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Vulnerability of state-interdependent networks under malware spreading

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HIGHLIGHTS

- A propagation model of modern malware using several vectors of infection is studied.
- We use SI dynamics on multilayer networks with the same state of nodes across layers.
- We develop a formalism to analytically deal with this model as in a single network.
- We use as case study the network of scientific collaborations/author affiliations.
- We find that the topology of this network makes it very vulnerable to modern malware.

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ABSTRACT

Computer viruses are evolving by developing spreading mechanisms based on the use of multiple vectors of propagation. The use of the social network as an extra vector of attack to penetrate the security measures in IP networks is improving the effectiveness of malware, and have therefore been used by the most aggressive viruses, like Conficker and Stuxnet. In this work we use interdependent networks to model the propagation of these kind of viruses. In particular, we study the propagation of a SIS model on interdependent networks where the state of each node is layer-independent and the dynamics in each network follows either a contact process or a reactive process, with different propagation rates. We apply this study to the case of existing interdependent networks, namely a Spanish scientific community of Statistical Physics, formed by a social network of scientific collaborations and a physical network of connected computers in each institution. We show that the interplay between layers increases dramatically the infectivity of viruses in the long term and their robustness against immunization.

1. Introduction

In response to a request from the UK Ministry of Defense, Anderson and coworkers estimated the global cost of malware at US $370 millions in the 2010 year [1]. In this report, they explain that some of the reasons of the inefficiency of war

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against cybercrime is that malware is global and have strong externalities. In this sense, during the last years computer viruses have developed complex spreading mechanisms that allow them to propagate using several mechanisms. There are noted examples, like Conficker [2] or Stuxnet [3], which had an enormous impact in the Internet network and use these new methods of spreading. For these kind of viruses the propagation is easy and quick within a Local Area Network (LAN). However, effective security measures [4] limit the propagation of these viruses to other LANs. To overcome this limit, these viruses also make use of other secondary vector of propagation such as the social relations between humans. Due to the complexity of the virus propagation and infection, re-infection is quite common after virus removal, so it is technically complicated to clean a whole LAN quickly enough to stop the re-infection.

Contagion and epidemic spreading have been widely studied in the scientific literature, usually considering only one network [5–12] and, more recently, using several interconnected layers of networks and multiplexes [13–18] and several infectious agents [19,20]. However, none of these formalisms suits the case we are dealing with, namely a single disease which spreads over a set of agents which are interconnected through several networks, each with a different propagation regime, but in which the state of each agent in every network must be the same. This scenario is quite common in disease propagation. Opinions may circulate around society, but each network of social ties (family, close friends, work-mates, followees, etc.) affects differently our opinion depending on the contact rate and our trust. Similarly, human diseases such as flu or venereal diseases propagate with rates of infection that clearly depend on social relationships. In the case of malware spreading computers are usually connected within a local network and also through a social network of contacts that involve receiving corporate, private and spam e-mails or plugging foreign pen drives in the computers.

In this paper we develop a new formalism that applies to the study of the epidemic spreading in these kind of systems. These can be understood as a special subset of *interdependent networks* [21] with no explicit links joining the networks but where the state of any node must be the same in every layer.[1] We will hereafter call them state-interdependent networks (SINs). Our case study is the propagation of a SIS epidemic model in SINs. We show that the disease dynamics can be described in terms of a single contagion matrix that subsumes the contagion processes of all layers. This matrix can be used to calculate any node or link-dependent magnitude concerning the epidemic spreading such as the centrality of nodes or links, and the community structure of the disease. Finally, based on our formalism, we show the effect of some immunization strategies to slow down or control the epidemic dynamics. An important part of our analysis includes the study of an actual SIN, a Spanish scientific community of Statistical Physics which is connected through the social network of scientific collaborations and the physical network of the university LANs, and simulate the spreading process of a SIS disease.

2. The model

Let us consider M layers of networks formed each one by N nodes. The usual adjacency matrix is replaced by a set of matrices, $A^{(\alpha)} = \left(A_{ij}^{(\alpha)}\right)$ with $\alpha = 1, \ldots, M$, that specifies the links between nodes in each layer α. Note that, in these SINs, the state of nodes with the same label must be the same, and the change in the state of one node in one layer changes automatically his state in all other layers (see Fig. 1).

In these SINs we will study a SIS epidemic spreading in which the contagion in every layer α may propagate differently. We will assume that the epidemic spreading in each layer may follow a contact process, a reactive process or something in between [9]. To this end we define the contagion matrix $C^{(\alpha)} = (C_{ij}^{(\alpha)})$ in layer α as

$$C_{ij}^{(\alpha)} = \beta_i^{(\alpha)} \left(1 - \left(1 - \frac{w_{ij}^{(\alpha)}}{w_i^{(\alpha)}} \right)^{\lambda_i^{(\alpha)}} \right), \tag{1}$$

where $w_{ij}^{(\alpha)}$ stands for the weight of the link between node i and j, $w_i^{(\alpha)} = \sum_j w_{ij}^{(\alpha)}$ is the total strength [23] of node i, $\beta_i^{(\alpha)}$ is a constant between 0 and 1, and $\lambda_i^{(\alpha)}$ is the parameter that defines the contagion process for node i, which varies from a reactive process for the limit $\lambda_i^{(\alpha)} \to \infty$ to a contact process for $\lambda_i^{(\alpha)} = 1$.

The system state is described by the vector state $\mathbf{x} = \{x_1, \ldots, x_N\}$, with $x_i = 0$ when node i is susceptible and $x_i = 1$ when is infected. The transition rate for node i from infected to susceptible is

$$q_i^-(\mathbf{x}) = \mu x_i \tag{2}$$

where μ is the recovery rate, which we assume layer-independent since the same healing mechanisms are available to all nodes (this assumption can, however, be easily relaxed). On the other hand, the transition rate from susceptible to infected is

$$q_i^+(\mathbf{x}) = \sigma(1 - x_i) \left[1 - \prod_{\alpha=1}^{M} \prod_{j=1}^{N} (1 - C_{ji}^{(\alpha)} x_j) \right], \tag{3}$$

[1] Other references to previous work and nomenclature can be found in Ref. [22].