

GLOBAL EPIDEMIC SPREADING PROCESSES IN COUPLED NETWORKS

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Abstract

We study the effect of coupling two random networks where an epidemic process propagates. A theoretical SIS model is applied and a new critical threshold for the existence of an endemic state is analytically calculated for the two coupled networks, under the assumption that there is total correlation between the outer degree and the inner degree of each node. Our main result is that a global endemic state can exist in the coupled networks, even though the epidemics does not propagate on each network separately. Finally, we checked these results by running large scale computer simulations.

1 Introduction

Recently, network models have become a powerful and attractive theoretical tool, not only for their non trivial properties but also for their large range of applications to real networks (Albert & Barabási, 2002): from the internet (Pastor-Satorras & Vespignani, 2004) to food webs (Dunne *et al.*, 2002), as well as interactions between proteins, genes or metabolites (Wagner & Fell, 2001). Also social interactions, as friendship (Amaral *et al.*, 2000) or sexual contacts (Liljeros *et al.*, 2001), can be treated as a network where nodes represent people and they are connected by an edge if they interact with each other. Dynamical processes, such as epidemic spreading, are specially interesting in this context and can be studied in networks in order to explain how networks topology affects the spreading of the diseases and its prevalence (Newman, 2003).

Current studies focus on the propagation of epidemics in isolated networks (Pastor-Satorras & Vespignani, 2001). However, many interesting cases involve the spreading of the disease in coupled networks (Buldyrev *et al.*, 2010). For example, sexually transmitted diseases can propagate both in heterosexual and homosexual networks of sexual contacts (Liljeros *et al.*, 2001). These two networks are not completely isolated due to the existence of bisexual individuals, which act as an effective coupling of the two networks.

In this master thesis, we consider the SIS epidemic spreading model (Pastor-Satorras & Vespignani, 2002) running on two coupled random networks. In the SIS model, nodes can be in the susceptible (S) or infected (I) state. Susceptible nodes are infected at a given rate λ when they are in contact with an infected node,

while the infected ones decay spontaneously to the susceptible state at a rate δ . The ratio between λ and δ and the topology of the network determine the prevalence of the epidemics. This epidemic process runs on two coupled networks, and each one will be in a range where the epidemics does not propagate. It is known that for a single network (and under the mean field approximation), the condition that must be satisfied in order to make the epidemic survive reaching a steady state is (Pastor-Satorras & Vespignani, 2002):

$$\Lambda \equiv \lambda \frac{\langle k^2 \rangle}{\langle k \rangle} > 1, \quad (1)$$

so the critical value of λ is:

$$\lambda_c = \frac{\langle k \rangle}{\langle k^2 \rangle}, \quad (2)$$

where k is the degree of a node (its total number of neighbours), $\langle k \rangle$ indicates the average of the degree distribution, and $\langle k^2 \rangle$ is its second moment, which quantifies the topological fluctuations. If λ is above this threshold λ_c , the epidemics prevails whereas if it is below this value, the epidemics dies out exponentially fast. Our goal in this master thesis is to find an equivalent condition in the case of two coupled random networks.

In Section 2, we prove analytically that there exists a threshold for the coupled networks such that a global endemic state exists, even though the epidemics does not propagate in each networks separately. It is shown that the way in which the networks are interconnected is crucial for the existence of the global endemic state. In particular, we study different levels of the correlation between the inner degree (the degree

between nodes belonging to the same network) and the outer degree (edges connecting nodes from distinct networks). If the correlation is maximum, the epidemics prevails.

In Section 3, we compare our theoretical results with numerical simulations. We create two large scale random networks with exponential degree distributions. We compute the time evolution of the prevalence for different values of the infection rate λ as well as the number of edges connecting the two networks. Finally, we check that an improvement is observed when the critical threshold for an isolated network is estimated more accurately. Conclusions are presented in Section 4.

2 SIS process in coupled networks

In a single network, the SIS process is an epidemic model where nodes can be in two different states: state S corresponds to the *susceptible* state and state I corresponds to the *infected* one. An infected node can infect one of its neighbours with rate λ if it is in the susceptible state and decays to the S state with rate δ . The ratio between λ and δ and the topology of the network determines the prevalence of the epidemics (Pastor-Satorras & Vespignani, 2002), as we have seen in Equations 1 and 2.

Now, let us consider the SIS process in two coupled networks, A and B : $\lambda^{\mathcal{X}\mathcal{Y}}$ denotes the rate that an infected node from network \mathcal{X} infects a susceptible node from network \mathcal{Y} , and $\delta^{\mathcal{X}}$ is the rate to turn to the susceptible state (\mathcal{X} and \mathcal{Y} can both be either networks A or B). $n_i(t)$ indicates the state of node i , and it can take the following values:

$$n_i(t) = \begin{cases} 1, & \text{if the node is infected} \\ 0, & \text{if the node is not infected} \end{cases} .$$

From now on we are focusing on nodes from network A , but the same equations can be obtained for network B by substituting A for B and vice versa.

It is possible to write the state of the node i from network A at time $t + dt$, $n_i^A(t + dt)$, in terms of its value in the previous time step $n_i^A(t)$ (Catanzaro *et al.*, 2005):

$$n_i^A(t + dt) = n_i^A(t)\eta^A(dt) + (1 - n_i^A(t))\xi^A(dt) , \quad (3)$$

where $\eta^A(dt)$ is a dichotomous random variable controlling the transition between the infected and susceptible states,

$$\eta^A(dt) = \begin{cases} 0 & \text{with probability } \delta^A dt \\ 1 & \text{with probability } 1 - \delta^A dt , \end{cases} \quad (4)$$

and $\xi^A(dt)$ is another dichotomous random variable controlling the transition between the susceptible and infected states,

$$\xi^A(dt) = \begin{cases} 1 & \text{with prob. } \lambda^{AA} dt \sum_{j \in A} a_{ij}^{AA} n_j^A(t) + \\ & \lambda^{BA} dt \sum_{j \in B} a_{ij}^{AB} n_j^B(t) \\ 0 & \text{with prob. } 1 - \lambda^{AA} dt \sum_{j \in A} a_{ij}^{AA} n_j^A(t) + \\ & \lambda^{BA} dt \sum_{j \in B} a_{ij}^{AB} n_j^B(t) , \end{cases} \quad (5)$$

where $\{a_{ij}^{\mathcal{X}\mathcal{Y}}\}$ is the adjacent matrix, with values equal to 1 if the node i from network \mathcal{X} is connected to node j from network \mathcal{Y} , and equal to 0 otherwise. Since $n_i^A(t + dt)$ can value 0 or 1, the first term of the right hand side of Equation 3 considers the case where the node is infected at the previous time step (the second term vanishes), and the second term considers the case when the node is, previously, susceptible to be infected (the first term vanishes).

By computing the conditional average of Equation 3, we obtain:

$$\begin{aligned} \langle n_i^A(t + dt) | n(t) \rangle &= n_i^A(t)(1 - \delta^A dt) + \\ & (1 - n_i^A(t)) \left[\lambda^{AA} dt \sum_{j \in A} a_{ij}^{AA} n_j^A(t) + \right. \\ & \left. \lambda^{BA} dt \sum_{j \in B} a_{ij}^{AB} n_j^B(t) \right] . \end{aligned} \quad (6)$$

This equation describes the evolution of the state of node i from network A by knowing the state of all nodes as well as its own state at the previous time step.

By multiplying this equation by the probability to be at state $n^A(t)$ at time t , and summing for all possible configurations, we can derive:

$$\begin{aligned} \frac{d\langle n_i^A(t) \rangle}{dt} &= -\delta^A \langle n_i^A(t) \rangle + \\ & \lambda^{AA} \sum_{j \in A} a_{ij}^{AA} \left(\langle n_j^A(t) \rangle - \langle n_i^A(t) n_j^A(t) \rangle \right) + \\ & \lambda^{BA} \sum_{j \in B} a_{ij}^{AB} \left(\langle n_j^B(t) \rangle - \langle n_i^A(t) n_j^B(t) \rangle \right) . \end{aligned} \quad (7)$$

If we define

$$\langle n_i(t) \rangle \equiv \rho_i(t), \quad \langle n_i(t) n_j(t) \rangle \equiv \rho_{ij}(t),$$

Equation 7 can be rewritten as follows:

$$\begin{aligned} \frac{d\rho_i^A(t)}{dt} &= -\delta^A \rho_i^A(t) + \lambda^{AA} \sum_{j \in A} a_{ij}^{AA} \left(\rho_j^A(t) - \rho_{ij}^{AA}(t) \right) + \\ & \lambda^{BA} \sum_{j \in B} a_{ij}^{AB} \left(\rho_j^B(t) - \rho_{ij}^{AB}(t) \right) . \end{aligned} \quad (8)$$

Now we will assume that all nodes with the same degree are statistically equivalent and we can treat

them equally (Boguñá *et al.*, 2003). Given a node in network \mathcal{X} , we will define $\vec{k}^{\mathcal{X}}$ as its degree vector in network \mathcal{X} , $\vec{k}^A = (k_a^A, k_b^A)$. The first component indicates the inner degree (number of connections of the node to other nodes from network A) and the second component corresponds to the outer degree (number of connections of the same node from network A connecting nodes from network B). With that assumption we can define:

$$\rho_i^{\mathcal{X}}(t) \equiv \rho_{\vec{k}^{\mathcal{X}}}(t), \quad \forall i \in \mathcal{V}(\vec{k}^{\mathcal{X}}) \quad (9)$$

$$\rho_{ij}^{\mathcal{X}\mathcal{Y}}(t) \equiv \rho_{\vec{k}^{\mathcal{X}}\vec{k}^{\mathcal{Y}}}(t), \quad \forall i \in \mathcal{V}(\vec{k}^{\mathcal{X}}), \quad j \in \mathcal{V}(\vec{k}^{\mathcal{Y}}), \quad (10)$$

where $\mathcal{V}(\vec{k}^{\mathcal{X}})$ and $\mathcal{V}(\vec{k}^{\mathcal{Y}})$ are the set of vertices from networks \mathcal{X} and \mathcal{Y} with degree \vec{k} and \vec{k}' , respectively. If we add all densities from nodes with the same degree and network, and we average over all of them (dividing by the total number of nodes with degree \vec{k} from network A , denoted by $N_{\vec{k}^A}$), we will obtain a differential equation for the fraction of infected nodes in degree class \vec{k}^A :

$$\begin{aligned} \frac{d\rho_{\vec{k}^A}}{dt} = & -\delta^A \rho_{\vec{k}^A} + \\ & \lambda^{AA} \sum_{\vec{k}'^A} (\rho_{\vec{k}'^A} - \rho_{\vec{k}^A \vec{k}'^A}) \frac{1}{N_{\vec{k}^A}} \sum_{i \in \mathcal{V}(\vec{k}^A)} \sum_{j \in \mathcal{V}(\vec{k}'^A)} a_{ij}^{AA} + \\ & \lambda^{BA} \sum_{\vec{k}'^B} (\rho_{\vec{k}'^B} - \rho_{\vec{k}^A \vec{k}'^B}) \frac{1}{N_{\vec{k}^A}} \sum_{i \in \mathcal{V}(\vec{k}^A)} \sum_{j \in \mathcal{V}(\vec{k}'^B)} a_{ij}^{AB}. \end{aligned} \quad (11)$$

With the assumption of all nodes with the same degree being statistically equivalent, we can also set:

$$\frac{1}{N_{\vec{k}'^A}} \sum_{i \in \mathcal{V}(\vec{k}^A)} \sum_{j \in \mathcal{V}(\vec{k}'^A)} a_{ij}^{AA} = k_a^A P(\vec{k}'^A | \vec{k}^A), \quad (12)$$

and

$$\frac{1}{N_{\vec{k}'^A}} \sum_{i \in \mathcal{V}(\vec{k}^A)} \sum_{j \in \mathcal{V}(\vec{k}'^B)} a_{ij}^{AB} = k_b^A P(\vec{k}'^B | \vec{k}^A). \quad (13)$$

By assuming the mean-field approximation, we can also consider that (Boguñá *et al.*, 2003):

$$\rho_{\vec{k}^A \vec{k}'^A} \approx \rho_{\vec{k}^A} \rho_{\vec{k}'^A}.$$

Finally, the time evolution equation of the prevalence can be expressed as follows:

$$\begin{aligned} \frac{d\rho_{\vec{k}^A}}{dt} = & -\delta^A \rho_{\vec{k}^A} + \\ & \lambda^{AA} (1 - \rho_{\vec{k}^A}) k_a^A \sum_{\vec{k}'^A} \rho_{\vec{k}'^A} P(\vec{k}'^A | \vec{k}^A) + \\ & \lambda^{BA} (1 - \rho_{\vec{k}^A}) k_b^A \sum_{\vec{k}'^B} \rho_{\vec{k}'^B} P(\vec{k}'^B | \vec{k}^A). \end{aligned} \quad (14)$$

2.1 Uncorrelated networks

If the networks are uncorrelated, i.e., there is no correlation between the degrees of the connected

nodes, it can be proved that

$$P(\vec{k}'^A | \vec{k}^A) = \frac{k_a^A P(\vec{k}'^A)}{\langle k_a^A \rangle}, \quad (15)$$

$$P(\vec{k}'^B | \vec{k}^A) = \frac{k_b^A P(\vec{k}'^B)}{\langle k_b^A \rangle}, \quad (16)$$

and the time evolution equation can be written:

$$\begin{aligned} \frac{d\rho_{\vec{k}^A}}{dt} = & -\delta^A \rho_{\vec{k}^A} + \\ & \lambda^{AA} \frac{k_a^A}{\langle k_a^A \rangle} (1 - \rho_{\vec{k}^A}) \sum_{\vec{k}'^A} k_a^A \rho_{\vec{k}'^A} P(\vec{k}'^A) + \\ & \lambda^{BA} \frac{k_b^A}{\langle k_b^A \rangle} (1 - \rho_{\vec{k}^A}) \sum_{\vec{k}'^B} k_b^A \rho_{\vec{k}'^B} P(\vec{k}'^B). \end{aligned} \quad (17)$$

Equation 17 represents a set of coupled differential equations for all values of \vec{k} , that can be expressed as $\dot{X} = MX$, where M is the coefficients matrix. Since we are interested in finding the prevalence of the epidemics at the steady state, we need to find the eigenvalues of the linearised system. Let $(\rho_{\vec{k}_1^A}^0, \rho_{\vec{k}_2^A}^0, \dots, \rho_{\vec{k}_n^A}^0)$ be the non-zero equilibrium point, which can be obtained by solving Equation 17 when time derivative is zero. It is known that the eigenvalues of matrix M give the stability of the equilibrium points: if $\Lambda_{\max} > 0$, at $t \rightarrow \infty$ the system will not converge at the equilibrium point, but if $\Lambda_{\max} < 0$, the system will converge at $(\rho_{\vec{k}_1^A}^0, \rho_{\vec{k}_2^A}^0, \dots, \rho_{\vec{k}_n^A}^0)$ and we will find stability around this solution. $\Lambda_{\max} = 0$ will denote the critical threshold.

For networks A and B , the linearised equations are:

$$\begin{aligned} \frac{d\rho_{\vec{k}^A}}{dt} = & -\delta^A \rho_{\vec{k}^A} + \lambda^{AA} \frac{k_a^A}{\langle k_a^A \rangle} \sum_{\vec{k}'^A} \rho_{\vec{k}'^A} P(\vec{k}'^A) k_a^A + \\ & \lambda^{BA} \frac{k_b^A}{\langle k_b^A \rangle} \sum_{\vec{k}'^B} \rho_{\vec{k}'^B} P(\vec{k}'^B) k_b^A, \end{aligned} \quad (18)$$

and

$$\begin{aligned} \frac{d\rho_{\vec{k}^B}}{dt} = & -\delta^B \rho_{\vec{k}^B} + \lambda^{BB} \frac{k_b^B}{\langle k_b^B \rangle} \sum_{\vec{k}'^B} \rho_{\vec{k}'^B} P(\vec{k}'^B) k_b^B + \\ & \lambda^{AB} \frac{k_a^B}{\langle k_a^B \rangle} \sum_{\vec{k}'^A} \rho_{\vec{k}'^A} P(\vec{k}'^A) k_a^B. \end{aligned} \quad (19)$$

The stationary system corresponds to the temporal derivative being equal to zero. In that case, the system can be written in the following matrix way:

$$0 = \begin{pmatrix} -\delta^A \delta_{\vec{k}^A, \vec{k}'^A} + \lambda^{AA} \frac{k_a^A}{\langle k_a^A \rangle} P(\vec{k}'^A) k'_a{}^A & \lambda^{BA} \frac{k_b^A}{\langle k_b^A \rangle} P(\vec{k}'^B) k'_a{}^B \\ \lambda^{AB} \frac{k_a^B}{\langle k_a^B \rangle} P(\vec{k}'^A) k'_b{}^A & -\delta^B \delta_{\vec{k}^B, \vec{k}'^B} + \lambda^{BB} \frac{k_b^B}{\langle k_b^B \rangle} P(\vec{k}'^B) k'_b{}^B \end{pmatrix} \begin{pmatrix} \rho_{\vec{k}^A} \\ \rho_{\vec{k}^B} \end{pmatrix} \quad (20)$$

As we said in the introduction, we can make $\delta^A = 1$. Moreover, we will assume that the rates of becoming susceptible are the same for both networks, that is, $\delta^A = \delta^B = 1$. In that case, the eigenvalue problem we have in Equation 20 can be written as:

$$(-\mathbb{I} + \mathbb{C})\vec{v} = -\vec{v} + \Lambda\vec{v} = (-1 + \Lambda)\vec{v}, \quad (21)$$

where \mathbb{I} is the identity matrix, \mathbb{C} is the matrix which contains the infection terms, and \vec{v} and Λ are its eigenvectors and eigenvalues, respectively.

In order to find stability, all eigenvalues must be negative, otherwise the system would be unstable. Instability in our problem means that the infection does not prevail and no nodes keep infected for large times. In that case, the critical value we are looking for is:

$$-1 + \Lambda = 0 \Rightarrow \Lambda = 1.$$

If $\Lambda > 1$ the infection prevails. To find the eigenvalues of matrix \mathbb{C} we can write:

$$\begin{aligned} \lambda^{AA} \frac{k_a^A}{\langle k_a^A \rangle} \sum_{\vec{k}'^A} P(\vec{k}'^A) k'_a{}^A v_{\vec{k}'^A} + \\ \lambda^{BA} \frac{k_b^A}{\langle k_b^A \rangle} \sum_{\vec{k}'^B} P(\vec{k}'^B) k'_a{}^B v_{\vec{k}'^B} = \Lambda v_{\vec{k}^A}, \end{aligned} \quad (22)$$

$$\begin{aligned} \lambda^{BB} \frac{k_b^B}{\langle k_b^B \rangle} \sum_{\vec{k}'^B} P(\vec{k}'^B) k'_b{}^B v_{\vec{k}'^B} + \\ \lambda^{AB} \frac{k_a^B}{\langle k_a^B \rangle} \sum_{\vec{k}'^A} P(\vec{k}'^A) k'_b{}^A v_{\vec{k}'^A} = \Lambda v_{\vec{k}^B}, \end{aligned} \quad (23)$$

where $v_{\vec{k}^A}$ and $v_{\vec{k}^B}$ are the eigenvectors. We can see that each summation term is constant, so the eigenvectors must have the form:

$$v_{\vec{k}^A} = m k_a^A + n k_b^A, \quad (24)$$

$$v_{\vec{k}^B} = p k_a^B + q k_b^B, \quad (25)$$

where m, n, p and q are constants. If we substitute Equations 24 and 25 into 22 and 23, and define:

$$\Lambda^{AA} \equiv \lambda^{AA} \frac{\langle k_a^A \rangle}{\langle k_a^A \rangle}, \quad \Lambda^{AB} \equiv \lambda^{AB} \frac{\langle k_b^A \rangle}{\langle k_b^A \rangle}, \quad (26)$$

$$\Lambda^{BB} \equiv \lambda^{BB} \frac{\langle k_b^B \rangle}{\langle k_b^B \rangle}, \quad \Lambda^{BA} \equiv \lambda^{BA} \frac{\langle k_a^B \rangle}{\langle k_a^B \rangle}, \quad (27)$$

$$F_1 \equiv \lambda^{AA} \frac{\langle k_a^A k_b^A \rangle}{\langle k_a^A \rangle}, \quad F_2 \equiv \lambda^{AB} \frac{\langle k_a^A k_b^A \rangle}{\langle k_b^A \rangle}, \quad (28)$$

$$F_3 \equiv \lambda^{BA} \frac{\langle k_a^B k_b^B \rangle}{\langle k_a^B \rangle}, \quad F_4 \equiv \lambda^{BB} \frac{\langle k_a^B k_b^B \rangle}{\langle k_b^B \rangle}, \quad (29)$$

we obtain, after some manipulations, the following equation for the eigenvalues:

$$\begin{aligned} (\Lambda^2 - \Lambda^{AB} \Lambda^{BA})(\Lambda - \Lambda^{AA})(\Lambda - \Lambda^{BB}) = F_1 F_2 F_3 F_4 + \\ F_1 F_2 \Lambda^{BA} (\Lambda - \Lambda^{BB}) + F_3 F_4 \Lambda^{AB} (\Lambda - \Lambda^{AA}). \end{aligned} \quad (30)$$

We can see that Λ^{AA} and Λ^{BB} are the eigenvalues shown in Equation 1 of networks A and B , respectively, if they are not coupled.

By solving this equation it is possible to determine the eigenvalues as a function of the average degrees and second moments of the networks and, therefore, the stability of the system.

2.2 Limit case: maximum correlation between outer and inner degrees

In this master thesis, we will consider the case where both networks do not propagate the epidemic by themselves. Hence, Λ^{AA} and Λ^{BB} are below the critical threshold:

$$\Lambda^{AA} < 1, \quad \Lambda^{BB} < 1.$$

Equations from 26 to 30 show that the prevalence of the epidemic for the coupled networks not only will depend on the outer and inner degrees of the nodes but also in their correlation. We study the coupling with maximum local correlation between the inner and outer degree of a node, such that:

$$k_b^A = \alpha k_a^A \Rightarrow \alpha = \frac{\langle k_b^A \rangle}{\langle k_a^A \rangle}, \quad (31)$$

$$k_a^B = \beta k_b^B \Rightarrow \beta = \frac{\langle k_a^B \rangle}{\langle k_b^B \rangle}, \quad (32)$$

where α and β are constants.

In order to go further, we will make all probabilities to infect to be equal ($\lambda^{AA} = \lambda^{AB} = \lambda^{BA} = \lambda^{BB}$). Then, after some basic operations, the eigenvalues equation can be expressed:

$$\begin{aligned} \Lambda^2 [\Lambda^2 - (\Lambda^{AA} + \Lambda^{BB})\Lambda - \\ \alpha\beta\Lambda^{AA}\Lambda^{BB} + \Lambda^{AA}\Lambda^{BB}] = 0. \end{aligned} \quad (33)$$

The solution of the maximum eigenvalue is then:

$$\Lambda = \frac{1}{2} \left\{ \Lambda^{AA} + \Lambda^{BB} + \sqrt{(\Lambda^{AA} - \Lambda^{BB})^2 + 4\Lambda^{AA}\Lambda^{BB} \frac{\langle k_b^A \rangle \langle k_a^B \rangle}{\langle k_a^A \rangle \langle k_b^B \rangle}} \right\}. \quad (34)$$

Since the infection prevails if $\Lambda > 1$, we get:

$$\frac{\langle k_b^A \rangle \langle k_a^B \rangle}{\langle k_a^A \rangle \langle k_b^B \rangle} > \frac{(1 - \Lambda^{AA})(1 - \Lambda^{BB})}{\Lambda^{AA}\Lambda^{BB}}. \quad (35)$$

If the two networks are statistically equivalent,

$$\frac{\langle k_b^A \rangle}{\langle k_a^A \rangle} > \frac{1 - \Lambda^{AA}}{\Lambda^{AA}}. \quad (36)$$

Then, the critical value from which the epidemic propagates is:

$$\alpha_c = \frac{1 - \Lambda^{AA}}{\Lambda^{AA}}, \quad (37)$$

where

$$\alpha \equiv \frac{\langle k_b^A \rangle}{\langle k_a^A \rangle} = \frac{\langle k_b^B \rangle}{\langle k_a^B \rangle}. \quad (38)$$

By controlling the fraction $\frac{\langle k_b^A \rangle}{\langle k_a^A \rangle}$ and using Equation 1 we will be able to find the critical value of Λ^{AA} (remember all rates have the same value) from which the infection propagates. Alternatively, this equation will allow us to determine the fraction of edges we will have to add connecting the two networks in order to make the epidemic survive if we know the infection rates (making the correlation to be maximum). In the following section, we compare these results with simulations.

3 Numerical simulation

In this section, we generate two exponential networks with inner minimum degree equal to 2. The number of internal edges of each node follows the probability distribution function:

$$f(k) = \begin{cases} \mu e^{-\mu(k_{in}-2)} & \text{if } k_{in} \geq 2 \\ 0 & \text{otherwise} \end{cases} \quad (39)$$

where we call k_{in} either k_a^A or k_b^B .

We made the inner average degree to be 10, $\langle k_a^A \rangle = \langle k_b^B \rangle = 10$. Moreover, we can easily prove that:

$$\langle k_{in} \rangle = 2 + \frac{1}{\mu}. \quad (40)$$

Then, by imposing the value of $\langle k_{in} \rangle$ we could determine μ . The value of $\langle k_{in}^2 \rangle$ could be easily obtained by computing:

$$\langle k_{in}^2 \rangle = \mu \int_2^\infty k_{in}^2 e^{-\mu(k_{in}-2)} dk = 4 + \frac{2}{\mu} \left(2 + \frac{1}{\mu} \right). \quad (41)$$

The algorithm used in order to implement this process is explained in Appendix and it is based on considering continuous time instead of discrete time, which makes the program more efficient and powerful and realistic. In the following simulations, we fixed different values of the infection rates (i.e. values of Λ). Then, by adding edges connecting the two networks making the correlation to be maximum, we could determine at which value of α the epidemic propagated when $t \rightarrow \infty$. Figure 1 shows the prevalence of the infection at large times. Arrows show the theoretical values of

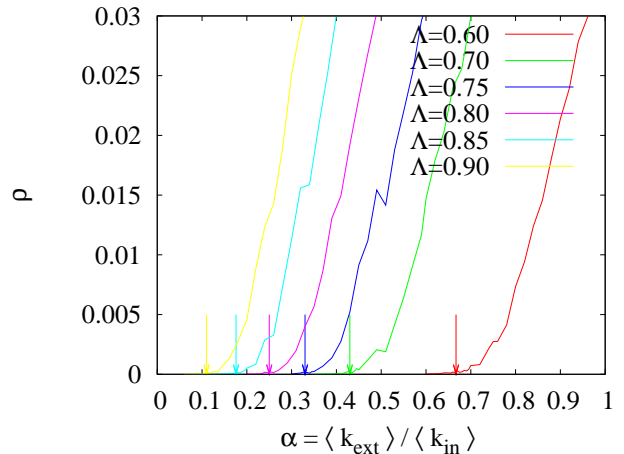


Figure 1: Prevalence of the epidemics as a function of α for different values of Λ . Arrows show the expected critical thresholds. We simulated the networks with 3×10^5 .

the critical threshold obtained in the previous section. We can observe that, qualitatively, the numerical results agree with the theoretical ones.

Given a certain infection rate, the critical value of α from which there is a phase transition and the infection propagates can also be determined by measuring the time evolution of the density of infected nodes. Right at the critical point the prevalence decays as a function of time as a power law function. Figure 2a shows, in logarithmic scale, the time evolution of the global prevalence for different values of α for $\Lambda = 0.8$. We can see that when $\alpha = 0.31$ the time evolution of the infected nodes shows a good power law behaviour. Below this value, the prevalence decays exponentially fast and the epidemic dies out and above it, there is always a fraction of nodes which remains infected.

Although $\alpha = 0.31$ is not the exact value for α_c (we should make a Finite Size Scaling to obtain, numerically, a really accurate result), we can see that the theoretical and the numerical results do not exactly coincide. This is due to the approximations we made to develop the critical value analytically. A first

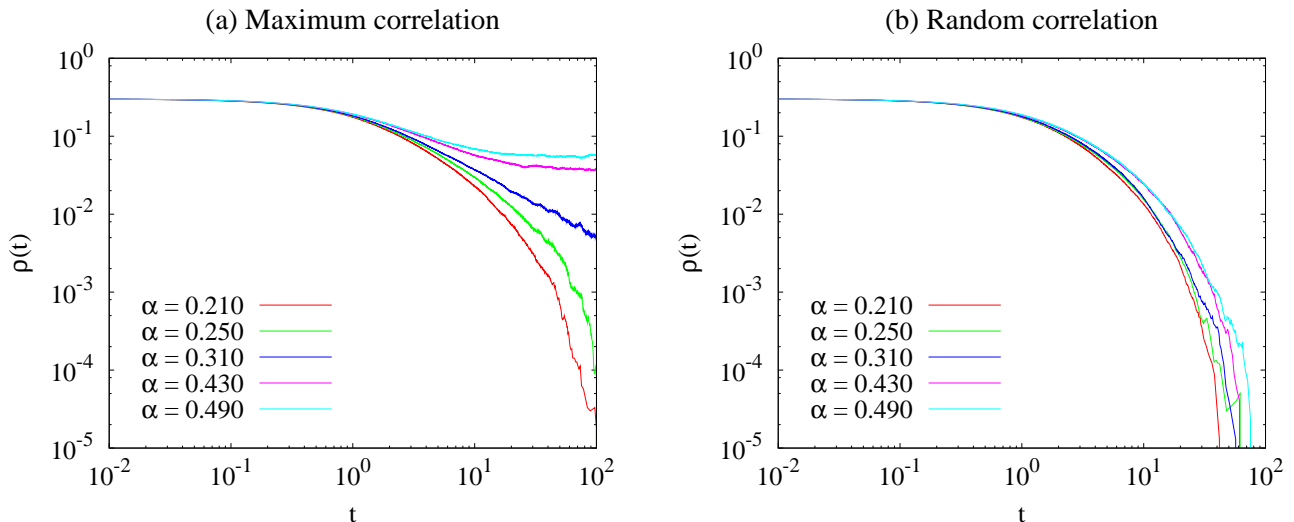


Figure 2: Time evolution of the infected nodes for different values for α when $\Lambda = 0.8$, when (a) local degrees are correlated and when (b) they are rewired randomly. We simulated the networks with 3×10^5 .

improvement we can make is the following:

It can be proved that, for a single network, a better result for the critical infection rate from which the epidemic propagates, instead of Equation 1, is:

$$\Lambda_c \equiv \lambda_c \frac{\langle k(k-1) \rangle}{\langle k \rangle} = 1 \quad (42)$$

By substituting the new value of Λ^{AA} into Equation 37 we could determine the new theoretical value of α_c . Figure 3 shows in a green line the dependence between Λ^{AA} and α_c without this correction. The red line shows that dependence using Equation 42, while blue line shows Equation 37. We can see that the new theoretical approximation gives a better result. We cannot expect to find the same curve for both results: although we have minimized the error for Λ_c , equation 37 is still an approximate solution by itself. Moreover, the numerical results we obtained for α_c are approximated. However, both results are very close to each other and that allowed us to check the expected behaviour of the epidemic spreading.

Finally, we have to point out that this critical threshold from which there is global propagation in the coupled networks is obtained under the condition of complete correlation between internal and external degrees for each node. In fact, if there is not correlation between them, there is not endemic state in the whole range of the studied values of α . To conclude this master thesis, we compared the results above against a null model. We computed the time evolution of the total fraction of infected nodes in the coupled networks without local correlation. We fixed the total number of external edges (for each value of α , we computed the total number of external edges connecting the two networks), we rewired them randomly in order to de-

stroy any correlation between internal and external degrees in each node and let the epidemic evolve. We checked that, even for values of α that were above the critical threshold, the epidemic does not propagate either. Figure 2b shows, for the same values of α and $\Lambda^{AA} = 0.8$, the time evolution of the fraction of infected nodes. We can see how the epidemics dies in the coupled networks, and it is not possible to observe a phase transition.

4 Conclusions and further work

In this master thesis, we have found a new threshold from which an epidemics propagates within the coupling of two random networks, that were below their critical threshold and did not propagate the epidemics by themselves.

By adding edges coupling the two networks under the condition that the correlation between inner and outer degrees of each node is maximum, we could observe that above a critical value of the number of edges connecting the two networks, the epidemics propagated globally.

Although all theoretical results are obtained under the heterogeneous mean field approximation, we compared them with some simulations and observed a good agreement. After using a better estimation for the threshold for isolated networks, we observed a great improvement of the results. On the other hand, the infection is supposed to spread on two infinite networks. However the networks we generated have 3×10^5 nodes, and one can expect finite size effects. We also used other simplifying assumptions, like considering that all infection rates were equal. Different results can be obtained if more realistic assumptions

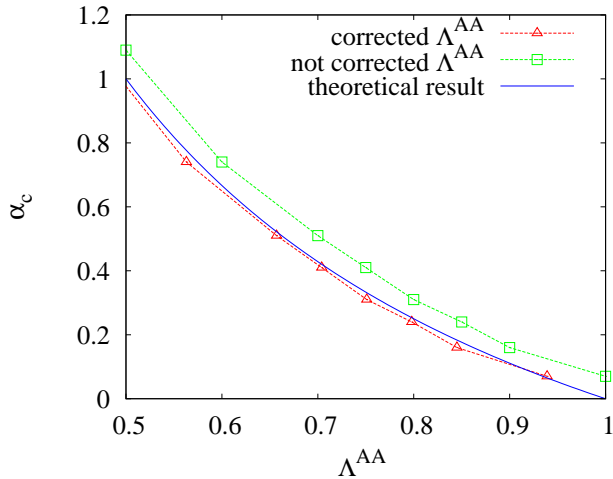


Figure 3: Dependence between Λ^{AA} and α_c . The blue line shows the theoretical equation, the green line shows each value of α_c we obtained for each value of Λ^{AA} without correction and the red line shows α_c once the theoretical critical threshold for a single network is modified for each value of Λ^{AA} .

are taken into account. We keep that for further work in order to complete the model.

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Appendix

The SIS model that we implemented uses an algorithm where time evolves continuously, where there are not two events taking place at the same time and they occur independently of one another. More precisely, they were assumed to follow a Poisson process. As we know, two classes of events were considered: recovery events and infectious events.

Since the simulations were taken in two coupled networks and their recovery rates δ^A and δ^B could take different values, it was needed to distinguish in which one the recovery event occurred. The total number of nodes that could be recovered in the network α (that could be either A or B) is the total number of infected nodes. Hence, the rate that one of these events occur is given by the total number of infected nodes times the rate for a single event:

$$\Omega_r^\alpha = \delta^\alpha \sum_{i \in \alpha} n_i^\alpha(t). \quad (43)$$

For each infectious event two nodes were involved, the node that was infected and the susceptible one that connected it by an edge. Since the rates to infect may depend on the network where they belong to, four different kinds of infectious events were distinguished. The total number of susceptible nodes from network β that can be infected by one of their infected neighbours from network α times the rate to infect, $\lambda^{\alpha\beta}$, gives the total rate this event occurs:

$$\Omega_i^{\alpha\beta} = \lambda^{\alpha\beta} \sum_{i \in \alpha} (1 - n_i^\alpha(t)) \sum_{j \in \beta} n_j^\beta(t) a_{ij}^{\alpha\beta}. \quad (44)$$

The initial system was supposed to have an infected fraction of the total nodes.

In order to compute the time evolution of the system, we created two lists with all infected nodes, one for each network, as well as four lists with all edges connecting an infected node with a node susceptible to be infected. Each list would contain a different combination between the two networks: infected node from

network A connected with a susceptible node from network A , infected node from network A connected with a susceptible node from network B , etc.

Using a random generator program, each event occurred with a certain probability. For example, the probability to recover an infected node from network A was given by:

$$\frac{\Omega_r^A}{\Omega_r^A + \Omega_r^B + \Omega_i^{AA} + \Omega_i^{AB} + \Omega_i^{BB} + \Omega_i^{BA}}, \quad (45)$$

and analogously for the other events. If in a iteration a node from network A was recovered, an infected node was chosen randomly and became in the susceptible state.

If, for example, in a given iteration an infection involving two nodes from network A was required, then, not one node was chosen randomly, but one of the edges connecting an infected node with a non infected one, both from network A . Then, the susceptible node turned to be infected.

At each iteration the list was actualized. It is important to point that, for example, in the case where an infection of a node from network A due to a node from the same network takes place, not only the list of infected nodes from network A and the list of both links of edges belonging to the network A must be actualized, but also the lists of edges connecting infected nodes from network A with susceptible nodes from network B , and the list connecting infected nodes from network B with susceptible nodes from network A . This algorithm needs to be computed more carefully than the continuous time simulations of the SIS model for one single network.

After each iteration, time evolved as:

$$t \rightarrow t + \frac{1}{\Omega_r^A + \Omega_r^B + \Omega_i^{AA} + \Omega_i^{AB} + \Omega_i^{BB} + \Omega_i^{BA}}. \quad (46)$$

After some iterations, process should evolve to the stationary state we were interested in. Our simulations finished when $t = 100$.