The robustness of interdependent networks under the interplay between cascading failures and virus propagation

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Abstract – Cascading failures and epidemic dynamics, as two successful application realms of network science, are usually investigated separately. How do they affect each other is still an open, interesting problem. In this letter, we couple both processes and put them into the framework of interdependent networks, where each network only supports one dynamical process. Of particular interest, they spontaneously form a feedback loop: virus propagation triggers cascading failures of systems while cascading failures suppress virus propagation (i.e., the interplay between cascading failures and virus propagation, also named CF-VP model). Under this novel model, the interdependent networks will collapse completely if virus transmissibility exceeds a crucial threshold. In addition, only when the network sustaining the epidemic dynamics has a larger average degree, will the interdependent networks become more vulnerable, which is opposite to the observation of traditional cascading models in interdependent networks. To protect interdependent networks we also propose control measures based on the identification capability: a stronger identification capability leads to more robust interdependent networks.

Introduction. – Over the past years, complex networks have proven to be a successful tool in describing a large variety of real-world complex systems, ranging from biological, technological, social infrastructures to information, engineering, and physical systems [1,2]. The investigations of the structure and dynamics of complex networks have triggered enormous interest, and a lot of remarkable results have been achieved [3–8]. However, the vast majority of the existing works mainly focus on single networks that are isolated from each other, despite the fact that many real-world networks usually interact with and depend on each other. In 2010, Buldyrev et al. [9] proposed a new model of networks, named interdependent networks, and developed a theoretical framework to study the cascading failures of interdependent systems caused by random node removal. Surprisingly, they found that systems made of interdependent networks would be intrinsically more fragile than each of the isolated components. After that, much research attention moves to more complicated yet more realistic multilayer networks, mainly including interdependent networks [10–14], interconnected networks [15–19] and multiplex networks [20–24].

Cascade of failures, as one of the hottest research topics in network science, has attracted great attention after the seminal idea of Buldyrev [10–14,25–30]. For example, Parshani et al. explored the influence of degree correlation...
on the robustness of interdependent networks to cascading failures, and found that the systems become more robust when they share higher inter-similarity [11]. By mapping the random attack to the targeted attack problem, Huang et al. evaluated the cascading failures in interdependent networks under an initial targeted attack [27]. Shao et al. developed a theoretical framework for understanding the cascade process of failures in interdependent networks with a random number of support and dependence relationships [28]. References [29,30] showed that when a small fraction of autonomous nodes are properly selected, the nature of the percolation transition changes from discontinuous to continuous fashion and the cascading failures could be largely suppressed.

Epidemic dynamics [4], as another rapidly developing research area in network science, is broadly used to mimic many real propagation processes, such as disease in human contact networks [31,32], information and rumor in social networks [33], and virus in computer or communication networks [34,35]. Understanding the epidemic spreading processes is thus crucial for developing efficient methods to either prevent propagation of disease, rumor and virus, or accelerate information dissemination [36–38]. At present, the most popular models to describe the propagation of epidemic include the susceptible-infected (SI) model, the susceptible-infected-susceptible (SIS) model, and the susceptible-infected-recovered (SIR) model [4]. Like other dynamic processes upon networks [23], the recent concerns of epidemic spreading also extend from single networks to multilayer networks [15,16,22,33].

In spite of the great progress of the recent years, cascading failures and epidemic dynamics are usually considered as two independent research topics and are studied separately. However, in many real-world systems, cascading failures and epidemic dynamics often influence and interact with each other. For example, the virus propagation on a communication network not only causes node failure or load redistribution of the communication network, but also triggers the collapse of other related networks like power grid due to the interdependence relationships between them, thus resulting in cascading failures in interdependent systems. The cascade of failure on other networks, in turn, enables more nodes or fragmentation to be removed in the communication network, which, however suppresses the propagation of the virus. In particular, if the virus is not completely suppressed, it will lead to a new crash of the nodes and thus triggers successive cascading failures in the interdependent networks. Compared with the existing research on the robustness of interdependent networks, the above case introduces a novel attack method for interdependent networks.

In this letter, we develop a new framework where the virus propagation could induce cascading failures and cascading failures are able to suppress virus propagation (i.e., forming feedback between cascade dynamics and epidemic dynamics). By means of numerous simulations, we will investigate the interplay of both processes in interdependent scale-free (SF) networks [39], and explore the robustness of interdependent networks under this novel setup.

**Model.** – Before defining the detailed model, we first survey the general cascading failures of interdependent networks and the SIR dynamics which we use as the paradigmatic example for the collapse process of interdependent systems under the attack from the spreading virus.

The general cascading failures in interdependent networks were first proposed in ref. [9], where there are two networks A and B with the same size of \( N \) nodes, coupled via one-to-one interdependence. If node \( A_i \) (\( B_i, i = 1, 2, \ldots, N \)) stops functioning owing to attack or failure, its inter-layer counterpart \( B_i \) (\( A_i \)) becomes non-functioning as well. When some nodes on network A (hereafter A-nodes) are removed, the nodes of network B (hereafter B-nodes) that connect to the non-functioning A-nodes will also be removed (because of the dependence between both networks), which further prunes connections of these B-nodes with the giant component of network B. Subsequently, A-nodes that connect to the non-functioning B-nodes will stop functioning and cut their connections with the new giant component of network A (only the nodes that belong to the giant component of the network remain functioning). These cascade processes repeat until no A-nodes and B-nodes could be removed.

The SIR model [4], as one of the most fundamental and important paradigms of epidemic dynamics, classifies the network nodes into three states: susceptible (S), infected (I), or recovered or removed (R). Susceptible nodes are free of epidemic and can get infection via direct contacts with infected counterparts. Infected nodes are assumed to carry the disease and pass it towards susceptible nodes. The recovered (removed) state means that the nodes recovered (died) from the disease so that these nodes neither diffuse the infection nor get infected again. In addition, classic SIR model considers a discrete time process: at each time step, the infected node can infect its susceptible neighbors with transmission rate \( \lambda \), and then becomes recovered or removed with recovery rate \( \delta \).

Now, we turn to our model: cascading failure and disease spreading are coupled via interdependent networks, namely, the interplay between cascading failures and virus propagation (CF-VP for short) in interdependent networks. Given the same interdependent networks as those in ref. [9], we give two additional hypotheses: 1) only one network (e.g., network A) supports the propagation of virus; 2) the time scale of cascading failures is much smaller than that of virus propagation, so that virus propagation can repeat until there is no failure node in the systems. Moreover, our model also considers a discrete time step: each time stage contains the virus propagation process and one general cascading failure process. Initially, one randomly chosen A-node is infected by the virus, then the infected node propagates the virus: it infects its susceptible neighbors with a probability \( \lambda \), and then gets
removed with a probability $\delta$ (without loss of generality, we use $\delta = 1$). In particular, if the removed nodes are assumed to be non-functioning, a general cascading failure process will be triggered in interdependent networks and more nodes may be pruned. If there still exist infected nodes in the networks after the cascading process, a new virus propagation process and the subsequently triggered cascading failure will repeat until no infected nodes exist in the network.

To get a better understanding, fig. 1 provides a schematic example for this novel CF-VP model. Assume $A_2$ being initially infected, it can infect its neighbors with probability $\lambda$. After the spreading process of stage 1 (i.e., stage 1-1), $A_2$ and $A_3$ become removed node and infected node, respectively. Due to interdependence, node $B_2$ will be non-functioning, which subsequently causes node $B_1$ to be removed since it does not belong to the giant component of network $B$. Similarly, $A_4$ is removed because of the removal of $B_1$. The first stage ends. Now there exists a new infected node $A_3$; it can bring infection to its neighbors $A_4$ and $A_5$. Since $A_3$ becomes non-functioning soon, a new cascade process is triggered: $B_3$ is removed due to the loss of the dependent counterpart; $A_4$ is removed because of the separation from the giant component of network $A$, which, in turn, causes $B_4$ to be non-functioning. At stage 3, even if $A_5$ fails to infect its neighbor, itself and its partner $B_5$ will still become non-functioning due to the state transition $1\rightarrow R$ of $A_5$. Because no giant component exists, $A_6$ and $B_6$ are finally removed and the systems are completely collapsed. From this illustration, it is clear that the virus propagation causes cascading failures, while the cascading failures suppress the virus propagation: the S-state node $A_1$ and the I-state node $A_4$ are isolated owing to the cascading failures. Of course, we notice that this simple model may be applicable to some empirical systems, such as interdependent networks composed of online social network and realistic contact network, which yet needs more interdisciplinary collaboration (especially combining with network data).

**Results.** – Results of computation simulations are obtained on interdependent scale-free (SF) networks with average degree $\langle k_A \rangle$ and $\langle k_B \rangle$ of networks $A$ and $B$. In each CF-VP process, we assume that, initially, only one randomly chosen node is infected on network $A$. What we are interested in is the robustness of interdependent networks against the CF-VP process, which is measured by the relative size $G$ of the remaining giant component of network $A$ (normalized by network size $N$) when the CF-VP ends. Here, it is worth mentioning that the CF-VP model finally generates the identical size of the remaining giant component on networks $A$ and $B$.

Figure 2 shows the evolution of the virus on network $A$ with the proposed CF-VP model, which is featured by the solid lines. To make a direct comparison, we also add the traditional case of virus propagation on single-layer networks (dashed lines, i.e., without the interplay of cascading failure and virus propagation). It is obvious that although the fraction of infected nodes in both scenarios is almost identical at the early stages, the following trend becomes greatly different. Comparing with the traditional case, the CF-VP model not only makes infection reach a peak faster, but also impedes the total infection risk. In fact, it is easy to explain these phenomena. At the early stages, only a small fraction of nodes are infected and removed, the interdependent networks are not broken...
and most nodes are still functioning. Thus, the virus propagates on network A almost as that on single-layer networks. But with continual propagation of virus, the triggered cascading failures cause more nodes to be removed and make interdependent networks collapse into the unconnected fragments. In particular, many infected and susceptible nodes are also removed due to the cascading failures, which, in turn, lead to the effective suppression of virus propagation (see also fig. 1). With the CF-VP framework, the role of the feedback loop becomes clear: virus propagation induces cascading failure, while cascading failure suppresses virus propagation.

Besides, another interesting observation from fig. 2 is that, similarly to the traditional case, the spreading scale of the virus is larger in network A with denser connections (i.e., the larger the average degree, the more obvious the infection peak), which makes the total transmission easier. Due to the feedback loop (refer to fig. 1), this should in turn cause larger-scale cascading failures in interdependent systems and make systems more vulnerable to the CF-VP model, which we will systematically discuss in what follows.

To explore the influence of the CF-VP model on the robustness of interdependent networks, we focus on two opposite cases. The first case is to fix the average degree of network B \(\langle k_B \rangle\) and yet vary the average degree of network A \(\langle k_A \rangle\); another case is to fix \(\langle k_A \rangle\) yet vary \(\langle k_B \rangle\). (Indeed, there exists the third case: keep \(\langle k_A \rangle\) and \(\langle k_B \rangle\) equal, i.e., \(\langle k_A \rangle = \langle k_B \rangle\), and simultaneous changing, like in fig. 2. But here we do not plot the curves of this case, which will be explained soon.) Interestingly, such a change that seems trivial will lead to greatly different outcomes. First, irrespectively of which case, increasing \(\lambda\) makes \(G\) smaller, namely, the fast propagation of virus will trigger a larger, stronger crash of the systems. In particular, there exists the critical threshold of virus transmissibility, \(\lambda_c\) (see footnote 1), above which the remaining giant component will be null. From fig. 3, we can see that \(\lambda_c\) becomes smaller with the increment of \(\langle k_A \rangle\), which means that denser connections of network A where virus spreading takes place will accelerate the propagation of the virus and thus the progression of cascading failures in the systems. By contrast, the change of \(\langle k_B \rangle\) has no obvious impact on the threshold \(\lambda_c\) (see fig. 4), i.e., the crash trend is nearly identical if only \(\langle k_B \rangle\) changes. Combining these observations, a significant finding poses itself: the interdependent systems will be more vulnerable only if the network layer supporting virus propagation has denser connections (i.e., larger average degree); otherwise the interdependent systems are robust against the change of connections in other layer(s). Along this discovery, it now becomes easy to understand that simultaneously changing \(\langle k_A \rangle\) and \(\langle k_B \rangle\) will generate the same results as fig. 3. In addition, this discovery also differs from previous framework of cascading failure in interdependent networks [11], where better robustness usually needs denser connections. Thus, our outcomes, to some extent, prove the necessity and significance of feedback loop when designing the interdependent networks.

**Control strategy.** — Up to now, it has been very clear that in interdependent networks virus propagation on a layer could lead to continuous cascading failures and fragmentation of systems. Along this line, the most intuitive method of protecting interdependent networks is to control the spread of virus when it appears. In reality, it seems hard to timely restrain the spreading of virus (especially the emerging virus) by using the well-known

\[1\] Here, the coupling between cascading failures and virus propagation makes the theoretical calculation of the threshold value rather complicated. We define \(\lambda_c\) as the maximum value of \(\lambda\) leading to no more than 1% nodes remaining in the final state of the network. In the computation simulation, the threshold value is obtained through increasing the value of \(\lambda\) with a step 0.01.
respectively. Moreover, if the emergence of the virus, the susceptible node is better isolated from their infected neighbors, but also decreases the average degree of the network layer which supports the virus propagation (see fig. 3 for its impact). With respect to the identification capability, regardless of which strategy. With a large identification probability, the infection source(s) can be controlled and isolated earlier. The removal of these infected nodes further slows down the cascading process, which also decreases the possibility of infection outbreak. This thus validates the importance of the feedback loop in the coupled disease-cascading model once again. Moreover, another similar phenomenon in figs. 5(a) and (b) is that network A possessing a large average degree needs larger $q$ to maintain the equivalent robustness with the case of small $\langle k_A \rangle$, which in fact is consistent with the prediction of fig. 3: larger $\langle k_A \rangle$ usually makes systems more vulnerable, thus requiring more powerful protection. Furthermore, we can observe that degree-based adaptive isolation performs much better than deterministic adaptive isolation. This actually agrees with our intuition, because (as single-layer networks) large-degree nodes play a more significant role in the propagation of a virus than small-degree nodes do. If there exist infected nodes among the neighborhood of large-degree nodes, they can easily prune the connections with infected neighbor(s) due to a great identification ability. With fast removal of infection sources, cascading will be controlled better (i.e., larger $G$ for the same $q$ value in degree-based adaptive isolation).

We now turn to another case: fixing $\langle k_A \rangle$ and yet varying $\langle k_B \rangle$ and we study how the isolation strategies improve the robustness of interdependent networks. As reflected in fig. 4, this case has no impact on the system crash. Although the relative size $G$ of the remaining giant component enhances with identification capability $q$ and the degree-based adaptive isolation performs better, only changing $\langle k_B \rangle$ will generate nearly identical results within each isolation strategy (i.e., the overlapped curves in fig. 6). This is because, for each $q$ value, the isolation probability of infected neighbors on a network supporting virus propagation is the same, irrespectively of the average degree in other network. Combining figs. 5 and 6, this seems to indicate that the best way of controlling the system crash is to eradicate the infection sources in the epidemic layer, which is specially useful when this layer has denser connections.

Fig. 4: (Colour online) The relative size $G$ of the remaining giant component of network $A$ vs. transmissibility probability $\lambda$. The interdependent networks are SF networks with average degree $\langle k_A \rangle = 8$, $\langle k_B \rangle = 4$ (squares), 6 (triangles), 8 (circles), 10 (diamonds), and 16 (stars), respectively. The inset features how the threshold $\lambda_c$ changes as a function of $\langle k_B \rangle$.

Fig. 5: (Colour online) The relative size $G$ of the remaining giant component of network $A$ vs. identification probability $q$ for the deterministic isolation case (a) and degree-based isolation $\langle k_B \rangle = 8$, $\langle k_A \rangle = 4$ (squares), 6 (triangles), 8 (circles), 10 (diamonds) and 16 (stars), respectively. The transmissibility probability is $\lambda = 0.5$.

Pre-immunization strategies [40–42], due to the absence of effective antivirus programs [35]. However, in the CF-VP model it seems feasible to identify the infected neighbor based on knowledge and abnormal behavior of infected nodes. Here we consider such a control strategy: after the emergence of the virus, the susceptible node $i$ can identify one infected neighbor with probability $q_i$, and then prunes its connection with this neighbor. This strategy not only isolates the healthy nodes from their infected neighbors, but also decreases the average degree of the network layer which supports the virus propagation (see fig. 3 for its impact). With respect to the identification capability $q_i$, we consider the two following cases.

1) Deterministic adaptive isolation: $q_i = q_j = q$ for $i \neq j$. That is, all of the nodes have the same ability to identify infected neighbors.

2) Degree-based adaptive isolation: $\{q_1, q_2, \cdots, q_N\}$ following Gaussian distribution. That is, $\{q_1, q_2, \cdots, q_N\} \sim N(q, \sigma)$, where $q$ and $\sigma$ are mean and standard deviation, respectively. Moreover, if $k_i \geq k_j$, we assume $q_i \geq q_j$, which means that large-degree nodes have a greater ability to identify infected neighbors. Considering that $q_i$ must be between 0 and 1, we assign $q_i$ as

$$q_i = \begin{cases} 0, & \text{if } q_i < 0, \\ q_i, & \text{if } 0 \leq q_i \leq 1, \\ 1, & \text{if } q_i > 1. \end{cases}$$

Subsequently, we explore how the control measures improve the robustness of interdependent networks under the CF-VP model, where we still use two opposite cases as shown in figs. 3 and 4. Figure 5 first shows the impact of isolation strategies when $\langle k_B \rangle$ is fixed and $\langle k_A \rangle$ changes. It is clear that the relative size $G$ of the remaining giant component increases with $q$, which indicates that the robustness of interdependent networks can be significantly improved by increasing the nodes’ identification capability, regardless of which strategy. With a large
be also worthy of our endeavors in the future. Between dynamics and interdependent network topology will terplay between dynamical processes, the co-evolution be-
the theoretical analysis framework, which may validate another point that deserves our attention is to consider may provide some new insights into understanding the in-
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more vulnerable, which is the opposite to the observation has denser connections, interdependent systems will be
propagation triggers continuous cascading failures and even complete fragmentation if the transmissibility probability is above a threshold, while cascading failures will break the connections of networks and thus suppress virus propagation. It is worth noting that, if the network layer supporting epidemic spreading has denser connections, interdependent systems will be more vulnerable, which is the opposite to the observation of the traditional cascading fashion in interdependent networks [9]. To protect interdependent networks, we further propose the control measures based on the capability to identify the infected neighbor. Interestingly, the larger the identification capability (especially for larger-degree node), the more robust the interdependent networks.

In spite of simplicity, our model describing the interplay between cascading failures and virus propagation in interdependent networks seems reasonable and also easily justifi-
able with realistic situations. For example, Internet and some social online networks could be encapsulated into the framework of multilayer networks. But how they in-
fluence each other will be a long-term question. This work may provide some new insights into understanding the inter-
play and proposing the protection measures. Besides, another point that deserves our attention is to consider the theoretical analysis framework, which may validate the present simulation findings. In addition to the inter-
play between dynamical processes, the co-evolution be-
tween dynamics and interdependent network topology will be also worthy of our endeavors in the future.

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