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EPIDEMIC MODELS FOR COMPLEX NETWORKS WITH DEMOGRAPHICS

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Abstract. In this paper, we propose and study network epidemic models with demographics for disease transmission. We obtain the formula of the basic reproduction number R_0 of infection for an SIS model with births or recruitment and death rate. We prove that if $R_0 \leq 1$, infection-free equilibrium of SIS model is globally asymptotically stable; if $R_0 > 1$, there exists a unique endemic equilibrium which is globally asymptotically stable. It is also found that demographics has great effect on basic reproduction number R_0. Furthermore, the degree distribution of population varies with time before it reaches the stationary state.

1. **Introduction.** Infectious diseases have serious impacts on human health and society, the study of emerging and reemerging epidemics has a long history, and many mathematical models, both deterministic and stochastic, have been employed to study such impacts [14, 1] . The modeling study of infectious diseases can be traced back to the work in 1925 by Kermack and McKendrick [19, 15]. Since then, the deterministic models have been served as main and powerful tools to study the dynamics of the emerging and reemerging infection diseases. These deterministic models and their many descendants are known as "mean-field" or "compartmental" models because homogeneous mixing were assumed. The overwhelming majority of epidemic models are based on a compartmentalization of individuals or hosts according to the evolution and courses of the diseases. The basic or essential variables

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describing the number of related individuals include susceptible, infected and recovered in terms of a particular disease. For those infectious diseases with a longer course, the related models usually involve demographics, including natural death and birth, as well as other dynamical factors, such as immigration or migration [14, 1, 25, 9, 6].

In a typical compartmental model for disease transmission, the individuals of the population considered are usually divided into a number of compartments, commonly denoted by S (susceptible) and I (infective). These type of SIS models usually consist of a set of differential equations describing the change rate in the number of individuals in the compartments over time. If the total host population size is N, hence $N = S + I$, then the total population size can be increased by new births or immigration, and reduced by natural deaths or due to infection of the disease. The birth and/or death rate will depend on the host population size. If we use $B(N)$ to denote the recruitment rate including birth and immigration, and use $D(N)$ for the per capita natural death rate, respectively, and use α to denote the mortality rate due to the infection, then the model for the total population N can be written as

$$N'(t) = B(N) - D(N)N - \alpha I. \tag{1}$$

In the absence of infectious disease(s), in general the population size tends over time to a positive equilibrium of (1) which is usually decided by the carrying capacity of the environment or supporting resources, that is, the population size at which birth and death rates are balanced.

In homogeneous mixing models, one usually assumes that the contact transmission between susceptible and infective individuals occur randomly, and that the I, infective individuals, make $\tau C(N)I$ random contacts with the susceptibles that could result in disease transmission per unit of time, here $C(N)$ is the average number of contacts each individual makes per unit of time, τ is the transmission probability per contact. Because the contacts are random, only a fraction S/N of contacts are with a susceptible individual. Therefore the rate at which individuals transfer from the susceptible to the infective compartment is $\tau C(N)IS/N$, (assuming no vertical transmission), we then have the following mean field equations

$$\begin{cases} S' = B(N) - \tau C(N)\frac{IS}{N} - D(N)S + \gamma I, \\ I' = \tau C(N)\frac{IS}{N} - \alpha I - D(N)I - \gamma I. \end{cases} \tag{2}$$

For the well known SIS type of model (2), there have been large amount of work contributing to study the dynamics with different demographic assumptions and incidence functions, we refer to the books [14, 1, 19] for extensive references. For a population with fixed demographics, the incidence function is the key factor to determine the disease dynamics. The most commonly used incidence functions are bilinear or standard.

The bilinear incidence βSI, also know as mass action, $\beta SI = \beta N\frac{S}{N}I$ implies that the contact rate is βN which is linearly proportional to the total population size N. This would be a good approximation when the total population size N is not too large because the number of contacts made by an individual per unit of time should increase as the total population size N increases.

The standard incidence starts with the assumption that the contact rate $C(N)$ is a constant. Anderson and May [1] assume a classical mass action incidence $C(N) = \beta N$, whereas Busenberg and van den Driessche [6] and Gao and Hethcote [9] all assume the contact transmission rate $C(N)$ is a constant. However, the spread

of an infectious diseases in a population depends not only on the character of the disease, but also on the structure and mix of the population. Deterministic models of infectious disease transmission often assume homogeneous random mixing which implies that all individuals are equally likely to contact each other and, therefore, if infected, are equally likely to infect susceptible members of the population, which is rarely observed in reality. In large populations, individuals typically contact only a small, clustered, subpopulation. The local correlations that result from transmission in such structured networks are not well captured by standard mean-field models. There have been extensive studies to overcome this shortcoming, one important effort along this direction is the use of complex network models, and since then great progresses have been made in the last decade [23, 24, 20, 21, 22, 12, 10, 13, 16, 26, 17, 30, 5].

The early work in this direction focus more on different net structures (small-world networks and scale-free networks) or the impact of different degree distribution on the threshold values [23, 24, 20, 21]. In 2001, Romualdo Pastor-Satorras and Alessandro Vespignani [24] find the absence of an epidemic threshold and its associated critical behavior, which implies that scale-free networks are prone to the spreading and the persistence of infections at whatever spreading rate the epidemic agents possess. The stability of such networks models was studied in some recent publications [26, 5].

If the member of a population is considered to be the number of nodes in a network with their own degree (i.e. the number of potential contacts with other members in the population), an undirected network of size N with node degree distribution $p(k)$ $(k = 1, 2, ..., n)$ is then well defined. The value $\langle k\rangle = \sum_k kp(k)$ is the average number of contacts each node can make. In an SI or SIS model, each node in the network at any time can be either susceptible (S) or infectious (I). Let us assume that the whole population is divided into n distinct groups of sizes $N_k(k = 1, 2, ..., n)$ such that each individual in group k has exactly k contacts, here n denotes the maximum degree value of all nodes. For simplicity , we call the group of size N_k group k. If the population size is $N(N = \sum_k^n N_k)$, then the probability that a uniformly chosen individual has k contacts is $p(k) = N_k/N$. If S_k and I_k represent the number of susceptible and infectious individuals within the group k, respectively, then

$$S_k + I_k = N_k, \quad S(t) = \sum_k^n S_k(t), \quad I(t) = \sum_k^n I_k(t).$$

Let $\rho_k = I_k/N_k$ be the relative density of infected nodes with given degree k, then $1 - \rho_k$ is the relative density of susceptible nodes. The following dynamical mean-field model was proposed and studied in [23, 24, 12, 3]:

$$\rho_k'(t) = -\rho_k + \tau k(1 - \rho_k)\Theta(t), \tag{3}$$

where $\Theta(t)$ stands for the probability that an edge emanating from a node of degree k points to an infected node, $\Theta(t) = \sum_l p(l/k)\rho_l(t)$, where $p(l/k)$ is the probability that a node with k degree connects to a node with degree l. For uncorrelated networks [17, 5], $p(l/k) = lp(l)/\langle k\rangle$, which means that the probability that a node points to a node with degree l is proportional to its degree and the degree distribution $p(l)$. From the definition of $\Theta(t)$, it is independent of k for uncorrelated

networks [5] :

$$\Theta(t) = \frac{\sum_k kp(k)\rho_k}{\langle k\rangle}. \tag{4}$$

In model (3), $N_k = Np(k)$ is a constant for the given degree distribution $p(k)$ provided the total population size N remains a constant, hence (3) can be reduced to

$$I_k'(t) = -I_k + \tau k(N_k - I_k)\Theta(t), \tag{5}$$

where

$$\Theta(t) = \frac{\sum\limits_{k}^{n} kp(k)\rho_k}{\langle k\rangle} = \frac{\sum\limits_{k=1}^{n} kI_k}{\sum\limits_{k=1}^{n} k(S_k + I_k)}. \tag{6}$$

Networks can be classified as homogeneous and heterogeneous based on the variation of the degree distribution [3] . Homogeneous networks usually assume that the networks of contacts among individuals has very small degree fluctuations. In other words, the degree k fluctuates very little and we can assume that $k \simeq \langle k\rangle$. Typically, such distribution includes Delta and Poissonian degree distribution. For Delta degree distribution, each individual shares the same degree k, then, $\Theta(t) = I/N$, and the rate at which individuals transfer from the susceptible to the infective of (3) is reduced to a homogeneous mixing model like (2), and if $k = N$, the incidence is bilinear, but if $k \neq N$, the incidence is similar to the standard incidence. The main difference lies in the lack of demographics. For a Poissonian degree distribution, the network is composed of a set of N different vertices, in which each one of the $N(N-1)/2$ possible edges is present with probability p (the connection probability), and is absent with probability $1 - p$. This procedure results in a random network with average degree $\langle k\rangle = pN$, and for a Poisson degree distribution in the limit of large N and constant $\langle k\rangle$, we have

$$p(k) = e^{-\langle k\rangle}\frac{\langle k\rangle^k}{k!}.$$

Heterogeneous networks assume that the degree of vertices is highly fluctuant and the average degree is no longer a meaningful characterization of such networks, for example, the power law distribution $p(k) \sim k^{-\gamma}$ for large k is scale-free networks [2].

Though model (3) or (5) describes the transmission dynamics of disease in terms of incidence probability $\Theta(t)$, the variation of population size was ignored, it also requires that the total number of population N and degree distribution $p(k)$ to be fixed. In other words, model (3) or (5) works only for a fixed network with constant degree distribution. Yet, in real world applications, for the infection disease which run over a longer course, the impact of the demographics including new born or death as well as the migration will definitely change the degree distribution $p(k)$, hence the degree distribution becomes time dependent. As far as we know that the network models for disease transmission with such important character of changing degree distribution has not been well addressed or investigated in previous studies.

In this paper, we shall incorporate the demographics of a population into the modeling for disease transmission in a complex network. By using the models with demographics, we will study the impact of the demographics on the degree distribution of the population and study the dynamical behave of some concrete SIS models for infectious transmission on complex networks.

2. **Epidemic models in complex networks with demographics.** Without loss of generality, we start with an SIS model, by dividing the population into two classes, susceptible S and infected I. Let N be the total population, then $N = S + I$. As in the introduction, let $B(N)$ be the recruitment rate and $D(N)$ be the per capita natural death rate of the population.

We consider all the population and their contacts as a network. Each person in a community can be regarded as a vertex in the network, and each contact between two individuals is represented as an edge (line) connecting the two vertices. The number of edges emanating from a vertex, that is, the number of contacts a person has, is called the degree of the vertex. We classify the population into groups based on the number of contacts the individual can make per unit of time. Let N_k be the total number of individuals who has k contacts, per unit of time, $k = 1, 2, \cdots n$, n is the maximum number of contact each individual can make per unit of time ($1 \leq n \leq N$). Let $\overrightarrow{N} = (N_1, N_2, N_3 \cdots, N_n)$. If we let S_k and I_k be the susceptible and infected individuals at time t, then

$$N_k = S_k + I_k, N = \sum_{k=1}^{n} N_k, S = \sum_{k=1}^{n} S_k, I = \sum_{k=1}^{n} I_k. \tag{7}$$

Correspondingly, the degree distribution $p(k,t) = N_k(t)/N(t)$ becomes time dependent.

We make the following basic assumptions about the network upon which the infectious diseases models will be established:

(A1) The recruitment rate $B(N)$ is distributed into group k at the probability $r_k(0 \leq r_k < 1)$, hence $\sum_{k=1}^{n} r_k = 1$.

We assume that each new member entering the network is susceptible. The natural death rate is denoted by $D(N)N$.

(A2) The recovery rate for each group are the same, which we denote by γ. For a disease, we assume that the contact transmission rate between the infected and susceptible node is the same across the whole network we denote this rate by τ.

(A3) We must consider how a new member enters and chooses the k other vertices to which it contacts. Let us define the attachment probability $\Pi_a(k, \overrightarrow{N})$ that a given edge of a newly added vertex attaches to a given preexisting vertex of degree k, and we need also consider that edges removed of other vertex of degree k due to the deaths of individuals. Let $\Pi_d(k, \overrightarrow{N})$ be the respective link removal probability for a node with degree k. Contributions from processes in which a vertex gains or loses two or more edges in a single unit of time will be neglected. Assume that each node has at least 1 and at most n links, $n \leq N$. This implies that when a link is attached to a node with degree n, since any node cannot has a degree greater n, we suppose this link immediate are rewired to a preexisting vertex of degree k with $k < n$, namely, $\Pi_a(n, \overrightarrow{N}) = 0$. Similarly, the link of a node with degree 1 is rewired when its edge is removed because of the death of another individuals, and maintain degree 1, namely, $\Pi_d(1, \overrightarrow{N}) = 0$. In these two cases, we ignore other individual degrees changes.

The sum of $\Pi_a(k, \overrightarrow{N})$ across all nodes is 1 (see [18]), namely,

$$\sum_{k=1}^{n} \Pi_a(k, \overrightarrow{N}) N_k = 1.$$

Similarly, the sum of $\Pi_d(k, \overrightarrow{N})$ across all nodes is 1, i.e.,

$$\sum_{k=1}^{n} \Pi_d(k, \overrightarrow{N}) N_k = 1.$$

(A4) Assume that the network does not have correlations, then the conditional probability $p(j|k)$ that a given vertex with degree k is linked to a vertex with degree j by one edge is proportional to $jp(j)$, which is independent of its own vertex degree k (see [23, 24]), hence we have

$$p(j|k) = \frac{jp(j)I_j/N_j}{\langle k \rangle}, \tag{8}$$

where $\langle k \rangle = \sum_{k=1}^{n} kp(k)$. The probability that a link from a site points to another site with at least one infected individual becomes

$$\Theta(t) = \frac{\sum_{j=1}^{n} jp(j)I_j/N_j}{\langle k \rangle} = \frac{\sum_{j=1}^{n} jI_j}{\sum_{k=1}^{n} kN_k}., \tag{9}$$

Based on the above assumptions and discussions, we can have the following SIS model when $n \geq 2$,

$$
\begin{cases}
S_1' =r_1B(N) - D(N)S_1 - \tau S_1\Theta + \gamma I_1 - B(N)\sum_{i=1}^{n} ir_i\Pi_a(1,\overrightarrow{N})S_1 \\
\quad + D(N)\sum_{i=1}^{n} iN_i\Pi_d(2,\overrightarrow{N})S_2, \\
I_1' =\tau S_1\Theta - D(N)I_1 - \gamma I_1 \\
\quad - B(N)\sum_{i=1}^{n} ir_i\Pi_a(1,\overrightarrow{N})I_1 + D(N)\sum_{i=1}^{n} iN_i\Pi_d(2,\overrightarrow{N})I_2, \\
S_k' =r_kB(N) - D(N)S_k - \tau kS_k\Theta + \gamma I_k \\
\quad + B(N)\sum_{i=1}^{n} ir_i[\Pi_a(k-1,\overrightarrow{N})S_{k-1} - \Pi_a(k,\overrightarrow{N})S_k] \\
\quad + D(N)\sum_{i=1}^{n} iN_i[\Pi_d(k+1,\overrightarrow{N})S_{k+1} - \Pi_d(k,\overrightarrow{N})S_k], \\
I_k' =\tau kS_k\Theta - D(N)I_k - \gamma I_k \\
\quad + B(N)\sum_{i=1}^{n} ir_i[\Pi_a(k-1,\overrightarrow{N})I_{k-1} - \Pi_a(k,\overrightarrow{N})I_k] \\
\quad + D(N)\sum_{i=1}^{n} iN_i[\Pi_d(k+1,\overrightarrow{N})I_{k+1} - \Pi_d(k,\overrightarrow{N})I_k], \\
S_n' =r_nB(N) - D(N)S_n - \tau nS_n\Theta + \gamma I_n \\
\quad + B(N)\sum_{i=1}^{n} ir_i\Pi_a(n-1,\overrightarrow{N})S_{n-1} - D(N)\sum_{i=1}^{n} iN_i\Pi_d(n,\overrightarrow{N})S_n, \\
I_n' =\tau nS_n\Theta - D(N)I_n - \gamma I_n + B(N)\sum_{i=1}^{n} ir_i\Pi_a(n-1,\overrightarrow{N})I_{n-1} \\
\quad - D(N)\sum_{i=1}^{n} iN_i\Pi_d(n,\overrightarrow{N})I_n, \\
k =2,...,n-1.
\end{cases}
\tag{10}
$$

and if $n = 1$, one can establish the model (2). It follows from (10) immediately that we have the total number of individuals in group k satisfies the following equations

$$
\begin{cases}
\frac{dN_1}{dt} =B(N)r_1 - D(N)N_1 - B(N)\sum_{i=1}^{n} ir_i\Pi_a(1,\overrightarrow{N})N_1 \\
\quad + D(N)\sum_{i=1}^{n} iN_i\Pi_d(2,\overrightarrow{N})N_2, \\
\frac{dN_k}{dt} =B(N)r_k - D(N)N_k + B(N)\sum_{i=1}^{n} ir_i[\Pi_a(k-1,\overrightarrow{N})N_{k-1} - \Pi_a(k,\overrightarrow{N})N_k] \\
\quad + D(N)\sum_{i=1}^{n} iN_i[\Pi_d(k+1,\overrightarrow{N})N_{k+1} - \Pi_d(k,\overrightarrow{N})N_k], \\
\frac{dN_n}{dt} =B(N)r_n - D(N)N_n + B(N)\sum_{i=1}^{n} ir_i\Pi_a(n-1,\overrightarrow{N})N_{n-1} \\
\quad - D(N)\sum_{i=1}^{n} iN_i\Pi_d(n,\overrightarrow{N})N_n, \\
k =2,...,n-1.
\end{cases}
\tag{11}
$$

where the term $B(N)r_k$ in (11) represents the addition of a individual of degree k to the network. The terms $B(N)\sum_{i=1}^{n} ir_i\Pi_a(k-1,\overrightarrow{N})N_{k-1}$ and $-B(N)\sum_{i=1}^{n} ir_i\Pi_a(k,\overrightarrow{N})N_k$ describe the flow of vertices from degree $k-1$ to k and from k to $k+1$ as they gain extra edges when newly added vertices are attached. The terms $D(N)\sum_{i=1}^{n} iN_i\Pi_d(k+1,\overrightarrow{N})N_{k+1}$ and $-D(N)\sum_{i=1}^{n} iN_i\Pi_d(k,\overrightarrow{N})N_k$ describe the flow from degree $k+1$ to k and from k to $k-1$ as vertices lose edges when one of their neighbors is removed from the network due to deaths, $\sum_{i=1}^{n} iD(N)N_i$ represents lost the total number of sides in the network by deaths.

From (11), we know that the total number of individuals $N(t)$ satisfies

$$N'(t) = B(N) - D(N)N(t). \tag{12}$$

We are interested to explore the impact of demographics on the transmission of a disease over a complex network, the new recruitment and death will change the connection transmission probability when a new member is introduced or removed from the network. There have been different ways of modeling the recruitment rate in dynamical modeling for a single population. In the book [19], the following two types of recruitment functions $B(N)$ and death functions $D(N)$ are commonly used:

(B1) $B(N) = A$, $D(N) = \mu$, $A > 0, \mu > 0$;
(B2) $B(N) = N(b + \frac{\mu N}{K})$ and $D(N) = \mu + \frac{bN}{K}$, $b > 0, \mu > 0, K > 0$.

With the above combinations of $B(N)$ and $D(N)$, respectively, the total number of individuals of the network $N(t)$ will satisfy respectively one of the following two equations

$$N'(t) = A - \mu N(t), \tag{13}$$

$$N'(t) = (b-\mu)N(1-\frac{N}{K}). \tag{14}$$

Each of the above equation has a positive equilibrium which is globally asymptotically stable. Therefore we only focus and consider the case that the equation (12) has a unique globally asymptotically stable equilibrium N^*. In this case, the total population approaches asymptotically to a constant.

Since we are only interested in the asymptotic dynamics of the transmission of the diseases on the network, we can then rewrite (11) as the following

$$\begin{cases} \dfrac{dN_1}{dt} =& B(N^*)r_1 - D(N^*)N_1 \\ & - B(N^*)\sum\limits_{i=1}^{n} ir_i\Pi_a(1,\overrightarrow{N})N_1 + D(N^*)\sum\limits_{i=1}^{n} iN_i\Pi_d(2,\overrightarrow{N})N_2, \\ \dfrac{dN_k}{dt} =& B(N^*)r_k - D(N^*)N_k \\ & + B(N^*)\sum\limits_{i=1}^{n} ir_i[\Pi_a(k-1,\overrightarrow{N})N_{k-1} - \Pi_a(k,\overrightarrow{N})N_k] \\ & + D(N^*)\sum\limits_{i=1}^{n} iN_i[\Pi_d(k+1,\overrightarrow{N})N_{k+1} - \Pi_d(k,\overrightarrow{N})N_k], \\ \dfrac{dN_n}{dt} =& B(N^*)r_n - D(N^*)N_n + B(N^*)\sum\limits_{i=1}^{n} ir_i\Pi_a(n-1,\overrightarrow{N})N_{n-1} \\ & - D(N^*)\sum\limits_{i=1}^{n} iN_i\Pi_d(n,\overrightarrow{N})N_n, \\ k =& 2,...,n-1. \end{cases} \tag{15}$$

3. **Dynamics of network models with demographics.** The two different types of recruitment function would lead to different dynamics over the networks, due to the complexity of the systems. We consider the simplest SIS model and its dynamics under the assumption $(B1)$, i.e. the recruitment and per capita natural death rats are constants A and μ respectively. We assume a new node is recruited and it links to a randomly selected node, i.e., the probability that a node is selected for attachment is uniformly distributed (see [18, 4]). Hence

$$\Pi_a(k,\overrightarrow{N}) \approx \frac{1}{\sum\limits_{i=1}^{n} N_i} = \frac{1}{N}, \quad k = 1,2,\cdots,n-1.$$

In this internal network, we assume that (A4) holds. In this case, the probability that each edge of a individual is pointing to other vertex of degree k is proportional to the fraction of edges emanated from these vertices. Thus, we can take

$$\Pi_d(k,\overrightarrow{N}) \approx \frac{k}{\sum\limits_{k=1}^{n} kN_k}, \quad k = 2,\cdots,n.$$

Then model (10) becomes,

$$\begin{cases} S_1' =r_1A-\mu S_1-\tau S_1\Theta+\gamma I_1-\dfrac{A\sum\limits_{k=1}^{n}kr_k}{N}S_1+2\mu S_2,\\ I_1' =\tau S_1\Theta-\mu I_1-\gamma I_1-\dfrac{A\sum\limits_{k=1}^{n}kr_k}{N}I_1+2\mu I_2,\\ S_k' =r_kA-\mu S_k-\tau kS_k\Theta+\gamma I_k\\ \qquad +\dfrac{A\sum\limits_{k=1}^{n}kr_k}{N}[S_{k-1}-S_k]+\mu[(k+1)S_{k+1}-kS_k],\\ I_k' =\tau kS_k\Theta-\mu I_k-\gamma I_k+\dfrac{A\sum\limits_{k=1}^{n}kr_k}{N}[I_{k-1}-I_k]+\mu[(k+1)I_{k+1}-kI_k],\\ S_n' =r_nA-\mu S_n-\tau nS_n\Theta+\gamma I_n+\dfrac{A\sum\limits_{k=1}^{n}kr_k}{N}S_{n-1}-n\mu S_n,\\ I_n' =\tau nS_n\Theta-\mu I_n-\gamma I_n+\dfrac{A\sum\limits_{k=1}^{n}kr_k}{N}I_{n-1}-n\mu I_n. \end{cases} \tag{16}$$

The flow diagram of the transmission of system (16) is depicted in Figure 1.

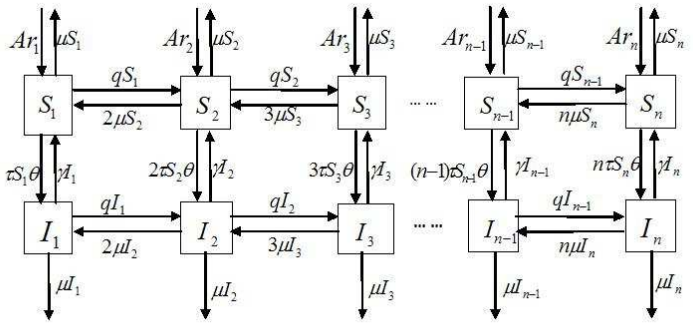


FIGURE 1. Flow diagram of the transmission dynamics in system (16), where $q=\frac{A}{N}\sum\limits_{k=1}^{n}kr_k$.

Correspondingly, model (11) for population in each group becomes,

$$\begin{cases} \frac{dN_1}{dt}=Ar_1-\frac{A}{N}\sum\limits_{i=1}^{n}ir_iN_1-\mu N_1+2\mu N_2,\\ \frac{dN_k}{dt}=Ar_k+\frac{A}{N}\sum\limits_{i=1}^{n}ir_i[N_{k-1}-N_k]-\mu N_k+\mu[(k+1)N_{k+1}-kN_k],\\ \frac{dN_n}{dt}=Ar_n+\frac{A}{N}\sum\limits_{i=1}^{n}ir_iN_{n-1}-\mu N_n-n\mu N_n,\\ k=2,\cdots,n-1. \end{cases} \tag{17}$$

Note that the total number of individuals $N(t)$ satisfies (13), which has a unique globally asymptotically stable equilibrium $N^* = A/\mu$. Since we are only interested in the asymptotic behavior of system (17), we can reduce (17) to following the equations:

$$\begin{cases} \frac{dN_1}{dt} = Ar_1 - \mu c N_1 - \mu N_1 + 2\mu N_2, \\ \frac{dN_k}{dt} = Ar_k + \mu c[N_{k-1} - N_k] - \mu N_k + \mu[(k+1)N_{k+1} - kN_k], \\ \frac{dN_n}{dt} = Ar_n + \mu c N_{n-1} - \mu N_n - n\mu N_n, \\ k = 2, \cdots, n-1. \end{cases} \tag{18}$$

where $c = \sum_{i=1}^{n} ir_i$.

In order to study the transmission dynamics of system (16), we start with system (18).

Let

$$J = -\begin{pmatrix} -(c+1)\mu & 2\mu & & & & & \\ c\mu & -(c+3)\mu & 3\mu & & & & \\ & c\mu & -(c+4)\mu & 4\mu & & & \\ & & \ddots & \ddots & \ddots & & \\ & & & c\mu & -(c+n)\mu & n\mu & \\ & & & & c\mu & -(1+n)\mu & \end{pmatrix}.$$

Let $T = \mathrm{diag}(\delta_1, \delta_2, \cdots, \delta_n)$, $\delta_1 = 1$, $\delta_i = \sqrt{\frac{c^{i-1}}{i!}}, 2 \le i \le n$, and $d_i = \mu\sqrt{c(1+i)}$, then one can verify that

$$T^{-1}JT = J^* = -\begin{pmatrix} -(c+1)\mu & d_1 & & & & & \\ d_1 & -(c+3)\mu & d_2 & & & & \\ & d_2 & -(c+4)\mu & d_3 & & & \\ & & \ddots & \ddots & \ddots & & \\ & & & d_{n-2} & -(c+n)\mu & d_{n-1} & \\ & & & & d_{n-1} & -(1+n)\mu & \end{pmatrix}.$$

Obviously, J and J^* have same eigenvalue, and J^* is real symmetry matrix. Since, a real symmetric matrix has real eigenvalues, we use Gerschgorin circles theorem for J to estimate its eigenvalues. Every eigenvalue of J lies within at least one of the Gershgorin discs:

$$D_1 = \{\lambda : -(2c+1)\mu \le \lambda \le -\mu\},$$

$$D_2 = \{\lambda : -\mu(2c+2k-1) \le \lambda \le -\mu, k = 2, 3, \cdots, n-1\},$$

$$D_3 = \{\lambda : -(2n+1)\mu \le \lambda \le -\mu\}.$$

By Theorem 1.2 in [27], we know that the system (18) has a unique endemic equilibrium $P_0 = (N_1^*, N_2^*, \cdots, N_n^*)$ and $\sum_{i=1}^{n} N_i^* = A/\mu$. If we denote $\overrightarrow{N}^* = (N_1^*, N_2^*, \cdots, N_n^*)^T$, $\overrightarrow{R} = (Ar_1, Ar_2, \cdots, Ar_n)^T$, then

$$\overrightarrow{N}^* = J^{-1}\overrightarrow{R}.$$

Thus, we have the following theorem.

Theorem 3.1. *System (18) has a unique equilibrium $P_0 = (N_1^*, N_2^*, \cdots, N_n^*)$, and it is globally asymptotically stable, i.e., $\lim_{t\to\infty} N_k(t) = N_k^*$.*

By Theorem 3.1, we will study the limiting system of (16):

$$\begin{cases} S_1' = r_1 A - \beta_1 S_1 \sum\limits_{i=1}^{n} iI_i + \mu(1+c)S_1 + \gamma I_1 + 2\mu S_2, \\ I_1' = \beta_1 S_1 \sum\limits_{i=1}^{n} iI_i - (\mu + \mu c + \gamma)I_1 + 2\mu I_2, \\ S_k' = r_k A - \beta_k S_k \sum\limits_{i=1}^{n} iI_i - \mu(1+k+c)S_k + \gamma I_k + \mu c S_{k-1} + \mu(k+1)S_{k+1}, \\ I_k' = \beta_k S_k \sum\limits_{i=1}^{n} iI_i - [\mu(1+k+c)+\gamma]I_k + \mu c I_{k-1} + \mu(k+1)I_{k+1}, \\ S_n' = r_n A - \beta_n S_n \sum\limits_{i=1}^{n} iI_i - \mu(1+n)S_n + \gamma I_n + \mu c S_{n-1}, \\ I_n' = \beta_n S_n \sum\limits_{i=1}^{n} iI_i - [\mu(1+n)+\gamma]I_n + \mu c I_{n-1} - \mu I_n, \\ k = 2, \cdots, n-1. \end{cases} \tag{19}$$

where $\beta_k = \frac{\tau k}{\sum\limits_{k=1}^{n} kN_k^*}$.

It follows from Theorem 3.1 that system (19) has a unique disease-free equilibrium $E_0 = (N_1^*, N_2^*, \cdots, N_n^*, 0, 0, \cdots, 0)$.

Note that only the compartments $I_k (k = 1, 2, \ldots, n)$ are involved in the calculation of the basic reproduction number R_0. We define $\mu + \mu c + \gamma = \theta$ and let

$$M = \begin{pmatrix} \sigma_1 - \theta & 2\sigma_1 + 2\mu & 3\sigma_1 & \cdots & k\sigma_1 & \cdots & n\sigma_1 \\ \sigma_2 + \mu c & 2\sigma_2 - \theta - 2\mu & 3\sigma_2 + 3\mu & \cdots & k\sigma_2 & \cdots & n\sigma_2 \\ \vdots & \vdots & \vdots & & \vdots & \vdots & \vdots \\ \sigma_k & 2\sigma_k & 3\sigma_k & \cdots & k\sigma_k - \theta - k\mu & \cdots & n\sigma_k \\ \vdots & \vdots & \vdots & & \vdots & \vdots & \vdots \\ \sigma_n & 2\sigma_n & 3\sigma_n & \cdots & k\sigma_n & \cdots & n\sigma_n - \gamma - (n+1)\mu \end{pmatrix},$$

where, $\sigma_i = \beta_i S_i^*$. Obviously, M is irreducible and has non-negative off-diagonal elements. Let

$$M_1 = \begin{pmatrix} \beta_1 & 2\beta_1 & \cdots & k\beta_1 & \cdots & n\beta_1 \\ \beta_2 & 2\beta_2 & \cdots & k\beta_2 & \cdots & n\beta_2 \\ \vdots & \vdots & & \vdots & \vdots & \vdots \\ \beta_k & 2\beta_k & \cdots & k\beta_k & \cdots & n\beta_k \\ \vdots & \vdots & & \vdots & \vdots & \vdots \\ \beta_n & 2\beta_n & \cdots & k\beta_n & \cdots & n\beta_n \end{pmatrix}, F = \begin{pmatrix} \sigma_1 & 2\sigma_1 & \cdots & k\sigma_1 & \cdots & n\sigma_1 \\ \sigma_2 & 2\sigma_2 & \cdots & k\sigma_2 & \cdots & n\sigma_2 \\ \vdots & \vdots & & \vdots & \vdots & \vdots \\ \sigma_k & 2\sigma_k & \cdots & k\sigma_k & \cdots & n\sigma_k \\ \vdots & \vdots & & \vdots & \vdots & \vdots \\ \sigma_n & 2\sigma_n & \cdots & k\sigma_n & \cdots & n\sigma_n \end{pmatrix},$$

$$V = -\begin{pmatrix} -\theta & 2\mu & 0 & 0 & 0 & 0 & 0 \\ \mu c & -\theta - 2\mu & 3\mu & 0 & 0 & 0 & 0 \\ 0 & \mu c & -\theta - 3\mu & 4\mu & 0 & 0 & 0 \\ \vdots & \vdots & \vdots & \vdots & \vdots & \vdots & \vdots \\ 0 & 0 & \cdots & \mu c & -\theta - k\mu & (k+1)\mu & 0 \\ \vdots & \vdots & \vdots & \vdots & \vdots & \vdots & \vdots \\ 0 & 0 & \cdots & 0 & 0 & \mu c & -\gamma - (n+1)\mu \end{pmatrix}.$$

Following the notations in van den Driessche and Watmough [29] and the concepts of next generation matrix, one can compute to get the basic reproduction number

$R_0 = \rho(FV^{-1})$, where ρ represents the spectral radius of the a matrix, and $R_0 < 1 \Leftrightarrow s(M) < 0$, $R_0 > 1 \Leftrightarrow s(M) > 0$, where

$$s(M) := \max\{Re\lambda : \lambda \text{ is an eigenvalue of } \quad M\}.$$

To calculate the expression for R_0, first, we will transforms the tridiagonal matrix $V = (v_{ij})_{n\times n}$ to a symmetric tridiagonal matrix.

Let

$T = \text{diag}(\delta_1, \delta_2, \cdots, \delta_n)$, $\delta_1 = 1$, $\delta_i = \sqrt{\frac{c^{i-1}}{i!}}, 2 \le i \le n$, and $d_i = \mu\sqrt{c(1+i)}$, then if we denote $V^* = T^{-1}VT = (h_{ij})_{n\times n}$, one can verify to yield that

$$V^* = -\begin{pmatrix} -\theta & d_1 & & & & & \\ d_1 & -\theta - 2\mu & d_2 & & & & \\ & d_2 & -\theta - 3\mu & d_3 & & & \\ & & \ddots & \ddots & \ddots & & \\ & & & d_{n-2} & -\theta - (n-1)\mu & d_{n-1} & \\ & & & & d_{n-1} & -\gamma - (n+1)\mu & \end{pmatrix},$$

where $v_{ij} = \frac{\delta_i}{\delta_j} h_{ij}$. Note that the sums of each column of matrix are the same $\mu+\gamma$, so V is an irreducible nonsingular M-matrix. Thus V^{-1} is a positive matrix. Using the methods in [8], for $V^* = (h_{ij})_{n\times n}$, one can obtain

$$h_{ij} = \begin{cases} \frac{|V^*_{i-1}|}{|V^*_i|} + |V^*_{i-1}|^2 \sum\limits_{k=i+1}^{n} \left(\frac{\prod\limits_{l=i}^{k-1} d_l^2}{|V^*_{k-1}||V^*_k|}\right), & i = j, \\ (-1)^{i+j} \frac{|V^*_{j-1}|}{|V^*_{i-1}|} \prod\limits_{l=j}^{i-1} d_l h_{ii}, & i \ne j, \end{cases}$$

where $|V^*_0| = 1$ and $|V^*_i| (1 \le i \le n)$ is the ith order principal minor determinant of V^*.

Now we can give the expression of R_0. Note F is a matrix of rank 1, so $R_0 = \rho(FV^{-1})$ is just the trace of FV^{-1}, i.e.,

$$R_0 = \rho(FV^{-1}) = \sum_{j=1}^{n} \sigma_j \left(\sum_{i=1}^{n} i v_{ji}\right) = \sum_{j=1}^{n} \sigma_j \delta_j \left(\sum_{i=1}^{n} \frac{i h_{ji}}{\delta_i}\right).$$

In general, it is difficult to give the implicit form of R_0 due to the fact of implicit form for the equilibrium. For the particular case of $n = 2$, one can calculate to get $S_1^* = \frac{A(r_1+2)}{\mu(3+c)}$, $S_2^* = \frac{A(1+2r_2)}{\mu(3+c)}$, and $\sigma_1 = \frac{\tau(r_1+2)}{r_1+4(1+r_2)}$, $\sigma_2 = \frac{2\tau(1+2r_2)}{r_1+4(1+r_2)}$, therefore to obtain the basic reproduction number

$$R_0 = \frac{\tau[(r_1+2)(\gamma+3\mu+2\mu c) + 4(1+2r_2)(\gamma+2\mu+\mu c)]}{[r_1+4(1+r_2)][\gamma(\gamma+4\mu+\mu c) + \mu^2(3+c)]},$$

where $c = r_1 + 2r_2$.

Remark 1. When $A \to 0, \mu \to 0$, it corresponds to the case with no demographics for the network models and the system (16) becomes the system (3), i.e., the network will be reduced from dynamic into static. By the references [23, 24], we know the basic reproduction number for the static network has the form of

$$R_0^* = \frac{\tau}{\gamma} \frac{\langle k^2 \rangle}{\langle k \rangle},$$

which depends only on the degree distribution. The degree distribution needs to be given in advance, and for different degree distribution, R_0^* has different values. However, for the network model with recruitment and deaths, the basic reproduction number R_0 depends on r_k, μ, γ and τ (see the case $n = 2$ for example) but does not degree distribution. In fact, the degree distribution function $p(k,t)$ varies as time increases. When $t \to \infty$, $\lim_{t\to\infty} p(k,t) = \lim_{t\to\infty} N_k/N = \mu N_k^*/A$. That is, our results are different from those in static network [23, 24, 20, 21, 22, 12, 13, 26, 5, 2, 4], therefore, demographics (including recruitment and death) contributes to the basic reproduction number, which is important to decide the endemics of the disease over the network.

Theorem 3.2. *For system (19), if $R_0 \le 1$, then the disease-free equilibrium $E_0 = (S_1^*, \cdots S_n^*, 0, \cdots 0)$ is globally attractive, where $S_k^* = N_k^*$ (k=1, 2, ..., n).*

Proof. We only need to prove that $\lim_{t\to\infty} I_k(t) = 0$.

By Theorem 3.1, we have that for any $\varepsilon > 0$, if t is sufficiently large,

$$N_k(t) = S_k(t) + I_k(t) < N_k^* + \epsilon, \qquad k = 1, 2, \cdots, n$$

holds. Thus, from (19), we have

$$\begin{cases} I_1' \le \beta_1(S_1^* + \epsilon)\sum_{i=1}^{n} iI_i - (\mu + \mu c + \gamma)I_1 + 2\mu I_2, \\ I_k' \le \beta_k(S_k^* + \epsilon)\sum_{i=1}^{n} iI_i - [\mu(1 + k + c) + \gamma]I_k + \mu c I_{k-1} + \mu(k+1)I_{k+1}, \\ I_n' \le \beta_n(S_n^* + \epsilon)\sum_{i=1}^{n} iI_i - [\mu(1 + n) + \gamma]I_n + \mu c I_{n-1} - \mu I_n, \\ k = 2, \cdots, n-1. \end{cases}$$

It then suffices to show that for the following system

$$\begin{cases} I_1' = \beta_1(S_1^* + \epsilon)\sum_{i=1}^{n} iI_i - (\mu + \mu c + \gamma)I_1 + 2\mu I_2, \\ I_k' = \beta_k(S_k^* + \epsilon)\sum_{i=1}^{n} iI_i - [\mu(1 + k + c + \gamma)I_k + \mu c I_{k-1} + \mu(k+1)I_{k+1}, \\ I_n' = \beta_n(S_n^* + \epsilon)\sum_{i=1}^{n} iI_i - [\mu(1 + n) + \gamma]I_n + \mu c I_{n-1} - \mu I_n, \\ k = 2, \cdots, n-1. \end{cases} \tag{20}$$

all the solutions tend to zero.

Since $s(M) < 0$ and $s(M + \varepsilon M_1)$ is continuous for small ε, we can fix an $\varepsilon > 0$ small enough such that $s(M + \varepsilon M_1) < 0$. As a consequence, solutions of equation (20) tend to zero as t goes to infinity. Then by the comparison theorem, $\lim_{t\to\infty} I_i(t) = 0$.

Next, we will show that when $R_0 > 1$, system (19) admits a unique endemic equilibrium which is globally attractive.

It follows from (16) that we can rewrite system (19) in an equivalent form:

$$\begin{cases} \frac{dN_1}{dt} = Ar_1 - \mu cN_1 - \mu N_1 + 2\mu N_2, \\ \frac{dN_k}{dt} = Ar_k + \mu c[N_{k-1} - N_k] - \mu N_k + \mu[(k+1)N_{k+1} - kN_k], \\ \frac{dN_n}{dt} = Ar_n + \mu cN_{n-1} - \mu N_n - n\mu N_n, \\ I_1' = \beta_1(N_1 - I_1)\sum\limits_{i=1}^{n} iI_i - (\mu + \mu c + \gamma)I_1 + 2\mu I_2, \\ I_k' = \beta_k(N_k - I_k)\sum\limits_{i=1}^{n} iI_i - [\mu(1+k+c) + \gamma]I_k + \mu cI_{k-1} + \mu(k+1)I_{k+1}, \\ I_n' = \beta_n(N_n - I_n)\sum\limits_{i=1}^{n} iI_i - [\mu(1+n) + \gamma]I_n + \mu cI_{n-1} - \mu I_n. \\ k = 2, \cdots, n-1. \end{cases} \tag{21}$$

To investigate the global dynamics of (21), we first show that (21) admits a compact and positively invariant set. □

Lemma 3.3. *For system (21), every forward orbit in R_+^{2n} eventually enters into*

$$G := \{(\overrightarrow{N}, \overrightarrow{I}) \in R^{2n} : \sum_{k=1}^{n} N_k \le \frac{A}{\mu}, 0 \le I_k \le N_k\},$$

where $\overrightarrow{N} = (N_1, N_2, \cdots, N_n), \overrightarrow{I} = (I_1, I_2, \cdots, I_n)$ and G is positively invariant for (21).

Note $N(t) = \sum\limits_{k=1}^{n} N_k$ satisfies (13), then Lemma 3.3 follows from straightforward calculations.

For (21), clearly, the first n equations are independent of the last n equations. By Theorem 3.1, we have $\lim\limits_{t\to\infty} N_k(t) = N_k^*$. Then (21) has the following limiting system:

$$\begin{cases} I_1' = \beta_1(N_1^* - I_1)\sum\limits_{i=1}^{n} iI_i - (\mu + \mu c + \gamma)I_1 + 2\mu I_2, \\ I_k' = \beta_k(N_k^* - I_k)\sum\limits_{i=1}^{n} iI_i - [\mu(1+k+c) + \gamma]I_k + \mu cI_{k-1} + \mu(k+1)I_{k+1}, \\ I_n' = \beta_n(N_n^* - I_n)\sum\limits_{i=1}^{n} iI_i - [\mu(1+n) + \gamma]I_n + \mu cI_{n-1} - \mu I_n, \\ k = 2, \cdots, n-1. \end{cases} \tag{22}$$

Lemma 3.4. *For system (22), the set*

$$G_1 := \{(I_1, I_2, \cdots, I_n) = \overrightarrow{I} \in R_+^n : 0 \le I_k \le N_k^*, k = 1, 2, \cdots, n\}$$

is positively invariant.

Proof. First we will show that $I_k(t) > 0$ for any $t > 0$ and $k = 1, 2, \cdots, n$, and initial value $\overrightarrow{I(0)} \in G_1$. Otherwise assume that exist a $k_0 \in \{1, 2, \cdots, n\}$ and $t_0 > 0$, such that $I_{k_0}(t_0) = 0$. Let $t^* = \inf\{t > 0, I_{k_0}(t) = 0\}$, it follow that

$$I_{k_0}'(t^*) = \beta_{k_0} N_{k_0}^* \sum_{i=1}^{n} iI_i(t^*) + \mu cI_{k_0-1}(t^*) + \mu(k_0+1)I_{k_0+1}(t^*),$$

then $I_{k_0}'(t^*) > 0$, but the definition of t^* implies $I_{k_0}'(t^*) \le 0$, this is a contradiction.

Second, we show that for any $t \geq 0$, $I_k(t) \leq N_k^*$ and $k = 1, 2, \cdots, n$.

For any initial value $\overrightarrow{I(0)} \in G_1$, let $x_k(t) = N_k^* - I_k(t)$. It follows from (22) that we have the following system:

$$\begin{cases} x_1' = -\beta_1 x_1 \sum_{i=1}^{n} i(N_i^* - x_i) + (\mu + \mu c + \gamma)(N_1^* - x_1) - 2\mu(N_2^* - x_2), \\ x_k' = -\beta_k x_k \sum_{i=1}^{n} i(N_i^* - x_i) + [\mu(1+k+c) + \gamma](N_k^* - x_k) \\ \qquad - \mu c(N_{k-1}^* - x_{k-1}) - \mu(k+1)(N_{k+1}^* - x_{k+1}), \\ x_n' = -\beta_n x_n \sum_{i=1}^{n} i(N_i^* - x_i) + [\mu(1+n) + \gamma](N_n^* - x_n) - \mu c(N_{n-1}^* - x_{n-1}), \\ k = 2, \cdots, n-1. \end{cases} \tag{23}$$

We will show that for any $t > 0$, $x_k(t) > 0$ and $k = 1, 2, \cdots, n$. If this were not true, there would exist a $k_0 (1 \leq k_0 \leq n)$ and $t_0 > 0$ such that $x_{k_0}(t_0) = 0$. Let $t^{**} = \inf\{t > 0, x_{k_0}(t) = 0\}$, it follows that

$$x_{k_0}'(t^{**}) = [\mu(1+k_0+c)+\gamma]N_{k_0}^* - \mu c N_{k_0-1}^* - \mu(k_0+1)N_{k_0+1}^* + \mu c x_{k_0-1}(t^{**}) + \mu c(k_0+1)x_{k_0+1}(t^{**}).$$

Since

$$[\mu(1+k_0+c)+\gamma]N_{k_0}^* - \mu c N_{k_0-1}^* - \mu(k_0+1)N_{k_0+1}^* = Ar_{k_0} > 0,$$

thus $x_{k_0}'(t^{**}) > 0$, but the definition of t^{**} implies $x_{k_0}'(t^{**}) \leq 0$, this is also a contradiction. Thus, it follows that $I_{k_0}(t) \leq N_{k_0}^*$. □

Theorem 3.5. *For the case when $R_0 > 1$, system (22) admits a unique endemic equilibrium $E_I^* = (I_1^*, \cdots, I_n^*)$ which is globally asymptotically stable with respect to $\overrightarrow{I}(0) \in G_1$.*

Proof. We will use the theory of cooperate system to prove the global stability, therefore we only need verify the assumption in Corollary 3.2 [33] for system (22).

In fact, let $\overrightarrow{f} : G_1 \to G_1$ be defined by the right-hand side of (22), $\overrightarrow{f} = (f_1, \cdots, f_n)$. Clearly $\overrightarrow{f}$ is continuously differentiable, $\overrightarrow{f}(0) = 0, f_i(\overrightarrow{I}) \geq 0$, for all $\overrightarrow{I} \in G_1$ with $I_i = 0$, and $\partial f_i/\partial I_j \geq 0, i \neq j$ for $\overrightarrow{I} \in G_1$, so $\overrightarrow{f}$ is cooperative. Clearly $D\overrightarrow{f} = (\partial f_i/\partial x_j)_{1 \leq i,j \leq n}$ is irreducible for every $\overrightarrow{I} \in G_1$. For every $\alpha \in (0,1)$ and $I_k > 0$.

Note that for $\forall \alpha \in (0,1)$ and $I_k > 0$,

$$\begin{aligned} f_k(\alpha \vec{I}) =& \alpha[(\beta_k N_k^* - \alpha I_k)\sum_{i=1}^{n} iI_i - [\mu(1+k+c)+\gamma]I_k + \mu c I_{k-1} + \mu(k+1)I_{k+1}] \\ \geq& \alpha[(\beta_k N_k^* - I_k)\sum_{i=1}^{n} iI_i - [\mu(1+k+c)+\gamma]I_k + \mu c I_{k-1} + \mu(k+1)I_{k+1}] \\ =& \alpha f_k(\overrightarrow{I}), \end{aligned}$$

thus $\overrightarrow{f}$ is strongly sublinear on G_1. By Lemma 2 and Corollary 3.2 [33] , one can conclude that (22) admits a unique endemic equilibrium $E_I^* = (I_1^*, \cdots, I_n^*)$ which is globally asymptotically stable with respect to $\overrightarrow{I}(0) \in G_1$. □

Next, by a similar proof as in Theorem 3.1 [11], we will prove the following theorem.

Theorem 3.6. *If $R_0 > 1$, then system (21) has a unique endemic equilibrium*

$$E^* = (N_1^*, \cdots, N_n^*, I_1^*, \cdots, I_n^*)$$

which is globally asymptotically stable with respect to $(\overrightarrow{N}(0), \overrightarrow{I}(0)) \in G$.

Proof. Let $\Phi(t) : R_+^{2n} \to R_+^{2n}$ be the solution semiflow of system (21), ω be omega limit set of $\Phi(\overrightarrow{N}(0), \overrightarrow{I}(0)), (\overrightarrow{N}(0), \overrightarrow{I}(0)) \in G$. By Lemma 1 and Lemma 1.2.1 in [32], ω is an internal chain transitive set for $\Phi(t)$. Obviously, for system (21) there are only two equilibria E_0 and E^* when $R_0 > 1$. By Theorem 3.1 and Theorem 3.5, it is not difficult to verify that $\Phi(t)$ satisfies the condition of Theorem 1.2.2 in [32] , thus, ω should be either E_0 or E^*.

Next, we prove that $\omega = \{E^*\}$. If this were not true, then $\omega = \{E_0\}$, then we should have $\lim\limits_{t\to+\infty} N_i = N_i^*$, $\lim\limits_{t\to+\infty} I_i = 0 (i = 1, 2, \cdots, n)$. Since $s(M) > 0$, we can choose a small $\varepsilon > 0$ such that $s(M - \varepsilon M_2) > 0$, where

$$M_2 = \begin{pmatrix} 1 & 2\times 1 & \cdots & k\times 1 & \cdots & n\times 1 \\ 2 & 2\times 2 & \cdots & k\times 2 & \cdots & n\times 2 \\ \vdots & \vdots & & \vdots & \vdots & \vdots \\ k & 2\times k & \cdots & k\times k & \cdots & n\times k \\ \vdots & \vdots & & \vdots & \vdots & \vdots \\ n & 2\times n & \cdots & k\times n & \cdots & n\times n \end{pmatrix}.$$

It follows that there exists a $\bar{t}$ such that $\beta_i(N_i(t) - I_i(t)) > \beta_i N_i^* - \varepsilon$, for $t > \bar{t}, i = 1, 2, \cdots, n$. Thus, we have

$$\begin{cases} I_1' > (\beta_1 S_k^* - \epsilon) \sum\limits_{i=1}^{n} iI_i - (\mu + \mu c + \gamma) I_1 + 2\mu I_2, \\ I_k' > (\beta_k S_k^* - \epsilon) \sum\limits_{i=1}^{n} iI_i - [\mu(1 + k + c) + \gamma] I_k + \mu c I_{k-1} + \mu(k+1) I_{k+1}, \\ I_n' > (\beta_n S_n^* - \epsilon) \sum\limits_{i=1}^{n} iI_i - [\mu(1 + n) + \gamma] I_n + \mu c I_{n-1} - \mu I_n, \\ k = 2, \cdots, n-1. \end{cases}$$

Let $\overrightarrow{v} = (v_1, \cdots, v_n)$ be a positive eigenvector of $M - \varepsilon M_2$ associated with $s(M - \varepsilon M_2)$. Choose an small number α such that $\overrightarrow{I} \geq \alpha \overrightarrow{v}$. Then by the comparison theorem,

$$\overrightarrow{I} \geq \alpha \exp[s(M - \varepsilon M_2)(t - \bar{t})] \overrightarrow{v}, \quad t \geq \bar{t},$$

and hence $I_i(t) \to +\infty (i = 1, 2, \cdots, n)$ which contradicts to $I_i(t) \to 0$. Consequently the unique endemic equilibrium E^* is globally attractive. □

By Theorems 3.2, 3.6 and Lemma 3.3, and limit system theory (see [28, 7]), for model (16), we have the following theorem.

Theorem 3.7. *When $R_0 < 1$, the disease-free equilibrium $E_0 = (N_1^*, \cdots, N_n^*, 0, \cdots, 0)$ is globally asymptotically stable in G. If $R_0 > 1$, the disease-free equilibrium is unstable and there is a unique endemic equilibrium*

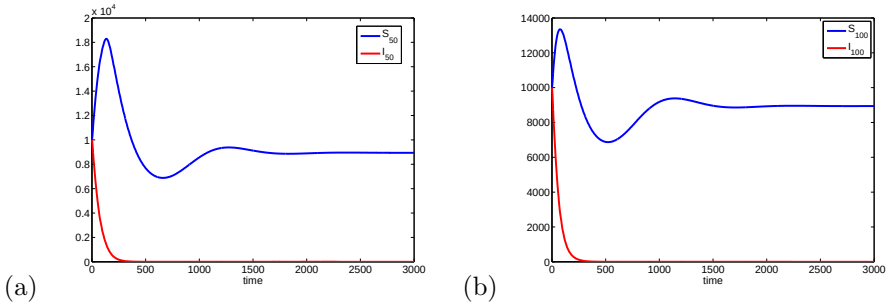
$$E^* = (S_1^*, \cdots, S_n^*, I_1^*, \cdots, I_n^*)$$

which is globally asymptotically stable with respect to $(\overrightarrow{N}(0), \overrightarrow{I}(0)) \in G$.

4. **Numerical simulations.** In this section, we will perform a series of numerical simulations to verify the mathematical analysis. For simulation purpose, we consider a network with constant recruitment rate $A = 5000$ and an initial size as $S_k(0) = I_k(0) = 10000$, and other parameters are chosen as: $\mu = 0.006$, $\gamma = 0.02$ (also see them in Table 1). The probability r_k of an individual entering group k, take two forms: poisson or scale-free distributions.

TABLE 1. Description of parameters in system (16).

Parameter	Value	Comments
A	5×10^3	recruitment rate of populations
μ	6×10^{-3}	natural death rate of populations
γ	2×10^{-2}	recovered rate of the infectives
τ	$0 \sim 1$	contact transmission rate between the infected and susceptible node
λ	4	exponent of poisson distribution
n	100	maximal degree



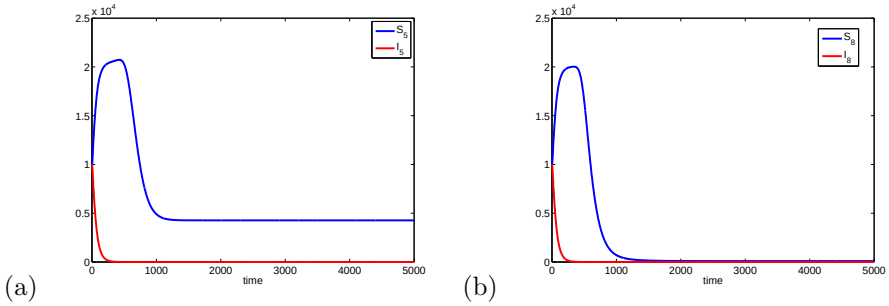
(a) (b)

FIGURE 2. (a) The numbers of the S_{50} and I_{50}; (b) The numbers of the S_{100} and I_{100}. Here, $\tau = 0.0000001$, $\lambda = 4$ and $R_0 \approx 7 \times 10^{-4} < 1$.

Firstly, we give the case that the basic reproduction number $R_0 < 1$ with poisson distribution $r_k = \frac{e^{-\lambda}\lambda^k}{k!} / \Sigma_{k=1}^{n} \frac{e^{-\lambda}\lambda^k}{k!}$ with $\lambda = 4$. Fig. 2 shows the numbers of the susceptibles and infectives with $k = 50$ and 100, respectively. As shown in Fig. 2, the disease will die out in both cases. If we consider $r_k = k^{-4}/\Sigma_{k=1}^{n} k^{-4}$, one can calculate to get $R_0 \approx 2 \times 10^{-3} < 1$. As shown in Fig. 3, the disease will also extinct for the population.

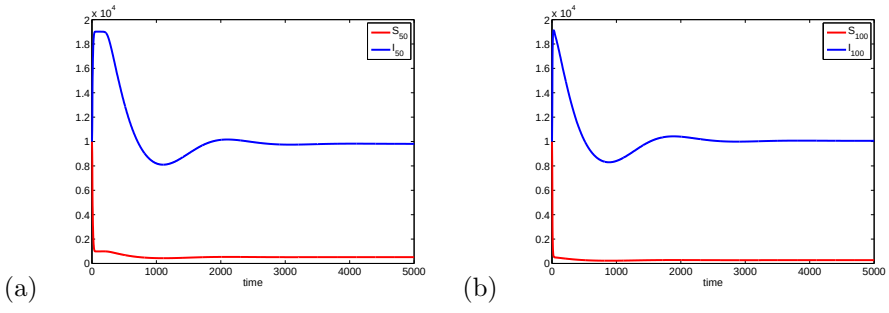
Now, we consider the case when $R_0 > 1$. We still use $r_k = \frac{e^{-4}4^k}{k!} / \Sigma_{k=1}^{n} \frac{e^{-4}4^k}{k!}$. In Fig. 4, we show the time series of S_{50}, I_{50}, S_{100} and I_{100} with $R_0 \approx 11$. One can see that the disease will persist and converge to a positive stationary state. If we change r_k as $k^{-4}/\Sigma_{k=1}^{n} k^{-4}$, we have $R_0 \approx 5 > 1$. Similarly, as shown In Fig. 5, the disease will also converge to a positive stationary state, which means that the endemic state is stable.

In Fig. 6, we show the degree distribution with respect to k and time. One can see that, when time is large enough, the degree distribution is stationary. However, for a short period, the degree distribution varied with time. In other words, the network in our model is dynamics not static. In Fig. 7, we give the time series



(a) (b)

FIGURE 3. (a) The number of the S_5 and I_5; (b) The number of the S_8 and I_8. Here, $\tau = 0.0000001$, $r_k = k^{-4}/\Sigma_{k=1}^{n} k^{-4}$ and $R_0 \approx 2 \times 10^{-3} < 1$.



(a) (b) (c)

FIGURE 4. (a) The number of the S_{50} and I_{50}; (b) The number of the S_{100} and I_{100}; (c) The total number of the infective. Here, $\tau = 0.008$ and $R_0 \approx 11 > 1$.

of $p(5)$ and $p(10)$ for two kinds of r_k and if time is small, $p(5)$ and $p(10)$ change as time increases. Furthermore, we found that the stationary degree distribution depends on the form of the probability r_k. If $r_k = \frac{e^{-4}4^k}{k!}/\Sigma_{k=1}^{n}\frac{e^{-4}4^k}{k!}$, then the stationary degree distribution is poisson distributions; if $r_k = k^{-4}/\Sigma_{k=1}^{n}k^{-4}$, then the stationary degree distribution is scale-free distributions, which can be seen from Fig. 8.

From the theoretical analysis, one can see that the basic reproduction number is a quantity to characterize the dynamics of network models. In order to show the impact of demographics, we look at basic reproduction number with respect to the parameters μ (death rate), λ (exponent of $r_k = \frac{e^{-\lambda}\lambda^k}{k!}/\Sigma_{k=1}^{n}\frac{e^{-\lambda}\lambda^k}{k!}$) and ν

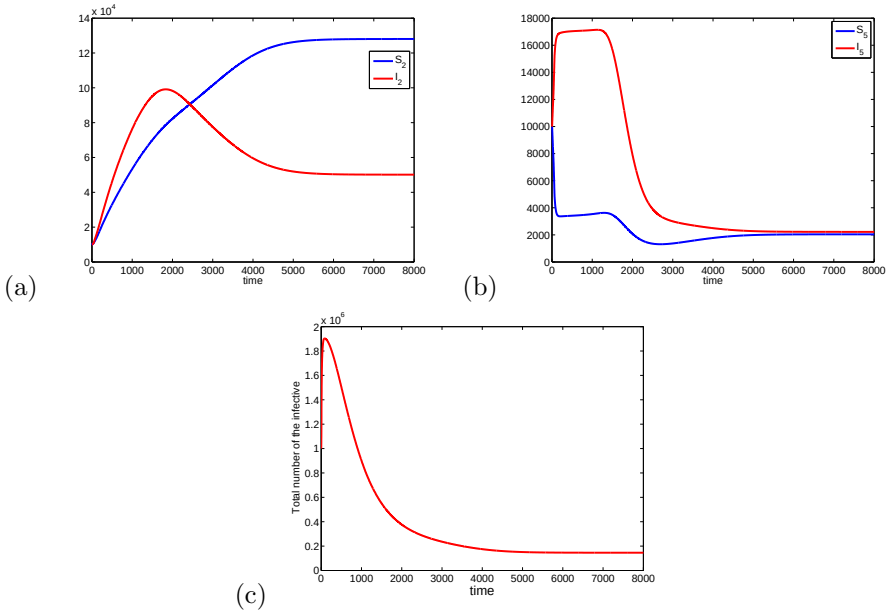


FIGURE 5. (a) The number of the S_2 and I_2; (b) The number of the S_5 and I_5; (c) The total number of the infective. Here, $\tau = 0.008$, $r_k = k^{-4}/\Sigma_{k=1}^{n} k^{-4}$ and $R_0 \approx 5 > 1$.

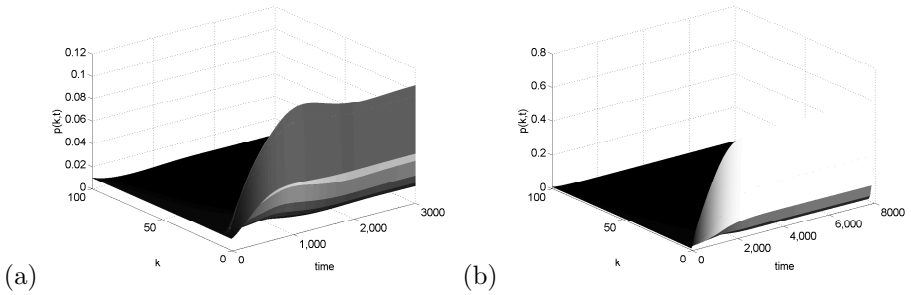
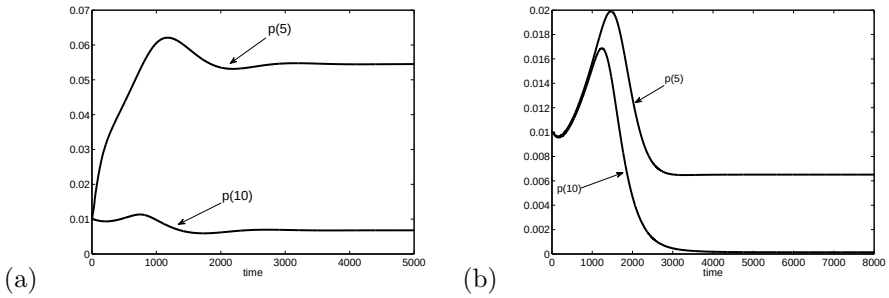


FIGURE 6. Degree distribution is regarded as a function of k and time. (a) $r_k = \frac{e^{-4}4^k}{k!}/\Sigma_{k=1}^{n}\frac{e^{-4}4^k}{k!}$; (b) $r_k = k^{-4}/\Sigma_{k=1}^{n} k^{-4}$. Other parameter values: $A = 5000$, $\gamma = 0.02$, $\mu = 0.006$ and $\tau = 0.008$.

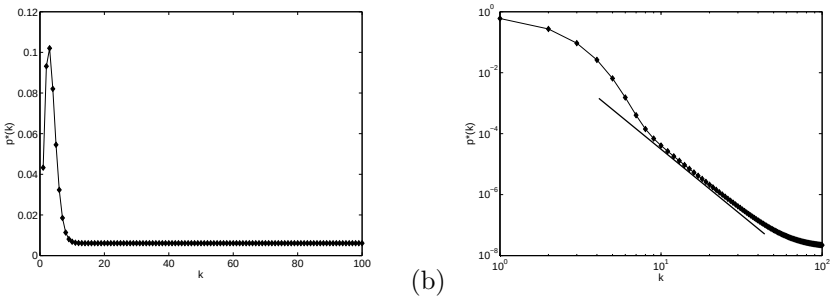
(exponent of $r_k = k^{-\nu}/\Sigma_{k=1}^{n} k^{-\nu}$). When r_k is in the form of poisson distribution, we give the plot of R_0 with respect to μ and λ in Fig. 9(a). One can see that R_0 is a decreasing function of μ and an increasing function of λ. That is to say, when μ is small or even zero, $R_0 > 1$ and thus the disease will persist. And when λ is small, the disease will die out due to that $R_0 < 1$. When $r_k = k^{-\nu}/\Sigma_{k=1}^{n} k^{-\nu}$, we give the plot of R_0 with respect to μ and ν in Fig. 9(b). As the death rate and ν increases, the basic reproduction number decreases.

5. **Discussions.** In this paper, we propose and study network epidemic models with demographics, and consider the effect of network topology due to population



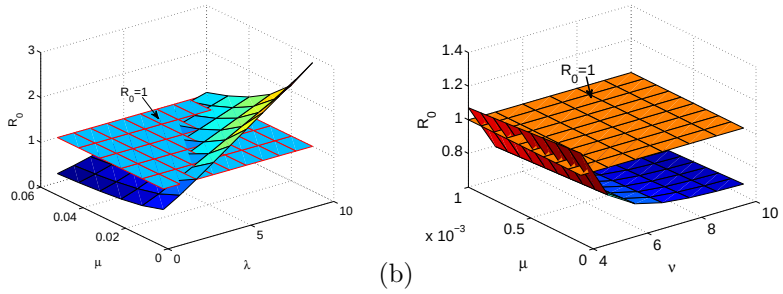
(a) (b)

FIGURE 7. Time series of $p(5)$ and $p(10)$. (a) $r_k = \frac{e^{-4}4^k}{k!}/\Sigma_{k=1}^{n}\frac{e^{-4}4^k}{k!}$; (b) $r_k = k^{-4}/\Sigma_{k=1}^{n}k^{-4}$. Other parameter values: $A = 5000$, $\gamma = 0.02$, $\mu = 0.006$ and $\tau = 0.008$.



(a) (b)

FIGURE 8. (a) Stationary degree distribution with $r_k = \frac{e^{-4}4^k}{k!}/\Sigma_{k=1}^{n}\frac{e^{-4}4^k}{k!}$; (b) Stationary degree distribution with $r_k = k^{-4}/\Sigma_{k=1}^{n}k^{-4}$ (log-log plot). Here, $\tau = 0.008$ and $R_0 > 1$.



(a) (b)

FIGURE 9. (a) The plot of R_0 as a function of μ and λ with $r_k = \frac{e^{-\lambda}\lambda^k}{k!}/\Sigma_{k=1}^{n}\frac{e^{-\lambda}\lambda^k}{k!}$; (b) The plot of R_0 as a function of μ and ν with $r_k = k^{-\nu}/\Sigma_{k=1}^{n}k^{-\nu}$). Other parameter values are taken as: $A = 5000$, $\gamma = 0.02$ and $\tau = 0.008$.

births or recruitment and deaths. We establish the basic reproduction number R_0 for SIS model by investigating the local stability of the infection-free equilibrium.

We further prove that the infection-free equilibrium of SIS model is globally asymptotically stable by qualitative analysis of the dynamics of the model system and by utilizing the method in [31]. We show that if the basic reproduction number is more than one, then there exists a unique endemic equilibrium and globally asymptotically stable for the SIS models.

It is known that, in the static network, basic reproduction number R_0 depends only on the degree distribution. However, in a dynamic network with recruitment and deaths, it does not depend on the degree distribution. And from the system (16), we know that the degree distribution of population varies with time before it reaches the stationary state (see Fig. 10), which is different from the previous work [23, 24, 20, 21, 22, 12, 13, 26, 5, 2, 4]. Furthermore, we found that basic reproduction number R_0 depends on the demographical parameters (μ or λ) (see Figs. 8-9). However, R_0 does not depend on the recruitment rate A (if recruitment rate is chosen as a different form, this conclusion may not hold). The reason is that the infection rate equals to the ratio of the edges connected with the infected in all the edges in the network, which is similar to the standard infection rate in the mean-filed epidemic models.

It should be noted that, in our model, we choose $\Pi_a(k, \overrightarrow{N}) = 1/N$. In fact, $\Pi_a(k, \overrightarrow{N}) = 1/(N-1)$ should be a better and realistic choice. Meanwhile, in system (10), the total population $N(t)$ follows the equation (13). In the future work, we will investigate the dynamical behavior of the network models $N(t)$ that satisfies the equation (14) or (15).

In this paper, we only consider the case of the network models with constant recruitment rate. If we consider cases of changing recruitment rate or other forms of demographics, one would expect much more complicated dynamics. It will also be interesting to use network models to study spreading of vector-borne diseases involving multiple-populations. We are working on to apply network modelings for vector-borne diseases.

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REFERENCES

[1] R. M. Anderson and R. M. May, *Infectious Diseases of Humans*, Oxford University Press, Oxford, 1992.

[2] A.-L. Barabasi and R. Albert, Emergence of scaling in random networks, *Science*, **286** (1999), 509–511.

[3] M. Barthelemy, A. Barrat, R. Pastor-Satorras and A. Vespignani, Dynamical patterns of epidemic outbreaks in complex heterogeneous networks, *Journal of Theoretical Biology*, **235** (2005), 275–288.

[4] E. Ben-Naim and P. L. Krapivsky, Addition-deletion networks, *J. Phys. A: Math. Theor.*, **40** (2007), 8607–8619.

[5] M. Boguna, R. Pastor-Satorras and A. Vespignani, Epidemic spreading in complex networks with degree correlations, e-print cond-mat/0301149, (2003).

[6] S. Busenberg and P. van den Driessche, Analysis of a disease transmission model in a population with varying size, *J. Math. Biol.*, **28** (1990), 257–270.

[7] C. Castillo-Chavez and H. R. Thieme, Asymptotically autonomous epidemic models, in *Mathematical Population Dynamics: Analysis of Heterogeneity* (eds. O. Arino, D. Axelrod, M. Kimmel and M. Langlais), Theory of Epidemics, 1, Wuerz, Winnipeg, 1993, 33–50.

[8] K. Emrah, Explicit formula for the inverse of a tridiagonal matrix by backward continued fractions, *Applied Mathematics and Computation*, **197** (2008), 345–357.

[9] L. Q. Gao and H. W. Hethcote, Disease transmission models with density-dependent demographics, *J. Math. Biol.*, **30** (1992), 717–731.
[10] L. Hufnagel, D. Brockmann and T. Geisel, Forecast and control of epidemics in a globalized world, *Proc. Natl. Acad. Sci. U.S.A.*, **101** (2004), 15124.
[11] Y. Jin and W. Wang, The effect of population dispersal on the spread of a disease, *J. Math. Anal. Appl.*, **308** (2005), 343–364.
[12] J. Joo and J. L. Lebowitz, Behavior of susceptible-infected-susceptible epidemics on heterogeneous networks with saturation, *Phys. Rev. E*, **69** (2004), 066105.
[13] M. J. Keeling and K. T. D. Eames, Networks and epidemic models, *J. R. Soc. Interface*, **2** (2005), 295–307.
[14] M. J. Keeling and P. Rohani, *Modeling Infectious Diseases in Humans and Animals*, Princeton University Press, 2007.
[15] W. O. Kermack and A. G. McKendrick, A contribution to the mathematical theory of epidemics, *Proc. R. Soc. A*, **115** (1927), 700–711.
[16] I. Z. Kiss, D. M. Green and R. R. Kao, Heterogeneity and multiple of transmission on final epidemic size, *Mathematical Biosciences*, **203** (2006), 124–136.
[17] I. Z. Kiss, P. L. Simon and R. R. Kao, A contact-network-based formulation of a preferential mixing model, *Bulletin of Mathematical Biology*, **71** (2009), 888–905.
[18] J. Lindquist, J. Ma, P. van den Driessche and F. H. Willeboords, Network evolution by different rewiring schemes, *Physica D*, **238** (2009), 370–378.
[19] Z. Ma and J. Li, *Dynamical Modeling and Anaylsis of Epidemics*, World Scientific, 2009.
[20] R. M. May and A. L. Lloyd, Infection dynamics on scale-free networks, *Phys. Rev. E*, **64** (2001), 066112.
[21] Y. Moreno, R. Pastor-Satorras and A. Vespignani, Epidemic outbreaks in complex heterogeneous networks, *Eur. Phys. J. B*, **26** (2002), 521–529.
[22] R. Olinky and L. Stone, Unexpected epidemic thresholds in heterogeneous networks: The role of disease transmission, *Phys. Rev. E*, **70** (2004), 030902.
[23] R. Pastor-Satorras and A. Vespignani, Epidemic dynamics and endemic states in complex networks, *Phys. Rev. E*, **63** (2001), 066117.
[24] R. Pastor-Satorras and A. Vespignani, Epidemic spreading in scale-free networks, *Phys. Rev. Let.*, **86** (2001), 3200.
[25] M. G. Roberta, An SEI model with density-dependent demographics and epidemiology, *IMA Journal of Mathematics Applied in Medicine & Biology*, **13** (1996), 245–257.
[26] L. B. Shaw and I. B. Schwartz, Fluctuating epidemics on adaptive networks, *Phys. Rev. E*, **77** (2008), 066101.
[27] H. L. Smith, On the asymptotic behavior of a class of deterministic models of cooperating species, *SIAM J. Appl. Math.*, **46** (1986), 368–375.
[28] H. R. Thieme, Asymptotically autonomous differential equations in the plane, *Rocky Mountain J. Math.*, **24** (1994), 351–380.
[29] P. van den Driessche and J. Watmough, Reproduction numbers and sub-threshold endemic equilibria for compartmental models of disease transmission, *Mathematical Biosciences*, **180** (2002), 29–48.
[30] L. Wang and G. Z. Dai, Global stability of virus spreading in complex heterogeneous networks, *Siam J. Appl. Math.*, **68** (2008), 1495–1502.
[31] W. Wang and X.-Q. Zhao, An epidemic model in a patchy environment, *Mathematical Biosciences*, **190** (2004), 97–112.
[32] X.-Q. Zhao, *Dynamical Systems in Population Biology*, Springer-Verlag, New York, 2003.
[33] X.-Q. Zhao and Z.-J. Jing, Global asymptotic behavior in some cooperative systems of functional differential equations, *Canad. Appl. Math. Quart.*, **4** (1996), 421–444.

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