Epidemic thresholds in a heterogenous population with competing strains*

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Among many epidemic models, one epidemic disease may transmit with the existence of other pathogens or other strains from the same pathogen. In this paper, we consider the case where all of the strains obey the susceptible-infected-susceptible mechanism and compete with each other at the expense of common susceptible individuals. By using the heterogenous mean-field approach, we discuss the epidemic threshold for one of two strains. We confirm the existence of epidemic threshold in both finite and infinite populations subject to underlying epidemic transmission. Simulations in the Barabasi–Albert (BA) scale-free networks are in good agreement with the analytical results.

Keywords: complex network, epidemic threshold, epidemic dynamics

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1. Introduction

Throughout history, epidemic diseases have been a constant threat: from tuberculosis (TB),[1] severe acute respiratory syndrome (SARS),[2] Asian-influenza,[3] swine-origin influenza A (H1N1)[4,5] or other epidemic diseases. Disease transmission in many epidemic systems can be represented as a graph where vertices denote individuals and an edge connecting a pair of vertices indicates interaction between individuals. But, patterns of interaction in such a system can be very complex and the connectivity between two vertices is usually not uniform but heterogeneous. The heterogeneous contact rate reflects the property that the vertex degree k, or, the number of contacts with other individuals for a given individual, is not uniform. Rather, it is necessary to invoke a degree distribution function $P(k)$. Real networks subject to underlying disease transmission can be represented not only by conventional graphs, such as lattices, regular trees or classical random graphs, but also by complex networks, such as Strogatz–Watts (SW) small-world networks[6] or Barabasi–Albert (BA) scale-free networks.[7] Research[8−11] has shown that the two kinds of heterogenous contact rates can reduce the epidemic threshold for the susceptible-infected-susceptible (SIS) model.

In the disease spreading processes, on the one hand, one pathogen sometimes generates many strains with different spreading features, and hence a detailed investigation of multi-strain epidemic dynamics has great relevance.[12−15] For example, the human immunodeficiency virus (HIV) (which causes acquired immune deficiency syndrome (AIDS)) has many genetic varieties, and can be divided into several distinct strains, such as strain HIV-1 and strain HIV-2.[16] On the other hand, one pathogen is always incorporated with other pathogens.[17] In March and early April 2009, a new swine-origin influenza A (H1N1) virus emerged in Mexico and the United Sates. This virus has the potential to develop into the first influenza pandemic of the twenty-first century,[4] and it is accompanied by seasonal influenza.[5]

Research on multi-strain epidemic models in heterogenous populations have been carried out previously. There,[12] an SIS model with pathogen competition and mutation in a heterogenous populations was studied on the assumption that any patient recovers at a rate 1, i.e., unit recovery rate, where the authors only analysed the effect of the mutation rate. In other

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work,[13] the author studied bond-percolation corresponding to the susceptible-infected-removed model with competing pantheon and found an interesting phenomenon: intervention in the case of two competing diseases could eradicate one of the diseases, but not both, in scale-free networks. Up to the present, we have not seen the complete work on the multi-strain SIS model with competing strains or pantheons.

In the present paper, a two-strain model with competing strains or pantheons is mainly studied, which is the basic two-strain model and can be extended to other complex models, such as mutation models[18] and super-infection models.[19]

2. SIS model with competing strains

Two kinds of strains from the same pathogen or two distinct pathogens can be represented by strain 1 and strain 2, and the two strains may have different spreading rates. Let the spreading rate of strain 1 be β_1 and that of strain 2 be β_2. Each individual is represented by a node of the network, and can be in three discrete states: susceptible, or infective by the strain 1, or infective by the strain 2. And for each strain, the transmission dynamics are of the SIS type.[20] That is, susceptible nodes may become infective owing to contact with other infected nodes, and infected nodes may also recover into the susceptible state, the recovery rates are β_1 and β_2 for strain 1 and strain 2, respectively.

In order to utilize the heterogenous mean-field approach,[8] we classify all of the nodes according to their degrees.[21] That is, those nodes with the same number of neighbours belong to the same class. Let $\rho_{1,k}(t)$ and $\rho_{2,k}(t)$ represent the densities at time t of nodes in class with degree k infected by strain 1 and strain 2, respectively.

The two-strain model with a competing mechanism can be described as

$$\begin{cases} \dfrac{\mathrm{d}\rho_{1,k}(t)}{\mathrm{d}t} = -\gamma_1\rho_{1,k}(t) + \beta_1 k[1-\rho_{1,k}(t)-\rho_{2,k}(t)]\Theta_1(t), \\ \dfrac{\mathrm{d}\rho_{2,k}(t)}{\mathrm{d}t} = -\gamma_2\rho_{2,k}(t) + \beta_2 k[1-\rho_{1,k}(t)-\rho_{2,k}(t)]\Theta_2(t), \end{cases} \tag{1}$$

where the probability $0 \leq \Theta_1(t) \leq 1$ describes a link pointing to an individual infected by strain 1. It satisfies the equality[22]

$$\Theta_1(k,t) = \sum_{k'} P(k'|k)\rho_{1,k'}(t). \tag{2}$$

Similarly, the probability $0 \leq \Theta_2(t) \leq 1$ describes a link pointing to an individual infected by strain 2, and it satisfies the equality

$$\Theta_2(k,t) = \sum_{k'} P(k'|k)\rho_{2,k'}(t). \tag{3}$$

The conditional probability $P(k'|k)$ denotes the probability with which a randomly selected edge emitting from a node with degree k points to the node with degree k'.

In the present paper, we mainly study epidemic transmissions on uncorrelated networks where the probability with which a link points to an infected node is considered independent of the connectivity of the node from which the link is emanating. By the conditional probability formula, $P(k'|k)$ can be computed from

$$P(k'|k) = \frac{k'P(k')}{\langle k\rangle},$$

where $\langle k\rangle = \sum_s sP(s)$ represents the average over connectivity distribution of the given network. In this case, Θ_1 and Θ_2 are not related to degree k, and can be transformed respectively into the following forms,

$$\Theta_1(t) = \frac{\sum_{k'} k'P(k')\rho_{1,k'}(t)}{\langle k\rangle}, \tag{4}$$

and

$$\Theta_2(t) = \frac{\sum_{k'} k'P(k')\rho_{2,k'}(t)}{\langle k\rangle}. \tag{5}$$

We remark here that in the above model, we can see that system (1) has four parameters, which can be reduced into three if the typical time-scale transformation is applied. So this multi-parameter property invokes some different dynamical behaviours, which are different from the case of one strain or only one

pathogen. In the following analysis, we can find that the combined parameters of $\lambda_i = \beta_i/\gamma_i$, $i = 1, 2$, are important, and they are referred to as the effective spreading rates for strain 1 and strain 2, respectively. According to actual meanings, it may be reasonable to assume that $\lambda_1 \neq \lambda_2$. Nonetheless, we find that the recovery rates γ_1 and γ_2 cannot all be eliminated. Hence, we also discuss the unusual case, i.e., $\lambda_1 = \lambda_2$.

2.1. The case of $\lambda_1 \neq \lambda_2$

Now we study phase transition in the case $\lambda_1 \neq \lambda_2$. In order to obtain this, we firstly define the following two parameters,

$$R_1 = \frac{\lambda_1\langle k^2\rangle}{\langle k\rangle}, \quad R_2 = \frac{\lambda_2\langle k^2\rangle}{\langle k\rangle}, \tag{6}$$

where $\langle k^2\rangle = \sum_s s^2 P(s)$. They are just the basic reproduction numbers (BRNs) for strain 1 and strain 2, respectively. The R_1 gives the average number of secondary infectious cases produced by the infected individual with strain 1 in the entire infectious period in a completely susceptible population. Similarly, the R_2 has the equivalent meaning for strain 2. As we can see, the two BRNs are related to the effective spreading rates. If $\lambda_1 = \lambda_2$, then $R_1 = R_2$. Otherwise, they are both not all identical.

By imposing steady state $\mathrm{d}\rho_{1,k}(t)/\mathrm{d}t = 0$ and $\mathrm{d}\rho_{2,k}(t)/\mathrm{d}t = 0$, from Eq. (1) we have

$$\begin{aligned}\rho_{1,k} &= \frac{\lambda_1 k\Theta_1}{1+\lambda_1 k\Theta_1+\lambda_2 k\Theta_2},\\ \rho_{2,k} &= \frac{\lambda_2 k\Theta_2}{1+\lambda_1 k\Theta_1+\lambda_2 k\Theta_2}.\end{aligned} \tag{7}$$

Substituting Eq. (7) into Eqs. (4) and (5), we can obtain two self-consistent equations as follows:

$$\Theta_1 = \frac{\lambda_1}{\langle k\rangle}\sum_{k'}\frac{k'^2P(k')\Theta_1}{1+\lambda_1 k'\Theta_1+\lambda_2 k'\Theta_2}, \tag{8}$$

and

$$\Theta_2 = \frac{\lambda_2}{\langle k\rangle}\sum_{k'}\frac{k'^2P(k')\Theta_2}{1+\lambda_1 k'\Theta_1+\lambda_2 k'\Theta_2}. \tag{9}$$

Obviously, $(\Theta_1, \Theta_2) = (0, 0)$ is a trivial solution of Eqs. (8) and (9). Note that $\lambda_1 \neq \lambda_2$, hence equations (8) and (9) have no positive solutions. So we need consider only the following two cases, $\Theta_1 = 0$ and $\Theta_2 = 0$. When $\Theta_2 = 0$, we can have only nontrivial solutions of Eq. (8). Note that equation (8) can be reduced to

$$1 = \frac{\lambda_1}{\langle k\rangle}\sum_{k'}\frac{k'^2P(k')}{1+\lambda_1 k'\Theta_1} \equiv f(\Theta_1). \tag{10}$$

Because

$$f(1) = \frac{\lambda_1}{\langle k\rangle}\sum_{k'}\frac{k'^2P(k')}{1+\lambda_1 k'} < \frac{1}{\langle k\rangle}\sum_{k'}k'P(k') = 1,$$

and

$$f'(\Theta_1) = -\frac{\lambda_1^2}{\langle k\rangle}\sum_{k'}\frac{k'^3P(k')}{(1+\lambda_1 k'\Theta_1)^2} < 0,$$

a nontrivial solution of Eq. (10) exits if and only if

$$f(0) > 1. \tag{11}$$

So we have $R_1 = f(0)$, i.e.,

$$R_1 = \frac{\lambda_1\langle k^2\rangle}{\langle k\rangle} = \frac{\beta_1\langle k^2\rangle}{\gamma_1\langle k\rangle},$$

which is just our result above. By a similar analysis, if we let $\Theta_1 = 0$ and define

$$R_2 = \frac{\lambda_2\langle k^2\rangle}{\langle k\rangle} = \frac{\beta_2\langle k^2\rangle}{\gamma_2\langle k\rangle},$$

a similar result can be obtained. Thus, we have the following conclusions:

C1 There exists always disease-free equilibrium $E_0 = (1, 0, 0)$.

C2 Strain 1 has exclusive equilibrium $E_1 = (s_1^*, \rho_1^*, 0)$, if and only if $R_1 > 1$.

C3 Strain 2 has exclusive equilibrium $E_2 = (s_2^*, 0, \rho_2^*)$, if and only if $R_2 > 1$.

In this model, there are three states for each node, i.e., 0: empty (susceptible), 1: occupied by strain 1 (infected by strain 1), and 2: occupied by strain 2 (infected by strain 2). Due to C1, C2 and C3, phases in this model may be $\{0\}$ (the steady state that contains only state 0), $\{1\}$ (the steady state that contains only state 1), $\{2\}$ (the steady state that contains only state 2), and $\{1, 2\}$ (the steady coexistence of state 1 and state 2). In order to separate the possible phases above, we must study the asymptotical properties of the equilibria E_0, E_1 and E_2.

Firstly, we study the stability of disease-free equilibrium. Define $D = \{k \in Z^+ | P(k) > 0\}$, where Z^+ is the set of all positive integers. Then $n = |D|$ denotes the size of D. Suppose that the Jacobian matrix of Eq. (1) at the disease-free equilibrium point is denoted by A,

$$A = \begin{pmatrix} A_1 & 0 \\ 0 & A_2 \end{pmatrix}, \tag{12}$$

where

$$(A_1)_{kk'} = -\gamma_1\delta_{kk'} + \beta_1 kP(k')/\langle k\rangle, \quad (13)$$

$$(A_2)_{kk'} = -\gamma_2\delta_{kk'} + \beta_2 kP(k')/\langle k\rangle. \quad (14)$$

Here $k, k' \in D$ and $\delta_{kk'}$ is defined as

$$\delta_{kk'} = \begin{cases} 1, & k = k', \\ 0, & k \neq k'. \end{cases} \quad (15)$$

Furthermore, we denote the unit matrix by E. Directly computing $|A_1 - \mu E| = 0$ and $|A_2 - \mu E| = 0$ leads to

$$-(\mu+\gamma_1)^{n-1}\left[-\mu-\gamma_1+\beta_1\frac{\langle k^2\rangle}{\langle k\rangle}\right] = 0, \quad (16)$$

and

$$-(\mu+\gamma_2)^{n-1}\left[-\mu-\gamma_2+\beta_2\frac{\langle k^2\rangle}{\langle k\rangle}\right] = 0 \quad (17)$$

respectively. According to the block property of the matrix, the zero solution E_0 is therefore locally asymptotically stable, if and only if $R_1 \leq 1$ and $R_2 \leq 1$.

Secondly, we further study the asymptotical properties of the equilibria E_1 and E_2. Perturbing the steady state $\rho^*_{1,k}, k \in D$ so that $\rho_{1,k} = \varepsilon_k + \rho^*_{1,k}$. Omitting the higher power terms of ϵ_k gives the linearization matrix H in the following form,

$$H = \begin{pmatrix} B_1 & B_2 \\ 0 & B_3 \end{pmatrix}. \quad (18)$$

Note that H is a block upper triangular matrix, and the eigenvalue spectral $\rho(H) = \rho(B_1)\bigcup\rho(B_3)$. By simple computation, $|B_1 - \mu E| = 0$ is equivalent to

$$\frac{\lambda_1}{\langle k\rangle}\sum_s \frac{sP(s)}{\Delta^1(s)} = 1, \quad (19)$$

where

$$\Delta^1(s) = \frac{\gamma_1+\mu+\beta_1 s\Theta^*_1}{1-\rho^*_{1,s}}.$$

Noting that i^*_k satisfies the identity

$$1 - \rho^*_{1,k} = \frac{\gamma_1\rho^*_{1,k}}{\beta_1 k\Theta^*_1}. \quad (20)$$

Thus, equation (19) can be transformed into

$$\sum_k \frac{kP(k)\rho^*_{1,k}}{\gamma_1+\mu+\beta_1 k\Theta^*_1} = \sum_k \frac{kP(k)\rho^*_{1,k}}{\gamma_1}, \quad (21)$$

which can determine n complex roots, $\mu_s, s = 1, \ldots, n$. Submitting one root $\mu_s = a_s + b_s i$ into Eq. (21), we can obtain the following two equations,

$$\sum_k \frac{P(k)i^*_k(\beta_1+a_s+\beta_1 k\Theta^*_1)}{(\gamma_1+a_s+\beta_1 k\Theta^*_1)^2+b_s^2} = \sum_k \frac{P(k)i^*_k}{\gamma_1}, \quad (22)$$

and

$$-b_s\sum_k \frac{P(k)i^*_k}{(\gamma_1+a_s+\beta_1 k\Theta^*_1)^2+b_s^2} i = 0. \quad (23)$$

The second equation (23) indicates $b_s = 0$. So the eigenvalues $\mu_s = a$ are all real numbers. From Eq. (21), we find that all eigenvalues of matrix B_1 are negative, that is, $\mu_s < 0, s = 1, \ldots, n$. which shows that $\mu < 0$.

Conversely, $|B_3 - \mu E| = 0$ can be transformed into

$$(\gamma_2+\mu)^{n-1} = 0, \quad (24)$$

or

$$\frac{\lambda_2}{\langle k\rangle}\sum_s \frac{sP(s)}{\Delta^2(s)} = 1, \quad (25)$$

where $\Delta^2(s) = (\gamma_2+\mu)/(1-\rho^*_{1,s})$. Furthermore, μ can be solved from Eq. (25) to obtain

$$\mu = \left(\frac{\beta_1\gamma_2}{\beta_1}\right)\frac{\langle k\rangle}{\langle k^2\rangle}(R_2 - R_1), \quad (26)$$

so the condition $R_1 < 1$ cannot ensure the local stability of E_1. To make E_1 stable, $R_2 < R_1$ must hold from Eq. (26).

According to the symmetry, we also apply the above analysis to E_2. Similarly, we conclude that E_2 is locally stable, if and only if $R_1 < R_2$.

2.2. The case of $\lambda_1 = \lambda_2$

Our discussion in the above section shows that there is no positive equilibrium, or in other words, for the case $\lambda_1 \neq \lambda_2$ it is impossible for the two strains to coexist. Now we consider the other case, that is, $\lambda_1 = \lambda_2 = \lambda$.

In this case, the self-consistent equations (8) and (9) respectively become

$$\Theta_1 = \frac{\lambda}{\langle k\rangle}\sum_{k'} \frac{k'P(k')\Theta_1}{1+\lambda k'(\Theta_1+\Theta_2)}, \quad (27)$$

and

$$\Theta_2 = \frac{\lambda}{\langle k\rangle}\sum_{k'} \frac{k'P(k')\Theta_2}{1+\lambda k'(\Theta_1+\Theta_2)}. \quad (28)$$

Let $R = \lambda\langle k^2\rangle/\langle k\rangle$, using the analysis similar to that for the above two equations, one can obtain that

the equilibrium solutions fall into the following two cases:

C4 When $R \leq 1$, there exists a disease-free equilibrium $E_0' = (1, 0, 0)$;

C5 When $R > 1$, there exist infinitely many equilibria, and the parameter points (Θ_1, Θ_2) of the equilibria form a line segment which connects two endpoints: $(0, \bar{\Theta})$ and $(\bar{\Theta}, 0)$, where $\bar{\Theta}$ satisfies

$$1 = \frac{\lambda}{\langle k \rangle} \sum_{k'} \frac{k' \varphi(k') P(k')}{1 + \lambda k' \bar{\Theta}}. \tag{29}$$

3. Epidemic threshold

3.1. In finite populations

In the real world, an epidemic always occurs on a finite network,[9] though the size of the network may be very large. For one-strain models[8−10] in a finite population, an epidemic disease possesses a finite epidemic threshold.[9]

For two-strain models, we regard strain 2 as the objective strain to study the epidemic threshold of it. According to our analysis, if $R_1 \leq 1$, the epidemic threshold for strain 2 satisfies $R_2 > 1$; while $R_1 > 1$, it satisfies $R_2 \geq R_1$.

3.2. In infinite populations

As we know, if there is only one strain or pathogen in the networks with scale-free connectivity and with any nonzero rate of infection, the presence of hubs in the network leads to the absence of any epidemic threshold.[8] And the growth of infected individuals is governed by a time scale τ, which is proportional to the ratio between the first and the second order moments of the network degree distribution,[23] i.e. $\tau \sim \langle k \rangle / \langle k^2 \rangle$, implying that for a sufficiently large scale-free network, a disease breaks out in a very short time period.

However, this is not the case in the networks with multiple strains or pathogens. Under the existence of another strain or pathogen, the above theoretical results ensure the presence of an epidemic threshold. In this case, the effective infection rate of strain 1 satisfies $R_2 \geq R_1$.

From the above discussion, we know that there exist two types of epidemic thresholds, that is, the network-based threshold determined by the network structure, and the competition-based threshold determined by the other competing strain. For finite populations, we can find the coexistence of these two types of thresholds. For infinite populations, there is only the competition-based threshold and thus the threshold is related to network size, which is similar to the case with a finite maximum dissemination.[24] So we conclude that the epidemic threshold exists regardless of populations, which is similar to the previous conclusion.[13]

4. Simulations

First of all, we briefly review the BA scale-free networks[2,7] subject to the underlying two strains or pathogens transmission. A BA network can be constructed as follows. Start from a small number, m_0, of nodes. At each time step, a new node is added to the existing network, bringing m new edges linking to the old nodes in such a way that it connects to node n_i with probability $\Pi_i = k_i / \sum_j k_j$, where k_i is the degree of the i-th node. After iterating this scheme sufficiently many times, a network is obtained, combining N nodes with node-degree distribution $p_k \sim k^{-3} \theta(k_c - k)$ and average degree $\langle k \rangle = 2m$, where $\theta(x)$ is the Heaviside step function, and k_c is a hard cutoff due to the network size.[9]

In the following simulations, we assume that $\gamma_1 = \gamma_2 = 1$, then the effective spreading rates $\lambda_1 = \beta_1$ and $\lambda_2 = \beta_2$. Since the state of each node in the epidemic process may be either susceptible or infected by strain 1 or strain 2, the probability with which one susceptible node n_i becomes an infected node at time step t can be computed from

$$p(i, t) = 1 - (1 - \beta_1)^x (1 - \beta_2)^y, \tag{30}$$

where x (y) is the number of n_i neighbouring nodes infected by strain 1 (2) at time step $t-1$. Furthermore, one can obtain the probability with which it may be infected by strain 1 at time step t to be

$$q_1(i, t) = \frac{x\beta_1}{x\beta_1 + y\beta_2}. \tag{31}$$

Accordingly, the probability with which it may be infected by strain 2 at time step t is just

$$q_2(i, t) = 1 - p_1(t) = \frac{y\beta_2}{x\beta_1 + y\beta_2}. \tag{32}$$

From the results for finite populations, one finds that strain 2 is outbroken only if

$$\beta_2 \begin{cases} > \dfrac{\langle k\rangle}{\langle k^2\rangle}, & \text{if} \quad \beta_1 \le \dfrac{\langle k\rangle}{\langle k^2\rangle}, \\ \ge \beta_1, & \text{if} \quad \beta_1 > \dfrac{\langle k\rangle}{\langle k^2\rangle}. \end{cases} \tag{33}$$

Figure 1 illustrates the steady density of nodes infected by strain 2, ρ_2, versus effective spreading β_2 in the BA network model with $m_0 = m = 2$ and network size $N = 3 \times 10^5$. For infection rates of strain 1: $\beta_1 = 0.02, 0.1$ and 0.16, there are epidemic thresholds $\beta_{2,c} = 0.04(2), 0.10(2)$ and $0.16(2)$, respectively, which are in good agreement with the results (33).

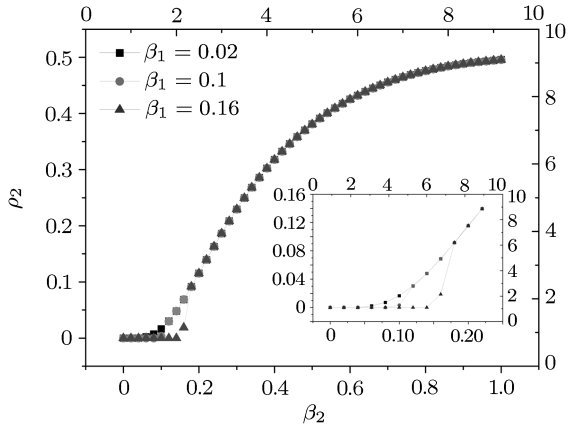


Fig. 1. Curves for infected density $\rho_2 = \sum_k P(k)\rho_{2,k}$ versus β_2 for $\beta_1 = 0.02, 0.1, 0.16$ in the BA networks of size $N = 3 \times 10^5$ and $m_0 = m = 2$. Without strain 1, say, $\beta_1 = 0$, the epidemic threshold of β_2 is just 0.04. This plot shows that simulations accord with the results obtained from mean-field equations. The inset is for the version of origin plot with β_2 limited in $[0, 0.25]$.

As shown in Fig. 1, the density of nodes infected by strain 2, ρ_2, as a function of β_2, is unrelated to parameter β_1 as $\beta_2 > \beta_1$. This can be seen in Eqs. (7) and (9). In fact, according to Eqs. (7) and (9), when $\rho_{1,k} = 0$, $k = k_0, \ldots, k_c$, one can obtain

$$\rho_{2,k} = \frac{\lambda_2 k \Theta_2}{1 + \lambda_2 k \Theta_2}, \tag{34}$$

where Θ_2 satisfies

$$1 = \frac{\lambda_2}{\langle k\rangle} \sum_{k'} \frac{k'^2 P(k')}{1 + \lambda_2 k' \Theta_2}. \tag{35}$$

So the dynamical behaviour in the epidemic model with competing strains is relatively simple, and can be regarded as the model with only one strain that possesses a big effective spreading rate.

Finally, we remark that $q_1(i,t)$ may be selected to be in some other forms, such as

$$q_1'(i,t) = \frac{1 - (1-\beta_1)^x}{2 - (1-\beta_1)^x - (1-\beta_2)^y}, \tag{36}$$

or

$$q_1''(i,t) = \frac{(1-\beta_2)^y}{(1-\beta_1)^x + (1-\beta_2)^y}. \tag{37}$$

But simulations show that the effect of the selecting expressions (31) and (32) is in better accordance with the mean-field equations.

5. Concluding remarks

Generally speaking, the forms of interaction between two epidemic particles in a multi-strain epidemic model may be classified into many types, including co-infection, which means that two strains can be hosted in one particle, super-infection, which means that one strain can host the particle with the other strain,[25] cross-immunity which means that two strains are perfectly competing and nodes infected by one strain cannot be infected by the other strain, mutation which means one strain can change into another strain[4,12] and so on. Some previous results have shown that different interaction mechanisms can generate different effects, such as co-infection, and can induce complex dynamical behaviour, such as chaotic attractors.[26] And the super-infection may generate a so-called strain replacement phenomenon.[19]

Now we give a brief summary of this paper. We have studied two-strain SIS models in scale-free networks. By theoretical analyses and numerical simulations, we described the effect of mean-field equations in modeling the spreading of multi-strain epidemics in heterogenous populations, and found the emergence of an epidemic threshold under the coexistence of the other strains. Meanwhile, our findings show the efficiency of the heterogenous mean-field approach,[8] which has been successfully applied in complex cases, i.e., the spread of sexually transmitted diseases.[27]

There still remains a vital problem that the global stability of endemic steady states should be proved rigorously, which seems difficult to solve by current methods.[28,29]

References

[1] Bleed D, Dye C and Raviglione M C 2000 *Curr. Opin. Plum. Med.* **6** 174

[2] Small M and Tse C K 2005 *Int. J. Bifur. Chaos* **15** 1745

[3] Small M, Walker D M and Tse C K 2007 *Phys. Rev. Lett.* **99** 188702

[4] Smith G J D, Vijaykrishna D, Bahl J, Lycett S J, Worobey M, Pybus O G, Ma S K, Cheung C L, Raghwani J, Bhatt S, Peiris J S M, Guan Y and Rambaut A 2009 *Nature* **459** 1122

[5] Maher B and Butler D 2009 *Nature* **462** 154

[6] Watts D J and Strogatz S H 1998 *Nature* **393** 440

[7] Barabasi A L and Albert R 1999 *Science* **286** 509

[8] Pastor-Satorras R and Vespignani A 2001 *Phys. Rev. Lett.* **86** 3200

[9] Pastor-Satorras R and Vespignani A 2002 *Phys. Rev.* E **65** 035108

[10] Santos F C, Rodrigues J F and Pacheco J M 2005 *Phys. Rev.* E **72** 056128

[11] May R M and Lloyd A L 2001 *Phys. Rev.* E **64** 066112

[12] Masuda N and Konno N 2006 *J. Theor. Biol.* **243** 64

[13] Newman M E J 2005 *Phys. Rev. Lett.* **95** 108701

[14] Thomasey D H and Martcheva M 2008 *J. Bio. Sys.* **16** 255

[15] NuNo M, Feng Z, Martcheva M and Castillo-Chavez C 2005 *SIAM J. Appl. Math.* **65** 964

[16] Balter M 1998 *Science* **281** 1425

[17] Castillo-Chavez C, Huang W and Li J 1996 *SIAM J. Appl. Math.* **56** 494

[18] Schinazi R B 2001 *Markov Processes Relat. Fields* **7** 595

[19] Martcheva M and Horst T R 2005 *Math. Biosci.* **195** 23

[20] Brauer F 2005 *Math. Biosci.* **198** 119

[21] Zhang H F, Small M, Fu X C and Wang B H 2009 *Chin. Phys.* B **18** 3633

[22] Fu X C, Small M, Walker D M and Zhang H F 2008 *Phys. Rev.* E **77** 036113

[23] Barthelémy M, Barrat A, Pastor-Satorras R and Vespignani A 2004 *Phys. Rev. Lett.* **92** 178701

[24] Pei W D, Liu Z X, Chen Z Q and Yuan Z Z 2008 *Acta Phys. Sin.* **57** 6777 (in Chinese)

[25] Cai L M, Li X Z and Yu J Y 2007 *Math. Appl.* **20** 328

[26] Kamo M and Sasaki A 2002 *Physica* D **165** 228

[27] Liu M X and Ruan J 2009 *Chin. Phys.* B **18** 2115

[28] Wang L and Dai G Z 2008 *SIAM J. Appl. Math.* **68** 1495

[29] d'Onofrio A 2008 *Nonlinear Analysis: RWA* **9** 1567