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Backward bifurcation and local dynamics of epidemic model on adaptive networks with treatment

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ABSTRACT

A Susceptible–Infected–Susceptible (SIS) model with limited treatment capacity on adaptive networks is presented to study the effects of the treatment on epidemic spreading and local stability and bifurcation behavior of the system. We derive the occurring condition of backward bifurcation or forward bifurcation at the disease-free equilibrium in the system. By theoretical analysis and numerical simulations, it is found (i) if a backward bifurcation occurs at the disease-free equilibrium of the system, then bistability exists regardless of the size of the capacity, and (ii) if a forward bifurcation occurs at the disease-free equilibrium of the system, then bistable endemic equilibria exist when the capacity is low. It is also shown that the range of bistability becomes smaller as the capacity increases when the capacity is smaller, otherwise unchanges for larger treatment capacity.

1. Introduction

Modeling of epidemic spreading and the asymptotic behavior of these epidemic models have been studied in recent years [1]. Pastor-Satorras and Vespignani studied SIS model on scale-free networks [2,3], showing that the epidemic threshold tend to 0 on an infinitely large scale-free network and remains to be low and decreases with increasing network size in the finite size scale-free networks. Zhou et al. [4] proposed a Susceptible–Infected (SI) model with identical infectivity on scale-free networks. Barrat et al. [5] studied dynamical processes on heterogeneous networks. Wang et al. [6] considered SIS model and studied the impact of mass media for the contact structure of a population network. Wang et al. [7] studied the global analysis of an SIS model with an infective vector on complex network. Hu et al. [8] discussed stability behaviors of discrete models. Yang et al. [9] and other experts [10–13] also considered stability on stochastic networks with different cases.

For many mathematical epidemiological models, it is common that the basic reproduction number R_0 is an important threshold parameter [14]. The basic reproduction number is the expected number of secondary cases produced, in a completely susceptible population, by a single infective individual [14]. If $R_0 < 1$, there is an asymptotically stable disease-free equilibrium, and the disease cannot grow. If $R_0 > 1$, there is an asymptotically stable endemic equilibrium and the disease persists and can invade the population. Thus, there usually occurs forward bifurcation at $R_0 = 1$ in the system [15]. A backward bifurcation can occur due to multiple groups with different susceptibilities and nonlinear incidence, etc. [16]. In this case, $R_0 < 1$ is not enough to control the spreading of infection and describe the elimination of disease, and R_0 must be reduced below the leftmost point on the backward bifurcation curve for which the critical value of an endemic equilibrium exists. Therefore, it is very important to identify forward and backward bifurcations for the control of diseases.

The treatment is one of the important and effective methods to prevent and control the spreading of diseases such as measles, tuberculosis and flu, etc. In classical epidemic models, the treatment rate of the infective is assumed to be proportional to the number of the infective individuals. But in fact, the treatment capacity is usually limited. Every country or city has usually a suitable treatment capacity. If it is too small, the risk of disease outbreak is larger. If it is too lager, the country or city will pay the additional cost. Thus, it is very important to determine a suitable treatment capacity. Wang and Ruan [17] adopted a constant treatment rate to understand the effect of limited treatment capacity. Wang [18] proposed the piecewise linear treatment function based on Susceptible–Infected–Removed (SIR) model as follows:

$$h(I) = \begin{cases} \delta I & 0 \le I \le I_0 \\ m = \delta I_0 & I > I_0 \end{cases} \tag{1.1}$$

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where I_0 is the infective level when treatment capacity reaches maximal value. This means that the treatment rate is proportional to the density of infected nodes when treatment capacity does not reach maximal value, otherwise takes the maximal capacity. Eq. (1.1) implies the situation where the number of hospital beds is limited. It also stands for the case where medicines are not sufficient. It was shown that there occurs backward bifurcation and exists bistable endemic equilibria if the capacity is low.

The piecewise linear treatment function has been used by some other researchers [19–22]. For instance, Li et al. [19] and Hu et al. [20] considered an SIR model with nonlinear incidence rate and the treatment function (1.1). Zhang and Liu [21] discussed an SIS model with general incidence rate and the treatment function (1.1). Hu et al. [22] studied an SIR epidemic model with standard incidence rate and the treatment function (1.1).

Other types of treatment functions have also been proposed. For example, Zhang and Liu [23] adopted a continuous and differentiable saturated treatment function $h(I)=\alpha I/(1+\beta I)$, $\alpha>0$ and $\beta\geq 0$, and found that backward bifurcation occurs when the delayed effect for treatment is strong. Eckalbar [24] proposed quadratic treatment function $h(I)=\max\{\gamma I-\mu I^2,0\}$, $\gamma>0$ and $\mu>0$, and found that the system has four equilibria with possible bi-stability, backward bifurcation and limit cycles.

The studies above are based on static networks where network topologies are not changed. But in real world, network topologies are changing with the states of nodes and vice versa. In this paper, we study the effect of limited treatment capacity for epidemic spreading in adaptive networks where the network topologies are changed. Recently studies have begun to focus on the epidemic dynamics on adaptive networks [25–28], in which there exists a feedback loop between the dynamics of networks and the dynamics on networks. Gross et al. [25] firstly proposed SIS model on adaptive networks, and found that adaptive rewiring can lead to bistability. The Susceptible–Infected–Removed–Susceptible (SIRS) model on adaptive networks was studied by Shaw and Schwartz [26]. Marceau et al. [29] and Taylor et al. [30] adopted the improved effective degree formalism and more precisely explored SIS epidemic dynamics on adaptive networks. Shaw and Schwartz [31] used a random vaccination to control disease spreading on adaptive networks. Juher et al. [32] studied the early dynamics of the SIS model and local stability analysis on adaptive networks.

In this paper, we study epidemic dynamics and asymptotic behavior on adaptive networks with limited treatment capacity. The rest of the paper is as follows. In Section 2, a modified SIS model with limited treatment capacity on adaptive networks is proposed. In Section 3, we analyze the modified SIS model and derive the existing condition of equilibria and bifurcations. In Section 4, we analyze the local stability of equilibria. Some numerical simulations are shown in Section 5. Finally, we give brief conclusion.

2. The SIS model with treatment

For the standard SIS epidemic model, the population can be divided into two types: susceptible or infected individuals. At each time step, each susceptible node is infected with probability p if it is connected to an infected individual; On the other hand, each infected node recovers and becomes susceptible node with probability r.

In this paper, each individual of the population can be one of the two states: susceptible S, infected I. In order to study epidemic spreading and topological evolution, we consider a random network with undirected links. Each of the nodes represents an individual, while each of the links stands for the physical interactions between nodes along which the disease can potentially spread. The average degree of the network is $<k>$. Double connections and self-connections are not allowed. Let S, I be the densities of susceptible, infected nodes at time t. The per capita densities of SS-links, SI-links and II-links can be denoted by l_{SS}, l_{SI} and l_{II}, respectively. These variables satisfy the following condition:

$$S+I=1$$
$$l_{SS}+l_{SI}+l_{II}=\frac{<k>}{2} \tag{2.1}$$

Assume that the rate for a susceptible node becomes infected as p along the SI-links and the natural recovery rate for an infected node as r. It is assumed that the nature recovery rate is unchanged as the treatment capacity varies. In this paper, we adopt the piecewise linear treatment functions (1.1). Based on the above assumptions, we have the following differential equation with treatment for node:

$$\frac{d}{dt}I=pl_{SI}-rI-h(I) \tag{2.2}$$

The equation contains the variable l_{SI} and therefore does not yet constitute a closed model. We will apply the moment closure approximation proposed in the references [33,34] and describe the time evolution of links density. In the pair approximation the density of all triples $l_{abc},(a,b,c\in[S,I])$ are approximated by $l_{abc}=l_{ab}l_{bc}/b$.

Assume that the susceptible nodes can rewire their links with probability ω. For every SI-links, the susceptible node breaks the link to the infected node and forms a new link to another randomly selected susceptible node. We can obtain the following differential equations for links:

$$\frac{d}{dt}l_{II}=pl_{SI}\left(1+\frac{l_{SI}}{S}\right)-2rl_{II}-2h(I)\frac{l_{II}}{I} \tag{2.3}$$

$$\frac{d}{dt}l_{SS}=-\frac{2pl_{SI}l_{SS}}{S}+rl_{SI}+h(I)\frac{l_{SI}}{I}+\omega l_{SI} \tag{2.4}$$

Eq. (2.3) describes the time evolution of density of II-link. II-links can be destroyed if the recovering node or the under treatment node was part of such links. The expected number of II-links in which a given infected node is involved is $2l_{II}/I$. Taking the rate of recovery event and treatment event into account, the II-links are destroyed at the total rate $2rl_{II}-2h(I)l_{II}/I$. In an infection event, epidemic spreads across SI-links. Therefore every infection event will create at least one II-link. However, additional II-links may be created if the newly infected node has other infected neighbors in addition to the infecting node. The II-links are treated at the total rate $pl_{SI}(1+l_{ISI}/l_{SI})=pl_{SI}(1+l_{SI}/S)$. Eq. (2.4) describes the time evolution of density of SS-link. Similar to the above description, SS-links are destroyed at the rate $pl_{SSI}=pl_{SI}l_{SS}/S$. Meanwhile, SS-links are created by treatment at the rate $h(I)l_{SI}/I$ and by recovery at the rate rl_{SI}. In addition SS-links can also be created by rewiring SI-links at the rate ωl_{SI}.

With the condition (2.1), Eqs. (2.2)–(2.4) can be simplified by

$$\begin{cases}\frac{d}{dt}I=pl_{SI}-rI-h(I)\\ \frac{d}{dt}l_{SI}=\frac{2pl_{SI}}{1-I}\left(\frac{<k>}{2}-l_{SI}-l_{II}\right)-pl_{SI}\left(1+\frac{l_{SI}}{1-I}\right)-(r+\omega)l_{SI}+2rl_{II}-h(I)\frac{l_{SI}}{I}+2h(I)\frac{l_{II}}{I}\\ \frac{d}{dt}l_{II}=pl_{SI}\left(1+\frac{l_{SI}}{1-I}\right)-2rl_{II}-2h(I)\frac{l_{II}}{I}\end{cases}. \tag{2.5}$$

3. The existence of equilibrium and bifurcations

In the steady state, let $D=\{(I,l_{SI},l_{II})|0\leq I\leq 1,\ 0\leq l_{SI},\ l_{II}\leq(<k>/2)\}$, which is invariant set under nonnegative initial