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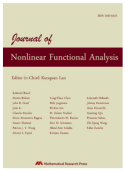

AN SEIS EPIDEMIC MODEL ON SCALE-FREE NETWORK WITH RECRUITMENT AND DEATH

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Abstract. Most epidemic models on networks do not involve demographical dynamics. Here, an SEIS epidemic model is introduced to describe sexual disease spreading on complex networks. Demographics are incorporate into the modeling. The basic reproduction number is calculated in terms of conditional probability for the correlated network models. The global asymptotic stability of the disease-free equilibrium is discussed. Simulations are given about the degree distributions under different demographical dynamics.

Keywords. Scale-free network; Demographical dynamics; Basic reproduction number; Global asymptotic stability.

1. Introduction

The knowledge of the mechanisms involved in disease spread and the relation between the network structure and the dynamical patterns of the spread process has improved in the last several years [1]. For instance, a very important example of scale-free networks is found in the web of human sexual contacts [2]. A scale-free connectivity distribution is associated with a large heterogeneity in the connectivity properties of the system. In scale-free networks, the

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number of contacts of a node with other nodes in the system, the degree (or connectivity) k, follows a power-law distribution, $P(k) \sim k^{-2-\gamma}$, with $0 < \gamma \leq 1$. For example, data from national sex surveys [2, 3] provide quantitative information on the number of sexual partners, i.e., the degree k, of an individual. Surveys turn out that the number of heterosexual partners reported from different populations is well described by power-law scale-free distributions [4, 5].

Many mathematical models about the spreading of epidemics on the scale-free networks have been studied [6, 7, 8, 9, 10, 11]. But most of them were based on static scale-free networks, i.e., they did not consider the recruitment and death of nodes and links. Some conclusions have been found for this kind of static infinite scale-free models. For example, reference [11] states that epidemic processes do not possess an epidemic threshold in infinite scale-free networks below which diseases cannot produce a major epidemic outbreak or the inset of an endemic state. The absence of an intrinsic epidemic threshold has been found in both the susceptible-infected-susceptible (SIS) model [11] and the susceptible-infected-removed (SIR) model [12] in infinite scale-free networks. Recently, some work have been done about the spread of disease with birth and death on networks [13, 14, 15]. Reference [13] considered a scale-free network with birth and death of nodes. Authors found that, the result was the same as that of the standard SIS model. This reminds us that the structure of the networks plays a very important role in the spreading properties of infectious disease. Reference [14] also compared immunize strategies, including proportional, targeted, and acquaintance immunization strategies.

References above mainly discussed SI or SIS model. But most STDs have latent periods. For example, the latent period of syphilis is 2-3 weeks and the latent period of poradenolymphitis is about 6-21 days, with an average 7-10 days. So in this paper, we build an SEIS model to analyze the spread of STDs in a scale-free network with recruitment and death of nodes and links. Our model relies on the following rough description of individuals in the population. Namely, each node of the graph represents an individual and each link is a connection along which the STDs can spread. We give theoretical analysis, For example, the basic reproduction number of the STDs spreading on this kind of network, the existence and global stability of the disease-free equilibrium.

This paper is organized as follows. In Section 2, we develop the mathematical model. In Section 3, we give the threshold for the STDs spread on scale-free network. We also analyse the stability of disease-free equilibrium. In Section 4, we give simulations about the dynamics behavior and finally in Section 5 we discuss the implications of the results.

2. The model

In this model, we use S_k, E_k and I_k represent the relative densities of nodes of degree k. They also denote the densities of the susceptible, the latent and the infectious respectively. Then we have the following dynamics model:

$$\begin{aligned}
S_1^{'} &= r_1A-\mu S_1-\tau S_1\Theta+\gamma I_1-\tfrac{A\sum_{i=1}^{n} ir_i}{N}S_1+2\mu S_2,\\
E_1^{'} &= \tau S_1\Theta-\mu E_1-\omega E_1-\tfrac{A\sum_{i=1}^{n} ir_i}{N}E_1+2\mu E_2,\\
I_1^{'} &= \omega E_1-\mu I_1-\gamma I_1-\tfrac{A\sum_{i=1}^{n} ir_i}{N}I_1+2\mu I_2,\\
&\cdots\cdots\cdots\\
S_k^{'} &= r_kA-\mu S_k-\tau kS_k\Theta+\gamma I_k+\tfrac{A\sum_{i=1}^{n} ir_i}{N}[S_{k-1}-S_k]+\mu[(k+1)S_{k+1}-kS_k]\\
E_k^{'} &= \tau kS_k\Theta-\mu E_k-\omega E_k+\tfrac{A\sum_{i=1}^{n} ir_i}{N}[E_{k-1}-E_k]+\mu[(k+1)E_{k+1}-kE_k],\\
I_k^{'} &= \omega E_k-\mu I_k-\gamma I_k+\tfrac{A\sum_{i=1}^{n} ir_i}{N}[I_{k-1}-I_k]+\mu[(k+1)I_{k+1}-kI_k],,k=2,...,n-1,\\
&\cdots\cdots\cdots\\
S_n^{'} &= r_nA-\mu S_n-\tau nS_n\Theta+\gamma I_n+\tfrac{A\sum_{i=1}^{n} ir_i}{N}S_{n-1}-\mu nS_n,\\
E_n^{'} &= \tau nS_n\Theta-\mu E_n-\omega E_n+\tfrac{A\sum_{i=1}^{n} ir_i}{N}E_{n-1}-\mu nE_n,\\
I_n^{'} &= \omega E_n-\mu I_n-\gamma I_n+\tfrac{A\sum_{i=1}^{n} ir_i}{N}I_{n-1}-\mu nI_n.
\end{aligned} \tag{3.1}$$

The meanings for each parameter or item of system (3.1) can be found in the following.

- Denote that

$$N=\sum_{k=1}^{n}N_k,\quad S=\sum_{k=1}^{n}S_k,\quad E=\sum_{k=1}^{n}E_k,\quad I=\sum_{k=1}^{n}I_k,$$

where $N_k=S_k+E_k+I_k$ and n is the maximum number of contact each individual can make per unit of time.

- Parameter A represents the recruitment nodes per unit time into the network and μ is the nature death (removed) rate of nodes. Parameter r_k represents the connection probability of the recruitment nodes with the kth subgroup. So $r_k A$ shows the addition of an individual of degree k to the network.
- Parameter τ represents the transmission rates for each sexual behavior of infected individuals and γ represents the recover rate for infected individuals.
- $\tau k S_k \Theta$ represents the loss of susceptible individuals with degree k because of infection, in which,

$$\Theta(t) = \frac{\sum_{j=1}^{n} jp(j)I_j/N_j}{< k >} = \frac{\sum_{j=1}^{n} jI_j}{\sum_{k=1}^{n} kN_k}.$$

  $\Theta(t)$ represents the expectation that any given link emanating from a node of connectivity k points to an infected node.
- Term $\frac{A\sum_{i=1}^{n} ir_i}{N} S_{k-1}$ describes the flow of nodes from degree $k-1$ to k as they gain extra edges when newly added nodes attach to them. Similarly, $-\frac{A\sum_{i=1}^{n} ir_i}{N} S_k$ represents the flow of nodes from degree k to $k+1$. Here, $\frac{1}{N}$ represents that the probability that a recruited node links to a node is uniformly distributed.
- Term $\mu(k+1)S_{k+1}$ describes the flow from degree $k+1$ to k as vertices lose edges when one of their neighbors is removed from the network by death. Similarly, term $-\mu k S_k$ describes the flow from degree k to $k-1$. We suppose each node can at most gain or lose one edge in a single unite time and there is no isolated node exist.
- Parameter γ represents the recovery rate of infected individuals, i.e., infected individuals recovery from STDs after time spam $\frac{1}{\gamma}$.

Note that, the degree distribution $p(k,t) = \frac{N_k(t)}{N(t)}$, i.e., the probability that a node chosen at random at time t have degree k, is time dependent under system (3.1). But in most previous studies, the number of population N and the degree distribution $p(k)$ is fixed.

3. Some results

Adding $S_i + E_i + I_i$ of system (3.1) and denote it as $N_i, i = 1, ...n$, then we get the following system:

$$\begin{aligned} N_1' &= Ar_1 - \tfrac{A\sum_{i=1}^{n} ir_i}{N} N_1 - \mu N_1 + 2\mu N_2, \\ N_k' &= Ar_k + \tfrac{A\sum_{i=1}^{n} ir_i}{N}[N_{k-1} - N_k] - \mu N_k + \mu[(k+1)N_{k+1} - kN_k], k = 2, \cdots, n-1, \\ N_n' &= Ar_n + \tfrac{A\sum_{i=1}^{n} ir_i}{N} N_{n-1} - \mu N_n - \mu n N_n. \end{aligned} \tag{3.2}$$

The sum of the above n equations is about the total nodes of this network, $N(t)$, which satisfies $N^{'}(t) = A - \mu N$. Then we get the unique globally asymptotically stable steady state of system (3.2): $N^* = A/\mu$. Since we are interest in asymptotic behavior, so we can write system (3.2) as the following simple system:

$$\begin{aligned} N_1' &= Ar_1 - \mu c N_1 - \mu N_1 + 2\mu N_2, \\ N_k' &= Ar_k + \mu c[N_{k-1} - N_k] - \mu N_k + \mu[(k+1)N_{k+1} - kN_k], k = 2, \cdots, n-1, \\ N_n' &= Ar_n + \mu c N_{k-1} - \mu N_n - n\mu N_n. \end{aligned} \tag{3.3}$$

In which, $c = \sum_{i=1}^{n} ir_i$. Let

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

$T = diag(1, \delta_1, \delta_2, ..., \delta_{n-1})$, $\delta_i = \sqrt{\frac{c^i}{(1+i)!}}$, $d_i = \mu\sqrt{c(1+i)}$. Then

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

Obviously, matrix J and J^* have the same eigenvalues. Also matrix J^* is a real symmetric matrix, which implies that it has real eigenvalues. So we can use Theorem Gerschgorin to estimate the eigenvalues of matrix J. According to Theorem Gerschgorin, every eigenvalue of matrix J should at least located in one of the following Gerschgorin disks:

$$\begin{aligned} D_1 &= \{\lambda : -(2c+1)\mu \le \lambda \le -\mu\}, \\ D_2 &= \{\lambda : -\mu(2c+2k-1) \le \lambda \le -\mu, k = 2, \cdots, n-1\}, \\ D_3 &= \{\lambda : -(2n+1)\mu \le \lambda \le -\mu\}. \end{aligned}$$

Let $X = \begin{pmatrix} N_1 \\ N_2 \\ \vdots \\ N_n \end{pmatrix}$. Then system (3.3) can be written as: $\frac{d}{dt}X = A \begin{pmatrix} r_1 \\ r_2 \\ \vdots \\ r_n \end{pmatrix} + JX$.

According to Theorem 1.2 of reference [16] , system (3.3) has a unique equilibrium ($P_0 = (N_1^*, N_2^*, \cdots, N_n^*)$, and $N = \sum_{i=1}^n N_i^* = A/\mu$. Then system (3.3) has a unique equilibrium $P_0 = (N_1^*, N_2^*, \cdots, N_n^*)$, which is globally asymptotically stable, i.e., $\lim_{t\to\infty} N_k(t) = N_k^*$. So the limit system of equations (3.3), i.e., $N_j = N_j^*, j = 1, 2, \cdots, n$, then becomes (we arrange the order of the equations):

$$\begin{aligned} E_1^{'} &= \beta_1 S_1 \textstyle\sum_{i=1}^n i I_i - [\mu(1+c)+\omega]E_1 + 2\mu E_2, \\ E_k^{'} &= \beta_k S_k \textstyle\sum_{i=1}^n i I_i - [\mu(1+c+k)+\omega]E_k + \mu c E_{k-1} + \mu(k+1)E_{k+1}, k = 2, ..., n-1, \\ E_n^{'} &= \beta_n S_n \textstyle\sum_{i=1}^n i I_i - [\mu(1+n)+\omega]E_n + \mu c E_{n-1}, \\ I_1^{'} &= \omega E_1 - [\mu(1+c)+\gamma]I_1 + 2\mu I_2, \\ I_k^{'} &= \omega E_k - [\mu(1+c+k)+\gamma]I_k + \mu c I_{k-1} + \mu(k+1)I_{k+1}, \quad k = 2, ..., n-1, \\ I_n^{'} &= \omega E_n - [\mu(1+n)+\gamma]I_n + \mu c I_{n-1}, \\ S_1^{'} &= r_1 A - \beta_1 S_1 \textstyle\sum_{i=1}^n i I_i - \mu(1+c)S_1 + \gamma I_1 + 2\mu S_2, \\ S_k^{'} &= r_k A - \beta_k S_k \textstyle\sum_{i=1}^n i I_i - \mu(1+c+k)S_k + \gamma I_k + \mu c S_{k-1} + \mu(k+1)S_{k+1}, k = 2, ..., n-1, \\ S_n^{'} &= r_n A - \beta_n S_n \textstyle\sum_{i=1}^n i I_i - \mu(1+n)S_n + \gamma I_n + \mu c S_{n-1}. \end{aligned} \tag{3.4}$$

where $c = \sum_{i=1}^{n} ir_i$, $\beta_k = \dfrac{\tau k}{\sum_{k=1}^{n} kN_k^*}$. Then system (3.4) has a unique disease free equilibrium $Q_0 = (0,\cdots,0,\cdots,0;0,\cdots,0,\cdots,0;N_1^*,\cdots N_k^*,\cdots,N_n^*)$.

The basic reproductive number $\mathscr{R}_0$ is the effective number of secondary cases produced by a typical infected individual during its entire period of infectiousness in a demographically steady susceptible population [17]. We found closed forms of $\mathscr{R}_0$ in terms of model parameters using the "next-generation operator" method of [17]:

$$\mathscr{R}_0 = \rho(\mathscr{F}\mathscr{V}^{-1}),$$

where matrix $\mathscr{F}$ and $\mathscr{V}$ are defined as the follows:

$$\mathscr{F} = \begin{pmatrix} 0_{n\times n} & M_{n\times n} \\ 0_{n\times n} & 0_{n\times n} \end{pmatrix},$$

here,

$$M_{n\times n} = \begin{pmatrix} \beta_1 N_1^* & 2\beta_1 N_1^* & \cdots & n\beta_1 N_1^* \\ \beta_2 N_2^* & 2\beta_2 N_2^* & \cdots & n\beta_2 N_2^* \\ \vdots & \vdots & \cdots & \vdots \\ \beta_n N_n^* & 2\beta_n N_n^* & \cdots & n\beta_n N_n^* \end{pmatrix}$$

and $\mathscr{V} = \begin{pmatrix} V_{n\times n}^1 & 0_{n\times n} \\ -\omega E_n & V_{n\times n}^2 \end{pmatrix}$, where E_n is unit matrix of $n\times n$ and

$$V_{n\times n}^1 = \begin{pmatrix} \theta_1 & -2\mu & & & & & \\ -\mu c & \theta_1+2\mu & -3\mu & & & & \\ & \ddots & \ddots & \ddots & & & \\ & & -\mu c & \theta_1+k\mu & -(k+1)\mu & & \\ & & & \ddots & \ddots & \ddots & \\ & & & & -\mu c & \theta_1+n\mu & -n\mu \\ & & & & & -\mu c & (n+1)\mu+\omega \end{pmatrix},$$

$$V_{n\times n}^2=\begin{pmatrix} \theta_2 & -2\mu & & & & & & \\ -\mu c & \theta_2+2\mu & -3\mu & & & & & \\ & \ddots & \ddots & \ddots & & & & \\ & & -\mu c & \theta_2+k\mu & -(k+1)\mu & & & \\ & & & \ddots & \ddots & \ddots & & \\ & & & & -\mu c & \theta_2+n\mu & -n\mu & \\ & & & & & -\mu c & (n+1)\mu+\gamma & \end{pmatrix}.$$

Here $\theta_1=\mu+\mu c+\omega$, $\theta_2=\mu+\mu c+\gamma$.

Then we know that the disease-free equilibrium Q_0 is locally stable when $\mathscr{R}_0<1$ and unstable when $\mathscr{R}_0>1$ [17].

Theorem 3.1 *The disease-free equilibrium Q_0 is globally stable when $\mathscr{R}_0<1$ and unstable when $\mathscr{R}_0>1$.*

Proof. Firstly, let $(E(t),I(t),S(t))$ be any solution of system (5), we will prove that $\lim_{t\to\infty}(E(t)+I(t))=0$.

From the discussion above we know that system (3.3) has one and only one globally stable steady state $P_0=(N_1^*,N_2^*,\cdots,N_n^*)$. So for any $\varepsilon>0$, there exists a sufficiently large t, such that $N(t)=S(t)+E(t)+I(t)<P_0+\overline{\varepsilon}$, in which, $\overline{\varepsilon}=(\varepsilon,\cdots,\varepsilon)$. So when t is sufficiently large, we have

$$\begin{aligned} E_1^{'} &\leq \beta_1(N_1^*+\varepsilon)\textstyle\sum_{i=1}^n iI_i-[\mu(1+c)+\omega]E_1+2\mu E_2,\\ E_k^{'} &\leq \beta_k(N_k^*+\varepsilon)\textstyle\sum_{i=1}^n iI_i-[\mu(1+c+k)+\omega]E_k+\mu cE_{k-1}+\mu(k+1)E_{k+1},k=2,...,n-1,\\ E_n^{'} &\leq \beta_n(N_n^*+\varepsilon)\textstyle\sum_{i=1}^n iI_i-[\mu(1+n)+\omega]E_n+\mu cE_{n-1}, \end{aligned}$$

Now we construct a system as follows:

$$\begin{aligned}
\overline{E_1'} &= \beta_1(N_1^*+\varepsilon)\textstyle\sum_{i=1}^n i\overline{I_i} - [\mu(1+c)+\omega]\overline{E_1} + 2\mu\overline{E_2},\\
\overline{E_k'} &= \beta_k(N_k^*+\varepsilon)\textstyle\sum_{i=1}^n i\overline{I_i} - [\mu(1+c+k)+\omega]\overline{E_k} + \mu c\overline{E_{k-1}} + \mu(k+1)\overline{E_{k+1}}, k=2,...,n-1,\\
\overline{E_n'} &= \beta_n(N_n^*+\varepsilon)\textstyle\sum_{i=1}^n i\overline{I_i} - [\mu(1+n)+\omega]\overline{E_n} + \mu c\overline{E_{n-1}},\\
\overline{I_1'} &= \omega\overline{E_1} - [\mu(1+c)+\gamma]\overline{I_1} + 2\mu\overline{I_2},\\
\overline{I_k'} &= \omega\overline{E_k} - [\mu(1+c+k)+\gamma]\overline{I_k} + \mu c\overline{I_{k-1}} + \mu(k+1)\overline{I_{k+1}}, \quad k=2,...,n-1,\\
\overline{I_n'} &= \omega\overline{E_n} - [\mu(1+n)+\gamma]\overline{I_n} + \mu c\overline{I_{n-1}}.
\end{aligned} \tag{3.5}$$

So, now we only need to prove that the solution of system (3.5) will tend to zero when time t tends to infinity.

Let matrix $M_2 = \begin{pmatrix} 0_{n\times n} & diag(\beta_1,\beta_2,\cdots,\beta_n) \\ 0_{n\times n} & 0_{n\times n} \end{pmatrix}$.

Since $\mathscr{R}_0 < 1$, we have $s(M_1) < 0$ ($M_1 = \mathscr{F} - \mathscr{V}$)[18], also $s(M_1+\varepsilon M_2)$ is continuous for small ε. So we can fix $\varepsilon > 0$ which is small enough, such that, $s(M_1+\varepsilon M_2) < 0$. So, the solution of system (3.5) will tend to zero when t is big enough.

It is easy to prove that, the off-diagonal elements of the Jacobi matrix of the right side of system (3.5) is non-negative, which means, the system is quasi-monotone. Using the comparison theorem, we have $\lim_{t\to\infty} E_i(t) = 0$ and $\lim_{t\to\infty} I_i(t) = 0$. So $\lim_{t\to\infty}(E(t)+I(t)) = 0$. That means, for any initial data $(E_0,I_0,S_0) \in R_+^n \times R_+^n \times (R_+^n \backslash \{0\})$, we have $N_0 = E_0+I_0+S_0 \in R_+^n\backslash\{0\}$. So

$$\lim_{t\to\infty} S(t) = \lim_{t\to\infty}(N(t)-E(t)-I(t)) = P_0 - 0 = P_0.$$

This completes the proof.

4. Simulations

In this section we present the results of numerical simulations investigating the dynamic of the complex network. Parameters that are used in the simulations are listed as follows: $A = 5000$, $\mu = 0.0167$, $\gamma = 0.02$, $\tau = 0.08$, $\omega = 0.1$, $p = 2.4$ and $\lambda = 100$. Figure 1 and Figure 2 show

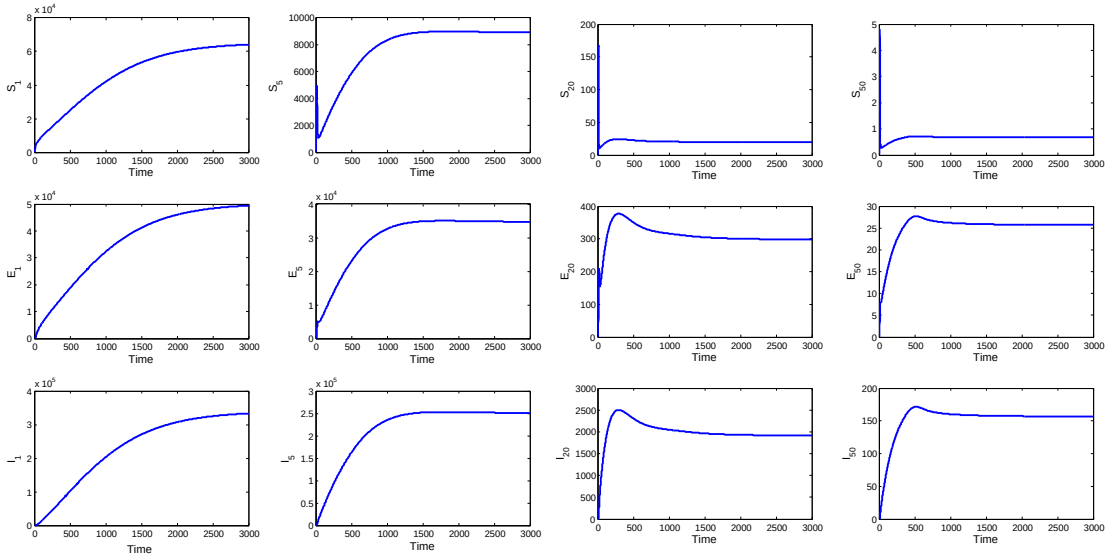

FIGURE 1. Dynamics of nodes of degree 1, 5, 20 and 50 respectively. The birth term follows power-law distribution and $\mathscr{R}_0 = 1.38$ under this situation.

the dynamics of nodes of degree 1, 5, 20 and 50 respectively. The birth term follows power-law distribution $r_k = 2k^{-p}$ (Figure 1, $\mathscr{R}_0 = 1.38$) and Poisson distribution $r_k = \frac{e^{-\lambda}\lambda^k}{k!}$ (Figure 2, $\mathscr{R}_0 = 2.07$) respectively. One can see that for both situations, the disease will persist and converge to a positive stationary state, which means that the endemic state is stable.

Also, it is interesting to know the evolution of the degree distribution along with time about the complex network. Let P_k be the cumulative degree distribution and $P(k)$ be the degree distribution. We compare the degree distributions and the cumulative degree distributions between the birth term following power-law distribution $r_k = 2k^{-p}$ and Poisson distribution $r_k = \frac{e^{-\lambda}\lambda^k}{k!}$ respectively. Parameters that are used in the simulations are s above. $\mathscr{R}_0 > 1$ under the two situations. Figure 3 shows the cumulative degree distribution of the sexual network under two different birth rate distribution. In both figures, the X-axis and Y-axis are both plotted in logarithmic forms. The curves in the two figures show that, after long time evolution, the degree of the sexual network does not show a simple power low distribution or Poisson distribution,

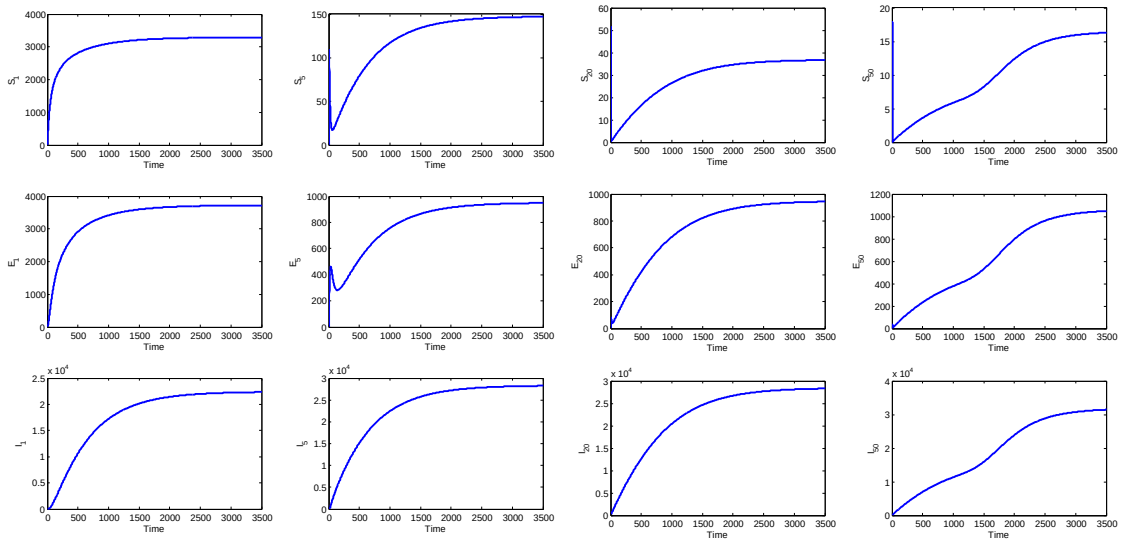

FIGURE 2. Dynamics of nodes of degree 1, 5, 20 and 50 respectively. The birth term follows Poisson distribution and $\mathscr{R}_0 = 2.07$ under this situation.

even if the new nodes link the network upon a Poisson or Power law distribution. It should be a combination of several different distributions.

5. Discussions

Recent research into the properties of human sexual-contact networks has suggested that the degree distribution of the contact graph exhibits power-law scaling. From a mathematical aspect, our models on networks can be viewed as multi-type SEIS models if the networks possess the bounded degree property.

Usually the network models do not involve demographical dynamics. In this model, we introduce the generalized epidemic models for modeling infectious diseases spreading in complex networks. We incorporate demographics and random mixing in to the modeling. The basic reproduction number is calculated in terms of conditional probability for the correlated network models. We also discuss the global asymptotic stability of the disease-free equilibrium.

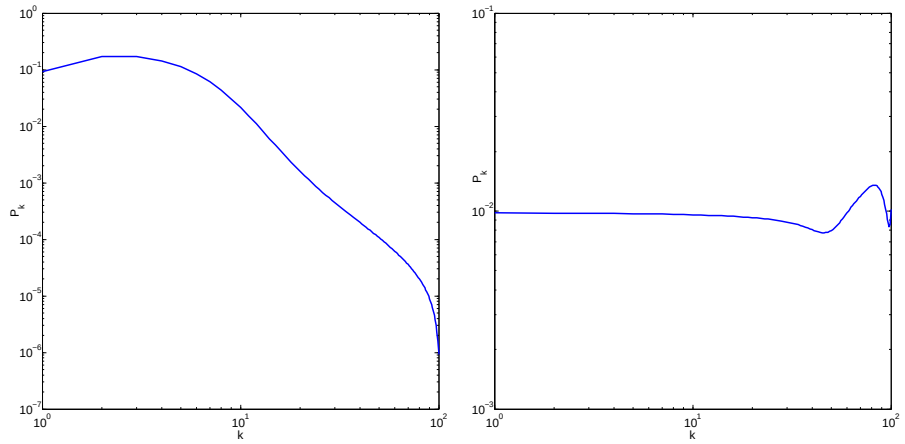

FIGURE 3. Comparation of degree distributions between the birth term following power-law distribution $r_k = 2k^{-2.4}$ and Poisson distribution $r_k = \frac{e^{-100}100^k}{k!}$ respectively. In both figures, the X-axis and Y-axis are both plotted in logarithmic forms.

Simulations show that, the degree of the sexual network does not keep a simple power low distribution or Poisson distribution after long time evolution. Furthermore, we found that dynamics depend on the demographical parameters p or λ when considering the birth term following power law or Poisson respectively. Also, the stationary degree distribution depends on the value of p and λ. For example, the stationary degree distribution follows a Poisson distribution when λ is small (For example 4, not shown in this paper) but more complex when it is big (Figure 3). In this sense, the results are similar to the *SIS* model, which is also sensitive to the birth term. In short, the structure of the complex network is not very clear, and need more work on it in the future.

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