpreted as the sum of the contributions from symptomatic individuals, asymptomatic individuals, and environmental pathogens to disease transmission.

Theorem 6.2. If $R_0 > 1$, system (6.1) admits a unique endemic equilibrium $Q^* = (S^*, V^*, E^*, I^*, A^*, R^*, B^*)$.

Proof. The endemic equilibrium $Q^* = (S^*, V^*, E^*, I^*, A^*, R^*, B^*)$ of system (6.1) is determined by setting the right-hand sides of the equations to zero. This leads to the following algebraic system:

$$\left\{ \begin{array}{l} \Lambda - \beta(I^*)\Gamma(I^*)S^* - (p + \mu)S^* = 0, \\ pS^* - \sigma\beta(I^*)\Gamma(I^*)V^* - \mu V^* = 0, \\ \beta(I^*)\Gamma(I^*)(S^* + \sigma V^*) - (\alpha_1 + \alpha_2 + \mu)E^* = 0, \\ \alpha_1 E^* - (\delta_1 + \eta + \mu)I^* = 0, \\ \alpha_2 E^* - (\delta_2 + \mu)A^* = 0, \\ \delta_1 I^* + \delta_2 A^* - \mu R^* = 0, \\ g_1 I^* + g_2 A^* - (d_B + \psi(I^*))B^* = 0, \end{array} \right. \quad (6.2)$$

Specifically, let $\beta(I) = \beta - \beta_1 f(I)$ and $\Gamma(I) = \beta_I I + \beta_A A + \beta_B B$. From the last four equations of (6.2), we can express E^*, A^*, R^* and B^* as functions of I^* :

$$E(I) = \frac{\delta_1 + \eta + \mu}{\alpha_1} I, \quad A(I) = \frac{\alpha_2(\delta_1 + \eta + \mu)}{\alpha_1(\delta_2 + \mu)} I, \quad R(I) = \frac{\delta_1 I + \delta_2 A(I)}{\mu}, \\ B(I) = \frac{g_1 I + g_2 A(I)}{d_B + \psi(I)} = \frac{I \left(g_1 + \frac{g_2 \alpha_2 (\delta_1 + \eta + \mu)}{\alpha_1 (\delta_2 + \mu)} \right)}{d_B + \psi(I)}.$$

From the first two equations of (6.2), we obtain

$$S(I) = \frac{\Lambda}{\beta(I)\Gamma(I) + p + \mu}, \quad V(I) = \frac{pS(I)}{\sigma\beta(I)\Gamma(I) + \mu}.$$

To analyze the existence and uniqueness of the positive solution to system (6.2), we substitute $S(I)$, $V(I)$ and $E(I)$ into the third equation of (6.2). By dividing the resulting equation by I , we define the following auxiliary function $H(I)$ for $I \in (0, +\infty)$:

$$H(I) := \frac{\beta(I)\Gamma(I)(S(I) + \sigma V(I))}{I} - \frac{(\alpha_1 + \alpha_2 + \mu)(\delta_1 + \eta + \mu)}{\alpha_1},$$

where $\Gamma(I) = \beta_I I + \beta_A A(I) + \beta_B B(I)$. According to Assumptions $(\mathbf{H}_1)$ – $(\mathbf{H}_3)$, $\beta(I)$ is a decreasing function and $\psi(I)$ is an increasing function with respect to I . By differentiating the above $H(I)$ and conducting a monotonicity analysis of the composite functions, it follows that $H(I)$ is strictly monotonically decreasing for $I \in (0, +\infty)$.

Next, we examine the limiting behavior. As $I \rightarrow 0$, utilizing the definition of the basic reproduction number R_0 , we obtain

$$H(0) = \frac{(\alpha_1 + \alpha_2 + \mu)(\delta_1 + \eta + \mu)}{\alpha_1} (R_0 - 1), \quad (6.3)$$

Obviously, $H(0) > 0$ whenever $R_0 > 1$. On the other hand, as $I \rightarrow +\infty$, since $S(I)$ and $V(I)$ approach zero and $\beta(I)$ is bounded, it is straightforward to show that $\lim_{I \rightarrow +\infty} H(I) < 0$. According to the Intermediate Value Theorem, there exists a unique $I^* > 0$ such that $H(I^*) = 0$. Once I^* is uniquely determined, the remaining components S^* , V^* , E^* , A^* , R^* and B^* are also uniquely determined and are all positive. Consequently, system (6.1) admits a unique endemic equilibrium Q^* when $R_0 > 1$. $\square$

Next, we discuss the global stability of the disease-free equilibrium Q^0 and the endemic equilibrium Q^* . Based on the discussion of Theorem 5.1, we define the following forward invariant set:

$$\Gamma = \{\phi = (\phi_1, \phi_2, \phi_3, \phi_4, \phi_5, \phi_6, \phi_7) \in X_+ : \phi_1(x) \leq S^0, \phi_2(x) \leq V^0, x \in \overline{\Omega}\}.$$

Theorem 6.3. *If $R_0 \leq 1$, the disease-free equilibrium Q^0 of system (6.1) is globally asymptotically stable within the region Γ .*

Proof. Define the following Lyapunov functional

$$W(t) = \int_{\Omega} \left[S^0 G\left(\frac{S}{S^0}\right) + V^0 G\left(\frac{V}{V^0}\right) + E + K_I I + K_A A + K_B B \right] dx,$$

where $G(x) = x - 1 - \ln x$. Choose the positive constants K_I , K_A and K_B as follows

$$K_B = \frac{\beta\beta_B(S^0 + \sigma V^0)}{d_B}, \quad K_I = \frac{\beta\beta_I(S^0 + \sigma V^0) + K_B g_1}{\delta_1 + \eta + \mu},$$

$$K_A = \frac{\beta\beta_A(S^0 + \sigma V^0) + K_B g_2}{\delta_2 + \mu}.$$

According to ([31], Theorem 3.7) and the homogeneous Neumann boundary conditions, we have

$$\begin{aligned}
\frac{dW(t)}{dt} &= \int_{\Omega} \left[\left(1 - \frac{S^0}{S}\right) (\Lambda - [\beta - \beta_1 f(I)]\Gamma(I)S - (p + \mu)S) \right. \\
&\quad + \left(1 - \frac{V^0}{V}\right) (pS - \sigma[\beta - \beta_1 f(I)]\Gamma(I)V - \mu V) \\
&\quad + [\beta - \beta_1 f(I)]\Gamma(I)(S + \sigma V) - (\alpha_1 + \alpha_2 + \mu)E \\
&\quad + K_I(\alpha_1 E - (\delta_1 + \eta + \mu)I) + K_A(\alpha_2 E - (\delta_2 + \mu)A) \\
&\quad \left. + K_B(g_1 I + g_2 A - (d_B + \psi(I))B) \right] dx \\
&= \int_{\Omega} \left[\mu S^0 \left(2 - \frac{S^0}{S} - \frac{S}{S^0}\right) + \mu V^0 \left(3 - \frac{S^0}{S} - \frac{V}{V^0} - \frac{SV^0}{S^0 V}\right) \right] \\
&\quad + [\beta - \beta_1 f(I)]\Gamma(I)(S^0 + \sigma V^0) - (\alpha_1 + \alpha_2 + \mu)E \\
&\quad + E(K_I \alpha_1 + K_A \alpha_2) - I(K_I(\delta_1 + \eta + \mu) - K_B g_1) \\
&\quad - A(K_A(\delta_2 + \mu) - K_B g_2) - B K_B d_B - B K_B \psi(I) dx \\
&\leq \int_{\Omega} \left[\mu S^0 \left(2 - \frac{S^0}{S} - \frac{S}{S^0}\right) + \mu V^0 \left(3 - \frac{S^0}{S} - \frac{V}{V^0} - \frac{SV^0}{S^0 V}\right) \right. \\
&\quad \left. + (\alpha_1 + \alpha_2 + \mu)(R_0 - 1)E - K_B \psi(I)B \right] dx,
\end{aligned}$$

From the arithmetic-geometric mean inequality, we have $\left(2 - \frac{S^0}{S} - \frac{S}{S^0}\right) \leq 0$ and $\left(3 - \frac{S^0}{S} - \frac{V}{V^0} - \frac{SV^0}{S^0 V}\right) \leq 0$. Additionally, $\psi(I) \geq 0$ and $B \geq 0$. Thus, when $R_0 \leq 1$, $\frac{dW(t)}{dt} \leq 0$, with $\frac{dW(t)}{dt} = 0$ if and only if $S = S^0, V = V^0, E(t, x) = 0$ and $B(t, x) = 0$. From the equations of model (6.1), it follows that if $E = 0$ and $B = 0$, then necessarily $I(t, x) = 0$ and $A(t, x) = 0$. This implies that the largest invariant set contained in $\{(S, V, E, I, A, R, B) \in \Gamma : \frac{dW(t)}{dt} = 0\}$ consists solely of the disease-free equilibrium Q^0 . According to LaSalle's invariance principle, we conclude that Q^0 is globally asymptotically stable for model (6.1) when $R_0 \leq 1$. $\square$

7 Numerical simulations

To reflect spatial heterogeneity, we consider a one-dimensional spatial domain $\Omega = [0, L]$ with $L = \pi$. The diffusion coefficients for each variable are set to $D_i(x) = 0.01 (i = 1, \dots, 7)$. The spatially heterogeneous parameters in model (1.1) are specified as follows

Table 2: All parameter values pertinent to model (1.1)

Parameters	Value	Parameters	Value
$\mu(x)$	$0.05 + 0.01 \cos(x)$	g_1	0.2
$\Lambda(x)$	$0.8 + 0.5 \cos(x)$	α_1	0.1
p	0.2	α_2	0.05
σ	0.3	δ_1	0.1
$\beta(x)$	$0.4 + 0.2 \sin(x)$	δ_2	0.1
d_B	0.5	η	0.02
g_2	0.1		

7.1 Spatiotemporal evolution of disease persistence

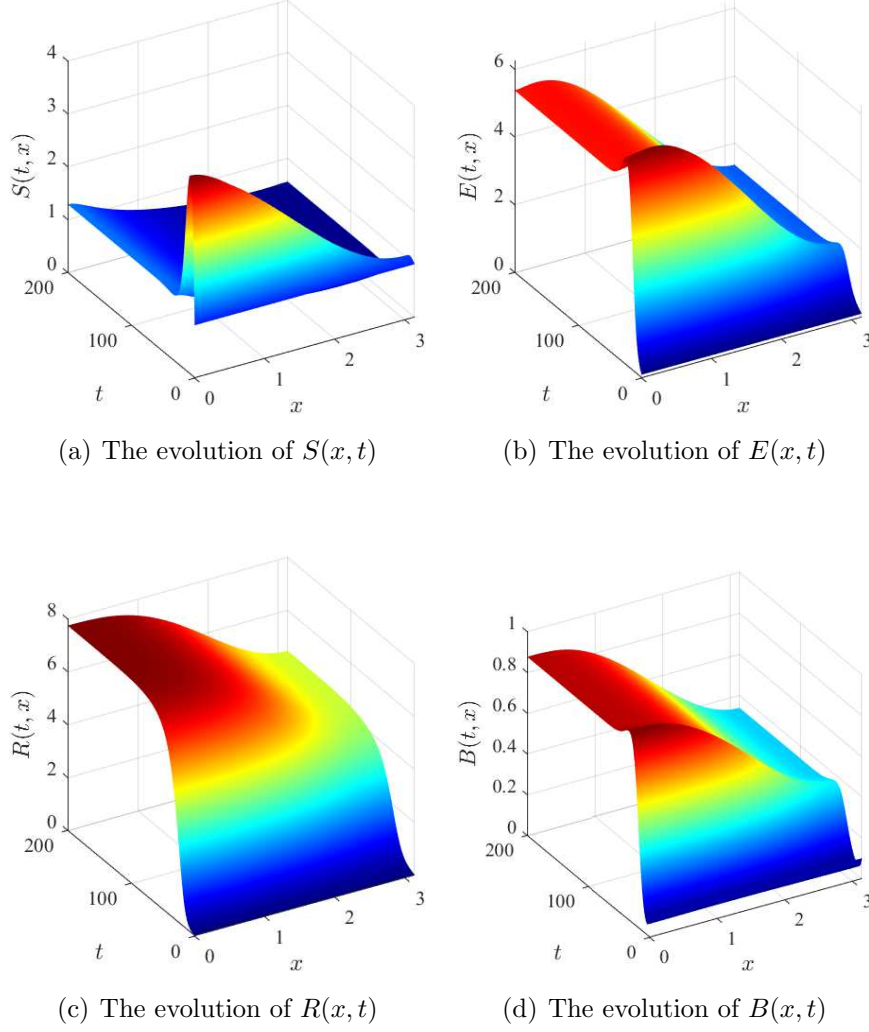


Fig. 1. Asymptotic behavior of each compartment for model (1.1) with $\mathcal{R}_0 > 1$.

In this subsection, the functions related to media intervention are taken as $f(I) = I/(1 + I)$ and $\psi(I) = 0.5I/(1 + I)$, and the relevant intensity parameter is $\beta_1 = 0.2$. Other parameter values are set as detailed in Table 2. Through calculation, we obtain the basic reproduction number $\mathcal{R}_0 > 1$. According to Theorem 6.3 and Lemma 5.5, the system should admit a positive steady-state solution, indicating that the disease will exhibit persistent survival under spatial heterogeneity. As shown in Fig.1, the three-dimensional spatiotemporal evolution of four main components in model (1.1) over time is presented.

To further reveal the synergistic effects of media coverage, vaccination, and environmental management on disease prevalence, we analyze the sensitivity of the basic reproduction number $\mathcal{R}_0$ with respect to parameter variations using the control variable method. The computational results are illustrated in Fig.2.

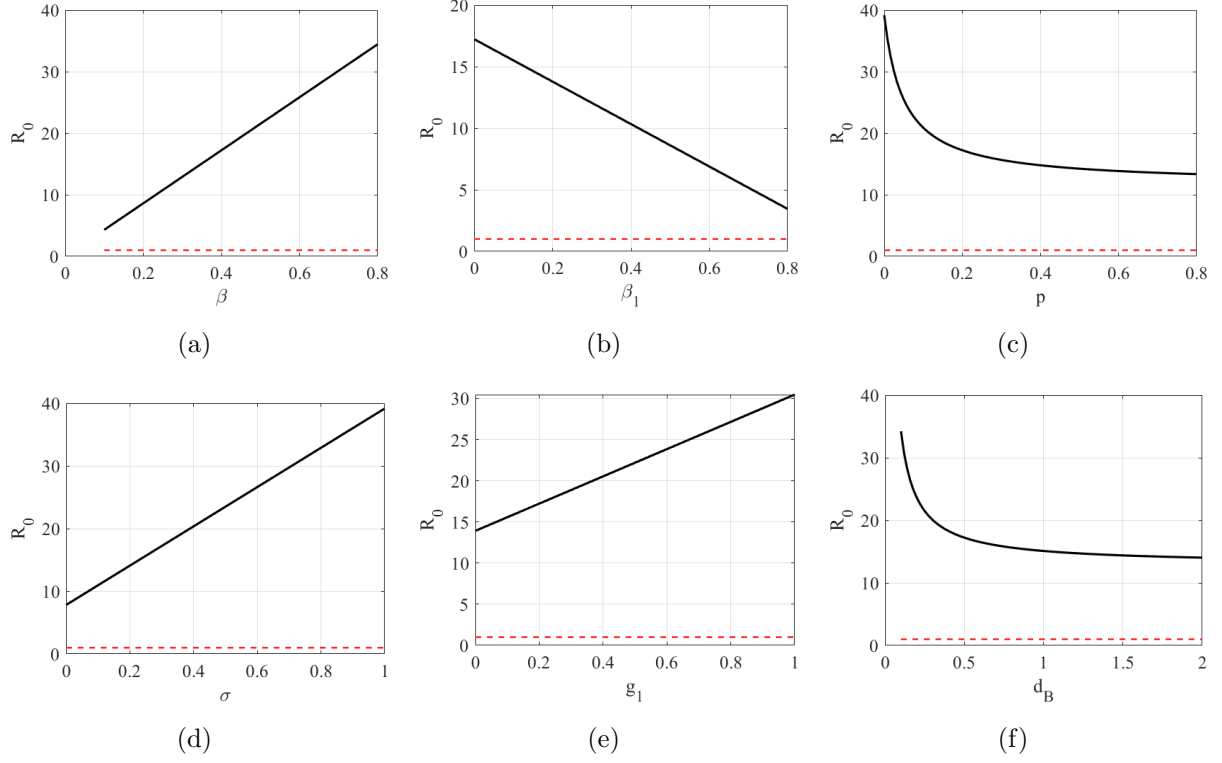
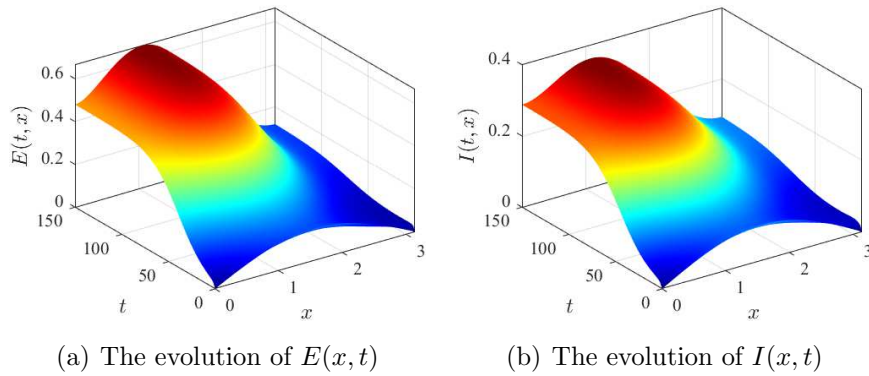


Fig. 2. Sensitivity of $\mathcal{R}_0$ with respect to parameter variations

7.2 Impact of media intervention intensity on disease elimination

In this subsection, we set the reduction intensity of the contact rate induced by media to $\beta_1 = 0.8$ and enhance the anthropogenic disinfection rate $\psi(I)$ in response to media reports by setting its intensity parameter to 0.9. Other parameter values are maintained as specified in Table 2. Through computation, the basic reproduction number is found to be $R_0 \approx 0.76 < 1$. As illustrated in Fig.3, the three-dimensional spatiotemporal distributions of the infected compartments are presented, demonstrating their evolution over time under strengthened media intervention and environmental governance.



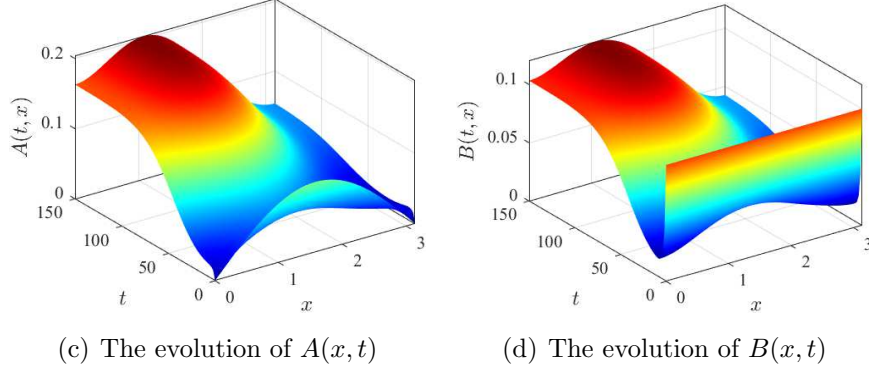


Fig. 3. Asymptotic behavior of each compartment for model (1.1) with $\mathcal{R}_0 < 1$.

To facilitate a more intuitive observation of the roles of various factors, the spatially averaged total population density is employed as the evaluation metric. Fig.4 illustrates the comparison of the system's temporal evolution curves under different parameter configurations.

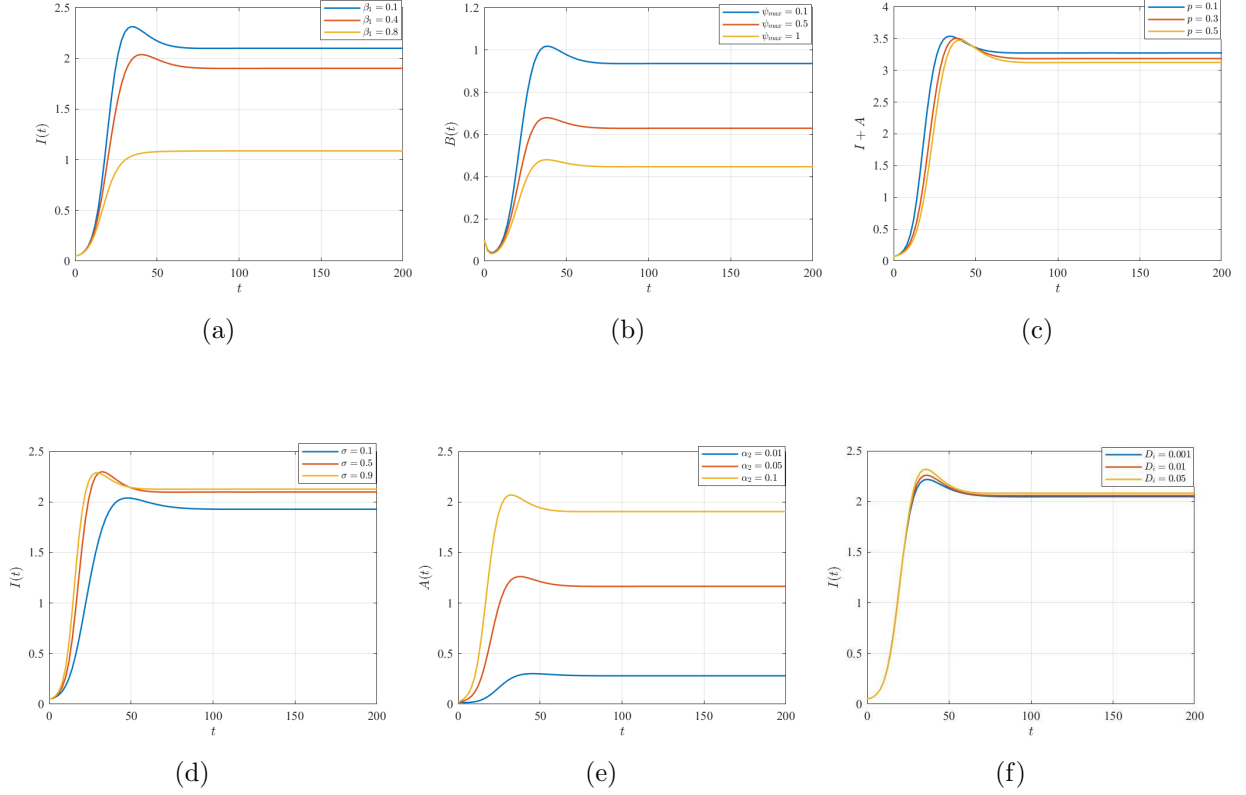


Fig. 4. Comparison of the effects of different parameters on the temporal evolution of infected compartment densities

7.3 The steady-state distribution of infection

To further elucidate how spatial heterogeneity shapes the transmission hotspots of the infectious disease, this subsection examines the long-term spatial distribution characteristics of the infected compartment $I(t, x)$ under the condition $R_0 > 1$. We assume that the baseline infection rate $\beta(x)$ exhibits a non-uniform pattern

with localized high incidence, and specify $\beta(x) = 0.4 + 0.35 \sin^2(x) + 0.1 \cos(2x)$ to simulate the empirical scenario of alternating urban centers and suburban areas. Additionally, relatively small diffusion coefficients D_i are chosen to capture the fine structures of the spatial patterns. As illustrated in Fig.5, the spatiotemporal evolution of the infected density $I(t, x)$ is presented, showing the process of eventually reaching a steady state.

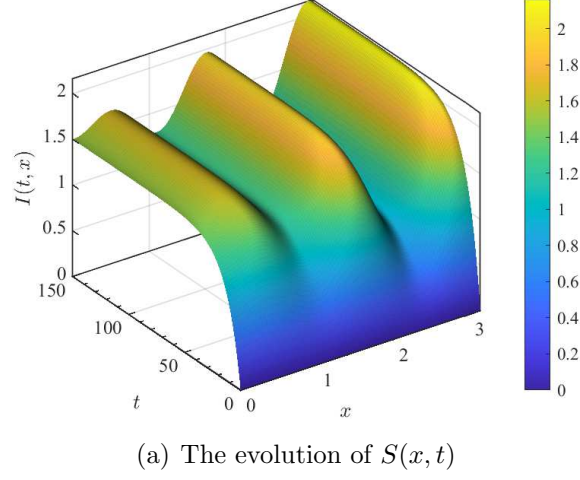


Fig. 5. Patchy distribution patterns of infection induced by spatial heterogeneity

8 Discussion

In this paper, we have proposed and investigated a class of SVEIAR-B reaction-diffusion epidemic models that incorporate both media intervention and spatial heterogeneity. By utilizing semigroup theory and the comparison principle for parabolic equations, the existence, uniqueness, and ultimate boundedness of non-negative classical solutions are established. The basic reproduction number R_0 is defined via the next-generation operator method, and the threshold dynamics of the system are analyzed: when $R_0 < 1$, the disease-free steady state is globally asymptotically stable within the system; when $R_0 > 1$, the system exhibits uniform persistence. Furthermore, under spatially homogeneous conditions, the global asymptotic stability of the endemic equilibrium is proved by constructing a suitable Lyapunov functional.

Numerical simulation results verify the aforementioned theoretical analysis and facilitate a discussion on the impact of relevant parameters on disease evolution. Our findings indicate that both the enhancement of media intervention and an increase in vaccination rates contribute to a decrease in the basic reproduction number, thereby reducing the scale of infection. Concurrently, the spatial non-uniformity of the baseline infection rate leads to a non-homogeneous patchy distribution of infected individuals. The analysis in this paper provides a theoretical foundation for infectious disease prevention and control. Future research could further explore the impacts of time delays or stochastic environmental perturbations on system dynamics.

Author Contributions

All authors contributed equally and significantly in preparing and writing this article. All authors read and approved the final manuscript.

Conflict of interest

The authors declare that there is no competing interests regarding the publication of this article.

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