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Research Article

Dynamics Modeling and Analysis of SIS Epidemic Spreading in Cluster Networks

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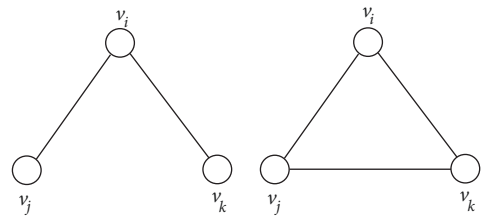
In this paper, we propose and study an SIS epidemic model with clustering characteristics based on networks. Using the method of the existence of positive equilibrium point, we obtain the formula of the basic reproduction number R_0. Furthermore, by constructing Lyapunov function, we also prove that the disease-free equilibrium of the model is globally asymptotically stable when $R_0 < 1$. When $R_0 > 1$, there is only one positive equilibrium point which is globally asymptotically stable. It is also shown that the infection proportion and the basic reproduction number R_0 increases as the clustering coefficient increases when the average degree of networks is fixed.

1. Introduction

A large number of complex systems in nature and human society can be described by complex networks. At present, the structure of complex networks and mathematical models based on networks in the field of biology and other fields has been deeply studied [1–3]. In the biological field, the spread of infectious diseases can affect people's physical and mental health. The spread of infectious diseases is not only related to the transmission mechanisms of diseases, but also related to the topology of the complex network. A lot of researches have shown that two topologies of the network can profoundly affect the dynamics of infectious diseases: one is the degree distribution (the number distribution of contact neighbors per individual) [4–6]; the other is contact clusters (such as households or school) [7, 8]. The effect of degree distribution on the spread of diseases in networks has been widely discussed and fully understood [9, 10]. The results of these studies clearly show that the basic reproduction number of diseases tends to infinity when the variance of the degree distribution tends to infinity in scale-free networks. This means that the disease can easily spread in scale-free networks regardless of how fast the epidemic spreads [9].

Clustering in a complex network means that two neighboring nodes of a given node also have a tendency to become neighbors, so a triangle is formed in the network. In the contact network, these triangles called clusters mean that two friends of one person are also friends with each other. The clustering coefficient is an indicator for measuring the level of clustering in the network. The average value of the clustering coefficients of all nodes in the network is called the clustering coefficient of the network [11]. Generally, when the degree distribution is fixed, the number of network clusters (namely, the number of triangles) can significantly increase as the clustering coefficient of the network increases. Network clusters not only affect the network structure, but also affect the dynamics of the disease transmission on the network.

In the past decade, some researchers have used different methods to study the impact of clustering on the spread of epidemics in weak clustering networks. Eames [12] studied the spread of epidemics in random networks when the number of triangles is given. The results show that sufficient clustering can increase the epidemic threshold. However, at the small and moderate levels, clustering appears not to change the final size of epidemics significantly. Miller found that clusters reduced the basic reproduction number and the final scale of the epidemic in weak clustering networks [13, 14]. Trapman constructed a random graph when degree distribution and the expected number of triangles were given [15] and studied the effects of the degree distribution and



(a) Open triple (b) Closed triple

Figure 1: Open triple and closed triple.

the expected number of triangles on disease transmissions in the network. The results show that clusters reduce the basic reproduction number and the final size of the epidemic. Newman found that, when the average degree of weak clustering network is fixed, the basic reproduction number of diseases increases and the final scale of epidemics decreases as the clustering coefficient increases [11]. In 2011, Volz et al. took advantage of the method of dynamical probability generating function based nodes in weak clustering networks to find that clustering always slows the spread of the epidemic, but simultaneously increasing clustering and the variance of the degree distribution can increase the final infection scale [8]. Li proposed a new SIS model that includes network clusters. It is pointed out that, due to the heterogeneity of infection, clusters always promote the spread of diseases in the network [16].

In this paper, we establish an SIS dynamical model on a class of clustered networks to further study how degree distribution and clustering influence the spread of disease. In Section 2, considering the infectivity heterogeneity of infective nodes located at different sites (at the end of single edge or the edge in the triangle), we derive an SIS dynamical model based on the mean-field method describing the transmission of diseases in networks with arbitrary degree distributions and clustering coefficients. In Section 3, we calculate the reproduction number R_0 of diseases and prove the local and global stability of disease-free equilibrium and the endemic equilibrium. In Section 4, the impacts of degree distributions and clusters on disease in the network are analyzed by simulations. The results show that the reproduction number and the relation size of infection individual always increase as the clustering coefficient increases.

2. Dynamic Modeling

We consider a class of weakly clustered network where there are no common edges between any two triangles. We assume that each node has some lines (or single edges) and triangles in the network. For convenience, we assume the numbers of lines and triangles of every node are independent. In the current clustered network, we consider that each individual exists only in two discrete states: S-susceptible and I-infected. At each time step, each susceptible (healthy) node is infected if it is contacted by one infected individual; at the same time, infected nodes are cured and become again susceptible with rate γ. Without lack of generality, we can set $\gamma = 1$. It is worth noting that each susceptible node is infected by its infected neighbors which are connected by the line or edge in triangles. If a susceptible node is not infected, then it must be not infected by any of all infected neighbors to which it is connected, neither by lines nor by triangles. Let β_1 be the infection probability that a susceptible node is infected by the random edge in triangles and β_2 be the infection probability that a susceptible node is infected by a random infected neighbor connected by a line.

Let N represents the total number of nodes in the network. $S_k(t)$ and $I_k(t)$ $(k = 1, 2, \ldots n)$, respectively, represent the number of susceptible and infected persons with degree k at time t. N_k represents the total number of nodes with degree k and is a constant in the static network. It is obvious that the degree distribution p_k is given by $p_k = N_k/N$. Then, the average degree $\langle k \rangle$ is given by $\langle k \rangle = \sum_{k=1}^{n} k p_k$. Let $s_k(t) = S_k(t)/N_k$ and $\rho_k(t) = I_k(t)/N_k$. Obviously, $s_k(t) + \rho_k(t) = 1$. For three different nodes $v_i - v_j - v_k$ in the network, they are called an open triple, where v_i is connected with v_j and v_k (see Figure 1(a)). If nodes v_j and v_k are also connected by an edge, the triple is called a closed triple or triangle (see Figure 1(b)).

We denote $N_{\triangle}^k$ as the number of triangles around a node of degree k and N_3^k as the number of connected all triples (open triple and triangle) around a node of degree k. The clustering coefficient C_k of nodes with degree k is defined by $C_k = N_{\triangle}^k / N_3^k = 2N_{\triangle}^k / k(k-1)$ [17]. Then, the number of edges connected with the nodes of degree k to the nodes in the triangle is $k(k-1)C_k$. The number of lines around a node with degree k is $k - k(k-1)C_k$. In degree uncorrelated networks, C_k is independent of k and so local clustering coefficient (C_k) is equal with global clustering coefficient (C) [17, 18]. Obviously,

$$\theta_1 = \frac{1}{\langle k \rangle} \sum_{k=1}^{n} Ck\,(k-1)\, p_k \rho_k,$$
$$\theta_2 = \frac{1}{\langle k \rangle} \sum_{k=1}^{n} [k - Ck\,(k-1)]\, p_k \rho_k \tag{1}$$

where θ_1 is the probability that any given edge points to an infected node in the triangles and θ_2 is the probability that any given link points to an infected node.

In degree uncorrelated networks, the dynamical mean-field (MF) reaction rate equations of disease transmission are established:

$$\frac{ds_k(t)}{dt} = -\beta_1 k s_k(t)\,\theta_1(t) - \beta_2 k s_k(t)\,\theta_2(t) + \rho_k(t),$$
$$\frac{d\rho_k(t)}{dt} = \beta_1 k s_k(t)\,\theta_1(t) + \beta_2 k s_k(t)\,\theta_2(t) - \rho_k(t). \tag{2}$$

Due to $s_k + \rho_k = 1$, then we can rewrite system (2) as

$$\frac{d\rho_k(t)}{dt} = k\left(1-\rho_k(t)\right)\left[\beta_1\theta_1(t) + \beta_2\theta_2(t)\right] - \rho_k(t). \quad (3)$$

Obviously, in real life, the probability that a susceptible node is infected by an infected node in the family is greater than the probability that a susceptible node is infected by an infected stranger. So we assume that $\beta_1 > \beta_2$ and $\beta_2 = l\beta_1$ $(0 < l < 1)$. Let $\widetilde{\theta}(t) = \sum_{k=1}^{n} \delta_k p_k \rho_k(t)/\langle k\rangle$, where $\delta_k = C(1-l)\beta_1 k(k-1) + l\beta_1 k$. Then, system (3) can be rewritten as

$$\dot{\rho}_k(t) = k\left(1-\rho_k(t)\right)\widetilde{\theta} - \rho_k(t). \quad (4)$$

3. Analysis of the Feasible Region

Lemma 1. *Suppose that the initial relative infected densities* $0 \le \rho_k(0) \le 1$ *satisfy* $\sum_{i=1}^{n} \delta_i p(i)\rho_i(0) > 0$. *Then, as* $t > 0$, *the solution* $\rho_k(t)$ *of system (4) satisfies* $0 < \rho_k(t) < 1, 0 < \widetilde{\theta}(t) < 1$.

Proof. According to system (4), $\widetilde{\theta}(t)$ satisfies the following equation:

$$\frac{d\widetilde{\theta}(t)}{dt} = -\widetilde{\theta}(t) + \frac{1}{\langle k\rangle}\sum_{k=1}^{n} k\delta_k p(k)\left(1-\rho_k(t)\right)\widetilde{\theta}(t). \quad (5)$$

For equation (5) from $0 \longrightarrow t$ integral, we get

$$\int_0^t \frac{d\widetilde{\theta}(s)}{\widetilde{\theta}(s)} = \int_0^t \left[-1 + \frac{1}{\langle k\rangle}\sum_{k=1}^{n} k\delta_k p(k)\left(1-\rho_k(s)\right)\right] ds. \quad (6)$$

Since $\widetilde{\theta}(0) = (1/\langle k\rangle)\sum_{k=1}^{n} \delta_k p(k)\rho_k(0) > 0$, we have

$$\widetilde{\theta}(t) = \widetilde{\theta}(0)\, e^{\{-t + \int_0^t (1/\langle k\rangle)\sum_{k=1}^{n} k\delta_k p(k)(1-\rho_k(s))ds\}} > 0, \quad (7)$$

where $t > 0$.

System (4) can be rewritten as

$$\frac{d\rho_k(t)}{dt} = -\left[1 + k\widetilde{\theta}(t)\right]\rho_k(t) + k\widetilde{\theta}(t). \quad (8)$$

Since $\widetilde{\theta}(t) > 0$, we have

$$\frac{d\rho_k(t)}{dt} + \left[1 + k\widetilde{\theta}(t)\right]\rho_k(t) > 0. \quad (9)$$

For the above inequality from $0 \longrightarrow t$ integral, we get

$$\rho_k(t) > \rho_k(0)\, e^{[-t-k\int_0^t \widetilde{\theta}(s)ds]} \ge 0, \quad t > 0. \quad (10)$$

On the other hand, it can be verified that the function $1-\rho_k(t)$ satisfies the equation

$$\begin{aligned}\frac{d\left[1-\rho_k(t)\right]}{dt} &= -\left[1 + k\widetilde{\theta}(t)\right]\left[1-\rho_k(t)\right] + 1 \\ &> -\left[1 + k\widetilde{\theta}(t)\right]\left[1-\rho_k(t)\right].\end{aligned} \quad (11)$$

For equation (11) from $0 \longrightarrow t$ integral, we get

$$1-\rho_k(t) > \left[1-\rho_k(0)\right] e^{[-t-k\int_0^t \widetilde{\theta}(s)ds]} > 0, \quad t > 0. \quad (12)$$

Thus, as $t > 0$, it follows that $0 < \rho_k(t) < 1$. Further, $0 < \widetilde{\theta} = \sum_{k=1}^{n} \delta_k p(k)\rho_k(t)/\langle k\rangle \le \sum_{k=1}^{n} \beta_1 k p(k)\rho_k(t)/\langle k\rangle = \beta_1 \sum_{k=1}^{n} k p_k \rho_k(t)/\langle k\rangle \le \beta_1 < 1$. □

4. Basic Reproduction Number

The equilibrium point of system (4) satisfies

$$\rho_k = \frac{k\widetilde{\theta}}{1 + k\widetilde{\theta}}. \quad (13)$$

Multiplying both sides of formula (13) by $\delta_k p_k/\langle k\rangle$ and summing of k, we obtain the following self-consistent equation about $\widetilde{\theta}$:

$$\widetilde{\theta} = \frac{1}{\langle k\rangle}\sum_{k=1}^{n} \delta_k p_k \frac{k\widetilde{\theta}}{1 + k\widetilde{\theta}} \triangleq f\left(\widetilde{\theta}\right). \quad (14)$$

Obviously, equation (14) has a zero solution $\widetilde{\theta} = 0$. System (4) has a disease-free equilibrium point $E_0 = (0, 0, \ldots, 0)$. Next, we discuss the existence of a unique positive solution. The first-order derivative of $f(\widetilde{\theta})$ is

$$\frac{df\left(\widetilde{\theta}\right)}{d\widetilde{\theta}} = \frac{1}{\langle k\rangle}\sum_{k=1}^{n} \frac{k\delta_k p_k}{\left(1 + k\widetilde{\theta}\right)^2}. \quad (15)$$

It is obviously that $df(\widetilde{\theta})/d\widetilde{\theta} > 0$, $f(\widetilde{\theta})$ is monotonically increasing with the increase of $\widetilde{\theta}$. The second derivative of $f(\widetilde{\theta})$ is

$$\frac{d^2 f\left(\widetilde{\theta}\right)}{d\widetilde{\theta^2}} = \frac{-2}{\langle k\rangle}\sum_{k=1}^{n} \frac{k^2\delta_k p_k}{\left(1 + k\widetilde{\theta}\right)^3}. \quad (16)$$

Because $d^2 f(\widetilde{\theta})/d\widetilde{\theta^2} < 0$, $f(\widetilde{\theta})$ is convex function. Note that $f(0) = 0$ and $f(1) < 1$. Thus, the sufficient and necessary condition which equation (14) has is a unique positive solution; namely, system (4) has a unique endemic disease equilibrium point $E^* = (\rho_1^*, \rho_2^*, \ldots, \rho_n^*)$; that is,

$$\left.\frac{df\left(\widetilde{\theta}\right)}{d\widetilde{\theta}}\right|_{\widetilde{\theta}=0} > 1. \quad (17)$$

Thus, we can get the basic reproduction number of system (4) from equation (17):

$$R_0 = \frac{C(1-l)\beta_1\left(\left\langle k^3\right\rangle - \left\langle k^2\right\rangle\right) + l\beta_1\left\langle k^2\right\rangle}{\langle k\rangle}, \quad (18)$$

where $\langle k^2\rangle = \sum_{k=1}^{n} k^2 p_k$ is the second moment of the degree distribution and $\langle k^3\rangle = \sum_{k=1}^{n} k^3 p_k$ is the third moment of degree distribution. In conclusion, we obtain the following theorem.

Theorem 2. *If the basic reproduction number* $R_0 < 1$, *system (4) has a disease-free equilibrium point* E_0; *if* $R_0 > 1$, *system (4) has a disease-free equilibrium point and a unique endemic equilibrium point* E^*.

5. Stability of Equilibrium Points

First, we analyse the global stability of the disease-free equilibrium point.

Theorem 3. *If $R_0 < 1$, the disease-free equilibrium point E_0 of system (4) is globally asymptotically stable within Ω.*

Proof. $\Omega = \{\rho_k \mid 0 \le \rho_k \le 1\}$ is an invariant region of system (4). The Lyapunov function can be constructed as

$$V\left(\rho_k(t)\right) = \sum_{k=1}^{n} \delta_k p_k \rho_k. \tag{19}$$

Taking the derivative along system (4) of formula (19), we can get

$$V'\left(\rho_k(t)\right)\Big|_{(4)} = -\widetilde{\theta}\left[\langle k\rangle\left(1 - R_0\right) + \sum_{k=1}^{n} k\delta_k p_k \rho_k\right]. \tag{20}$$

Therefore, for all $\rho_k \ge 0$, when $R_0 < 1$, $dV/dt \le 0$. Furthermore, $V'(t) = 0$ if and only if $\rho_k = 0$. So, we can obtain the global stability of the disease-free equilibrium. □

Next, the local stability of the endemic equilibrium is analyzed.

Theorem 4. *If $R_0 > 1$, then the endemic equilibrium E^* of system (4) is locally asymptotically stable.*

Proof. Let $x_k = \rho_k - \rho_k^*$; we can get the following equation from system (4):

$$\frac{dx_k(t)}{dt} = k\left(1 - x_k\right)\widetilde{\theta} - x_k, \tag{21}$$

where $\overline{\theta} = (1/\langle k\rangle)\sum_{k=1}^{n} \delta_k p_k x_k$, $\widetilde{\theta}^* = (1/\langle k\rangle)\sum_{k=1}^{n} \delta_k p_k \rho_k^*$.

Let us consider the linear systems

$$\frac{d}{dt}\begin{pmatrix} x_1(t) \\ x_2(t) \\ \vdots \\ x_n(t) \end{pmatrix} = A\begin{pmatrix} x_1(t) \\ x_2(t) \\ \vdots \\ x_n(t) \end{pmatrix}, \tag{22}$$

where

$$A = \begin{pmatrix} \frac{1}{\langle k\rangle}\delta_1 p(1) - 1 & \frac{1}{\langle k\rangle}\delta_2 p(2) & \cdots & \frac{1}{\langle k\rangle}\delta_n p(n) \\ \frac{2}{\langle k\rangle}\delta_1 p(1) & \frac{2}{\langle k\rangle}\delta_2 p(2) - 1 & \cdots & \frac{2}{\langle k\rangle}\delta_n p(n) \\ \vdots & \vdots & \ddots & \vdots \\ \frac{n}{\langle k\rangle}\delta_1 p(1) & \frac{n}{\langle k\rangle}\delta_2 p(2) & \cdots & \frac{n}{\langle k\rangle}\delta_n p(n) - 1 \end{pmatrix}. \tag{23}$$

Next, we perform a similar transformation on matrix A; that is, multiply the first row by $-2, -3, \cdots, -n$ and add it to the $2, 3, \cdots, n$ row, and then multiply the $2, 3, \cdots, n$ column by $2, 3, \cdots, n$ to the first column; then, matrix A becomes B:

$$B = \begin{pmatrix} \frac{1}{\langle k\rangle}\sum_{k=1}^{n} k\delta_k p(k) - 1 & \frac{1}{\langle k\rangle}\delta_2 p(2) & \cdots & \frac{1}{\langle k\rangle}\delta_n p(n) \\ 0 & -1 & \cdots & 0 \\ \vdots & \vdots & \ddots & \vdots \\ 0 & 0 & \cdots & -1 \end{pmatrix}. \tag{24}$$

Since matrices A and B have the same characteristic roots, we only need to consider the eigenvalues of matrix B. The characteristic equation of matrix B is

$$(\lambda + 1)^{n-1}\left[\lambda - \left(R_0 - 1\right)\right] = 0. \tag{25}$$

Obviously, when $R_0 > 1$, the real part of all the eigenvalues of matrix B is negative. The eigenvalues of B are also the eigenvalues of the Jacobian matrix of system (4) at E^*, so the endemic equilibrium point E^* of system (4) is locally asymptotically stable. □

Lemma 5. *Suppose that the initial relative infected densities $0 \le \rho_k(0) \le 1$ satisfy $\sum_{i=1}^{n} \delta_i p(i)\rho_i(0) > 0$ and that basic reproduction number $R_0 > 1$; then, the solution $\rho_k(t)$ of system (4) satisfies $\inf_{t\ge 0}\theta(t) > 0$; for any $\tau > 0$, $\inf_{t\ge\tau}\rho_k(t) > 0$.*

Proof. Similar to proving the global stability of the disease-free equilibrium, the following equation can be obtained:

$$\frac{d\widetilde{\theta}}{dt} = \widetilde{\theta}\left[R_0 - 1 - \frac{1}{\langle k\rangle}\sum_{k=1}^{n} k\delta_k p(k)\rho_k\right]. \tag{26}$$

Thus,

$$\frac{d\widetilde{\theta}}{dt} \ge \widetilde{\theta}\left[R_0 - 1 - n\widetilde{\theta}\right]. \tag{27}$$

Note that system

$$\frac{dx(t)}{dt} = x\left[R_0 - 1 - nx\right] \tag{28}$$

is logistic equation. Due to $x(0) > 0$, system (28) is consistent. By the comparison theorem, it is easy to see that $\inf_{t\ge 0}\widetilde{\theta} > 0$. Let $\alpha = \inf_{t\ge 0}\widetilde{\theta} > 0$. By system (2), we have, for all $t \ge 0$,

$$\frac{d\rho_k}{dt} = -(1 + k)\widetilde{\theta}\rho_k + k\widetilde{\theta} \ge -(1 + k)\rho_k + k\alpha. \tag{29}$$

By comparison theorem,

$$\rho_k(t) \ge \rho_k(0)e^{-(1+k)t} + \frac{k\alpha}{1 + k}\left(1 - e^{-(1+k)t}\right). \tag{30}$$

Hence, for $\forall\tau > 0$, $\inf_{t\ge\tau}\rho_k(t) > 0$. □

Theorem 6. *Suppose that the initial relative infected densities $0 \le \rho_k(t) \le 1$ satisfy $\sum_{i=1}^{n} \delta_i p(i)\rho_i(0) > 0$ and that $R_0 > 1$; then any solution of $\rho_k(t)$ from the invariant set Ω must satisfy $\lim_{t\to\infty}\rho_k(t) = \rho_k^*$, where $\rho_1^*, \rho_2^*, \cdots, \rho_n^*$ are the unique nonzero stationary points of system (4).*

Proof. In order to prove this theorem, we first prove that the limit $\lim_{t\longrightarrow\infty}\rho_k(t)$ exists. For this purpose, we have to prove that

$$\lim_{t\longrightarrow+\infty}\inf\rho_k(t)=\lim_{t\longrightarrow+\infty}\sup\rho_k(t). \tag{31}$$

Letting $u_k^{(1)}=1$, define the sequence

$$u_k^{(m+1)}=\frac{k\langle k\rangle^{-1}\sum_{i=1}^{n}\delta_i p(i)u_i^{(m)}}{1+k\langle k\rangle^{-1}\sum_{i=1}^{n}\delta_i p(i)u_i^{(m)}},\quad 1\le k\le n,\ m=1,2,\cdots. \tag{32}$$

According to Lemma 1, for $1\le k\le n$, $\lim_{t\longrightarrow+\infty}\sup\rho_k(t)\le 1=u_k^{(1)}$. Therefore, for $\forall\varepsilon>0$, $\exists\tau>0$, we have $\rho_k(t)=u_k^{(1)}+\varepsilon$, for $t\ge\tau$. From Lemma 1 and system (4), we have

$$\begin{aligned}\frac{d\rho_k(t)}{dt}&\le-\rho_k(t)+k\left[1-\rho_k(t)\right]\langle k\rangle^{-1}\sum_{i=1}^{n}\delta_i p(i)\left(u_i^{(1)}+\varepsilon\right)\\&=-\rho_k(t)+k\langle k\rangle^{-1}\sum_{i=1}^{n}\delta_i p(i)\left(u_i^{(1)}+\varepsilon\right)-k\langle k\rangle^{-1}\sum_{i=1}^{n}\delta_i p(i)\rho_k(t)\left(u_i^{(1)}+\varepsilon\right).\end{aligned} \tag{33}$$

We integrate the above inequality from $\tau\longrightarrow t$, getting

$$\rho_k(t)\le\frac{\rho_k(\tau)}{e^{[1+k\langle k\rangle^{-1}\sum_{i=1}^{n}\delta_i p(i)(u_i^{(1)}+\varepsilon)](t-\tau)}}+\frac{k\langle k\rangle^{-1}\sum_{i=1}^{n}\delta_i p(i)\left(u_i^{(1)}+\varepsilon\right)}{1+k\langle k\rangle^{-1}\sum_{i=1}^{n}\delta_i p(i)\left(u_i^{(1)}+\varepsilon\right)}. \tag{34}$$

Taking the limit as $t\longrightarrow+\infty$, we obtain

$$\lim_{t\longrightarrow+\infty}\sup\rho_k(t)\le\frac{k\langle k\rangle^{-1}\sum_{i=1}^{n}\delta_i p(i)\left(u_i^{(1)}+\varepsilon\right)}{1+k\langle k\rangle^{-1}\sum_{i=1}^{n}\delta_i p(i)\left(u_i^{(1)}+\varepsilon\right)}. \tag{35}$$

By the arbitrariness of ε, letting $\varepsilon\longrightarrow+\infty$, we have

$$\lim_{t\longrightarrow+\infty}\sup\rho_k(t)\le\frac{k\langle k\rangle^{-1}\sum_{i=1}^{n}\delta_i p(i)u_i^{(1)}}{1+k\langle k\rangle^{-1}\sum_{i=1}^{n}\delta_i p(i)u_i^{(1)}}=u_k^{(2)}. \tag{36}$$

Consider the convergence of (32) defined sequences. For all k, $u_k^{(2)}\le 1=u_k^{(1)}$.

$$u_k^{(m+1)}\le u_k^{(m)}. \tag{37}$$

$$\begin{aligned}u_k^{(m+2)}&=\frac{k\langle k\rangle^{-1}\sum_{i=1}^{n}\delta_i p(i)u_i^{(m+1)}}{1+k\langle k\rangle^{-1}\sum_{i=1}^{n}\delta_i p(i)u_i^{(m+1)}}\\&\le\frac{k\langle k\rangle^{-1}\sum_{i=1}^{n}\delta_i p(i)u_i^{(m)}}{1+k\langle k\rangle^{-1}\sum_{i=1}^{n}\delta_i p(i)u_i^{(m)}}=u_k^{(m+1)}.\end{aligned} \tag{38}$$

Similarly, it can be obtained that

$$\lim_{t\longrightarrow\infty}\sup\rho_k(t)\le\frac{k\langle k\rangle^{-1}\sum_{i=1}^{n}\delta_i p(i)u_i^{(1)}}{1+k\langle k\rangle^{-1}\sum_{i=1}^{n}\delta_i p(i)u_i^{(1)}}=u_k^{(m)},\quad 1\le k\le n,\ m=1,2,\cdots. \tag{39}$$

By induction, we know that for each k, the sequence $u_k^{(m)}$ is decreasing, so its limit exists and is denoted by $\lim_{m\longrightarrow\infty}u_k^{(m)}=u_k$. Letting $m\longrightarrow\infty$ on both sides of formulas (32) and (39), we deduce that $\lim_{m\longrightarrow\infty}u_k^{(m)}=u_k$ satisfies the following stability equation:

$$-u_k+k(1-u_k)\frac{1}{\langle k\rangle}\sum_{i=1}^{n}\delta_i p(i)u_i=0. \tag{40}$$

Then,

$$\lim_{t\longrightarrow\infty}\sup\rho_k(t)\le u_k,\quad 1\le k\le n. \tag{41}$$

On the other hand, we consider the function

$$f(x)=\frac{1}{\langle k\rangle}\sum_{k}^{n}\frac{k\delta_k p(k)x}{1+kx}-x. \tag{42}$$

By simple calculations, we obtain

$$f(0)=0. \tag{43}$$

$$\begin{aligned}f'(0)&=\frac{1}{\langle k\rangle}\sum_{k}^{n}k\delta_k p(k)-1\\&=\frac{C(1-l)\beta_1\left(\left\langle k^3\right\rangle-\left\langle k^2\right\rangle\right)+l\beta_1\langle k\rangle}{\langle k\rangle}-1\\&>0.\end{aligned} \tag{44}$$

By the definition of derivatives, if $x>0$ is sufficiently small, then $f(x)>f(0)=0$. According to Lemma 5 and formula (44), we can take $l_k^{(1)}$ such that

$$0<l_k^{(1)}<\lim_{t\longrightarrow\infty}\inf\rho_k(t),\qquad f\left(\frac{1}{\langle k\rangle}\sum_{i=1}^{n}\delta_i p(i)l_i^{(1)}\right)>0,\quad\forall k. \tag{45}$$

We define the following sequence:

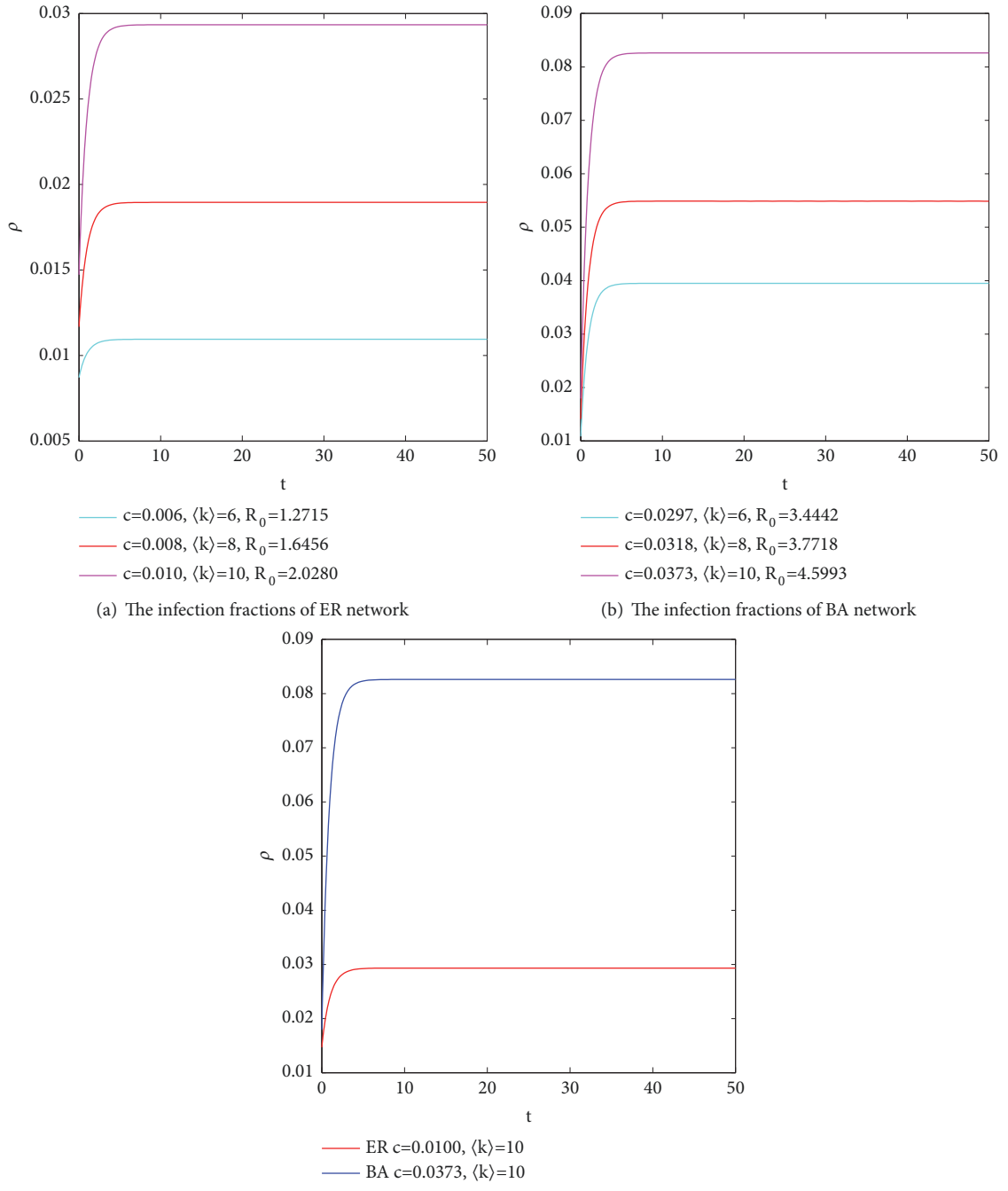
$$l_k^{(m+1)}=\frac{k\langle k\rangle^{-1}\sum_{i=1}^{n}\delta_i p(i)l_i^{(m)}}{1+k\langle k\rangle^{-1}\sum_{i=1}^{n}\delta_i p(i)l_i^{(m)}},\quad 1\le k\le n,\ m=1,2,\cdots. \tag{46}$$

Similar to $u_k^{(m)}$, proved by induction,

$$\lim_{t\longrightarrow+\infty}\inf\rho_k(t)\ge l_k^{(m)},\quad 1\le k\le n,\ m=1,2,\cdots. \tag{47}$$

Now, consider the convergence of the sequence defined by formula (46). First, according to formulas (41), (45), and (46), we have

$$\frac{1}{k}\sum_{i=1}^{n}\delta_i p(i)l_i^{(2)}>\frac{1}{k}\sum_{i=1}^{n}\delta_i p(i)l_i^{(1)}. \tag{48}$$



(a) The infection fractions of ER network

(b) The infection fractions of BA network

(c) Comparison of infection fractions between ER network and BA network

FIGURE 2: (a) In the ER network, the infection fractions with clustering coefficients $c = 0.006$, $\langle k\rangle = 6$, $c = 0.008$, $\langle k\rangle = 8$, and $c = 0.010$, $\langle k\rangle = 10$ are described, respectively. (b) In the BA network, the infection fractions with clustering coefficients $c = 0.0297$, $\langle k\rangle = 6$, $c = 0.0318$, $\langle k\rangle = 8$, and $c = 0.0373$, $\langle k\rangle = 10$ are described, respectively. (c) When $\langle k\rangle = 10$, the infection fractions in the random network with the clustering coefficient $c = 0.01$ and in the scale-free network with the clustering coefficient $c = 0.0373$ are described, respectively.

By formulas (46) and (48), we obtain

$$l_k^{(3)} > \frac{k\langle k\rangle^{-1}\sum_{i=1}^{n}\delta_i p(i)\, l_i^{(1)}}{1 + k\langle k\rangle^{-1}\sum_{i=1}^{n}\delta_i p(i)\, l_i^{(1)}} = l_k^{(2)}, \quad \forall k. \tag{49}$$

If for all k, $l_k^{(m+1)} > l_k^{(m)}$, it follows from formula (46) that

$$l_k^{(m+2)} > \frac{k\langle k\rangle^{-1}\sum_{i=1}^{n}\delta_i p(i)\, l_i^{(m)}}{1 + k\langle k\rangle^{-1}\sum_{i=1}^{n}\delta_i p(i)\, l_i^{(m)}} = l_k^{(m+1)}, \quad \forall k. \tag{50}$$

Thus, by induction, we know that, for each k, the sequence $l_k^{(m)}$, $m \geq 2$ is increasing, so its limit exists and is denoted by $l_k = \lim_{m\to\infty} l_k^{(m)}$. Letting $m \longrightarrow \infty$ on both sides of formulas

(46) and (47), we deduce that the limit $l_k = \lim_{m\longrightarrow\infty} l_k^{(m)}$ satisfies the following relations:

$$-l_k + k\left(1 - l_k\right)\frac{1}{\langle k\rangle}\sum_{i=1}^{n}\delta_i p\left(i\right) l_i = 0, \tag{51}$$

$$l_k \leq \lim_{t\longrightarrow+\infty} \inf \rho_k\left(t\right), \quad 1 \leq k \leq n. \tag{52}$$

By formulas (41) and (52), both $u_k = \lim_{m\longrightarrow\infty} u_k^{(m)}$ and $l_k = \lim_{m\longrightarrow\infty} l_k^{(m)}$ are positive stationary points of system (4); thus, by the uniqueness of the positive stationary point of system (4), we have that $u_k = l_k = \rho_k$ and

$$\rho_k \leq \lim_{t\longrightarrow+\infty} \inf \rho_k\left(t\right) \leq \lim_{t\longrightarrow+\infty} \sup \rho_k\left(t\right) \leq \rho_k, \quad 1 \leq k \leq n. \tag{53}$$

That is, $\lim_{t\longrightarrow+\infty}\rho_k(t) = \rho_k$, and Theorem 6 is proved. □

6. Numerical Simulation and Conclusion

In this section, we present some numerical simulations of system (3) in a random network and a scale-free network to study the effect of clustering coefficients on disease transmission. We first consider the degree distribution of the network. In the random network, p_k obeying Poisson distribution is expressed as $p_k = e^{-\lambda}\lambda^k/k!$, where λ indicates the average degree of nodes in the network. In the scale-free network, p_k obeying a Power-law distribution is expressed as $p_k = 2m^2/k^3$, where m represents the number of edges generated for each new node introduced in the network.

In Figure 2, we take the total number of nodes in the network $N = 1000$, $\beta_1 = 0.22$, and $\beta_2 = 0.18$. Figures 2(a) and 2(b) show that the infection fractions ρ and the basic reproduction number R_0 will be increasing with the clustering coefficient in the ER random network and the BA scale-free network. This means that the increase in clustering coefficient can easily cause some nodes to connect with their neighbor's neighbors. So some triangles are formed; the final epidemic size will be increasing. From Figure 2(c), we compare the infection fractions in the ER random network and the BA scale-free network where the average degree is fixed and the clustering coefficient is changed. It is found that the increase in clustering coefficient always promotes the disease spreading.

In conclusion, the network model we presented more accurately depicts the special local relationships between individuals in the contact network. We study the influence of clustering coefficients on the basic reproduction number and the infection fractions in the network. The basic reproduction number can change larger as the clustering coefficient increases. Thus, the disease is more easy to spread. Simulations indicate that the final infection fraction can increase when the clustering coefficient is larger. From the perspective of sociology and biology, the reduction of household or school clusters will effectively impede the disease spreading.

Data Availability

No data were used to support this study. The values of parameters that appeared in the simulations are assumed by us.

Conflicts of Interest

The authors declare that they have no conflicts of interest.

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