

Collective synchronization induced by epidemic dynamics on complex networks with communities

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Much recent empirical evidence shows that *community structure* is ubiquitous in the real-world networks. In this paper we propose a growth model to create scale-free networks with the tunable strength (noted by Q) of community structure and investigate the influence of community strength upon the collective synchronization induced by Susceptible-Infected-Recovery-Susceptible (SIRS) epidemiological process. Global and local synchronizability of the system is studied by means of an order parameter and the relevant finite-size scaling analysis is provided. The numerical results show that a phase transition occurs at $Q_c \approx 0.835$ from global synchronization to desynchronization and the local synchronization is weakened in a range of intermediately large Q . Moreover, we study the impact of mean degree $\langle k \rangle$ upon synchronization on scale-free networks.

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I. INTRODUCTION

The study of networked systems, including technological, social, and biological networks of various kinds, has attracted much attention in the physics community [1–4]. How the properties of networks, such as the lengths of the shortest paths between vertices, degree distribution, clustering coefficient, degree-degree correlation, and so on, affect dynamical processes taking place upon the networks [5–10] has been one of the most important subjects of the body of this work. Recently, it has been determined that many real-world networks show *community structure* [11,12], i.e., groups of vertices that have a high density of edges within them, but a lower density of edges between groups. However, there are few works about the influences of various degree of community structure upon dynamics.

In this paper we intend to fill this gap by investigating synchronization behavior induced by the SIRS epidemiological dynamics [13,14] on the scale-free networks with various strength (noted by Q) of community structure. In Ref. [15], the authors have studied the SIRS on small-world networks and found that when p , which characterizes the degree of disorder of the network, reaches an intermediately large value p_c , synchronization of the system emerges. Comparatively, we focus on global and local (inside each community) dynamics, and discover that no synchronization comes forth when the network possesses a strong enough community structure, i.e., the communities are connected by a few edges among them. Moreover, the vertices inside each community behave with weaker synchronization when Q is in a range of intermediately large values.

II. Network Model

To generally characterize the community structure of scale-free networks, we propose a growth model to create a

network with a tunable parameter denoting the strength of community structure. Inspired by two ingredients of the Barabasi-Albert model (BA for short), i.e., growth and preferential attachment [16], the rules of our model are as follows: Starting with c communities, noted by $U_1, U_2, \dots, U_{c-1}, U_c$, and each community with a small number (m_0) of vertices, and at every time step, we add into each community a new vertex with $m (< m_0)$ edges that link the new vertex to n different vertices in this community and $m-n$ different vertices in other $c-1$ communities already existing in the system. The initial $m_0 \times c$ vertices link to each other to keep the connectivity of the network. The values of m and n are not necessary integers (take m , for example, the fractional part of m denotes the probability to link $m'+1$ different vertices, where m' is the integral part of m). When adding a new vertex into community U_l , first choose n different vertices in community U_l according to “preferential attachment,” which means the probability Π that the new vertex will connect to vertex i ($i \in U_l$) depend on the degree k_i of vertex i , such that $\Pi(k_i) = k_i / \sum_{j \in U_l} k_j$. Then for each one of the other $m-n$ edges of the new vertex, choose a community $U_h (\neq U_l)$ randomly and connect the new vertex to one vertex in U_h following the preferential attachment mechanism referred to above.

The scaling behavior of the degree distribution can be calculated by using several approaches [17–19]. In our model, the degree distributions $p(k)$ of vertices of the global network, as well as the local vertices (inside each community), are power law with exponent 3.0, i.e., $p(k) \propto k^{-3.0}$ (see Fig. 1). The analytic procedure is simple and not shown here.

As proposed by Newman and Girvan [20] and modified by Kashtan and Alon [21], the strength of community structure can be quantified by

$$Q = \sum_1^c \left[\frac{l_s}{L} - \left(\frac{d_s}{2L} \right)^2 \right], \quad (1)$$

where c is the number of communities, L is the number of edges in the network, l_s is the number of edges between

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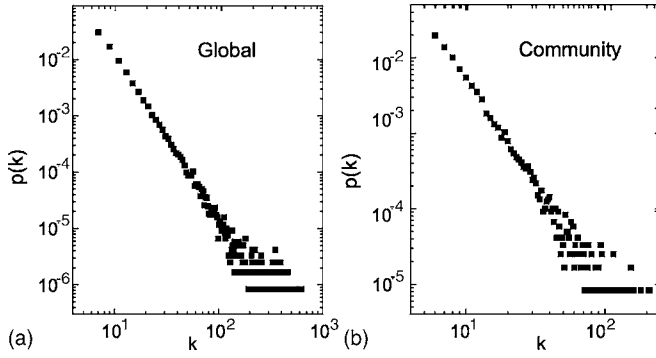


FIG. 1. The global (left) and local (right) degree distribution of the network with $N=10^5$, $c=10$, $m=4.0$, and $n=3.0$, where $Q=0.65$. It is worthwhile to point out that, for different values of Q , the distributions do not change.

nodes in community U_s , and d_s is the sum of the degrees of the nodes in community U_s . Roughly speaking, Q is the ratio of the number of edges intra-community to the total number of the edges. Obviously, if the network is divided into some communities more clearly, i.e., there are fewer edges among different communities, the value of Q is larger. In our model, for large N (the number of all vertices), $L=mN$, $l_s = \frac{nN}{c}$, and $d_s = 2n + (m-n) + (m-n)(c-1)/(c-1) = \frac{2mN}{c}$. Substituting these results into Eq. (1), we obtain

$$Q = \frac{n}{m} - \frac{1}{c}. \quad (2)$$

Thus, for fixed m and c , we modulate the value of n to obtain the networks with various community strength Q .

III. EPIDEMIC MODEL

We analyze SIRS epidemic model and aim to point out the role of community structure on the temporal dynamics of the epidemic spreading. The disease has three stages: susceptible (S), infected (I), and refractory (R). A vertex of the networked population is described by a single dynamical variable adopting one of these three values. Susceptible elements can pass to the infected state through contagion by an infected one. Infected elements pass to the refractory state after an infection time T_I . Refractory elements return to the susceptible state after a recovery time T_R . The contagion is possible only during the S phase, and only by an I element. During the R phase, the elements are immune and do not infect. The system evolves with discrete time steps. Each vertex in the network is characterized by a time counter $\tau_i(t)=0, 1, \dots, T_I+T_R \equiv T$, describing its phase in the cycle of the disease. The epidemiological state π_i (S , I , or R) of the vertex depends on the phase in the following way:

$$\begin{aligned} \pi_i(t) &= S & \text{if } \tau_i(t) &= 0, \\ \pi_i(t) &= I & \text{if } \tau_i(t) \in [1, T_I], \\ \pi_i(t) &= R & \text{if } \tau_i(t) \in [T_I+1, T]. \end{aligned} \quad (3)$$

The state of a vertex in the next step depends on its current phase in the cycle, and the state of its neighbors in the

network. A susceptible vertex stays as such, at $\tau=0$, until it becomes infected. Once infected, it goes (deterministically) over a cycle that lasts T time steps. During the first T_I time steps, it is infected and can potentially transmit the disease to a susceptible neighbor. During the last T_R time steps of the cycle, it remains in state R , immune and not contagious. After the cycle is complete, it returns to the susceptible state. As mentioned in Ref. [15], if vertex i is susceptible and it has k_i neighbors, of which k_{inf} are infected, then i will become infected with probability k_{inf}/k_i .

IV. RESULTS AND ANALYSIS

Specifically we study the behavior of the infected sites with respect to Q . A typical realization starts with the generation of the network characterized by Q and the initialization of the states of the vertices. The initial fraction of infected vertices $n_{inf}(0)=0.1$ and the rest susceptible, was used in all the simulations here.

After a transient period, a stationary state is achieved. We find that the pronounced fluctuations of the fraction of infected vertices is a function of time. Figure 2 shows three time series displaying the fraction of infected vertices in the network with varying community strength Q . When $Q=0.46$ [see Fig. 2(a₁)], the network has a weak strength of community structure. It is similar to the real-world networks where the community strength Q falls in the range from about 0.3 to 0.7 [20]. In such a condition, the fraction of infected vertices exhibits large amplitude oscillations. For strong community structure, such as $Q=0.81, 0.935$ [see Figs. 2(b₁ and c₁), respectively], the time series have regular periods, but the amplitudes are small and disordered. In addition, we study the local dynamics that is the epidemic process inside each community. Figures 2(a₂), 2(b₂), and 2(c₂) show the time evolution of the fraction of infected vertices in a community, for $Q=0.46, 0.81$, and 0.935 , respectively. The amplitudes are almost the same. Since the amplitude is related to the synchronization of the system, we will give a measure below and make it clearer. Figures 2(d₁) and 2(d₂) show the clear periodic oscillations of the fraction of local and global infected vertices vs the scaled time t/T while $Q=0.46$, respectively. One can see that the period T_0 is larger than the natural period T of the infection cycle, which is different from the result on small-world networks presented in Ref. [22]. Moreover, we have done the Fourier power analysis for different Q and find that there is a sharp peak in the frequency $1/T_0$ (not shown here), which reveals that the series have regular temporal periods although the amplitudes are variable. As the difference between T_0 and T is the time staying at the state S and T is of the same value for all vertices, we could analyze the reason of regular T_0 by estimating the probability $P_i(t)$ of a vertex i changing state from S to I at time t . We obtain here $p_i(t)$ by using the mean-field estimation as the following: $p_i(t) = k_{inf}/k_i \propto n_{inf}(t)$. That result implies that all the vertices update their states at almost the same time which induces the regular temporal cycles. Besides the parameter-free infection mechanism, there may be other reasonable choices. For example, if the susceptible had a probability λ of contagion with each infected neighbor,

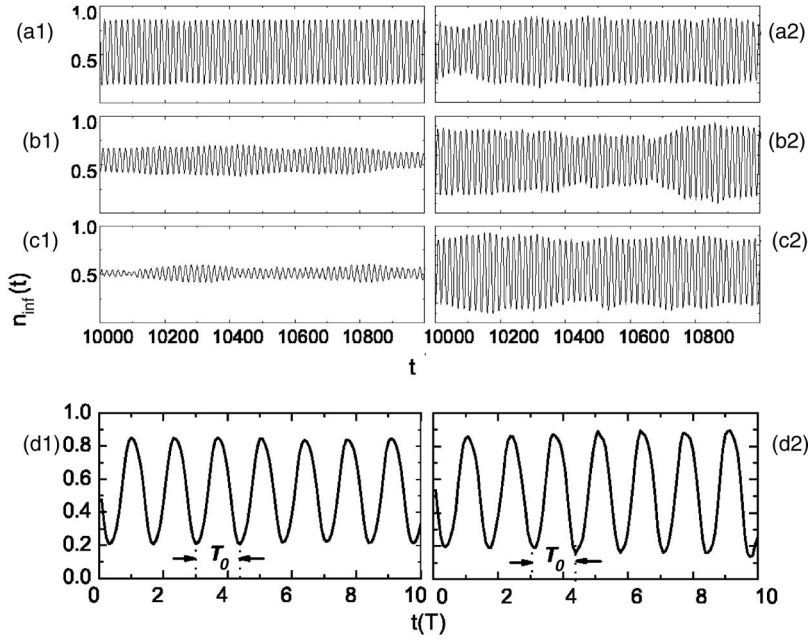


FIG. 2. The time series of the fraction of infected vertices. The systems have $N=10^4$, $c=25$, and $m=4.0$, and the infection cycle with $T_I=8$ and $T_R=5$. The left three figures (a₁), (b₁) and (c₁) showed the global fluctuations of $n_{inf}(t)$ on the network with $Q=0.46$, 0.81 , and 0.935 , respectively. The right three figures show the local fluctuations correspondingly. It is obvious that the global and local fluctuations are very different. The detailed analysis is presented in the text. The two bottom figures show the clear global (d₁) and local (d₂) periodic oscillations on the network with weak community structure ($Q=0.46$). The time steps have been scaled by the natural period T of the infection cycle. T_0 is the period of the oscillations. It is manifest that $T_0 > T$, which is different from the result $T_0 = T$ presented for small-world (SW) networks in Ref. [22].

then the probability of infection is $[1 - (1 - \lambda)^{k_{inf}}]$. For small λ , we have $p_i(t) = [1 - (1 - \lambda)^{k_{inf}}] \approx \lambda k_{inf} \propto n_{inf}(t)$. Obviously, that does not affect the qualitative results here.

To quantify the amplitudes of the oscillation series, we define the relevant order parameter

$$\sigma(t) = \left| \frac{1}{N} \sum_{j=1}^N e^{i\phi_j(t)} \right|, \quad (4)$$

where $\phi_j = 2\pi(\tau_j - 1)/T$ is a geometrical phase corresponding to τ_j . The states $\tau=0$ have been left out of the sum in Eq. (4). We obtain the synchronization order parameter σ by averaging over 10^4 time steps after the transient to a stationary state and subsequently by averaging over 400 different realizations of the system. Here the synchronization is a measure of the collective order. If at any time t all the vertices are almost

at the same state, i.e., τ_i is equal to the same value for all i , the system is synchronous. While the vertices are at different states equally, the system is not synchronous. Obviously, when the system is not synchronized, the phases are widely spread in the cycle and the complex numbers $e^{i\phi}$ are correspondingly spread in the unit circle which leads to a low value of σ . In contrast, when a significant part of the vertices are synchronized in the cycle, σ is large. The full synchronization, i.e., $\sigma=1$, will be achieved only when all the vertices enter the same state simultaneously. For the local synchronization, we calculate the above order parameter over the vertices in one community.

Precise calculation of the critical community strength Q_c separating synchronized and desynchronized states requires considering the finite-size effect. In the thermodynamic limit, the order parameter displays the critical behavior $\sigma \sim (Q - Q_c)^\beta$, with the critical exponent β . While in a finite system

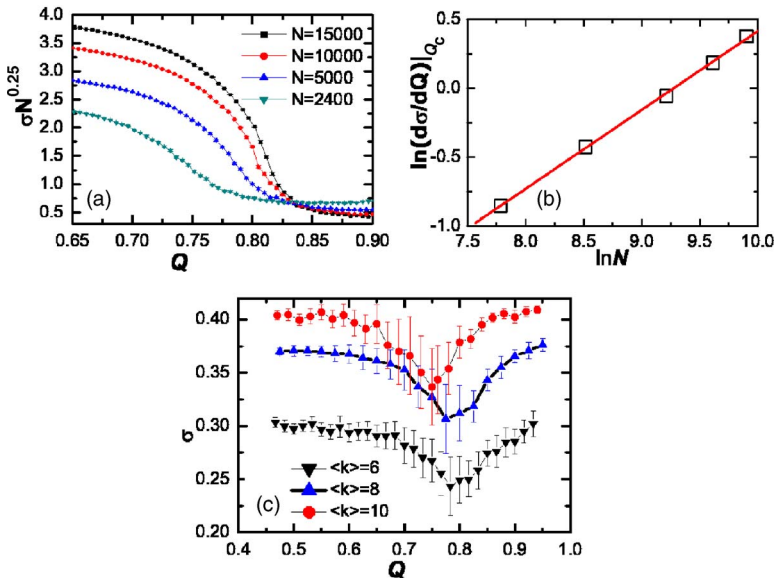


FIG. 3. (Color online) (a) The order parameter for global synchronization plotted as $\sigma N^{\beta/\bar{\nu}}$ with $\beta/\bar{\nu}=0.25$ vs Q for different network size N with fixed $Nc=200$, where Nc is the number of vertices in each community. There is given a unique crossing point at $Q_c=0.83(5)$. From (b) we obtained $(1-\beta)/\bar{\nu}=0.57(2)$. These yield $\beta \approx 0.30$ and $\bar{\nu} \approx 1.22$. (c) The order parameter for local synchronization vs Q for different network mean degree $\langle k \rangle = 6, 8, 10$ (from bottom to top).

with size much larger than the additional length scale, the critical scaling form is

$$\sigma = N^{-\beta/\bar{\nu}} F[(Q - Q_c)N^{1/\bar{\nu}}], \quad (5)$$

where the exponent ν describes the divergence of correlation volume ξ_v at Q_c , $\xi_v \sim |Q - Q_c|^{-\bar{\nu}}$. Since at $Q = Q_c$ the function F in Eq. (5) has a value independent of N , plotting $\sigma N^{\beta/\bar{\nu}}$ vs Q for various sizes, one can obtain the value of $\beta/\bar{\nu}$ that gives a unique crossing point at Q_c . One then uses

$$\ln \left(\frac{d\sigma}{dQ} \right)_{Q_c} = \frac{1 - \beta}{\bar{\nu}} \ln N + \text{const} \quad (6)$$

in order to determine the value of $(1 - \beta)/\bar{\nu}$. Then the exponent β and $\bar{\nu}$ can be figured out. Figure 3(a) displays the determination of Q_c for the global synchronization using the finite-scale form Eq. (5). Varying the value of $\beta/\bar{\nu}$ we find that $\beta/\bar{\nu} \approx 0.25$ gives a well-defined crossing point at $Q_c \approx 0.835$. In Fig. 3(b), the least-square fit to Eq. (6) gives $(1 - \beta)/\bar{\nu} \approx 0.572$. These yield $\beta \approx 0.30$ and $\bar{\nu} \approx 1.22$. Moreover, as shown in Fig. 3(c), for different mean degree $\langle k \rangle$, the local synchronization parameter falls into the pit around a value $Q \approx 0.75$. This implicates that, when the communities are almost unattached (i.e., for very large Q) the local dynamic lies on the inner structure of community independently, while the communities couple each other strongly (for small Q), the local dynamic is almost the same as the global one, and in the midst the local synchronization is the weakest.

Furthermore, we have studied the impact of the mean degree $\langle k \rangle$ of scale-free networks upon the synchronization. We start the simulation with a generation of scale-free networks with $Q = 0$, that is the BA model or our model with $n/m = 1/c$, and the initial fraction of infected vertices $n_{\text{inf}}(0) = 0.1$. We let the number of vertices m that a new added vertex will connect be real number, as referred in Sec. II. Obviously, $\langle k \rangle = 2m$. Figure 4(a) shows the order parameter σ vs m for different period T of the infection cycle. For a fixed period T , a transition in the synchronization can be observed as m increases. Moreover, the larger the period T , the less the critical value of m_c at which the transition occurs. We set $T = 10$ to analyze the critical scaling by using standard finite-size analysis mentioned above. Figure 4(b) shows when $\beta/\bar{\nu} = 0.16$ the curves with different sizes N give a unique crossing point at $m_c \approx 2.8$ (where $\langle k \rangle_c \approx 5.6$). In Fig. 4(c), the fit gives $(1 - \beta)/\bar{\nu} \approx 0.35$. Hence $\beta \approx 0.31$ and $\bar{\nu} \approx 1.95$.

V. CONCLUSION

To summarize, we have investigated the influence of the strength of community structure (Q) on global and local synchronization induced by the SIRS epidemic dynamics. The

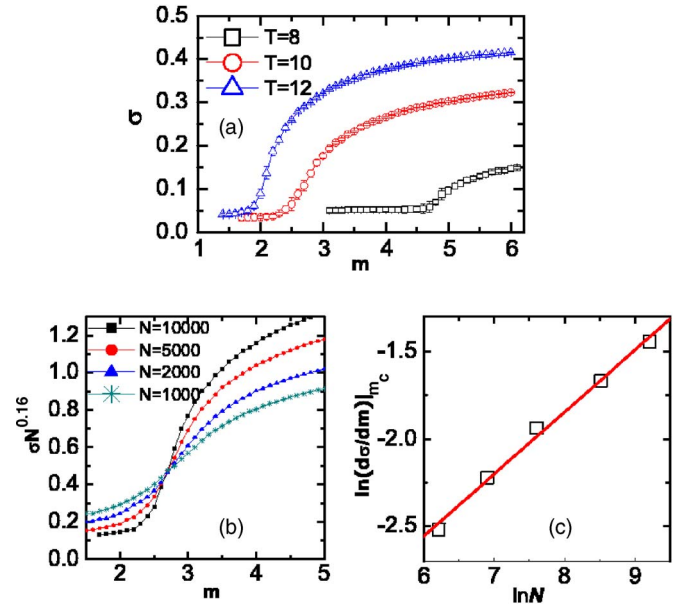


FIG. 4. (Color online) (a) The order parameter vs m for different natural period $T=8, 10, 12$, where $m = \langle k \rangle / 2$. (b) The order parameter σ for natural period $T=10$ plotted as $\sigma N^{\beta/\bar{\nu}}$ with $\beta/\bar{\nu} = 0.16$ vs m for different network size N . There is given a unique crossing point at $m_c \approx 2.80$. From (c) we obtained $(1 - \beta)/\bar{\nu} = 0.35(0)$. These yield $\beta \approx 0.31$ and $\bar{\nu} \approx 1.95$.

numerical results have shown that small Q induces better global synchronization and a phase transition occurs at $Q_c \approx 0.835$ estimated by using finite-size analysis, while for the local synchronization there exists a minimal value of order parameter σ around $Q \approx 0.75$. This result is in accordance with Ref. [23] in which a modified simulated annealing algorithm is applied to optimize the synchronizability and well-defined communities do not exit in the emerging networks. That implies the networks with small Q are of strong synchronizability. It is also worth mentioning that, as in the synchronization process, well-defined communities of nodes emerge in different time scales, Arenas *et al.* have used the synchronization to reveal the community structure [24].

Moreover, we have studied the synchronization order parameter vs $\langle k \rangle$ on scale-free networks with $Q = 0$. The simulation results demonstrate that, for a fixed period T , a transition in the synchronization can be observed as $\langle k \rangle$ increases. The larger period T corresponds to a smaller critical value of transition point $\langle k \rangle_c$.

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