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Self-organized criticality and scale-free properties in emergent functional neural networks

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Recent studies on complex systems have shown that the synchronization of oscillators, including neuronal ones, is faster, stronger, and more efficient in small-world networks than in regular or random networks. We show that the functional structures in the brain can be self-organized to both small-world and scale-free networks by synaptic reorganization via spike timing dependent synaptic plasticity instead of conventional Hebbian learning rules. We show that the balance between the excitatory and the inhibitory synaptic inputs is critical in the formation of the functional structure, which is found to lie in a self-organized critical state.

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The brain is one of the most challenging complex systems. Massively interconnected to one another, the neurons respond to external stimuli in a correlated and highly complex manner in order to process information. Understanding the neural complexity requires understanding the network structure on which the functional self-organization of neural firing activities are supported.

Recent studies on diverse complex networks describe real networks by simply defining a set of nodes and connections between them. Examples range over social, information, technological, and biological networks [1]. They lie between regular and fully random networks. A wide variety of such systems are scale-free with a power-law connectivity distribution, and their topology and evolution are governed by a common mechanism such as preferential attachment and growth [1–4].

The topological structure of simple nervous systems, for example, in *Caenorhabditis elegans* has been proven to be an inhomogeneous small-world network [5]. However, in the brain, the functional structure is more important as the direct carrier of the neuronal information in the form of spikes and synaptic conductances. Moreover, it changes adaptively due to the inputs from external stimuli and the internal dynamics of neurons, which in turn affect the neural responses. This feedback process of synaptic plasticity is believed to be closely linked to the mechanisms for learning and memory in the brain. Recently, spike timing dependent plasticity (STDP) has been observed experimentally in various brain regions, such as neocortical slices [6], hippocampal slices [7] and cell cultures [8], and the ELL (electrosensory lateral line lobe) of the electric fish [9]. That is, long term synaptic modifications arise from repeated pairings of presynaptic and postsynaptic action potentials, the sign and the degree of which depend on their relative timing. For example, in the hippocampal CA3 region and neocortical slices, STDP strengthens a synapse if the presynaptic spike is followed by postsynaptic action potentials within about 50 ms and weakens it if the presynaptic action potential follows postsynaptic spikes.

In this paper, we report that STDP can reorganize a globally connected neural network spontaneously into a functional network, which is both small-world and scale-free. This functional structure is formed by the activity dependent synaptic plasticity depending on the spatiotemporal dynamics of the neurons rather than a commonly used explicit preferential attachment rule. We find that the functional complex network arises when the excitatory and inhibitory connection strengths between neurons are balanced. The neuronal activities in this small-world, scale-free neural functional network are found to lie in a self-organized critical state as in the case of sandpile and forest fire models [10]. The neuronal oscillators in the functionally organized structure show fast synchronous responses to external stimuli, making the neural network more reliable in information transformation and stable from epileptic overexcitation. Our results are quite robust and general, and hold for a wide class of neuron models, including the Hodgkin-Huxley (HH) model, in a wide range of control parameters, such as the strength of the external stimulus, and parameters related to STDP, independent of initial conditions.

The model neuron used is the FitzHugh-Nagumo (FHN) model [11], which is a two dimensional relaxation oscillator with two time scales but contains the essential ingredients of nervous excitation and fast action potential generation followed by a slow refractory period

$$\epsilon \dot{v} = I_{\text{ion}} + I_{\text{syn}} + I_{\text{ext}},$$

$$\dot{w} = v - w - b,$$

$$I_{\text{ion}} = v(v-a)(1-v) - w, \tag{1}$$

where, with $\epsilon \ll 1$, v is a fast voltagelike variable, w a slow recovery variable, I_{ion} the ionic current through the membrane with cubic nonlinearity, and I_{ext} the external current stimulus. Neurons are coupled through synapses, and, if a presynaptic neuron makes an action potential, it generates synaptic currents in the postsynaptic neurons. The synaptic current input to the ith neuron is the sum of excitatory and inhibitory currents from pre-synaptic neurons

$$I_{\text{syn}}(t) = \sum_{j \neq i} \{ g_{ij}(t)[V - v_i(t)] + \bar{g}_{ij}(t)[\bar{V} - v_i(t)] \}, \tag{2}$$

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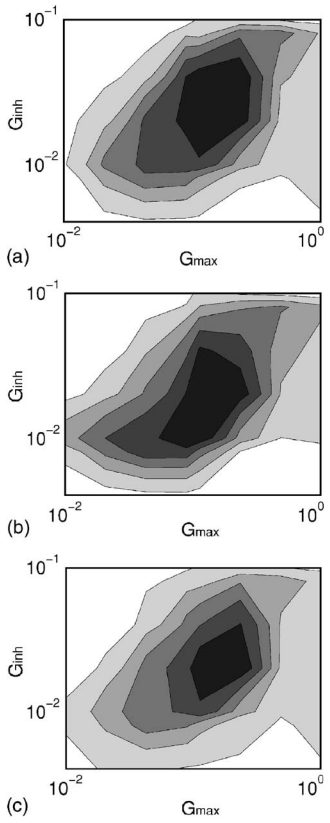

FIG. 1. (a) Connection probability of the STDP network $\langle k/N \rangle$ in the range between 0.03 (dark) and 0.6 (light), (b) phase coherence Γ between 0.4 (dark) and 1.0 (light), and (c) the ratio of the clustering coefficients between our model and the random network with the same connection probability C/C_{rand} between 0.5 (light) and 7.5 (dark) in the parameter space of $G_{\max}$ and G_{inh} in log-log scale. The results are averaged over 30 different initial conditions.

where g_{ij} ($\bar{g}_{ij}$) is the excitatory (inhibitory) synaptic conductance from the jth neuron to the ith neuron and V ($\bar{V}$) the excitatory (inhibitory) synaptic reversal potential, respectively. The synaptic conductances decay exponentially in the absence of synaptic stimulus from presynaptic neuron

$$\tau_{\mathrm{syn}} \frac{dg_{ij}}{dt} = -g_{ij} \quad \text{and} \quad \bar{\tau}_{\mathrm{syn}} \frac{d\bar{g}_{ij}}{dt} = -\bar{g}_{ij}. \tag{3}$$

If the jth presynaptic neuron makes an action potential at time t^*, it increases the post-synaptic conductances by the amount of the peak conductances at time t^* normalized by the number of neurons, $g_{ij} \to g_{ij} + G_{ij}(t^*)/(N-1)$ and $\bar{g}_{ij} \to \bar{g}_{ij} + \bar{G}_{ij}(t^*)/(N-1)$. $G_{ij}(t)$ $[\bar{G}_{ij}(t)]$ is the maximal excitatory (inhibitory) conductance from the jth to the ith neuron generated by one action potential at time t, which can be regarded as the synaptic coupling strength between two neurons and modified by STDP.

In our STDP neural network model, we assume that inhibitory synaptic coupling strengths remain constant [8], $\bar{G}_{ij}(t) = G_{\mathrm{inh}}$, while excitatory synaptic strengths change multiplicatively at every firing event [12–14]:

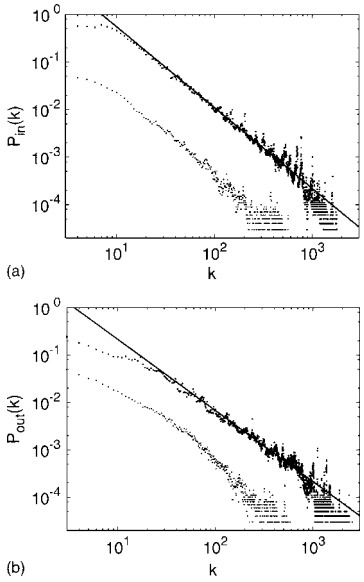

FIG. 2. Log-log plots of degree distributions of the functional STDP network for the optimal synaptic couplings. (a) In-degree probability distribution and (b) out-degree probability distribution for $N=1000$ and $N=10\,000$. The scaling exponents are $\gamma_{\mathrm{in}} \approx 1.7$ and $\gamma_{\mathrm{out}} \approx 1.5$ in the intermediate k. The results are averaged over 100(40) runs with different initial conditions for $N=1000$ (10 000).

$$\Delta G_{ij} = G_{ij} W(\Delta t). \tag{4}$$

The amount of the synaptic modification by STDP depending on the time difference between presynaptic and postsynaptic spikes, $\Delta t = t_{\mathrm{post}} - t_{\mathrm{pre}}$, is modeled by the STDP modification function

$$W(\Delta t) = \begin{cases} A_+ \exp(-\Delta t/\tau_+) & \text{if } \Delta t > 0, \\ -A_- \exp(\Delta t/\tau_-) & \text{if } \Delta t < 0 \end{cases} \tag{5}$$

and $W(\Delta t = 0) = 0$. The parameters $\tau_\pm$ determine the temporal window of the spike intervals, and $A_\pm$ determine the maximum amount of synaptic modification. It has been shown experimentally that in most situations, $A_+ > A_-$, $\tau_+ < \tau_-$, and the integral of the function W is usually negative [14]. Here, the parameter values are chosen to be $A_+ = 0.01$, $A_- = 0.006$, $\tau_+ = 1.0$, and $\tau_- = 2.0$. It is assumed that $0 < G_{ij} \leq G_{\max}$ and if G_{ij} increases over the maximal value, G_{ij} is set to $G_{\max}$. Other parameters are set to $a=0.5$, $b=0.12$, $\epsilon=0.005$, $V=0.7$, $\bar{V}=0.0$, and $\tau_{\mathrm{syn}} = \bar{\tau}_{\mathrm{syn}} = 0.2$.

We start with a globally coupled network with 1000 neurons and random initial coupling strengths, $0 < G_{ij} \leq G_{\max}$, and investigate how the functional structure develops spontaneously in time. We apply external dc current, $I_{\mathrm{ext}} = 0.2$, which is suprathreshold stimulus for spontaneous generation of action potentials. After a period of relaxation by STDP, some population of synapses are strengthened to the maximum conductance $G_{\max}$ while the others are weakened to near zero. This is similar to the bimodal distribution in the case of the balanced excitation of synapses from many inputs to a single neuron [15]. As a result, even if the neurons are morphologically connected all-to-all by synapses, their func-

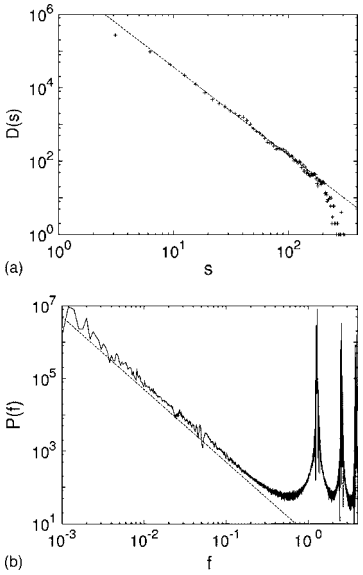

FIG. 3. (a) Distribution of the size of the change of the total synaptic coupling strengths and (b) the power spectrum of the fluctuation of the synaptic strengths in log-log scale showing power-law decays with exponents $\gamma_s \approx 2.5$ and $\gamma_f \approx 2.0$.

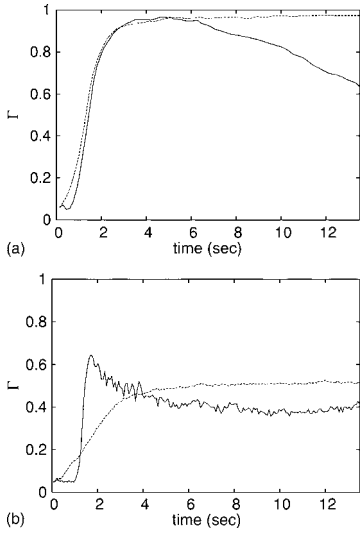

FIG. 4. Phase coherence Γ of the STDP network (solid lines) and the random networks (dashed lines) when a suprathreshold external stimulus is subjected to all the neurons (a) and only half of neurons (b), with a random initial phase of the neurons.

tional structure can be reorganized by STDP to a smaller functional network involving only a small population of neurons.

Each synapse is regarded as functionally connected if the synaptic conductance is larger than a critical value $G_{ij} > G_c$. In Fig. 1(a), the average connection probability, the ratio of the number of strengthened synapses to the total number of synapses, $\langle k/N \rangle$, is shown in the parameter space of $G_{\max}$ and G_{inh}. In this figure, there exists a region along the diagonal, where the connection probability is very small, and on either side of this region the connection probability becomes relatively large. The states of the STDP networks can be classified as synchronized, clustering, and dispersed states. To characterize the dynamical properties of the functional network, we define the phase of a neuron at time t between firing times piecewise linearly [16]:

$$\phi(t) = 2\pi\left(\frac{t - t_n^*}{t_{n+1}^* - t_n^*} + n\right), \tag{6}$$

where t_n^* is the nth firing time. The phase coherence Γ of neurons is defined as

$$\Gamma = \max\left\{\left|\lim_{T\to\infty}\frac{1}{T}\int_0^T \frac{1}{N-1}\sum_{j\neq k}^{N} e^{im\Delta_{jk}(t)}dt\right|\right\}_m, \tag{7}$$

where $\Delta_{jk}(t)$ is the difference between the instantaneous phases of the jth and kth neurons at time t. Note that Γ saturates to 1 if the firing times of all neurons are coherent with m clusters and 0 if they are random. The dependence of the phase coherence Γ in Fig. 1(b) is similar to that of the average connection probability in Fig. 1(a). On the lower side of the diagonal, where the excitatory input becomes more dominant, all the neurons in the network fire synchronously. On the other hand, when the inhibitory input dominates the excitatory input, the clustering state is formed, in which the neurons are decomposed into synchronized groups of neurons that fire asynchronously. In the diagonal region, the excitatory and inhibitory inputs are balanced, and the firing pattern of the network widely is dispersed but not entirely random.

To characterize the structural properties of the functional STDP network, we calculate the clustering coefficient C the fraction of connections that actually exist between neighbors of each neurons with respect to all allowed connections, and the average path length L the number of synaptic connections in the shortest path between two neurons averaged over all pairs of neurons. The phase diagram of the clustering coefficient relative to that of the random network with the same connection probability C/C_{rand} in Fig. 1(c) is similar to those for the average connection probability and the phase coherence. In the middle of the diagonal region, the connection probability is very small with $\langle k/N \rangle \approx 0.03$, but there is one spanning cluster involving almost all the neurons in the network. The clustering coefficient for the functional STDP network is large ($C \approx 0.23$), whereas for a random network $C_{\text{rand}} \approx 0.03$. In this region, the average path length is $L \approx 3.19$, while for a random network, $L_{\text{rand}} \approx 2.03$. These results show that the functional structure organized by STDP in the case of balanced excitations has typical small-world characteristics: its clustering coefficient is much larger than that of the random network with the same connection probability $C \gg C_{\text{rand}}$ and the average path length is similar to that of the random network $L \sim L_{\text{rand}}$.

We also find that the degree distributions of the functional STDP network are scale-free. Figure 2 shows that the degree distributions for optimal synaptic coupling follow power-law decays with exponentially decaying cutoffs for large k: $P_{\text{in}}(k) \sim k^{-\gamma_{\text{in}}}$, and $P_{\text{out}}(k) \sim k^{-\gamma_{\text{out}}}$, where P_{in} and P_{out} are the frequency distributions of nodes with the same number of in-coming and out-going synaptic connections, respectively.

In the middle of the diagonal region in Fig. 1, the scaling exponents are $\gamma_{\text{in}} \approx 1.7$ and $\gamma_{\text{out}} \approx 1.5$, and do not depend much on the synaptic parameters $G_{\max}$ and G_{inh} nor the size of the network N, while the size of the scaling regions is reduced when the synaptic parameters move away from the optimal value. By STDP, due to the negative integral of the STDP modification function (5), the synapses between incoherently firing neurons decays, whereas in a group of coherently spiking neurons, the synapses from neurons firing in advance to the postsynaptic neurons can be strengthened, and the synapses between the two neurons and their neighbors also tend to be strengthened. In this synaptic reorganization process, the more connections a neuron has, the higher probability of making new connections it has. This is effectively the preferential attachment rule for our dynamical network.

After a period of relaxation, the system reaches a quasisteady state and the average global properties of the functional STDP network, such as the clustering coefficient, the average path length, and the degree distributions, remain constant. However, the synaptic coupling strengths continue to fluctuate, as the neurons under the suprathreshold stimulus generate action potentials spontaneously. Figure 3(a) shows that the frequency distribution of the synaptic modification event sizes shows a power-law decay $D(s) \sim s^{-\gamma_s}$, with the exponent $\gamma_s \approx 2.5$, where s is the total amount of the change of the synaptic strengths in the network per unit time interval. The power spectrum $P(f)$ of the fluctuation $s(t)$ has peaks at the natural frequency of the FHN neuron and its harmonics, but in the low frequency range it follows a power-law decay $P(f) \sim 1/f^{\gamma_f}$, with the exponent $\gamma_f \approx 2.0$. This implies that the fluctuations are random with no time correlations. These facts suggest that functional STDP network lies in a self-organized critical state. In this critical state, the phase differences between neurons change slightly in time, as nonuniform excitatory (inhibitory) synaptic stimuli tend to slightly enhance (delay) the phase of the postsynaptic neurons. If a neuron overtakes its presynaptic partner or, in other words, if there is a phase flip between the two neurons, their synaptic conductances begin to change more rapidly and reverse the direction of the synaptic interaction, which may in turn induce larger changes in phase differences between them and their neighbors. In this way, phase flips and STDP events can propagate through the network in an avalanchelike manner. The frequency distribution of the number of phase flip events per unit time was also computed, which follows a power-law decay $D(p) \sim p^{-\gamma_p}$, with the exponent $\gamma_p \approx 2.0$.

Our results show that a neural network can be spontaneously organized by STDP to a small-world and scale-free functional structure in a self-organized critical state. The balance between excitation and inhibition in the network dynamics is critical to the formation of the complex network structure. Recent experimental studies using f-MRI (functional magnetic resonance imaging) and MEG (magnetoencephalography) in human brain sites also show that the functional networks in the brain after thresholding are in fact scale-free, small-world networks [17,18]. In a small-world network, due to the large clustering and the short average path length, faster and larger synchronization can be achieved with only a small number of connections [19–21]. We also find that, under a suprathreshold stimulus the STDP network shows as fast and high a coherence as a random network with the same connection probability. In the case that only a part of neurons are subjected to the stimulus, the STDP network shows much faster and stronger synchronization than random networks, as in Fig. 4. It should also be noted that, after a period of relaxation, the coherence decreases and saturates, exhibiting the adaptation often found in the nervous system.

In the case of conventional Hebbian networks, all the synaptic connections between the neurons under the common external stimulus increase, whereas the others are weakened. However, in our STDP network, the neurons under common stimulus need not be fully connected with only a small portion of the strengthened synapses forming a sparse, small-world, scale-free network, which is dynamically more effective and structurally more robust. This neural mechanism may be utilized in modeling and controlling the neural network more efficiently. This work provides insights useful to the studies of the formation of functional complex networks in the brain due to the activity dependent synaptic plasticity and the developmental process of neural circuits, as in the learning and memory models.

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