Self-sustained firing activities of the cortical network with plastic rules in weak AC electrical fields*

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Both external and endogenous electrical fields widely exist in the environment of cortical neurons. The effects of a weak alternating current (AC) field on a neural network model with synaptic plasticity are studied. It is found that self-sustained rhythmic firing patterns, which are closely correlated with the cognitive functions, are significantly modified due to the self-organizing of the network in the weak AC field. The activities of the neural networks are affected by the synaptic connection strength, the external stimuli, and so on. In the presence of learning rules, the synaptic connections can be modulated by the external stimuli, which will further enhance the sensitivity of the network to the external signal. The properties of the external AC stimuli can serve as control parameters in modulating the evolution of the neural network.

Keywords: spike-timing-dependent plasticity (STDP), weak electrical field, rhythmic activity

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1. Introduction

In human beings, the brain performs many sophisticated computational tasks with a high speed and precision that is unparalleled by present computers, in areas such as image and voice recognition. Investigating the mechanism with which the brain achieves such a function is helpful to both better understand the brain and to design applications based on it.[1] Recent advances in brain research have generated a renewed awareness and appreciation that the brain operates as a complex non-linear dynamic system, and that synchronous and phase-locked oscillations may play a crucial role in information processing, such as feature grouping, saliency enhancing, and phase-dependent coding of objects in the short-term memory.[2,3] Learning is central to understanding neuronal information processing, which has been studied from the molecular level up to the behavioral level. In more recent years, a new concept where the temporal order, not frequency, is more important in cellular learning has been proposed. This new learning paradigm, which is known as spike-timing-dependent plasticity (STDP), has rapidly got wide attention.[1,4] The synaptic plasticity describes the mechanisms that take place at the connection site between two neurons (synapse) when the synaptic weight related to the post-synaptic response to a single pulse is strengthened (potentiation) or weakened (depression).[5] The STDP model is a departure from the traditional Hebbian models of learning, which state that the neurons that fire action potentials together will have their interconnections strengthened.[4] Perhaps because of STDP's combination of elegant simplicity, biological plausibility, and computational power, research into STDP has provided great progress in many aspects. Previous studies have shown how the STDP rules implement input selectivity according to the spike-time correlation structure of the input spike trains for single neurons and feedforward networks.[6−8] The STDP has also been supposed to play a role in the hippocampus theta phase precession phenomenon.[9,10] Several more recent studies of the STDP have focused on parameterizing the STDP with respect to factors such as rate, higher-order spiking motifs, and dendritic location.[11] For example, Froemke and Dan reported that the first spike pairing in a train of triplet or quadruplet spike-pairings determines whether long-term potentiation (LTP) or long-term depression (LTD) ensues in the layer-2/3 pyramidal cells.[12] Similar findings have

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been acquired in the hippocampal cell culture by Bi *et al.*[13] In addition, the computational role and the functional implications of the STDP have been explored from many points of view.[14−16]

The rhythms of brain activity represent the oscillations of postsynaptic potentials in the neocortex. For example, the α rhythm (8–13 Hz) mainly presents at the resting eyes-closed state, and the θ (4–7 Hz) and β (14–30 Hz) rhythms show the reduction of power from the eyes-closed state to the eyes-open state and can therefore serve as an indicator of the brain activity state.[17] The switches between the different firing patterns of neurons due to different parameters such as external stimuli have been widely studied.[18−21] However, the effects of weak external stimuli on network evolution have not been well studied.

Transcranial electrical stimulation (TES) with weak currents is actively investigated to treat a range of neurological and psychiatric disorders.[22,23] Indeed, there is evidence that a low-intensity TES can affect ongoing brain activity.[24,25] Animal studies have indicated that electric fields will incrementally polarize somatic membranes at the levels below synaptic background activity,[26] and that there is increasing evidence that very weak electric fields can acutely modulate coherent network activity, indicating that a small incremental polarization of individual neurons can entrain the ongoing network dynamics when operating coherently on a population of coupled neurons.[27,28] Evidence that the endogenous electrical field can modulate the firing activities in the neocortex has also been found experimentally.[29]. The endogenous electrical field and most external electrical fields are believed to be weak stimuli for cortical neurons and could depolarize neurons.[30] As the effects of the electrical field on a single neuron are not obvious, a plausible hypothesis can be proposed that the effects of electrical field can be amplified by the network connections and the plastic rules. The work in this paper aims to testify this hypothesis based on a cortical network consisting of Izhikevich neurons.

The synaptic connection presented in a neural network exerts significant impacts on its function. Traditionally, a predefined topological structure is adopted in neural network modeling, which may not reflect the true situation in real-world networks, such as the brain network. Furthermore, in most studies, the synaptic delays are often neglected or taken to be an identical value. Here, the synaptic delays obey the distributions that are consistent with the real cortical networks. We investigate how the external weak alternating current (AC) stimuli influence the dynamical evolution and the emergent topology of the delayed network.

The rest of this paper is organized as follows. We first introduce the neural network's constitution and the STDP rule. Then we study the learning process of the neural network. The effects of the external weak alternating current stimuli on the learning process of the network are also investigated. We further study the changes of the average firing rate and the excitatory neurons' average synaptic strength in the learning process of the network. In the final section, the conclusions are given.

2. Neural network model and the STDP rule

Each neuron in the network is described by the simple spiking model (Izhikevich model)[31]

$$\begin{aligned} \dot{v} &= 0.04v^2 + 5v + 140 - u + I + I_{\text{ext}}, \\ \dot{u} &= a(bv - u), \end{aligned} \tag{1}$$

with the auxiliary after-spike resetting

$$\text{if } v \geq 30 \text{ mV}, \quad \text{then} \begin{cases} v \leftarrow c, \\ u \leftarrow u + d, \end{cases} \tag{2}$$

where v represents the membrane potential of the neuron, and u represents the membrane recovery variable, which accounts for the activation of K^+ ionic currents and the inactivation of Na^+ ionic currents, and also provides a negative feedback to v. Synaptic or injected currents are delivered via variable I. Parameters a, b, c, and d are dimensionless. Parameter a describes the time scale of recovery variable u, b describes the sensitivity of recovery variable u to the subthreshold fluctuation of membrane potential v, c describes the after-spike reset value of membrane potential v caused by the fast high-threshold K^+ conductances, and d describes the after-spike reset of recovery variable u caused by slow high-threshold Na^+ and K^+ conductances. According to Eq. (2), the membrane voltage and the recovery variable are reset when the spike reaches its apex at 30 mV. Depending on the values of the parameters, the model can exhibit many properties of biological neurons.[32] The model does not have a fixed threshold, and its resting potential is between −70 mV and −60 mV according to the value of b. We use $(b, c) = (0.2, -65)$ for all neurons in the network. The polarization of the single-neuron model of Izhikevich by an external electrical field exhibits

low-pass characteristics.[31] So the electrical stimulation in the model is implemented with a low-pass filter with time constant 10 ms. For excitatory neurons, we use the values $(a, d) = (0.02, 8)$, and for inhibitory neurons, we use the values $(a, d) = (0.1, 2)$.

We simulate an anatomically realistic network model consisting of 1000 cortical spiking neurons with axonal delays. The network is composed by excitatory (80%) and inhibitory (20%) neurons, and any two neurons are connected with a probability of 0.1. The synaptic connections among the neurons have fixed conduction delays, which are random integers between 1 ms and 20 ms. In the network model, synaptic connections are modified according to the STDP rule.[15] Figure 1 shows the modification function of the STDP rule. The function $F(\Delta t)$ determines the amount of synaptic modification arising from a single pair of pre- and post-synaptic spikes separated by time Δt. The STDP rules are governed by the following equations:

$$F(\Delta t) = \begin{cases} A_+ \exp(\Delta t/\tau_+), & \Delta t < 0, \\ -A_- \exp(-\Delta t/\tau_-), & \Delta t \geq 0, \end{cases} \tag{3}$$

where $\Delta t = t_{\rm pre} - t_{\rm post}$, parameters τ_+ and τ_- determine the ranges of pre-to-postsynaptic interspike intervals over which synaptic strengthening and weakening occur, respectively, and A_+ and A_- (both positive) determine the maximum amounts of synaptic modification that occur when Δt is close to zero. These equations provide a reasonable approximation of the dependence of synaptic modification on the spike timing observed experimentally. Here, we set $\tau_+ = \tau_- = 20$ ms, $A_+ = 0.1$, and $A_- = 0.12$. If a spike from an excitatory presynaptic neuron arrives at a postsynaptic neuron (possibly making the postsynaptic neuron fire), then the synapse is potentiated (strengthened). In contrast, if the spike arrives right after the postsynaptic neuron has fired, the synapse is depressed (weakened). If pre- and post-synaptic neurons fire uncorrelated Poissonian spike trains, there are moments in which the weight of the synaptic connection is potentiated or depressed. We choose the parameters of the STDP rule so that the depression is stronger than the potentiation. In contrast, if the presynaptic neuron often fires before the postsynaptic neuron, then the synaptic connection slowly potentiates. Indeed, such a connection causes the postsynaptic spikes and should be strengthened. In this way, the STDP strengthens the causal interactions in the network. The magnitude of potentiation or depression depends on the time interval between the spikes. For each fired neuron, we consider all its presynaptic neurons and determine the timings of the last excitatory spikes arriving from these neurons. Since these spikes make the neuron fire, the synaptic weights are potentiated according to the value of STDP at the presynaptic neuron adjusted for the conduction delay. Notice that the largest increase occurs for the spikes that arrive right before the postsynaptic neuron firing, that is, the spikes that actually cause the postsynaptic spike. Otherwise, when an excitatory spike arrives at a postsynaptic neuron, we depress the synapse according to the value of STDP at the postsynaptic neuron. Indeed, such a spike arrives after the postsynaptic neuron has fired, and hence the synapse between the neurons should be weakened. We artificially keep the synaptic weights between 0 and the maximum synaptic strength $s_{\rm max}$, where $s_{\rm max}$ is 10 here. The other parameters used in this paper are given in each case.

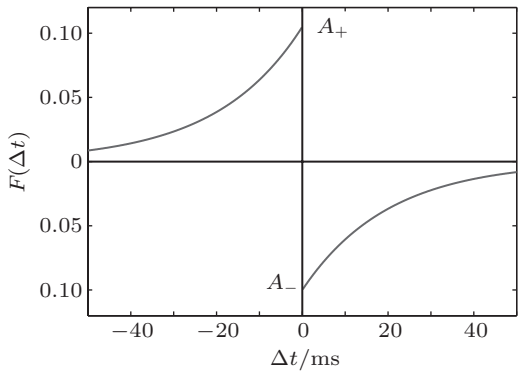


Fig. 1. The modification function of the STDP rule.

3. Effects of weak AC field on the learning process of the cortical network

In this section, we study the learning process of the neural network. The neural network can spontaneously self-organize into groups and generate patterns of stereotypical polychronous activity, as shown in Ref. [33]. Here, we mainly investigate the learning process of the network with external AC stimuli. We focus on how the firing rate and the synaptic connection strength of the network are affected by the AC stimuli during the learning process.

3.1. Network activity affected by weak AC stimuli

When the neural network is not under stimuli, it exhibits obvious self-sustained rhythmic activity at the beginning of the evolution, and the 200 inhibitory neurons (upper) in the network fire more frequently than the excitatory neurons (below), which can be seen clearly in Fig. 2(a). As the synaptic connections of the network evolve according to the STDP rule, the high-amplitude rhythmic activity disappears in the evolution process. It is shown clearly in Fig. 2(b) that the spiking activity of the neurons becomes more Poissonian and uncorrelated after an evolution time of 100 s. These phenomena arise from the competition of the pre- and post-synaptic neurons' spikings that regulate the synaptic connection strength between the neurons.

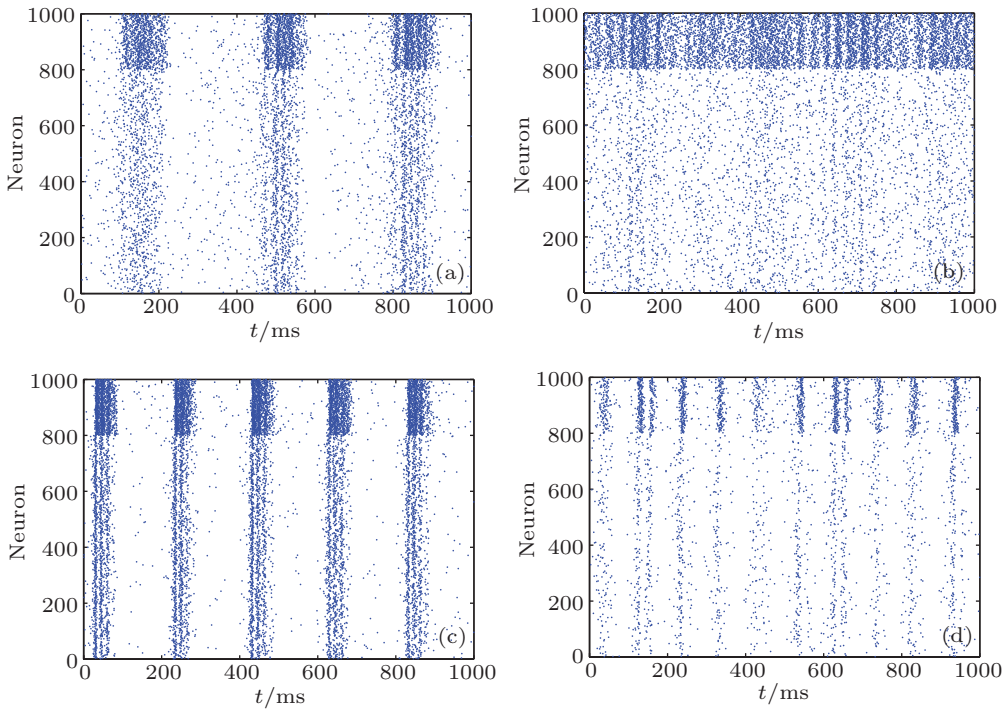


Fig. 2. (colour online) The spike rasters of the network without ((a) and (b)) and with ((c) and (d)) weak AC stimuli, where the 200 neurons on the upper side represent the inhibitory neurons, the others are the excitatory neurons, and the parameters are $A_s = 0.5$ mV, $f_s = 10$ Hz. The learning time is 2 s in panels (a) and (c), and 100 s in panels (b) and (d).

Then we study the evolution process of the neural network when we apply external weak AC stimuli to it, where all of the weak AC stimuli are in the form of $I_{ext} = A_s \sin(2\pi f_s t)$. Here, we choose $A_s = 0.5$ mV and $f_s = 10$ Hz. Figure 2(c) shows the spiking activity of the network at the beginning of the learning process. It exhibits obvious rhythmic activity, which is similar to the spiking activity of the network under no stimuli (Fig. 2(a)). After a learning time of 100 s, the neural network converges to an approximate balance state, and it still exhibits the rhythmic activity with a higher rate, as shown in Fig. 2(d). This rhythm tends to be correlated with the frequency of the external weak AC field.

3.2. The firing rate affected by weak AC stimuli

To study how the weak AC field affects the firing patterns of the network in the parameter space in detail, we investigate the evolution of the average firing rate of the whole neural network under the AC stimuli. Figure 3 shows the effects of the amplitude of the AC stimulus on the firing rate of the whole neural network. It can be seen from Fig. 3 that a long time is needed for the firing rate to achieve a stable state when there is no AC stimulus. However, the AC stimuli make it quicker. Besides, the firing rate that the network achieves after learning increases with the increase in the amplitude of the AC stimulus. It is

also interesting that the AC field with a small amplitude ($A_m < 1.8$ mV) can decrease the firing rate of the network after the learning period.

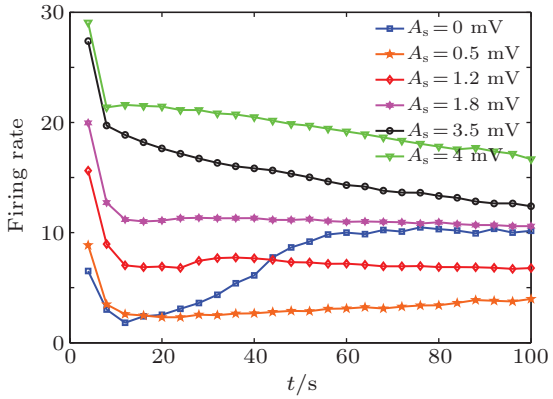


Fig. 3. (colour online) The firing rates of the network under AC stimuli of different amplitudes, and $f_s = 10$ Hz.

The frequency, which is another important characteristic of the AC stimulus, also influences the firing rate of the network. Here we discuss the effect of the low frequency on the firing rate, which is shown in Fig. 4. It is found that the firing rate will decrease after it reaches a peak when the frequency is very low. However, the firing rate can be kept on a plateau (the peak value) when the frequency is relatively high. The average firing rate of the neural network is enhanced with the increasing frequency of the AC stimulus, though the fluctuation is the relatively significant factor at the beginning of the learning process.

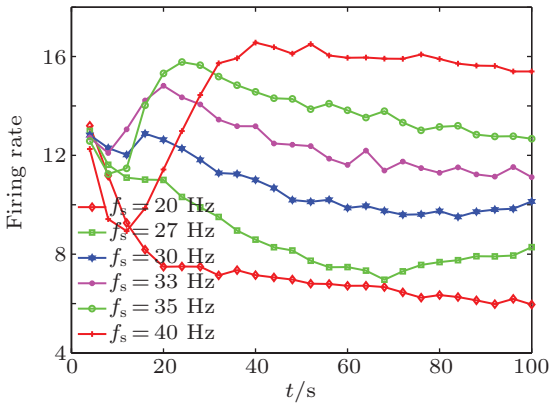


Fig. 4. (colour online) The firing rates of the network under AC stimuli of different frequencies, and $A_s = 1$ mV.

To further study the effects of the AC stimuli on the firing rate, we consider the firing rate as a function of the amplitude and the frequency of the AC stimulus. The simulation results are shown in Fig. 5. It can be seen that the firing rate is related to both the amplitude and the frequency. When the amplitude and the frequency are in proper ranges, we can obtain the optimum firing rate. Furthermore, the frequency selectivity of the AC field is not obvious when A_s is relatively low. When $A_s > 2$ mV, the phenomenon becomes obvious, and the optimal frequency is near 30 Hz, which may be the intrinsic frequency of the cortical network.

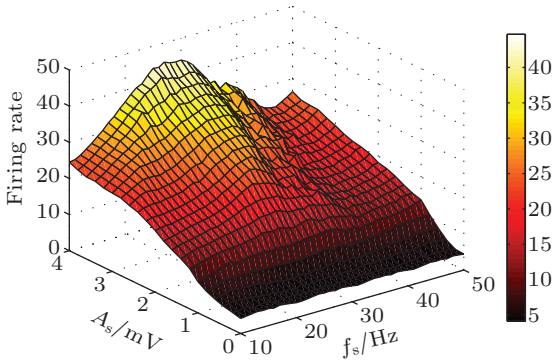


Fig. 5. (colour online) The firing rate of the network under external AC stimuli as a function of the amplitude and frequency of the AC field.

The tendency of the firing rate curve may be caused by the structure of the network, such as the axonal conduction delays and the changes in the synaptic connection. So in the next subsection, we will discuss the changes in synaptic strength when the network is under the AC stimuli.

3.3. Synaptic connection strength affected by weak AC stimuli

We now check how the synaptic connection of the neural network evolves when we apply the weak AC stimuli on it. Because the inhibitory synaptic weights are not plastic, and the excitatory synaptic weights evolve according to the STDP rule, we take the excitatory synapses into account.

Figure 6 shows the average synaptic strengths of the excitatory neurons under the AC stimuli of different frequencies. The firing rate decreases sharply at the beginning of the evolution when the frequency is low, however, the decrease trend becomes slow as the frequency increases.

To study the effects of the amplitude of the AC stimulus on the synaptic strength, we divide the synaptic strength of the excitatory neuron into three parts and calculate the percentage of the average synaptic strength under different stimuli in each part. As shown in Fig. 7, the percentages of the strong

and the medium synaptic strengths decrease, while the percentage of the weak synaptic coupling strength increases with the increasing amplitude of the stimulus, thus the average synaptic strength decreases. It is also found that the percentages of the different synaptic strength values almost remain stable when the amplitude of the AC stimulus becomes larger than 2 mV.

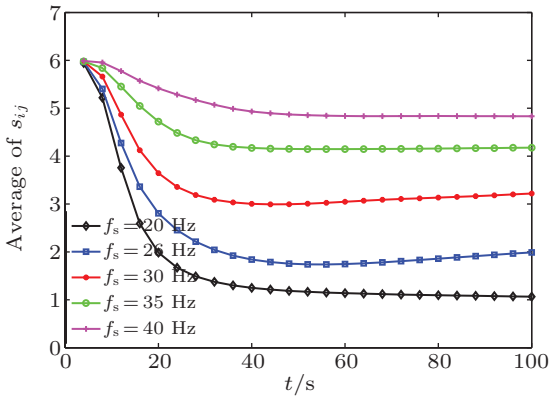


Fig. 6. (colour online) The average synaptic strengths of the neural network's excitatory neurons under AC stimuli of different frequencies. The amplitude of the stimulus is 1 mV.

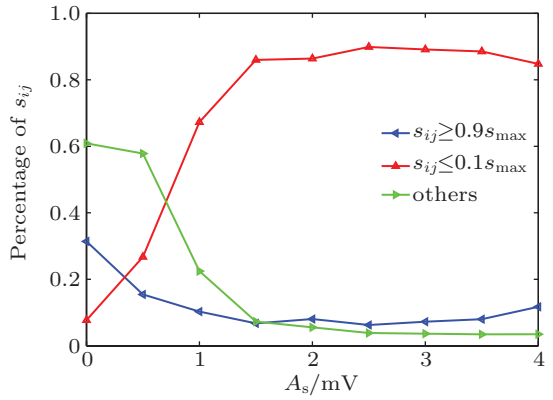


Fig. 7. (colour online) The percentages of different synaptic strengths each as a function of the amplitude of the AC stimulus when the evolution time of the network is 100 s.

It can be seen from Fig. 8 that the amplitude of the AC stimulus plays the main role in the evolution of the average synaptic strength of the network's excitatory neurons. And the synaptic strength decreases with the increasing amplitude in the evolutionary process. In contrast, the increase in the frequency of the AC stimulus can enhance the synaptic strength of the network's excitatory neurons. Hence the synaptic strength needed can be obtained by modulating the amplitude and the frequency in proper ranges.

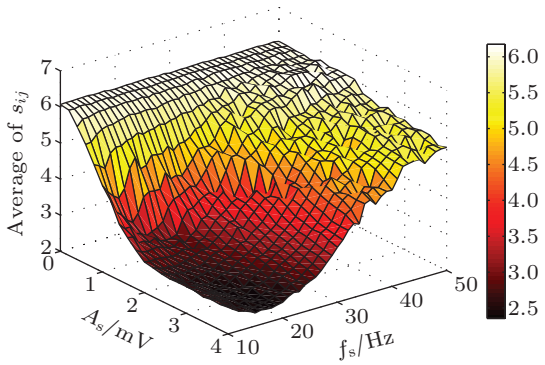


Fig. 8. (colour online) The average synaptic strength of the network's excitatory neurons as a function of the amplitude and frequency of the AC stimulus.

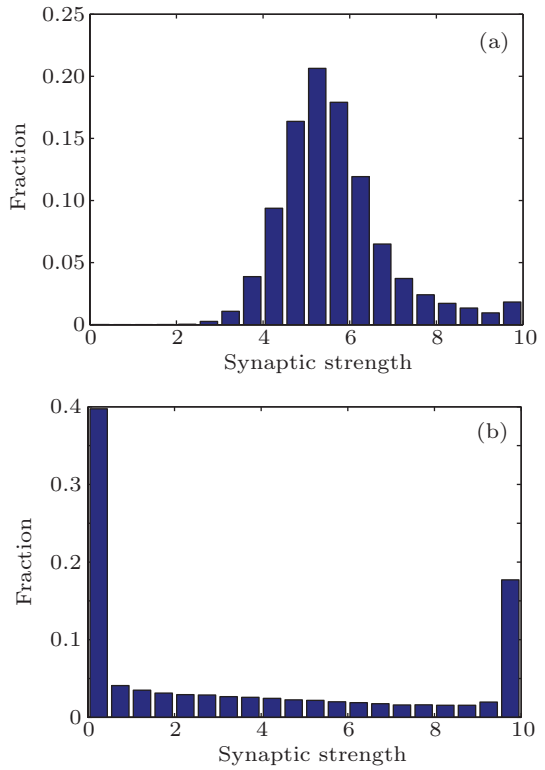


Fig. 9. (colour online) The distributions of the excitatory neurons' synaptic strength for learning times of (a) 4 s and (b) 100 s. The AC stimuli have the form $I_{ext} = A_s \sin(2\pi f_s t)$, where $A_s = 0.5$ mV, and $f_s = 10$ Hz.

Figures 9(a) and 9(b) show the distributions of the excitatory neurons' synaptic strength under the same stimulus during the evolution process. We set 6 mV as the excitatory neurons' initial synaptic strength. Comparing Fig. 9(a) with Fig. 9(b), we can find that at the beginning of the evolution, the distribution of the synaptic strength concentrates at the initial value. However, after a learning time of 100 s, it is shown in Fig. 9(b) that most of the synaptic connections are polarized to either 0 or the maximum s_{max} from the initial values. The underlying mechanism

is the competition between the synapses during the learning process. The stronger synapses tend to make it easier for the presynaptic spikes to induce the corresponding postsynaptic firings, which will strengthen this synapse according to the STDP rules shown in Fig. 1. The weaker synapses will also be weakened because the presynaptic and postsynaptic spikes tend to be irrelevant. Finally, the synaptic strength values will be saturated at the bounding values.

4. Discussion and conclusion

The effects of the external AC field on the learning process of a network are investigated in detail. It has been shown that networks without any external AC stimulus display uncorrelated Poissonian firing activity at the end of the learning process. However, a new self-sustained rhythmic firing activity is found at the end of the learning process when the network is under external weak AC stimuli. We have found that both the amplitude and frequency of the AC stimuli can influence the firing patterns of the network. Synaptic plasticity plays an important role in the learning process of the network. We illustrate that the external AC stimuli change the synaptic connection strength according to the STDP rule, and that the evolution enhances the sensitivity of the cortical network to the weak AC field.

Our findings in this paper may be significant in neuroscience, and helpful for further study of the evolution of the neural network. Several properties of the neuronal network, such as synaptic connection strength and different external stimuli, can serve as parameters in modulating the firing activities in the cortical neural network. This may be significant in neural disease therapy.

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