

Intermittent social distancing strategy for epidemic control

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We study the critical effect of an intermittent social distancing strategy on the propagation of epidemics in adaptive complex networks. We characterize the effect of our strategy in the framework of the susceptible-infected-recovered model. In our model, based on local information, a susceptible individual interrupts the contact with an infected individual with a probability σ and restores it after a fixed time t_b . We find that, depending on the network topology, in our social distancing strategy there exists a cutoff threshold σ_c beyond which the epidemic phase disappears. Our results are supported by a theoretical framework and extensive simulations of the model. Furthermore we show that this strategy is very efficient because it leads to a “susceptible herd behavior” that protects a large fraction of susceptible individuals. We explain our results using percolation arguments.

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I. INTRODUCTION

The study of the topology of complex networks, and the dynamical processes that use these networks as substrate to spread, has recently generated great interest in the scientific community [1–3]. In the past, studies of dynamic processes such as the spreading of rumors, opinions, and diseases on static networks were concentrated on how their topology affects these processes [1–3]. However, it is known that these processes evolve on top of networks where the topology changes with time [4]. As a consequence, recently many researchers began to study dynamic networks and the interplay between the dynamic process and the network dynamics. Those networks in which the topology changes regardless of the process taking place on top of them are called evolvable networks, while those networks that change their topology to mitigate or promote these processes are called adaptive networks [4,5]. Adaptive networks have been investigated in many disciplines such as social sciences, epidemiology, biology, etc. [4,6,7]. In these networks there is a coevolution between the link dynamics and the state of the nodes which leads to a collective phenomenon on adapting networks. As an example, in a network of routes, where the nodes are cities and the links are the routes connecting them, some overloaded paths become dysfunctional and new paths between cities are built to avoid the congestion. In the analysis of opinion formation on social networks, nodes usually tend to rewire or break their links with individuals with different opinions, leading in some models to a network fragmentation into components or clusters in which all members have the same opinion [7–9]. In biological networks, such as the vascular system, after an arterial occlusion collateral vessels grow in order to increase the blood flow to neighboring tissues [10]. Similarly, in the widely studied epidemic models on static networks, adaptive processes are used to model strategies that reduce the impact of the disease spreading [5,11,12].

One of the most popular models in epidemiology that reproduces seasonal diseases is the susceptible-infected-recovered model (SIR) [1,13], where individuals can be in one of three states, susceptible (S), infected (I), or recovered (R). In its classical formulation, an infected node infects a susceptible

neighbor with probability β and recovers with a certain fixed probability, which implies an exponential distribution of times for which individuals remain infected. However, this distribution is rarely realistic and for most seasonal diseases it has a sharp peak around an average value [14]. As a consequence, some studies [15–17] have used a different version of the SIR model in which an infected individual recovers after a fixed time t_r , called the recovery time.

It is well known that in the SIR model on static networks, the size of the infection is governed by the effective probability of infection or transmissibility T of the disease, where $T = 1 - (1 - \beta)^{t_r}$. In turn, it was shown that this model can be mapped into a link percolation process [16,18], where T plays the role of the link occupancy probability p in percolation. In a percolation process, there is a critical probability p_c where the finite cluster size distribution n_s behaves as $n_s \sim s^{-\tau}$ in the thermodynamic limit. Above this threshold a “giant component” appears. As a consequence of the mapping between percolation and the SIR model, in the latter there is an epidemic threshold at $T_c = p_c$ below which the disease is an outbreak where the infection reaches a small fraction of the population, which is equivalent to having finite clusters in percolation, while above T_c an epidemic develops corresponding to the emergence of a percolating giant component [19,20]. This threshold, in uncorrelated static networks, depends only on the degree distribution $P(k)$, where k is the degree or the number of links that a node can have. In particular, for Erdős-Rényi (ER) networks, $P(k) = e^{-\langle k \rangle} \langle k \rangle^k / k!$ where $\langle k \rangle$ is the mean connectivity, the threshold is $T_c = 1/\langle k \rangle$. However, in pure scale-free (SF) networks $P(k) \sim k^{-\lambda}$, where λ is the broadness of the distribution, in the thermodynamic limit $T_c \rightarrow 0$ for $\lambda < 3$, which means that the epidemic spreads for any value of T . This last result indicates that highly heterogeneous networks, such as many theoretical social networks, are very likely to develop an epidemic [16,21].

While it is well known how the topology affects the SIR process in static networks, there is very little literature about this model on adaptive networks. Recently, Lagorio *et al.* [5] studied two different strategies to mitigate the spread of a disease in an adaptive SIR model. In that model a susceptible

node disconnects the link with an infected individual with a rewiring probability w , and creates a link with another susceptible node. The authors found that there is a phase transition at a critical rewiring threshold w_c separating an epidemic from a nonepidemic phase, which can be related to static link percolation. A feature of this rewiring process is that links between susceptible individuals can be established independently of their previous relationship. In that strategy, the nodes have no memory because two individuals can be connected independently of their past. Even though this adaptive process could be representative of casual contacts between individuals such as those generated in public buildings like shopping, theaters, etc., that strategy will not work for other types of interaction such as friendship and working partners, where individuals preserve their closer contacts. Therefore, if an individual is separated from its closer neighbors, it will tend to reconnect with them, at some time, more often than with an unknown individual. On the other hand, recently Wang *et al.* [22] and Van Segbroeck *et al.* [23] proposed strategies to stop the spread in the SIR model where the susceptible individuals driven by fear disconnect their links with their neighbors, infected or not, without creating a new link. This strategy reduces the number of contacts permanently, which is efficient but also very inconvenient from an economical point of view.

In this paper we propose a strategy based on “intermittent social distancing” in adaptive networks and study its efficiency in stopping the spread of diseases in theoretical and real networks. We found theoretically that in our model there exists a cutoff threshold that prevents an epidemic phase. Our results are supported by extensive simulations. Moreover, we found that the intermittent social distancing strategy is efficient to protect a large susceptible cluster. The paper is organized as following: In Sec. II, we derive the theoretical transmissibility for our model and show how our strategy diminishes the epidemic phase. In Sec. III, we show how the epidemic size is reduced with our strategy and the agreement between our theoretical approach and the simulations. In Sec. IV, we present a study of the “susceptible herd behavior” that we use as a criterion to evaluate the effectiveness of our strategy. In Sec. V, we present our conclusions.

II. ANALYTICAL APPROACH

We propose a SIR model in an adaptive network where an infected individual transmits the disease to a susceptible neighbor with probability β and, if he fails, with probability σ the susceptible individual breaks the link with the infected one for a period t_b . Thus, the effective probability of breaking a link is $(1 - \beta)\sigma$. After a time t_b both nodes are reconnected and the process is repeated until the infected node recovers at a fixed time $t_r > t_b$, i.e., there is an intermittent connection between a susceptible node and its infected neighbor. This mimics a behavioral adaptation of the society to avoid contacts with infected individuals by imposing a social distancing during one or more periods of duration t_b . Notice that in our model a susceptible node breaks its links using only local information and not global knowledge as provided by communication media. In our model, the time is increased by 1 after every

infected node tries to infect its neighbors and the updates are done after each time step. In this process the dynamic transmissibility $T(\beta, \sigma, t_r, t_b) \equiv T_\sigma$ can be written as

$$T_\sigma = \sum_{n=1}^{t_r} \beta(1 - \beta)^{n-1}(1 - \sigma)^{n-1} + \beta \sum_{n=t_b+2}^{t_r} \phi(n, t_b, \sigma, \beta), \quad (1)$$

where $\phi(n, t_b, \sigma, \beta)$ is given by

$$\phi(n, t_b, \sigma, \beta) = \sum_{u=1}^{\lfloor \frac{n-1}{t_b+1} \rfloor} \binom{n - u t_b - 1}{u} \sigma^u \times (1 - \sigma)^{n-1-u(t_b+1)}(1 - \beta)^{n-1-u t_b} \quad (2)$$

and $\lfloor \cdot \rfloor$ denotes the integer part function. The first term of Eq. (1) is the probability that a node in the I state transmits the disease to any neighbor node in the S state (before it recovers), considering that the link $S-I$ has never been broken. The second term represents the probability for a node in the I state to transmit the disease to an S node after that pair has been disconnected u times for a period t_b . The binomial coefficient of the second term takes into account the number of ways to arrange u intermittent disconnected periods before the susceptible individual becomes infected at time n . Notice that $u t_b$ is the total times that the pair $S-I$ is broken and represent a temporal social distancing. In Table I, we illustrate the element $n = 8$ of the second term of Eq. (1) with $t_r = 10$ and $t_b = 2$.

It is known that the disease becomes an epidemic if the basic reproductive number $R_0 \geq 1$, where R_0 is the number of secondary infections. For uncorrelated networks, R_0 is related to the connectivity distribution $P(k)$ through the branching factor κ ,

$$R_0 = (\kappa - 1)T_\sigma, \quad (3)$$

where $\kappa \equiv \langle k^2 \rangle / \langle k \rangle$, and $\langle k \rangle$ and $\langle k^2 \rangle$ are the first and the second moments of $P(k)$ respectively. The branching factor is a measure of the heterogeneity of the network that diverges for SF networks with $\lambda < 3$ in the thermodynamic limit because $\langle k^2 \rangle \rightarrow \infty$ [21]. Then the critical cutoff σ_c , above which the disease dies, as a function of β , t_r , and t_b , can be found through the condition $R_0 = 1$, which yields

$$T_{\sigma_c} = \frac{1}{\kappa - 1} = T_c, \quad (4)$$

[21,24] where T_c is the critical transmissibility for the SIR model in static networks, and consequently our dynamic process in the steady state is related to a static topological property of the network. This is expected due to the fact that in our model the network topology does not change globally in the characteristic time scale of the disease spreading, and hence our strategy can be understood as a SIR model on a static network but with a transmissibility $T_c = T_{\sigma_c}$.

From Eq. (1) it is straightforward that in the limit $\sigma \rightarrow 0$, $T_\sigma = T = 1 - (1 - \beta)^{t_r}$. On the other hand, when $\sigma \rightarrow 1$, as

TABLE I. Disconnected periods for a pair $S-I$ with $t_r = 10$ (recovery time), $t_b = 2$ (disconnection period), and $n = 8$ (time of infection). The first column represents the number of disconnected periods u before $n = 8$, the second column is a typical configuration, the third column is the probability of that configuration, and the fourth column is the number of ways to arrange u disconnected periods. In the second column, each cell corresponds to a unit time. The white cells represent the time unit where a link between the S and the I node exists, the gray ones correspond to the disconnection period, and in the black cells there is no dynamic for the pair $S-I$ because the S has been infected and now the pair becomes $I-I$. Notice that initially the link cannot be broken because this disconnection happens only after the I individual fails to infect the susceptible one, with probability $(1 - \beta)$. Similarly, two disconnection periods must be separated by at least a white cell. When the infection occurs at time n , the maximum number of disconnected periods is $u = [(n - 1)/(t_b + 1)]$.

u	Example	Probability	Binomial coefficient
$u = 1$		$\beta \sigma (1 - \sigma)^4 (1 - \beta)^5$	$\binom{8-2-1}{1} = 5$
$u = 2$		$\beta \sigma^2 (1 - \sigma)^1 (1 - \beta)^3$	$\binom{8-4-1}{2} = 3$

the only terms that survive in Eq. (2) are those which fulfill the condition $n - 1 - u(t_b + 1) = 0$, we obtain

$$T_\sigma = \beta \left(1 + \sum_{u=1}^{\lfloor \frac{t_r-1}{t_b+1} \rfloor} (1 - \beta)^u \right),$$

$$= 1 - (1 - \beta)^{\lfloor \frac{t_r-1}{t_b+1} \rfloor + 1}. \tag{5}$$

In Fig. 1, we plot the plane $\sigma-T$ [$T \equiv T(\sigma = 0)$] in order to show that with our strategy the epidemic phase is reduced compared to the static case. Notice that for $t_b = t_r/2$ the epidemic phase shrinks substantially compared to the case $t_b = 1$.

In the figure, the light-gray area, delimited between the curves corresponding to the blocking periods $t_b = 1$ and $t_b = t_r - 1$, displays the region of parameters controlled by the intervention strategy. We can see that even the milder intervention $t_b = 1$ expands the epidemic-free area compared

to the static case. On the other hand, the stronger intervention $t_b = t_r - 1$ greatly shrinks the epidemic phase as σ increases. From the figure, we can also see that for $T_c \neq 0$, as the social distancing increases the epidemic-free phase increases; meanwhile, for a theoretical SF network with $\lambda \leq 3$, as $T_c = 0$, when $N \rightarrow \infty$ there is no epidemic-free phase for any values of σ and t_b . However, real networks are finite and they are not generally pure uncorrelated SF networks, which implies that our intermittent social distancing strategy could be applied in these networks.

In order to determine how the heterogeneity of the network affects the strategy performance, in Fig. 2 we plot a phase diagram in the plane $t_b-\sigma$ for fixed t_r and different values of κ .

We can see that as the heterogeneity κ increases, the social distancing t_b and σ have to increase in order to prevent the epidemic phase. Surprisingly, in highly heterogeneous networks, we find that for $t_b \geq t_r/2$, the critical cutoff

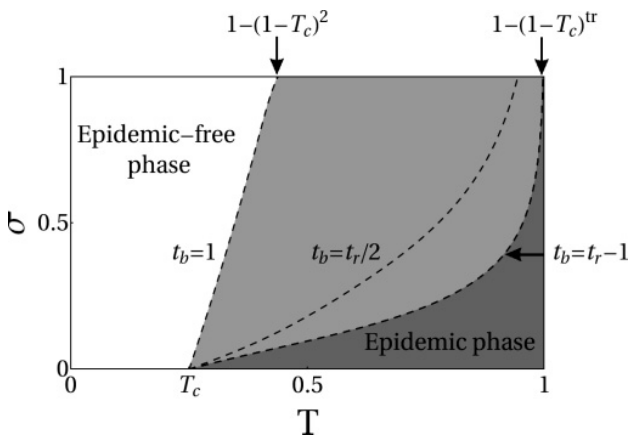


FIG. 1. Plot of the epidemic phases in the plane $\sigma-T$ for $t_r = 20$ and static $T_c = 0.25$, where T corresponds to the transmissibility in a nonadaptive network. The dashed lines correspond to the critical dynamic transmissibility T_{σ_c} for (from left to right) $t_b = 1$, $t_b = t_r/2$, and $t_b = t_r - 1$. For $t_b = 1$ and $\sigma = 1$, the maximum transmissibility for the static SIR model for which the epidemic phase is $T = 1 - (1 - T_c)^{t_r/(t_r+1)/2}$ or $T \approx 1 - (1 - T_c)^2$, and the t_r dependence disappears.

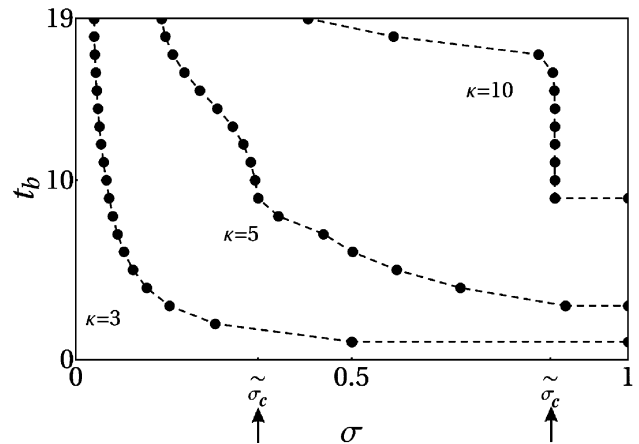


FIG. 2. Phase diagram for σ and t_b for $\beta = 0.05$ and $t_r = 20$ for different heterogeneities κ . The dashed lines with circles represent the interface between the nonepidemic (right) and epidemic (left) phases for the different values of κ . Notice that $\tilde{\sigma}_c = 0.33$ and 0.86 correspond to the critical cutoff probabilities where in some region the interface is a vertical line for $\kappa = 5$ and 10 , respectively.

probability $\sigma_c = \tilde{\sigma}_c$ is almost constant (see the Appendix) with

$$\tilde{\sigma}_c \sim \frac{\beta}{1-\beta} \left(\frac{1}{T_c} - 1 + \sqrt{\frac{1-T_c}{T_c^2}} \right) \quad (6)$$

$$\sim \frac{\beta}{(1-\beta)} \left[\kappa - 2 + \sqrt{(\kappa-2)(\kappa-1)} \right], \quad (7)$$

i.e., for very heterogeneous networks above $t_b = t_r/2$, σ_c does not change the transmissibility; then the best and least expensive strategy is to reconnect at $t_b = t_r/2$. This means that if we know the average duration of a disease t_r , individuals can return to their activities with low risk just after half of the characteristic time. From Eq. (7), it is straightforward that for $\tilde{\sigma}_c = 1$ there exists an upper value of $\kappa \equiv \kappa^{\text{lim}}$ that depends only on β , where κ^{lim} is given by

$$\kappa^{\text{lim}} = \frac{2 - (1-\beta)^2}{1 - (1-\beta)^2}, \quad (8)$$

where this strategy can be applied. From Eq. (6) the limit $\tilde{\sigma}_c = 1$ can also be expressed in terms of the minimum critical transmissibility,

$$T_{\sigma_c}^{\text{lim}} = 2\beta - \beta^2. \quad (9)$$

As a consequence, for very low infection rates, our strategy predicts an epidemic-free phase even for highly heterogeneous networks with a finite epidemic threshold. Notice that since real networks have degree correlations and clustering, then the relation $T_c = 1/(\kappa - 1)$ does not hold and the only magnitude that matters is T , which has to be measured by the peak of the second giant component [25]. In those cases, for $\tilde{\sigma}_c = 1$ we have to use Eq. (9) instead of Eq. (8). We found that for the condensed matter coauthorship network [26] ($T_c = 0.026$) and mathematics coauthorship network [27] ($T_c = 0.050$) the epidemic spread can be stopped in diseases with $\beta \leq 0.013$ and $\beta \leq 0.025$, respectively. Similar values of β were used for real networks by Kitsak *et al.* [28].

III. NUMERICAL RESULTS

In our model at the initial stage, all nodes are susceptible and we infect a randomly chosen node (patient zero) in the biggest component of our network. From the patient zero the disease spreads to its neighbors according to the rules of our model described above: an infected individual transmits the disease to a susceptible neighbor with probability β and, if it fails, with probability σ the susceptible individual breaks the link with the infected one for a period t_b . After a time t_b both nodes are reconnected and the process is repeated until the infected node recovers at a fixed time $t_r > t_b$. The time is increased by 1 after every infected node tries to infect its neighbors and the updates are done after each time step.

All our results are presented for $t_r = 20$ but qualitatively all the results are the same for $t_r > 1$.

In Fig. 3, we plot the relative epidemic size $M_I/M_I(0) \equiv M_I(\sigma; t_b)/M_I(\sigma = 0)$ as a function of σ for ER and SF networks for different values of t_b and $t_r = 20$. From the plot we can see that $M_I(\sigma)$ decreases as σ and t_b increase compared to the static case $M_I(0)$. We can also see that a critical probability σ_c exists, which can be obtained theoretically from Eq. (4), above which the disease dies. Then, depending on how

virulent is the disease without intervention, we can control t_b in order to stop the spread. In the insets of the figures, we collapse all the curves using the value of σ_c obtained from Eq. (4). The collapse close to the critical value shows the excellent agreement between the theoretical value of σ_c and the simulations.

IV. SUSCEPTIBLE HERD BEHAVIOR

With our intermittent social distancing strategy, the susceptible nodes dynamically reduce their contact with the nodes in the infected cluster, mitigating the spread of the disease, i.e., our strategy produces a resistance to the disease which we call susceptible herd behavior. As a result of the coevolutionary process, at the end of the spreading there is only one cluster composed of recovered individuals and, depending on T_σ , one or more susceptible clusters that give rise to a cluster size distribution of susceptible individuals or “voids” [29]. In our model, the cluster size distribution of voids or susceptible individuals is important since the formation of a susceptible herd behavior induced by the network dynamics also measures how effective our strategy is to preserve a whole part of the society safe from the disease. Next, we derive the value of the transmissibility for which a susceptible crowding develops, i.e., the value below which our strategy is efficient.

Describing the growth of an epidemic cluster as a Leath process [30,31] for a value of the link occupancy probability $p \equiv T_\sigma$ and denoting by $f_n(p)$ the probability that a cluster reaches the n th generation following a link, then the probability $f_\infty(p)$ that a link leads to a giant component when $n \rightarrow \infty$ is given by

$$f_\infty(p) = \sum_{k=1}^{\infty} \frac{kP(k)}{\langle k \rangle} [1 - p f_\infty(p)]^{k-1}, \quad (10)$$

where $f_\infty(p)$ is the solution of

$$f_\infty(p) = 1 - G_1[1 - p f_\infty(p)], \quad (11)$$

and $G_1(x) = \sum_{k=1}^{\infty} kP(k)/(k)x^{k-1}$.

When the “epidemic” cluster grows, the size of the void clusters is reduced as in a node dilution process, since when a link is occupied a void cluster loses a node and all its edges. Then $f_\infty(p)$ is the probability that a void cluster loses a node. If we denote by $1 - p^v$ the fraction of void nodes removed, the following relation holds [32]:

$$1 - p^v = f_\infty(p). \quad (12)$$

For node percolation, it is known that [33] $P_\infty^v(p^v) + \sum s n_s^v = p^v$, where $P_\infty^v(p^v)$ is the fraction of nodes in the giant void component and n_s^v the number of finite void clusters of size s . The fraction of remaining nodes, below which the giant void cluster is destroyed, corresponds to the critical probability of node void percolation $p^v = p_c^v$. At this value $P_\infty^v(p_c^v) = 0$; then $\sum s n_s^v = p_c^v$. This means that at the void transition only a fraction p_c^v of the nodes belong to void clusters. As a consequence, the fraction of links p^* needed to reach this point fulfills

$$p_c^v = 1 - f_\infty(p^*). \quad (13)$$

Therefore, from Eqs. (11) and (13)

$$p_c^v = G_1[1 - p^*(1 - p_c^v)], \quad (14)$$

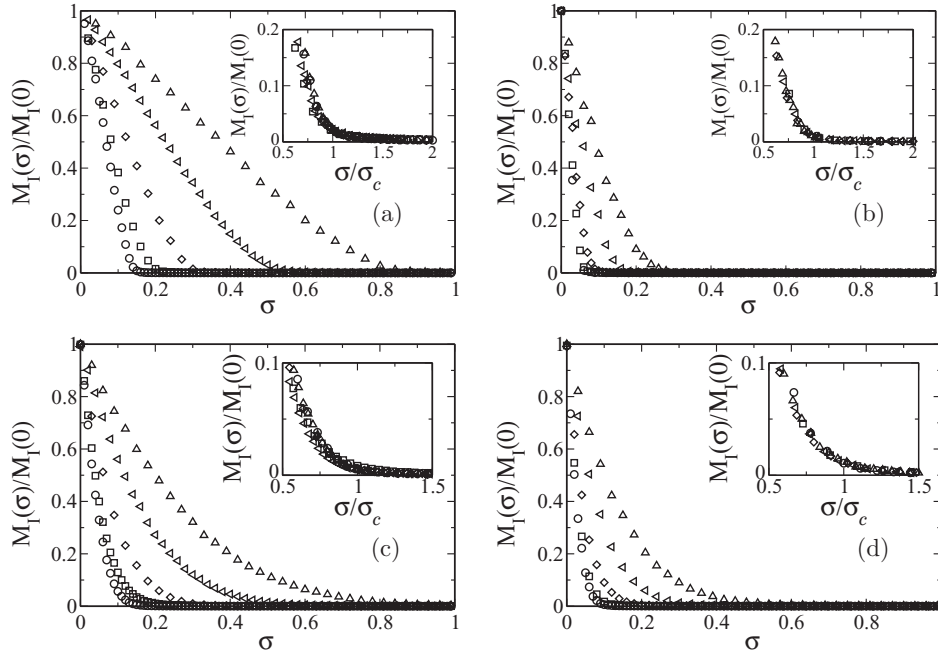


FIG. 3. $M_I(\sigma, t_b)/M_I(\sigma=0)$ vs σ in an ER network with $\langle k \rangle = 4$, $N = 10^4$, $t_r = 20$, for different values of t_b : $t_b = 3$ (Δ), 5 (\triangleleft), 10 (\diamond), 15 (\square), and 19 (\circ) for $\beta = 0.05$ with original transmissibility $T = 0.64$ (a) and $\beta = 0.025$ with $T = 0.40$ (b); $M_I(\sigma, t_b)/M_I(\sigma=0)$ vs σ in a SF network with $\lambda = 3.5$ and minimal connectivity $k_{\min} = 2$ for $t_r = 20$, $\beta = 0.075$ with original transmissibility $T = 0.79$ (c) and $\beta = 0.05$ with original transmissibility $T = 0.64$ (d). In the insets we show enlargements of the main plot, rescaled in the abscissa by the factor σ_c , obtained from Eq. (1). Our simulations were averaged over 10^4 realizations.

where $p_c^v = G_1[(G_1')^{-1}(1)]$ and p^* is the solution of Eq. (14). Notice that at the void transition $G_1'(1 - p^* + p_c^v p^*) = 1$. A similar result was also found by Newman for a process of spreading of two pathogens [34], employing a different approach. Using the mapping between our adaptive SIR model and link percolation, the transmissibility T_c^* needed to create a giant component of crowded susceptible individuals is $T_c^* = p^*$. Thus, for a disease spreading in an ER network, the dynamical critical transmissibility for the giant susceptible cluster is given by $T_\sigma^* = -T_{\sigma_c} \ln(T_{\sigma_c}) / (1 - T_{\sigma_c})$.

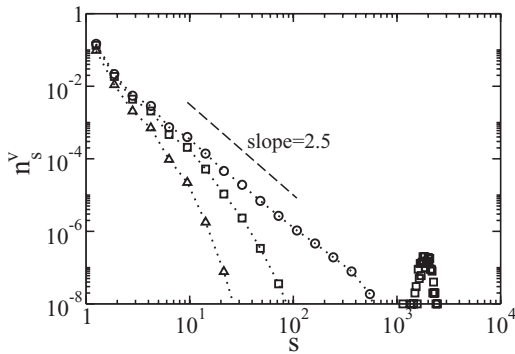


FIG. 4. Log-log plot of cluster size distribution of susceptible individuals for an ER network with $\langle k \rangle = 4$ ($T_\sigma^* = 0.46$) and $N = 10^4$ at $T_\sigma = 0.64$ (Δ), $T_\sigma = 0.46$ (\circ), and $T_\sigma = 0.40$ (\square). The dotted lines are a guide to the eye and the dashed line is the result of a power law fitting for T_σ^* . Notice that we add in n_s^v the giant component.

In Fig. 4, we compare the cluster size distribution of susceptible individuals or voids n_s^v for different values of the transmissibility, where we distinguish outbreaks from epidemics [15,16,35] on an ER network with $\langle k \rangle = 4$.

For $T_\sigma > T_\sigma^*$ we obtain only small clusters of susceptible nodes that decay faster than exponentially. On the other hand, for $T_\sigma < T_\sigma^*$ a giant susceptible cluster appears, meaning that our strategy is efficient in producing large connected clusters of susceptible nodes that crowd in order to protect themselves. When with our strategy the transmissibility is reduced to $T_\sigma = T_\sigma^*$, the epidemic spreading slows down and the distribution n_s^v decays as a power law with the same exponent $\tau = 5/2$ as in a void node percolation transition, in contrast with the distribution of infected sizes of outbreaks, which at criticality goes as $n_s^I \sim s^{-\tau+1}$ as in a Leath process [30]. These results confirm the importance of the interplay between void nodes and link percolation in our model.

V. CONCLUSIONS

In this paper we present an adaptive strategy based on intermittent social distancing in an adaptive SIR model. In the intermittent social distancing strategy a susceptible individual breaks the link with the infected neighbor with a cutoff probability σ and then both individuals are reconnected after a time t_b before the infected individual recovers at t_r . Using the framework of percolation theory, we derive the dynamical transmissibility, and we find that there exists a critical cutoff σ_c where the epidemic spread is stopped for non-highly-heterogeneous networks. We show that in some

real networks our intermittent social distance strategy could stop the epidemic spreading for not very virulent diseases. We find an excellent agreement between the theory and the simulations. For heterogeneous networks, we find that a very high value of t_b does not lead to a decrease of the epidemic, which implies that the less expensive strategy is to chose $t_b = t_r/2$. Finally we verify that our strategy reduces the transmissibility below a value where a susceptible crowding is produced. Any clever strategy used to reduce the disease from spreading should protect the population at the least economic cost. We believe that our strategy, which allows us to control the disconnection of periods through t_b , is a very convenient strategy because it creates a susceptible herd cluster. Our present findings could be used as a support and reference guidance for the development of further strategies to stop diseases in real networks.

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APPENDIX

For $t_b \geq [(t_r - 1)/2] \approx t_r/2$ and $t_b < t_r - 1$, in Eq. (2) the summation has only one term, because for $t_b \geq t_r/2$ we have only one period of disconnection, i.e., $[(n - 1)/(t_b + 1)] = 1$. Intuitively, if $t_b \geq t_r/2$ then in order to compute the

transmissibility, we have to consider at most only one break period since otherwise the transmission of the disease is not possible. Denoting by $\delta = (1 - \sigma)(1 - \beta)$, Eq. (1) reduces to

$$\begin{aligned} T_\sigma &= \sum_{n=1}^{t_r} \beta \delta^{n-1} + \beta(1 - \beta)\sigma \sum_{n=t_b+2}^{t_r} (n - t_b - 1)\delta^{n-2-t_b} \\ &= \frac{\beta(1 - \delta^{t_r})}{1 - \delta} + \frac{\beta\sigma}{(1 - \sigma)(1 - \delta)^2} \\ &\quad \times \{1 + \delta^{t_r - t_b - 1}[\delta(t_r - t_b - 1) + t_b - t_r]\}. \end{aligned} \quad (\text{A1})$$

Neglecting the higher powers of δ (which hold for $\sigma \rightarrow 1$, $\beta \rightarrow 1$, or high values of $t_r - t_b$), we obtain

$$T_\sigma = \frac{\beta}{1 - \delta} + \frac{\beta\sigma(1 - \beta)}{(1 - \delta)^2}. \quad (\text{A2})$$

Notice that T_σ loses all the dependence on t_r and t_b as shown in Fig. 2. With this approximation and using the fact that $T_{\sigma_c} = T_c = 1/(1 - \kappa)$, the critical cutoff $\sigma_c = \tilde{\sigma}_c$ is given by

$$\tilde{\sigma}_c = \frac{\beta}{1 - \beta} \left(\frac{1}{T_c} - 1 + \sqrt{\frac{1 - T_c}{T_c^2}} \right). \quad (\text{A3})$$

If Eq. (4) holds, then

$$\tilde{\sigma}_c = \frac{\beta}{1 - \beta} [\kappa - 2 + \sqrt{(\kappa - 1)(\kappa - 2)}]. \quad (\text{A4})$$

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