Analytical investigation of self-organized criticality in neural networks

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Dynamical criticality has been shown to enhance information processing in dynamical systems, and there is evidence for self-organized criticality in neural networks. A plausible mechanism for such self-organization is activity dependent synaptic plasticity. Here, we model neurons as discrete-state nodes on an adaptive network following stochastic dynamics. At a threshold connectivity, this system undergoes a dynamical phase transition at which persistent activity sets in. In a low dimensional representation of the macroscopic dynamics, this corresponds to a transcritical bifurcation. We show analytically that adding activity dependent rewiring rules, inspired by homeostatic plasticity, leads to the emergence of an attractive steady state at criticality and present numerical evidence for the system's evolution to such a state.

Information processing systems often exhibit optimal computational capabilities when their parameters are tuned to *critical states* associated with phase transitions [1–4]. It therefore appears likely that our brains operate at criticality [5]. Although still hotly debated in neuroscience, the hypothesis of neural criticality is supported by recent experiments. Power-law distributions indicative of critical behavior were observed in slices of rat cortex [6–8] as well as EEG [9–11], fMRI [12], and EcoG [11] measurements in humans.

In the light of the experimental corroboration of neural criticality it is interesting to ask how a biological system can robustly self-tune its parameters to a critical state. A likely answer is found in the study of adaptive networks [13, 14], a class of models in which the dynamics on a network coevolves with the network structure. Already in 1998, it was noted that adaptive networks with slowly evolving topology can self-organize to a state where the dynamics on the network are critical [15]. Adaptive self-organized criticality (aSOC) was subsequently demonstrated conclusively in a simple boolean network model [16] and then studied in detail in neural models [17–23].

We note that insights in aSOC may not only advance our understanding of natural neural networks, but may reveal an important design principle for electronic computers: Within the next ten years, current manufacturing processes will hit fundamental boundaries [24]. Continued progress will require utilizing nanoscale components (e.g. nanotubes, nanowires, biomolecules) that cannot be positioned precisely with present-day techniques for manufacturing large-scale integrated systems. It is thus conceivable that future computers may consist of active (nano-)elements that are deposited randomly and then left to self-tune to a critical state, where meaningful information processing is possible. A promising mechanism for such self-tuning is provided by aSOC. Importantly, this mechanism does not require the rewiring of physical interconnections, but can be achieved already by local changes of conductivity between elements [22] that have recently been demonstrated experimentally [25].

Despite the prominent role of aSOC for information processing systems in both biology and technology, our understanding of the phenomenon is still limited. In many aSOC models, the critical state is identified by showing that certain quantities follow power-law distributions. However, power laws can appear due to other mechanisms besides criticality. It is thus desirable to make the corresponding dynamical phase transition directly accessible to analytical investigation.

The aim of the present paper is to develop a better understanding into aSOC by means of a simple conceptual model. We start in Sec. I with a brief review of the biological background. In Sec. II, introduce a simple neuron model. In Sec. III, we use a moment closure approximation [26] for deriving a low-dimensional system of ordinary differential equations (ODEs) that capture the emergent dynamics. By analyzing the bifurcation structure of these ODEs, we show in Sec. IV that the model exhibits a non-equilibrium phase-transition. In Sec. V, we discuss the conditions under which the chosen topological update rules drive the system toward the phase transition. Finally, in Sec. VI we show analytically and numerically that the model indeed exhibits aSOC.

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I. BIOLOGICAL BACKGROUND

In biology, transmission of information between neurons occurs via cell contacts known as synapses. Up to the order of 10^5 such connections (with different partners) can exist on a single neuron. Synapses allow a pre-synaptic neuron to depolarize a post-synaptic neuron, similar to polar devices that transmit current only in one direction. The topology of interconnections can thus be captured by a directed network, in which the nodes correspond to neurons and the directed links correspond to synapses.

On a short timescale, the neurons encode information in an electrical potential across their cell membrane. In the absence of inputs a neuron approaches a *resting state* with a characteristic membrane voltage. Depolarization of the membrane due to input from other neurons can lead to a *firing state* in which active mechanisms are used to emit a strong voltage pulse, which in turn excites connected neurons. After firing, the neuron enters a *refractory state* in which no excitation is possible before it finally returns to the resting state.

On a longer timescale, the strength of synapses changes depending on the activity of the connected neurons. Thus, from an engineering perspective the synapse is a memristive element [27]. In biological terms the processes affecting synaptic strength are collectively known as synaptic plasticity. Here, we specifically consider *homeostatic synaptic plasticity* (HSP) [28, 29], which decreases the strength of synapses if the activity of a neuron is high, and increases the strength if the activity is low.

II. DISCRETE NEURAL MODEL

In the present paper we consider a directed network of N nodes. At any time a given node is in one of three different states: resting (inactive, I), firing (F), or refractory (R). We start from a random network in which a majority of the nodes are in the inactive state, whereas a small fraction is in the firing state. The node states are then evolved according to the following rules: Firing nodes become refractory at a rate i, refractory nodes become inactive at rate r, and, for every link pointing form a firing neuron to an inactive neuron, the target neuron is set to the firing state at rate p.

The network topology is evolved according to a rule modeling synaptic plasticity: A firing neuron looses an incoming link at the rate l whereas new links are established between neurons at the rate g. The creation and deletion of links is reminiscent the formation of synaptic contacts in the developing brain, but is used here as a discretized model of the continuous changes of synaptic weight in the adult brain. By formulating the model in terms of discrete linking and unlinking events we avoid additional complications caused by real-valued link dynamics.

III. LOW-DIMENSIONAL APPROXIMATION

Let us now derive a low-dimensional model that describes the time evolution of macroscopic quantities in the limit of infinite N. We denote the densities of nodes in a certain state by $[F], [I], [R]$, respectively. Similarly, we denote the per-neuron density of links from a node in state X to a node in state Y by $[XY]$. By *moment closure approximation* (MCA)[26, 30] on the link level, we obtain the following ODE description of the system:

$$[\dot{F}] = p[FI] - i[F] \tag{1a}$$

$$[\dot{R}] = i[F] - r[R] \tag{1b}$$

$$[\dot{FF}] = -2i[FF] + p\left(\tfrac{1}{2}\tfrac{[FI]^2}{[I]} + [FI]\right) - l[FF] + g[F][F] \tag{1c}$$

$$[\dot{FI}] = -i[FI] + p\left(\tfrac{[II][FI]}{[I]} - \tfrac{1}{2}\tfrac{[FI]^2}{[I]} - [FI]\right) + r[FR] + g[F][I] \tag{1d}$$

$$[\dot{FR}] = i\left([FF] - [FR]\right) + p\tfrac{[IR][FI]}{[I]} - r[FR] + g[F][R] \tag{1e}$$

$$[\dot{II}] = -p\left(1+\kappa\right)\tfrac{[II][FI]}{[I]} + r\left([RI] + [IR]\right) + g[I][I] \tag{1f}$$

$$[\dot{IR}] = i[IF] - p\tfrac{[IR][FI]}{[I]} + r\left([RR] - [IR]\right) + g[I][R] \tag{1g}$$

$$[\dot{RF}] = i\left([FF] - [RF]\right) + p\tfrac{[RI][FI]}{[I]} - r[RF] - l[RF] + g[R][F] \tag{1h}$$

$$[\dot{RI}] = i[FI] - p\tfrac{[RI][FI]}{[I]} + r([RR] - [RI]) + g[R][I] \tag{1i}$$

$$[\dot{RR}] = i\left([FR] + [RF]\right) - 2r[RR] + g[R][R] \tag{1j}$$

$$\dot{k} = g - l[F] \tag{1k}$$

where the last equation explicitly captures the change of the mean degree k of the network. Writing the the equation for k saves us from writing the longer equation for IF, due to

$$[IF] = k - [FF] - [FI] - [FR] - [II] - [IR] \tag{2}$$
$$- [RF] - [RI] - [RR]\ . \tag{3}$$

Similarly $[I]$ follows from the conservation relation for nodes

$$[I] = 1 - [F] - [R]\ . \tag{4}$$

IV. NON-EQUILIBRIUM PHASE TRANSITION

Before we address the dynamics of the full system, let us first focus on the case $l = g = 0$, where the network is static. In this case, the right hand side of the equation of motion for k vanishes such that k becomes a control parameter. Below, we will refer to the system without topological evolution as the *static network model* and to the complete system as the *adaptive network model*.

The static network has a trivial steady state $\mathbf{x}^0 = ([F]^0, \cdots, [RR]^0)$ in which all nodes are inactive, i.e $[I]^0 = 1, [II]^0 = k, [F]^0 = [R]^0 = [FF]^0 = \cdots = [RR]^0 = 0$. Depending on parameters, it may moreover have a

non-trivial, active steady state, in which $[I] < 1$. The stability of the trivial steady state is determined by the spectrum of the Jacobian matrix $\mathbf{J}|_{\mathbf{x}^0} \in \mathbb{R}^{10\times 10}$, where $J_{ij} = \partial \dot{x}_i / \partial x_j$. The steady state is asymptotically stable if all eigenvalues of $\mathbf{J}|_{\mathbf{x}^0}$ have a negative real part [31].

If the variables x_i are ordered as in Eqns. (1), the non-vanishing entries of $\mathbf{J}|_{\mathbf{x}^0}$ are

$$\begin{aligned}
J_{1,1} &= J_{7,3} = J_{7,4} = J_{7,5} = J_{7,6} = J_{7,8} = J_{7,9} = -i \ , \\
J_{1,4} &= J_{3,4} = p \ , \\
J_{2,1} &= J_{5,3} = J_{8,3} = J_{9,4} = J_{10,5} = J_{10,8} = i \ , \\
J_{2,2} &= J_{4,5} = J_{6,7} = J_{6,9} = J_{9,10} = r \ , \\
J_{3,3} &= -2i \ , \\
J_{4,4} &= -i + (k-1)p \ , \\
J_{5,5} &= J_{7,7} = J_{8,8} = -i - r \ , \\
J_{6,4} &= -2kp \ , \\
J_{7,10} &= -i + r \ , \\
J_{9,9} &= -r \ , \\
J_{10,10} &= -2r \ .
\end{aligned}$$

The characteristic polynomial can be factored into 7 linear factors with negative real roots and a remaining third order polynomial

$$P(\lambda) = (-r-\lambda)^3(-i-\lambda)^2(-i-r-\lambda)(-2r-\lambda)f(\lambda) \ , \quad (5)$$

where

$$\begin{aligned}
f(\lambda) = i\left(i^2 + 2i\left((1-k)p + r\right) + (1-2k)pr\right) \\
+ \left(5i^2 + (1-k)pr + 3i\left((1-kp) + r\right)\right)\lambda \\
+ \left(4i + (1-k)p + r\right)\lambda^2 + \lambda^3 \ .
\end{aligned} \quad (6)$$

In other to assess the stability of $\mathbf{x}^0$ without explicitly calculating the roots of $f(\lambda)$, we use the Routh-Hurwitz theorem [32]. It states that the roots of a polynomial $p(x) = b^n + a_1 x^{n-1} + \cdots + b_{n-1}x + b_n$ all have negative real parts if the *Hurwitz determinants*

$$\Delta_k = \begin{vmatrix} b_1 & 1 & 0 & 0 & \dots & 0 \\ b_3 & b_2 & b_1 & 1 & \dots & 0 \\ \vdots & \vdots & \vdots & \vdots & \ddots & \vdots \\ b_{2k-1} & b_{2k-2} & b_{2k-3} & b_{2k-4} & \dots & b_k \end{vmatrix}, \quad (7)$$

with $k = 1 \dots n$, are all positive. Calculating the Hurwitz determinants of the Jacobian matrix $\mathbf{J}|_{\mathbf{x}^0}$ and evaluating the positivity conditions reveals that the trivial steady state is stable if and only if

$$k < \frac{i}{p} + \frac{i + r/2}{i + r} =: k_c. \quad (8)$$

At $k = k_c$ the trivial steady state becomes unstable as the systems undergoes a transcritical bifurcation. In this bifurcation a nontrivial steady state enters the positive cone of the state space, becoming a physical solution. The transcritical bifurcation thus marks a transition between the trivial inactive state and an active state in which ongoing activity is observed.

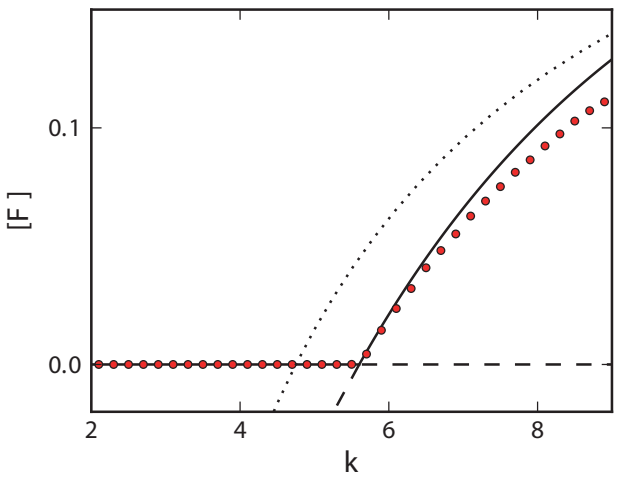

FIG. 1. Bifurcation diagram for the static network. Plotted is the steady state density of firing neurons $[F]$ over the network's mean degree k. The solid line marks stable steady states of the static system, the dashed line unstable ones. At $k = k_c \approx 5.6$, the inactive steady state loses stability in a transcritical bifurcation. The respective transition from an inactive to an active phase is already observed in individual-based simulations with $N = 10^6$ neurons (circles). Note that the critical k is nicely predicted by the link-level approximation Eqns. (1), but underestimated by a MCA at mean-field level (dotted lines). Parameters used were $p = 0.2, i = 0.95, r = 0.4$. Nontrivial steady states were calculated using `AUTO`[33].

The role of the transcritical bifurcation is illustrated in a representative bifurcation diagram in Fig. 1. The diagram shows that the analytically predicted bifurcation point is in very good agreement with numerical results from agent-based simulations of the system. Further, numerical continuation of the ODEs (1) shows good agreement close to the bifurcation point. By contrast, a MCA at mean-field level yields a much poorer approximation and underestimates the bifurcation point.

V. LOCAL INFORMATION AND TIME-SCALE SEPARATION

The previous section showed that the static network model exhibits a transcritical bifurcation. In the language of statistical physics this bifurcation constitutes a phase transition. To establish that the adaptive network model shows self-organized criticality we have to show that the evolution of the connectivity drives the system to the critical point.

In the discussion leading up to Fig. 1 we treated k as a parameter of the system. For studying the evolution of the connectivity we now consider k as a dynamical variable that evolves according to Eq. (1k). By introducing dynamics in k we change the dynamical system, which can potentially lead to a changed bifurcation diagram.

Indeed, in general a diagram analogous to Fig. 1 cannot be drawn for the adaptive network model because k is not available as a parameter axis anymore.

The pitfall described above is inherent in the concept of SOC: Criticality can only be cleanly defined in a different system, where the self-organization is absent. To circumvent this pitfall one has to demand that the self-organization acts on a slower timescale. In the example studied here this implies,

$$g, l \ll p, i, r \ . \tag{9}$$

In this case, the bifurcation diagram of the static network model reappears as the bifurcation diagram of the fast-subsystem of the adaptive network model, where k can again be treated as a parameter.

The genesis of aSOC in our model requires additionally a second time scale separation. To see this let us us revisited a widely held plausibility argument for aSOC[13, 18]. It is argued that in adaptive networks robust self-organized criticality is possible because the dynamics on the networks make information on the global topology available in every node. Based on this information the nodes can then infer the global phase and adjust the local topology accordingly. Indeed, in previous publications[16, 18] the networks nodes extracted the global phase information by integrating their dynamics over a long time.

In our model, the local accessibility of the global phase information is limited, as both the dynamics on the static network as well as the topological updates are memoryless processes that depend only on the current state of the system. Hence, neurons in the firing state can infer the global phase from their local state, as their occurrence is restricted to the active phase. By contrast, neurons in the resting state do not possess any information about the global phase, as they occur in both phases. To achieve aSOC despite the limited local accessibility of the global phase, links have to be created *tentatively* as long as no definitive information is known, but destroyed *decisively* once information about the global phase is available. This corresponds to a separation of timescale between the link creation and link deletion process

$$\epsilon := \frac{g}{l} \ll 1 \ . \tag{10}$$

Note, that the twofold separation of time scales constituted by Eq. (9) and Eq. (10) is typical for SOC. However, as remarked in [34, 35], it is often hidden in a nonlocal model definition.

VI. SELF-ORGANIZED CRITICALITY

Let us now show that the adaptive network model has a stable steady state, which in the double limit $l \to 0$, $\epsilon \to 0$ coincides with the bifurcation of the static network model. We start by defining $\mathbf{x}^* = ([F]^*, \cdots, [RR]^*, k^*)$ as a steady state of the adaptive network model (1). Evaluating the stationarity conditions $\dot{k}|_{\mathbf{x}^*} = 0, [\dot{F}]|_{\mathbf{x}^*} = 0$ and $[\dot{R}]|_{\mathbf{x}^*} = 0$, we can immediately read off the steady state values

$$[F]^* = \epsilon, \quad [R]^* = \epsilon i/r, \quad [FI]^* = \epsilon i/p \ . \tag{11}$$

Therewith, the remaining equations ($[\dot{FF}]|_{\mathbf{x}^*} = 0, \cdots, [\dot{RR}]|_{\mathbf{x}^*} = 0$) become linear and can be solved by a computer algebra system. Due to the time-scale separation, higher-order terms in ϵ and l can be dropped, and we obtain

$$[FF]^* = \frac{1}{2}\epsilon \tag{12a}$$

$$[FR]^* = [RF]^* = \frac{1}{2}\frac{i}{i+r}\epsilon \tag{12b}$$

$$[II]^* = k_\mathrm{c} + \frac{r}{4i(i+r)}l - \frac{i}{i+r}\left(k_\mathrm{c} + \frac{1}{2}\right)\epsilon \tag{12c}$$

$$[IR]^* = \frac{i}{r}\left(k_\mathrm{c} + \frac{1}{2}\right)\epsilon \tag{12d}$$

$$[RI]^* = \frac{i}{r}\left(k_\mathrm{c} - \frac{1}{2}\right)\epsilon \tag{12e}$$

$$[RR]^* = \frac{1}{2}\frac{i}{r}\frac{i}{i+r}\epsilon \tag{12f}$$

$$\begin{aligned} k^* = {} & k_\mathrm{c} + \frac{r}{4i(i+r)}l \\ & + \left(\frac{i+r}{r}\left(\frac{1}{2} + 2k_\mathrm{c}\right) - \frac{i}{i+r}\left(1 + k_\mathrm{c}\right)\right)\epsilon \ . \end{aligned} \tag{12g}$$

For $\epsilon, l \ll 1$, the steady state $\mathbf{x}^*$ is always stable, which can be shown by calculating the characteristic polynomial of $\mathbf{J}|_{\mathbf{x}^*}$ and then checking the Hurwitz determinants for positivity (again, dropping higher-order terms in ϵ and l). Moreover, the Eqns. (12) reveal that in the double limit $l \to 0, \epsilon \to 0$, $[II]^* \to k_\mathrm{c}$, while $[F]^*, \ldots, [RR]^* \to 0$, i.e., $\mathbf{x}^*$ converges towards the transcritical bifurcation of the static network model. We thus conclude that for $N \to \infty$ and $l \to 0, \epsilon \to 0$, the HSP-inspired update rule self-organizes the adaptive system to criticality.

To confirm aSOC in the discussed model, we ran individual-based simulations. We start with a network of N nodes, each of which has an outgoing connection to any other node with probability k_0/N. We initialize 5% of all nodes in the firing state, all others in the inactive state. Then, nodes and links are evolved according to the rules described in Sec. II using the Gillespie algorithm[36].

For finite N, the simulations tend toward the absorbing inactive state due to demographic stochasticity. To compensate for the finite size effect, we include an additional process: Inactive nodes fire spontaneously with probability s. This process has an immediate biological interpretation as it reminds of the spontaneous activity observed in neurons. To reconcile the simulations with the low-dimensional description (1), s has to vanish in the $N \to \infty$ limit. A plausible assumption is $s = c/N$,

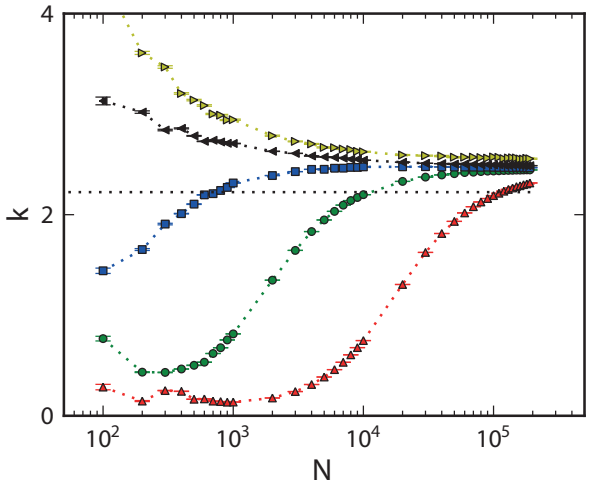

FIG. 2. Mean degree k of evolved networks for different network sizes N and different spontaneous firing rates $s = 1/(1000N)$ (yellow triangles pointing right), $s = 1/(100N)$ (black triangles pointing left), $s = 1/(10N)$ (blue squares), $s = 1/N$ (green circles), $s = 10/N$ (red triangles pointing up). The dotted line marks the analytical steady state value for $l = 0.01, \epsilon = 10^{-4}$. Simulations were run for 10^7 time units, plotted connectivities are averages over the last $5 \cdot 10^6$ time units. Other parameters: $p = 0.7, i = 0.95, r = 0.4$.

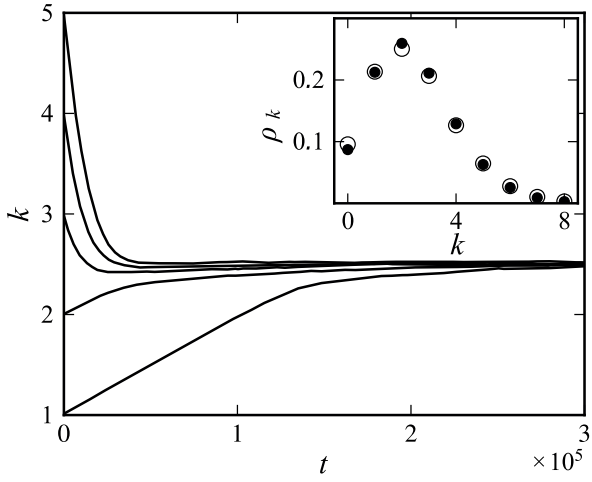

FIG. 3. Time evolution of the mean degree of networks ($N = 10000, l = 10^{-3}, \epsilon = 0.01, s = 10^{-4}$), starting from different initial connectivities. Inset: In-degree distribution of an evolved network after $5 \cdot 10^6$ time steps ($N = 10^5, l = 10^{-3}, \epsilon = 10^{-3}, s = 10^{-5}$, open circles) and Poissonian distribution around the same mean (filled circles). Other parameters: $p = 0.7, i = 0.95, r = 0.4$ in both cases.

where c can be chosen arbitrarily. We have verified that for sufficiently large system sizes the particular choice of c does not significantly influence the evolving connectivity (cf. Fig. 2).

We start in Fig. 3 by plotting the time evolution of the mean degree k in networks with different initial configurations. All networks approach the same connectivity, which corroborates that the adaptive network model has exactly one stable steady state to which it converges irrespective of initial conditions.

The inset of Fig. 3 shows the in-degree distribution of an exemplary evolved network. The degree distribution is of interest as it affects the validity of the MCA approach and therefore the validity of out analytical treat-

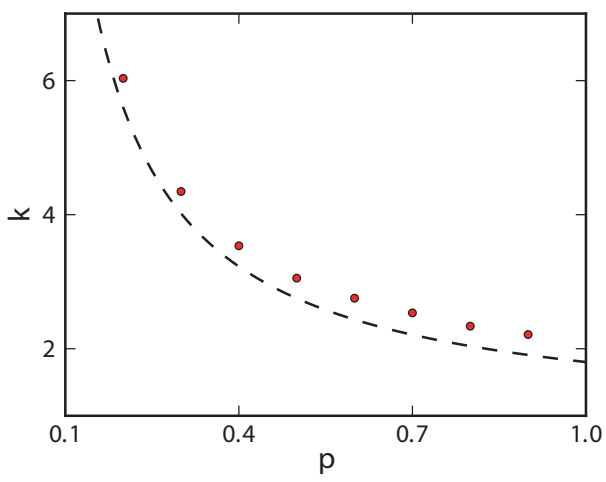

FIG. 4. Mean degree k of evolved networks for different values of p. The dashed line marks the analytically obtained critical connectivity k_c. Simulations were run for 10^8 time units. Parameters: $N = 10^4, l = 10^{-6}, \epsilon = 0.0015, s = 10^{-7}, i = 0.95, r = 0.4$

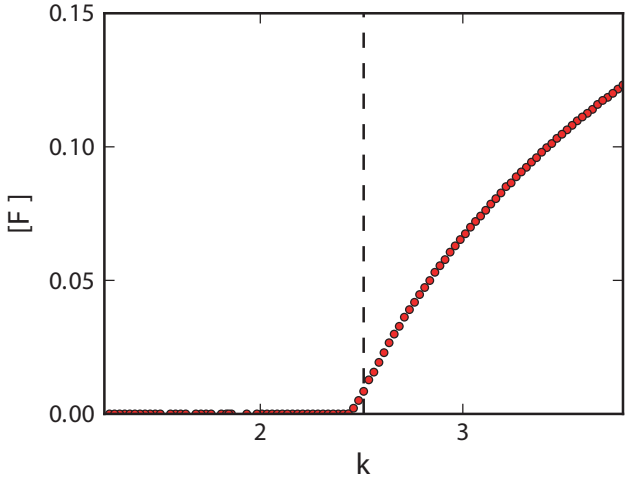

FIG. 5. Average density of firing neurons (after transients) around the evolved connectivity (marked by vertical dashed line). Values above or below the evolved connectivity were obtained by adding (removing) random links to (from) the evolved topology. The network was evolved over $5 \cdot 10^6$ time units. Parameters: $N = 10^5, p = 0.7, i = 0.95, r = 0.4, l = 0.01, \epsilon = 10^{-4}, s = 1/(100N)$.

The inset of Fig. 3 shows the in-degree distribution of an exemplary evolved network. The degree distribution is of interest as it affects the validity of the MCA approach and therefore the validity of out analytical treat-

ment. The approximation performs poorly for networks with long-tailed degree distribution, such as scale-free networks. But is known to yield good results for Poissonian distributions. Thus, the observed degree distributions retrospectively justify our analytical treatment.

So far we have shown that the network self-organizes to a unique value of the connectivity. It remains to test whether this value is indeed the critical connectivity. As a first test, we compare the connectivity of the networks evolved in numerical simulations with the critical connectivity k_c predicted by the analytical approximation from Eq. (8). As shown in Fig. 4 the numerical result agrees with the analytical estimate of the critical state except for a small but systematic deviation. This discrepancy can be understood considering Eq. (12g). The second and third term of this equation are positive for all $i, r, k_c, l, \epsilon > 0$. Hence, the systematic deviation $k^* > k_c$ is consequence of the networks' topologies being evolved with small but finite rates ϵ, l.

As a second test, we directly probe the dynamics on the evolved networks. For this purpose, we first evolve the topology until the mean degree has reached a stationary state. Then, we deactivate the adaptive addition and deletion of links but manually add (delete) links from the network. After every addition (deletion) we let the dynamics on the network a stationary level and record the average number of active neurons. As shown in Fig. 5, this procedure recreates the phase diagram of the system numerically. It thereby provides direct evidence of the criticality of the evolved state. The slight displacement from the critical point can be understood recalling the analytical results given above: According to Eq. (11), $[F]^* = \epsilon$ in the evolved network. Hence, the displacement toward the active regime can be attributed to the small but finite rates ϵ, l used in the simulations.

VII. CONCLUSIONS

In summary, we have shown that activity-dependent synaptic plasticity self-organizes a neural network to criticality, provided that driving is slow and there is an appropriate separation of timescales between potentiation and depression.

We have considered a simple discrete neural network model and used moment-closure approximation to derive a low-dimensional ODE representation, in which the dynamical phase transition becomes manifest as a transcritical bifurcation. By adding activity dependent rewiring rules we obtained an adaptive network model that has one attractive steady state. We have shown that, in the limit of infinitesimally slow topological adaptation, this steady state coincides with the bifurcation of the static network model, and, thus, that the adaptive model displays aSOC.

Our work provides a conceptual angle on self-organized criticality of neurally inspired models. Although the phenomenon has been demonstrated in several earlier works [19, 37], the simplified model studied here is the first system in which it is analytically tractable. Using dynamical systems and bifurcation theory, aSOC can be established more rigorously than in more realistic models and experiments, where evidence for criticality is mainly provided by the numerical observation of power laws. We believe that this approach will prove useful in the context of other, more complex dynamical phase transitions. For example, it is conceivable that a similar strategy may help to elucidate the recently observed self-organization to the onset of synchronous activity[22]. We hope that the approach can thus contribute to settling an ongoing debate regarding the existence and function of criticality in the brain [5] and potentially contribute to the development of self-organizing electronic circuits.

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