Dynamic behavior of the interaction between epidemics and cascades on heterogeneous networks

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Abstract – Epidemic spreading and cascading failure are two important dynamical processes on complex networks. They have been investigated separately for a long time. But in the real world, these two dynamics sometimes may interact with each other. In this paper, we explore a model combined with the SIR epidemic spreading model and a local load sharing cascading failure model. There exists a critical value of the tolerance parameter for which the epidemic with high infection probability can spread out and infect a fraction of the network in this model. When the tolerance parameter is smaller than the critical value, the cascading failure cuts off the abundance of paths and blocks the spreading of the epidemic locally. While the tolerance parameter is larger than the critical value, the epidemic spreads out and infects a fraction of the network. A method for estimating the critical value is proposed. In simulations, we verify the effectiveness of this method in the uncorrelated configuration model (UCM) scale-free networks.

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Introduction. – Epidemic spreading and cascading failure are extensively investigated in the research of complex networks. The study of epidemics on networks is an area of special interest including the spreading of infectious diseases, rumors [1] and computer viruses [2], and has deepened our insights into the interplay between topologies and the spreading dynamics [3–7]. Cascading failure is another phenomenon that random failures or intentional attacks lead to severe chain reactions propagating through links in networks. It is widely found in power transmission [8,9], communication [10], economic [11], and biological [12] networks.

Traditionally, these two dynamics correspond to independent topics in the research of complex networks, i.e., when one of them is studied, the other is considered to be irrelevant. But in practice, there are many cases in which they interact with each other and neither of their effects can be ignored. For example, consider a digital virus spreading over a data communication network. In this scenario, an infected router may not deliver data packets normally and cause overloading of nearby routers. These routers then redistribute their loads to other routers. Eventually, a large cascading failure occurs. In turn, the cascading failure also influences the spreading of the virus, because failed nodes cannot deliver data properly, especially they cannot deliver copies of the virus properly. A real-world case in which computer viruses CODE RED and Nima caused widely reported BGP storms, which is a cascading failure, was observed in September 2001 [13].

In our previous work [14], we proposed a model to study the interplay between epidemic spreading and cascading failure. In this model, when the two dynamical processes stop in a steady state, the nodes both uninfected and un-failed form several clusters. We considered the relative size $G$ of the largest one, i.e. the giant component, and found that in both Erdős-Rényi (ER) random networks and scale-free networks, with an infectivity over some threshold, a giant component forms only if the tolerance parameter $\alpha$, which captures the capacities of nodes, is within some interval $(\alpha_l, \alpha_u)$. In ref. [14], we have stated the reason for this —with $\alpha$ under $\alpha_l$, a large-scale cascade occurs and almost all nodes fail, while with $\alpha$ over $\alpha_u$, epidemics and cascading failure together kill almost all nodes.

In this paper, we explore this model from the perspective of epidemic spreading, while our previous work inspected it from the perspective of cascading failure. We consider the situation when the infection probability is

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sufficiently large. In this case, when $\alpha$ is extremely small, the epidemic dies out locally since the cascading failure cuts off almost all the paths in the network. When $\alpha$ is sufficiently large, nodes will never be overloaded, and the epidemic thus can spread out. Therefore, there exists a critical value $\alpha_c$ for which the epidemic with high infection probability can spread out. The critical value $\alpha_c$ is different from $\alpha_0$ in our previous work. The $\alpha_c$ indicates the critical moment at which epidemic starts to spread out but not to die out locally, while $\alpha_0$ implies the critical moment at which the epidemic and cascading failure kill all nodes in the network together. In the rest of this paper, we devote our efforts to further studying the interplay between epidemics and cascades, and pay special attention to the critical value $\alpha_c$.

Model. – In ref. [14], we link the epidemiological SIR model [15,16] together with a local load sharing cascading failure model to form a hybrid model.

In the SIR model, nodes are in one of the three states: susceptible (S), infected (I), and removed (R). At every time step, the susceptible node becomes infected with probability $\beta$ for every link connecting an infected node with a susceptible node. The infected node becomes removed with probability $\gamma$. The effective spreading rate is $\lambda = \beta/\gamma$. For simplicity, we let $\gamma = 1$.

In our model, removed nodes in the epidemic spreading are assumed to be not functioning and cannot manipulate loads. So the spreading of the epidemic will lead to load redistribution, which may cause the cascading failure in the network. The failed nodes in the cascading failure are not functioning and cannot manipulate loads either, and they cannot infect or be infected by others. Because both removed and failed nodes no longer interact with other nodes in the consequent process, they are said to be inactive. All nodes both uninformed and un-failed are said to be active.

Inspired by previous works [17,18], we assume that when a node becomes failed or removed, a fixed positive load $\Delta$ is transferred to each of its active neighbors. Overloaded nodes are those whose load exceeds their capacity. It is natural to assume that the capacity $C_v$ of a node $v$ is proportional to its initial load $L_v$ [19–22]

$$C_v = (1 + \alpha)L_v, \quad (1)$$

where the constant $\alpha$ is the tolerance parameter.

The numerical simulation of the hybrid process in a degree-degree uncorrelated network with $N$ nodes is summarized as follows:

1) The initial load of each node is randomly generated according to a uniform distribution on the interval $[L_{\text{min}}, L_{\text{max}}]$ and its capacity is defined by eq. (1). Without loss of generality, we let $L_{\text{min}} = 0$ and $L_{\text{max}} = 1$.

2) Randomly select a few nodes in the network as the initial infected nodes.

3) For every link connecting an infected node with a susceptible node, the susceptible node becomes infected with probability $\lambda$, and for every infected node, it becomes removed with probability 1.

4) The removal may cause load redistribution and possibly cascading failure. Repeat the cascades until there is no overloaded node in the network.

5) If there exist infected nodes in the network, go back to 3) to start another step of epidemic spreading; otherwise, the process halts.

Note that we make another assumption, i.e., that the time scale of the cascading failure is much smaller than that of epidemic spreading, so every single step of epidemic spreading is executed until there are no overloaded nodes in the network.

Analysis. – When the process ends in a steady state, the active nodes in the network form several clusters. In ref. [14], we use the fraction $G$ of the largest cluster, namely, the giant component, to measure the performance of the network. But here, for now, we examine the fraction $S_\alpha$ of the active nodes rather than the largest cluster formed by them, because it is closely related to $G$ and more likely to be theoretically tractable. Figure 1 shows $S_\alpha$ as a function of the tolerance parameter $\alpha$ in ER and uncorrelated configuration model (UCM) scale-free networks [23]. When $\alpha$ is extremely small, the cascading failure kills almost all nodes and the epidemic dies out locally, thus $S_\alpha$ is very close to zero. When $\alpha$ increases, the severity of the cascading failure reduces and $S_\alpha$ increases. When $\alpha$ is over some critical value, $\alpha_c$, the epidemic starts to spread out and infects a large number of nodes, making $S_\alpha$ decrease.

Preliminaries. Before we introduce the method to solve the critical value $\alpha_c$ in our model, we present some preliminary remarks.

The spread of a disease is equivalent to a bond percolation process [24,25]. With the effective spreading rate $\lambda$, the equivalent process is as follows: select an edge from the network with probability $\lambda$, and the outbreak of the disease means that the set of chosen edges forms a connectivity cluster. The cascading failure is a site percolation process [26,27]. There are several nodes participating in both bond percolation and site percolation processes. We assume that these nodes belong to the site percolation process rather than to the bond percolation process. The reason for this assumption is that, although the cascading failure is triggered by the spreading of epidemic, once cascade starts with a small value of $\alpha$, most of the paths linked to infected nodes are cut off by the cascading failure, and thus the number of failed nodes from the cascading failure will be much larger than that of newly infected nodes. Therefore, most of the nodes participating in both bond percolation and site percolation processes are considered to be failed in cascades eventually, rather than removed in epidemic spreading. Note that there is a
that a failed node shares with each of its neighbors is set to 0.01.

In the simulations, $\Delta$, the number of units of load $N$ in the end. Denote $s$ as the probability that a node of degree $k$ happens to be a quasi-failed node, and it becomes inactive as shown in fig. 2:

- **Quasi-infected** node, a node at least one of whose edges participates in the bond percolation process.

- **Quasi-failed** node, a node takes part in the site percolation process.

A quasi-failed node will fail in cascades undoubtedly, while a quasi-infected node will become removed or failed in the end. Denote $s_k$ as the probability that a node of degree $k$ happens to be a quasi-failed node, and $b_k$ as the probability that a node of degree $k$ happens to be a quasi-infected node. Eventually, this node happens to be failed with probability $s_k$, or be removed with probability $(1 - s_k)b_k$.

Furthermore, in this model, we call a node:

- an **$m$-vulnerable** node, if and only if it has $m$ quasi-failed or quasi-infected neighbors, and it becomes inactive if all of the $m$ neighbors fail and share load with it;

- a **vulnerable** node, if it is an $m$-vulnerable node, where $m \geq 1$.

The cascading failure is a site percolation process, thus, considering the locally tree-like approximation in percolation, the probability that a node of degree $k$ happens to be a vulnerable node is

$$f_k = \sum_{m=1}^{k-1} f_k^{(m)},$$

where $f_k^{(m)}$ is the probability that it is an $m$-vulnerable node.

When $\alpha$ is small, the number of quasi-failed nodes is larger than that of quasi-infected nodes, as the time scale of cascading failure is much smaller than that of epidemic spreading. Thus, vulnerable nodes can be approximated to quasi-failed nodes, as $f_k \approx s_k$.

**Critical value of the tolerance parameter.** In a degree-degree uncorrelated network, consider a node $v$ of degree $k$ and any of its neighbors $w$. The probability of node $w$ with degree $j$ is $\sum_{w} j p_j = \langle j \rangle/k$, where $\langle k \rangle$ is the average degree of the network. Node $w$ becomes vulnerable or infected with probability $1 - (1 - s_j)(1 - b_j) = s_j + (1 - s_j)b_j$. Therefore, the probability of node $v$’s arbitrary neighbor being quasi-failed or quasi-infected is

$$\sum_{j} j p_j (s_j + (1 - s_j)b_j) / \langle k \rangle \leq \sigma_f + \sigma_r,$$

where $\sigma_f$ and $\sigma_r$ are the probabilities that node $v$’s arbitrary neighbor happens to be a failed or removed node in a steady state, respectively, as follows:

$$\sigma_f \triangleq \sum_{j} j p_j s_j / \langle k \rangle,$$

$$\sigma_r \triangleq \sum_{j} j p_j (1 - s_j)b_j / \langle k \rangle.$$
According to eq. (3), the probability that exactly $m$ of node $v$’s neighbors are quasi-failed is

\[
\binom{k-1}{m} (\sigma_f + \sigma_r)^m (1 - \sigma_f - \sigma_r)^{k-1-m} \varphi \left( \frac{m\Delta}{\alpha} \right).
\]

(6)

For node $v$, its initial load $L_v$ is uniformly distributed in the range $[0, 1]$, so the cumulative distribution function of $L_v$ is

\[
P\{L_v < l\} \triangleq \varphi(l) = \begin{cases} 
0, & l \leq 0, \\
1, & l > 1.
\end{cases}
\]

(7)

The probability that node $v$’s failure is triggered by the failure and loads sharing from exact $m$ of its neighbors is

\[
P\{L_v + m\Delta > (1 + \alpha)L_v\} = P\{L_v < \frac{m\Delta}{\alpha}\} = \varphi \left( \frac{m\Delta}{\alpha} \right).
\]

(8)

Then, the probability that node $v$ is an $m$-vulnerable node is

\[
f_k^{(m)} = \binom{k-1}{m} (\sigma_f + \sigma_r)^m (1 - \sigma_f - \sigma_r)^{k-1-m} \varphi \left( \frac{m\Delta}{\alpha} \right).
\]

(9)

We substitute eq. (9) into eq. (2), and get

see eq. (10) above

where $\lfloor \cdot \rfloor$ is the floor function.

Equation (10) provides the probability that a node of degree $k$ turns out to be a quasi-failed node. Then, we will handle the probability that nodes of degree $k$ participate in bond percolation. The node $v$ will be infected with probability $\lambda$ for each edge connecting to a quasi-infected node. Considering the cavity probability in percolation, the probability that node $v$ arrives to be a quasi-infected node is

\[
b_k = 1 - (1 - \lambda\sigma_r)^{k-1}.
\]

(11)

According to eqs. (10) and (11), we have the probability that a node of degree $k$ is a removed node in epidemic spreading finally as

\[
r_k = (1 - s_k)b_k = (1 - f_k)(1 - (1 - \lambda\sigma_r)^{k-1}).
\]

(12)

Now we complete the derivation by eqs. (4), (5), (10), and (12). These four equations depend on each other. We can get $f_k$, $r_k$, the fraction of removed nodes $S_r$, and the fraction of failed nodes $S_f$ in a steady state from these four equations by the following iteration process:

1) Set the initial values. Assign a very small initial value to $r_{k=0}$, where $k_{min}$ represents the minimum degree of all nodes. Let $r_k = 0$ ($k > k_{min}$) and $f_k = 0$. Substitute $f_k$ and $r_k$ into eq. (4) and eq. (5) to get the initial values of $\sigma_f$ and $\sigma_r$.

2) Substitute $\sigma_f$ and $\sigma_r$ into eq. (10) and eq. (12) to get the values of $f_k$ and $r_k$. Substitute the new values of $f_k$ and $r_k$ into eq. (4) and eq. (5), update $\sigma_f$ and $\sigma_r$. Repeat this step until convergence.

3) Get the fraction of removed and failed nodes in a steady state by $S_r = \sum_k p_k r_k$ and $S_f = \sum_k p_k f_k$, respectively. When $\alpha$ increases, $S_r$ will change from zero to a positive value at $\alpha_c$ and this indicates when the epidemic starts to spread out.

Simulation. – We mainly focus on the fraction of active nodes in simulations, and validate the theoretical results of the critical value $\alpha_c$. We carry out the simulations in networks generated by UCM [23] with 5000 nodes, minimum degree value as 3, and degree distribution $p(k) \sim k^{-\gamma}$, where $\gamma = 3$. The results are averaged over 50 realizations in each of which the network is independently generated. The average degree of these networks is $\langle k \rangle = 4.88$. The unit of load shared in the cascading failure, $\Delta$, is fixed at 0.01.

Before exploring the fraction of active nodes, we firstly investigate the complementary states consisting of failed and removed nodes. Figure 3 presents the fractions of failed nodes $S_f$ and removed nodes $S_r$ with varied effective spreading rate $\lambda$ in UCM scale-free networks. When $\alpha$ is small, the theoretical results are in good agreement with the simulation results. While $\alpha$ is large, there is a deviation between the theoretical result and the simulation result. The reason for this deviation is that nodes which participate both in the bond percolation (epidemic spreading) and in the site percolation processes (cascading failure) are supposed to belong to the latter one in the theoretical prediction. In the simulations, a considerable amount of nodes are infected and become removed when
out locally. While $\alpha$ at abscissa is large. We can easily find that each of the curves of $\alpha$ scale-free networks with failed (b) nodes after the dynamics are terminated in UCM Fig. 3: (Color online) The fractions of removed (a) and theoretical results, the turning points (whose abscissas are $\alpha_c$) with $\lambda = 0.5, 0.6, 0.75, 1$ are marked by a solid diamond (♦), a circle (●), a square (■), and a down-pointing triangle (▼), respectively.

$\alpha$ is large. We can easily find that each of the curves of the simulation and theoretical results has a turning point at abscissa $\alpha_c$. In fig. 3(a), when $\alpha$ is smaller than $\alpha_c$, $S_f$ is very close to zero, which means the epidemic dies out locally. While $\alpha$ is larger than $\alpha_c$, $S_f$ is larger than zero, indicating the epidemic spreads out and infects a portion of the network. In fig. 3(b), when $\alpha$ is smaller than $\alpha_c$, $S_f$ decreases with increasing $\alpha$. While $\alpha$ is larger than $\alpha_c$, $S_f$ decreases more slowly, indicating the cascading failure slows down, and the epidemic spreads out and infects a fraction of the network. This is an interesting phenomenon, i.e., that even if the infection probability is very large, the epidemic still dies out when $\alpha$ is smaller than $\alpha_c$. This is because, when $\alpha$ is small, very few initial infected nodes can lead to a severe cascading failure, which cuts off the spreading paths of the epidemic.

Then we explore the fraction of active nodes $S_a$ and the fraction of the giant component $G$ formed by them when the two dynamics of epidemic spreading and cascading failure are terminated, as shown in fig. 4. Simulation and theoretical results of $S_a$ are both plotted. Since it is in a data communication network, the fraction of the largest connected sub-network formed by active nodes (the giant component of active nodes, $G$) is the important one to capture the robustness of the network with respect to the structural damage and maintain the network functions [28]. Though the sizes of $G$ and $S_a$ are different, their humps meet the peaks at almost the same $\alpha$ value which is very close to the critical value $\alpha_c$. This means that our theoretical method can be used to estimate the critical value $\alpha_c$ as well as the optimal value of $\alpha$ at which most active nodes remain. It is worth noting that the fraction of active nodes is remarkably larger than that of the standard SIR model (the size for the remaining active nodes is very close to zero with large $\lambda$ in the standard SIR model), because in our model, the cascading failure blocks the spreading of the epidemic, as we have mentioned above.

Conclusion. – In this paper, we explore a model based on the SIR epidemic spreading model and a local load sharing cascading failure model. With the change of the tolerance parameter $\alpha$, there exists a critical value $\alpha_c$ at which the epidemic with large $\lambda$ is blocked by cascading failure. When $\alpha < \alpha_c$, the cascading failure cuts off the abundance of paths and blocks the spreading of the epidemic locally. While $\alpha > \alpha_c$, the epidemic spreads out and infects a fraction of the network. We propose a method for estimating $\alpha_c$ which is applicable in degree-degree uncorrelated networks. In simulations, we validate the effectiveness of this method in UCM scale-free networks.

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