Research Article

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Global analysis and control for a vector-borne epidemic model with multi-edge infection on complex networks

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Abstract: In this study, a vector-borne epidemic model with multi-edge infection on complex networks is built. Using the method of next-generation matrix, the basic reproduction number R_0 is calculated, and if $R_0 < 1$, the disease-free equilibrium E_0 is globally asymptotically stable; if $R_0 > 1$, there exists a unique endemic equilibrium $i^* = (i_1^*, i_2^*, \ldots, i_n^*)$ that is globally attractive. Moreover, three control strategies are proposed to control the spread of infectious diseases. Finally, some numerical simulations are given to illustrate our theoretical results.

Keywords: vector-borne epidemic model, global analysis, control strategy, complex networks

MSC 2020: 34D20, 35A25, 92C37

1 Introduction

Vector-borne disease is a kind of disease that spreads pathogens to people through animals (e.g., mosquitoes and fleas). Common vector-borne infectious diseases include avian influenza, malaria, plague, Japanese encephalitis, rabies, schistosomiasis, etc. A vector-borne disease model was built by Cooke [1] for the first time. Subsequently, this model was extended and modified by Marcati and Pozio [2], Volz [3], Beretta [4–6], and Busenberg and Cooke [7]. Some traditional models consider only the propagation of vector-borne disease in homogeneous mixing models. These models [8–11] ignore the influence of population structure on the transmission of infectious diseases, which is inconsistent with reality [12]. Actually, population structure, personal behavior, geographical distance, and contact patterns may lead to diversity. Therefore, it is more practical to study the spread of infectious diseases on complex networks. Pastor-Satorras and Vespignani [13,14] established an infectious disease model on complex networks and obtained the infection threshold, which is important for persistence, but they also found that there may be no infection threshold on scale-free networks, which is a very interesting phenomenon. Then, they considered immunization on complex networks. Their results laid the foundation for the future study of the spread of infectious diseases on complex networks.

In recent years, the study of the spread of infectious disease on complex networks has attracted wide attention [15–21]. Two edge-based epidemic models are proposed on heterogeneous networks [22,23]. Wei et al. [24] and Guo et al. [25] studied the coupled epidemic model in two-layered interconnected network. Wang et al. [26] and Bodó and Simon [27] studied the epidemic model on adaptive networks. Wang et al. [17],

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Wang et al. [28], and Zhu et al. [29] studied three SIS epidemic models on complex networks. Lately, the model with vector-borne were studied widely on networks [30–34]. But most models ignored the assumption that the infection rates of multi-edge interfere with one another in complex networks, especially for the vector-borne diseases. Hence, few people pay attention to the vector-borne disease with multi-edge infection on complex networks. Therefore, this article considers a new SIS-SI epidemic model (vector-borne disease model) in complex networks under the assumption that the infection rates of multiple edges interfere with one another. When $k = 1$, our model becomes the model of reference [28]. The method and conclusion used in this article are generalizations of the study by Wang et al. [28]. Such an approach may provide new insights into the construction of other similar epidemic spreading models on complex networks, such as SIR and SEIR models with a vector.

The objectives of this article are as follows. First, a vector-borne model with multi-edge infection on complex networks is built. Second, using the concepts of next-generation matrix, the basic reproduction number R_0 is calculated. In addition, some conditions for the existence and stability of the disease-free equilibrium and the endemic equilibrium are given. Third, control strategies are proposed to control the transmission of infectious diseases. Finally, some numerical simulations are implemented to illustrate the theoretical results.

The rest of the article is organized as follows: in Section 2, a vector-borne model with multi-edge infection on complex networks is built; in Section 3, dynamics analysis is discussed, containing the basic reproduction number and the existence and stability of the disease-free equilibrium and the endemic equilibrium; in Section 4, three control strategies are proposed; in Section 5, some numerical simulations are implemented to support the theoretical prediction; in Section 6, conclusions are given.

2 Model derivation

In [28], by the mean-field method, they proposed the following SIS–SIS model with vector-borne on complex networks:

$$\begin{cases} \dfrac{\mathrm{d}S_k(t)}{\mathrm{d}t} = -r_1 k S_k(t)\Theta(i(t)) - r_2 S_k(t) I^M(t) + \nu I_k(t), \\ \dfrac{\mathrm{d}I_k(t)}{\mathrm{d}t} = r_1 k S_k(t)\Theta(i(t)) + r_2 S_k(t) I^M(t) - \nu I_k(t), \\ \dfrac{\mathrm{d}S^M(t)}{\mathrm{d}t} = -r_3 S^M(t) I(t) + \mu I^M(t), \\ \dfrac{\mathrm{d}I^M(t)}{\mathrm{d}t} = r_3 S^M(t) I(t) - \mu I^M(t), \end{cases} \tag{1}$$

where $S_k(t)$ and $I_k(t)$ denote the number of susceptible and infective nodes of degree k at time t, respectively. $S^M(t)$ and $I^M(t)$ denote the number of susceptible and infective female mosquitoes at time t. r_1 is the transmission rate from infected humans to susceptible humans. r_2 is the transmission rate from infected mosquitoes to susceptible humans. ν represents the probability that an infected individual is cured and becomes susceptible. r_3 denotes the transmission rate from infected humans to susceptible mosquitoes through biting. μ is recover rate of female mosquitoes.

Let S_k and I_k denote susceptible and infected individuals with degree k, $k = 1, 2, \ldots, n$, respectively. If S_2 is a susceptible individual with two neighbor, and if we suppose that two adjacent nodes are infected nodes, then the probability of S_2 being infected is $1 - (1 - r_1)^2$, where r_1 represents the infection rate and $0 < r_1 < 1$. S_3 is a susceptible individual with three neighbor, and if we also suppose that three adjacent nodes are infected nodes, then the probability of S_3 being infected is $1 - (1 - r_1)^3$. As a result, we obtain that the probability of S_k being infected is $1 - (1 - r_1)^k$.

Now we consider the spread of infectious diseases in heterogeneous networks. Let $P(k)$ be a degree distribution, $k = 1, 2, \ldots, n$. $P(k'|k)$ be the probability that any node with degree k points to an node of

degree k'. Denote $N_k = S_k + I_k$ as the number of nodes with degree k. Then, $\Theta(I(t)) = \frac{\sum_{k=1}^{n} kI_k}{\sum_{k=1}^{n} kN_k} = \sum_{k=1}^{n} P(k'|k)\frac{I_k}{N_k}$ describes the probability that any node with degree k is connected to the infection node of degree k'. Using the above method, we obtain that the probability of S_k being infected is $1 - (1 - r_1\Theta(I(t)))^k$. Motivated by system (1), the following vector-borne infectious disease model with multi-edge infection is established:

$$\begin{cases} \dfrac{\mathrm{d}S_k(t)}{\mathrm{d}t} = -[1 - (1 - r_1\Theta(I(t)))^k]S_k(t) - r_2 S_k(t) I^M(t) + \nu I_k(t), \\ \dfrac{\mathrm{d}I_k(t)}{\mathrm{d}t} = [1 - (1 - r_1\Theta(I(t)))^k]S_k(t) + r_2 S_k(t) I^M(t) - \nu I_k(t), \\ \dfrac{\mathrm{d}S^M(t)}{\mathrm{d}t} = \mu N^M - r_3 S^M(t) I(t) - \mu S^M(t), \\ \dfrac{\mathrm{d}I^M(t)}{\mathrm{d}t} = r_3 S^M(t) I(t) - \mu I^M(t), \end{cases} \tag{2}$$

where μ is the birth (death) rate of female mosquitoes. $I(t) = \sum_{k=1}^{n} P(k) I_k(t)$, $N^M = S^M(t) + I^M(t)$, $N = \sum_{k=1}^{n} N_k$.

Let $s_k(t) = \frac{S_k(t)}{N_k}$, $i_k(t) = \frac{I_k(t)}{N_k}$, $s^M(t) = \frac{S^M(t)}{N^M}$, $i^M(t) = \frac{S^M(t)}{N^M}$, $\beta_1 = r_1$, $\beta_2 = r_2 N^M$, $\beta_3 = r_3 N$, $i(t) = \sum_{k=1}^{n} P(k) i_k(t)$, and $\langle k \rangle = \sum_{k=1}^{n} kP_k$. If we suppose that the network is uncorrelated, namely, $P(k'|k) = k'P(k')/\langle k \rangle$ and $\theta(i(t)) = \frac{1}{\langle k \rangle}\sum_{k=1}^{n} kP(k) i_k(t)$, then, by the normalization condition, equation (2) can be rewritten as follows:

$$\begin{cases} \dfrac{\mathrm{d}i_k(t)}{\mathrm{d}t} = [1 - (1 - \beta_1\theta(i(t)))^k](1 - i_k(t)) + \beta_2(1 - i_k(t)) i^M(t) - \nu i_k(t), \\ \dfrac{\mathrm{d}i^M(t)}{\mathrm{d}t} = \beta_3(1 - i^M(t)) i(t) - \mu i^M(t). \end{cases} \tag{3}$$

Generally, the mosquito population is huge, so the number of infected mosquitoes depends on the number of infected people. We further suppose that the number of infected mosquitoes is proportional to the number of infected people [1,35]. Let $i^M(t) = \alpha i(t)$, where α is the scale factor. Then, equation (2) is further simplified as follows:

$$\frac{\mathrm{d}i_k(t)}{\mathrm{d}t} = [1 - (1 - \beta_1\theta(i(t)))^k](1 - i_k(t)) + \beta_4(1 - i_k(t)) i(t) - \nu i_k(t), \tag{4}$$

where $\beta_4 = \alpha\beta_2$.

3 Dynamical analysis

In this section, we will study the dynamics of the new proposed system (4).

3.1 Basic reproduction number

Theorem 3.1. $R_0 = \frac{1}{2}\left(\frac{\beta_1\langle k^2 \rangle}{\nu\langle k \rangle} + \frac{\beta_4}{\nu}\right) + \frac{1}{2}\sqrt{\Delta}$ *is the basic reproduction number of system* (4), *where* $\Delta = \left(\frac{\beta_1\langle k^2 \rangle}{\nu\langle k \rangle} - \frac{\beta_4}{\nu}\right)^2 + \frac{4\beta_1\beta_4\langle k \rangle}{\nu^2}$.

Proof. Obviously, there is a unique disease-free equilibrium $E_0 = (0, 0, \ldots, 0)$ for system (4). In the following, we use the method proposed by van den Driessche and Watmough [36] (the next generation matrix) to compute the basic reproduction number, $R_0 = \rho(FV^{-1})$. The matrices F and V are denoted as follows:

$$F = \begin{pmatrix} \frac{1P(1)}{\langle k\rangle}\beta_1 + P(1)\beta_4 & \frac{2P(2)}{\langle k\rangle}\beta_1 + P(2)\beta_4 & \cdots & \frac{nP(n)}{\langle k\rangle}\beta_1 + P(n)\beta_4 \\ \frac{1P(1)}{\langle k\rangle}2\beta_1 + P(1)\beta_4 & \frac{2P(2)}{\langle k\rangle}2\beta_1 + P(2)\beta_4 & \cdots & \frac{nP(n)}{\langle k\rangle}2\beta_1 + P(n)\beta_4 \\ \vdots & \vdots & \vdots & \vdots \\ \frac{1P(1)}{\langle k\rangle}(n-1)\beta_1 + P(1)\beta_4 & \frac{2P(2)}{\langle k\rangle}(n-1)\beta_1 + P(2)\beta_4 & \cdots & \frac{nP(n)}{\langle k\rangle}(n-1)\beta_1 + P(n)\beta_4 \\ \frac{1P(1)}{\langle k\rangle}n\beta_1 + P(1)\beta_4 & \frac{2P(2)}{\langle k\rangle}n\beta_1 + P(2)\beta_4 & \cdots & \frac{nP(n)}{\langle k\rangle}n\beta_1 + P(n)\beta_4 \end{pmatrix}$$

and

$$V = \begin{pmatrix} \nu & 0 & \cdots & 0 \\ 0 & \nu & \cdots & 0 \\ \vdots & \vdots & \vdots & \vdots \\ 0 & 0 & \cdots & \nu \end{pmatrix}.$$

Denote $g(\lambda) = |FV^{-1} - \lambda E|$, where E is identity matrix and $|FV^{-1} - \lambda E|$=

$$\begin{vmatrix} \frac{1P(1)}{\nu\langle k\rangle}\beta_1 + \frac{P(1)\beta_4}{\nu} - \lambda & \frac{2P(2)}{\nu\langle k\rangle}\beta_1 + \frac{P(2)\beta_4}{\nu} & \cdots & \frac{nP(n)}{\nu\langle k\rangle}\beta_1 + \frac{P(n)\beta_4}{\nu} \\ \frac{1P(1)}{\nu\langle k\rangle}2\beta_1 + \frac{P(1)\beta_4}{\nu} & \frac{2P(2)}{\nu\langle k\rangle}2\beta_1 + \frac{P(2)\beta_4}{\nu} - \lambda & \cdots & \frac{nP(n)}{\nu\langle k\rangle}2\beta_1 + \frac{P(n)\beta_4}{\nu} \\ \vdots & \vdots & \vdots & \vdots \\ \frac{1P(1)}{\nu\langle k\rangle}n\beta_1 + \frac{P(1)\beta_4}{\nu} & \frac{2P(2)}{\nu\langle k\rangle}n\beta_1 + \frac{P(2)\beta_4}{\nu} & \cdots & \frac{nP(n)}{\nu\langle k\rangle}n\beta_1 + \frac{P(n)\beta_4}{\nu} - \lambda \end{vmatrix}.$$

When $l = 2, 3, \ldots, n$, the lth column multiplied by l adds to the first column, and we can obtain

$$g(\lambda) = \left(\frac{\beta_1\langle k^2\rangle}{\nu\langle k\rangle} - \lambda\right)g_1(\lambda) + \frac{\beta_4\langle k\rangle}{\nu}g_2(\lambda),$$

where

$$g_1(\lambda) = \begin{vmatrix} 1 & \frac{2P(2)}{\nu\langle k\rangle}\beta_1 + \frac{P(2)\beta_4}{\nu} & \cdots & \frac{nP(n)}{\nu\langle k\rangle}\beta_1 + \frac{P(n)\beta_4}{\nu} \\ 2 & \frac{2P(2)}{\nu\langle k\rangle}2\beta_1 + \frac{P(2)\beta_4}{\nu} - \lambda & \cdots & \frac{nP(n)}{\nu\langle k\rangle}2\beta_1 + \frac{P(n)\beta_4}{\nu} \\ \vdots & \vdots & \vdots & \vdots \\ n & \frac{2P(2)}{\nu\langle k\rangle}n\beta_1 + \frac{P(2)\beta_4}{\nu} & \cdots & \frac{nP(n)}{\nu\langle k\rangle}n\beta_1 + \frac{P(n)\beta_4}{\nu} - \lambda \end{vmatrix}$$

and

$$g_2(\lambda) = \begin{vmatrix} 1 & \frac{2P(2)}{\nu\langle k\rangle}\beta_1 + \frac{P(2)\beta_4}{\nu} & \cdots & \frac{nP(n)}{\nu\langle k\rangle}\beta_1 + \frac{P(n)\beta_4}{\nu} \\ 1 & \frac{2P(2)}{\nu\langle k\rangle}2\beta_1 + \frac{P(2)\beta_4}{\nu} - \lambda & \cdots & \frac{nP(n)}{\nu\langle k\rangle}2\beta_1 + \frac{P(n)\beta_4}{\nu} \\ \vdots & \vdots & \vdots & \vdots \\ 1 & \frac{2P(2)}{\nu\langle k\rangle}n\beta_1 + \frac{P(2)\beta_4}{\nu} & \cdots & \frac{nP(n)}{\nu\langle k\rangle}n\beta_1 + \frac{P(n)\beta_4}{\nu} - \lambda \end{vmatrix}.$$

For the determinant $g_1(\lambda)$, the first column multiplied by $-\frac{\beta_1 lP(l)}{\nu\langle k\rangle}$ adds to the lth column and the first row multiplied by -1 adds to the lth row. When $l = 2, 3, \ldots, n$, the lth column multiplied by $\frac{l-1}{\lambda}(\lambda \neq 0)$ adds to the first column. We have

$$g_1(\lambda) = (-\lambda)^{n-1}\left(1 + \frac{\beta_4\langle k\rangle}{\lambda\nu} - \frac{\beta_4}{\lambda\nu}\right).$$

For the determinant $g_2(\lambda)$, when $l = 2, 3, \ldots, n$, the first column multiplied by $-\frac{\beta_1 lP(l)}{\nu\langle k\rangle} - \frac{\beta_4 P(l)}{\nu}$ adds to the lth column, and expanding the determinant according to the first row, we obtain

$$g_2(\lambda) = \begin{vmatrix} \frac{2P(2)}{\nu\langle k\rangle}\beta_1 - \lambda & \frac{3P(3)}{\nu\langle k\rangle}\beta_1 & \cdots & \frac{nP(n)}{\nu\langle k\rangle}\beta_1 \\ \frac{2P(2)}{\nu\langle k\rangle}2\beta_1 & \frac{3P(3)}{\nu\langle k\rangle}\beta_1 - \lambda & \cdots & \frac{nP(n)}{\nu\langle k\rangle}2\beta_1 \\ \vdots & \vdots & \vdots & \vdots \\ \frac{2P(2)}{\nu\langle k\rangle}(n-1)\beta_1 & \frac{3P(3)}{\nu\langle k\rangle}(n-1)\beta_1 & \cdots & \frac{nP(n)}{\nu\langle k\rangle}(n-1)\beta_1 - \lambda \end{vmatrix},$$

which is equivalent to

$$g_2(\lambda) = \begin{vmatrix} 1 & \frac{2P(2)}{\nu\langle k\rangle}\beta_1 & \cdots & \frac{nP(n)}{\nu\langle k\rangle}\beta_1 \\ 0 & \frac{2P(2)}{\nu\langle k\rangle}2\beta_1 - \lambda & \cdots & \frac{nP(n)}{\nu\langle k\rangle}2\beta_1 \\ \vdots & \vdots & \vdots & \vdots \\ 0 & \frac{2P(2)}{\nu\langle k\rangle}(n-1)\beta_1 & \cdots & \frac{nP(n)}{\nu\langle k\rangle}(n-1)\beta_1 - \lambda \end{vmatrix}.$$

The first row multiplied by $(1 - l)$ adds to the lth row. When $l = 2, 3, \ldots, n$, the lth column multiplied by $\frac{1-l}{\lambda}(\lambda \neq 0)$ adds to the first column, and we obtain

$$g_2(\lambda) = (-\lambda)^{n-1}\left(1 - \frac{\beta_1\langle k^2\rangle}{\lambda\nu\langle k\rangle} + \frac{\beta_1}{\lambda\nu}\right).$$

Namely,

$$g(\lambda) = (-\lambda)^{n-2}\left[\lambda^2 - \left(\frac{\beta_1\langle k^2\rangle}{\nu\langle k\rangle} + \frac{\beta_4}{\nu}\right)\lambda + \left(\frac{\beta_1\beta_4\langle k^2\rangle}{\nu^2\langle k\rangle} - \frac{\beta_1\beta_4\langle k\rangle}{\nu^2}\right)\right].$$

Denote

$$f(\lambda) = \lambda^2 - \left(\frac{\beta_1\langle k^2\rangle}{\nu\langle k\rangle} + \frac{\beta_4}{\nu}\right)\lambda + \left(\frac{\beta_1\beta_4\langle k^2\rangle}{\nu^2\langle k\rangle} - \frac{\beta_1\beta_4\langle k\rangle}{\nu^2}\right).$$

Then, $f(\lambda)$ is a quadratic equation, and its discriminant is

$$\Delta = \left(\frac{\beta_1\langle k^2\rangle}{\nu\langle k\rangle} - \frac{\beta_4}{\nu}\right)^2 + \frac{4\beta_1\beta_4\langle k\rangle}{\nu^2} > 0.$$

Hence,

$$\lambda_1 = 0, \lambda_{2,3} = \frac{1}{2}\left(\frac{\beta_1\langle k^2\rangle}{\nu\langle k\rangle} + \frac{\beta_4}{\nu}\right) \pm \frac{1}{2}\sqrt{\Delta}.$$

Then, we can obtain

$$R_0 = \frac{1}{2}\left(\frac{\beta_1\langle k^2\rangle}{\nu\langle k\rangle} + \frac{\beta_4}{\nu}\right) + \frac{1}{2}\sqrt{\Delta}. \qquad \square$$

Remark 3.1. If $\beta_4 = 0$, then $R_0 = \frac{\beta_1\langle k^2\rangle}{\nu\langle k\rangle}$, which is consistent with the basic reproduction number in the studies by [14,37]

3.2 The stability for E_0

In this part, we first provide a lemma.

Lemma 3.1. [38] *Consider the following system*

$$\frac{\mathrm{d}i}{\mathrm{d}t} = Ai + N(i), \tag{5}$$

where A is an $n \times n$ matrix and $N(i)$ is continuously differentiable in a region $C \in R^n$. Suppose that

(1) *the compact convex set $D \in C$ is positively invariant with respect to system* (5) *and* $(0, 0, \ldots, 0) \in D$,
(2) $\lim_{t\to 0}\|N(i)\|/\|i\| = 0$,
(3) *there exist $w_0 > 0$ and a (real) eigenvector $w = (w_1, w_2, \ldots, w_n)^T$ of A^T such that $(w \cdot i) \geq w_0\|i\|$ for all $i \in D$,*
(4) $(w \cdot N(i)) \leq 0$ *for all $i \in D$,*
(5) *$i = (0, 0, \ldots, 0)$ is the largest positively invariant set for system* (5) *contained in $H = \{i \in D | w \cdot N(i) = 0\}$. Then, either $i = (0, 0, \ldots, 0)$ is globally asymptotically stable in D or, for any $i_0 \in D - (0, 0, \ldots, 0)$, the solution $\varphi(t, i_0)$ of* (5) *satisfies* $\liminf_{t\to\infty}\|\varphi(t, i_0)\| \geq m > 0$, *where $m > 0$, independent of the initial value i_0. Moreover, there exists a constant solution of* (5), $i^* \in D - (0, 0, \ldots, 0)$.

Denote $\Phi_n = \prod_{i=1}^{n}[0, 1]$.

Theorem 3.2. *Φ_n is a positively invariant set for system* (4).

Proof. We prove that Φ_n is a positively invariant set of system (4) by using the method in the study by Yorke [39]. Set two sets

$$\partial\Phi_n^1 = \{i = (i_1, i_2, \ldots, i_n) | i_k = 0 \text{ for some } k\},$$
$$\partial\Phi_n^2 = \{i = (i_1, i_2, \ldots, i_n) | i_k = 1 \text{ for some } k\}.$$

Denote η_k^1 and η_k^2 as outer normals, where $\eta_k^1 = (\overbrace{0, 0, \ldots, 0, -1}^{k}, 0, \ldots, 0)$ and $\eta_k^2 = (\overbrace{0, 0, \ldots, 0, 1}^{k}, 0, \ldots, 0)$. We have

$$\left.\frac{\mathrm{d}i}{\mathrm{d}t}\right|_{i_k=0} \cdot\eta_k^1 = -\sum_{j=0}^{k-1} C_k^j\left(-\frac{\beta_1}{\langle k\rangle}\sum_{l\neq k} lP(l)i_l(t)\right)^{k-j} - \beta_4\sum_{l\neq k} P(l)i_l(t) \leq 0, \quad k = 1, 2, \ldots, n,$$
$$\left.\frac{\mathrm{d}i}{\mathrm{d}t}\right|_{i_k=1} \cdot\eta_k^2 = -\nu \leq 0, \quad k = 1, 2, \ldots, n.$$

Therefore, any solution, starting in $\partial\Phi_n^1 \bigcup \partial\Phi_n^2$, remains inside Φ_n, i.e., Φ_n is a positively invariant set of the system (4). $\square$

Let $i = (i_1, i_2, \ldots, i_n)$ be a solution of system (4), then system (4) can be written as the compact vector form as follows:

$$\frac{\mathrm{d}i}{\mathrm{d}t} = Ai + N(i), \tag{6}$$

where $A = F - V$, Ai is the linear part, and $N(i) \leq 0$ is the nonlinear part.

Theorem 3.3. *If $R_0 < 1$, the disease-free equilibrium E_0 is globally asymptotically stable; otherwise, there at least exists a positive constant solution $i^* \in D - (0, 0, \ldots, 0)$.*

Proof. We will prove that system (6) satisfies all the conditions of Lemma 3.1. Let $D = \Phi_n$, then condition (1) of Lemma 3.1 is satisfied. Obviously, the condition (2) of Lemma 3.1 is satisfied.

Note that A^T is irreducible, and $a_{ij} \geq 0$ is non-diagonal element. It is known from the study by Varga [40] that there exists an eigenvector $w = (w_1, w_2, \ldots, w_n)^T > 0$. If we choose $w_0 = \min_{1 \leq k \leq n}\{w_k\}$, then for any $i = (i_1, i_2, \ldots, i_n) \in \Phi_n$, we obtain

$$(w \cdot i) \geq w_0\left(\sum_{k=1}^{n} i_k\right) \geq w_0\left(\sum_{k=1}^{n} i_k^2\right)^{\frac{1}{2}},$$

namely, $(w \cdot i) \geq w_0\|i\|$ for all $i \in \Phi_n$. Condition (3) is satisfied.

Because

$$-\sum_{j=0}^{k-1} C_k^j(-\beta_1\theta(i(t)))^{k-j} \leq k\beta_1\theta(i(t)), \quad 1 - i_k(t) \leq 1,$$

$$-\sum_{j=0}^{k-1} C_k^j(-\beta_1\theta(i(t)))^{k-j}(1 - i_k(t)) \leq k\beta_1\theta(i(t)).$$

Since

$$-\beta_4 i_k(t)\sum_{l=1}^{n} P(l)i_l(t) \leq 0,$$

we can have $N(i) \leq 0$. Condition (4) is satisfied.

Let $H = \{i \in \Phi_n | w \cdot N(i) = 0\}$. Because $w > 0$ and $N(i) \leq 0$, then for any $i \in \Phi_n$, we obtain $N(i) = 0$, namely,

$$\left[-\sum_{j=0}^{k-2} C_k^j(-\beta_1\theta(i(t)))^{k-j}(1 - i_k(t)) - \beta_1 k\theta(i(t))i_k(t) - \beta_4 i_k(t)\sum_{l=1}^{n} P(l)i_l(t)\right]w_k = 0.$$

Since $w_k > 0$ and

$$-\sum_{j=0}^{k-2} C_k^j(-\beta_1\theta(i(t)))^{k-j}(1 - i_k(t)) \leq 0, \quad -\beta_1 k\theta(i(t))i_k(t) \leq 0, \quad -\beta_4 i_k(t)\sum_{l=1}^{n} P(l)i_l(t) \leq 0.$$

We obtain that $N(i) = 0$ if and only if $i_k = 0$, namely, $i = (0, 0, \ldots, 0)$. So condition (5) is satisfied.

Hence, all conditions of Lemma (3.1) are satisfied, and if $R_0 < 1$, the disease-free equilibrium E_0 is globally asymptotically stable; otherwise, there at least exists a positive constant solution $i^* \in D - (0, 0, \ldots, 0)$. □

3.3 The uniqueness of endemic equilibrium

Theorem 3.4. *If $R_0 > 1$, there exists a unique endemic equilibrium $i^* = (i_1^*, i_2^*, \ldots, i_n^*)$ for system (4).*

Proof. By Theorem 3.3, there at least exists a endemic equilibrium $i^* = (i_1^*, i_2^*, \ldots, i_n^*)$ for system (4). Suppose $z^* = (z_1^*, z_2^*, \ldots, z_n^*)$ is another endemic equilibrium and $i^* \neq z^*$, then there exists $k_0 = 1, 2, \ldots, n$ such that $i_{k_0}^* \neq z_{k_0}^*$. Without loss of generality, we assume that $i_{k_0}^* > z_{k_0}^*$ and

$$\frac{i_{k_0}^*}{z_{k_0}^*} = \max_{1 \leq k \leq n}\left\{\frac{i_k^*}{z_k^*}\right\}.$$

Since i^* and z^* are two positive equilibrium points of system (4), we have

$$\left[1-(1-\beta_1\theta(i^*))^{k_0}\right]\left(1-i^*_{k_0}\right)+\beta_4\left(1-i^*_{k_0}\right)\sum_{k=1}^{n}P(k)i^*_k-\nu i^*_{k_0}=0 \tag{7}$$

and

$$\left[1-(1-\beta_1\theta(z^*))^{k_0}\right]\left(1-z^*_{k_0}\right)+\beta_4\left(1-z^*_{k_0}\right)\sum_{k=1}^{n}P(k)z^*_k-\nu z^*_{k_0}=0, \tag{8}$$

namely,

$$\begin{aligned}&\left[1-(1-\beta_1\theta(i^*))^{k_0}\right]\left(1-i^*_{k_0}\right)+\beta_4\left(1-i^*_{k_0}\right)\sum_{k=1}^{n}P(k)i^*_k\\&=\left[1-(1-\beta_1\theta(z^*))^{k_0}\right]\left(1-z^*_{k_0}\right)\frac{i^*_{k_0}}{z^*_{k_0}}+\beta_4\left(1-z^*_{k_0}\right)\sum_{k=1}^{n}P(k)z^*_k\frac{i^*_{k_0}}{z^*_{k_0}}.\end{aligned} \tag{9}$$

Two possibilities are considered.

Case (a): If $\theta(i^*)=\theta(z^*)$. Because $i^*_k\le\frac{i^*_{k_0}}{z^*_{k_0}}z^*_k$ and $1-i^*_{k_0}<1-z^*_{k_0}$, by equations (7) and (8), we have

$$\begin{aligned}&\left[1-(1-\beta_1\theta(i^*))^{k_0}\right]\left(1-i^*_{k_0}\right)+\beta_4\left(1-i^*_{k_0}\right)\sum_{k=1}^{n}P(k)i^*_k,\\&<\left[1-(1-\beta_1\theta(z^*))^{k_0}\right]\left(1-z^*_{k_0}\right)\frac{i^*_{k_0}}{z^*_{k_0}}+\beta_4\left(1-z^*_{k_0}\right)\sum_{k=1}^{n}P(k)z^*_k\frac{i^*_{k_0}}{z^*_{k_0}},\end{aligned} \tag{10}$$

which is in contradiction with (9).

Case (b): If $\theta(i^*)\neq\theta(z^*)$. Suppose $\theta(i^*)>\theta(z^*)$. We will prove

$$[1-(1-\beta_1\theta(z^*))^k]\frac{i^*_{k_0}}{z^*_{k_0}}>1-(1-\beta_1\theta(i^*))^k,\quad k=1,2,\ldots,n. \tag{11}$$

When $k=1$, equation (11) holds. Assume that

$$[1-(1-\beta_1\theta(z^*))^{k-1}]\frac{i^*_{k_0}}{z^*_{k_0}}>1-(1-\beta_1\theta(i^*))^{k-1} \tag{12}$$

holds. Then,

$$[1-(1-\beta_1\theta(z^*))^k]\frac{i^*_{k_0}}{z^*_{k_0}}=\{(1-\beta_1\theta(z^*))[1-(1-\beta_1\theta(z^*))^{k-1}]+\beta_1\theta(z^*)\}\frac{i^*_{k_0}}{z^*_{k_0}}$$

and

$$1-\beta_1\theta(z^*)>1-\beta_1\theta(i^*),\ \beta_1\theta(z^*)\frac{i^*_{k_0}}{z^*_{k_0}}>\beta_1\theta(i^*).$$

Hence, equation (11) holds.

Especially, let $k=k_0$, we can obtain

$$\left[1-(1-\beta_1\theta(z^*))^{k_0}\right]\frac{i^*_{k_0}}{z^*_{k_0}}>1-(1-\beta_1\theta(i^*))^{k_0}. \tag{13}$$

By equations (7) and (8), we can obtain

$$\begin{aligned}&\left[1-(1-\beta_1\theta(i^*))^{k_0}\right]\left(1-i^*_{k_0}\right)+\beta_4\left(1-i^*_{k_0}\right)\sum_{k=1}^{n}P(k)i^*_k\\&<\left[1-(1-\beta_1\theta(z^*))^{k_0}\right]\left(1-z^*_{k_0}\right)\frac{i^*_{k_0}}{z^*_{k_0}}+\beta_4\left(1-z^*_{k_0}\right)\sum_{k=1}^{n}P(k)z^*_k\frac{i^*_{k_0}}{z^*_{k_0}},\end{aligned} \tag{14}$$

which is in contradiction with equation (9).

Therefore, if $R_0>1$, there exists a unique endemic equilibrium $i^*=(i^*_1,i^*_2,\ldots,i^*_n)$ for system (4). □

3.4 The global attractiveness of endemic equilibrium

Theorem 3.5. *If $R_0 > 1$, the endemic equilibrium $i^* = (i_1^*, i_2^*, \ldots, i_n^*)$ is globally attractive.*

Proof. Define two functions

$$G : \Phi_n \to R, \quad g : \Phi_n \to R,$$

where

$$G(i) = \max_{1\le k\le n}\left\{\frac{i_k}{i_k^*}\right\}, \quad g(i) = \min_{1\le k\le n}\left\{\frac{i_k}{i_k^*}\right\},$$

$i = (i_1, i_2, \ldots, i_n)$ is a solution of system (4). Obviously, $G(i)$ and $g(i)$ are continuous, and the right-hand derivative exists along solutions of (4).

Define

$$G'(i) = \limsup_{h\to 0}\frac{G(i(t+h)) - G(i(t))}{h}.$$

Assume that

$$G(i(t)) = \frac{i_{k_0}(t)}{i_{k_0}^*},$$

then

$$i_{k_0}^*\frac{i_{k_0}'(t)}{i_{k_0}(t)} = \left[1 - (1 - \beta_1\theta(i(t)))^{k_0}\right]\left(1 - i_{k_0}(t)\right)\frac{i_{k_0}^*}{i_{k_0}(t)} + \beta_4\left(1 - i_{k_0}(t)\right)\sum_{k=1}^{n}P(k)i_k(t)\frac{i_{k_0}^*}{i_{k_0}(t)} - \nu i_{k_0}^*.$$

Suppose that $\theta(i(t)) > \theta(i^*)$, then $G(i(t)) > 1$.

Since

$$\begin{aligned} i_{k_0}^*\frac{i_{k_0}'(t)}{i_{k_0}(t)} &= \left[1 - (1 - \beta_1\theta(i(t)))^{k_0}\right]\left(1 - i_{k_0}(t)\right)\frac{i_{k_0}^*}{i_{k_0}(t)} + \beta_4\left(1 - i_{k_0}(t)\right)\sum_{k=1}^{n}P(k)i_k(t)\frac{i_{k_0}^*}{i_{k_0}(t)} - \nu i_{k_0}^* \\ &= \frac{i_{k_0}^*}{i_{k_0}(t)}[\beta_1\theta(i(t))]\left(1 - i_{k_0}(t)\right)\sum_{j=1}^{k_0-1}(1 - \beta_1\theta(i(t)))^j + \beta_4\left(1 - i_{k_0}(t)\right)\sum_{k=1}^{n}P(k)i_k(t)\frac{i_{k_0}^*}{i_{k_0}(t)} - \nu i_{k_0}^*, \end{aligned}$$

and

$$\frac{i_{k_0}^*}{i_{k_0}(t)}\beta_1\theta(i(t)) < \beta_1\theta(i^*), 1 - i_{k_0}(t) < 1 - i_{k_0}^*, 1 - \beta_1\theta(i(t)) < 1 - \beta_1\theta(i^*).$$

Then,

$$i_{k_0}^*\frac{i_{k_0}'(t)}{i_{k_0}(t)} < \left[1 - (1 - \beta_1\theta(i^*))^{k_0}\right]\left(1 - i_{k_0}^*\right) + \beta_4\left(1 - i_{k_0}^*\right)\sum_{k=1}^{n}P(k)i_k^* - \nu i_{k_0}^* = 0.$$

That is to say, $G'(i(t)) < 0$. Similarly, we can obtain $G'(i(t)) \le 0$, if $\theta(i(t)) = \theta(i^*)$.

Suppose $\theta(i(t)) < \theta(i^*)$. By the above method, we have $g'(i(t)) > 0$. If $\theta(i(t)) = \theta(i^*)$, then $g'(i(t)) \ge 0$.

Define two functions

$$U(i) = \max\{G(i) - 1, 0\}, \quad u(i) = \max\{1 - g(i), 0\}.$$

Then, for any $i \in \Phi_n$, functions $U(i)$ and $u(i)$ are non-negative and continuous and

$$U'(i(t)) \le 0, \quad u'(i(t)) \le 0.$$

Denote

$$V_U = \{i \in \Phi_n | U'(i) = 0\}, \quad V_u = \{i \in \Phi_n | u'(i) = 0\}.$$

Then,

$$V_U = \{i \in \Phi_n | 0 \le i_k \le i_k^*\}, \quad V_u = \{i \in \Phi_n | i_k^* \le i_k \le 1\} \bigcup \{(0, 0, \ldots, 0)\}.$$

Therefore, $V_U \bigcap V_u = \{i^* = (i_1^*, i_2^*, \ldots, i_n^*)\} \bigcup \{(0, 0, \ldots, 0)\}$.

According to the LaSalle invariant set principle, any solution of system (4), starting in Φ_n, will tend to Φ_n. By Lemma 3.1, if $i(t) \neq 0$, then $\liminf_{t\to\infty}\|i(t)\| \ge m > 0$. For arbitrary $i_0 \in D - (0, 0, \ldots, 0)$, we obtain $\lim_{t\to\infty} i(t) = i^* = (i_1^*, i_2^*, \ldots, i_n^*)$, where $i(t) = (i_1(t), i_2(t), \ldots, i_n(t))$ is a solution of system (4). Hence, if $R_0 > 1$, the endemic equilibrium $i^* = (i_1^*, i_2^*, \ldots, i_n^*)$ is globally attractive. □

4 Control strategies

In this section, we will propose three control strategies to prevent the spread of the disease.

4.1 Uniform immunization strategy

A uniform immunization strategy is the simplest immunization strategy. Let p be immunization rate in the network. Then, system (4) becomes

$$\frac{\mathrm{d}i_k(t)}{\mathrm{d}t} = [1 - (1 - \beta_1\theta(i(t)))^k](1 - p)(1 - i_k(t)) + \beta_4(1 - p)(1 - i_k(t))i(t) - \nu i_k(t).$$

Using the same method as in Section 3.1, we can calculate the basic reproductive number

$$R_0^U = \frac{1}{2}\left(\frac{(1-p)\beta_1\langle k^2\rangle}{\nu\langle k\rangle} + \frac{(1-p)\beta_4}{\nu}\right) + \frac{1}{2}\sqrt{\Delta_1},$$

where $\Delta_1 = \left(\frac{(1-p)\beta_1\langle k^2\rangle}{\nu\langle k\rangle} - \frac{(1-p)\beta_4}{\nu}\right)^2 + \frac{4(1-p)^2\beta_1\beta_4\langle k\rangle}{\nu^2}$.

4.2 Drug treatment strategy

The drug treatment strategy is to control the spread of disease by increasing the cure rate. Assume that the improved cure rate from drug treatment is $r \in (0, 1 - \nu)$. Then, system (4) becomes

$$\frac{\mathrm{d}i_k(t)}{\mathrm{d}t} = [1 - (1 - \beta_1\theta(i(t)))^k](1 - i_k(t)) + \beta_4(1 - i_k(t))i(t) - (\nu + r)i_k(t).$$

Using the same method in Section 3.1, we can calculate the basic reproductive number

$$R_0^M = \frac{1}{2}\left(\frac{\beta_1\langle k^2\rangle}{(\nu + r)\langle k\rangle} + \frac{\beta_4}{\nu + r}\right) + \frac{1}{2}\sqrt{\Delta_2},$$

where $\Delta_2 = \left(\frac{\beta_1\langle k^2\rangle}{(\nu+r)\langle k\rangle} - \frac{\beta_4}{\nu+r}\right)^2 + \frac{4\beta_1\beta_4\langle k\rangle}{(\nu+r)^2}$.

4.3 Targeted immunization strategy

A targeted immunization strategy is to immunize the nodes with high degree. We give lower and upper thresholds k_1 and k_2 and define the immunization rate η_k as follows:

$$\eta_k = \begin{cases} 1, & k > k_2, \\ \rho_k, & k_1 < k < k_2, \\ 0, & k < k_1. \end{cases}$$

Then, system (4) becomes

$$\frac{\mathrm{d}i_k(t)}{\mathrm{d}t} = [1 - (1 - \beta_1\theta(i(t)))^k](1 - \eta_k)(1 - i_k(t)) + \beta_4(1 - \eta_k)(1 - i_k(t))i(t) - \nu i_k(t).$$

Using the same method in Section 3.1, we can calculate the basic reproductive number

$$R_0^T = \frac{1}{2}\left(\frac{\beta_1\langle k^2(1-\eta_k)\rangle}{\nu\langle k\rangle} + \frac{\beta_4\langle(1-\eta_k)\rangle}{\nu}\right) + \frac{1}{2}\sqrt{\Delta_3},$$

where $\Delta_3 = \left(\frac{\beta_1\langle k^2(1-\eta_k)\rangle}{\nu\langle k\rangle} - \frac{\beta_4\langle(1-\eta_k)\rangle}{\nu}\right)^2 + \frac{4\beta_1\beta_4\langle k(1-\eta_k)^2\rangle}{\nu^2}$.

5 Numerical simulations

In this section, we give two examples to support the theoretical prediction. Assume that the degree distribution is $P(k) = mk^{-3}$, $k = 1, 2, \ldots, 100$. The value of parameter m satisfies the equation $\sum_{k=1}^{100} mk^{-3} = 1$.

Example 5.1. Choose $\beta_1 = 0.08$, $\beta_4 = 0.07$, and $\nu = 0.5$. Then, $R_0 \approx 0.058 < 1$. Let the initial value $i_{20} = 1 - 0.2h$, $i_{40} = 1 - 0.2h$, $i_{60} = 1 - 0.2h$, and $i_{80} = 1 - 0.2h$, $h = 1, 2, 3, 4, 5$. By Theorem 3.3, the disease-free equilibrium E_0 is globally asymptotically stable (Figure 1).

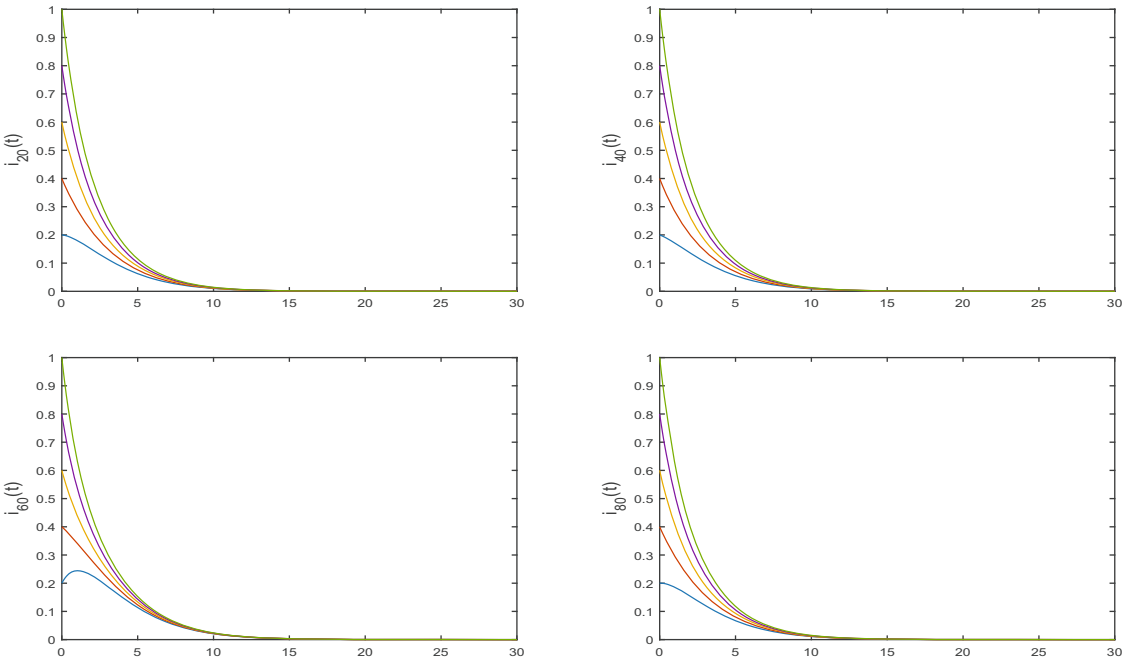

Figure 1: The disease-free equilibrium is globally asymptotically stable.

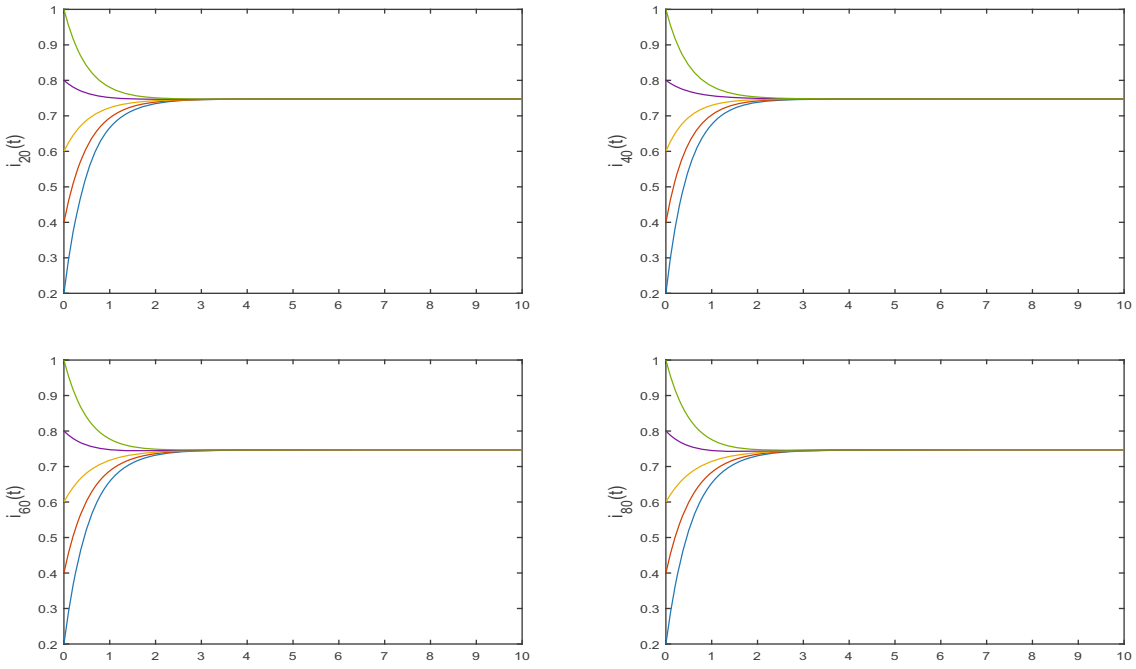

Figure 2: The endemic equilibrium is unique and globally attractive.

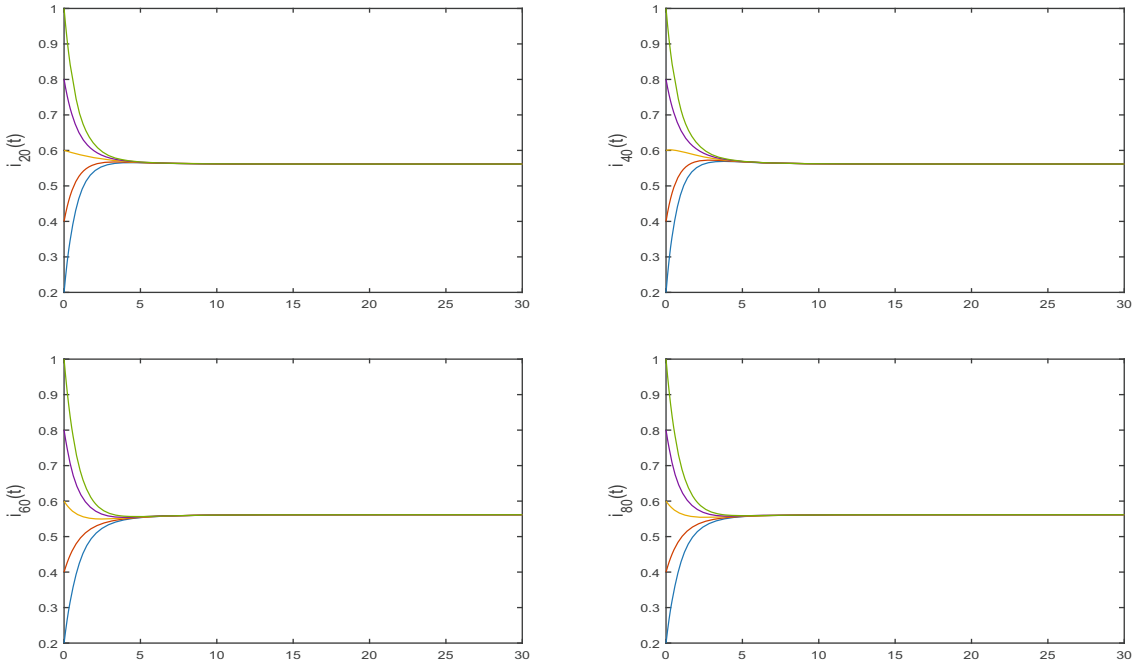

Figure 3: The density of infected nodes under uniform immunization strategy.

Example 5.2. Choose $\beta_1 = 0.8$, $\beta_4 = 0.7$, and $\nu = 0.5$. Then, $R_0 \approx 5.773 > 1$. Let the initial value $i_{20} = 1 - 0.2h$, $i_{40} = 1 - 0.2h$, $i_{60} = 1 - 0.2h$, and $i_{80} = 1 - 0.2h$, $h = 1, 2, 3, 4$. By Theorem 3.5, the endemic equilibrium $i^* = (i_1^*, i_2^*, \ldots, i_n^*)$ is globally attractive (Figure 2).

We consider three control strategies. In Example 5.2, let $p = 0.5$, the numerical simulation results are given in Figure 3. Let $r = 0.5$, then the numerical simulation results are given in Figure 4. Let $\eta_k = \eta = 0.6$,

the numerical simulation results are given in Figure 5. When $R_0 > 1$, the endemic equilibrium $i^* = (i_1^*, i_2^*, \ldots, i_n^*)$ is globally attractive. At this very moment, the numerical simulation results show that the density of infected nodes with a control strategy is smaller than that without a control strategy. In this sense, the control strategy is effective. Especially, using a uniform immunization strategy and a drug treatment strategy at the same time, the numerical simulation results are given in Figure 6, which demonstrate that the density of infected nodes decreases significantly and indicates that the combination of the two strategies is more effective.

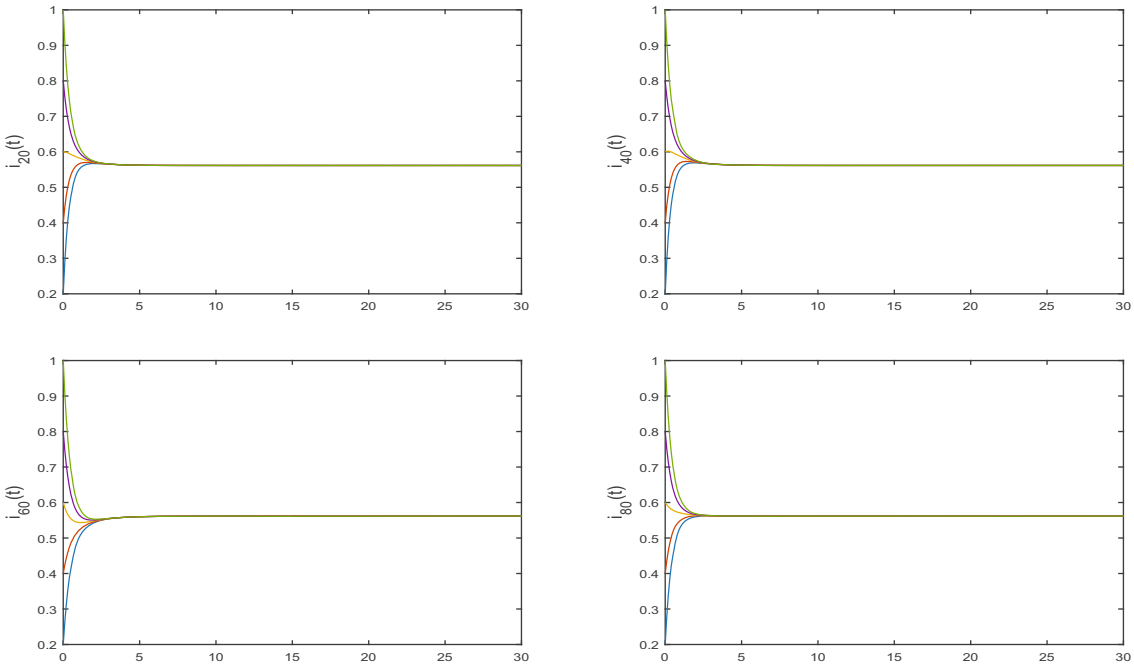

Figure 4: The density of infected nodes under drug treatment strategy.

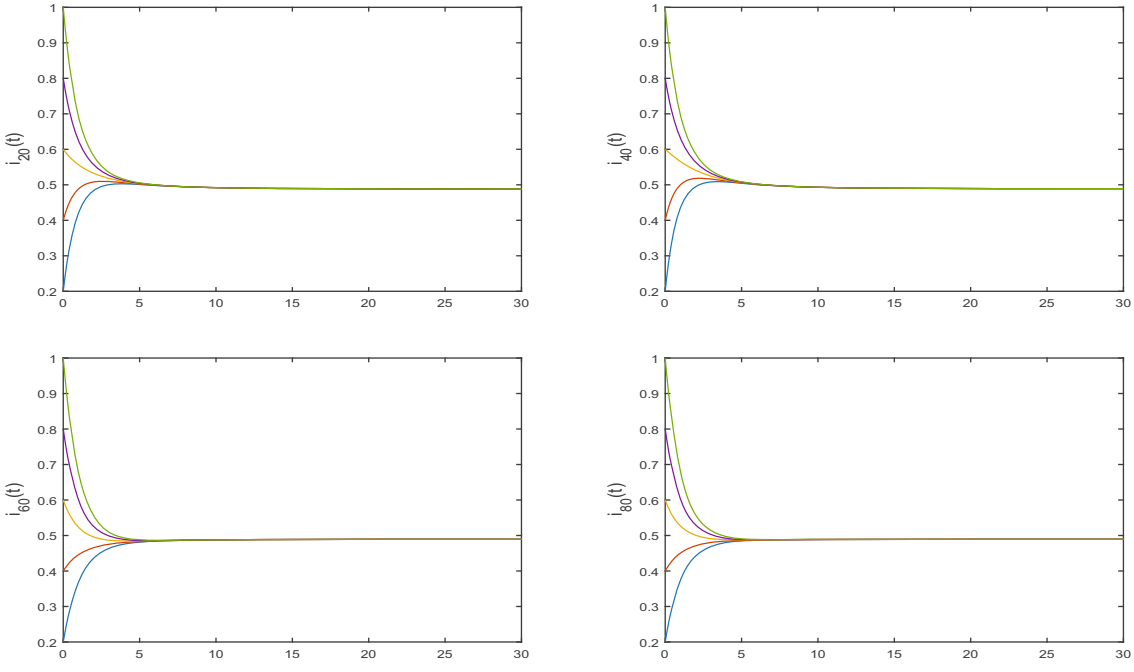

Figure 5: The density of infected nodes under targeted immunization strategy.

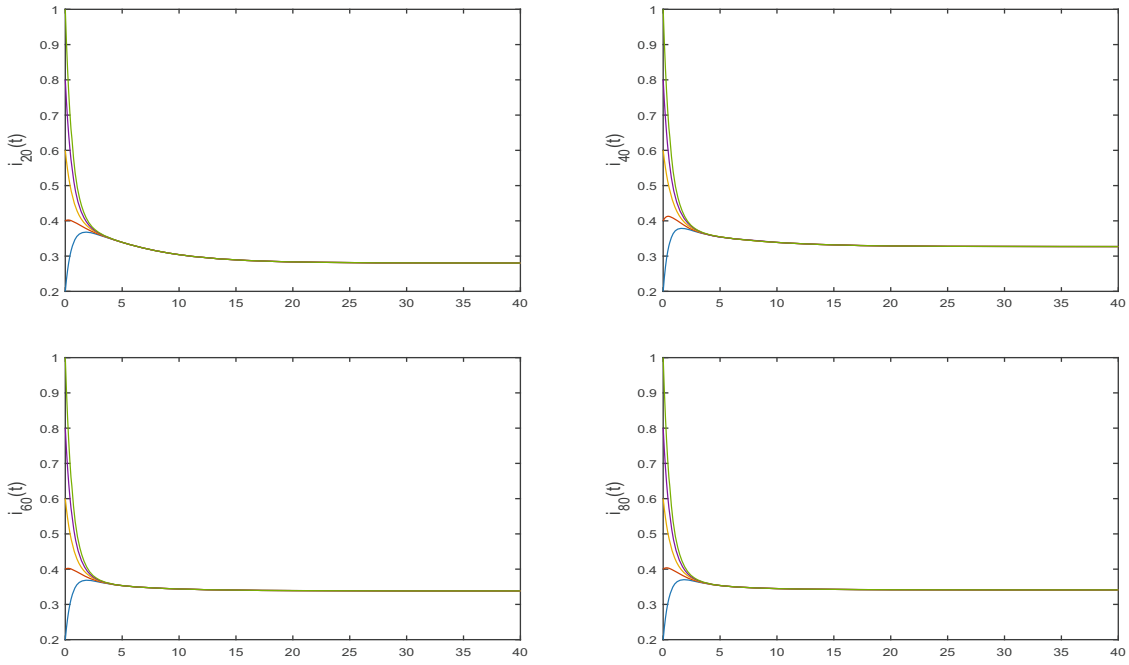

Figure 6: The density of infected nodes under uniform immunization strategy and drug treatment strategy.

6 Conclusion

In this study, a vector-borne epidemic model with multi-edge infection on complex networks is proposed. Using the next generation matrix, the basic reproduction number is obtained, which is an important threshold. If $R_0 < 1$, the disease-free equilibrium is globally asymptotically stable. If $R_0 > 1$, system (4) has a unique positive equilibrium, which is globally attractive. Moreover, three control strategies are studied to control the spread of epidemic disease.

It is worth noting that vector-borne diseases may be affected by environmental changes, which is a very interesting topic. A vector-borne disease with periodic infection rate on complex networks may be considered in the future.

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References

[1] K. L. Cooke, *Stability analysis for a vector disease model*, Rocky Mountain J. Math. **9** (1979), no. 1, 31–42, DOI: https://doi.org/10.1216/RMJ-1979-9-1-31.

[2] P. Marcati and A. M. Pozio, *Global asymptotic stability for a vector disease model with spatial spread*, J. Math. Biol. **9** (1980), no. 2, 179–187, DOI: https://doi.org/10.1007/BF00275920.

[3] R. Volz, *Global asymptotic stability of a periodic solution to an epidemic model*, J. Math. Biol. **15** (1982), no. 3, 319–338, DOI: https://doi.org/10.1007/BF00275691.

[4] E. Beretta and Y. Takeuchi, *Convergence results in SIR epidemic model with varying population size*, Nonlinear Anal. **28** (1997), no. 12, 1909–1921, DOI: https://doi.org/10.1016/S0362-546X(96)00035-1.
[5] Y. Takeuchi, W. Ma, and E. Beretta, *Global asymptotic properties of a delay SIR epidemic model with finite incubation times*, Nonlinear Anal. **42** (2000), no. 6, 931–947, DOI: https://doi.org/10.1016/S0362-546X(99)00138-8.
[6] E. Beretta, T. Hara, W. Ma, and Y. Takeuchi, *Global asymptotic stability of an SIR epidemic model with distributed time delay*, Nonlinear Anal. **47** (2001), no. 6, 4107–4115, DOI: https://doi.org/10.1016/S0362-546X(01)00528-4.
[7] S. Busenberg and K. L. Cooke, *Periodic solutions of a periodic nonlinear delay differential equation*, SIAM J. Appl. Math. **35** (1978), no. 4, 704–721, DOI: https://doi.org/10.1137/0135059.
[8] H. Cao, D. Yan, S. Zhang, and X. Wang, *Analysis of dynamics of recurrent epidemics: periodic or non-periodic*, Bull. Math. Biol. **81** (2019), no. 12, 4889–4907, DOI: https://doi.org/10.1007/s11538-019-00638-5.
[9] P. Liu and H. X. Li, *Global behavior of a multi-group SEIR epidemic model with age structure and spatial diffusion*, Math. Biosci. Eng. **17** (2020), no. 6, 7248–7273, DOI: https://doi.org/10.3934/mbe.2020372.
[10] W. Guo, Q. Zhang, X. Li, and W. Wang, *Dynamic behavior of a stochastic SIRS epidemic model with media coverage*, Math. Methods Appl. Sci. **41** (2018), 5506–5525, DOI: https://doi.org/10.1002/mma.5094.
[11] S. Anita, M. Banerjee, S. Ghosh, and V. Volpert, *Vaccination in a two-group epidemic model*, Appl. Math. Lett. **119** (2021), 107197, DOI: https://doi.org/10.1016/j.aml.2021.107197.
[12] G. Zhu, X. Fu and G. Chen, *Spreading dynamics and global stability of a generalized epidemic model on complex heterogeneous networks*, Appl. Math. Model. **36** (2012), no. 12, 5808–5817, DOI: https://doi.org/10.1016/j.apm.2012.01.023.
[13] R. Pastor-Satorras and A. Vespignani, *Epidemic spreading in scale-free networks*, Phys. Rev. Lett. **86** (2001), no. 14, 3200, DOI: https://doi.org/10.1103/PhysRevLett.86.3200.
[14] R. Pastor-Satorras and A. Vespignani, *Epidemic dynamics and endemic states in complex networks*, Phys. Rev. E **63** (2001), no. 6, 066117, DOI: https://doi.org/10.1103/PhysRevE.63.066117.
[15] J. Yang, L. Wang, X. Li, and F. Zhang, *Global dynamical analysis of a heroin epidemic model on complex networks*, J. Appl. Anal. Comput. **6** (2016), no. 2, 429–442, DOI: https://doi.org/10.11948/2016032.
[16] X. Wei, G. Xu, L. Liu, and W. Zhou, *Global stability of endemic equilibrium of an epidemic model with birth and death on complex networks*, Phys. A **477** (2017), 78–84, DOI: https://doi.org/10.1016/j.physa.2017.02.050.
[17] X. Wang, Z. Wang, and H. Shen, *Dynamical analysis of a discrete-time SIS epidemic model on complex networks*, Appl. Math. Lett. **94** (2019), 292–299, DOI: https://doi.org/10.1016/j.aml.2019.03.011.
[18] W. Lv, Q. Ke, and K. Li, *Dynamic stability of an SIVS epidemic model with imperfect vaccination on scale-free networks and its control strategy*, J. Franklin Inst. **357** (2020), no. 11, 7092–7121, DOI: https://doi.org/10.1016/j.jfranklin.2020.05.029.
[19] Y. Xie, Z. Wang, J. Lu, and Y. Li, *Stability analysis and control strategies for a new SIS epidemic model in heterogeneous networks*, Appl. Math. Comput. **384** (2020), 125381, DOI: https://doi.org/10.1016/j.amc.2020.125381.
[20] K. Li, G. Zhu, Z. Ma, and L. Chen, *Dynamic stability of an SIQS epidemic network and its optimal control*, Commun. Nonlinear Sci. Numer. Simul. **66** (2019), 84–95, DOI: https://doi.org/10.1016/j.cnsns.2018.06.020.
[21] M. Xueyu, C. Zhiqiang, S. Shubin, and D. Dongli, *Analysis of epidemic vaccination strategies on heterogeneous networks: Based on SEIRV model and evolutionary game*, Appl. Math. Comput. **403** (2021), 126172, DOI: https://doi.org/10.1016/j.amc.2021.126172.
[22] C. P. Alota, C. P. C. Pilar-Arceo, and A. A. de los Reyes V, *An edge-based model of SEIR epidemics on static random networks*, Bull. Math. Biol. **82** (2020), 96, DOI: https://doi.org/10.1007/s11538-020-00769-0.
[23] Y. Wang, J. Cao, X. Li, and A. Alseadi, *Edge-based epidemic dynamics with multiple routes of transmission on random networks*, Nonlinear Dyn. **91** (2018), no. 1, 403–420, DOI: https://doi.org/10.1007/s11071-017-3877-3.
[24] X. Wei, X. Wu, S. Chen, J-an. Lu, and G. Chen, *Cooperative epidemic spreading on a two-layered interconnected network*, SIAM J. Appl. Dyn. Syst. **17** (2018), no. 2, 1503–1520, DOI: https://doi.org/10.1137/17M1134202.
[25] H. Guo, Z. Wang, S. Sun, and C. Xia, *Interplay between epidemic spread and information diffusion on two-layered networks with partial mapping*, Phys. Lett. A **398** (2021), 127282, DOI: https://doi.org/10.1016/j.physleta.2021.127282.
[26] X. Wang, Z. Wang, J. Lu, and B. Meng, *Stability, bifurcation and chaos of a discrete-time pair approximation epidemic model on adaptive networks*, Math. Comput. Simulation **182** (2021), 182–194, DOI: https://doi.org/10.1016/j.matcom.2020.10.019.
[27] Á. Bodó and P. L. Simon, *Analytic study of bifurcations of the pairwise model for SIS epidemic propagation on an adaptive network*, Differ. Equ. Dyn. Syst. **28** (2020), no. 4, 807–826, DOI: https://doi.org/10.1007/s12591-017-0348-8.
[28] Y. Wang, Z. Jin, Z. Yang, Z.-K. Zhang, T. Zhou, and G.-Q. Sun, *Global analysis of an SIS model with an infective vector on complex networks*, Nonlinear Anal. Real World Appl. **13** (2012), no. 2, 543–557, DOI: https://doi.org/10.1016/j.nonrwa.2011.07.033.
[29] L. Zhu, G. Guan, and Y. Li, *Nonlinear dynamical analysis and control strategies of a network-based SIS epidemic model with time delay*, Appl. Math. Model. **70** (2019), 512–531, DOI: https://doi.org/10.1016/j.apm.2019.01.037.
[30] H. Yang, H. Wei, and X. Li, *Global stability of an epidemic model for vector-borne disease*, J. Syst. Sci. Complex. **23** (2010), no. 2, 279–292, DOI: https://doi.org/10.1007/s11424-010-8436-7.
[31] G. R. Hosack, P. A. Rossignol, and P. van den Driessche, *The control of vector-borne disease epidemics*, J. Theoret. Biol. **255** (2008), no. 1, 16–25, DOI: https://doi.org/10.1016/j.jtbi.2008.07.033.

[32] X. Wang, Y. Chen, M. Martcheva, and L. Rong, *Asymptotic analysis of a vector-borne disease model with the age of infection*, J. Biol. Dyn. **14** (2020), no. 1, 332–367, DOI: https://doi.org/10.1080/17513758.2020.1745912.
[33] Y. X. Dang, Z. P. Qiu, X. Z. Li, and M. Martcheva, *Global dynamics of a vector-host epidemic model with age of infection*, Math. Biosci. Eng. **14** (2017), no. 5–6, 1159–1186, DOI: https://doi.org/10.3934/mbe.2017060.
[34] X. Wang, Y. Chen, and S. Liu, *Global dynamics of a vector-borne disease model with infection ages and general incidence rates*, Comput. Appl. Math. **37** (2018), 4055–4080, DOI: https://doi.org/10.1007/s40314-017-0560-8.
[35] H. Kang and X. Fu, *Epidemic spreading and global stability of an SIS model with an infective vector on complex networks*, Commun. Nonlinear Sci. Numer. Simul. **27** (2015), no. 1–3, 30–39, DOI: https://doi.org/10.1016/j.cnsns.2015.02.018.
[36] P. van den Driessche and J. Watmough, *Reproduction numbers and sub-threshold endemic equilibria for compartmental models of disease transmission*, Math. Biosci. **180** (2002), no. 1–2, 29–48, DOI: https://doi.org/10.1016/S0025-5564(02)00108-6.
[37] Y. Xie, Z. Wang, J. Lu, and Y. Li, *Stability analysis and control strategies for a new SIS epidemic model in heterogeneous networks*, Appl. Math. Comput. **383** (2020), 125381, DOI: https://doi.org/10.1016/j.amc.2020.125381.
[38] A. Lajmanovich and J. A. Yorke, *A deterministic model for gonorrhea in a nonhomogeneous population*, Math. Biosci. **28** (1976), no. 3–4, 221–236, DOI: https://doi.org/10.1016/0025-5564(76)90125-5.
[39] J. A. Yorke, *Invariance for ordinary differential equations*, Math. Syst. Theory **1** (1967), no. 8, 353–372, DOI: https://doi.org/10.1007/BF01695169.
[40] R. Varga, *Iterative Analysis*, Prentice-Hall, Englewood, New Jersey, 1962.