Enhancing resilience of interdependent networks by healing

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Interdependent networks are characterized by two kinds of interactions: The usual connectivity links within each network and the dependency links coupling nodes of different networks. Due to the latter links such networks are known to suffer from cascading failures and catastrophic breakdowns. When modeling these phenomena, usually one assumes that a fraction of nodes gets damaged in one of the networks, which is followed possibly by a cascade of failures. In real life the initiating failures do not occur at once and effort is made replace the ties eliminated due to the failing nodes. Here we study a dynamic extension of the model of interdependent networks and introduce the possibility of link formation with a probability $w$, called healing, to bridge non-functioning nodes and enhance network resilience. A single random node is removed, which may initiate an avalanche. After each removal step healing sets in resulting in a new topology. Then a new node fails and the process continues until the giant component disappears either in a catastrophic breakdown or in a smooth transition. Simulation results are presented for square lattices as starting networks under random attacks of constant intensity. We find that the shift in the position of the breakdown has a power-law scaling as a function of the healing probability with an exponent close to 1. Below a critical healing probability, catastrophic cascades form and the average degree of surviving nodes decreases monotonically, while above this value there are no macroscopic cascades and the average degree has first an increasing character and decreases only at the very late stage of the process. These findings facilitate to plan intervention in case of crisis situation by describing the efficiency of healing efforts needed to suppress cascading failures.

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I. INTRODUCTION

Robustness is one of the key issues for network maintenance and design \cite{1,2,3}. The representation of complex systems has been limited to single networks for a long time \cite{4}. In many cases, however, coupling between several networks takes place \cite{5,6}. An important case is that of interdependency \cite{7,8} where there are two kinds of links: connectivity and dependency links. An example of interdependent networks is the ensemble of the Internet and the power supply grid where telecommunication is used to control power plants and electric power is needed to supply communication devices \cite{9}. Connectivity links model the relation of the entities within the same sector, spanning in the above example a power supply network and a telecommunication network. Dependency links depict the basic supplies an entity depends on which are supplied by entities in the other network. If a supplier fails its dependent nodes fail as well. The system is viable if a giant component of interconnected units exists in both networks. In the 28 September 2003 blackout in Italy it came to evidence that the interdependency of the two networks makes them more vulnerable than ever thought before \cite{10}. Similar relations occur in the economics between banks and firms or funds. Banks are related through interbank loans, firms through supply chains and the interdependence comes from loans and securities. Inappropriate asset proportions can also lead to global avalanches as seen in the subprime mortgage crisis \cite{11}.

Interconnecting similar subsystems used to increase capacity was shown beneficial as long as it does not open pathways to cascades \cite{12}. However, in interdependent networks, the aspect of robustness was considered with the conclusion that broadening the degree distribution of the initial networks enhances vulnerability \cite{13}. A cost-intensive intervention to strengthen robustness is to upgrade nodes to be autonomous on some resources \cite{14}.

Because failures propagate rapidly in infrastructure networks, they cannot be stopped by installing backup devices during the spreading of the damage, but rather they require already existing systems. After the cascade of failures, damaged devices or elements can be replaced by new, functioning ones identical to the originals \cite{15}. In contrast to engineered systems, social or economic networks are highly responsive and may react quickly \cite{16,17}. When a failure occurs considerable effort is made to reorganize the network and rearrange the load of failing elements among functioning ones. The role of the failing entities is taken over by similar participants. Such processes can be modeled by healing, i.e., substituting some of the failed elements by new ones. The timescale of an economic crisis is wide enough for the network to completely restructure itself \cite{18}. So far such mechanisms have only been studied for simple net-
Figure 1. a) Failures, represented by red dots, affect the nodes one by one in a random order. Whenever a node fails, its counterpart, that is, the node in the other network which depends on it, fails as well. In both networks, only the largest connected component (LCC) survives. This constraint can cause further nodes to fail in both networks, which trigger further shrinking of the LCC, and so on, illustrated by the shaded areas. b) The neighbors of a failing node try to heal the network, such that two functioning neighbors of a removed node establish a connectivity link with probability $w$.

The outline of the paper is as follows. In Sec. II we define the node failure process in a dynamic way. We introduce initial failures one by one to be able to apply healing at every failure event. Then we relate the original version of cascading failures to our model as a special case and give formulas for comparing the order parameter of the two models. The scaling properties of the healing are explained along with the numeric results in Sec. III. In Sec. IV we discuss the properties of the cascades with microscopic insight to the model. Finally we conclude our findings in Sec. V.

II. THE MODEL

In the standard model of interdependent networks the computer-generated model-system is built up of two topologically identical networks $A$ and $B$, e.g., square lattices of size $N = L \times L$, where each node has connectivity links within the same network. In addition, dependency links couple between the networks, which are bidirectional one-to-one relationships connecting randomly selected pairs of nodes from the two networks. If any of the nodes fails its dependent pair fails too. A node in any network can function only if it is connected to the largest connected component of that network the node which it depends on is also functional, otherwise it fails, i.e., it is removed from the network.

The existence of a macroscopic connected component in a single network is treated by percolation theory. In the usual case, for a lattice it describes a second-order phase transition between the phases with and without the existence of a giant component. Adding interdependency allows cascades of failures to propagate between the two networks. The threshold the network can survive without collapse decreases considerably in this setting.

The collapse due to cascades was shown to be a first order transition if the dependency links have unlimited range while the transition is of second order if the range is less than a critical length $r_c$. Moreover, the first order transition has a hybrid character with scaling on one of its sides.

As mentioned in the Introduction we first introduce a dynamic process on the interdependent network model. In the setting of two interdependent networks of general topology this dynamic process consists of the repetition of attacks and relaxations to a rest via cascades. (See Fig. 1.) Let us suppose that failures affect the nodes one by one in a random order which defines a timeline. One time step is identified with the external attack of one node. Time is measured by the number of time steps normalized by $N$ for systems of different sizes to be com-
parable:

\[
elapsed\ time = 1 - p = \frac{\text{number of time steps}}{N}
\]

The externally introduced failure in network \( A \) may separate the largest connected component (LCC) into two or more parts where only the largest one survives. All the failed nodes have dependency connections to nodes of the network \( B \) causing their failure. Again, the LCC of \( B \) may get fragmented and only the largest part survives. This cascading procedure is repeated until no more failures happen. Of course, our model can easily be generalized to any number of interdependent networks and any density of dependency links.

Our aim is to introduce healing into this dynamic model. The procedure is as follows: After an externally introduced failure (which may cut off a part of the LCC) the healing step follows. Two remaining, functioning neighbors of a removed node establish a connectivity link with an independent probability \( w \). (See part b) in Fig. 1.) Then the dependent nodes of the removed nodes are removed from the other network. After the propagation of the failure there, again, two functioning neighbors of a removed node establish a connectivity link probability \( w \). Due to the separation of small components, further damages might propagate back and forth within the network, always followed by a healing step. Here, the healing step means that all pairs of neighbors of each failed node is considered as a candidate for a new connectivity link with an independent probability \( w \). Then, after having selected the candidates, the connectivity links are established simultaneously. The process goes on until no more separation of components occurs. The healing links may change the topology considerably, bridging larger and larger distances as the time goes on (Fig. 2). Once a critical fraction \( (1 - p_c) \) of nodes are removed, a catastrophic cascade destroys the remaining system.

The \( w = 0 \) case is simply the dynamic version of the well studied model of Li et al. In [8] a fraction \( (1 - q) \) of the original network is destroyed in the first step then the size of the giant component after the relaxation of the original network is destroyed in the first step then randomly shuffling the end of the links. The \( p_c \)-s are then obtained averaging over the vertical axis: for a given number of surviving nodes \( P_\infty(p,w) \), we averaged the proportion of nodes \( 1 - p \) attacked one-by-one. Fig. 3 shows the averaged curves for different values of \( w \). The shape of the \( P_\infty(p,w) \) curves suggests the scaling in the form of anisotropic resizing from the \( S(p = 1, P_\infty = 1) \):

\[
1 - P_\infty(1 - p, w) = 1 - a(w) P_\infty \left( \frac{1 - p}{c(w)}, 0 \right)
\]

which is asymptotically satisfied in the \( w \rightarrow 0 \) limit.

In the infinite lattice limit, the initial few attacks almost surely occur in different parts of the lattice and do not raise cascades, only the attacked points fail, \( P_\infty(p) = p \) if \( p \sim 1 \). The unit slope at \( S \) with respect to \( p \) can be expressed by differentiation and yields \( a(w) \equiv c(w) \). Let us express the fraction of unattacked nodes relative to the threshold without healing: \( \Delta p = p - p_{c,0} \leq 0 \). The change in the threshold value \( \Delta p_c(\omega) = p_c(\omega) - p_{c,0} \) can be identified by the largest \( \Delta p \) where \( P_\infty \) has an infinite slope (see Fig. 4): \( \lim_{\Delta p \rightarrow \Delta p_c(\omega) + 0} \frac{P_\infty(1 - p_{c,0} - \Delta p, w)}{\Delta p} \). Substituting it into (2) yields \( a(w) = \frac{1 - p_{c,0} - \Delta p_c(\omega)}{1 - p_{c,0}} \). The increase in lifetime, \( -\Delta p_c(\omega) \), has a general scaling behavior expressed in

\[
-\Delta p_c(\omega) = h w^\gamma
\]

for small \( w \)-s, in the range \([0.000, 0.050]\). For the purpose of precise measurement we created simulation data for all system sizes with step size 0.001 for \( w \in [0.000, 0.010] \) additional to that shown in Fig. 4. The measurement is hampered by large fluctuations of the small systems, therefore we extrapolated to infinite system size using standard finite size scaling [24]. We used both systems.
nodes not attacked externally. Note: In order to sharply mark the breakdown, averaging in variable $p$ is done for a given $P_\infty(p)$ over 60 simulations. (right) The same curves scaled on each other using relation (3). In (both) parts, plots from the right to the left correspond to the range $w = 0.00$ to $w = 0.38$ respectively with a step size 0.02, solid lines indicate steps of 0.06.

Figure 3. (left) The fraction $P_\infty(p)$ of remaining nodes of the original $N = 320 \times 320$ nodes as a function of the fraction $p$ of nodes not attacked externally. Note: In order to sharply mark the breakdown, averaging in variable $p$ is done for a given $P_\infty(p)$ over 60 simulations. (right) The same curves scaled on each other using relation (3). In (both) parts, plots from the right to the left correspond to the range $w = 0.00$ to $w = 0.38$ respectively with a step size 0.02, solid lines indicate steps of 0.06.

The critical healing is defined as the lowest $P_\infty$ which the network size (number of nodes involved compared to the starting size network) of typical cascades is small up to the point of breakdown. The healing dynamics changes the network topology and the average degree as well. Fig. 6 allows us to describe a transition: below a critical healing threshold $w_c$ we find a sharp breakdown in the number of surviving nodes. The critical healing is defined as the lowest $w$ for which the $P_\infty(p)$ function does not have an infinite slope. In our simulation we observe $w_c = 0.351 \pm 0.002$. For $w > w_c$ also there is no macroscopic cascade and $P_\infty(p)$ goes smoothly to zero in a second-order transition as $p$ decreases (see also Fig. 3).

### IV. CASCADES CHANGE TOPOLOGY

We call cascades all events involving more nodes than the attacked one and its dependency counterpart. The size (number of nodes involved compared to the starting lattice size) of typical cascades is small up to the point of breakdown.

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The average degree on the horizontal axis as a function of the fraction of dead nodes on the vertical axis for the starting $N = 320 \times 320$ system size. The average degree remains constant for $w_c = 0.348 \pm 0.003$. Plotted lines from the right to the left correspond to the range $w = 0.340$ to $w = 0.360$ respectively with a step size $0.001$, solid lines indicate steps of 0.005. The shaded areas represent 0.33 standard deviations in the left part. In the inset, shaded areas are only plotted for solid lines and represent 1.00 standard deviation. (right) The fraction $P_\infty(p)$ of failing nodes as a function of the fraction $p$ of nodes not attacked externally using the same averaging as in Fig. 6. Above $w_c = 0.351 \pm 0.002$ there is no breakdown.

The healing performed by the $k$ neighbors introduces $w(k)$ new links on average. A rough mean-field estimate of $w_c$ is the healing probability, which conserves the average degree in the initial settings, leading to $2w_c(k) = k$ (each link joins 2 nodes). As the square lattice has $k = 4$, the result is $w_c^{\text{mean-field}} = 1/3$. According to the left plot in Fig. 6 we find that the average degree $k = 4$ changes least through the simulation for $w_c = 0.348 \pm 0.004$, which agrees well with the critical healing determined from the $P_\infty$ curves. The change in the topology along with the trend of the average degree can be observed in Fig. 2. Below the critical healing $w_c$, the average degree is monotonically decreasing function of $1 - P_\infty$ and the connectivity links remain local, conserving the disordered lattice-like topology. Thorough inspection shows that all simulations end with a cascade wiping out all of the remaining network at $p_c(w)$. Above $w_c$, the healing promotes the formation of densely connected regions and connectivity links begin to join distant nodes. We remark that in the terminal stage the defined dynamics removes all nodes and links in both cases. In summary, the difference is that for $w < w_c$ the process terminates with a macroscopic cascade, while for $w > w_c$ there is no macroscopic cascade. In the latter case the average degree increases until it has to decrease due to the small number of remaining nodes.

V. CONCLUSIONS

We examined the consequences of healing by edge formation in interdependent networks under random attack. We found that the increase in resilience of the network, measured in the number of survived attacks, has power-law scaling with the probability $w$ of healing. By establishing new random links in the neighborhood of the failed nodes, we delayed the collapse of the network through the hindering of cascades. We found that it is possible to completely suppress macroscopic cascading failures for healing probabilities higher than a critical value $w_c$. We demonstrated that this critical healing probability keeps the average degree of the nodes close to the initial value while the network topology changes. By analyzing healing efficiency, these findings can aid in the development of intervention strategies for crisis situations. The presented model contains a number of unrealistic features, like the starting lattice, the unbounded range and the high density of dependency links and the non-locality of the healing links. Further studies should clarify the role of these simplifications.

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