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Modeling and analysis of epidemic spreading on community network with node’s birth and death

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Abstract

In this paper, a modified susceptible infected susceptible (SIS) epidemic model is proposed on community structure networks considering birth and death of node. For the existence of node’s death would change the topology of global network, the characteristic of network with death rate is discussed. Then we study the epidemiology behavior based on the mean-field theory and derive the relationships between epidemic threshold and other parameters, such as modularity coefficient, birth rate and death rates (caused by disease or other reasons). In addition, the stability of endemic equilibrium is analyzed. Theoretical analysis and simulations show that the epidemic threshold increases with the increase of two kinds of death rates, while it decreases with the increase of the modularity coefficient and network size.

Keywords community structure, epidemic dynamics, complex networks, mean-field theory

1 Introduction

With the development of complex network theory, many social, biological and technological systems, such as the transportation networks, Internet and social network, can be properly analyzed from the perspective of complex network. And many common characteristics of most real-life networks have been found out, e.g., small-world effect and scale-free property. For some kind of networks, the degree distributions have small fluctuations, and they are called as homogeneous networks [1], e.g., random networks, small world networks and regular networks. In contrary to the homogeneous networks, heterogeneous networks [2] show power law distribution.

Based on the mean-field theory, many epidemic models, such as susceptible-infected (SI), SIS and susceptible-infected-recovered/ removed (SIR), have been proposed to describe the epidemic spreading process and investigate the epidemiology. It has been demonstrated that a threshold value exists in the homogeneous networks, while it is absent in the heterogeneous networks with sufficiently large size [3]. Compared to the lifetime of individuals, the infectious period of the majority of infectious diseases is short. Therefore, in most of the epidemic models, researchers generally choose to ignore the impact of individuals’ birth and death on epidemic spreading. However, in real life, some infectious diseases have high death rate and may result in people’s death in just a few days or even a few hours, such as severe acute respiratory syndrome (SARS), Hemagglutinin 7 Neuraminidase 9 (H7N9) and the recent Ebola. And some infectious diseases may have longer spreading time, like HBV, Tuberculosis. Besides, on the Internet, nodes’ adding and removing every time can also be treated as nodes’ birth and death. In Ref. [4], Liu et al. analyzed the spread of diseases with individuals’ birth and death on regular and scale-free networks. They find that on a regular network the epidemic threshold increases with the increase of the treatment rate and death rate, while for a power law degree distribution network the epidemic threshold is absent in the thermodynamic limit. Sanz et al. have investigated a Tuberculosis-like infection epidemiological model with
constant birth and death rates [5]. It is found that the constant change of the network topology which caused by the individuals’ birth and death enhances the epidemic incidence and reduces the epidemic threshold. Zhang et al. considered the epidemic thresholds for a staged progression model with birth and death on homogeneous and heterogeneous networks respectively [6]. In Ref. [7], an SIS model with nonlinear infection rate, as well as birth and death of nodes, is investigated on heterogeneous networks. In Ref. [8], Zhu et al. proposed a modified SIS model with a birth-death process and nonlinear infection rate on an adaptive and weighted contact network. It is indicated that the fixed weights setting can raise the disease risk, and that the variation of the weight cannot change the epidemic threshold but it can affect the epidemic size.

Recently, it has been revealed that many real networks have the so-called community structure [9], such as social networks, Internet and citation networks. A lot of researchers focus on the study of epidemic spreading on community structure networks. Liu et al. investigated the epidemic propagation in the SIS model on homogeneous network with community structure. They found that community structure suppress the global spread but increase the threshold [10]. Many researchers studied the epidemic spreading in scale-free networks with community structure based on different epidemic model, such as SI model [11], SIS model [12], SIR model [13–14] and susceptible exposed asymptotically infected recovered (SEAIR) model [15]. Chu et al. investigated the epidemic spreading in weighted scale-free networks with community structure [16]. In Ref. [17], Shao et al. proposed a traffic-driven SIS epidemic model in which the epidemic pathway is decided by the traffic of nodes in community structure networks. It is found that the community structure can accelerate the epidemic propagation in the traffic-driven model, which is different from the traditional model.

The social network has the property of community structure and some infectious diseases have high mortality rates or long infection period, while the previous studies only consider the impact of one of the aforementioned factors. So in this paper, we study the epidemic spreading in a modified SIS epidemic model with birth and death of individuals on a community structure network. The rest of this paper is organized as follows. In Sect. 2, we introduce in detail the network model and epidemic spreading process, and discuss the network characteristics either. In Sect. 3, mean-field theory is utilized to analyze the spreading properties of the modified SIS epidemic model. Sect. 4 gives some numerical and simulations which support the theoretical analysis. At last, Sect. 5 concludes the paper.

2 The community structure network model

As there exists the phenomena of the individual’s birth and death in real networks, the topology of the network changes over time. We consider undirected and un-weighted graphs in this paper. The generating algorithm of the network with community structure can be summarized as follows:

1. There are m different communities with \( n_i \) (\( i = 1, 2, \ldots, m \)) sites, the network size \( N = \sum_i n_i \). We assume that each site of this network is empty or occupied by only one individual.

2. The probability to have a link between the individuals (non-empty sites) in the same community is \( p_e \).

3. We create a link between two nodes (non-empty sites) belonging to different communities with probability \( p_c \).

4. Every site has its own state and may change with the evolution of epidemic. In each time step, susceptible individuals and infected individuals may respectively die with probability \( \alpha \) and \( \beta \), meanwhile, the corresponding site becomes empty, and the links of these sites are broken.

5. For each empty site, a susceptible individual may be born with probability \( b \), and then it create links with other individuals with probability \( p_i \) in the same community or \( p_c \) belonging to different communities.

Suppose the initial number of edges is \( K \), then we have:

\[
K = \sum_{i=1}^{m} n_i (n_i - 1) p_e + \sum_{i<j} n_i n_j p_c \tag{1}
\]

The state transition rules of the transmission process are schematically shown in Fig. 1. All the sites of the network are described as parameters: \( E, S \) or \( I \), which respectively represent the empty states, susceptible individual occupations and infected individual occupations. The specific process are as follows: an empty site can give birth to a healthy individual at rate \( b \); a healthy individual can be infected by contacting with infected neighbors at rate \( \lambda \) or die at rate \( \alpha \) (due to other reasons); an infected individual can be cured at rate \( \gamma \) or die at rate \( \beta \) (on account of the disease). When an individual dies, this site becomes
empty. In general, $\beta > \alpha$, and all parameters above are non-negative.

\[
\text{Fig. 1} \quad \text{The schematic diagram of state transition rules}
\]

An important measurement for community structure networks is the modularity coefficient [18]. It is defined as follows:

\[
Q = \sum_i \left[ e_i \left( \sum_j e_{ij} \right)^2 \right]
\]

where $e_{ij}$ denotes the proportion of edges between community $i$ and $j$ in the total network edges. So $e_i$ and $\sum_j e_{ij}$ can be described as follows:

\[
e_i = \frac{n_i(n_i-1)p_i}{2K}, \quad \sum_j e_{ij} = \frac{n_i(n_i-1)p_i}{2K} + \sum_{j \neq i} \frac{n_in_jp_z}{K}
\]

where $K$ represents the total edge number. Thus, for our model the modularity coefficient is:

\[
Q = \sum_i \left[ \frac{n_i(n_i-1)p_i}{2K} - \left( \frac{n_i(n_i-1)p_i}{2K} + \sum_{j \neq i} \frac{n_in_jp_z}{K} \right) \right]^2
\]

Therefore, for the given parameters of $m$, $n_i$ and $K$, combining Eqs. (1) and (5), we can adjust the values of $p_i$ and $p_z$ to get community structure networks with various modularity $Q$.

For the network has time-varying topology, it is necessary to characterize the network’s characteristics. We plot the curves of average degree $\langle k \rangle$, average path length $L$ and average clustering coefficient $C$ of networks changing with time. In Fig. 2, the lateral axis denotes time step, a time step is equal to one second. According to the statistics of birth and death rates of our country in recent years, we can approximately assume that the birth rate $b = 0.01$ and the natural death rate $\alpha = 0.01$. For different infectious diseases have different mortality rate and the mortality rate is affected by many factors (such as the region and personal habit), so we set the disease death rate $\beta = 0.005$ in the following simulations.

\[
\text{Fig. 2} \quad \text{The evolution curves of average clustering coefficient, average path length, average degree with the different edge numbers of networks}
\]

In addition, the network size is 1 000. As shown in Fig. 2, the larger the network's link number $K$ is, the higher the clustering coefficient $C$ is, and the smaller the average path length $L$ is. And the statistical property values remain unchanged with small $\beta$. This is because isolated nodes are not easily generated when the disease death rate is sufficiently small. The simulation results are averaged
3 The modified SIS epidemic model

Let parameters $S$, $I$ represent the density of healthy individuals and infected individuals of the entire network. $S$, $I$ are respectively the density of the susceptible and infected nodes within community $i$. Based on the classical SIS model [19], we establish a modified SIS epidemic model considering the characteristic of community structure. In addition, the circumstances of node’s birth and death are taken into consideration either in this model. Therefore, this epidemic model can be established as follows:

$$\frac{dS}{dt} = -\lambda \sum_{i=1}^{m} \langle k_i \rangle p_i I_i S_i + \langle k_i \rangle p_e \sum_{j \neq i} I_j S_i$$
$$\alpha S + b(1 - S - I) + \gamma I$$
(6)

$$\frac{dI}{dt} = \lambda \sum_{i=1}^{m} \langle k_i \rangle p_i I_i S_i + \langle k_i \rangle p_e \sum_{j \neq i} I_j S_i - \gamma I - \beta I$$
(7)

In Eq. (6), $(1 - S - I)$ denotes the density of empty sites; $p_i I_i$ and $p_e \sum_{j \neq i} I_j$ respectively are the probability of a healthy individual in community $i$ being connected with the inner-community or external-community infected individuals; and $\langle k_i \rangle = \frac{\sum_{n_i} n_i}{n_i}$ is the average number of the neighbors of one node in community $i$. As the network is homogeneous, when the epidemic spreads to the entire network, we can consider that the transmission sizes in each community are approximately the same, namely, $\sum_{j} I_j = (m-1)I_i$.

Therefore, Eqs. (6) and (7) can be written as:

$$\frac{dS}{dt} = -\lambda \frac{m^2}{(\alpha + b)} (p_i (m-1) p_e([p_i (N-m) + p_e (m-1) N] S) - \alpha S + b(1 - S - I) + \gamma I$$
(8)

$$\frac{dI}{dt} = \lambda \frac{m^2}{(\alpha + b)} (p_i (m-1) p_e([p_i (N-m) + p_e (m-1) N] S) - \gamma I - \beta I$$
(9)

Let $dS/dt = 0$ and $dI/dt = 0$, we get two steady state solutions:

$$S = \frac{b}{\alpha + b}$$
$$I = 0$$
(10)

and

$$S = \frac{m^2 (\beta + \gamma)}{\lambda ([p_i (m-1) p_e([p_i (N-m) + p_e (m-1) N] S) - \alpha S + b(1 - S - I) + \gamma I}}$$

$$I = \frac{m^2 (\beta + \gamma)}{\lambda ([p_i (m-1) p_e([p_i (N-m) + p_e (m-1) N] S) - \gamma I - \beta I$$

(11)

For the first solution, the Jacobin matrix is:

$$J = \begin{bmatrix} -\alpha - b & g - b - A \\ 0 & A - (g + b) \end{bmatrix}$$
(12)

where $A = b\lambda ([p_i (m-1) p_e([p_i (N-m) + p_e (m-1) N] S) - \alpha S + b(1 - S - I) + \gamma I$.

The determinant and the trace of $J$ are:

$$|J| = -(\alpha + b)(A - (\gamma + \beta))$$
(13)

$$\text{tr} J = A - (\alpha + b + \gamma + \beta)$$
(14)

If $|J| > 0$, then $\text{tr} J < 0$, and the solution is stable.

Then we can get the critical value:

$$\lambda_e = \frac{(\alpha + b)(\gamma + \beta) m^2}{b([p_i (N-m) + p_e (m-1) N] S) - \alpha S + b(1 - S - I) + \gamma I}$$
(15)

For the second solution, the Jacobin matrix is:

$$J = \begin{bmatrix} -\alpha - b(\alpha + b)(\gamma + \beta) - A & -\beta - b \\ (\alpha + b)(\gamma + \beta) - A & 0 \end{bmatrix}$$
(16)

where $A$ is the same as above.

Clearly, if $I = (\alpha + b)(\gamma + \beta) - A \approx 0$, we can also get $\lambda_e$:

$$\lambda_e = \frac{(\alpha + b)(\gamma + \beta) m^2}{b([p_i (N-m) + p_e (m-1) N] S) - \alpha S + b(1 - S - I) + \gamma I}$$
(17)

When $\lambda > \lambda_e$, the second solution is stable, and the disease will diffuse in the network, otherwise the disease will die out. From Eq. (17), we find that the threshold value is governed by $\alpha$, $\beta$ and $b$ in a given network.

4 Simulations and discussions

In this section, we make a set of Monte-Carlo simulations on $N$-node networks to find the relationships between epidemic size and different parameters, such as modularity coefficient, death rate, birth rate and total edge number. The following simulation results are averaged over 100 configurations with different set of random numbers $n_i$ ($i=1, 2, \ldots, m$). And for each configuration, 200 simulations are taken with one randomly chosen seed.
node initially.

Fig. 3 shows the time evolution curves of epidemic size, where $\beta$ equals to 0, 0.001 and 0.005 respectively. Some related parameters are $N=1000$, $m=10$, $K=10000$, $Q=0.3$, $\lambda=0.1$, $b=0.01$, $\alpha=0.01$. It is shown that when $\beta \neq 0$, the epidemic size increases to a peak value then decays to tend a stable value, otherwise the epidemic size keeps increase and finally reach a steady state. The existence of disease death rate can prevent the spread of the disease by decreasing the infected fraction directly. The maximum prevalence of epidemic spreading without considering nodes’ disease deaths is the largest. In addition, larger $\beta$ corresponds to smaller stable epidemic size, which agrees well with the reality.

Fig. 4 indicates the effect of the variation of birth rate on epidemic threshold and prevalence. We keep death rates $\alpha$ and $\beta$ to be constant ($\alpha=0.005$, $\beta=0.05$), and the edge number $K=40000$.

Fig. 4 shows the critical epidemic value decreases with the increase of birth rate, while the epidemic prevalence increases with the increase of birth rate. The arrows in Fig. 4 indicate the theoretic epidemic threshold calculated through Eq. (17). Eq. (17) clearly shows that the birth rate is inversely proportional to the critical value, which is consistent with the simulation results in Fig. 4. In real life, with the increase of birth rate, the density of whole population and healthy proportion increases, which makes it easier for infectious disease to diffuse.

Next, we plot the curves to indicate the influence of two kinds of death rates (natural death rate $\alpha$ and disease death rate $\beta$) on the epidemic threshold and average disease prevalence. The arrows in Fig. 5 and 6 indicate the theoretic epidemic threshold.

In Fig. 5, $\beta$ constantly equal to 0.05. For some infectious diseases, such as acquired immune deficiency syndrome (AIDS), it is necessary to consider the situation of individuals’ natural deaths. From Fig. 5, we find that the existence of natural death rate $\alpha$ is conducive to prevent the spread of the disease, and the increase of threshold and
decrease of epidemic size are expected with the increase of $\alpha$. Individuals’ natural death decreases the density of total population, thus restrains the propagation of epidemic. The arrows in Fig. 5 indicate the theoretic epidemic threshold.

Fig. 6 shows the effect of the existence of individuals’ death caused by disease on epidemic threshold. The related parameters are $b=0.005$, $Q=0.3$, $K=5000$, and $\alpha=0.005$. By comparisons, it is found that the epidemic threshold increases with the growing of $\beta$, while the epidemic size decreases with the growing of $\beta$. The existing of disease deaths can rapidly reduce the number of infected individual in populations, thus the existence of disease death rate can inhibits the epidemic spreading.

In Fig. 7, we study the effects of both modularity coefficient $Q$ and the edge number of network $K$ on the epidemic threshold. Larger $K$ represents that the individuals in network are linked more closely. It is found that the epidemic threshold decreases with the increase of the modularity coefficient of the network, and the epidemic size of the network with higher modularity coefficient is larger around the epidemic threshold, while the inverse situation occurs when the infection rate is far greater than the threshold.

![Fig. 7 The relationship between $I_\infty$ and $\lambda$ with different modularity coefficient $Q$ and edge number $K$.](image)

This is because the infectious disease is mainly transmitted within the community, and when the propagation rate is sufficiently, the infectious disease spreads throughout the network through the edges between communities. The edge density of network with higher modularity coefficient is small, this is not conducive to the spread between communities, thereby reducing the spreading size of the entire network. In addition, the epidemic threshold has inverse correlation with the total edge number $K$. This is consistent with the real network circumstances.

## 5 Conclusions

Considering the circumstances of node’s birth and death that may exist in real networks, a modified epidemic model based on the classical SIS model is proposed in a community structure network. An approximate formula for the epidemic threshold is obtained by mathematical analysis to find the relative relationships between different parameters. Then the stability of endemic equilibrium is analyzed. The simulations in this study illustrate that the epidemic threshold $\lambda$ increases with the increase of the death rate (natural death or disease death), while it decreases with the increase of the birth rate, modularity coefficient and edge number. Through this study, it is helpful to predict the spreading trend of some infectious diseases that may cause the deaths of individuals (such as Ebola and H7N9) more accurately than ever before.

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