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Crossover from weak to strong disorder regime in the duration of epidemics

C. Buono [a,*], C. Lagorio [a], P.A. Macri [a], L.A. Braunstein [a,b]

[a] 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

[b] Center for Polymer Studies, Physics Department, Boston University, Boston, MA 02215, USA

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ABSTRACT

We study the susceptible–infected–recovered (SIR) model in complex networks, considering that not all individuals in the population interact in the same way. This heterogeneity between contacts is modeled by a continuous disorder. In our model, the disorder represents the contact time or the closeness between individuals. We find that the duration time of an epidemic has a crossover with the system size, from a power-law regime to a logarithmic regime depending on the transmissibility related to the strength of the disorder. Using percolation theory, we find that the duration of the epidemic scales as the average length of the branches of the infection. Our theoretical findings, supported by simulations, explains the crossover between the two regimes.

1. Introduction

Complex networks have became a topic of interest among scientists in recent years, due to the fact that many real systems such as protein interaction, the Internet, communication systems [1–3], among others, can be properly described by complex networks, making this theoretical framework inherently interdisciplinary. On complex networks, nodes can represent the individuals of a population in the case of social networks, computers in communication systems, and so on, and the links represent the interactions. The research on networks goes from the study of its topology to the study of transport processes that use the networks as the underlying substrate on which to propagate. In particular, many researchers have focused on the propagation of seasonal diseases on social networks due to the appearance of new infections such as SARS, Avian Flu, and the recent A(H1N1) influenza epidemic.

Several models have been developed to characterize the spreading of these kinds of disease. One of the most used models is the susceptible–infected–recovered (SIR) model, first introduced by Kermack and McKendrick [4] in the full mixing approximation and then extended to complex networks [5,6]. In this model, the individuals of the population can be in three different states: S (susceptible), I (infected), or R (recovered or removed). An S individual becomes infected with a probability β by contact with an infected individual. Infected individuals recover after t_r time steps since they were infected, and cannot infect or change their state thereafter. The system reaches the steady state when all the infected individuals recover. In this model, the size of the disease, defined as the number of recovered individuals, depends only on the effective probability of transmission between individuals, given by

$$T = 1 - \int (1-\beta)^{t_r} P(\beta)\, \mathrm{d}\beta, \tag{1}$$

where $P(\beta)$ is the density distribution of β, with $P(\beta) = \delta(\beta - \beta_0)$ for a constant probability of infection.

* Corresponding author. Tel.: +54 2234945669.
E-mail addresses: cbuono@mdp.edu.ar, camilabuono@yahoo.com.ar (C. Buono).

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It has been shown that the steady state of the SIR model on static complex networks can be mapped into a link percolation problem [6,7] in which the order parameter of the SIR is the fraction of recovered individuals [8], and the control parameter is the transmissibility T, which plays the role of the fraction of links p in percolation. Thus there exists a critical threshold T_c (or p_c) in the SIR (or percolation) model above which a nonzero fraction of individuals (size of biggest cluster) are infected (is finite). It was shown that, in finite systems, the critical threshold $T_c \equiv p_c$ of link percolation depends strongly on the network size N with $T_c(N) - T_c(N \to \infty) \sim N^{-1/3}$ in the mean field (MF) approximation; i.e., finite size effects are strong [9]. Then, in finite systems, for $T > T_c(N)$, the disease becomes an epidemic, while for $T < T_c(N)$ the disease reaches a small fraction of the population (outbreaks) [6,10–12].

Usually, the study of disease transmission or any type of transport process such as information flow [13] or rumor spreading [14] through a network is made assuming that all the contacts are equivalent. However, this assumption does not give a very realistic description of real networks, such as social networks [15], where not all individuals in a society have the same interaction. A way to improve the description of real networks is to consider the heterogeneity of the social contacts. This can be done by considering weighted (disordered) networks, where the weights in the contacts could represent the closeness or the contact time between the individuals [16–18]. The contact time is a parameter that can be controlled by health policies as a strategy to mitigate the duration of the spreading of the disease. Using different strategies such as broadcasting, brochures, etc., the public health agencies can induce people to change their contact time or the closeness of any contact, for example, encouraging people to reduce their contact time. This strategy, which is a social distancing, was used by some governments in the recent wave of the A(H1N1) influenza epidemic in 2009 [19]. It is known that disorder can dramatically alter some topological properties of networks such as the average length of the optimal paths [16,20–22]. In the optimal path problem, defined as the path between any two nodes that minimizes the total weight along the path [16,20,21], it was shown that the average length of the optimal path l_{opt} scales as $N^{\nu_{opt}}$ in the strong disorder (SD) limit, where $\nu_{opt} = 1/3$ for homogeneous networks (MF), and as $l_{opt} \sim \ln N$ in the weak disorder (WD) regime, where the SD limit is related to percolation at criticality [16,20,23]. However, the exact mapping between the order parameter of both second-order phase transitions (percolation and SIR) is not affected by the disorder when the disorder is not correlated [6,11]. Nevertheless, the disorder could affect the duration of a disease.

In this paper, we introduce disorder in the links and show how a broad disorder affects the duration of an epidemic in the SIR model.

Using theoretical arguments, supported by intensive simulations, we find that the average time of the duration of the epidemic t_f goes as the average length of the branches l_b of the infection. Thus, relating l_b with the optimal path problem, we find that t_f has a crossover from the WD regime to the SD regime with the same exponent as in the optimal path problem [21].

The paper is organized as follows. In Section 2, we present our model, and in Section 3 we show theoretically, and by simulation, the crossover for the duration time of the disease from the WD regime to the SD regime. Finally, in Section 4, we present our conclusions.

2. SIR model with disorder

In our model, we assign to each link i between any two nodes a random number β_i drawn from the distribution

$$P(\beta_i) = \frac{1}{a\beta_i}, \tag{2}$$

where a is the parameter that controls the broadness of the distribution of link weights, i.e., the strength of the disorder, and β_i is defined in the interval $[\,\mathrm{e}^{-a}, 1]$. With this distribution, we randomly assign a weight to each link of the network, of the form

$$\beta_i = \mathrm{e}^{-a\, r_i}, \tag{3}$$

where r_i is a random number taken from an uniform distribution $r_i \epsilon [0, 1]$. Thus β_i is the probability of infection between any pair of nodes. This type of weight has been widely used [20,23,22,24] because it is a well-known example of many distributions that produce WD and SD crossover [25]. In a disordered medium, the SD limit can be thought of as a potential barrier ϵ_i such that τ_i is the time to cross this barrier in a thermal activation process; then $\tau_i = \mathrm{e}^{\epsilon_i/k\hat{T}}$, where k is the Boltzmann constant and $\hat{T}$ is the absolute temperature. Therefore, we can associate β_i as the inverse of the time needed to cross this barrier, τ_i.

With this weight distribution, the transmissibility T (see Eq. (1)) is given by

$$T = 1 - \int_{\mathrm{e}^{-a}}^{1} \frac{(1-\beta)^{t_r}}{a\beta}\, \mathrm{d}\beta. \tag{4}$$

In our initial stage, all the individuals are in the susceptible state. We choose a node at random from the biggest connected cluster, or giant component (GC) and infect it (patient zero); then the process follows the rules of the SIR model but in a weighted network. After the system reaches the steady state, we compute the duration of the infection t^*, defined as the time at which the last infected individual recovers, and the length of the branches of the infection as a function of t^*. As the substrate for disease spreading we use only an Erdös–Rényi network (ER) [26], characterized by a Poisson degree distribution

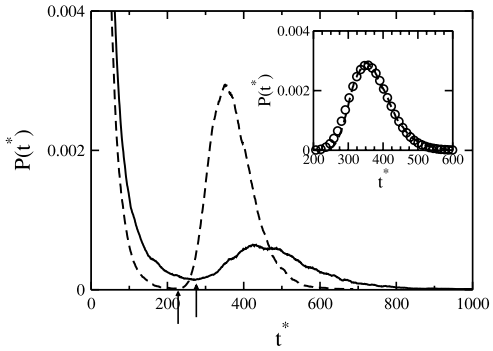

Fig. 1. $P(t^*)$ as a function of t^* for an ER network with $\langle k\rangle = 2$, $N = 2^{14}$, $t_r = 19$, $a = 4.36$ (full line) and $a = 3.24$ (dashed line). The arrows indicate the separation between outbreaks (left) and epidemics (right). In the inset, we show in symbols the fitting with Eq. (5) of the data for $a = 3.24$ (see main plot), from which we obtain $t_f \simeq 363.8$. All simulations were done over 10^5 realizations.

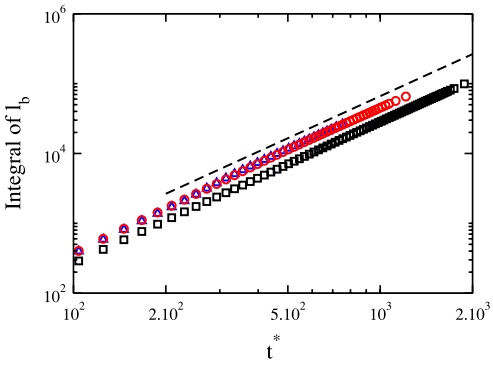

Fig. 2. Log–log plot of the integral of l_b as a function of t^*, for an ER network with $\langle k\rangle = 2$, $N = 2^{14}$ and $t_r = 19$. For the undisordered problem $T = T_c$ (□), and for the disordered problem with $T = T_c$ and $a = 6.55$ (◯), and $T > T_c$ and $a = 3.97$ (△). The dashed line is used as a guide to show the slope 2. All simulations were done over 10^5 realizations. (Color online).

$P(k) = \mathrm{e}^{\langle k\rangle}\langle k\rangle^k k!$, where k is the connectivity and $\langle k\rangle$ is the average degree. However, the results obtained are similar for all networks with a finite value of T_c in the MF approach.

3. Crossover from WD regime to SD regime in the duration of epidemics

We only consider those propagations that lead to epidemic states, and disregarded the outbreaks (see Fig. 1). Fig. 1 shows $P(t^*)$ as a function of t^*; the arrows show the separation between the outbreak and epidemic regimes. The criterion used to distinguish between outbreaks and epidemics is the observation of the behavior of the cluster size distribution, which decays as a power law close to T_c for the outbreaks and has a maximum for the epidemics. The same criterion is used in percolation to distinguish the finite cluster sizes from the giant component with size $\sim N$. We find that $P(t^*)$ in the epidemic regime can be well represented by a log-normal distribution,

$$P(t^*) = \frac{1}{t^*\sigma\sqrt{2\pi}}\,\mathrm{e}^{-\frac{\ln(t^*/t_f)^2}{2\sigma^2}}, \tag{5}$$

where t_f and σ are the average and the standard deviation of the distribution of t^*. Log-normal distributions have been observed in several phenomena, such as the size of crushed ore [27], fragmentation of glass [28], income distribution [29], events in medical histories [30], and food fragmentation by human mastication [31]. By fitting our data with a log-normal distribution, we obtain t_f (see the inset of Fig. 1).

From Fig. 1, we can see that t_f increases with a for a fixed value of t_r. This behavior can be understood if we take into account that, when a increases, the barrier that the disease needs to overcome in order to propagate is bigger. Therefore, even though the transmissibility decreases as a increases for fixed t_r, the epidemic will last longer due to the disorder, allowing the health services to make earlier interventions.

We also study the average length of the branches of the infection, l_b. The length of a branch is defined as the number of links between patient zero and the last patient in that branch. In Fig. 2, we plot the integral of l_b as a function of t^* in log–log scale, from which it is easy to see that, independent of the disorder, in the epidemic region the integral of l_b has slope 2; thus $l_b \sim t^*$. If we compare l_b between the disordered and the undisordered substrates, we can see that, in the disordered

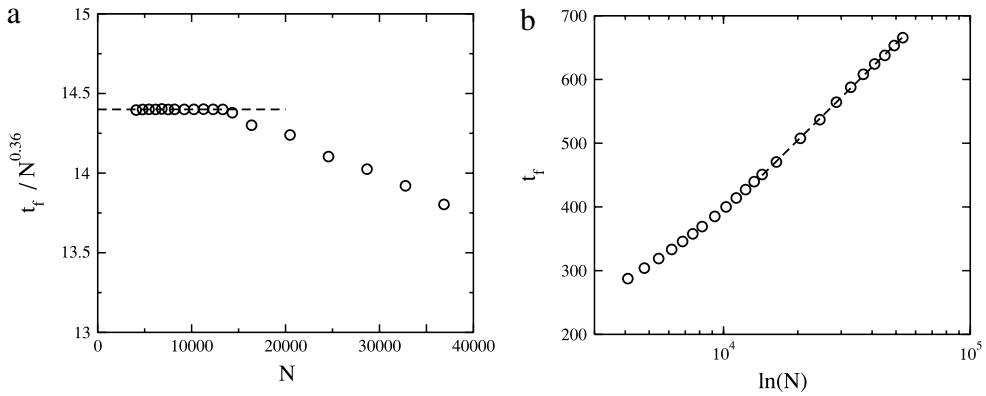

Fig. 3. Results for an ER network with $\langle k\rangle = 2$, $t_r = 19$ and $T = T_c(N = 2^{12})$. (a) Linear–linear plot of $t_f/N^{0.36}$ as a function of N; the dashed line is used as a guide to show that the exponent 0.36 is correct. (b) Linear–log plot of t_f as a function of $\ln(N)$; the dashed line correspond to a logarithmic fit. Notice that, in the WD regime, $T_c(N = 2^{12}) > T_c(N > 2^{12})$. All simulations were done over 10^5 realizations.

case, the lengths of the branches are larger than in the undisordered case, even for the same values of T and t_r. This is due to the fact that the link of the smallest crossing probability e^{-a} that the disease has to traverse is smaller than the required value of β for the same values of T and t_r in the undisordered case (see Eq. (1)).

For the SIR problem, at the threshold T_c in the MF approximation, the fraction of recovered individuals R grows with time t^* as $R \sim t^{*2}$ [32] up to t_f, then, at t_f, $R \sim t_f^2$, and, for $t^* > t_f$, $R \approx const.$, due to finite size effects. Percolation in complex networks predicts that, at criticality, $T \approx T_c$, the size R of the epidemic scales with the chemical length l, as shown in [33].

$$R \sim \ell^{d_l} \sim t_f^2, \tag{6}$$

with $d_l = 2$ in the MF approximation,

$$t_f \sim \ell, \tag{7}$$

where $\ell \sim N^{1/3}$. For the WD regime,

$$R \sim \begin{cases} e^{t^*} & \text{for } t^* \lesssim t_f; \\ const. & \text{for } t^* > t_f. \end{cases} \tag{8}$$

Then, at t_f,

$$R \sim \exp(t_f). \tag{9}$$

Using the fact that, in the WD regime, $R \sim N$ and $\ell \sim \ln N$, we obtain that also in this regime

$$t_f \sim \ell. \tag{10}$$

Thus, from the theoretical arguments presented above,

$$t_f \sim \begin{cases} N^{1/3} & \text{if } T \simeq T_c \\ \ln(N) & \text{if } T > T_c. \end{cases} \tag{11}$$

In order to corroborate Eq. (11), we compute t_f as a function of N for fixed values of $T = T_c(N = 2^{12})$, a, and t_r, and found that, for $T \simeq T_c(N)$, t_f behaves as a power law with exponent 0.36, compatible with the one found for the SD regime in the optimal path problem. The slight difference between the exponent obtained and the theoretical one $(1/3)$ is due to finite size effects [9]. Notice that the SD power-law regime holds only until $N \simeq 15\,000$. In Fig. 3(a), we plot the ratio $t_f/N^{0.36}$ as a function of N. We can see that, for values of N for which $T \simeq T_c(N)$, this ratio goes to a constant, in agreement with our prediction. However, for $T > T_c(N)$, we can see from Fig. 3(b) that t_f behaves logarithmically with N, in agreement with the WD regime $T > T_c$ of Eq. (11). Thus, we can see clearly that there is a crossover from WD to SD, in full agreement with our theoretical prediction.

4. Conclusions

In this paper, we study the SIR model on a broad disordered network, where the disorder represents the duration of the interactions between the individuals of a population, and the broadness of the disorder is a control parameter.

We found that, as the broadness of the disorder increases, the spreading of the disease is delayed. Thus, recommending people to decrease the duration of their contacts as a social distancing could be a good strategy to delay the spreading of an

epidemic. Moreover, this social distancing strategy is more suitable than a quarantine, where contacts are broken, due to the fact that it is less expensive from an economic point of view.

Using percolation arguments, we found that the duration time of the epidemic goes as the average length of the branches of the infection. Our theoretical results are strongly supported by simulations. Thus, in the same way as in the optimal path problem, the duration of the disease has a crossover with the system size, from a power-law regime (SD) to a logarithmic regime (WD).

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