



Research article

Modeling epidemic in metapopulation networks with heterogeneous diffusion rates

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Abstract: In this paper, the process of the infectious diseases among cities is studied in metapopulation networks. Based on the heterogeneous diffusion rate, the epidemic model in metapopulation networks is established. The factors affecting diffusion rate are discussed, and the relationship among diffusion rate, connectivity of cities and the heterogeneity parameter of traffic flow is obtained. The existence and stability of the disease-free equilibrium and the endemic equilibrium are analyzed, and epidemic threshold is also obtained. It is shown that the more developed traffic of the city, the greater the diffusion rate, which resulting in the large number of infected individuals; the stronger the heterogeneity of the traffic flow, the greater the threshold of the disease outbreak. Finally, numerical simulations are performed to illustrate the analytical results.

Keywords: epidemic; metapopulation; reaction diffusion process; diffusion rate; stability

1. Introduction

Metapopulation model is not only used to describe population reproduction, migration, competition and death [1–4], but also describe the spread of disease in real networks [5–9]. Colizza et al. have done a lot of representative results on metapopulation models with heterogeneous degree distribution, which mainly consider the spread of epidemic influenced by individual movement [10–16]. Juher et al. have given threshold for metapopulation epidemic model with uncorrelated network [17, 18]. The reaction-diffusion equation is usually used to describe the propagation process, which assumed that the processes of reaction and diffusion occur simultaneously [14, 17, 19, 20]. Furthermore, there is becoming up-front trend that concerns not only disease spreading but also human decision making; whether he is committing vaccination or not [21–25].

In an SIS dynamic system, there are two kinds of processes between individual states: $I \xrightarrow{\mu} S, I +$

$S \xrightarrow{\beta} 2I$. Here μ is the recovery rate and β is the transmission rate across an infective contact. The influence of individuals diffusion was considered on disease transmission in papers [26, 27]. Here the node represents a city or an airport, and the edge represents the connection between cities. For a system with N nodes, which include two types of individuals S, I . The diffusion rate of infected individuals and susceptible individuals in different subpopulations are D_I, D_S (D_I, D_S are all nonnegative). Thus the dynamic system is as follows:

$$\begin{cases} \frac{d\rho_{S,i}}{dt} = -\beta\rho_{S,i}\rho_{I,i} + \mu\rho_{I,i} - D_S\rho_{S,i} + D_S \sum_{j=1}^N \frac{A_{ji}}{k_j} \rho_{S,j}, \\ \frac{d\rho_{I,i}}{dt} = \beta\rho_{S,i}\rho_{I,i} - \mu\rho_{I,i} - D_I\rho_{I,i} + D_I \sum_{j=1}^N \frac{A_{ji}}{k_j} \rho_{I,j}, \end{cases}$$

where $\rho_{S,i}(\rho_{I,i})$ is the average density of susceptible individuals (infected individuals) in node i , A_{ji} ($1 \leq j, i \leq N$) is an adjacency matrix, and k_j is the degree of node j . If there is a connection between node j and i , then $A_{ji} = 1$; otherwise $A_{ji} = 0$. In the first equation of above system, the first term represents the number of susceptible individuals becoming infected, and the second term is the number of infected individuals recovering susceptible, and the third term is the number of individuals that diffuse away from the node; the last term is the number of individuals which diffuse into the node.

The data of the International Air Transport Association was analyzed in [26, 28, 29]. It is shown that there is a strong heterogeneity about the airport connectivity and traffic capacity. The different carrying capacity of route is considered in [11, 12, 30], which is reflected in the size of the traffic flow. In paper [11], the metapopulation epidemic model is established in the mean field, and the average traffic flow in the subpopulation with degree k in per unit time is $T_k = k \sum_{k'} p(k'/k) \omega_{kk'}$, where $p(k'/k)$ is the conditional probability that the node with degree k connects the node with degree k' (in the uncorrelated network $p(k'/k) = k' p(k')/\langle k \rangle$). Here $\omega_{kk'} = \omega_0(kk')^\nu$ is the average weight, where ω_0 is the coefficient of a particular system, and ν is the heterogeneity parameter of traffic flow ($0 \leq \nu \leq 1$). In the uncorrelated network T_k can be wrote in the form of $T_k = \omega_0 k^{1+\nu} \langle k^{1+\nu} \rangle / \langle k \rangle$. The diffusion rate of nodes with degree k is $D_k = T_k / \rho_k$. The effects of diffusion on the disease due to traffic driving have also been studied in [31].

In this paper we consider an epidemic model in metapopulation network with heterogeneous diffusion rate. The heterogeneity includes the difference of urban scale, the discrepancy of traffic conditions and so on [11, 32–34]. We use D_S^{ij}, D_I^{ij} to express the diffusion rate of susceptible and infected individuals from node i to node j , which is more in line with the heterogeneity of metapopulation network. In fact, when the traffic of a city is more developed, the number of people flowing into and out of the city is larger. So, for the convenience of studying the problem, we assume that $D_S^{ij} = D_S^{ji} = D_S^i$. From the view of travel, infected individuals will reduce the amount of travel, thus the relation is taken as follows: $D_I^i = rD_S^i$, where $0 < r \leq 1$ is constant. In this paper, we also give the relationship among the diffusion rate, connectivity and the heterogeneity parameter of traffic flow. The results show that, if considering the heterogeneity of the degree and the heterogeneity of the traffic flow at the same time, the relationship can be obtained among the diffusion rate, connectivity and the heterogeneity of traffic flow. It can be got a more real conclusion than before.

The paper is organized as follows. The model is formulated in section 2 and the disease-free equilibrium is obtained. Some mathematical analysis are given in section 3 and the stability of the disease-free equilibria of the model is investigated. The existence and stability of the endemic

equilibria of the model are studied in sections 4 and 5 respectively. In section 6, numerical simulations are illustrated.

2. The model

Considering an SIS model with nonlimited transmission, the master equation is obtained as follows

$$\begin{cases} \frac{d\rho_{S,i}(t)}{dt} = -\beta\rho_{S,i}\rho_{I,i} + \mu\rho_{I,i} - D_S^i\rho_{S,i} + \sum_{j=1}^N D_S^i \frac{A_{ji}}{k_j} \rho_{S,j}, \\ \frac{d\rho_{I,i}(t)}{dt} = \beta\rho_{S,i}\rho_{I,i} - \mu\rho_{I,i} - D_I^i\rho_{I,i} + \sum_{j=1}^N D_I^i \frac{A_{ji}}{k_j} \rho_{I,j}. \end{cases} \quad (2.1)$$

In uncorrelated networks, A_{ji} can be approximately expressed in the form $A_{ji} \simeq k_j k_i / (N\langle k \rangle)$ [26], where $\langle k \rangle = \sum_{i=1}^N k_i / N$ is the average degree of the network, thus one obtains the following equations for the epidemic spread in metapopulation networks:

$$\begin{cases} \frac{d\rho_{S,i}(t)}{dt} = -\beta\rho_{S,i}\rho_{I,i} + \mu\rho_{I,i} - D_S^i\rho_{S,i} + D_S^i \frac{k_i}{\langle k \rangle} \rho_S, \\ \frac{d\rho_{I,i}(t)}{dt} = \beta\rho_{S,i}\rho_{I,i} - \mu\rho_{I,i} - D_I^i\rho_{I,i} + D_I^i \frac{k_i}{\langle k \rangle} \rho_I. \end{cases} \quad (2.2)$$

It is easy to see that the total density of individuals $\rho(t) = \rho_S(t) + \rho_I(t)$ remains constant and equal to ρ_0 , the initial average number of individuals per cities, where $\rho_S = \sum_{j=1}^N \rho_{S,j} / N$, $\rho_I = \sum_{j=1}^N \rho_{I,j} / N$. In these metapopulation networks, the connectivity matrix Φ is given by

$$\Phi = \frac{1}{N\langle k \rangle} \begin{pmatrix} k_1 & k_1 & \cdots & k_1 \\ k_2 & k_2 & \cdots & k_2 \\ \vdots & \vdots & \ddots & \vdots \\ k_N & k_N & \cdots & k_N \end{pmatrix}.$$

3. The stability of disease-free equilibrium

The disease-free equilibrium of system (2.2) is $E_0 = (k_1\rho_0/\langle k \rangle, \dots, k_N\rho_0/\langle k \rangle, 0, \dots, 0)$. The local stability of the disease-free equilibrium can be determined by Jacobian matrix. The Jacobian matrix of system (2.2) at the disease-free equilibrium is

$$J_{E_0} = \begin{pmatrix} A & B \\ O & C \end{pmatrix},$$

where A, B, O, C are matrix blocks of $N \times N$, $A = \text{diag}(D_S^i)(\Phi - I_d)$, Φ is the connectivity matrix, I_d is the identity matrix, $B = -\text{diag}(\beta k_i \rho_0 / \langle k \rangle - \mu)$, O is the null matrix, and $C = \text{diag}(\beta k_i \rho_0 / \langle k \rangle - \mu) + \text{diag}(D_I^i)(\Phi - I_d)$. Then, the characteristic polynomial of J_{E_0} are the product of the characteristic polynomial of diagonal matrix blocks $P_{J_{E_0}}(\lambda) = P(\lambda^A)P(\lambda^C)$. Taking $D_S^{\min} = \min\{D_S^i, i = 1, 2, \dots, N\}$ and we consider another matrix $\bar{A} = D_S^{\min}(\Phi - I_d)$, which has the maximum eigenvalue $\lambda_{\max}^{\bar{A}} = 0$.

Noticed that $\lambda_{\max}^A \leq \lambda_{\max}^{\bar{A}} = 0$, it can be proved that the eigenvalues of the matrix block A are all non-positive.

By using the eigenvalue perturbation theorem in [35], the relationship between the eigenvalues of C and $\text{diag}(\beta k_i \rho_0 / \langle k \rangle - \mu)$ is given by

$$\lambda_{\max}^C > \max_{1 \leq i \leq N} \left\{ \frac{\beta k_i \rho_0}{\langle k \rangle} - \mu - D_I^i \right\}, \quad i = 1, 2, \dots, N. \quad (3.1)$$

Therefore, the maximum eigenvalue of J_{E_0} is $\lambda_{\max} = \max\{0, \lambda_{\max}^C\}$. If $\lambda_{\max} > 0$, the disease-free equilibrium of system (2.2) is unstable, and the system has an endemic equilibrium. Thus we have the following theorem:

Theorem 3.1. *The sufficient condition of the disease-free equilibrium to be unstable is*

$$\max_{1 \leq i \leq N} \left\{ \frac{\beta k_i \rho_0}{\langle k \rangle} - \mu - D_I^i \right\} \geq 0. \quad (3.2)$$

Theorem 3.2. *It can be seen from Eq (3.2), for fixed μ , even if β is sufficient small, if ρ_0 is large enough, the disease-free equilibrium is also unstable. While for fixed ρ_0 , the diffusion rates D_I^i will affect the spread of the disease.*

As mentioned in the first section, the traffic flow is the physical quantity in the mean field. Here, we assume that all nodes with the same degree are one class [14, 15]. In this paper T_{k_i} represent traffic flow with degree k_i in node i . According to the previous discussion in the first section, the diffusion rate of node i can be expressed as

$$D_i = \frac{T_{k_i}}{\rho_i} = \frac{\langle k^{1+\nu} \rangle \omega_0 k_i^\nu}{\rho_0}, \quad (3.3)$$

where $\rho_i = \frac{k_i \rho_0}{\langle k \rangle}$ is the average density of node i . From Eq (3.3), it is not difficult to see that D_i is a function of k_i . Similarly, we can get the diffusion rates D_I^i, D_S^i . Based on $D_i = \frac{T_{I,k_i} + T_{S,k_i}}{\rho_{I,i} + \rho_{S,i}}$, the relationship can be got about D_i, D_I^i, D_S^i :

$$D_i \rho_i = D_I^i \rho_{I,i} + D_S^i \rho_{S,i}, \quad (3.4)$$

Substituting $\rho_{S,i} = \rho_i - \rho_{I,i}$ into the Eq (3.4), it can be simplified

$$D_I^i = \frac{1}{h_i} D_i + \left(1 - \frac{1}{h_i}\right) D_S^i, \quad (3.5)$$

(where $h_i = \frac{\rho_{I,i}}{\rho_i}$). Substituting (3.3) into (3.5), we can get

$$D_I^i = \frac{\langle k^{1+\nu} \rangle \omega_0}{h_i \rho_0} k_i^\nu + \left(1 - \frac{1}{h_i}\right) D_S^i. \quad (3.6)$$

It can be seen that D_I^i is a function of k_i and parameter ν .

Theorem 3.3. *Let $D_I^i = r D_S^i$, ($0 < r \leq 1$), then*

$$D_S^i = \frac{\langle k^{1+\nu} \rangle \omega_0 k_i^\nu}{[1 - h_i(1 - r)] \rho_0}, \quad D_I^i = \frac{r \langle k^{1+\nu} \rangle \omega_0 k_i^\nu}{[1 - h_i(1 - r)] \rho_0}.$$

When $r = 1$, then $D_I^i = D_S^i = \frac{\langle k^{1+\nu} \rangle \omega_0 k_i^\nu}{\rho_0}$, which is satisfying (3.4).

Theorem 3.4. *The unstable condition of the disease-free equilibrium is $\beta > \beta_c$, where*

$$\beta_c = \frac{\mu \langle k \rangle}{\rho_0 k_{\max}} + \frac{\langle k \rangle \langle k^{1+\nu} \rangle \omega_0}{\rho_0^2 k_{\max}^{1-\nu}}. \quad (3.7)$$

Here k_{\max} is the maximum degree of all nodes in the metapopulation network. When $\nu = 0$, $\beta_c = \frac{\langle k \rangle (\mu \rho_0 + \langle k \rangle \omega_0)}{\rho_0^2 k_{\max}}$; And when $\nu = 1$, there is $\beta_c = \frac{\langle k \rangle (\mu \rho_0 + \langle k^2 \rangle \omega_0 k_{\max})}{\rho_0^2 k_{\max}}$.

Comparing the above two thresholds, it is shown that the epidemic threshold increases with the traffic flow heterogeneity parameter increase.

4. The existence and uniqueness of endemic equilibrium

The endemic equilibrium $E^* = (\rho_{S,1}^*, \rho_{S,2}^*, \dots, \rho_{S,N}^*, \rho_{I,1}^*, \rho_{I,2}^*, \dots, \rho_{I,N}^*)$ of system (2.2) is satisfied

$$D_S^i \rho_{S,i}^* + D_I^i \rho_{I,i}^* = D_S^i \frac{k_i}{\langle k \rangle} \rho_S^* + D_I^i \frac{k_i}{\langle k \rangle} \rho_I^*,$$

where $\rho_S^* = \frac{1}{N} \sum_{i=1}^N \rho_{S,i}^*$, $\rho_I^* = \frac{1}{N} \sum_{i=1}^N \rho_{I,i}^*$, and $\rho_{S,i}^*$ can be expressed

$$\rho_{S,i}^* = \frac{k_i}{\langle k \rangle} \left[\rho_0 + \left(\frac{D_I^i}{D_S^i} - 1 \right) \rho_I^* \right] - \frac{D_I^i}{D_S^i} \rho_{I,i}^*. \quad (4.1)$$

Putting Eq (4.1) into the second equation of system (2.2) and solving a quadratic equation, then we get

$$\rho_{I,i}^* = \frac{\beta \frac{k_i}{\langle k \rangle} \rho_0 - (\mu + D_I^i) + \delta + \sqrt{[\beta \frac{k_i}{\langle k \rangle} \rho_0 - (\mu + D_I^i) + \delta]^2 + \frac{D_I^i}{D_S^i} \theta}}{2\beta \frac{D_I^i}{D_S^i}}, \quad (4.2)$$

where $\delta = \beta \frac{k_i}{\langle k \rangle} \rho_I^* (\frac{D_I^i}{D_S^i} - 1)$, $\theta = 4\beta \frac{k_i}{\langle k \rangle} \rho_I^* D_I^i$. The negative root of equation has been taken out.

Now, $\rho_{I,i}^* > 0$ need to be proven. Taking the summation over i and multiplying $\frac{1}{N}$ for (4.2), it can be obtained as follows

$$\rho_I^* = \frac{1}{N} \sum_{i=1}^N \frac{\beta \frac{k_i}{\langle k \rangle} \rho_0 - (\mu + D_I^i) + \delta + \sqrt{[\beta \frac{k_i}{\langle k \rangle} \rho_0 - (\mu + D_I^i) + \delta]^2 + \frac{D_I^i}{D_S^i} \theta}}{2\beta \frac{D_I^i}{D_S^i}},$$

where $0 \leq \rho_I^* \leq \rho_0$. We define function

$$F(\rho_I) = \frac{1}{N} \sum_{i=1}^N \frac{\beta \frac{k_i}{\langle k \rangle} \rho_0 - (\mu + D_I^i) + \delta' + \sqrt{[\beta \frac{k_i}{\langle k \rangle} \rho_0 - (\mu + D_I^i) + \delta']^2 + \frac{D_I^i}{D_S^i} \theta'}}{2\beta \frac{D_I^i}{D_S^i}} - \rho_I, \quad (4.3)$$

where $\delta' = \beta \frac{k_i}{\langle k \rangle} \rho_I (\frac{D_I^i}{D_S^i} - 1)$, $\theta' = 4\beta \frac{k_i}{\langle k \rangle} \rho_I D_I^i$.

When $D_I^i = rD_S^i$ ($0 < r \leq 1$), we get

$$F(\rho_I) = \frac{1}{N} \sum_{i=1}^N \frac{\beta_{\langle k \rangle}^{k_i} \rho_0 - (\mu + D_I^i) + \delta' + \sqrt{[\beta_{\langle k \rangle}^{k_i} \rho_0 - (\mu + D_I^i) + \delta']^2 + \theta'}}{2\beta r} - \rho_I,$$

where $\delta' = \beta_{\langle k \rangle}^{k_i} \rho_I (r - 1)$, $\theta' = 4\beta_{\langle k \rangle}^{k_i} \rho_I D_I^i$. Especially, when $r = 1$, then

$$\rho_{I,i}^* = \frac{\beta_{\langle k \rangle}^{k_i} \rho_0 - (\mu + D_I^i) + \sqrt{[\beta_{\langle k \rangle}^{k_i} \rho_0 - (\mu + D_I^i)]^2 + \theta'}}{2\beta},$$

$$\rho_{S,i}^* = \frac{k_i}{\langle k \rangle} \rho_0 - \rho_{I,i}^*,$$

$$F(\rho_I) = \frac{1}{N} \sum_{i=1}^N \frac{\beta_{\langle k \rangle}^{k_i} \rho_0 - (\mu + D_I^i) + \sqrt{[\beta_{\langle k \rangle}^{k_i} \rho_0 - (\mu + D_I^i)]^2 + \theta'}}{2\beta} - \rho_I.$$

$F(\rho_0) < 0$ is always satisfied.

When $\beta = \beta_c$, we can obtain $F(0) = 0$;

$$F'(\rho_I) = \frac{1}{N} \sum_{i=1}^N \frac{D_I^i \frac{k_i}{\langle k \rangle}}{\sqrt{4\beta D_I^i \frac{k_i}{\langle k \rangle} \rho_I}} - 1;$$

$F''(\rho_I) < 0$. So, there is a ρ_I^* that makes $F(\rho_I^*) = 0$.

When $\beta > \beta_c$, we can get $F(0) > 0$. Thus, there is also a ρ_I^* that makes $F(\rho_I^*) = 0$.

Theorem 4.1. *There is a unique endemic equilibrium $E^* = (\rho_{S,1}^*, \rho_{S,2}^*, \dots, \rho_{S,N}^*, \rho_{I,1}^*, \rho_{I,2}^*, \dots, \rho_{I,N}^*)$ of system (2.2), if the infection rate is larger than epidemic threshold $\beta \geq \beta_c$.*

Theorem 4.2. *In fact it can be proved that the existence of ρ_I^* that make (4.3) satisfied, when $0 < r < 1$. Therefore, the existence and uniqueness of the solution are independent of the relation about D_I^i, D_S^i .*

5. The stability of endemic equilibrium

The Jacobian matrix of system (2.2) at the endemic equilibrium is

$$J_{E^*} = \begin{bmatrix} D_S^i(\Phi - I_d) - \text{diag}(\beta \rho_{I,i}^*) & \text{diag}(\mu - \beta \rho_{S,i}^*) \\ \text{diag}(\beta \rho_{I,i}^*) & D_I^i(\Phi - I_d) + \text{diag}(\beta \rho_{S,i}^* - \mu) \end{bmatrix}.$$

When $r = 1$, the above matrix can be transformed into a new matrix as follows:

$$J_{E^*}' = \begin{bmatrix} A' & O \\ B' & C' \end{bmatrix}.$$

Thus the characteristic polynomial of J_{E^*}' can be given by $P_{J_{E^*}'}(\lambda') = P(\lambda'_{A'})P(\lambda'_{C'})$.

For the matrix $A' = D_S^i(\Phi - I_d)$, $\lambda'_{A'} \leq 0$ is also satisfied according to section 3. $C' = \text{diag}[\beta(\rho_{S,i}^* - \rho_{I,i}^*) - \mu] + D_I^i(\Phi - I_d)$ is equivalent to the sum of a diagonal matrix and a perturbation matrix. So, using the general formula of secular equation [35], we can get

$$\sum_{i=1}^N \frac{D_I^i k_i}{\langle k \rangle N} \frac{1}{(\alpha_i - \lambda'_{C'})} + 1 = 0 \quad (1 \leq i \leq N), \quad (5.1)$$

where

$$\alpha_i = [\beta(\rho_{S,i}^* - \rho_{I,i}^*) - (\mu + D_I^i)] = -\sqrt{\left\{ \frac{\beta k_i}{\langle k \rangle} \rho_0 - (\mu + D_I^i) \right\}^2 + 4\beta D_I^i \frac{k_i}{\langle k \rangle} \rho_I^*} < 0,$$

are the eigenvalues of $\text{diag}[\beta(\rho_{S,i}^* - \rho_{I,i}^*) - \mu - D_I^i]$, and $\lambda'_{C'}$ is an eigenvalue of C' . Even $\alpha_i < \lambda'_{C'}$, but $\lambda'_{C'} < 0$ can not be judged. Then, we prove $\lambda'_{C'} < 0$ is satisfied.

Making right side of the second formula of system (2.2) is 0, we can obtain $\rho_{I,i}^*(\beta\rho_{S,i}^* - \mu) = D_I^i(\rho_{I,i}^* - \frac{k_i}{\langle k \rangle})\rho_I^*$, which is simplified to

$$\langle k \rangle \rho_{I,i}^* = \frac{D_I^i k_i}{(\mu + D_I^i) - \beta \rho_{S,i}^*} \rho_I^*. \quad (5.2)$$

Summing over i and dividing by N for Eq (5.2), we obtain $\langle k \rangle N = \sum_{i=1}^N \frac{D_I^i k_i}{(\mu + D_I^i) - \beta \rho_{S,i}^*}$. So, it can be judged that

$$\sum_{i=1}^N \frac{D_I^i k_i}{(\mu + D_I^i) - \beta(\rho_{S,i}^* - \rho_{I,i}^*)} < \langle k \rangle N. \quad (5.3)$$

Eq (5.3) can be rewritten as

$$\sum_{i=1}^N \frac{D_I^i k_i}{\langle k \rangle N} \frac{1}{\alpha_i} + 1 > 0. \quad (5.4)$$

From Eqs (5.1) and (5.4), it can be inferred that $\lambda'_{C'} < 0$. So the eigenvalues of J_{E^*} is negative. According to the above statement, we have the following theorem.

Theorem 5.1. *The endemic equilibrium E^* of system (2.2) is always locally asymptotically stable, if it exists.*

6. Numerical simulation and sensitivity analysis

In the above section, we get the equilibrium existence and stability of system (2.2) through theoretical analysis, and get the epidemic threshold. The following is the numerical simulations.

6.1. On scale-free network

We simulate the spread of disease among N interacting nodes, here $N = 100$. It is found that there are two main factors that affect the diffusion rate and epidemic threshold: One is the connectivity of nodes, and another is the heterogeneity parameter of the traffic flow. On scale-free network, the adjacency A of order $N \times N$ is generated randomly (A is a symmetric matrix with the diagonal elements are all 0, the other elements are 0 or 1). Then, the degree of node i is the sum of the elements of all

the rows in line i of A_{ij} . On scale-free network, the degree distribution obeys power-law distribution $p(k) \sim k^{-\gamma}$. Therefore, the effect of the parameter ν for the diffusion rate and epidemic threshold should be explored by some sensitivity analysis about ν . In the real world network, the traffic flow have a certain heterogeneity. It can be seen from Figure 1a, D_i^i increase with the increase of ν , and the heterogeneity of the diffusion rate also increase. At the same time, the threshold β_c is also increase in Figure 1b. That is to say, the parameter ν increases the epidemic threshold and suppresses the outbreak of diseases. In the case of $\beta > \beta_c$, the endemic equilibrium of the system is locally asymptotically

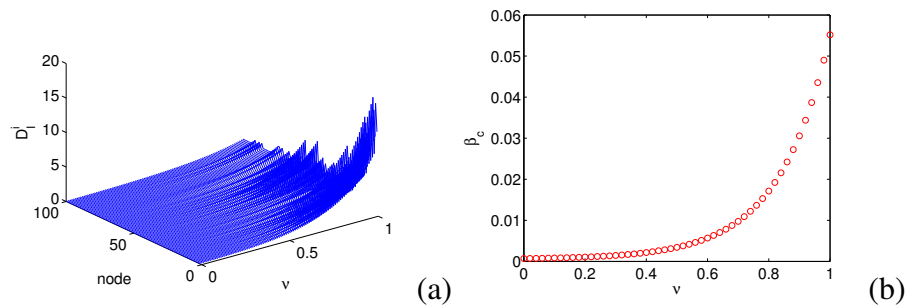


Figure 1. Results for SIS model on scale-free networks with $\rho_0 = 80$, $\mu = 0.212$, $\omega_0 = 1$. (a) Relationship between D_i^i and ν . (b) Relationship between β_c and ν .

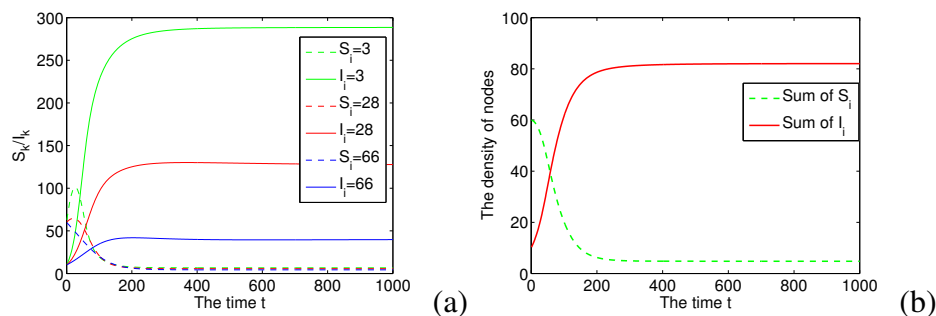


Figure 2. The time evolutions of the density of three different nodes $i = 3, 28, 66$ on scale-free networks respectively. Here $\rho_0 = 80$, $\beta = 0.0425$, $\mu = 0.212$, $\omega_0 = 1$.

stable. When $\nu = 0.6$, the number of susceptible individuals tends to be stable; At the same time, the number of infected individuals gradually tends to be stable, thus the disease is prevalent. It is shown that the more developed of city traffic is, the more people will be infected (Figure 1a). And for the whole system, the change of average density of overall S, I population is similarity (Figure 1b).

For one city, the heterogeneity of traffic flow will also affect the infection rate. For the cities with larger degree, the greater the ν is, the more the infected individuals are. That is to say, the heterogeneity of traffic flow has an influence on the spread of the disease. For the cities with smaller degree, with the increase of ν , the disease will go extinct (Figure 2). In general, the traffic heterogeneity of cities is

about $\nu \approx 0.5$ [11], which can inhibit the prevalence of the disease.

6.2. On small-world network

In this section, the spread of disease on the small-world network is studied. The influence of ν for the diffusion rate and epidemic threshold are shown in Figure 3. In Figure 4, it is the change of individuals (susceptible and infected) and the overall average density over time. From Figure 5, it is shown that the influence of ν for the infected individuals and the infection rate. In Figure 7 the comparison of the epidemic between on the small-world and scale-free networks is given.

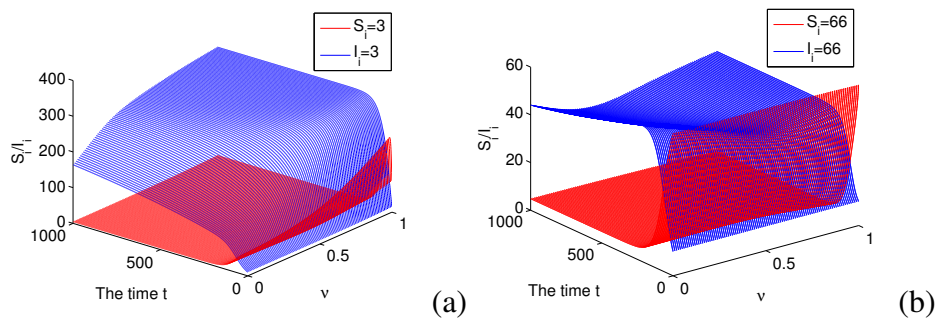


Figure 3. The time evolutions of the density of two different nodes $i = 3, 66$ with ν , respectively. Here $\rho_0 = 80$, $\beta = 0.0425$, $\mu = 0.212$, $\omega_0 = 1$.

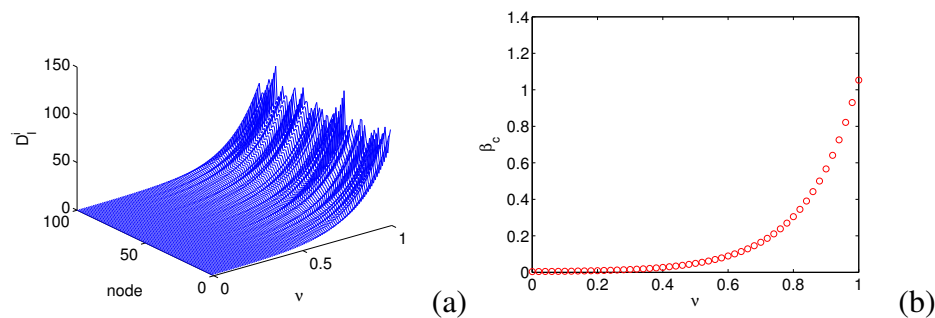


Figure 4. Results for SIS model on small-world network with $\rho_0 = 80$, $\mu = 0.212$, $\omega_0 = 1$. (a) Relationship between D_i^i and ν . (b) Relationship between β_c and ν .

It can be seen that for the appropriate ν , the greater the degree of the city is, the greater the diffusion rate (Figure 4). With the increase of ν , D_i^i and the epidemic threshold β_c all increase. That is also to say, the parameter ν increases the epidemic threshold and suppresses outbreak of the disease (Figure 5). In the case of $\beta > \beta_c$, the endemic equilibrium of the system is locally asymptotically stable. If the epidemic threshold is reached, the prevalence of the disease will be promoted with increase of parameter ν (Figure 6). From Figure 7, the system is locally asymptotically stable at the disease-free equilibrium on the small-world network, while the disease is spreading on the scale-free network. It can

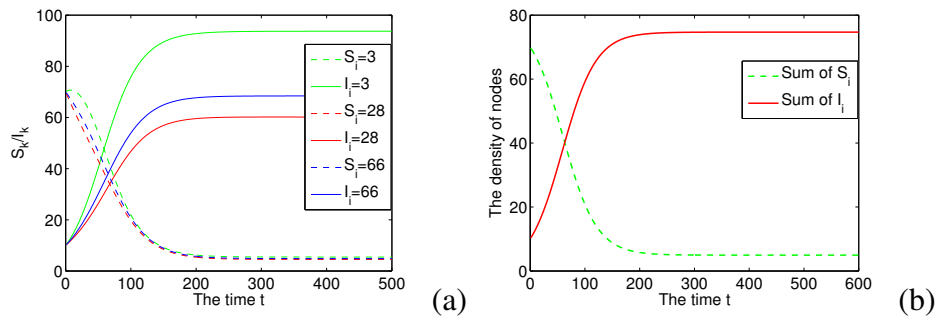


Figure 5. The time evolutions of the density of three different nodes $i = 3, 28, 66$ on small-world network respectively. Here $\nu = 0.4$, $\rho_0 = 80$, $\beta = 0.0425$, $\mu = 0.212$, $\omega_0 = 1$.

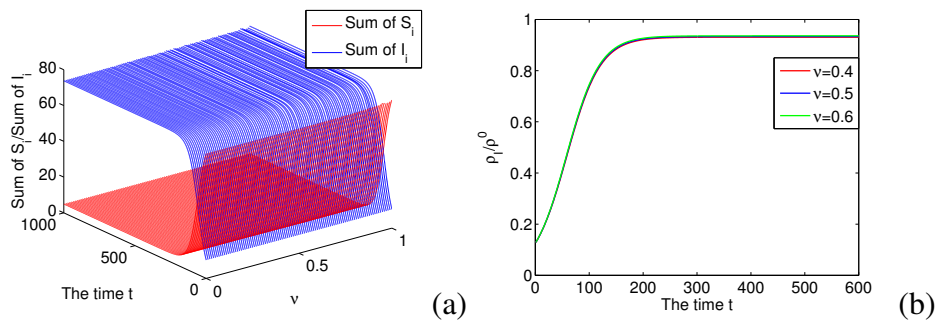


Figure 6. The time evolutions of the density of three different nodes $i = 3, 28, 66$ on small-world network respectively. (a) is the change overall average density of S , I with ν . (b) is the change of the infection rate for different ν .

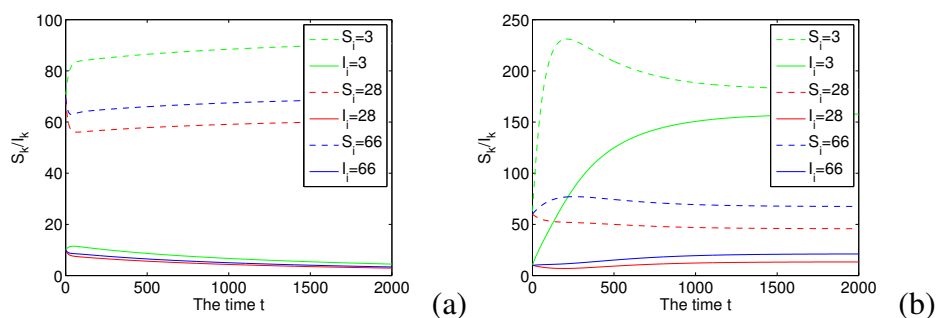


Figure 7. The time evolutions of the density of three different nodes $i = 3, 28, 66$ on different networks. (a) is on the small-world network. (b) is on scale-free networks.

be seen that the disease is more prevalent on the scale-free network than on the small-world network at the same parameters. The results show that the heterogeneity of traffic flow has a greater impact on the disease, and the epidemic is easier to be controlled on the small-world network.

7. Conclusion

In this paper, an SIS model in metapopulation networks with heterogeneous diffusion rates is established. According to the qualitative analysis of dynamics, the existence and stability of the disease-free equilibrium are analyzed, and the epidemic threshold is obtained. When the epidemic threshold is reached, the disease-free equilibrium is unstable, and the system has an endemic equilibrium. It is proved that it is locally asymptotically stable, if the endemic equilibrium is existing.

Due to there is a big difference in the traffic level and population density in each city, the diffusion rate of each city also have heterogeneity. In this paper, we consider the spread of disease with heterogeneity of the degree and heterogeneity of diffusion rate, which is more suitable for the real world. Based on this study, the relationship among the diffusion rate, connectivity and the heterogeneity of traffic flow are given. There are two conclusions: On the one hand, for the larger degree nodes, the number of infected individuals is higher than the smaller one. When the epidemic threshold is reached, the more developed of city is, the higher the diffusion rate is, which result in the large number of individuals enter from other nodes. Therefore, this increases the spread of disease; on the other hand, from the view of disease control, heterogeneity of the traffic flow by increase the epidemic threshold, can improve the ability to control the disease. Finally, numerical simulations are compared on the scale-free network and the small-world network.

In this paper, because the eigenvalues of the Jacobian matrixes at the equilibria could not be directly calculated, we estimated them by using other methods. In addition, we consider that the diffusion rate from different cities is the same, and the diffusion rate, which is proportion to the traffic flow, will be discussed in the future.

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Conflict of interest

The authors declare there is no conflict of interest.

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