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Preventive behavioural responses and information dissemination in network epidemic models

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Abstract— Human behavioural responses have an important impact on the spread of epidemics. To deal with them, some epidemic models consider that individuals are aware of the risk of contagion and adopt preventive responses when they learn about the existence of the disease. If awareness is assumed to be transmitted from individual to individual, the information dissemination can be thought to spread over a second network where links are defined according to a certain type social relationship (friends, acquaintances, etc.), with the same set of nodes as the contact network. Here we present a simple model for epidemic spreading with awareness defined on a two-layer network which includes the overlap between these two layers as a parameter. This formulation leads to an expression of the epidemic threshold as a function of the network overlap.

Keywords: Epidemic modelling, multilayer networks.

1 Introduction

Human behavioural responses have an important impact on the spread of epidemics. One way to include them into epidemic models is to consider individuals who are aware of the risk of contagion and adopt preventive responses when they get informed about the existence of the disease. Some models include a new class of individuals, the so-called aware or alerted ones, and derive the corresponding equations for the transmission of the disease and recovery (see, for instance, [7] and references therein). More sophisticated models consider specific features of the information transmission process. For example, in contrast to disease transmission, the quality of information passed on to other people decreases at each transmission event. This leads to more complex models with several classes of aware individuals (see [3]). On the other hand, the routes of information transmission do not need to be the same as those for the spread of a disease. In this case, information dissemination is modelled by means of a second network, with the same set of nodes as the contact network but with a distinct set of links, over which information spreads.

In a more general context, the simultaneous spread of awareness and infectious diseases is an example of interacting spreading processes taking place on multilayer networks. Other examples are competitive viruses propagating in a host population where each virus has different routes of transmission, i.e., a distinct network for propagation (see [4, 11]). In such an instance, the interaction among virus species is determined by the type of competition existing between them (exclusive, reinforcing, weakeing, etc.) when they coincide in the same host. A challenging issue related to these processes is to elucidate the effect of the cross-layer interrelation on the dynamics of simultaneous propagation of contagious agents. Some analytical results relating different features of the adjacency matrices of a two-layer network have been recently obtained in [11].

Following the studies of the impact of network overlap on the coexistence of competing viral agents in [3, 4, 9], we derive a simple mean-field epidemic model defined on a two-layer network where the overlap between the two layers appears as a parameter of the model equations. This fact allows to express the basic reproduction number R_0 as a function of the overlap of the two networks and, hence, to derive a simple analytical expression of the epidemic threshold which involves the network overlap. Certainly, the overlap between two layers offers an incomplete description of the cross-layer interrrelation but, in addition to degree-degree correlations between layers, is a basic statistical descriptor of its topology. Finally, model's predictions are tested against the output of stochastic simulations of epidemic spreading carried out in continuous time on partially overlapped networks generated using the so-called network configuration model and a cross-rewiring algorithm.

2 An SIS epidemic model defined on a two-layer network

As usual in epidemic modelling, we describe the spread of infectious diseases on populations by classifying their individu-

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als into two classes or compartments. Here we consider the class of susceptible (S) individuals and the class of infectious (I) ones. The routes of transmission of some infectious diseases, in particular those infections that are sexually transmitted, reveal that a suitable description of populations must take into account the network A of physical contacts among individuals, with nodes representing individuals and links corresponding to physical contacts along which disease can propagate. On the other hand, it seems natural to assume that the probability of getting infected through an infectious contact S-I depends on the awareness state of the susceptible individual. In such a case, a second network B over which information about the infection state of individuals circulates can be considered. This dissemination network has the same set of nodes as the one of physical contacts but, in contrast, it has a different set of links representing, for instance, relationships with friends and acquaintances. So, if two individuals, one susceptible and the other infectious, are connected to each other on both networks, we assume that the transmission rate β_c of the disease though a physical contact between them will be smaller than the standard transmission rate β. The reason is that susceptible individuals have information on the health state of their infected partners and adopt preventive measures in order to diminish the risk of infection.

Under this scenario, we derive a mean-field susceptible-infectious-susceptible (SIS) epidemic model which implicitly assumes the spread of both information and an infectious agent over a two-layer network. Following the standard modelling approach of sexually transmitted diseases in which the variance in the number of contacts (sexual partners) is a basic ingredient [1], individuals are classified according to their infection state and their number of physical contacts. So, the model will take into account the network layer A of physical contacts in terms of its degree distribution $p_A(k) = N_k/N$ where N_k is the number of individuals having degree k. Analogously, the dissemination network (network layer B) is described by its degree distribution $p_B(k)$. A key assumption of the model will be the existence of a *uniform* (but not complete) overlap between the links of both layers, which means that the probability of finding two connected nodes in both networks does not depend on their degrees. A pair of such nodes is said to share a *common link*, although the connections are of different nature.

In each layer, no degree-degree correlation is assumed, i.e., neighbours in each layer are randomly sampled from the population according to the so-called proportionate mixing of individuals [2]. Therefore, in each layer, the probability $P(k'|k)$ that a node of degree k is connected to a node of degree k' is independent of the degree k and it is given by the fraction of links pointing to nodes of degree k', i.e., $P(k'|k) = k'p(k')/\langle k\rangle$ [2]. So, the expected degree of a node reached through a randomly chosen link, i.e., the expected degree of a neighbour in a population with proportionate mixing, is $\langle k^2\rangle/\langle k\rangle$. On the other hand, let I_k denote the number of infectious nodes of degree k in network layer A. Although the links are unordered pairs of connected nodes by definition, let us consider that every link $\{u, v\}$ gives rise to two *oriented links* $u \to v$ and $v \to u$. Then, the probability that a randomly chosen oriented link of A leads to an infectious node is given by the fraction of oriented links in A pointing to infectious nodes, that is,

$$\Theta_I = \frac{1}{\langle k_A\rangle N}\sum_k k\, I_k = \frac{1}{\langle k_A\rangle}\sum_k k\, i_k$$

where $\langle k_A\rangle$ is the average degree in A, and $i_k := I_k/N$ is the fraction of nodes that are both infectious and of degree k in A.

Now, let L_A, L_B, and $L_{A\cap B}$ be the number of links of A, B, and common links, respectively. Then the probability $p_{B|A}$ that a randomly chosen link of A, an A-link, connects two nodes that are also connected in B is $p_{B|A} = \frac{L_{A\cap B}}{L_A}$. Similarly, $p_{A|B} = \frac{L_{A\cap B}}{L_B}$ is the probability that a randomly chosen B-link is a common link to both networks. With all these quantities, the epidemic spreading is described in terms of the following system of differential equations for the number of infectious nodes of degree k in layer A:

$$\frac{dI_k}{dt} = k(1-p_{B|A})\beta\, S_k\, \Theta_I + k\, p_{B|A}\, \beta_c S_k\, \Theta_I - \mu I_k \tag{1}$$

where $S_k = N_k - I_k$ is the number of susceptible nodes of degree k in layer A, β is the transmission rate through a non-common infectious link, and β_c is the transmission rate through a common infectious link.

The first term in the rhs of equation (1) is the rate of creation of new infectious nodes of degree k in A by infections through links that only belong to layer A, whereas the second term is the rate of creation of new infectious nodes from transmissions through common links. The last term accounts for the recoveries of infectious nodes, which occur at a recovery rate μ. Here $\langle k_A\rangle p_{B|A}$ is the expected number of common oriented links. Therefore, since this number is the same regardless the network we use to compute it, the following consistency relationship must follow:

$$\langle k_A\rangle p_{B|A} = \langle k_B\rangle p_{A|B}. \tag{2}$$

Now let us express $p_{B|A}$ and $p_{A|B}$ in terms of the overlap α, which is defined as $\alpha := \frac{L_{A\cap B}}{L_{A\cup B}}$ where $L_{A\cup B}$ is the set of links of the union network $A \cup B$. It follows that $p_{B|A}$ can be expressed in terms of α as:

$$\begin{aligned} p_{B|A} &= \frac{L_{A\cap B}}{L_A} = \frac{L_{A\cap B}}{L_{A\cup B}}\,\frac{L_{A\cup B}}{L_A} \\ &= \alpha\,\frac{L_A + L_B - L_{A\cap B}}{L_A} \\ &= \alpha\left(1 + \frac{\langle k_B\rangle}{\langle k_A\rangle} - p_{B|A}\right). \end{aligned}$$

From this relationship it follows that

$$p_{B|A} = \left(1 + \frac{\langle k_B\rangle}{\langle k_A\rangle}\right)\frac{\alpha}{1+\alpha}. \tag{3}$$

and, also, that $p_{A|B} = \left(1 + \frac{\langle k_A \rangle}{\langle k_B \rangle}\right) \frac{\alpha}{1+\alpha}$. Note that, as expected, $p_{B|A}$ and $p_{A|B}$ fulfil relationship (2).

Uppn introducing (3) into Eq. (1), α appears as a new model parameter. In terms of the fraction i_k of nodes that are both infectious and of degree k, the model reads

$$\frac{di_k}{dt} = \frac{k}{1+\alpha}\left(\beta\left(1 - \frac{\langle k_B \rangle}{\langle k_A \rangle}\alpha\right) + \beta_c\left(1 + \frac{\langle k_B \rangle}{\langle k_A \rangle}\right)\alpha\right)(p_A(k) - i_k)\,\Theta_I - \mu i_k. \quad (4)$$

This equation corresponds to the standard SIS model for heterogeneous populations with proportionate mixing, but with an averaged transmission rate which depends on α.

Remarks:

1. If $\langle k_B \rangle > \langle k_A \rangle$, the non-negativity of the factor multiplying β is guaranteed because the overlapping α is bounded from above by

   $$\alpha = \frac{L_{A\cap B}}{L_{A\cup B}} \leq \frac{\langle k_A \rangle N}{\langle k_A \rangle N + \langle k_B \rangle N - \langle k_A \rangle N} = \frac{\langle k_A \rangle}{\langle k_B \rangle}$$

   because $L_{A\cap B} \leq L_A = \langle k_A \rangle N/2$. An upper bound for the maximum overlap coefficient is then given by $\min\{\langle k_A \rangle, \langle k_B \rangle\}/\max\{\langle k_A \rangle, \langle k_B \rangle\}$. Note that, since the factor $\alpha/(1+\alpha)$ in (3) is increasing in α, when $\langle k_A \rangle \leq \langle k_B \rangle$ it follows that $p_{B|A} \leq 1$ whereas, for $\langle k_A \rangle > \langle k_B \rangle$, we have $p_{B|A} \leq \langle k_B \rangle / \langle k_A \rangle < 1$. An improved upper bound for α is derived in [8].

2. If $\beta_c = \beta$ or $\alpha = 0$, the previous equation reduces to the classic SIS-model, as expected, because awareness plays no role in the infection spread. If $\alpha = 1$, we actually have one network and again Eq. (4) reduces to the SIS-model but now with β replaced with β_c.

In order to assess the impact of the network overlap on the initial epidemic growth, we linearise system (4) about the disease-free equilibrium $i_k^* = 0\,\forall k$ and obtain that the elements of the Jacobian matrix J^* evaluated at this equilibrium are

$$J^*_{kk'} = \frac{\beta_0(\alpha)}{\langle k_A \rangle} k k' p_A(k) - \mu \delta_{kk'}$$

where $\beta_0(\alpha) := \left(\beta\left(1 - \frac{\langle k_B \rangle}{\langle k_A \rangle}\alpha\right) + \beta_c\left(1 + \frac{\langle k_B \rangle}{\langle k_A \rangle}\right)\alpha\right)/(1+\alpha)$ and $\delta_{kk'}$ is the Kronecker delta. Since the eigenvalue of the matrix $(kk'p_A(k))$ with the largest real part is equal to $\langle k_A^2 \rangle = \sum_k k^2 p_A(k)$ (with an eigenvector whose components v_k are proportional to $kp_A(k)$), it follows that the dominant eigenvalue of J^* is

$$\lambda_1 = \frac{\langle k_A^2 \rangle}{\langle k_A \rangle}\beta_0(\alpha) - \mu,$$

which corresponds to the initial growth rate of the epidemic (cf. [1, 10] for $\alpha = 0$). From this expression we get that λ_1 decreases with α when $\beta_c < \beta$.

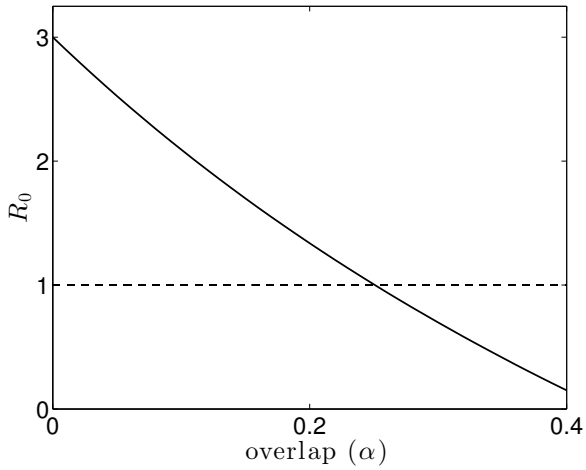

Figure 1: R_0 of the mean-field SIS model as a function of the overlap α between network layers. Parameters values: $\mu = 1$, $\beta = 0.1$, $\beta_c = 0.005$, $\langle k_A \rangle = 20$, $\langle k_A^2 \rangle = 600$, and $\langle k_B \rangle = 50$. For these mean degrees, $\alpha \in [0, 2/5]$.

We can also measure the initial epidemic growth in terms of the basic reproduction number R_0, i.e., the average number of secondary infections caused by a typical infectious individual at the beginning of an epidemic in a wholly susceptible population [2]. Interpreting $\beta_0(\alpha)$ as an averaged transmission rate weighted by the overlap coefficient α and recalling that $\langle k_A^2 \rangle / \langle k_A \rangle$ is the expected degree of a neighbour in a population with proportionate mixing, R_0 is given by

$$R_0 = \frac{\langle k_A^2 \rangle}{\langle k_A \rangle}\frac{\beta_0(\alpha)}{\mu} = \frac{\langle k_A^2 \rangle}{\langle k_A \rangle(1+\alpha)\,\mu}\left(\beta\left(1 - \frac{\langle k_B \rangle}{\langle k_A \rangle}\,\alpha\right) + \beta_c\left(1 + \frac{\langle k_B \rangle}{\langle k_A \rangle}\right)\alpha\right).$$

Therefore, as λ_1, R_0 is a decreasing function of the overlap coefficient between the two layers as long as $\beta_c < \beta$. This expression of R_0 is, indeed, a straightforward extension of the one obtained in [1] for heterogeneous populations and STDs.

Figure 1 shows this relationship when layer A has a degree distribution with mean degree $\langle k_A \rangle = 20$ and mean square degree $\langle k_A^2 \rangle = 600$. Note that there is no quantity related to the variance of the degree distribution of network B (as, for instance $\langle k_B^2 \rangle$) in the expression of R_0. This fact reflects the asymmetry of the roles of both networks in the disease propagation.

3 Stochastic simulations

We test the accuracy of the model by comparing the numerical integration of equations (4) with the output of stochastic simulations. It is worth noting that, in the derivation of (4), we have assumed that the edge overlap over the whole network layers is uniform. So, networks with dissimilar topological features will not satisfy such a hypothesis and, hence, they are not suitable to check the model.

Since we are interested in the overlap α as a critical parameter for the epidemic dynamics, we generate two-layer networks of a given size N by using the configuration model from two empirical degree sequences that are obtained from two distributions $p_A(k)$ and $p_B(k)$ with similar variances. Then, in order to have networks with a prescribed degree of overlap, we use an algorithm based on a selective cross-rewiring of two pairs of connected nodes. The cross-rewiring is done if it increases the overlap between layers (see [8] for details of the algorithm and properties of the resulting overlap). Finally, for several values of α, we perform continuous-time stochastic simulations on these networks based on the Gillespie's algorithm.

In simulations, the number of nodes is $N = 5000$, $p_A(k)$ is a power law ($p_A(k) = Ck^{-3}$), and $p_B(k)$ is a Poisson distribution of expected degree $\langle k_B \rangle = 25$ (Figure 2), or an exponential distribution of the same expected degree (Figure 3). In order to avoid the degree-degree correlations within a layer that appear when very high degrees are present in the degree sequence generated from a power law distribution, we normalize $p(k)$ to have a minimum degree $k_{\min}$ and a maximum degree given by the cut-off $k_c(N) = k_{\min} N^{1/2}$. This value is defined as the degree above which one expects to find at most one node in the network. This expression of $k_c(N)$ leads to the normalization constant $C = (\gamma - 1) k_{\min}^{\gamma - 1} N/(N-1)$ and an expected degree $\langle k \rangle = 2k_{\min} N/(N-1) \approx 2k_{\min}$.

The numerical integration of (4) is performed with an initial condition given by $i_k(0) = 0.1 p_A(k)\ \forall\, k$, which corresponds to uniformly infect 10% of nodes, the same initial fraction of infected nodes as the one used in stochastic simulations. Moreover, the integration is carried out by feeding the model with the empirical degree distribution obtained from the degree sequence of layer A. The reason for not using the theoretical distribution $p(k)$ is that, when the variance of $p(k)$ is large (i.e., in highly heterogeneous networks), there can be noticeable differences among distinct finite samples of $p(k)$. In particular, this is the case with respect to the values of the highest degrees in the generated degree sequences, which have a noticeable impact on the epidemic dynamics.

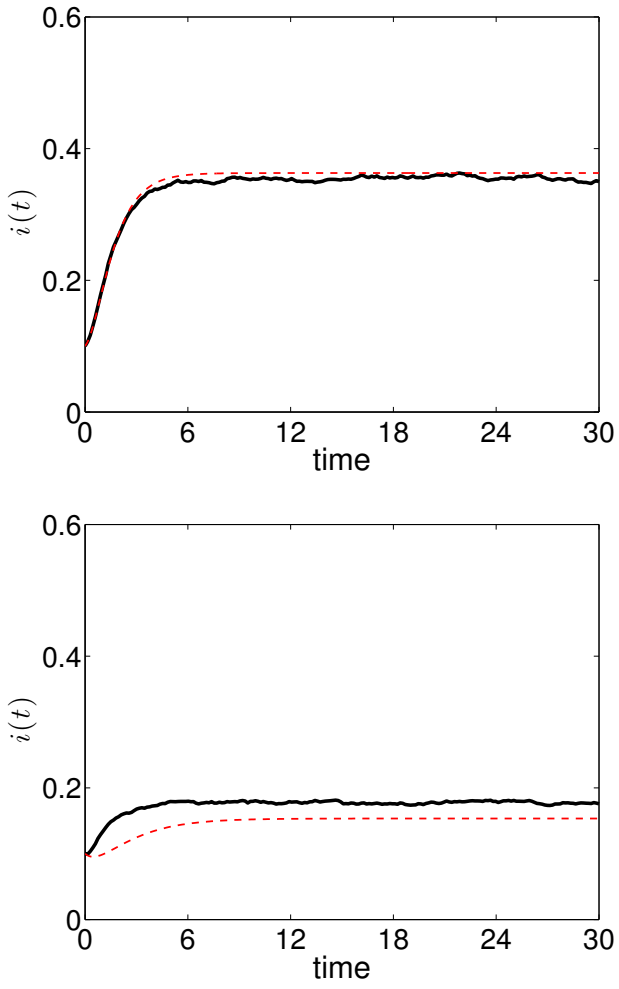

Figure 2: Fraction of infectious nodes averaged over 10 runs of stochastic simulations carried out on a two-layer network of size $N = 5000$ for $\alpha = 0.2$ (top), and 0.56 (bottom). In both panels, $p_A(k) \sim k^{-3}$ with $k_{\min} = 10$ ($\langle k_A \rangle = 20$), and layer B has a Poisson degree distribution of $\langle k_B \rangle = 25$. Dashed line shows the prevalence ($\sum_k i_k$) predicted by the SIS model. Initial fraction of infected nodes: 10%. Parameters: $\mu = 1$, $\beta = 0.1$, and $\beta_c = 0.03$.

4 Discussion

The derivation of the model is based on the hypothesis of a uniform distribution of the overlap over the set of nodes. This means that those nodes with high degrees in layer A have the same fraction of overlapped links than those with lower degrees. Clearly, this will not be the case if there is a large asymmetry between the degree distributions. One can observe the differences when layer A, the one over which physical contacts occur, has a power-law degree distribution whereas dissemination layer B has a Poisson degree distribution. In particular, when both degree distributions have similar mean degrees,

those nodes with the highest degrees in layer A only have a small fraction of overlapped links because of the low variance of the Poisson distribution. This amounts to an underestimation of the epidemic prevalence by the mean-field SIS model (4) because those nodes acting as a superspreaders in layer A have proportionally much less contacts with a low transmission rate (see Figure 2). In contrast, by increasing the variance of the degree distribution of layer B, disease transmission is reduced and the epidemic evolution is closer to the one predicted by the the mean-field model (see Figure 3).

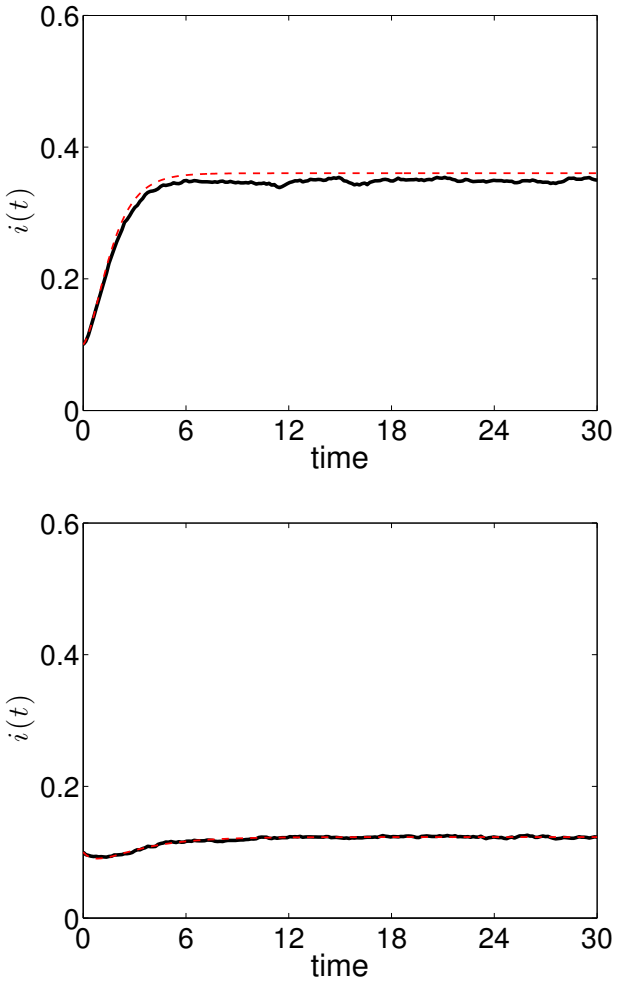

Figure 3: Fraction of infectious nodes averaged over 10 runs of stochastic simulations carried out on a two-layer network of size $N = 5000$ for $\alpha = 0.2$ (top), and 0.6 (bottom). In both panels, $p_A(k) \sim k^{-3}$ with $k_{\min} = 10$ ($\langle k_A \rangle = 20$), and layer B has an exponential degree distribution of $\langle k_B \rangle = 25$. Dashed line shows the prevalence ($\sum_k i_k$) predicted by the SIS model. Initial fraction of infected nodes: 10%. Parameters: $\mu = 1$, $\beta = 0.1$, and $\beta_c = 0.03$.

Stochastic simulations confirm that, when the mean-field assumptions are met, the proposed mean-field SIS model is suitable for modelling two interacting contagious processes like epidemic spreading and awareness dissemination. As it is usually the case for mean-field models, the analytical predictions of the SIS model is not accurate close to the epidemic threshold $R_0(\alpha) = 1$ (not shown here). However, when the overlap coefficient is not so close to its critical value, the predicted relationship between prevalence and network overlap shows a good agreement with stochastic simulations.

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