The role of bridge nodes between layers on epidemic spreading

L D Valdez\(^{1}\), H H Aragão Rêgo\(^{2}\), H E Stanley\(^{4}\), S Havlin\(^{3}\) and L A Braunstein\(^{4,1}\)

\(^{1}\) Center for Polymer Studies, Boston University, Boston, MA 02215, United States of America
\(^{2}\) Departamento de Física, Instituto Federal de Educação Tecnológica do Maranhão, São Luís, MA, 65030-005, Brazil
\(^{3}\) Department of Physics, Bar-Ilan University, Ramat-Gan 52900, Israel
\(^{4}\) Instituto de Investigaciones Físicas de Mar del Plata (IFIMAR)-Departamento de Física, Facultad de Ciencias Exactas y Naturales, Universidad Nacional de Mar del Plata-CONICET, Funes 3350, (7600) Mar del Plata, Argentina

E-mail: ldvaldez@bu.edu

Keywords: multilayer networks, epidemic modeling, percolation, SIR model

Abstract
Real networks, like the international airport network and the Internet, are composed of interconnected layers (or communities) through a small fraction of nodes that we call here ‘bridge nodes’. These nodes are crucial in the spreading of epidemics because they enable the spread of the disease to the entire system. In this work we study the effect of the bridge nodes on the susceptible-infected-recovered model in a two layer network with a small fraction \( r \) of these nodes. In the dynamical process, we theoretically determine that at criticality and for the limit \( r \to 0 \), the time \( t_\infty \) at which the first bridge node is infected diverges as a power-law with \( r \), while above criticality, it appears a crossover between a logarithmic and a power-law behavior. Additionally, in the steady state at criticality, the fraction of recovered nodes scales with \( r \) as a power-law whose exponent can be understood from the finite size cluster distribution at criticality. We also test our model on the real international airline network and show that ‘high-degree bridge nodes’ reduce the time \( t_\infty \).

1. Introduction
One of the main aspects of the spread of contagious diseases in society, such as the Influenza [1], Ebola [2], and syphilis [3] is that these processes rely not only on the pathogen-specific characteristics but also on the structure of the network of interactions among individuals. Consequently, in the last two decades, many researchers have studied the effect of the structure of complex networks on the spread of epidemics, in order to improve the epidemic forecast and to propose efficient strategies to mitigate their effects on the population [4, 5]. Initially, most researches have focused on isolated networks, but in the recent years, in particular, since 2010 [6] multilayer networks or network-of-networks (NON) attracted much interest, since they are more general and suitable to model more realistic interactions between people. In a NON, the nodes in a network which interact with nodes in other networks are called here ‘bridge nodes’ [7] and the links that connect bridge nodes of different networks are called ‘external links’. On the other hand, within each layer, all the nodes (including the bridge ones) are connected by internal links. One of the most fundamental microscopic measures that characterize the topology or internal structure of a network is the degree distribution \( P(k) \), which is the fraction of nodes with \( k \) internal contacts within the same network [8]. A particular class of NON is the multilayer networks [9, 10] in which a fraction \( r \leq 1 \) of nodes are bridge nodes in each network layer, and each of these nodes are connected or ‘interacts’ with only one bridge node in the other layers [11]. This structure that we call a simple multilayer network, is suitable for example, for modeling the propagation of a rumor in which an individual can transmit the rumor both in a virtual layer (for example Facebook) and in a physical network or layer of face-to-face contact [12]. The individual who participates in both layers is represented by a bridge node in each layer, which is connected to each other through an external link. Several studies showed that simple multilayer networks boost the propagation of information [12], accelerate diffusion processes [13], delay reactions [14] and increase the virulence of diseases with respect to an isolated network [15].
In the last two decades, several mathematical models have been developed using complex networks as a substrate to describe the spread of diseases [4, 16, 17]. One of the most studied models is the Susceptible-Infected-Recovered (SIR) which is suitable for diseases that confer permanent immunity [18]. In this model, initially each node is in the susceptible state (S), that is, it is healthy but not immunized to the disease, except for an infected individual (I), which is called the index case. Each individual in state I can transmit the disease to his/her contacts in state S with probability $\beta$ during a time $t$, since he/she became infected. After that period, the individual $I$ goes to the recovered state (R) and stops transmitting the disease. The epidemics continues spreading until it reaches the steady state in which there are no more infected individuals. Depending on the context, we also refer to $S$, $I$ and $R$ as the fraction of susceptible, infected and recovered individuals, respectively. One of the main features of this model in the steady state, is that it undergoes a second order phase transition between two regimes that are governed by the transmissibility $T$ [19]. This quantity represents the effective probability of contagion, given by $T = 1 - (1 - \beta)^r$ and it is the control parameter of the transition. This transition occurs at a critical value $T = T_c$ which depends on the topology of the system. On the other hand, the order parameter of the system is the fraction of recovered individuals $R$ in the steady state. It is known that in isolated networks as well as in simple multilayer networks, the SIR model in the steady state can be exactly mapped onto link percolation [15, 19, 20]. In this framework, the fraction of recovered nodes $R$ is analogous to the fraction of nodes in the giant component (GC), and $T$ plays the role of the probability $p$ of link occupation. In isolated single networks, there is only one single component or cluster of recovered nodes in the steady state. Similarly, in simple multilayer networks, there is only one cluster of recovered nodes, formed by both internal and external links that are used to transmit the disease [15].

Although the spread of diseases in simple multilayer networks has been extensively investigated in recent years, the effect of bridge nodes with multiple external links has received little attention. On the other hand, in some real multilayer networks, there are usually few bridge nodes in each layer and in turn they have many external connections compared to the average degree in their layer. This kind of structure appears when only few nodes in one layer have sufficient infrastructure, and the necessary economic and human resources to connect with nodes in other layers. For example, in social networks, only a few individuals of one community or layer may have the necessary skills to establish commercial and cultural relationships with individuals from other layers [21]. In another example, in a national and international airport network, most of the airports in a country (layer) only serve national flights, while a small set of airports serve international flights from and to other countries (layers) [22]. One additional feature of these international airports (bridge nodes) is that they may have many external links, i.e. flights to many nodes in other layers since they have the necessary infrastructure to support the air traffic to and from many countries. These bridge nodes with a large number of external links are called ‘central bridge nodes’, which could be considered as influential spreaders or superspreaders [23–25]. Hereafter, for simplicity, we will refer to the ‘central bridge nodes’ as ‘bridge nodes’. This type of structure is more general than the simple multilayer model. Very recently, Dong et al [11] studied a node percolation process on this structure and obtained that the fraction of nodes in the GC behaves as a power law with the fraction of bridge nodes $r$, which is analogous to the relation between the magnetization and the external field in the Ising model.

In this work, we study the SIR model in a two-layered network with a fraction $r$ of central bridge nodes during the dynamic process and in the steady state. In section 2 we present our model and the dynamic equations. In sections 3 and 4 we show the scaling properties of the time $t_0$, at which the disease reaches the first bridge node. In section 5 we focus on the steady state of the epidemic process at criticality, i.e. at the critical point and explain how the structure of the recovered clusters explains the scaling relation between the fraction of recovered nodes and the fraction of bridge nodes. In section 6 we apply our model to real flight networks. Finally in section 7 we present our conclusions.

2. Model and dynamic equations

In this paper we study the SIR model in a system of two layers or networks with the same number of nodes $N$, in which a fraction $r$ of nodes in each layer are ‘bridge nodes’, that is, they have external links which connect to bridge nodes in the other layer. Additionally, we consider that the external connectivity follows a Poisson or Erdős Rényi (ER) distribution with a mean external connectivity equal to $\langle k_{ext} \rangle = c/r$, where $c$ is a constant. This implies that $r \langle k_{ext} \rangle = c$, so the total number of external links $M$ is constant and equal to $cN$. The Poisson distribution in the external connectivity will allow us to study the effect of an increasing mean external

---

Note that the structure of our model with two layers can be interpreted as a network with two communities. Heuristically, a community is a subnetwork in which the density of its internal links is greater than its external links. However, since in our model the number of external links could be comparable to the number of internal links, we prefer to use the term of layers and multilayer networks, instead of network with communities.
connectivity while the number of external links is constant. Unless otherwise indicated, the bridge nodes are chosen randomly and the external links only connect bridge nodes from different layers. The nodes that do not have external links are called ‘internal nodes’. In the main text of this work, we present our results for \( M_1 = N \), but in the appendix the reader can find our results for \( M_1 < N \). For the stochastic simulations of the SIR model, we randomly choose the index case in one layer, which can be also a bridge node.

In order to study the evolution of the states of the individuals in the SIR with fixed recovery time, we use the edge-based compartmental model (EBCM) [26–28]. In single networks, this approach is based on using two generating functions. The first one is the generating function of the node degree distribution \( P(k) \) which is given by \( G_0(x) = \sum_k P(k)x^k \), with \( k_{\text{min}} \leq k \leq k_{\text{max}} \). Here, \( k_{\text{min}} \) and \( k_{\text{max}} \) are the minimum and maximum values of the degree distribution. The second one is the generating function of the degree distribution of the first neighbors of a node, \( P_1(k) \equiv kP(k)/\langle k \rangle \), given by \( G_1(x) = \sum_k kP(k)/\langle k \rangle x^{k-1} \), where \( \langle k \rangle \) is the first moment of \( P(k) \). Here, \( P_1(k) \) is the probability to reach a neighbor of a node with degree \( k \), following a random chosen link. For simplicity, we will assume in this work that both networks have the same degree distribution in order to reduce the number of equations.

The EBCM approach describes the evolution of the fraction of susceptible (\( S(t) \)), infected (\( I(t) \)) and recovered (\( R(t) \)) individuals at time \( t \) by computing an auxiliary probability \( \theta(t) \). Note that this approach is only valid in the deterministic regime of the epidemic spreading, that is, when the fluctuations of the number of infected nodes at time \( t \) are negligible with respect to the mean number of infected nodes. In a single network, \( \theta(t) \equiv \theta \), stands for the probability that a randomly chosen node through a link has not transmitted the disease towards this link. This could be due to the following cases:

(i) the node is susceptible with probability \( \Phi_S(t) \), so it cannot transmit the disease,

(ii) the node is infected, but it has not transmitted the disease yet, with probability \( \Phi_I(t) \),

(iii) the node is recovered and it did not transmit the disease while it was infected, with probability \( \Phi_R(t) \).

Therefore, \( \theta_i \) is given by, \( \theta_i = \Phi_S(t) + \Phi_I(t) + \Phi_R(t) \).

Then, given a node with degree \( k \), the probability that none of its neighbors has transmitted the disease to him/her at time \( t \) is \( \theta_i^k \). If this node is not the index case, i.e. it is not an infected node at the initial condition, then it is in the susceptible state at the beginning of the dynamic process. Since, the fraction of index cases is negligible, then the fraction of susceptible nodes is \( S(t) = \sum_i P(k)\theta_i^k = G_0(\theta_i) \) for all \( t \).

Similarly, in a two layer networks with the same degree distribution, the following relations hold:

\[ \theta_i^k = \Phi_S^k(t) + \Phi_I^k(t) + \Phi_R^k(t) \]
\[ \theta_i^\beta = \Phi_S^\beta(t) + \Phi_I^\beta(t) + \Phi_R^\beta(t) \]

where \( \Phi_S^k \) and \( \Phi_R^\beta \) correspond to the internal and external links or internal and bridge nodes, respectively. Note that the equations do not have an index that indicates the number of the layer because we assume, as mentioned earlier, that both layers have the same degree distribution. Using the EBCM adapted to SIR with fixed \( t_r \), the evolution of \( \theta_i^k \), \( \Phi_S^k(t) \), \( \Phi_I^k(t) \), \( \Phi_R^k(t) \) and \( \Phi_I^\beta(t) \) are given by the deterministic equations

\[ \theta_i^{k+1} = \theta_i^k - \beta \Phi_I^k(t) \]
\[ \theta_i^{\beta+1} = \theta_i^\beta - \beta \Phi_I^\beta(t) \]

\[ \Delta \Phi_S^k = (1 - r)[G^k_i(\theta_i^{k+1}) - G^k_i(\theta_i^k)] + r[G^k_i(\theta_i^{k+1})G^\beta_j(\theta_i^{\beta+1}) - G^k_i(\theta_i^k)G^\beta_j(\theta_i^\beta)] \]

\[ \Delta \Phi_R^k = G^k_i(\theta_i^{k+1})G^\beta_j(\theta_i^{\beta+1}) - G^k_i(\theta_i^k)G^\beta_j(\theta_i^\beta) \]

\[ \Delta \Phi_I^k = -\beta \Phi_I^k(t)^i - \Delta \Phi_S^k + (1 - T) \Delta \Phi_S^k(t - t_r) \]

\[ \Delta \Phi_I^\beta = -\beta \Phi_I^\beta(t)^\beta - \Delta \Phi_S^\beta + (1 - T) \Delta \Phi_S^\beta(t - t_r) \]

where \( \Delta \) is the discrete change of the variables between times \( t \) and \( t + 1 \). Equation (1) computes the change in \( \theta_i^k \) when an infected internal node transmits the disease with probability \( \beta \). Equation (3) represents the change in \( \Phi_S^k \) when an internal node is infected (first term) and a bridge node is infected (second term). Note that \( \Delta \Phi_S^k(t)^i < 0 \). Finally, equation (5) computes the variation of \( \Phi_I^k \) due to: (i) an infected node transmits the disease which decreases \( \Phi_I^k \) (first term), (ii) there are new links that lead to the new infected nodes (second term), and (iii) the infected nodes which have not transmitted the disease have probability \( 1 - T \) during a period \( t_r \) are recovered. Equations (2), (4), and (6) have similar interpretations for the bridge nodes.

Using the equations given above, we can compute the fraction of susceptible and infected nodes as,

\[ \Delta S^i = (1 - r)[G^i_0(\theta_i^{k+1}) - G^i_0(\theta_i^k)] \]
\[ \Delta S^\beta = r[G^\beta_0(\theta_i^{k+1})G^\beta_j(\theta_i^{\beta+1}) - G^\beta_0(\theta_i^k)G^\beta_j(\theta_i^\beta)] \]
\[ \Delta I^i = -\Delta S^i + \Delta S^i(t - t_r) \]
In this section we study the relation between different characteristic times, as seen clearly in the following sections, we will study the dependency of the fraction of infected internal nodes $I_i(t)$ and infected bridge nodes $I_b(t)$ for two layers with a ER distribution with $\langle k \rangle = 4$. $T_i(r = 0) = 0.25$ (this is the critical transmissibility for an isolated network with the same degree distribution), $t_b = 10$ and: (a) $r = 0.20$, (b) $r = 0.05$, and (c) $r = 0.01$. The colored lines correspond to 100 stochastic realizations with $N = 10^5$ for $I_i$ (pink), $I_b$ (light blue) and $I_p$ (orange), and the black lines correspond to the theoretical solutions obtained from equations (1)–(10). In the main plot we shifted the time to $t = 0$ when the fraction of total nodes in the infected state is equal to 0.01, in order to compare the theoretical solution with the simulations [26, 28]. In the insets we show 100 stochastic realizations of $I_i$ (pink) in log-linear scale. In the insets we shifted for each realization, the time to $t = 0$ (indicated by a dashed vertical line) when the first bridge node is infected at time $t_b$ in the simulations.

$$\Delta I_b = -\Delta S^b + \Delta S^i(t - t_b),$$  \hspace{1cm} (10)$$

where $S^i(I_i)$ and $S^b(I_b)$ are the fractions of susceptible (infected) internal and bridges nodes, respectively.

In order to understand the effect of the bridge nodes on the epidemic spreading, in figure 1 we plot the evolution of the fraction of infected internal nodes $I_i$, infected bridge nodes $I_b$, and the total fraction of infected nodes $I = I_i + I_b$, for different values of $r$, at $T_i(r = 0) = \langle k \rangle / \langle (k^2) - \langle k \rangle \rangle$, i.e. at the critical transmissibility value for a single network. Here $\langle k^2 \rangle$ is the second moment of the degree distribution.

From figure 1 we can see that as the fraction of bridge nodes $r$ decreases, a second sharp peak appears in $I$. This sharp peak is caused by the fast spreading of the infection between the bridge nodes because they have a large connectivity $\langle k_{ca} \rangle = 1/r$ and they are connected to each other, so when one bridge node is infected the disease spreads very fast among them. This represents the high vulnerability for disease spreading of international airports during an epidemic spreading. After these nodes are infected, the disease continues spreading to the rest of the network. That is, for very small $r$, bridge nodes and internal nodes are infected at two different characteristic times, as seen clearly in figure 1(c), since the curve $I(t)$ has two peaks. The first one corresponds to the bridge nodes while the second one to the internal nodes.

From the insets of the figures, we also can see that as $r$ decreases, the disease or epidemics ‘explodes’ only after the first bridge node is infected at time $t_b$. This magnitude is also related to the ‘arrival time’ which is the moment at which the first infected node appears in the other community, metapopulation or layer [7, 29]. Since in such networks, the ‘explosion’ of the epidemics is governed by the bridge nodes, it is of interest to compute the average time $t_b$ at which the first bridge node is infected, because after that, the fraction of infected nodes will rise very quickly. Unlike our dynamical equations (1)–(10) for the deterministic evolution of the fraction infected nodes which assume that both layers are of the same size, the computation of $t_b$ can be applied to layers of different sizes, because $t_b$ depends only on the topology of the layer in which the disease originates. In the following sections, we will study the dependency of $t_b$ on $r$ for $T_i = T_c$ and $T > T_c$ (note again that $T_c$ is for a single network with $r = 0$).

3. Time to reach the first bridge node $t_b$ at $T = T_c(r = 0)$

In this section we study the relation between $t_b$ and the fraction of bridge nodes, $r$, when the disease has started in one layer before reaching the other layer. It is important to remark that $t_b$ only depends on the network topology.
theoretical that the relation is a power law:

\[ \frac{\text{the fraction of bridge nodes of the layer in which the disease originates, because when } t \leq t_0 \text{ the disease has not reached the other layer.}}{\text{by partitioning the cluster into bridge nodes is infected, is a decreasing function with the fraction }} \]

\[ \text{and at } T = T_c, \text{ the average time at which the first bridge node is infected, is a decreasing function with the fraction } r, \text{ as expected. Moreover, we obtain theoretically that the relation is a power law: } t_0 \sim r^{-1/2} \text{ for homogeneous networks and } t_0 \sim r^{-(\lambda - 3)/(\lambda - 2)} \text{ for SF networks with degree distribution } P(k) \sim k^{-\lambda} \text{ and } 3 < \lambda < 4 \text{ (see appendix C). In figure 2 we show the simulations and the theoretical solutions of } t_0 \text{ versus } r \text{ at } T = T_c (r = 0) \text{ for these networks, and we obtain that the exponent is consistent with our theoretical predictions.}} \]

and the fraction of bridge nodes of the layer in which the disease originates, because when \( t \leq t_0 \), the disease has not reached the other layer.

Using the branching formalism (see appendix C) we obtain that at \( T = T_c \), the average time at which the first bridge node is infected, is a decreasing function with the fraction \( r \), as expected. Moreover, we obtain theoretically that the relation is a power law: \( t_0 \sim r^{-1/2} \) for homogeneous networks and \( t_0 \sim r^{-(\lambda - 3)/(\lambda - 2)} \) for SF networks with degree distribution \( P(k) \sim k^{-\lambda} \) and \( 3 < \lambda < 4 \) (see appendix C). In figure 2 we show the simulations and the theoretical solutions of \( t_0 \) versus \( r \) at \( T = T_c (r = 0) \) for these networks, and we obtain that the exponent is consistent with our theoretical predictions.

This relation between \( t_0 \) and \( r \) can also be obtained using scaling theory [30]. Considering a Leath [31] or link percolation process at \( T = T_c \), a finite cluster of occupied links leads to the set of recovered nodes in an outbreak which is originated from an index case. The size of this cluster is \( \mathcal{R} \equiv R N \), where \( R \) is the fraction of recovered nodes and \( N \) is the size of the network. The average shortest path between all the nodes in that cluster is denoted by \( \ell' \), which is expected to be proportional to the duration of the outbreak. It is known that at criticality (i.e. at the critical point), \( \mathcal{R} \sim \ell'^d \), where \( d_l \) is the chemical dimension [32, 33]. We are interested in the time \( t_0 \) that takes the disease to reach the first bridge node as a function of \( r \) (assuming that the cluster has at least one bridge node). Let us consider a finite cluster of size \( \mathcal{R} \) with \( n \) bridge nodes, then we expect that if a node of the cluster is the index patient or the source of the epidemic, then the time \( t_0 \) will be proportional to the distance between this index case and the nearest bridge node. The minimum chemical distance to the nearest bridge node can be found by partitioning the cluster into \( n \) subclusters, one for each bridge node (see figure 3(a)). Thus a node \( v \) in the cluster belongs to the subcluster of the bridge node \( u \) when the node \( u \) is the nearest bridge node of \( v \) among all the bridge nodes in the cluster. The average minimum chemical distance in each subcluster \( \ell_{\text{subcluster}} \) behaves as \( \ell' \) since the cluster of infected nodes is a fractal at criticality [33]. Additionally, the average mass of each subcluster is \( \mathcal{R}/n \) and at \( T = T_c \) we expect that (see also, figure 3(b))
the behavior distances to reach a bridge node and it behaves like above the criticality, i.e. the distance between two nodes is small and the system is like at criticality. While for small values of

integrating the equations of time evolution for the stochastic regime for 4. Crossover regimes of

obtain that

where \( \mathcal{R} \) is the average shortest path from a node to the bridge node of this subcluster. Since the probability that a node is a bridge is uniform, the density of bridge nodes in the network is equal to the density of bridge nodes in the cluster of size \( \mathcal{R} \), i.e. \( n/\mathcal{R} \approx r \), which leads to

\[
\frac{\mathcal{R}}{n} \sim \ell_{\text{subcluster}}^{d_1},
\]

where \( \ell_{\text{subcluster}} \) is the average shortest path from a node to the bridge node of this subcluster. Since the probability that a node is a bridge is uniform, the density of bridge nodes in the network is equal to the density of bridge nodes in the cluster of size \( \mathcal{R} \), i.e. \( n/\mathcal{R} \approx r \), which leads to

\[
r^{-1} \sim \ell_{\text{subcluster}}^{d_1}.
\]

On the other hand, we expect that \( \ell_{\text{subcluster}} \sim t_0 \) [34], so finally

\[
t_0 \sim r^{-1/d_1}.
\]

For ER networks \( d_1 = 2 \) and for SF networks with \( 3 < \lambda < 4, d_1 = (\lambda - 2)/(\lambda - 3) \) [35].

4. Crossover regimes of \( t_0 \) for \( T \gtrsim T_c (r = 0) \)

Integrating the equations of time evolution for the stochastic regime for \( T \gtrsim T_c (r = 0) \) (see appendix C) we obtain that \( t_0 \) as a function of \( r \) has two regimes, which are separated by a crossover at \( r = \tilde{r} \) (see figures 4(a) and (b)). For \( r > \tilde{r} \) we obtain that \( t_0 \sim r^{-1/d_1} \) as in the previous section. On the other hand, for \( r < \tilde{r} \), the time \( t_0 \) is a logarithmic function with \( r \). This behavior is expected since for large \( r \), the distance between the bridge nodes is small and the system is like at criticality. While for small values of \( r \), the disease needs to cross longer distances to reach a bridge node and it behaves like above the criticality, i.e. the distance between two nodes is a logarithmic function of \( N \) [36]. Therefore, using similar arguments to the previous section, we can obtain the behavior \( t_0 \sim -\ln(r) \) for \( r < \tilde{r} \). Indeed, from the numerical results we obtain that above criticality

\[
t_0 \sim A(T_c, T_c) + B \ln(r)
\]

for ER networks and SF networks with \( 3 < \lambda < 4 \), i.e. the expected \( r \) dependence. In addition, we get that \( A(T_c, T_c) \) is a function of the distance to the criticality \( T - T_c \), and \( B = (T - T_c)^{-1} \) for both topologies. Therefore, \( t_0 \) behaves as

Figure 4. Time \( t_0 \) as a function of \( r \) obtained from the equations of the appendix C. (a) for an ER network with \( (k) = 4 \) for \( T = 0.250 \, 625 \) (black), \( T = 0.250 \, 78 \) (red), \( T = 0.250 \, 976 \) (green) and \( T = 0.251 \, 22 \) (blue). (b) SF network with \( k_{\text{max}} = 2 \) and \( \lambda = 3.5 \) for \( T = 0.269 \, 295 \) (black), \( T = 0.269 \, 481 \) (red), \( T = 0.269 \, 922 \) (green), and \( T = 0.270 \, 226 \) (blue). The main figures in panels (a) and (b) are in log-log scale for identifying the slope of the logarithmic term in equation (14). The symbols are obtained from a logarithmic fit of the curves in the main plot for small values of \( r \), and the dashed line is a power law fit. Panels (c) and (d) show the same curves as in (a) and (b), respectively, in log-log scale. The dashed lines are a power law fit with exponent \( -0.35 \) for the ER network and \(-0.35 \) for the SF network, which are consistent with the exponent \(-1/d_1 \) predicted in section 3.
since bridge nodes have a high external degree. However, an increase in the fraction of bridge nodes generates a large number of infected nodes in our model with two layers. The steady state of the system is characterized by a power-law behavior in the fraction of infected nodes, as shown in Figure 5.

Figure 5. Collapse of the curves $d_i/dt$ as a function of $t$, (a) ER network with $\langle k \rangle = 4$ for $T = 0.250625$ (black), $T = 0.25078$ (red), $T = 0.250976$ (green) and $T = 0.2512207$ (blue), (b) SF network with $k_{\text{min}} = 2$ and $\lambda = 3.5$ for $T = 0.269325$ (black), $T = 0.269481$ (red), $T = 0.26992$ (green), and $T = 0.270226$ (blue). The dotted line indicates the position of the crossover. The figures are in log-log scale.

The scaling results of equation (14) are supported by the numerical solution of the theory in the appendix C as seen in figure 4.

In the following, we study in more detail the transition between the logarithmic regime and the power-law regime.

In order to avoid to work with the $y$-intercept (i.e. the function $A(T, T_c)$) and the logarithmic function, instead of using the equation (14), we will work with the derivative of $h_b$ with respect to $r$.

Based on equation (14) we propose the following scaling Anzat for $d h_b / d r$,

$$\frac{d h_b}{d r} \sim \begin{cases} \frac{A(T, T_c) - (T - T_c)^{-1} \ln(r)}{r^{-1/d_i}} & \text{if } r \ll r^* \\ \frac{1}{d_i} r^{-(1/d_i + 1)} & \text{if } r > r^* \end{cases}$$  \hspace{1cm} (15)

The crossover is defined by the point $r = r^*$ at which both regimes intersects, and is given by

$$r^* \sim (\Delta T)^{d_i}.\hspace{1cm} (16)$$

In addition, we have that at $r = r^*$,

$$\frac{d h_b}{d r} \bigg|_{r = r^*} \sim \Delta T^{-(d_i + 1)}.\hspace{1cm} (17)$$

Applying to the function $d h_b / d r$ the transformations: $r \rightarrow r/r^*$ and $d h_b / d r \rightarrow d h_b / d r \Delta T^{d_i + 1}$ (where $\Delta T \equiv T - T_c$) we obtain that the curves $d h_b / d r$ collapse (see figure 5). Therefore, equation (15) can be written as

$$\frac{d h_b}{d r} = \Delta T^{-(d_i + 1)} F \left( \frac{r}{r^*} \right),$$  \hspace{1cm} (18)

where $F(x)$ is given by

$$F(x) \sim \begin{cases} x^{-1}; & \text{if } x \ll 1 \\ x^{-(d_i + 1)/d_i}; & \text{if } x \gg 1 \end{cases}$$  \hspace{1cm} (19)

with $x = r/r^*$.

Besides the rich behavior of $h_b$ which corresponds to the beginning of the epidemic, in the next section we will show that in the steady state, the bridge nodes also generates a power-law behavior in the fraction of recovered nodes as a function of $r$, similar to Dong et al [11]. However, unlike [11] which interpreted that power-law as an external field, we will explain this behavior from a geometrical point of view.

5. The steady state

5.1. $T \leq T_c$

In our model with two layers, increasing fraction of bridge nodes generates a large number of infected nodes since bridge nodes have a high external degree. However, an increase in $r$ decreases the external connectivity...
which reduces the disease propagation. Therefore, there exist a nonlinear relation between $R$ and $r$, as can be seen in figure 6. In the appendix A we show that this behavior does not depend on the value of $\langle k_{\text{ext}} \rangle r$. In addition, for $r \to 0$, we observe (see inset of figure 6(b)) that $R$ as a function of $r$ is a power-law with exponent $1/\delta = 1/2$ for $T = T_\tau(r = 0)$ and $1/\delta = 1$ for $T < T_\tau(r = 0)$.

The origin of the exponent at $T = T_\tau(r = 0)$ is due to the particular structure of the cluster of recovered nodes since the infection tree is composed by finite clusters of recovered nodes in each layer, that are connected through bridge nodes (see figure 7(a)). In figure 7(b) we show the probability of recovered clusters $P(s)$ of size $s$ in each layer (obtained from stochastic simulations in a ER network and SF network with $\lambda = 3.5$ at $T = T_\tau(r = 0)$) which follows a power-law distribution with exponent $\tau - 1$, where $\tau = 5/2$ for ER networks and $\tau = 2 + 1/(\lambda - 2) = 2.66$ for SF networks with $\lambda = 3.5$ [37].

In the following, we present a geometric interpretation of our model and show theoretically that the structure of the infection tree explains the exponent of the power-law between $R$ and $r$, using the probability $P(s)$.

In our model it is expected that as $r \to 0$ at $T = T_\tau(r = 0)$, almost every bridge node will be infected since they have a large external connectivity. Each of these nodes will be the seed of the finite outbreak or cluster in each layer. In this case, the infection tree is composed by these finite clusters and hence the fraction of recovered nodes is proportional to the sum of the sizes of these finite clusters in which one node of each cluster is a bridge node. This can be written as

\[
R = P(s = 1)(1 - (1 - r)^3)x + P(s = 2)(1 - (1 - r)^3)x^2 \\
+ P(s = 3)(1 - (1 - r)^3)x^3 + ..., \tag{20}
\]

where $P(s)$ is the probability that a randomly chosen node belongs to a cluster of size $s$ and $(1 - (1 - r)^3)$ is the probability that this cluster has a bridge node (with $x = 1$). We can approximate $R$ as
While above criticality, so with

\[
R = 1 - \sum_{i=1}^{\infty} P(s) (1 - r)^i
\]

(21)

\[
\approx 1 - A \int_1^{\infty} P(s) (1 - r)^i ds
\]

(22)

\[
\approx 1 - A \int_1^{\infty} P(s) \exp(s(1 - r)) ds.
\]

(23)

If \( r \ll 1 \) then \( \exp(s(1 - r)) = (1 - r)^s \approx 1 - rs \), and when \( T < T_c \), it holds that

\[
P(s) \sim s^{-\tau + 1} \exp(-s/s_{\max}) [38], \text{ where } s_{\max} \sim |T - T_c|^{-1/\alpha}. \text{ Thus,}
\]

\[
R \approx 1 - A \int_1^{\infty} (1 - rs)s^{-\tau + 1} \exp(-s/s_{\max}) ds,
\]

(24)

where

\[
A = \frac{1}{\int_1^{\infty} s^{-\tau + 1} \exp(-s/s_{\max}) ds}.
\]

(25)

Since for \( T < T_c \), \( \int_1^{\infty} s^{-\tau + 2} \exp(-s/s_{\max}) ds \) does not diverge when \( r \to 0 \), then we obtain that, \( R \sim r \), i.e.

\[
1/\delta = 1,
\]

(26)

for \( T < T_c \).

On the other hand, for \( T = T_c \), \( s_{\max} \to \infty \); and if \( r \ll 1 \) then \( \ln(1 - r) \approx -r \). In this case \( R \) can be approximated by

\[
\approx 1 - A \int_1^{\infty} s^{-\tau + 1} \exp(-rs) ds,
\]

(27)

with

\[
A = \frac{1}{\int_1^{\infty} s^{-\tau + 1} ds} = -\tau + 2.
\]

(28)

Denoting \( u = rs \), and integrating by part

\[
\approx 1 - \frac{-\tau + 2}{r^{-\tau + 2}} \int_r^{\infty} u^{-\tau + 1} \exp(-u) du
\]

\[
\approx 1 + \frac{-\tau + 2}{r^{-\tau + 2}} \left( \frac{1}{-\tau + 2} - \int_r^{\infty} u^{-\tau + 2} \exp(-u) du \right)
\]

\[
\approx \frac{-\tau + 2}{r^{-\tau + 2}} \int_r^{\infty} u^{-\tau + 2} \exp(-u) du \sim r^{\tau - 2},
\]

(29)

so finally, \( R \sim r^{\tau - 2} \), i.e.

\[
1/\delta = \tau - 2.
\]

(30)

Thus, for \( T = T_c \), the exponent of the scaling relation between \( R \) and \( r \) is related to the Fisher exponent of the finite size cluster distribution. Therefore at criticality, alternatively to the interpretation of the exponent \( 1/\delta \) as the result of an ‘external field’ [11], here we show theoretically that this exponent can be understood from the distribution of finite cluster sizes in one layer. This exponent was also studied in the context of semiconductors and percolation in the presence of a ‘ghost field’ [30, 39, 40].

5.2. \( T > T_c \)

While above criticality, \( R \) does not go to zero when \( r \to 0 \), we obtain theoretically that \( \frac{dR}{dr} |_{r \to 0} \sim (\Delta T)^{-1} \) for ER and SF networks. Here, \( \frac{dR}{dr} |_{r \to 0} \) is related to the divergence of the average size of the finite clusters (see appendix B).

On the other hand, above criticality the contribution of finite clusters to the GC is dependent on two factors. First, note that in equation (27) the contribution of the finite size cluster distribution \( P(s) \) that compose the GC is constrained by an exponential function \( \exp(-s/s_c) = \exp(-rs) \), where \( s_c = 1/r \) is the cutoff imposed by the fraction of bridge nodes. This is due to the fact that during the dynamic process, the finite clusters cannot grow without any constrain because these clusters interfere with each other. Second, for single networks, it is known that above criticality, \( P(s) \sim s^{-\tau} \exp(-s/s_{\max}) \) where \( s_{\max} \approx |T - T_d|^{-1/\alpha} \), i.e. there is another cutoff imposed by the transmissibility. Therefore in a bilayer network for \( T > T_c \), the cutoff of the finite size distribution in one layer can be imposed either by the fraction of bridge nodes or by the transmissibility. When the transmissibility is

\footnote{Note that we do not use the approximation \((1 - r)^s \approx 1 - rs \) because it would lead to a divergent integral.}
very close to $T_c$, or $r$ is not small, then $s_t \ll s_{max}$, i.e. the cutoff imposed by the bridge nodes dominates the finite cluster distribution, and the system behaves as at criticality, since it does not ‘see’ the cutoff imposed by the transmissibility. The opposite occurs when $T$ is well above $T_c$, or $r \ll 1$. Denoting $r^\#$ as the value of the fraction of bridge nodes at which both cutoffs are comparable, then

$$r^\# \sim |T - T_c|^\nu,$$

and using that $\nu = 1/(d\nu)$ [35], we obtain that

$$r^\# \sim (|T - T_c|^\nu)^d,$$

Finally, since for uncorrelated homogeneous networks and SF networks $\nu_l = 1$ [35],

$$r^\# \sim |T - T_c|^d,$$

which is the same relation as in equation (16), i.e. the crossover $r^\#$ of the time $t_b$ between the logarithmic regime and the power law regime. Thus, the crossover of the time $t_b$ scales with $T - T_c$ as the crossover $r^\#$ between the cutoff imposed by the transmissibility and the cutoff imposed by the bridge nodes. Therefore $r^\# \sim r^\$. Moreover, since the correlation length behaves as $\xi \sim |T - T_c|^{-\nu_l}$ (where $\nu_l = 1$ for ER and SF networks, as mentioned above), this result suggests that the coefficient of the logarithmic term in equation (14) is related to the correlation length.

6. Real networks

We next examine how the topology and the selection of bridge nodes (targeted or random) affects $t_b$.

Flight transportation data are widely available through different online sources. In this work, we considered the Flightradar24 flight tracker [41] as the main source of data in order to demonstrate our results on a real world example of a two layer complex network. In this work, we consider our flight networks as being the networks where the airports are represented by nodes and the routes among them by links. According to its own website description, Flightradar24 is a flight tracker that shows live air traffic from around the world. Flightradar24 combines data from several data sources including automatic dependent surveillance-broadcast (ADS-B), Multilateration (MLAT) and radar data. The ADS-B, MLAT and radar data is aggregated together with schedule and flight status data from airlines and airports to create their flight tracker datasets with live online access. For security and privacy reasons information about some aircraft is limited or blocked. This includes most military aircraft and certain high profile aircraft. The site provides information of current and historical flights, such as the destination and origin airports, the flight status (flight codes, arrival and departures times, etc), airlines and aircraft models. For the purpose of this work, we build two different networks. One considering only those flights within the United States, another within Europe, both connected by links represented by the flights among them (between bridge nodes). It was shown that these flights networks typically have a highly connected set of core airports (mainly hubs) and a few connections to and among periphery airports, in a so called core-periphery structure, typical for real-world airline networks [42]. In figure 8 we show both networks and their actual degree distribution $P(k)$.

Since these networks have a finite size, we use the effective critical transmissibility which is obtained by measuring the position of the peak of the second largest cluster size in a link percolation process as a function of the link occupation probability. In figure 9 we plot $t_b$ as a function of $r$ for $T = T_c$ of each network in the main figure and for $T \geq T_c$ in the insets obtained from the simulations. In these subfigures we find that when $T > T_c$ the time $t_b$ behaves as a logarithmic function with $r$. However, for $T = T_c$ we cannot observe a power law behavior as predicted by our theory in section 4 due to finite size effects ($N \sim 10^5$). On the other hand, from the main figures, we can see that the real network with the real bridge nodes has a smaller $t_b$ than expected for randomly chosen bridge nodes. This is due to the fact that the actual bridge nodes have an average degree well above $\langle k \rangle$. Specifically, $\langle k \rangle = 26.5$ for Europe and $\langle k \rangle = 17.9$ for USA, while the average connectivities of the actual bridge nodes are: 77.3 and 90.2, respectively. Since these bridges airports have a higher degree than the average $\langle k \rangle$, then they can be infected more quickly than other airports. To test this effect of the degree of the bridge nodes on $t_b$, we analyze in figure 9 the time $t_b$ as a function $r$, in which the fraction $r$ corresponds to the nodes with the highest degree, called ‘targeted bridge nodes’. Indeed, we obtain that the disease reaches a bridge node earlier that in the case of randomly chosen bridge nodes. Furthermore, the time $t_b$ for the targeted case is in agreement with the case for the actual fraction of bridge nodes (see the green cross in figure 9).

On the other hand, to study the effect of the topology on $t_b$ in real finite networks, we also compute $t_b$ versus $r$ for an infinite synthetic uncorrelated network with the actual degree distribution as a ‘null model’ (i.e. as a baseline to compare with the real network), using the equations of the appendix C. For randomly chosen bridge nodes, we obtain that for small values of $r$ a finite network has a smaller time $t_b$ than an infinite network, which is expected since the loops or cycles in a finite network would tend to decrease the distance between nodes and
hence the time to reach a bridge node. Notably, for larger values of $r$, $t_b$ versus $r$ is almost the same for an infinite uncorrelated network and a finite real networks. This result indicates that in this regime the topology magnitudes like the degree–degree correlation and clustering are less important to predict $t_b$. Indeed, as $r \to 1$, the probability that the first infected node is a bridge, increases. Therefore, the effect of the core-periphery structure, clustering and degree–degree correlation on $t_b$ should not affect the value of $t_b$.

7. Conclusions

Since in real networks, like in the flight network, the bridge nodes are vulnerable and are able to boost the spreading of a disease, it is important to understand their role in the spreading. In this paper, we study the effect
of the bridge nodes on the dynamic spreading in a two layer network. We obtain that at criticality, these nodes are crucial for spreading the disease to the entire global system and their presence induces a double peak on the number of infected individuals during the dynamic which corresponds to the infection of the bridge and non-bridge nodes. Moreover, the fraction of infected nodes increases rapidly after the disease reaches the first bridge node, so the time \( t_0 \) at which the epidemic reaches this node is of great importance to predict the ‘explosion’ of the epidemic. We showed that at criticality in the stochastic regime of the spreading process the time \( t_0 \) behaves as a power law with the fraction of bridge nodes \( r \), with an exponent related to the chemical dimension.

Additionally, above criticality, \( t_0 \) follows a scaling function with two regimes separated by a crossover \( r = r^* \). For \( r < r^* \) \( t_0 \) behaves a logarithmic function while for \( r > r^* \), it follows a power-law function. We showed that this behavior emerges as the result of the ‘competition’ between two scales: one imposed by the transmissibility through the correlation length and the other imposed by the fraction of bridge nodes which constrain the size of a finite infected cluster. On the other hand, in the steady state, we showed that at criticality the fraction of recovered nodes obeys a power law function with \( r \). We find that the origin of the exponent is related to the finite cluster size distribution since the structure of the epidemic cluster is composed by a distribution of finite clusters in each layer which are connected by the bridge nodes. Finally, we applied our model on real flight networks and obtained that a targeted fraction of bridge nodes reduces the time \( t_0 \), and as \( r \) increases, the structure of the network becomes less relevant to predict the value of \( t_0 \).

The model and results presented in this paper could be generalized. For example, our model could be extended to more layers and other epidemic models like the susceptible-infected-susceptible model, in order to evaluate the role of the bridge nodes in more complex structures and different dynamic processes. In addition, different effective control methods could be studied with the goal of increasing the time \( t_0 \) in real-world networks.

**Acknowledgments**

SH thanks the Israel Science Foundation, ONR, Army Research Office (ARO), the Israel Ministry of Science and Technology (MOST) with the Italy Ministry of Foreign Affairs, BSF-NSF, MOST with the Japan Science and Technology Agency, the BIU Center for Research in Applied Cryptography and Cyber Security, and DTRA (Grant no. HDTRA-1-10-1-0014) for financial support. LAB wishes to thank to UNMdP and CONICET (PIP 00443/2014) for financial support. HHAR also acknowledges the financial support from INTERNACIONAL No. 100/2018 PRPGI/IFMA; and UNIVERSAL-01429/16 FAPEMA. Work at Boston University is supported by NSF Grants PHY-1505000, CMMI1125290, and CHE-1213217, and by DTRA Grant HDTRA1-14-1-0017. HES thanks Project 71601112 by National Science Foundation of China for financial support. We thank Dr Gaogao Dong for useful discussions.

**Appendix A. R versus \( r \)**

In figure A1 we show the relation between \( R \) and \( r \) for different number of external links, i.e \( \langle k_{ext} \rangle = c \) (see section 2).

**Appendix B. Scaling of \( dR/dr \) for \( T \gg T_c \)**

In this section we will obtain the scaling relation between \( dR/dr \) and the distance to criticality \( T - T_c \) in the limit of \( r \to 0 \). i.e. \( dR/dr \sim (T - T_c)^{-\gamma} \) where \( \gamma \) is the critical exponent of the mean size of finite clusters. We apply our equations for homogeneous networks and SF networks with \( 3 < \lambda < 4 \). Note that in our calculation the external connectivity distribution always follows a Poisson distribution with \( \langle k_{ext} \rangle \sim 1/r \).

For any value of \( r \), the fraction of nodes that belong to the GC \( R \) is given by

\[
R = 1 - [(1 - r)G^b_0(1 - T\lambda^i_{\infty}) + rG^i_0(1 - T\lambda^i_{\infty})g^b_0(1 - T\lambda^b_{\infty})],
\]

where \( f^i_{\infty} \) and \( f^b_{\infty} \) satisfy the following equations,

\[
f^i_{\infty} = 1 - [(1 - r)G^i_0(1 - T\lambda^i_{\infty}) + rG^i_0(1 - T\lambda^i_{\infty})g^b_0(1 - T\lambda^b_{\infty})],
\]

\[
f^b_{\infty} = 1 - G^b_0(1 - T\lambda^b_{\infty})G^i_0(1 - T\lambda^i_{\infty}).
\]

In the limit of \( r \to 0 \), \( \langle k_{ext} \rangle \to \infty \) and since \( G^b_0(x) = G^i_0(x) = \exp(\langle k_{ext} \rangle (x - 1)) \), then for any value of \( x < 1 \), it is straightforward that \( G^b_0(x) = G^i_0(x) \to 0 \). Therefore in this limit the fraction of nodes in the GC can be approximated by,
Note that equation (B.5) is the same as in a single network with a perturbation \( r \). Additionally in this limit, equation (B.3) reduces to \( f^i_\infty = 1 \), i.e. the probability that an external link leads to a bridge node that belong to the GC is equal to one because these nodes have an increasing number of external links \((k_{\text{ext}}) \to \infty\). Since these equations depend only in variables related to the internal links, for simplicity we will omit the index \( i \).

Taking the derivative of the equations (B.4) and (B.5) with respect to \( r \), we obtain for \( r \to 0 \):

\[
\frac{dR}{dr} \bigg|_{r=0} = G_0(1 - T f^i_\infty) + TG'_0(1 - T f^i_\infty) \frac{df^i_\infty}{dr} \bigg|_{r=0},
\]

\[
\frac{df^i_\infty}{dr} \bigg|_{r=0} = \frac{G_0(1 - T f^i_\infty)}{1 - TG'_0(1 - T f^i_\infty)}.
\]

In the following, we will apply these equations for homogeneous and SF networks with \( 3 < \lambda < 4 \).

### B.1. Homogeneous networks

For the case of homogeneous networks, i.e. with finite third-order moment, we can expand the self-consistent equation (B.5) around \( f^i_\infty = 0 \) up to the quadratic term for \( T \) near \( T_c \), obtaining

\[
f^i_\infty = \frac{2G'_0(1)}{TG'_0(1)}(T - T_c).
\]

Similarly, expanding \( G'_0(1 - T f^i_\infty) \) and \( G'_0(1 - T f^i_\infty) \) in equation (B.7), we obtain

\[
\frac{df^i_\infty}{dr} \bigg|_{r=0} = \frac{1 - TG'_0(1)f^i_\infty}{1 - T(G'_0(1) - TG'_0(1)f^i_\infty)}.
\]

Then, using equation (B.8), the derivative of \( f^i_\infty \) with respect to \( r \) when \( r \to 0 \) behaves as

\[
\frac{df^i_\infty}{dr} \bigg|_{r=0} \approx \frac{1}{G'_0(1)(T - T_c)}.
\]
In order to compute the time it takes the disease to reach a bridge node, we describe the disease spreading with Appendix C. Mean time i.e.

\[ \lim_{T \to T_c} \frac{dT}{dt} = G_0(1 - T f_\infty) + G_0(1 - T f_\infty) \left( 1 - \frac{T}{T_c} f_\infty \right) \frac{df_\infty}{dr} \bigg|_{r=0}, \]  

and replacing equation (B.10) in the last expression, we obtain

\[ \frac{dR}{dr} \bigg|_{r=0} = G_0(1 - T f_\infty) + \frac{T_c}{T - T_c} \frac{G_0'(1)}{G_1(1)}, \]  

where

\[ \gamma = 1 \]  

for homogeneous networks.

**B.2. SF networks with** \( 3 < \lambda < 4 \)

For the case of SF networks with \( 3 < \lambda < 4 \) and using Tauberian theorems [43], the expansion of equation (B.5) around \( f_\infty = 0 \) is

\[ f_\infty = 1 - (1 - G_1(1) T f_\infty + c_1 f_\infty^{2/3}), \]  

where \( c_1 \) is a constant. This expression leads to

\[ f_\infty \sim (T - T_c)^{\lambda^{2/3}}. \]  

Similarly, expanding \( G_1(1 - T f_\infty) \) and \( G_1(1 - T f_\infty) \) in equation (B.7), we obtain

\[ \frac{df_\infty}{dr} \bigg|_{r=0} \approx \frac{1 - G_1(1) T f_\infty + c_1 f_\infty^{2/3}}{1 - T (G_1(1) - c_2 f_\infty^{2/3})}, \]  

where \( c_1 \) and \( c_2 \) are constants. Using that \( T_c = 1/G_1(1) \), and the equation (B.15), the last equation can be approximated by,

\[ \frac{df_\infty}{dr} \bigg|_{r=0} \approx \frac{1 - G_1(1) T c_3 (T - T_c)^{\lambda^{2/3}} + c_4 (T - T_c)^{\lambda^{2/3}/(\lambda - 3)}}{c_5 (T - T_c)} \]  

where \( c_3, c_4, c_5 \) are constants.

Since \( \lambda > 3 \) then for \( T \to T_c \), in the numerator of the last equation only the first term remains, and hence

\[ \frac{df_\infty}{dr} \bigg|_{r=0} \approx \frac{1}{c_5 (T - T_c)} \sim (T - T_c)^{-1}. \]  

On the other hand, applying Tauberian theorems [43], and inserting equations (B.15) and (B.18) into equation (B.6), leads to the following relation

\[ \frac{dR}{dr} \bigg|_{r=0} \approx G_0(1 - T f_\infty) + (G_0'(1) - G_0''(1) T f_\infty) \]  

\[ + c_6 (T - T_c)^{\lambda^{2/3}/(\lambda - 3)} \frac{T}{T - T_c}. \]  

In the limit \( T \to T_c \), the first term is finite while the second one diverges, hence the first term can be disregarded. In turn, the numerator of the second term is also finite, which finally leads to

\[ \frac{dR}{dr} \bigg|_{r=0} \sim (T - T_c)^{-1}. \]  

i.e. \( \gamma = 1 \).

In figure B1 we show \( dR/dr \) as a function of \( |T - T_c| \) for an ER network and a SF network with \( \lambda = 3.5 \), in which we can observe that the exponent obtained is consistent with the predicted value.

**Appendix C. Mean time \( t_b \) to reach a bridge node**

In order to compute the time it takes the disease to reach a bridge node, we describe the disease spreading with \( t_r = 1 \) as a branching process.
Let us consider an infected individual in generation \( n \), then the generating function of the probability to not reach a bridge node in the following generation through a link is given by

\[
G_{1b}(x) \equiv 1 - T + T(1 - r)G_1(x),
\]

where \( T \) is the transmissibility and \( G_1(x) \) is the generating function of the excess degree distribution. Similarly the generating function of the probability to not reach a bridge node in the following \( n \)-generations through a link is given by

\[
g_n(x) \equiv G_{1b}(G_{1b}(...G_{1b}(x)(x))),
\]

for \( n \geq 1 \), and \( g_0(x) = x \) for \( n = 0 \). On the other hand, the generating function of the probability to not reach a bridge node in the following \( n \)-generations from the index case is \( G_0(g_0(x)) \), where \( G_0(x) \) is the generating function of the degree distribution. Therefore, the probability that in generation \( n + 1 \) the disease reaches a bridge node is,

\[
P(t = n + 1) = (1 - r)[G_0(g_n(1)) - G_0(g_n(1 - Tr))]
\]

\[
= (1 - r)[G_0(g_{n+1}(1)) - G_0(g_{n+1}(1))],
\]

where \( G_0(g_n(1)) \) is the probability that the disease reaches the generation \( n + 1 \), i.e. is the probability that there is an infected node in generation \( n + 1 \), and \( G_0(g_n(1 - Tr)) \) is the probability to not reach a bridge node in generation \( n + 1 \). For \( n = 0 \) we set \( P(t = 0) = r \).

Since, we only consider those realizations in which one of the infected node is a bridge, we normalize the probability \( P(t = n) \):

\[
Q(t = n) \equiv \frac{P(t = n)}{\sum_{i=0}^n P(t = i)}.
\]

Finally, the average time \( t_b \) to reach a bridge node is given by

\[
t_b = \sum_{n=0}^{\infty} nQ(t = n).
\]

### C.1. Scaling relation between \( t_b \) and \( r \) at \( T = T_c \)

In the following we will study the asymptotic behavior of \( P(t = n) \) for large values of \( n \) and \( r \ll 1 \) at \( T = T_c \).

Since \( g_n \) is the recursive iteration of the function \( G_{1b}(x) \), the value of \( g_n \) tends to the solution of

\[
x = G_{1b}(x),
\]

as in a fixed point iteration process. We denote as \( g_\infty \) the solution of this equation. Therefore, for large values of \( n \) we can approximate equation (C.3) to

\[
P(t = n + 1) \approx G_0(g_\infty)(g_n - g_{n+1}).
\]

Since \( g_n = G_{1b}(g_{n-1}) \), \( g_{n+1} = G_{1b}(g_n) \) and \( g_n \approx g_{n+1} \approx g_\infty \) we can approximate the last equation to

\[
P(t = n + 1) \approx G_0(g_\infty)G_{1b}(g_\infty)(g_n - g_{n+1}).
\]

If we use the approximation \( g_{n-1} - g_n \approx G_{1b}(g_\infty)(g_{n-2} - g_{n-1}) \) then equation (C.8), can be rewritten as

\[
P(t = n + 1) \approx G_0(g_\infty)G_{1b}(g_\infty)G_{1b}(g_\infty)(g_{n-2} - g_{n-1}).
\]
and using equation (C.8) at $t = n - 1$ we obtain that
\[
P(t = n + 1) \approx G(t(n_{G}))P(t = n).
\] (C.10)

This relation shows that for large values of $n$, the distribution $P(t)$ corresponds to an exponential distribution with a mean time value of $b_0 = 1/(\ln G(t(n_{G})))).$. Since for $r \to 0$ and $T = T_c$, we have that \(g_\infty \to 1\) and $G(t(n_{G})) \to 1$, then $\ln(G(t(n_{G})) \approx 1 - G(t(n_{G}))$, i.e.
\[
t_b \approx 1 \over 1 - G(t(n_{G})).
\] (C.11)

In the following we will explore the relation between $G(t(n_{G}))$ and $r$ for homogeneous networks, i.e. with finite third order moment in the degree distribution, and SF networks with $3 < \lambda < 4$.

C.1.1. Homogeneous networks. As $r \to 0$, the solution $g_\infty$ tends to the value $g_\infty = 1$. Therefore, expanding equation (C.6) around this value we have that
\[
g_\infty = 1 - T_c + T_c (1 - r) \left( 1 + G(t(n_{G})) - 1 \right) + \frac{1}{2} G(t(n_{G})) (g_\infty - 1)^2,
\]
\[
= 1 - T_c + T_c (1 - r) + (1 - r)(g_\infty - 1)
\]
\[
+ T_c (1 - r) \frac{1}{2} G(t(n_{G})) (g_\infty - 1)^2,
\] (C.12)

in which the physical solution of this equation is
\[
g_\infty = 1 + \frac{r - r_1/2 \sqrt{2 + 2(1 - r) T_c^2 G(t(n_{G}))}}{T_c (1 - r) G(t(n_{G}))}.
\] (C.13)

In the limit of $r \to 0$ we have $g_\infty$ behaves as
\[
g_\infty = 1 - \sqrt{\frac{2}{G(t(n_{G}))}} r_1/2.
\] (C.14)

Since $g_\infty \approx 1$, we can approximate $G(t(n_{G}))$ by
\[
G(t(n_{G})) \approx \frac{T_c (1 - r)}{G(t(n_{G}))} (1 - G(t(n_{G})) + G(t(n_{G})) (g_\infty - 1) + \cdots),
\]
\[
\approx 1 - T_c \sqrt{2G(t(n_{G}))} r_1/2.
\] (C.15)

Therefore, according to equation (C.11) the mean time $t_b$ scales with $r$ as,
\[
t_b \sim r_1/2,
\] (C.16)

for homogeneous networks, i.e. with a finite third order moment of the degree distribution.

C.1.2. SF networks with $3 < \lambda < 4$. Similarly, for SF networks with $3 < \lambda < 4$ and using Tauberian theorems [43], the expansion of equation (C.11) is given by
\[
g_\infty = 1 - T_c + T_c (1 - r) \left( 1 - G(t(n_{G})) (1 - g_\infty) + c_0 (1 - g_\infty)^{1/3 - 2},
\] (C.17)

where $c_0$ is a constant.

In the limit $r \to 0$, $g_\infty \to 1$, and hence $(1 - g_\infty) \ll T_c$. Therefore, the solution of equation (C.17) is
\[
g_\infty \approx 1 - c_1 r_1/3,
\] (C.18)

where $c_1$ is a constant.

In order to find the value of $t_b$, we have to insert this solution in equation (C.11). Note that for SF networks with $3 < \lambda < 4$ the following relation holds,
\[
G(t(n_{G})) \approx G(t(n_{G})) - c_2 (1 - x)^{1/3 - 3}.
\] (C.19)

Therefore, the expansion of $G(t(n_{G}))$ around $x = 1$ is
\[
G(t(n_{G})) \approx T_c (1 - r) (G(t(n_{G})) - c_2 (1 - x)^{1/3 - 3}),
\]
\[
\approx (1 - r) - c_2 (1 - x)^{1/3 - 3},
\] (C.20)

where $c_2$ and $c_3$ are constants. For $x = g_\infty, r \ll 1$ and using the equation (C.18) we obtain,
\[
G(t(n_{G})) \sim 1 - c_4 r_1/3.
\] (C.22)
Thus, the mean time $t_h$ for SF networks with $3 < \lambda < 4$ is

$$t_h \sim r^{-\frac{1}{\lambda-2}}.$$  \hfill (C.23)

References

[27] Miller J C 2018 A primer on the use of probability generating functions in infectious disease modeling Infect. Dis. Mod. 3 192–248
[34] Buono C, Lagorio C, Macri P A and Braunstein L A 2012 Crossover from weak to strong disorder regime in the duration of epidemics Physica A 391 481–5
[38] Stauffer D and Aharony A 2014 Introduction to Percolation Theory 2nd edn (Boca Raton, FL: CRC Press)