

# Dynamics of a competing two-strain SIS epidemic model on complex networks with a saturating incidence rate

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## Abstract

This paper studies a two-strain SIS epidemic model with a competing mechanism and a saturating incidence rate on complex networks. This type of incidence rate could be used to reflect the crowding effect of the infective individuals. We first obtain the associated reproduction numbers for each of the two strains which determine the existence of the boundary equilibria. Stability of the disease-free and boundary equilibria are further examined. Besides, we also show that the two competing strains can coexist under certain conditions. Interestingly, the saturating incidence rate can have specific effects on not only the stability of the boundary equilibria, but also the existence of the coexistence equilibrium. Numerical simulations are presented to support the theoretical results.

*Keywords:* Complex network, epidemic model, saturating incidence, stability, coexistence

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## 1. Introduction

To control and prevent the spread of infectious diseases is one of the greatest global challenges in our time. Although the mechanism of transmission of infections is now known for most diseases, for example, influenza, measles, and severe acute respiratory syndrome (SARS) are transmitted by viral agents; tuberculosis (TB) and gonorrhea are transmitted by bacteria, how to prevent the transmission of infection effectively is still an important and ongoing problem. Mathematical modeling of infectious diseases has been widely agreed that it is an effective tool for studying and predicting the spreading dynamics of various communicable diseases. Subsequently, appropriate strategies could be developed to control possible outbreaks of diseases.

In the disease spreading processes, a common phenomenon is pathogen mutation. One example is the influenza in 2009, in addition to the seasonal influenza, the H1N1 influenza emerged in Mexico and the United States. The disease then spread very rapidly and had caused about 17,000 deaths by the start of 2010. Another widely-spread disease is the human immunodeficiency virus (HIV). It is well known that HIV can cause HIV infection and acquired immunodeficiency syndrome (AIDS). Two types of HIV (i.e., HIV-1 and HIV-2) have been characterized. Both of these two examples cause serious problems in treating the resulting diseases. As a result, the study of disease dynamics with multiple strains has great relevance and related research in recent years has increased noticeably [1, 2, 3, 5, 6, 8, 9, 12, 14, 15, 16, 17, 18, 20, 21, 24, 25]. An important question in studying multi-strain epidemic models is to identify the conditions that lead to the coexistence of different strains. Various mechanisms have been discussed in this issue, including the effect of age [18], super-infection [6, 9, 25], cross-immunity [1, 5], co-infection [17, 20], and mutation [16].

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Most of aforementioned studies mainly focus on the model with the homogeneous mixing assumption, that is, each susceptible individual within a population has the same probability to contact with an infected one. However, it has been widely accepted that the effect of contact heterogeneity should be incorporated into consideration in reality. Therefore, the disease transmission should be modeled over complex networks. Although there have been many efforts on one-strain epidemic model [7, 11, 13, 22, 23, 27, 28, 29, 30, 31], to the best of our knowledge, comparatively little research has focused on the multi-strain models on complex networks [24, 25]. In [24], Wu et al. proposed a two-strain SIS model in scale-free networks [4] with a competing mechanism, which means that two strains from one pathogen cannot co-infect in a single host at any time. The authors found the emergence of an epidemic threshold under the coexistence of the other strain. Besides, a two-strain epidemic model with superinfection on scale-free networks was proposed in [25]. This work indicates that the superinfection is a key mechanism for the coexistence of the two strains. More specifically, superinfection can allow a strain to persist even if the strain reproduction number is less than one.

The purpose of the present paper is to study other mechanism that permit coexistence of different strains. Motivated by Li et al. [12], we will investigate the dynamics of a two-strain epidemic model on complex networks with a nonlinear incidence rate. More precisely, the incidence rate considered in [12] is a saturating incidence rate. In contrast to linear incidence rate which is due to the mass action law for infection, the saturating one is more reasonable as it reflects the crowding effect of the infective individuals. Moreover, there has thus far been relatively little research into network epidemic models with nonlinear incidence rate [11, 29]. Therefore, in this paper we are mainly concerned with a two-strain SIS epidemic model with a competing mechanism and a saturating incidence rate on complex networks. Each of the two strains obeys an SIS epidemiological process and the associated reproduction numbers for each strain are obtained. The stability of the disease-free and boundary equilibria are examined by the linear stability analysis. Besides, we also proposed some conditions that can allow one strain to coexist with the other one. We will show that the saturating incidence rate can have specific effects on not only the stability of the boundary equilibria, but also the existence of the coexistence equilibrium. Numerical examples with a finite size of scale-free network will be proposed to support our theoretical analysis.

The rest of this paper is organized as follows: In Section 2, we propose a two-strain SIS epidemic model on complex networks. In Section 3, the existence and the stability of the disease-free and boundary equilibria are discussed. In Section 4, we deal with the strain coexistence. In Section 5, six numerical examples are given to demonstrate the theoretical results. Finally, concluding remarks are made in Section 6.

## 2. Model formulation

In this section, we first review the model proposed in [24] and subsequently, introduce our modified model. From a mathematical viewpoint, a complex network is composed by a set of nodes and edges. Each node represents an individual in its corresponding epidemiological state, and each edge between two nodes stands for an interaction that may allow disease transmission. All the nodes in a given network can be firstly classified into  $n$  groups such that the nodes in the same group have equal degree. That is, each node in the  $k$ -th group has degree  $k$  for  $k = 1, 2, \dots, n$ , where  $n$  is the maximal degree of the network. Let  $\rho_k(t)$  and  $\vartheta_k(t)$  be the densities of nodes in the group with degree  $k$  infected by strain 1 and strain 2, respectively. Then the competing two-strain SIS model proposed in [24] is given by:

$$\begin{cases} \rho'_k(t) = -\gamma_1\rho_k(t) + \beta_1k(1 - \rho_k(t) - \vartheta_k(t))\Theta_1(t), \\ \vartheta'_k(t) = -\gamma_2\vartheta_k(t) + \beta_2k(1 - \rho_k(t) - \vartheta_k(t))\Theta_2(t), \end{cases} \quad k = 1, 2, \dots, n, \quad (2.1)$$

where  $\beta_i$  and  $\gamma_i$  are respectively the transmission and the cure rates for strain  $i$  ( $i = 1, 2$ ). The term  $\Theta_1$  (resp.,  $\Theta_2$ ) stands for the probability of a link pointing to a node infected by strain 1 (resp., 2). To study epidemic transmission on uncorrelated networks, the coupled terms  $\Theta_1$  and  $\Theta_2$  can be formed as

$$\Theta_1(t) = \frac{\sum_{k=1}^n kP(k)\rho_k(t)}{\langle k \rangle} \quad \text{and} \quad \Theta_2(t) = \frac{\sum_{k=1}^n kP(k)\vartheta_k(t)}{\langle k \rangle}, \quad (2.2)$$

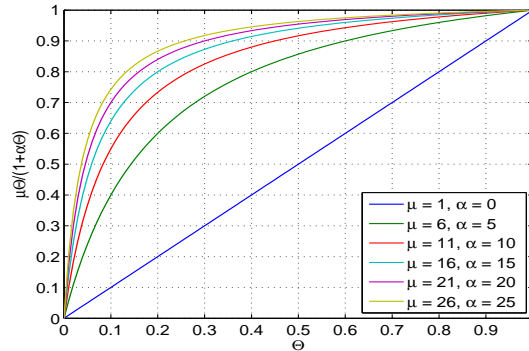
where  $P(k)$  is the fraction of nodes with degree  $k$  in the network with  $\sum_{k=1}^n P(k) = 1$ , and  $\langle k \rangle = \sum_{h=1}^n hP(h)$  is the averaged degree of the network. The conclusion in [24] is that for  $\beta_1/\gamma_1 \neq \beta_2/\gamma_2$  it is impossible for the two strain to coexist. In other words, only competitive exclusion occurs in this case. On the other hand, both strains can coexist only for the degenerate case, namely,  $\beta_1/\gamma_1 = \beta_2/\gamma_2$ . A natural question that arises regarding this model is that ‘‘Does there exist other mechanism that can allow two strains to coexist?’’ The answer is positive, for example, Wu et al. [25] show that superinfection is one of such mechanisms. However, the incidence rate considered in [25] is still a linear one. Since it has been pointed out that nonlinear incidence rates might be more reasonable as they can reflect the behavioral change of individuals in the population [26]. Thus, motivated by the work [12], we now incorporate a nonlinear incidence rate into the competing two-strain model which is governed by

$$\begin{cases} \rho'_k(t) = -\gamma_1\rho_k(t) + \beta_1k(1 - \rho_k(t) - \vartheta_k(t))g(\Theta_1(t)), \\ \vartheta'_k(t) = -\gamma_2\vartheta_k(t) + \beta_2k(1 - \rho_k(t) - \vartheta_k(t))g(\Theta_2(t)), \end{cases} \quad k = 1, 2, \dots, n, \quad (2.3)$$

where  $g(\Theta_i)$  are the nonlinear incidence rates for  $i = 1, 2$ . To reflect the crowding effect of the infective individuals, in the present work, the nonlinear incidence rates  $g$  will be given by

$$g(\Theta_1) = \frac{\mu_1\Theta_1}{1 + \alpha_1\Theta_1} \quad \text{and} \quad g(\Theta_2) = \frac{\mu_2\Theta_2}{1 + \alpha_2\Theta_2},$$

where the parameters  $\mu_i > 0$  and  $\alpha_i \geq 0$  for  $i = 1, 2$ . Since  $0 \leq \Theta_1, \Theta_2 \leq 1$ , these parameters can determine the saturation levels (cf. Figure 1). We also remark here that if  $\mu_1 = \mu_2 = 1$  and  $\alpha_1 = \alpha_2 = 0$ , then the system (2.3) can be reduced to system (2.1).



**Figure 1.** The graphs of  $g(\Theta)$  with various values of  $\mu$  and  $\alpha$ .

Throughout this paper, we will focus on the dynamics of solutions of system (2.3) in the following bounded region:

$$\Omega := \{(\rho_1, \rho_2, \dots, \rho_n, \vartheta_1, \vartheta_2, \dots, \vartheta_n) : 0 \leq \rho_k, \vartheta_k \leq 1, 1 \leq k \leq n\}.$$

We can show that system (2.3) is positively invariant in  $\Omega$ . The proof of this result is analog to the discussion of Lemma 3.1 proposed in [10], and we shall omit here.

### 3. Existence and stability of the disease-free and boundary equilibria

In this section, we investigate the existence and stability of the disease-free equilibrium and the boundary equilibria of the system (2.3). Now, we are going to compute the equilibria and determine the reproduction numbers corresponding to each strain. Let  $\tau_i = \beta_i/\gamma_i$  for  $i = 1, 2$ . From  $\rho'_k(t) = 0$  and  $\vartheta'_k(t) = 0$ , we have

$$\begin{cases} \rho_k = \frac{\tau_1\mu_1k\Theta_1(1 + \alpha_2\Theta_2)}{(1 + \alpha_1\Theta_1)(1 + \alpha_2\Theta_2) + \tau_1\mu_1k\Theta_1(1 + \alpha_2\Theta_2) + \tau_2\mu_2k\Theta_2(1 + \alpha_1\Theta_1)}, \\ \vartheta_k = \frac{\tau_2\mu_2k\Theta_2(1 + \alpha_1\Theta_1)}{(1 + \alpha_1\Theta_1)(1 + \alpha_2\Theta_2) + \tau_1\mu_1k\Theta_1(1 + \alpha_2\Theta_2) + \tau_2\mu_2k\Theta_2(1 + \alpha_1\Theta_1)}. \end{cases} \quad (3.1)$$

Substituting (3.1) into (2.2), we can obtain

$$\Theta_1 = \frac{\tau_1 \mu_1}{\langle k \rangle} \sum_{h=1}^n \frac{h^2 P(h) \Theta_1 (1 + \alpha_2 \Theta_2)}{(1 + \alpha_1 \Theta_1)(1 + \alpha_2 \Theta_2) + \tau_1 \mu_1 h \Theta_1 (1 + \alpha_2 \Theta_2) + \tau_2 \mu_2 h \Theta_2 (1 + \alpha_1 \Theta_1)}, \quad (3.2)$$

and

$$\Theta_2 = \frac{\tau_2 \mu_2}{\langle k \rangle} \sum_{h=1}^n \frac{h^2 P(h) \Theta_2 (1 + \alpha_1 \Theta_1)}{(1 + \alpha_1 \Theta_1)(1 + \alpha_2 \Theta_2) + \tau_1 \mu_1 h \Theta_1 (1 + \alpha_2 \Theta_2) + \tau_2 \mu_2 h \Theta_2 (1 + \alpha_1 \Theta_1)}. \quad (3.3)$$

Obviously,  $(\Theta_1, \Theta_2) = (0, 0)$  is always a solution of (3.2) and (3.3). It follows from (3.1) that there is always a disease-free equilibrium  $E_0 : \rho_k = 0, \vartheta_k = 0$  ( $k = 1, 2, \dots, n$ ) for system (2.3). To find other equilibria, let us define

$$f_1(\Theta_1, \Theta_2) = \frac{\tau_1 \mu_1}{\langle k \rangle} \sum_{h=1}^n \frac{h^2 P(h)(1 + \alpha_2 \Theta_2)}{(1 + \alpha_1 \Theta_1)(1 + \alpha_2 \Theta_2) + \tau_1 \mu_1 h \Theta_1 (1 + \alpha_2 \Theta_2) + \tau_2 \mu_2 h \Theta_2 (1 + \alpha_1 \Theta_1)} - 1, \quad (3.4)$$

and

$$f_2(\Theta_1, \Theta_2) = \frac{\tau_2 \mu_2}{\langle k \rangle} \sum_{h=1}^n \frac{h^2 P(h)(1 + \alpha_1 \Theta_1)}{(1 + \alpha_1 \Theta_1)(1 + \alpha_2 \Theta_2) + \tau_1 \mu_1 h \Theta_1 (1 + \alpha_2 \Theta_2) + \tau_2 \mu_2 h \Theta_2 (1 + \alpha_1 \Theta_1)} - 1. \quad (3.5)$$

Then (3.2) and (3.3) are equivalent to  $\Theta_1 f_1(\Theta_1, \Theta_2) = 0$  and  $\Theta_2 f_2(\Theta_1, \Theta_2) = 0$ . Now, we identify the boundary cases, namely,  $\Theta_1 > 0, \Theta_2 = 0$  and  $\Theta_1 = 0, \Theta_2 > 0$ . For the former case, we look for  $0 < \Theta_1 < 1$  that satisfies  $f_1(\Theta_1, 0) = 0$ . That is,

$$f_1(\Theta_1, 0) = \frac{\tau_1 \mu_1}{\langle k \rangle} \sum_{h=1}^n \frac{h^2 P(h)}{1 + \alpha_1 \Theta_1 + \tau_1 \mu_1 h \Theta_1} - 1 = 0. \quad (3.6)$$

Because

$$f_1(1, 0) = \frac{\tau_1 \mu_1}{\langle k \rangle} \sum_{h=1}^n \frac{h^2 P(h)}{1 + \alpha_1 + \tau_1 \mu_1 h} - 1 < \frac{1}{\langle k \rangle} \sum_{h=1}^n h P(h) - 1 = 0,$$

and

$$\frac{\partial}{\partial \Theta_1} f_1(\Theta_1, 0) = \frac{\tau_1 \mu_1}{\langle k \rangle} \sum_{h=1}^n \frac{-h^2 P(h)(\alpha_1 + \tau_1 \mu_1 h)}{(1 + \alpha_1 \Theta_1 + \tau_1 \mu_1 h \Theta_1)^2} < 0.$$

Thus, a unique nontrivial solution of (3.6) exists if and only if  $f_1(0, 0) > 0$ , that is,

$$R_1 := \frac{\tau_1 \mu_1 \langle k^2 \rangle}{\langle k \rangle} = \frac{\beta_1 \mu_1 \langle k^2 \rangle}{\gamma_1 \langle k \rangle} > 1,$$

where  $\langle k^2 \rangle = \sum_{h=1}^n h^2 P(h)$  and  $R_1$  is the associated reproduction number for strain 1. This means that there exists a unique boundary equilibrium of strain 1 at  $E_1 : \bar{\rho}_k > 0, \bar{\vartheta}_k = 0$  ( $k = 1, 2, \dots, n$ ) if and only if  $R_1 > 1$ . A similar analysis for the later case (i.e.,  $\Theta_1 = 0, \Theta_2 > 0$ ) yields the existence of the other unique boundary equilibrium of strain 2 at  $E_2 : \tilde{\rho}_k = 0, \tilde{\vartheta}_k > 0$  ( $k = 1, 2, \dots, n$ ) if and only if  $R_2 > 1$ . Here

$$R_2 := \frac{\tau_2 \mu_2 \langle k^2 \rangle}{\langle k \rangle} = \frac{\beta_2 \mu_2 \langle k^2 \rangle}{\gamma_2 \langle k \rangle}$$

is the associated reproduction number for strain 2. Summarizing the above discussion, we have the following theorem for the existence of the disease-free and boundary equilibria.

**Theorem 3.1.** *Consider the two-strain model (2.3).*

- (1) *There exists always a disease-free equilibrium at  $E_0$ .*
- (2) *There exists a unique strain 1-only boundary equilibrium  $E_1$  if and only if  $R_1 > 1$ .*
- (3) *There exists a unique strain 2-only boundary equilibrium  $E_2$  if and only if  $R_2 > 1$ .*

Now, we are going to discuss the stability of each of the equilibria. By applying the linear stability analysis, we show that each of the equilibria exhibits stability under some threshold conditions. The following lemma is introduced to facilitate the stability analysis.

**Lemma 3.1.** ([25]) *For a real  $n \times n$  matrix  $A = (a_{ij})$  where  $a_{ij} = \delta_{ij}\sigma_i + p_iq_j$  ( $p_i, q_j \geq 0, i, j = 1, 2, \dots, n$ ) and  $\delta_{ij}$  is the Kronecker symbol, we have that the determinant of  $A$  is*

$$\det(A) = \sigma_1\sigma_2 \cdots \sigma_n + p_1q_1\sigma_2 \cdots \sigma_n + \sigma_1p_2q_2\sigma_3 \cdots \sigma_n + \cdots + \sigma_1\sigma_2 \cdots \sigma_{n-1}p_nq_n. \quad (3.7)$$

Specially, if  $\sigma_k \neq 0$  for all  $k$ , then

$$\det(A) = \left(1 + \sum_{k=1}^n \frac{p_kq_k}{\sigma_k}\right) \prod_{k=1}^n \sigma_k; \quad (3.8)$$

if  $\sigma_k = \sigma$  for all  $k$ , then

$$\det(A) = \sigma^{n-1} \left(\sigma + \sum_{k=1}^n p_kq_k\right). \quad (3.9)$$

We begin with the disease-free equilibrium  $E_0$ . Let  $R_0 = \max\{R_1, R_2\}$ , which defines the basic reproduction number for the system (2.3). The following two theorems relate the stability of  $E_0$  to the value of  $R_0$ .

**Theorem 3.2.**  $E_0$  is asymptotically stable if  $R_0 < 1$ ; and it becomes unstable if  $R_0 > 1$ .

*Proof.* The Jacobian matrix of system (2.3) evaluated at  $E_0$  is given by

$$A = \begin{pmatrix} A_1 & 0 \\ 0 & A_2 \end{pmatrix},$$

where

$$(A_1)_{ij} = -\gamma_1\delta_{ij} + \beta_1\mu_1ijP(j)/\langle k \rangle \quad \text{and} \quad (A_2)_{ij} = -\gamma_2\delta_{ij} + \beta_2\mu_2ijP(j)/\langle k \rangle.$$

Let  $\lambda(A)$  be the spectrum of the square matrix  $A$ . Since  $\lambda(A) = \lambda(A_1) \cup \lambda(A_2)$ , it suffices to determine the eigenvalues of matrix  $A_1$  and  $A_2$  respectively. In order to assess the eigenvalues of matrix  $A_1$  by Lemma 3.1, we denote the entries of matrix  $A_1 - \lambda I$  be  $a_{ij} = -(\gamma_1 + \lambda)\delta_{ij} + \beta_1\mu_1ijP(j)/\langle k \rangle$  for  $1 \leq i, j \leq n$ . Let  $\sigma_i = -(\gamma_1 + \lambda)$ ,  $p_i = \beta_1\mu_1i$ , and  $q_j = jP(j)/\langle k \rangle$ . From (3.9), we can derive that

$$\det(A_1 - \lambda I) = (-\gamma_1 - \lambda)^{n-1} \left(-\gamma_1 - \lambda + \frac{\beta_1\mu_1\langle k^2 \rangle}{\langle k \rangle}\right) = 0. \quad (3.10)$$

Similarly, we can obtain that

$$\det(A_2 - \lambda I) = (-\gamma_2 - \lambda)^{n-1} \left(-\gamma_2 - \lambda + \frac{\beta_2\mu_2\langle k^2 \rangle}{\langle k \rangle}\right) = 0. \quad (3.11)$$

It follows from (3.10) and (3.11) that all eigenvalues of  $A_1$  and  $A_2$  are real and negative if  $R_0 < 1$  and there exists a positive eigenvalue if  $R_0 > 1$ . Therefore, the disease-free equilibrium  $E_0$  is asymptotically stable if  $R_0 < 1$  and it is unstable if  $R_0 > 1$ . This completes the proof.  $\square$

**Remark 3.1.** *From Theorem 3.1 and 3.2, we can conclude that the existence of either boundary equilibrium implies the instability of the disease-free equilibrium  $E_0$ .*

**Theorem 3.3.** *If  $R_0 < 1$ , then  $E_0$  is indeed globally asymptotically stable.*

*Proof.* It suffices to show that  $E_0$  is globally attractive. Let  $\Theta(t) = \Theta_1(t) + \Theta_2(t)$ . According to (2.2) and (2.3), we have

$$\begin{aligned}
\Theta'(t) &= \Theta_1'(t) + \Theta_2'(t) \\
&= \frac{1}{\langle k \rangle} \sum_{h=1}^n hP(h) \{-\gamma_1 \rho_h(t) + \beta_1 h(1 - \rho_h(t) - \vartheta_h(t))g(\Theta_1(t)) - \gamma_2 \vartheta_h(t) + \beta_2 h(1 - \rho_h(t) - \vartheta_h(t))g(\Theta_2(t))\} \\
&\leq -\gamma_1 \Theta_1(t) + \frac{\beta_1 \mu_1 \langle k^2 \rangle}{\langle k \rangle} \frac{\Theta_1(t)}{1 + \alpha_1 \Theta_1(t)} - \gamma_2 \Theta_2(t) + \frac{\beta_2 \mu_2 \langle k^2 \rangle}{\langle k \rangle} \frac{\Theta_2(t)}{1 + \alpha_2 \Theta_2(t)} \\
&\leq -\gamma_1(1 - R_1)\Theta_1(t) - \gamma_2(1 - R_2)\Theta_2(t) \\
&\leq -\min\{\gamma_1(1 - R_1), \gamma_2(1 - R_2)\}\Theta(t).
\end{aligned}$$

Since  $R_0 < 1$ , we have  $\min\{\gamma_1(1 - R_1), \gamma_2(1 - R_2)\} = \zeta > 0$ . This implies that  $\Theta'(t) < -\zeta\Theta(t)$  and one can obtain  $\lim_{t \rightarrow \infty} \Theta(t) = 0$ . Due to  $\Theta_1(t) \geq 0$  and  $\Theta_2(t) \geq 0$ , we can further obtain  $\lim_{t \rightarrow \infty} \Theta_1(t) = 0$  and  $\lim_{t \rightarrow \infty} \Theta_2(t) = 0$ . Consequently, we get  $\lim_{t \rightarrow \infty} \rho_k(t) = 0$  and  $\lim_{t \rightarrow \infty} \vartheta_k(t) = 0$  for  $k = 1, 2, \dots, n$ . This proves that  $E_0$  is globally attractive if  $R_0 < 1$  and it follows from Theorem 3.2 that  $E_0$  is indeed globally asymptotically stable if  $R_0 < 1$ . This completes the proof.  $\square$

Our task now is to claim the stability of the boundary equilibria  $E_1$  and  $E_2$ . As we will see, the nonlinear incidence rate can have specific effects on the stability of  $E_1$  and  $E_2$ .

**Theorem 3.4.** *The stability of the boundary equilibria  $E_1$  and  $E_2$  is described as following:*

- (1) *Suppose the boundary equilibrium  $E_1$  exists (i.e.,  $R_1 > 1$ ), it is asymptotically stable if  $\alpha_1 < (R_1 - R_2)/(R_2 \bar{\Theta}_1)$ , and it becomes unstable if  $\alpha_1 > (R_1 - R_2)/(R_2 \bar{\Theta}_1)$ . Here  $\bar{\Theta}_1 = \sum_{h=1}^n hP(h)\bar{\rho}_h/\langle k \rangle$ .*
- (2) *Suppose the boundary equilibrium  $E_2$  exists (i.e.,  $R_2 > 1$ ), it is asymptotically stable if  $\alpha_2 < (R_2 - R_1)/(R_1 \bar{\Theta}_2)$ , and it becomes unstable if  $\alpha_2 > (R_2 - R_1)/(R_1 \bar{\Theta}_2)$ . Here  $\bar{\Theta}_2 = \sum_{h=1}^n hP(h)\bar{\vartheta}_h/\langle k \rangle$ .*

*Proof.* The Jacobian matrix of system (2.3) evaluated at  $E_1$  is now described as

$$B = \begin{pmatrix} B_1 & B_3 \\ 0 & B_2 \end{pmatrix},$$

where

$$(B_1)_{ij} = -\left(\gamma_1 + \beta_1 \mu_1 i \frac{\bar{\Theta}_1}{1 + \alpha_1 \bar{\Theta}_1}\right) \delta_{ij} + \frac{\beta_1 \mu_1}{\langle k \rangle (1 + \alpha_1 \bar{\Theta}_1)^2} ijP(j)(1 - \bar{\rho}_i) \quad \text{and} \quad (B_2)_{ij} = -\gamma_2 \delta_{ij} + \frac{\beta_2 \mu_2}{\langle k \rangle} ijP(j)(1 - \bar{\rho}_i).$$

To determine the stability of  $E_1$ , it is sufficiently to determine the eigenvalues of matrix  $B_1$  and  $B_2$  respectively. We now denote the entries of  $B_1 - \lambda I$  by  $b_{ij} = \sigma_i \delta_{ij} + p_i q_i$  for  $1 \leq i, j \leq n$ , where  $\sigma_i = -(\lambda + \gamma_1 + \beta_1 \mu_1 i \bar{\Theta}_1 / (1 + \alpha_1 \bar{\Theta}_1))$ ,  $p_i = \beta_1 \mu_1 i (1 - \bar{\rho}_i)$ , and  $q_i = iP(i) / (\langle k \rangle (1 + \alpha_1 \bar{\Theta}_1)^2)$ . According to (3.7),  $\sigma_i \neq 0$  for each  $i$ , otherwise there exist  $i \neq j$  such that  $\sigma_i = \sigma_j = 0$  (this is impossible in view of the expression for  $\sigma_i$ ). From (3.8),  $\det(B_1 - \lambda I) = 0$  is equivalent to

$$\frac{\beta_1 \mu_1}{\langle k \rangle} \sum_{h=1}^n \frac{h^2 P(h)}{\Delta(h)} = 1, \quad (3.12)$$

where

$$\Delta(h) = \frac{(1 + \alpha_1 \bar{\Theta}_1)^2}{1 - \bar{\rho}_h} \left( \lambda + \gamma_1 + \beta_1 \mu_1 h \frac{\bar{\Theta}_1}{1 + \alpha_1 \bar{\Theta}_1} \right).$$

Due to

$$1 - \bar{\rho}_h = \frac{\gamma_1 \bar{\rho}_h (1 + \alpha_1 \bar{\Theta}_1)}{\beta_1 \mu_1 h \bar{\Theta}_1}, \quad (3.13)$$

Equation (3.12) becomes the following equality

$$\sum_{h=1}^n \frac{hP(h)\bar{\rho}_h}{(1 + \alpha_1\bar{\Theta}_1)(\lambda + \gamma_1 + \beta_1\mu_1 h\bar{\Theta}_1/(1 + \alpha_1\bar{\Theta}_1))} = \sum_{h=1}^n \frac{hP(h)\bar{\rho}_h}{\gamma_1}, \quad (3.14)$$

which establishes  $n$  complex roots  $\lambda_\nu$  for  $\nu = 1, 2, \dots, n$ . Let  $a_\nu = \text{Re}(\lambda_\nu)$  and  $b_\nu = \text{Im}(\lambda_\nu)$ . Substituting each root  $\lambda_\nu$  into (3.14), we obtain

$$\sum_{h=1}^n \frac{hP(h)\bar{\rho}_h(\gamma_1 + a_\nu + \beta_1\mu_1 h\bar{\Theta}_1/(1 + \alpha_1\bar{\Theta}_1))}{(1 + \alpha_1\bar{\Theta}_1)\left(\left(\gamma_1 + a_\nu + \beta_1\mu_1 h\bar{\Theta}_1/(1 + \alpha_1\bar{\Theta}_1)\right)^2 + b_\nu^2\right)} = \sum_{h=1}^n \frac{hP(h)\bar{\rho}_h}{\gamma_1},$$

and

$$-b_\nu \sum_{h=1}^n \frac{hP(h)\bar{\rho}_h}{(1 + \alpha_1\bar{\Theta}_1)\left(\left(\gamma_1 + a_\nu + \beta_1\mu_1 h\bar{\Theta}_1/(1 + \alpha_1\bar{\Theta}_1)\right)^2 + b_\nu^2\right)} = 0. \quad (3.15)$$

Equation (3.15) implies that  $b_\nu = 0$  for all  $\nu$  and hence the eigenvalues  $\lambda_\nu$  are all real numbers. From (3.14), we can conclude that  $\lambda_\nu < 0$  for all  $\nu$ , that is, all eigenvalues of  $B_1$  are negative.

On the other hand, the entries of  $B_3 - \lambda I$  can be denoted as  $\bar{b}_{ij} = \bar{\sigma}_i \delta_{ij} + \bar{p}_i \bar{q}_i$  for  $1 \leq i, j \leq n$ , where  $\bar{\sigma}_i = -(\gamma_2 + \lambda)$ ,  $\bar{p}_i = \beta_2 \mu_2 i(1 - \bar{\rho}_i)$ , and  $\bar{q}_i = iP(i)/\langle k \rangle$ . Thanks to (3.9), we obtain

$$\det(B_3 - \lambda I) = (-\gamma_2 - \lambda)^{n-1} \left( -\gamma_2 - \lambda + \frac{\beta_2 \mu_2}{\langle k \rangle} \sum_{h=1}^n h^2 P(h)(1 - \bar{\rho}_h) \right) = 0.$$

Thus, the stability of  $E_1$  is completely determined by the term

$$-\gamma_2 - \lambda + \frac{\beta_2 \mu_2}{\langle k \rangle} \sum_{h=1}^n h^2 P(h)(1 - \bar{\rho}_h) = 0.$$

Using (3.13) we can derive that

$$\lambda = -\gamma_2 + \frac{\beta_2 \mu_2}{\langle k \rangle} \sum_{h=1}^n h^2 P(h)(1 - \bar{\rho}_h) = -\gamma_2 \left( 1 - \frac{R_2}{R_1} (1 + \alpha_1 \bar{\Theta}_1) \right).$$

Thus if  $\alpha_1 < (R_1 - R_2)/(R_2 \bar{\Theta}_1)$ , then  $\lambda < 0$  and this implies that the boundary equilibrium  $E_1$  is asymptotically stable. Obviously, it becomes unstable provided  $\alpha_1 > (R_1 - R_2)/(R_2 \bar{\Theta}_1)$ . According to the symmetry of system (2.3), we can also apply the above analysis to the equilibrium  $E_2$ . Therefore, we can conclude that if  $E_2$  exists, then  $E_2$  is asymptotically stable provided  $\alpha_2 < (R_2 - R_1)/(R_1 \bar{\Theta}_2)$  and unstable provided  $\alpha_2 > (R_2 - R_1)/(R_1 \bar{\Theta}_2)$ . This completes the proof.  $\square$

**Remark 3.2.** Notice that the parameters  $\tau_i = \beta_i/\gamma_i$ ,  $i = 1, 2$ , can be referred to as the effective spreading rates for strain 1 and strain 2, respectively. Furthermore, it is easy to see that  $\tau_i$  is proportional to the associated reproduction number  $R_i$  for each  $i$ . Therefore, the biological meaning about the theoretical results can be stated as follows:

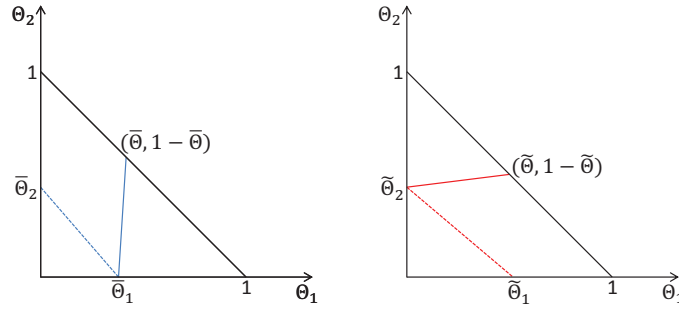
- (1) Either small transmission rate  $\beta_i$  or large cure rate  $\gamma_i$  can reduce the corresponding effective spreading rate  $\tau_i$ . Thus, if  $\tau_1$  and  $\tau_2$  could be reduced so that  $R_1$  and  $R_2$  are both less than one, then Theorem 3.3 indicates that both strains will eventually die out.
- (2) If  $R_j < 1 < R_i$  for  $i, j = 1, 2$  and  $i \neq j$ , then the boundary equilibrium  $E_j$  does not exist from Theorem 3.1. Furthermore, Theorem 3.4 indicates that the boundary equilibrium  $E_i$  is asymptotically stable provided that the parameter  $\alpha_i$  is small enough. This means that competitive exclusion occurs (i.e., the strain with the larger reproduction number will persist and eliminate the other strain).



(3) If  $R_1 > 1$  and  $R_2 > 1$ , the situation becomes more complicated. In this case, either competitive exclusion or strain coexistence could occur. For example, if  $1 < R_2 < R_1$ , then Theorem 3.4 shows that  $E_2$  is unstable and  $E_1$  is asymptotically stable whenever  $\alpha_1$  is small enough. Consequently, a competing-exclusion exists. On the other hand, if  $\alpha_1$  is large so that  $E_1$  is unstable, then the two strains could coexist under some additional conditions which will be given in Section 4. Therefore, we shall emphasize here that the conditions  $R_1 > 1$  and  $R_2 > 1$  cannot guarantee the strain coexistence, and we will address this issue in the following section.

#### 4. Coexistence equilibrium

In this section, we will propose some conditions that ensure the existence of the coexistence equilibrium  $E^* : \rho_k^* > 0, \vartheta_k^* > 0$  ( $k = 1, 2, \dots, n$ ). To this end, we need to find a positive solution (i.e.,  $\Theta_1 > 0$  and  $\Theta_2 > 0$ ) of (3.2) and (3.3). This is equivalent to find a positive solution of  $f_1(\Theta_1, \Theta_2) = 0$  and  $f_2(\Theta_1, \Theta_2) = 0$  where the functions  $f_1$  and  $f_2$  are defined in (3.4) and (3.5), respectively. In the following, we assume that  $R_1 > 1$  and  $R_2 > 1$ , and so both boundary equilibria exist. Now we start to find the existence condition for  $E^*$  based on the idea of [19]. Since  $\partial f_1(\Theta_1, \Theta_2)/\partial \Theta_1 < 0$  and  $\partial f_1(\Theta_1, \Theta_2)/\partial \Theta_2 < 0$ , there is a curve  $f_1(\Theta_1, \Theta_2) = 0$  in the  $\Theta_1\Theta_2$ -plane that looks like the dashed line if  $f_1(0, 1) < 0$ , or solid line if  $f_1(0, 1) > 0$  in the left panel of Figure 2. Similarly, we have  $\partial f_2(\Theta_1, \Theta_2)/\partial \Theta_1 < 0$  and  $\partial f_2(\Theta_1, \Theta_2)/\partial \Theta_2 < 0$ , and hence a curve  $f_2(\Theta_1, \Theta_2) = 0$  is located as the dashed line if  $f_2(1, 0) < 0$ , or the solid line if  $f_2(1, 0) > 0$  in the right panel of Figure 2.



**Figure 2.** (Left) The curves  $f_1(\Theta_1, \Theta_2) = 0$ :  $f_1(0, 1) < 0$  (blue dashed line) or  $f_1(0, 1) > 0$  (blue solid line). (Right) The curves  $f_2(\Theta_1, \Theta_2) = 0$ :  $f_2(1, 0) < 0$  (red dashed line) or  $f_2(1, 0) > 0$  (red solid line).

Since  $R_1 > 1$  and  $R_2 > 1$ , it follows from Theorem 3.1 that there exist  $0 < \bar{\Theta}_1 < 1$  and  $0 < \bar{\Theta}_2 < 1$  so that  $f_1(\bar{\Theta}_1, 0) = 0$  and  $f_2(0, \bar{\Theta}_2) = 0$ . Furthermore, we can derive that

$$f_1(\bar{\Theta}_1, 0) = 0 \Leftrightarrow \frac{1}{\langle k \rangle} \sum_{h=1}^n \frac{h^2 P(h)}{1 + \alpha_1 \bar{\Theta}_1 + \tau_1 \mu_1 h \bar{\Theta}_1} = \frac{1}{\tau_1 \mu_1}, \quad (4.1)$$

and

$$f_2(0, \bar{\Theta}_2) = 0 \Leftrightarrow \frac{1}{\langle k \rangle} \sum_{h=1}^n \frac{h^2 P(h)}{1 + \alpha_2 \bar{\Theta}_2 + \tau_2 \mu_2 h \bar{\Theta}_2} = \frac{1}{\tau_2 \mu_2}. \quad (4.2)$$

Note that the coexistence equilibrium  $E^*$  is the intersection of the two curves  $f_1(\Theta_1, \Theta_2) = 0$  and  $f_2(\Theta_1, \Theta_2) = 0$ . We consider four separate groups of assumptions that can each lead to the existence of  $E^*$ :

- (a) If  $f_1(0, 1) > 0$  and  $f_2(1, 0) < 0$ , then there exist  $0 < \bar{\Theta}_1 < 1$  and  $0 < \bar{\Theta}_2 < 1$  satisfying  $f_1(\bar{\Theta}_1, 1 - \bar{\Theta}_2) = 0$  and  $f_2(\bar{\Theta}_1, 0) = 0$ . Therefore,  $f_1(\Theta_1, \Theta_2) = 0$  and  $f_2(\Theta_1, \Theta_2) = 0$  cross when  $f_2(\bar{\Theta}_1, 0) > 0$  (cf. Figure 3(a)). From (4.1), one can derive that

$$f_2(\bar{\Theta}_1, 0) = \frac{\tau_2 \mu_2}{\langle k \rangle} \sum_{h=1}^n \frac{h^2 P(h)(1 + \alpha_1 \bar{\Theta}_1)}{1 + \alpha_1 \bar{\Theta}_1 + \tau_1 \mu_1 h \bar{\Theta}_1} - 1 = \frac{\tau_2 \mu_2}{\tau_1 \mu_1} (1 + \alpha_1 \bar{\Theta}_1) - 1 > 0 \Leftrightarrow \alpha_1 > \frac{R_1 - R_2}{R_2 \bar{\Theta}_1}.$$

From Theorem 3.4(1), this is the instability condition for  $E_1$ . Hence,  $E^*$  exists if the boundary equilibrium  $E_1$  is unstable in this case.



- (b) If  $f_1(0, 1) < 0$  and  $f_2(1, 0) < 0$ , then there exist  $0 < \tilde{\Theta}_1 < 1$  and  $0 < \tilde{\Theta}_2 < 1$  satisfying  $f_1(0, \tilde{\Theta}_2) = 0$  and  $f_2(\tilde{\Theta}_1, 0) = 0$ . In particular, we have two subcases to make the two curves  $f_1(\Theta_1, \Theta_2) = 0$  and  $f_2(\Theta_1, \Theta_2) = 0$  cross: (i)  $f_1(0, \tilde{\Theta}_2) < 0$  and  $f_2(\tilde{\Theta}_1, 0) < 0$  (cf. Figure 3(b)); (ii)  $f_1(0, \tilde{\Theta}_2) > 0$  and  $f_2(\tilde{\Theta}_1, 0) > 0$  (cf. Figure 3(c)). We now claim that subcase(i) is impossible. Indeed, it follows from (4.1) and (4.2) that

$$f_1(0, \tilde{\Theta}_2) < 0 \Leftrightarrow \alpha_2 < \frac{R_2 - R_1}{R_1 \tilde{\Theta}_2} \quad \text{and} \quad f_2(\tilde{\Theta}_1, 0) < 0 \Leftrightarrow \alpha_1 < \frac{R_1 - R_2}{R_2 \tilde{\Theta}_1}.$$

This implies that either  $\alpha_1 < 0$  or  $\alpha_2 < 0$ , which is apparently a contradiction. For subcase(ii), one can easily obtain that

$$f_1(0, \tilde{\Theta}_2) > 0 \Leftrightarrow \alpha_2 > \frac{R_2 - R_1}{R_1 \tilde{\Theta}_2} \quad \text{and} \quad f_2(\tilde{\Theta}_1, 0) > 0 \Leftrightarrow \alpha_1 > \frac{R_1 - R_2}{R_2 \tilde{\Theta}_1}.$$

Hence, in this case, it can be concluded from Theorem 3.4 that  $E^*$  exists if both  $E_1$  and  $E_2$  are unstable.

- (c) If  $f_1(0, 1) > 0$  and  $f_2(1, 0) > 0$ , then there exist  $0 < \bar{\Theta} < 1$  and  $0 < \tilde{\Theta} < 1$  satisfying  $f_1(\bar{\Theta}, 1 - \bar{\Theta}) = 0$  and  $f_2(\tilde{\Theta}, 1 - \tilde{\Theta}) = 0$ . Therefore,  $f_1(\Theta_1, \Theta_2) = 0$  and  $f_2(\Theta_1, \Theta_2) = 0$  cross when  $f_1(\tilde{\Theta}, 1 - \tilde{\Theta}) < 0$  and  $f_2(\bar{\Theta}, 1 - \bar{\Theta}) < 0$  (cf. Figure 3(d)). Using  $f_2(\tilde{\Theta}, 1 - \tilde{\Theta}) = 0$ , we have

$$f_1(\tilde{\Theta}, 1 - \tilde{\Theta}) < 0 \Leftrightarrow \frac{1 + \alpha_2(1 - \tilde{\Theta})}{1 + \alpha_1 \tilde{\Theta}} < \frac{R_2}{R_1}.$$

Similarly, the condition  $f_1(\bar{\Theta}, 1 - \bar{\Theta}) = 0$  implies that

$$f_2(\bar{\Theta}, 1 - \bar{\Theta}) < 0 \Leftrightarrow \frac{1 + \alpha_2(1 - \bar{\Theta})}{1 + \alpha_1 \bar{\Theta}} > \frac{R_2}{R_1}.$$

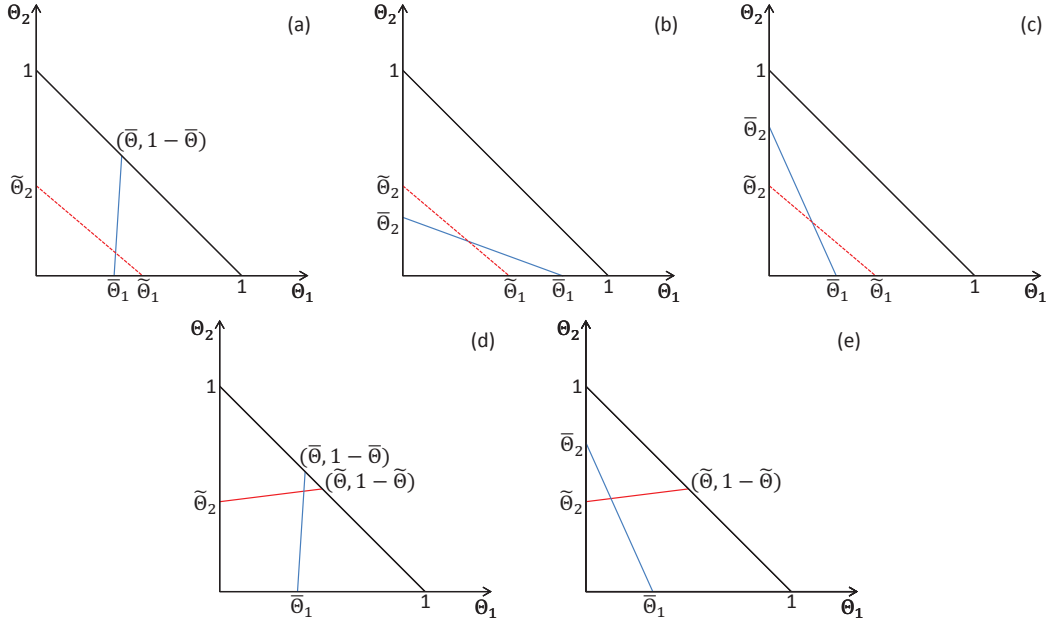
As a result,  $E^*$  exists if

$$\frac{1 + \alpha_2(1 - \tilde{\Theta})}{1 + \alpha_1 \tilde{\Theta}} < \frac{R_2}{R_1} < \frac{1 + \alpha_2(1 - \bar{\Theta})}{1 + \alpha_1 \bar{\Theta}}.$$

- (d) If  $f_1(0, 1) < 0$  and  $f_2(1, 0) > 0$ , then there exist  $0 < \bar{\Theta}_2 < 1$  and  $0 < \tilde{\Theta} < 1$  satisfying  $f_1(0, \bar{\Theta}_2) = 0$  and  $f_2(\tilde{\Theta}, 1 - \tilde{\Theta}) = 0$ . Therefore,  $f_1(\Theta_1, \Theta_2) = 0$  and  $f_2(\Theta_1, \Theta_2) = 0$  cross when  $f_1(0, \bar{\Theta}_2) > 0$  (cf. Figure 3(e)). From (4.2), one can derive that

$$f_1(0, \bar{\Theta}_2) = \frac{\tau_1 \mu_1}{\langle k \rangle} \sum_{h=1}^n \frac{h^2 P(h)(1 + \alpha_2 \bar{\Theta}_2)}{1 + \alpha_2 \bar{\Theta}_2 + \tau_2 \mu_2 h \bar{\Theta}_2} - 1 = \frac{\tau_1 \mu_1}{\tau_2 \mu_2} (1 + \alpha_2 \bar{\Theta}_2) - 1 > 0 \Leftrightarrow \alpha_2 > \frac{R_2 - R_1}{R_1 \bar{\Theta}_2}.$$

Therefore, in this case,  $E^*$  exists if the boundary equilibrium  $E_2$  is unstable.



**Figure 3.** Five possibilities for the intersection of the two curves  $f_1(\Theta_1, \Theta_2) = 0$  and  $f_2(\Theta_1, \Theta_2) = 0$ .

We summarize these findings in the following theorem:

**Theorem 4.1.** *Assume that  $\min\{R_1, R_2\} > 1$ . This implies that there exist  $\bar{\Theta}_1, \bar{\Theta}_2 \in (0, 1)$  so that  $f_1(\bar{\Theta}_1, 0) = 0$  and  $f_2(0, \bar{\Theta}_2) = 0$ . If one of the following conditions holds, then a coexistence equilibrium of system (2.3) exists.*

- (1)  $f_1(0, 1) > 0$ ,  $f_2(1, 0) < 0$ , and  $\alpha_1 > (R_1 - R_2)/(R_2\bar{\Theta}_1)$ .
- (2)  $f_1(0, 1) < 0$ ,  $f_2(1, 0) < 0$ ,  $\alpha_1 > (R_1 - R_2)/(R_2\bar{\Theta}_1)$ , and  $\alpha_2 > (R_2 - R_1)/(R_1\bar{\Theta}_2)$ .
- (3)  $f_1(0, 1) > 0$ ,  $f_2(1, 0) > 0$ , and  $(1 + \alpha_2(1 - \bar{\Theta})) / (1 + \alpha_1\bar{\Theta}) < R_2/R_1 < (1 + \alpha_2(1 - \bar{\Theta})) / (1 + \alpha_1\bar{\Theta})$  where  $\bar{\Theta}, \bar{\Theta} \in (0, 1)$  are respectively satisfy  $f_1(\bar{\Theta}, 1 - \bar{\Theta}) = 0$  and  $f_2(\bar{\Theta}, 1 - \bar{\Theta}) = 0$ .
- (4)  $f_1(0, 1) < 0$ ,  $f_2(1, 0) > 0$ , and  $\alpha_2 > (R_2 - R_1)/(R_1\bar{\Theta}_2)$ .

Before the end of this section, we make some comparison of our results with other references in the following remark.

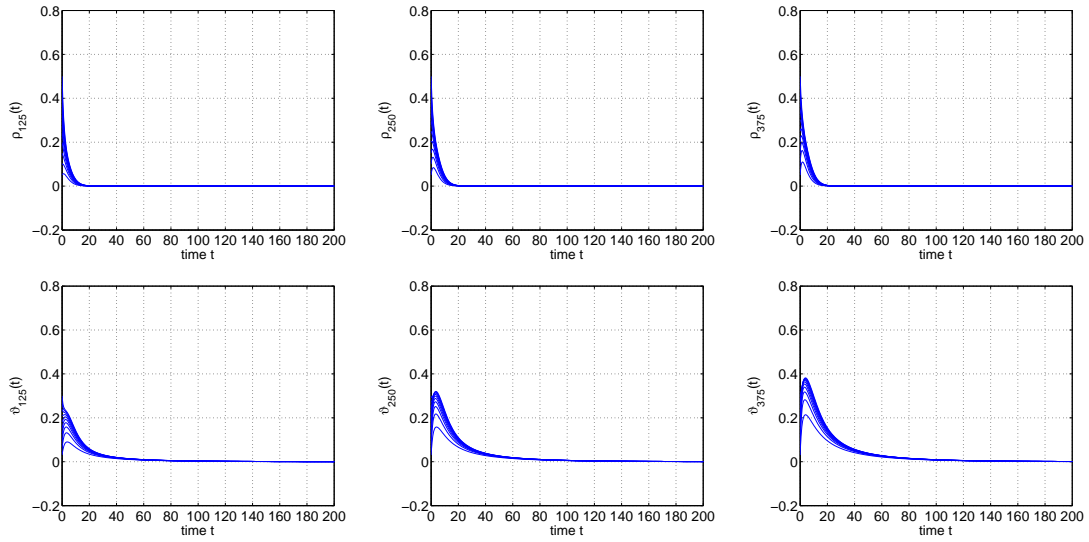
**Remark 4.1.**

- (1) *If  $\mu_1 = \mu_2 = 1$  and  $\alpha_1 = \alpha_2 = 0$ , then system (2.3) can be reduced to system (2.1), which is proposed in [24]. The main results in [24] indicate that if the effective spreading rates  $\tau_1 \neq \tau_2$ , only competitive exclusion can occur and strain coexistence is impossible except for the degenerate case (i.e.,  $\tau_1 = \tau_2$ ). However, our results show that strain coexistence is possible for non-degenerate case when the saturating incidence rate has been incorporated.*
- (2) *Wu et al. [25] studied the role of the superinfection rate on the epidemic spreading. They showed that two forms of dynamical behaviors, that is, strain coexistence and strain replacement, can be generated with the superinfection rate increasing from 0 to 1. However, sufficient conditions for the existence of the coexistence equilibrium does not obtain therein. In the present paper, we investigate another mechanism to allow the coexistence of different strains. Though our two-strain model cannot exhibit the strain replacement dynamics, some sufficient conditions that ensure the existence of the coexistence equilibrium are derived.*

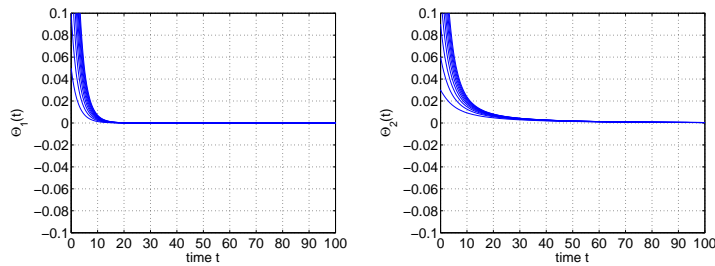
## 5. Numerical experiments

In this section, we will give some simulations to illustrate our theoretical results. We consider the system (2.3) on a scale-free network [4] with degree distribution  $P(k) = \eta k^{-2.5}$  for  $k = 1, 2, \dots, 500$ , and constant  $\eta$  makes sure that  $\sum_{k=1}^{500} P(k) = 1$ . In this situation, we can numerically obtain  $\langle k \rangle = 1.8808$  and  $\langle k^2 \rangle = 32.2666$ .

**Example 5.1.** We first consider the stability of the disease-free equilibrium  $E_0$ . In this example, we choose  $\alpha_1 = 9, \alpha_2 = 10, \beta_1 = 0.001, \beta_2 = 0.002, \gamma_1 = 0.5, \gamma_2 = 0.4, \mu_1 = 10, \mu_2 = 11$ . Thus, one can obtain  $R_1 = 0.3431 < 1$  and  $R_2 = 0.9436 < 1$ . It follows from Theorem 3.3 that the disease-free equilibrium  $E_0$  is globally asymptotically stable. We use 10 different initial conditions to plot the trajectories of  $\rho_k(t)$  and  $\vartheta_k(t)$  for  $k = 125, 250, 375$  in Figure 4. And we also display  $\Theta_1(t)$  and  $\Theta_2(t)$  in Figure 5 with the same initial conditions. As we can see in Figure 4 and Figure 5, it suggests that  $E_0$  is indeed globally asymptotically stable.



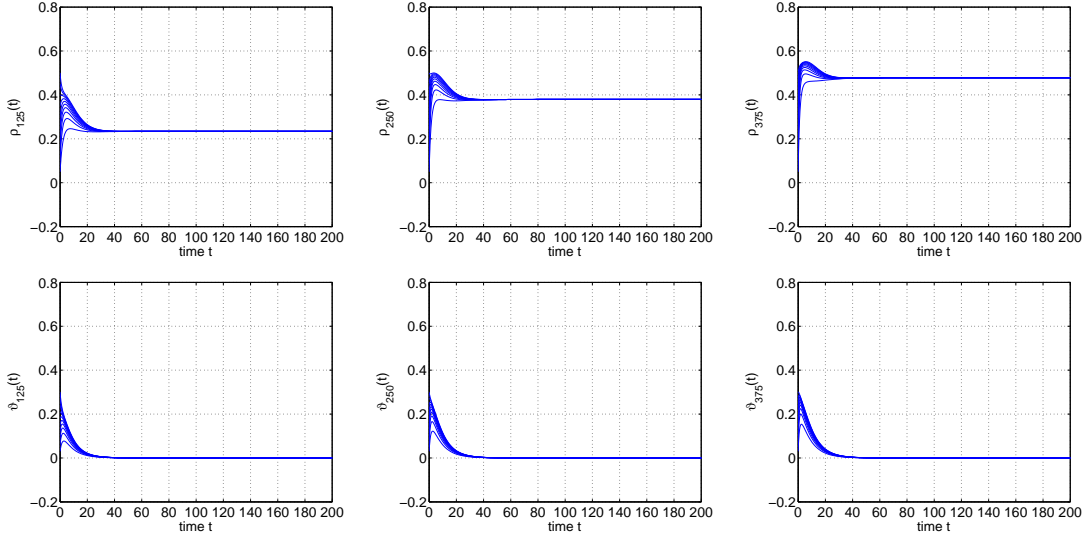
**Figure 4.** Results for system (2.3) with  $\alpha_1 = 9, \alpha_2 = 10, \beta_1 = 0.001, \beta_2 = 0.002, \gamma_1 = 0.5, \gamma_2 = 0.4, \mu_1 = 10, \mu_2 = 11$ . The 10 different initial conditions are given by  $\rho_k(0) = 0.05i$  and  $\vartheta_k(0) = 0.03i$  for  $i = 1, 2, \dots, 10$ .



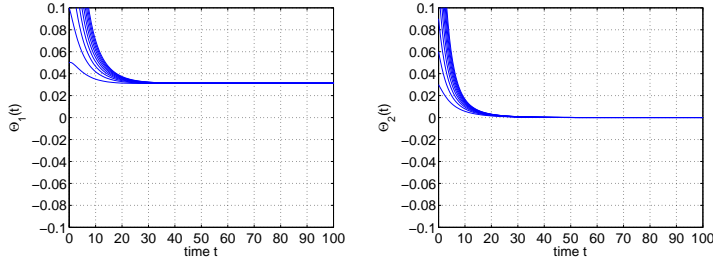
**Figure 5.** Time evolutions of  $\Theta_1(t)$  and  $\Theta_2(t)$  in Example 5.1.

**Example 5.2.** In this example, we choose  $\alpha_1 = 9, \alpha_2 = 10, \beta_1 = 0.003, \beta_2 = 0.002, \gamma_1 = 0.3, \gamma_2 = 0.4, \mu_1 = 10, \mu_2 = 11$ . Thus, one can obtain  $R_1 = 1.7156 > 1$  and  $R_2 = 0.9436 < 1$ . In this situation,  $E_0$  is unstable and  $E_2$  does not exist. Besides, we can obtain  $\bar{\Theta}_1 = 0.0313$  numerically. Thus, we have  $\alpha_1 = 9 < (R_1 - R_2)/(R_2 \bar{\Theta}_1) = 26.1388$ . It follows from Theorem 3.4 that  $E_1$  is asymptotically stable. We plot the trajectories of  $\rho_k(t)$  and  $\vartheta_k(t)$  for  $k = 125, 250, 375$  in Figure 6. The time evolutions of  $\Theta_1(t)$  and  $\Theta_2(t)$  are depicted in Figure 7. One can observe that only strain 1 dominates the prevalence.

**Example 5.3.** In this example, the parameters are chosen as  $\alpha_1 = 10, \alpha_2 = 9, \beta_1 = 0.003, \beta_2 = 0.003, \gamma_1 = 0.4, \gamma_2 = 0.5, \mu_1 = 11, \mu_2 = 10$ . Subsequently, we have  $R_1 = 1.4153 > 1$  and  $R_2 = 1.0293 > 1$ . In this situation,  $E_0$  is unstable and both  $E_1$  and  $E_2$  exist. In addition, we can get  $\bar{\Theta}_1 = 0.0185$  and  $\tilde{\Theta}_2 = 0.0015$  numerically. Therefore, we can check that  $\alpha_1 < (R_1 - R_2)/(R_2 \bar{\Theta}_1) = 20.2709$  and  $\alpha_2 > (R_2 - R_1)/(R_1 \tilde{\Theta}_2) = -181.8225$ . By Theorem 3.4, we know that  $E_1$  is asymptotically stable and  $E_2$  is unstable. Hence, we predict that strain 1 prevails over the network. The trajectories of  $\rho_k(t)$  and  $\vartheta_k(t)$  for  $k = 125, 250, 375$  are drawn in Figure 8. The time evolutions of  $\Theta_1(t)$  and  $\Theta_2(t)$  are depicted in Figure 9. Indeed, only strain 1 spreads eventually.



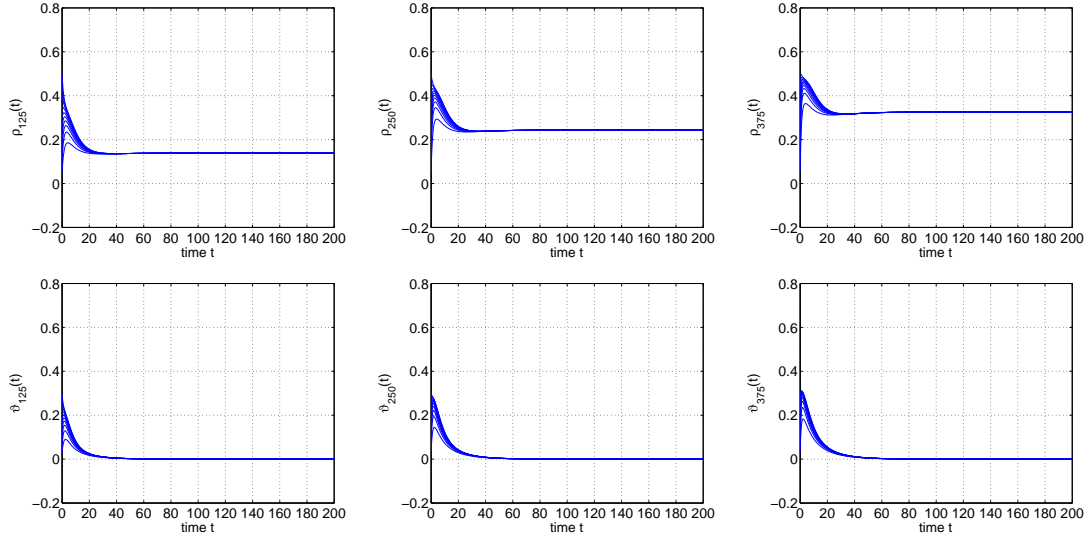
**Figure 6.** Results for system (2.3) with  $\alpha_1 = 9, \alpha_2 = 10, \beta_1 = 0.003, \beta_2 = 0.002, \gamma_1 = 0.3, \gamma_2 = 0.4, \mu_1 = 10, \mu_2 = 11$ . The 10 different initial conditions are given by  $\rho_k(0) = 0.05i$  and  $\vartheta_k(0) = 0.03i$  for  $i = 1, 2, \dots, 10$ .



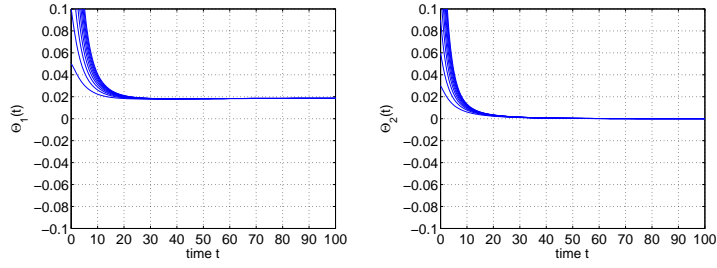
**Figure 7.** Time evolutions of  $\Theta_1(t)$  and  $\Theta_2(t)$  in Example 5.2.

In the following three examples, we will demonstrate the existence of the coexistence equilibrium  $E^*$  discussed in Section 4. Since case (a) and (d) are symmetric, we only give an example (i.e., Example 5.4) for case (a).

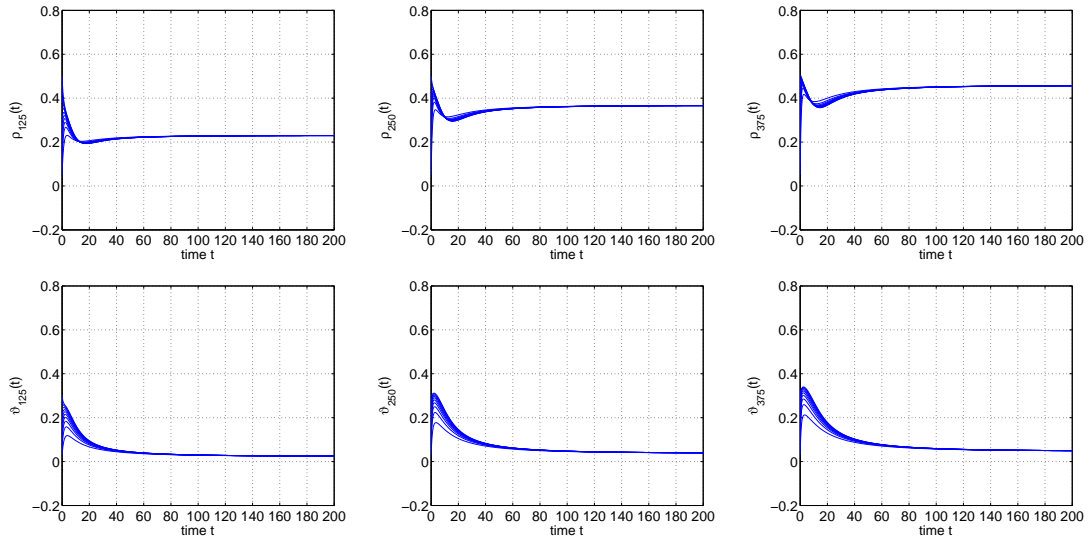
**Example 5.4.** Let  $\alpha_1 = 13, \alpha_2 = 10, \beta_1 = 0.004, \beta_2 = 0.003, \gamma_1 = 0.5, \gamma_2 = 0.4, \mu_1 = 14, \mu_2 = 11$ . We can obtain  $R_1 = 1.9214 > 1, R_2 = 1.4153 > 1, f_1(0, 1) = 0.0571 > 0$ , and  $f_2(1, 0) = -0.2380 < 0$ . In this situation, we know that  $E_0$  is unstable and both  $E_1$  and  $E_2$  exist. Besides, we can obtain  $\bar{\Theta}_1 = 0.0325$  numerically, and subsequently, one can claim that  $\alpha_1 > (R_1 - R_2)/(R_2 \bar{\Theta}_1) = 11.0028$ . By Theorem 4.1(1), the coexistence equilibrium  $E^*$  exists. The trajectories of  $\rho_k(t)$  and  $\vartheta_k(t)$  for  $k = 125, 250, 375$  are plotted in Figure 10. The time evolutions of  $\Theta_1(t)$  and  $\Theta_2(t)$  are depicted in Figure 11. We can observe that two competing strains co-exist. Moreover, numerical results suggest that  $E^*$  is asymptotically stable.



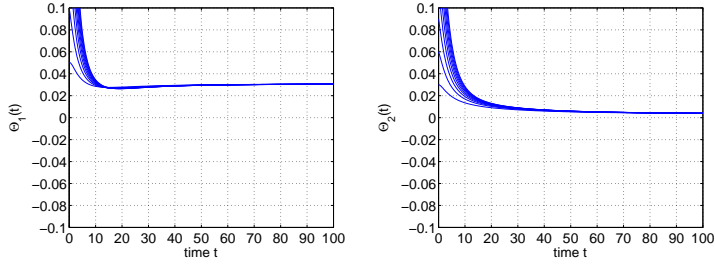
**Figure 8.** Results for system (2.3) with  $\alpha_1 = 10, \alpha_2 = 9, \beta_1 = 0.003, \beta_2 = 0.003, \gamma_1 = 0.4, \gamma_2 = 0.5, \mu_1 = 11, \mu_2 = 10$ . The 10 different initial conditions are given by  $\rho_k(0) = 0.05i$  and  $\vartheta_k(0) = 0.03i$  for  $i = 1, 2, \dots, 10$ .



**Figure 9.** Time evolutions of  $\Theta_1(t)$  and  $\Theta_2(t)$  in Example 5.3.

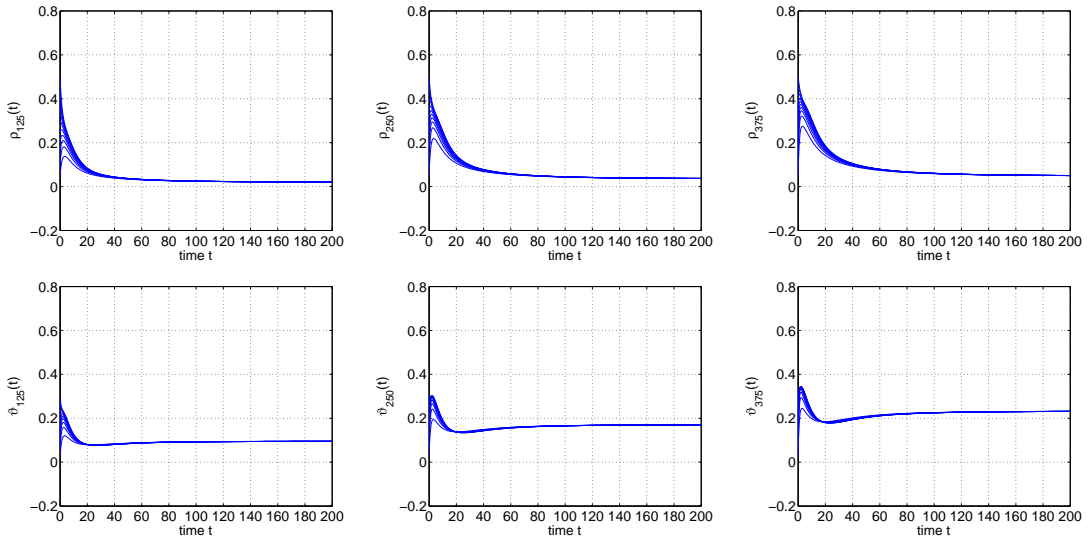


**Figure 10.** Results for system (2.3) with  $\alpha_1 = 13, \alpha_2 = 10, \beta_1 = 0.004, \beta_2 = 0.003, \gamma_1 = 0.5, \gamma_2 = 0.4, \mu_1 = 14, \mu_2 = 11$ . The 10 different initial conditions are given by  $\rho_k(0) = 0.05i$  and  $\vartheta_k(0) = 0.03i$  for  $i = 1, 2, \dots, 10$ .

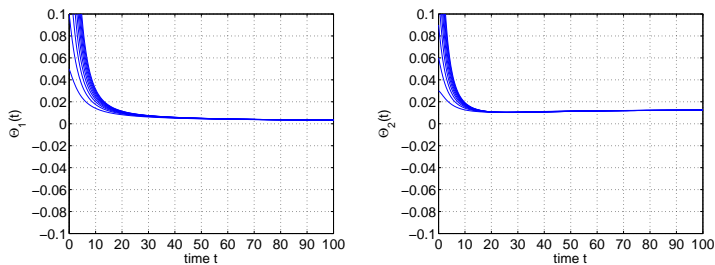


**Figure 11.** Time evolutions of  $\Theta_1(t)$  and  $\Theta_2(t)$  in Example 5.4.

**Example 5.5.** Now, we set  $\alpha_1 = 13, \alpha_2 = 12, \beta_1 = 0.002, \beta_2 = 0.003, \gamma_1 = 0.4, \gamma_2 = 0.5, \mu_1 = 14, \mu_2 = 13$ . One can derive that  $R_1 = 1.2009 > 1, R_2 = 1.3381 > 1, f_1(0, 1) = -0.2901 < 0$ , and  $f_2(1, 0) = -0.1645 < 0$ . Hence, both  $E_1$  and  $E_2$  exist. Furthermore, we can obtain  $\bar{\Theta}_1 = 0.0082$  and  $\bar{\Theta}_2 = 0.0140$ . Therefore, one can claim that  $\alpha_1 > (R_1 - R_2)/(R_2 \bar{\Theta}_1) = -12.5041$  and  $\alpha_2 > (R_2 - R_1)/(R_1 \bar{\Theta}_2) = 8.1605$ . It follows from Theorem 4.1(2) that the coexistence equilibrium  $E^*$  exists. We plot the trajectories of  $\rho_k(t)$  and  $\vartheta_k(t)$  for  $k = 125, 250, 375$  in Figure 12. The time evolutions of  $\Theta_1(t)$  and  $\Theta_2(t)$  are depicted in Figure 13. Indeed, the strain coexistence can be observed therein and it also seems that  $E^*$  is asymptotically stable in this case.



**Figure 12.** Results for system (2.3) with  $\alpha_1 = 13, \alpha_2 = 12, \beta_1 = 0.002, \beta_2 = 0.003, \gamma_1 = 0.4, \gamma_2 = 0.5, \mu_1 = 14, \mu_2 = 13$ . The 10 different initial conditions are given by  $\rho_k(0) = 0.05i$  and  $\vartheta_k(0) = 0.03i$  for  $i = 1, 2, \dots, 10$ .



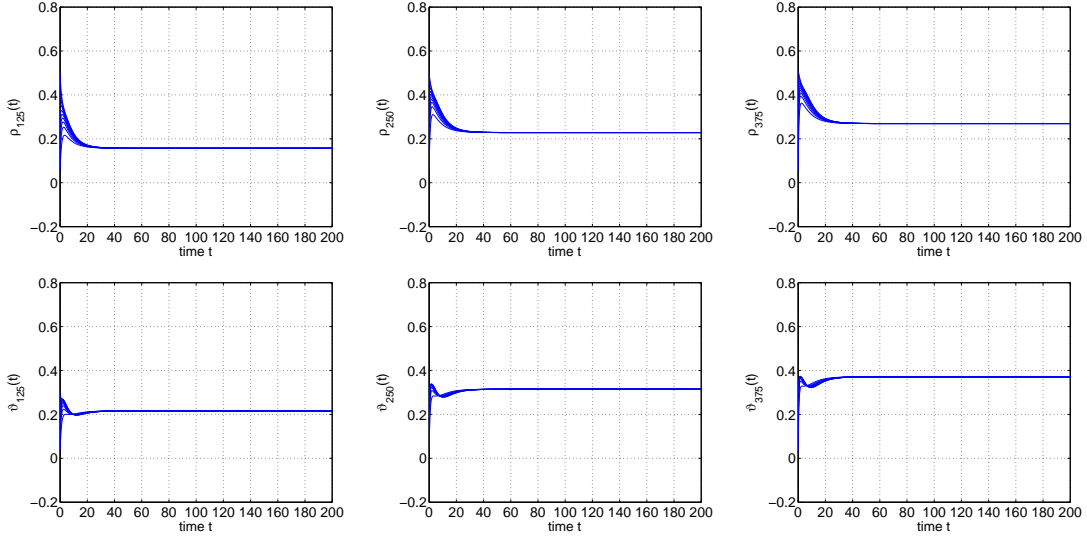
**Figure 13.** Time evolutions of  $\Theta_1(t)$  and  $\Theta_2(t)$  in Example 5.5.

**Example 5.6.** In the final example, we set  $\alpha_1 = 14, \alpha_2 = 15, \beta_1 = 0.004, \beta_2 = 0.005, \gamma_1 = 0.5, \gamma_2 = 0.6, \mu_1 = 15, \mu_2 = 16$ . It can be verified that  $R_1 = 2.0587 > 1, R_2 = 2.2874 > 1, f_1(0, 1) = 0.0930 > 0$ , and  $f_2(1, 0) = 0.2315 > 0$ .

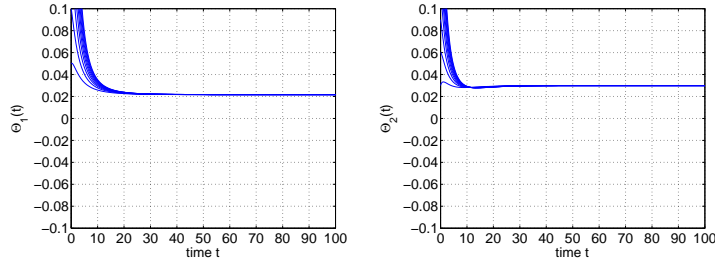
Hence, both  $E_1$  and  $E_2$  exist. Furthermore, we can obtain  $\bar{\Theta} = 0.0049$  and  $\tilde{\Theta} = 0.9887$ . Therefore, we have

$$\frac{1 + \alpha_2(1 - \bar{\Theta})}{1 + \alpha_1\tilde{\Theta}} = 0.0788 < \frac{R_2}{R_1} = 1.1111 < \frac{1 + \alpha_2(1 - \bar{\Theta})}{1 + \alpha_1\bar{\Theta}} = 14.9041.$$

It follows from Theorem 4.1(3) that the coexistence equilibrium  $E^*$  exists. Again, one can see the coexistence behavior in Figure 14 and 15. Besides, in this case, numerical results also suggest that  $E^*$  is asymptotically stable.



**Figure 14.** Results for system (2.3) with  $\alpha_1 = 14, \alpha_2 = 14, \beta_1 = 0.004, \beta_2 = 0.005, \gamma_1 = 0.5, \gamma_2 = 0.6, \mu_1 = 15, \mu_2 = 16$ . The 10 different initial conditions are given by  $\rho_k(0) = 0.05i$  and  $\vartheta_k(0) = 0.03i$  for  $i = 1, 2, \dots, 10$ .



**Figure 15.** Time evolutions of  $\Theta_1(t)$  and  $\Theta_2(t)$  in Example 5.6.

## 6. Concluding remarks

In this paper, we have studied the dynamics of a two-strain epidemic model with a competing mechanism and a saturating incidence rate on complex networks. The associated reproduction numbers for each strain are obtained. Furthermore, we show that if the maximum of the associated reproduction numbers of the two strains is less than one, then the disease-free equilibrium  $E_0$  is globally asymptotically stable, and so the disease dies out. Otherwise, there exists either a unique strain 1-only or a strain 2-only boundary equilibrium. The stability of each boundary equilibrium is also derived under some threshold condition. We also propose some conditions that ensure the existence of the coexistence equilibrium. Several numerical simulations are given to support our theoretical results. From the numerical results, we have observed that the coexistence equilibrium seems probable to be asymptotically stable. However, the detailed analysis of its stability of remains a challenge problem. In summary, the saturating incidence rate not only can have specific effects on the stability of the boundary equilibria, but also can allow the emerge of coexistence.



As aforementioned, the associated reproduction numbers for the two strains play an important role in whether or not a two-strain epidemic spreads in complex networks by Theorem 3.1. The sensitivity of the associated reproduction numbers with respect to parameters can be analyzed by the elasticities of the reproduction numbers. Thus, the elasticity of  $R_1$  with respect to different parameters (i.e.,  $\beta_1$ ,  $\gamma_1$ , and  $\langle k^2 \rangle / \langle k \rangle$ ) are defined as follows:

$$\varepsilon_{\beta_1}^{R_1} = \frac{\beta_1}{R_1} \frac{\partial R_1}{\partial \beta_1} = 1, \quad \varepsilon_{\gamma_1}^{R_1} = \frac{\gamma_1}{R_1} \frac{\partial R_1}{\partial \gamma_1} = -1, \quad \text{and} \quad \varepsilon_{\frac{\langle k^2 \rangle}{\langle k \rangle}}^{R_1} = \frac{\langle k^2 \rangle}{R_1} \frac{\partial R_1}{\partial \frac{\langle k^2 \rangle}{\langle k \rangle}} = 1.$$

Note that the positive value of the elasticity means  $R_1$  increases with the increase in corresponding parameter, while a negative value says that  $R_1$  decreases with the increase in corresponding parameter. The elasticity of  $R_2$  with respect to different parameters can be defined in a similar way. Therefore, improving the cure rate and decreasing the transmission rate are the regular methods for controlling the disease spread like a classical model. The degree distribution is another important factor impacting a two-strain spread in a heterogeneous networks. Specially, if the degree distribution is large the disease will spread even if initial infected individual is few or the transmission rate is low. The topology of a network should be caused our high attention to controlling a disease transmission on complex networks. The three main parameters have same status in controlling a disease spread in one strain. Coexistence and competing mechanism are the main mechanisms in multi-strain epidemic models. Recently, there are many mechanisms resulting in coexistence of multi-strain, such as superinfection, co-infection, and cross-immunity and so on. We note that if  $\alpha_1 = \alpha_2 = 0$ , then all the conditions for coexistence are not satisfied in Theorem 4.1. This implies the model just has a simple competing-exclusion, i.e., only the strain with a higher reproduction number can prevail and persist. Therefore, the saturating incidence rate is another mechanism for coexistence of a two-strain model.

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