Branching process in a stochastic extremal model

S. S. Manna[*]

Max-Planck-Institute für Physik Komplexer Systeme, Nöthnitzer Str. 38, D-01187 Dresden, Germany
and Satyendra Nath Bose National Centre for Basic Sciences, Block-JD, Sector-III, Salt Lake, Kolkata 700098, India
(Received 16 April 2009; revised manuscript received 5 July 2009; published 28 August 2009)

We considered a stochastic version of the Bak-Sneppen model (SBSM) of ecological evolution where the number M of sites mutated in a mutation event is restricted to only two. Here the mutation zone consists of only one site and this site is randomly selected from the neighboring sites at every mutation event in an annealed fashion. The critical behavior of the SBSM is found to be the same as the BS model in dimensions $d=1$ and 2. However on the scale-free graphs the critical fitness value is nonzero even in the thermodynamic limit but the critical behavior is mean-field like. Finally $\langle M \rangle$ has been made even smaller than two by probabilistically updating the mutation zone, which also shows the original BS model behavior. We conjecture that a SBSM on any arbitrary graph with any small branching factor greater than unity will lead to a self-organized critical state.

In a seminal paper Bak and Sneppen (BS) introduced a self-organized critical (SOC) [1] model for the ecological evolution of interacting species, known as the Bak-Sneppen (BS) [2] model. In this model an entire species is represented by a single fitness variable. Using the spirit of Darwinian principle the minimally fit species is mutated. This however disturbs the stability of the ecological system. There are some other species, which are dependent on the minimally fit species for example as a part of the food web. These species are also mutated. The ecological evolution takes place in a series of such events.

The phenomenon of SOC is the spontaneous emergence of fluctuations of all length and time scales in a slowly driven system. This concept was first introduced to describe the formation of a sandpile of a fixed shape [1]. Later the idea of SOC has been applied to a large number of different physical systems [3]. A number of models have been introduced to describe SOC in different systems. In the Bak, Tang, and Wiesenfeld (BTW) model [1] the dynamics is described in terms of spreading of sand grains on a sandpile. Toppling of an unstable sand column distributes sand grains to all neighboring sites. This model is also known as the Abelian sandpile model since the stationary state is independent of the sequence of grain additions [4]. In a stochastic version of the sandpile model grains are distributed to randomly selected neighboring sites [5]. In the SOC models fluctuations are described in terms of avalanches of activities and their size distributions assume power-law decaying functions for large system sizes. The BS model is regarded as a simple but nontrivial SOC system.

The BS model is described as follows. The ecosystem consists of N species located at the sites $i=1, N$ of an one dimensional lattice. A fitness variable f_i is associated with every site. Initially uniformly distributed random numbers within the range $\{0, 1\}$ are assigned for the fitness values. The dynamical evolution of the ecosystem takes place in a series of mutation events. Each event consists of two steps:

(i) the "active" site i_o is searched out, which has the minimal fitness f_o. This site is mutated i.e., the value of f_o is replaced by a new random number. (ii) All sites of a fixed mutation zone in the local neighborhood are mutated as well. E.g., in $d=1$ the fitness values at two neighboring sites of i_o are also refreshed. These two steps complete a single mutation event. After that the active site is located at some other site where the next mutation event takes place and so on. The sequential time is measured by the number of mutation events. The system eventually reaches a steady state in which the associated statistical distributions assume their time independent stationary forms. The recurrent culling of the globally minimal fitness values leads to a step like form of the probability distribution $P(f)$ so that in the limit of $N \rightarrow \infty$: $P(f)=0$ for $f < f_c$ and $P(f)=\mathcal{C}$ a constant otherwise, where f_c is a critical fitness threshold [6].

In the steady state the fluctuations are described in terms of avalanches. A critical avalanche is a sequence of successive mutation events with $f_o < f_c$. The lifetime s of the avalanche is the total number of events in the avalanche. The distribution of the avalanche lifetimes has a power-law tail in the limit of $N \rightarrow \infty$: $D(s) \sim s^{-\tau}$. The BS model has been studied on hypercubic lattices, e.g., the values of f_c and τ are found to be 0.66702(8) and 1.073(3) [7] and 0.328855(4) and 1.245(10) [8,9] in $d=1$ and 2, respectively. The upper critical dimension has been argued to be 4 [10] and 8 [11] where τ assumes its mean-field value of 3/2.

It has been observed that increasing the size of the mutation zone modifies the critical fitness f_c but not the critical behavior [12]. It has also been shown that the BS model with isotropic and the anisotropic mutation zones have different critical behaviors [13]. Variants of the BS model with exponentially and power-law distributed random numbers have been studied [14,15]. BS model has also been studied on different heterogeneous graphs as well, e.g., on random graphs [16] and on an adoptive networks [17]. However, the critical fitness threshold is zero for BS model on infinitely large scale-free graphs [18]. In a scale-free graph the degree distribution $P(k)$ decays as a power law (with degree k being the number of edges meeting a vertex) as: $P(k) \sim k^{-\gamma}$ and the cutoff $k_{max} \sim N^x$, N being the size of the graph. The Barabási-

Albert (BA) network [19] is a well-known scale-free graph with $\gamma=3$ and $x=1/2$ [20]. For the BS model on BA network, the value of $f_c(N)$ decreases to zero in $N\rightarrow\infty$ limit as $1/\log(N)$ [18,21–23].

First, let us consider two limiting cases. Suppose the mutation zone has size zero so that only the active site is updated in every mutation event. Then the chance that the next f_o will be less than the present f_o arises due to refreshing this site only. Consequently f_o increases almost monotonically leading to $f_c=1$. The other limiting case is the N-clique graph where each vertex is connected to all other $N-1$ vertices in the graph [24]. The mutation zone consists of $N-1$ nodes and therefore in a mutation event fitness values of all N vertices are refreshed, as a result both f_o, $f_c\rightarrow0$ as $N\rightarrow\infty$. Therefore when the size of the mutation zone is in between 0 and $N-1$, there is a competition between the mechanisms of these two limiting processes and consequently f_c assumes a nontrivial value between {0, 1}.

Since a random graph has Possionian degree distribution, which decays very fast the threshold fitness f_c has a fixed value [16]. On the other hand in scale-free networks whenever a mutation event initiates at a hub vertex its large number $\sim N^x$ of neighbors are refreshed. Qualitatively this is a similar mechanism as the N-clique graph, but since $x<1$, the $f_c(N)\rightarrow0$ inverse logarithmically. Therefore if the size of the mutation zone is reduced, the number of refreshed sites is less; consequently the chance of creating new f_o smaller than the present minimal is less. As a result f_c goes up.

The dynamics in the Bak-Sneppen model is well known to be described by a branching process [8,25]. A branching process [26] is defined by a population where each individual in one generation produces randomly a number of offsprings in the next generation. The average number of offsprings is called the basic reproduction rate. Here in BS model we call the sites with fitness values $f<f_c$ as the critical sites. Every mutation event produces randomly a number of critical sites. If the total number of sites refreshed in a mutation event is M, which is the number of sites in the mutation zone plus one for the active site, then the average number of critical sites produced in a mutation event is $r_b=Mf_c$, which we call as the branching factor. It may be noted that all critical sites produced in a mutation event may not be fresh critical sites. An existing critical site may be refreshed again to a critical site with a different fitness value. It is known that a nontrivial branching process needs a value of the basic reproduction rate greater than unity. For BS model a similar condition may be that the branching factor should be greater than one. This implies that if one reduces the size of the mutation zone to only one site, it may still be possible to achieve a self-organized critical state only if $r_b=2f_c>1$. In the following we will present numerical evidence, which indicates that indeed this is likely to be true. In a particular anisotropic case the fixed one member mutation zone has already been studied in one dimension [12].

In this paper we study a stochastic Bak-Sneppen model (SBSM) with the simplest possible mutation zone. In addition to the usual procedure of randomly refreshing the fitness values the stochasticity is introduced in randomly selecting the sites of the mutation zone as well. The size (the number of sites) of the mutation zone is kept fixed at the minimal
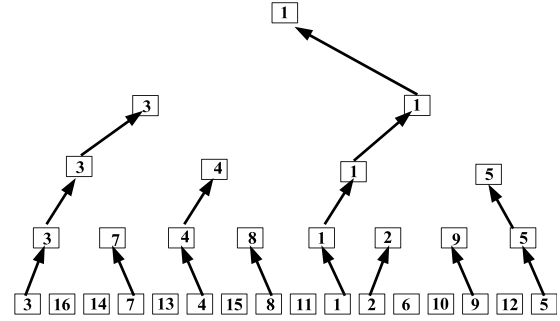

FIG. 1. Hierarchical organization of the data structure to search for the global minimum [7]. An $1d$ array of 16 sites has 16 random numbers. Successive pairs of sites form blocks in level 1. The smaller number of every block is forwarded to level 2 and a pointer is attached. Similar block pairs are also formed in level 2 and pointers are attached toward level 3 and so on. One moves opposite to the pointer directions starting from the top level to reach the minimal site at the lowest level.

value, i.e., unity and for different mutation events different mutation zones are randomly selected only from the nearest neighbors of the active site.

We follow the Grassberger algorithm for our study [7]. Searching for the minimal fitness using a brute-force algorithm needs to test the fitness values of all sites requiring CPU $\sim N$. Grassberger used a hierarchical organization of the data in block structure where CPU $\sim\log(N)$. Sites of an one dimensional lattice of $N=2^n$ sites are divided into $N/2$ block pairs such as $(1,2)$, $(3,4)$,… $(N-1,N)$. For each block, the smaller fitness value of two sites is stored in a site of another lattice of $N/2$ sites in a higher level and a pointer is assigned to this site. This procedure is repeated for the next higher level as well. Finally only one site in the $(n+1)$th level contains the global minimal fitness value (Fig. 1). To locate the minimal fitness site one moves opposite to the pointer directions starting from the top level. During the mutation of every site at the lowest level one needs to update the fitness values and the pointer directions up to the top level.

Indeed we observe that the simple SBSM preserves all characteristics of the original BS model. In $d=1$, the step form of $P(f)$ has been observed in all system sizes from $N=2^7$ to 2^{14}, increased by a factor of 2. The jump in $P(f)$ at $f_c(N)$ is rather smooth for the small N but becomes more and more sharper on increasing N [Fig. 2(a)]. The f value at the intersection of two successive $P(f)$ curves gradually shifts to higher values with N. The value of $f_c(\infty)$ is estimated as follows. In $N\rightarrow\infty$ limit the normalization of $P(f)$ gives $\mathcal{C}(\infty)=1/(1-f_c(\infty))$. The average fitness per site is then $\langle f(\infty)\rangle=\int_0^1 fP(f)df=(\mathcal{C}(\infty)/2)(1-f_c^2(\infty))=(1+f_c(\infty))/2$, which gives $f_c(\infty)=2\langle f(\infty)\rangle-1$. The whole fitness profile is sampled at the interval of every N mutation events and $f_c(N)$ values are extrapolated with $N^{-\kappa}$ with $\kappa=0.79\pm0.01$ to obtain $f_c(\infty)=0.7894(10)$ [Fig. 2(b)]. The most suitable value of κ is decided by trying different trial values of it and then selecting that particular value for which the fitting error is minimum. On a similar extrapolation of $\langle f(N)\rangle$ s we get $\langle f(\infty)\rangle=0.8947(10)$ implying that the branching factor $r_b=1.5788>1$.

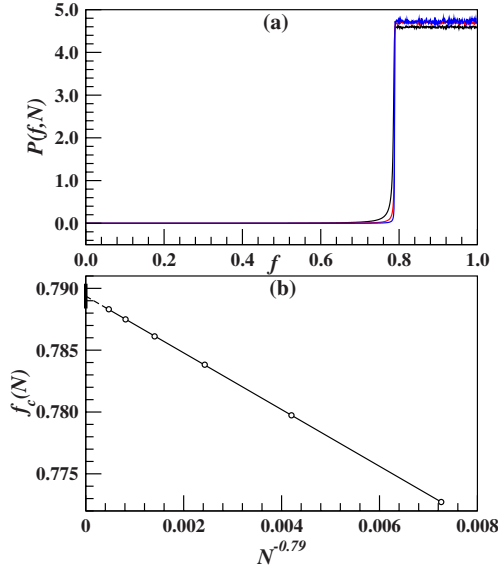The lifetime distribution exponent τ is estimated using the

FIG. 2. (Color online) (a) The distribution $P(f,N)$ of fitness values are shown for $1d$ SBSM for system sizes $N=2^{10}$ (black), 2^{12} (red), and 2^{14} (blue). The jump in the distribution becomes gradually sharper with increasing the system sizes. (b) The threshold fitness values for different system sizes are plotted with $N^{-0.79}$ and extrapolated to obtain the $f_c(\infty)=0.7894(10)$. The solid line is a least square fit of the data and the thick line indicates the extent of the error.

method of finite-size scaling analysis. Large number of mutation events are studied: $\approx 4 \times 10^{10}$ for $N=2^7$ to $\approx 110 \times 10^{10}$ for $N=2^{14}$. In Fig. 3(a) the binned probability $P(s,N)$ distribution data for $N=2^{10}$, 2^{12}, and 2^{14} are only plotted. A direct measurement of the slopes of these three curves gives
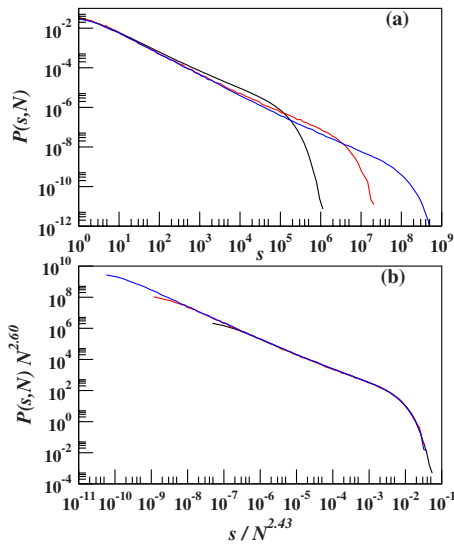

FIG. 3. (Color online) The avalanche lifetime distributions $P(s,N)$ for the $1d$ SBSM. In (a) we show the plots for the system sizes $N=2^{10}$ (black), 2^{12} (red), and 2^{14} (blue) (from left to right). (b) A finite-size scaling of this data shows an excellent data collapse for $\eta_1=2.60$ and $\zeta_1=2.43$ giving the value of the avalanche lifetime exponent to be $\tau_1 \approx 1.07$. The left end of each curve shifts to the left on increasing system size.
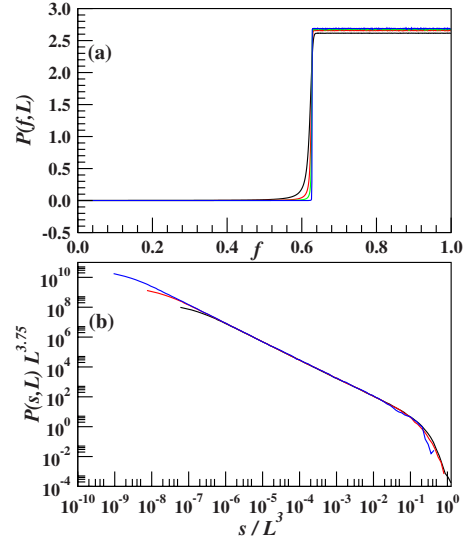

FIG. 4. (Color online) Data for the $2d$ square lattice of size L. (a) The fitness distributions $P(f,L)$ vs f for the system sizes $L=2^5$ (black), 2^6 (red), 2^7 (green), and 2^{10} (blue). The jump in the distribution becomes gradually sharper with increasing the system sizes. (b) The finite-size scaling of the avalanche size distribution for $L=2^8$ (black), 2^9 (red) and 2^{10} (blue) giving $\eta_2=3.75$, $\zeta_2=3.00$, and $\tau_2=1.25$. The left end of each curve shifts to the left on increasing system size.

0.94, 1.02 and 1.05 for the estimates of τ for the three system sizes, respectively. A finite-size scaling analysis has been done using the following scaling form:

$$P(s,N) \propto N^{-\eta} \mathcal{G}(s/N^{\zeta}), \tag{1}$$

where the scaling function $\mathcal{G}(x) \sim x^{-\tau}$ in the limit of $x \to 0$ and $\mathcal{G}(x)$ approaches zero very fast for $x \gg 1$. The exponents η and ζ fully characterize the scaling of $P(s,N)$ in this case. An immediate way to check the validity of this equation is to attempt a data collapse by plotting $P(s,N)N^{\eta}$ vs s/N^{ζ} with trial values of the scaling exponents. The values for obtaining the best data collapse are found to be $\eta_1=2.60$ and $\zeta_1=2.43$; here we have used the subscripts to denote the dimension of the system [Fig. 3(b)]. The lifetime exponent for $1d$ SBSM is therefore $\tau_1=\eta_1/\zeta_1 \approx 1.07(2)$. This exponent is very close to the value of τ_1 in the BS model [7].

Next, we have studied the SBSM on a two-dimensional square lattice of size $L \times L$ so that total number of sites $N=L^2$. The fitness distribution profiles have been shown in Fig. 4(a) for different system sizes. The critical fitness value f_c has been obtained as 0.628(1) on extrapolating the $f_c(L)$ values of $L=2^7$, 2^8, 2^9, and 2^{10} with $L^{-1.29}$ and $r_b=1.256>1$. Another finite-size scaling has been done in a similar way to obtain the scaling function exponents for the lifetime distribution as shown in Fig. 4(b). The values obtained for the best data collapse are $\eta_2=3.75$ and $\zeta_2=3.0$ yielding the value of $\tau_2=1.25(2)$. This exponent is also very close to the value of τ obtained for two-dimensional BS model [8,9].

The SBSM is also studied on the scale-free BA network. In this growing graph every new vertex comes up with m edges and gets connections to m distinct vertices of the ex-
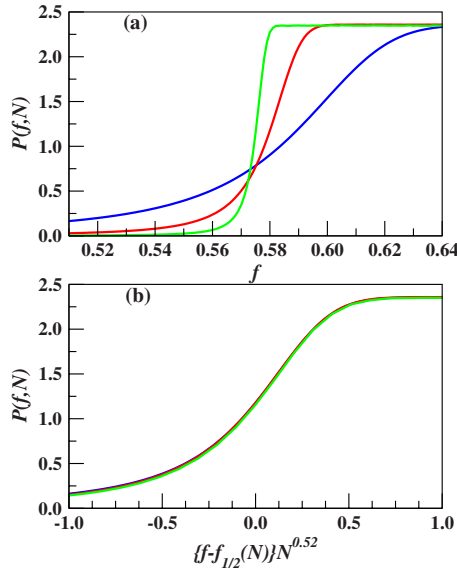
FIG. 5. (Color online) The distributions $P(f,N)$ of the fitness values in the steady state of SBSM on the Barabási-Albert scale-free graph. (a) Distribution plots for the graph sizes $N=2^7$ (blue), 2^{10} (red), and 2^{13} (green). The jump in the distribution becomes gradually sharper with increasing the system sizes. (b) A finite-size scaling of this data shows an excellent collapse.
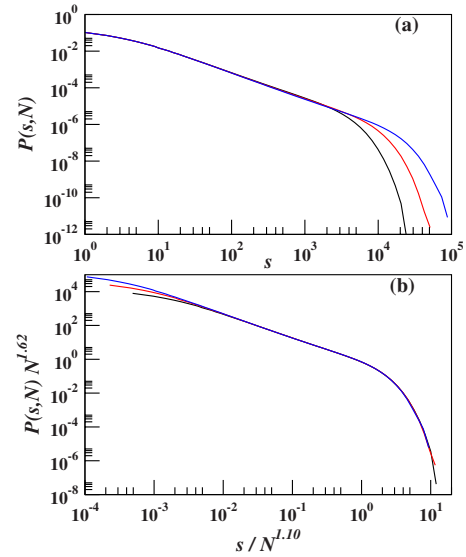

FIG. 6. (Color online) The avalanche lifetime distributions $P(s,N)$ of the SBSM on the BA graphs. In (a) we show the plots for the system sizes $N=2^{10}$ (black), 2^{11} (red), and 2^{12} (blue) (from left to right). (b) A finite-size scaling of this data shows an excellent data collapse for $\zeta_{BA}=1.62$ and $\eta_{BA}=1.10$ giving the value of the avalanche lifetime exponent to be $\tau_{BA} \approx 1.47$. The left end of each curve shifts to the left on increasing system size.

isting graph. Initially the growth starts from a $(m+1)$ clique. The actual growth process is executed by the improved algorithm [27]. In this method a new vertex selects one of the existing edges with uniform probability and gets a connection to one of its end vertices with probability 1/2 to generate the BA graph. For every vertex this process is repeated m times for attaching m links keeping track that all m vertices must be distinct. We have used $m=2$ in our studies.

For the SBSM on a BA graph, the mutation zone is a single randomly selected vertex out of all the k neighboring vertices. As expected this system also gradually evolves to a steady state. The stationary fitness distribution $P(f)$ shows up the characteristic jump at a certain value of f_c. For the small graphs $P(f)$ grows continuously across the critical fitness value from a low value to a high value. All quantities measured are averaged over different independent realizations of BA graphs. Both the fitness function and avalanche size distribution differ little from one graph to the other so that when the number of configurations are increased the fluctuations in the averaged plot gradually reduced. The dynamics is followed till $\approx 12 \times 10^{10}$ mutation events including 2500 ($N=2^7$) to 152 ($N=2^{13}$) uncorrelated BA graphs. The arrival of the steady state is ensured by keeping track of the average fitness value $\langle f \rangle$ per vertex, which initially grows but eventually becomes steady. The stationary-state data is collected skipping the first 25 million mutation events as the relaxation time. For the fitness distribution, the fitness data is collected from all vertices of the network at the interval of every N mutation events. In Fig. 5(a) the fitness distribution $P(f)$ has been plotted for the system sizes $N=2^7$, 2^{10}, and 2^{13}. The average fitness values $\langle f(N) \rangle$ are measured to obtain the critical fitness thresholds for different N values. These on extrapolation with $N^{-4/3}$ gives $f_c=0.5751(10)$. Therefore for

SBSM on BA graph the branching factor $r_b=1.1502>1$.

For BA graph we have done a finite-size scaling of the fitness distribution data to exhibit explicitly that the value of the fitness threshold $f_c(\infty)$ is indeed nonzero even in the thermodynamic limit. To make a data collapse we need to stretch different curves of Fig. 5(a) to different amounts along the f axis. For that we need to keep one point fixed on every curve. We selected this point, which has $P(f,N)=\mathcal{C}(N)/2$. The corresponding value of f is denoted by $f_{1/2}(N)$ and is calculated by an interpolation. The f axes are then shifted by $f_{1/2}(N)$ amounts for all three plots and have been rescaled by $N^{0.52}$ to obtain a nice data collapse, which is shown in Fig. 5(b).

The avalanche size distributions have been studied for the same values of $f_c(N)$ obtained above. The raw distribution data has been shown in Fig. 6(a) for the three graph sizes $N=2^{10}$, 2^{11}, and 2^{12}. A similar finite-size scaling analysis yields $\eta_{BA}=1.62$ and $\zeta_{BA}=1.10$ giving the value of the lifetime exponent $\tau_{BA} \approx 1.47(3)$ [Fig. 6(b)]. This value is close to the mean-field value of 1.5 obtained by [18].

The branching factor can be reduced even further. During every mutation event we first decide with a probability $p=1/2$ if we update the single site in the mutation zone or not. If it is favored only then one of the nearest neighbors is selected randomly and updated as in SBSM, otherwise the active site is only updated and therefore $\langle M \rangle = 1.5$. In $d=1$, we get an enhanced value of $f_c \approx 0.883(5)$, which means $r_b=\langle M \rangle f_c \approx 1.325>1$. The critical exponents found to be very consistent with SBSM at $d=1$. Figure 7(a) shows the fitness distribution plot whereas Fig. 7(b) exhibits the finite-size scaling analysis of the avalanche size distributions. Our expectation is that on further reduction of the branching factor by reducing p, the f_c will go up but the critical behavior
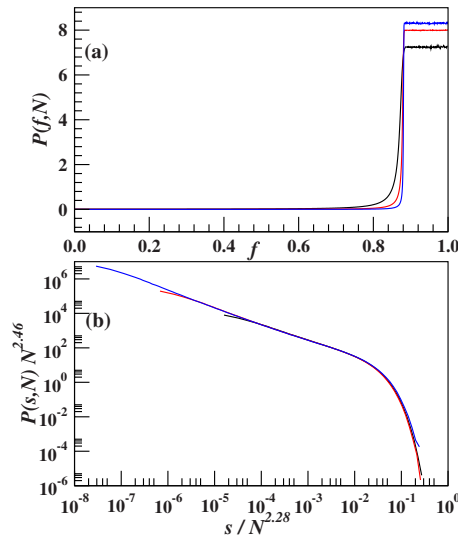
FIG. 7. (Color online) Data for the case when the average number of sites updated in a mutation event $\langle M \rangle = 1.5$ in $1d$ for the system sizes $N = 2^7$ (black), 2^9 (red), and 2^{11} (blue). (a) The fitness distributions $P(f, N)$ vs f. The jump in the distribution becomes gradually sharper with increasing the system sizes. (b) The finite-size scaling of the avalanche size distribution for the same system sizes giving $\eta = 2.46$, $\zeta = 2.28$, and $\tau \approx 1.08$. The left end of each curve shifts to the left on increasing system size.

would remain intact. To check it we simulated only one system size of $N = 2^{11}$ for $\langle M \rangle = 1.75$, 1.25, 1.125, and 1.0625 and obtained $f_c(2^{11})$ values 0.828, 0.935, 0.966, and 0.982, respectively, corresponding to branching factors 1.449, 1.168, 1.087, and 1.044, respectively, all values larger than

unity. Direct measurement of τ exponents from $D(s)$ vs s plots gives 1.07, 1.05, 1.04, and 1.01, respectively. Therefore it seems likely that for any branching factor greater than unity, the SBSM would exhibit a nontrivial SOC state.

Lastly we studied the SBSM on the N-clique graph. Our single member mutation zone is a special case of the random BS model studied in [28]. For all finite-size systems the $f_c(N)$ values are larger than 1/2 but approach to it as N increases. The results in the asymptotic limit are consistent with [28], i.e., $f_c = 0.5$, $\eta_{NC} = 1.5$, and $\zeta_{NC} = 1.0$ giving $\tau_{NC} = 1.5$, a complete mean-field behavior. Here $r_b = 1$ holds good only in the asymptotic limit, for all finite system sizes $r_b > 1$.

To summarize, the ecological evolution process described in the Bak-Sneppen SOC model has been widely regarded as a branching process. Here each mutation event generates randomly on the average r_b offsprings with fitness values under the threshold. Similar to the basic reproduction rate in the theory of branching process we propose that a nontrivial branching process, and thus a nontrivial SOC state in BS model is achieved only when the branching factor $r_b > 1$. To justify this idea we have considered a stochastic version of the BS model where other than minimal fitness site, only one neighboring site is updated. This model is numerically studied on $1d$ and $2d$ regular lattices, Barabási-Albert scale-free networks and N-clique graphs and in all these cases $r_b > 1$ and nontrivial SOC states are observed. In addition we have seen that in $1d$ where on the average 1.5 neighbors are updated in a mutation event, one still has a SOC state. These evidences led us to conjecture that in a stochastic BS model studied on any arbitrary graph where the average branching factor is greater than unity would lead to a nontrivial SOC state.

[1] P. Bak, C. Tang, and K. Wiesenfeld, Phys. Rev. Lett. **59**, 381 (1987).
[2] P. Bak and K. Sneppen, Phys. Rev. Lett. **71**, 4083 (1993).
[3] P. Bak, *How Nature Works: The Science of Self-Organized Criticality* (Copernicus, New York, 1996).
[4] D. Dhar, Phys. Rev. Lett. **64**, 1613 (1990).
[5] S. S. Manna, J. Phys. A **24**, L363 (1991).
[6] See a video in http://cmol.nbi.dk/models/bs/bs.html
[7] P. Grassberger, Phys. Lett. A **200**, 277 (1995).
[8] M. Paczuski, S. Maslov, and P. Bak, Phys. Rev. E **53**, 414 (1996).
[9] S. N. Dorogovtsev, J. F. F. Mendes, and Y. G. Pogorelov, Phys. Rev. E **62**, 295 (2000).
[10] S. Boettcher and M. Paczuski, Phys. Rev. Lett. **84**, 2267 (2000).
[11] P. De Los Rios, M. Marsili, and M. Vendruscolo, Phys. Rev. Lett. **80**, 5746 (1998).
[12] G. J. M. Garcia and R. Dickman, Physica A **342**, 516 (2004).
[13] S. Maslov, P. De Los Rios, M. Marsili, and Y.-C. Zhang, Phys. Rev. E **58**, 7141 (1998).
[14] M. Vergeles, Phys. Rev. Lett. **75**, 1969 (1995).
[15] R. Cafiero, P. De Los Rios, F.-M. Dittes, A. Valleriani, and J. L. Vega, Phys. Rev. E **58**, 3993 (1998).

[16] K. Christensen, R. Donangelo, B. Koiller, and K. Sneppen, Phys. Rev. Lett. **81**, 2380 (1998).
[17] G. Caldarelli, A. Capocci, and D. Garlaschelli, Eur. Phys. J. B **64**, 585 (2008).
[18] Y. Moreno and A. Vazquez, Europhys. Lett. **57**, 765 (2002).
[19] A.-L. Barabási and R. Albert, Science **286**, 509 (1999).
[20] R. Albert and A.-L. Barabási, Rev. Mod. Phys. **74**, 47 (2002).
[21] K. E. Lee, B. H. Hong, and J. W. Lee, e-print arXiv:cond-mat/0510067.
[22] S. Lee and Y. Kim, Phys. Rev. E **71**, 057102 (2005).
[23] N. Masuda, K.-I. Goh, and B. Kahng, Phys. Rev. E **72**, 066106 (2005).
[24] N. Deo, *Graph Theory* (Prentice-Hall of India, New Delhi, India, 2004).
[25] M. Felici, G. Caldarelli, A. Gabrielli, and L. Pietronero, Phys. Rev. Lett. **86**, 1896 (2001).
[26] K. Christensen and N. R. Moloney, *Complexity and Criticality* (Imperial College Press, London, 2005).
[27] G. Mukherjee and S. S. Manna, Phys. Rev. E **74**, 036111 (2006).
[28] H. Flyvbjerg, K. Sneppen, and P. Bak, Phys. Rev. Lett. **71**, 4087 (1993).