Epidemic Spread in Weighted Scale-Free Networks *

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We investigate the detailed epidemic spreading process in scale-free networks with link weights that denote familiarity between two individuals. It is found that the spreading velocity reaches a peak quickly then decays in a power-law form. Numerical study exhibits that the nodes with larger strength is preferential to be infected, but the hierarchical dynamics are not clearly found, which is different from the well-known result in the unweighed network case. In addition, also by numerical study, we demonstrate that larger dispersion of weight of networks results in slower spreading, which indicates that epidemic spreads more quickly on unweighted scale-free networks than on weighted scale-free networks with the same condition.

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Many social, biological, and communication systems can be properly described as complex networks with vertices representing individuals or organizations and links mimicking the interactions among them. Recently, the ubiquity of a power-law degree distribution in real-life networks has attracted much attention.[1] Examples of such networks (scale-free (SF) networks) are numerous: these include the Internet, the World Wide Web, social networks of acquaintance or other relations between individuals, metabolic networks, integer networks, food webs, etc.[2] The ultimate goal of the current studies on topological structures of networks is to understand and explain the workings of systems built upon those networks, for instance, to understand how the topology of the World Wide Web affects Web surfing and search engines, how the structure of social networks affects the spread of diseases, information, rumors or other things, how the structure of a food web affects population dynamics, and so on.

Recent studies on epidemic spreading in SF networks indicate a particular relevance in the case of networks characterized by complex topologies and very heterogeneous structures[1,3] that in many cases present us with new epidemic propagation scenarios,[4,5] such as absence of any epidemic threshold,[4] hierarchical spread of epidemic outbreaks,[5] and so on. The new scenarios are of practical interest in computer virus diffusion and the spreading of diseases in heterogeneous populations. Furthermore, they also raise new questions on how to protect the networks and find optimal strategies for the deployment of immunization resources.[6] However, so far, studies of epidemic spread just focus on unweighted SF networks, and a detailed inspection of epidemic spreading process in weighted SF networks is still missing while real networks, such as population and Internet, are obviously scale-free and with link weights that denote familiarity between two individuals (such as people or computers), respectively. One can easily take cognizance of how the link weights affect the epidemic spreading process. For instance, if your little son gets flu, then you will be infected in all probability, since you two contact each other very frequently (i.e. of large familiarity). In contrast, it is impossible that you will be infected by your unfamiliar colleague just because of saying hello to him this morning.

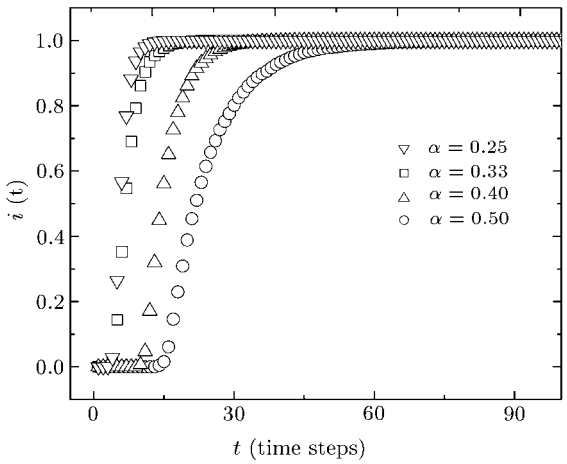

Fig. 1. Density of infected individuals versus time in a BBV network with $N = 10^4$, $\delta = 3.0$, $\omega_0 = 1.0$ and $m = 3$, the four numerical curves $i(t)$ correspond to parameter $\alpha = 0.5$, 0.4, 0.33 and 0.25, respectively, form bottom to top.

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In this Letter, we intend to provide a first analysis of the time evolution of epidemic spreading in weighted SF networks. The weighted SF network model used is one of the most well-known model introduced by Barrat, Barthelemy, and Vespignani (BBV networks),[7] whose degree, strength and weight distributions are power-law distributions with heavy tails. The BBV model suggests that two main ingredients of self-organization of a network in a weighted scale-free structure are strength preferential attachment and weight dynamics. These point to the facts that most networks continuously grow by the addition of new vertices, new vertices are preferentially attached to existing vertices with larger strength, and the creation of new links will introduce variations of the existing weight distribution. More precisely, the weight of each new edge is fixed to value ω_0; if a new vertex linked to an existing vertex i, then the local rearrangement of weights between i and its neighbours j will be taken according to the simple rule,

$$\omega_{ij} \to \omega_{ij} + \Delta\omega_{ij}, \tag{1}$$

where

$$\Delta\omega_{ij} = \delta\frac{\omega_{ij}}{s_i}, \tag{2}$$

with s_i being the strength of node i, expressed by $s_i = \sum_j \omega_{ij}$. This rule considers that the establishment of a new edge of weight ω_0 with the vertex i induces a total increase of traffic δ that is proportionally distributed among the edges departing from the vertex according to their weights. Since BBV networks are of the same properties (e.g. power-law distribution of degree, strength and weight) as many social networks (e.g. friendship networks and scientists collaboration networks) and technical networks (e.g. the Internet and WWW), it is reasonable to investigate epidemic spreading on BBV networks.

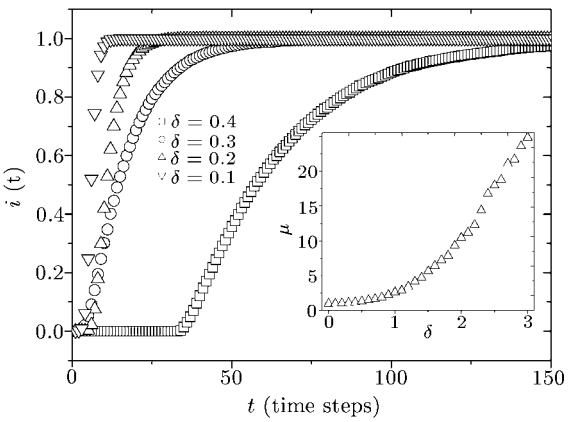

Fig. 2. Density of infected individuals versus time in a BBV network with $N = 10^4$, $\alpha = 2.0$, $\omega_0 = 1.0$ and $m = 3$. The four numerical curves $i(t)$ correspond to parameter $\delta = 0.4$, 0.3, 0.2 and 0.1 respectively, from bottom to top. The inset shows the relationship between the dispersion of weight (μ) and the value of δ.

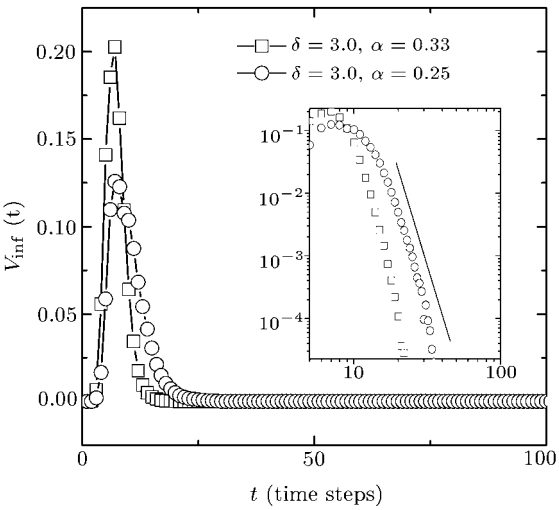

Fig. 3. Spreading velocity at each time t in a BBV network with $N = 10^4$, $\delta = 3.0$, $\omega_0 = 1.0$ and $m = 3$, when $\alpha = 0.33$ (square) and $\alpha = 0.25$ (circle). The inset shows the curves in a log-log plot. The data are averaged over 200 experiments.

In order to study the dynamical evolution of epidemic spreading we shall focus on the susceptible-infected (SI) model in which individuals can be in two discrete states, either susceptible or infected.[8] Each individual is represented by a vertex of the network and the links are the connections between individuals along which the infection may spread. The total population (the size of the network) N is assumed to be constant if $S(t)$ and $I(t)$ are the number of susceptible and infected individuals at time t, respectively, then $N = S(t) + I(t)$. In weighted networks, we define the infection transmission by the spreading rate,

$$\lambda_{ij} = \left(\frac{\omega_{ij}}{\omega_M}\right)^{\alpha}, \quad \alpha > 0, \tag{3}$$

at which susceptible individual i acquires the infection from the infected neighbour j, where α is a positive constant and ω_M is the largest value of w_{ij} in the network. Obviously, more familiar two individuals (i.e. with larger weight) may infect each other with greater probability. According to Eq. (3), one can quickly obtain the probability that an susceptible individual i will be infected at the present time step is

$$\lambda_i(t) = 1 - \prod_{j\in N_i(t)} (1 - \lambda_{ij}), \tag{4}$$

where $N_i(t)$ is the set of all the infected neighbours of node i at time t.

We start by selecting one vertex randomly and assume it is infected. The diseases or computer virus will spread in the networks in according with the rule of Eq. (4). In Fig. 1, we plot the density of infected individuals versus time in a BBV network with $N = 10^4$, $\delta = 3.0$, $\omega_0 = 1.0$ and $m = 3$. Since $\dfrac{\omega_{ij}}{\omega_M} \leq 1$, the smaller the α value is, the more quickly the infection

spreads. It is natural that a larger value of δ will induce larger dispersion of weight of networks. Then, a direct question is how the value of δ impacts epidemic spreading behaviour. In Fig. 2, we show that epidemic spreads more quickly while δ is smaller. In other words, larger dispersion of weight of networks results in slower spreading. This means that epidemic spreads more quickly on unweighted scale-free networks than that on weighted scale-free networks under the same condition.

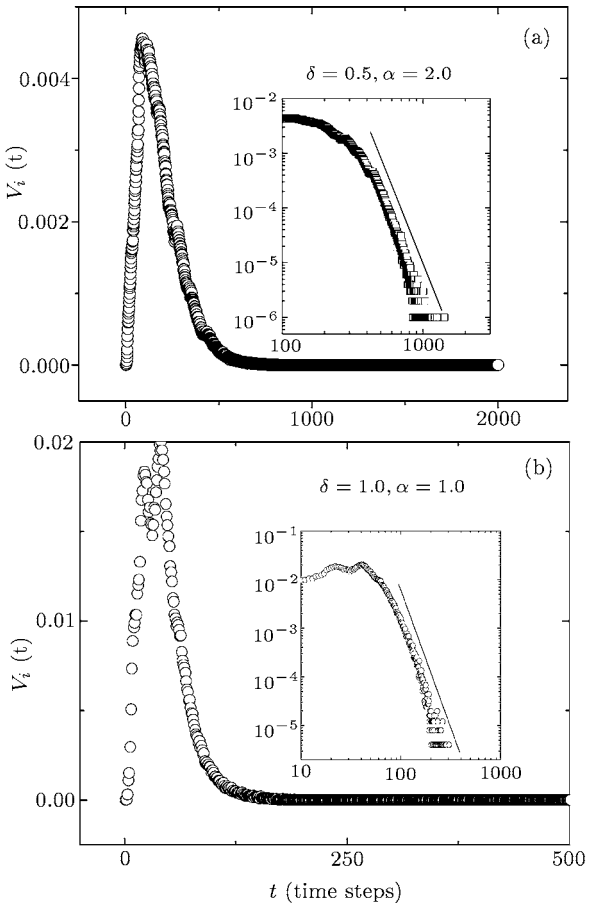

Fig. 4. Consider the cases of $\delta < 1.0$ and $\delta = 1.0$. Here (a) and (b) show the spreading velocity at each time t in a BBV network with $N = 10^4$, $\omega_0 = 1.0$ and $m = 3$, when $\delta = 0.5$, $\alpha = 2.0$ and $\delta = 1.0$, $\alpha = 1.0$, respectively. The inset shows the curves in a log–log plot. The data are averaged over 200 experiments.

Obviously, all the individuals will be infected in the limit of long time as $\lim_{t\to\infty} i(t) = 1$. For the sake of finding optimal strategies to protect individuals from being infected, we will study the details of spreading velocity at the outbreak moment. The spreading velocity is defined as

$$V_{\rm inf}(t) = \frac{di(t)}{dt} \approx \frac{I(t) - I(t-1)}{N}, \qquad (5)$$

where $i(t) = \dfrac{I(t)}{N}$. We account the number of newly infected vertices at each time step and report the spreading velocity in Fig. 3. Apparently, the spreading velocity goes up to a peak quickly that is similar to the unweighted network cases,[5] leaving us very short response time to develop control measures. Moreover, and what is new and interesting, the velocity decays following a power-law form after the "peak time". At the moment of infection outbreak, the number of infected individuals is very small; a very long time after the outbreak, the number of susceptible individuals is also very small. Thus, when t is very small (close to zero) or very large, the spreading velocity is close to zero; one can see the corresponding simulation result in Fig. 3. One may think that the velocity follows the power-law behaviour just because of the extreme case of $\delta > 1.0$. Now we consider the cases of $\delta < 1.0$ and $\delta = 1.0$. Figure 4 shows spreading velocity at each time t in a BBV network with $N = 10^4$, $\omega_0 = 1.0$ and $m = 3$, when $\delta = 0.5$, $\alpha = 2.0$ and $\delta = 1.0$, $\alpha = 1.0$, respectively. It is obvious that epidemic spreading behaviour does not show sensitive dependence on the parameter δ, the reason will be explored deeply in our future studies.

In order to give a more precise characterization of the epidemic diffusion through the weighted networks, we measure the average strength of newly infected vertices at time t, defined as

$$\bar{S}_{\rm inf}(t) = \frac{\sum_s s[I_{\rm s}(t) - I_{\rm s}(t-1)]}{I(t) - I(t-1)}, \qquad (6)$$

where $I_{\rm s}(t)$ is the number of infected vertices with strength s. Figure 5 shows the average strength of newly infected vertices $\bar{S}_{\rm inf}(t)$ as a function of time t, and the curves exhibit that $\bar{S}_{\rm inf}(t)$ displays power-law behaviour for large t, $\bar{S}_{\rm inf}(t) \propto t^{-\gamma}$, which is remarkably different from the clear hierarchical feature on unweighted networks.[5]

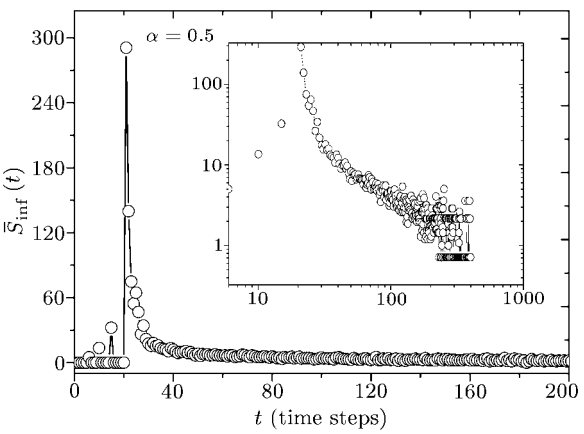

Fig. 5. Behaviour of average strength of the newly infected vertices at time t for the SI model spreading in a BBV network with $N = 10^4$, $\delta = 3.0$, $\omega_0 = 1.0$ and $m = 3$. The inset shows that $\bar{S}_{\rm inf}(t)$ represents power-law behaviour, $\bar{S}_{\rm inf}(t) \propto t^{-\gamma}$.

It is explicit that the individuals with larger strength are much more dangerous when they are infected, rather than the ones with smaller strength,

thus if one want to protect most individuals being infected, the susceptible individuals with larger strength must be protected foremost. In Fig. 5, one can find that the individuals with larger strength are preferential to be infected, which means that there is little time leaving us to find the "large individuals" and to isolate them. Therefore, at the outbreak moment of disease or computer virus, the dense crowd or pivotal servers must be protected primarily. Of course, the outcome is not good news for practical operators, but it may be related to the development of containment strategies.

In summary, we have studied the epidemic spreading process in BBV networks. The present results provide a clear picture of the infection propagation in weighted SF networks. The numerical studies show that spreading velocity $V_{\rm inf}(t)$ and average strength of newly infected vertices $\bar{S}_{\rm inf}(t)$ present a power-law time behaviour for large t, which is remarkably different from infection propagation in unweighted networks. Also by numerical study, we demonstrate that larger dispersion of weight of networks results in slower spreading, which indicates that an epidemic spreads more quickly on unweighted scale-free networks than that on weighted scale-free networks under the same condition. These results indicate that not only the topological structures of networks but also the link weights affect the epidemic spreading process. Furthermore, the detailed study of behaviour of the average strength of the newly infected vertices may be related to development of containment strategies.

However, up to now, there have been a great many important and fundamental problems that puzzle us and have not been referred to in the present study. Some of them have been partially solved and will be publicized in further publications, and others are still unanswered. As an end of this Letter, we list some of them. How should one analyse the average density of infected individuals versus time at the outbreak moment in weighted SF networks, and likewise the dynamic behaviour after "peak time"? Is the mean-field theory appropriate to solve this problem? How should one design an optimal containment strategy, and assess the effectiveness of various strategies, such as to protect vertices at random, to protect vertices purposefully, to cut off links at random, to cut off links purposefully, and so on?

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