Epidemic spreading in a variety of scale free networks

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We have shown that the epidemic spreading in scale-free networks is very sensitive to the statistics of degree distribution characterized by the index γ, the effective spreading rate λ, the social strategy used by individuals to choose a partner, and the policy of administrating a cure to an infected node. Depending on the interplay of these four factors, the stationary fractions of infected population F_γ as well as the epidemic threshold properties can be essentially different. We have given an example of the evolutionary scale-free network which is disposed to the spreading and the persistence of infections at any spreading rate $\lambda > 0$ for any γ. Probably, it is impossible to obtain a simple immunization program that can be simultaneously effective for all types of scale-free networks. We have also studied the dynamical solutions for the evolution equation governed by the epidemic spreading in scale-free networks and found that for the case of vanishingly small cure rate $\delta \ll 1$ the initial configuration of infected nodes would feature the solution for very long times.

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I. INTRODUCTION

Many systems in biology, sociology, and economics are best described by complex networks of agents linked by various physical and informational connections. Detailed studies of their connectivity properties [1–3] have revealed that the probability that a node of these networks has degree *k* usually follows a power law

$$P(k) \propto k^{-\gamma} \tag{1}$$

over a large range of *k*, with an exponent γ that ranges between 1 and 3 depending on the system [4]. Among various practical applications, the *scale-free* (SF) networks exhibiting the property (1) is of great interest for epidemiology [5] and computer virus spreading [6]. The key model used in the epidemiological studies is the susceptible-infected-susceptible (SIS) model [5], in which individuals represented by nodes exist in either "healthy" or "infected" discrete states, and each link represents a connection along, with which the infection can spread. At each time step, each healthy node is infected at rate ν if it is connected at least to one infected node. At the same time, infected nodes are cured at rate δ, regaining susceptibility. One defines an effective spreading rate as $\lambda = \nu/\delta$.

The first step in understanding epidemic spreading in SF networks has been made in Ref. [7], where the SIS model has been defined on the Barabási-Albert (BA) scale-free graphs [2]. The BA graphs are generated by a random process such that the vertices are added to the graph one at a time and connected to a fixed number of earlier vertices, selected with probabilities proportional to their degrees, so that a new site is more likely to link to existing sites which are "popular" at the time the site is added. An important conclusion reported in Ref. [7] on the critical behavior observed in the BA scale-free graphs for $\gamma \leqslant 3$ states the absence of a critical epidemic threshold, $\lambda_c = 0$. It implies that the BA networks are disposed to the spreading and the persistence of infections at whatever spreading rate the epidemic agents possess if $\gamma \leqslant 3$. For $\gamma > 4$, epidemics on the BA networks have the same properties as on random networks, i.e. there exists $\lambda_c > 0$ such that the infection spreads and becomes persistent if $\lambda \geqslant \lambda_c$ and dies out fast when $\lambda < \lambda_c$ [7].

Recently, it has been demonstrated that the BA algorithm is not a unique model generating scale-free graphs. An alternative flexible model based on the principle of evolutionary selection of a common large-scale structure of biological networks [3] has been proposed in Ref. [8]. Among the clear advantages of the algorithm discussed in Ref. [8], one can mention that the possible value of index γ can vary in the wide range $(1,\infty)$ and that the in-degree and out-degree statistics can be tuned independently from an exponential decay to a power law. Studying numerically the epidemic spreading in evolutionary scale-free networks, we have found that it differs essentially from those predicted in Ref. [7] for the BA networks. In particular, we find the absence of a critical epidemic threshold for any $\gamma > 1$. This numerical observation presents evidence of the sensitivity of epidemic spreading to the topological properties of SF graphs, which are obviously distinctive for the graphs generated by the different algorithms even though they enjoy the same probability degree statistics.

In Ref. [9], it has been supposed that the individual structural properties of SF graphs generated in accordance with a particular algorithm can be characterized by a pair-formation process, in which each vertex v of degree $d(v) = k$ chooses a set of partners according to a specified *k*-dependent rule describing the preferential choice (a social strategy) of an individual located at v. It was also shown that the critical behavior and epidemic threshold properties depend very much on the particular social strategy chosen by individuals.

In Ref. [10], it was shown that the *k*-dependent immunization strategy would change the epidemic threshold in the BA networks. In particular, it has been demonstrated that if the likelihood of identifying and administering a cure to an

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infected node with k links depends on the node's degree as k^{α}, the critical epidemic threshold $\lambda_c > 0$ would be restored.

In a sense, our paper presents a generalization of the approaches given in Refs. [9] and [10]. In the following section, we introduce the classes of vertices C_k possessing the same degrees k. The structure of SF networks is determined by the specification of social strategy of individuals, which is described by a coupling between different classes of vertices. It is important to note that different social strategies can generate the SF networks characterized by the same index γ of the power law statistics of the degree distribution. In spite of that their structural properties rising in accordance to the distinct social strategies of individuals can be rather different.

In this paper, we consider two alternative models for the partner choice preference. In the first model, we assume that the society is unstructured in a sense that an individual is chosen as a partner depending only on its connectivity degree k. This type of model has been the focus of studies so far (see Refs. [7,9]). However, from the sociological point of view, the second model, taking a possible social structure into account, seems definitely much more natural. In the second model, the coupling strength between vertices $v \in C_k$ and $w \in C_s$ belonging to different classes is supposed to depend on the difference $|k-s|$ and fades out if $|k-s| \gg 1$. We demonstrate that the epidemic spreadings in these two types of SF networks are dramatically different even if their indices γ are equal. Among the striking distinctions, we can point out that an average fraction of infected individuals in the unstructured societies grows up with γ for any immunization administrative policy, but in the structured communities it decreases with γ; that is good news, indeed.

We write down an exact evolution equation for the probability distribution of infected nodes over the classes of vertices. In spite of popularity of such an approach in the literature, this equation has not been discussed before. Assuming that the infection of an individual is a rare event, one arrives at a simplified version of the evolution equation which is usually discussed in the related papers as the "mean-field approach" (see, for instance, [7,10] and many others). Generally speaking, the solutions of the exact equation differ from those of the simplified one, nevertheless, the critical epidemic threshold λ_c predicted by these equations is the same.

We study the stationary solutions of the simplified equation in detail; we are interested in the dependence of behavior of the average fraction of infected individuals upon the effective spreading rate λ, the power law exponent γ, and two affinity parameters characterizing the social strategy chosen by individuals and the immunization policy in both structured and unstructured societies. These stationary solutions are indeed independent of the initial distribution of infected individuals in SF networks. However, in practical epidemiology, the knowledge of epidemic dynamics (i.e., of the transient processes) is of vital importance since it may help to devise an optimal dynamical immunization strategy to avoid the approach of stationary asymptotic solutions characterized by the large fraction of infected population.

We study the dynamical solutions of the simplified equation and find the relaxation time $T(k)$ within which the initial epidemic outbreaks develop into a stationary state. For $t \leqslant T$, the actual disease spreading depends crucially upon the initial distribution of infected individuals over the classes C_k since the occasional infection of hubs accumulating a large number of links at the very onset of spreading process can accelerate it overwhelmingly. The transient processes can feature the spread of diseases with the vanishingly small cure rate $\delta \ll 1$.

We conclude the paper with an example of the epidemic spreading process in the evolutionary SF network. This network is disposed to the spreading and the persistence of infections at whatever spreading rate the epidemic agents possess for any index of degree statistics γ. The initial configuration of the infected nodes influences the epidemic spreading in such networks even for very long times.

II. DEFINITION OF THE EPIDEMIC SPREADING PROBLEM IN SCALE-FREE NETWORKS

Let us consider a scale-free network of N nodes spanned with the graph $\mathbb{G}(N,\gamma)$ with $\gamma > 1$. In general, one can partition the set of vertices V ($|V| = N$) into $N-1$ different classes C_k comprising vertices having the same degree k,

$$C_k = \{v \in V : \deg(v) = k\}, \quad k \in [1 \ldots N-1]. \tag{2}$$

A *configuration* of the graph $\mathbb{G}(N,\gamma)$ is the string $\xi = (n_1, \ldots n_k \ldots n_{N-1})$ where $n_k = |C_k|$ are the random variables distributed in accordance to the power law,

$$p_{\gamma}(k) = \frac{\gamma-1}{1-(N-1)^{1-\gamma}} k^{-\gamma} \sim_{N\to\infty} (\gamma-1) k^{-\gamma}. \tag{3}$$

The structural properties of SF networks depend upon the social strategy chosen by individuals establishing a pair formation process generating edges of the graph $\mathbb{G}(N,\gamma)$. It can be defined by the matrix $\hat{\sigma}_{sk}$ of which the elements are the probabilities that the vertex $\mathrm{v} \in C_s$ chooses some other vertex $\mathrm{w} \in C_k$ as a partner. For instance, in the popular preference attachment model of the scale-free random graphs proposed by Barabási and Albert [2], the elements of $\hat{\sigma}$ depend only on one variable k,

$$\sigma_{sk} = \frac{k}{\langle k \rangle}, \quad \text{for any } s, \tag{4}$$

where $\langle k \rangle$ is the average number of connections between vertices. For $1 < \gamma < 2$, the average connectivity $\langle k \rangle$ diverges, and therefore the BA graphs do not exist. However, for other generating algorithms with alternative $\hat{\sigma}$ the SF networks exist even for $1 < \gamma < 2$.

In the present paper, we assume that $N \to \infty$ and treat the connectivity k as a continuous variable taking values in between 1 and ∞. The edge generating rule is given by an arbitrary positive integrable function (generally speaking, of two variables), σ satisfying the normalization condition

$$1=\int_1^\infty\int_1^\infty \sigma(k,s)p_\gamma(s)dk\,ds. \tag{5}$$

In the problem of epidemic spreading in SF networks, we are interested in the fraction of infected individuals at time t, $0<F(t)<1$, which is given by

$$F_\gamma(t)=\int_1^\infty p_\gamma(k)\phi(t,k)dk, \tag{6}$$

where $\phi(t,k)$ is the probability function that an arbitrary node $v\in C_k$ is infected. We suppose that the initial probability function $\phi(0,k)$ is known.

In accordance with the SIS model [5], at each time step the total fraction of infected individuals in a scale-free community is changed by the quantity

$$\Delta F=\nu F_h-\delta F_i, \tag{7}$$

where $0<\nu<1$ is the infection rate, $0<\delta<1$ is the rate at which the infected nodes are cured, F_h is the fraction of healthy nodes connected to at least one infected node, F_i is the fraction of infected nodes. In general, the probability of being linked to an infected node depends upon the social strategy of individuals σ,

$$\Theta_\gamma(t,k)=\int_1^\infty \sigma(k,s)p_\gamma(s)\phi(t,s)ds. \tag{8}$$

Since the balance equation (7) is satisfied for any $p_\gamma(k)$, one can write down the evolution equation for the probability functions $\phi(t,k)$ in the following form:

$$\partial_t\phi(t,k)=-\delta\cdot\mu(k)\phi(t,k)+(1-\phi(t,k))\{1-[1-\nu\Theta_\gamma(t,k)]^k\}. \tag{9}$$

The infecting term considers the probability that a node with k links is healthy $[1-\phi(t,k)]$ and gets infected in proportion to the rate $\nu>0$, via an infected connected node chosen with the probability $\sigma(k,s)$. The recovering term describes the probability that an infected node chosen with the probability $\mu(k)$ is cured in proportion to the rate $\delta>0$. One can think of $\mu(k)$ as a distribution of funds destined for a recovering of individuals from the class C_k provided the total scope is taken as 1.

The stationary solution of the Eq. (9) $[\partial_t\phi_{\rm st}(k,t)=0]$ is given by the formula

$$\phi_{\rm st}(k)=\frac{1-[1-\nu\Theta_\gamma(k)]^k}{1-[1-\nu\Theta_\gamma(k)]^k+\delta\mu(k)}, \tag{10}$$

in which the stationary probability function $\Theta_\gamma(k)$ satisfies the self-consistency equation

$$\Theta_\gamma(k)=\int_1^\infty\frac{\sigma(k,s)p_\gamma(s)\{1-[1-\nu\Theta_\gamma(k)]^k\}}{1-[1-\nu\Theta_\gamma(k)]^k+\delta\mu(k)}ds. \tag{11}$$

The above equation has a countable number of solutions, but only one of them belongs to the unit interval. For large connectivities $k\gg1$, the solution (10) behaves like

$$\phi_{\rm st}\simeq_{k\gg1}\begin{cases}1, & \text{if } \delta\cdot\mu(k)=o(1),\\ 0, & \text{if } \delta\cdot\mu(k)=O(1).\end{cases} \tag{12}$$

III. SIMPLIFIED EQUATION FOR THE LOW INFECTION RATES

In almost all papers devoted to the problem of epidemic spreading in the SF networks, a simplified version of the Eq. (9) is considered. Namely, it is assumed that the probability that a chosen node will be infected via connection with other infected nodes is very small, $\nu\Theta_\gamma(k,t)\ll1$. In this case, the right hand side of Eq. (9) is expanded into the power series in $\nu\Theta_\gamma$ and then only the linear term is retained. As a result, one arrives at the following simplified equation:

$$\partial_t\phi(t,k)=-\delta\cdot\mu(k)\phi(t,k)+\nu(1-\phi(t,k))k\Theta_\gamma(k,t). \tag{13}$$

Let us note that the solutions of the exact equation (9) differ from those of (13); nevertheless, the critical epidemic threshold λ_c predicted by these equations is the same.

A. Stationary solution of the epidemic equation for low infection rates

The stationary solutions, $\partial_t f_\gamma(\lambda,k)=0$, of the Eq. (13) is given by the function

$$f_\gamma(\lambda,k)=\frac{\lambda k\Theta_\gamma(k)}{\lambda k\Theta_\gamma(k)+\mu(k)}. \tag{14}$$

The asymptotic behavior of $f_\gamma(\lambda,k)$ as $k\gg1$ depends essentially upon the large-scale behavior of $\mu(k)$ and $\sigma(k,s)$,

$$f_\gamma(\lambda,k)\simeq_{k\gg1}\begin{cases}1, & \mu(k)/\lambda k\Theta_\gamma(k)=o(1)\\ 0, & \mu(k)/\lambda k\Theta_\gamma(k)=O(1).\end{cases} \tag{15}$$

The stationary probability that any given link points to an infected node, $0<\Theta_\gamma(k)\le1$, satisfies the self-consistency equation

$$\Theta_\gamma(k)=\int_1^\infty\frac{\lambda s\Theta_\gamma(s)\sigma(k,s)p_\gamma(s)}{\lambda s\Theta_\gamma(s)+\mu(s)}ds. \tag{16}$$

Trivial solution $\Theta_\gamma(k)=0$ always satisfies the above equation and gives a zero stationary prevalence, $f_\gamma=0$. A nonzero stationary prevalence $f_\gamma(k)\neq0$ is obtained when the Eq. (16) has a nontrivial solution in the interval $0<\Theta_\gamma(k)\le1$ that takes place if

$$\delta_\Theta\left[\int_1^\infty\frac{\lambda s\Theta_\gamma(s,\lambda)\sigma(k,s)p_\gamma(s)}{\lambda s\Theta_\gamma(s,\lambda)+\mu(s)}ds\right]\Bigg|_{\Theta=0}\ge1. \tag{17}$$

The above inequality defines the critical epidemic threshold such that $f_\gamma(k)>0$ as $\lambda>\lambda_c(k)$,

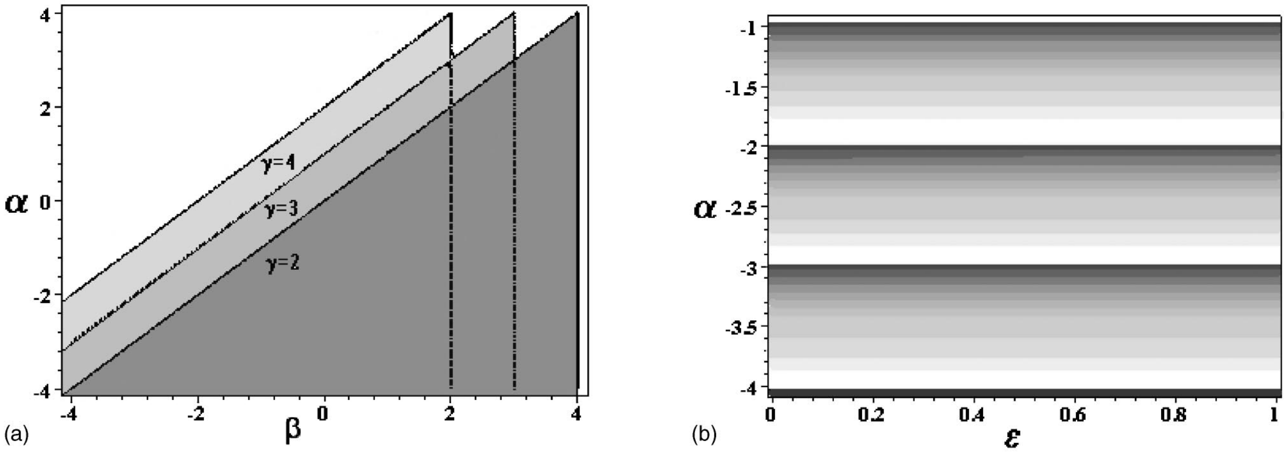

FIG. 1. The critical domains in which the critical epidemic threshold exists for (a) the power law model (19), for $\gamma=3$, where the phase diagram passes through the point $(\beta=1,\alpha=0)$ that corresponds to the Barabási-Albert SF network exactly as predicted in Ref. [7]; and (b) the hierarchical society with the social strategy of individuals given by the function (21). The critical epidemic threshold is infinite as α takes negative integer values and has zeros somewhere between negative integers. The parameter ε determines the width of the band in critical domains.

$$\lambda_c(k)\cdot\left[\int_1^{\infty}\frac{s\sigma(k,s)p_{\gamma}(s)}{\mu(s)}ds\right]=1. \tag{18}$$

Equation (18) shows that if $\sigma(k,s)$ depends on two variables, the critical epidemic threshold depends on k, i.e., in a structured society, the different classes of vertices C_k can possess different critical epidemic thresholds. Otherwise, if the probability of being chosen as a partner depends merely on the connectivity of a node, the same critical epidemic threshold λ_c holds for all vertices of the network.

As an example of the epidemic spreading in such a homogeneous SF network modeling an unstructured society, let us consider a generalized power law model with the social and immunization preference functions given by

$$\sigma(k)=\frac{\gamma-\beta-1}{\gamma-1}k^{\beta},\quad \mu(k)=\frac{\gamma-\alpha-1}{\gamma-1}k^{\alpha},$$
$$\alpha,\beta<\gamma-1,\quad \gamma\geqslant 1. \tag{19}$$

Then the integral in the Eq. (18) defining the critical epidemic threshold converges if $\gamma+\alpha-\beta>2$ and results in

$$\lambda_c=\frac{(\gamma-\alpha-1)(\gamma+\alpha-\beta-2)}{(\gamma-\beta-a)(\gamma-1)}. \tag{20}$$

In Fig. 1(a), we have presented the critical domains in which the critical epidemic threshold exists for different values of γ. Let us note that one of the phase (α,β) diagrams presented in Fig. 1(a) (for $\gamma=3$) passes through the point $(\beta=1,\alpha=0)$ relevant to the Barabási-Albert SF network exactly as predicted in Ref. [7].

In Fig. 2, we have sketched out some patterns of complicated behavior of the stationary fraction of infected individuals $F_{\gamma}>0$ depending on the affinity parameters α and β characterizing the immunization policy and the social strategy chosen by individuals, effective spreading rate λ, and the index of the degree statistics γ. These figures reveal the absence of a plain immunization program which can be effective simultaneously for all types of SF networks and for all effective spreading rates of viruses. One can see that, in general, the stationary fraction of infected individuals for the model (19) increases with λ and γ. The model predicts that the healing of certain classes of individuals does not help much in eradication of epidemics except for the small virus spreading rates $\lambda<0.2$. An effective immunization program in this case would assume a decentralization of the network providing a course of remedial treatment to everybody.

In a structured society, the coupling between vertices of different classes C_k and C_s depends on the distance $|k-s|$ between them and fades out for $|k-s|\gg 1$. A possible social strategy of individuals can be modeled by the function of two variables

$$\sigma(k,s)=\frac{(\gamma-\varepsilon-1)\Gamma(\gamma)}{(\gamma-1)\Gamma(\gamma-\varepsilon)\Gamma(\varepsilon)}\frac{\theta(k-s)}{(k-s)^{1-\varepsilon}},\quad 0<\varepsilon<1, \tag{21}$$

satisfying the normalization condition (5). Here $\theta(x)$ is the step function [we need to include it to make the normalization integral (5) converge at ∞]. It is also required that $\gamma>1+\varepsilon$. We use the power law model (19) for the immunization preference function $\mu(k)$ with $\alpha<\gamma-1$. Then the critical epidemic threshold given by the Eq. (18) is

$$\lambda_c(k)=\frac{(\gamma-\alpha-1)}{(\gamma-1)(\gamma-\varepsilon-1)}\cdot\frac{\Gamma(\varepsilon)\Gamma(\gamma-\varepsilon)\Gamma(\gamma+\alpha-1)}{\Gamma(\gamma)\Gamma(\gamma+\alpha-\varepsilon-1)}\times k^{\gamma+\alpha-\varepsilon-1} \tag{22}$$

and is different for the different classes of vertices C_k exhibiting the power law behavior with k. The domains where the nontrivial critical epidemic threshold exists are determined by the poles of $\Gamma(x)$ and have a bandlike structure. They are

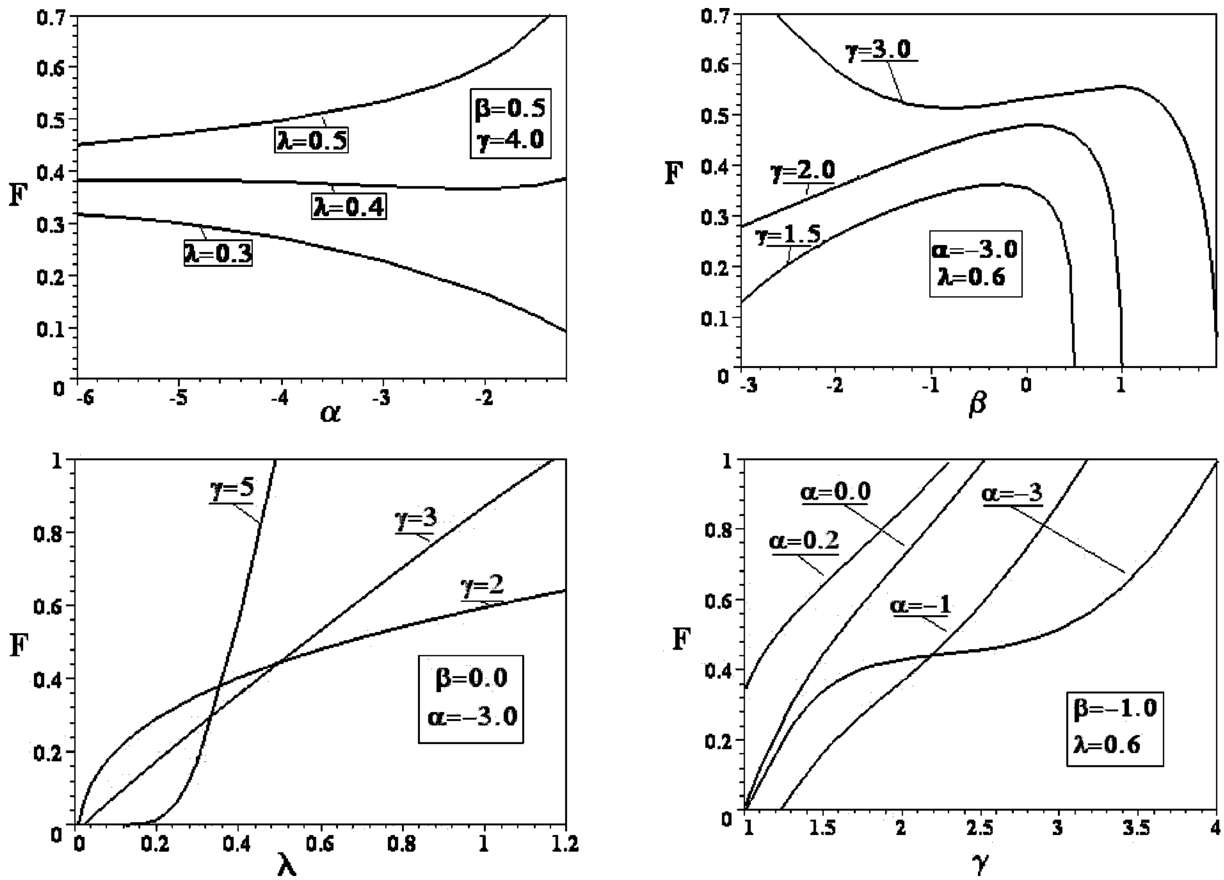

FIG. 2. The stationary fraction of infected individuals $F_\gamma > 0$ in the "power law" model (19) depending on the affinity parameters α and β characterizing the immunization policy and the social strategy chosen by individuals, effective spreading rate λ, and the index of the degree statistics γ. The figures reveal the absence of a plain immunization strategy which can be efficient simultaneously for all types of SF networks with power law preferences (19). The stationary fraction F_γ increases with the effective spreading rate of viruses λ and the index of degree statistics γ. The model predicts that the healing of certain classes of individuals does not help much in eradication of epidemics except for the small virus spreading rates $\lambda < 0.2$.

displayed in Fig. 1(b). The critical epidemic threshold is infinite as α takes negative integer values and has zeros somewhere between negative integers. The parameter ε determines the width of the band in critical domains.

The behavior of the stationary fraction of infected population at different values of λ, γ, α, and ε is displayed in Fig. (3). In general, it is rather different from the behavior observed for the unstructured community described by the power law model (19). Similarly to model (19), the fraction F_γ increases with the effective spreading rate λ, but decreases with γ and α. An efficient immunization program would be to vaccinate hubs and consolidate them to enlarge γ.

B. Dynamical solution of the evolution equation for low infection rates

Given the initial probability distribution of infected nodes $\phi(0,k)$, the dynamical solution $\phi(t,k)$ of Eq. (13) can be obtained in the following form:

$$\phi(t,k) = \nu k \int_0^t \Theta(k,\tau) \exp\left[\frac{\tau - t}{T(k,\tau)}\right] d\tau + \phi(k,0) \times \exp\left[-\frac{t}{T(k,t)}\right], \tag{23}$$

where the inverse relaxation time is

$$\frac{1}{T(k,t)} = \frac{1}{T(k)} + \frac{\nu k}{t}\int_0^t \tilde{\Theta}(k,\tau)\, d\tau,$$

$$\frac{1}{T(k)} = \delta\mu(k) + \nu k \Theta(k), \tag{24}$$

and $\Theta(k,t)$ is presented as the sum of the stationary probability $\Theta(k)$ satisfying the self-consistency equation (16) and the time-dependent part $\tilde{\Theta}(k,t)$, $\Theta(k,t) = \Theta(k) + \tilde{\Theta}(k,t)$.

Neglecting this time-dependent part $\tilde{\Theta}(k,t)$, we obtain

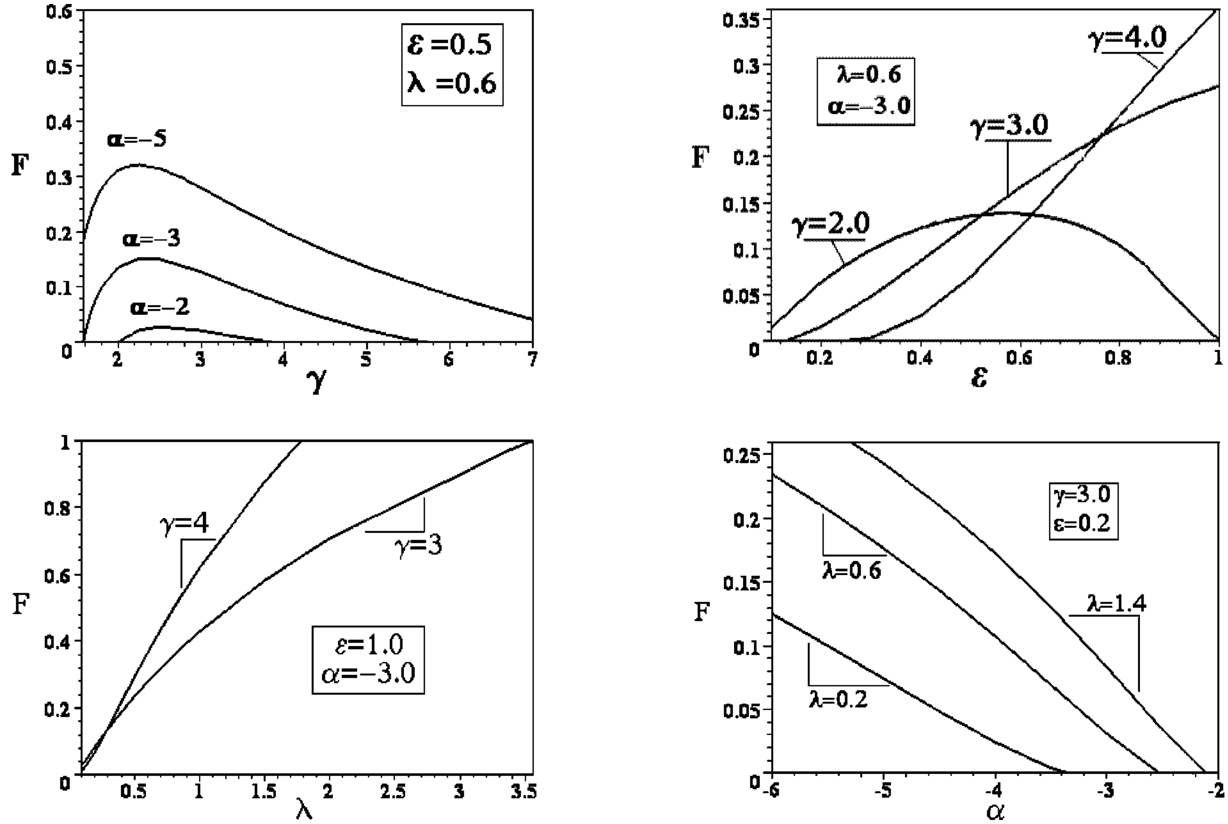

FIG. 3. The behavior of the stationary fraction of infected population at different values of λ, γ, α, and ε for model (21) in which individuals chose their partners from the classes of similar communication ability. Similarly to model (19), the fraction F_γ increases with the effective spreading rate λ but decreases with γ and α.

$$\phi(k,t)=f(\lambda,k)+\Delta(k)\exp\left[-\frac{t}{T(k)}\right], \tag{25}$$

where $\Delta(k)=\phi(k,0)-f(\lambda,k)$ is the departure of the initial probability distribution of infected population from the stationary solution.

The solution (25) is trivial as $\lambda<\lambda_c$. If $T(k)\gg 1$, the contribution coming from the initial distribution drives $\phi(k,t)$ out from the stationary solution even for very large time *t*. For the low infection rates $|\nu\Theta|\ll 1$, and if $\delta\ll 1$, the initial distribution $\phi(k,0)$ features the epidemic spreading over almost all vertices except maybe a few hubs accumulating a considerable fraction of connections.

IV. EPIDEMIC SPREADING IN EVOLUTIONARY SCALE-FREE NETWORKS

We conclude our paper with an example of a scale-free network, which has no critical epidemic threshold for any γ. A flexible algorithm generating SF networks based on the principle of evolutionary selection of a common large-scale structure of biological networks [3] has been discussed in Ref. [8] recently. Here we briefly reproduce this algorithm for the convenience of our readers, referring them to Ref. [8] for details.

One considers the three random variables x, y, and z that are the real numbers distributed in accordance with the distributions *f*, *g*, and v within the unit interval $[0,1]$. We assume that *x* represents the current performance of a biological network (say, the protein-protein interaction map), while *y* and *z* are the thresholds for outgoing and incoming edges, respectively. The network is supposed to be stable until $x<y$ and $x<z$, and is condemned otherwise. Fluctuations of thresholds reflect the changes in an environment.

The random process begins on the set of *N* vertices with no edges at time 0, at a chosen vertex *i*. Given two fixed numbers $\eta\in[0,1]$ and $\nu\in[0,1]$, the variable *x* is chosen with respect to probability distribution function (pdf) *f*, *y* is chosen with pdf *g*, and *z* is chosen with pdf v. We draw the e_{ij} edge leaving the *i* vertex and entering the *j* vertex if $x<y$ and $x<z$ and continue the process to time $t=1$. Otherwise, if $x\geq y$ $(x\geq z)$, the process moves to other vertices having no outgoing (incoming) links yet.

At time $t\geq 1$, one of three events happens.

(i) With probability η, the random variable *x* is chosen with pdf *f* but the thresholds *y* and *z* keep the values they had at time $t-1$.

(ii) With probability $1-\eta$, the random variable *x* is chosen with pdf *f*, and the thresholds *y* and *z* are chosen with pdf *g* and v, respectively.

(iii) With probability ν, the random variable *x* is chosen with pdf *f*, and the threshold *z* is chosen with pdf v, but the threshold *y* keeps the value it had at time $t-1$.

If $x\geq y$, the process stops at the *i* vertex and then starts at

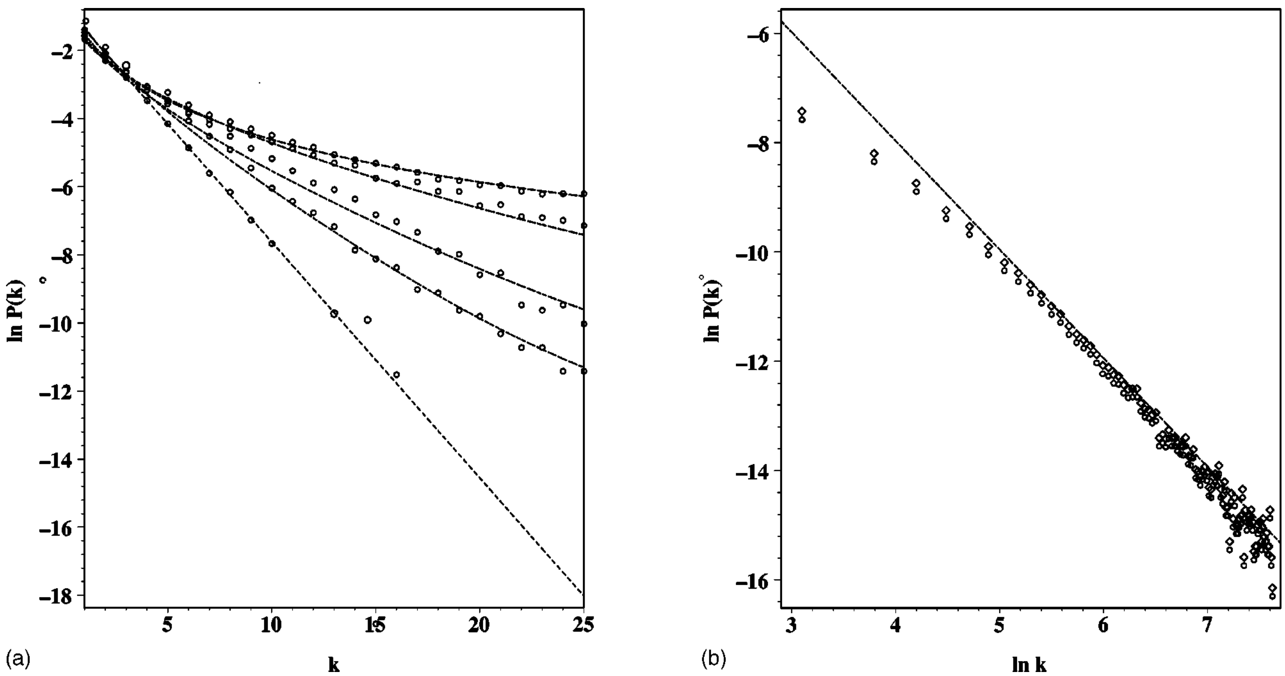

FIG. 4. Statistics of the evolutionary scale-free random graph. (a) The probability degree distributions $p(k)$ in the log-linear scale for different values of the parameter η, $\eta=0$, $\eta=0.5$, $\eta=0.7$, $\eta=0.9$, and $\eta=1.0$ (bottom to top). Straight line (bottom) corresponds to the pure exponential decay ($\sim 2^{-k}$) observed in the case $\eta=0$; the top line ($\eta=1.0$) corresponds to the power law decay $\sim k^{-2}$. (b) The probability degree distribution $p(k)$ vs k in the log-log scale generated on $N=10^5$ nodes for $f=g=v=1$, $\eta=1$, $\nu=0$. Here, the circles stay for the outgoing degrees $k_{\rm out}$ and diamonds are for the incoming degrees $k_{\rm in}$. Both profiles enjoy a power law decay with $\gamma=2$.

some other vertex having no outgoing edges yet. If $x \geqslant z$, the accepting vertex j is blocked and does not admit any more incoming links (provided it has any). If $x<y$ and $x<z$, the process continues at the same vertex i and goes to time $t+1$.

It has been shown in Ref. [8] that the above model exhibits a multivariant behavior depending on the probability distribution functions f, g, and v chosen and values of relative frequencies η and ν. In particular, if $\nu=0$, both thresholds y and z have synchronized dynamics, and sliding the value of η from 0 to 1, one can tune the statistics of out-degrees and in-degrees simultaneously out from the pure exponential decay (for $\eta=0$) to the power laws (at $\eta=1$) provided f, g, and v belong to the class of power law functions. For instance, by choosing the probability distribution functions in the following forms,

$$f(u)=(1+\alpha)u^{\alpha}, \quad \alpha>-1,$$
$$v(u)\equiv g(u)=(1+\beta)(1-u)^{\beta}, \quad \beta>-1, \tag{26}$$

one obtains that

$$p_{\eta=1}(k) \simeq_{k\gg 1} \frac{(1+\beta)\Gamma(2+\beta)(1+\alpha)^{-1-\beta}}{k^{2+\beta}}\left[1+0\left(\frac{1}{k}\right)\right]. \tag{27}$$

For different values of β, the exponent of the threshold distribution, one gets all possible power law decays of $p_{\eta=1}(k)$. Notice that the exponent $\gamma=2+\beta$ characterizing the decay of $p_{\eta=1}(k)$ is independent of the distribution $f(u)$ of the state variable x. In the uncorrelated case, $\eta=0$, the degree distribution function decays exponentially (for instance, $p_{\eta=0}=2^{-k}$ for $f=g=v=1$) [8]. For the intermediate values of η, the decay rate is mixed.

In Fig. 4(a), we have plotted these asymptotic profiles $p(k)$ vs k in the log-linear scale for the case of uniform densities $f=g=v=1$, for the consequent frequency values $\eta=0$, $\eta=0.5$, $\eta=0.7$, $\eta=0.9$, $\eta=1$ (bottom to top). In Fig. 4(b), we have presented the distribution $p(k)$ vs k in the log-log scale over $N=10^5$ vertices for $f=g=v=1$, $\eta=1$, $\nu=0$. Here, the circles stay for outgoing degrees, and diamonds are for incoming degrees. For $k \gg 1$, both profiles enjoy a power law decay with $\gamma_{\rm in}=\gamma_{\rm out}=2$.

Interestingly, in epidemic spreading properties in such an evolutionary network, we note that the preferred function for the above model is

$$\sigma(k)=(1+\beta)\left(1-\frac{k}{N-1}\right)^{\beta}, \quad \beta>-1. \tag{28}$$

Expanding the binomial in the above equation, one gets the leading term $\propto (k/N-1)^{\beta}$. Consequently, the integral determining the critical epidemic threshold diverges and $\lambda_c=0$ for any γ.

Figures 5(a) and 5(b) illustrate the absence of the critical epidemic threshold for model (28) (we have checked this fact for γ up to several tenths) and reveal the complexity of the epidemic spreading in an evolutionary SF network as a dynamical system. We have presented the results of numerical simulations for the epidemic spreading process in the above

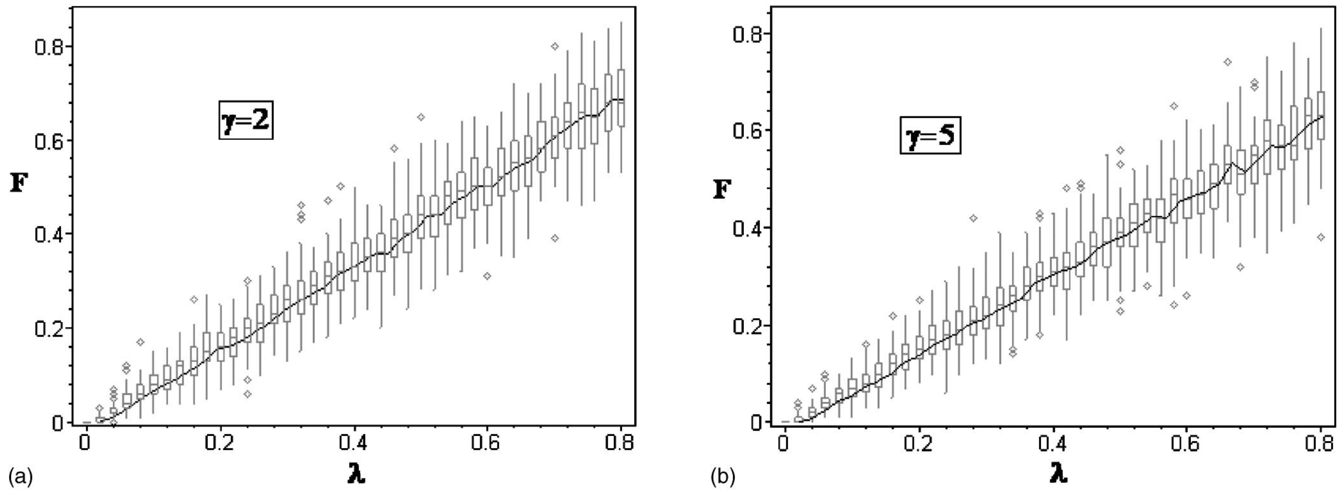

FIG. 5. The fraction of infected agents in the evolutionary SF networks vs the effective spreading rate λ for different values of γ. (a) $p(k)\propto k^{-2}$, (b) $p(k)\propto k^{-5}$. At the onset of the process, the initial state (''healthy'' or ''infected'') has been assigned to each of 1000 nodes by the coin tossing procedure. Starting from such a random configuration of initially infected individuals, each infection spreading process was simulated in 1000 consequent iterations and then started again with a different random initial configuration. The bold lines represent the mean infected fraction averaged over 500 spreading processes vs the effective spreading rate λ. The error bars correspond to the standard deviations. Here, a lower line of boxes shows the first quartile of data and an upper line shows the third quartile.

network (for $\gamma=2$ and $\gamma=5$) starting from the randomly chosen configuration of initially infected individuals. At the onset of the process, the initial state (''healthy'' or ''infected'') has been assigned to each of $N=10^3$ nodes by the coin tossing procedure with a probability 1/2. Each infection spreading process takes 1000 consequent iterations and then starts again with a different random initial configuration of infected individuals. The bold lines represent the mean infected fraction averaged over 500 spreading processes vs the effective spreading rate λ. The error bars correspond to the standard deviations. Here, a lower line of boxes shows the first quartile of data, and an upper line shows the third quartile.

V. CONCLUSION

In the present paper, we have shown that epidemic spreading in scale-free networks is very sensitive to the statistics of degree distribution characterized by the index γ, the effective spreading rate of a virus, λ, the social strategy using by individuals to choose a partner, and the policy of administrating a cure to an infected node. Depending on the interplay of these four factors, the stationary fractions of infected population F_γ as well as the epidemic threshold properties can be essentially different.

We have considered two alternative models for the partner choice preference. In the first model, the society is unstructured in the sense that an individual is chosen as a partner depending only on its connectivity degree k. In the second model, the coupling strength between vertices $v\in C_k$ and $w\in C_s$ belonging to different classes is assumed to depend on the difference $|k-s|$ and fades out if $|k-s|\gg 1$. We have demonstrated that the epidemic spreadings in these two types of SF networks are dramatically different even if their indices γ are equal. Probably, it is impossible to obtain a simple immunization program that can be simultaneously effective for all types of SF networks.

We have also studied the dynamical solutions for the evolution equation governed by the epidemic spreading in SF networks and found an expression for the relaxation time $T(k)$ such that if $t\leqslant T(k)$, the disease spreading depends crucially upon the initial distribution of infected individuals in the network. In particular, we have shown that for the case of vanishingly small cure rate $\delta\ll 1$ the initial configuration would feature the solution for very long times.

Finally, we have given the example of the evolutionary SF network which is disposed to the spreading and the persistence of infections at any spreading rate $\lambda>0$ for any value of the index γ. We have demonstrated that such a network is strongly influenced by the initial configuration of the infected nodes even for long times.

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