

Link equations for discrete-time epidemic processes in complex network

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Abstract

Epidemic spreading in complex networks has been intensely studied because of its practical implications. Predicting epidemic spreading is essential to perform containment campaigns. The vast majority of the literature has concentrate the efforts on determining the state of the nodes according to the epidemics, however the “states of the links” has received little attention. The state of links inform about the probability of spreading the disease from infected to susceptible individuals. These states depend on the full structure of the network, and its determination is not straightforward from the knowledge of nodes' states. In particular, we wonder about its computation in discrete-time epidemics, which is the approach used in basically all large-scale simulation of epidemics at the world-scale. Here, we confront this challenge and propose a set of discrete-time governing equations that can be closed and analyzed, assessing the contribution of links to spreading processes in complex networks. We validate our approach in synthetic and real networks, obtaining a better determination of critical thresholds. Moreover, our approach allows new schemes for the contention of epidemics based on link deactivation that outperform previous approaches.

The problem of modeling the spread of a disease among individuals has been studied in deep over many years. The development of compartmental models, models that divide the individuals among a set of possible states, has given rise to a new collection of techniques that enables, for instance, the

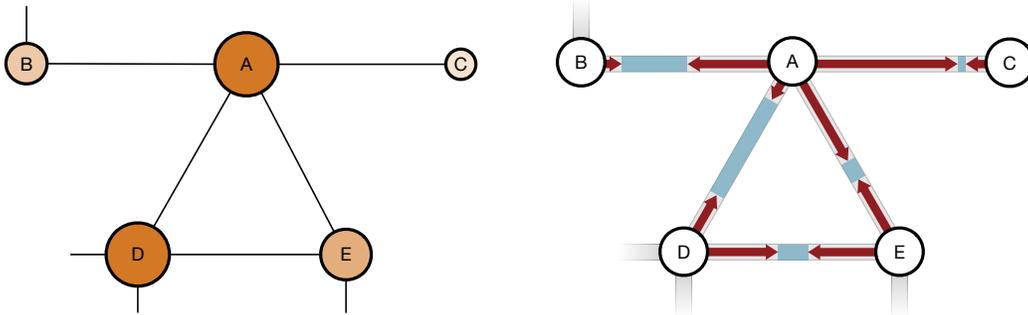


Figure 1: **Representation of spreading processes in networks.** Left, state of the epidemic spreading at node level, where the size and the color of the nodes is proportional to the probability of being in an infected state. Right, quantification of the role of the links in the dynamics: the length of the arrow represents the conductance (Φ) of the link, i.e. the probability that the link is in a state that enables the spreading of the disease, and the blue space between the arrows is proportional to the probability that a given link stays in a non-transmittable state ($\Theta^I + \Theta^S$). Both representations are complementary and help to understand the spreading processes in complex networks.

analysis of the epidemic threshold or the study of the impact of a prophylactic campaign. It is worth mentioning the Susceptible-Infected-Susceptible (SIS) model, in which individuals that are susceptible become infected and after a time they become susceptible again.

This article is based on the works by Pastor-Satorras et al. [20, 21], Newman [19, 18], Chakrabarti et al. [5], Moreno et al. [17], Hethcote [15], Anderson et al. [1], Daley et al. [6], Stanley [22], Dorogotsev et al. [8], Barabási [2], Mata et al. [16], Gleeson [9], Cai et al. [4, 3], and Gómez et al. [10, 11].

Here, we want to go one step forward and focus on the microscopic characteristics of the epidemics at the level of links of the network, see Fig. 1.

We define a new property, the epidemic *conductance* of a link, Φ_{ij} , as the probability that an edge is in a state that enables the spreading of a disease from node j to node i ; the higher the conductance, the larger the likelihood that the disease propagates in that direction. It is worth mentioning that this feature is asymmetrical, meaning that the propagation of the illness can be more probable from j to i than the other way around. These descriptors can

be used to model the hostility of the network, and to design countermeasures based on the removal of the edges with the largest conductance.

Let us consider a discrete time SIS dynamics that runs on top of a complex network of N nodes and L edges, with adjacency matrix A , and where each node i can be in one of two different states σ_i , either susceptible (S) or infected (I), i.e. $\sigma_i \in \{S, I\}$. The parameters of the SIS dynamics are the infection and recovery probabilities, β and μ , respectively. Since epidemics flow from infected nodes to their susceptible neighbors, we can define the conductance of the link between nodes i and j as the joint probability $\Phi_{ij} = P(\sigma_i = S, \sigma_j = I)$. In the same way, the epidemic is restrained by edges where the nodes are in the same state, thus it is convenient to define the coupling probabilities $\Theta_{ij}^S = P(\sigma_i = \sigma_j = S)$ and $\Theta_{ij}^I = P(\sigma_i = \sigma_j = I)$ for all pairs of neighboring nodes.

The evolution of the conductance of one link depends on the conductance and couplings to the rest of the neighboring links, and the infection rules of the SIS dynamics. Thus, we can write the following equation for each link:

$$\begin{aligned}
\Phi_{ij}(t+1) &= \Theta_{ij}^S(t)q_{ij}(t)(1 - q_{ji}(t)) \\
&+ \Phi_{ij}(t)((1 - \beta)q_{ij}(t))(1 - \mu) \\
&+ \Phi_{ji}(t)\mu(1 - (1 - \beta)q_{ji}(t)) \\
&+ \Theta_{ij}^I(t)\mu(1 - \mu)
\end{aligned} \tag{1}$$

where we have taken into account all the possible changes of state of the nodes i and j . The first term considers the susceptible coupling between nodes i and j , node i remaining susceptible, and node j is being infected by any of its other neighbors. The second term accounts for both nodes remaining in the same state, node i is not infected by any of its neighbors and node j is not recovered from the infection. Then, the third represents the transition in which node i is infected and recovers while node j is susceptible and it is infected by any of its other neighbors. Finally, the fourth term takes into account that both nodes are infected but node i recovers while node j does not. The asymmetry of the conductance implies there are a total of $2L$ conductance equations, since for each link between nodes i and j we need an equation for $\Phi_{ij}(t+1)$ and another for $\Phi_{ji}(t+1)$.

Similarly we can obtain an expression for the infected coupling Θ_{ij}^I :

$$\begin{aligned}
\Theta_{ij}^I(t+1) &= \Theta_{ij}^S(t) (1 - q_{ij}(t)) (1 - q_{ji}(t)) \\
&\quad + \Phi_{ij}(t) (1 - (1 - \beta) q_{ij}(t)) (1 - \mu) \\
&\quad + \Phi_{ji}(t) (1 - \mu) (1 - (1 - \beta) q_{ji}(t)) \\
&\quad + \Theta_{ij}^I(t) (1 - \mu)^2
\end{aligned} \tag{2}$$

In this case we have only L equations, one per link, due to the symmetry of the coupling. There is no need of extra equations for the susceptible coupling since the normalization leads to $\Theta_{ij}^S = 1 - \Phi_{ij} - \Phi_{ji} - \Theta_{ij}^I$.

The $q_{ij}(t)$ in Eqs. (1) and (2) stands for the probability that a susceptible node i is not infected by any of its neighbors (excluding node j):

$$q_{ij}(t) = \prod_{\substack{r=1 \\ r \neq j}}^N (1 - \beta A_{ri} h_{ir}) \tag{3}$$

where h_{ij} defines the hostility of j against i , i.e., the probability that node j is infected when node i is susceptible, $h_{ij} = P(\sigma_j = I | \sigma_i = S)$. The hostility can be obtained in terms of Θ_{ij}^S and Φ_{ij} as:

$$h_{ij} = \frac{\Phi_{ij}}{\Phi_{ij} + \Theta_{ij}^S} \tag{4}$$

Note that the denominator in Eq. (4) is a property of node i given that $\Phi_{ij} + \Theta_{ij}^S = P(\sigma_i = S)$ for all neighboring nodes j of vertex i .

We call this system of $3L$ equations and unknowns (conductances and infected couplings) our Epidemic Link Equations (ELE) model. It can be solved by iteration, starting from any meaningful initial condition, e.g. $\Theta_{ij}^I(0) = \rho_0^2$ and $\Phi_{ij}(0) = \Phi_{ji}(0) = \rho_0(1 - \rho_0)$ (for any $0 < \rho_0 \leq 1$), until fixed values are found. Apart from the solution where all nodes are susceptible, $\Theta_{ij}^S = 1$ for all the links, a non-trivial one appears when the system is above the critical value of the epidemic spreading. When that happens, the incidence of the epidemic process, the average number of infected nodes in the whole system, has a positive value, that can be computed as:

$$\rho = \frac{1}{N} \sum_{i=1}^N \frac{1}{k_i} \sum_{j=1}^N A_{ji} (\Phi_{ji} + \Theta_{ij}^I) \tag{5}$$

The determination of the epidemic threshold is performed by considering a state of the system in which the epidemic incidence is very small ($\Phi_{ij}, \Phi_{ji}, \Theta_{ij}^I \ll 1$, for all links), thus the system of equations can be linearized, resulting in:

$$\Theta_{ij}^I = \beta(1 - \mu)\Phi_{ij} + \beta(1 - \mu)\Phi_{ji} + (1 - \mu)^2\Theta_{ij}^I \quad (6)$$

$$\begin{aligned} \Phi_{ij} &= \beta \sum_r (A_{rj} - (1 - \mu)\delta_{ri})\Phi_{jr} \\ &+ (1 - \beta)(1 - \mu)\Phi_{ij} + \mu(1 - \mu)\Theta_{ij}^I \end{aligned} \quad (7)$$

Here we have removed the dependence on time, to emphasize we are considering the steady state. From Eq. (6) we can write

$$\Theta_{ij}^I = \frac{\beta(1 - \mu)}{\mu(2 - \mu)}(\Phi_{ij} + \Phi_{ji}) \quad (8)$$

Now, calling $\epsilon_i = \Phi_{ji} + \Theta_{ij}^I \ll 1$, which does not depend on node j since $P(\sigma_i = I, \sigma_j = S) + P(\sigma_i = I, \sigma_j = I) = P(\sigma_i = I)$, we make the following ansatz:

$$\Theta_{ij}^I = \Upsilon(\epsilon_i + \epsilon_j) \quad (9)$$

$$\Phi_{ij} = X\epsilon_i + Z\epsilon_j \quad (10)$$

where Υ , X and Z are constants independent of the link. These ansatz take into account the symmetry of the infected coupling and the asymmetry of the conductance. We can determine the constants by substitution in Eq. (8) and using the definition of ϵ_i , which leads to

$$\Upsilon = \frac{\beta(1 - \mu)}{\mu(2 - \mu) + 2\beta(1 - \mu)} \quad (11)$$

$$X = -\Upsilon \quad (12)$$

$$Z = 1 - \Upsilon \quad (13)$$

Finally, we build equations for the ϵ_i by substituting Eqs. (6) and (7) in $\epsilon_i = \Phi_{ji} + \Theta_{ij}^I$, and using the ansatz. The result is

$$\frac{\mu}{\beta}\epsilon_i = \sum_j B_{ji}\epsilon_j \quad (14)$$

where B is a matrix whose elements depend on the adjacency matrix of the network, on Υ and on the degrees k_i of the nodes:

$$B_{ij} = (1 - \Upsilon)A_{ij} - \Upsilon k_i \delta_{ij} \quad (15)$$

The δ_{ij} stands for the Kronecker delta function, which is 1 if $i = j$, and 0 otherwise. If μ/β is an eigenvalue of matrix B , Eq. (14) has a non-trivial solutions. Hence, the onset of the epidemics β_c , the lowest value of β that yields non-trivial solutions of Eq. (14), is given by

$$\beta_c = \frac{\mu}{\Lambda_{\max}(B)} \quad (16)$$

where $\Lambda_{\max}(B)$ is the largest eigenvalue of matrix B . Note that matrix B depends on β and μ , thus Eq. (16) is an implicit equation for β_c , which can be solved by iteration.

To test the agreement between our approach and empirical simulations we have analyzed the incidence of the epidemics, ρ , in different synthetic and real network structures, covering the full range of infection probabilities, β , see Fig. 2. The results in Fig. 2 show a perfect agreement between our ELE model and the Monte Carlo simulations, and a good prediction of the epidemic threshold for all synthetic and real networks, pointing out the validity of our model to describe the global impact of the epidemics.

Using Eq. (4) we can define the average hostility of the network H ,

$$H = \frac{1}{N} \sum_i \frac{1}{k_i} \sum_j A_{ji} \frac{\Phi_{ij}}{\Phi_{ij} + \Theta_{ij}^S} \quad (17)$$

which accounts for the average fraction of infected individuals in the neighborhood of a susceptible node, see Fig. 3.

Comparing this hostility $H(\beta)$ with the epidemic incidence $\rho(\beta)$, and averaging over all values of the infection probability, we end up with a global hostility measure,

$$\mathcal{H} = \int_0^1 (H(\beta) - \rho(\beta)) d\beta \quad (18)$$

that can be used to compare the hostility that nodes find in their neighborhoods for different topological configurations of the network. This is relevant to model the perception that susceptible individuals have about their environment, that can be different to the real level of incidence of the epidemics.

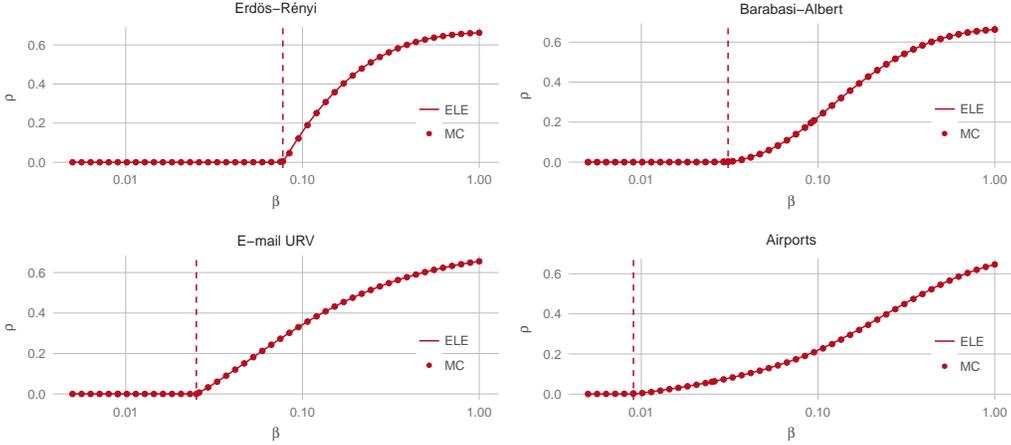


Figure 2: **Incidence of the epidemic process ρ as a function of the infection probability β .** We show the incidence level for the ELE model (solid lines) and for Monte Carlo simulations (circles). The theoretical epidemic threshold calculated using Eq. (16) is pointed out using a vertical line. We have used the following networks: two syntetic networks of 5000 nodes, an Erdős-Rényi with $\langle k \rangle = 5$ (up-left) and a Barabási-Albert with $\langle k \rangle = 6$ (up-right), and two empirical networks, the URV email network [13] (bottom-left) and the air transportation network [14] (bottom-right). We have set the recovery probability to $\mu = 0.5$ and the initial epidemic incidence to $\rho_0 = 0.5$. To ensure statistical significance, in the case of syntetic networks we have computed averages over 36 different network initializations (errors bars are smaller than the size of the symbols). The Monte Carlo simulations have been performed using a Quasi-Stationary approach [7], averaging the last 1×10^4 steps of a total of 2.5×10^4 steps with 50 active configurations and a probability of configuration update of 0.2.

In Fig. 4 we explore the dependency of the global hostility on the heterogeneity of the network. We make use of the method described in Ref. [12], that enables us to generate a series of networks with structures that interpolate between Erdős-Rényi and Barabási-Albert networks, preserving the average degree. The results show a clear increasing effect of the heterogeneity of the network on \mathcal{H} independently of the average degree of the network, meaning that individuals on a highly heterogeneous network have, on average, a

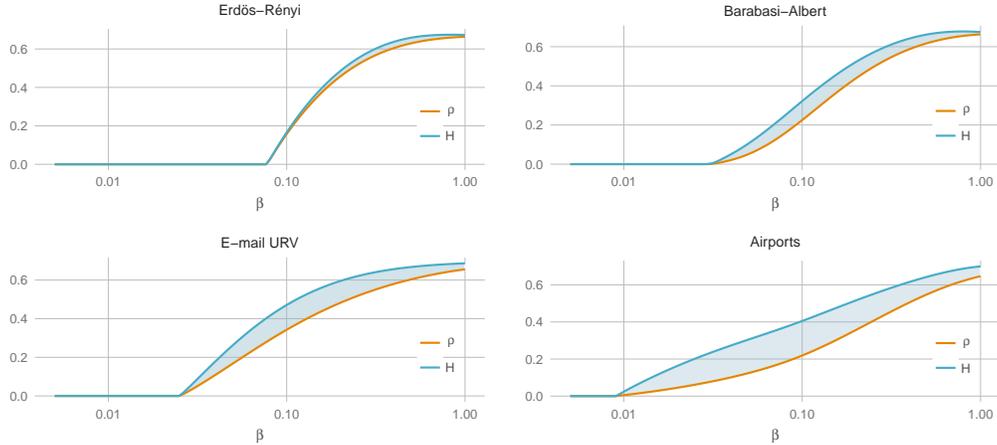


Figure 3: **Average hostility H (dashed line), and incidence ρ (solid line) as a function of the infection probability β .** We make use of the same methodological parameters detailed in Fig. 2 to compute ρ using ELE approach and H using the Eq. (4).

higher perception of a hostile environment around them.

To fully understand the impact of link properties on spreading dynamics, we consider the problem of reducing the incidence of the epidemics through link removal. If we identify the links which are more involved in the propagation of a disease, it is possible to design targeted countermeasures which affect just specific links instead of whole nodes, while being more effective. This can be illustrated by a hypothetical pandemic disease propagated using the air transportation network: the isolation of one airport is a dramatic measure that is socially and politically difficult to accept and put into practice, but the suspension of just a few connections between selected airports could be more easily assumed, and at the same time achieving a better contention of the disease.

Within our scheme, a natural solution is to remove at each step of the process as much conductance as possible. The node oriented alternative would consist in the removal of all the edges of the node with higher probability of being infected. We compare both options in Fig. 5, together with a random edge removal as a reference. The results clearly show that the removal of the edges with largest total conductance leads to a faster extinction of the

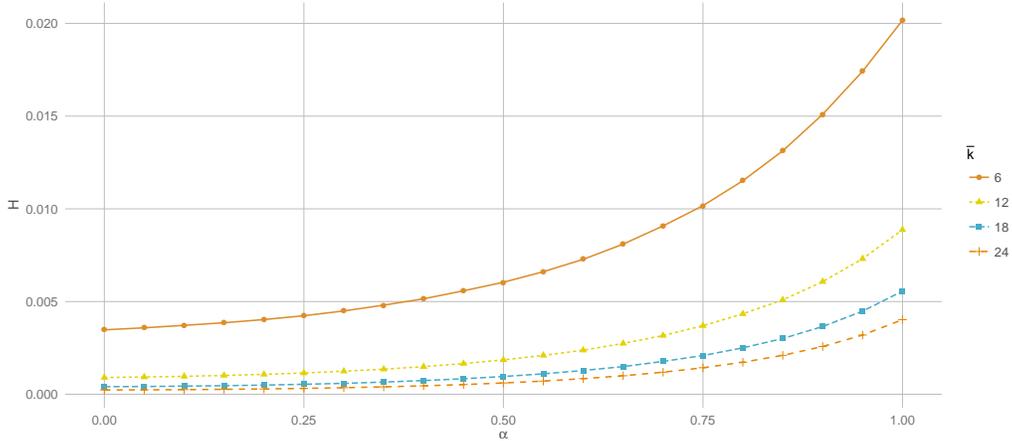


Figure 4: **Global hostility \mathcal{H} as function of the heterogeneity of the network and the average degree $\langle k \rangle$.** To generate a set of networks with different heterogeneity, we use the model described in [12] that let us parameterize the amount of heterogeneity with a parameter α , going from an ER network at $\alpha = 0$ to a BA network if $\alpha = 1$. For each value of α we average \mathcal{H} over 36 different networks, each made of 5000 nodes. We compare the results for different average connectivity setups.

epidemics.

In summary, we have introduced an Epidemic Link Equations (ELE) model capable of describing a SIS epidemic process in complex networks at the level of links, instead of focusing our attention on the nodes as in the previous literature. This ELE model allows the calculation of the epidemic threshold with larger precision than previous approaches, and opens the possibility of analyzing the response of different network architectures to the epidemics, e.g. by defining new measures such as the conductance and coupling of the links, or the hostility of the whole network. We have also shown that an edge percolation of the network guided by maximum conductance leads to a faster extinction of the epidemics.

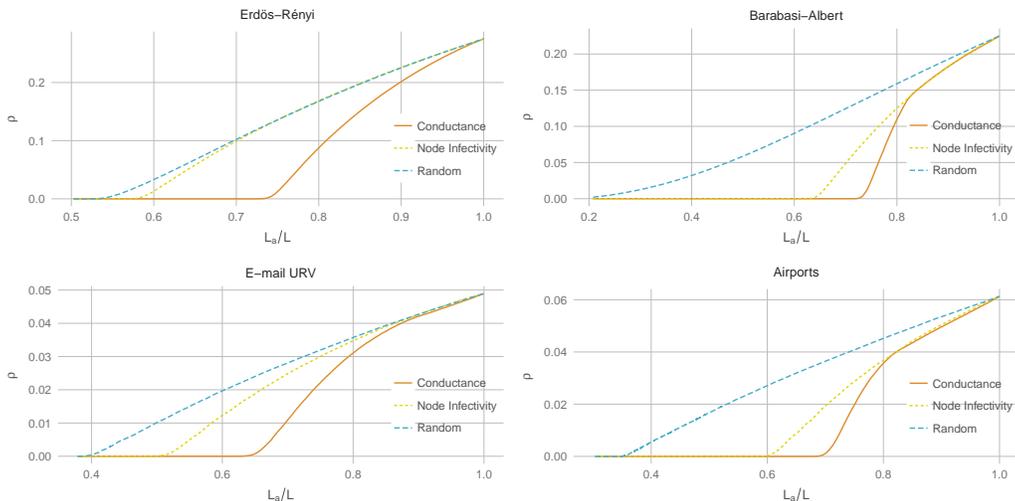


Figure 5: **Targeted edge percolation.** We show the incidence of the epidemics, ρ , as function of the occupation probability, L_a/L , where L_a is the current number of active edges in the percolation process. We compare three different percolation strategies: a random edge removal (blue dashed line); removing the edges of the node with highest probability of being infected, $P(\sigma_i = I)$ (yellow dotted line); and removing the edge that has the largest total conductance (orange solid line). We have made use of the total conductance since the percolation process is undirected and we try to remove as much conductance as possible.

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