Effects of local and global network connectivity on synergistic epidemics

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Epidemics in networks can be affected by cooperation in transmission of infection and also connectivity between nodes. An interplay between these two properties and their influence on epidemic spread are addressed in the paper. A particular type of cooperative effects (called synergy effects) is considered, where the transmission rate between a pair of nodes depends on the number of infected neighbors. The connectivity effects are studied by constructing networks of different topology, starting with lattices with only local connectivity and then with networks that have both local and global connectivity obtained by random bond-rewiring to nodes within a certain distance. The susceptible-infected-removed epidemics were found to exhibit several interesting effects: (i) for epidemics with strong constructive synergy spreading in networks with high local connectivity, the bond rewiring has a negative role in epidemic spread, i.e., it reduces invasion probability; (ii) in contrast, for epidemics with destructive or weak constructive synergy spreading on networks of arbitrary local connectivity, rewiring helps epidemics to spread; (iii) finally, rewiring always enhances the spread of epidemics, independent of synergy, if the local connectivity is low.

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I. INTRODUCTION

Dynamical processes on networks is a subject of broad interdisciplinary interest and intensive study [1,2]. In particular, network models provide a unique framework to describe a wide range of spreading processes, including the spread of infectious diseases, social phenomena, or biological species [2–4]. Such models assume a graph representation of systems with nodes (vertices) that can be in several states specific to the spreading process (e.g., a host infected by a pathogen or a patch occupied by some species). The state of nodes can change due to interactions with other nodes connected by links (edges or bonds) to a recipient. For instance, such interactions may represent transmission of infection, opinions, behaviors, or ecological migrations.

The chances for a spreading phenomenon to affect a large number of nodes in a network, i.e., to invade a network, depend on both the dynamics of interaction between nodes and the topology of the network [2,5–9]. The simplest type of interaction is a pairwise interaction when only two nodes are involved in transmission, e.g., an infected donor and a susceptible recipient. The process of transmission can be characterized by two parameters, namely the rate of transmission (or probability of transmission in discrete-time description) and the time of interaction (time of existence of the contact or link between the donor and recipient), which can be a random variable. If the transmission rate is a fixed constant parameter, then the transmission of infection is a homogeneous Poisson process, i.e., simple transmission. However, in real situations the transmission can be a much more complex process. In particular, both the lifetime of nodes in different states and the transmission rates can depend not only on the characteristics of the donor-recipient pair but also on the characteristics of other nodes. In other words, cooperative effects due to multiple-node interactions, which are called synergistic effects below, can affect the values of parameters characterizing transmission. For example, the transmission rate can depend on the number of infected neighbors of a recipient. This number can change with time throughout the course of an epidemic, and thus the transmission rate can change with time. These abrupt stepwise changes in transmission rate due to cooperative effects have significant and nontrivial effects on the spreading process.

The role of network topology in the ability of epidemics to invade the network can be of crucial importance. For example, assuming simple transmission, an invasion can be much easier in globally connected networks such as complete or random graphs than in lattices where the nodes are locally connected to their nearest neighbors in space. However, it is not clear a priori what the effects of cooperative or interfering synergistic phenomena in transmission would be on invasion in networks of different topology. For example, if the local connectivity in lattices is reduced by the rewiring of some bonds, which enhances global connectivity, would this necessarily result in increased invasion ability for epidemics with synergistic effects? This and other related questions are addressed in the paper. Before we describe our model, we give a brief overview of existing models accounting for simple and complex transmission in spreading phenomena in networks of different topology.

Models assuming simple transmission dynamics have provided good insight into some aspects of the interplay between...
the features of transmission and network topology. This is indeed the case for network models for epidemic spread, which often assume that the transmission of infection between a pair of donor-recipient nodes is independent of the rest of the nodes connected to the pair [10,11]. A similar assumption was made in some models for the spread of social phenomena [12]. These models predict that invasions are facilitated in networks with small local clustering [12–15] and long links that can act as bridges for the transmission of infection [12,16–18]. The size of invasions with simple transmission is therefore minimized in regular lattices (with relatively large clustering and no shortcuts) and maximized in random graphs (small clustering and many shortcuts). Small-world (SW) network topologies bridge the gap between lattices and random graphs by means of either random bond rewiring [16,19,20] or adding random shortcuts [17,18]. The size and chance of simple invasions increase with the probability of either rewiring (i.e., by reducing local and increasing global connectivity) or adding shortcuts (increasing global connectivity) [16–18,21–24].

Many social and biological systems involve complex transmission dynamics that are often characterized by synergistic effects for donor-recipient pairs of nodes. These effects are not captured by simple epidemiological models, but they can significantly change the dynamics of spreading processes. Knowledge and understanding of complex transmission dynamics on spreading and its interplay with network topology is rather limited and is a topic of active research [7,25–36].

Synergistic effects can be either constructive or interfering (destructive). Constructive synergistic effects from the neighborhood of a recipient node were explicitly observed in experiments on the spread of behavior [27] and fungal invasion [30]. Social reinforcement was proposed as a key synergistic effect making invasions of social phenomena more likely and larger in clustered networks than in random graphs (i.e., opposite to the predictions obtained assuming simple transmission). This conclusion was supported by models involving social reinforcement from multiple neighbors [26,28,31,33]. The authors of Ref. [31] went a step further suggesting that these types of invasions are, in fact, optimal on SW networks rather than in fully clustered lattices. Interfering synergistic effects associated with, e.g., behavioral responses to epidemic spread [26–28,31,33], we show that rewiring systematically leads to larger invasions for weak local connectivity. We illustrate these and other effects with numerical simulations and analytical results for a simple model [32] based on an approximate mapping SIR synergistic spread to uncorrelated dynamical percolation (such mapping is exact in the absence of synergy [10,40–42]).

The structure of the paper is the following. The model is introduced in Sec. II, and the results of its numerical analysis are given in Sec. III. The analytical results are presented and compared with the results of numerical simulations in Sec. IV. The conclusions are given in Sec. V. Some technical details are discussed in Appendices A, B, C, and D.

II. MODEL

Let us consider a network consisting of $N$ nodes arranged on a regular two-dimensional lattice, with each node connected to the same number of nearest neighbors, $q > 2$. In particular, we studied honeycomb ($q = 3$), square ($q = 4$), and triangular ($q = 6$) lattices in which the bonds connecting nearest neighbors can be rewired with probability $\phi$ to a randomly chosen node under the constraint of no self- or double bonds. The probability of rewiring was assumed to be dependent of the states of the nodes (cf. Refs. [25,43]). Two types of models for bond rewiring were considered: (i) Spatial small-world (SSW) networks with rewiring to a random node within a finite distance, $R \in [R_{\min}, R_{\max}]$, where $R_{\min} > 0$ and $R_{\max}$ are parameters of the model, which are assumed to be independent of the lattice size [see Fig. 1(a)]; (ii) SW networks with rewiring to any random node within the system [see Fig. 1(b)], which is the limiting case of a spatial SW if $R_{\min} = 1$ and $R_{\max}(L) \sim L \rightarrow \infty$. Here, $L$ is the linear size of the system. In SSW networks, the rewiring is local, which contrasts with SW networks where it is global. The bond rewiring was performed in the following way. Consider for concreteness a square lattice (see Fig. 1). It can be constructed by an $N$ times repeated translation of a node with two bonds attached to it (horizontal and vertical) over a distance $a = 1$ in both the horizontal and vertical directions, producing $N = L \times L$ nodes on a square grid. These two bonds attached to each node are then rewired with probability $\phi$ (per bond) to a random node within the range $[R_{\min}, R_{\max}]$ subject to no double bonds. Such a bond-rewiring algorithm similar to that used
in Ref. [16] does not preserve the degree distribution, in contrast to degree-preserving algorithms used in analysis of homogeneous SW networks [22,44], and it results in degree distribution [20], which differs from that of a random graph (see Appendix C for more details).

The dynamics of the SIR process is defined by the lifetime $\tau = 1$ of any node in the infected state and the rate $\lambda_{ij}$ of stochastic transmission of infection from node $i$, infected at time $t_i$, to an attached node $j$ in the S state. The SIR process with simple nonsynergistic Poisson transmission is described by a constant transmission rate, $\lambda_{ij} = \alpha$. This model has been extensively studied on networks, and it is well established that an SIR epidemic spreads (invades the network) if the transmission rate is greater than a critical value, $\alpha \geq \alpha_c$, which marks the epidemic or, equivalently, the invasion threshold [2,6,10,45]. In the presence of synergy, the transmission rate of infection from an infected node, $i$, to a susceptible neighbor, $j$, is a piecewise constant function. Stepwise changes occur after infection or removal events involving nodes $i,j$ or any other in their neighborhood. Such changes can be conveniently incorporated in numerical simulations using the event-driven continuous-time algorithm described in Appendix A.

Our aim was to investigate the effects of synergistic transmission and rewiring (varying both the probability and the range of rewiring) on the invasion threshold. We focused on a specific type of synergy associated with the number of infected neighbors of a susceptible recipient node (referred to as $r$-synergy in [29]). In this case, the individual transmission of infection from node $i$ to node $j$ occurs with the rate $\lambda_{ij}(t)$, which depends on the number $n_j(t)$ of infected neighbors of recipient node $j$ excluding the attacker $i$ [29,32]. The individual transmission between nodes $i$ and $j$ starts at time $t_i$ when the node $i$ is infected, and it stops at $t'_i$, the time of infection of node $j$, not necessarily by node $i$, or the time of recovery of node $i$, i.e., $t'_i = t_i + \tau$. The number $n_j(t)$ of infected neighbors of $j$ can vary with time in a stepwise manner for $t \in [t_i,t'_i)$. The time locations of the steps correspond to the stochastic infection and deterministic recovery events for the neighbors of node $j$ excluding $i$, and they depend on the history of the system at $t < t_i$.

In particular, we analyzed invasions for the following three functional forms of $\lambda_{ij}(t)$ given as implicit functions of $n_j(t)$: (i) Exponential rate,

$$\lambda_{ij}(t) = \alpha e^{\beta n_j(t)},$$

(ii) the linear approximation to the exponential rate in Eq. (1),

$$\lambda_{ij}(t) = [\alpha + \alpha \beta n_j(t)]\theta[1 + \beta n_j(t)],$$

(iii) and a linear rate [29,32],

$$\lambda_{ij}(t) = [\alpha + \beta n_j(t)]\theta[\alpha + \beta' n_j(t)].$$

Here, the Heaviside function takes the value $\theta(x) = 1$ for $x \geq 0$ and is zero otherwise. The expressions for these rates are valid for $t \in [t_i,t'_i)$. For times outside this interval, $\lambda_{ij} = 0$.

The rate $\alpha$ in Eqs. (1)–(3) refers to the inherent (synergy-free) transmission rate. The coefficient $\beta$ in Eqs. (1) and (2) accounts for constructive ($\beta > 0$) or destructive ($\beta < 0$) synergy and is assumed to be independent of $\alpha$. If $\beta > 0$ ($\beta < 0$), then the rate $\lambda_{ij}(t)$ can exceed (be smaller than) the inherent rate $\alpha$ in the presence of a finite number of infected neighbors of node $j$ (see Fig. 2). The choice of the exponential function ensures positive values of the transmission rate for all values of $\beta$. In the linear approximation to the exponential rate, the synergy contribution is proportional to the product of the inherent rate and synergy coefficient, i.e., $\alpha \beta$. However, it is possible that the synergistic effects do not depend on the inherent rate, which is described by the functional form given by Eq. (3), where the synergy rate, $\beta'$, is independent of $\alpha$.

III. NUMERICAL RESULTS

To study the effects of the model parameters ($\beta$ or $\beta'$, $R_{\text{min}}$, $R_{\text{max}}$, and $\phi$) on the invasion threshold, $\alpha_c$, we numerically analyzed the SIR process with synergistic rates defined by Eqs. (1)–(3) on both SSW and SW networks with periodic boundary conditions. All our simulations correspond to a linear
phase boundaries corresponding to different values of \( R \), the exponential rate given by Eq. (1) on a square lattice, \( β \) transmission rate depends on the synergy parameter \( λ \).

For any given set of parameters, the value of \( α_∗ \) was estimated by using finite-size scaling analysis for one-dimensional spanning epidemics \([29,46,47]\) in the case of SSW networks with finite-range rewiring, \( R_{\text{max}} \ll L \). For SW networks with infinite-range rewiring, we used a linear fit \([21]\) and/or infection point for the mass of the infinite cluster versus the inherent transmission rate \([11,17]\) (see Appendix B for details).

Figure 3 shows how the critical value of the inherent transmission rate depends on the synergy parameter \( β \) for the exponential rate given by Eq. (1) on a square lattice \( (q = 4) \). Results are shown for SSW [panel (a)] and SW [panel (b)] networks. Each line in the figures gives the invasion threshold \( α_∗ \) as a function of \( β \) for given \( φ \) and rewiring range. SIR epidemics in systems with \( α \) and \( β \) above (below) the invasion line are invasive (noninvasive). For fixed values of \( φ \), the critical transmission rate \( λ_∗ \) depends on two parameters \( α_∗ \) and \( β \) in such a way [see Eqs. (1)–(3)] that if \( β \) increases, then the value of \( α_∗ \) should decrease in order to keep the same value of \( λ_∗ \). Therefore, as expected, \( α_∗ \) decreases with increasing \( β \) for any fixed \( φ < 1 \), meaning that increasing synergistic cooperation systematically makes systems less resilient to epidemic invasion. The effect of rewiring probability on \( α_∗ \) is more involved, and the trend depends on the value of \( β \). For small rewiring probability \( (φ \ll 1) \), all the phase-separation lines intersect at a single model-dependent point \( (β_c, α_∗) \). This means that for a certain value of \( β = β_c \), the critical inherent transmission rate \( α_∗ = α_c \) does not depend on the rewiring probability \( φ \) [see the horizontal lines in Fig. 4 and dependence of the mass of the infinite cluster on \( α \) in Fig. 10(b) in Appendix B]. For values of \( β < β_c \), the critical threshold in \( α \) decays with increasing \( φ \) [see Figs. 4 and 10(a)]. This is the expected behavior for synergy-free epidemics in SW networks \([18]\). In contrast, for relatively strong synergy, \( β > β_c \), the critical value of \( α \) increases with rewiring probability [see Figs. 4 and 10(c)]. In other words, the more bonds rewired in the system, the more resilient it becomes to strongly synergistic SIR epidemics. This finding is in qualitative agreement with the social reinforcement effects discussed by Centola \([27]\). It is important to note, however, that this effect not only requires the synergistic effects to be constructive (i.e. \( β > 0 \)), but it also requires that they are strong enough so that \( β > β_∗ > 0 \).

To interpret the results presented above, it is helpful to analyze separately the effects of cutting local bonds and adding local or global connections, which are the two basic operations involved in rewiring. For large positive \( β \), removing short-range bonds alone increases the resilience of the system, while adding bonds on its own \([11,21]\) decreases its resilience to SIR epidemics (see Fig. 5). In the rewiring scenario, these two tendencies compete and the resulting effects on invasion depend on the synergy strength. The effect of enhanced resilience with increasing rewiring is observed only for relatively large values of the synergy parameter \( β \) when the constructive synergy helps the SIR process to evolve locally where support from the infected neighbors is strongest. The rewired bonds diminish the local connectivity and bring the infection to such remote parts of the system where there are practically no infected nodes that could support a further spread of infection. The loss in the ability to spread locally is more significant than the gain due to jumps to remote places.
where the advantages of high constructive synergy cannot be used efficiently. This is the reason why the addition of new rewired shortcuts can make the system more resilient.

The arguments presented above relied on the interesting property of the intersection point \((\beta_\ast, \alpha_\ast)\), which does not depend significantly on rewiring with \(\phi \ll 1\). However, the conclusions remain valid for any value of \(\phi\). In general, the intersection point of the phase boundaries for arbitrary \(\phi \in (0, 1)\) and for \(\phi = 0\) occurs at a point \((\hat{\beta}(\phi),\hat{\alpha}(\phi))\), which depends on \(\phi\). The function \(\hat{\alpha}(\phi)\) decreases monotonically with \(\phi\) from \(\hat{\alpha}(0) = \alpha_\ast\) to \(\hat{\alpha}(1) = \alpha_\ast < \alpha_\ast\). In contrast, \(\hat{\beta}(\phi)\) is a monotonically increasing function taking values between \(\hat{\beta}(0) = \beta_\ast\) and \(\hat{\beta}(1) = \beta_\ast > \beta_\ast\). These trends can be seen in Figs. 3(a) and 6(a) for networks with underlying square and triangular lattices, respectively.

Considering a fully rewired lattice with \(\phi = 1\) allows the effect of enhanced resilience to SIR epidemics with strong synergy to be predicted independently from the analysis of SW networks. Indeed, for \(\phi = 1\), the network becomes similar to a random (Erdős–Rényi) graph where local lattice connections are rare, resulting in the absence of small loops. Therefore, in this limit, the simultaneous presence of more than one infected node in the neighborhood of the recipient is very unlikely, and thus the synergy effects should be negligible. In other words, the epidemic threshold does not depend significantly on \(\beta\). Consequently, the phase boundary is close to a horizontal line in the \(\beta-\alpha\) plane and, importantly, this line can intersect the phase boundary for \(\phi = 0\) at the point \((\beta_\ast, \alpha_\ast)\) [see Figs. 3 and 6(a)]. This means that for \(\beta \gtrsim \beta_\ast\), the fully rewired system is more resilient to invasion than the original lattice without rewiring. Again, this result contrasts with the prediction from models with simple transmission, suggesting that, given a mean node degree, invasions are more likely in random graphs than in regular lattices [16–18,21–24]. For instance, the critical transmissibility \(T_c = 1 - e^{-\alpha}\) (a complementary quantity marking epidemic threshold, see Sec. IV for more detail) on a square lattice \((q = 4)\) is \(T_c = 1/2\), whereas it is \(T_c = 1/(q - 1) = 1/3\) in a random graph with \((k) = q = 4\). Our model reproduces this traditional behavior for \(\beta < \beta_\ast\). Such an effect becomes more pronounced for regular lattices with a higher coordination number. For example, in a triangular lattice \((q = 6)\), all the epidemics with an exponential transmission rate are invasive for relatively large values of \(\beta \gtrsim \beta_{\max} \simeq 4\). However, in the case of a fully rewired triangular lattice, the presence of an almost horizontal phase boundary at \(\alpha = \tilde{\alpha} \approx \alpha_\ast\) (with \(\alpha_\ast \approx 0.21 \pm 0.03\) for both the SW network and the SSW network with \(R \in [1, 1.3]\)) clearly demonstrates that all the epidemics become noninvasive for \(\alpha \lesssim \alpha_\ast\), including those that were invasive in nonrewired networks for \(\beta \gtrsim \beta_{\max}\) [see Fig. 6(a)].

In lattices with a relatively small coordination number (i.e., with weak local connectivity), the effect of rewiring-enhanced resilience to synergistic epidemics is not observed. This is due to the fact that the effects of synergy in such lattices are not very strong, and the phase boundaries in the lattices without rewiring are almost horizontal. This effect is illustrated in...
 rewiring, including relatively small ones. Similarly, the effect was observed for models with linear transmission rates given by Eqs. (2) and (3). Figure 7 summarizes our findings for the variety of models investigated. Here, we show the location of three sets of characteristic points \((\beta_c, \alpha_c)\) found for different models with the exponential [Eq. (1)], the linear approximation to the exponential [Eq. (2)], and linear [Eq. (3)] transmission rates defined on a square lattice. These points, as expected (see Sec. IV), belong to the corresponding phase separation lines, \(\alpha_c(\beta, \phi = 0)\), for models without rewiring \((\phi = 0)\).

It should be noticed that the position of the characteristic point \((\beta_c, \alpha_c)\) changes in a systematic way, moving down along the phase boundary (for lattices without rewiring, see the solid curve in Fig. 7) with increasing rewiring range, tending to the point corresponding to the infinite rewiring range. This can be understood as follows. For given \(\beta\) and \(\phi \ll 1\), increasing the maximum rewiring radius, \(R_{\text{max}}\), will increase the global connectivity while the local connectivity

Fig. 6(b) for the honeycomb lattice \((q = 3)\), where the invasion threshold is in the range \(1.02 \lesssim \alpha_c \lesssim 1.15\). In the fully rewired lattice, the critical inherent rate takes values \(\alpha_c = \tilde{\alpha} \approx 0.57 \pm 0.07\) for infinite-range rewiring and \(\alpha_c = \tilde{\alpha} \approx 0.62 \pm 0.06\) for finite-range rewiring with \(R \in [1, 13]\). In both cases, \(\alpha_c\) is practically independent of \(\beta\) and the invasion boundary is an almost horizontal line located at \(\tilde{\alpha}\), below the range of the invasion boundary corresponding to \(\phi = 0\). Therefore, it is not surprising that rewiring decreases the resilience of the system irrespective of the value of \(\beta\) [see Fig. 6(b)]. These results show that the effect of local social reinforcement in networks with weak local connectivity is not strong enough to compete with the gain in the spread efficiency achieved by the shortcuts and rewiring in the networks. This effect is not captured by existing models with social reinforcement [26–28, 31, 33].

The critical value of \(\tilde{\alpha}\), defined by the position of the horizontal phase boundary in the \((\beta, \alpha)\) plane for fully rewired lattices, can be found in terms of the bond-percolation threshold [10], \(T_c\), as

\[
\tilde{\alpha} = \ln(1 - T_c)^{-1},
\]

where \(T_c = \langle k \rangle / (\langle k \rangle - 1)\) [48–50]. The values of \(\tilde{\alpha}\) obtained numerically for infinite-range fully rewired lattices with triangular \((\tilde{\alpha} \approx 0.21 \pm 0.03)\), square \((\tilde{\alpha} \approx 0.35 \pm 0.04)\), and honeycomb \((\tilde{\alpha} \approx 0.57 \pm 0.07)\) geometries agree well with the values calculated from Eq. (4) of \(\tilde{\alpha} \approx 0.201 \pm 0.001\), \(\tilde{\alpha} \approx 0.336 \pm 0.002\), and \(\tilde{\alpha} \approx 0.558 \pm 0.005\) for the same lattice types, respectively.

The effect of rewiring-enhanced resilience to invasion does not change qualitatively for the variety of the models given a sufficiently high coordination number of the underlying lattice. In particular, we found this resilience for different ranges of rewiring, including relatively small ones. Similarly, the effect was observed for models with linear transmission rates given by Eqs. (2) and (3). Figure 7 summarizes our findings for the variety of models investigated. Here, we show the location of three sets of characteristic points \((\beta_c, \alpha_c)\) found for different models with the exponential [Eq. (1)], the linear approximation to the exponential [Eq. (2)], and linear [Eq. (3)] transmission rates defined on a square lattice. These points, as expected (see Sec. IV), belong to the corresponding phase separation lines, \(\alpha_c(\beta, \phi = 0)\), for models without rewiring \((\phi = 0)\).

It should be noticed that the position of the characteristic point \((\beta_c, \alpha_c)\) changes in a systematic way, moving down along the phase boundary (for lattices without rewiring, see the solid curve in Fig. 7) with increasing rewiring range, tending to the point corresponding to the infinite rewiring range. This can be understood as follows. For given \(\beta\) and \(\phi \ll 1\), increasing the maximum rewiring radius, \(R_{\text{max}}\), will increase the global connectivity while the local connectivity

FIG. 6. (Color online) Invasion phase diagrams for (a) triangular \((q = 6)\) and (b) honeycomb \((q = 3)\) lattices with finite-range rewiring with \(R \in [1, 13]\). An exponential rate given by Eq. (1) was used. The same line styles as in panel (a) for \(\phi \in [0, 1]\) were used in panel (b). The crossing point in (a) between the phase boundaries for networks without rewiring \((\phi = 0)\) and complete rewiring \((\phi = 1)\) corresponds to \(\beta = \beta_c \approx 0.9 \pm 0.1\).

FIG. 7. (Color online) The set of characteristic parameters \((\beta_c, \alpha_c)\) for several SSW models and the SW model (with small probability of rewiring, \(\phi \leq 4.5 \times 10^{-2} \ll 1\)) defined on a square lattice with (a) exponential [Eq. (1)], (b) linear approximation to the exponential [Eq. (2)], and (c) linear [Eq. (3)] transmission rates of infection for SIR epidemics. In each panel, the solid line shows the invasion threshold separating noninvasive (below the line) and invasive (above the line) regimes in networks without rewiring \((\phi = 0)\). In panel (a), the continuous line corresponds to part of the curve in Fig. 3. Different symbols refer to different models of rewiring within the range \(R \in [R_{\text{min}}, R_{\text{max}}]\) as marked in the figure legend.
remains similar. This has the effect of making the system more susceptible to invasion, and thus the larger the rewiring range, the smaller the critical inherent rate. Therefore, the crossing point with the monotonically decaying curve corresponding to \(\alpha_c(\beta, \phi = 0)\) shifts downward along this curve.

### IV. Analytical Results for a Model Without Correlations in Transmission

Synergistic epidemics with removal can be viewed as correlated dynamical bond-percolation [29]. In this mapping, the bond probability between two nodes \(i\) and \(j\) corresponds to the probability that node \(i\) infects \(j\) during its infectious period, \(\tau\),

\[
T_{ij} = 1 - \exp \left( - \int_{t_i}^{t_i + \tau} \lambda_{ij}(t) dt \right),
\]

where \(t_i\) is the infection time of node \(i\). The probability \(T_{ij}\) is usually called the transmissibility from node \(i\) to node \(j\). Synergy makes the transmission rate \(\lambda_{ij}(t)\) dependent on the infection history of the pair \(i-j\) and its neighborhood. Accordingly, transmissibility will in general be different for different pairs of hosts, i.e., transmissibility is heterogeneous over the set of pairs of hosts. This heterogeneity is annealed, i.e., it varies with time, as opposed to quenched heterogeneity, which is well studied for epidemics [41,51–57], and its effect on the spread of epidemics is not obvious. In addition, transmissibilities of sufficiently close donor-recipient pairs have common nodes in their neighborhoods, and they are not independent from each other if transmission is synergistic (since the infection history of close donor-recipient pairs neighborhoods can overlap). In Ref. [29], we showed that correlations in transmissibility can have a significant effect on invasion for large synergy. In spite of that, we found that the main features of invasion phase diagrams on lattices can be qualitatively described by a model that ignores spatial correlations but accounts for crucial spatial heterogeneity in transmissibilities [29,32]. Here, we extend this approach to obtain approximate analytical results that explain the rewiring-enhanced resilience reported above.

The critical transmissibility in rewired networks, \(T_c(\phi)\) (i.e., the bond-percolation threshold), coincides with the mean transmissibility, \(\langle T(\phi)\rangle\), in the system:

\[
\langle T(\phi) \rangle = T_c(\phi).
\]

The value of \(T_c(\phi)\) depends on the topology of the network through the rewiring probability, but it does not depend on \(\alpha\) or \(\beta' (\beta')\). The expression for the mean transmissibility, \(\langle T(\phi)\rangle\), involves averaging over degree distribution,

\[
\langle T(\phi) \rangle = \sum_k p_k(T_k),
\]

and averaging over possible challenge histories of recipients with a fixed number \(k\) of nearest neighbors, accounted for by \(T_k\) in Eq. (7).

Equation (6) is valid for heterogeneous transmissibilities [21,50,51], but it assumes the absence of correlations in transmissibilities for different bonds, which is true for non-synergistic SIR processes with a fixed removal time [53]. As argued above, such correlations are inherent for the spread of the synergistic SIR processes, and condition (6) does not hold in general [41,53,55,56]. However, assuming that Eq. (6) holds even for synergistic SIR processes leads to a quantitatively correct invasion phase diagram for small values of \(\beta' (\beta')\) and a qualitatively correct picture for relatively large values of \(\beta' \sim 1 (\beta' \sim \alpha)\) [29,32]. To analytically study the consequences of Eq. (6), we linearize the dependence of \(T(\phi))\) and \(T_c(\phi)\) on \(\phi\). This leads to an approximate condition for epidemic threshold that reads (see Appendix C for more details)

\[
T_c(\phi) - A_q \phi = 1 - e^{-\alpha \left[ 1 - q (\alpha, \beta', 0) B(\beta') \right]^{q-1}}
\]

\[
- 2 \frac{e^{-\alpha \left[ 1 - q (\alpha, \beta', 0) B(\beta') \right]^{q-1}}}{(q - 1) \phi} B(\beta') \frac{\partial \varphi}{\partial \phi}
\]

\[
\times \left[ \left( 1 - q (\alpha, \beta', 0) B(\beta') \right)^{q-2} \right],
\]

where \(T_c(\phi)\) is the bond-percolation threshold for a regular lattice with coordination number \(q\), and the non-negative functions \(s_q(\alpha, \beta', \phi), \hat{B}(\beta'), \text{ and } A_q\) are introduced in Appendix C.

Equation (8) can be solved for \(\phi(\alpha, \beta')\), resulting in

\[
\phi = \frac{T_c(\phi) - 1 - e^{-\alpha \varphi} \left[ 1 - q (\alpha, \beta', 0) B(\beta') \right]^{q-1}}{F_q(\alpha, \beta')}
\]

(9)

if \(F_q(\alpha, \beta') \neq 0\), where

\[
F_q(\alpha, \beta') = \left( A_q - e^{-\alpha} \left[ 2 \left( 1 - q (\alpha, \beta', 0) B(\beta') \right)^{q-2} \right.ight.
\]

\[
+ 2 \left[ 1 - q (\alpha, \beta', 0) B(\beta') \right]^{q-1}
\]

\[
- 4 \left( 1 - q (\alpha, \beta', 0) B(\beta') \right)^{q-1}
\]

\[
+ (q - 1) B(\beta') \left[ 1 - q (\alpha, \beta', 0) B(\beta') \right]^{q-2} \frac{\partial \varphi}{\partial \phi} \right].
\]

(10)

For a fixed value of \(\beta'\), Eq. (9) defines how the critical value of the inherent transmission rate varies with rewiring probability.

For synergy-free epidemics with \(\beta' = 0\), the values of \(q = 0\) and \(F_q(\alpha, \beta') = 0 = A_q\). Accordingly, the critical inherent rate decreases with rewiring probability,

\[
\alpha_c = \alpha_c(0) - \ln \left( 1 + \frac{A_q \phi}{1 - T_c(\phi)} \right) \approx \alpha_c(0) - \frac{A_q \phi}{1 - T_c(\phi)},
\]

(11)

where \(\alpha_c(0) = -\ln(1 - T_c(\phi))\) is the critical transmission rate in the lattice without rewiring and synergy. However, for increasing synergy, the decay of \(\alpha_c\) with increasing rewiring probability becomes less pronounced, and eventually, depending on \(s_q(\alpha, \beta')\), it can become an increasing function. This happens at a characteristic value of \(\beta' = \beta_0\), when \(\alpha_c = \alpha_0\) does not depend on \(\phi\). Within the linear approximation for \(\phi \ll 1\), this is possible when both the numerator and the denominator in Eq. (9) are simultaneously equal to zero, i.e.,

\[
T_c(\phi) - 1 - e^{-\alpha \varphi} \left[ 1 - s_q(\alpha, \beta', 0) B(\beta') \right]^{q-1} = 0,
\]

(12a)

\[
F_q(\alpha, \beta') = 0.
\]

(12b)
On the one hand, Eq. (12a) gives the phase boundary for invasion in the absence of rewiring. On the other hand, the condition imposed by Eq. (12b) ensures that the solution (\(\beta_c, \alpha_c\)) does not depend on \(\phi\), as was found numerically (see Fig. 7).

For given \(\beta\), the solution of Eq. (9) for \(\alpha_c(\phi)\) agrees qualitatively with numerical simulations. In the case of triangular [see Fig. 8(a)] and square [see Fig. 8(b)] lattices, the change from a decrease of \(\alpha_c\) with increasing \(\phi\) for \(\beta' < \beta_c\) to an increase for \(\beta' > \beta_c\) is observed. At a characteristic value \(\beta' = \beta_c^*\), the inherent rate does not depend on \(\phi\). For honeycomb lattices [see Fig. 8(c)], no such transition is seen and the critical inherent rate only decreases with rewiring probability \(\phi\).

The model is accurate for \(\beta = 0\) when there are no synergy effects and thus no correlations in the transmission between different pairs of nodes (see the top lines in Fig. 8). Significant deviations between numerical data and model predictions are seen for larger values of \(\beta\) and \(\phi\), and they are due to approximations ignoring correlations in transmission. In spite of that, the model still provides qualitatively correct tendencies in \(\alpha_c(\phi)\) for different values of \(\beta'\) in various lattices.

Interestingly, in the case of just cutting or adding bonds, the condition given by Eq. (12b) is not satisfied for any \(\beta'\), meaning that \(\alpha_c\) varies with \(\phi\) as a monotonically decreasing or increasing function for just cutting or adding bonds, respectively (see Appendix D).

V. CONCLUSIONS

To conclude, we have investigated the effects of local and global connectivity on the spread of synergistic epidemics. The underlying networks used in the analysis were two-dimensional lattices with different coordination numbers (honeycomb, square, and triangular). The local and global connectivity in these networks were changed by means of local (finite-range) and global (infinite-range) random bond rewiring. The global bond rewiring produced two-dimensional small-world networks, while the local rewiring created spatial small-world networks with geographical constraints on the finite length of rewired bonds. SIR epidemics with constructive and destructive synergy transmissions were analyzed on such networks. Our main findings are the following:

(i) Bond rewiring enhances resilience to synergistic epidemics if two conditions are satisfied. First, the synergy effects are sufficiently strong and, second, the local connectivity is high enough. More specifically, the effect of rewiring-enhanced resilience is found only on lattices with a high coordination number (\(q \geq 4\)) and synergy strength \(\beta > \beta_s > 0\). This finding is in line with those in Refs. [26–28,31,33].

(ii) Independent of local connectivity, if constructive synergy is not strong enough, i.e., \(0 < \beta < \beta_s\), or if synergy is destructive (\(\beta < 0\)), rewiring enhances the spread of (reduces the resilience to) epidemics. In other words, destructive and weakly constructive synergy do not change qualitatively the behavior of synergy-free epidemics in rewired (small-world) networks [17,18,21,23,24]. In particular, the fact that the traditional framework is recovered for \(0 < \beta < \beta_s\) challenges the statement of Refs. [26–28,31,33] showing that rewiring-enhanced resilience of epidemic invasion does not occur for every constructive synergistic mechanism.

(iii) Independent of the strength of the synergy (constructive or destructive), if the local connectivity is small enough, the rewiring always decreases the resilience of the network to SIR epidemics. In particular, we have demonstrated this effect for epidemics in rewired honeycomb lattices (\(q = 3\)).

All three effects are quite robust to changes in the functional form of the synergy transmission rate. In our approach, synergy is modeled by the dependence of the transmission rate of infection between a donor-recipient pair on the number of infected neighbors of the recipient. Three types of functional dependence of the transmission rate on the number of infected neighbors of donor-recipient pairs were investigated. Similar effects of local and global connectivity on the spread of synergistic epidemics were found for all of them. The strength of synergistic effects was controlled with a single parameter, \(\beta\), which allowed both constructive (\(\beta > 0\)) and destructive (\(\beta < 0\)) synergy effects to be studied. This might be considered as an advantage of our model relative to other approaches typically studying one type of synergy, either constructive or destructive [26,28,31]. The effects reported here correspond to small-world networks obtained with a rewiring strategy, which brings heterogeneity in the node degree. However, such heterogeneity is expected to play a secondary role in synergistic effects (e.g., rewiring-enhanced resilience) compared to rewiring-induced changes in local and global connectivity. In particular, we
expect a similar interplay between synergy and local and global topology when using a rewiring strategy leading to small-world networks with a homogeneous node degree \[22,44\].

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**APPENDIX A: ALGORITHM**

In this appendix, we describe the rules of the SIR process and algorithm used in the simulations. The SIR process can be described as a trajectory in discrete state space with the state vector having \(N\) components that can have three discrete values corresponding to different states of the nodes (S, I, and R) resulting in \(3^N\) states in total, \([S]\). The process evolves by means of instantaneous jumps between the states \(S_i\). These jumps occur at times \(t_i\) (elapsed from the start of the process at \(t = 0\)) when the system rests in state \(S_i\) and the trajectory is an ordered-in-time sequence of states \(S_i(t_i)\). The jumps can occur only between the states described by state vectors that differ in one component only, i.e., only one node changes its individual state after the jump. Only one of two changes is possible in one event: infection, i.e., \(S \rightarrow I\) or removal, i.e., \(I \rightarrow R\).

The time intervals between jumps are defined by the dynamical rules of the process, namely by the rules for infection and removal. The removal rule states that a node becomes deterministically removed after time \(\tau\) (the parameter of the model) elapsed since the moment of infection of this node. For example, if node \(j\) has been infected at time \(t_j\) with the system being in state \(S_j\), it is removed at time \(t_k = t_j + \tau\) when the system is in the state \(S_k\). The states \(S_j\) and \(S_k\) can be separated on the trajectory of the process by many other system states that correspond to infection and removal of other nodes. The individual transmission of infection from an infected node to a susceptible one connected to the infected node occurs stochastically at times given by a Poisson process. The rate of such processes remains constant during quiescent intervals of time between consecutive states, but, in the presence of synergy, its value can change after transition events.

For example, assume that the system jumps from the state \(S_{i-1}\) to the next state on the trajectory \(S_i\) at time \(t_i\) and the infection event occurs at time \(t_i\), i.e., a susceptible node \(k\) becomes infected. The process of infection of node \(k\) is a superposition of independent individual infection transmissions from all infected neighbors connected to \(k\), and it takes place with the rate \(\lambda_k = \sum_{m} \lambda_{mk}\), where \(m\) runs over all infected neighbors of \(k\). The value of \(\lambda_k\) does not depend on time for \(t \in [t_{i-1}, t_i)\), and it is fully defined by the state of the system \(S_{i-1}\) at \(t = t_{i-1}\), i.e., it does not depend on the previous history of the system at \(t < t_{i-1}\). In particular, the rate \(\lambda_k\) depends on the number of infected neighbors and on individual rates \(\lambda_{mk}\). In general, the rates \(\lambda_{mk}\) can also depend on the number of infected neighbors of node \(k\) (only for nonsynergistic epidemics are the values of \(\lambda_{mk}\) independent of the infected neighbors of \(k\)). After the infection event at time \(t = t_i\), infection rates between any infected-susceptible pairs of connected nodes may have changed and should be updated. Similarly, if a deterministic removal rather than infection event takes place at \(t_i\), then all the individual infection rates should also be updated.

Numerically, we aimed to sample without bias all possible trajectories of the SIR process. This can be achieved by means of kinetic Monte Carlo \(\[38\]\) simulations exploiting the Gillespie algorithm (direct method \(\[59,60\]\)) with modifications accounting for deterministic recovery events. Within this algorithm, the SIR trajectory was sampled as follows:

(i) Start simulations at \(t = 0\) by infecting a small number of nodes, \(N_0 \sim O(1) < N\), distributed randomly within the network. Create a list of infection events, i.e., the list of susceptible nodes linked to the infected nodes and the cumulative infection rate (sum of all individual infection rates) for each node in the list. Create a list of nodes in the infected state with their recovery times.

(ii) For a current time step \(t\), calculate the cumulative infection rate, \(R = \sum_{i,j} \lambda_{ij}\), where \(t\) runs over all infected nodes and \(j\) runs over susceptible neighbors of infected nodes connected by the links to them, i.e., the sum is evaluated over all possible individual infection transmissions in the network.

(iii) If \(R > 0\), calculate a uniformly distributed random number \(r_1 \in (0,1]\).

(iv) Calculate a step time until the next possible infection event, \(\Delta t = -\ln(r_1)/R\).

(v) Compare \(t + \Delta t\) with the time of the earliest deterministic recovery event, \(t_r(>1)\). If \(t + \Delta t \geq t_r\) or \(R = 0\), perform the recovery event at \(t_r\), update the list of the individual infection rates, set the current time \(t\) to \(t_r\), and return to step (ii).

(vi) If \(t + \Delta t < t_r\), calculate a uniform random number, \(r_2 \in (0,1]\).

(vii) Add the individual infection rates for nodes from the list of infection events cumulatively until it exceeds \(r_2R\). Infect the node for this event, update the infection rates and recovery times and set \(t = t + \Delta t\).

(viii) Return to step (ii).

The simulation stops when there are no nodes in the infected state. This algorithm is valid for both synergistic and synergy-free SIR processes. For a synergy-free process, the infection rates \(\lambda_{ij} = \alpha\) are identical for all the individual infection processes. In the case of synergy, the individual infection rates \(\lambda_{ij}\) entering the expression for \(R\) depend on the neighborhood of susceptible nodes as described in Sec. II.

**APPENDIX B: SCALING ANALYSIS**

In this appendix, we present the results for the finite-size scaling analysis performed to find the invasion threshold in the case of finite- and infinite-range rewiring. To find the invasion threshold, we exploit the fact that it corresponds to a critical point of the system. Following this property, in models with finite-range rewiring, we determined the invasion threshold using finite-size scaling for one-dimensional spanning clusters \(\[29,32,46,47\]\). The relative number of one-dimensional spanning clusters, \(N_1(\alpha, L)\), exhibits a maximum near the critical value of the inherent transmission rate, \(\alpha_\theta\) [see Fig. 9(a)], and the values of \(N_1(\alpha, L)L^{-\theta}\) for varying \(L\) should collapse onto a single master curve, \(\tilde{N}_1(\alpha)\), if plotted against \(\log_2 L = (\alpha/\alpha_\theta)^{1/\nu}\). The exponents \(\nu\) and \(\theta\) and the critical transmission rate, \(\alpha_\nu\), are found from the scaling collapse [see Fig. 9(b)]. The value of the universal exponent \(\nu \approx 1.3 \pm 0.1\)
is consistent with $v = 4/3$ [61,62] known for percolation in lattices without rewiring. This is due to the restriction that the maximum rewiring distance in FSS must be much smaller and independent of the linear system size, $R_{\text{max}} \ll L$. The finite-size scaling used in the case of finite-range rewiring models cannot be applied to infinite-range rewiring. This is due to the existence of an additional length scale related to the distance between the shortcuts [11,21,63]. Therefore, we used two complementary methods for estimating the critical threshold, $\alpha_c$. The first method is based on the fact that small-world networks can be described by a mean-field approximation, and thus the relative mass of the infinite cluster, $M$, depends linearly on $(\alpha - \alpha_c)$ near the critical point. A linear fit [21] was then used to estimate $\alpha_c$ [see the solid line in Fig. 10(a)]. Alternatively, the critical value of the inherent transmission rate can be found from the location of the inflection point on the curve for the mass of the infinite cluster, $M(\alpha)$ [11,17] [see the vertical dashed line through the inflection point in Fig. 10(a)]. The big error bars seen in Figs. 3 and 4 are due to the limited world sizes available in the small-world simulations. The position of the inflection point gives an upper estimate on the value of $\alpha_c$, while the linear fit provides a lower bound estimate. The restricted system sizes are due to the limited availability of processing power, which is required for large worlds with synergistic effects within the continuous-time kinetic Monte Carlo algorithm.

The different tendencies in $M(\alpha)$ with increasing rewiring probability, $\phi$, for small-world networks for different values of the synergy parameter are seen in Fig. 10: (a) $\beta < \beta_*$, the system becomes less resilient with increasing $\phi$; (b) $\beta \simeq \beta_*$, the mass of the infinite cluster practically does not depend on $\phi$; and (c) $\beta > \beta_*$, the system becomes more resilient with increasing $\phi$.

APPENDIX C: TECHNICAL DETAILS IN THE ANALYSIS OF THE ANALYTICAL MODEL

In this appendix, first we derive an approximate condition for the epidemic threshold given by Eq. (8), and then we give a simplified version for it. The condition for the epidemic threshold given by Eq. (6) depends on the bond-percolation threshold and mean transmissibility. To leading order in $\phi$ (with $\phi \ll 1$), the bond-percolation threshold for networks with rewiring in a finite range is given by

$$T_c(\phi) \simeq T_{c0} - A_q(R_{\text{min}}, R_{\text{max}})\phi,$$

where $T_{c0}(q)$ is the bond-percolation threshold for a regular lattice, and $A_q(R_{\text{min}}, R_{\text{max}}) > 0$ is a model-dependent constant.

To obtain a linear approximation for the dependence of $(T(\phi))$ on $\phi$, we first derive the expression for degree

FIG. 9. (Color online) (a) The relative number of one-dimensional spanning clusters, $N_1(\alpha, L)$, vs the inherent transmission rate, $\alpha$, for several lattice sizes, $L$, as indicated in the figure legend. (b) The scaling collapse of $N_1(\alpha, L)L^{-\nu}$ when plotted against $(\alpha - \alpha_c)L^\nu$ with $\alpha_c = 0.727$, $\nu \simeq 1.3 \pm 0.1$, and $\theta = -0.0522$ for SIR epidemics on a square lattice with finite-range rewiring $R \in [1,8]$ with $\phi = 0.025$ for an exponential form of the rate given by Eq. (1) with synergy parameter, $\beta = -1$. The different symbols in both panels correspond to different lattice sizes as indicated in the legend with each point averaged over 20 000 realizations of the epidemics.

FIG. 10. (Color online) The relative mass of the infinite cluster, $M$, for an SIR process on a square lattice of size $N = 201 \times 201$ with infinite-range rewiring vs the inherent transition rate, $\alpha$, for different values of the synergy parameter: (a) $\beta = -2$, (b) $\beta = 2 \simeq \beta_*$, and (c) $\beta = 5$ and for varying rewiring probability, $\phi$, as indicated in the legends (the same symbols and line styles are used in all panels). An exponential form of the synergistic transmission rate given by Eq. (1) was used. An example of the linear fit used for estimating the critical inherent transmission rate is shown by the solid line in (a) for $\phi = 0.015$, and the vertical dashed line goes through the inflection point for the $\phi = 0.015$ curve.
distribution in a network with rewiring (following Ref. [20]), and then we obtain an approximation for it in the case of small \( \phi \ll 1 \). For example, we consider the case of the square lattice \( q = 4 \) with bonds rewired according to the Watts-Strogatz rewiring algorithm as described in Sec. II, i.e., a SW network. According to this algorithm, for each node, \( Q = q/2 \) out of \( q = Q + Q_1 \) bonds attached to this node are rewired with probability \( \phi \). This means that an arbitrary node has at least \( Q \) bonds attached to it. The remaining \( Q_1 \) bonds can be broken by rewiring, so that only \( n_1 \) out of \( Q_1 \) are still attached to the node. The random number \( n_1 \) is distributed according to the binomial distribution, \( B_{n_1}(Q, \phi) \). In addition to \( Q + n_1 \) bonds, a random number \( n_2 \) of new bonds can be attached to the node as a result of bond rewiring from other nodes. This number is also distributed with binomial distribution \( B_{n_2}(NQ, \phi/N) \), with \( NQ \) being the total number of bonds in the system [the terms \( \sim O(N^{-1}) \) were ignored]. Therefore, the node degree \( k = Q + n_1 + n_2 \) is given by

\[
p_k(N, \phi) = \sum_{n=0}^{\min(k-Q, Q)} B_{n_1}(Q, \phi) B_{n_2}(NQ, \phi/N), \tag{C2}
\]

if \( k \geq Q \) and \( p_k = 0 \) otherwise. In the limit of large \( N \gg 1 \), the binomial distribution \( B_{k-Q-n}(NQ, \phi/N) \) tends to the Poisson one and

\[
p_k(N, \phi) \to p_k(\phi) \simeq \sum_{n=0}^{\min(k-Q, Q)} \left( \frac{Q}{n} \right)^n (1 - \phi)^{Q-n} \phi^n \left( \frac{k}{(k-n)!} \right) e^{-\phi}, \tag{C3}
\]

which does not depend on the system size and coincides with the degree distribution obtained for a ring with nodes connected to \( q \) nearest neighbors [20]. The convergence of \( p_k(N, \phi) \) to \( p_k(\phi) \), as demonstrated in Ref. [20], is rather fast, and for \( N \sim 10^3 \) the numerical data almost perfectly reproduce the limiting distribution given by Eq. (C3).

The limiting case for small rewiring probabilities, \( \phi \ll 1 \), follows from Eq. (C3). In this limit, mainly nodes with coordination numbers \( k = q - 1, q \) and \( q + 1 \) are present in the network, and \( p_k(\phi) \) is given by

\[
p_k(\phi) \simeq (1 - 4\phi)\delta_{k,q} + 2\phi\delta_{k,q-1} + 2\phi\delta_{k,q+1}. \tag{C4}
\]

The value of \( \langle T_k \rangle \) in the expression for mean transmissibility given by Eq. (7) takes into account the synergy effects, i.e., that the transmission of infection from a donor to a recipient can occur in the presence of different numbers of infected neighbors (excluding the donor) of the recipient \( n = 1, 2, \ldots, q - 1 \), which can affect the transmission rate and thus the transmissibility. Within the model of a time-dependent environment with the linear transmission rate given by Eq. (3), the mean transmissibility with fixed node degree \( k \) for \( \beta' > -\alpha/(k-1) \) is given by [32]

\[
\langle T_k \rangle = 1 - e^{-\alpha}(1 - s_k B(\beta'))^{\frac{k-1}{\beta'}} - \frac{s_k B(\beta')}{\beta'}, \tag{C5}
\]

where \( s_k = s_k(\alpha, \beta', \phi) \) (parameter of the model) is the probability that a neighbor of a recipient node (excluding the donor, i.e., one out of \( k - 1 \) neighbors) has been infected within the time interval \([\tau, \tau]\) if the donor became infected and started to challenge the recipient at \( t = 0 \). The function \( B(\beta') = 1 - (1 - e^{-\beta'})/\beta' \) increases monotonically from 0 to 1 with increasing \( \beta' \to \infty \).

![FIG. 11. (Color online) Dependence of (a) \( s_4 \), (b) \( s_5 \), and (c) \( s_6 \) on the inherent rate, \( \alpha \), for different values of the synergy parameter, \( \beta' \) [as marked in the legend in (c)] in square lattices with \( \phi = 0 \) in (b) and a small value of rewiring probability \( \phi = 2.5 \times 10^{-3} \) and \( R \in [1.4] \) in (a) and (c). The unlabeled curves show values of \( \beta' \) varying stepwise with unity from \( \beta' = 0 \) (lowest curve) to \( \beta' = 5 \) (topmost curve) except for the \( \beta'_s \) curve. Part (d) shows the variation of \( s_4(\alpha, \beta'_s) \) with rewiring probability \( \phi \). The open circles in panels (a)–(c) represent the location of the critical inherent rate, \( \alpha_c \), for each value of \( \beta' \), while the solid circles show the location of \( \alpha_c \).](image-url)
Combining Eqs. (7), (C5), and (C4) gives the desired linear approximation for \( T(\phi) \). Introducing this expression and Eq. (C1) into Eq. (6) leads to an approximate condition for the invasion threshold, \( \alpha = \alpha_c(\beta, \phi) \), given by Eq. (8), which can be transformed to Eq. (12).

The solution of Eq. (12b) depends significantly on the functional form of the infection probabilities \( s_q(\alpha, \beta', \phi) = 0 \), which can be found numerically in the same way as described in Ref. [32]. The dependence of \( s_q(\alpha, \beta', \phi = 0) \) on \( \alpha \) for \( q = 3, 4, \) and 5 and different values of \( \beta' \) are shown in Fig. 11. It follows from this figure that \( s_q(\alpha, \beta', \phi = 0) \ll 1 \) for all values of \( q \) (at least for the values close to the invasion boundaries).

As a consequence, the function \( s_q(\alpha, \beta', 0)B(\beta') \ll 1 \), and Eq. (12b) can be simplified as follows:

\[
T_{c0} = \{1 - e^{-\alpha(1 - (q - 1) s_q(\alpha, \beta, \phi) B(\beta'))}\} = 0. \tag{C6a}
\]

\[
A_q - e^{-\alpha} B(\beta') \left[ 4(q - 1)s_q(\alpha, \beta, 0) - 2(q - 2)s_q(\alpha, \beta, 0) - 2qs_q(\alpha, \beta, 0) + (q - 1) \frac{\partial s_q(\alpha, \beta, 0)}{\partial \phi} \right] = 0. \tag{C6b}
\]

Here, the derivative \( \partial s_q(\alpha, \beta, 0)/\partial \phi \) is typically much smaller than \( s_q(\alpha, \beta, 0) \), as seen from Fig. 11(d). If the expression in the square brackets is positive, Eq. (C6b) can be solved, with \( (\alpha, \beta', \phi) \) lying on the phase boundary for \( \phi = 0 \), which follows from Eq. (C6a).

**APPENDIX D: MODELS WITH ADDED AND CUT BONDS**

In this appendix, we study analytically and numerically two additional models by just adding and cutting the bonds instead of rewiring. In a finite-range rewiring model, each bond can be rewired with probability \( \phi \), i.e., the original bond is removed from the system and a new bond connecting two nodes within rewiring range is created. Let us modify this model in such a way that the original bond is kept in the network in addition to the added bond. This is a model with just added bonds [17,18]. For small values of the probability of adding a bond, \( \phi_{\text{add}} \ll 1 \), the node degree distribution for this model is given by the following equation:

\[
p_q = (1 - 4\phi_{\text{add}})\delta_{q,4} + 4\phi_{\text{add}}\delta_{q,5}. \tag{D1}
\]

The corresponding expression for \( F_q(\alpha, \beta' \) is given by

\[
F_q(\alpha, \beta') = A_q - e^{-\alpha} \left[ 4(1 - s_{q+1}(\alpha, \beta', 0)B(\beta'))^q - 4(1 - s_q(\alpha, \beta', 0)B(\beta'))^q - 2(q - 1)B(\beta')(1 - s_q(\alpha, \beta, 0)B(\beta'))^q - 2qs_q(\alpha, \beta', 0) + (q - 1) \frac{\partial s_q(\alpha, \beta', 0)}{\partial \phi_{\text{add}}} \right]. \tag{D2}
\]

The dependence of \( \alpha_c(\phi) \) is given by Eq. (9) with \( F_q(\alpha, \beta') \) obeying Eq. (D2). The results of its numerical solution are shown in Fig. 12(a). It can be seen from this figure that the critical inherent rate decreases with probability \( \phi_{\text{add}} \) irrespective of the value of \( \beta \). Such a monotonic trend is expected and agrees with the fact that Eq. (8), which gives a necessary condition for \( \alpha_c \), to be independent of \( \beta \), is not satisfied for any \( \beta \). The analytical results are well supported by the results of the numerical simulations for \( \phi_{\text{add}} \ll 1 \) and \( \beta \ll 1 \).

Alternatively, the original rewiring model can be modified in such a way that the original bonds are cut with probability \( \phi_{\text{cut}} \) but new bonds are not added to the network. This is a model with just removed bonds. The node degree distribution for this model is given by the following equation:

\[
p_q = (1 - 4\phi_{\text{cut}})\delta_{q,4} + 4\phi_{\text{cut}}\delta_{q,3}. \tag{D3}
\]
The corresponding expression for $F_q$ is given by
\[
F_q(\alpha, \beta') = A_q - e^{-\alpha} \left[ 4(1 - s_{q-1}(\alpha, \beta', 0)B(\beta'))^{q-2} - 4(1 - s_q(\alpha, \beta', 0)B(\beta'))^{-q-1} + (q-1)B(\beta')(1 - s_q(\alpha, \beta', 0)B(\beta'))^{q-2} \frac{\partial s_q(\alpha, \beta', 0)}{\partial \phi_{\text{cut}}} \right] \approx A_q - e^{-\alpha} B(\beta') \left[ 4(q-1)s_q(\alpha, \beta', 0) - 4(q-2)s_{q-1}(\alpha, \beta', 0) + (q-1) \frac{\partial s_q(\alpha, \beta', 0)}{\partial \phi_{\text{cut}}} \right],
\]
\[(D4)\]

The dependence of $\alpha_c(\phi)$ is given by Eq. (9) with $F_q(\alpha, \beta')$ obeying Eq. (D4). The results of its numerical solution are shown in Fig. 12(b). In this case, the critical inherent rate expectedly increases with $\phi_{\text{cut}}$. The numeric results are again supportive of the analytics for relatively small values of $\beta$, and they show qualitatively the same behavior for $\beta \gtrsim 1$.