Epidemic reemergence in adaptive complex networks

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The dynamic nature of a system gives rise to dynamical features of epidemic spreading, such as oscillation and bistability. In this paper, by studying the epidemic spreading in growing networks, in which susceptible nodes may adaptively break the connections with infected ones yet avoid being isolated, we reveal a phenomenon, epidemic reemergence, where the number of infected nodes is incubated at a low level for a long time and then erupts for a short time. The process may repeat several times before the infection finally vanishes. Simulation results show that all three factors, namely the network growth, the connection breaking, and the isolation avoidance, are necessary for epidemic reemergence to happen. We present a simple theoretical analysis to explain the process of reemergence in detail. Our study may offer some useful insights, helping explain the phenomenon of repeated epidemic explosions.

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I. INTRODUCTION

Infectious diseases have caused tremendous losses in human health and lives and they remain as a serious threat to mankind today. To resist infectious disease, theoretical investigations have been engaged to study epidemic behaviors [1–5] and immunization strategies were suggested to prevent epidemic spreading when vaccine resources are limited [6–9]. Recently, large-scale agent-based simulations have been applied to get more detailed descriptions of disease outbreaks [10–12]. A prominent development among these studies was to abstract the complex social relations into networks, where nodes represent individuals and links represent the contacts among them. It is found that the basic reproductive number R_0, a key factor determining whether a disease can spread out or not, depends strongly on the variance of the distribution of the contacts [2,3,13]. Extensive results show that the social contacts typically have a fat-tail degree distribution where a small number of people have a very large number of contacts [14,15]. This property typically leads to a much higher R_0 than that in a network with the same average degree but homogeneous degree distribution [2].

The nontrivial features of social networks such as small-world property and fat-tail degree distribution, and the complexity of the dynamics of infectious diseases, lead to some interesting properties of epidemic spreading. For example, it is found that for a linearly growing network, the evolution of the number of the infected nodes has oscillatory behaviors when the susceptible-infected-recovered (SIR) model is adopted [16]. An adaptive mechanism is studied in Ref. [17], where a susceptible individual may avoid contact with his infected neighbors and rewire these contacts to other susceptible individuals. An important observation is that the interplay of the epidemic dynamics and the network topology may cause a bistable phenomenon. That is, the persistence of a disease is difficult when the infection density is low, yet may enter

an endemic state when the infection density is high. Network growth is a fundamental property for any healthy system [18] and adaptive changes widely exist in most systems when faced with infection. Adaptations to avoid an undesirable outcome can sometimes postpone the onset of the undesirable outcome, at the same time making it more severe [19,20]. Hence it is necessary to study the influences of both network growth and adaptive dynamics to the dynamics of epidemic spreading.

In this paper, we study epidemic spreading in linearly growing networks assuming that the susceptible nodes may break the contacts with the infected nodes. Considering the fact that in general an individual cannot survive when he or she is fully isolated in a modern society, the contact breaking process takes place only when both of the two end nodes of a contact still have other neighbors. Interestingly, we observe an epidemic reemergence phenomenon, where the number of infected nodes may stay at a low level for a long time and then surges to a high level. The process may repeat for a long time before the disease finally dies out. In Sec. II we present the epidemic model. Simulation results are presented in Sec. III. In Sec. IV we give some theoretical analysis to explain our observations. Section V presents some further discussion and concludes the paper.

II. MODEL

Consider a Barabasi-Albert (BA) model [14] with N nodes as the initial network, where the average nodal degree $\langle k \rangle = 2m$ and m is the number of links attached by each newly added node. We use the SIS model to describe the epidemiological process, which is widely adopted to describe infectious diseases [1,19,21,22]. In this model agents can be in two distinct states: susceptible or infected. A susceptible agent may get infected if he has infected neighbors. Suppose a susceptible node has neighbors, of which k_{inf} are infected,

and the probability of having contagion with each infected neighbor is p. Then the susceptible node may get infected with probability $1 - (1 - p)^{k_{\inf}}$, which is approximately $pk_{\inf}$ when p is small. At the same time, each infected agent will become susceptible at a rate of r. In this paper, we set $r = 0.04$ unless otherwise specified.

III. SIMULATION RESULTS

We first consider the case in which the network is continually growing during epidemic spreading. Assume on average there are α susceptible nodes joining the networks at each time step and each new node brings m links connected to the existing nodes in a preferential attachment manner. We assume that the newly added nodes know the infectious states of the existing nodes. Thus they only connect to susceptible ones. The probability that a susceptible node e is connected to a newly added node is $mk_e^S / \sum_{e'} k_{e'}^S$, where k_e^S denotes the degree of the susceptible node e. Such a network growth model has also been adopted in Ref. [23]. Figure 1 shows the relation between the fraction of infected nodes i after transient process and the disease transmission probability p for different values of α. In this figure, all the curves overlap completely. Therefore, the fraction of infected nodes will not be influenced when we change the network growing rate.

Now we consider the case that susceptible nodes may remove their links connected to infected nodes when the network is not growing. Specifically, in each time step each susceptible node may break the link connected to an infected neighbor at a probability ω. Considering that an individual seldom can exist in isolation in a modern society, we set the constraint that a link removal can happen only when both of the two end nodes of the link have other neighbors. We term such a constraint as isolation avoidance. Obviously, without isolation avoidance, the link removal process can finally lead the network to a disease-free status, since it is equivalent to reducing the effective infection rate [17], in the extreme case to zero when all the links to the infected nodes are removed. With the isolation avoidance, however,

FIG. 2. (Color online) The evolution of the number of infected nodes in the BA model without network growth. We set $p = 0.09$ and all the other parameters are the same as those in Fig. 1. The red hollow circles (black hollow squares) represent the results when the isolation avoidance is (is not) considered.

the link-removal process cannot guarantee the clearance of the disease, since the mechanism lets each node retain at least one connection and a disease may spread out in the condition of $p > r$ as the reproductive number $R_0 > 1$. Figure 2 shows the evolution of the number of infected nodes for the cases with and without isolation avoidance, respectively. The infectious disease disappears after a transient process when the isolation avoidance mechanism is not considered. However, when the isolation avoidance is adopted, the number of infected nodes may remain at a low level rather than disappear.

Next we consider the case in a growing network where susceptible nodes may remove their links connected to infected nodes without isolation avoidance. Figure 3 shows the
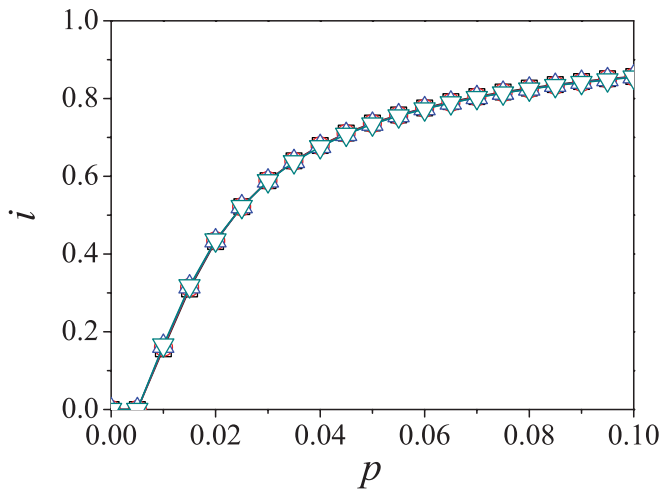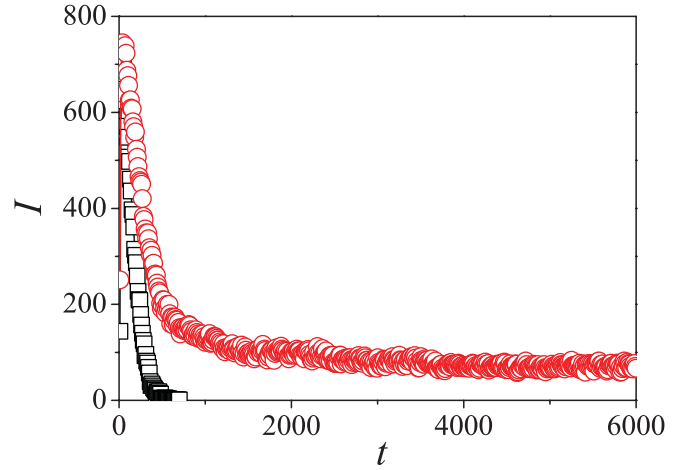

FIG. 1. (Color online) The fraction of infected nodes after transient time as a function of parameter p for different growing rates $\alpha = 0.02, 0.05, 0.1,$ and 0.2, where $r = 0.04$ and $m = 2$.
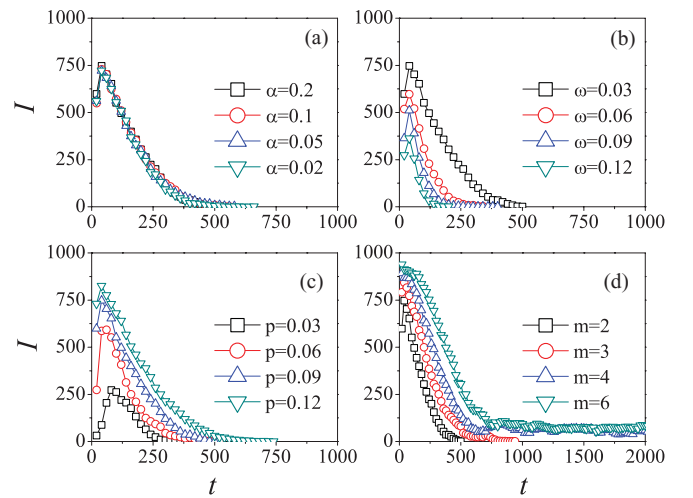

FIG. 3. (Color online) Evolution of the number of infected nodes in a network with growth and link removal but without isolation avoidance. (a) Results when $\alpha = 0.2, 0.1, 0.05,$ and 0.02 for $m = 2$, $\omega = 0.03$, $p = 0.09$. (b) Results when $\omega = 0.03, 0.06, 0.09,$ and 0.12 for $m = 2$, $p = 0.09$, $\alpha = 0.2$. (c) Results when $p = 0.03, 0.06, 0.09,$ and 0.12 for $m = 2$, $\alpha = 0.2$, $\omega = 0.03$. (d) Results when $m = 2, 3,$ $4,$ and 6 for $\alpha = 0.2$, $\omega = 0.03$, $p = 0.09$.
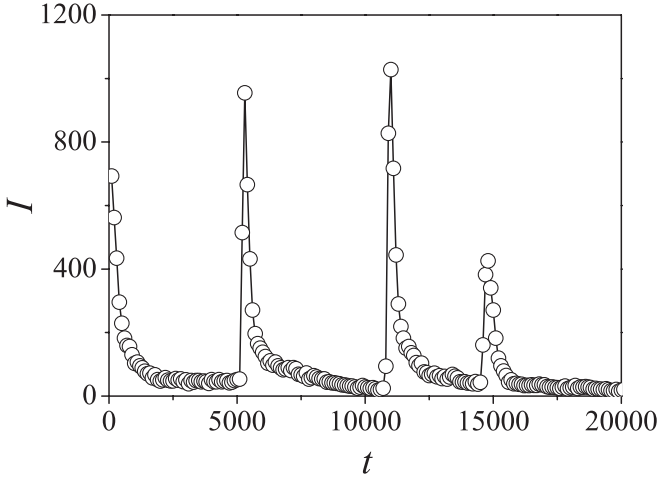
FIG. 4. The evolution of the number of infected nodes when the network growth, the link-removal process, and isolation avoidance are all considered, $\alpha = 0.2$, $\omega = 0.03$, $p = 0.09$, and $m = 2$.

FIG. 5. Evolution of the number of infected nodes in the giant component, I_G, the size of giant component, S_G, and the number of components, N_C. Parameters are the same as those in Fig. 4.

evolution of the number of infected nodes, I, with different parameters. It shows that network growing rate α does not have significant influence to epidemic dynamics; and in all the cases, the infection will finally either become extinct or stay at a low level.

Finally we study the case in which network growth, link-removal process, and the mechanism of isolation avoidance are all involved. Interestingly, we observe the phenomenon of epidemic reemergence. As shown in Fig. 4 where $\alpha = 0.2$, $\omega = 0.03$, $p = 0.09$, and $m = 2$, the number of infected nodes stays at a low level for a long time, then suddenly surges to a high level before decaying to a low level again. This process may repeat at certain times before the infection finally dies out.

This observation can be explained as follows: on one hand, the link-removal process can suppress the epidemic spreading, while the isolation avoidance lets each node have at least one neighbor. The interplay between these two processes makes the number of infected nodes remain at a low level. On the other hand, the newly added nodes connect to susceptible nodes when they join the network. Due to the small number of infected nodes and the isolation avoidance, the infection cannot easily reach these newly added nodes. Therefore, the newly added nodes can cumulate, connecting susceptible nodes into a large component. Once the infection invades into this component, however, the infection size can quickly erupt.

To reveal the mechanism of the epidemic reemergence in more detail, we illustrate the evolution of the number of infected nodes in the giant component, I_G, the size of the giant component, S_G, and the number of components, N_C, respectively, in Fig. 5. Referring to Fig. 4, we can see that when the number of the infected nodes I is very small, the size of the giant component increases linearly along time and I_G is about zero. This means that the giant component is basically in the disease-free status. Meanwhile, N_C decreases along time, meaning that the newly added nodes continually merge the existing components into larger ones. However, once the disease invades into the giant component, the infection may quickly spread over the whole component, leading to a high
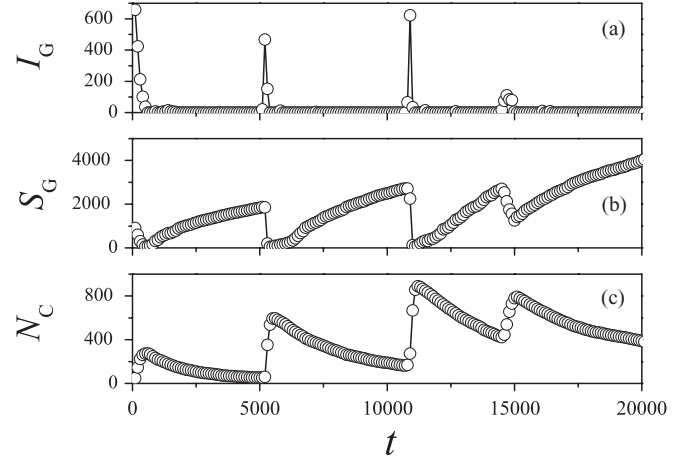
value of I_G. Then the component quickly breaks into small pieces due to the link removals, evidenced by an increasing number of N_C.

The growth of a large giant component plays an important role in inducing the reemergence phenomenon. For a component that is totally composed of susceptible nodes, referred as an S component, the size of it may keep growing during the network growing process. However, if a newly added node connects the S component to a component containing infected nodes, referred as an I component, the infection may reach the S component by going through the newly added node. If the size of the S component is small, it may not make a large impact when it is infected. When a large S component is infected, however, the disease may quickly spread over it, causing a sharp increase in the number of infected nodes.

It is interesting to have a closer look at how the small I components survive the long interepidemic periods and how the infection finally invades a large S component (in most cases, the giant component). We take the second explosion, which happens at around $t = 5000$ as an example. At $t = 5034$, there are totally 46 infected nodes left in the network and they belong to 5 I components. One of these I components is an 18-node star network, which is composed of 11 infected nodes including the hub of the star and 7 susceptible nodes. This explains how the infection remains endemic: the links in the I component cannot be removed due to isolation avoidance. At $t = 5035$, the 18-node I component is connected to the giant component through one of its susceptible nodes. A few time steps later the infection invades the giant component, leading to an epidemic. In fact, similar observations apply to all the other epidemics: the long-lived I components seeding the epidemics almost always have star or starlike topologies. The isolation avoidance mechanism prevents such I components from being further fragmented. The infection therefore has a chance to survive over a long time. Finally, by chance the I component may be connected to the giant or big S component, which may lead to an explosion.

Figure 6 illustrates snapshots of network structures right before and after an epidemic explosion. Specifically, when $t = 5000$, which is just before the epidemic explosion, as we
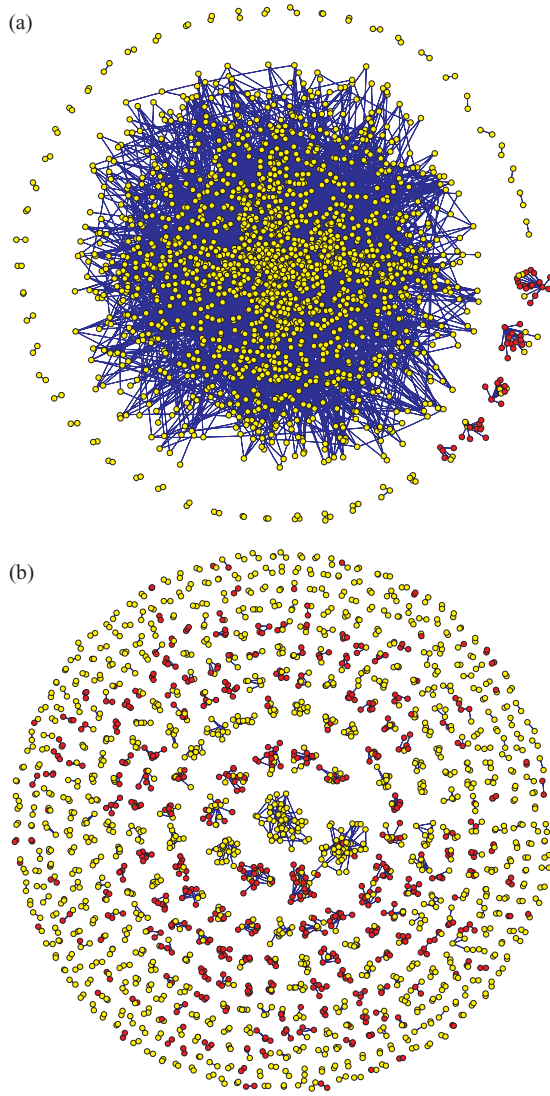
(a)

(b)

FIG. 6. (Color online) Snapshots of the network topology when (a) $t = 5000$ and (b) $t = 5400$. The yellow (red) circles denote the susceptible (infected) nodes.

can see in Fig. 6(a), there is a giant component containing most of the network nodes and all the nodes in this component are susceptible. Meanwhile, a small number of infected nodes exist in several small components. This observation is accordant with our previous discussion that due to the network growth and isolation avoidance, the network can form a giant component that is basically disease free, while a small number of infected nodes may remain in small clusters. Figure 6(b) shows the network structure when $t = 5400$, which is right after the epidemic explosion. We see that the network breaks into many small pieces, most of which with no more than five nodes. In this stage, on one hand, the network still has many infected nodes, thus some I components may be further decomposed to suppress the infection spreading. On the other hand, the network has several S components with relatively large size, which indicates that a new giant component starts to form.

Note that a reconnection between an I component and the giant or big S component does not always lead to an explosion. In fact, in most cases, the giant or big component is

finally disconnected from any infected nodes again without any noticeable increase in the number of infected nodes in between. An explosion typically only happens when the infection manages to reach a high-degree hub in the giant component. In the next section, we will show in more detail the strong randomness in the reconnection process.

IV. THEORETICAL ANALYSIS

Now we do some theoretical analysis to explain the observations in the simulation. We start with the status that the network has just suffered from a large-scale infection and the susceptible nodes have cut off a large number of connections to protect themselves. Consequently, the network is broken into pieces. In such a situation, though most nodes have recovered to susceptible status, due to the isolation avoidance, there are still a small number of infected nodes remaining in the network. Because the number of infected nodes and the number of the corresponding I components are very small, the probability that a newly added node connects to a susceptible node belonging to an I component is low. Hence, to study the growing speed of the giant component, we can ignore the effects of the infected nodes and the corresponding I components. Upon obtaining the growing behavior of the giant component, we can then calculate the expected time that the giant component is connected to an I component.

We assume that there are N_0 nodes and $M_0/2$ links remaining in a network that just suffered from a large-scale infection. Therefore on average each node has a degree M_0/N_0. Denote the average component size as $\langle g \rangle$. We have $\langle g \rangle = \sum_j g_j/N_G$ where g_j denotes the size of component j and N_G the number of the components. Since the probability that a new node connects to a component is proportional to the number of links remaining in the component and most nodes in the small components are of very low and similar nodal degrees, the probability that a new node connects to a component is approximately proportional to the size of this component. Therefore the average size of the components, other than the giant one, that are chosen by a new node is $h = \langle g^2 \rangle/\langle g \rangle$, where $\langle g^2 \rangle = \sum_j g_j^2/N_G$. Ideally, if a new node connects to only a single node in the giant component and connects to other $m - 1$ nodes from other different components, the size of the giant component may be increased by $(m - 1)h + 1$. However, with the growing of the giant component, the probability of having the new node making multiple connections to the giant component increases. Thus the giant component size may be increased at a speed of $(hl + 1)\alpha$, where l denotes the number of components connected to the new node other than the giant one, $l \in [0, m - 1]$. We assume that each new node connects the giant component by at least one link when he joins the network. This assumption is reasonable: on one hand, when all components are of small sizes, a new node may not connect to the largest component at that time. However, since the new node connects a few small components into a larger one, the newly formed component stands a better chance to be connected to more new nodes arriving later and become even bigger. With the growing of the network, the newly formed component has a high chance to be finally included into the largest component. Once this happens, the new node eventually contributes to increasing the size of the giant component.

On the other hand, when the giant component is large, the probability that it is connected to a new node is high. Based on the above consideration, the evolution of the giant component size, denoted as S_G, can be expressed as follows:

$$\frac{dS_G}{dt'} = \alpha \sum_{l=0}^{m-1} C_{m-1}^l \left(1 - \frac{M_G(t')}{M(t')}\right)^l \left(\frac{M_G(t')}{M(t')}\right)^{m-l-1} (hl+1),$$
(1)

where t' is the elapsed time after the network breaks into pieces, $M(t') = M_0 + 2m\alpha t'$ is the sum of the degrees of all the nodes at time t', and $M_G(t') = (S_G - \alpha t')M_0/N_0 + 2m\alpha t'$ is the sum of the degrees of all the nodes in the giant component at time t'. Note that $S_G - \alpha t'$ equals the number of nodes when $t' = 0$. The term in the first (second) bracket in the summation of Eq. (1) indicates the probability that a link sourced from a new node does not (does) choose the giant component, in which the preferential attachment is adopted and the effects of I components are ignored. For the case of $m = 2$, Eq. (1) goes to

$$\frac{dS_G}{dt'} = \left(1 - \frac{(S_G - \alpha t')\frac{M_0}{N_0} + 4\alpha t'}{M_0 + 4\alpha t'}\right)(h+1)\alpha$$
$$+ \frac{(S_G - \alpha t')\frac{M_0}{N_0} + 4\alpha t'}{M_0 + 4\alpha t'}\alpha.$$
(2)

Solving Eq. (2), we have

$$S_G = (N_0 + \alpha t') + (M_0 + 4\alpha t')^{-\frac{hM_0}{4N_0}} \left(hM_0^{\frac{hM_0}{4N_0}} - N_0 M_0^{\frac{hM_0}{4N_0}}\right),$$
(3)

where we set $S_G(0) = h$.

Figure 7 shows the results of Fig. 5(b) in the range of $t \in [500,5000]$. When t is around 500, the network breaks up into a large number of small pieces. After that the network grows continually until a giant component forms. In this figure, the circles show the simulation results and the red solid curve
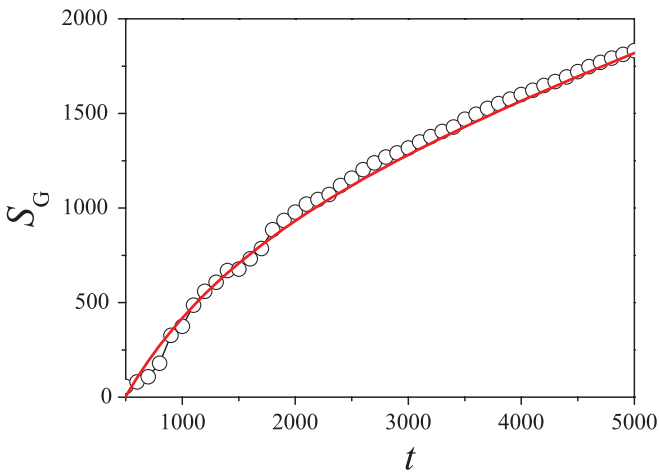

FIG. 7. (Color online) A detailed plot of Fig. 5(b) for $t \in [500,5000]$. The circles show the simulation results and the red solid curve shows the theoretical results obtained from Eq. (3). In the theoretical analysis, we adopt the calculation results that at time $t = 500$ (i.e., $t' = 0$), $N_0 = 1100$, $M_0 = 1648$, $h = 4.15$. Other parameter values include $\alpha = 0.2$ and $m = 2$.

shows the theoretical results obtained from Eq. (3). We can see they match fairly well.

In the following we analyze the average duration when the giant component can keep growing without connecting to an I component. Denote $P(T)$ as the probability that the giant component connects to I component for the first time at time T. We have

$$P(T) = \left(\prod_{t'=0}^{T-1} \Theta(t')^\alpha\right)[1 - \Theta(T)^\alpha],$$
(4)

where

$$\Theta(t') = \left(\frac{M(t') - M_I}{M(t')}\right)^m + \left(\frac{M(t') - M_G(t')}{M(t')}\right)^m$$
$$- \left(\frac{M(t') - M_I - M_G(t')}{M(t')}\right)^m.$$
(5)

In Eq. (5), M_I is the number of susceptible nodes in the I components, which does not change much during the network growing process until the infection invades the giant component, and hence is treated as a constant value here. The first (second) term on the right-hand side indicates the probability that none of the m links of the new node connects to the I components (giant component), while the third term indicates the probability that none of the m links connects to either the I components or the giant component. Therefore, $\Theta(t')$ presents the probability that a node newly added at time t' does not connect any I component to the giant component. As α is the growing rate of the new nodes, $\Theta(t')^\alpha$ represents the probability that all the newly added nodes do not connect any I component to the giant component at time t'. Hence, the expected value of the time that the giant component gets connected to an I component for the first time is

$$E(T) = \sum_{T=1}^\infty T P(T)$$
$$= \sum_{T=1}^\infty T \left(\prod_{t'=0}^{T-1} \Theta(t')^\alpha\right)[1 - \Theta(T)^\alpha],$$
(6)

which can be simplified to

$$E(T) = \sum_{T=0}^\infty \left(\prod_{t'=0}^T \Theta(t')^\alpha\right).$$
(7)

Setting $M_I = 20$, which is obtained from the simulation, and all the other parameters the same as those in Fig. 7, we substitute Eq. (3) into Eq. (7) and then solve it numerically. The result is $E(T) \simeq 723$. Using a similar method, we can calculate the standard deviation as 522. The very large standard deviation shows that there exist strong fluctuations in the time intervals between reconnections of the I component and the giant component. To demonstrate such strong fluctuations, we plot in Fig. 8 the evolution of the overall size of all the I components represented by the thin red curve. It is easy to understand that the overall infection size would jump up when a small I component is connected to the giant component (which makes the giant component itself an I component, evidenced by the heights of the jumps close to the size of the giant component). As we can see, after the transient process, several jumps take place at around time 1970,
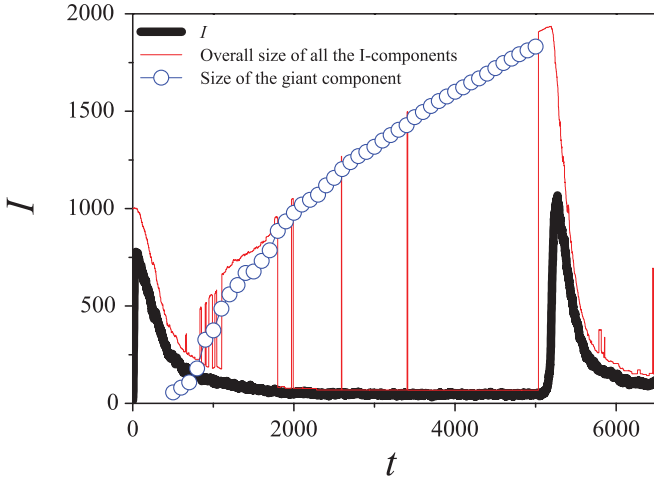
FIG. 8. (Color online) The evolution of the overall size of all the I components indicated by the thin red curve, the number of infected nodes indicated by the thick black curve, and the size of the giant component indicated by the blue circles. All parameters are the same as those in Fig. 7.

2590, 3405, and 5035 and the time intervals in between are 620, 815, and 1630, respectively. Such results are accordant with our theoretical estimation on the expected value of the time interval and its deviation. The observation on the large deviation value has its significance: The strong fluctuations of the reconnection process make it difficult to predict when the next reconnection of an I component and the giant component would happen though we could estimate the long-term average of the intervals. Further, consider the fact that a reconnection (reflected as a peak of the thin red curve in Fig. 8) does not always lead to an explosion (a peak of the thick black curve in Fig. 8), it would be very difficult, if not impossible, to predict when a reemergence would happen.

Once the infection does invade the giant component, the disease may spread over the whole component very quickly and further transmit to other smaller components. In fact, in the beginning stage of the outbreak, the fraction of infected nodes grows exponentially fast [4]. Specifically, with mean-field approximation the evolution of i and $\langle k \rangle$ can be expressed as

$$\begin{cases} \frac{di}{dt} = p\langle k \rangle(1-i)i - ri \\ \frac{d\langle k \rangle}{dt} = -2\omega\langle k \rangle(1-i)i \end{cases}. \qquad (8)$$

Here we ignore the network growing process since we mainly focus on the drastic change of the infection size in which the time span is short and the number of newly added nodes is very limited. Since i is very small in the beginning stage of an epidemic explosion, we neglect the high-order terms of i. Besides, because of the small time interval during this process, we regard $\langle k \rangle$ as a constant. Thus, the first equation in Eq. (8) can be simplified as [4]

$$\frac{di}{dt} = p\langle k \rangle i - ri. \qquad (9)$$

Solving Eq. (9), we have

$$i(t) = i(0)e^{(p\langle k \rangle - r)t}. \qquad (10)$$

We see that $i(t)$ grows exponentially, which proves that the number of infected nodes can increase very quickly in a short time.

Figure 4 illustrates that after each infection peak, the number of infected nodes decreases gradually and this decreasing process sustains much longer than the increasing process. To study the behaviors of the decreasing process, we first consider the dynamics of $i(t)$ during a short period of time where $\langle k \rangle$ can be approximately regarded as a constant. Solving the first equation in Eq. (8) by assuming $\langle k \rangle$ as a constant, we have

$$i(t) = (p\langle k \rangle - r)\left(1 - \frac{1}{1 + Cp\langle k \rangle e^{(p\langle k \rangle - r)t}}\right), \qquad (11)$$

where $C = \frac{i(0)}{p\langle k \rangle[1-i(0)]-r}$.

The exponential function in Eq. (11) reveals that the value of $p\langle k \rangle - r$ determines the typical time it takes for $i(t)$ to go to the stable state for a constant value $\langle k \rangle$. A larger $p\langle k \rangle - r$ corresponds to a shorter time. For the specific case as shown in Figs. 4 and 8 where $m = 2$, $p = 0.09$, $r = 0.04$, and $\omega = 0.03$, when $i(t)$ starts to decrease, $\langle k \rangle \approx 3$ and $(p\langle k \rangle - r)/\omega \approx 8$. This shows that the spreading process is much faster than the link-removal process. Such observation allows us to simplify our analysis by considering the decreasing process as composed of a series of quasistatic processes. That is, we divide the decreasing process into a series of very short time intervals and regard each short interval as composed of two different parts: first some links are removed, then $i(t)$ quickly converges to a temporally stable state where $di/dt = p\langle k \rangle(1-i)i - ri = 0$ for the updated value of $\langle k \rangle$. Hence, suppose $\langle k \rangle$ has a small change in each time interval as $\langle k \rangle \rightarrow \langle k \rangle - \varepsilon$, where ε is a small positive quantity. We have

$$\frac{di}{dt} = p(\langle k \rangle - \varepsilon)(1-i)i - ri = -\varepsilon p(1-i)i. \qquad (12)$$

By solving Eq. (12), the evolution of $i(t)$ can be expressed as $i(t) = 1/(C'e^{\varepsilon pt} + 1)$, where C' is a constant depending on
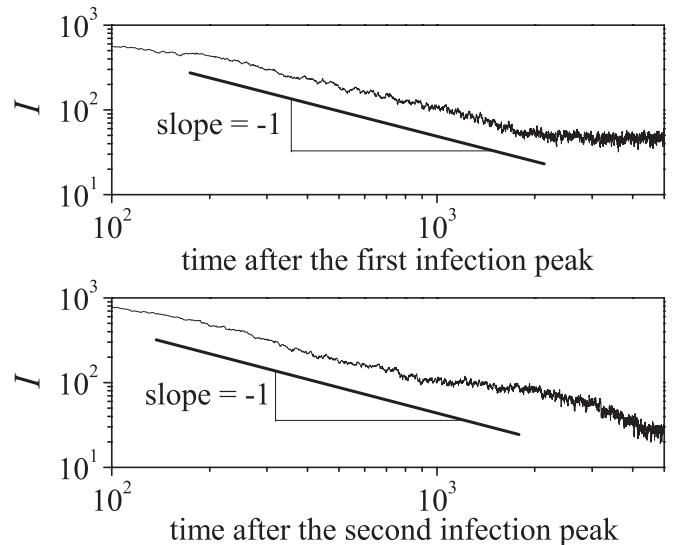

FIG. 9. The evolutions of the number of infected nodes after the first and second infection peak. Parameters are the same as those in Fig. 7.
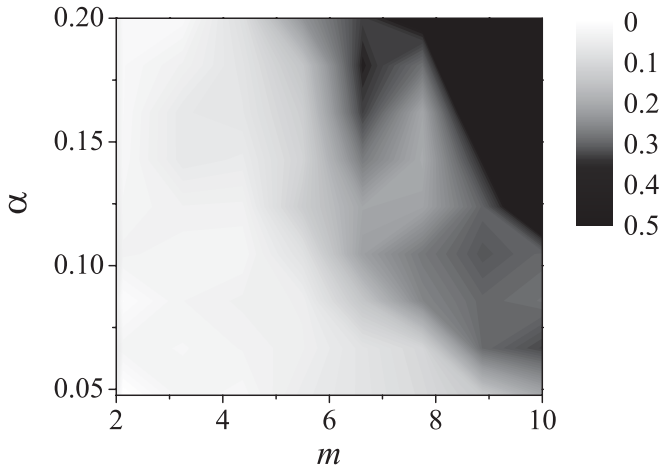
FIG. 10. Gray plot of parameter F on the $m - \alpha$ plane. Results are obtained from 100 realizations. Other parameters are the same as those in Fig. 4.

the initial condition. As ε is of a very small positive value, $i(t)$ can be further approximated as

$$i(t) = \frac{1}{1 + C'\varepsilon pt + C'} \sim \frac{1}{t}. \tag{13}$$

Therefore, we have that approximately $i(t)$ evolves inverse proportionally with time t after the infection peak. The log-log plot in Fig. 9 shows the simulation results of the evolution of the number of infected nodes I after the first and second infection peaks for the case in Fig. 8, where the first (second) peak happens around the time $t = 100$ ($t = 5270$). As expected, I evolves approximately inverse proportionally with time when it decays from the peak.

When the number of infected nodes decreases to a small value, it will become stable. In this stage, the network breaks to an extent that the sizes of I components are small enough so that the breaking process almost ceases due to the isolation avoidance. Consequently a small number of infected nodes may be preserved in the I components for a long time until the next epidemic explosion happens.

It has been observed that epidemic reemergence may happen for a wide range of α and m. To manifest the influences of α and m, we introduce a parameter F to measure the explosiveness of the epidemic reemergence, which is defined as

$$F = \frac{T\left(I > \frac{I_{\max}}{2}\right)}{T\left(I > 0\right)}, \tag{14}$$

where $I_{\max}$ is the maximum value of I in the whole disease spreading process. A smaller value of F corresponds to a more abrupt burst in the number of infected nodes. Figure 10

shows the results of parameter F on the $m - \alpha$ plane. It can be seen that generally speaking slower network growing speed α and smaller m lead to smaller F value. This phenomenon can be understood as follows. Having a higher network growing speed and more links added to the network by each newly added node makes the S components connect to I components in a larger probability; consequently the susceptible nodes may get infected more easily. If a significant number of S components are invaded by infection before they form into a large component, the reemergence shall become less explosive. Note that in all our simulations, the infection finally dies out: small fluctuations at a very low infection size sooner or later will push the infection into extinction. Reemergence cannot be repeated forever unless it has a stable pool outside the system we have been considering.

V. CONCLUSION

Based on a simple model that captures basic characteristics of epidemic spreading, we revealed the mechanism of a phenomenon, epidemic reemergence. Specifically, we considered the epidemic spreading in a growing network where a susceptible node may remove the link connecting to an infected neighbor. Such a link-removal process, however, is subject to the isolation avoidance constraint. It is observed that epidemic reemergence may happen in such a simple model: The number of infected nodes may be suppressed to a low level for a long time and then suddenly erupt into a high level. Our findings indicate that an infectious disease may break out repeatedly, and between two explosions the disease may be incubated for a long time by a small number of carriers who are not connected to the main body of the society but are confined in a small area. The small number of disease carriers, however, stands a high chance to induce a large-scale epidemic explosion even when most people are intentionally avoiding the disease.

In the current model, S-I links are simply removed unless the removal will cause isolation. In real life, however, people may tend to enhance existing S-S links or even build up new ones to make compensations for the social connections they have lost. For example, they may tend to spend more time with their family members while reducing social gatherings. How such rewiring operations and connection enhancements will affect the epidemic spreading and reemergence is of future research interest.

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