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Global Dynamics and Continuum of Equilibria in a Two-Strain SAIR Epidemic Model with Asymptomatic Transmission

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Abstract

In this paper, we develop a two-strain SAIR epidemic model with asymptomatic transmission to investigate the mechanisms governing strain competition and coexistence. The basic reproduction numbers are derived, and threshold conditions for disease extinction and persistence are established. When the reproduction numbers differ, the strain with the larger value dominates, leading to competitive exclusion. In contrast, when the two strains have identical transmission potential, the model admits a continuum of endemic equilibria, representing a regime of neutral competition. The global dynamics of the system are rigorously characterized using Lyapunov functions and center manifold theory, including the stability of the disease-free equilibrium, boundary equilibria, and the coexistence equilibrium set. Numerical simulations are performed to support the analytical results and to illustrate the effects of key parameters on the system dynamics. These findings reveal how asymptomatic transmission and parameter balance shape multi-strain interactions, providing new insights into the persistence and coexistence of competing pathogens.

Keywords: two-strain epidemic model; asymptomatic transmission; basic reproduction number; global dynamics; Lyapunov function

MSC: 34D23; 37N25; 92D30; 34C60

1. Introduction

The spread of infectious diseases is often characterized by the coexistence and interaction of multiple pathogen strains, which may differ in transmissibility, pathogenicity, or resistance to treatment [1–4]. Understanding the mechanisms governing strain competition, exclusion, and coexistence has become a central topic in mathematical epidemiology [5,6]. In particular, multi-strain epidemic systems provide a rich framework for investigating nonlinear dynamical phenomena, including threshold effects, bifurcations, and complex equilibrium structures [7–10].

Early studies have revealed key biological and epidemiological mechanisms underlying multi-strain interactions. For instance, Blower and Chou introduced the concept of “hot zones” to describe the localized amplification of resistant strains induced by treatment insufficiency and transmission heterogeneity [11]. In addition, Gagneux et al. demonstrated that antibiotic resistance is typically associated with a fitness cost, which plays a decisive role in determining competitive outcomes between sensitive and resistant strains [12]. These findings provide fundamental biological insights into the mechanisms governing strain competition and persistence.



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Subsequently, some researchers further explored the dynamics of multi-strain epidemic models [13–17]. For example, Meskaf et al. developed a two-strain model with non-monotone incidence rates and established global stability results using Lyapunov methods, showing that behavioral responses and saturation effects can significantly modify transmission thresholds and dynamical structures [13]. Poonia et al. investigated the impact of drug adherence in HIV transmission and demonstrated that treatment compliance critically influences strain competition through its effect on effective transmission rates [15]. Moreover, Gavish revisited the classical competitive exclusion principle and showed that coexistence may arise even under strong competitive asymmetry [16], whereas Wang rigorously proved that, under standard assumptions, the strain with the larger basic reproduction number dominates, thereby confirming competitive exclusion [17].

In recent years, multi-strain epidemic models have increasingly incorporated more realistic biological mechanisms to capture the complex processes of strain competition and evolution [18–24]. For instance, Basaiti et al. systematically analyzed the combined effects of cross-immunity, waning immunity, and non-pharmaceutical interventions on strain competition dynamics [18]. Pell et al. introduced cross-immunity time delays and validated their model using wastewater surveillance data, thereby revealing the critical role of immune delay in strain replacement and coexistence dynamics [19]. Meanwhile, data-driven and evolutionary approaches have become an important complement to multi-strain modeling. Recent studies infer viral fitness and antigenic relationships from genomic surveillance data via strain frequency dynamics, thereby linking evolutionary processes with population-level transmission [20]. These developments provide a more comprehensive and realistic framework for understanding multi-strain epidemic dynamics.

At the same time, epidemic modeling generally involves two complementary research directions: data-driven parameter inference and qualitative dynamical analysis. While parameter estimation is essential for practical prediction and epidemic surveillance, theoretical analysis remains important for identifying threshold mechanisms, global stability properties, and qualitative dynamical structures that are independent of particular datasets. In particular, rigorous analytical results can reveal general mechanisms governing strain competition and persistence that may not be directly observable from numerical simulations alone. The present work mainly focuses on the latter perspective and aims to provide a dynamical systems analysis of strain competition in the presence of asymptomatic transmission.

Another important feature influencing epidemic dynamics is the presence of asymptomatic infections. Asymptomatic individuals may contribute substantially to disease transmission due to their hidden nature and large population size [25–27]. Related studies have shown that asymptomatic classes can sustain transmission even when symptomatic infections are limited, thereby altering threshold conditions and facilitating endemic persistence [28–30]. These findings highlight the necessity of explicitly incorporating asymptomatic transmission into epidemic models, particularly in multi-strain settings.

Despite these advances, relatively few studies have systematically investigated the combined effects of multi-strain competition and asymptomatic transmission from a nonlinear dynamical systems perspective. In particular, the emergence of non-isolated equilibria and degenerate structures remains insufficiently understood. From the viewpoint of dynamical systems theory, such features are closely related to non-hyperbolicity and may lead to qualitatively different long-term behaviors compared with classical epidemic models.

Motivated by these observations, we propose and analyze a two-strain SAIR epidemic model incorporating asymptomatic transmission. The population is divided into susceptible, asymptomatic infectious, symptomatic infectious, and recovered compartments, with two competing strains co-circulating. The two-strain framework is adopted as the minimal

nontrivial setting capable of capturing competitive exclusion, coexistence, and parameter degeneracy while remaining analytically tractable. Our aim is to characterize the global dynamical behavior of the system and to elucidate the mechanisms underlying strain competition and coexistence.

The main contributions of this work are summarized as follows. First, we derive the basic reproduction numbers for each strain and establish threshold conditions for disease invasion and persistence. Second, we show that, under certain parameter regimes, the system admits a continuum of endemic equilibria, revealing a degenerate structure associated with neutral competition. Third, by combining Lyapunov function techniques with center manifold theory, we rigorously analyze the local and global stability properties of the equilibrium set. Finally, numerical simulations and sensitivity analysis are conducted to illustrate the theoretical results and to assess the influence of key parameters on long-term dynamics.

The remainder of this paper is organized as follows. Section 2 presents the model formulation and basic properties. Section 3 analyzes the equilibria and their stability. Section 4 is devoted to sensitivity analysis. Section 5 provides numerical simulations to support the theoretical findings. Finally, Section 6 concludes the paper with a discussion and future research directions.

2. Model Description and Theoretical Analysis

2.1. Model Formulation

We consider a compartmental epidemic model describing the transmission dynamics of two competing pathogen strains in a homogeneous population. Let $N(t)$ denote the total population at time t . The population is divided into susceptible (S), asymptomatic infectious (A_i), symptomatic infectious (I_i), and recovered (R) compartments ($i = 1, 2$). Susceptible individuals become infected through effective contact with either asymptomatic or symptomatic infectious individuals carrying strain i . Upon infection, a proportion enters the asymptomatic class A_i , while the remainder progresses directly to the symptomatic class I_i . Individuals in the asymptomatic class may either develop symptoms or recover, whereas symptomatic individuals recover at strain-dependent rates. A schematic diagram of the model is shown in Figure 1.

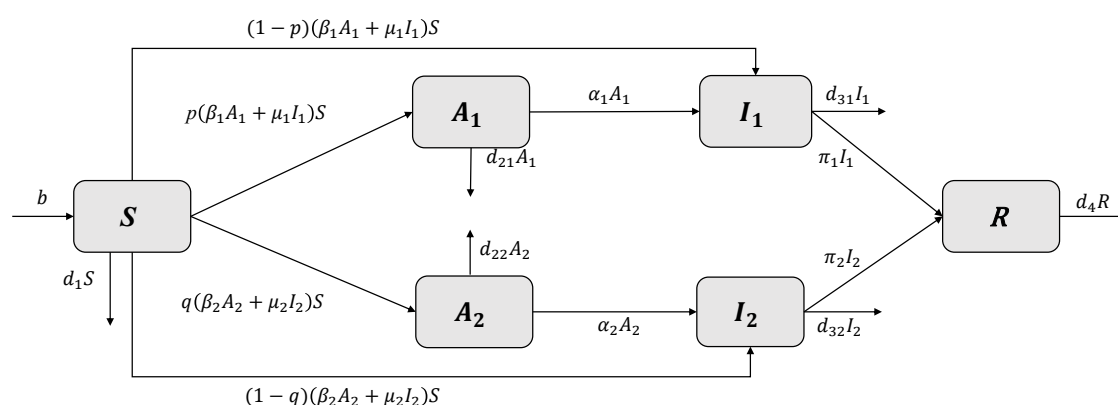


Figure 1. Schematic diagram of the two-strain SAIR epidemic model.

Based on the above assumptions, the model is governed by the following system, where all parameters and their biological interpretations are summarized in Table 1.

$$\begin{cases} \frac{dS}{dt} = b - (\beta_1 A_1 + \mu_1 I_1)S - (\beta_2 A_2 + \mu_2 I_2)S - d_1 S, \\ \frac{dA_1}{dt} = p(\beta_1 A_1 + \mu_1 I_1)S - (d_{21} + \alpha_1)A_1, \\ \frac{dI_1}{dt} = (1-p)(\beta_1 A_1 + \mu_1 I_1)S + \alpha_1 A_1 - (d_{31} + \pi_1)I_1, \\ \frac{dA_2}{dt} = q(\beta_2 A_2 + \mu_2 I_2)S - (d_{22} + \alpha_2)A_2, \\ \frac{dI_2}{dt} = (1-q)(\beta_2 A_2 + \mu_2 I_2)S + \alpha_2 A_2 - (d_{32} + \pi_2)I_2, \\ \frac{dR}{dt} = \pi_1 I_1 + \pi_2 I_2 - d_4 R. \end{cases} \quad (1)$$

Here, b denotes the recruitment rate of susceptible individuals. The parameters β_i and μ_i represent the transmission rates associated with asymptomatic and symptomatic infectious individuals of strain i , respectively. The parameter α_i denotes the progression rate from A_i to I_i , while π_i is the recovery rate of symptomatic individuals. The parameters p and q denote the proportions of newly infected individuals who enter the asymptomatic class for strains 1 and 2, respectively. Natural death rates are denoted by d_1 for susceptibles, d_{2i} for asymptomatic individuals, d_{3i} for symptomatic individuals, and d_4 for recovered individuals.

Table 1. Model parameters, descriptions, baseline values, and sources.

Parameter	Description	Value	Source
b	Recruitment rate of susceptible individuals	140	Assumed
β_1	Transmission rate from asymptomatic individuals (strain 1)	6.5×10^{-5}	Assumed
β_2	Transmission rate from asymptomatic individuals (strain 2)	6×10^{-5}	Assumed
μ_1	Transmission rate from symptomatic individuals (strain 1)	4.3×10^{-5}	[31]
μ_2	Transmission rate from symptomatic individuals (strain 2)	4×10^{-5}	[31]
p	Proportion entering asymptomatic class (strain 1)	0.3	[31]
q	Proportion entering asymptomatic class (strain 2)	0.3	[31]
α_1	Progression rate from A_1 to I_1	0.2	Assumed
α_2	Progression rate from A_2 to I_2	0.2	Assumed
π_1	Recovery rate of symptomatic individuals (strain 1)	0.75	[31]
π_2	Recovery rate of symptomatic individuals (strain 2)	0.7	Assumed
d_1	Natural death rate of susceptible individuals	0.007	Assumed
d_{21}	Natural death rate of asymptomatic individuals (strain 1)	0.007	Assumed
d_{22}	Natural death rate of asymptomatic individuals (strain 2)	0.007	Assumed
d_{31}	Natural death rate of symptomatic individuals (strain 1)	$0.007 + 7.79 \times 10^{-5}$	[32]
d_{32}	Natural death rate of symptomatic individuals (strain 2)	$0.007 + 7.79 \times 10^{-5}$	[32]
d_4	Natural death rate of recovered individuals	0.007	Assumed

2.2. Positivity of Solutions

We first show that all state variables of system (1) remain nonnegative.

Theorem 1. For any nonnegative initial conditions

$$S(0), A_1(0), I_1(0), A_2(0), I_2(0), R(0) \geq 0,$$

the solution of system (1) satisfies

$$S(t), A_1(t), I_1(t), A_2(t), I_2(t), R(t) \geq 0, \quad \forall t \geq 0.$$

Proof of Theorem 1. From the first equation of (1), we have $\frac{dS}{dt} = b - \Phi(t)S$, where

$$\Phi(t) = (\beta_1 A_1 + \mu_1 I_1) + (\beta_2 A_2 + \mu_2 I_2) + d_1 \geq 0.$$

By the comparison principle, it follows that $S(t) \geq 0$ for all $t \geq 0$.

Similarly, each of the remaining equations can be written in the form $\frac{dx}{dt} \geq -cx$, for some $c > 0$, which implies $x(t) \geq 0$ whenever $x(0) \geq 0$. Hence, all state variables remain nonnegative for all $t \geq 0$. $\square$

2.3. Invariant Region

Let $N(t) = S(t) + A_1(t) + A_2(t) + I_1(t) + I_2(t) + R(t)$ denote the total population. Summing all equations in (1), we obtain

$$\frac{dN}{dt} = b - d_1 S - d_{21} A_1 - d_{31} I_1 - d_{22} A_2 - d_{32} I_2 - d_4 R \leq b - \bar{d} N,$$

where $\bar{d} = \min\{d_1, d_{21}, d_{31}, d_{22}, d_{32}, d_4\}$. It follows that $\limsup_{t \rightarrow \infty} N(t) \leq b/\bar{d}$.

Moreover, from the first equation of (1),

$$\frac{dS}{dt} \leq b - d_1 S,$$

which yields

$$\limsup_{t \rightarrow \infty} S(t) \leq \frac{b}{d_1}.$$

Since the recovered class R does not affect the first five equations, the dynamics can be reduced to the following subsystem:

$$\begin{cases} \frac{dS}{dt} = b - (\beta_1 A_1 + \mu_1 I_1)S - (\beta_2 A_2 + \mu_2 I_2)S - d_1 S, \\ \frac{dA_1}{dt} = p(\beta_1 A_1 + \mu_1 I_1)S - (d_{21} + \alpha_1)A_1, \\ \frac{dI_1}{dt} = (1-p)(\beta_1 A_1 + \mu_1 I_1)S + \alpha_1 A_1 - (d_{31} + \pi_1)I_1, \\ \frac{dA_2}{dt} = q(\beta_2 A_2 + \mu_2 I_2)S - (d_{22} + \alpha_2)A_2, \\ \frac{dI_2}{dt} = (1-q)(\beta_2 A_2 + \mu_2 I_2)S + \alpha_2 A_2 - (d_{32} + \pi_2)I_2. \end{cases} \quad (2)$$

Therefore, the feasible region is defined as

$$\Gamma = \left\{ (S, A_1, I_1, A_2, I_2) \in \mathbb{R}_+^5 \mid S \leq \frac{b}{d_1}, N \leq \frac{b}{\bar{d}} \right\}.$$

It is straightforward to verify that Γ is positively invariant for system (2).

2.4. Equilibria and the Basic Reproduction Number

2.4.1. Disease-Free Equilibrium

System (2) always admits a unique disease-free equilibrium (DFE), denoted by P_0 , located on the boundary of the feasible region Γ . It is given by

$$P_0 = (S_0, 0, 0, 0, 0), \quad \text{with} \quad S_0 = \frac{b}{d_1}.$$

2.4.2. Basic Reproduction Number

Following the next-generation matrix approach [33], we consider the infected state vector $X = (A_1, I_1, A_2, I_2)^T$. Let F and V denote the matrices of new infection terms and tran-

sition terms, respectively. Then, the basic reproduction number is given by $\mathcal{R}_0 = \rho(FV^{-1})$, where $\rho(\cdot)$ denotes the spectral radius. Due to the block structure of the system, $\mathcal{R}_0$ can be decomposed as $\mathcal{R}_0 = \max\{\mathcal{R}_{01}, \mathcal{R}_{02}\}$, where

$$\mathcal{R}_{01} = \left[\frac{p\beta_1}{d_{21} + \alpha_1} + \frac{p\mu_1\alpha_1}{(d_{31} + \pi_1)(d_{21} + \alpha_1)} + \frac{(1-p)\mu_1}{d_{31} + \pi_1} \right] \frac{b}{d_1},$$

$$\mathcal{R}_{02} = \left[\frac{q\beta_2}{d_{22} + \alpha_2} + \frac{q\mu_2\alpha_2}{(d_{32} + \pi_2)(d_{22} + \alpha_2)} + \frac{(1-q)\mu_2}{d_{32} + \pi_2} \right] \frac{b}{d_1}.$$

Here, $\mathcal{R}_{01}$ and $\mathcal{R}_{02}$ correspond to the reproduction numbers of strain 1 (drug-sensitive) and strain 2 (drug-resistant), respectively.

2.4.3. Endemic Equilibria

Theorem 2. *The existence of endemic equilibria of system (2) is characterized as follows:*

(i) *If $\mathcal{R}_{01} > \max\{1, \mathcal{R}_{02}\}$, then there exists a unique strain-1 endemic equilibrium*

$$P_1^* = (S_1^*, A_{11}^*, I_{11}^*, 0, 0).$$

(ii) *If $\mathcal{R}_{02} > \max\{1, \mathcal{R}_{01}\}$, then there exists a unique strain-2 endemic equilibrium*

$$P_2^* = (S_2^*, 0, 0, A_{22}^*, I_{22}^*).$$

(iii) *If $\mathcal{R}_{01} = \mathcal{R}_{02} = \mathcal{R}_0 > 1$, then system (2) admits a continuum of coexistence equilibria*

$$\mathcal{E}_* = \left\{ P_3^*(S_3^*, A_{13}^*, I_{13}^*, A_{23}^*, I_{23}^*) : \begin{array}{l} S_3^* = \frac{b}{d_1 \mathcal{R}_0}, \\ I_{13}^* = \frac{(1-p)(d_{21} + \alpha_1) + p\alpha_1}{p(d_{31} + \pi_1)} A_{13}^*, \\ I_{23}^* = \frac{(1-q)(d_{22} + \alpha_2) + q\alpha_2}{q(d_{32} + \pi_2)} A_{23}^*, \\ C_1 A_{13}^* + C_2 A_{23}^* = d_1(\mathcal{R}_0 - 1), \\ A_{13}^*, A_{23}^* \geq 0 \end{array} \right\}.$$

where

$$C_1 = \beta_1 + \mu_1 \frac{(1-p)(d_{21} + \alpha_1) + p\alpha_1}{p(d_{31} + \pi_1)},$$

and

$$C_2 = \beta_2 + \mu_2 \frac{(1-q)(d_{22} + \alpha_2) + q\alpha_2}{q(d_{32} + \pi_2)}.$$

Consequently, $\mathcal{E}_*$ forms a one-dimensional smooth manifold in the feasible region.

Proof of Theorem 2. (i) At the equilibrium P_1^* , all variables associated with strain 2 vanish. Solving the reduced subsystem yields

$$S_1^* = \frac{b}{d_1 \mathcal{R}_{01}}, \quad A_{11}^* = \frac{pb}{d_{21} + \alpha_1} \left(1 - \frac{1}{\mathcal{R}_{01}} \right),$$

$$I_{11}^* = \frac{((1-p)(d_{21} + \alpha_1) + p\alpha_1)b}{(d_{31} + \pi_1)(d_{21} + \alpha_1)} \left(1 - \frac{1}{\mathcal{R}_{01}} \right).$$

These quantities are positive if and only if $\mathcal{R}_{01} > 1$. Moreover, the invasion reproduction number of strain 2 satisfies $\mathcal{R}_{02} < 1$, which ensures that strain 2 cannot persist. Hence, the condition $\mathcal{R}_{01} > \max\{1, \mathcal{R}_{02}\}$ guarantees the existence of P_1^* .

(ii) The existence of P_2^* follows analogously by symmetry.

(iii) For coexistence equilibria, assume that both strains persist, namely $A_{13}^* > 0$, $I_{13}^* > 0$, $A_{23}^* > 0$, $I_{23}^* > 0$. At equilibrium, from system (2), we obtain $S_3^* = \frac{b}{d_1 \mathcal{R}_0}$, where $\mathcal{R}_{01} = \mathcal{R}_{02} = \mathcal{R}_0 > 1$. Moreover, the equilibrium relations imply

$$I_{13}^* = \frac{(1-p)(d_{21} + \alpha_1) + p\alpha_1}{p(d_{31} + \pi_1)} A_{13}^*,$$

and

$$I_{23}^* = \frac{(1-q)(d_{22} + \alpha_2) + q\alpha_2}{q(d_{32} + \pi_2)} A_{23}^*.$$

Setting the right-hand sides of system (2) to zero leads to a linear system in A_{13}^* and A_{23}^* :

$$\begin{cases} d_1(\mathcal{R}_0 - 1) = C_1 A_{13}^* + C_2 A_{23}^*, \\ b\left(1 - \frac{1}{\mathcal{R}_0}\right) = D_1 A_{13}^* + D_2 A_{23}^*, \end{cases}$$

where

$$C_1 = \beta_1 + \mu_1 \frac{(1-p)(d_{21} + \alpha_1) + p\alpha_1}{p(d_{31} + \pi_1)}, \quad C_2 = \beta_2 + \mu_2 \frac{(1-q)(d_{22} + \alpha_2) + q\alpha_2}{q(d_{32} + \pi_2)},$$

$$D_1 = \frac{d_{21} + \alpha_1}{p}, \quad D_2 = \frac{d_{22} + \alpha_2}{q}.$$

When $\mathcal{R}_{01} = \mathcal{R}_{02} = \mathcal{R}_0 > 1$, the coefficient matrix becomes singular with rank one. Therefore, the system admits infinitely many solutions, forming a one-dimensional continuum of equilibria. $\square$

The qualitative dynamical structure in the (R_{01}, R_{02}) parameter space is illustrated in Figure 2. In particular, competitive exclusion occurs when $R_{01} \neq R_{02}$, while the degenerate case $R_{01} = R_{02} = \mathcal{R}_0 > 1$ gives rise to the continuum of equilibria.

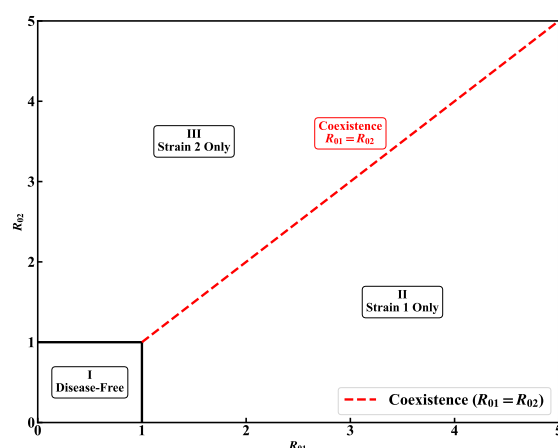


Figure 2. Qualitative dynamical regimes of the system in the (R_{01}, R_{02}) plane. Competitive exclusion occurs when $R_{01} \neq R_{02}$, while the degenerate case $R_{01} = R_{02} = \mathcal{R}_0 > 1$ leads to coexistence and a continuum of equilibria.

3. Global Dynamical Analysis

3.1. Stability of the Disease-Free Equilibrium

We first investigate the stability of the disease-free equilibrium P_0 .

Theorem 3. If $\mathcal{R}_0 < 1$, then the disease-free equilibrium P_0 of system (2) is locally asymptotically stable in Γ . If $\mathcal{R}_0 > 1$, it is unstable.

Proof of Theorem 3. The Jacobian matrix of system (2) at P_0 is given by

$$J(P_0) = \begin{pmatrix} -d_1 & -\beta_1 S_0 & -\mu_1 S_0 & -\beta_2 S_0 & -\mu_2 S_0 \\ 0 & p\beta_1 S_0 - \hat{d}_1 & p\mu_1 S_0 & 0 & 0 \\ 0 & (1-p)\beta_1 S_0 + \alpha_1 & (1-p)\mu_1 S_0 - \hat{d}_2 & 0 & 0 \\ 0 & 0 & 0 & q\beta_2 S_0 - \hat{d}_3 & q\mu_2 S_0 \\ 0 & 0 & 0 & (1-q)\beta_2 S_0 + \alpha_2 & (1-q)\mu_2 S_0 - \hat{d}_4 \end{pmatrix},$$

where $\hat{d}_1 = d_{21} + \alpha_1$, $\hat{d}_2 = d_{31} + \pi_1$, $\hat{d}_3 = d_{22} + \alpha_2$, $\hat{d}_4 = d_{32} + \pi_2$. It is clear that one eigenvalue is $\lambda_1 = -d_1 < 0$. The remaining eigenvalues are determined by two decoupled 2×2 blocks corresponding to the two strains:

$$C = \begin{pmatrix} p\beta_1 S_0 - \hat{d}_1 & p\mu_1 S_0 \\ (1-p)\beta_1 S_0 + \alpha_1 & (1-p)\mu_1 S_0 - \hat{d}_2 \end{pmatrix}, \quad D = \begin{pmatrix} q\beta_2 S_0 - \hat{d}_3 & q\mu_2 S_0 \\ (1-q)\beta_2 S_0 + \alpha_2 & (1-q)\mu_2 S_0 - \hat{d}_4 \end{pmatrix}.$$

For matrix C , direct computation yields $\det(C) = \hat{d}_1 \hat{d}_2 (1 - \mathcal{R}_{01})$, $\text{tr}(C) < 0$ if $\mathcal{R}_{01} < 1$. Similarly, $\det(D) = \hat{d}_3 \hat{d}_4 (1 - \mathcal{R}_{02})$, $\text{tr}(D) < 0$ if $\mathcal{R}_{02} < 1$. Thus, all eigenvalues have negative real parts if and only if $\mathcal{R}_0 = \max\{\mathcal{R}_{01}, \mathcal{R}_{02}\} < 1$. The result follows from the Hurwitz criterion. $\square$

Theorem 4. If $\mathcal{R}_0 < 1$, then the disease-free equilibrium P_0 is globally asymptotically stable in Γ .

Proof of Theorem 4. Consider the Lyapunov function

$$L_0 = a_1 A_1 + a_2 I_1 + a_3 A_2 + a_4 I_2,$$

where

$$a_1 = \frac{\beta_1}{d_{21} + \alpha_1} + \frac{\mu_1 \alpha_1}{(d_{21} + \alpha_1)(d_{31} + \pi_1)}, \quad a_2 = \frac{\mu_1}{d_{31} + \pi_1},$$

$$a_3 = \frac{\beta_2}{d_{22} + \alpha_2} + \frac{\mu_2 \alpha_2}{(d_{22} + \alpha_2)(d_{32} + \pi_2)}, \quad a_4 = \frac{\mu_2}{d_{32} + \pi_2}.$$

Differentiating L_0 along solutions gives

$$\frac{dL_0}{dt} = (\beta_1 A_1 + \mu_1 I_1) \left(\frac{\mathcal{R}_{01} d_1 S}{b} - 1 \right) + (\beta_2 A_2 + \mu_2 I_2) \left(\frac{\mathcal{R}_{02} d_1 S}{b} - 1 \right).$$

Since $S \leq \frac{b}{d_1}$ in Γ , it follows that $\frac{dL_0}{dt} \leq 0$ whenever $\mathcal{R}_0 < 1$. Moreover, $\frac{dL_0}{dt} = 0$ if and only if $A_1 = I_1 = A_2 = I_2 = 0$. By LaSalle's invariance principle, the solution converges to P_0 . Hence, P_0 is globally asymptotically stable. $\square$

3.2. Stability of the Boundary Endemic Equilibrium

Theorem 5. If $\mathcal{R}_{01} > \max\{1, \mathcal{R}_{02}\}$, then the strain-1 endemic equilibrium P_1^* is globally asymptotically stable in Γ .

Proof of Theorem 5. Consider the Lyapunov function

$$L_1 = x_1 \left(S - S_1^* \ln \frac{S}{S_1^*} \right) + x_2 \left(A_1 - A_{11}^* \ln \frac{A_1}{A_{11}^*} \right) + x_3 \left(I_1 - I_{11}^* \ln \frac{I_1}{I_{11}^*} \right) + x_4 A_2 + x_5 I_2,$$

where $x_i > 0$ ($i = 1, \dots, 5$). Differentiating L_1 along solutions of system (2), we obtain

$$\frac{dL_1}{dt} = V_{11} + V_{12} + V_{13},$$

where

$$V_{11} = d_1 S_1^* \left(2 - \frac{S_1^*}{S} - \frac{S}{S_1^*} \right) \leq 0,$$

$$V_{12} = -\frac{S_1^{*2}}{S} (\beta_1 A_{11}^* + \mu_1 I_{11}^*) - x_2 \frac{S}{A_1} p A_{11}^* (\beta_1 A_1 + \mu_1 I_1) \\ - x_3 \left[\frac{S}{I_1} (1-p) I_{11}^* (\beta_1 A_1 + \mu_1 I_1) - \frac{A_1}{I_1} I_{11}^* \alpha_1 \right],$$

$$V_{13} = (\beta_1 A_{11}^* + \mu_1 I_{11}^*) S_1^* + x_2 A_{11}^* (d_{21} + \alpha_1) + x_3 (d_{31} + \pi_1) I_{11}^*.$$

The coefficients x_i are chosen such that

$$\begin{cases} -x_1 + px_2 + (1-p)x_3 = 0, \\ -x_1 + qx_4 + (1-q)x_5 = 0, \\ x_1 \beta_1 S_1^* - x_2 (d_{21} + \alpha_1) + x_3 \alpha_1 = 0, \\ x_1 \mu_1 S_1^* - x_3 (d_{31} + \pi_1) = 0, \\ x_1 \beta_2 S_1^* - x_4 (d_{22} + \alpha_2) + x_5 \alpha_2 = 0, \\ x_1 \mu_2 S_1^* - x_5 (d_{32} + \pi_2) = 0. \end{cases} \quad (3)$$

A direct computation yields the positive solution

$$x_1 = 1, \quad x_2 = \frac{(d_{31} + \pi_1) \beta_1 S_1^* + \mu_1 \alpha_1 S_1^*}{(d_{31} + \pi_1)(d_{21} + \alpha_1)}, \quad x_3 = \frac{\mu_1 S_1^*}{d_{31} + \pi_1}, \\ x_4 = \frac{(d_{32} + \pi_2) \beta_2 S_1^* + \mu_2 \alpha_2 S_1^*}{(d_{32} + \pi_2)(d_{22} + \alpha_2)}, \quad x_5 = \frac{\mu_2 S_1^*}{d_{32} + \pi_2}.$$

Using the relation $-x_1 + px_2 + (1-p)x_3 = 0$, the term V_{12} can be rearranged as

$$V_{12} = \left[-px_2 \frac{S_1^{*2}}{S} \beta_1 A_{11}^* - x_2 Sp \beta_1 A_{11}^* \right] \\ + \left[-(1-p)x_3 \frac{S_1^{*2}}{S} \mu_1 I_{11}^* - x_3 S(1-p) \mu_1 I_{11}^* \right] \\ + \left[-upx_2 \frac{S_1^{*2}}{S} \mu_1 I_{11}^* - ux_2 \frac{SI_1}{A_1} p \mu_1 A_{11}^* - x_3 \frac{A_1}{I_1} I_{11}^* \alpha_1 \right] \\ + \left[-(1-p)x_3 \frac{S_1^{*2}}{S} \beta_1 A_{11}^* - x_3 \frac{SA_1}{I_1} (1-p) I_{11}^* \beta_1 \right] \\ + \left[-(1-u)x_2 \frac{SI_1}{A_1} p \mu_1 A_{11}^* - (1-u)px_2 \frac{S_1^{*2}}{S} \mu_1 I_{11}^* \right] \\ \leq -2x_2 Sp \beta_1 A_{11}^* - 2x_3 S(1-p) \mu_1 I_{11}^* - 3(u^2 p^2 x_2^2 x_3 \mu_1^2 I_{11}^{*2} A_{11}^* \alpha_1 S_1^{*2})^{1/3} \\ - 4 \left[(1-u)^2 p^2 (1-p)^2 x_2^2 x_3^2 \mu_1^2 \beta_1^2 I_{11}^{*2} A_{11}^{*2} S_1^{*2} \right]^{1/4},$$

where

$$u = \frac{\alpha_1}{[(1-p)d_{21} + \alpha_1]x_2}, \quad 1-u = \frac{(1-p)\beta_1 S_1^*}{[(1-p)d_{21} + \alpha_1]x_2}.$$

Similarly, we further obtain

$$V_{13} = 2px_2\beta_1 A_{11}^* S_1^* + 2(1-p)x_3\mu_1 I_{11}^* S_1^* + 3x_3\alpha_1 A_{11}^* + 4(1-p)x_3\beta_1 A_{11}^* S_1^*,$$

By the arithmetic mean–geometric mean inequality,

$$a_1 + a_2 + \cdots + a_n \geq n(a_1 a_2 \cdots a_n)^{1/n}, \quad a_i > 0,$$

we obtain $V_{12} + V_{13} \leq 0$. Therefore, $\frac{dL_1}{dt} \leq 0$ in Γ , and equality holds if and only if $(S, A_1, I_1, A_2, I_2) = (S_1^*, A_{11}^*, I_{11}^*, 0, 0)$. Hence, the largest invariant set contained in $\{x \in \Gamma : \frac{dL_1}{dt} = 0\}$ is the singleton $\{P_1^*\}$. By LaSalle's invariance principle, P_1^* is globally asymptotically stable. $\square$

Theorem 6. If $\mathcal{R}_{02} > \max\{1, \mathcal{R}_{01}\}$, then the drug-resistant strain endemic equilibrium $P_2^* = (S_2^*, 0, 0, A_{22}^*, I_{22}^*)$ is globally asymptotically stable in the feasible region Γ .

Proof of Theorem 6. Consider the Lyapunov function

$$L_2 = x_1 \left(S - S_2^* \ln \frac{S}{S_2^*} \right) + x_6 A_1 + x_7 I_1 + x_8 \left(A_2 - A_{22}^* \ln \frac{A_2}{A_{22}^*} \right) + x_9 \left(I_2 - I_{22}^* \ln \frac{I_2}{I_{22}^*} \right),$$

where $x_i > 0$. Differentiating L_2 along the solutions of system (2), we obtain

$$\frac{dL_2}{dt} = V_{21} + V_{22} + V_{23},$$

where

$$V_{21} = d_1 S_2^* \left(2 - \frac{S_2^*}{S} - \frac{S}{S_2^*} \right) \leq 0,$$

$$V_{22} = -\frac{S_2^{*2}}{S} (\beta_2 A_{22}^* + \mu_2 I_{22}^*) - x_8 \frac{S}{A_2} q A_{22}^* (\beta_2 A_2 + \mu_2 I_2) \\ - x_9 \left[\frac{S}{I_2} (1-q) I_{22}^* (\beta_2 A_2 + \mu_2 I_2) - \frac{A_2}{I_2} I_{22}^* \alpha_2 \right],$$

$$V_{23} = (\beta_2 A_{22}^* + \mu_2 I_{22}^*) S_2^* + x_8 A_{22}^* (d_{22} + \alpha_2) + x_9 (d_{32} + \pi_2) I_{22}^*.$$

The coefficients are chosen such that

$$\begin{cases} -x_1 + px_6 + (1-p)x_7 = 0, \\ -x_1 + qx_8 + (1-q)x_9 = 0, \\ x_1\beta_1 S_2^* - x_6(d_{21} + \alpha_1) + x_7\alpha_1 = 0, \\ x_1\mu_1 S_2^* - x_7(d_{31} + \pi_1) = 0, \\ x_1\beta_2 S_2^* - x_8(d_{22} + \alpha_2) + x_9\alpha_2 = 0, \\ x_1\mu_2 S_2^* - x_9(d_{32} + \pi_2) = 0. \end{cases} \quad (4)$$

A direct computation gives

$$x_6 = \frac{(d_{31} + \pi_1)\beta_1 S_2^* + \mu_1 \alpha_1 S_2^*}{(d_{31} + \pi_1)(d_{21} + \alpha_1)}, \quad x_7 = \frac{\mu_1 S_2^*}{d_{31} + \pi_1},$$

$$x_8 = \frac{(d_{32} + \pi_2)\beta_2 S_2^* + \mu_2 \alpha_2 S_2^*}{(d_{32} + \pi_2)(d_{22} + \alpha_2)}, \quad x_9 = \frac{\mu_2 S_2^*}{d_{32} + \pi_2}.$$

Using the relation $qx_8 + (1 - q)x_9 = x_1$, the term V_{22} can be rewritten as

$$\begin{aligned} V_{22} &= \left[-qx_8 \frac{S_2^{*2}}{S} \beta_2 A_{22}^* - x_8 Sq \beta_2 A_{22}^* \right] + \left[-(1 - q)x_9 \frac{S_2^{*2}}{S} \mu_2 I_{22}^* - x_9 S(1 - q) \mu_2 I_{22}^* \right] \\ &\quad + \left[-u^* qx_8 \frac{S_2^{*2}}{S} \mu_2 I_{22}^* - u^* x_8 \frac{SI_2}{A_2} q \mu_2 A_{22}^* - x_9 \frac{A_2}{I_2} I_{22}^* \alpha_2 \right] \\ &\quad + \left[-(1 - q)x_9 \frac{S_2^{*2}}{S} \beta_2 A_{22}^* - x_9 \frac{SA_2}{I_2} (1 - q) I_{22}^* \beta_2 \right] \\ &\quad + \left[-(1 - u^*) x_8 \frac{SI_2}{A_2} p \mu_2 A_{22}^* - (1 - u^*) qx_8 \frac{S_2^{*2}}{S} \mu_2 I_{22}^* \right] \\ &\leq -2x_8 Sq \beta_2 A_{22}^* - 2x_9 S(1 - q) \mu_2 I_{22}^* - 3 \left(u^{*2} q^2 x_8^2 x_9 \mu_2^2 I_{22}^{*2} A_{22}^* \alpha_2 S_2^{*2} \right)^{1/3} \\ &\quad - 4 \left[(1 - u^*)^2 q^2 (1 - q)^2 x_8^2 x_9^2 \mu_2^3 \beta_2^2 I_{22}^{*2} A_{22}^* S_2^{*2} \right]^{1/4}, \end{aligned}$$

where

$$u^* = \frac{\alpha_2}{[(1 - q)d_{22} + \alpha_2]x_8}, \quad 1 - u^* = \frac{(1 - q)\beta_2 S_2^*}{[(1 - q)d_{22} + \alpha_2]x_8}.$$

Similarly, using the balance relations (4), V_{23} can be further simplified as

$$V_{23} = 2qx_8 \beta_2 A_{22}^* S_2^* + 2(1 - q)x_9 \mu_2 I_{22}^* S_2^* + 3x_9 \alpha_2 A_{22}^* + 4(1 - q)x_9 \beta_2 A_{22}^* S_2^*.$$

Consequently, we obtain $V_{22} + V_{23} \leq 0$, which implies $\frac{dL_2}{dt} \leq 0$ in Γ . Moreover, $\frac{dL_2}{dt} = 0$ if and only if $(S, A_1, I_1, A_2, I_2) = (S_2^*, 0, 0, A_{22}^*, I_{22}^*)$. Therefore, the largest invariant set contained in $\left\{ x \in \Gamma : \frac{dL_2}{dt} = 0 \right\}$ is the singleton $\{P_2^*\}$. By LaSalle's invariance principle, P_2^* is globally asymptotically stable. $\square$

3.3. Stability of the Coexistence Equilibrium Set

In this subsection, we analyze the local stability of the coexistence endemic equilibrium set of system (2). The analysis relies on center manifold theory due to the presence of a zero eigenvalue.

3.3.1. Preliminary Lemmas

We first recall several classical results from invariant manifold theory.

Lemma 1 ([34]). Consider the dynamical system

$$\frac{dx}{dt} = f(x), \quad x \in \mathbb{R}^n,$$

where f is sufficiently smooth and $f(0) = 0$. Let $\lambda_1, \dots, \lambda_n$ be the eigenvalues of the Jacobian matrix $J = Df(0)$. Define

$$\sigma_s = \{\lambda_i : \operatorname{Re}(\lambda_i) < 0\}, \quad \sigma_u = \{\lambda_i : \operatorname{Re}(\lambda_i) > 0\}, \quad \sigma_c = \{\lambda_i : \operatorname{Re}(\lambda_i) = 0\}.$$

Then there exist locally invariant manifolds tangent to the corresponding eigenspaces, namely the stable manifold W^s , unstable manifold W^u , and center manifold W^c .

Lemma 2 ([35]). Consider the system

$$\begin{cases} \frac{dx}{dt} = Ax + f(x, y), & x \in T^c, \\ \frac{dy}{dt} = By + g(x, y), & y \in T^s, \end{cases}$$

where $f(0, 0) = 0$, $g(0, 0) = 0$, and $Df(0, 0) = Dg(0, 0) = 0$. Then there exists a locally invariant center manifold W^c tangent to T^c at the origin, which can be expressed as

$$W^c = \{(x, y) \in T^c \times T^s : y = h(x)\},$$

where $h(0) = 0$ and $Dh(0) = 0$. Moreover, the dynamics restricted to the center manifold are governed by the reduced system

$$\frac{du}{dt} = Au + f(u, h(u)), \quad u \in T^c.$$

Lemma 3 ([34]). Let T^{su} denote the $(n - 1)$ -dimensional eigenspace associated with all nonzero eigenvalues of J . Then $y \in T^{su}$ if and only if

$$\langle l, y \rangle = 0,$$

where l is the left eigenvector corresponding to the zero eigenvalue of J .

3.3.2. Jacobian Matrix and Spectral Structure

Let P_3^* be a coexistence equilibrium with $\mathcal{R}_{01} = \mathcal{R}_{02} = \mathcal{R}_0 > 1$. Denote

$$t_3 = \beta_1 A_{13}^* + \mu_1 I_{13}^*, \quad t_4 = \beta_2 A_{23}^* + \mu_2 I_{23}^*.$$

The Jacobian matrix $J(P_3^*)$ is given by

$$J(P_3^*) = \begin{pmatrix} -d_1 \mathcal{R}_0 & -\beta_1 S^* & -\mu_1 S^* & -\beta_2 S^* & -\mu_2 S^* \\ pt_3 & p\beta_1 S^* - \hat{d}_1 & p\mu_1 S^* & 0 & 0 \\ (1-p)t_3 & (1-p)\beta_1 S^* + \alpha_1 & (1-p)\mu_1 S^* - \hat{d}_2 & 0 & 0 \\ qt_4 & 0 & 0 & q\beta_2 S^* - \hat{d}_3 & q\mu_2 S^* \\ (1-q)t_4 & 0 & 0 & J_{54}^* & J_{55}^* \end{pmatrix}.$$

where

$$J_{54}^* = (1-q)\beta_2 S^* + \alpha_2, \quad J_{55}^* = (1-q)\mu_2 S^* - \hat{d}_4.$$

A direct computation shows that the characteristic polynomial can be factorized as

$$\det(\lambda I - J(P_3^*)) = \lambda P_2(\lambda) Q_2(\lambda),$$

where

$$P_2(\lambda) = \lambda^2 + a_1 \lambda + a_2, \quad Q_2(\lambda) = \lambda^2 + b_1 \lambda + b_2,$$

with coefficients

$$\begin{aligned} a_1 &= \hat{d}_1 + \hat{d}_2 - S^*(p\beta_1 + (1-p)\mu_1), \\ a_2 &= \hat{d}_1 \hat{d}_2 - S^*(\hat{d}_2 p\beta_1 + \hat{d}_1 (1-p)\mu_1), \\ b_1 &= \hat{d}_3 + \hat{d}_4 - S^*(q\beta_2 + (1-q)\mu_2), \\ b_2 &= \hat{d}_3 \hat{d}_4 - S^*(\hat{d}_4 q\beta_2 + \hat{d}_3 (1-q)\mu_2). \end{aligned}$$

It follows immediately that $\lambda = 0$ is an eigenvalue of $J(P_3^*)$. Moreover, under the condition

$\mathcal{R}_{01} = \mathcal{R}_{02} = \mathcal{R}_0 > 1$, the coexistence equilibrium satisfies $S^* = \frac{b}{\hat{d}_1 \mathcal{R}_0}$. Using the definition of $\mathcal{R}_{01}$, we obtain

$$1 = S^* \left[\frac{p\beta_1}{\hat{d}_1} + \frac{p\mu_1\alpha_1}{\hat{d}_1\hat{d}_2} + \frac{(1-p)\mu_1}{\hat{d}_2} \right].$$

Multiplying both sides by $\hat{d}_1\hat{d}_2$ gives

$$\hat{d}_1\hat{d}_2 = S^* (\hat{d}_2 p\beta_1 + p\mu_1\alpha_1 + \hat{d}_1(1-p)\mu_1).$$

Hence, $a_2 = \hat{d}_1\hat{d}_2 - S^* (\hat{d}_2 p\beta_1 + \hat{d}_1(1-p)\mu_1) = S^* p\mu_1\alpha_1 > 0$.

Next, we verify the positivity of a_1 . Since

$$\hat{d}_1\hat{d}_2 = S^* (\hat{d}_2 p\beta_1 + p\mu_1\alpha_1 + \hat{d}_1(1-p)\mu_1) > S^* (\hat{d}_2 p\beta_1 + \hat{d}_1(1-p)\mu_1).$$

Dividing both sides by $\hat{d}_1\hat{d}_2 > 0$ yields

$$1 > S^* \left(\frac{p\beta_1}{\hat{d}_1} + \frac{(1-p)\mu_1}{\hat{d}_2} \right).$$

Since

$$\frac{1}{\hat{d}_1} > \frac{1}{\hat{d}_1 + \hat{d}_2}, \quad \frac{1}{\hat{d}_2} > \frac{1}{\hat{d}_1 + \hat{d}_2},$$

we further obtain

$$1 > S^* \frac{p\beta_1 + (1-p)\mu_1}{\hat{d}_1 + \hat{d}_2},$$

which implies $S^* (p\beta_1 + (1-p)\mu_1) < \hat{d}_1 + \hat{d}_2$.

Similarly, one can prove that $b_1 > 0, b_2 > 0$. Therefore, by the Routh–Hurwitz criterion, all roots of $P_2(\lambda) = 0$ and $Q_2(\lambda) = 0$ have strictly negative real parts. Consequently, the Jacobian matrix $J(P_3^*)$ admits a simple zero eigenvalue, while all remaining eigenvalues lie in the open left half-plane.

Hence, the coexistence equilibrium P_3^* is non-hyperbolic with a one-dimensional center subspace and a four-dimensional stable subspace.

3.3.3. Main Result

Theorem 7. *If $\mathcal{R}_{01} = \mathcal{R}_{02} = \mathcal{R}_0 > 1$, then the coexistence endemic equilibrium P_3^* of System (2) is locally stable in the feasible region Γ .*

Proof of Theorem 7.

Step 1. Center manifold reduction

Let $x = (S, A_1, I_1, A_2, I_2)^T$ and denote by x^* the coexistence equilibrium. Let $J(x^*)$ be the Jacobian matrix at x^* . Under the condition $\mathcal{R}_{01} = \mathcal{R}_{02} = \mathcal{R}_0 > 1$, it follows from Lemmas 1 and 2 that $J(x^*)$ admits a simple zero eigenvalue, while all other eigenvalues have strictly negative real parts. Hence, there exists a one-dimensional center manifold W^c in a neighborhood of x^* , which can be expressed as

$$W^c(x^*) = \{(x, y) \in T^c \times T^s : y = \omega(x), |x - x^*| < \delta, \omega(x^*) = x^*, D\omega(x^*) = 0\},$$

Let r and l be the right and left eigenvectors associated with the zero eigenvalue of $J(x^*)$, respectively.

The right eigenvector r is obtained from $J(x^*)r = 0$. Choosing $r_1 = 0$ and normalizing by $r_2 = 1$, we obtain

$$r = \left(0, 1, \frac{(1-p)d_{21} + \alpha_1}{p(\pi_1 + d_{31})}, -\frac{q(d_{21} + \alpha_1)}{p(d_{22} + \alpha_2)}, -\frac{(d_{21} + \alpha_1)}{p(d_{22} + \alpha_2)} \cdot \frac{(1-q)d_{22} + \alpha_2}{\pi_2 + d_{32}} \right)^T.$$

The left eigenvector l is determined by $l^T J(x^*) = 0$. Choosing $l_1 = 0$ and $l_2 = 1$, we obtain $l = (0, 1, l_3, l_4, l_5)^T$, where

$$l_3 = \frac{p\beta_1 S_3^* - \hat{d}_1}{(1-p)\beta_1 S_3^* + \alpha_1}, l_4 = -\frac{\left[p + (1-p)l_3 \right] t_3}{\left[q + (1-q) \frac{q\beta_2 S_3^* - \hat{d}_3}{(1-q)\beta_2 S_3^* + \alpha_2} \right] t_4}, l_5 = l_3 \cdot l_4.$$

Then, let $c = l^T r \neq 0$. According to Lemma 3, the deviation of the state variable from the equilibrium can be decomposed as

$$x = x^* + \frac{1}{c}ur + \omega(u).$$

Here, $u = l^T(x - x^*)$ represents the projection of $(x - x^*)$ onto the critical direction, while $ur \in T^c$, $\omega(u) \in T^s$, and the function $\omega(u)$ satisfies $\omega(0) = 0$, $D\omega(0) = 0$. Thus, the pair $(u, \omega(u))$ defines a local coordinate system in a neighborhood of x^* .

Since $\omega(u)$ can be expanded around $u = 0$ as $\omega(u) = h_2 u^2 + h_3 u^3 + O(u^4)$, by Lemma 2, the dynamics of System (2) restricted to the center manifold are governed by the reduced scalar equation

$$\frac{du}{dt} = l^T \frac{dx}{dt}.$$

Step 2. Derivation of the reduced equation

We now determine the dynamics of the reduced system. To this end, we expand the vector field $f(x)$ in a neighborhood of x^* :

$$\frac{dx}{dt} = f(x) = f(x^*) + J(x^*)(x - x^*) + G(x) = J(x^*)(x - x^*) + G(x),$$

where $G(x)$ collects all higher-order nonlinear terms and satisfies

$$G(x) = \left(\frac{dS}{dt}, \frac{dA_1}{dt}, \frac{dI_1}{dt}, \frac{dA_2}{dt}, \frac{dI_2}{dt} \right)^T - J(x^*)(S - S^*, A_1 - A_{13}^*, I_1 - I_{13}^*, A_2 - A_{23}^*, I_2 - I_{23}^*)^T,$$

By Taylor expansion, we have

$$G(x) = G(x^*) + DG(x^*)(x - x^*) + \frac{1}{2}(x - x^*)^T H(x - x^*).$$

Since $G(x^*) = DG(x^*) = 0$, it follows that $G(x) = \frac{1}{2}(x - x^*)^T H(x - x^*)$. Here $H = (H_1, H_2, H_3, H_4, H_5)^T$, where

$$H_1 = \begin{pmatrix} 0 & -\beta_1 & -\mu_1 & -\beta_2 & -\mu_2 \\ -\beta_1 & 0 & 0 & 0 & 0 \\ -\mu_1 & 0 & 0 & 0 & 0 \\ -\beta_2 & 0 & 0 & 0 & 0 \\ -\mu_2 & 0 & 0 & 0 & 0 \end{pmatrix}, \quad H_2 = \begin{pmatrix} 0 & p\beta_1 & p\mu_1 & 0 & 0 \\ p\beta_1 & 0 & 0 & 0 & 0 \\ p\mu_1 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{pmatrix},$$

$$H_3 = \begin{pmatrix} 0 & (1-p)\beta_1 & (1-p)\mu_1 & 0 & 0 \\ (1-p)\beta_1 & 0 & 0 & 0 & 0 \\ (1-p)\mu_1 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{pmatrix}, \quad H_4 = \begin{pmatrix} 0 & 0 & 0 & q\beta_2 & q\mu_2 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ q\beta_2 & 0 & 0 & 0 & 0 \\ q\mu_2 & 0 & 0 & 0 & 0 \end{pmatrix},$$

$$H_5 = \begin{pmatrix} 0 & 0 & 0 & (1-q)\beta_2 & (1-q)\mu_2 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ (1-q)\beta_2 & 0 & 0 & 0 & 0 \\ (1-q)\mu_2 & 0 & 0 & 0 & 0 \end{pmatrix}.$$

Define $B : \mathbb{R}^5 \times \mathbb{R}^5 \rightarrow \mathbb{R}^5$. Given two vectors $a, b \in \mathbb{R}^5$, the output is a vector $B(a, b) \in \mathbb{R}^5$. Its i -th component is given by $B(a, b)_i = \sum_{j=1}^5 \sum_{k=1}^5 H_{ijk} a_j b_k$, where H_{ijk} are the components of the Hessian tensor. Substituting $x = x^* + ur + \omega(u)$ into the above equation yields

$$G\left(x^* + \frac{1}{c}ur + \omega(u)\right) = \frac{1}{c}u^3B(r, h_2) + u^4\left[\frac{1}{c}B(r, h_3) + \frac{1}{2}B(h_2, h_2)\right] + O(u^4),$$

Thus, the reduced system becomes

$$\begin{aligned} \frac{du}{dt} &= l^T \left[J(x^*) \left(\frac{1}{c}ur + \omega(u) \right) + G\left(x^* + \frac{1}{c}ur + \omega(u)\right) \right] \\ &= l^T \left[\frac{1}{c}u^3B(r, h_2) + u^4\left(\frac{1}{c}B(r, h_3) + \frac{1}{2}B(h_2, h_2)\right) + O(u^4) \right]. \end{aligned}$$

Step 3. Stability analysis

From $x = x^* + \frac{1}{c}ur + \omega(u)$, $u = l^T(x - x^*)$, we obtain

$$\frac{dx}{dt} = f(x) = \frac{1}{c} \frac{du}{dt} r + \frac{d\omega}{dt} = \frac{1}{c} l^T \frac{dx}{dt} r + \frac{d\omega}{dt} = \frac{1}{c} l^T f(x) r + \frac{d\omega}{dt},$$

which further yields

$$\begin{aligned} \frac{d\omega}{dt} &= \left(I - \frac{rl^T}{c} \right) f(x) \\ &= \left(I - \frac{rl^T}{c} \right) \left[J(x^*) \left(\frac{1}{c}ur + \omega(u) \right) + G\left(x^* + \frac{1}{c}ur + \omega(u)\right) \right] \\ &= J(x^*)\omega(u) + \left(I - \frac{rl^T}{c} \right) \left[\frac{1}{c}u^3B(r, h_2) + u^4\left(\frac{1}{c}B(r, h_3) + \frac{1}{2}B(h_2, h_2)\right) + O(u^4) \right], \end{aligned}$$

Since $\omega(u) = h_2u^2 + h_3u^3 + O(u^4)$, we have

$$\begin{aligned} \frac{d\omega}{dt} &= \frac{d\omega}{du} \frac{du}{dt} \\ &= (2h_2u + 3h_3u^2 + O(u^3)) l^T \left[\frac{1}{c}u^3B(r, h_2) + u^4\left(\frac{1}{c}B(r, h_3) + \frac{1}{2}B(h_2, h_2)\right) + O(u^4) \right]. \end{aligned}$$

Then, we obtain $h_2 = 0$. By recursively applying the same procedure to higher-order terms, we further obtain $h_3 = 0, h_4 = 0$ and so on. Consequently, all higher-order coefficients vanish, leading to $\frac{du}{dt} = 0$ on the center manifold. Hence, every point on the center manifold is an equilibrium, the center direction is neutrally stable, and the stability

of the original system is determined solely by the stable eigenvalues, which are all negative. Therefore, the coexistence endemic equilibrium is locally stable. $\square$

Theorem 8. *If $R_{01} = R_{02} = R_0 > 1$, then the coexistence endemic equilibrium is globally attractive in Γ .*

Proof of Theorem 8. We construct the following Volterra-type Lyapunov function

$$L = x_1 \left(S - S_3^* \ln \frac{S}{S_3^*} \right) + \hat{x}_2 \left(A_1 - A_{13}^* \ln \frac{A_1}{A_{13}^*} \right) + \hat{x}_3 \left(I_1 - I_{13}^* \ln \frac{I_1}{I_{13}^*} \right) \\ + \hat{x}_8 \left(A_2 - A_{23}^* \ln \frac{A_2}{A_{23}^*} \right) + \hat{x}_9 \left(I_2 - I_{23}^* \ln \frac{I_2}{I_{23}^*} \right),$$

where $x_1, \hat{x}_2, \hat{x}_3, \hat{x}_8, \hat{x}_9 > 0$ are constants to be determined. Differentiating $L(t)$ along the trajectories of system (2) yields

$$\frac{dL}{dt} = x_1 \left[d_1 S_3^* \left(2 - \frac{S_3^*}{S} - \frac{S}{S_3^*} \right) + (\beta_1 A_{13}^* + \mu_1 I_{13}^*) \left(S_3^* - \frac{S_3^{*2}}{S} \right) + (\beta_2 A_{23}^* + \mu_2 I_{23}^*) \left(S_3^* - \frac{S_3^{*2}}{S} \right) \right] \\ + \hat{x}_2 \left[A_{13}^* (d_{21} + \alpha_1) - \frac{S}{A_1} p A_{13}^* (\beta_1 A_1 + \mu_1 I_1) \right] \\ + \hat{x}_3 \left[(d_{31} + \pi_1) I_{13}^* - \frac{S}{I_1} (1 - p) I_{13}^* (\beta_1 A_1 + \mu_1 I_1) - \frac{A_1}{I_1} I_{13}^* \alpha_1 \right] \\ + \hat{x}_8 \left[A_{23}^* (d_{22} + \alpha_2) - \frac{S}{A_2} q A_{23}^* (\beta_2 A_2 + \mu_2 I_2) \right] \\ + \hat{x}_9 \left[(d_{32} + \pi_2) I_{23}^* - \frac{S}{I_2} (1 - q) I_{23}^* (\beta_2 A_2 + \mu_2 I_2) - \frac{A_2}{I_2} I_{23}^* \alpha_2 \right],$$

where the above equality holds under the condition

$$\begin{cases} -x_1 + p\hat{x}_2 + (1-p)\hat{x}_3 = 0, \\ -x_1 + q\hat{x}_8 + (1-q)\hat{x}_9 = 0, \\ x_1\beta_1 S_3^* - \hat{x}_2(d_{21} + \alpha_1) + \hat{x}_3\alpha_1 = 0, \\ x_1\mu_1 S_3^* - \hat{x}_3(d_{31} + \pi_1) = 0, \\ x_1\beta_2 S_3^* - \hat{x}_8(d_{23} + \alpha_2) + \hat{x}_9\alpha_2 = 0, \\ x_1\mu_2 S_3^* - \hat{x}_9(\pi_2 + d_{32}) = 0. \end{cases} \quad (5)$$

We choose the positive constants $\hat{x}_2, \hat{x}_3, \hat{x}_8, \hat{x}_9$ as

$$\hat{x}_2 = \frac{(d_{31} + \pi_1)\beta_1 S_3^* + \mu_1 \alpha_1 S_3^*}{(d_{31} + \pi_1)(d_{21} + \alpha_1)}, \quad \hat{x}_3 = \frac{\mu_1 S_3^*}{(d_{31} + \pi_1)}, \\ \hat{x}_8 = \frac{(d_{32} + \pi_2)\beta_2 S_3^* + \mu_2 \alpha_2 S_3^*}{(d_{32} + \pi_2)(d_{22} + \alpha_2)}, \quad \hat{x}_9 = \frac{\mu_2 S_3^*}{(d_{32} + \pi_2)}.$$

Therefore, $\frac{dL}{dt}$ can be rewritten as $\frac{dL}{dt} = V_1 + V_2 + V_3 + V_4 + V_5$, where

$$V_1 = d_1 S_3^* \left(2 - \frac{S_3^*}{S} - \frac{S}{S_3^*} \right) \leq 0,$$

$$V_2 = -\frac{S_3^{*2}}{S}(\beta_1 A_{13}^* + \mu_1 I_{13}^*) - \hat{x}_2 \frac{S}{A_1} p A_{13}^* (\beta_1 A_1 + \mu_1 I_1) \\ - \hat{x}_3 \left[\frac{S}{I_1} (1-p) I_{13}^* (\beta_1 A_1 + \mu_1 I_1) - \frac{A_1}{I_1} I_{13}^* \alpha_1 \right],$$

$$V_3 = (\beta_1 A_{13}^* + \mu_1 I_{13}^*) S_3^* + \hat{x}_2 A_{13}^* (d_{21} + \alpha_1) + \hat{x}_3 (d_{31} + \pi_1) I_{13}^*,$$

$$V_4 = -\frac{S_3^{*2}}{S}(\beta_2 A_{23}^* + \mu_2 I_{23}^*) - \hat{x}_8 \frac{S}{A_2} q A_{23}^* (\beta_2 A_2 + \mu_2 I_2) \\ - \hat{x}_9 \left[\frac{S}{I_2} (1-q) I_{23}^* (\beta_2 A_2 + \mu_2 I_2) - \frac{A_2}{I_2} I_{23}^* \alpha_2 \right],$$

$$V_5 = (\beta_2 A_{23}^* + \mu_2 I_{23}^*) S_3^* + \hat{x}_8 A_{23}^* (d_{22} + \alpha_2) + \hat{x}_9 (d_{32} + \pi_2) I_{23}^*.$$

For V_2 , using $p\hat{x}_2 + (1-p)\hat{x}_3 = x_1$, we can rewrite it as

$$V_2 = \left[-p\hat{x}_2 \frac{S_3^{*2}}{S} \beta_1 A_{13}^* - \hat{x}_2 S p \beta_1 A_{13}^* \right] + \left[-(1-p)\hat{x}_3 \frac{S_3^{*2}}{S} \mu_1 I_{13}^* - \hat{x}_2 S (1-p) \mu_1 I_{13}^* \right] \\ + \left[-u_1 p \hat{x}_2 \frac{S_3^{*2}}{S} \mu_1 I_{13}^* - u_1 \hat{x}_2 \frac{S I_1}{A_1} p \mu_1 A_{13}^* - \hat{x}_3 \frac{A_1}{I_1} I_{13}^* \alpha_1 \right] \\ + \left[-(1-p)\hat{x}_3 \frac{S_3^{*2}}{S} \beta_1 A_{13}^* - \hat{x}_3 \frac{S A_1}{I_1} (1-p) I_{13}^* \beta_1 \right] \\ + \left[-(1-u_1)\hat{x}_2 \frac{S I_1}{A_1} p \mu_1 A_{13}^* - (1-u_1)p\hat{x}_2 \frac{S_3^{*2}}{S} \mu_1 I_{13}^* \right] \\ \leq -2\hat{x}_2 S p \beta_1 A_{13}^* - 2\hat{x}_3 S (1-p) \mu_1 I_{13}^* - 3 \left(u_1^2 p^2 \hat{x}_2^2 \hat{x}_3 \mu_1^2 I_{13}^{*2} A_{13}^* \alpha_1 S_3^{*2} \right)^{1/3} \\ - 4 \left[(1-u_1)^2 p^2 (1-p)^2 \hat{x}_2^2 \hat{x}_3^2 \mu_1^2 \beta_1^2 I_{13}^{*2} A_{13}^{*2} S_3^{*2} \right]^{1/4},$$

where

$$u_1 = \frac{\alpha_1}{[(1-p)d_{21} + \alpha_1]\hat{x}_2}, \quad 1-u_1 = \frac{(1-p)\beta_1 S_3^*}{[(1-p)d_{21} + \alpha_1]\hat{x}_2}.$$

For V_4 , using $q\hat{x}_8 + (1-q)\hat{x}_9 = x_1$, we obtain

$$V_4 = \left[-q\hat{x}_8 \frac{S_3^{*2}}{S} \beta_2 A_{23}^* - \hat{x}_8 S q \beta_2 A_{23}^* \right] + \left[-(1-q)\hat{x}_9 \frac{S_3^{*2}}{S} \mu_2 I_{23}^* - \hat{x}_8 S (1-q) \mu_2 I_{23}^* \right] \\ + \left[-u_2 q \hat{x}_8 \frac{S_3^{*2}}{S} \mu_2 I_{23}^* - u_2 \hat{x}_8 \frac{S I_2}{A_2} q \mu_2 A_{23}^* - \hat{x}_9 \frac{A_2}{I_2} I_{23}^* \alpha_2 \right] \\ + \left[-(1-q)\hat{x}_9 \frac{S_3^{*2}}{S} \beta_2 A_{23}^* - \hat{x}_9 \frac{S A_2}{I_2} (1-q) I_{23}^* \beta_2 \right] \\ + \left[-(1-u_2)\hat{x}_8 \frac{S I_2}{A_2} p \mu_2 A_{23}^* - (1-u_2)q\hat{x}_8 \frac{S_3^{*2}}{S} \mu_2 I_{23}^* \right] \\ \leq -2\hat{x}_8 S q \beta_2 A_{23}^* - 2\hat{x}_9 S (1-q) \mu_2 I_{23}^* - 3 \left(u_2^2 q^2 \hat{x}_8^2 \hat{x}_9 \mu_2^2 I_{23}^{*2} A_{23}^* \alpha_2 S_3^{*2} \right)^{1/3} \\ - 4 \left[(1-u_2)^2 q^2 (1-q)^2 \hat{x}_8^2 \hat{x}_9^2 \mu_2^2 \beta_2^2 I_{23}^{*2} A_{23}^{*2} S_3^{*2} \right]^{1/4},$$

where

$$u_2 = \frac{\alpha_2}{[(1-p)d_{22} + \alpha_2]\hat{x}_8}, \quad 1 - u_2 = \frac{(1-q)\beta_2 S_3^*}{[(1-p)d_{22} + \alpha_2]\hat{x}_8}.$$

Using relations (5), V_3 can be further expressed as

$$V_3 = 2p\hat{x}_2\beta_1 A_{13}^* S_3^* + 2(1-p)\hat{x}_3\mu_1 I_{13}^* S_3^* + 3\hat{x}_3\alpha_1 A_{13}^* + 4(1-p)\hat{x}_3\beta_1 A_{13}^* S_3^*,$$

Similarly, we can rewrite V_5 as

$$V_5 = 2q\hat{x}_8\beta_2 A_{23}^* S_3^* + 2(1-q)\hat{x}_9\mu_2 I_{23}^* S_3^* + 3\hat{x}_9\alpha_2 A_{23}^* + 4(1-q)\hat{x}_9\beta_2 A_{23}^* S_3^*,$$

by the arithmetic mean–geometric mean inequality, we derive $V_2 + V_4 \leq 0$, $V_3 + V_5 \leq 0$.

Combining the above results, we obtain $\frac{dL}{dt} \leq 0$, and equality holds if and only if $(S_3^*, A_{13}^*, I_{13}^*, A_{23}^*, I_{23}^*)$ belongs to the set of coexistence endemic equilibria in the feasible domain Γ . Therefore, by the Lyapunov function method, we conclude that all solutions of system (2) converge to the set of coexistence endemic equilibria, i.e., the system is globally stable in Γ . $\square$

4. Sensitivity Analysis

In this section, a global sensitivity analysis is performed using the Partial Rank Correlation Coefficient (PRCC) method combined with Latin Hypercube Sampling (LHS), with R_{01} and R_{02} as outputs. As illustrated in Figure 3, the parameters μ_1 , β_1 , and p exhibit a strong positive correlation with R_{01} , while π_1 and α_1 are negatively correlated with R_{01} . Similarly, μ_2 , β_2 , and q show significant positive correlations with R_{02} , whereas π_2 and α_2 are negatively correlated with R_{02} . These results provide important insights for disease control. In particular, reducing transmission rates or increasing recovery rates can effectively suppress disease spread by shortening the infectious period and lowering the overall transmission potential. The consistency of these effects across both strains highlights the robustness of the proposed control strategies.

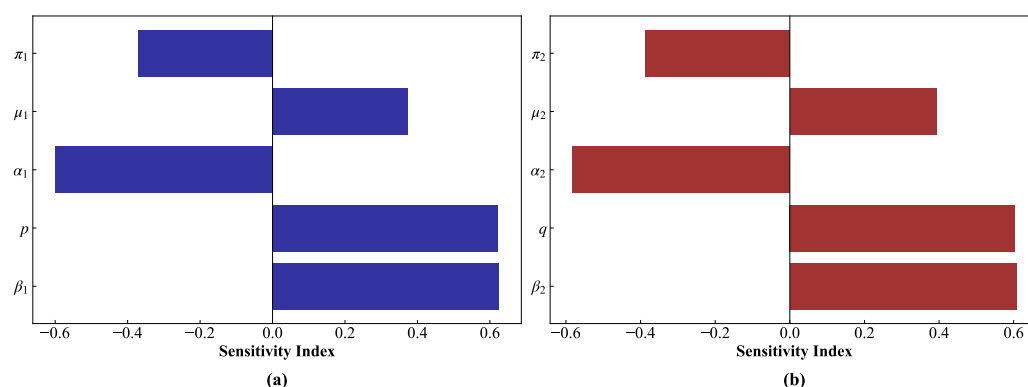


Figure 3. Partial rank correlation coefficients (PRCCs) of the basic reproduction numbers with respect to key model parameters. (a) PRCC values of R_{01} with respect to the key parameters. (b) PRCC values of R_{02} with respect to the key parameters.

5. Numerical Simulations

In this section, numerical simulations are performed to illustrate and complement the theoretical results obtained in the previous sections. In particular, the simulations are used to demonstrate the global dynamical behavior of System (2) under different threshold conditions, including convergence toward the disease-free equilibrium, single-strain endemic equilibria, and the coexistence equilibrium manifold arising in the degenerate case. All simulations in this section are carried out numerically using Python (3.14). The

system of ordinary differential equations is solved with the `solve_ivp` function from the `scipy.integrate` package.

The parameter values used in the simulations are listed in Table 1. Some parameters are adopted from the existing literature whenever available, while the remaining ones are chosen for illustrative purposes in the absence of reliable epidemiological data. It should be emphasized that the main theoretical results of this paper, including the existence of equilibria, stability properties, and the continuum of coexistence equilibria, are established analytically and do not depend on any specific parameter choice. Different parameter values may affect the quantitative behavior of the solutions, but the qualitative dynamical properties remain valid provided the corresponding threshold conditions are satisfied.

Moreover, the numerical examples are intended to visualize the distinct asymptotic behaviors predicted by the analysis and to highlight the role of parameter degeneracy in generating a continuum of coexistence equilibria, which cannot be observed in classical epidemic models with isolated endemic states.

Figure 4 illustrates the global dynamics under different parameter regimes. The system converges to the disease-free equilibrium when $R_{01} < 1$ and $R_{02} < 1$, to a boundary equilibrium when one strain dominates, and to a coexistence equilibrium when $R_{01} = R_{02} > 1$. To further illustrate the global dynamics, three-dimensional phase portraits are constructed in the (A_1, A_2, S) and (I_1, I_2, S) spaces (see Figures 5 and 6). The numerical results are consistent with the theoretical analysis: solutions converge to the disease-free equilibrium when $R_0 < 1$, and to a unique boundary endemic equilibrium when one strain dominates the other. In particular, when $R_{01} = R_{02} > 1$, trajectories converge to different equilibrium points lying on a one-dimensional manifold, providing numerical evidence for the existence of a continuum of endemic equilibria.

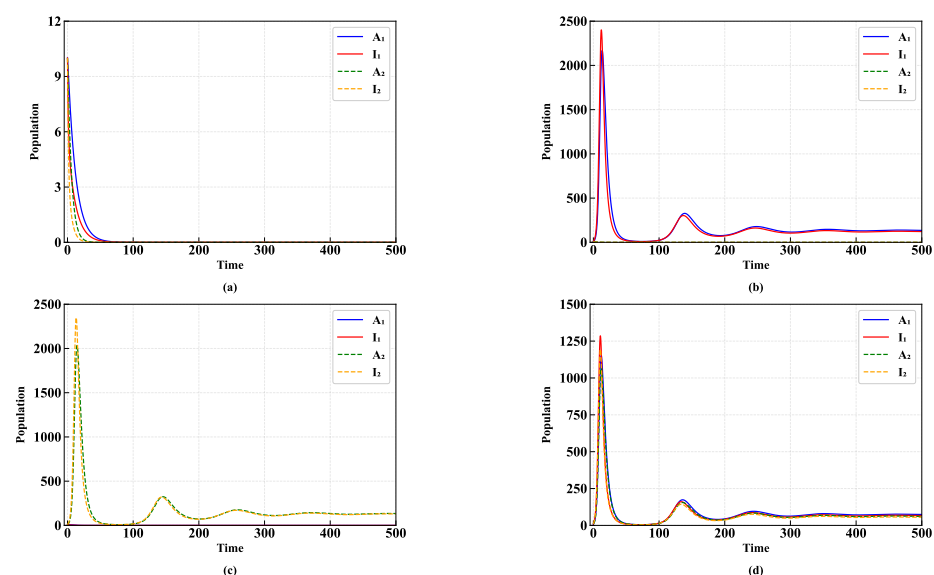


Figure 4. Dynamic behaviors of system (2) under different parameter regimes, with baseline values given in Table 1. (a) When $R_{01} < 1$ and $R_{02} < 1$ (e.g., $\beta_1 = 1.5 \times 10^{-5}$, $\beta_2 = 2 \times 10^{-6}$, $\mu_1 = \mu_2 = 1 \times 10^{-5}$, $\pi_1 = 0.95$, $\pi_2 = 0.9$), the system converges to the disease-free equilibrium ($R_{01} = 0.64$, $R_{02} = 0.28$). (b) When $R_{01} > \max\{1, R_{02}\}$ (e.g., $\beta_2 = 2 \times 10^{-6}$, $\mu_2 = 1 \times 10^{-5}$, $\pi_2 = 0.9$), the sensitive strain dominates ($R_{01} = 3.01$, $R_{02} = 0.28$). (c) When $R_{02} > \max\{1, R_{01}\}$ (e.g., $\beta_1 = 1.5 \times 10^{-5}$, $\mu_1 = 1 \times 10^{-5}$, $\pi_1 = 0.95$), the drug-resistant strain dominates ($R_{01} = 0.64$, $R_{02} = 2.86$). (d) When $R_{01} = R_{02} > 1$ (e.g., $\beta_2 = 6.67 \times 10^{-5}$, $\mu_1 = 4.5 \times 10^{-5}$, $\mu_2 = 4.3 \times 10^{-5}$, $\pi_2 = 0.75$), both strains coexist at an endemic equilibrium ($R_{01} = R_{02} = 3.06$).

To examine the influence of key parameters, simulations are conducted for transmission, recovery, and transition rates (Figures 7–10). As shown in Figure 7, decreasing μ_1

leads to a substantial reduction in both asymptomatic and symptomatic populations of the drug-sensitive strain, accompanied by a delayed outbreak and a lower endemic level. In contrast, decreasing μ_2 weakens the competitive pressure imposed by the drug-resistant strain, resulting in an increase in the prevalence of the sensitive strain. Similarly, a reduction in μ_2 significantly suppresses the infection levels of the resistant strain, while decreasing μ_1 promotes its persistence due to reduced competition from the sensitive strain. These results highlight that the relative transmission advantages between strains play a decisive role in determining competitive outcomes.

Figure 9 shows that increasing the recovery rates π_1 and π_2 reduces infection levels and lowers endemic equilibria, thereby shortening the infectious period. However, asymmetry in recovery rates may shift the competitive balance between strains. Figure 10 illustrates the effects of transition (screening) rates α_1 and α_2 . Higher transition rates reduce the asymptomatic population and influence the distribution between asymptomatic and symptomatic classes, thereby affecting overall transmission dynamics. This suggests that enhanced screening and early detection are crucial for controlling diseases with significant asymptomatic transmission.

Overall, the numerical simulations reveal that transmission rates primarily determine strain dominance, recovery rates regulate infection persistence, and transition rates control hidden transmission pathways. These findings provide important insights into the design of effective intervention strategies for multi-strain infectious diseases.

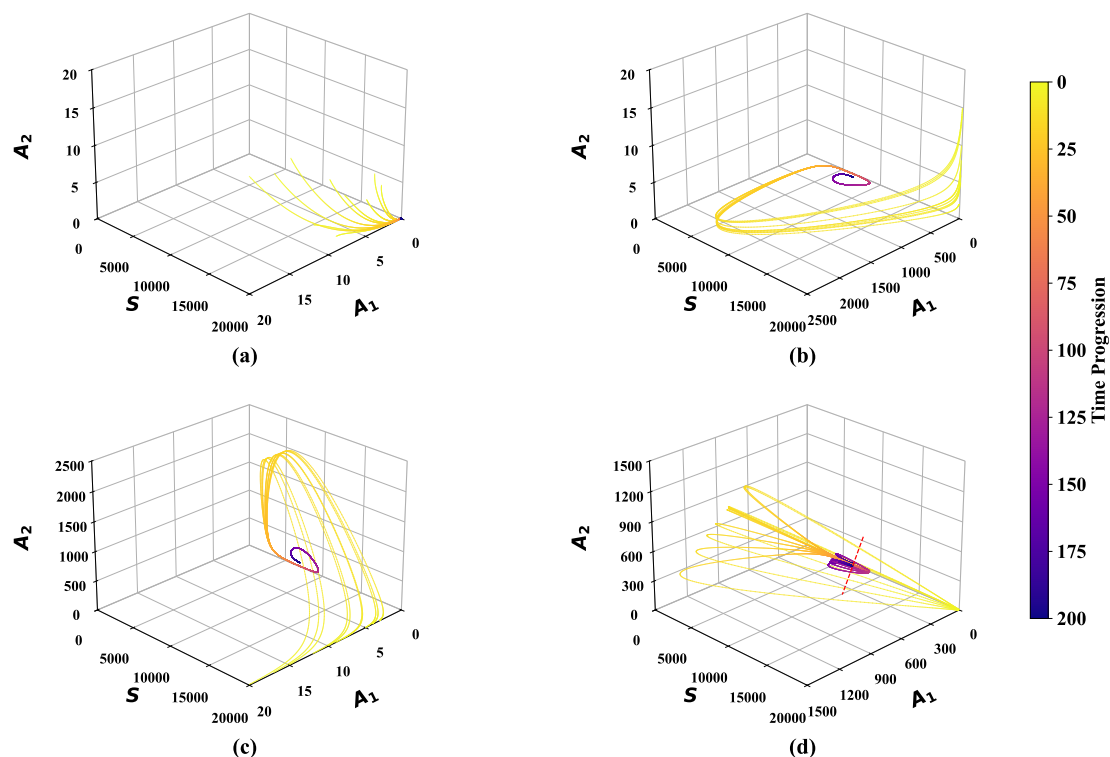


Figure 5. Phase portraits of system (2) in the (A_1, A_2, S) space under the same parameter regimes as in Figure 4. (a) When $R_{01} < 1$ and $R_{02} < 1$, the system converges to the disease-free equilibrium. (b) When $R_{01} > \max\{1, R_{02}\}$, the sensitive strain dominates. (c) When $R_{02} > \max\{1, R_{01}\}$, the drug-resistant strain dominates. (d) When $R_{01} = R_{02} > 1$, both strains coexist at an endemic equilibrium.

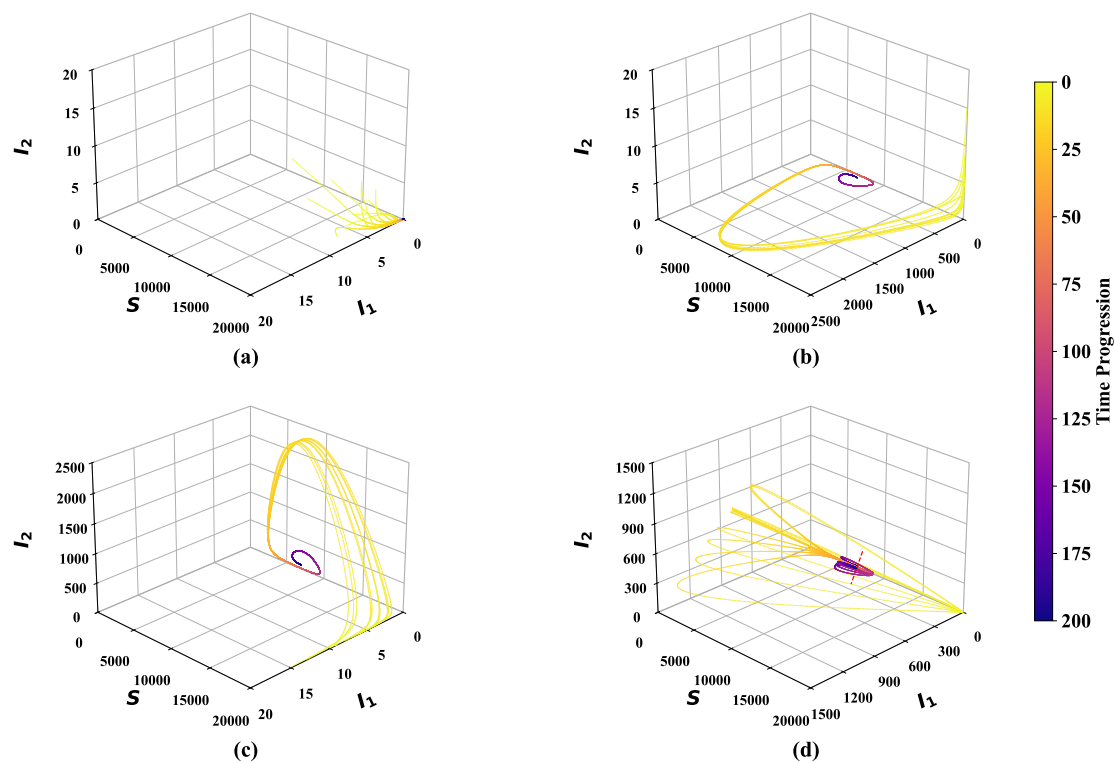


Figure 6. Phase portraits of system (2) in the (I_1, I_2, S) space under the same parameter regimes as in Figure 5. (a) When $R_{01} < 1$ and $R_{02} < 1$, the system converges to the disease-free equilibrium. (b) When $R_{01} > \max\{1, R_{02}\}$, the sensitive strain dominates. (c) When $R_{02} > \max\{1, R_{01}\}$, the drug-resistant strain dominates. (d) When $R_{01} = R_{02} > 1$, both strains coexist at an endemic equilibrium.

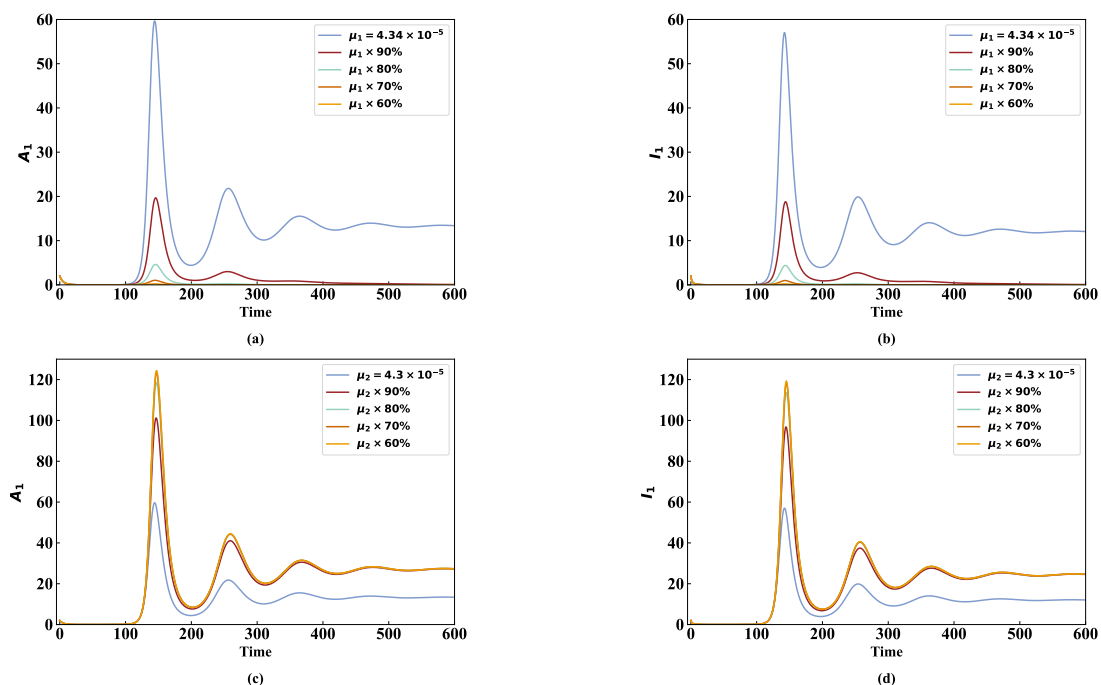


Figure 7. Effects of transmission rates on the dynamics of the drug-sensitive strain, with parameter values partly taken from Table 1 and partly specified for simulation purposes (e.g., $\mu_1 = 4.34 \times 10^{-5}$, $\mu_2 = 4.3 \times 10^{-5}$, $\beta_2 = 6.54 \times 10^{-5}$, $\pi_2 = 0.75$). (a) Effects of varying μ_1 on the asymptomatic population of the sensitive strain. (b) Effects of varying μ_1 on the symptomatic population of the sensitive strain. (c) Cross effects of μ_2 on the asymptomatic population of the sensitive strain. (d) Cross effects of μ_2 on the symptomatic population of the sensitive strain.

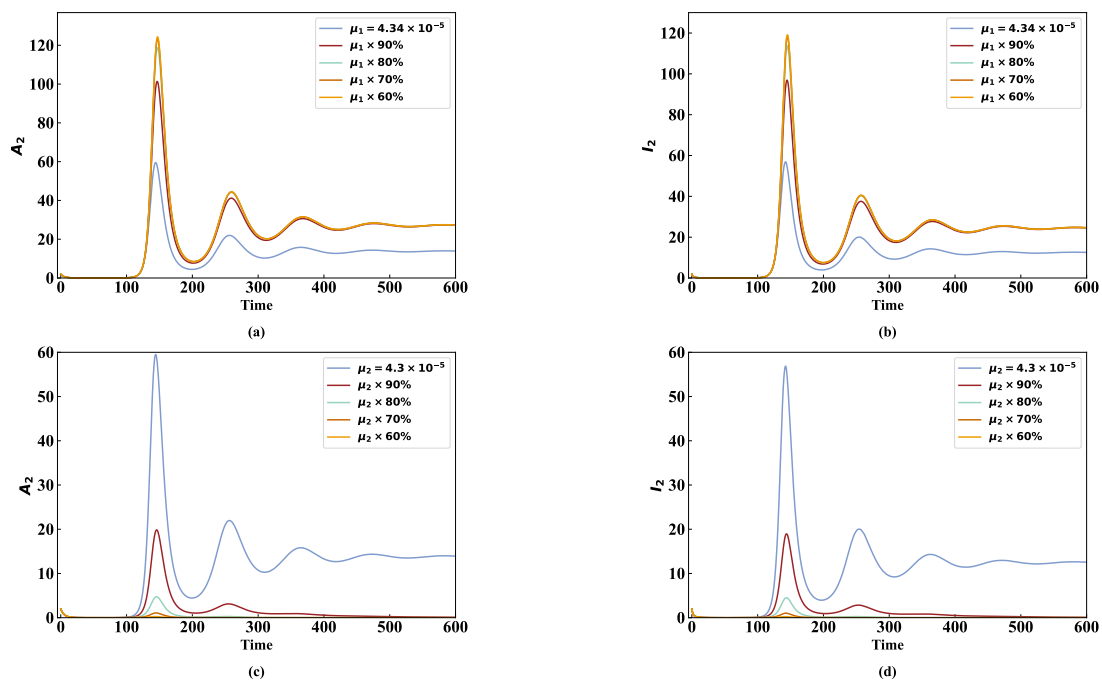


Figure 8. Effects of transmission rates on the dynamics of the drug-resistant strain. (a) Effects of varying μ_1 on the asymptomatic population of the drug-resistant strain. (b) Effects of varying μ_1 on the symptomatic population of the drug-resistant strain. (c) Cross effects of μ_2 on the asymptomatic population of the drug-resistant strain. (d) Cross effects of μ_2 on the symptomatic population of the drug-resistant strain. Parameter values are the same as in Figure 7.

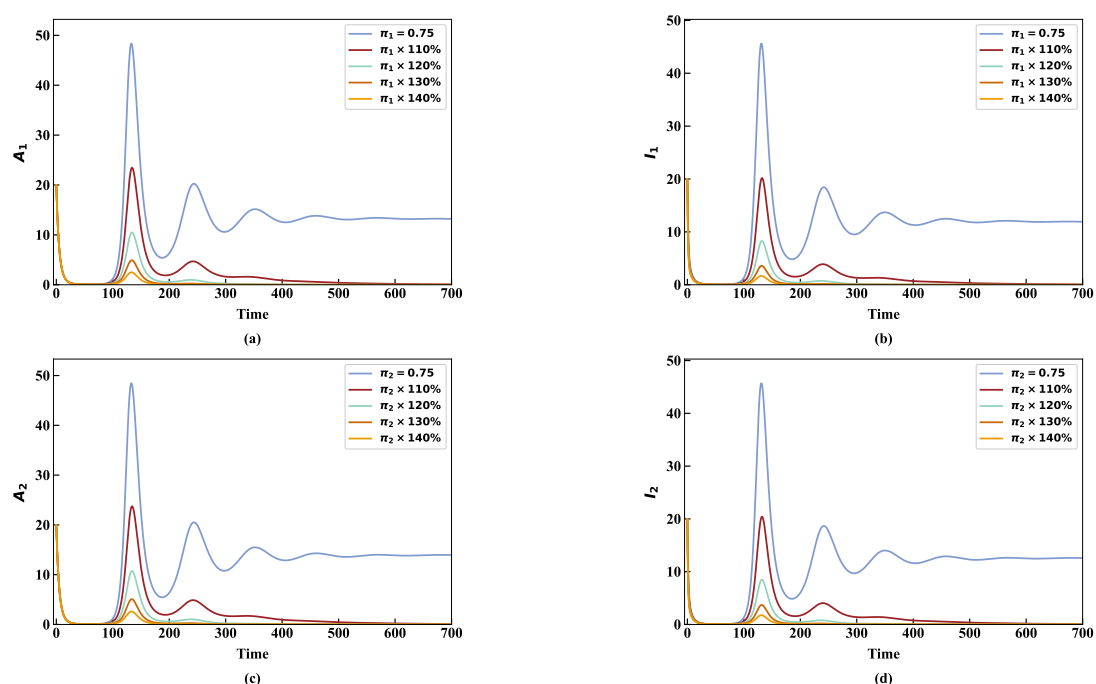


Figure 9. Effects of recovery rates on the infection dynamics of the two strains. (a,b) Effects of varying π_1 on the asymptomatic and symptomatic populations of the sensitive strain. (c,d) Effects of varying π_2 on the drug-resistant strain. Parameter values are the same as in Figure 7.

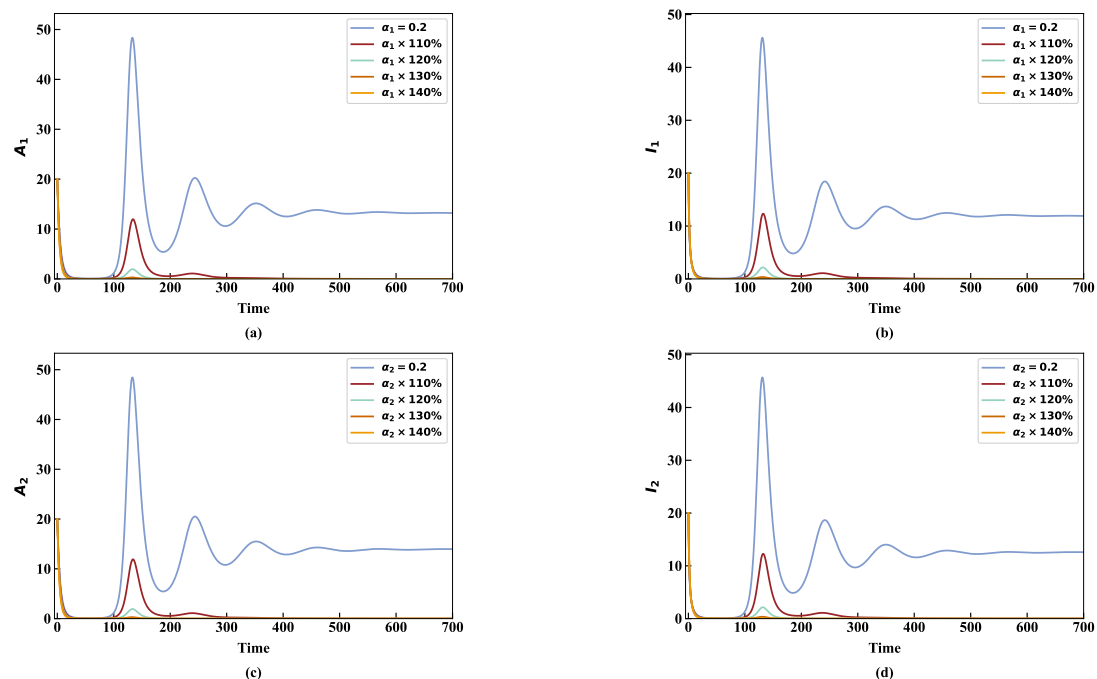


Figure 10. Effects of transition (screening) rates on the infection dynamics. (a,b) Effects of varying α_1 on the asymptomatic and symptomatic populations of the sensitive strain. (c,d) Effects of varying α_2 on the drug-resistant strain. Parameter values are the same as in Figure 7.

6. Discussion

This study develops and analyzes a two-strain SAIR epidemic model with asymptomatic transmission and reveals a degenerate dynamical structure characterized by a continuum of endemic equilibria. This feature departs from the classical framework of isolated equilibria in multi-strain epidemic models.

The results show that the basic reproduction numbers $\mathcal{R}_{01}$ and $\mathcal{R}_{02}$ determine the global dynamics. When $\mathcal{R}_{01} \neq \mathcal{R}_{02}$, the system exhibits competitive exclusion, with the strain of larger reproduction number dominating. When $\mathcal{R}_{01} = \mathcal{R}_{02} > 1$, the system admits a continuum of coexistence equilibria forming a one-dimensional manifold. These equilibria are non-hyperbolic and possess neutral stability along a center direction. By combining Lyapunov methods with center manifold analysis, the equilibrium set is shown to be globally attractive.

From an epidemiological perspective, this degenerate structure corresponds to a regime of neutral competition, where competing strains have identical transmission potential. In this case, the long-term distribution of strains depends on initial conditions rather than parameter differences. This mechanism provides a possible explanation for the coexistence of pathogen variants, particularly in the presence of asymptomatic transmission.

The analysis also highlights the critical role of transmission-related parameters and asymptomatic infection in influencing epidemic outcomes. Variations in transmission rates and recovery processes determine whether exclusion or coexistence occurs, while asymptomatic transmission can sustain hidden infection pathways and promote persistence. These findings suggest that effective control strategies should simultaneously reduce transmission and enhance the detection and management of asymptomatic infections.

Although the present study is primarily theoretical, the analytical results provide general threshold criteria and qualitative mechanisms that may help interpret long-term epidemic behaviors in multi-strain systems. In particular, the existence of a continuum of coexistence equilibria suggests that small parameter perturbations or estimation un-

certainties may substantially influence the asymptotic distribution of competing strains. Therefore, the present analysis complements data-driven epidemic studies by clarifying the underlying dynamical structures governing strain competition and persistence.

Despite these contributions, several limitations should be noted. The model assumes homogeneous mixing and constant parameters, which may not fully reflect realistic contact structures or time-varying interventions. In addition, mutation and evolutionary dynamics between strains are not explicitly incorporated, which could influence the stability and structure of the coexistence regime.

Furthermore, the present study focuses on a two-strain framework as a fundamental and analytically tractable setting for investigating strain competition and coexistence mechanisms. Although epidemic systems with multiple strains are also important, the two-strain model already captures the essential dynamical features associated with competitive exclusion, coexistence, and parameter degeneracy. Moreover, the mathematical analysis of the continuum of equilibria already requires delicate treatment based on Lyapunov methods and center manifold theory. Extending the present framework to a general n -strain system is therefore not a straightforward addition of variables, since the equilibrium structure, invariant manifolds, and degeneracy conditions become substantially more complicated in higher-dimensional settings. Such extensions will be considered in future work.

Future work may extend this framework by incorporating heterogeneous contact patterns, time-dependent parameters, and stochastic perturbations to assess the robustness of the equilibrium manifold. It would also be of interest to include evolutionary dynamics or mutation mechanisms, as well as intervention strategies such as vaccination or treatment, to investigate how these factors may break the degeneracy and alter long-term epidemic outcomes.

7. Conclusions

In conclusion, this work reveals how asymptomatic transmission and parameter symmetry can generate a continuum of endemic equilibria and induce neutral competition in multi-strain epidemic systems. The study demonstrates that even within a two-strain framework, rich non-hyperbolic dynamical structures may arise, leading to qualitative behaviors that differ fundamentally from classical epidemic models with isolated equilibria. These findings enrich the theoretical understanding of epidemic dynamics and provide useful insights for the control of competing infectious diseases.

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