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Epidemics on evolving networks with varying degrees

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Abstract

Epidemics on complex networks is a widely investigated topic in the last few years, mainly due to the last pandemic events. Usually, real contact networks are dynamic, hence much effort has been invested in studying epidemics on evolving networks. Here we propose and study a model for evolving networks based on varying degrees, where at each time step a node might get, with probability r, a new degree and new neighbors according to a given degree distribution, instead of its former neighbors. We find analytically, using the generating functions framework, the epidemic threshold and the probability for a macroscopic spread of disease depending on the rewiring rate r. Our analytical results are supported by numerical simulations. We find that the impact of the rewiring rate r has qualitative different trends for networks having different degree distributions. That is, in some structures, such as random regular networks the dynamics enhances the epidemic spreading while in others such as scale free (SF) the dynamics reduces the spreading. In addition, we unveil that the extreme vulnerability of static SF networks, expressed by zero epidemic threshold, vanishes for only fully evolving network, $r = 1$, while for any partial dynamics, i.e. $r < 1$, this zero threshold exists. Finally, we find the epidemic threshold also for a general distribution of the recovery time.

1. Introduction

Following the fundamental works on epidemic processes [1–4], the study of epidemics on *complex networks* [5–7] has attracted significantly the network science community, and yielded many studies [8–18], which explore the epidemic threshold for a major outbreak as well as the epidemic dynamic information for a variety of static networks and for several epidemic models. However, real networks of epidemiological contacts are usually not stationary but show significant dynamic patterns, which challenge the theory for epidemics on static networks [19–22].

Hence, many studies have investigated epidemic processes on *temporal networks* [23–36]. Few temporal contact network models have been proposed and studied in the context of epidemic spread. Volz and Meyers [25] examined the effect of social mixing on susceptible–infectious–recovered (SIR) disease. They considered *neighbor exchanges*, in which pairs of edges are selected uniformly randomly and swapped, at a fixed mixing rate. Thus, each individual maintains a fixed number of concurrent contacts while the identities of the contacts change stochastically over time. They found that the epidemic threshold depends also on the rate at which the network changes over time, in addition to the properties of the disease and the network topology. Further studies [29–31] assumed, rather than an exchange between two random edges, that one side of an edge might be rewired to a random node, or that the edge might be deleted, resulting in non-stationary degrees. Perra *et al* [32] introduced the *activity-driven network* model, where each node is assigned a time-invariant activity rate. Then, at each time step, each node becomes active with its activity rate, and forms fixed m connections with random nodes. All connections are cleared between time steps. Several works have been done on epidemics on the activity-driven network model and its extensions [33–35]. Prakash *et al* [36] examined the epidemic threshold under SIS dynamics for arbitrary temporal

networks, represented by a sequence of T static network snapshots with adjacency matrices $A = \{A_1, A_2, \ldots, A_T\}$. They showed that the epidemic threshold is then characterized by the maximal eigenvalue of the product of T matrices.

Here, we propose and analyze a model of temporal network based on any degree distribution, where according to some rewiring rate, r, each node samples new neighbors according to a *new degree* sampled from the given degree distribution. This assumption represents a real scenario where people change, at some points of time, their neighbors and number of neighbors, e.g. when they take part in different events which might have remarkable different sizes and different participants, or when they play a varying social roles within events which might lead them to considerably varying exposure. We keep our model simple and focused on this evolution characteristic, in order to fully analyze and recognize the effects of the rewiring phenomenon. We study on this evolving network model the SIR model. Using an analytical approach based on the generating functions framework, we find the epidemic threshold and the probability of a major outbreak, depending on the rewiring rate r, as well as the properties of the network and the infection rate β. One of the main questions we address is: does the dynamics in the connections enhance the capability of the epidemic to spread, or the opposite, mitigate it? We find that the answer interestingly varies for different structures of the network, which implies two opposite effects of the rewiring on the epidemic as we discuss below. While the dynamics *strengthens* the epidemic spread in random regular (RR) networks, it *weakens* the spread in scale free (SF) networks. In Erdős–Rényi (ER) network the trend depends surprisingly on the value of r. We derived analytical theory which predicts quantitatively these surprising phenomena. Another interesting result that we obtain, is about SF networks. They are known, in the static case, to be extremely vulnerable to epidemics, such that for any non-zero infection rate the epidemic can spread, that is, $\beta_c = 0$ [8, 37]. However, in the dynamic case, we find that if $r = 1$, that is, the network changes fast relative to the infection time, then β_c becomes nonzero, namely the evolving network is dramatically more robust against epidemics for this case. In marked contrast, as long as $r < 1$ (even very close to 1) the epidemic threshold is zero for large enough network. We also discuss the relation between epidemics and directed percolation (DP) [38, 39] which can be regarded as the inspiration for our model. We find that the SIR model on evolving networks can be rigorously mapped to a modification of the DP model. This new mapping completes a picture within statistical physics, since SIS model on static and evolving networks is mapped to DP [38], and SIR model on *static* networks is mapped to *bond percolation* [9, 40, 41], but mapping for SIR on *evolving* networks was missing. Finally, we further explore the case of a general nonuniform recovery time and particularly where there is a recovery rate γ, instead of taking the recovery time to be a single time step. We find for this case the phase diagrams that show the conditions for an epidemic spread depending on the rewiring, infection, and recovery rates, (r, β, γ) and the degree distribution p_k.

2. Model

We consider the stochastic SIR model in discrete time. According to this model, if an agent is susceptible and it has a contact with an infectious node at some time step, then it is infected by the infective node with probability β. Once it is infected, it can infect others at the following time step. For simplicity, we analyze first the case in which an infected node can infect others only at the next time, and then it is recovered and cannot infect nor become infected anymore. Below, we further analyze the more complicated case that an infected node is recovered with probability γ at each time step.

We present here our model for the evolving network on which the SIR epidemic process takes place. Initially, we build a random network with some degree distribution p_k. Then, at each time step, each node is chosen with probability r to switch its degree and neighbors. Next, we shuffle the neighbors of these chosen nodes in the following way. First, we shuffle the degrees of these nodes, hence each node gets randomly a new degree from the pool of degrees of all chosen nodes, thus the degree distribution is conserved. This is since the nodes not selected preserve their degree and the chosen nodes just exchange degrees. Next, we shuffle the neighbors of the chosen nodes, such that each one gets random neighbors according to its new degree from the pool of the neighbors of the chosen nodes. See figure 1 for a demonstration of our model for the evolution of the network.

Next, we describe how the epidemic process and the evolution process of the network integrate with each other. The infections take place at each time step according to SIR model, whereas the rewiring occurs between time steps. We also assume the recovery occurs between time steps.

To summarize, we have the following parameters governing the system behavior: β infecting rate, r rewiring rate, and for the case that the recovery time is not one, γ recovery rate.

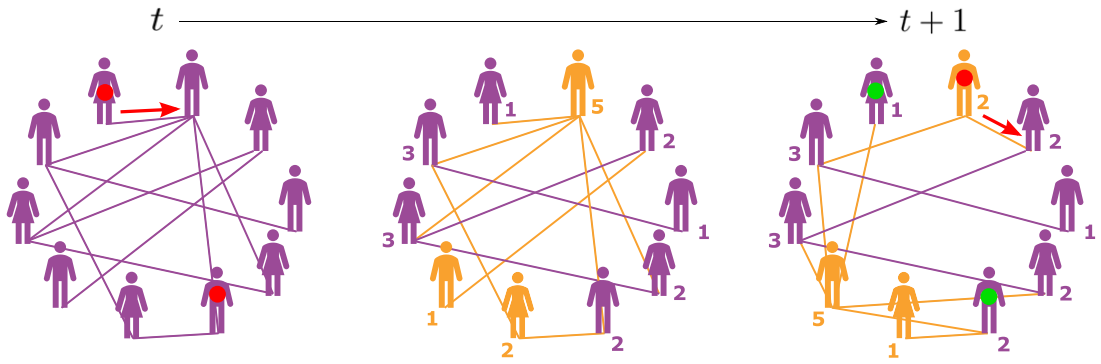

Figure 1. Illustration of our evolving network model. When rewiring, first we choose randomly r nodes (in orange) to switch neighbors and degree, while other nodes (purple) conserve their degree. Here $r = 0.3$, hence, the outgoing links from the three chosen nodes (orange) are rewired while other links remain. The degrees of the chosen nodes are shuffled randomly, and also their neighbors are shuffled randomly between them. One can see that the degrees of the chosen nodes changed while the degrees of the other nodes were preserved. In such a way, the degree distribution p_k is conserved, and a fraction r of nodes samples new degrees and neighbors. However, notice that also other nodes get some changes in the identities of their neighbors, since if they have a neighbor which is a chosen node, it abandons and leaves someone else instead. The red circles and arrows demonstrate an effect of the rewiring on the epidemic spread. At time t there are two infectious people (red circles). The red arrows represent actual infections. A person with many neighbors is more likely to get infected by one of them. Here comes a major difference between static and dynamic networks. While in the former, the infected node preserves its degree, which tends to be high, in the latter it gets a new degree which is probably smaller. This reduces the epidemic capability to be spread. An opposite effect is that the parent of the infected node is now immunized (green circles) and thus cannot spread the disease, thus switching neighbors has some advantage for spreading. We take all these considerations into account and provide formulas to predict the epidemic spread.

3. Fixed infectious time $\tau = 1$

For simplicity, we consider first the case in which a node can infect only at the following time after it gets infected, then it gets recovered. That is, the recovery time is $\tau = 1$. We look for the probability P_{inf} that a random node infected from outside the system will lead to a macroscopic infection, namely, at the end of the process, an extensive fraction of nodes has been infected in an infinite network. We further look for the critical infection rate β_c for the spread of disease, which represents the transition from zero probability of infinite infection to a nonzero probability.

The probability P_{inf} that a random infected node will lead to an outbreak, that is, spreads the epidemic to an infinite group, is the likelihood that it infects at least one of its neighbors and the latter will spread the contagion to infinite, see demonstration in figure 2(a). Clearly, it depends on its degree k. Thus, the probability, $1 - P_{\text{inf}}$, that the infectious node will not spread the epidemic to an infinite number of nodes is,

$$1 - P_{\text{inf}} = \sum_{k=0}^{\infty} p_k \left((1-r) v_p^k + r v_{np}^k \right). \tag{1}$$

The rhs captures the probability that the infectious node (node p in figure 2(a)) does not spread the epidemic to infinity through anyone of its k neighbors, whether it preserves with probability $1 - r$ its neighbors *after* it infects (bottom left in figure 2(a)), or it switches with probability r its neighbors just *after* it infects (bottom right in figure 2(a)). Here, p_k is the degree distribution. The quantity v_p is the probability that the epidemic does not spread to an infinite group *through a random neighbor*, given the parent node was not chosen to switch immediately *after* it had the chance to infect its neighbors. The quantity v_{np} is the same except that the parent node did switch its neighbors the moment *after* it had the chance to infect its neighbors, leaving its children with no parent (np) and with another node instead. The reason we separate this likelihood into two cases, is that otherwise the chances to spread through each neighbor are dependent via the question of whether their parent was chosen to switch or not, since the parent is not susceptible, unlike others.

Defining the generating function [42, 43] of the degree distribution p_k,

$$G_0(x) = \sum_{k=0}^{\infty} p_k x^k, \tag{2}$$

we obtain

$$1 - P_{\text{inf}} = (1-r) G_0(v_p) + r G_0(v_{np}). \tag{3}$$

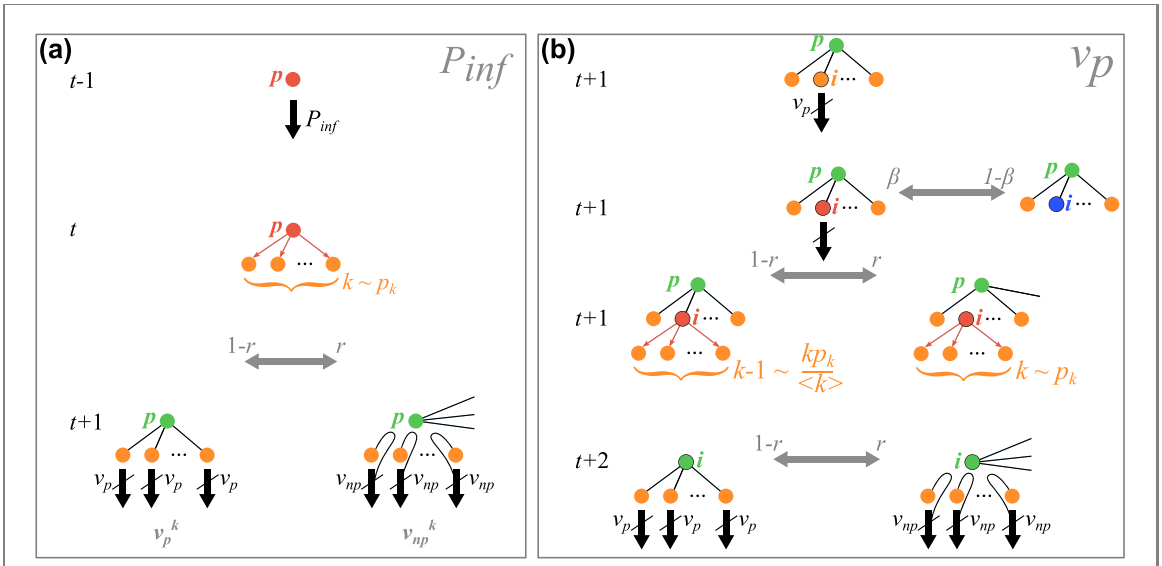

Figure 2. Diagrams demonstrating the possibilities considered in theory. (a) A diagram for demonstrating the derivation of equation (1). The probability, P_{inf}, of a random infected node (node p) to cause a major outbreak, is the likelihood that it will happen through at least one of its k neighbors. The orange nodes shown at time t represent nodes that came in contact with an infectious node (p) at time t, but it is unknown whether they were infected. Node p, that was infected at time $t-1$, will not lead to a macroscopic epidemic if and only if all its neighbors at time t will not spread the disease to an infinite group. However, their probability to cause an outbreak depends on whether before they infect, at time $t+1$, their parent node p switched neighbors. (b) A diagram for demonstrating the derivation of equation (4). The neighbor i, that was in contact with the infectious parent p at time t, now at time $t+1$ might spread the epidemic to cause an outbreak. In case it is known that the parent p did not switch neighbors before $t+1$, the probability that i will not make an outbreak is v_p. This v_p depends first on whether i was infected by the parent p at all. It is also dependent on whether i switched neighbors before it infects it neighbors ($t+1$), since a switching impacts the distribution of i's susceptible neighbors number. Finally, v_p depends also on whether i switches neighbors after it infects others (before $t+2$), since it influences the probability of its neighbors to spread the epidemic (v_p or v_{np}).

Note that we do not consider whether the infectious node performed neighbors-switch *before* it infected, since it does not have any impact.

Next, we find v_p. We follow the diagram in figure 2(b) to consider all the possibilities. Node i was in contact with the infectious parent node p at time t, and at $t+1$ it might spread a major outbreak. The probability that it does not happen is v_p. Note first that if the parent (node p) did not infect its arbitrary neighbor (node i), with probability $1-\beta$, then obviously the disease will not spread through this neighbor (blue node i on right in figure 2(b)). Thus, we analyze what happens if the neighbor (i) did got infected (red i). In case of v_p (figure 2(b)) it is known that the parent (p) did not switch neighbors. However, we distinguish between two cases: i switched neighbors (with likelihood $1-r$) after it *was infected* before time $t+1$, or it did not. When switching neighbors it gets the degree distribution of a random node p_k, while when not switching it preserves its *neighbor* degree distribution which is $kp_k/\langle k\rangle$. We also separate between the case it switches neighbors *after* it infects before time $t+2$, and the case it does not. Switching *after* infecting leaves its former neighbors with no parent (figure 2(b) bottom right), consequently gives them the chances v_{np}, while not switching (figure 2(b) bottom left) gives its neighbors the probability v_p for not spreading the epidemic to infinity. Finally, the only difference between v_p and v_{np} is where the neighbor, i, did not switch neighbors before it infected (before time $t+1$), then if the parent p was not replaced, there are $k-1$ susceptible neighbors, since the parent is recovered, while if the parent node p, was replaced by a random node, i has k susceptible neighbors to infect. Thus, we obtain two self consistent equations,

$$v_p = 1-\beta+\beta\left[(1-r)^2 G_1(v_p)+(1-r)rG_1(v_{np})+r(1-r)G_0(v_p)+r^2G_0(v_{np})\right], \tag{4}$$

and

$$v_{np} = 1-\beta+\beta\left[(1-r)^2 G_2(v_p)+(1-r)rG_2(v_{np})+r(1-r)G_0(v_p)+r^2G_0(v_{np})\right], \tag{5}$$

where G_1 and G_2, the generating functions of the residual degree and the neighbor degree distributions correspondingly, are defined by

$$\begin{aligned} G_1(x) &= \sum_{k=0}^{\infty}\frac{kp_k}{\langle k\rangle}x^{k-1}, \\ G_2(x) &= \sum_{k=0}^{\infty}\frac{kp_k}{\langle k\rangle}x^{k}. \end{aligned} \tag{6}$$

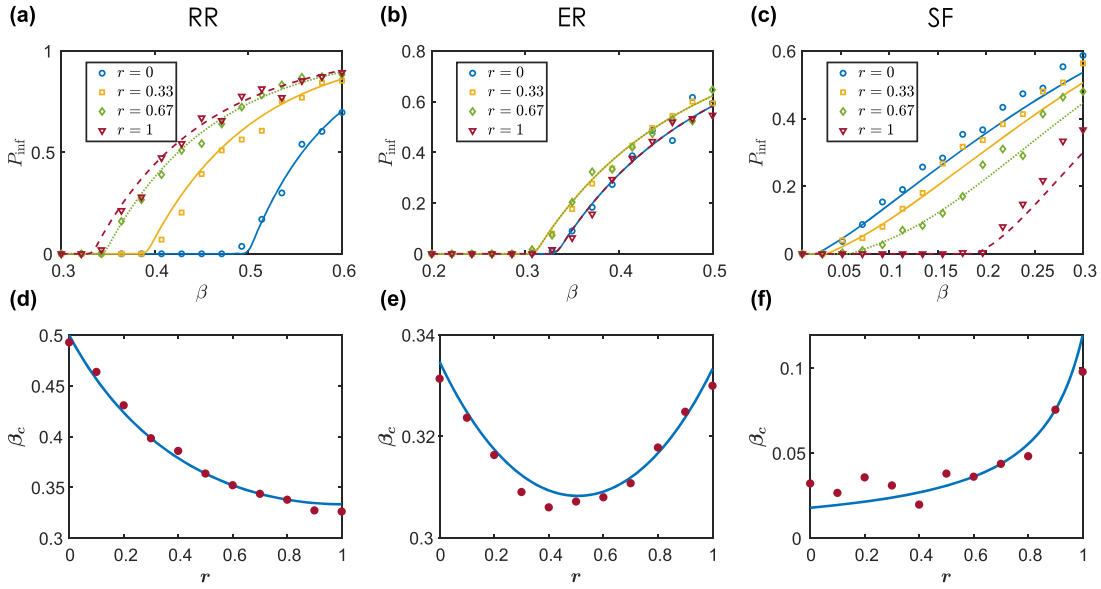

Figure 3. Epidemic spread on evolving networks when the recovery time, τ, is one unit. (a)–(c) The likelihood for large (infinite) infection spreading, $P_{\rm inf}$, versus the infection rate, β, using equations (3)–(5) (lines) and simulations (symbols). The recovery time is $\tau = 1$. In our simulations we set $N = 10^4$ and averaged over 300 realizations. The results are for (a) RR network with degree $k = 3$, (b) ER network with degree $\langle k\rangle = 3$, and (c) SF network with $k_0 = 2$ and $\lambda = 2.5$. In (d)–(f) we show the critical infection rate, β_c, versus the rewiring rate, r, using equations (7)–(10) (lines) and simulations (symbols) for (d) RR, (e) ER, and (f) SF. For SF, the simulations agree with equation (7) presented by the line. However, there is some deviation from equation (10) due to the finite size effect. See in SI (https://stacks.iop.org/NJP/24/053002/mmedia) figure S1 that $\beta_c \to 0$ for $N \to \infty$ except at $r = 1$.

Note that $G_1(x) = G_0'(x)$ and $G_2(x) = xG_0'(x)$. The rhs of equations (4) and (5) consider in fact four options regarding switching before and after infecting others. For each option, there is the corresponding degree distribution (*before*-switch determines) and the corresponding variable v_p or v_{np} (*after*-switch determines). Note that in equations (4) and (5) we assume that all *random* neighbors (except the parent node) are susceptible because we look on the very first steps of the spread, and at this point, in a large network the probability to catch randomly the recovered or infectious nodes is negligible.

Notice that the SIS model is different from the SIR model considered here, even at the first steps of the epidemic. The difference is that in SIS the parent node becomes susceptible when its 'child' infects others, instead of recovered in SIR. This fact has an impact since there is a non-negligible chance of $1 - r^2$ that they are still connected at the time after the parent infected the child. However, for $r = 1$ SIS and SIR are equivalent at the very beginning of the process since the parent is not anymore a neighbor of its child after one step and random nodes are susceptible. We further mention that for $r < 1$, the SIS model is not solvable by our analysis [8, 44] since the presence of the susceptible parent makes all its 'children' dependent on each other, what prevents us from writing equations such as equations (3)–(5).

Solving equations (4) and (5) and substituting v_p and v_{np} in equation (3) we obtain the probability that a random infectious node spreads the epidemic to an infinite group. Figure 3 shows the analytical results of $P_{\rm inf}$ vs β according to equations (3)–(5) compared with simulations, showing excellent agreement. One can see that below β_c the chance of infinite infection is negligible, while above β_c there is a nonzero probability for it.

3.1. Critical threshold

Below the criticality ($\beta < \beta_c$), only the single solution ($P_{\rm inf} = 0, v_p = 1, v_{np} = 1$) satisfies equations (3)–(5), representing no outbreak of the contagion. Above criticality, in contrast, there is another solution representing the existence of a major outbreak, in which $P_{\rm inf} > 0$, in addition to the $P_{\rm inf} = 0$ solution. At criticality, there exists a phase transition between the two states described above, a single solution and a pair of solutions. This determines (see SI section 1.2) that the derivative of both sides of equation (4) with respect to v_p are equal, leading to,

$$\beta_c = \frac{1}{(1-r)(\kappa - 1 + r) + r\langle k\rangle}, \tag{7}$$

where $\langle k\rangle = G_0'(1)$ is the average degree, and $\kappa = \langle k^2\rangle/\langle k\rangle = G_2'(1)$ is the average neighbor degree. The last equation is pretty intuitive because the denominator represents the average number of susceptible neighbors

which are in touch with an infected node. Thus, it is equivalent to the known condition [3] of the critical reproductive ratio $R_0 = 1$.

We can further recognize in this equation that in fact there are two effects of the rewiring. One, the second term in the denominator, $r\langle k\rangle$, is a change of the degree from a typical degree of a neighbor to that of a random node. The second, the adding of r to $\kappa - 1$ in the first term, is that the parent node, which infected the current node, might be switched, and replaced by a susceptible node rather than recovered.

Notice that the limit case $r = 0$ recovers the well known result [8, 37, 45] for a static network $\beta_c = 1/(\kappa - 1)$. The other limit of full rewiring, $r = 1$, gives $\beta_c = 1/\langle k\rangle$ which recovers the result of branching process since a new degree and neighbors are sampled at each time as in branching process.

The above results, equations (1)–(7), are general for any degree distribution. Next, we analyze this result for several model networks. We are interested in the dependence of β_c on the rewiring rate r. This behavior changes qualitatively for different networks since the relation between κ and $\langle k\rangle$ varies. For RR network, $\kappa = \langle k\rangle = k$, and substituting this in equation (7) gives,

$$\beta_c^{\mathrm{RR}} = \frac{1}{k - (1-r)^2}, \tag{8}$$

which implies that β_c decreases when r increases (see figure 3(d)), namely, the dynamics strengthens the contagion in RR. This happens because it is better for mitigating the spread of the disease not to switch neighbors, since when switching, the infectious node is exposed to more susceptible nodes by replacing the contact with the parent node which is recovered. For ER network, $\kappa - 1 = \langle k\rangle$, hence

$$\beta_c^{\mathrm{ER}} = \frac{1}{\langle k\rangle + r(1-r)}, \tag{9}$$

which exhibits an interesting and surprising non-monotonic behavior (see figure 3(e)). The explanation is the following. In ER the average excess degree is equal to the mean degree. Hence, for $r = 0$ and $r = 1$, the threshold is the same, since the parent node is recovered, thus the number of susceptible neighbors is the same in average. In the between range $0 < r < 1$, the threshold is lower due to the option that our infectious node does not switch neighbors, and the parent switches and thus leaves someone else instead. In this case, the infectious node has another susceptible neighbor in addition to the excess degree. The probability of this option is $r(1-r)$, which is non-monotonic, because increasing r, on one hand, increases the chance that the parent switches and adds a susceptible neighbor. On the other hand, increasing r decreases the probability that the infectious node does not switch to gain this extra-susceptible neighbor. For small $r < 0.5$ the first effect is dominant while for large $r > 0.5$ the second effect is dominant. This explains also the symmetry of $r \to 1 - r$. The most interesting case is SF network whose degree distribution is $p_k = Ak^{-\lambda}$ for $k \geqslant k_0$ with $\lambda \leqslant 3$. This distribution has a divergent second moment, and therefore $\kappa \to \infty$. If $\lambda \leqslant 2$ then also the first moment diverges, $\langle k\rangle \to \infty$. Thus,

$$\beta_c^{\mathrm{SF}} = \begin{cases} 0, & r < 1 \text{ or } \lambda \leqslant 2 \\ \dfrac{1}{\langle k\rangle}, & r = 1 \text{ and } \lambda > 2 \end{cases}. \tag{10}$$

The reason that $\beta_c = 0$ for a static SF network is that when spreading the epidemic the infection goes through neighbors. This leads the disease very fast to the hubs, whose degrees are very large, therefore they spread the contagion even if the infection rate β is very small. However, in evolving network, when the rewiring rate is one, once an hub gets infected it immediately switches its neighbors and samples a new degree from p_k, such that at the moment it comes to infect it is no more a hub. Hence β_c becomes nonzero. This phenomenon exists in reality when people are likely infected in large events when they have a contact with many potential infectious people, however, only few days later (e.g. in COVID-19), they can infect, but at this time, they probably do not take part in a mass event. Interestingly, this finite value of β_c is also valid for SIS on evolving networks with $r = 1$ as for SIR. This is since for $r = 1$, both SIR and SIS are identical at the beginning of the epidemic spreading, see above discussion after equation (6). Interestingly, in contrast, as long as $r < 1$ (even very close to 1) the epidemic threshold is zero for large enough network since there exists a finite probability to have an average neighbor's degree which diverges. Notice that $\beta_c^{\mathrm{SF}} = 0$ since $\kappa \to \infty$ for $N \to \infty$. However, for a finite system, β_c^{SF} given by equation (7) is finite and dependent of N as shown in figure 3, and in SI figure S1.

For SF, switching neighbors helps a lot to curb the epidemic, because then the infectious node has a typical degree of a random node which is much lower than the typical degree of neighbors which tend to be hubs. Therefore, when r increases β_c also increases in contrast to RR, see figure 3. Namely, SF and RR exhibit qualitatively opposite responses to dynamics of the structure. While in SF, dynamics mitigates the contagion, in RR, it helps the disease to propagate.

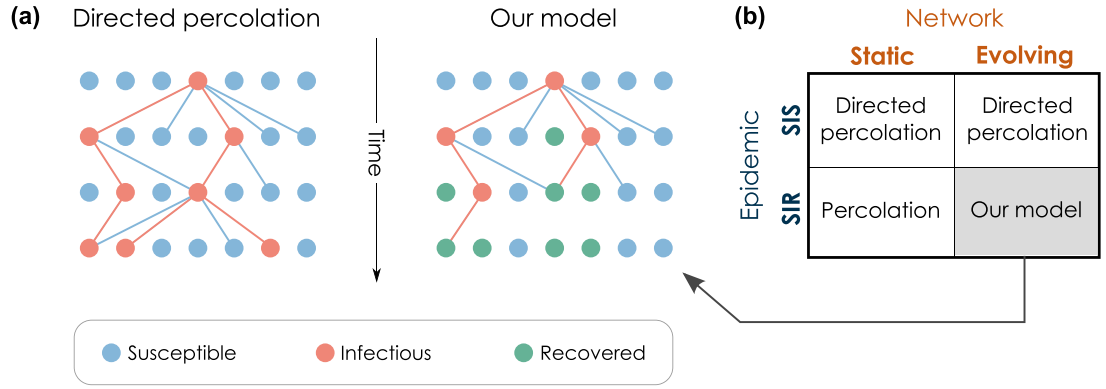

Figure 4. Comparison of SIR on evolving networks to DP model. (a) On the left, we see the DP model which is equivalent to SIS model. The first row represents the network at time 0, the second at time 1, and so on. The red links represent traverses (in DP) or infections (in SIS), and the blue links indicate contacts that were not traversed (DP) or did not lead to infection (SIS). A link is traversed randomly with likelihood β. Note that the same node can be infected again and again, allowing the spread of disease. Also the same node might have different degree and neighbors at different times due to the network dynamics. On the right, we show our model (SIR on evolving network) which is identical to the model on left, except that an infectious node recovers after one step and cannot be infected anymore. While on the left (SIS or DP) the epidemic is spread by going through the same nodes few times, on the right the epidemic stops due to the recovery of the spreaders. Our model is like DP modified to SIR. (b) A table showing the mapping from the four epidemic models on static/dynamic networks to percolation models.

4. Comparison to directed percolation

It was shown [9, 40, 41] that SIR model with a fixed recovery time *on static network* is mapped exactly to *bond percolation*, where the chance p of a link to be occupied replaces the probability β to infect a neighbor before recovering, see equation (13). The disease will spread to the whole connected component of the source node. Thus, the chance of a major outbreak equals to the relative size of the giant connected component.

SIS model, on the contrary, cannot be mapped to a percolation since a link can be traversed many times. This feature cannot be captured by the single probability p of a link occupation in percolation. However, SIS model can be rigorously mapped [38] to directed percolation (DP) [39], for a fixed recovery time $\tau = 1$, where each time step gets a layer which contains a copy of the network [38], see figure 4(a). Between the layers there reside the edges of the network at the corresponding time. Each link is traversed with chance $p = \beta$. This mapping covers both static and evolving networks. If the network is static the links between all the layers are identical, while for an evolving network the connections between layers might be different. Of course all links have one direction representing the flow of time.

The only case without mapping to percolation is the SIR model on an evolving network, which can be described nor by percolation since the latter is static, neither by DP in which a node can be traversed many times in contrary to SIR model where an agent can be infected one single time.

Thus, the SIR model on evolving networks proposed in this manuscript, for recovery time $\tau = 1$, is actually a modification of DP with the additional following condition. When tracking the cluster reachable from a source, one can go through each node only once. This is since once a node is traversed (infected) it is recovered and immunized and cannot be traversed again. In our model, the connections from a layer to the next layer are rewired such that each node changes randomly its neighbors and degree with rate r. See in figure 4(a) an illustration of the classic DP compared to our modified DP which represents the SIR model on an evolving network.

Our new mapping completes a picture within statistical physics, giving for SIR or SIS on static or evolving networks a mapping in percolation models, see figure 4(b).

5. Nonuniform infectious time

Next we consider a general distribution for the recovery time τ, and particularly the case in which the probability of an infected node to recover before each time step is γ. In contrast to the case we analyzed above in which the infectious time was fixed $\tau = 1$, now the infectious time is random, distributed exponentially as

$$\phi(\tau) = (1-\gamma)^{\tau}\gamma. \tag{11}$$

Note that also $\tau = 0$ is a possible option capturing the scenario of immediate recovery after being infected before infecting others. The mean of this distribution, equation (11), is $\langle\tau\rangle = (1-\gamma)/\gamma$, ranging from 0 to

infinity depending on γ. The generating function of $\phi(\tau)$ is

$$G_\tau(x) = \sum_{\tau=0}^{\infty} \phi(\tau) x^\tau = \frac{\gamma}{1-(1-\gamma)x}. \tag{12}$$

Because of the complexity of this case, we find as above the outbreak probability P_{inf} analytically in the extremes of static ($r = 0$) and fast-evolving ($r = 1$) networks. For the range in between, we solve only the critical conditions for a major epidemic in the 3D (r, γ, β) space.

5.1. Static network ($r = 0$)

An infectious node spends a time τ in contact with its constant neighbors. The chance to infect each neighbor, p, depends on τ as

$$p = 1 - (1-\beta)^\tau, \tag{13}$$

which is the complementary probability of no infection in any time during the contact. The infection probabilities of neighbors are dependent on each other through the recovery time of their parent τ, requiring us to separate between different values of τ. The probability P_{inf} of a major outbreak starting with a random infectious node is

$$1 - P_{\text{inf}} = \sum_{\tau=0}^{\infty} \phi(\tau) G_0(1-p+pv), \tag{14}$$

where the sum is over all possible recovery times τ and requiring that any neighbor does not get infected $(1-p)$ or does not spread the epidemic to infinity (v) even though it got infected. The probability v that an infected neighbor does not spread the epidemic to a macroscopic group is obtained by

$$v = \sum_{\tau=0}^{\infty} \phi(\tau) G_1(1-p+pv). \tag{15}$$

Here G_0 is replaced by G_1 which corresponds to the residual degree distribution (for v) rather than the degree distribution (for P_{inf}). Equations (14) and (15) are the equivalent of equations (3)–(5) for any recovery time distribution $\phi(\tau)$ and $r = 0$.

At criticality, the derivative of both sides of equation (15) are equal, and substituting equation (13), we obtain (see SI section 2.1)

$$\beta_c = 1 - G_\tau^{-1}\left(1 - \frac{1}{\kappa - 1}\right), \tag{16}$$

which is valid for any recovery time distribution $\phi(\tau)$. For our case of recovery probability γ in every step, using equation (12), we obtain,

$$\beta_c = \frac{\gamma}{1-\gamma}\frac{1}{\kappa - 2}. \tag{17}$$

The above case of uniform $\tau = 1$, is recovered since G_τ is simply the identity function, therefore, $\beta_c = 1/(\kappa - 1)$. For the general case of uniform τ, $G_\tau(x) = x^\tau$, thus $\beta_c = 1 - [1 - 1/(\kappa - 1)]^{1/\tau}$. We recognize from equation (17) the value of γ_c that makes $\beta_c = 1$. That is for $\gamma > \gamma_c$ there is no macroscopic outbreak, for any infection rate β, where $\gamma_c = (\kappa - 2)/(\kappa - 1)$.

5.2. Fully temporal network ($r = 1$)

For the case of fully temporal networks, $r = 1$, the probability of infecting a neighbor is just β independent on τ since the parent switches its neighbors at each time step. However, the recovery time τ determines how many neighbors the parent meets before it recovers. Let q be the number of neighbors that an infectious node meets until it recovers. This number, q, satisfies $q = \sum_{i=1}^{\tau} k_i$, where k_i are sampled from p_k and τ is sampled from $\phi(\tau)$. Even if the infectious node is a random *neighbor*, its degree distribution is p_k when it comes to infect, since $r = 1$, namely it has new random degree and neighbors. Thus, as a sum of random variables [46], q has the average $\langle q \rangle = \langle \tau \rangle \langle k \rangle = (1-\gamma)/\gamma \langle k \rangle$, and using equation (12), its generating function is (see SI section 2.2)

$$G_q(x) = G_\tau\left(G_0(x)\right) = \frac{\gamma}{1-(1-\gamma)G_0(x)}. \tag{18}$$

Due to the fully switching, it does not matter if the spreader is a random node or a random infected neighbor. Hence, $P_{\text{inf}} = 1 - v$, and

$$v = G_q(1 - \beta + \beta v), \tag{19}$$

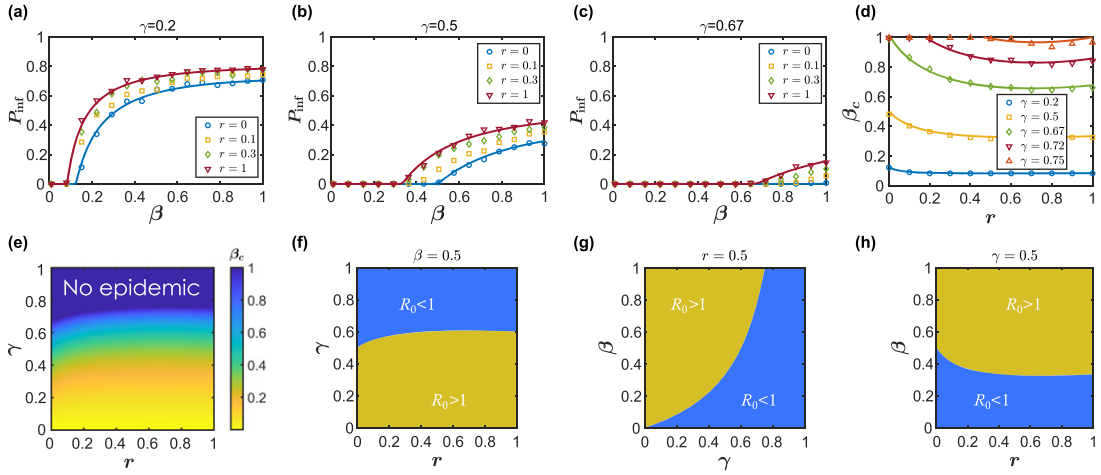

Figure 5. Epidemic spread on evolving networks for nonuniform recovery time. (a)–(c) The likelihood of a major outbreak, P_{inf}, versus the infection rate, β, for different rewiring rates r. Lines represent the theory for $r = 0, 1$ using equations (14), (15) and (19), and symbols represent simulations results. The recovery rate is presented for values, (a) $\gamma = 0.2$ (b) $\gamma = 0.5$ and (c) $\gamma = 0.67$. In our simulations, we constructed ER networks with average degree $\langle k\rangle = 3$ and size of $N = 10^4$. We averaged the results over 10^3 realizations. Since the recovery rate $\gamma = 0.5$ yields $\langle\tau\rangle = 1$, it is comparable to the above case of fixed $\tau = 1$, figure 3(b). Note that for $\gamma = 0.67$ and $r = 0$ there is no transition since $P_{\text{inf}} = 0$ for any value of β, see panel (e). (d) The critical infection rate, β_c for a major outbreak, versus the rewiring rate, r, for different values of γ using equations (24) and (25) and simulations. (e)–(h) Equations (24) and (25) provide a 3D phase diagram in (r, γ, β) space which splits into the outbreak phase and the no-outbreak phase. R_0 determines the boarders of these phases, $R_0 > 1$ is the condition for an outbreak. (e) β_c below which there is no macroscopic epidemic. Note that in the blue area $\beta_c = 1$ i.e. there is no epidemic for any β. (f)–(h) Cross-sections in the sub-spaces of (r, γ, β) for one fixed value (blue—no epidemic, yellow—epidemic). In SI figures S2 and S3, we present the results also for RR and SF networks.

which yields at criticality,

$$\beta_c = \frac{1}{\langle\tau\rangle}\frac{1}{\langle k\rangle} = \frac{\gamma}{1-\gamma}\frac{1}{\langle k\rangle}. \tag{20}$$

The above case of fixed recovery time $\tau = 1$ is included by substituting $\langle\tau\rangle = 1$ to get $\beta_c = 1/\langle k\rangle$. Here $\gamma_c = \langle k\rangle/(\langle k\rangle + 1)$, above which there can not be a major outbreak for any β.

Comparing equations (17) and (20) for nonuniform recovery time, we recognize that for ER network where $\kappa = \langle k\rangle + 1$, it follows that $\beta_c^{\text{ER}}(r = 0) > \beta_c^{\text{ER}}(r = 1)$, implying that the contagion is spread better in fully-evolving network, rather than in a static network. It is interesting that this result is in contrast to the fixed recovery time $\tau = 1$, for which β_c^{ER} is symmetric for substituting $1 - r$ instead of r (equation (9)). That is, not fixed unit recovery time, causes the dynamics of the network to enhance more the spread of the epidemic. The reason can be understood as follows. Suppose that a node is infectious for a time longer than one unit, then if it switches neighbors it has the opportunity to infect more nodes, while if it stays with the same neighbors it can at most infect all of them, it cannot infect the same node twice. This effect of rewiring does not appear of course at fixed recovery time $\tau = 1$. However, equations (24) and (25) imply that also fixed recovery time for any $\tau > 1$ and recovery rate γ show for ER a specific value of r between $0 < r < 1$ at which β_c is minimal similar to the case of $\tau = 1$, see figure 5(d).

5.3. Partial temporal network ($0 < r < 1$)

For the partial temporal network, $0 < r < 1$, we calculate directly the critical conditions for a macroscopic outbreak. To this end, we track the reproductive ratio, R_0, defined as the average number of neighbors that a random infected node infects until it recovers. Let us denote by I the random variable of the number of infections that a random infected node performs. I satisfies

$$I = \sum_{j=1}^{\tau} I_j, \tag{21}$$

where I_j is the number of infections our node acts at time j after it was infected. We seek for R_0, which is just

$$R_0 = \langle I\rangle. \tag{22}$$

To this end, we write a recurrence relation for the degree, k_j, and the number of susceptible neighbors, S_j, of the infectious node at time j after it got infected (see details in SI section 2.3),

$$\begin{aligned}\langle k_j\rangle &= (1-r)\langle k_{j-1}\rangle + r\langle k\rangle,\\ \langle S_j\rangle &= (1-r)^2(1-\beta)\langle S_{j-1}\rangle + r(1-r)\langle k_{j-1}\rangle + r\langle k\rangle,\end{aligned} \tag{23}$$

for $1 < j \leqslant \tau$. These equations are based on considering both the possibility that our node switches neighbors just before time j, and the possibility that it does not. Even if not, each one of its neighbors might switch and get replaced by another node. We assume as above that any random new neighbor is susceptible since we look at the very first steps of the disease, thus the amount of infectious and recovered nodes is negligible. Solving this recurrence relation, we manage to obtain an expression for the reproductive ratio (see details in SI section 2.3),

$$R_0 = \alpha_1 r\beta\frac{1-G_\tau(\lambda_1)}{1-\lambda_1} + \alpha_2\beta\frac{1-G_\tau(\lambda_2)}{1-\lambda_2} + \langle\tau\rangle\beta\alpha, \tag{24}$$

where $\lambda_1 = 1-r$, $\lambda_2 = (1-r)^2(1-\beta)$, $\alpha = r(2-r)\langle k\rangle/(1-(1-r)^2(1-\beta))$, $\alpha_1 = (1-r)(\kappa-\langle k\rangle)/(1-(1-r)(1-\beta))$, $\alpha_2 = y_1 - r\alpha_1$, and $y_1 = (1-r)(\kappa-1+r) + r\langle k\rangle - \alpha$. Thus, R_0 captures all the parameters of the problem. It includes the structure-related attributes as well as the epidemic-related characteristics. The structure is represented by $\langle k\rangle$ and κ coming from the static pattern of the network and also by the rewiring rate r governing the network dynamics. The epidemic properties are the infection and recovery rates, β and γ. Next, we use the well-known [3] critical condition, to get the equation for the critical transition from $P_{\text{inf}} = 0$ to $P_{\text{inf}} > 0$,

$$R_0 = 1. \tag{25}$$

Note that equations (24) and (25) converge, for fixed $\tau = 1$, to equation (7) since for this case $G_\tau(x) = x$, and $\langle\tau\rangle = 1$, thus $R_0 = \beta[\alpha_1 r + \alpha_2 + \alpha] = \beta[(1-r)(\kappa-1+r) + r\langle k\rangle]$. For a general fixed τ, to obtain R_0, we just have to substitute in equation (24) $G_\tau(x) = x^\tau$.

In figure 5 we show the results for the case of nonuniform recovery time with recovery rate γ for ER network (see SI figures S2 and S3 for RR and SF networks). Using equations (14), (15) and (19), we show the probability of a macroscopic spread, P_{inf}, depending on the recovery rate β for $r = 0, 1$, showing a good agreement with computer simulations results. For other values of r only computer simulations are presented. Note that larger γ gives smaller β_c as expected (figures 5(a)–(c)), and for large enough γ, and certain values of r, there is no macroscopic epidemic spread for any β (figure 5(c)). The dependence of β_c on r, for different γ values (figure 5(d)), is different from the dependence for the fixed one unit recovery time, $\tau = 1$, shown in figure 3(e). While the latter shows non-monotonic and symmetric behavior, the former shows a general decreasing pattern except for γ close to one. The reason is related, as explained above, to the additional effect of the rewiring when the infectious time is larger than one unit. For the full range of $0 < r < 1$, we present (figures 5(e)–(h)), using equations (24) and (25) the 3D phase diagram in (r, γ, β) space which splits into the outbreak phase and the no-outbreak phase. This 3D space combines the structure sub-space (r) and the epidemic sub-space (γ, β). R_0 is the parameter predicting the borders of these phases, $R_0 > 1$ leads to an outbreak, while if $R_0 < 1$ the epidemic will not spread macroscopically. A heat map of β_c below which there is no a macroscopic epidemic reveals a region (figure 5(e), in blue) in which $\beta_c = 1$ i.e. there is no epidemic for any β. We further present some cross sections of the above mentioned phases in the sub-spaces of (r, γ, β) where one is fixed (figures 5(f)–(h)).

6. Discussion

We explored how an evolving network in which the degree of each node changes randomly according to a fixed degree distribution, responds to epidemics. We found that different network structures show cardinal distinguished behaviors regarding the question: does dynamics in network structure aid the epidemic spread or restrain it? We also found that the well-known character of SF networks, the existence of a major outbreak under any infection probability, is broken in a fast enough evolving network, in which the epidemic does not occur for small infection rates. We further found an equation giving the critical condition for a major outbreak depending on all the parameters both of the structure and of the epidemic.

Yet, some realistic and influential characteristics of epidemics on evolving networks were not taken into account in this study which are worth a future research. We propose here two characters, correlations and non-homogeneity, to be investigated as extensions of our model. Degree correlations emerge between neighbors either due to participating in same events or because of homophily [47]. Hence, the rewiring

should not be random. Homophily might cause also other correlations between neighbors [47]. Non-homogeneity can be considered in several directions. For example, people often have a small set of quite *constant* contacts, as family and colleagues. These connections are close to be *static*, while connections with the rest of population are usually very *dynamic*. Another case is when the population comprises of few classes of people having different degree distributions. Each person preserves its degree *distribution* during all the epidemic process though its *degree* varies in time. Moreover, the infection rate might be nonuniform among the population e.g. since part of the people are vaccinated while the rest not as happened in the last waves of the COVID-19 pandemic during 2020–2021. Another interesting direction to explore is the impact of the evolving structure on the efficiency of different vaccinating strategies. Other aspects in our model that demand future studies are the accumulative size of the disease as well as the dynamic information of the epidemic propagation, such as that investigated in reference [26].

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Data availability statement

No new data were created or analysed in this study.

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