Network-induced nonequilibrium phase transition in the "game of Life"

Sheng-You Huang, Xian-Wu Zou,* Zhi-Jie Tan, and Zhun-Zhi Jin
Department of Physics, Wuhan University, Wuhan 430072, People's Republic of China
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A cellular automation model of the ''game of Life'' on a two-dimensional small-world network is presented in order to count in long-range interactions among living individuals in social or biological systems. The density of the life and its fluctuation are calculated, respectively. The present model exhibits a nonequilibrium phase transition from an ''inactive-sparse'' state to an ''active-dense'' one at a certain intermediate value of the network disorder. Employing finite-size scaling analysis, we estimate the location of the critical point with $p_c(\infty) \simeq 0.3685$. The transition is of the ''second-order'' type with power-law diverging length. We obtain the critical exponents $1/\nu \simeq 1.70$, $\beta \simeq 0.50$, and $\beta/\nu \simeq 0.85$. The calculated results indicate that the present model may belong to the universality class of directed percolation.

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I. INTRODUCTION

For many years, the cellular automation has been extensively studied because of its relevant application in many social, biological, and physical processes [1–4]. Conway's ''game of Life'' (GL) is probably the best known cellular automation, which has been suggested to mimic aspects of complexity in nature [5–10]. The original GL is a society of cells (on a two-dimension lattice), in which the state (''dead'' or ''alive'') of each cell depends on deterministic local rules [5]. The evolution is determined by the number of living cells among its eight nearest and next-nearest neighbors: (i) A live cell that has four or more live neighbors will die in the next time step (decrease by overcrowding). Also a live cell will die in the next time step if it has one or zero live neighbor (decrease by isolation). However, if the live cell has two or three live neighbors it will remain alive. (ii) At a dead site, a new cell will only be born in the next time step if it has exactly three live neighbors. Starting from random initial conditions, ''Life'' will evolve through complex patterns eventually settling down in a stationary state. In spite of its simple algorithm, the GL simulates the dynamic evolution of a society of living individuals, including processes such as growth, death, survival, self-propagation, and competition.

All the work about Life (deterministic or stochastic) takes into account only local interactions, i.e., the state of each cell depends on its 8 neighbors [5–10]. However, real populations rarely fall into this simple category. For example, each individual is not only dependent on its neighbors, but also may depend on a distant individual because of the developed highways or convenient information communications in real society. Recently introduced by Watts and Strogatz, the small-world network (SWN) [11] attempts to translate the complex topology of social interactions into an abstract model. Small worlds have been found to play an important role in the study of the influence of the network structure upon the dynamics of many complex processes in nature, such as disease spreading, formation of public opinion, distribution of wealth, biological evolution, etc. [12–22].

The SWN model consists of a regular lattice, typically a one-dimensional lattice with periodic boundary conditions, although lattices of two or more dimensions have been studied as well, with each bond in the original lattice rewired at random with probability p. The model exhibits unusual connection properties. On one hand it shows high network clustering, like regular graph. On the other hand it shows a very small average shortest path through the network between any two sites. It has been shown that geometrical properties, as well as certain statistical mechanics properties, show a first-order transition at $p=0$ in the limit of large systems, $N \to \infty$ [23,24]. That is, any finite value of the disorder induces the small-world behavior [14,23]. In this paper we study the cellular automation of the game of Life on the small-world network in order to investigate the effect of the population structure on the evolution of Life. It shows that there exists a nonequilibrium phase transition in the behavior of ''Life'' dynamics at a finite value of the network disorder.

II. MODEL AND METHOD

We investigate the effect of the topology of populations on the game of Life. The interactions between the individuals of the population are described by a two-dimensional small-world network, and the link between two sites represents that there exists interaction between two individuals which are located at these two sites, respectively. To build a small-world network, we start with a regular square lattice of size $L \times L$ with periodic boundary conditions. Each site in the lattice is linked to its 8 nearest and next-nearest neighbors in order to incorporate the local rule of the original GL. Then each link in the original lattice is rewired at random, with probability p, to another site of the system. With probability $(1-p)$ the original link is preserved. Self-connections and multiple connections are prohibited. With this procedure, we have a regular lattice at $p=0$ and progressively random graph for $p>0$, with the average coordination number 8.

Based the built small-world network, we describe how the Life evolves. Each site in the lattice may be in two states,

*Author to whom any correspondence should be addressed. Email address: xwzou@whu.edu.cn

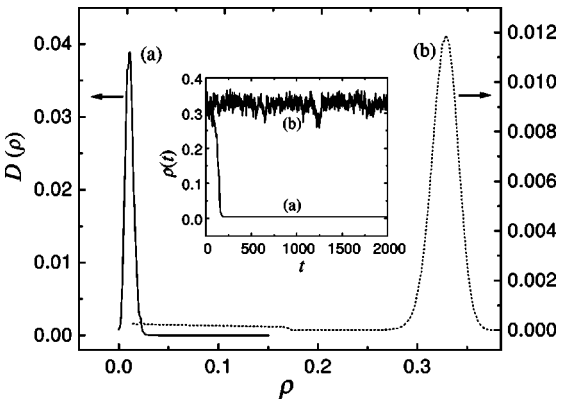

FIG. 1. The stationary density distribution of life $D(\rho)$ at the network disorders $p=0.37$(a) and $p=0.44$(b) for the lattice size $L=50$. Inset: the density of life $\rho(t)$ as a function of time t at two corresponding p values.

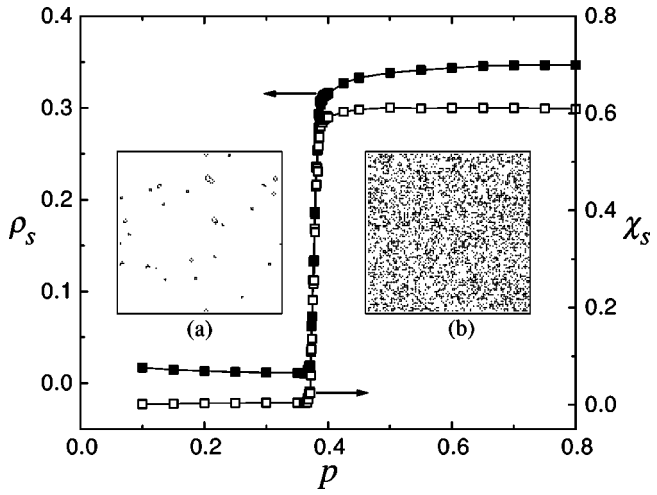

FIG. 2. The stationary density of life ρ_s (solid square) and its normalized fluctuation χ_s (open square) as a function of the network disorder p for the lattice size $L=100$. Insets: the patterns of living individuals at the network disorders $p=0.35$ (a) and $p=0.45$ (b).

representing the presence or absence of a live individual. The fate of each state depends on its neighbors. In the present model, we define that two sites are neighboring if there exists a link between them. So the rule of the evolution for the Life is as follows. A dead individual will only come to life if it has exactly three living neighbors. A living individual will stay alive if it has two or three living neighbors, otherwise it will die. We start at time $t=0$ with a random distribution of living sites with density ρ_0. As in standard cellular automation procedure, at each time step all sites are updated simultaneously according to the rule described above, until the GL reaches a stationary state. It should be noted that the stationary state of the original GL model depends on the initial conditions [25,26]. The average stationary density of living individuals ρ_s approximates to a constant for initial densities in the range $0.15 \leqslant \rho_0 \leqslant 0.75$ [25,26]. We focus on the effect of the network structure on the evolution of living individuals. Without loss of generality, the initial density is chosen to be $\rho_0=0.35$, around which the mean-field theory predicts a maximum stationary density of living individuals [26].

III. RESULTS AND DISCUSSION

We have performed extensive numerical simulations of the described model on the small-world network with sizes ranging from $L=20$ to $L=500$ and different rewiring probabilities $p \in [0,1]$. For every system with size L, the calculated results are averaged over both n different realizations of the network and 10 independent runs for each network realization, in such a way that $n \times L^2 \approx 2.5 \times 10^5$.

Figure 1 shows the density of life $\rho(t)$ as a function of time t at the network disorder $p=0.37$ and 0.44 for the system with size $L=50$, respectively. As we can see, at $p=0.37$ the density of life $\rho(t)$ rapidly drops toward a very small value. However, at $p=0.44$ the density $\rho(t)$ decreases a little and then approaches a large fluctuating value. In Fig. 1, we also plot the stationary density distribution $D(\rho)$ for $p=0.37$ and 0.44, respectively. From the density distribution $D(\rho)$ a peak is easily identified. The positions of the peaks show a very large difference corresponding to two different p values (see Fig. 1). The drastic change of the density $\rho(t)$ and distribution $D(\rho)$ within such a small range of p indicates that there may exist a phase transition of Life at a certain intermediate value of p. From Fig. 1, we also find that the stationary state of Life has been reached after about 2000 time steps. Therefore, all the results are sampled over the time interval ranging from 2000 to 2100 time steps for each independent run in the simulations.

In addition to the density of life $\rho(t)$, we also calculate the fluctuation of the density $\chi(t)$ in order to characterize the activity of life. The parameter $\chi(t)$ is defined as the square value of the difference between the densities corresponding to two sequential time steps, i.e.,

$$\chi(t) \equiv [\rho(t) - \rho(t-1)]^2. \tag{1}$$

Figure 2 shows the average stationary density of the life $\rho_s = \langle \rho(t \to \infty) \rangle$ and its normalized fluctuation $\chi_s = L \langle \chi(t \to \infty) \rangle$ as a function of the network disorder p for the system with size $L=100$, respectively. It can be seen from Fig. 2 that there exists a sharp jump for both ρ_s and χ_s in the vicinity of a critical value of $p_c \simeq 0.375$. When $p<0.375$, the present model is similar to the GL in a regular lattice [25,26] and the density of life ρ_s have a very small value of about 0.02. When $p>0.375$, the present model presents the results of the GL in a random graph, and the density $\rho_s \simeq 0.347$, which is consistent with the mean-field theory [25,26]. Correspondingly, the pattern consisting of living individuals transits from a sparse state to a dense one at the critical value 0.375 (see the insets of Fig. 2). The results in Fig. 2 confirm that the present model exhibits a nonequilibrium phase transition from an inactive-sparse phase to an "active-dense" one at a intermediate p value. The fluctuation of the density χ_s, which transits from $\chi_s=0$ to $\chi_s>0$ at the critical value p_c, serves as the order parameter in the present model.

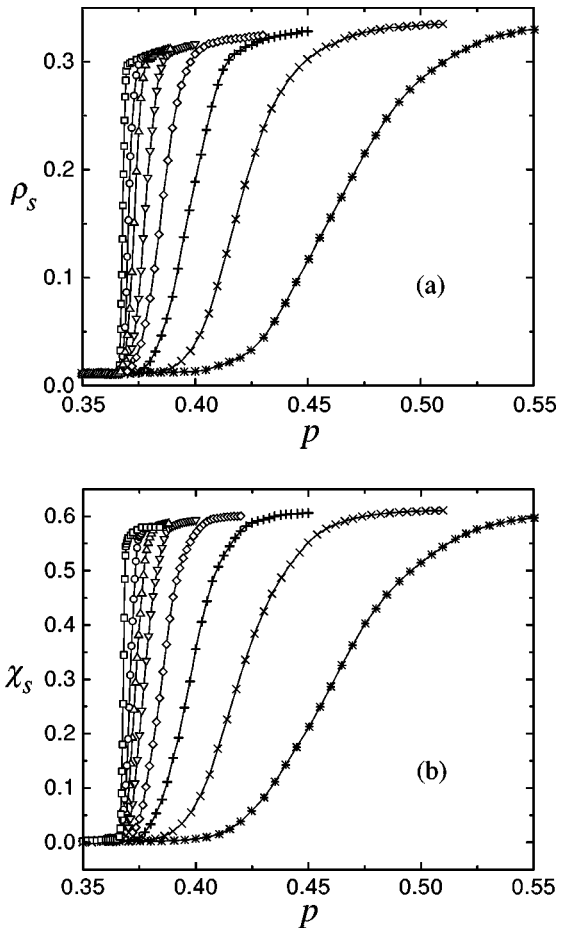

FIG. 3. (a) The stationary density of life ρ_s and (b) its normalized fluctuation χ_s as a function of the network disorder p for several systems with different sizes. From left to right the size $L=500$, 200, 140, 100, 70, 50, 35, and 25.

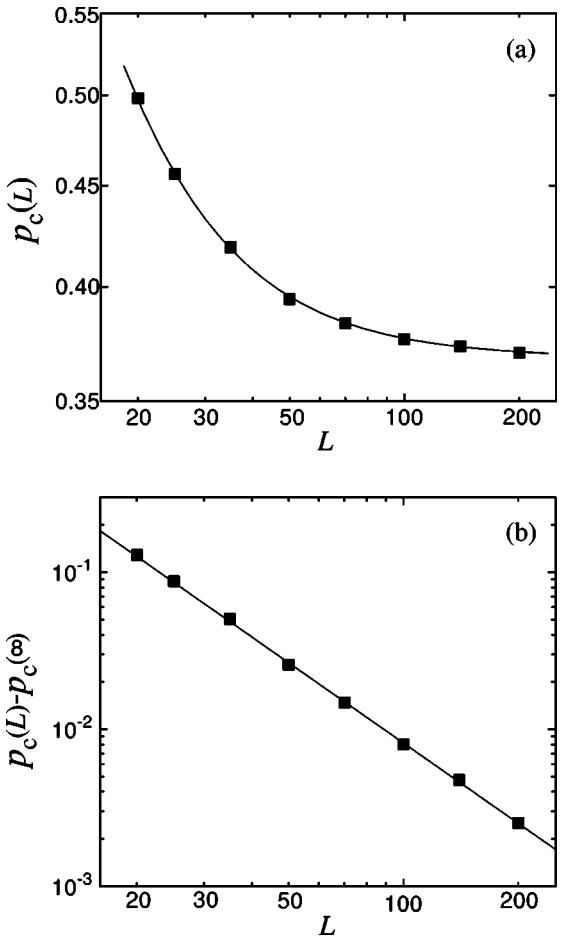

FIG. 4. (a) The critical network disorder $p_c(L)$ for finite-size systems as a function of the system size L on a log-log plot. The symbols are the simulation results and the line is guided to eye. (b) The deviation $p_c(L)-p_c(\infty)$ from the true critical value as a function of size L on a log-log plot, where $p_c(\infty)$ is chosen to be 0.3685. The symbols are the simulation results, and the line is the least-square fit to the data.

In the simulations, the systems with finite sizes are used. The determined critical probability p_c depends the size of the system. To obtain the true critical point $p_c(\infty)$, which corresponds the critical value for very large systems, we study the stationary behavior of Life for several systems with different sizes. Figures 3(a) and 3(b) plot the density of life ρ_s and its fluctuation χ_s as a function of the network disorder p for the systems with sizes from $L=25$ to $L=500$, respectively. From Fig. 3 we can see that there exists a transition at a certain finite p value for each system. In the present model, the fluctuation of the system is very strong (see Fig. 1). The smaller the system size, the stronger is the fluctuation of the density. For a system with small size, a large fluctuation in $\rho(t)$ may cause the system to go extinct, i.e., to enter the completely ''inactive-sparse'' state within the simulation time. Therefore, the transition is smoother for the smaller system. The location of the critical point $p_c(L)$ in a finite-size lattice also shows a deviation from the true critical value $p_c(\infty)$. From Fig. 3, we can estimate the critical values $p_c(L)$ for the systems with different sizes corresponding to the inflexions of the curves. The errors in determining $p_c(L)$ is due to locating the inflexions of the transition curves. Thus, one can reduce the errors of $p_c(L)$ by means of averaging the results over more independent runs to obtain smoother curves. The results of $p_c(L)$ are shown in Fig. 4(a) on a log-log plot. It can be seen from Fig. 4(a) that with the increase of the system size L the critical value $p_c(L)$ decreases toward a constant value, which corresponds to the true critical value $p_c(\infty)$ for the infinite-size system. According to the finite-size effects of the systems, the apparent critical point $p_c(L)$ and true critical point $p_c(\infty)$ are expected to scale with size L as [27]

$$p_c(L)-p_c(\infty)\sim L^{-1/\nu}, \tag{2}$$

where ν is the critical shift exponent. To obtain the values of the true critical point $p_c(\infty)$ and critical exponent ν, Fig. 4(b) shows the critical deviation $p_c(L)-p_c(\infty)$ as a function of the system size L on a log-log plot. When the true critical value is chosen to be $p_c(\infty)\simeq 0.3685$, we obtain the best power-law relation of the data [see Fig. 4(b)]. The excellent linear dependence in Fig. 4(b) indicates that within the uncertainties, the finite-size scaling relation Eq. (2) is reason-

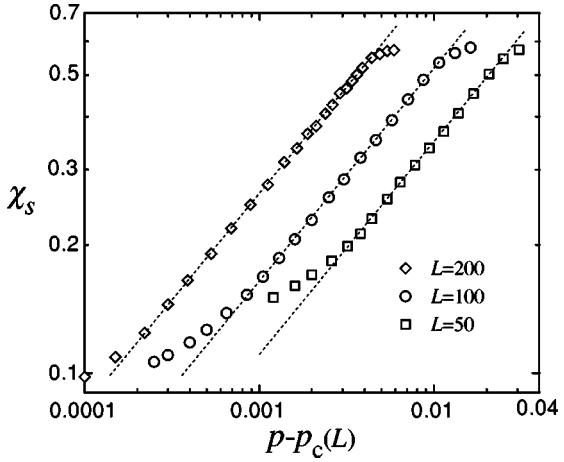

FIG. 5. The log-log plot of the fluctuation χ_s as a function of $[p-p_c(L)]$ for several system sizes. The slope of the line fitted to the data is associated with the critical exponent β.

able for describing the present simulation results. From Fig. 4(b) we obtain the critical exponent $1/\nu=1.70(5)$ by means of the least-square fit to the data.

Going a step further, we investigate the behavior of the systems in the vicinity of the critical point p_c. By analogy with Ref. [8], we assume that the present phase transition is a second-order continuous one. Thus, the order parameter is expected to have a power-law behavior near the critical point

$$\chi_s(p\to p_c^+)\propto(p-p_c)^\beta, \tag{3}$$

where β is the critical exponent of the order parameter. Note that Eq. (3) only holds true in the system size $L\to\infty$. However, the lattice model of SWN essentially determines that it is impossible to built a very large network, and also it is difficult to obtain accurate simulation results near the critical point because of the topology fluctuation in the network and also the life fluctuation in the evolution. Thus, we will focus on the systems with size $L\leqslant 200$ to save the computation time. For finite-size systems, we can determine β corresponding to the vanishing rate of the order parameter from the log-log plot of χ_s as a function of $[p-p_c(L)]$ [28]. In the simulations, we have used more network realizations, i.e., $n\times L^2\approx 5\times 10^5$, to obtain a accurate β value. These results are plotted in Fig. 5. From Fig. 5 we can see that the data show a good power-law dependence in a certain region of disorder values for different system sizes. The slope of the line fitted to the data can be associated with the critical exponent for which we obtain $\beta=0.50(8)$. The error in determining β is due to the uncertainties in the χ_s and the $p_c(L)$ values. Since the scaling plots in Fig. 5 depend sensitively on the choice of the critical disorder $p_c(L)$ and our method of determining their values is indirect, we give rather conservative estimate for the error of β.

With the critical exponents $1/\nu=1.70(5)$ and $\beta=0.50(8)$, we can obtain another critical exponent $\beta/\nu=0.85(13)$. For $(2+1)$-dimensional directed percolation, the values of critical exponents are $\beta=0.60$ and $\beta/\nu_\perp=0.82$ [29,30]. Thus, it can be concluded that the present model may belong to the universality class of directed percolation.

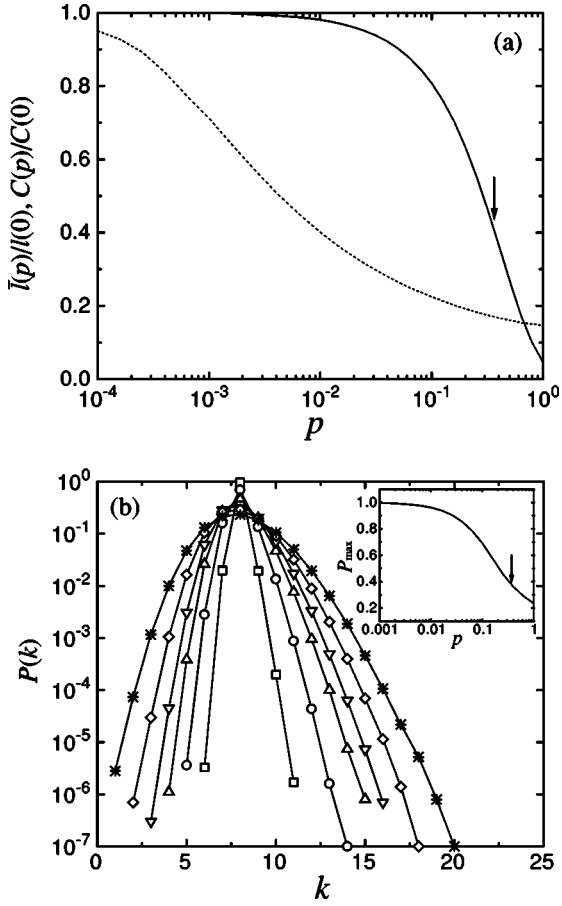

FIG. 6. (a) The normalized clustering coefficient C (solid line) and average shortest-path length $\bar{\ell}$ (dashed line) as a function of the disorder p for the small-world network with size $L=100$, and the arrow indicates the critical point $p_c\simeq 0.37$. (b) The degree distribution $P(k)$ for the small-world network with the disorder $p=0.01(\square)$, $0.1(\bigcirc)$, $0.251(\triangle)$, $0.398(\nabla)$, $0.631(\diamond)$, and $1.0(*)$. The data are sampled from 1000 independent network realizations. Inset: the peak value P_{max} of the degree distribution as a function of p, and the arrow indicates the critical point $p_c\simeq 0.37$.

As for the reason that the present critical transition takes place at a intermediate p value but not at $p=0$, it is not still very clear up to date. It is difficult to obtain a analytical expression to describe this phenomenon because of the strongly nonlinear effects in the systems. As mentioned before, a mean-field theory can be shown to predict the high density of Life, but this can be expected to describe only the small-world network at $p=1$, and it cannot explain the nature of the transition at the lower values of disorder. Here, we will present some conjectural explanations for the transition from our observation of the dynamical behavior of the system. As we know, there exists a typical length $L^*(p)\sim p^{-1/d}$ in the small-world network such that for the system size above which the network is indeed a small world and below which it behaves as a regular lattice [14]. For the present model, the critical transition occurs at the network

disorder of $p_c \simeq 0.37$. Obviously, the present system size L is far larger than $p_c^{-1/2}$, which indicates that the present transition cannot result from the finite-size effects. An explanation involving the average shortest-path length $\overline{\ell}(p)$ is also not reasonable, since $\overline{\ell}$ is known to behave critically with the disorder of $p=0$ [23,24], and we observe the critical transition at $p_c > 0$.

In addition to $\overline{\ell}$, the small-world network can be described by the clustering coefficient $C(p)$, which characterizes the closest environment of a site. At low p values, the networks are rather regular and highly clustered. As p approaches 1, C decreases. The crossover from high to low clusterization occurs at a higher p value, compared to that observed in the decay of $\overline{\ell}$ [see Fig. 6(a)]. Moreover, the change in the clustering coefficient $C(p)$ is accompanied by a corresponding one in the degree distribution $P(k)$, where $P(k)$ denotes the probability of the number of sites with k edges in the network [see Fig. 6(b)]. Therefore, it is expected that the present transition maybe result from the change of the clustering coefficient C and degree distribution $P(k)$ in the SWN. When p is small ($<p_c$), the network is high clustered as the regular lattice, and the degree distribution $P(k)$ is very narrow, centered on the average coordination number of $\langle k \rangle = 8$ (see Fig. 6). In this case, most of living individuals will die because of overcrowding or isolation, and only a small number of individuals survive as the form of gliders, blinks, ponder, etc. [25,26]. When p is large ($>p_c$), the clustering coefficient is small and the network have a wide degree distribution $P(k)$ with k ranging from $k \approx 1$ to $k \approx 20$ (see Fig. 6). In this situation, there exists a certain number of sites with long-range links, through which each site can be easily connected to each other in the network. On the one hand, the birth probability of new individuals will greatly increase because of the wide degree distribution, which can be comparable to the dead one of the living individuals. On the other hand, the birth of a new individual can rapidly affect the whole system through the long-range links. Therefore, the living individuals have a high fluctuating density as that in the random graph, as expected. When the clustering coefficient $C(p)$ crosses over from the higher value to a smaller one, it is expected that the transition occurs. From Fig. 6(a) we obtain the crossover point $p_{cr} \simeq 0.35$, which is consistent with the present critical value $p_c \simeq 0.37$. From Fig. 6(b) we can also find that as p approaches p_c, the range of the degree distribution begins to include the low degree of $k \approx 3$, and the peak value P_{max} of $P(k)$ also shows a corresponding transition.

IV. CONCLUSION

In summary, we have investigated the cellular automation ''game of Life'' on a two-dimensional small-world network by extensive numerical simulations. The results show that with the increase of the network disorder p, the present model exhibits a second-order phase transition at a intermediate disorder value p_c. When $p<p_c$, the stationary behaviors of systems are close to those of Life on a regular lattice with a very small static density of life. When $p>p_c$, the stationary behaviors of systems are consistent with those of Life on a random graph with a fluctuating high density of living individuals, and the present model presents the mean-field results of game of Life. The location of the critical point is precisely estimated with $p_c \simeq 0.3685$ by means of the finite-size scaling analysis. The critical exponents are also obtained, which are found to be consistent with those of directed percolation. The present method can also be applied to study the other cellular automation models. From a practical point of view, the present critical transition is a useful guide for building a network. Since the long-range connection usually costs more than the local one, it is advantageous to obtain the value of p_c in advance, above which the individuals in the system have a fluctuating high density, so that one can establish a high-quality network with least consumption of resources.

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