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Epidemics with temporary link deactivation in scale-free networks

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Abstract

During an epidemic, people may adapt or alter their social contacts to avoid infection. Various adaptation mechanisms have been studied previously. Recently, a new adaptation mechanism was presented in [1], where susceptible nodes temporarily deactivate their links to infected neighbors and reactivate when their neighbors recover. Considering the same adaptation mechanism on a scale-free network, we find that the topology of the subnetwork consisting of active links is fundamentally different from the original network topology. We predict the scaling exponent of the active degree distribution and derive mean-field equations by using improved moment closure approximations based on the conditional distribution of active degree given the total degree. These mean field equations show better agreement with numerical simulation results than the standard mean field equations based on a homogeneity assumption.

Keywords

adaptive networks; epidemics model; moment-closure approximation

I. INTRODUCTION

In recent years, modeling spread of diseases on social networks has attracted great attention [2–7]. The complex topology of a real social network is often approximated by using simpler network models. One of the simplest is the Erdős-Rényi (ER) random graph [8], where the links are generated randomly between the nodes with a fixed probability. As a result of the random generation of links, the degree distribution is Poisson in the limit of large network size. Epidemic models on ER random graphs have been studied previously [9, 10]. It has been shown that there is a threshold infection rate, below which the infection dies out and above which the infection will persist.

However, in real social networks the links are not generated uniformly randomly. Many real world networks display a power law in the tail of their degree distribution, i.e., $P(k) \sim k^{-\gamma}$, where $P(k)$ is the fraction of nodes with degree k and γ is the exponent of the distribution [4, 11]. Such networks are called scale-free (SF). In contrast to ER networks, there is a significant probability that nodes with a large number of connected links (hubs) will exist in SF networks. It has been shown that static random SF networks do not have an epidemic

threshold in the limit of large network size and thus are prone to infection spreading [2, 3, 12].

In the case where the dynamics of the disease is much faster than the dynamics of the network, the network topology can be treated as static. However, during an epidemic, people may respond to the infection by changing their social contacts. Thus, the network of the social contacts may also evolve in time. Epidemic models on dynamic networks have been studied previously (e.g., [13]). If the evolution of the network depends on the state of the nodes, and the state of nodes depends on the network geometry, then the network is called an adaptive network (e.g., [14–17]).

Various adaptation rules have been considered. An adaptation mechanism depending on rewiring of the links is introduced in [7], where the susceptible nodes are allowed to rewire from their infected neighbors to a randomly chosen susceptible node. Here the total number of links is preserved while the links are rewired to reduce infection spread. In [18], susceptible nodes are allowed to rewire to a randomly chosen node, whereas in [19] the infected nodes are also allowed to rewire to a randomly chosen node. Other studies have also considered epidemic models with a rewiring type mechanism (e.g., [20]). In these models, the original network topology is not preserved. Indeed, Marceau *et al.* [21] argue that the model of [7] appears to reach a universal steady state, independent of the initial network structure.

Other models have considered rewiring with some constraints on network structure. In [22], non-adaptive rewiring based on random swapping of edges was introduced, such that the degree distribution is preserved. However, the original neighborhood is not preserved. It is expected that in real life, people tend to keep their connections with their friends. With this motivation, we presented a new adaptation mechanism in [1] based on temporary deactivation of the links between susceptible and infected nodes. The deactivated links are reactivated when the infected neighbors have recovered. The initial network topology in [1] was limited to the ER random graph.

In this paper, we study the temporary link deactivation mechanism for a susceptible-infected-susceptible (SIS) epidemic model on scale-free networks. The model of [1] is reviewed in Section II. In Section III, we investigate the probability distribution for the number of active links of a node, termed the “active degree distribution,” when the network has a SF initial structure. Previously, mean field equations based on a homogeneity assumption showed good agreement with the simulations for an ER random network in [1]. However, the homogeneity assumption is invalid for heterogeneous networks, such as scale-free networks. In Section IV, improved mean field equations are derived for the model and show good agreement with simulations in predicting the behavior of the scale-free system.

II. MODEL

In this section, we review the model introduced in [1]. The disease follows susceptible-infected-susceptible (SIS) dynamics. A susceptible node becomes infected with rate pN_{nbr} , where p is the infection rate per SI link and N_{nbr} is the number of infected neighbors of the

node that are connected by active links. An infected node becomes susceptible again with recovery rate r .

Links behave as follows. As a strategy to avoid infection, a susceptible node temporarily deactivates its links to its infected neighbors with deactivation rate d [1]. When the infected neighbor recovers, the deactivated links are reactivated with reactivation rate a . The adaptation mechanism is shown diagrammatically in Fig. 1(a), and all possible link transitions are summarized in Fig. 1(b). Note that this adaptation mechanism preserves the underlying network topology, i.e., the links are only temporarily deactivated and no links are ever deleted or created.

In [1], we found two distinct dynamical regimes for the model on ER networks. When network dynamics are slow, the adaptation merely reduces the average number of contacts per individual, and the system can be described by mean field equations for a static network with a lower mean degree. When network dynamics are fast, more efficient adaptation further reduces the spread of disease by targeting dangerous connections. The epidemic threshold, where the disease free state becomes unstable, is a forward transcritical bifurcation as a function of infection rate. The link deactivation rate affects the threshold, but reactivation rate does not. The mean active degree displays non-monotonic dependence on the link deactivation rate, as connectivity initially drops but increases at higher deactivation rates because the infection is being controlled and the number of links that can be deactivated is lessened. All of these phenomena are well described by mean field equations for node and link dynamics based on a homogeneity assumption [1].

For our studies of scale-free networks, stochastic simulations are performed using a continuous time Monte Carlo algorithm [23] on a Barabási-Albert (BA) type scale-free network [11] with total number of nodes $N = 10^5$ and fixed recovery rate $r = 1$. The total degree distribution of the network, on average, is given by $P_k(k) = 2mk^{-3}$, where m is the minimum number of links a node has in the BA growing network algorithm [11].

III. ACTIVE DEGREE DISTRIBUTION

The dynamics of a node's infection status depends on the number of infected nodes actively connected to it. The adaptation mechanism under consideration reduces this number by spontaneously deactivating links between susceptible and infected nodes. Both the disease propagation and the network adaptation depend on the total number of connections a given node can have. That is, low degree nodes are less likely to have an infected node as a neighbor, which in turn means that their active degree will be less affected by the adaptation. On the other hand, nodes with a large total degree are strongly affected. In particular, those nodes are more likely to have infected neighbors and thus have their links deactivated.

The extent of the deactivation is of particular interest when considering networks with complex topology such scale-free networks. In the following sections we show that the interplay between the disease dynamics and the adaptation leads to nontrivial changes in the topology of the active part of the network. Specifically, we show that in the network under consideration, the tail of the active degree distribution is very different from the tail of the total degree distribution. Here we present direct numerical simulations investigating the

topology of the active part of the network and discuss a heuristic explanation for the observed properties of the active network topology.

A. Asymptotic nature of active degree distribution: heuristic explanation

We present a heuristic argument for the asymptotic behavior of the active degree of a node with total degree k , as a function of k . We consider the limit of $d, a \rightarrow 0$ where we expect just a few link events, i.e., events associated with link activation/deactivation, over the period of time when a given node changes its status from susceptible to infected and back to susceptible. For slow network dynamics, the fluctuations in the active degree are expected to be less than that in fast network dynamics. Figure 2 shows sample results for fixed $d/a = 1$, where the fluctuations of the active degree are much higher when $d = a = 10$. On the other hand, the fluctuations are much smaller for d and a close to zero. (Recall that the recovery rate $r = 1$ sets a time scale for the disease dynamics.)

We claim that at the steady state we must balance the number of link events that take place while the node is in the infected state with the number of such events while the node is in the susceptible state. We use the notation $[XY]_k$ and $[X\hat{Y}]_k$, respectively, to denote the expected number of Y -nodes actively and inactively connected to an X -node with degree k . We treat these as approximately constant in the slow network limit. Thus, the average time a node spends in the susceptible state is approximated by $1/(p[SI]_k)$. In this time, the expected number of deactivated links that are reactivated is approximately $(a[\hat{S}\hat{S}]_k)/(p[SI]_k)$ and the expected number of active links that are deactivated is approximately $(d[SI]_k)/(p[SI]_k)$. On the other hand, the average time interval a node spends in the infected state is $1/r$. In this time, the expected number of active links that are deactivated is $(d[IS]_k)/r$. Balancing these numbers we obtain the relation:

$$\frac{1}{p[SI]_k} [a[\hat{S}\hat{S}]_k - d[SI]_k] = (d/r) [IS]_k, \quad (1)$$

which implies that

$$[\hat{S}\hat{S}]_k = \frac{dp}{ra} [SI]_k [IS]_k + \frac{d}{a} [SI]_k. \quad (2)$$

Let the function $f(k)$ denote the expected active degree of a node with total degree k . With additional assumptions that $[SI]_k$ are proportional to $f(k)$, and assuming that $[\hat{S}\hat{S}]_k$ is proportional to the deactivated degree of a node of degree k (i.e., $k - f(k)$), we obtain

$$k - f(k) \propto [f(k)]^2 + cf(k), \quad (3)$$

where c is a combination of constants. For high degree nodes $f(k)$ is large, so it is valid to assume that $f(k) \ll [f(k)]^2$ as the degree of the node increases. As a result, we conclude that $f(k) \sim k^{1/2}$ for large k .

This heuristic argument suggests that the tail of the active degree distribution may have different asymptotic behavior than the tail of the total degree distribution. The active degree distribution is obtained from the total degree distribution as follows:

$$P_A(A) = \sum_k P_{A|k}(A|k) P_k(k), \quad (4)$$

where P_k and P_A are the total and the active degree distributions, respectively, and $P_{A|k}$ is the function describing the probability that a node with degree k has A active links. In the limit where the nodes change their infection status much more frequently than links change their activity status, we assume that the conditional distribution $P_{A|k}$ is narrow and can be approximated by a delta distribution centered at the mean of $P_{A|k}$, which is equal to $f(k)$. With the additional assumption that k can be treated as continuous, the function P_A can be found by evaluating the integral

$$P_A(A) = \int \delta[A - f(k)] P_k(k) dk. \quad (5)$$

Substituting $u = f(k)$:

$$P_A(A) = \int \delta[A - u] P_k(f^{-1}(u)) \frac{1}{f'(f^{-1}(u))} du = \frac{P_k(f^{-1}(A))}{|f'(f^{-1}(A))|}. \quad (6)$$

Using that $f(k) \sim k^{1/2}$, $f^{-1}(A) \sim A^2$, and $P_k(k) \sim k^{-3}$ for a BA network, we obtain $P_A(A) \sim A^{-5}$. Thus we conclude that the active degree distribution has $\sim A^{-5}$ asymptotic behavior in the tail of the distribution.

B. Results: Active degree distribution

We perform numerical investigation in various parameter regimes to test the results obtained using the heuristic argument. Numerical results are averaged over 100 different realizations of BA scale-free networks generated using the algorithm in [11] with $N = 10^5$ nodes and $m = 4$ (average degree 8). In each run, we discard the first 10^9 events to avoid considering the transient regime. Then, we select 10^3 equally spaced sample points out of further 10^8 events, i.e., with a time step of 10^5 events between each sample.

In Fig. 3(a) we can see that as we fix d/a and p , then for low values of d , i.e., whenever the network dynamics is slow, the expected active degree A as a function of the node's total degree k has a square root asymptotic dependence as predicted. As the values of d and a are simultaneously increased, thus increasing the speed of the network dynamics, the asymptotic nature of $f(k)$ becomes less clear for the larger values of d and a . As we consider the active degree distribution, the results in Fig. 3(b) show that the tail of the distribution has the predicted k^{-5} asymptotic behavior for small values of d and a . The slope of the tail deviates from -5 increasingly as the link activity is increased. Thus, we can see that the asymptotic behavior of the distribution is consistent with our prediction in the considered limit of slow network dynamics but is inconsistent away from that limit.

In Fig. 4 we consider the asymptotic behavior of $f(k)$ and the active degree distribution P_A as we fix d to be slow, fix p , and vary the value of d/a (thus varying the activation rate). As

shown in Fig. 4(a), the asymptotic behavior of $f(k)$ is consistent with our $\sim k^{1/2}$ prediction, even for the large values of a (small value of d/a). As we can see from Fig. 4(b), while the tail of P_A appears to have the k^{-5} asymptotic behavior for small values of a , this prediction is less accurate for the large values of a , possibly due to fast link activity.

Finally, in Fig. 5 we consider the consistency of our prediction as we vary the values of the infection rate p . We expect our prediction about the asymptotic behavior of $f(k)$ to improve as the value of p is increased, meaning the network dynamics are slower relative to the disease dynamics, which is in fact the behavior shown in Fig. 5(a). Similarly, as shown in Fig. 5(b), the distribution P_A has $\sim k^{-5}$ asymptotic behavior for large values of i , but not for the smaller values of p , where the node activity is slow compared to the link activity.

We note that when link deactivation is frequent, finite size effects make observation of high active degree nodes difficult. As we have shown previously for ER networks [1], the mean active degree in the network can be significantly reduced. For parameter values such as the blue crosses in Figs. 4(b) and 5(b), the active degree distribution falls off around degree 20 due to finite size effects, and obtaining a greater range of power law scaling would require prohibitively large network sizes. However, these distributions are fit better by A^{-5} scaling than other simple distributions such as exponential. The relationship between mean active degree and total degree is much less sensitive to finite size effects, enabling us to test most of our assumptions.

IV. MEAN FIELD MODEL

We now turn to finding a low dimensional description of the system. In the usual mean field approach for networks, equations capturing the evolution of nodes and links are derived [7, 18, 19, 24]. Since the evolution of links (pairs) depends on higher order terms (triples), the system is open. To close the system, in a moment closure approximation method, triples are derived in terms of the other variables (nodes, links) under a homogeneity assumption. In ER networks, a typical node has degree close to the average degree, and the variation in degree between nodes can be ignored. However, this assumption is no longer valid for networks with heterogeneous structure, such as scale-free networks where the variance grows as the size of the network increases. To overcome the failure of the homogeneity assumption in scale-free networks, heterogeneous mean field (HMF) equations were introduced previously [3], where the nodes are classified depending on their degrees, and ordinary differential equations are derived for each degree. Although these equations capture the dynamics of the system, the number of equations is large and increases with the network size. In this section, we introduce new mean field equations based on an improved moment closure approximation derived by considering the active degree distribution conditioned on the total degree of the nodes.

A. Moment closure approximation: beyond homogeneous closure

We first review the mean field equations derived in [1]. The following system of equations describes the evolution of the number of each type of node:

$$\frac{dN_S}{dt} = rN_I - pN_{SI}, \quad (7a)$$

$$\frac{dN_I}{dt} = -rN_I - pN_{SI}, \quad (7b)$$

and each type of link:

$$\frac{dN_{SS}}{dt} = rN_{SI} - pN_{SSI} + aN_{SS}, \quad (8a)$$

$$\frac{dN_{SI}}{dt} = r(2N_{II} - N_{SI}) + p(N_{SSI} - N_{ISI}) - dN_{SI}, \quad (8b)$$

$$\frac{dN_{II}}{dt} = -2rN_{II} + pN_{ISI} \quad (8c)$$

$$\frac{dN_{\dot{S}\dot{S}}}{dt} = rN_{\dot{S}I} - pN_{\dot{S}SI} - aN_{\dot{S}\dot{S}}, \quad (8d)$$

$$\frac{dN_{\dot{S}I}}{dt} = r(2N_{\dot{I}\dot{I}} - N_{\dot{S}I}) + p(N_{\dot{S}SI} - N_{\dot{I}SI}) + dN_{\dot{S}I}, \quad (8e)$$

$$\frac{dN_{\dot{I}\dot{I}}}{dt} = -2rN_{\dot{I}\dot{I}} + pN_{\dot{I}SI}, \quad (8f)$$

where N_X denotes the expected number of nodes in state X, while $N_{XY} (N_{\dot{X}\dot{Y}})$ denotes the expected number of active (deactivated) links connecting nodes in states X and Y, with $X \in \{S, I\}$. The notation N_{XYZ} denotes the expected number of triples of connected nodes of type X, Y, and Z, with a circumflex over nodes that are connected by a deactivated link. In other words, N_{XYZ} counts the node triples such that a node in state X is linked to a node in state Y, which in turn is linked to a node in state Z. Note that our definition of an XYX type of a triple includes the degenerate triples, i.e., those triples formed by a single Y-node and an X-node. The values of N_X , N_{XY} and $N_{\dot{X}\dot{Y}}$ are treated as continuous in the limit of large network size.

Equations (7) reflect the transitions of nodes between susceptible and infected states. For example, infected nodes are lost due to recovery and are created when infection spreads along SI links (Eq. 7b). Eqs. (8) reflect the link transitions indicated in Fig. 1(b). For example, in Eq. 8c, II links are lost when the I node on either end recovers with rate r . An II link is created when infection transmits along an SI link, but links between the S-node and its other I neighbors also become II links. Thus the rate of formation of II links is pN_{ISI} , where the former process is accounted for by the degenerate ISI triples (triples formed by a

single I-node and an S-node) and the latter by the non-degenerate ISI triples. For more details on derivations, [25] shows for similar models how node and link equations may be derived from a set of heterogeneous mean field equations.

The system of equations (7) and (8) is open since it contains unknown quantities corresponding to the statistics of higher order node formations, i.e., the number of connected triples of nodes where at least one of the links is of SI type. By definition, the expected number of XSI triples, N_{XSI} , can be found using the following equation for the number of XSI triples per S-node:

$$\frac{N_{XSI}}{N_S} = \sum_{x,i} xi P_{X|I|S} (X=x, I=i|S) = \sum_{x,i} xi P_{X|I,S} (x|i, S) P_{I|S} (i|S), \quad (9)$$

where x indicates the number of XS links stemming from an S-node under consideration, with $XS \in \{\hat{S}\hat{S}, SS, \hat{I}\hat{S}, IS\}$. Further, $P_{X,Y|Z} (X=x, Y=y|Z)$ is the probability a node of type Z has x neighbors of type X and y neighbors of type Y , $P_{X|Y,Z}(x|y, Z)$ is the probability a Z node has x neighbors of type X given that it has y neighbors of type Y , and $P_{Y|Z} (y|Z)$ is the probability a Z node has y neighbors of type Y . One way to close the system is to make an ad hoc assumption that the information about the neighborhood of a susceptible node is independent of the number of infected nodes actively connected to it. In other words, we assume that $P_{X|I,S}(x|i, S) = P_{X|S}(x/S)$, and, therefore, for $XS \in \{\hat{S}\hat{S}, SS, \hat{I}\hat{S}\}$,

$$\frac{N_{XSI}}{N_S} = \sum_{x,i} xi P_{X|S} (x|S) P_{I|S} (i|S) = \sum_x x P_{X|S} (x|S) \sum_i i P_{I|S} (i|S) = \frac{N_{SX}}{N_S} \frac{N_{SI}}{N_S}. \quad (10)$$

In the case of an ISI triple, a closure can be obtained by assuming that the distribution of number of infected neighbors of an S-node is Poisson, in which case $P_{X|I,S}(x|i, S) = \delta_{x,i}$ and

$$\frac{N_{ISI}}{N_S} = \sum_i i^2 P_{I|S} (I=i|S) = \left[\sum_i i P_{I|S} (I=i|S) \right]^2 + \sum_i i P_{I|S} (I=i|S) = \left[\frac{N_{SI}}{N_S} \right]^2 + \frac{N_{SI}}{N_S}. \quad (11)$$

Note that the i^2 factor (instead of $i(i-1)$) in the first summation occurs because N_{ISI} includes degenerate triples. The closures in Eqs. (10) and (11) were successfully used in [1] for ER networks. However, such an approach disregards the total degree of the node under consideration, an assumption which may be especially poor in a scale-free network, where the node degree is a highly heterogeneous quantity.

A simple way to take into account the degree of the susceptible node is to introduce additional conditioning on the total degree in Eq. (9):

$$\frac{N_{XSI}}{N_S} = \sum_{x,i,k} xi P_{X|I,k,S} (x|i, k, S) P_{I|k,S} (i|k, S) P_{k|S} (k|S), \quad (12)$$

where the k in $P_{X|I,k,S}(x|i, k, S)$ and $P_{I|k,S}(i|k, S)$ indicates that the node under consideration has total degree k , and $P_{k|S}(k/S)$ is the probability that the total degree of an S-node is k . We

can now close this relation by a homogeneity assumption that is conditioned on the total degree of nodes. In other words, for a given degree k of an S-node, the number x of XS-links stemming from the S-node is assumed independent of the number i of IS-links stemming from it, i.e.,

$$P_{X|I,k,S}(x|i,k,S) = P_{X|k,S}(x|k,S). \quad (13)$$

Then for $XS \in \{\hat{S}\hat{S}, SS, I\hat{S}\}$,

$$\frac{N_{XSI}}{N_S} = \sum_k \left[\sum_x x P_{X|k,S}(x|k,S) \right] \left[\sum_i i P_{I|k,S}(i|k,S) \right] P_{k|S}(k|S), \quad (14)$$

and

$$\frac{N_{ISI}}{N_S} = \sum_{k,i} i^2 P_{I|k,S}(i|k,S) P_{k|S}(k|S). \quad (15)$$

The second assumption that we make on the way to closing the mean field system of equations is that the expected number of XS-links stemming from an S-node is proportional to the S-node's degree, i.e.,

$$[XS]_k \equiv \sum_x x P_{X|k,S}(x|k,S) = C_{SX} k, \quad (16)$$

where the proportionality constant C_{SX} corresponds to the probability that by following a link stemming from an S-node we will encounter an X-node. C_{SX} is found by averaging over the total degree k :

$$\frac{N_{SX}}{N_S} = \sum_{x,k} x P_{X|k,S}(x|k,S) P_{k|S}(k|S) = \sum_k C_{SX} k P_{k|S}(k|S) = C_{SX} \langle k \rangle_S. \quad (17)$$

Here $\langle \cdot \rangle_S$ denotes the average taken over the total degree of susceptible nodes. As a result, the expected number of triples per S-node is found as follows:

$$\frac{N_{XSI}}{N_S} = \sum_k C_{SI} C_{SX} k^2 P_{k|S}(k|S) = \frac{\langle k^2 \rangle_S}{\langle k \rangle_S^2} \frac{N_{SX}}{N_S} \frac{N_{SI}}{N_S}. \quad (18)$$

The additional assumption that the IS-links are homogeneously distributed among S-nodes, i.e., links stemming from an S-node are equally likely to be an SI-link. This is equivalent to $P_{I|k,S}(i|k,S)$ being given by a binomial distribution:

$$P_{I|k,S}(i|k,S) = \binom{k}{i} C_{SI}^i (1 - C_{SI})^{k-i}, \quad (19)$$

which leads to the following closure:

$$\frac{N_{ISI}}{N_S} = \sum_k \left[C_{SI}^2 k^2 + C_{SI} (1 - C_{SI}) k \right] P_{k|S}(k|S) = \left[\frac{N_{SI}}{N_S} \right]^2 \frac{(\langle k^2 \rangle_S - \langle k \rangle_S^2)}{\langle k \rangle_S^2} + \frac{N_{SI}}{N_S}. \quad (20)$$

In order to complete the closure, we must estimate the value of the ratio $\alpha \equiv \langle k^2 \rangle_S / \langle k \rangle_S^2$. We present the estimate of these moments in two limits: near bifurcation and in the limit of large infection rate p .

Our numerical simulations allow us to conclude that the solution undergoes a transcritical bifurcation at a threshold infection rate in the case of finite size networks. Therefore, in the limit as p approaches the threshold from above, the expected number of infected nodes approaches zero, and as a result in this limit the degree distribution of susceptible nodes must approach the network's total degree distribution $P_k(k) = 2m^2 k^{-3}$. In this case the value of will depend on the size of the network, and will approach infinity in the limit of infinite network size.

In order to estimate in the limit of large values of p , we consider the following relation:

$$P_{k|S}(k|S) = P_{S|k}(S|k) \frac{P_k(k)}{P_S(S)}, \quad (21)$$

where the probability that the degree of a randomly chosen node is k is given by the total degree distribution, $P_k(k)$, and the probability $P_S(S)$ that a randomly chosen node is susceptible given by $N_S/(N_S + N_I)$, i.e., the fraction of susceptible nodes in the system. Function $P_{S|k}(S|k)$ is the probability that a node with total degree k is susceptible.

We approximate its value by estimating the fraction of time a node with degree k spends in a susceptible state:

$$P_{S|k}(S|k) = \frac{1/(p[SI]_k)}{1/r + 1/(p[SI]_k)} = \frac{1}{(p/r)[SI]_k + 1} \sim [SI]_k^{-1} \quad \text{in the limit of large } p, \quad (22)$$

where $1/(p[SI]_k)$ is the expected amount of time it takes for an S-node with $[SI]_k$ infected neighbors to be infected, and $1/r$ is the expected amount of time it takes an infected node to recover. In the limit of large p we obtain:

$$P_{k|S}(k|S) \sim [SI]_k^{-1} P_k(k) \sim k^{-1} P_k(k), \quad (23)$$

where we used the approximation of Eq. (16) for SI-links.

Using $P_k(k) = 2m^2 k^{-3}$ for BA scale-free network, where m is the minimal node degree, we get

$$P_{k|S}(k|S) \sim k^{-4} = ck^{-4}, \quad (24)$$

for some normalization constant c . Then treating k as continuous, we derive the normalization constant:

$$\int_{k_{min}=m}^{\infty} c k^{-4} dk = 1 \Rightarrow c = 3m^3. \quad (25)$$

Using Eq. (25) we obtain the following approximation for α :

$$\alpha = \frac{\langle k^2 \rangle_S}{\langle k \rangle_S^2} = \frac{\int_m^{\infty} k^2 P_{k|S}(k|S) dk}{\left[\int_m^{\infty} k P_{k|S}(k|S) dk \right]^2} = \frac{4}{3}. \quad (26)$$

We would like to point out that one may expect the linear approximation in Eq. (16) to be accurate in a range of values $k < K$, for some K , as a consequence of Taylor expansion of $[XS]_k$. In other words, Eq. (14) can be presented as

$$\begin{aligned} \frac{N_{XSI}}{N_S} = & \sum_{\{k < K\} \cup \{k \geq K\}} \left[\sum_x x P_{X|k,S}(x|k, S) \right] \left[\sum_i i P_{I|k,S}(i|k, S) \right] P_{k|S}(k|S) \approx \\ & \sum_{k < K} C_{SI} \quad C_{SX} \quad k^2 P_{k|S}(k|S) + \sum_{k \geq K} \left[\sum_x x P_{X|k,S}(x|k, S) \right] \left[\sum_i i P_{I|k,S}(i|k, S) \right] P_{k|S}(k|S). \end{aligned} \quad (27)$$

We argue that the proposed closure implicitly assumes that the second moment of $P_{k|S}(k|S)$ is a convergent quantity in the limit of large network size, and that the proportionality approximation in Eq. (16) is valid for $k < K$, where K is large enough for the sum over $k < K$ to be negligible. This is because the terms in the brackets in Eq. (27) are bounded by the total degree k , and therefore

$$\sum_{k \geq K} \left[\sum_x x P_{X|k,S}(x|k, S) \right] \left[\sum_i i P_{I|k,S}(i|k, S) \right] P_{k|S}(k|S) \leq \sum_{k \geq K} k^2 P_{k|S}(k|S). \quad (28)$$

As long as the second moment of $P_{k|S}(k|S)$ converges, there exists a value of K such that the second sum can be made arbitrarily small, which leads to the approximation in Eq. (18).

B. Results: Mean field

In Fig. 6 we present the results of simulating the spread of disease on a realization of the scale-free network generated according to the algorithm in [11] for $m = 10$. To locate the endemic branch, we first simulated the system for a long time for $p = 10$. Then p is swept from $p = 10$ to lower values. The steady state infection level for each p value is obtained using the sampling described in Section III B. The final state of each run is used as the initial state for the next p value. The results of the direct simulations are compared to the mean field approximation found in Eqs. (7)-(8), which were closed using two different assumptions: firstly, infected nodes are distributed homogeneously in the neighborhood of susceptible nodes as in Eqs. (10)-(11), and secondly, the distribution of the infected nodes is homogeneous when conditioned on the total degree of the susceptible nodes. The second closure is completed by

- closure A: approximating the value α of with that for the total degree distribution for all the nodes, which is only expected to perform well near the threshold where almost all of the nodes are expected to be susceptible. ($\alpha = 2.9$ for the particular network realization used here.)

- closure B: with the expression derived in Eq. (26) for the large infection rate limit
- closure C: using the value of α measured directly from the simulations.

As we can see from Fig. 6, the usual moment closure based on homogeneity rarely performs well. Closure C performs well everywhere, suggesting that the homogeneity assumption with the total degree conditioning is a good approximation. However, this approach is limited as it is based on the information obtained from the simulations and cannot be used for analytical study of the system. The two remaining closures perform well in their respective derivation limits. Thus, closure B shows excellent agreement with the simulations except near the epidemic threshold. On the other hand, closure A is the most accurate in its prediction of the threshold location, which appears to be nonzero for the finite network size. Thus, we conclude that the approximation in Eq. (13) is a minor source of error and completing the closure relies heavily on how well we can approximate the value of α for a broad range of parameters. Similar to what was observed in [1] for Erdős-Rényi random graphs, Fig. 6 illustrates that the epidemic threshold depends much more strongly on the deactivation rate than on the activation rate.

V. CONCLUSIONS

We applied an adaptation mechanism based on the temporary deactivation/reactivation of links [1] to scale-free networks. Although the underlying topology of the original network does not change, the active subnetwork is dynamic under this adaptation mechanism. We studied how the active degree distribution of the network evolves as the disease spreads in the network. We observed that the active degree distribution is a power law with a different exponent than the original degree distribution of the network. A heuristic argument explaining that observation is presented. We observe that the predicted exponent of the tail of the active degree distribution shows good agreement with the simulation results in the limit where network dynamics is slow relative to disease dynamics.

A related study of adaptation in scale-free networks was presented in [17]. In contrast to our model with temporary link deactivation, Demirel et al. studied a susceptible-infected-removed (SIR) epidemic in a growing network, in which removed nodes and their links were permanently deleted from the system. In both systems, high degree nodes (hubs) are more likely to become infected than low degree nodes. However, the effect on the network structure is different. Removal of infected nodes leads to a more drastic suppression of high degrees, and the resulting degree distribution quickly becomes exponential as the infection rate increases.

In order to derive mean field equations for our system, we improved the commonly used homogeneous moment closure approximation. The moment closure approximation in previous studies is based on the assumption that infected and susceptible neighbors of a susceptible node are independent of each other and independent of node degree. This assumption is more valid for a random network where the degree distribution is narrow. To improve that approximation, we take into account different node-type distributions conditioned on total degree, and we obtain a closure with an additional factor depending on the degree distribution of susceptible nodes. We approximated analytically the value of this

factor in two different regions. Direct numerical simulations of the full network system indicate that the system undergoes a forward bifurcation where the disease free state loses stability. In the region where the system is near the bifurcation, we assumed that the degree distribution of susceptibles is close to the degree distribution of the original network. Therefore, we were able to predict the factor approximately. We were also able to approximate the factor in the region where infection rate is large using asymptotic arguments. Our improved mean field showed better agreement with simulation than the homogeneous mean field in both of these regions. We also showed good agreement between the mean field equations and the simulations when the factor was directly measured via simulation.

A typical way to predict dynamics on complex networks is by utilizing a high dimensional approach, often referred to as heterogeneous mean field (e.g., [3]). In this approach, the number of equations scales with the maximum degree in the network and thus with the network size. Here we have developed a method that both takes into account the heterogeneity of the network and is low dimensional, with only eight differential equations describing the disease dynamics on an adaptive network. The closure approach derived here is sufficiently general and can be applied to a broader spectrum of problems of dynamics on evolving networks.

Epidemic dynamics on finite scale-free networks with our link deactivation mechanism were qualitatively similar to those observed previously in Erdős-Rényi random graphs [1], in that the epidemic threshold increased with increasing deactivation rate but did not depend strongly on activation rate. However, future studies may explore the epidemic dynamics in more detail, for example the effects of large fluctuations in the degree of hubs (cf. Fig. 2(b)) on infection levels. Another question of interest for the current model is how the epidemic threshold scales in the limit of infinite system size. Finally, we note that our predicted relationship between total degree and active degree is expected to apply to networks with other total degree distributions, and we have presented a conjecture for the active degree distribution (Eq. 6) that can be tested in other networks, such as scale-free networks with different exponents.

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FIG. 1.

(a) Schematic of the adaptation mechanism. An active SI link (solid line) is deactivated with deactivation rate d , and a deactivated SS link (dashed line) is reactivated with reactivation rate a . (b) Flow diagram of link transitions, with circumflex indicating inactive links. Horizontal arrows indicate infection of a node (arrows toward right) and recovery of a node (arrows toward left).

FIG. 2.

(a) Time series of the active degree of the node with largest total degree for $d/a = 1$, $d = 0.1$ and $a = 0.1$. (b) Time series of the active degree of the node with largest total degree for $d/a = 1$, $d = 10$ and $a = 10$.

FIG. 3.

(a) Mean active degree as a function of total degree. (b) Probability distribution of active degree (log-binned for the values of active degree greater than 10.) In both panels $d/a = 1$ and $p = 1$, and d increases monotonically from lowermost to uppermost data curve. Black points (crosses in (b)): $d = 0.1$; red points (circles in (b)): $d = 1$; green points (squares in (b)): $d = 5$; and blue points (x's in (b)): $d = 10$. In (a), red (lower) and blue (upper) dashed lines indicate slopes $1/2$ and 1 , respectively. In (b), red (lower) and blue (upper) dashed lines indicate slopes -5 and -3 , respectively.

FIG. 4.

(a) Mean active degree as a function of total degree. (b) Probability distribution of active degree (log-binned for the values of active degree greater than 10.) In both panels $d = 0.1$ and $p = 1$, and d/a increases monotonically from uppermost to lowermost data curve. Black points (crosses in (b)): $d/a = 0.1$; red points (circles in (b)): $d/a = 1$; green points (squares in (b)): $d/a = 2$; and blue points (x's in (b)): $d/a = 5$. In (a), red (lower) and blue (upper) dashed lines indicate slopes $1/2$ and 1 , respectively. In (b), red (lower) and blue (upper) dashed lines indicate slopes -5 and -3 , respectively.

FIG. 5.

(a) Mean active degree as a function of total degree. (b) Probability distribution of active degree (log-binned for the values of active degree greater than 10.) In both panels $d = 0.1$ and $a = 0.1$, and p increases monotonically from uppermost to lowermost data curve. Black points (crosses in (b)): $p = 0.1$; red points (circles in (b)): $p = 1$; green points (squares in (b)): $p = 5$; and blue points (x's in (b)): $p = 10$. In (a), red (lower) and blue (upper) dashed lines indicate slopes $1/2$ and 1 , respectively. In (b), red (lower) and blue (upper) dashed lines indicate slopes -5 and -3 , respectively.

FIG. 6.

Steady state fraction of infected nodes as a function of infection rate p for different d and a values. Circles: simulation results; red dot-dashed curve: solutions of the standard mean field equations with homogeneity closure; green dashed curve: solutions of the mean field equations closed using closure A (near bifurcation); black solid curve: solutions of the mean field equations closed using closure B (limit of large p); blue solid curve: solutions of the mean field equations closed using closure C (using simulations). Insets show closeup near epidemic threshold.