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Epidemic spreading and immunization strategy in multiplex networks

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Abstract. A more connected world has brought major consequences such as facilitate the spread of diseases all over the world to quickly become epidemics, reason why researchers are concentrated in modeling the propagation of epidemics and outbreaks in multilayer networks. In this networks all nodes interact in different layers with different type of links. However, in many scenarios such as in the society, a multiplex network framework is not completely suitable since not all individuals participate in all layers. In this paper, we use a *partially overlapped multiplex* network where only a fraction of the individuals are shared by the layers. We develop a mitigation strategy for stopping a disease propagation, considering the Susceptible-Infected-Recover model, in a system consisted by two layers. We consider a random immunization in one of the layers and study the effect of the overlapping fraction in both, the propagation of the disease and the immunization strategy. Using branching theory, we study this scenario theoretically and via simulations and find a lower epidemic threshold than in the case without strategy.

1. Introduction

In the last years the complex networks analysis has been focused in no further considering networks as isolated entities, but characterizing how networks interact with other networks and how this interaction affects processes that occurs on top of them. A system composed of interconnected networks is called a *Network of Networks* (NoN) [1, 2, 3, 4]. In NoN there are connectivity links within each individual network, and external links that connect each network to other networks in the system. Very recently physicists have begun to consider a particular class of NoN in which the nodes have multiple types of links across different *layers* called *multiplex or multilayer* networks [5, 6, 7, 8, 9, 10, 11].

Recently, the study of the effect of multiplexity of networks in propagation processes such as epidemics has been the focus of many recent researches [12, 13, 14, 15]. In Ref [16] the research concentrated in the propagation of a disease in partially overlapped multilayer networks, owing to the fact that individuals are not necessarily present in all the layers of a society and this has an impact in the epidemic propagation. For the epidemic model they used the susceptible-infected-recovered (SIR) model [17, 18, 19] that describes the propagation of non recurrent diseases in which infected individuals either die or, after recovery, become immune to future infections. In the SIR model each individual of the population can be in one of three different states:

Susceptible, Infected, or Recovered. Infected individuals transmit the disease to its susceptible neighbors with a probability β and recover after a fixed period of time t_r. The spreading process stops when there is only susceptible and/or recovered nodes. The dynamic of the epidemic is controlled by the transmissibility $T = 1 - (1 - \beta)^{t_r}$, which is a measure of the disease virulence, i.e., the effective probability that the disease will be transmitted by an infected individual across any given link. At the final state of this process, the fraction of recovered individuals R is the order parameter of a second order phase transition with a control parameter T. For $T < T_c$, where T_c is the epidemic threshold, there is an epidemic-free phase with only small outbreaks. However, for $T \geq T_c$, an epidemic phase develops. In isolated networks the epidemic threshold is given by $T_c = 1/(\kappa - 1)$, where κ is the branching factor that is a measure of the heterogeneity of the network. The branching factor is defined as $\kappa = \langle k^2 \rangle / \langle k \rangle$, where $\langle k^2 \rangle$ and $\langle k \rangle$ are the second and first moment of the degree distribution, respectively. Since the SIR model presents a local tree structure we can employ the branching theory approach within a generating function formalism [20, 21] that holds in the thermodynamic limit. In [16] the SIR model was studied, with β and t_r constant, in a system composed of two overlapping layers in which only a fraction q of individuals can act in both layers. In their model, the two layers represent contact networks in which only the overlapping nodes enable the propagation between layers, and thus the transmissibility T is the same in both layers. They found that decreasing the overlap decreases the risk of an epidemic compared to the case of full overlap ($q = 1$). They also found that the critical threshold increases as q decreases, and that in the limit of small overlapping fraction, the epidemic threshold is dominated by the most heterogeneous layer, this effect could have important implications in the implementation of mitigation strategies.

Motivated by this, in this work we study a disease spreading process in overlapped multiplex networks and an immunization strategy for the epidemic spreading. For the strategy, we use a random immunization of individuals in one layer of the network. Those immunized overlapped individuals will remain immunized in all layers of the network.

2. Epidemic propagation process

In our model we use an overlapping multiplex network formed by two layers, A and B, of the same size N, where an overlapping fraction q of *shared* individuals is active in both layers. Figure 1(a) shows schematically the partially overlapped network. The dashed lines that represent the fraction q of shared individuals should not to be interpreted as interacting or interdependent links but as the shared nodes and their counterpart in the other layer.

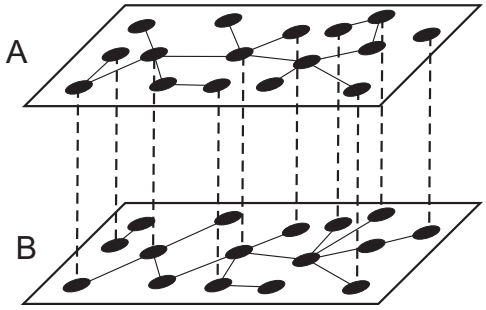

Figure 1. Partially overlapped multiplex network with layer size $N = 16$ and fraction of shared nodes $q = 0.625$. The total size of the network is $(2 - q)N = 22$ individuals. The dashed lines are used as a guide to show the fraction q of shared nodes. Before the spreading dynamics, all individuals are in the susceptible stage represented by black circles.

For the simulation, we construct each layer using the Molloy Reed algorithm [22], and we

choose randomly a fraction q of nodes in each of the layers that represent the same nodes. In our model we assume that the transmissibility is the same in both layers because there is only one disease and all individuals in the system spread with the same probability. We begin by infecting a randomly chosen individual in layer A. The spreading process then follows the SIR dynamics in both layers, the overlapped nodes in both layers have the same state because they represent the same individuals. After all infected nodes infect their susceptible neighbors with probability β in both layers, the time is increased in one, and the states of the nodes are updated simultaneously. Note that because there are shared nodes the branches of infection can cross between the two layers. Thus the probability that, following a random link, a node belonging to the infected branch will be reached in each layer can be written as,

$$f_A = (1-q)\,[1 - G_1^A(1-Tf_A)] + q\,[1 - G_1^A(1-Tf_A)\,G_0^B(1-Tf_B)]\,, \tag{1}$$
$$f_B = (1-q)\,[1 - G_1^B(1-Tf_B)] + q\,[1 - G_1^B(1-Tf_B)\,G_0^A(1-Tf_A)]\,, \tag{2}$$

where $G_0^i(x) = \sum_{k=k_{\min}}^{k_{\max}} P_i(k)x^k$ is the generating function of the degree distribution and $G_1^i = \sum_{k=k_{\min}}^{k_{\max}} P_i(k)\; kx^{k-1}$ is the generating function of the excess degree distribution in layer i, with $i = A, B$ [21].

Equation (1) has two terms, since the probability f_A to expand an infected branch following a random chosen link in layer A, can be written as the probability to reach one of the $(1-q)$ non-overlapped individuals and that the branch of infection expands through the $k-1$ remaining connections of the individual in layer A, combined with the probability of reaching one of the q overlapped individuals and that the branch of infection expands through the $k-1$ remaining connections of the individual in layer A and through the k connections of the individual in layer B. An analogous interpretation holds for the equation (2).

The solution of the system of equations (1) and (2) for all T above and at criticality is given by the intersection of the curves f_A and f_B. At criticality, this intersection can be derived by solving the determinant equation $|J - I| = 0$, where I is the identity and J is the Jacobian matrix of the system of equations (1) and (2). The only possibility to have a non-epidemic regime is that none of the branches of infection spread, *i.e.* $f_A = f_B = 0$, therefore below and at criticality $f_A = f_B = 0$. The evaluation of the Jacobian matrix $J_{ij} = (\partial f_i/\partial f_j)|_{f_A=f_B=0}$ allow us to obtain a quadratic equation for T_c with only one stable solution [23] given by,

$$T_c = \frac{[(\kappa_A - 1) + (\kappa_B - 1)] - \sqrt{[(\kappa_A - 1) - (\kappa_B - 1)]^2 + 4q^2\langle k_A\rangle\langle k_B\rangle}}{2(\kappa_A - 1)(\kappa_B - 1) - 2q^2\langle k_A\rangle\langle k_B\rangle}, \tag{3}$$

where $\kappa = 1 + 1/T_c$ is the total branching factor of the system and κ_A, κ_B are the isolated branching factors of layer A and B respectively. For $q \to 0$ we recover the isolated network result $T_c = 1/(\kappa_A - 1)$, which is compatible with our model in which the infection starts in layer A and the disease never reaches layer B. In contrast, when $q \to 1$, we find that $T_c = 1/\sqrt{[(\kappa_A - \kappa_B)]^2 + 4\langle k_A\rangle\langle k_B\rangle}$. Note that $T_c(q \to 1) < T_c(q \to 0)$. In general, T_c decreases with q. This is the case because an increase in the overlapping between layers increases the total branching factor, and therefore the total system becomes more heterogeneous in degree, *i.e.*, the total branching factor is equal to or bigger than the branching factor of the isolated layers.

3. Immunization strategy

We study a random immunization strategy on the partially overlapped multiplex network. We start by immunizing a random fraction m of individuals in layer A, before the epidemic spreading take place. An immunized individual will be immune to the disease in all layers, and therefore can not be infected or infect during all the propagation process. Note that, due to the presence

of the overlapped individuals, in layer B there will be a random fraction mq of immunized individuals.

After immunizing, we spread a disease in the network, starting by infecting a random susceptible non-immunized individual in layer A (patient zero). Thus, the probability that reaching a node by following a randomly chosen link, it belongs to a branch of infection is given by the system of equations (1) and (2), using a node diluted degree distribution in each layer [24] due to the immunization strategy. Thus with the diluted degree distribution we have that the branching factor of the diluted layers are,

$$\widetilde{\kappa_A} = (1-m)\,\kappa_A \quad (4)$$
$$\widetilde{\kappa_B} = (1-qm)\,\kappa_B\ , \quad (5)$$

where κ_A and κ_B are the branching factor of the original layers respectively. Note that the branching factor is reduced due to the immunization strategy increasing the epidemic threshold and thus hindering the diseases propagation.

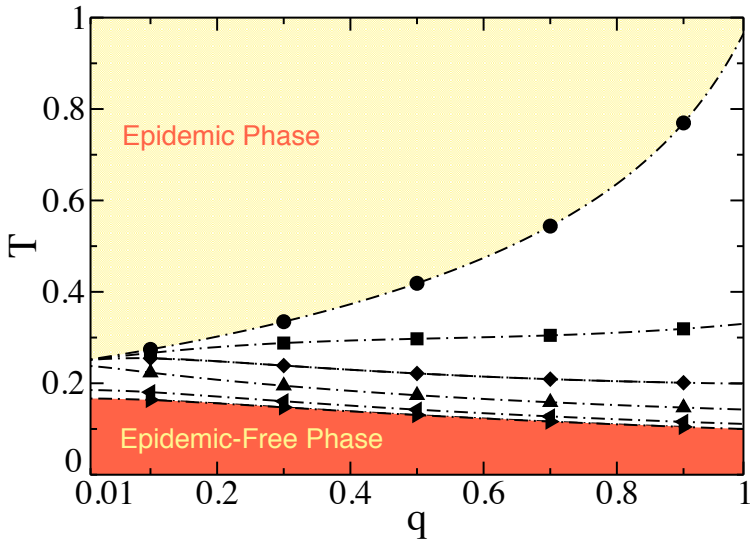

Figure 2. Phase diagram in the plane $T-q$ for the SIR model in the multiplex network, when the random immunization strategy is applied, for different values of the immunized fraction m. Both, layer A and B, have Erdős Rényi degree distributions with mean values of connectivity $\langle k_A\rangle = 6$ and $\langle k_B\rangle = 4$ for layer A and B respectively. Symbols corresponds to the value of T_c for different values of m obtained by numerical simulation with layer size $N = 10^5$, while the lines denote the theoretical results obtained numerically from Eqs. (1) and (2) using $\widetilde{\kappa_A}$ and $\widetilde{\kappa_B}$ given by Eqs. (4) and (5). From top to bottom $m = 0.9; 0.7; 0.5; 0.3; 0.1; 0$. Above the lines the system is in the epidemic phase for each value of m, and below it is in the epidemic-free phase where the disease can not propagate. All simulations were done over 10^5 network realizations.

In Figure 2 we show the phase diagram in the plane $T-q$ for different values of the immunization fraction m. We consider that both layers have Erdős Rényi degree distributions with mean values of connectivity $\langle k_A\rangle = 6$ and $\langle k_B\rangle = 4$ for layer A and B respectively, and we use $k_{min} = 1$ and $k_{max} = 40$ as the minimum and maximum connectivity respectively in each

layer. The lines represent T_c for many values of m obtained theoretically from Eqs. (1) and (2) while symbols denote the numerical simulation results. Above T_c there is an epidemic phase and below T_c only outbreaks exists (non-epidemic phase). Fig. 2 shows that T_c has different behaviors with q depending on the value of m. From Figure 2 we can see a good agreement between the theoretical predictions and the numerical simulation results.

Note that when $q = 0$ (not shown) the critical threshold corresponds to an isolated layer in which the disease starts, *i.e.* layer A and where the critical threshold is given by $T_c = 1/(\kappa_A - 1)$. For $q \to 0$ ($q = 0.01$) the epidemic threshold converges to the threshold of the layer with the bigger branching factor, since in this limit the process is dominated by the most heterogeneous layer [16]. We can observe from Fig. 2 that as the immunization fraction increases, the epidemic-free phase widens. When $m < 0.7$ (see Fig. 2) T_c decreases with q owing to the fact that as the overlapping between the layers increases the total branching factor of the network increases. However, for $m \geq 0.7$ T_c increases as q increases. This last effect can be understood taking into account that for $m > 0.7$ layer A is very diluted, thus the disease spreads mostly through layer B, as q increases the immunization fraction mq of layer B increases, hindering the propagation through that layer. It is expected that for more heterogeneous networks this strategy has less impact in the spreading process, due to the fact that the more heterogeneous the network is, the more harder it is to dilute with this strategy.

4. Discussion

In this work we study, theoretically and via simulations, an epidemic spreading and a random immunization strategy in a partially overlapped multiplex network composed by two layer with an overlapping fraction q. We immunize a fraction m of individuals in one layer of the network and study how this process affects the propagation of the disease through all layers. We found that for $q \to 0$ the critical threshold of the epidemic is dominated by the threshold of the most heterogeneous layer for all $m > 0$. We found that there is a regime in which T_c decreases with q due to the fact that the total branching factor of the system increases. This behavior stands for $m < 0.7$, however for bigger values of m, T_c increases as q increases, hindering the disease propagation. This last effect can be understood taking into account that when $m > 0.7$, layer A is diluted, and as q increases the immunization fraction mq of layer B increases, and the effect of the immunized individuals in that layer is stronger.

We can observe from Fig. 2 that as the immunization fraction increases, the epidemic-free phase widens. When $m < 0.7$ we can see that T_c decreases with q owing to the fact that as the overlapping between the layers increases the total branching factor of the network increases. However, for $m \geq 0.7$, T_c increases as q increases, hindering the disease propagation. This last effect can be understood taking into account that as q increases the immunization fraction mq of layer B increases and for $m > 0.7$ the effect of the immunized individuals in that layer is stronger. Our study suggests that vaccinating or isolating only in one layer with the higher propagation capacity, can reduce drastically the total branching factor of the network. As a consequence, the epidemic threshold of the system increases significantly, reducing the risk of a disease epidemic in the system.

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