Influence of dynamical condensation on epidemic spreading in scale-free networks

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Considering the accumulation phenomenon in public places, we investigate how the condensation of moving bosonic particles influences the epidemic spreading in scale-free metapopulation networks. Our mean-field theory shows that condensation can significantly enhance the effect of epidemic spreading and reduce the threshold for epidemic to survive, in contrast to the case of without condensation. In the stationary state, the number of infected particles increases with the degree k linearly when $k<k_c$ and nonlinearly when $k>k_c$, where k_c denotes the crossover degree of the nodes with unity particle. The dependence of critical infective rate β_c on the parameters $k_{\max}$, μ, and δ, is figured out, where $k_{\max}$, μ, and δ denote the largest degree, recovery rate, and jumping exponent, respectively. Numerical simulations have confirmed the theoretical predictions.

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I. INTRODUCTION

In the last decade, complex networks have provided an increasingly challenging framework for the study of collective behaviors based on the interplay between the topological structures and the dynamics at nodes. Much progress has been achieved, such as in the aspects of synchronization, packet delivering, epidemic spreading, and energy transportation, etc. [1–5]. We here focus on the topic of epidemic spreading.

Epidemic spreading has been well studied in two typical models: Susceptible-infected-susceptible (SIS) and susceptible-infected-refractory (SIR) models [6–9]. In these models, each susceptible node is infected with probability β at each time step if it is connected to one infected node. At the same time, the infected nodes are cured and become susceptible again with probability μ, defining an effective spreading rate $\lambda=\beta/\mu$. A key problem in epidemic spreading is how to figure out the threshold for epidemic to survive. In random homogeneous networks the most significant result is the general prediction of a nonzero epidemic threshold $\lambda_c=1/\langle k\rangle$ with $\langle k\rangle$ denoting the average degree [6,7]. If the value of λ is above the threshold, i.e., $\lambda>\lambda_c$, the infection spreads and becomes persistent in time; otherwise, the infection dies out exponentially fast.

It has been revealed that most of the realistic networks are scale-free (SF) networks and the results obtained on the random homogeneous networks do not work there. For studying how the SF topology influences the epidemic spreading, much attention has been paid recently [1–3] and some important results have been achieved, such as the infection in SF network can spread to the entire network even if the probability of transmission is infinitely small, i.e., $\lambda_c=\langle k\rangle/\langle k^2\rangle\approx 0$ for large network size [10–12]. This results is in sharp contrast to the well-known threshold phenomenon in epidemiology [6,7].

The above studies are mainly focused on networked static systems where each node corresponds to a single individual and the individuals cannot move. However, the real social networks are based on the heterogeneous topologies of bosonic systems, where nodes can be occupied by any number of particles and individuals can move from one node to another. A good model to characterize this situation is the so-called metapopulation model [13] where particles represent people moving across different subpopulations (nodes) such as city or urban areas. Metapopulation models rely on the basic assumption that the system under study is characterized by a highly fragmented environment where the population is structured and localized in relatively isolated discrete subpopulations connected by some degree of migration. Especially for disease spreading in the urban networks, the spatial structure of populations is a key element in the understanding of the large scale spreading of epidemics. For studying epidemic spreading in social networks, Colizza *et al.* has recently studied a reaction-diffusion process in SF network where the reaction (infection) takes place when particles stay in the same node [14,15]. They find that the epidemic persists only when the total density of particles satisfy the condition $\rho>\rho_c$, with $\rho_c=\frac{\langle k\rangle^2}{\langle k^2\rangle}\frac{\mu}{\beta}$. In other words, there is a critical infection rate $\beta_c=\frac{\langle k\rangle^2}{\langle k^2\rangle}\frac{\mu}{\rho}$ for a fixed density ρ and epidemic survives when $\beta>\beta_c$. In the thermodynamic limit, we have $\langle k^2\rangle\to\infty$ for SF network and thus $\beta_c=0$. Zhou *et al.* has recently studied the epidemic spreading in dynamical communities with different densities and found that both direct and indirect contacts can seriously influence the safety of communities [16]. These results open a new window of studying the epidemic spreading from the angle of reaction-diffusion process. We here try to widen this window a little.

On the other hand, the moving of particles may result in accumulation and/or condensation at the hub to some extent. In statistical physics, condensation means that a finite fraction of the total particles will be condensed to a single state, i.e., the ground state, when the temperature approaches to zero, which is called as the Bose-Einstein condensation. We here keep this meaning and think that there is condensation in complex networks once a finite fraction of total particles accumulates at the hub node. This problem was first studied in regular lattice [17–21] and recently studied in SF networks [22–25]. For the former, one of the necessary conditions for condensation to occur is that the density of particles in lattice must be over some threshold. While for the latter, it is found

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that the condensation may occur at any finite value of density [23]. For studying the influence of network structure on condensation, Noh *et al.* considered a zero range process (ZRP) of interaction in SF network and found that most of the particles may be condensed to the hub nodes [23,24]. We have investigated how the weights of links influence the condensation and found that it is possible for the weights to make the condensation occur even when there is no attraction among particles [25]. Considering the ZRP interaction as a reaction and the moving of particles as a diffusion, the condensation phenomenon is in fact a consequence of reaction-diffusion process. This phenomenon of condensation can be also observed very often in the social networks or metapopulation models where the individuals sometimes prefer to accumulate at the public places and hence result in high density localization, such as at schools and malls, etc. If there is a seed of virus and/or disease in the population, the reaction-diffusion process will become an epidemic spreading process and the occurred condensation will definitely influence the epidemic spreading. Then an interesting question is how the accumulation and/or condensation influences the epidemic spreading.

In this paper, we will combine together the two phenomena, i.e., epidemic spreading and condensation, and aim to understand how the accumulation and/or condensation influences the epidemic spreading. Our results show that the condensation can significantly enhance the effect of epidemic spreading and reduce the threshold for epidemic to survive, in contrast to the case of without condensation. Moreover, we have figured out the influence of parameters, such as the largest degree, recovery rate, and jumping exponent, etc., on the critical infective rate β_c. Numerical simulations have completely confirmed the theoretical predictions. We organize the paper as follows. In Sec. II, we present a model to describe the epidemic spreading in the framework of ZRP and figure out its solution through a mean-field approach. Then in Sec. III, we make numerical simulations to confirm the predictions given in Sec. II. Finally, we give discussions and conclusions in Sec. IV.

II. MODELING THE EPIDEMIC SPREADING IN THE FRAMEWORK OF ZRP

We here consider the uncorrelated configuration model (UCM) with power-law degree distribution $P(k) \sim k^{-3}$ [26]. We let each node of the network have an infinite capacity and randomly set n_i particles at node i. Suppose the total number of nodes is N, then the density of particles in the network is $\rho = \sum_{i=1}^{N} n_i / N$. At each time step, part of the n_i particles at the node i can jump out with jumping exponent δ in [0,1]. Let $p(n_i)$ be the number of people diffusing out of a given place and/or node at a time step. Then $p(n_i)/n_i$ represents the jumping rate. We here take $p(n_i)$ as

$$p(n_i) = n_i^{\delta}. \tag{1}$$

$\delta = 0$ means that only one of the n_i particles will jump out per each time step, indicating that the particles are attracting each other. While in the case of $\delta = 1$, all the n_i particles will jump out, implying that they are moving independently. For the middle δ between 0 and 1, the jumping out particles will be in between 1 and n_i.

At the same time, each particle jumping out from the node i will hop randomly to one of its neighboring nodes j, i.e., the hopping rate takes the expression

$$T_{j \leftarrow i} = \frac{1}{k_i}. \tag{2}$$

It has been shown that a complete condensation occurs when $\delta < \delta_c = 1/2$; otherwise, no condensation [23,24]. In condensation, the hub nodes are occupied by macroscopic numbers of particles, while the other nodes are occupied by negligible numbers of particles.

Considering the heterogeneous structure of UCM, the hub nodes will have larger probability to get particles than the nodes with small links. There are two ways to describe the evolution of particles at a node. One is the approach of canonical ensemble [17,22–24] and the other is the mean-field approach [25]. We here take the second one. In the mean-field approach, we transfer the description of n_i for each node to the description of the mean occupation number $m_k(t)$ for the nodes with the same degree k, i.e., $m_k(t)$ is the average of all the n_i at the nodes with degree k. Hence $m_k(t)$ is no longer necessary to be an integer. Correspondingly, we transfer the jumping rate $p(n_i)$ to $p(m_k) = m_k^{\delta}$.

When $m_k \leqslant 1$, we have $m_k^{\delta} \geqslant m_k$ and the number of jumping particles is in fact m_k but not m_k^{δ} as we do not have so many average particles m_k^{δ} at those nodes with degree k. When $m_k > 1$, we have $m_k^{\delta} < m_k$ and hence the number of jumping particles is m_k^{δ}. Let k_c be the crossover degree with $m_{k_c} = 1$. Except the aspect of jumping out, at the same time, a node accepts particles from its neighbors. The incoming particles can be classified into two parts: One from the node with $m_k < 1$ and the other from the nodes with $m_k \geqslant 1$. The incoming particles from one neighboring node with degree k' is $P(k'|k) m_{k'}(t)/k'$ when $m_{k'}(t) < 1$ and $P(k'|k) m_{k'}^{\delta}(t)/k'$ when $m_{k'}(t) \geqslant 1$, where the conditional probability $P(k'|k) = k' P(k')/\langle k \rangle$ for the UCM.

We now consider the SIS model for the mobile particles. Suppose a particle represents an agent with two states, i.e., susceptible and infectious, and a node represents a location to be occupied by agents. The agents may interact with each other only when they stay at the same location and/ or node, thus we obtain an agent-based model. Let $m_{I,k}$ and $m_{S,k}$ be the mean number of infectious and susceptible particles and/or agents at those nodes with degree k, respectively. Obviously, we have

$$m_k = m_{I,k} + m_{S,k}. \tag{3}$$

Following Ref. [14] we divide the reaction-diffusion process into two steps. In the first step, the susceptible particles take infection with the infected particles at the same node. In the second step, both the susceptible and infected particles take diffusion. Suppose the infection rate is denoted by β, which means that each susceptible node can be infected by a probability β if it contacts an infectious. We also suppose that the infected node can be cured automatically by a probability μ.

When the susceptible and infected agents are fully mixed, the number of new infected agents is

$$\Gamma_k = m_{S,k}[1-(1-\beta)^{m_{I,k}}], \tag{4}$$

where the factor $(1-\beta)^{m_{I,k}}$ represents the uninfected probability when a susceptible contacts with $m_{I,k}$ infected agents [27]. Equation (4) becomes $\Gamma_k=\beta m_{S,k}m_{I,k}$ when $\beta \ll 1$. Right after the reaction or right before the diffusion, the particles with infectious status are $(1-\mu)m_{I,k}+\Gamma_k$ and the particles with the susceptible status are $\mu m_{I,k}+m_{s,k}-\Gamma_k$. After a finite number of evolutionary steps, the system will arrive at a stationary distribution where we have $m_k<1$ for $k<k_c$ and $m_k>1$ for $k>k_c$. We here discuss the evolution of particles based on the stationary solution. Considering the different forms of number of jumping particles for $k<k_c$ and $k>k_c$, i.e., $p(m_k)=m_k$ for $k<k_c$ and $p(m_k)=m_k^\delta$ for $k>k_c$, we distinguish the evolution of particles at the nodes with $k<k_c$ from that with $k>k_c$. For the nodes with $k<k_c$, all of the m_k particles will be diffused to the surroundings, thus each of the infectious and susceptible particles will have the possibility of unity to be diffused. While for the nodes with $k>k_c$, only a part of m_k particles, i.e., m_k^δ particles, will be diffused to the surroundings, indicating that each of the infectious and susceptible particles will have possibility $m_k^\delta/m_k=m_k^{\delta-1}<1$ to be diffused. Therefore, at each time step, the diffused infectious particles at a node are $[(1-\mu)m_{I,k}+\Gamma_k]m_k^{\delta-1}$ and the diffused susceptible particles are $(\mu m_{I,k}+m_{s,k}-\Gamma_k)m_k^{\delta-1}$. Based on this analysis, the mean-field equations for the evolution of $m_{I,k}$ and $m_{S,k}$ with $k<k_c$ can be given as follows:

$$\frac{\partial m_{I,k}}{\partial t} = -m_{I,k} + \frac{k}{\langle k\rangle}\left(\sum_{k'=k_{\min}}^{k_c} P(k')[(1-\mu)m_{I,k'}+\Gamma_{k'}] + \sum_{k'=k_c}^{k_{\max}} P(k')[(1-\mu)m_{I,k'}+\Gamma_{k'}]m_{k'}^{\delta-1}\right),$$

$$\frac{\partial m_{S,k}}{\partial t} = -m_{S,k} + \frac{k}{\langle k\rangle}\left(\sum_{k'=k_{\min}}^{k_c} P(k')(\mu m_{I,k'}+m_{S,k'}-\Gamma_{k'}) + \sum_{k'=k_c}^{k_{\max}} P(k')(\mu m_{I,k'}+m_{S,k'}-\Gamma_{k'})m_{k'}^{\delta-1}\right), \tag{5}$$

where the first term $-m_{I,k}$ or $-m_{S,k}$ comes from the fact that all the particles at the nodes with $k<k_c$ will jump out at the next step, the second term denotes the gained particles from the surrounding neighbors, and $k_{\min}$ and $k_{\max}$ denote the minimum and maximum degrees, respectively. The part with $\Sigma_{k'=k_{\min}}^{k_c}$ represents the gained particles from the neighbors with $k<k_c$ and the part with $\Sigma_{k'=k_c}^{k_{\max}}$ represents the gained particles from the neighbors with $k>k_c$. Similarly, the mean-field equations for the evolution of $m_{I,k}$ and $m_{S,k}$ with $k>k_c$ are

$$\frac{\partial m_{I,k}}{\partial t} = -m_{I,k} + [(1-\mu)m_{I,k}+\Gamma_k](1-m_k^{\delta-1}) + \frac{k}{\langle k\rangle}\left(\sum_{k'=k_{\min}}^{k_c} P(k')[(1-\mu)m_{I,k'}+\Gamma_{k'}] + \sum_{k'=k_c}^{k_{\max}} P(k')[(1-\mu)m_{I,k'}+\Gamma_{k'}]m_{k'}^{\delta-1}\right),$$

$$\frac{\partial m_{S,k}}{\partial t} = -m_{S,k} + (\mu m_{I,k}+m_{S,k}-\Gamma_k)(1-m_k^{\delta-1}) + \frac{k}{\langle k\rangle}\left(\sum_{k'=k_{\min}}^{k_c} P(k')(\mu m_{I,k'}+m_{S,k'}-\Gamma_{k'}) + \sum_{k'=k_c}^{k_{\max}} P(k')(\mu m_{I,k'}+m_{S,k'}-\Gamma_{k'})m_{k'}^{\delta-1}\right), \tag{6}$$

where the factor $1-m_k^{\delta-1}$ denotes the possibility for individuals to remain at their positions, the sum of the first two terms denotes the part of jumping out, and the third term denotes the part gained from the neighbors.

Equations (5) and (6) are the evolution equations of infectious and susceptible particles and will reach the stationary state when they evolve enough time. In the stationary state, we may solve Eqs. (5) and (6) through the condition $\partial m_{I,k}/\partial t=0$ and $\partial m_{S,k}/\partial t=0$ to get the stabilized $m_{I,k}$ and $m_{S,k}$. By doing this we have

$$m_{I,k} = kA,$$

$$m_{S,k} = kB, \tag{7}$$

for $k<k_c$ and

$$m_{I,k} = [(1-\mu)m_{I,k}+\Gamma_k](1-m_k^{\delta-1}) + kA,$$

$$m_{S,k} = (\mu m_{I,k}+m_{S,k}-\Gamma_k)(1-m_k^{\delta-1}) + kB, \tag{8}$$

for $k>k_c$, where

$$A = \frac{1}{\langle k\rangle}\{\sum_{k'=k_{\min}}^{k_c} p(k')[(1-\mu)m_{I,k'}+\Gamma_{k'}] + \sum_{k'=k_c}^{k_{\max}} p(k')[(1-\mu)m_{I,k'}+\Gamma_{k'}]m_{k'}^{\delta-1}\}$$

and

$$B = \frac{1}{\langle k\rangle}[\sum_{k'=k_{\min}}^{k_c} p(k')(\mu m_{I,k'}+m_{S,k'}-\Gamma_{k'}) + \sum_{k'=k_c}^{k_{\max}} p(k')(\mu m_{I,k'}+m_{S,k'}-\Gamma_{k'})m_{k'}^{\delta-1}].$$

From the definition of k_c and Eq. (7) we have $A+B=1/k_c$.

Equations (7) and (8) are the stationary solution of $m_{I,k}$ and $m_{S,k}$. From Eq. (7) it is easy to see that both $m_{I,k}$ and $m_{S,k}$

increases linearly with k for $k<k_c$. Thus, the infected fraction

$$\frac{m_{I,k}}{m_k} = \frac{A}{A+B} \tag{9}$$

is a constant for $k<k_c$. While for $k>k_c$, it is generally difficult to get the solution of Eq. (8). When $\beta \ll 1$, however, we can obtain the approximate solution by substituting $\Gamma_k=\beta m_{S,k} m_{I,k}$ and the condition $m_{S,k}=m_k-m_{I,k}$ into Eq. (8). Through simple calculation we obtain

$$m_{I,k} = \frac{F(m_k) + \sqrt{F^2(m_k) + 4\beta k A(1-m_k^{\delta-1})}}{2\beta(1-m_k^{\delta-1})}, \tag{10}$$

where $F(m_k)=(\beta m_k-\mu)(1-m_k^{\delta-1})-m_k^{\delta-1}$. Considering $m_k>1$ for $k>k_c$, we have approximately $m_{I,k}\sim m_k$. By summing the two equations of Eq. (8) we have $m_k=(k/k_c)^{1/\delta}$, thus we obtain

$$m_{I,k} \sim \left(\frac{k}{k_c}\right)^{1/\delta} - \frac{\mu}{\beta} - \frac{1}{\beta[(k/k_c)^{(1-\delta)/\delta}-1]} \sim k^{1/\delta} \quad \text{for } k>k_c, \tag{11}$$

indicating $m_{I,k}$ increases nonlinearly with k for $k>k_c$. The threshold k_c can be determined by the self-consistent condition $\Sigma_{k=k_{\min}}^{k_c} m_k P(k) N + \Sigma_{k=k_c}^{k_{\max}} m_k P(k) N = \rho N$, which gives

$$\int_{k_{\min}}^{k_c} dk P(k) \frac{k}{k_c} + \int_{k_c}^{k_{\max}} dk P(k) \left(\frac{k}{k_c}\right)^{1/\delta} = \rho. \tag{12}$$

Thus, k_c depends on the density ρ. A larger density ρ corresponds to a smaller k_c.

Furthermore, from Eq. (10) we obtain

$$\frac{m_{I,k}}{m_k} \approx 1 - \frac{\mu}{\beta m_k} - \frac{1}{\beta m_k (m_k^{1-\delta}-1)} = 1 - \frac{\mu}{\beta}\left(\frac{k_c}{k}\right)^{1/\delta} - \frac{1}{\beta}\left(\frac{k_c}{k}\right)^{1/\delta} \frac{1}{\left(\frac{k}{k_c}\right)^{(1-\delta)/\delta}-1}, \tag{13}$$

indicating that the infected fraction $m_{I,k}/m_k$ will increase with the degree k and the infection rate β but decrease with the recovery rate μ and the jumping exponent δ. Especially, from Eq. (13) we have $m_{I,k}/m_k \approx 1$ for $k \gg k_c$. Obviously, Eq. (13) does not work for the case of $\delta=1$. In this situation, we should go back to the approach used by Colizza *et al.* in Ref. [14] as there is no accumulation.

Without condensation, the threshold for epidemic to survive is $\beta_c \sim \langle k \rangle^2 / \langle k^2 \rangle$ [14]. With condensation, the threshold can be figured out by letting $m_{I,k_{\max}}>0$, which gives

$$\beta_c \sim \left(\frac{k_c}{k_{\max}}\right)^{1/\delta} \left(\mu + \frac{1}{\left(\frac{k_{\max}}{k_c}\right)^{(1-\delta)/\delta}-1}\right). \tag{14}$$

Considering that $k_c \sim (k_{\max})^{1-\delta/\delta_c}$ for $\delta<\delta_c$ [24], Eq. (14) becomes

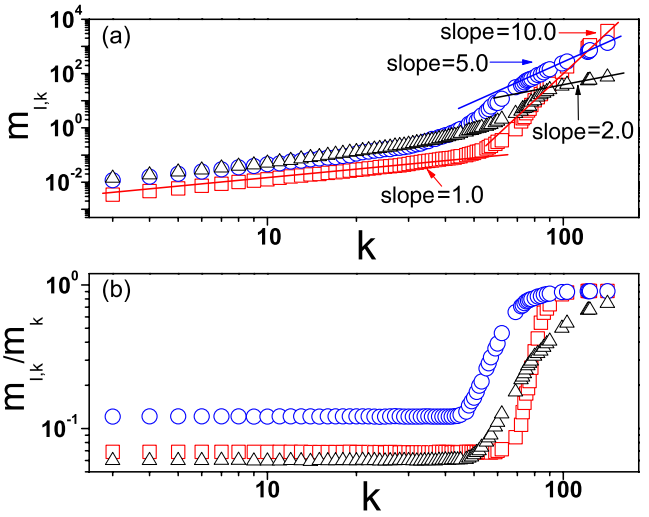

FIG. 1. (Color online) slope--> Infected particles in the stationary state with "squares," "circles," and "triangles" denoting the cases of $\delta=0.1$, 0.2, and 0.5, respectively. The parameters are chosen as $N=20\,000$, $\rho=0.5$, $\beta=0.01$, and $\mu=0.1$. (a) $m_{I,k}$ versus k; (b) the fraction $m_{I,k}/m_k$ versus k.

$$\beta_c \sim k_{\max}^{-1/\delta_c}\left(\mu + \frac{1}{k_{\max}^{(1-\delta)/\delta_c}-1}\right) \sim k_{\max}^{-1/\delta_c}\mu \tag{15}$$

for $\delta<\delta_c$. That is, β_c will be a small constant value for $\delta<\delta_c$ and then gradually increase with δ according to Eq. (14) for $\delta>\delta_c$. Equations (7), (9), (11), (13), and (15) are our main results. We will confirm them numerically in the next section.

III. NUMERICAL SIMULATIONS

In numerical simulations, we would like to use an uncorrelated heterogeneous network, i.e., the UCM network, to confirm the theoretical predictions. We first construct a UCM network with degree distribution $P(k)\sim k^{-3}$, size $N=2\times10^4$, and the constraints $m=3\leqslant k\leqslant N^{1/2}$, according to the algorithm given in [26]. Then, we randomly put 1×10^4 particles at the N nodes, i.e., the density $\rho=0.5$, and let them evolve according to Eqs. (1) and (2). After the transient time, it is easy to observe the particle condensation when $\delta<\delta_c=0.5$. For understanding how the condensation influences the epidemic spreading, we randomly choose a few particles as the seeds of virus and/or disease, i.e., they are the infectious. Then, the infection process begins. After the transient process of infection, the number of infected particles will be stabilized. As the stationary state is a dynamical equilibrium, we make time average to reduce the fluctuation of $m_{I,k}$ and $m_{S,k}$. That is, we let $m_{I,k}=\frac{1}{T}\Sigma_{t=1}^{T} m_{I,k}(t)$ and $m_{S,k}=\frac{1}{T}\Sigma_{t=1}^{T} m_{S,k}(t)$ with $T=10^4$. For reducing the random effect caused by the initial conditions, we make average of $m_{I,k}$ and $m_{S,k}$ on 100 realizations of different initial infectious particles. Figure 1 shows the results for $\mu=0.1$ and $\beta=0.01$, where (a) represents the distribution of $m_{I,k}$ on different k and (b) the fraction $m_{I,k}/m_k$ on different k, and the "squares," "circles," and "triangles" in both (a) and (b) denote the cases

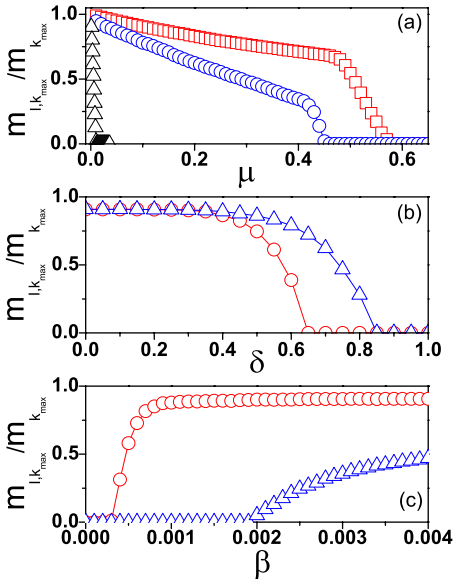

FIG. 2. (Color online) Influence of parameters μ, δ, and β on the fraction $m_{I,k_{\max}}/m_{k_{\max}}$ with $N=20\,000$ and $\rho=0.5$. (a) $m_{I,k_{\max}}/m_{k_{\max}}$ versus μ for $\beta=0.01$ and the "squares," "circles," and "triangles" denote the cases of $\delta=0.2$, 0.5, and 1.0, respectively. (b) $m_{I,k_{\max}}/m_{k_{\max}}$ versus δ for $\mu=0.1$ and the "circles" and "triangles" denote the cases of $\beta=0.01$ and 0.05, respectively. (c) $m_{I,k_{\max}}/m_{k_{\max}}$ versus β for $\mu=0.1$ and the "circles" and "triangles" denote the cases of $\delta=0.2$ and 0.5, respectively.

of $\delta=0.1$, 0.2, and 0.5, respectively. From Fig. 1(a) it is easy to see that all the slopes of the three cases are unity for $k<k_c$ and the slope is approximate 10.0 for the case of $\delta=0.1$, 5.0 for the case of $\delta=0.2$ and 2.0 for the case of $\delta=0.5$ when $k>k_c$, i.e., they are approximate $1/\delta$ for $k>k_c$, confirming Eqs. (7) and (11). From Fig. 1(b) we see that the fraction $m_{I,k}/m_k$ is a constant for $k<k_c$ and a nonlinear increasing function for $k>k_c$, confirming Eqs. (9) and (13). Especially, the fraction $m_{I,k}/m_k$ approaches to unity for those nodes with the largest k when $\delta<\delta_c$, indicating that most of the particles at the hub nodes are infected because of the condensation.

For confirming the influence of parameters μ, δ, and β on the fraction $m_{I,k}/m_k$ in Eq. (13), we take $k=k_{\max}$ as an example. Figure 2 shows the results where (a) represents $m_{I,k_{\max}}/m_{k_{\max}}$ versus μ, (b) $m_{I,k_{\max}}/m_{k_{\max}}$ versus δ, and (c) $m_{I,k_{\max}}/m_{k_{\max}}$ versus β. In Fig. 2(a) we fix $\beta=0.01$ and let the "squares," "circles," and "triangles" denote the case of $\delta=0.2$, 0.5, and 1.0, respectively. Obviously, the fraction $m_{I,k_{\max}}/m_{k_{\max}}$ in the case of $\delta=1$ survives only for a very small μ; while the cases for $\delta=0.2(<\delta_c=0.5)$ and $\delta=0.5(=\delta_c)$ survive for large μ, indicating that the condensation enhances the effect of the epidemic spreading significantly. This point can be seen more clear in Fig. 2(b) where we fix $\mu=0.1$ and let the "circles" and "triangles" denote the cases of $\beta=0.01$ and 0.05, respectively. It is easy to see that the fraction $m_{I,k_{\max}}/m_{k_{\max}}$ is approximate unity for $\delta<\delta_c$ and gradually decreases to zero for $\delta>\delta_c$. In Fig. 2(c) we fix $\mu=0.1$ and let the "circles" and "triangles" denote the cases of $\delta=0.2$ and 0.5, respectively. It is easy to see that the needed minimum β for the surviving of $m_{I,k_{\max}}/m_{k_{\max}}$ is much larger in the case of $\delta=0.5$ than that of $\delta=0.2$, confirming the condensation enhanced epidemic spreading again. These results are completely consistent with Eq. (13).

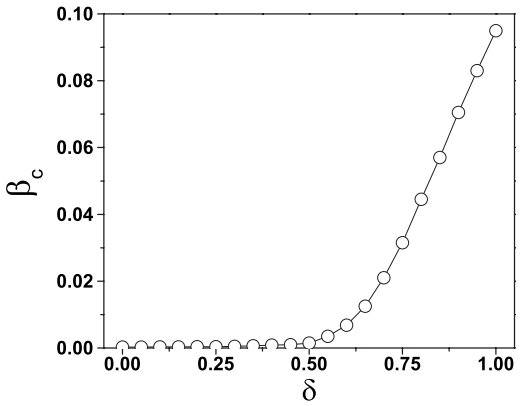

FIG. 3. The critical β_c versus the jumping exponent δ with $N=20\,000$, $\rho=0.5$, and $\mu=0.1$.

Finally, we check the relationship between the critical β_c and the jumping exponent δ for a fixed μ. By gradually increasing β from zero, we check the ratio $m_{I,k_{\max}}/m_{k_{\max}}$. β_c is the transition point when the ratio $m_{I,k_{\max}}/m_{k_{\max}}$ changes from zero to positive. Figure 3 shows the result. It is easy to see that β_c is a very small constant value for $\delta<\delta_c$ and increases with δ for $\delta>\delta_c$, confirming Eqs. (14) and (15). Furthermore, from Fig. 3 we see that the β_c for $\delta<\delta_c$ is much smaller than that of $\delta=1$ with no condensation, indicating that the condensation reduces β_c significantly.

IV. DISCUSSIONS AND CONCLUSIONS

Condensation favorite epidemic spreading can be understood as follows. In the condensation, most of the particles are condensed at the hub nodes. Thus, the surviving of epidemic in SF network is equivalent to the surviving of epidemic at the hub. Each particle at the hub contacts the other $m_{k_{\max}}-1$ particles and can be considered as having $m_{k_{\max}}-1$ links, hence the threshold is $\beta_c \sim 1/(m_{k_{\max}}-1) \sim (k_c/k_{\max})^{1/\delta} = k_{\max}^{-1/\delta_c} = 1/k_{\max}^2$, which is much less than $\beta_c = \frac{\langle k\rangle^2}{\langle k^2\rangle}\frac{\mu}{\rho} \sim 1/\langle k^2\rangle$ for the case of without condensation [14]. The result of Ref. [14] is a specific case of our model with $\delta=1$.

Our results may be useful in controlling the epidemic spreading. Consider the fact that people are moving for some purposes and accumulate at the public places to a certain degree at sometimes and disperse at another time, which corresponds to different degrees of condensation with varying δ from zero to unity. Reducing epidemic spreading means keeping δ around one. Therefore, our results provide a theoretical evidence for reducing the epidemic spreading by preventing people to accumulate at the public places.

In conclusion, through both theoretical analysis and numerical simulations, we show that the dynamical condensation favors the spread of the infectious disease. Our study is based on a reaction-diffusion process where the reaction is

modeled by a SIS dynamics inside the nodes of the metapopulation network, and the diffusion is defined by a mobility rate out of a given node that depends on the population size of that node. We find that the infected fraction is a constant for the nodes with $k < k_c$ but close to unity for the hub nodes. The threshold β_c is a very small constant for $\delta < \delta_c$ and then gradually increase with δ for $\delta > \delta_c$. Our results are based on the condensation in SF networks but the benefit of condensation on epidemic spreading is not limited to the SF networks but also works for other networks, provided that there is condensation there.

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