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Dynamical analysis of an SIRS network model with direct immunization and infective vector

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Abstract

With the awareness of risk in infective disease spreading, healthy individuals (the susceptible ones) will take some measures to acquire temporary immunity. This paper addresses an SIRS model with direct immunization and an infective vector in complex networks and performs the dynamical analysis for this model. By theoretical analysis, we obtain the epidemic threshold λ_c and prove that if infection rate $\lambda < \lambda_c$, the disease-free equilibrium is globally asymptotically stable; if $\lambda > \lambda_c$, there exists a unique endemic equilibrium, and it is globally attractive. These theoretical results are confirmed by numerical simulations.

Keywords: epidemic threshold; dynamical analysis; epidemic equilibrium; SIRS model; global attraction

1 Introduction

In recent years, many epidemic models on complex networks, such as SIS (susceptible-infected-susceptible) [1–8] and SIR (susceptible-infected-removed) [9–16] and so on, have been widely studied by researchers from different subjects. Classical studies have revealed that there is an epidemic threshold λ_c for an epidemic model on homogeneous networks, below which the disease will die out; otherwise there will exist a persistence. However, Pastor-Satorras and Vespignani further showed a striking result that the epidemic threshold λ_c will vanish for a heterogenous network with sufficiently large sizes [1, 2, 4].

In fact, apart from the human behavior [17, 18] and the external environment [19], the infection vector (*e.g.*, mosquitoes) may also play an important role in epidemic transmission [3, 20, 21]. The infective vector generally acts as a carrier of an infective disease and can transmit it to a human. By considering the disease spreading on a human network caused by an infection vector, Cooke and Busenberg [22, 23] have addressed some epidemic compartment models. As we known, some diseases spread not only by contacts between people and infected vectors but also by blood contacts within human. By noting this fact, Shi *et al.* [21] proposed a new SIS model with an infective medium on complex networks, which models the spread of a class of infectious diseases. Then a modified SIS model is proposed in [3] by assuming that the human contacts can be considered as a scale-free network, but the infective media may contact a person without any selectivity. The epidemic threshold and the stability of endemic equilibrium are investigated theoretically. A more general modified SIS model with an infective medium on complex networks was introduced in [20], and the authors investigated the global attraction of endemic equilibrium by the basic reproduction number R_0. However, the direct relation between the

epidemic threshold λ_c in [3] and the basic reproduction number R_0 in [20] has not been revealed.

In the real world, with the awareness of an infectious disease spreading, some healthy individuals will usually take some protective measures (*e.g.*, vaccine inoculation) to acquire temporary immunity. By investigating an SIRS epidemic model [24] with direct immunization on complex networks, the result shows that the direct immunization can increase the epidemic threshold and reduce the prevalence of infectious disease.

In this paper, we propose a new SIRS model with direct immunization and an infective vector on complex networks. We get the epidemic threshold λ_c, below which the disease-free equilibrium is globally stable; otherwise, the disease-free equilibrium is unstable and a unique endemic equilibrium exists, and it is also globally attractive. More importantly, according to our method, one can directly determine the relation between the epidemic threshold and the basic reproduction number.

The rest of the paper is organized as follows. In Section 2, we propose a new SIRS model with direct immunization and an infective vector on complex networks. Section 3 analyzes the dynamics of the model and shows some theoretical results. Some numerical simulations are performed to confirm our theoretical predictions in Section 4.

2 The model

Based on the real mechanism of some relevant epidemic networks, our model will be constructed with the following context:

- Two infection mechanisms: the disease spreads not only by contacts between individuals, but also by contacts between individuals and infective vectors.
- Two removed ways: (i) with awareness of risk infective disease spreading, some health individuals may make some protective measures (vaccination) to acquire temporary immunity; (ii) an infected individual becoming a removed individual after cure may acquire temporary immunity.

In addition, we further suppose that the individuals' contacts can be treated as heterogeneous, but the contacts between individuals and vectors can be considered as homogeneous. This assumption results from the selective contacts in a human network and non-selective contacts between people and vectors [3, 20].

Let $S_k(t)$, $I_k(t)$ and $R_k(t)$ be the densities of susceptible, infected and removed nodes with degree k at time t respectively, and let $V(t)$ be the density of the infective medium at time t. Let $\rho(t) = \sum p(k)I_k(t)$ denote the density of infected individuals on the network, and Θ represents the probability that a randomly chosen link emanating from a node of degree k leads to infected nodes. In this paper, we consider the situation of uncorrelated networks, then Θ can be written as $\Theta = \frac{1}{\langle k\rangle}\sum_{k'} k'p(k')I_{k'}(t)$ [1, 2], where $p(k)$ denotes the degree distribution of the network, $\langle k\rangle = \sum kp(k)$ is its average degree.

We assume that the susceptible nodes become the removed nodes with rate α for acquiring temporary immunity. At the same time, each susceptible (health) node is infected with rates λ and γ_1 if it is contacted to infected nodes and infective vectors, respectively. Infected nodes are cured with rate β and removed nodes again become susceptible with rate δ for immunization-lost. Health vectors are infected with rate γ_2 if they are contacted to infected individuals and infected vectors recover with rate ξ.

In the real world, an epidemic always occurs on a finite network [4] even though the size of the network is very large. Hence, we consider disease transmission in a finite population in this paper, and let n be the maximum degree. Then, neglecting of contact duration,

the proposed SIRS propagation model can be described by the following differential equations:

$$\begin{cases} \frac{dS_k(t)}{dt} = -\lambda k S_k(t)\Theta + \delta R_k(t) - \gamma_1 V(t) S_k(t) - \alpha S_k(t), \\ \frac{dI_k(t)}{dt} = \lambda k S_k(t)\Theta - \beta I_k(t) + \gamma_1 V(t) S_k(t), \\ \frac{dR_k(t)}{dt} = \beta I_k(t) - \delta R_k(t) + \alpha S_k(t), \\ \frac{dV(t)}{dt} = -\xi V(t) + \gamma_2 (1 - V(t))\rho(t). \end{cases} \tag{1}$$

Without loss of generality, assume that the infective vector has unit recovery, *i.e.*, $\xi = 1$. In addition, the variables $S_k(t), I_k(t), R_k(t)$ satisfy the normalization condition $S_k(t) + I_k(t) + R_k(t) = 1$. Then Eq. (1) can be rewritten as

$$\begin{cases} \frac{dI_k(t)}{dt} = [\lambda k\Theta + \gamma_1 V(t)][1 - I_k(t) - R_k(t)] - \beta I_k(t), \\ \frac{dR_k(t)}{dt} = (\beta - \alpha) I_k(t) - (\alpha + \delta) R_k(t) + \alpha, \\ \frac{dV(t)}{dt} = -V(t) + \gamma_2 (1 - V(t))\rho(t). \end{cases} \tag{2}$$

3 Epidemic threshold and global analysis

3.1 Epidemic threshold

Theorem 1 *Let* $\lambda_c = \frac{[\beta(\alpha+\delta) - \delta\gamma_1\gamma_2]\beta(\alpha+\delta)\langle k\rangle}{\delta^2\gamma_1\gamma_2(\langle k\rangle^2 - \langle k^2\rangle) + \delta\beta(\delta+\alpha)\langle k^2\rangle}$ *and* $\beta - \frac{\delta}{\alpha+\delta}\gamma_1\gamma_2 > 0$, *if* $\lambda > \lambda_c$, *then one and only one endemic equilibrium solution of system* (2) *exists, i.e., the epidemic propagation may outbreak on complex networks.*

Proof By letting the right-hand side of system (2) be zero, we have

$$\beta I_k(t) = \big[\lambda k\Theta + \gamma_1 V(t)\big]\big[1 - R_k(t) - I_k(t)\big], \tag{3}$$

$$(\beta - \alpha) I_k(t) - (\alpha + \delta) R_k(t) + \alpha = 0 \tag{4}$$

and

$$V(t) = \frac{\gamma_2 \rho(t)}{1 + \gamma_2 \rho(t)}. \tag{5}$$

Substituting (4) into (3), one obtains

$$\beta I_k(t) = \big[\lambda k\Theta + \gamma_1 V(t)\big]\left[\frac{\delta}{\delta + \alpha} - \frac{\beta + \delta}{\delta + \alpha} I_k(t)\right]. \tag{6}$$

Then substituting (5) into (6), we have

$$I_k(t) = \frac{\delta[\lambda k\Theta + \lambda k\gamma_2\rho(t)\Theta + \gamma_1\gamma_2\rho(t)]}{\beta(\delta + \alpha)[1 + \gamma_2\rho(t)] + (\beta + \delta)[\lambda k\Theta + \lambda k\gamma_2\rho(t)\Theta + \gamma_1\gamma_2\rho(t)]}. \tag{7}$$

Let

$$\mathcal{F}_1 = \delta\big[\lambda k\Theta + \lambda k\gamma_2\rho(t)\Theta + \gamma_1\gamma_2\rho(t)\big]$$

and

$$\mathcal{F}_2 = \beta(\delta + \alpha)\big[1 + \gamma_2\rho(t)\big] + (\beta + \delta)\big[\lambda k\Theta + \lambda k\gamma_2\rho(t)\Theta + \gamma_1\gamma_2\rho(t)\big].$$

Then one has a self-consistency equation as follows:

$$\Theta(t) = \frac{1}{\langle k\rangle}\sum_{k} kp(k)I_k(t) = \frac{1}{\langle k\rangle}\left\langle k\frac{\mathcal{F}_1}{\mathcal{F}_2}\right\rangle \equiv \mathcal{F}(\Theta). \tag{8}$$

It is obvious that $\Theta(t) = 0$ is a trivial solution to Eq. (8). What we are interested in is the condition under which the epidemic propagation outbreaks. Since $\Theta \in [0,1]$, $\mathcal{F}(0) = 0$ and $\mathcal{F}(\Theta) \in [0,1)$, then Eq. (8) must have a non-trivial solution if $\frac{d\mathcal{F}}{d\Theta}|_{\Theta=0} > 1$.

By computing the following expression

$$\left.\frac{d\mathcal{F}}{d\Theta}\right|_{\Theta=0} = \frac{1}{\langle k\rangle}\left\langle k\frac{(\frac{\partial\mathcal{F}_1}{\partial\rho}\cdot\frac{d\rho}{d\Theta}+\frac{\partial\mathcal{F}_1}{\partial\Theta})\mathcal{F}_2 - (\frac{\partial\mathcal{F}_2}{\partial\rho}\cdot\frac{d\rho}{d\Theta}+\frac{\partial\mathcal{F}_2}{\partial\Theta})\mathcal{F}_1}{\mathcal{F}_2^2}\right\rangle\right|_{\Theta=0} > 1, \tag{9}$$

we have the epidemic threshold λ_c taking the following expression in the case where $\beta - \frac{\delta}{\alpha+\delta}\gamma_1\gamma_2 > 0$,

$$\lambda_c = \frac{[\beta(\alpha+\delta) - \delta\gamma_1\gamma_2]\beta(\alpha+\delta)\langle k\rangle}{\delta^2\gamma_1\gamma_2(\langle k\rangle^2 - \langle k^2\rangle) + \delta\beta(\delta+\alpha)\langle k^2\rangle}.$$

Inequality (9) holds if and only if $\lambda > \lambda_c$. Furthermore, we will ascertain the uniqueness of endemic equilibrium in a similar way as paper [20]. Assume that $I = (I_1, I_2, \ldots, I_n, V)$ and $I^* = (I_1^*, I_2^*, \ldots, I_n^*, V^*)$ are two different roots to Eqs. (5) and (6). Let

$$\eta = \max\left\{\max_{k=1,\ldots,n}\left\{\frac{I_k}{I_k^*}\right\}, \frac{V}{V^*}\right\}.$$

Moreover, assume that $\eta > 1$ without loss of generality. We will complete the proof in two cases as follows.

Case 1: If there exists a natural number $k_0 \in \{1,2,\ldots,n\}$ such that $\eta = \frac{I_{k_0}}{I_{k_0}^*}$, then we, by (6), have

$$\beta I_{k_0} = \left[\lambda k_0\Theta(I) + \gamma_1 V\right]\left[\frac{\delta}{\delta+\alpha} - \frac{\beta+\delta}{\delta+\alpha}I_{k_0}\right], \tag{10}$$

$$\beta I_{k_0}^* = \left[\lambda k_0\Theta\left(I^*\right) + \gamma_1 V^*\right]\left[\frac{\delta}{\delta+\alpha} - \frac{\beta+\delta}{\delta+\alpha}I_{k_0}^*\right], \tag{11}$$

where $\Theta(I) = \frac{1}{\langle k\rangle}\sum kp(k)I_k$ and $\Theta(I^*) = \frac{1}{\langle k\rangle}\sum kp(k)I_k^*$. From (10) and (11), we can obtain

$$\left[\lambda k_0\Theta(I) + \gamma_1 V\right]\left[\frac{\delta}{\delta+\alpha} - \frac{\beta+\delta}{\delta+\alpha}I_{k_0}\right] = \eta\cdot\left[\lambda k_0\Theta\left(I^*\right) + \gamma_1 V^*\right]\left[\frac{\delta}{\delta+\alpha} - \frac{\beta+\delta}{\delta+\alpha}I_{k_0}^*\right], \tag{12}$$

while we have the following inequalities according to the definition of η:

$$\eta\cdot\left[\lambda k_0\Theta\left(I^*\right) + \gamma_1 V^*\right] = \lambda k_0\Theta\left(I^*\right)\cdot\eta + \gamma_1 V^*\cdot\eta > \lambda k_0\Theta(I) + \gamma_1 V, \tag{13}$$

$$\frac{\delta}{\delta+\alpha} - \frac{\beta+\delta}{\delta+\alpha}I_{k_0}^* > \frac{\delta}{\delta+\alpha} - \frac{\beta+\delta}{\delta+\alpha}I_{k_0}. \tag{14}$$

It is obvious that (13) and (14) contradict (12).

Case 2: If $\eta = \frac{V}{V^*}$, according to (5), then we have

$$V = (1 - V)\gamma_2 \rho(I), \tag{15}$$

$$V^* = \left(1 - V^*\right)\gamma_2 \rho\left(I^*\right), \tag{16}$$

where $\rho(I) = \sum p(k)I_k$ and $\rho(I^*) = \sum p(k)I_k^*$. From (15), (16) and the definition of η, one obtains

$$(1 - V)\gamma_2 \rho(I) = \left(1 - V^*\right)\gamma_2 \rho\left(I^*\right) \cdot \eta > (1 - V)\gamma_2 \sum p(k)\left(I_k^* \cdot \eta\right) > (1 - V)\gamma_2 \rho(I). \tag{17}$$

A contradiction appears. In conclusion, when $\lambda > \lambda_c$, one and only one endemic equilibrium solution exists for system (1). □

Letting $\tau = \frac{\delta}{\delta+\alpha}$, the epidemic threshold λ_c can be rewritten as

$$\lambda_c = \frac{[\beta - \tau\gamma_1\gamma_2]\beta\langle k\rangle}{\tau^2\gamma_1\gamma_2(\langle k\rangle^2 - \langle k^2\rangle) + \tau\beta\langle k^2\rangle}. \tag{18}$$

Note that the assumption that $\beta - \tau\gamma_1\gamma_2 > 0$, which is a default condition below, ensures that the threshold λ_c is larger than zero for a finite size network. One can see that the critical threshold vanishes for a scale-free network with sufficiently large sizes, which agrees with the previous papers [1, 2, 4].

The reader should find that the model parameters are general in this model. If γ_1 or γ_2 vanishes, the proposed model may become an SIRS model with direct immunization via one infection mechanism (contacts between individuals), and the epidemic $\lambda_c = \frac{\beta\langle k\rangle}{\tau\langle k^2\rangle}$ which agrees with the one of paper [24]. In addition, the proposed model may become one SIRS model with an infection vector via two infection mechanisms when $\alpha = 0$. And the epidemic threshold $\lambda_c = \frac{[\beta-\gamma_1\gamma_2]\beta\langle k\rangle}{\gamma_1\gamma_2(\langle k\rangle^2-\langle k^2\rangle)+\beta\langle k^2\rangle}$, especially, $\lambda_c = \frac{[1-\gamma_1\gamma_2]\langle k\rangle}{\gamma_1\gamma_2(\langle k\rangle^2-\langle k^2\rangle)+\langle k^2\rangle}$ if $\beta = 1$, which is in accordance with the one of paper [3].

3.2 Global stability of disease-free equilibrium

Theorem 2 *For system* (1), *let* λ_c *be the epidemic threshold defined as* (18). *If* $\lambda < \lambda_c$, *then the disease-free equilibrium is globally asymptotically stable. Otherwise, there exists a unique endemic equilibrium.*

Proof For convenience, system (1) can be rewritten as follows:

$$\begin{cases} \frac{dS_k(t)}{dt} = -[\lambda k\Theta + \gamma_1 V(t) + \delta + \alpha]S_k(t) - \delta I_k(t) + \delta, \\ \frac{dI_k(t)}{dt} = [\lambda k\Theta + \gamma_1 V(t)]S_k(t) - \beta I_k(t), \\ \frac{dV(t)}{dt} = -V(t) + \gamma_2(1 - V(t))\rho(t). \end{cases} \tag{19}$$

The disease-free equilibrium of Eq. (19) is $S_k = \frac{\delta}{\delta+\alpha} = \tau$, $I_k = 0$, $V = 0$, $k = 1, 2, \ldots, n$. Moreover, the Jacobian matrix at disease-free equilibrium can be represented as

$$J = \begin{bmatrix} D & F \\ 0 & M \end{bmatrix}, \tag{20}$$

where $D = -(\delta + \alpha)E_n$, and E_n is an nth identity matrix, F and M are $n \times (n+1)$ and $(n+1) \times (n+1)$ matrices taking the following forms, respectively:

$$F = \begin{bmatrix} -\frac{\tau\lambda}{\langle k\rangle}p(1) - \delta & -\frac{\tau\lambda}{\langle k\rangle}2 \cdot p(2) & \cdots & -\frac{\tau\lambda}{\langle k\rangle}n \cdot p(n) & -\gamma_1\tau \\ -\frac{\tau\lambda}{\langle k\rangle}2 \cdot p(1) & -\frac{\tau\lambda}{\langle k\rangle}2^2 \cdot p(2) - \delta & \cdots & -\frac{\tau\lambda}{\langle k\rangle}2n \cdot p(n) & -\gamma_1\tau \\ \vdots & \vdots & \ddots & \vdots & \vdots \\ -\frac{\tau\lambda}{\langle k\rangle}n \cdot p(1) & -\frac{\tau\lambda}{\langle k\rangle}n \cdot 2 \cdot p(2) & \cdots & -\frac{\tau\lambda}{\langle k\rangle}n^2 \cdot p(n) - \delta & -\gamma_1\tau \end{bmatrix},$$

$$M = \begin{bmatrix} \frac{\tau\lambda}{\langle k\rangle}p(1) - \beta & \frac{\tau\lambda}{\langle k\rangle}2 \cdot p(2) & \cdots & \frac{\tau\lambda}{\langle k\rangle}n \cdot p(n) & \gamma_1\tau \\ \frac{\tau\lambda}{\langle k\rangle}2 \cdot p(1) & \frac{\tau\lambda}{\langle k\rangle}2^2 \cdot p(2) - \beta & \cdots & \frac{\tau\lambda}{\langle k\rangle}2n \cdot p(n) & \gamma_1\tau \\ \vdots & \vdots & \ddots & \vdots & \vdots \\ \frac{\tau\lambda}{\langle k\rangle}n \cdot p(1) & \frac{\tau\lambda}{\langle k\rangle}n \cdot 2 \cdot p(2) & \cdots & \frac{\tau\lambda}{\langle k\rangle}n^2 \cdot p(n) - \beta & \gamma_1\tau \\ \gamma_2 p(1) & \gamma_2 p(2) & \cdots & \gamma_2 p(n) & -1 \end{bmatrix}.$$

It is obvious that all of eigenvalues of J have negative part if and only if all of eigenvalues of M have negative part. Denote $A = M - \mu \cdot E$ and $\nu = \mu + \beta$, where E is an $(n+1)$-order unit matrix, and μ is eigenvalue, then

$$\det A = \begin{vmatrix} \frac{\tau\lambda}{\langle k\rangle}p(1) - \nu & \frac{\tau\lambda}{\langle k\rangle}2 \cdot p(2) & \cdots & \frac{\tau\lambda}{\langle k\rangle}n \cdot p(n) & \gamma_1\tau \\ \frac{\tau\lambda}{\langle k\rangle}2 \cdot p(1) & \frac{\tau\lambda}{\langle k\rangle}2^2 \cdot p(2) - \nu & \cdots & \frac{\tau\lambda}{\langle k\rangle}2n \cdot p(n) & \gamma_1\tau \\ \vdots & \vdots & \ddots & \vdots & \vdots \\ \frac{\tau\lambda}{\langle k\rangle}n \cdot p(1) & \frac{\tau\lambda}{\langle k\rangle}n \cdot 2 \cdot p(2) & \cdots & \frac{\tau\lambda}{\langle k\rangle}n^2 \cdot p(n) - \nu & \gamma_1\tau \\ \gamma_2 p(1) & \gamma_2 p(2) & \cdots & \gamma_2 p(n) & -\nu - (1-\beta) \end{vmatrix}.$$

It follows that all of eigenvalues of M have negative real part if and only if all of roots of the characteristic polynomial $\det A = 0$ have real part of at most β. By some elementary transformations, one obtains that the issue above holds if and only all of roots of the following cubic polynomial have real part of at most β:

$$g(\nu) = \nu^3 - a_2\nu^2 - a_1\nu + a_0 = 0, \tag{21}$$

where

$$a_2 = \frac{\tau\lambda\langle k^2\rangle - (1-\beta)\langle k\rangle}{\langle k\rangle},$$

$$a_1 = \frac{(1-\beta)\tau\lambda\langle k^2\rangle + \gamma_1\gamma_2\tau\langle k\rangle}{\langle k\rangle},$$

$$a_0 = \frac{\gamma_1\gamma_2\tau^2\lambda[\langle hk^2\rangle - \langle k\rangle^2]}{\langle k\rangle}.$$

It is easy to obtain that

$$\lim_{\nu\to-\infty} g(\nu) = -\infty, \qquad \lim_{\nu\to+\infty} g(\nu) = +\infty,$$

$$g(0) = \frac{\gamma_1\gamma_2\tau^2\lambda[\langle k^2\rangle - \langle k\rangle^2]}{\langle k\rangle} > 0, \qquad g\left(\lambda\tau\frac{\langle k^2\rangle}{\langle k\rangle}\right) = -\gamma_1\gamma_2\tau^2\lambda\langle k\rangle < 0,$$

since $\langle k^2\rangle \gg \langle k\rangle^2$ for a sufficiently large network. We will investigate the roots to the cubic equation (21) below:

$$g(\beta) = \beta^2 - \beta\tau\lambda\frac{\langle k^2\rangle}{\langle k\rangle} - \gamma_1\gamma_2\tau\beta + \gamma_1\gamma_2\tau^2\lambda\frac{\langle k^2\rangle - \langle k\rangle^2}{\langle k\rangle} \begin{cases} > 0, & \text{if } \lambda < \lambda_c, \\ < 0, & \text{if } \lambda > \lambda_c. \end{cases} \tag{22}$$

If $\lambda < \lambda_c$, then $\lambda\tau\frac{\langle k^2\rangle}{\langle k\rangle} < \beta$ since $\lambda_c < \frac{\beta\langle k\rangle}{\tau\langle k^2\rangle}$. It implies that the cubic equation (21) has three roots ν_1, ν_2 and ν_3 satisfying

$$-\infty < \nu_1 < 0 < \nu_2 < \lambda\tau\frac{\langle k^2\rangle}{\langle k\rangle} < \nu_3 < \beta.$$

If $\lambda > \lambda_c$, then the cubic equation (21) has three roots satisfying

$$-\infty < \nu_1 < 0 < \nu_2 < \beta < \nu_3 < +\infty.$$

In a word, there exists a unique positive eigenvalue of J if and only if $\lambda > \lambda_c$, below which the unique epidemic equilibrium exists. Otherwise all real-valued eigenvalues of J are negative, this implies that the disease-free equilibrium is globally stable according to Lemma 1 in paper [14]. □

In paper [20], the authors follow the concepts of next-generation matrix (NGM) to give a threshold - the basic reproduction number R_0, by which the global stability of a modified SIS model is studied. The NGM is a matrix that relates the numbers of newly infected individuals in various categories in consecutive generation, and the basic reproduction number R_0 is the spectral radius of the NGM (refer to the papers [25, 26] for details). However, the direct relationship between the epidemic threshold and the basic reproduction number is not clearly revealed. In fact, we can reveal that $\lambda = \lambda_c$ if and only if $R_0 = 1$ by the same way as above.

3.3 Global attraction of endemic equilibrium

In this part, we show a proposition and prove the global attraction of the endemic equilibrium by the same way as the one in [3, 8]. Inequalities (27) and (28) in Proposition 2 are helpful to prove the main result (Theorem 3).

Proposition 1 *Suppose that* $\alpha \geq \beta$ *and the solution* $I_k(t)$ *of system* (2) *satisfies* $\limsup_{t\to\infty} I_k(t) \leq U_k$, $\liminf_{t\to\infty} I_k(t) \geq L_k$, *then*

$$\limsup_{t\to\infty} V(t) \leq \frac{\gamma_2\langle U_k\rangle}{1+\gamma_2\langle U_k\rangle}, \qquad \liminf_{t\to\infty} V(t) \geq \frac{\gamma_2\langle L_k\rangle}{1+\gamma_2\langle L_k\rangle}$$

and

$$\limsup_{t\to\infty}\big(1-R_k(t)\big) \leq \frac{(\alpha-\beta)U_k+\delta}{\alpha+\delta}, \qquad \liminf_{t\to\infty}\big(1-R_k(t)\big) \geq \frac{(\alpha-\beta)L_k+\delta}{\alpha+\delta}.$$

Proof Without loss of generality, we only verify the upper limit inequalities. From $\limsup_{t\to\infty} I_k(t) \leq U_k$, we obtain that for any $\epsilon > 0$, there exists $\tau_0 > 0$ such that $I_k(t) \leq$

$U_k + \epsilon$ for $t > \tau_0$. It follows that

$$\begin{aligned}\frac{dV(t)}{dt} &= -V(t) + \gamma_2\big(1 - V(t)\big)\rho(t) \le -V(t) + \gamma_2\big(1 - V(t)\big)\langle U_k + \epsilon\rangle \\ &= -\big(1 + \gamma_2\langle U_k + \epsilon\rangle\big)V(t) + \gamma_2\langle U_k + \epsilon\rangle \end{aligned} \tag{23}$$

for $t > \tau_0$. Therefore, the following inequality holds since $\epsilon > 0$ is arbitrarily small:

$$\limsup_{t\to\infty} V(t) \le \frac{\gamma_2\langle U_k\rangle}{1 + \gamma_2\langle U_k\rangle}. \tag{24}$$

At the same time, it follows from the second equation of system (2) and $\alpha \ge \beta$ that

$$\begin{aligned}\frac{d(1 - R_k(t))}{dt} &= -(\alpha + \delta)\big(1 - R_k(t)\big) + (\alpha - \beta)I_k(t) + \delta \\ &\le -(\alpha + \delta)\big(1 - R_k(t)\big) + (\alpha - \beta)(U_k + \epsilon) + \delta. \end{aligned} \tag{25}$$

Consequently,

$$\limsup_{t\to\infty}\big(1 - R_k(t)\big) \le \frac{(\alpha - \beta)U_k + \delta}{\alpha + \delta}. \tag{26}$$

□

Proposition 2 *Suppose that* $\alpha \ge \beta$ *and the solution* $I_k(t)$ *of system* (2) *satisfies* $\limsup_{t\to\infty} I_k(t) \le U_k$, $\liminf_{t\to\infty} I_k(t) \ge L_k$, *then*

$$\limsup_{t\to\infty} I_k(t) \le \frac{[\lambda\frac{k}{\langle k\rangle}\langle kU_k\rangle + \frac{\gamma_1\gamma_2\langle U_k\rangle}{1+\gamma_2\langle U_k\rangle}][\frac{\delta+(\alpha-\beta)U_k}{\alpha+\delta}]}{\beta + \lambda\frac{k}{\langle k\rangle}\langle kU_k\rangle + \frac{\gamma_1\gamma_2\langle U_k\rangle}{1+\gamma_2\langle U_k\rangle}}, \tag{27}$$

$$\liminf_{t\to\infty} I_k(t) \ge \frac{[\lambda\frac{k}{\langle k\rangle}\langle kL_k\rangle + \frac{\gamma_1\gamma_2\langle L_k\rangle}{1+\gamma_2\langle L_k\rangle}][\frac{\delta+(\alpha-\beta)L_k}{\alpha+\delta}]}{\beta + \lambda\frac{k}{\langle k\rangle}\langle kL_k\rangle + \frac{\gamma_1\gamma_2\langle L_k\rangle}{1+\gamma_2\langle L_k\rangle}}. \tag{28}$$

Proof From $\limsup_{t\to\infty} I_k(t) \le U_k$ and Proposition 1, one obtains that for any $\epsilon > 0$, there exists large enough τ such that the following inequalities hold for $t > \tau$:

$$\Theta(t,k) \le \frac{\langle k(U_k + \epsilon)\rangle}{\langle k\rangle}, \tag{29}$$

$$V(t) \le \frac{\gamma_2 U_k}{1 + \gamma_2 U_k} + \epsilon, \tag{30}$$

$$1 - R_k(t) \le \frac{(\alpha - \beta)U_k + \delta}{\alpha + \delta} + \epsilon. \tag{31}$$

Considering the first equation of system (2), for $t > \tau$, it follows from (29), (30) and (31) that

$$\begin{aligned}\frac{dI_k(t)}{dt} &= \big[\lambda k\Theta(t,k) + \gamma_1 V(t)\big]\big[1 - I_k(t) - R_k(t)\big] - \beta I_k(t) \\ &\le \left[\lambda\frac{k}{\langle k\rangle}\big\langle k(U_k + \epsilon)\big\rangle + \gamma_1\left(\frac{\gamma_2 U_k}{1 + \gamma_2 U_k} + \epsilon\right)\right] \\ &\quad\times\left[\frac{(\alpha - \beta)U_k + \delta}{\alpha + \delta} + \epsilon - I_k(t)\right] - \beta I_k(t)\end{aligned}$$

$$= -\left[\beta + \lambda\frac{k}{\langle k\rangle}\langle k(U_k+\epsilon)\rangle + \gamma_1\left(\frac{\gamma_2 U_k}{1+\gamma_2 U_k}+\epsilon\right)\right]I_k + \left[\lambda\frac{k}{\langle k\rangle}\langle k(U_k+\epsilon)\rangle + \gamma_1\left(\frac{\gamma_2 U_k}{1+\gamma_2 U_k}+\epsilon\right)\right]\left[\frac{(\alpha-\beta)U_k+\delta}{\alpha+\delta}+\epsilon\right]. \tag{32}$$

Since $\epsilon > 0$ is arbitrary small, we get

$$\limsup_{t\to\infty} I_k(t) \le \frac{[\lambda\frac{k}{\langle k\rangle}\langle kU_k\rangle + \frac{\gamma_1\gamma_2\langle U_k\rangle}{1+\gamma_2\langle U_k\rangle}][\frac{\delta+(\alpha-\beta)U_k}{\alpha+\delta}]}{\beta+\lambda\frac{k}{\langle k\rangle}\langle kU_k\rangle + \frac{\gamma_1\gamma_2\langle U_k\rangle}{1+\gamma_2\langle U_k\rangle}}.$$

Similarly, we can prove inequality (28). □

Denote $\triangle_k = \{(I_k, R_k) | 0 \le I_k + R_k \le 1, I_k \ge 0, R_k \ge 0\}$, $k = 1, 2, \ldots, n$, and $\triangle = \prod_{k=1}^{n} \triangle_k \times [0,1]$.

Theorem 3 *If $\lambda > \lambda_c$ and $\alpha \ge \beta$, then system* (2) *has a unique endemic equilibrium $E^* = \{I_1^*, R_1^*, \ldots, I_n^*, R_n^*, V^*\}$ which is of global attraction in $\triangle - \{F^*\}$, where $F^* = \{0, \frac{\alpha}{\alpha+\delta}, 0, \frac{\alpha}{\alpha+\delta}, \ldots, 0, \frac{\alpha}{\alpha+\delta}, 0\}$ is the disease-free equilibrium of system* (2).

Proof Define a map $\mathcal{G} = \{\mathcal{G}_1, \mathcal{G}_2, \ldots, \mathcal{G}_n\} : \mathbb{R}^n \mapsto \mathbb{R}^n$ as follows:

$$\mathcal{G}_k(x_1, x_2, \ldots, x_n) = \frac{[\lambda\frac{k}{\langle k\rangle}\langle kx_k\rangle + \frac{\gamma_1\gamma_2\langle x_k\rangle}{1+\gamma_2\langle x_k\rangle}][\frac{\delta+(\alpha-\beta)x_k}{\alpha+\delta}]}{\beta+\lambda\frac{k}{\langle k\rangle}\langle kx_k\rangle + \frac{\gamma_1\gamma_2\langle x_k\rangle}{1+\gamma_2\langle x_k\rangle}} \quad (k = 1, 2, \ldots, n). \tag{33}$$

Let $U_k^{(1)} = 1$ for all $k = 1, 2, \ldots, n$ and $U_k^{(m+1)} = \mathcal{G}_k(U_1^{(m)}, U_2^{(m)}, \ldots, U_n^{(m)})$, it is obvious that $\limsup_{t\to\infty} I_k(t) \le U_k^{(1)} = 1$. According to Proposition 2, we then obtain

$$\limsup_{t\to\infty} I_k(t) \le U_k^{(m)}, \quad k = 1, 2, \ldots, n, m = 1, 2, \ldots.$$

Moreover, for all $k = 1, 2, \ldots, n$, we can testify the convergence of the sequences $\{U_k^{(m)}\}_{m=1}^{+\infty}$ by induction. First, it is obvious that $U_k^{(2)} < U_k^{(1)} = 1$. Secondly, if $U_k^{(m+1)} \le U_k^{(m)}$, then the reader can easily verify that $U_k^{(m+2)} \le U_k^{(m+1)}$. It implies that the sequence $\{U_k^{(m)}\}$ is convergent. Denoted by $U_k = \lim_{m\to\infty} U_k^{(m)}$, we then have $\limsup_{t\to\infty} I_k(t) \le U_k$, $k = 1, 2, \ldots, n$.

Let $\mathcal{H}(\Theta) = \frac{1}{\langle k\rangle}\langle k\mathcal{G}_k\rangle = \frac{1}{\langle k\rangle}\langle k\frac{[\lambda\frac{k}{\langle k\rangle}\langle kx_k\rangle + \frac{\gamma_1\gamma_2\langle x_k\rangle}{1+\gamma_2\langle x_k\rangle}][\frac{\delta+(\alpha-\beta)x_k}{\alpha+\delta}]}{\beta+\lambda\frac{k}{\langle k\rangle}\langle kx_k\rangle + \frac{\gamma_1\gamma_2\langle x_k\rangle}{1+\gamma_2\langle x_k\rangle}}\rangle$, where $\Theta = \frac{1}{\langle k\rangle}\langle kx_k\rangle$. One obtains that $\mathcal{H}'(\Theta)|_{\Theta=0} > 1$ if $\lambda > \lambda_c$. It implies that $\frac{1}{\langle k\rangle}\langle k\mathcal{G}_k\rangle > \frac{1}{\langle k\rangle}\langle kx_k\rangle$ when $x_k > 0$ $(k = 1, 2, \ldots, n)$ is small enough.

Denote $L_k^{(m+1)} = \mathcal{G}_k(L_1^{(m)}, L_2^{(m)}, \ldots, L_n^{(m)})$ for each $k = 1, 2, \ldots, n$. According to Lemma 1 in paper [14], we can take $L_k^{(1)}$ small enough such that $\forall k$, $0 < L_k^{(1)} < \liminf_{t\to\infty} I_k(t)$ and $L_k^{(2)} > L_k^{(1)}$. If $L_k^{(m)} \ge L_k^{(m-1)}$, it is easy to testify that $L_k^{(m+1)} \ge L_k^{(m)}$. In result, the sequences $\{L_k^{(m)}\}_{m=1}^{+\infty}$ are convergent for all k, and denote $L_k = \lim_{m\to\infty} L_k^{(m)}$.

Both $\{L_k\}$ and $\{U_k\}$ satisfy the following equation:

$$I_k = \frac{[\lambda\frac{k}{\langle k\rangle}\langle kI_k\rangle + \gamma_1\frac{\gamma_2\langle I_k\rangle}{1+\gamma_2\langle I_k\rangle}][\frac{\delta+(\alpha-\beta)I_k}{\alpha+\delta}]}{\beta+\lambda\frac{k}{\langle k\rangle}\langle kI_k\rangle + \gamma_1\frac{\gamma_2\langle I_k\rangle}{1+\gamma_2\langle I_k\rangle}}. \tag{34}$$

After some transformations, we can find that both $\{L_k\}$ and $\{U_k\}$ satisfy Eq. (7). Thus one obtains that $U_k = L_k = I_k^*$ and $\lim_{t\to\infty} I_k(t) = I_k^*$ according to the uniqueness of endemic equilibrium of system (1). The proof of Theorem 3 is completed. □

4 Simulations

In the section above, some theoretical results for the proposed mean-field equations are revealed. For a finite size network, there exists an epidemic threshold λ_c, if the infection rate $\lambda < \lambda_c$, then the disease-free equilibrium is globally asymptotically stable. Otherwise, the disease-free equilibrium is unstable and a unique globally attracting endemic equilibrium exists.

In this section, we will perform some numerical simulations to confirm the theoretical results over BA (Barabási-Albert) scale-free networks which are generated by the preferential attachment algorithm [27]. All the networks used in the simulations were built using $N = 10^4$ nodes.

The epidemics are seeded with randomly chosen fraction of nodes to avoid stochastic extinction. The probability of a susceptible node with n ($\leq$ degree k) infectious neighbors being infected in small interval of time h is $1 - (1 - \lambda h)^n(1 - \gamma_1 hV(t))$ at step $t + 1$. The term $(1 - \lambda h)^n$ represents the probability that a susceptible node cannot be infected by his (her) infectious neighbors, and the term $1 - \gamma_1 hV(t)$ represents the probability that a susceptible node cannot be infected by infectious vectors. In this paper, we give the numerical simulations from different angles:

- The epidemic threshold λ_c that changes as functions of different model parameters.
- The final density of the infected nodes that changes as functions of different model parameters.

In stochastic simulations, the dynamics are totally evolved for 2,000 time steps, we set the time interval $h = 0.1$ and let $\rho = \frac{1}{T}\sum_{t=t_0}^{t_0-1+T} \rho(t)$ (here, $T = 100$, $t_0 = 1{,}901$) be the time average to reduce the fluctuation of $\rho(t)$. At the same time, to minimize random fluctuation caused by the initial conditions, we make average of ρ over 100 realizations of different initial infectious nodes. Let λ increase systematically by $\Delta\lambda$ beginning with $\lambda = 0$, if $\rho > 0.0005$ as $\lambda = \lambda^*$ and $\rho < 0.0005$ as $\lambda < \lambda^*$, we set $\lambda_c = \lambda^* - \Delta\lambda$.

In Figure 1, we illustrate the variation of the epidemic threshold λ_c with respect to the parameters γ_1 and γ_2 both for stochastic simulations (SS) and also for mean-field (MF)

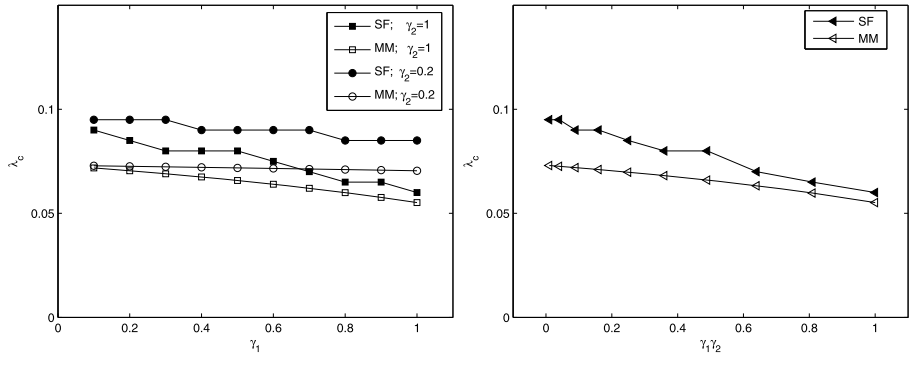

Figure 1 Illustration of the variation of the epidemic threshold with respect to the parameters γ_1 and γ_2. Left: Plot of λ_c *versus* γ_1 for different γ_2. Right: Plot of λ_c *versus* $\gamma_1\gamma_2$. Other parameters are chosen as $\alpha = 1.05$, $\beta = 0.9$, $\delta = 0.8$ and $\langle k\rangle = 10$. 'SF' means stochastic simulations and 'MM' means mean-field predictions.

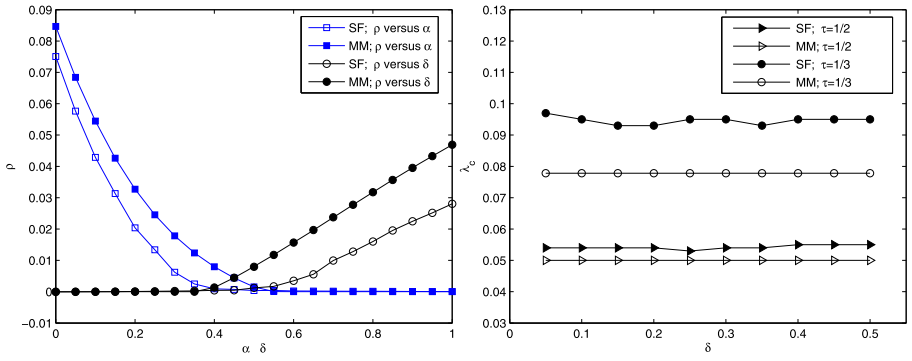

Figure 2 Illustration of the variations of the final infected density and the epidemic threshold with respect to the parameters α and δ. Left: Plot of ρ *versus* α and δ. When considering ρ *versus* α, we set $\delta = 0.5$; when considering ρ *versus* δ, we set $\alpha = 0.4$. Right: The variation of the infection threshold λ_c with respect to δ over BA scale-free network for different $\tau = \frac{\delta}{\alpha+\delta}$. Other parameters are chosen as $\gamma_1 = \gamma_2 = 0.6$, $\beta = 0.8$ and $\langle k \rangle = 10$.

predictions formula (18). It is clear that the epidemic threshold λ_c decreases as γ_1 (or γ_2) increases. We thus conclude that reducing the contacts between individuals and vectors can effectively control the spread of the disease over networks. From this angle, the stochastic simulations agree well with the mean-field predictions. The discrepancy between these is also shown in our simulations, we can see the stochastic simulations are slightly larger than the theoretical predictions for threshold λ_c, which is likely to be due to a distribution cutoff effect on a finite size network [28] and other neglected factors (for instance, network is static and has degree-correlations). Moreover, one can find that for larger infection rates γ_1 and γ_2, the error between stochastic simulation and theoretical predictions is smaller, which is likely to be attributed to the contacts homogeneity between individuals and vectors.

Figure 2 illustrates the variations of the final infected density ρ (left) and the epidemic threshold λ_c (right) with respect to parameters α and δ, respectively. It is clear that the discrepancy remains for the effect of a finite size network. The theoretical prediction of formula (18) shows that for different parameters α and δ, the epidemic threshold λ_c is unchanged as long as $\tau = \frac{\delta}{\alpha+\delta}$ is unchanged. From Figure 2 we can see that the stochastic simulations are in accordance with mean-field predictions disregarding the slight fluctuations for the effect of the stochastic factor. Also, one can see that the larger direct immunization rate α, the larger the epidemic threshold is from Figure 2 (right). Namely, the direct immunization can increase the critical threshold of epidemic spreading on complex networks and reduce the prevalence of infectious disease, which agrees with the results of paper [24]. Consequently, we can prevent the disease spreading by improving immunization strength.

Figure 3 reflects the relation between the final densities of infected nodes and infection rate λ for different average degree. One can see that the larger average degree, the lower the discrepancy rate is. We simulate the time series of total densities of infected nodes on the BA network with $\langle k \rangle = 20$ in Figure 4. We can obtain that the epidemic threshold $\lambda_c = 0.032$ from formula (18). It is clear that if $\lambda < \lambda_c$, the disease will disappear quickly; otherwise $\lambda > \lambda_c$ the disease will persist in this system. Considering the factor that the epidemic threshold λ_c of stochastic simulations is larger than the one of the mean-field

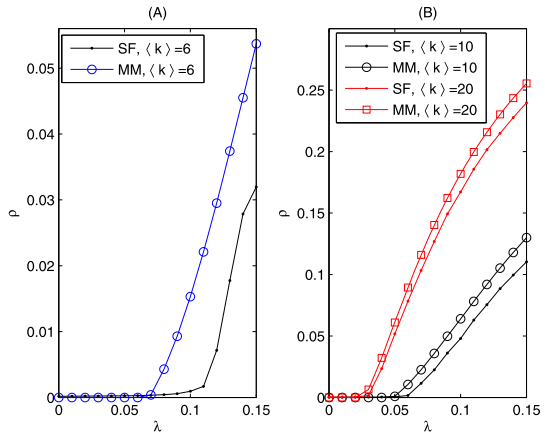

Figure 3 The variation of the stable densities of infected nodes with respect to infection rate λ for BA scale-free network, where $\alpha = 0.83$, $\beta = 0.7$, $\delta = 0.71$, $\gamma_1 = 0.45$, $\gamma_2 = 0.54$.

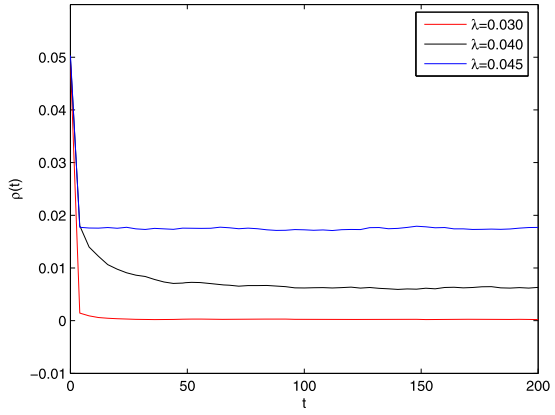

Figure 4 The total densities of infected nodes that change as time series on BA scale-free network with $\langle k \rangle = 20$, where $\alpha = 0.83$, $\beta = 0.8$, $\delta = 0.7$, $\gamma_1 = 0.60$, $\gamma_2 = 0.60$.

predictions, we think the stochastic simulations are reasonably consistent with the theoretic results.

5 Conclusions

In order to better explain the mechanism of spreading of epidemics, we have investigated a novel SIRS model with direct immunization via two infection mechanisms in this paper. The model is approximately described by the mean-field method neglecting contact duration.

The disease-free equilibrium and endemic equilibrium and their dynamics are discussed in this paper. Our theoretic results show that for finite size networks, there exists epidemic threshold λ_c, below which the disease-free equilibrium is globally asymptotically stable; otherwise a unique endemic equilibrium exists. We prove theoretically that the endemic equilibrium is of attraction when $\lambda > \lambda_c$ under the assumption that $\alpha \geq \beta$. To go a step further, we perform some numerical simulations to test and verify our theoretical results.

From simulations as above, we can find that the discrepancies between stochastic simulations and theoretical predictions remain for the effect of a finite size network [28] and stochastic factors. Disregarding these slight errors, we think that the numerical simulations confirm to the theoretical results, and the mean-field approach is of effectiveness. Especially, one can see that the larger transmission rates from infected vectors to susceptible individuals γ_1 or from infected individuals to susceptible vectors γ_2, the better the simulations accord with the mean-field predictions. Just as mentioned above, this may attribute to the homogeneity of contacts between individuals and vectors.

Competing interests
The authors declare that they have no competing interests.

Authors' contributions
All authors contributed equally to the writing of this paper. All authors read and approved the final manuscript.

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