Chaos, Solitons and Fractals 104 (2017) 215–221

Contents lists available at ScienceDirect

Chaos, Solitons and Fractals

Nonlinear Science, and Nonequilibrium and Complex Phenomena

journal homepage: www.elsevier.com/locate/chaos

Frontiers

Analysis of competitive infectious diseases with multiple strains

Jian-Qin Qiao[a,b], Li Li[c,*]

[a] Complex Systems Research Center, Shanxi University, Taiyuan, Shanxi 030006, China
[b] Shanxi Key Laboratory of Mathematical Techniques and Big Data Analysis on Disease Control and Prevention, Shanxi University, Shanxi 030006, China
[c] School of Computer and Information Technology, Shanxi University, Taiyuan, Shanxi 030006, China

ARTICLE INFO

Article history:
Received 21 July 2017
Revised 19 August 2017
Accepted 21 August 2017

Keywords:
Three strains
Competitive exclusion principle
Global stability
Comparison theorem

ABSTRACT

As we all known, there are many kinds of strains for a disease. However, the transmission dynamics of such disease is far from being well understood. In this paper, we established a SIS multi-strain model on scale-free network and the dynamics of multi-strain disease was studied by mean-field method. It is proved that the disease-free equilibrium is globally asymptotically stable when the basic reproduction number $R_0 < 1$. It is proved that the equilibrium point with the largest basic reproduction number is globally stable. Our results indicate that competitive exclusion principle also holds for the disease with multiple strains.

1. Introduction

Infectious diseases, including humans, animals, plant infectious diseases, especially human infectious diseases, are the most important biosecurity issues. Infectious diseases are still the first cause of global death. In China, infectious diseases are still a serious threat to people's health and national security. According to the World Health Organization statistics, in the past 20 years there have been at least 30 kinds of new infectious diseases. In addition to SARS, some new infectious diseases such as AIDS (HIV / AIDS, since 1981, the United States reported the disease since the first time, as of January 1992, the global health report to the World Health Organization reported a total of 189 countries and regions, In 2002 the world a total of about 70 million AIDS, killing 20 million people, each year more than 560 million people infected with AIDS [1–3]. In addition, H1N1, H7N9, Ebola, dengue and other infectious diseases are still in some parts of the country, some countries and regions in the world epidemic or outbreak [4–9]. In recent years, the spread of disease on complex networks has been studied extensively, and has been a wealth of research results [10]. Complex network is composed of a large number of nodes and nodes between the edge, it has the topology and complexity of the dynamics of infectious diseases can be described in the process of more refined. There are already many studies on disease in the scale-free network [11–15]. In 2001, Pastor-Satorras et al. used the mean field theory to study the SIS epidemic model on the general network and applied it to the scale-free network. It was proved that the scale-free network had no threshold under the appropriate parameters, which means even if only very few individuals affected by the disease, the disease can also exist in the network for a long time [14].

In the past, most of the research work on mathematical modeling and research in infectious dynamics has assumed that only pathogens (viruses or bacteria) that cause infectious diseases have only one form of expression [10]. However, many infectious diseases are not caused by a single strain. For example, influenza and Neisseria gonorrhoeae, its pathogens have many forms, and there is a high degree of variability, according to media reports, there have been mutated strains. In addition, there are many genetic varieties of HIV, such as HIV-1 and HIV-2. Strains compete with each other, common infection, and mutation will affect the spread of infectious diseases [16]. So it is clear that it is necessary to clarify the mechanism of transmission of multiple strains of disease, which is of great significance to the study of the spread and epidemic of diseases.

In recent years, we have studied a lot about the propagation dynamics of multiple strains on the network. For example, in 2005, Newman studied the propagation threshold of two competing strains on the network by the bond percolation approach. It also shows that the two strains can coexist under certain conditions [17]. In 2011, Wu et al. [18], established a perfectly competitive two-strain SIS model on a scale-free network, demonstrating the existence of two strains competing thresholds on a finite network and an infinite network. Two strains of disease have been studied a lot of articles [17–25], but the actual situation in most of the disease has three or even more strains caused. In this paper,

* Corresponding author.
E-mail address: lili831113@sxu.edu.cn (L. Li).

http://dx.doi.org/10.1016/j.chaos.2017.08.017
0960-0779/

we use the method of mean-field to study the epidemic threshold of three competitive strains.

The paper is organized as follows: in Section 2, we develop a SIS model of three competing stain. In Section 3, we calculate the basic regeneration number and study the existence and stability of equilibrium points. In Section 4, we present numerical simulations to confirmed our theoretical predictions in Section 3. In Section 5, we have made some summaries and discussions.

2. Dynamical model

In order to carry out our research, we give the following two main assumptions.

(i) In this article, we studied three strains, which we call strain 1, 2 and strain 3. We used SIS dynamics to study each strain separately. Each node on the network represents a person, and we can divide these people into four states: susceptible, infected by strain 1, infected by strain 2, and infected by strain 3. A susceptible person can be infected by strain 1 with probability β_1 and can recover with probability γ_1. Similarly, β_2 and β_3 are the infection rates of strain 2 and strain 3, and their recovery rate is γ_2 and γ_3 . And the three strains compete with each other.

(ii) In this paper, we study the spread of disease on scale-free networks. In this scale-free network, $P(k)$ represents the probability of a randomly chosen node having a degree k. We assume that the network is uncorrelated. And $P(k)$ follows power-law distribution, $P(k) \sim k^{-2-\gamma}$ with $0 < \gamma \leq 1$.

In our uncorrelated network, the $N(k)$ represents the number of nodes with degree k. Then the number of four states at time t is represented as $S_k(t)$, $I_{1k}(t)$, $I_{2k}(t)$, $I_{3k}(t)$, and $s_k(t)$, $\rho_{1k}(t)$, $\rho_{2k}(t)$, $\rho_{3k}(t)$, as their densities respectively. Obviously $s_k(t) = S_k(t)/N(k)$, $\rho_{1k}(t) = I_{1k}(t)/N(k)$, $\rho_{2k}(t) = I_{2k}(t)/N(k)$, $\rho_{2k}(t) = I_{3k}(t)/N(k)$. Then we give the following equation:

$$\begin{cases} ds_k(t)/dt = -\beta_1 k s_k(t)\theta_1(t) - \beta_2 k s_k(t)\theta_2(t) - \beta_3 k s_k(t)\theta_3(t) \\ \qquad\qquad + \gamma_1\rho_{1k}(t) + \gamma_2\rho_{2k}(t) + \gamma_3\rho_{3k}(t), \\ d\rho_{1k}(t)/dt = \beta_1 k s_k(t)\theta_1(t) - \gamma_1\rho_{1k}(t), \\ d\rho_{2k}(t)/dt = \beta_2 k s_k(t)\theta_2(t) - \gamma_2\rho_{2k}(t), \\ d\rho_{3k}(t)/dt = \beta_3 k s_k(t)\theta_3(t) - \gamma_3\rho_{3k}(t). \end{cases} \tag{2.1}$$

Among them

$$\Omega = \{(s_1, \rho_{11}, \rho_{21}, \rho_{31}, \ldots, s_M, \rho_{1M}, \rho_{2M}, \rho_{3M}), \\ \rho_{1k} + \rho_{2k} + \rho_{3k} \leq 1, 1 \leq k \leq M\}.$$

M is the maximum number of contacts for each individual. Ω is a positive invariant set. In steady state, $s_k(t) + \rho_{1k}(t) + \rho_{2k}(t) + \rho_{3k}(t) = 1$. The system (2.1) can be converted to that

$$\begin{cases} d\rho_{1k}(t)/dt = \beta_1 k(1 - \rho_{1k}(t) - \rho_{2k}(t) - \rho_{3k}(t))\theta_1(t) - \gamma_1\rho_{1k}(t), \\ d\rho_{2k}(t)/dt = \beta_2 k(1 - \rho_{1k}(t) - \rho_{2k}(t) - \rho_{3k}(t))\theta_2(t) - \gamma_2\rho_{2k}(t), \\ d\rho_{3k}(t)/dt = \beta_3 k(1 - \rho_{1k}(t) - \rho_{2k}(t) - \rho_{3k}(t))\theta_3(t) - \gamma_3\rho_{3k}(t). \end{cases} \tag{2.2}$$

And

$$\Omega = \{(\rho_{11}, \rho_{21}, \rho_{31}, \ldots, \rho_{1M}, \rho_{2M}, \rho_{3M}), \\ \rho_{1k} + \rho_{2k} + \rho_{3k} \leq 1, 1 \leq k \leq M\}.$$

Where θ_1, θ_2, θ_3 represent the probability that each susceptible individual of degree k is in contact with strain 1, strain 2, and strain 3, respectively. It can be expressed as

$$\theta_1 = \sum_{k'} P(k'|k)\rho_{1k'}/< k >.$$

In the unrelated network, the conditional probability $p(k'/k)$ of a node with a degree of k randomly contacts with a node degree k' does not depend on k, which is proportional to $k'p(k')$. That is to say

$$p(k'/k) = k'p(k')/< k >.$$

Then

$$\theta_1 = \sum_{k'=1}^{M} k'p(k')\rho_{1k'}/< k >.$$

Similarly available

$$\theta_2 = \sum_{k'=1}^{M} k'p(k')\rho_{2k'}/< k >, \theta_3 = \sum_{k'=1}^{M} k'p(k')\rho_{3k'}/< k >.$$

3. Analysis of the model

3.1. The stability of disease-free equilibrium

System (2.2) has a disease-free equilibrium $E_0 = (\underbrace{0, \cdots, 0}_{3M})$. We use the next generation matrix method [26] to calculate its threshold. We can get that

$$F = \begin{pmatrix} F_{11} & O & O \\ O & F_{22} & O \\ O & O & F_{33} \end{pmatrix}_{3M\times\ 3M},$$

$$V = \begin{pmatrix} V_{11} & O & O \\ O & V_{22} & O \\ O & O & V_{33} \end{pmatrix}_{3M\times\ 3M},$$

where F is non-negative, and V is a non-singular M-matrix. And

$$F_{11} = \beta_1 \begin{pmatrix} \begin{pmatrix} 1 \\ 2 \\ \cdots \\ M \end{pmatrix} \begin{pmatrix} 1\times P(1) & 2\times P(2) & \cdots & M\times P(M) \end{pmatrix} \end{pmatrix} /< k >,$$

$$F_{22} = \beta_2 \begin{pmatrix} \begin{pmatrix} 1 \\ 2 \\ \cdots \\ M \end{pmatrix} \begin{pmatrix} 1\times P(1) & 2\times P(2) & \cdots & M\times P(M) \end{pmatrix} \end{pmatrix} /< k >,$$

$$F_{33} = \beta_3 \begin{pmatrix} \begin{pmatrix} 1 \\ 2 \\ \cdots \\ M \end{pmatrix} \begin{pmatrix} 1\times P(1) & 2\times P(2) & \cdots & M\times P(M) \end{pmatrix} \end{pmatrix} /< k >,$$

and

$$V_{11} = \gamma_1 I, V_{22} = \gamma_2 I, V_{33} = \gamma_3 I.$$

I represents the M-dimensional unit matrix, and the O represents the M-dimensional zero matrix. Then we can get the basic regeneration number of the model, $R_0 = \rho(FV^{-1})$, where $\rho(A)$ is defined as the spectral radius of the matrix A. We can get

$$R_0 = \max\{R_1, R_2, R_3\},$$

where

$$R_1 = \beta_1 < k^2 >/\gamma_1 < k >, R_2 = \beta_2 < k^2 >/\gamma_2 < k >,$$
$$R_3 = \beta_3 < k^2 >/\gamma_3 < k >.$$

Theorem 1. *The disease-free equilibrium point of the system (2.2) is locally asymptotically stable, when $R_0 < 1$.*

Proof. Assume that the Jacobian matrix of system (2.2) at disease-free equilibrium is as follows:

$$A = \begin{pmatrix} A_1 & O & O \\ O & A_2 & O \\ O & O & A_3 \end{pmatrix}_{3M\times\ 3M}.$$

where

$$(A_1)_{kk'} = -\gamma_1\delta_{kk'} + \beta_1 kp(k')/ < k >,$$