Introducing a real time scale into the Bak-Sneppen model

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Recently, a simple model of evolution has been proposed by Bak and Sneppen [Phys. Rev. Lett. **71**, 4083 (1993)]. This model self-organizes into a critical state for nearest- and random-neighbor interactions. The Bak-Sneppen (BS) model has no explicit time scale, because time steps are always identified with an evolutionary step. Therefore, we introduce at each time step a local stochastical update rule. Hence it is possible to observe time steps in which no species are removed from the system. In the following, the durations of time steps in which no further evolution occurs are called interevent intervals. We study a random-neighbor version of the model and derive the steady-state distribution of the fitnesses. The distributions are the same for synchronous and asynchronous updating rules and resemble the solutions obtained for the mean field BS model. We give an interpretation of the modified BS model as a neural network with random connections. For a concrete choice of the stochastical updating rule, we derive the distribution of the interevent or interspike intervals. It turns out that for parallel updating we get a power law decay, whereas in the case of random sequential updating the distribution is simply an exponential in the limit $N \to \infty$. N is the system size. All analytical results are supported by numerical simulations.

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I. INTRODUCTION

Based on morphological observations, it has been speculated that the evolution of life is concentrated in very rapid events of speciation [1] rather than in a gradual manner as supposed by Darwin. The fitness of most species hardly changes and fluctuates only mildly. This concept is called *punctuated equilibria.* Intermittent bursts of evolutionary activity are separated by inactive periods or stasis. Observations of Raup [2] suggest that extinctions of species are episodic on all scales. Therefore it has been conjectured that the ecology of interacting species has evolved to a self-organized critical state [3,4]. Considering the NKC model for special parameters of K and C, it was found that the time for the system to reach Nash equilibrium decays algebraically [3]. (N is the number of genes in a genotype, K is the number of other genes in the same genotype to which one gene is connected, and C is the number of genes in other genotypes to which one gene is connected).

Recently a simple model of evolution was introduced by Bak and Sneppen that reveals indeed self-organized criticality [5]. Their model is defined as follows. The ecology consists of N species on a d-dimensional lattice. Each species is assigned a random fitness $0 \leq x_i \leq 1$ chosen from a uniform distribution $\hat{P}(x_i) = \vartheta(1 - x_i)\vartheta(x_i)$, where ϑ is the step function. At each step the site with the lowest fitness x_i and its $2d + 1$ nearest neighbors are changed to new random numbers ξ_i, which are drawn from a uniform distribution $\hat{P}(\xi_i)$. After a number of steps the system reaches a dynamical steady state. Bak and Sneppen measured the distances r on the lattice between subsequently active sites and found for the one-dimensional nearest-neighbor model a power-law decay for the distribution $P(r) = r^{-3.15\pm0.05}$. In the random-neighbor version of the Bak-Sneppen (BS) model, where the $2d + 1$ neighbors are chosen anew at each step, the steady-state distribution of the mean fitness can be derived analytically [6]. One obtains a step function with the discontinuity located at $1/K$, where $K-1$ is the number of neighbors. An *avalanche* is defined in this model as follows. If the smallest fitness $x_{\min}$ falls below a value q, with $0 \leq q \leq 1$, then the number of subsequent steps needed for $x_{\min}$ to exceed q again is defined as the length of the avalanche. If q is exactly $1/K$ an avalanche can be identified in the limit $N \to \infty$ with the length of a tree in a critical branching process and the distribution $\Pi(l)$ of the lengths l scales as $\Pi(l) = l^{-3/2}$ [7]. For $K = 2$ this can be explained in terms of a one-step process. For $N \to \infty$ the total number S^t of sites i with fitness x_i below $1/2$ at step t performs simply a symmetrical random walk. In the case $K = 2$ we see that $S^{t+1} = S^t$ with probability 0.5, because there are two possibilities that exactly one of the two new random values is smaller than q. With probability 0.25 both new uniform distributed random values are below or above q and we obtain $S^{t+1} = S^t + 1$ or $S^{t+1} = S^t - 1$, respectively. Therefore the considered avalanches are in fact the recurrence times of the renewal process S^t. For the random walk it is well known [8] that the number of steps needed to reach the starting point again scales with the exponent $-3/2$. In [9] the finite-size effects are calculated, which cut off the power-law behavior. If $q < 1/K$ the recurrence times decay exponentially, whereas for $q > 1/K$ it is unlikely that S^t returns to zero. The critical behavior of the recurrence time occurs only for $q = 1/K$.

The BS model does not describe evolution on a physical time scale, because an update step *always* corresponds to a mutation of the species with the smallest fitness and its neighbors. This implies that we would observe a constant extinction intensity in morphological data and that

there will never be periods in which the system does not change. In contrast to this, the biological extinction intensity exhibits a large variability [2]. Therefore we extend the BS model by introducing a local stochastic variable η_i, which simulates unspecified degrees of freedom that can lead to an extinction of a species i. Hence we get a *local* mutation or update probability for each site at each time step. The update probability depends on the fitness of the considered species. In Sec. II we solve the random-neighbor version of our model for a general distribution $A(\eta_i)$, which is normalized. If η_i is larger than the fitness x_i of species i, species i and its neighbors will be mutated. Giving that updating rule, it is more likely that a species with high fitness will survive and a species with low fitness becomes extinct and is replaced by a new species. So there is no need to mutate in every step exactly the species with the lowest fitness value. Furthermore, it is possible to observe time steps in which no evolution takes place. Therefore the system evolves in real time and not simply in mutation steps. Henceforth we can measure the distribution of the interevent intervals within the evolving system, which is interrupted by bursts of adaptation or speciation as proposed in [1].

There are hints that processes in the brain exhibit critical behavior [10]. We propose in Sec. III a simple neural network with random connections, which is related to our mean-field model. In this case, η_i obeys the Boltzmann distribution $A(\eta_i) = \beta e^{-\beta\eta_i}$. The distribution of the interevent periods or interspike intervals is calculated for this model. In the case of parallel update we observe power-law behavior with the exponent -1. The cutoff of the algebraic decay depends, in the limit $N \to \infty$, on the parameter β and the connectivity K. For the sequential updating rule the distribution of the interevent intervals decays exponentially. In Sec. IV we discuss several conclusions and summarize our results.

II. MODEL

We consider N sites with real *fitnesses* $x_i \in [0,1]$. If the fitness x_i^t, where t denotes time, is smaller than η_i^t, then in the next time step x_i^t changes to $x_i^{t+1} = \xi_i^t$, where ξ_i^t is a new uniformly distributed random value between 0 and 1, with the probability distribution $\hat{P}(\xi_i) = \vartheta(\xi_i)\vartheta(1-\xi_i)$. ϑ denotes the unit step function and η_i is a stochastic variable, which models all not explicitly known degrees of freedom of the system and is chosen anew at every time step from a normalized distribution $A(\eta_i)$, $\int d\eta_i\, A(\eta_i) = 1$, for each site. We denote the number of sites that change their fitness due to the fact that $x_i^t \le \eta_i^t$ per unit of time by L. Together with a site i that is changed in this fashion, its $K-1$ neighboring sites, denoted by $\{r_i\}$, will also obtain new randomly chosen fitnesses ξ_{r_i} that are independent of the actual fitness $x_{r_i}^t$. Thus there are two causes for a change of fitness.

In contrast to the BS model, we have to deal only with a local update rule. There is no need to select the smallest fitness. On the other hand, time steps are possible in which no update occurs. If we consider parallel updating, more than one site can be mutated directly, i.e., via $x_i^t < \eta_i^t$ in a single time step. Hence coevolution occurs in parallel at many different sites within the whole system.

In this paper we analyze a random-neighbor model, where for each species i we choose $K-1$ neighbors at random in each time step. This is usually called "annealed" disorder instead of "quenched" disorder for which the randomly chosen neighbors are the same for all time steps. Note that if L sites are changed via $x_i < \eta_i$, a total number of LK sites are involved in the update process. The equations of motion for the process are given by

$$x_i^{t+1} = \begin{cases} \xi_i^t & \text{if } \Theta_i^t > 0 \\ x_i^t & \text{if } \Theta_i^t = 0, \end{cases} \tag{1}$$

where

$$\Theta_i^t = 1 - \prod_{k \in M_i^t} \left(1 - \vartheta_k^t\right) . \tag{2}$$

ϑ_k^t is an abbreviation for $\vartheta(\eta_k^t - x_k^t)$ and M_i^t denotes the set of $\{i\}$ with its neighbors $\{r_i\}$ at time step t. From Eq. (2) it follows that Θ_i itself is a unit step function.

We consider synchronous and asynchronous updating rules for our model. For asynchronous updating only one randomly chosen fitness x_i^t is compared to η_i^t per unit of time. Note that this means that altogether K sites, i.e., i and its $K-1$ neighbors, are updated instantaneously. For synchronous or parallel updating we compare all x_i^t's with their related η_i^t's at each time step.

For both update rules we derive the steady-state distribution $p(x)$ in the mean-field approximation. We denote the joint probability distribution at time t by $P^t(x_1, ..., x_N) = P^t(\vec{x})$ and the conditional transition density for the system to be at $\vec{x}$ if it was previously at $\vec{x}'$ by $K(\vec{x}|\vec{x}')$. The time evolution of the system is given by

$$P^{t+1}(\vec{x}) = \int d\vec{x}' K(\vec{x}|\vec{x}') P^t(\vec{x}'), \tag{3}$$

where

$$K(\vec{x}|\vec{x}') = \left\langle \prod_i \delta(x_i - \xi_i\Theta_i' - x_i'(1-\Theta_i')) \right\rangle_{\vec{\xi},\vec{\eta}} \tag{4}$$

and $\langle\,\rangle_{\vec{\xi},\vec{\eta}}$ is an abbreviation for the annealed average with the distributions $\hat{P}(\vec{\xi}) = \prod_i \hat{P}(\xi_i)$ and $A(\vec{\eta}) = \prod_i A(\eta_i)$, over a random neighborhood and in the case of sequential updating over the randomly chosen site i. Now we use the fact that any function $f(x)$, with $x = 0, 1$, can be decomposed as $f(x) = f(0)(1-x) + f(1)\,x$ and obtain

$$K(\vec{x}|\vec{x}') = \left\langle \prod_i \{\delta(x_i - \xi_i)\Theta_i' + \delta(x_i - x_i')(1-\Theta_i')\} \right\rangle_{\vec{\xi},\vec{\eta}} . \tag{5}$$

A. Asynchronous updating

Under the assumption that only one site could be updated directly per time step, i.e., a total of K sites could change their fitness, Eq. (5) reduces to

$$K(\vec{x}|\vec{x}') = \left\langle \prod_k \delta(x_k - x'_k) \right\rangle_{\vec{\xi},\vec{\eta}} + \left\langle \vartheta'_i \, \delta(x_i - \xi_i) \prod_{\{r_i\}} \delta(x_{r_i} - \xi_{r_i}) \times \prod_{k \notin M_i} \delta(x_k - x'_k) - \vartheta'_i \prod_k \delta(x_k - x'_k) \right\rangle_{\vec{\xi},\vec{\eta}} . \quad (6)$$

Note that transitions in which more than one and fewer than K sites get new random values cannot happen. Equation (6) is valid in the case of asynchronous updating for *any* distribution $A(\eta_i)$, because only one site is selected for comparison. But if we focus on parallel updating it is possible that more than K sites change in a single time step. Therefore, in order to apply the results we will derive from Eq. (6), we must restrict the choice for the updating function $A(\eta_i)$. We will discuss this point later in this section.

If we take the average over $\prod_i A(\eta_i)$ and introduce $B(x_i) = \int_{x_i}^{\infty} d\eta_i A(\eta_i)$ we obtain

$$K(\vec{x}|\vec{x}') = \prod_k \delta(x_k - x'_k) + \left\langle B(x'_i) \, \delta(x_i - \xi_i) \prod_{\{r_i\}} \delta(x_{r_i} - \xi'_{r_i}) \times \prod_{k \notin M_i} \delta(x_k - x'_k) \right\rangle_{\vec{\xi}} - \left\langle B(x'_i) \prod_k \delta(x_k - x'_k) \right\rangle_{\vec{\xi}} . \quad (7)$$

Since $\langle \delta(x - \xi) \rangle_\xi = 1$ we obtain

$$K(\vec{x}|\vec{x}') = \prod_k \delta(x_k - x'_k) + \left\langle B(x'_i) \prod_{k \notin M_i} \delta(x_k - x'_k) - B(x'_i) \prod_k \delta(x_k - x'_k) \right\rangle . \quad (8)$$

By using the mean-field ansatz that $P(\vec{x})$ factorizes, we derive in Appendix A the master equation for the reduced density $p^t(x)$, which is defined as

$$p^t(x) = \frac{1}{N} \sum_{l=1}^{N} \int d\vec{x} \, \delta(x - x_l) P^t(\vec{x}) = \frac{1}{N} \sum_{l=1}^{N} P_l^t(x), \quad (9)$$

and obtain

$$p^{t+1}(x) = p^t(x) - \frac{1}{N} B(x) p^t(x) - \frac{K-1}{N} C^t(B) p^t(x) + \frac{K}{N} C^t(B) + O\left(1/N^2\right), \quad (10)$$

where C^t is a functional of $B(x)$ and $p^t(x)$

$$C^t[B, p^t] = \int dx \, B(x) \, p^t(x). \quad (11)$$

C^t is the probability that the fitness x_i for a randomly selected site i is smaller than η_i at time step t. By neglecting terms of $O(1/N^2)$ and because C is independent of x, we can calculate from Eq. (10) the equilibrium distribution for $p(x) = \lim_{t \to \infty} p^t(x)$ and obtain

$$p(x) = \frac{K/(K-1)}{1 + B(x)/(K-1)C[B,p]}. \quad (12)$$

This is the main result of this section.

B. Synchronous updating

As mentioned above, Eq. (6) and therefore the master equation (10) will be valid for parallel updating only if no more than one site is updated by a direct mutation per unit of time. Now C^t measures the mean activity of all sites that are changed via $x_i^t \leq \eta_i^t$. Roughly stated, in the case of parallel updating we have to choose an updating function $A(\eta)$ such that C is of order $1/N$. But suppose that L sites are selected for comparison; then a total of LK sites could change their fitness. If we demand that all sites i and their related neighbors $\{r_i\}$ are distinct, we can extend Eq. (6) and obtain the master equation

$$p^{t+1}(x) = p^t(x) - L\left(\frac{1}{N} B(x) p^t(x) - \frac{(K-1)}{N} C^t(B) p^t(x) + \frac{K}{N} C^t(B) \right) + O\left(1/N^2\right). \quad (13)$$

This leads to the same steady-state distribution $p(x)$ as in Eq. (12). It is obvious that $L \leq N/K$, since no more than N sites could change their fitness in one time step. Hence we can weaken our restriction for the distribution $A(\eta)$ and conclude that C should only be of order $1/K$, i.e., independent of N.

For parallel updating the equilibrium is approached faster than for sequential updating. This can be seen in the following example. Suppose that at the beginning of the process all fitnesses are uniformly distributed and that at each update step only the smallest fitness is mutated like for the BS model. We denote by R^s the number of species with fitness below $1/K$. Hence, on the average, we obtain $R^0 = N/K$ and

$$R^s \simeq R^0 \left(1 - \frac{K-1}{N-1}\right)^s + O(1/N) \quad (14)$$

fitnesses remain smaller than $1/K$ at an updating step s for an asynchronous updating rule. If we assume that the average number of update steps $\bar{s}$ needed to reach equilibrium is at least of order $O(N)$, a rough estimation leads to

$$\bar{s}_{\text{asyn}} \propto \frac{\ln \frac{K}{N-1}}{\ln\left(1-\frac{K-1}{N-1}\right)}$$
$$\propto \frac{N}{K-1}\ln\frac{N-1}{K}, \qquad N \gg K\,. \tag{15}$$

Therefore the duration to reach equilibrium scales with $N \ln N$ for asynchronous updating and is reduced if K is increased. Assume that in the case of parallel updating $LK \leq N$ species are mutated together and that the mutated species do not interact. Then the mean number of updating steps reduces to

$$\bar{s}_{\text{syn}} \propto \frac{\ln \frac{K}{N-1}}{\ln\left(1-\frac{L(K-1)}{N}\right)}. \tag{16}$$

Therefore $\bar{s}_{\text{syn}} \ll \bar{s}_{\text{asyn}}$ in the case $1 \ll L \leq N/K$.

III. INTERPRETATION OF THE MODEL AS A NEURAL NETWORK

In this section we map our mean-field model to a neural model. To gain analytical results, we will choose a special function for $A(\eta_i)$ and derive the steady-state distribution $p(x)$. The result are valid for the BS model with a real time scale. Table I explains the correspondence between both models.

We define our neural network as follows. Consider N interacting neurons. From physiology it is known that cells in the central nervous system do not have a unique resting potential. The actual depolarization depends on the activity within a neuronal assembly, the potassium and transmitter concentration in the outside of the cell, and the metabolic exchange with the surroundings.

Now we make an enormous simplification of the complicated microphysical processes by considering only two variables for each neuron. One variable is the resting potential d_i and the second represents all mechanisms that have a short-time impact on the membrane of the neuron and do not lead to a long-time depolarization. We describe these charge fluctuations on the membrane of neuron i by a stochastic variable η_i. Furthermore, we restrict the resting potential d_i to vary between -1 and 0 and approximate d_i by a constant that does not alter until the neuron fires or the neuron receives input from another neuron. If $d_i + \eta_i$ exceeds the threshold, which we choose without restrictions to be 0, neuron i fires and many processes begin to restore the resting potential.

At this point we make the conjecture that the neuron does not get the same depolarization as before spiking, but instead a value $-\xi_i$, which is drawn randomly from a distribution $\hat{P}(\xi_i)$. For simplicity we assume that $\hat{P}(\xi_i)$ is a uniform distribution between 0 and 1, but this will not affect our main results. Moreover, neuron i will change the resting potentials of $K-1$ other neurons to which it is connected. The interaction is mediated via transmitters that can excite or inhibit, i.e., depolarize or hyperpolarize, other neurons. This process by itself is quite complicated and we approximate the influence by the assumption that the neighboring cells get new depolarizations d_i, which we choose in the same way as for the firing neuron. If we replace d_i by $-x_i$ and ξ by $-\xi$ and choose annealed random neighbors of each firing neuron, we can apply all results derived in Sec. II. Without restriction we consider only positive values of η_i because negative η_i's will never fire a neuron. For a suitable choice of $A(\eta_i)$ we can obtain a process where it is much more likely that a neuron with a high resting potential (near the threshold at 0) spikes than a neuron with a low one (near -1). For concreteness we choose the

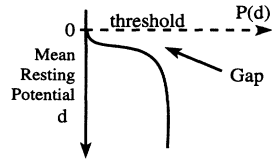

FIG. 1. Distribution of the mean resting potential of a neuronal assembly that is connected and updated according to our model proposed in Sec. I. If the system reaches the steady-state distribution, one observes a gap $\sim 1/K$, where $K-1$ is the connectivity within the network.

TABLE I. Comparison between the values in the evolution model and the neural network model.

Evolution model	Neural model
fitness x	resting potential $d = -x$
new fitness ξ	new resting potential $-\xi$
environmental fluctuation η	charge fluctuation η

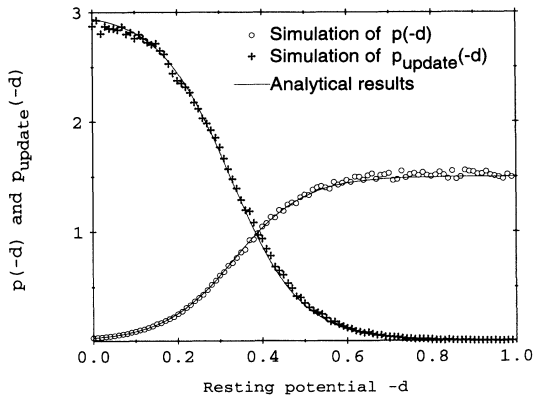

FIG. 2. Results for sequential updating of our model. Both the equilibrium distribution of the mean resting potential $p(-d)$ and the equilibrium distribution of the updated resting potentials $p_{\text{update}}(-d)$ are Fermi functions with the threshold at $\sim 1/K$. All curves are displayed with $\beta = 12$, $N = 100$, and $K = 3$.

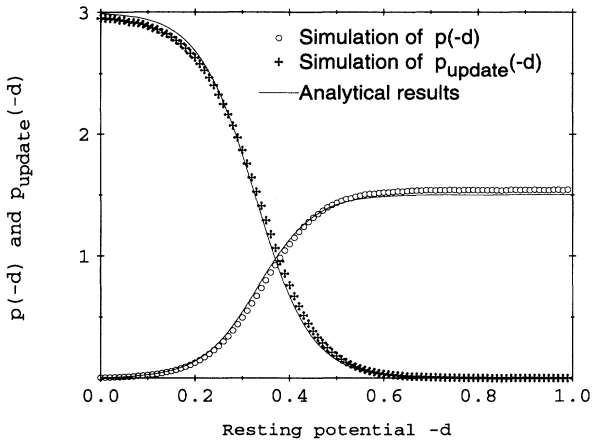

FIG. 3. Analytical results and the simulations for $p(-d)$ and $p_{\text{update}}(-d)$ in the case of parallel updating. Like in the asynchronous updating model, the simulations and the analytically predicted curves are in good agreement. The chosen parameters are $\beta = 17$, $N = 100$, and $K = 3$.

Boltzmann distribution for thermal activated potentials

$$A(\eta_i) = \beta\, e^{-\beta\eta_i}. \tag{17}$$

Inserting this into Eq. (12) yields (see Appendix B)

$$p(x) \simeq \frac{K/(K-1)}{1+\exp\left[-\beta\,(x-1/K)\right]} \qquad \text{for} \quad \beta \gg 1. \tag{18}$$

In order to get the distribution of the resting potentials $p(d)$ one simply has to reflect the function $p(x)$ at the y axis. The mean-field equilibrium distribution of the resting potentials of an interconnected assembly of neurons self-organizes to a Fermi function (Figs. 1–3). The results do not depend on the updating rule for the neuronal assembly, as long as in the case of synchronous update one chooses $\beta > K$ to ensure that $C \leq 1/K$ (or $\beta > K \ln N$ if we demand $C \leq 1/N$, respectively). From the distribution one sees that only a few neurons have depolarizations that are in the range $[-1/K, 0]$, whereas all others have resting potentials that are smaller than $-1/K$. Hence there is a gap of width $1/K$ between the threshold at 0 and nearly all resting potentials. Remember that $K-1$ is the connectivity within the network. The distribution of the updated resting potentials $p_{\text{update}}(x)$ can also be calculated. We get

$$p_{\text{update}}(x) = \frac{e^{-\beta x}p(x)}{C(\beta)} = \frac{K}{1+e^{-\beta(1/K-x)}}. \tag{19}$$

This is just a reversed Fermi function and therefore only a few neurons with resting potentials d smaller than $-1/K$ are actually spiking, whereas almost all spiking neurons have resting potentials between 0 and $-1/K$. Figures 2 and 3 show a comparison of the analytical and the simulated steady-state distributions for $p(d)$ and $p_{\text{update}}(d)$.

IV. DISTRIBUTION OF INTERSPIKE INTERVALS

We derive the interspike interval (ISI) distribution for our mean-field model driven by the stochastic process of Eq. (17). The ISIs directly correspond to the interevent intervals in the BS model with a physical time scale as proposed in Sec. II.

There are indications that neurons exhibit a high variability of their ISIs [11]. It is therefore interesting to look at the interevent intervals of our simple model. If the network consists of only one neuron, it will exhibit essentially a renewal process. Every time the neuron is updated, it starts with a new random value ξ chosen from the uniform distribution $\hat{P}(\xi)$. We have to calculate the expectation value for the number of subsequent events in which the noise η^t is smaller than ξ. Hence the distribution of the ISIs is given by

$$L_\beta(\tau) = \sum_{\tau'} \left\langle \delta\left(\tau - \tau' \prod_{t=1}^{\tau'-1} \vartheta(\xi-\eta^t)\vartheta(\eta^{\tau'}-\xi)\right)\right\rangle_{\vec{\eta},\xi}, \tag{20}$$

which is the probability that the resting potential $d = -\xi$ approaches the threshold at 0 after exactly τ time steps. After averaging over the stochastically independent noise η^t (see Appendix C) we obtain

$$L_\beta(\tau) = \int d\xi \left(1-e^{-\beta\xi}\right)^{\tau-1} e^{-\beta\xi}. \tag{21}$$

Keeping ξ fixed, the integrand is just an exponential function that is equal to the interevent distribution of a discrete point process with the event probability $p = \exp(-\beta\xi)$. To solve the integral we substitute $z = 1-e^{-\beta\xi}$ and obtain the ISI distribution of our neural network model consisting of only one neuron

$$L_\beta(\tau) = \frac{\left(1-e^{-\beta}\right)^\tau}{\beta\tau}. \tag{22}$$

For large β we can rewrite Eq. (22) and get

$$L_\beta(\tau) = (\beta\tau)^{-1} e^{-\tau/\bar{\tau}}, \tag{23}$$

with $\bar{\tau} \simeq e^\beta$. In Fig. 4 a comparison between the analyt-

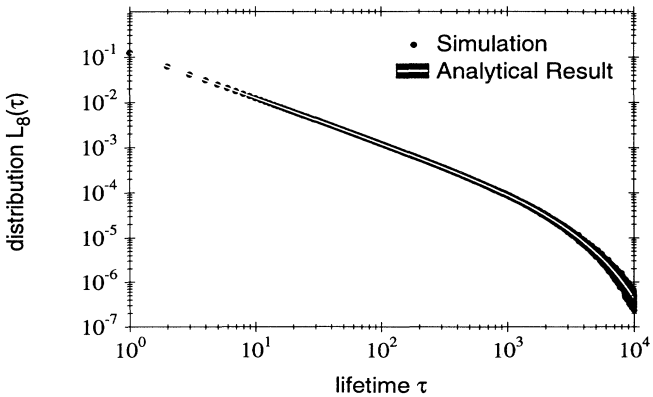

FIG. 4. Analytical results and simulated distribution $L_8(\tau)$ of the time intervals between updates with parameters $N = 1$ and $\beta = 8$.

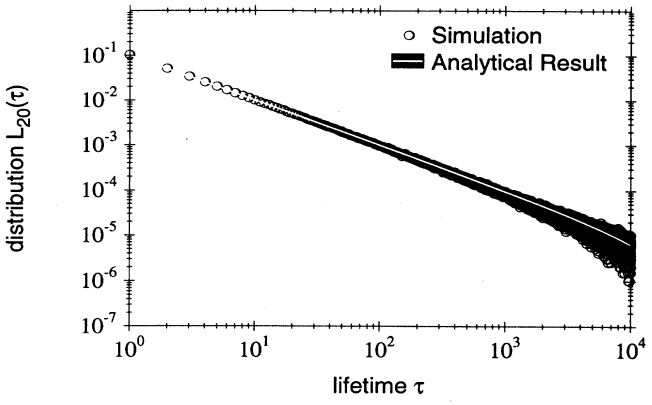

FIG. 5. Analytical results and simulated distribution $L_{20}(\tau)$ of the time intervals between parallel updates with parameters $\beta = 20$, $N = 10$, and $K = 2$.

ical result and a simulation is displayed. For one neuron and hence $K = 1$ we can derive the equilibrium distribution from Eq. (10)

$$p(x) = \beta \frac{e^{\beta x}}{e^{\beta} - 1} \simeq \beta e^{\beta(x-1)}. \tag{24}$$

We summarize that one observes power-law-distributed interspike intervals for isolated neurons in our model, i.e., neurons without interaction to other neurons. The power law exhibits a finite-size scaling and is exact if the system is in the frozen state at $\beta \to \infty$. In this limit we see from Eq. (24) that the mean resting potential $d = -x$ is located maximally away from the threshold and lies at $d = -1$.

The results differ only slightly if we introduce interaction in the neuronal assembly. If all neurons are updated in parallel (see Fig. 5) we obtain roughly the same distribution as for only one neuron, but with a different finite-size scaling. We found $\bar{\tau} \simeq e^{\beta/K}$ for large β (see Appendix C) and hence the strict power-law behavior of the ISI is not so far extended as in the case of a single neuron. In both cases the power spectrum of the ISI is flat, because $S(f) \sim 1/f^D$. Following [12], the fractal dimension D is defined by $D = -1+\gamma$, where γ is the exponent of the ISI distribution $L_\beta(\tau)$. In the limit $\beta \to \infty$ the exponent γ tends to -1 and hence the fractal dimension to 0. For synchronous update we have to give a lower bound for β to take care that the restriction stated above ($C \leq 1/K$ or $C \leq 1/N$) is fulfilled. We obtain $\beta \geq K$ and $\beta \geq K \ln N/(K-1)$, respectively. The crossover in the distribution $L_\beta(\tau)$ has a simple explanation. The variable η_i is distributed in the interval $[0, \infty)$, whereas the fitness is distributed in the interval $[0, 1]$. Therefore the state of maximal fitness of the system has only a limited lifetime. If we choose the Boltzmann distribution confined and normalized to the interval $[0, 1]$, we observe no crossover in the power-law distribution $L_\beta(\tau)$. The $1/\tau$ scaling within a finite range for the mean-field BS model had been derived also in [13] without the finite-size effect.

Now we turn to the case of sequential update. The distribution of interevent intervals is

$$L_\beta(\tau) = \sum_{\tau'} \left\langle \delta\left(\tau - \tau' \prod_{t=1}^{\tau'-1} \vartheta(x_t - \eta^t)\vartheta(\eta^{\tau'} - x_{\tau'})\right)\right\rangle_{\vec{\eta},\vec{x}}, \tag{25}$$

where x_t denotes the fitness of the randomly chosen site at time step t. Using the same procedure as for Eq. (20), we obtain

$$L_\beta(\tau) = \left\langle \prod_{t=1}^{\tau-1} \left(1 - e^{-\beta x_t}\right) e^{-\beta x_t} \right\rangle_{\vec{x}}. \tag{26}$$

Under the assumption that the distribution of the fitnesses is the same for all time steps, which is valid only in the limit $N \to \infty$, we get

$$L_\beta(\tau) = \int dx\, p(x)\, e^{-\beta x} \left\{ \int dx\, p(x) \left(1 - e^{-\beta x}\right) \right\}^{\tau-1}$$
$$= C_1(\beta)\, [C_2(\beta)]^{\tau-1}, \tag{27}$$

where C_1 and C_2 are constants. For finite N the distribution $p(x)$ fluctuates and the decay should differ from

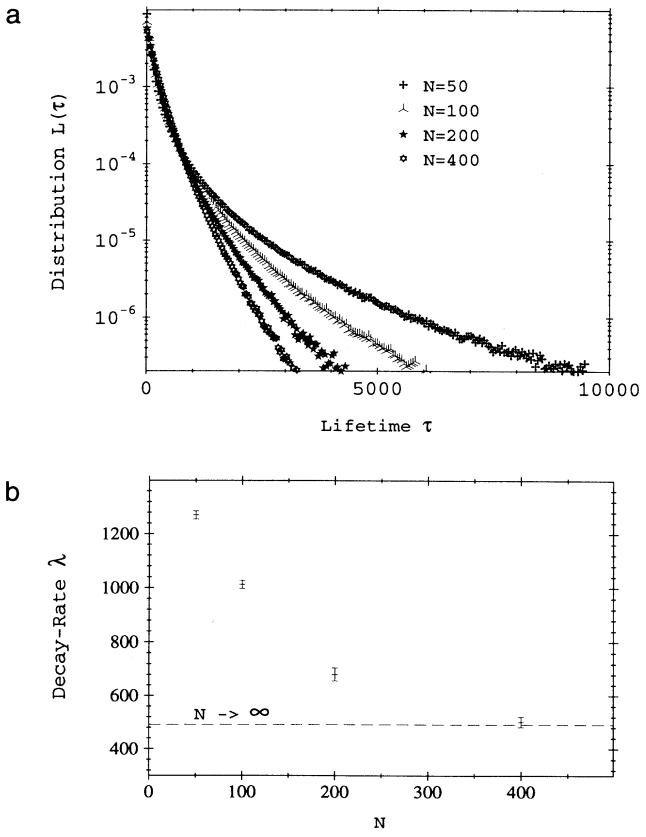

FIG. 6. (a) Simulated distributions of the time intervals between sequential updates for several values of N. (b) The exponential decay rates λ converge for increasing system size N to the analytical solution $\ln(1/C_2)$ (dashed line). All simulations are done with parameters $\beta = 17$ and $K = 3$.

an exponential function. For $N = 1$ one gets the result of Eq. (22) independent of the chosen updating rule. Therefore, in the case of asynchronous updating for increasing N we expect a transition from algebraic to exponential decay in the interevent statistics. In Fig. 6(a) a simulation with four different values of N is shown. In Fig. 6(b) the decay rate vs system size N is displayed. The decay rate λ approaches the limiting value $\ln(1/C_2)$ for large N.

V. CONCLUSION AND SUMMARY

We have proposed a random-neighbor model that is for a special choice of the updating function $A(\eta)$ capable of producing self-organized critical behavior with a finite-size scaling. The critical state corresponds to a phase transition at zero temperature. Indeed, in the limit $\beta \to \infty$ the system freezes and further dynamical changes are unlikely to occur.

Nevertheless, our model has two interesting applications. It is possible to introduce a real time scale into the BS model by assigning each species i a local updating probability $B(x_i^t)$ depending on its actual fitness x_i^t. In our interpretation of the BS model, mutation will occur only if $x_i^t < \eta_i^t$, where the stochastic variable η_i models all influences on species i that are not explicitly known. It reveals that the mean-field BS model corresponds to our model in the limit $\beta \to \infty$ and by regarding only the updating points on the real time scale.

Furthermore, we investigated a neural network with random connections that is driven by weak noise. We found a self-organized gap $\sim 1/K$ between the neuronal threshold and nearly all resting potentials. $K-1$ is the connectivity of the neurons. The interspike intervals of the network obey a power-law distribution with the mean-field exponent -1. The crossover in the interspike statistics depends on a parameter β, which controls the updating or spike probability of each neuron.

All results derived for the neural network model apply to the BS model also. Therefore Figs. 2 and 3 display the mean equilibrium distribution of the fitness in an ecosystem and the mean fitness of the updated species respectively. The distributions differ only slightly from the results in [6] as long β is large enough. This is not astonishing because the local stochastic updating procedure converges to the global updating rule used in the BS model for $\beta \to \infty$. However, as mentioned above, in this limit the ecology will be in a frozen state where adaptation rarely occurs.

The main idea in [1] is the concept of *punctuated equilibrium*. This means that the ecology remains in a stasis for long times and gets interrupted by bursts of activity in which species adapt rapidly. If we identify the stasis periods with the interevent intervals, then Eq. (23) is the desired distribution (see also Figs. 4 and 5). Therefore the periods obey a power law and show finite-size scaling.

It is likely to find other applications of our mean-field model proposed in Sec. II. Furthermore, it is possible to apply different types of updating functions $A(\eta)$ and look for the steady-state distribution and the interevent statistics. It would be interesting to see for which choices of the noise η the process will or will not generate a power law in the interevent statistics.

APPENDIX A

In this appendix we derive the mean-field master equation for the reduced density $p(x)$. We insert the integral kernel Eq. (8) in Eq. (3) and assume that $P(\vec{x})$ factorizes. We get

$$\begin{aligned} P^{t+1}(\vec{x}) &= \int d\vec{x}'\, K(\vec{x}|\vec{x}')P^t(\vec{x}') \\ &= P^t(\vec{x}) + \left\langle C_i^t \prod_{k\notin M_i} P_k^t(x_k) \right. \\ &\quad \left. - B(x_i)P_i^t(x_i)\prod_{k\neq i} P_k^t(x_k) \right\rangle, \end{aligned} \tag{A1}$$

where we have introduced $C_i^t[B] = \int dx' B(x')P_i^t(x')$ and used the definition of M_i from Sec. II. $\langle\,\rangle$ denotes now the average over the randomly chosen site i and its $K-1$ annealed random neighbors $\{r_i\}$.

Now we use the definition for the reduced density $p^t(x)$ from Eq. (9) and obtain

$$\begin{aligned} p^{t+1}(x) &= p^t(x) + \left\langle \frac{1}{N} C_i^t \left(\sum_{k\notin M_i} P_k^t(x) + K \right) \right. \\ &\quad \left. - \frac{1}{N} B(x)P_i^t(x) - \frac{1}{N} C_i^t \sum_{k\neq i} P_k^t(x) \right\rangle \\ &= p^t(x) - \frac{1}{N} B(x)p^t(x) + \frac{K}{N} C^t \\ &\quad - \left\langle \frac{1}{N} C_i^t \sum_{k\in\{r_i\}} P_k^t(x) \right\rangle. \end{aligned} \tag{A2}$$

For large N we get

$$\begin{aligned} p^{t+1}(x) &= p^t(x) - \frac{1}{N} B(x)p^t(x) \\ &\quad - \frac{K-1}{N} C^t p^t(x) + \frac{K}{N} C^t + O\left(1/N^2\right), \end{aligned} \tag{A3}$$

where we have abbreviated $C^t = \frac{1}{N}\sum_i C_i^t$.

APPENDIX B

We calculate the equilibrium distribution $p(x)$ for the special choice $A(\eta_i) = \beta\, e^{-\beta\eta_i}$. The updating probability of x is given by

$$B(x) = e^{-\beta\, x}. \tag{B1}$$

Integration of Eq. (18) yields

$$\frac{K}{K-1}\int_0^1 dx\, \frac{1}{1 + D\, e^{-\beta\, x}} = 1, \tag{B2}$$

where $D = \frac{1}{(K-1)C}$. We substitute $y = e^{-\beta x}$ and get

$$\frac{K}{(K-1)\beta}\int_{e^{-\beta}}^{1} dy\,\frac{1/D}{y\,(1/D+y)} = 1. \tag{B3}$$

We write $\frac{1}{D}/y\left(\frac{1}{D}+y\right)$ as $1/y - 1/(\frac{1}{D}+y)$ and integrate Eq. (B3). We solve for D and obtain

$$D = \frac{1-\exp\left[-\beta\,(1-1/K)\right]}{\exp\left[-\beta\,(1-1/K)\right]-\exp(-\beta)}. \tag{B4}$$

We get for $\beta \gg 1$

$$\frac{1}{C} = (K-1)\exp\left[\beta\,(1-1/K)\right]. \tag{B5}$$

After inserting $1/C$ into Eq. (12) we obtain the distribution displayed in Eq. (18).

APPENDIX C

We derive in this appendix the distribution of the time intervals $L_\beta(\tau)$ between two successive updates. For $N = 1$ the result is exact; for large N we make an approximation that will be valid for large β's. The product over the ϑ functions can only be 0 or 1. By using the relation $f(x) = f(0)(1-x) + f(1)\,x$ for $x \in \{0,1\}$, Eq. (20) is transformed into

$$L_\beta(\tau) = \sum_{\tau'}\left\langle \delta(\tau-\tau')\prod_{t=1}^{\tau'-1}\vartheta(\xi-\eta^t)\vartheta(\eta^{\tau'}-\xi)\right\rangle_{\vec\eta,\xi} + \sum_{\tau'}\left\langle\delta(\tau)\left(1-\prod_{t=1}^{\tau'-1}\vartheta(\xi-\eta^t)\vartheta(\eta^{\tau'}-\xi)\right)\right\rangle_{\vec\eta,\xi}. \tag{C1}$$

The last term of the sum is 0, because τ is always larger than 0. Hence we have to calculate

$$L_\beta(\tau) = \sum_{\tau'}\left\langle \delta(\tau-\tau')\prod_{t=1}^{\tau'-1}\vartheta(\xi-\eta^t)\vartheta(\eta^{\tau'}-\xi)\right\rangle_{\vec\eta,\xi}. \tag{C2}$$

Since the noise $\vec\eta$ is stochastically independent, the average can be easily done:

$$\begin{aligned} L_\beta(\tau) &= \sum_{\tau'}\delta(\tau-\tau')\Big\langle \langle\vartheta(\xi-\eta^1)\rangle_{\eta^1}\langle\vartheta(\xi-\eta^2)\rangle_{\eta^2}\cdots \\ &\quad\times\left\langle\vartheta(\xi-\eta^{\tau'-1})\right\rangle_{\eta^{\tau'-1}}\left\langle\vartheta(\eta^\tau-\xi)\right\rangle_{\eta^\tau}\Big\rangle_\xi \\ &= \sum_{\tau'}\delta(\tau-\tau')\left\langle\left(1-e^{-\beta\xi}\right)^{\tau'-1}e^{-\beta\xi}\right\rangle_\xi \\ &= \int d\xi\left(1-e^{-\beta\xi}\right)^{\tau-1}e^{-\beta\xi} = \frac{\left(1-e^{-\beta}\right)^\tau}{\beta\tau}. \end{aligned} \tag{C3}$$

This is the exact result for only one site. Let us now look at what happens in the case of large N and synchronous updating. From Eq. (19) we know the distribution of x_{update}. It follows from the considerations for the case $N = 1$ that we have to replace the average over $\hat P(\xi) = 1$ by the average over the distribution $p_{\text{update}}(x)$ in Eq. (C3). We get

$$L_\beta(\tau) = \sum_{\tau'}\delta(\tau-\tau')\int d\xi\,p_{\text{update}}(\xi) \times\left(1-e^{-\beta\xi}\right)^{\tau'-1}e^{-\beta\xi}. \tag{C4}$$

For large β's, $p_{\text{update}}(x)$ tends to a rectangle and can be approximated by

$$p_{\text{update}}(\xi) \simeq K\vartheta(1/K-\xi). \tag{C5}$$

We insert this into Eq. (C4) and obtain

$$\begin{aligned} L_\beta(\tau) &= \int_0^1 d\xi\,K\,\vartheta(1/K-\xi)\left(1-e^{-\beta\xi}\right)^{\tau-1}e^{-\beta\xi} \\ &= K\int_0^{1/K} d\xi\left(1-e^{-\beta\xi}\right)^{\tau-1}e^{-\beta\xi} \\ &= K\,\frac{\left(1-e^{-\beta/K}\right)^\tau}{\beta\tau} \simeq \frac{K}{\beta\tau}\,e^{-\tau/\bar\tau}, \end{aligned} \tag{C6}$$

where $\bar\tau = e^{\beta/K}$.

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