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Immunization strategy for epidemic spreading on multilayer networks

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Abstract – In many real-world complex systems, individuals have many kinds of interactions among them, suggesting that it is necessary to consider a layered-structure framework to model systems such as social interactions. This structure can be captured by multilayer networks and can have major effects on the spreading of process that occurs over them, such as epidemics. In this letter we study a targeted immunization strategy for epidemic spreading over a multilayer network. We apply the strategy in one of the layers and study its effect in all layers of the network disregarding degree-degree correlation among layers. We found that the targeted strategy is not as efficient as in isolated networks, due to the fact that in order to stop the spreading of the disease it is necessary to immunize more than 80% of the individuals. However, the size of the epidemic is drastically reduced in the layer where the immunization strategy is applied compared to the case with no mitigation strategy. Thus, the immunization strategy has a major effect on the layer were it is applied, but does not efficiently protect the individuals of other layers.

Introduction. – The new insights in the complex-networks analysis, are no further considering networks as isolated entities, but characterizing how networks interact with other networks and how this interaction affects processes that occur on top of them. A system composed by many networks is called *Network of Networks* (NoN), a terminology introduced a few years ago [1–4]. In NoN, there are connectivity links within each individual network, and external links that connect each network to other networks in the system. A particular class of Network of Networks in which the nodes have multiple types of links across different *layers* [5–11], are called *Multiplex or Multilayer Networks* [12]. The multiplex network approach has proven to be a successful tool in modeling a number of very wide real-world systems, such as the Indian air and train transportation networks [13] and the International Trade Network [14,15].

In the last couple of years, the study of the effect of multiplexity of networks in propagation processes such as epidemics has been the focus of many recent researches [12,16–20]. In ref. [21] the research concentrated on the propagation of a disease in partially overlapped multilayer networks, because the fact that individuals are not necessarily present in all the layers of a society impacts the propagation of the epidemic. For the epidemic model they used the susceptible-infected-recovered (SIR) model [22–24] that describes the propagation of non-recurrent diseases for which ill 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 their susceptible neighbors with a probability β and recover after a fixed time t_r. The spreading process stops when all the infected individuals are recovered. The dynamic of the epidemic is controlled by the transmissibility T, that is the effective probability that the disease will be transmitted across any given contact. As in the SIR model an individual cannot be reinfected, the disease spreads through branches of infection that have a local tree-like structure, and thus, this model, can be described using the branching theory approach within a generating function formalism [25,26] that holds in the thermodynamic limit. In [21], they found, theoretically and via

simulations, that in the partially overlapped multiplex network, the epidemic threshold decreases as the overlapped fraction between layers increases, due to the fact that, when the overlapping between layers increases, the number of paths the disease can take increases. They also found 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.

In a real context, the immunization strategy in social networks is not made at random. It is a well-known fact that the bigger spreaders in social networks are those individuals with higher degrees. Some of the mitigation strategies used in society nowadays are based on these phenomena, for example, it is mandatory for all hospital staff to get the vaccine against flu every year, since they are (in average) the most connected and exposed individuals in the population. This suggests that health agencies always try to immunize those individuals that have, somehow, more chances to get infected and to propagate the disease. Motivated by this, in this letter we study a strategy in overlapped multiplex networks where the most connected individuals in one layer are identified and vaccinated, which is called targeted immunization strategy. Those immunized overlapped individuals will remain immunized in all layers of the network.

Model and results. –

Immunization strategy. In our model we use as the substrate for the epidemic spreading a multiplex network formed by two layers, called A and B, of the same size N, and with degree distribution $P_A(k)$ and $P_B(k)$ which are the probability that a random chosen node in layer A and B, respectively, has degree k. An overlapping fraction q of *shared individuals* is active in both layers.

For the targeted immunization strategy, we start by immunizing a fraction p of the highest connected individuals in layer A, and as we assume no degree correlation between layers, the immunization in layer B will be at random. Immunized individuals cannot be infected by the disease and will remain in the susceptible state during all the propagation process.

Let $\psi(k)$ be the probability that a node is not immunized given that it has degree k, then $P_A(k)\psi(k)$ is the probability of a node in layer A to have degree k and not being immunized, and

$$F_0^A(x) = \sum_{k=k_{min}}^{k_{max}} P_A(k)\psi(k)x^k \tag{1}$$

is the probability generating function for this distribution [25] and k_{min} and k_{max} are the minimum and maximum values of the degrees. Note that $F_0^A(1) = 1 - p$, where $1 - p$ is the fraction of the non-immunized individuals in layer A.

If we follow a randomly chosen link in layer A, the node we reach has degree distribution proportional to $kP_A(k)$, rather than just $P_A(k)$, because a randomly chosen link is more likely to lead to a node with higher degree. Hence the equivalent of eq. (1) for such a node is [25]

$$F_1^A(x) = \frac{\sum_k kP_A(k)\psi(k)x^{k-1}}{\sum_k kP_A(k)} = \frac{F_0^{A\prime}(x)}{\langle k_A\rangle}, \tag{2}$$

where $\langle k_A\rangle$ is the average node degree in layer A, and $F_0^{A\prime}(x) = \mathrm{d}F_0^A(x)/\mathrm{d}x$.

We need to define the function $\psi(k)$ that will depend on the immunization strategy used. For the targeted immunization, in layer A we immunize a fraction p of the higher degree nodes, thus, there will be a degree cutoff k_s in that layer such that all individuals with degree higher than k_s, and a fraction w of individuals with degree k_s in layer A are immunized. Therefore, for this strategy, $\psi(k)$ is

$$\psi(k) = \begin{cases} 0, & \text{if } k > k_s, \\ 1, & \text{if } k < k_s, \\ w, & \text{if } k = k_s. \end{cases} \tag{3}$$

The total fraction of immunized individuals p can be written as

$$p = wP_A(k_s) + \sum_{k=k_s+1}^{k_{max}} P_A(k), \tag{4}$$

using the normalization property of the degree distribution $\sum_{k=0}^{k_{max}} P_A(k) = \sum_{k=0}^{k_s} P_A(k) + \sum_{k=k_s+1}^{k_{max}} P_A(k) = 1$, we can write w as

$$w = \frac{p - 1 + \sum_{k=0}^{k_{max}} P_A(k)}{P_A(k_s)}. \tag{5}$$

In layer B, there is not a direct immunization strategy, however, the overlapped individuals that were immunized in layer A, will be also immunized in layer B but at random. Thus, there is a fraction pq of random immunized individuals in layer B.

Propagation process over the immunized multiplex network. After the immunization strategy takes place, we start the propagation process by infecting one randomly chosen susceptible (non-immunized) individual in layer A. The spreading process then follows the SIR dynamics in both layers, and the disease spreads through branches of infection. We assume that the transmissibility is the same in both layers and thus all individuals in the system spread equally. The overlapped nodes in both layers have the same state because they represent the same individuals.

One parameter that contains all the information about the branching process is the probability Q_i, that choosing a random selected link, it does not leads to the infinite branch of infected individuals in layer i, with $i = A, B$. The probabilities Q_A and Q_B satisfies the following self-consistent equations:

$$\begin{aligned} Q_A &= 1 - F_1^A(1) + (1-q)\, F_1^A(1 - T + TQ_A) \\ &\quad + q\, F_1^A(1 - T + TQ_A)\, G_0^B(1 - T + TQ_B), \end{aligned} \tag{6}$$

$$\begin{aligned} Q_B &= pq + (1-q)\, G_1^B(1 - T + TQ_B) \\ &\quad + q\, G_1^B(1 - T + TQ_B)\, F_0^A(1 - T + TQ_A), \end{aligned} \tag{7}$$

$$T_c = \frac{F_1^{A\prime}(1) + (\kappa_B - 1)(1 - pq) - \sqrt{(F_1^{A\prime}(1) - (\kappa_B - 1)(1 - pq))^2 + 4q^2 F_1^A(1)^2 \langle k_A \rangle \langle k_B \rangle}}{2F_1^{A\prime}(1)(\kappa_B - 1)(1 - pq) - 2q^2 F_1^A(1)^2 \langle k_A \rangle \langle k_B \rangle}, \tag{8}$$

where $G_0^B(x) = \sum_{k=k_{min}}^{k_{max}} P_B(k) x^k$ is the generating function of the probability to reach a node with degree k in layer B and $G_1^B(x) = \sum_{k=k_{min}}^{k_{max}} \frac{kP_B(k)}{\langle k_B \rangle} x^{k-1}$ is the generating function for the probability to reach a node of degree k in layer B by following a random chosen link.

Equation (6) has three terms, since the probability Q_A to not reach the infected branches following a random chosen link in layer A, can be written as the probability that an immunized individual is reached $(1 - F_1^A(1))$, plus the conditional probability that the reached individual does not have spread the disease given that it is not immunized. This last conditional probability is split into two terms, depending on whether the reached individual is one of the q overlapped fraction or not. If the individual is only present in one layer with probability $1 - q$, the branch will never reach layer B while if the individual is present in both layers with probability q, the branch will reach a node in layer B and can expand through the k connections of the reached node in that layer. An analogous interpretation can be made for eq. (7).

The solution of the system (6) and (7) above is given by the intersection of Q_A and Q_B. In the criticality, this intersection can be derived by solving the equation $|J - I| = 0$, where $|\,|$ denotes the determinant, I is the identity and J is the Jacobian matrix of the system of equations (6) and (7), whose elements are $J_{ij} = \partial Q_i / \partial Q_j$, with $i = A, B$ and $j = A, B$. The Jacobian has to be evaluated in $Q_A = Q_B = 1$, since at criticality the disease does not spread and there are no branches of infection. There are two different eigenvalues for each one of the possible solutions of the system. The stability of each solution can be analyzed by the behavior of the eigenvalues, *i.e.* sink, source or saddle [27]. We find that only one of the possible solutions is stable and, therefore, the epidemic threshold is given by $T_c(q) \equiv T_c$:

see eq. (8) *above*

where $F_1^{A\prime}(1) = \mathrm{d}F_1^A(x)/\mathrm{d}x|_{x=1}$ and $T_c = 1/(\kappa - 1)$, where κ is the total branching factor of the multilayer networks.

In fig. 1 we plot the plane T-q obtained from eq. (8), for different values of p. We use a power law degree distribution $P_{A/B} \sim k^{-\gamma_{A/B}}$ in both layers with exponents $\gamma_A = 2.5$ and $\gamma_B = 3.5$ in layer A and B, respectively, where $k_{min} = 2$ and $k_{max} = 250$ are the minimum and maximum connectivity. Note that layer A in which the immunization is applied is the most heterogeneous layer, however similar results are found using different degree distributions on each layer. The lines represent T_c for many values of p, above the lines there is an epidemic phase and below T_c only outbreaks exists (non-epidemic phase). Figure 1 shows that T_c has different behaviors with q depending on the value of p.

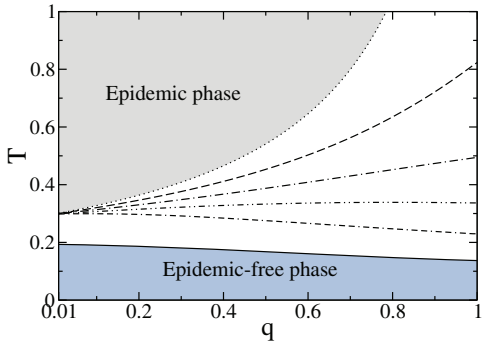

Fig. 1: (Color online) Plane T-q for the SIR model in the multiplex network, when the targeted immunization strategy is applied, for different values of the immunized fraction p. Both layers A and B have power law degree distributions $P_{A/B} \sim k^{-\gamma_{A/B}}$ with $\gamma_A = 2.5$ and $\gamma_B = 3.5$ with $k_{min} = 2$ and $k_{max} = 250$. The lines denote the theoretical values of T_c for different values of q obtained numerically from eqs. (6) and (7). From top to bottom $p = 0.9; 0.7; 0.5; 0.3; 0.1; 0.01$. Above the lines the system is in the epidemic phase for each value of p, and below it is in the epidemic-free phase where the disease dies out.

For $q = 0$ (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 $T_c = 1/F_1^{A\prime}(1)$, where $F_1^{A\prime}$ is not the branching factor of layer A, but gives a measure of the heterogeneity of the layer. For $q \to 0$ the process is dominated by the most heterogeneous layer [21], therefore, the epidemic threshold converges to the threshold of that layer. In fig. 1 we can see that for $p = 0.01$ and $q \to 0$, $T_c = 1/F_1^{A\prime}(1)$, due to the fact that the most heterogeneous layer is A, while for $p \geq 0.1$, $T_c = 1/G_1^{B\prime}(1) = 1/\kappa_B - 1$ were κ_B is the branching factor of layer B.

From the phase diagram (see fig. 1) we can see that for $p < 0.2$, T_c decreases with q, this behavior agrees with the expected non-immunized behavior, since as q increases, the total branching factor of the network increases and thus T_c decreases [21]. For $p > 0.2$, T_c increases with q, due to the fact that layer A gets fragmented and the disease spreads through layer B, and as q increases, the fraction pq of the random immunized individuals in layer B increases hindering the spreading through layer B and thus, T_c increases with q. When the fraction of immunized individuals $pq > 0.72$, layer B also gets fragmented, and the disease cannot spread at all, thus, the epidemic regime disappears as shown in fig. 1 for $p = 0.9$ and $q \gtrsim 0.79$. We can understand this behavior using percolation theory, for the targeted percolation process. For this process it was found that [28] the critical value of the percolation fraction $\hat{p_c}$ in scale-free networks with exponent $\gamma = 2.5$ is

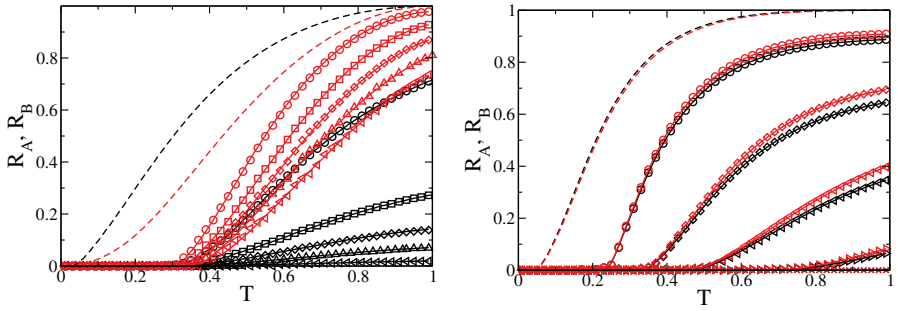

Fig. 2: (Color online) Fraction of recovered individuals in the final state of the epidemics for layer A, R_A (black), and layer B, R_B (red), as a function of T. Both layers have power law degree distributions $P_{A/B} \sim k^{-\gamma_{A/B}}$ with $\gamma_A = 2.5$ and $\gamma_B = 3.5$ for layer A and B, respectively. Lines denote the theoretical results obtained from eqs. (9) and (10) while symbols denote numerical simulation results for layer size $N = 10^5$ and over 10^5 network realization. (a) $q = 0.2$ the dashed lines correspond to the case without immunization strategy $p = 0$, and $p = 0.1; 0.3; 0.5; 0.7; 0.9$ from top to bottom; (b) $q = 0.9$, the dashed lines correspond to the case without immunization strategy $p = 0$, and the full lines and symbols corresponds to $p = 0.1; 0.3; 0.5; 0.7; 0.9$ from top to bottom.

$\hat{p_c} \approx 0.2$ such that for $p > \hat{p_c}$ the networks is fragmented and for $p < \hat{p_c}$ there is a giant connected cluster, that is also the critical threshold corresponding to the targeted immunization strategy in layer A. In layer B, there is a random immunization equivalent to a random percolation process, for which the critical value of percolation fraction $\hat{p_c}$ in scale-free networks with exponent $\gamma = 3.5$ is $\hat{p_c} \approx 0.72$ [28] and corresponds to the critical threshold due to the random immunization strategy in layer B. Despite that, the targeted immunization strategy is the best strategy to stop propagation in isolated networks, in overlapped multiplex networks it is not as efficient due to the fact that the threshold is dominated by the most heterogeneous network. From the phase diagram we can observe that in order to suppress the epidemic phase one has to immunize more than 80% of the population in layer A. Thus even if network A is fragmented ($p > 0.2$) the disease can still propagate in network B which is more heterogeneous than the fragmented layer A. Notice that layer B is not fragmented for $pq < 0.72$.

However, even if it is hard to stop the epidemic, its size can be drastically reduced compared to the case where no strategy is applied. The size of the epidemic can be computed as the total number of recovered individuals in the final state of the epidemic, and is given by

$$R_A = q\left[1-p-F_0^A(1-T+T\,Q_A^*)G_0^B(1-T+T\,Q_B^*)\right] + (1-q)\left[1-p-F_0^A(1-T+T\,Q_A^*)\right], \quad (9)$$

$$R_B = q\left[1-p-F_0^A(1-T+T\,Q_A^*)G_0^B(1-T+T\,Q_B^*)\right] + (1-q)\left[1-G_0^B(1-T+T\,Q_B^*)\right], \quad (10)$$

where Q_A^* and Q_B^* are the non-trivial solutions of eqs. (6) and (7) for $T \gtrsim T_c$.

In figs. 2(a) and (b) we plot the results of R_A and R_B as a function of T, obtained both theoretically from eqs. (9) and (10) and from the numerical simulation. We found a good agreement between the theoretical results (lines) and the numerical simulations (symbols). In fig. 2(a) we show the results for $q = 0.2$ and different values of p. If we compare the results with and without strategy (dashed line) we can see that the immunization strategy not only affects the epidemic threshold, but also decreases the impact of the disease in both layers, since at fixed T, both R_A and R_B decreases with p. For $p \geq 0.2$ layer A gets fragmented and the disease never reaches more than 30% of the individuals in that layer, however the impact of the disease in layer B is significant and even for $p = 0.9$, more than 60% of the individuals in layer B can be infected. Therefore, for an overlapping fraction between layers $q = 0.2$, the immunization strategy has a major effect on the layer were it is applied, but does not protect the individuals of other layers.

In fig. 2(b), we show R_A and R_B for $q = 0.9$, and we can see that even though one needs to immunize more than 80% of the individuals to suppress the epidemic phase, the impact of the disease in both layers decreases significantly with p. We compare R_A and R_B with and without strategy (dashed line) and see that, in this regime, due to the high overlapping between layers, the effect of the immunization strategy on layer B is stronger, making the propagation through that layer difficult. Therefore when the overlapping between layers is high, the strategy is more efficient to protect the individuals of the whole network.

Conclusions. – In this letter we study, theoretically and via simulations, a targeted immunization strategy for epidemic spreading in a partially overlapped multiplex network composed by two layers with an overlapping fraction q. We immunize a fraction p 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 the branching theory gives a good approach of the phenomena. For $q \to 0$ the critical threshold of the epidemic is dominated by the threshold of the most heterogeneous layer for all p. When p is smaller than the critical percolation threshold of layer A, T_c decreases with

q, as in the non-immunized model presented in [21]. When p is above the criticality of layer A, this layer gets fragmented and thus the epidemic can only spread through layer B. The fraction of immunized individuals in layer B is pq, thus as q increases T_c increases. This behavior holds until the fraction pq exceeds the critical percolation threshold of layer B. Above this threshold layer B also gets fragmented and thus, the disease cannot spread at all, suppressing the epidemic phase. This regime can only be reached if one immunizes more than 80% of the individuals. However, even if it is hard to stop the epidemic, its size can be drastically reduced compared to the case where no strategy is applied. We found that the immunization strategy has a major effect on the layer were it is applied, but does not efficiently protect the individuals of other layers.

Real networks of networks such as the worldwide port network and a worldwide airport network [29] have assortative degree-degree correlation between networks, *i.e.* biggest airports are connected with bigger ports. In order to have a realistic scenario we should consider degree-degree correlation between layers. If the correlation is assortative, then the immunization strategy in layer B will be also targeted and thus the strategy would be more effective since layer B will get fragmented easier. In a future work, we will study deeply the effects of correlations between layers in the epidemic spreading and immunization strategies in multilayer networks.

After this letter was submitted a similar strategy was published by Zhao *et al.* in [30].

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