Quarantine-generated phase transition in epidemic spreading

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We study the critical effect of quarantine on the propagation of epidemics on an adaptive network of social contacts. For this purpose, we analyze the susceptible-infected-recovered model in the presence of quarantine, where susceptible individuals protect themselves by disconnecting their links to infected neighbors with probability w and reconnecting them to other susceptible individuals chosen at random. Starting from a single infected individual, we show by an analytical approach and simulations that there is a phase transition at a critical rewiring (quarantine) threshold w_c separating a phase ($w < w_c$) where the disease reaches a large fraction of the population from a phase ($w \geqslant w_c$) where the disease does not spread out. We find that in our model the topology of the network strongly affects the size of the propagation and that w_c increases with the mean degree and heterogeneity of the network. We also find that w_c is reduced if we perform a preferential rewiring, in which the rewiring probability is proportional to the degree of infected nodes.

I. INTRODUCTION

The representation of interactions in a human society as a complex network, where nodes and links play the respective roles of individuals and their contacts, has been useful for modeling, studying, and understanding many problems in epidemiology [1,2]. Usually it is assumed that diseases evolve more rapidly than the topological evolution of the underlying network, so that the links of the network can be regarded as static. With modern mass media, however, the presence of an epidemic can be broadcast at a rate much faster than that of disease propagation. This information will inevitably change the behavior of the individuals comprising this network, as, for example, they try to avoid contacts with infected people. In this way a feedback loop between the state of individuals and the topology of the network is formed. Networks that exhibit such feedback are called adaptive or coevolutionary networks [3–6].

Public health services are constantly searching for new ways to try to reduce the spread of diseases. Interventions like vaccination [7] and total closure of workplaces and schools are very effective, but come with a high economic cost. As a less expensive alternative we examine here the effectiveness of a "quarantine" strategy, where healthy people are "advised" to avoid contacts with individuals that carry the disease. That is, a healthy person has a chance to suppress a contact with an infected neighbor and form a new tie with another healthy peer (rewiring). The value of this rewiring probability could depend, for instance, on the concern that the society has about the disease, and as suggested, in a globalized world this concern will depend on the broadcast news. The degree of media attention to the disease is a parameter that could be controlled by public health services. Indeed, it is known that spontaneous quarantine in the recent H1N1 pandemic was

found to have a large impact in reducing the final size of the epidemic [8].

Based on these observations, we propose two strategies for the propagation of epidemics on an adaptive network, in which individuals alter their local neighborhoods with constant quarantine probability w as described and systematically study the effect that the quarantine has on epidemic spreading. We find a phase transition at a critical threshold w_c above which the epidemic is stopped from spreading. We show also how the epidemic spreading in the presence of quarantine depends on the contagion and recovery parameters. More importantly, we find that the initial structure of the network plays an essential role in disease propagation at criticality, unlike in previous related models [6,9–11] where results depend only on the average network connectivity. We also introduce a generalized form of quarantine, where the probability of rewiring is proportional to the degree of infected nodes, producing a more efficient isolation of the nodes with a high degree. This preferential rewiring is more efficient than the case with w constant.

II. ANALYTICAL APPROACH

We consider the susceptible-infected-recovered (SIR) epidemic spreading model, which is well established and accurately describes diseases such as seasonal influenza, SARS, and AIDS [7,12]. Initially, all nodes are in the susceptible state (s), with one node chosen at random (seed) in the infected state (i). In the first strategy, strategy A, at each time unit, every infected node i in the network tries to transmit the disease to each susceptible neighbor s with infection probability β. If s does not get infected, then with rewiring probability w it disconnects its link from i and reconnects it to another randomly chosen susceptible node, different from its present neighbors. Thus, the rewiring probability w measures how strongly susceptible nodes react to the disease (the quarantine probability). Infected nodes recover (r) after a fixed recovery

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time t_R since they first became infected, remaining in the recovered state forever.

To estimate w_c we start by assuming that the network has a tree structure [13], so that the disease spreads out from the seed and reaches a susceptible node s through only one of its neighbors i. Then, if i becomes infected at time t_0, the probability that s becomes infected by i at time $t_0 + n$, with $n = 1, 2, \ldots t_R$, is $\beta(1-\beta)^{n-1}(1-w)^{n-1}$. This is the probability that s has neither become infected nor disconnected from i up to time $t_0 + n - 1$, times the probability that i succeeds in transmitting the disease to s at time $t_0 + n$. Therefore, the overall probability that s becomes infected before i recovers is given by the sum

$$T_{\beta,w}^A \equiv \sum_{n=1}^{t_R} \beta(1-\beta)^{n-1}(1-w)^{n-1}$$
$$= \frac{\beta\{1 - [(1-\beta)(1-w)]^{t_R}\}}{1 - (1-\beta)(1-w)}. \tag{1}$$

This expression for the transmissibility $T_{\beta,w}^A$ is equivalent to the corresponding expression in the standard SIR model $\sum_{n=1}^{n=t_R} \beta(1-\beta)^{n-1}$ [1], but with a noninfection probability $(1-\beta)(1-w)$, instead of $(1-\beta)$. When $w = 0$, Eq. (1) reproduces the known value for the transmissibility $T = 1 - (1-\beta)^{t_R}$ for the SIR model on static networks [1]. In this formulation w plays the role of a control parameter of the transmissibility: for fixed values of t_R and β, $T_{\beta,w}^A$ can be reduced by increasing w. By reducing $T_{\beta,w}^A$ we can go from a regime in which the epidemic spreads over the population (epidemic phase) to another regime where the disease cannot spread (disease-free phase). The transition from the disease-free phase to the epidemic phase corresponds to the average number of secondary infections per infected node becoming larger than 1, allowing the long-term survival of the disease and thus ensuring that the epidemic spreads to a large fraction of the population. In our problem, the expected number of susceptible neighbors that a node has when it just becomes infected is given by $\kappa - 1$, where $\kappa - 1 \equiv \langle k^2 \rangle / \langle k \rangle - 1$ is called the *branching factor*, and $\langle k \rangle$ and $\langle k^2 \rangle$ are the first and second moments, respectively, of the degree distribution $P(k)$. Since $T_{\beta,w}^A$ is the overall probability of infecting a neighbor, the mean number of secondary infections per infected node is

$$N_I^A(w) = (\kappa - 1)T_{\beta,w}^A. \tag{2}$$

The infection will die out if each infected node does not spawn, on average, at least one replacement, so for a very large system, the critical point is given by the relation $(\kappa - 1)T_{\beta,w_c}^A = 1$, or

$$\frac{(\kappa - 1)\beta\{1 - [(1-\beta)(1-w_c)]^{t_R}\}}{1 - (1-\beta)(1-w_c)} = 1. \tag{3}$$

The transition between the free-disease phase and the epidemic phase is analogous to the *static link percolation* problem [14], in which each link in a network is occupied with probability p and empty with probability $q = (1-p)$. When p becomes lower than a percolation threshold p_c, the giant connected component disappears. In general, p_c depends on the size of the network N [15], but in the thermodynamic

limit it can be expressed as $p_c = 1/(\kappa - 1)$ [16]. Identifying p with the transmissibility $T_{\beta,w}^A$, and using the relation between p_c and κ, we find that on the epidemic/disease-free transition line,

$$T_{\beta,w_c}^A = p_c. \tag{4}$$

This result shows that the transition point depends on the initial topology of the network through the moments of the degree distribution, as we shall confirm via simulation.

A better strategy would be try to avoid the contact between susceptible and infected individuals before the attempt to infection, that is, avoid the contact only when you know an individual is sick. In this second strategy, strategy B, at each time unit every susceptible node attached to an infected node disconnects its link from i with probability w and reconnects it to another randomly chosen susceptible node, different from its present neighbors. If s does not rewire its link, the infected node i tries to infect it with infection probability β. Notice that $N_I^B(w) = (1-w)N_I^A(w)$. For this strategy Eq. (1) is replaced with

$$T_{\beta,w}^B \equiv \frac{\beta(1-w)\{1 - [(1-\beta)(1-w)]^{t_R}\}}{1 - (1-\beta)(1-w)}. \tag{5}$$

Comparing Eqs. (1) and (5) we see that $T_{\beta,w}^B < T_{\beta,w}^A$ for identical values of β and w. The new condition for disease to die out in strategy B follows from Eq. (5):

$$\frac{(\kappa - 1)\beta(1-w_c)\{1 - [(1-\beta)(1-w_c)]^{t_R}\}}{1 - (1-\beta)(1-w_c)} = 1. \tag{6}$$

The two strategies represent different scenarios depending on the knowledge of the state of infection of the nodes. For strategy A, susceptible nodes have no information about the state of their neighbors until they are in physical contact. In contrast, for strategy B susceptible nodes know the state of their neighbors before they are in physical contact. We will show that this difference in the knowledge of the states of the nodes results in strategy B being more effective at stopping epidemic spreading.

Figure 1 shows phase diagrams in the w-β plane for strategy A and strategy B, obtained by the numerical solutions of Eqs. (3) and (6), respectively, for a network with $\kappa = 5$ ($p_c = 0.25$). Two phases emerge: the epidemic phase for $T_{\beta,w}^{A,B} > p_c$ and the disease-free phase for $T_{\beta,w}^{A,B} \leqslant p_c$. The location of the critical line separating the two phases depends on the recovery time t_R. Figure 1(a) illustrates the two limiting cases for strategy A: $t_R = 1$ (upper curve) and $t_R = \infty$ (lower curve), which are given by the expressions $\beta(t_R = 1) = 1/(\kappa - 1)$ and $\beta(t_R = \infty) = w/(\kappa + w - 2)$. Therefore, the pairs of (w,β) values in the region above the curve $\beta(t_R = 1)$ are always in the epidemic phase, and below the curve $\beta(t_R = \infty)$ they are always in the disease-free phase. A striking consequence is that if β is larger than the percolation threshold $1/(\kappa - 1)$, the propagation of an epidemic cannot be stopped, even with the largest rewiring probability $w = 1$. Thus, strategy A is not an efficient mechanism to control an epidemic when β is higher than p_c.

For strategy B, Fig. 1(b) illustrates the two limiting cases $t_R = 1$ (upper curve) and $t_R = \infty$ (lower curve), which are
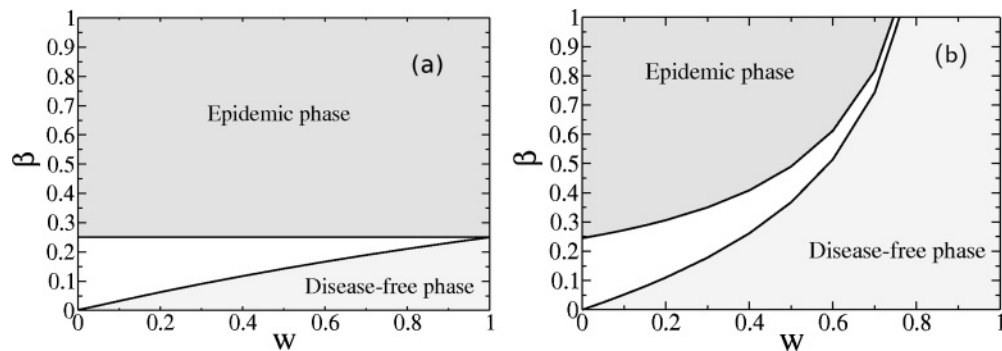
FIG. 1. β-w phase diagram for $p_c = 0.25$. Curves correspond to $t_R = 1$ (upper) and $t_R = \infty$ (lower). The darker gray region will always be an epidemic phase, and the light gray region always a disease-free phase: (a) strategy A; (b) strategy B.

given by the expressions $\beta(t_R = 1) = 1/(\kappa - 1)(1 - w)$ and $\beta(t_R = \infty) = w/(\kappa - \kappa w + 2w - 2)$. In contrast to strategy A, if κ does not diverge, that is, the network has finite p_c, the epidemic can always be stopped, even for $\beta \to 1$. Notice that the maximum value of w needed to stop an epidemic is $w_c = (\kappa - 2)/(\kappa - 1) = 1 - p_c$.

Our theoretical predictions illustrate a novel feature about the dynamics of adaptive networks in SIR models. While previous adaptive SIS and SIRS models have transition values that depend only on the average connectivity of the network $\langle k \rangle$, and are independent of the heterogeneity or structural correlations of the initial topology [3], our SIR model predicts dependence on the topological structure through the higher order moments of the degree distribution.

III. SIMULATION RESULTS

Our analytical approach predicts, through Eqs. (3) and (6), a dependence of w_c on the initial network. To test and explore this dependence, we performed extensive numerical simulations of our model starting from different topologies. We first used Erdös-Rényi (ER) networks with Poissonian degree distribution $P(k) = e^{-\langle k \rangle} \langle k \rangle^k / k!$. Even though these

FIG. 2. Plot of $n_I(w)/n_I(0)$ as a function of w for strategy A ($\bigcirc$) and strategy B ($\square$), for an ER network with $\langle k \rangle_{GC} = 4.07$; $N = 10^4$ and 10^4 realizations. As we predicted previously, strategy B is better than strategy A, shown by $w_c^B < w_c^A$.

types of homogeneous networks are common in nature, many real social networks are well represented by heterogeneous networks. Thus, we also used finite scale-free (SF) networks with degree distribution $P(k) = k^{-\lambda} \exp(-k/K)/\mathrm{Li}_\lambda(e^{-1/K})$, where K is the degree cutoff. This distribution represents networks with a finite threshold p_c and appears in a variety of real-world networks [17,18]. We only consider epidemic propagation in the largest connected cluster of the network, the giant component (GC).

In Fig. 2 we compare both strategies for ER networks. We plot, as a function of w, the average fraction of infected nodes $n_I(w) = N_I(w)/N_{GC}$, where N_{GC} is the size of the GC, on an ER network. This fraction is normalized by the corresponding fraction on an identical fixed network, that is, for the SIR model without quarantine, $n_I(0)$. We can see that w_c for strategy B is lower than w_c for strategy A, as expected. This relation will hold for any topology with the same κ [see Eqs. (3) and (6)]. Since strategy B is more effective, henceforth we show only simulation results for strategy B.

Figure 3 shows $n_I(w)/n_I(0)$ vs. w for different β. The figure shows the strong effect of quarantine and the critical threshold w_c above which $n_I(w)$ approaches 0. Thus, in the disease-free phase ($w \geqslant w_c$), only a small number of individuals get infected and the disease quickly dies out. We observe, as expected, that the values of w_c increase with β. This behavior matches the phase diagram in Fig. 1(b), where the critical line has a positive slope. Scaling the horizontal axis by the values of w_c obtained by numerically solving Eq. (6) collapses the curves, showing an excellent agreement between theory and simulations, as well as a scaling behavior of the form $n_I(w) = n_I(0) f(w/w_c)$, as shown in the inset in Fig. 3.

We also explored the dependence of w_c on the connectivity of the network, by computing $n_I(w)/n_I(0)$ vs. w for different $\langle k \rangle$ (see Fig. 4). We observe that w_c increases with $\langle k \rangle$, due to the fact that propagation is facilitated by having more neighbors, as in the original SIR dynamics. The inset in Fig. 4 shows the collapse of all curves. Again, the good agreement between theory and simulation confirms that Eq. (6) is a valid expression of the transition point for the adaptive SIR model on ER networks.

Given that the second moment, and therefore κ, is large in heterogeneous networks, the critical value w_c turns out to be larger in heterogeneous networks than in homogeneous networks with the same mean degree $\langle k \rangle$ and size N, as seen
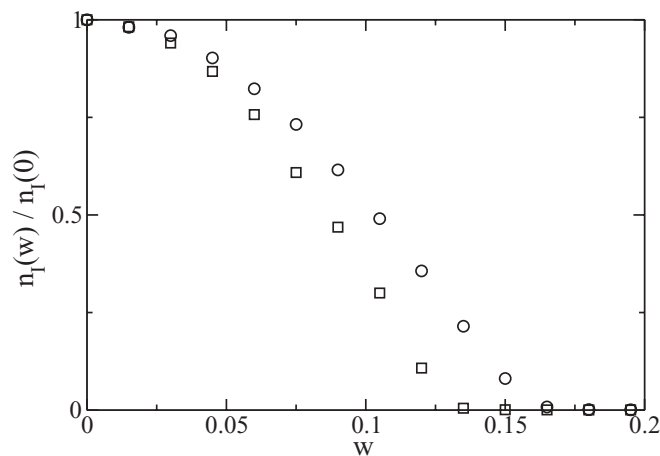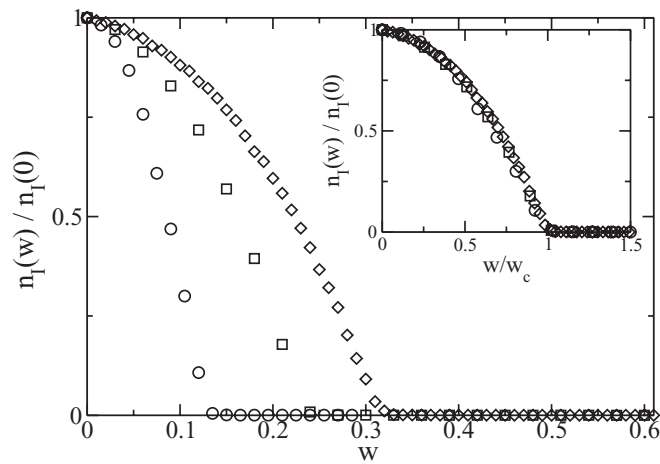
FIG. 3. Fraction of nodes infected as a function of the applied quarantine parameter w divided by the fraction of infected nodes at $w = 0$ for three infection probabilities β in an ER network. ($\bigcirc$) $\beta = 0.05$, ($\square$) $\beta = 0.1$, and ($\diamond$) $\beta = 0.15$. Above a threshold value of w, no finite fraction of the network becomes infected. Inset: Data with w rescaled by the appropriate w_c calculated from Eq. (6). The curves collapse very well, showing universal behavior and good agreement with theory. In all simulations, $\langle k \rangle_{GC} = 4.07$ ($\langle k \rangle = 4$ over all nodes), $N = 10^4$, and averages are over 10^4 realizations.

by considering Eq. (6) in the $t_R \gg 1$ limit, where

$$w_c \simeq \frac{1}{\frac{1}{\beta(\kappa-2)} + 1}. \qquad (7)$$

Since κ for heterogeneous networks is much bigger than for homogeneous networks, we expect that w_c increases as the heterogeneity increases with larger K. For $\kappa \to \infty$ we expect that $w_c \to 1$ and that the transition will eventually disappear for large heterogeneous networks.
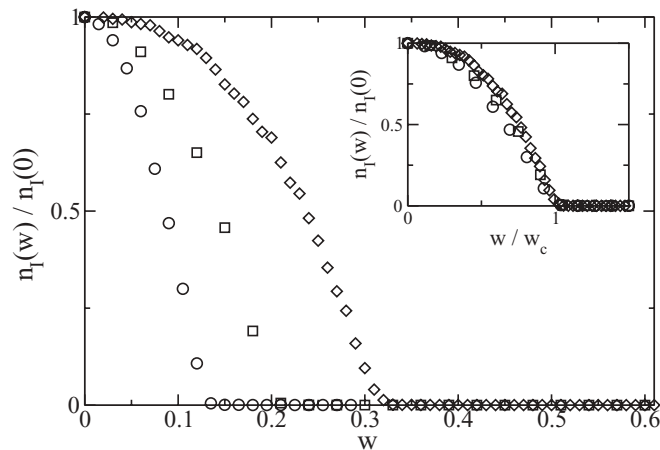

FIG. 4. Fraction of nodes infected as a function of the applied quarantine parameter w divided by the fraction of infected nodes at $w = 0$ for ER networks with three $\langle k \rangle$ values: $\langle k \rangle_{GC} = 4.07$ ($\bigcirc$), $\langle k \rangle_{GC} = 6.015$ ($\square$), and $\langle k \rangle_{GC} = 10$ ($\diamond$). Again, while below w_c a finite fraction of the network become infected, above w_c the epidemic vanishes. Inset: Data rescaled by w_c calculated from Eq. (6). In all simulations, $\beta = 0.05$, $N = 10^4$, and averages are over 10^4 realizations.
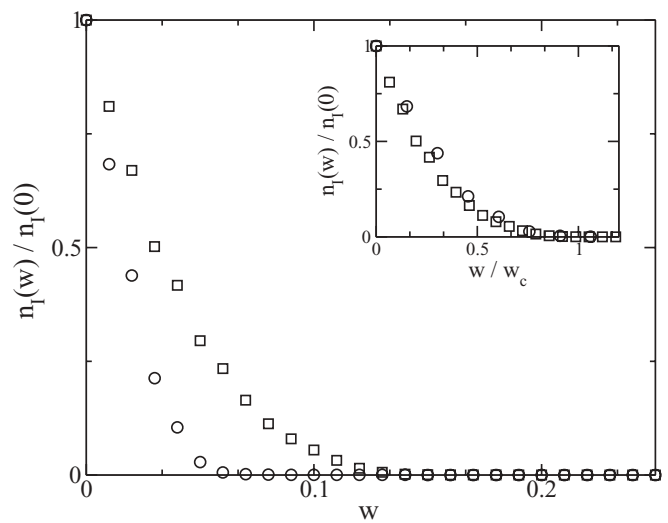

FIG. 5. Fraction of infected nodes still infected as a function of w for $\beta = 0.05$, $t_R = 20$ on an SF network with $K = 10$ ($\bigcirc$) and $K = 20$ ($\square$); $\lambda = 2.1$, with $N = 10^5$. $p_c \cong 0.375$ for $K = 10$ and $p_c \cong 0.215$ for $K = 20$. Inset: Rescaled by w_c. The result shows an excellent agreement between theory and simulation.

In Fig. 5, we show simulations on SF networks for different values of K [19]. In good agreement with our predictions from Eq. (7), as κ increases with K, w_c increases. In the inset in Fig. 5 we rescale by w_c, obtained from Eq. (6), and find good collapse, confirming the general validity of Eq. (6) for heterogeneous SF networks.

In very heterogeneous networks it is well known that due to high-degree nodes, propagation processes are very difficult to stop [14,20]. Thus, a constant probability of rewiring is not effective for controlling epidemics in these networks, because such a strategy assumes all nodes have the same importance in an epidemic, ignoring the function of higher degree nodes as superspreaders. It is also well known that removing the high-degree nodes in the network (targeted percolation) [14,21,22] is a more efficient method for stopping propagation processes than random removal. This type of strategy is expected to be superior but requires global information about the network.

To test this prediction, we propose a new strategy of type B where w depends on the degree k of the infected node, with w_k given by the general form

$$w_k \equiv w_k(\alpha) = \gamma k^{\alpha}, \qquad (8)$$

where γ is a constant that controls the highest possible value of w_k, and it is equal to or smaller than $k_{max}^{-\alpha}$, where k_{max} is the largest degree of the network. For $\alpha = 0$ and $\gamma \in [0,1]$ we recover the results for a constant value of w, with $w = \gamma$. For $\alpha > 0$ and $\gamma = k_{max}^{-\alpha}$ the rewiring increases with the degree k of the infected node and decreases with increasing α. In the limit of $\alpha \to \infty$ the rewiring process is equivalent to a targeted rewiring where only the links of the highest connected node(s) are rewired. To compare the cases with $\alpha = 0$ and $\alpha > 0$, we use the average $\langle w \rangle$ over the network, choosing α for the targeted case such that $\langle w \rangle$ is equal to w in the uniform case.
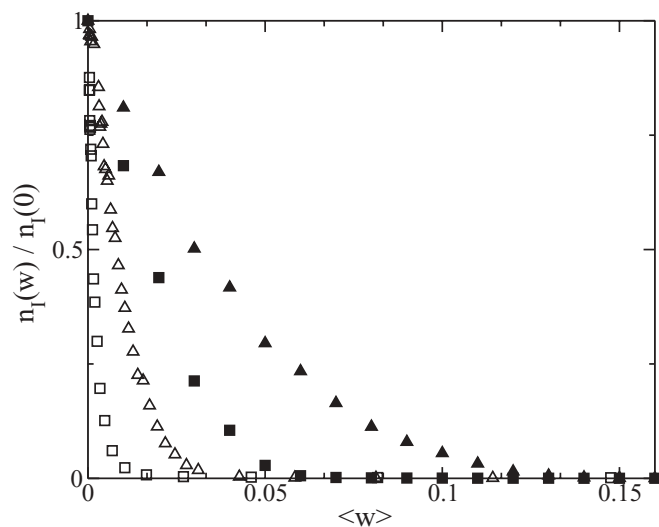
FIG. 6. Fraction of infected nodes still infected as a function of $\langle w \rangle$ for $\beta = 0.05$, $t_R = 20$ on an SF network; $\lambda = 2.1$, $N = 10^4$, and $K = 10, 20$. For general strategy B with $\alpha = 0$ [$K = 10$ (■) and $K = 20$ (▲)] and with $\alpha > 0$ [$K = 10$ (□) and $K = 20$ (△)], each $\langle w \rangle$ represents a different α.

In Fig. 6 we plot $n_I(w)/n_I(0)$ as a function of $\langle w \rangle$ for SF networks with $K = 10$ and $K = 20$. As expected, w_c is lower for $\alpha > 0$ than for $\alpha = 0$. The preferential rewiring reduces the value of w_c because quarantine is more effective at isolating the superspreaders.

IV. SUMMARY AND CONCLUSIONS

We have introduced and studied two strategies for the propagation of epidemics on evolving networks of social contacts. The states of the individuals are changed according to SIR dynamics, while the network evolves according to a quarantine mechanism based on local information, in which susceptible individuals replace their infected neighbors with other susceptible peers with probability w. We demonstrated by an analytical approach, and confirmed by numerical simulations, that the size of the epidemics can be largely reduced by increasing the probability of rewiring and that propagation can eventually be stopped by using high enough values of w. In other words, quarantine is an effective way to halt the appearance of epidemics that would otherwise emerge in the case of a static network. For strategy A, when the infection probabliy is higher than a threshold, the quarantine mechanism is not effective any more and disease propagation becomes unavoidable. This is because the quarantine model only breaks contact after allowing for a chance of infection, thus for a high enough infection probability, the spreading is irreversible, even with a rewiring probability of unity. For strategy B, the quarantine mechanism is effective in homogeneous networks and finite heterogeneous networks, even for an infection probability equal to unity. The transition disappears only for large, very heterogeneous networks. In these SF networks, the critical rewiring probability becomes very high and the transition eventually disappears for large enough systems, thus only the epidemic phase is observed. This is likely due to the presence of individuals with a very high connectivity that can spread the disease over a large fraction of the population, even for low infection probabilities. To confirm this, we introduced a generalized form of strategy B, where the quarantine depends on the degree of the infected nodes. This preferential rewiring isolates the superspreaders more efficiently, reducing w_c and preserving a finite transition even for SF networks. Finally, in SIR dynamics the final frozen state, where each individual is either recovered or susceptible, is reached rather quickly—in a time of order $\ln N$ according to our simulations—and thus the network does not evolve much, so the initial topology is preserved during the entire evolution of the system. In contrast, in adaptive models with SIS or SIRS dynamics the system evolves for a very long time in the epidemic phase—times that grow exponentially larger with N—thus the network moves toward a stationary topology similar to an ER network, independent of the initial topology, rendering the initial topology irrelevant. Therefore, unlike other adaptive network models of epidemic spreading, in our model the epidemic threshold has a strong correlation with the topology of the network, which remains relatively unchanged at criticality, and the social structure of connections when epidemics begin to propagate is crucial in the state of the final outbreak. We are currently extending this work to weighted networks [23].

[1] M. E. J. Newman, Phys. Rev. E **66**, 016128 (2002).

[2] A. Vespignani and G. Caldarelli, *Large Scale Structure and Dynamics of Complex Networks* (World Scientific, Singapore, 2007).

[3] T. Gross and H. Sayama, *Adaptive Networks: Theory, Models and Applications* (Springer, New York, 2009).

[4] I. B. Schwartz and L. B. Shaw, Physics **3**, 17 (2010).

[5] T. Gross and B. Blasius, J. R. Soc. Interface **5**, 259 (2008); F. Vazquez, V. Eguíluz and M. San Miguel, Phys. Rev. Lett. **100**, 108702 (2008)

[6] T. Gross, C. D. J. D'Lima, and B. Blasius, Phys. Rev. Lett. **96**, 208701 (2006).

[7] R. M. Anderson and R. M. May, *Infectious Diseases of Humans* (Oxford University Press, Oxford, 1992).

[8] K. Eastwood, D. N. Durrheim, M. Butler, and A. Jon, Emerg. Infect. Dis. **16**(8), 1211 (2010).

[9] L. B. Shaw and I. B. Schwartz, Phys. Rev. E **77**, 066101 (2008).

[10] D. H. Zanette and S. Risau-Gusman, J. Biol. Phys. **34**, 135 (2008).

[11] S. Funk, E. Gilad, C. Watkins, and V. A. A. Jansen, Proc. Natl. Acad. Sci. USA **106**, 6872 (2009).

[12] V. Colizza, A. Barrat, M. Barthélemy, and A. Vespignani, Proc. Natl. Acad. Sci. USA **103**, 2015 (2006).

[13] This assumption is valid at and below criticality, as the low density of infected nodes makes collisions between infected branches (loops) unlikely.

[14] R. Cohen and S. Havlin, *Complex Networks: Structure, Robustness and Function* (Cambridge University Press, Cambridge, 2010).

[15] Z. Wu, C. Lagorio, L. A. Braunstein, R. Cohen, S. Havlin, and H. E. Stanley, Phys. Rev. E **75**, 066110 (2007).

[16] R. Cohen, K. Erez, D. ben-Avraham, and S. Havlin, Phys. Rev. Lett. **85**, 4626 (2000).

[17] W. Aiello, F. Chung, and L. Lu, Exp. Math. **10**(1), 53 (2001).

[18] R. Albert and A. L Barabàsi, Rev. Mod. Phys. **74**, 47 (2002).

[19] For $K \to \infty$ we have to replace p_c with $p_c(N)$ in Eq. (4). Recently, Wu *et al.* [15] observed that the aproximation $p_c \cong p_c(N)$ is only valid in networks larger than 10^9 nodes.

[20] R. Pastor-Satorras and A. Vespignani, Phys. Rev. Lett. **86**, 3200 (2001).

[21] R. Albert, H. Jeong, and A.-L. Barabási, Nature **401**, 130 (1999).

[22] R. Cohen, K. Erez, D. ben-Avraham, and S. Havlin, Phys. Rev. Lett. **86**, 3682 (2001).

[23] L. A. Braunstein, S. V. Buldyrev, R. Cohen, S. Havlin, and H. E. Stanley, Phys. Rev. Lett. **91**, 168701 (2003).