Environmental Persistence and Threshold Dynamics in a Multi-Region Cholera Model

Original Research Article

Abstract

Cholera remains a major global health concern, particularly in regions with limited sanitation and high population mobility. Motivated by the 2010 Haiti earthquake and subsequent outbreak, this study examines the combined roles of environmental reservoirs and spatial coupling in cholera transmission using a coupled SEIR-B and hydrodynamic modeling framework. Human disease dynamics are integrated with environmental bacterial transport via advection and diffusion, together with cross-regional coupling mechanisms. Analysis of the basic reproduction number $\mathcal{R}_0$, stability properties, and numerical simulations identifies thresholds separating bacterial elimination from persistence and demonstrates how hydrological transport, exogenous contamination, and upstream sources shape downstream risk. These results underscore the need for coordinated sanitation and water-management interventions in hydrologically connected regions and illustrate the value of mathematical modeling for informing effective disease control strategies.

Keywords: cholera; waterborne transmission; environmental diffusion; exogenous contamination; PDE epidemiology; $\mathcal{R}_0$; global stability.

2010 Mathematics Subject Classification: 53C25; 83C05; 57N16

1 Introduction

Cholera is an acute, waterborne infectious disease caused by the bacterium *Vibrio cholerae*, transmitted primarily through ingestion of contaminated water or food. Clinical outcomes range from mild diarrhea to severe dehydration and shock, and without prompt rehydration therapy, death may occur within hours. Because *V. cholerae* proliferates in aquatic environments, particularly in regions with inadequate drinking water and sanitation, cholera transmission is closely linked to environmental conditions and population exposure to contaminated water sources (World Health Organization, 2024; Médecins Sans Frontières, 2024; Centers for Disease Control and Prevention, 2024a,b).

Unlike many directly transmitted diseases, cholera outbreaks are frequently sustained by persistent environmental reservoirs in rivers, wells, and community water systems rather than by person-to-person contact alone. Hydrological processes such as rainfall, flooding, and river flow transport bacteria across geographic regions, allowing environmental pathways to dominate epidemic dynamics. These mechanisms have motivated mathematical models incorporating diffusion, advection, and spatial heterogeneity through partial differential equations (Cai et al., 2016; Gobbert and Agheksanterian, 2007; Jung et al., 2016; Shu et al., 2021a; Yamazaki and Wang, 2017; Shu et al., 2021b; Rosa and Torres, 2021).

Because environmental contamination can persist long after clinical cases decline, sustaining transmission and enabling re-emergence, understanding how water movement, infrastructure failure, and sanitation influence bacterial persistence is essential for accurate modeling and effective intervention design.

Haiti provides a particularly instructive case study for environmentally driven cholera transmission. The 2010 epidemic began when *Vibrio cholerae* was introduced into the Artibonite River near Mirebalais, after which contaminated water spread rapidly downstream to communities such as Gonaïves that rely on the river for drinking and agriculture. Seasonal rainfall, flooding, and watershed flow amplified bacterial transport, underscoring the central role of hydrological forcing in cholera persistence and re-emergence.

Structural vulnerabilities further magnified these dynamics. Limited sanitation infrastructure, high population density in urban centers such as Port-au-Prince, and constrained medical capacity influenced both transmission intensity and the timing of interventions. These conditions make Haiti well suited for mechanistic models that couple human epidemiological dynamics with environmental bacterial transport via diffusion and advection (Cai et al., 2016; Gobbert and Agheksanterian, 2007; Jung et al., 2016; Shu et al., 2021a; Yamazaki and Wang, 2017; Shu et al., 2021b; Rosa and Torres, 2021).

Despite extensive public health responses, including surveillance, treatment, and vaccination campaigns implemented by the MSPP, WHO, PAHO, UNICEF, CDC, and Médecins Sans Frontières (World Health Organization, 2024; Médecins Sans Frontières, 2024; Centers for Disease Control and Prevention, 2024a,b), environmental contamination persisted long after reported cases declined, highlighting the need for models that explicitly capture bacterial survival in aquatic reservoirs and hydrological drivers of sustained transmission.

A defining feature of the Haitian outbreak was the role of exogenous environmental contamination. Represented here by the parameter Λ_i, exogenous contamination captures bacterial inputs originating outside the local human-environment system. In 2010, improper disposal of human waste by UN peacekeepers near the Mirebalais base resulted in the direct release of contaminated sewage into the Artibonite River, abruptly initiating widespread downstream transmission. This event illustrates that Λ_i represents a documented mechanism capable of fundamentally altering epidemic trajectories.

Mathematically, Λ_i enters the environmental compartment as an external source term independent of local human shedding. When combined with hydrological transport, diffusion, and flood-season dynamics, such inputs can generate nonlinear amplification effects and sustain bacterial persistence (Cai et al., 2016; Gobbert and Agheksanterian, 2007; Jung et al., 2016; Shu et al., 2021a; Yamazaki and Wang, 2017; Shu et al., 2021b; Rosa and Torres, 2021).

Although many existing cholera models incorporate spatial structure or environmental transmission, important gaps remain. Few frameworks explicitly represent seasonally varying diffusion despite strong empirical evidence linking rainfall and flooding to bacterial transport, and even fewer include exogenous environmental forcing through terms such as Λ_i, despite documented cases where external contamination dominates outbreak dynamics. Moreover, while prior studies provide threshold and stability results (Cai et al., 2016; Gobbert and Agheksanterian, 2007; Jung et al., 2016; Shu et al., 2021a; Yamazaki and Wang, 2017; Shu et al., 2021b; Rosa and Torres, 2021), few derive analytical persistence conditions that simultaneously account for spatial heterogeneity, hydrological variability, and external inputs.

To address these gaps, we develop a coupled ODE-PDE model in which human disease dynamics are governed by ODEs and environmental *V. cholerae* concentrations evolve according to a PDE with seasonally varying diffusion and explicit exogenous forcing. We derive conditions for global asymptotic stability of the disease-free equilibrium and identify an environmental persistence threshold capturing the combined effects of hydrological transport and external contamination. Numerical simulations illustrate how water-driven mechanisms and exogenous inputs can initiate, sustain, and propagate outbreaks across connected regions.

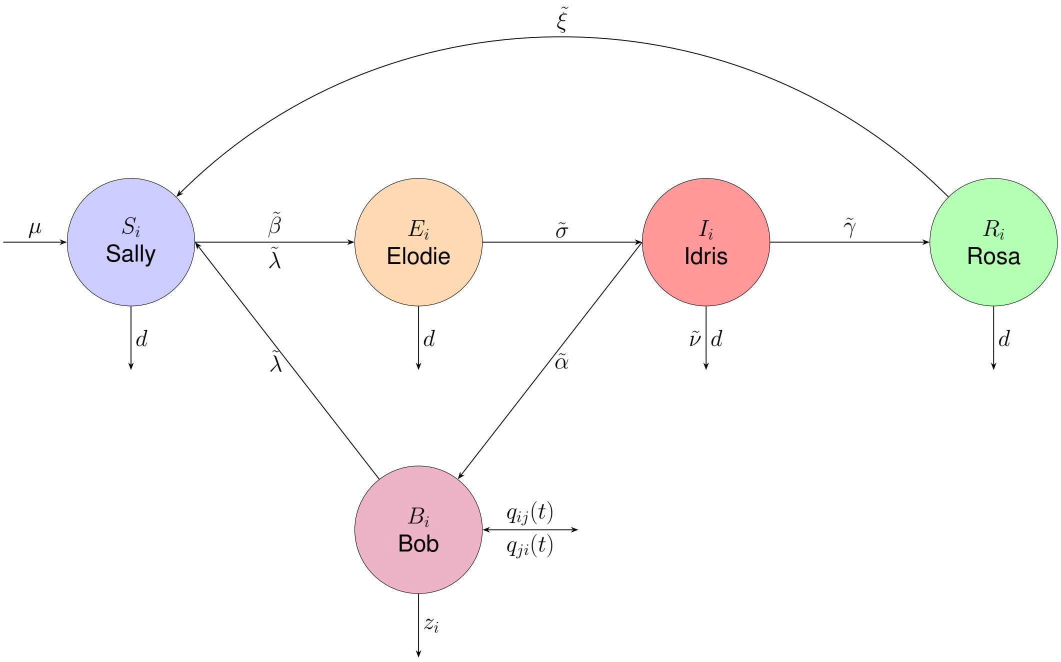
The remainder of the paper is organized as follows. Section 2 presents the multi-region SEIR-B model. Section 3.1 establishes well-posedness of the system. Section 3 analyzes the basic reproduction number and stability of the disease-free equilibrium. Section 4 presents numerical simulations, and Section 5 discusses epidemiological and policy implications.

2 Model Formulation

2.1 Visualization and Narrative Overview of the Model

We first present a schematic representation of the population compartments and their interactions, followed by the governing equations describing disease progression, bacterial transport, and inter-regional coupling. This framework builds on classical compartmental epidemic theory and cholera-specific extensions incorporating environmental reservoirs (Hethcote, 2000; Anderson and May, 1991; Kermack and McKendrick, 1927; Codeço, 2001). Figure 1 depicts transitions among susceptible (S_i), exposed (E_i), infectious (I_i), and recovered (R_i) compartments across multiple regions, driven by direct transmission, environmental exposure, disease progression, and demographic processes.

Figure 1: Flowchart of the 3–Region SEIR–B Cholera Model.



Within each region i, human disease dynamics are coupled to an environmental reservoir representing waterborne *Vibrio cholerae*. Susceptible individuals enter through recruitment at rate μ or loss of immunity at rate $\tilde{\xi} R_i$. Infection occurs via direct contact with infectious individuals at rate $\tilde{\beta} \frac{I_i}{N_i} S_i$ (Kermack and McKendrick, 1927) or environmental exposure to contaminated water at rate $\tilde{\eta} \frac{B_i}{K+B_i} S_i$ (Codeço, 2001).

Exposed individuals represent a latent stage and progress to infectiousness at rate $\tilde{\sigma} E_i$, while all human compartments experience natural mortality at rate d (Hethcote, 2000). Infectious individuals recover at rate $\tilde{\gamma} I_i$, experience disease-induced mortality at rate $\tilde{\nu} I_i$, and shed bacteria into the environment at rate $\tilde{\alpha} I_i$ (Codeço, 2001). Immunity following recovery is temporary and wanes at rate $\tilde{\xi} R_i$ (Anderson and May, 1991).

Environmental bacteria evolve according to an advection-diffusion-reaction equation. Transport is governed by hydrological advection with velocity $\mathbf{v}_i$ and diffusion with coefficient D_i, capturing directed flow and local mixing in riverine systems (Tien and Earn, 2010; Bertuzzo et al., 2010). Bacterial inputs arise from human shedding, while removal occurs at rate $z_i B_i$, reflecting natural decay and sanitation-related processes (Hartley et al., 2006; King et al., 2008). Inter-regional coupling is modeled through time-dependent transport terms $q_{ij}(t)$, representing hydrological export and upstream inflow (Bertuzzo et al., 2010). Exogenous contamination is incorporated via $\Lambda_i(t)$, capturing external inputs such as flooding, runoff, or infrastructure failure (Mari et al., 2012; Chin et al., 2011).

Although bacterial transport is inherently three-dimensional, we adopt a depth-averaged two-dimensional formulation appropriate for rivers, floodplains, and shallow surface waters in cholera-endemic regions. Vertical mixing driven by turbulence, suspended particulates, and flood-season hydrodynamics justifies this approximation while reducing computational complexity (Huq and et al., 1983; Colwell and et al., 2003; King et al., 2008; Hartley et al., 2006; Bertuzzo et al., 2010).

By coupling human disease dynamics with spatially explicit environmental transport, the SEIR-B framework captures how hydrological connectivity and environmental persistence can sustain outbreaks independently of local population density. This structure enables systematic evaluation of sanitation, vaccination, and water-based intervention strategies in interconnected regions (Zhu et al., 2023; Iyaniwura et al., 2023; Wang et al., 2022). Model parameters are summarized in Table 1.

2.2 Mathematical Model Formulation

The SEIR system is defined as follows:

$$
\begin{aligned}
\frac{dS_i}{dt} &= \mu + \tilde{\xi} R_i - \tilde{\beta}\frac{I_i}{N_i} S_i - \tilde{\eta}\frac{B_i}{K + B_i} S_i - dS_i, \\
\frac{dE_i}{dt} &= \tilde{\beta}\frac{I_i}{N_i} S_i + \tilde{\eta}\frac{B_i}{K + B_i} S_i - \tilde{\sigma} E_i - dE_i + \Pi_i(t), \\
\frac{dI_i}{dt} &= \tilde{\sigma} E_i - \tilde{\gamma} I_i - \tilde{\nu} I_i - dI_i, \\
\frac{dR_i}{dt} &= \tilde{\gamma} I_i - \tilde{\xi} R_i - dR_i.
\end{aligned}
\tag{2.1}
$$

Environmental bacterial concentration in region i evolves according to:

$$
\frac{\partial B_i}{\partial t} + \mathbf{v}_i \cdot \nabla B_i = D_i \nabla^2 B_i + \tilde{\alpha}\, I_i + \sum_{j \neq i} q_{ji}(t)\, B_j - \sum_{j \neq i} q_{ij}(t)\, B_i - z_i B_i + \Lambda_i(t), \qquad i \in \{1, 2, 3\}. \tag{2.2}
$$

Table 1: **Model Variables and Parameters with Embedded Vaccination Effects**

Symbol	Description
i	Region index, $i \in \{1, 2, 3\}$.
$S_i(t)$	Susceptible population in region i
$E_i(t)$	Exposed population in region i
$I_i(t)$	Infectious population in region i
$R_i(t)$	Recovered population in region i
$N_i(t)$	Total population in region i; $N_i(t) = S_i(t) + E_i(t) + I_i(t) + R_i(t)$
$B_i(x, t)$	Environmental bacterial concentration in region i at location x and time t
K	Half-saturation constant for environmental transmission
μ	Birth/Recruitment rate.
d	Natural death rate

$\tilde{\beta} = \beta(1 - \epsilon_S)$	Human-to-human transmission rate accounting for reduced susceptibility.
$\tilde{\eta} = \eta(1 - \epsilon_B)$	Environment-to-human transmission rate modified by interventions limiting exposure.
$\tilde{\sigma} = \sigma(1 - \epsilon_E)$	Progression rate from exposed to infectious modified by intervention effects.
$\tilde{\gamma} = \gamma(1 + \epsilon_I)$	Recovery rate modified by intervention effects.
$\tilde{\nu} = \nu(1 - \epsilon_I)$	Disease-induced mortality rate modified by interventions.
$\tilde{\xi} = \xi(1 - \epsilon_R)$	Immunity waning rate modified by interventions.
$\tilde{\alpha} = \alpha(1 - \epsilon)$	Bacterial shedding rate modified by interventions.
$\mathbf{v}_i$	Advection (velocity) vector representing directional water flow in region i
D_i	Diffusion coefficient for bacterial spread within region i.
$q_{ij}(t) := a_{ji}\, r_{ij}(t)$	Time-dependent bacterial transport rate from region i to region j.
a_{ij}	Binary hydrological connectivity indicator between regions i and j.
$r_{ij}(t)$	Time-dependent runoff-driven transport intensity from region i to region j
z_i	Net bacterial removal rate in region i
$\Lambda_i(t)$	Local external bacterial input in region i

2.3 Model Structure and Assumptions

The SEIR-B cholera transmission model (2.1) is formulated under the following biological, environmental, and structural assumptions:

1. **Homogeneous mixing within regions.** Within each region i, individuals mix uniformly. Susceptible individuals experience equal contact with infectious individuals and equal exposure to environmental bacteria. No within-region heterogeneity (e.g., age or behavior) is represented.
2. **Deterministic compartmental dynamics.** Human populations evolve according to deterministic ODEs, and environmental bacteria evolve according to deterministic advection-diffusion PDEs, representing mean-field dynamics. Stochastic effects are neglected.
3. **Interventions act through rate modification.** Public health interventions are modeled via scaling parameters $\epsilon_S, \epsilon_B, \epsilon_E, \epsilon_I$, and ϵ_R, modifying susceptibility, environmental exposure, disease progression, severity, and immunity duration.
4. **Saturating environmental transmission.** Environmental transmission follows a Michaelis-Menten-type response $B_i/(K + B_i)$, reflecting saturation of infection risk at high bacterial concentrations.
5. **Population turnover without explicit human mobility.** Regional populations change only through recruitment μ and natural or disease-induced mortality d. Human migration between regions is not explicitly modeled in the SEIR compartments.
6. **Latent infection and temporary immunity.** Exposed individuals are infected but not infectious and progress to the infectious class at rate $\tilde{\sigma}$. Immunity following recovery is temporary and wanes at rate $\tilde{\xi}$.

7. **Uniform natural mortality and no reinfection during infection.** All human compartments experience the same natural death rate d, and reinfection does not occur during the exposed or infectious stages.

8. **Environmental bacteria as the sole indirect transmission pathway.** Waterborne *Vibrio cholerae* constitutes the only environmental reservoir. No alternative indirect transmission routes are considered.

9. **Advection-diffusion-reaction environmental dynamics.** Environmental bacteria evolve via advection, diffusion, shedding from infectious individuals, hydrological transport $q_{ij}(t)$, linear removal at rate $z_i B_i$, and exogenous input $\Lambda_i(t)$. No latent or persistent bacterial states are modeled.

10. **No healthcare capacity or behavioral feedback.** Rates of recovery, mortality, and treatment effectiveness are assumed constant and independent of disease prevalence (Hethcote, 2000).

11. **Region-specific importation, hydrology, and forcing.** Human-mediated importation occurs only into Port-au-Prince: $\Pi_1 = \Pi_2 = 0$, $\Pi_3 = m_{13} I_1 + m_{23} I_2$. Hydrological coupling connects Mirebalais (Region 1) to Gonaïves (Region 2), while Port-au-Prince (Region 3) is hydrologically isolated. External environmental forcing occurs only in Mirebalais, with $\Lambda_1 \neq 0$ and $\Lambda_2 = \Lambda_3 = 0$.

Updated model incorporating assumptions:

$$
\begin{aligned}
\frac{dS_1}{dt} &= \mu + \tilde{\xi}R_1 - \tilde{\beta}\frac{I_1}{N_1}S_1 - \tilde{\eta}\frac{B_1}{K+B_1}S_1 - dS_1, \\
\frac{dE_1}{dt} &= \tilde{\beta}\frac{I_1}{N_1}S_1 + \tilde{\eta}\frac{B_1}{K+B_1}S_1 - \tilde{\sigma}E_1 - dE_1, \\
\frac{dI_1}{dt} &= \tilde{\sigma}E_1 - \tilde{\gamma}I_1 - \tilde{\nu}I_1 - dI_1, \\
\frac{dR_1}{dt} &= \tilde{\gamma}I_1 - \tilde{\xi}R_1 - dR_1, \\
\frac{\partial B_1}{\partial t} + \mathbf{v}_1 \cdot \nabla B_1 &= D_1\nabla^2 B_1 + \tilde{\alpha}I_1 - q_{12}(t)B_1 - z_1B_1 + \Lambda_1(t), \\
\frac{dS_2}{dt} &= \mu + \tilde{\xi}R_2 - \tilde{\beta}\frac{I_2}{N_2}S_2 - \tilde{\eta}\frac{B_2}{K+B_2}S_2 - dS_2, \\
\frac{dE_2}{dt} &= \tilde{\beta}\frac{I_2}{N_2}S_2 + \tilde{\eta}\frac{B_2}{K+B_2}S_2 - \tilde{\sigma}E_2 - dE_2, \\
\frac{dI_2}{dt} &= \tilde{\sigma}E_2 - \tilde{\gamma}I_2 - \tilde{\nu}I_2 - dI_2, \\
\frac{dR_2}{dt} &= \gamma I_2 - \tilde{\xi}R_2 - dR_2, \\
\frac{\partial B_2}{\partial t} + \mathbf{v}_2 \cdot \nabla B_2 &= D_2\nabla^2 B_2 + \tilde{\alpha}I_2 + q_{12}(t)B_1 - z_2B_2, \\
\frac{dS_3}{dt} &= \mu + \tilde{\xi}R_3 - \tilde{\beta}\frac{I_3}{N_3}S_3 - \tilde{\eta}\frac{B_3}{K+B_3}S_3 - dS_3, \\
\frac{dE_3}{dt} &= \tilde{\beta}\frac{I_3}{N_3}S_3 + \tilde{\eta}\frac{B_3}{K+B_3}S_3 - \tilde{\sigma}E_3 - dE_3 + m_{13}I_1(t) + m_{23}I_2(t). \\
\frac{dI_3}{dt} &= \tilde{\sigma}E_3 - \tilde{\gamma}I_3 - \tilde{\nu}I_3 - dI_3, \\
\frac{dR_3}{dt} &= \tilde{\gamma}I_3 - \tilde{\xi}R_3 - dR_3, \\
\frac{\partial B_3}{\partial t} + \mathbf{v}_3 \cdot \nabla B_3 &= D_3\nabla^2 B_3 + \tilde{\alpha}I_3 - z_3B_3.
\end{aligned}
\tag{2.3}
$$

3 Epidemiological Analysis

3.1 Existence, Uniqueness, & Positivity of Solutions

Theorem 3.1 (Well-posedness of the Bacterial Transport Subsystem)**.** *Let* $\Omega \subset \mathbb{R}^n$ *be a bounded domain with smooth boundary and* $T > 0$*. The bacterial transport system* (2.2)*, subject to nonnegative initial data* $B_i(0, x) \geq 0$ *and homogeneous Neumann boundary conditions, admits* *a unique weak solution on* $[0, T]$ *satisfying*

$$B_i \in C([0,T]; L^2(\Omega)) \cap L^2(0,T; H^1(\Omega)).$$

Moreover, solutions remain nonnegative for all $t \in [0, T]$.

Proof. The full proof is given in Appendix A.

Theorem 3.2 (Well-posedness of the Human SEIR Subsystem)**.** *For each Region* $i \in \{1, 2, 3\}$, *the human SEIR system* (2.1) *admits a unique global solution for all* $t \geq 0$ *whenever the initial conditions satisfy*

$$S_i(0),\ E_i(0),\ I_i(0),\ R_i(0) \geq 0 \quad \textit{with} \quad N_i(0) > 0.$$

Moreover, solutions remain nonnegative for all time, and the total population $N_i(t)$ *remains bounded on finite time intervals. Consequently, the human SEIR subsystem is well posed.*

Proof. The full proof is provided in Appendix B.

3.2 Global Asymptotic Stability of the DFE, $\Lambda_i = \Pi_i = 0$

Theorem 3.3 (Local Stability of the Disease-Free Equilibrium)**.** *Consider the cholera transmission model defined by System* (2.3)*, which admits a disease-free equilibrium (DFE)* $\mathcal{E}_0$. *Let* $\mathcal{R}_0$ *denote the basic reproduction number, defined as the spectral radius of the associated next-generation matrix. Then* $\mathcal{E}_0$ *is locally asymptotically stable if* $\mathcal{R}_0 < 1$ *and unstable if* $\mathcal{R}_0 > 1$.

Proof. The Disease-Free Equilibrium (DFE) of System 2.3 is

$$\begin{aligned}\mathcal{E}_0 &= \Big(N_1^*, E_1^*, I_1^*, R_1^*, B_1^*, N_2^*, E_2^*, I_2^*, R_2^*, B_2^*, N_3^*, E_3^*, I_3^*, R_3^*, B_3^*\Big)\\ &= \Big(\frac{\mu}{d}, 0, 0, 0, 0, \frac{\mu}{d}, 0, 0, 0, 0, \frac{\mu}{d}, 0, 0, 0, 0\Big)\end{aligned}$$

To assess the local stability of the DFE, we apply the next-generation matrix method of van den Driessche and Watmough (van den Driessche and Watmough, 2002). We define the infected state vector as $\mathbf{x} = \begin{bmatrix} E_i \\ I_i \\ B_i \end{bmatrix}$. Recall, $N_i = S_i + E_i + I_i + R_i$. We define the Jacobian Matrix, $\mathcal{J}$, with respect to $\mathbf{x}$ and evaluate it at the DFE:

$$J(\mathcal{E}_0) = \begin{bmatrix} -\sigma - d & \beta & \frac{\eta\mu}{Kd} & 0 & 0 & 0 & 0 & 0 & 0 \\ \sigma & -d-\gamma-\nu & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & \alpha & -q_{12} - z_1 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & -\sigma - d & \beta & \frac{\eta\mu}{Kd} & 0 & 0 & 0 \\ 0 & 0 & 0 & \sigma & -d-\gamma-\nu & 0 & 0 & 0 & 0 \\ 0 & 0 & q_{12} & 0 & \alpha & -z_2 & 0 & 0 & 0 \\ 0 & m_{13} & 0 & 0 & m_{23} & 0 & -\sigma - d & \beta & \frac{\eta\mu}{Kd} \\ 0 & 0 & 0 & 0 & 0 & 0 & \sigma & -d-\gamma-\nu & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & \alpha & -z_3 \end{bmatrix}$$

Let $\mathcal{F}(\mathbf{x})$ denote the vector of new infection terms, and $\mathcal{V}(\mathbf{x})$ represent the vector of transitions between infected compartments. At the DFE, we compute the Jacobian matrix, $\mathcal{J}$ with respect to $\mathbf{x}$ and .

$$\mathcal{J}(\mathcal{E}_0) = \mathcal{F} - \mathcal{V}$$

where:

$$\mathcal{F} = \begin{bmatrix} 0 & \beta & \frac{\eta\mu}{Kd} & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & \beta & \frac{\eta\mu}{Kd} & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & \beta & \frac{\eta\mu}{Kd} \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \end{bmatrix}$$

$$
\mathcal{V} = \begin{bmatrix}
\sigma + d & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
-\sigma & d + \gamma + \nu & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & -\alpha & q_{12} + z_1 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & \sigma + d & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & -\sigma & d + \gamma + \nu & 0 & 0 & 0 & 0 \\
0 & 0 & -q_{12} & 0 & -\alpha & z_2 & 0 & 0 & 0 \\
0 & -m_{13} & 0 & 0 & -m_{23} & 0 & \sigma + d & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & -\sigma & d + \gamma + \nu & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & -\alpha & z_3
\end{bmatrix}
$$

The Basic Reproduction Number, $\mathcal{R}_0$, is defined as the spectral radius, maximum eigenvalue, of the matrix FV^{-1}:

$$
\begin{aligned}
\mathcal{R}_0 &= \max \left\{ \frac{\tilde{\sigma}\left(\tilde{\beta} K d z_3 + \tilde{\eta}\mu\alpha\right)}{Kd\,(d+\gamma+\tilde{\nu})\,(\tilde{\sigma}+d)\,z_3}, \frac{\tilde{\sigma}\left(K\tilde{\beta} d z_2 + \tilde{\eta}\mu\tilde{\alpha}\right)}{Kd\,(d+\tilde{\gamma}+\tilde{\nu})\,(\tilde{\sigma}+d)\,z_2}, \frac{\tilde{\sigma}\left(K\beta d q_{12} + K\tilde{\beta} d z_1 + \tilde{\eta}\mu\tilde{\alpha}\right)}{Kd\,(d+\tilde{\gamma}+\tilde{\nu})\,(\tilde{\sigma}+d)\,(q_{12}+z_1)} \right\} \\
&= \max \Bigg\{ \frac{\tilde{\beta}\tilde{\sigma}}{(\tilde{\sigma}+d)\,(d+\tilde{\gamma}+\tilde{\nu})} + \frac{\tilde{\eta}\mu\tilde{\alpha}\tilde{\sigma}}{Kd\,(d+\tilde{\gamma}+\tilde{\nu})\,(\tilde{\sigma}+d)\,z_3}, \\
&\qquad \frac{\tilde{\beta}\tilde{\sigma}}{(\tilde{\sigma}+d)\,(d+\tilde{\gamma}+\tilde{\nu})} + \frac{\tilde{\eta}\mu\tilde{\alpha}\tilde{\sigma}}{Kd\,(d+\gamma+\nu)\,(\tilde{\sigma}+d)\,z_2}, \\
&\qquad \frac{\tilde{\beta}\tilde{\sigma}}{(\tilde{\sigma}+d)\,(d+\tilde{\gamma}+\tilde{\nu})} + \frac{\tilde{\eta}\mu\alpha\sigma}{Kd\,(d+\gamma+\nu)\,(\sigma+d)\,(q_{12}+z_1)} \Bigg\} \\
&= \frac{\tilde{\beta}\tilde{\sigma}}{(\tilde{\sigma}+d)\,(d+\tilde{\gamma}+\tilde{\nu})} + max \left\{ \frac{\eta\mu\tilde{\alpha}\sigma}{Kd\,(d+\tilde{\gamma}+\tilde{\nu})\,(\tilde{\sigma}+d)\,z_3}, \frac{\eta\mu\alpha\sigma}{Kd\,(d+\tilde{\gamma}+\tilde{\nu})\,(\tilde{\sigma}+d)\,z_2}, \frac{\tilde{\eta}\mu\tilde{\alpha}\tilde{\sigma}}{Kd\,(d+\tilde{\gamma}+\tilde{\nu})\,(\tilde{\sigma}+d)\,(q_{12}+z_1)} \right\} \\
&= \frac{\beta\sigma}{(\sigma+d)(d+\gamma+\nu)} + \frac{\eta\mu\tilde{\alpha}\tilde{\sigma}}{Kd(d+\tilde{\gamma}+\tilde{\nu})(\tilde{\sigma}+d)} \max\left\{ \frac{1}{z_3}, \frac{1}{z_2}, \frac{1}{q_{12}+z_1} \right\} \\
&=: R_H + R_B \cdot \Psi_B.
\end{aligned}
$$

where,

$$
R_H := \frac{\tilde{\beta}\tilde{\sigma}}{(\tilde{\sigma}+d)(d+\tilde{\gamma}+\tilde{\nu})}, \quad R_B := \frac{\tilde{\eta}\mu\tilde{\alpha}\tilde{\sigma}}{Kd(d+\tilde{\gamma}+\tilde{\nu})(\tilde{\sigma}+d)}, \quad \Psi_B := \max\left\{ \frac{1}{z_3}, \frac{1}{z_2}, \frac{1}{q_{12}+z_1} \right\}
$$

A result of the Next Generation Method assures us that if $\mathcal{R}_0 < 1$, the DFE is Locally Asymptotically Stable. Conversely, if $\mathcal{R}_0 > 1$, the DFE is unstable. □

The basic reproduction number decomposes as $\mathcal{R}_0 = R_H + R_B\,\Psi_B$, separating direct human transmission from environmentally mediated transmission. The term R_H captures person-to-person spread, while $R_B\,\Psi_B$ represents environmental amplification governed by the dominant bacterial persistence pathway, reflecting a bottleneck determined by the slowest effective removal or flushing process.

This structure shows that cholera persistence arises from the interaction of human transmission and environmental amplification shaped by hydrological transport and spatial heterogeneity. Effective control therefore requires simultaneous reduction of direct transmission and environmental persistence, particularly in regions with slow bacterial clearance.
Notably, $\mathcal{R}_0$ is independent of the external importation term $\Pi_i(t)$, reflecting a limitation of threshold quantities derived from linearization about the disease-free equilibrium. While $\mathcal{R}_0 < 1$ guarantees

elimination in the absence of external inputs, sustained importation ($\Pi_i(t) \neq 0$) can maintain transmission under subcritical local dynamics. In the Haitian context, $\Pi_i(t)$ may represent infection pressure from population movement or external intervention, indicating reducing $\mathcal{R}_0$ below unity is necessary but not sufficient for eradication.

3.3 Global Asymptotic Stability of the DFE $(\Lambda_i = \Pi_i = 0)$

Theorem 3.4 (Global Asymptotic Stability of the DFE)**.** *Consider the three-region SEIR-B cholera model* (2.3)*. Assume there is no exogenous environmental contamination or external infection importation, i.e.,*

$$\Lambda_i(t) \equiv 0 \quad \textit{and} \quad \Pi_i(t) \equiv 0, \qquad i = 1, 2, 3.$$

Define the Lyapunov function

$$\mathcal{L}(t) = a \sum_{i=1}^{3} E_i^2 + b \sum_{i=1}^{3} I_i^2 + \sum_{i=1}^{3} c_i B_i^2,$$

with constants $a, b, c_i > 0$ chosen appropriately. If the basic reproduction number satisfies $\mathcal{R}_0 < 1$, then

$$\dot{\mathcal{L}}(t) \leq 0,$$

with equality only at the DFE $\mathcal{E}_0$. Consequently, $\mathcal{E}_0$ is globally asymptotically stable. The proof is given in Appendix C.

3.4 Final Epidemic Size Relation

The *final size relation* links the susceptible population at the beginning and end of an epidemic, providing an implicit measure of cumulative outbreak burden without resolving full transient dynamics. If u_i denotes the proportion of individuals in Region i remaining susceptible at epidemic termination, then $1 - u_i$ represents the total fraction infected. In models with both direct and environmentally mediated transmission, the final size relation decomposes into human and environmental components, yielding a transcendental equation for u_i that depends on key epidemiological parameters, including $\mathcal{R}_0$.

Theorem 3.5 (Final Epidemic Size Relation)**.** *Let*

$$u_i := \frac{S_i(T)}{S_i(0)} \in (0, 1)$$

denote the proportion of individuals in Region i remaining susceptible at the end of a single epidemic wave over $[0, T]$. Under the epidemic time-scale approximation, the final size relation satisfies

$$\ln\left(\frac{1}{u_i}\right) = \mathcal{R}_0 \,(1 - u_i) + \mathcal{E}_i,$$

where $\mathcal{R}_0$ is the basic reproduction number associated with endogenous transmission and

$$\mathcal{E}_i := \tilde{\lambda} \int_0^T \frac{B_i(t)}{K + B_i(t)} \, dt$$

is the cumulative environmental infection pressure in Region i. In the absence of environmental exposure ($\mathcal{E}_i = 0$), this reduces to the classical final size equation $\ln(1/u_i) = \mathcal{R}_0(1 - u_i)$. The derivation is given in Appendix D.

Corollary 3.6 (Threshold Behavior of the Basic Reproduction Number)**.** *Suppose the final size relation*

$$\ln\left(\frac{1}{u_i}\right) = \mathcal{R}_0(1 - u_i) + \mathcal{E}_i, \qquad \mathcal{E}_i \geq 0,$$

holds. Then:

- *If $\mathcal{R}_0 < 1$ and $\mathcal{E}_i = 0$, the only solution is $u_i \to 1$, yielding a negligible final epidemic size.*
- *If $\mathcal{R}_0 > 1$, there exists a solution $u_i < 1$, corresponding to a nontrivial outbreak.*
- *If $\mathcal{R}_0 < 1$ but $\mathcal{E}_i > 0$, environmental exposure can still induce a positive final epidemic size despite subcritical endogenous transmission.*

Thus, $\mathcal{R}_0 = 1$ remains the threshold for invasion driven by local transmission, while environmental forcing modifies outbreak magnitude without altering the intrinsic threshold.

This approximation is most accurate for large populations and single-wave epidemics, where stochastic effects, demographic turnover, reinfection, and spatial heterogeneity are negligible. Despite these limitations, the final size relation provides useful analytical insight for short- to moderate-duration outbreaks in homogeneous settings. In contrast to prior work on COVID-19 dynamics (Thomas, 2024), where the final size relation was obtained as an inequality using age-of-infection models and bounding arguments, the present study employs a deterministic compartmental framework and derives an exact equality by direct integration of the susceptible equation.

3.5 Thresholds for Bacterial Persistence and Elimination

Theorem 3.7 (Global Stability of the Bacteria-Free State)**.** *Consider the bacterial concentration $B_i(t, x)$ in Region $i \in \{1, 2, 3\}$ governed by*

$$\frac{\partial B_i}{\partial t} + \mathbf{v}_i \cdot \nabla B_i = D_i \nabla^2 B_i + \tilde{\alpha}\, I_i + \sum_{j \neq i} q_{ji}(t)\, B_j - \sum_{j \neq i} q_{ij}(t)\, B_i - z_i B_i + \Lambda_i(t),$$

with nonnegative initial data $B_i(0, x) \geq 0$. Define the bacterial persistence threshold

$$\mathcal{Q}_B := \max_{i \in \{1,2,3\}} \frac{\tilde{\alpha}\, I_{\max} + \Lambda_{\max}}{z_i + \sum_{j \neq i} \underline{q}_{ij}},$$

where $I_{\max}$ and $\Lambda_{\max}$ are uniform upper bounds on $I_i(t, x)$ and $\Lambda_i(t)$, and $\underline{q}_{ij}$ is a positive lower bound on $q_{ij}(t)$. If $\mathcal{Q}_B < 1$, then the bacteria-free equilibrium

$$B_i^*(x) \equiv 0, \qquad i \in \{1, 2, 3\},$$

is globally asymptotically stable, i.e., $B_i(t, x) \to 0$ as $t \to \infty$ for all admissible initial conditions. The proof is given in Appendix E.

The threshold $\mathcal{Q}_B$ quantifies the balance between bacterial input and effective removal. The numerator represents bacterial introduction through human shedding and exogenous environmental contamination, while the denominator captures removal via natural decay, sanitation, and hydrological export. When $\mathcal{Q}_B < 1$, bacterial concentrations decay to zero for all admissible initial conditions; when $\mathcal{Q}_B > 1$, environmental contamination persists and can sustain transmission.

From a control perspective, elimination can be achieved by reducing shedding, enhancing bacterial clearance, increasing hydrological flushing, or limiting external inputs. Accordingly, $\mathcal{Q}_B$ provides a practical criterion for determining whether local interventions suffice or coordinated multi-region control is required.

3.6 Intervention Effects and Structural Risk

We analyze the effects of interventions on environmental bacterial persistence using analytical tools including Lyapunov stability, monotonicity arguments, and threshold analysis. This framework identifies conditions under which interventions successfully eliminate environmental reservoirs, as well as regimes in which well-intended external actions may inadvertently sustain bacterial persistence by introducing persistent environmental forcing.

Theorem 3.8 (Monotone Effect of Exogenous Contamination on Environmental Persistence)**.** *Consider the bacterial concentration in Region i governed by*

$$\frac{\partial B_i}{\partial t} + \mathbf{v}_i \cdot \nabla B_i = D_i \nabla^2 B_i + \tilde{\alpha}\, I_i + \sum_{j \neq i} q_{ji}(t)\, B_j - \sum_{j \neq i} q_{ij}(t)\, B_i - z_i B_i + \Lambda_i(t),$$

where all parameters are nonnegative, $z_i > 0$, and $\Lambda_i(t) \geq 0$ represents exogenous environmental contamination. Assume spatial homogeneity (or spatial averaging with zero-flux boundary conditions), bounded transport rates $q_{ij}(t)$, and uniformly bounded $I_i(t)$. Then the reduced mean bacterial dynamics admit a unique nonnegative equilibrium B_i^*, which depends strictly monotonically *on Λ_i. In particular,*

$$\frac{\partial B_i^*}{\partial \Lambda_i} > 0 \qquad \text{whenever } B_i^* > 0.$$

Moreover, if $I_i \equiv 0$ and $\Lambda_i > 0$, the system admits a unique positive environmental equilibrium, whereas if $I_i \equiv 0$ and $\Lambda_i \equiv 0$, the bacteria-free state $B_i^* = 0$ is globally asymptotically stable. The proof is given in Appendix F.

Theorem 3.8 establishes that any persistent external input $\Lambda_i > 0$ sustains a nonzero environmental bacterial reservoir, even in the absence of local human infection. Exogenous contamination therefore acts as a *structural forcing mechanism*, elevating long-term environmental burden independently of endogenous transmission dynamics.

In the Haitian context, the 2010 introduction of cholera by UN peacekeepers (Katz, 2016; Frerichs, 2016; Orata et al., 2014) may be interpreted as a sustained increase in Λ_i due to contaminated waste discharge into the Artibonite River system. Under transport-driven dynamics, such forcing necessarily shifts the system to a higher environmental equilibrium, facilitating persistence and downstream spread. This provides a mathematical formalization of observations documented in epidemiological, historical, and sociological studies: well-intended external interventions can introduce structural risks that persist beyond the initiating event. Within this framework, the Western savior complex is not modeled as intent, but as a *mechanism of exogenous environmental forcing*, whereby externally imposed actions alter system dynamics in ways local control measures alone cannot reverse (Farmer, 2011; Dubois, 2012; Jean-Louis, 2020).

4 Numerical Simulations

4.1 Parameter Values and Simulation Setup

We specify the baseline parameter values, initial conditions, and numerical methods used in all simulations, including the simulation horizon, integration scheme, spatial structure, and implementation of hydrological transport $q_{ij}(t)$ and exogenous environmental contamination $\Lambda_i(t)$.

All simulations use the baseline parameter values listed in Tables 2-3, which define demographic dynamics, disease progression, environmental transmission, bacterial decay, and hydrological coupling across the three regions. Initial conditions were chosen to reflect heterogeneous regional disease burden and environmental exposure. Unless stated otherwise, simulations were conducted over a one-year horizon $t \in [0, 365]$ using the stiff solver `ode15s` with relative tolerance 10^{-7} and absolute tolerance 10^{-9}.

Hydrological transport was modeled through a constant downstream coupling rate q_{12} from Region (1) to Region (2), while Region (3) was treated as environmentally isolated. Exogenous contamination was introduced via a constant forcing term Λ_1 applied to the upstream region. Sensitivity to external contamination was examined by varying Λ_1 while holding all other parameters fixed.

Table 2: Baseline parameter values used in numerical simulations. City indices correspond to (1) Mirebalais, (2) Gonaïves, and (3) Port-au-Prince.

Parameter	Value	Source
μ	$1/(65 \times 365)$	(Codeço, 2001; World Health Organization, 2023)
d	$1/(70 \times 365)$	(World Health Organization, 2023) (United Nations, 2022)
β	0.04-0.08	(Tuite et al., 2011; Eisenberg et al., 2013)
η	0.2-0.4	(Mukandavire et al., 2011)
σ	$1/5$	(Centers for Disease Control and Prevention, 2023)
γ	$1/7$	(Centers for Disease Control and Prevention, 2023; Codeço, 2001)
ν	0.001-0.005	(World Health Organization, 2011–2019; Pan American Health Organization, 2019)
ξ	$1/(3\text{-}5 \times 365)$	(Koelle et al., 2005).
α	0.05-0.2	(Hartley et al., 2006).
K	10^4	(Codeço, 2001).
z_1	0.3-0.5	(Hartley et al., 2006).

Continued on next page

Parameter	Value	Source
z_2	0.3-0.5	(Mukandavire et al., 2011)
z_3	0.3	(Eisenberg et al., 2013)
q_{12}	0.1	(Tuite et al., 2011)
m_{13}	0.005	(International Organization for Migration, 2011; Tuite et al., 2011)
m_{23}	0.002	(International Organization for Migration, 2011)

Table 3: Initial conditions for epidemiological compartments and bacterial concentrations used in numerical simulations. City indices correspond to (1) Mirebalais, (2) Gonaïves, and (3) Port-au-Prince.

Region	$S_i(0)$	$E_i(0)$	$I_i(0)$	$R_i(0)$	$B_i(0)$	Justification / Source
Mirebalais (1)	5.0×10^2	8	2	0	10^{-6}	(Piarroux et al., 2011; Tuite et al., 2011)
Gonaïves (2)	8.0×10^2	10	5	0	10^{-6}	(Barzilay et al., 2013; Tuite et al., 2011)
Port-au-Prince (3)	9.0×10^2	15	10	0	10^{-6}	(Piarroux et al., 2011; Tuite et al., 2011; Ivers and Walton, 2013)

We vary the exogenous contamination input $\Lambda_1(t)$ over magnitudes scaled to endogenous shedding,

$$\Lambda_1(t) = L\,\mathbb{I}_{[t_0,t_1]}(t), \qquad L \in \{0, 0.1, 0.25, 0.5, 1, 2, 5\} \times (\alpha I_{\text{ref}}),$$

so external forcing ranges from negligible to several-fold larger than the typical internal contribution $\tilde{\alpha} I$. Initial conditions reflect relative outbreak timing and hydrological connectivity rather than exact case counts, and results are robust to moderate variation in these values.

4.2 Numerical Results in a Three-Region Hydrological Network

Figures 2 and 3 show the baseline dynamics of the three-region SEIR-B system with no exogenous environmental contamination ($\Lambda_i(t) \equiv 0$). Figure 2 displays the infectious populations $I_1(t)$, $I_2(t)$, and $I_3(t)$, while Figure 3 shows the corresponding bacterial concentrations $B_1(t)$, $B_2(t)$, and $B_3(t)$. Under baseline conditions, transmission arises solely from endogenous processes, including direct human-to-human spread and locally generated environmental reservoirs. The infection trajectories in Figure 2 exhibit distinct regional epidemic patterns driven by heterogeneous initial conditions and asymmetric network coupling. Although Region (3) receives no environmental input via hydrological transport, its infection dynamics are indirectly influenced by upstream regions through human migration.

The bacterial dynamics in Figure 3 reflect the underlying hydrological network: Region (1) acts as an upstream bacterial source, Region (2) receives downstream inflow, and Region (3) remains environmentally isolated, with bacterial concentrations governed solely by local shedding and natural decay.

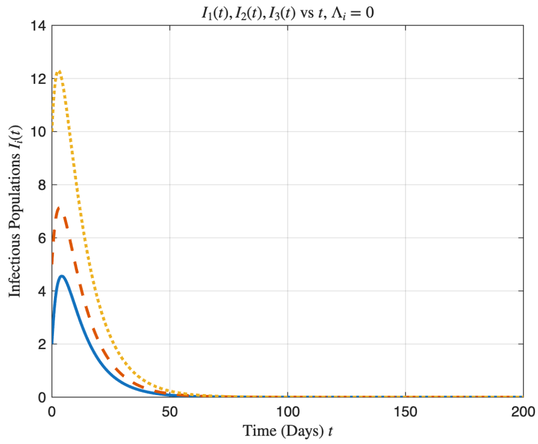


Figure 2: Temporal evolution of the infectious populations $I_1(t)$, $I_2(t)$, and $I_3(t)$ in the three-region SEIR-B model under baseline conditions with no exogenous environmental contamination ($\Lambda_i(t) \equiv 0$).

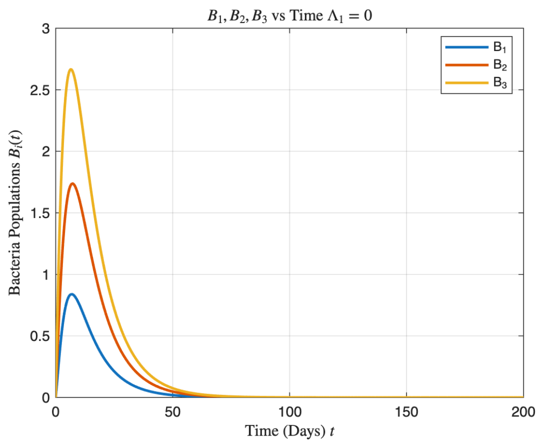


Figure 3: Time evolution of environmental bacterial concentrations $B_1(t)$, $B_2(t)$, and $B_3(t)$ under baseline conditions with $\Lambda_i(t) \equiv 0$.

Together, these simulations characterize the intrinsic coupling between infection and environmental contamination in the absence of external forcing, providing a baseline for assessing the effects of exogenous contamination, control interventions, and transport intensity in subsequent sections.

Figures 4-6 illustrate the effects of time-dependent exogenous environmental contamination on bacterial dynamics in the three-region hydrological network. An external input $\Lambda_1(t)$ is applied to the upstream region while all other parameters remain at baseline values, isolating the impact of exogenous contamination from endogenous transmission.

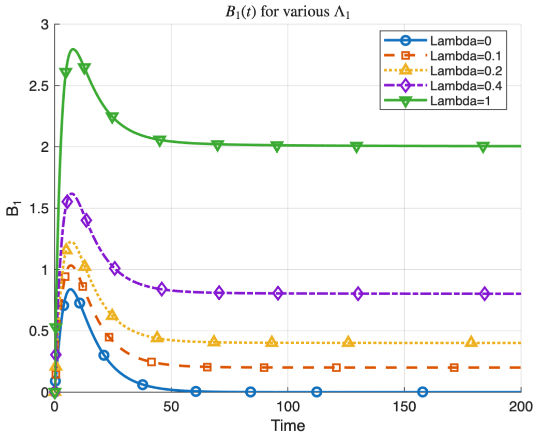


Figure 4: Environmental bacterial concentration $B_1(t)$ in the upstream region for increasing magnitudes of the exogenous contamination input $\Lambda_1(t)$.

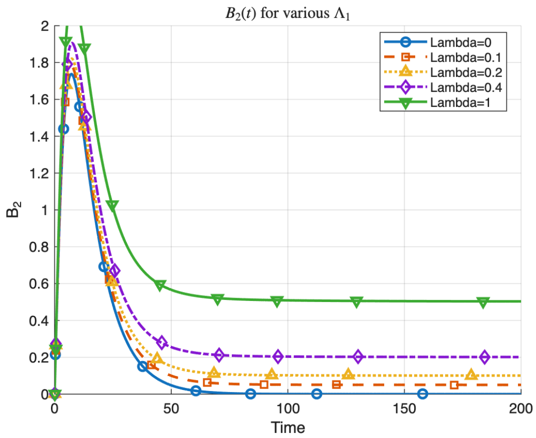


Figure 5: Environmental bacterial concentration $B_2(t)$ in the downstream region under increasing upstream contamination $\Lambda_1(t)$.

As shown in Figure 4, increasing the magnitude of $\Lambda_1(t)$ produces a monotone increase in the bacterial concentration $B_1(t)$ in the upstream region. Elevated bacterial levels propagate

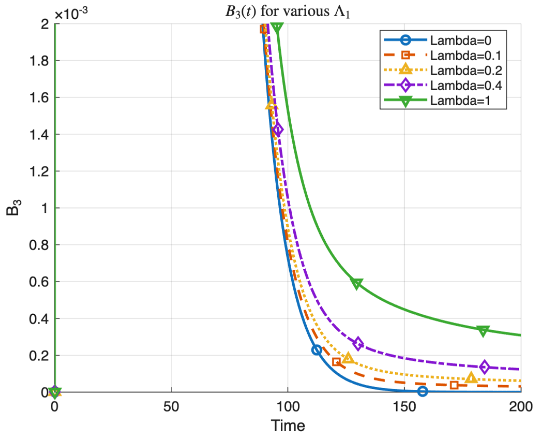


Figure 6: Environmental bacterial concentration $B_3(t)$ in the environmentally isolated region.

downstream to Region (2) via hydrological transport (Figure 5), while Region (3) shows no direct bacterial response, reflecting its environmental isolation (Figure 6). The corresponding infection dynamics (Figures 7-9) exhibit only modest sensitivity to $\Lambda_1(t)$, as exogenous contamination influences prevalence indirectly through environmental and migratory coupling, particularly in regions without direct environmental inflow.

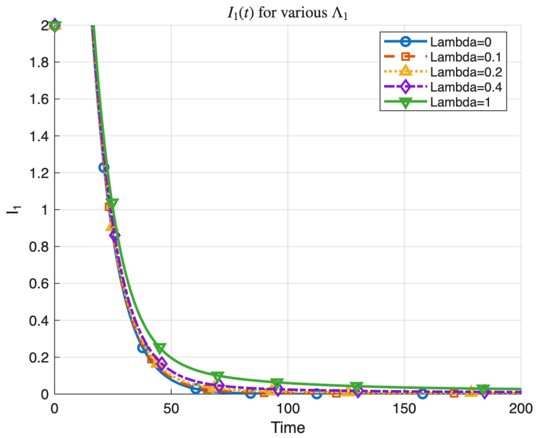


Figure 7: Infectious population $I_1(t)$ in the upstream region for varying magnitudes of $\Lambda_1(t)$.

These numerical results are consistent with the analytical monotonicity result in Theorem 3.8, which predicts sustained external contamination prevents bacterial elimination in affected regions. Together, the simulations confirm exogenous inputs act as a dominant driver of environmental persistence, while downstream epidemiological effects depend critically on network structure and coupling pathways. The analytical results identify conditions under which environmental reservoirs of *Vibrio cholerae* can be eliminated through reductions in bacterial shedding and environmental

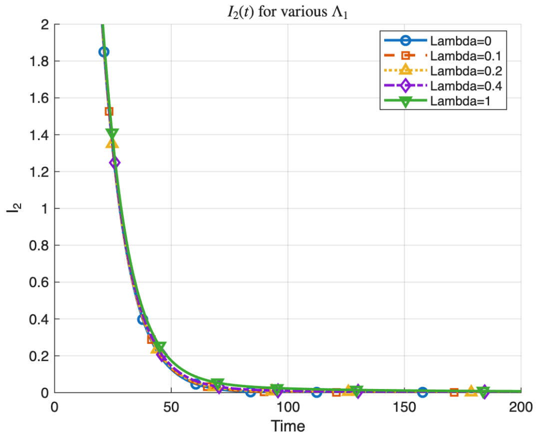


Figure 8: Infectious population $I_2(t)$ in the downstream region under increasing upstream contamination.

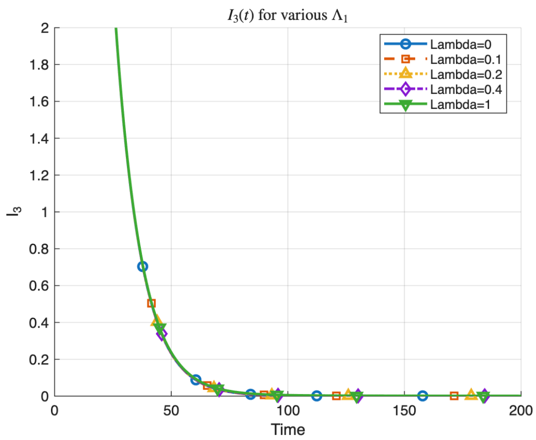


Figure 9: Infectious population $I_3(t)$ in the environmentally isolated region.

transmission, revealing a threshold separating persistence from elimination. Although the numerical simulations do not explicitly vary sanitation or shedding parameters, the strong sensitivity of bacterial dynamics to environmental inputs highlights the central role of these mechanisms in sustaining infection. In the absence of sufficient control, bacterial reservoirs persist at positive endemic levels, whereas sufficiently strong reductions in shedding or environmental transmission are predicted to suppress persistence and drive elimination. Taken together, the theoretical and numerical results underscore environmental control measures, such as improved sanitation and reduced pathogen shedding, are essential components of effective intervention strategies, even in the absence of ongoing exogenous contamination.

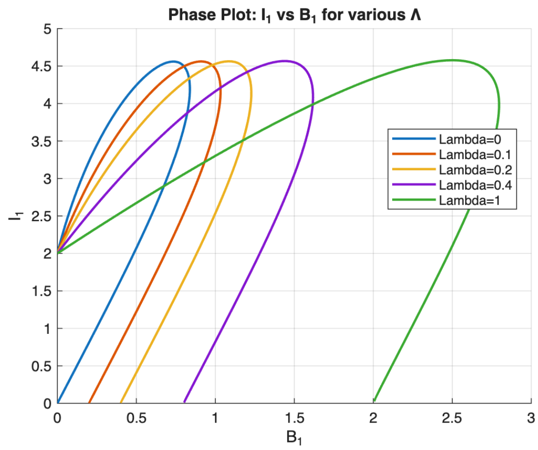


Figure 10: Phase-plane trajectories of $I_1(t)$ versus $B_1(t)$ in the upstream region under varying $\Lambda_1(t)$.

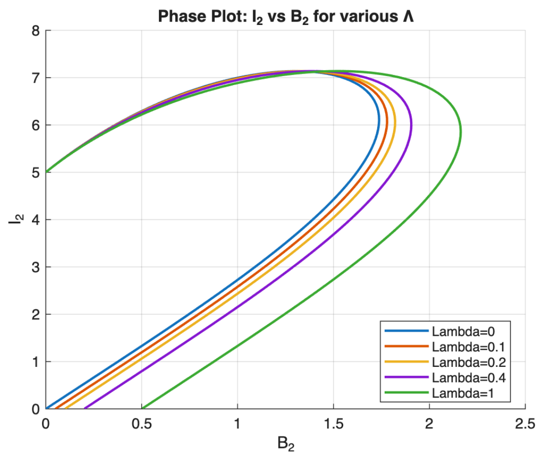


Figure 11: Phase-plane trajectories of $I_2(t)$ versus $B_2(t)$ in the downstream region.

Although the analytical results identify conditions for eliminating environmental reservoirs through sanitation and shedding control, the numerical simulations here focus on exogenous contamination and hydrological coupling. Accordingly, explicit variation of sanitation or shedding parameters is

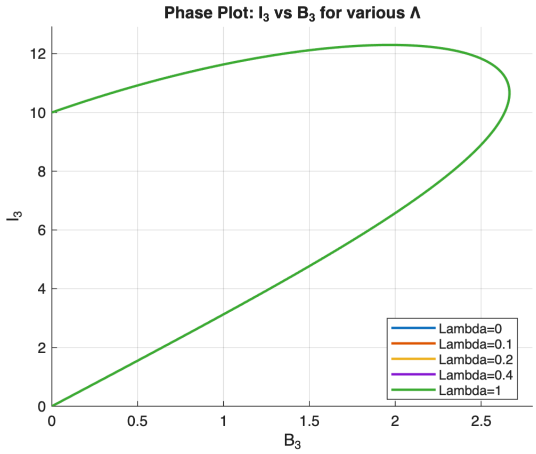


Figure 12: Phase-plane trajectories of $I_3(t)$ versus $B_3(t)$ in the environmentally isolated region.

not shown. Nonetheless, the simulations demonstrate bacterial persistence is strongly governed by environmental feedback, suggesting that reductions in shedding and environmental transmission would be decisive for elimination. A systematic numerical study of sanitation-driven thresholds is deferred to future work.

5 Discussion and Conclusions

5.1 Interpretation of Numerical Results

We examine cholera dynamics in a three-region system where transmission occurs through both direct contact and environmental exposure, and environmental bacteria evolve via shedding, decay, and hydrological transport. The upstream region includes both an outflow term $-q_{12}(t)B_1$ and an external forcing term $\Lambda_1(t)$, while the downstream region receives bacterial inflow through $q_{12}(t)B_1$. This structure distinguishes persistence driven by endogenous transmission from persistence maintained by sustained exogenous contamination.

Baseline simulations (Figures 2-3) show when exogenous contamination is absent $(\Lambda_i \equiv 0)$, bacteria arise solely from infection-driven shedding and are attenuated by decay and hydrological loss. Under these conditions, large transient outbreaks may still occur, accompanied by a rise-and-fall response in environmental reservoirs (Figure 3), demonstrating the amplifying role of environmental feedback even in the absence of external forcing.

Forcing experiments show upstream environmental inputs propagate downstream through hydrological coupling. Increasing $\Lambda_1(t)$ elevates the upstream reservoir $B_1(t)$ (Figure 4), which in turn increases the downstream reservoir $B_2(t)$ via the transport term $q_{12}(t)B_1$ (Figure 5). Correspondingly, infection trajectories exhibit increased outbreak magnitude and persistence as $\Lambda_1(t)$ increases (Figures 7-9), indicating local disease control can be undermined by persistent upstream contamination.

In contrast, Region 3 is environmentally isolated and receives neither hydrological inflow nor direct forcing. As a result, its bacterial reservoir $B_3(t)$ is governed primarily by local shedding and decay and exhibits weak sensitivity to changes in Λ_1 (Figures 6, 12). More generally, these results highlight in environmentally uncoupled regions, upstream contamination affects local dynamics primarily through human mobility rather than waterborne pathways.

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Appendix

A Existence, Uniqueness, and Positivity (Bacteria)

Proof. Let's define

$$\mathbf{B}(t,x) := (B_1(t,x), B_2(t,x), B_3(t,x))^\top \implies \tag{A.1}$$

$$\partial_t \mathbf{B}(t) = \mathcal{L}\mathbf{B}(t) + \mathcal{Q}(t)\mathbf{B}(t) + \mathbf{F}(t), \qquad t \in (0,T), \tag{A.2}$$

where $\mathcal{L}$ is the (block-diagonal) advection-diffusion operator

$$(\mathcal{L}\mathbf{B})_i = -\mathbf{v}_i \cdot \nabla B_i + D_i \Delta B_i - z_i B_i, \qquad i = 1,2,3,$$

with homogeneous Neumann boundary conditions $\nabla B_i \cdot \mathbf{n} = 0$ on $\partial\Omega$, and $\mathcal{Q}(t)$ is the (time-dependent) migration operator defined by

$$(\mathcal{Q}(t)\mathbf{B})_i = \sum_{j \neq i} q_{ji}(t)\, B_j - \sum_{j \neq i} q_{ij}(t)\, B_i, \qquad i = 1,2,3.$$

Finally, the forcing term is

$$\mathbf{F}(t,x) = \big(\tilde{\alpha} I_1(t,x) + \Lambda_1(t,x),\ \tilde{\alpha} I_2(t,x) + \Lambda_2(t,x),\ \tilde{\alpha} I_3(t,x) + \Lambda_3(t,x)\big)^\top.$$

I. Existence and Uniqueness.

For each $i = 1,2,3$, the operator

$$-\mathbf{v}_i \cdot \nabla + D_i \Delta - z_i$$

with homogeneous Neumann boundary conditions generates a strongly continuous (indeed analytic) semigroup on $L^2(\Omega)$. Hence, the block-diagonal operator $\mathcal{L}$ generates an analytic semigroup on $L^2(\Omega)^3$.

Moreover, since $q_{ij} \in L^\infty(0,T)$ for $i \neq j$, the operator $\mathcal{Q}(t)$ is bounded on $L^2(\Omega)^3$ for almost every $t \in (0,T)$, with

$$\|\mathcal{Q}(t)\|_{\mathcal{L}(L^2(\Omega)^3)} \leq C \max_{i\neq j} \|q_{ij}\|_{L^\infty(0,T)},$$

for some constant $C > 0$ depending only on the number of regions. In addition, the forcing term $\mathbf{F} \in L^2(0,T;L^2(\Omega)^3)$ under the stated assumptions on I_i and Λ_i.

Therefore, (A.2) is a linear nonautonomous evolution problem on $L^2(\Omega)^3$. By standard semigroup theory for linear parabolic systems (see Pazy (Pazy, 1983) and Amann (Amann, 1995)), there exists a unique mild solution satisfying

$$\mathbf{B}(t) \in C([0,T];L^2(\Omega)^3) \cap L^2(0,T;H^1(\Omega)^3).$$

II. Positivity.

We now show the solution $\mathbf{B} = (B_1, B_2, B_3)$ constructed above remains nonnegative for all $t \in [0,T]$, provided the initial data and source terms are nonnegative.

Each equation in the system is linear and parabolic, with homogeneous Neumann boundary conditions. Moreover, the source terms satisfy

$$\tilde{\alpha} I_i(t,x) + \Lambda_i(t,x) \geq 0 \quad \text{for almost every } (t,x) \in (0,T) \times \Omega,$$

by assumption.
The coupling (migration) terms have the form

$$\sum_{j\neq i} q_{ji}(t)\, B_j - \sum_{j\neq i} q_{ij}(t)\, B_i,$$

where $q_{ij}(t) \geq 0$ for all $i \neq j$. Thus, the off-diagonal terms enter each equation with nonnegative coefficients, while the diagonal terms are non-positive. Consequently, the system is *cooperative* in the sense of monotone dynamical systems.

Equivalently, the migration operator $\mathcal{Q}(t)$ generates a positive linear operator on $L^2(\Omega)^3$; that is, if $\mathbf{B} \geq 0$ componentwise, then $\mathcal{Q}(t)\mathbf{B}$ has no negative contributions that could create sign changes. The advection-diffusion operator with Neumann boundary conditions also preserves positivity.

Therefore, the full evolution operator associated with

$$\partial_t \mathbf{B} = \mathcal{L}\mathbf{B} + \mathcal{Q}(t)\mathbf{B} + \mathbf{F}(t)$$

generates a positive semigroup on $L^2(\Omega)^3$. Since $\mathbf{B}(0) \geq 0$ and $\mathbf{F}(t) \geq 0$, standard results for cooperative parabolic systems and the weak maximum principle (see Evans (Evans, 2010)) imply

$$B_i(t,x) \geq 0 \quad \text{for almost every } x \in \Omega,\ \forall\, t \in [0,T],\ i = 1,2,3.$$

□

B Existence, Uniqueness, and Positivity (Human)

Proof. **I. Existence of Solutions**.

Fix $i \in \{1, 2, 3\}$. Let
$$X_i(t) := (S_i(t), E_i(t), I_i(t), R_i(t))^\top.$$
The right-hand side of (2.1) is continuous in t and locally Lipschitz in X_i on any set where $N_i(t) = S_i + E_i + I_i + R_i$ is bounded away from 0.

Indeed, the only potentially singular term is $\frac{I_i}{N_i}$, and for nonnegative states we have $0 \le I_i \le N_i$, hence $\frac{I_i}{N_i} \in [0, 1]$ whenever $N_i > 0$.) Therefore, by the Picard-Lindelöf theorem, for any initial condition $X_i(0) \in \mathbb{R}^4_{\ge 0}$ with $N_i(0) > 0$, there exists a unique local solution $X_i(t)$ on some interval $[0, T_{\max})$.

To extend the local solution globally, we show the total population remains bounded in time.
$$N_i(t) := S_i(t) + E_i(t) + I_i(t) + R_i(t).$$
Summing the equations in (2.1) gives
$$\frac{dN_i}{dt} = \mu - dN_i - \tilde{\nu} I_i + \Pi_i(t). \tag{B.1}$$
Since $I_i(t) \ge 0$ and $\tilde{\nu} \ge 0$, we obtain the differential inequality
$$\frac{dN_i}{dt} \le \mu + \Pi_i(t) - dN_i. \tag{B.2}$$
Assuming $\Pi_i \in L^\infty(0, T)$ and $\Pi_i(t) \ge 0$, Grönwall's inequality (or the comparison principle for scalar ODEs) implies that for all $t \in [0, T]$,
$$0 \le N_i(t) \le e^{-dt} N_i(0) + \int_0^t e^{-d(t-s)} \big(\mu + \Pi_i(s)\big)\, ds \le N_i(0) + \frac{\mu + \|\Pi_i\|_{L^\infty(0,T)}}{d}. \tag{B.3}$$
Thus $N_i(t)$ remains bounded on finite time intervals and cannot blow up in finite time.

Next we note the nonnegative orthant is forward invariant. Indeed, on the boundary where $S_i = 0$ we have
$$\left.\frac{dS_i}{dt}\right|_{S_i=0} = \mu + \tilde{\xi} R_i \ge 0,$$
on the boundary where $E_i = 0$ we have
$$\left.\frac{dE_i}{dt}\right|_{E_i=0} = \tilde{\beta} \frac{I_i}{N_i} S_i + \tilde{\eta} \frac{B_i}{K + B_i} S_i + \Pi_i(t) \ge 0,$$
on the boundary where $I_i = 0$ we have
$$\left.\frac{dI_i}{dt}\right|_{I_i=0} = \tilde{\sigma} E_i \ge 0,$$

and on the boundary where $R_i = 0$ we have

$$\frac{dR_i}{dt}\bigg|_{R_i=0} = \tilde{\gamma} I_i \geq 0.$$

Hence $S_i(t), E_i(t), I_i(t), R_i(t)$ remain nonnegative for as long as the solution exists, and in particular $N_i(t) \geq 0$.

Finally, the bound (B.3) rules out finite-time blow-up of solutions, and therefore the local solution extends to a unique global solution on $[0, T]$ (and hence for all $t \geq 0$). This completes the proof of existence for the human SEIR subsystem.

II. Uniqueness of Solutions.

Fix $i \in \{1, 2, 3\}$ and define the state vector

$$\mathbf{X}(t) = (S_i(t), E_i(t), I_i(t), R_i(t))^\top, \qquad N_i(t) = S_i(t) + E_i(t) + I_i(t) + R_i(t).$$

Write (2.1) in the standard form

$$\frac{d\mathbf{X}}{dt} = F\big(t, \mathbf{X}\big),$$

where $F : [0, T] \times \mathbb{R}^4 \to \mathbb{R}^4$ is given by the right-hand sides of (2.1). We prove uniqueness by showing $F(t, \cdot)$ is locally Lipschitz in $\mathbf{X}$ on any positively invariant set where $N_i(t)$ is bounded away from 0.

Let $\mathcal{D} \subset \mathbb{R}^4_{\geq 0}$ be a bounded set such that

$$0 < N_{\min} \leq S + E + I + R \leq N_{\max} \quad \text{for all } (S, E, I, R) \in \mathcal{D}.$$

(Existence and positivity imply that solutions with $N_i(0) > 0$ remain in such a region on $[0, T]$.) Assume also $B_i(t) \geq 0$ and $\Pi_i(t) \geq 0$ are given bounded functions on $[0, T]$ (as ensured by the bacterial subsystem and the hypotheses on Π_i). Then the map

$$(S, E, I, R) \;\mapsto\; \frac{I}{S + E + I + R}$$

is Lipschitz on $\mathcal{D}$, since the denominator is bounded below by $N_{\min}$.

To illustrate, consider the susceptible component

$$F_S(t, \mathbf{X}) = \mu + \tilde{\xi}(1 - \epsilon_R)R - \tilde{\beta}(1 - \epsilon_S)\frac{I}{N}S - \tilde{\eta}(1 - \epsilon_B)\frac{B_i(t)}{K + B_i(t)}S - dS,$$

where $N = S + E + I + R$. Let $\mathbf{X}_1 = (S_1, E_1, I_1, R_1)$ and $\mathbf{X}_2 = (S_2, E_2, I_2, R_2)$ be in $\mathcal{D}$. Using the triangle inequality and boundedness of S and N^{-1} on $\mathcal{D}$, we obtain

$$\begin{aligned} |F_S(t, \mathbf{X}_1) - F_S(t, \mathbf{X}_2)| \leq{}& \tilde{\xi}(1 - \epsilon_R)|R_1 - R_2| + d|S_1 - S_2| \\ &+ \tilde{\beta}(1 - \epsilon_S)\left|\frac{I_1}{N_1}S_1 - \frac{I_2}{N_2}S_2\right| + \tilde{\eta}\frac{B_i(t)}{K + B_i(t)}|S_1 - S_2|. \end{aligned}$$

Moreover, since $0 \leq \dfrac{B_i(t)}{K + B_i(t)} \leq 1$, the environmental term is bounded by $\tilde{\eta}|S_1 - S_2|$.

For the incidence term, write

$$\frac{I_1}{N_1}S_1 - \frac{I_2}{N_2}S_2 = \frac{I_1}{N_1}(S_1 - S_2) + S_2\left(\frac{I_1}{N_1} - \frac{I_2}{N_2}\right).$$

On $\mathcal{D}$, $S_2 \le N_{\max}$ and $N_1, N_2 \ge N_{\min}$, hence

$$\left|\frac{I_1}{N_1}(S_1 - S_2)\right| \le |S_1 - S_2| \quad \text{and} \quad \left|\frac{I_1}{N_1} - \frac{I_2}{N_2}\right| = \left|\frac{I_1 N_2 - I_2 N_1}{N_1 N_2}\right| \le \frac{1}{N_{\min}^2}\Big(|I_1 - I_2|\, N_{\max} + |N_1 - N_2|\, N_{\max}\Big).$$

Since $|N_1 - N_2| \le |S_1 - S_2| + |E_1 - E_2| + |I_1 - I_2| + |R_1 - R_2|$, it follows:

$$\left|\frac{I_1}{N_1}S_1 - \frac{I_2}{N_2}S_2\right| \le C\Big(|S_1 - S_2| + |E_1 - E_2| + |I_1 - I_2| + |R_1 - R_2|\Big),$$

for a constant $C > 0$ depending only on $N_{\min}$ and $N_{\max}$. Therefore, for each fixed $t \in [0, T]$,

$$|F_S(t, \mathbf{X}_1) - F_S(t, \mathbf{X}_2)| \le L\|\mathbf{X}_1 - \mathbf{X}_2\|$$

for some constant $L > 0$ independent of $\mathbf{X}_1, \mathbf{X}_2 \in \mathcal{D}$.

The same argument applies to $F_E(t, \mathbf{X})$, since it contains the same incidence terms and otherwise only linear terms, while F_I and F_R are linear in (E, I, R). Hence $F(t, \cdot)$ is locally Lipschitz on $\mathcal{D}$ uniformly in $t \in [0, T]$. By the Picard-Lindelöf theorem, solutions to (2.1) with the same initial data are unique on $[0, T]$.

III. Positivity of Solutions.

We now show solutions of the human SEIR system (2.1) remain nonnegative for all $t \ge 0$, provided they start from nonnegative initial data. Assume

$$S_i(0) \ge 0, \qquad E_i(0) \ge 0, \qquad I_i(0) \ge 0, \qquad R_i(0) \ge 0,$$

with $N_i(0) > 0$.

We verify the nonnegative orthant $\mathbb{R}^4_{\ge 0}$ is forward invariant by examining the vector field on each coordinate hyperplane.

- **Susceptible compartment.** If $S_i = 0$, then

$$\frac{dS_i}{dt}\Big|_{S_i=0} = \mu + \tilde{\xi}(1 - \epsilon_R)R_i \ge 0,$$

  since $\mu \ge 0$ and $R_i \ge 0$.
- **Exposed compartment.** If $E_i = 0$, then

$$\frac{dE_i}{dt}\Big|_{E_i=0} = \tilde{\beta}(1 - \epsilon_S)\frac{I_i}{N_i}S_i + \tilde{\eta}(1 - \epsilon_B)\frac{B_i}{K + B_i}S_i + \Pi_i(t) \ge 0,$$

  because $S_i, I_i, B_i, \Pi_i(t) \ge 0$.

- **Infectious compartment.** If $I_i = 0$, then

$$\frac{dI_i}{dt}\Big|_{I_i=0} = \tilde{\sigma}(1-\epsilon_E)E_i \geq 0,$$

since $E_i \geq 0$.

- **Recovered compartment.** If $R_i = 0$, then

$$\frac{dR_i}{dt}\Big|_{R_i=0} = \gamma(1+\epsilon_I)I_i \geq 0,$$

since $I_i \geq 0$.

Thus, on each boundary face of the nonnegative orthant, the vector field points inward or is tangent. Consequently, no component of the solution can cross into negative values. Therefore,

$$S_i(t) \geq 0, \quad E_i(t) \geq 0, \quad I_i(t) \geq 0, \quad R_i(t) \geq 0 \qquad \text{for all } t \geq 0.$$

This shows the human SEIR subsystem preserves positivity.

Since existence, uniqueness, and positivity have been established, the human population subsystem is well posed.

□

C Lyapunov Stability Derivations

Proof. We consider the infected and environmental subsystems of the three-region SEIR-B model. Assume there is *no exogenous contamination*, i.e.

$$\Lambda_i(t) \equiv 0, \qquad i = 1, 2, 3.$$

Note: Only Region 1 has hydrological outflow to Region 2 through $q_{12}(t)$, and Region 3 receives infection pressure through migration terms $m_{13}I_1$ and $m_{23}I_2$ entering E_3.

Let is define

$$L(t) = a\,(E_1^2 + E_2^2 + E_3^2) + b\,(I_1^2 + I_2^2 + I_3^2) + c_1B_1^2 + c_2B_2^2 + c_3B_3^2, \tag{C.1}$$

where $a, b, c_1, c_2, c_3 > 0$. Then $L(t) > 0$ for all nonzero states and $L(t) = 0$ only at the disease-free state in the (E, I, B) variables.

Differentiating (C.1) gives

$$\dot{L}(t) = 2a\sum_{i=1}^{3} E_i\dot{E}_i + 2b\sum_{i=1}^{3} I_i\dot{I}_i + 2c_1B_1\dot{B}_1 + 2c_2B_2\dot{B}_2 + 2c_3B_3\dot{B}_3.$$

Using Young's inequality and recognizing that $\frac{B_i}{K+B_i} \leq \frac{B_i}{K}$ and $S_i \leq N_i \leq \frac{\mu}{d}$ and $\frac{S_i}{N_i} < 1$, we can rewrite $\frac{dL}{dt}$ as the following inequality:

$$
\begin{aligned}
\frac{dL}{dt} \leq & \left(2Ka\beta\mu - 2Ka\,d^2 - 2Kad\sigma + 2Kbd\sigma + 2a\eta\mu\right) E_1^2 \\
& + \left(2Ka\beta\mu - 2Ka\,d^2 - 2Kad\sigma + 2Kbd\sigma + 2a\eta\mu\right) E_2^2 \\
& + \left(2Ka\beta\mu - 2Ka\,d^2 + 2Kadm_{13} + 2Kadm_{23} - 2Kad\sigma + 2Kbd\sigma + 2a\eta\mu\right) E_3^2 \\
& - 2dK\left(-am_{13} - \alpha c_1 + bd + b\gamma + b\nu - b\sigma\right) I_1^2 \\
& - 2dK\left(-am_{23} - \alpha c_2 + bd + b\gamma + b\nu - b\sigma\right) I_2^2 \\
& - 2dK\left(-\alpha c_3 + bd + b\gamma + b\nu - b\sigma\right) I_3^2 \\
& + \left(2K\alpha c_1 d - 2Kc_1 dq_{12} - 2Kc_1 dz1 + 2Kc_2 dq_{12} + 2a\eta\mu\right) B_1^2 \\
& + \left(2K\alpha c_2 d + 2Kc_2 dq_{12} - 2Kc_2 dz2 + 2a\eta\mu\right) B_2^2 \\
& + \left(2K\alpha c_3 d - 2Kc_3 dz3 + 2a\eta\mu\right) B_3^2
\end{aligned}
$$

Let us define the following coefficients:

$$
a = R_H = \frac{\beta\sigma}{(\sigma + d)(d + \gamma + \nu)}; \quad b = \frac{1}{\gamma + \nu + d}; \quad c_1 = \frac{1}{q_{12} + z_1}; \quad c_2 = \frac{1}{z_2}; \quad c_3 = \frac{1}{z_3};
$$

When $\mathcal{R}_0 < 1 \implies \mathcal{R}_H < 1$ thereby making all of the coefficients before the E_i^2, I_i^2, and B_i^2 terms negative. Thus, $\dot{L} \leq 0$, therefore, by LaSalle's Invariance Principle the DFE is globally asymptotically stable whenever $\mathcal{R}_0 < 1$. ☐

D Final Epidemic Size

Proof. We begin with the susceptible equation in region i:

$$
\frac{dS_i}{dt} = \mu + \tilde{\xi} R_i - \tilde{\beta}\frac{I_i}{N_i} S_i - \tilde{\lambda}\frac{B_i}{K + B_i} S_i - dS_i.
$$

Following the classical final-size derivations (Hethcote, 2000; Diekmann et al., 2013; Ma and Earn, 2006; Brauer et al., 2019), we consider a single epidemic wave on a time window $[0, T]$ and assume, over this window, demographic turnover and other slow processes (births, natural deaths, waning immunity, reinfection terms, and migration) contribute negligibly compared to the infection process. Under this epidemic-time-scale approximation,

$$
\frac{dS_i}{dt} \approx -\left(\tilde{\beta}\frac{I_i(t)}{N_i(t)} + \tilde{\lambda}\frac{B_i(t)}{K + B_i(t)}\right) S_i(t) \implies \tag{D.1}
$$

$$
\int_{S_i(0)}^{S_i(T)} \frac{dS_i}{S_i} = -\int_0^T \left(\tilde{\beta}\frac{I_i(t)}{N_i(t)} + \tilde{\lambda}\frac{B_i(t)}{K + B_i(t)}\right) dt, \implies \tag{D.2}
$$

$$
\ln\left(\frac{S_i(T)}{S_i(0)}\right) = -\tilde{\beta}\int_0^T \frac{I_i(t)}{N_i(t)}\,dt - \tilde{\lambda}\int_0^T \frac{B_i(t)}{K + B_i(t)}\,dt. \tag{D.3}
$$

Define the final susceptible fraction (final size variable)

$$
u_i := \frac{S_i(T)}{S_i(0)} \in (0, 1],
$$

and the cumulative infection pressures

$$\mathcal{H}_i := \tilde{\beta} \int_0^T \frac{I_i(t)}{N_i(t)}\, dt, \qquad \mathcal{E}_i := \tilde{\lambda} \int_0^T \frac{B_i(t)}{K + B_i(t)}\, dt.$$

Then the final size identity becomes the exact relation

$$\ln\left(\frac{1}{u_i}\right) = \mathcal{H}_i + \mathcal{E}_i. \tag{D.4}$$

The quantity $\mathcal{H}_i$ represents the *cumulative human-to-human infection pressure* experienced by susceptibles in Region i over the interval $[0, T]$, while $\mathcal{E}_i$ denotes the *cumulative environmental infection pressure* arising from exposure to contaminated water over the same period. Equation (D.4) therefore states that the logarithmic depletion of susceptibles equals the total accumulated force of infection, human and environmental, during a single epidemic wave.

If, in addition, the total population size remains approximately constant ($N_i(t) \approx N_i(0)$ on $[0, T]$) and the epidemic wave is driven by endogenous transmission characterized by $\mathcal{R}_0$, then (D.4) reduces to the familiar attack-rate form

$$\ln\left(\frac{1}{u_i}\right) = \mathcal{R}_0\,(1 - u_i) + \mathcal{E}_i,$$

where $1 - u_i$ is the fraction infected during the outbreak and $\mathcal{E}_i$ captures the additional contribution of environmental exposure. In the absence of environmental transmission ($\mathcal{E}_i = 0$), this expression recovers the classical final size relation $\ln(\frac{1}{u_i}) = \mathcal{R}_0(1 - u_i)$.

□

E Global Stability of the Bacteria-Free State

Proof. We consider the bacterial concentration $B_i(t, x)$ in region $i \in \{1, 2, 3\}$ governed by the transport-diffusion equation

$$\frac{\partial B_i}{\partial t} + \mathbf{v}_i \cdot \nabla B_i = D_i \nabla^2 B_i + \tilde{\alpha}\, I_i + \sum_{j \neq i} q_{ji}(t)\, B_j - \sum_{j \neq i} q_{ij}(t)\, B_i - z_i B_i + \Lambda_i(t),$$

subject to nonnegative initial data $B_i(0, x) \geq 0$.

We analyze the global stability of the bacteria-free equilibrium

$$B_i^*(x) \equiv 0, \qquad i \in \{1, 2, 3\}.$$

Assume the infectious populations $I_i(t, x)$ and external inputs $\Lambda_i(t)$ are uniformly bounded, so that

$$0 \leq I_i(t, x) \leq I_{\max}, \qquad 0 \leq \Lambda_i(t) \leq \Lambda_{\max},$$

for all $t \geq 0$ and $x \in \Omega$.

Define the Lyapunov functional

$$L(t) := \frac{1}{2}\sum_{i=1}^{3}\int_\Omega B_i(t,x)^2\,dx.$$

Clearly, $L(t) \geq 0$ with equality if and only if $B_i(t,x) \equiv 0$ for all i. Differentiating $L(t)$ along solutions gives

$$\begin{aligned}\frac{dL}{dt} &= \sum_{i=1}^{3}\int_\Omega B_i \frac{\partial B_i}{\partial t}\,dx \\ &= \sum_{i=1}^{3}\int_\Omega B_i\Big(D_i\nabla^2 B_i - \mathbf{v}_i\cdot\nabla B_i + \tilde{\alpha} I_i + \sum_{j\neq i} q_{ji}(t)B_j - \sum_{j\neq i} q_{ij}(t)B_i - z_i B_i + \Lambda_i(t)\Big)\,dx.\end{aligned}$$

We impose homogeneous Neumann boundary conditions $\partial B_i/\partial n = 0$ on $\partial\Omega$ and assume $\mathbf{v}_i\cdot\mathbf{n} = 0$ on $\partial\Omega$ with $\nabla\cdot\mathbf{v}_i = 0$. Then, by Green's identity and the divergence theorem,

$$\int_\Omega B_i\nabla^2 B_i\,dx = -\int_\Omega |\nabla B_i|^2\,dx, \qquad \int_\Omega B_i\,\mathbf{v}_i\cdot\nabla B_i\,dx = 0.$$

Substituting these identities yields

$$\begin{aligned}\frac{dL}{dt} = &-\sum_{i=1}^{3} D_i\int_\Omega |\nabla B_i|^2\,dx - \sum_{i=1}^{3} z_i\int_\Omega B_i^2\,dx - \sum_{i=1}^{3}\sum_{j\neq i}\int_\Omega q_{ij}(t)B_i^2\,dx \\ &+ \sum_{i=1}^{3}\sum_{j\neq i}\int_\Omega q_{ji}(t)B_iB_j\,dx + \sum_{i=1}^{3}\int_\Omega B_i\big(\tilde{\alpha} I_i + \Lambda_i(t)\big)\,dx.\end{aligned}$$

The transport terms represent redistribution between regions. Using the inequality $B_iB_j \leq \frac{1}{2}(B_i^2 + B_j^2)$ and assuming uniform lower bounds $\underline{q}_{ij}$ on the outflow rates $q_{ij}(t)$, we obtain

$$\sum_{i,j\neq i}\int_\Omega q_{ji}(t)B_iB_j\,dx \;\leq\; \sum_{i=1}^{3}\sum_{j\neq i}\underline{q}_{ij}\int_\Omega B_i^2\,dx.$$

Consequently,

$$\frac{dL}{dt} \leq -\sum_{i=1}^{3}\left(z_i + \sum_{j\neq i}\underline{q}_{ij}\right)\int_\Omega B_i^2\,dx + \sum_{i=1}^{3}\int_\Omega B_i\big(\tilde{\alpha} I_{\max} + \Lambda_{\max}\big)\,dx.$$

Applying Young's inequality to the source terms and defining the bacterial reproduction threshold

$$\mathcal{Q}_B := \max_{i\in\{1,2,3\}} \frac{\tilde{\alpha} I_{\max} + \Lambda_{\max}}{z_i + \sum_{j\neq i}\underline{q}_{ij}},$$

we conclude

$$\frac{dL}{dt} \le -C\,L(t) \quad \text{whenever} \quad \mathcal{Q}_B < 1,$$

for some constant $C > 0$. Therefore, $L(t) \to 0$ as $t \to \infty$, which implies

$$B_i(t, x) \to 0 \quad \text{for all } i \in \{1, 2, 3\} \text{ and } x \in \Omega.$$

Hence, the bacteria-free equilibrium is globally asymptotically stable whenever $\mathcal{Q}_B < 1$. □

F Monotonicity of Environmental Contamination

Proof. We consider the bacterial concentration in region i, governed by

$$\frac{\partial B_i}{\partial t} + \mathbf{v}_i \cdot \nabla B_i = D_i \nabla^2 B_i + \tilde{\alpha}\, I_i + \sum_{j \ne i} q_{ji}(t)\, B_j - \sum_{j \ne i} q_{ij}(t)\, B_i - z_i B_i + \Lambda_i(t). \tag{F.1}$$

We assume zero–flux boundary conditions and either spatial homogeneity or consider the spatial average over a bounded domain Ω. Define the spatial mean

$$\bar{B}_i(t) := \frac{1}{|\Omega|} \int_\Omega B_i(t, x)\, dx.$$

Under these assumptions, the diffusion and advection terms vanish upon integration, yielding the reduced mean–field equation

$$\frac{d\bar{B}_i}{dt} = \tilde{\alpha}\, \bar{I}_i + \sum_{j \ne i} \bar{q}_{ji}\, \bar{B}_j - \sum_{j \ne i} \bar{q}_{ij}\, \bar{B}_i - z_i \bar{B}_i + \bar{\Lambda}_i, \tag{F.2}$$

where bars denote time–averaged quantities and all coefficients are nonnegative.

Fix all parameters except $\bar{\Lambda}_i$. At equilibrium,

$$0 = \tilde{\alpha}\, \bar{I}_i + \sum_{j \ne i} \bar{q}_{ji}\, \bar{B}_j^* - \sum_{j \ne i} \bar{q}_{ij}\, \bar{B}_i^* - z_i \bar{B}_i^* + \bar{\Lambda}_i.$$

Rearranging gives

$$\bar{B}_i^* = \frac{\tilde{\alpha}\, \bar{I}_i + \sum_{j \ne i} \bar{q}_{ji}\, \bar{B}_j^* + \bar{\Lambda}_i}{z_i + \sum_{j \ne i} \bar{q}_{ij}}.$$

The denominator is strictly positive. Differentiating implicitly with respect to $\bar{\Lambda}_i$ yields

$$\frac{\partial \bar{B}_i^*}{\partial \bar{\Lambda}_i} = \frac{1}{z_i + \sum_{j \ne i} \bar{q}_{ij}} \;>\; 0.$$

Thus, the equilibrium bacterial concentration in region i depends *strictly monotonically* on the exogenous environmental input $\bar{\Lambda}_i$. In particular, if $\bar{\Lambda}_i^{(2)} > \bar{\Lambda}_i^{(1)}$, then

$$\bar{B}_i^*(\bar{\Lambda}_i^{(2)}) > \bar{B}_i^*(\bar{\Lambda}_i^{(1)}).$$

Finally, if $\bar{I}_i = 0$ and $\bar{\Lambda}_i > 0$, then

$$\bar{B}_i^* = \frac{\bar{\Lambda}_i}{z_i + \sum_{j \neq i} \bar{q}_{ij}} \; > \; 0,$$

so a strictly positive environmental equilibrium persists even in the absence of local human infection. If $\bar{I}_i = 0$ and $\bar{\Lambda}_i = 0$, then $\bar{B}_i^* = 0$ is the unique equilibrium. This establishes monotone dependence of the environmental bacterial equilibrium on exogenous contamination and completes the proof. □