The brain is one of the most challenging complex systems. The neurons, massively interconnected to each other, show highly complex and correlated responses to the external stimuli, which help the brain to extract relevant patterns from sensory inputs, coordinate movements and control behaviors. To understand the complexity of the nervous system, we need to characterize its network structure on which the spatio-temporal firing activities are supported.

Recent studies on the complex networks in a variety of systems describe real networks by simply defining a set of nodes and connections between them. Examples range over social networks, information networks, technological networks, and biological networks. They lie between regular networks and fully random networks. A wide variety of such systems are scale-free, where the connectivity distributions take a power-law form, and the topology and evolution of such networks are governed by the common mechanism such as the preferential attachment and the growth regardless of the detailed nature of the specific networks.

In the context of the complex network, the topological structure of simple nervous systems, for example, in the worm Caenorhabditis elegans neural network, have been proved to be an inhomogeneous small-world network. However, for the network in the brain, more important is the functional structure than the morphological one because the former is the direct carrier of the neuronal information in the form of spikes. Moreover, the functional structure changes through the adaptive variation in the synaptic conductances due to the inputs from external stimuli and the internal dynamics of neurons in the network, which in turn leads to the change in the responses of the network. This feedback process of synaptic modification in the brain is believed to be closely connected to the learning and memory. Recently, this kind of synaptic changes have been observed experimentally in various brain regions, such as neocortical slices, hippocampal slices and cell cultures, and the EEL of the electric fish, where long term synaptic modifications, both long term potentiation (LTP) and long term depression (LTD), arise from repeated pairings of pre- and post-synaptic action potentials. The sign and the degree of synaptic modification depend on their relative timing, called the spike timing dependent plasticity (STDP). For example, in the hippocampal CA3 region and neocortical slices, by STDP a synapse is strengthened if the presynaptic spike is followed by postsynaptic action potentials within about 50ms and weakened if the postsynaptic action potential follows postsynaptic spikes.

In this Letter, we report that STDP reorganizes a globally connected neural network spontaneously into a functional network which is both small-world network and scale-free. This complex network arises when the excitatory and inhibitory connection strengths between neurons are balanced. The neuronal activities on this small-world scale-free neural functional network is found to lie in a self-organized critical state. The neuronal oscillators in the functional network which is both small-world scale-free network and

\[ \text{PACS numbers: 87.18.Bb 87.18.Sn 87.19.La 89.75.Da 89.75.Fb} \]
The amount of the synaptic modification by STDP depending on the time difference between pre- and postsynaptic spikes, \( \Delta t = t_{\text{post}} - t_{\text{pre}} \), is modeled by the STDP modification function:

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

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 \cite{13}. Here, the parameter values are chosen to be \( A_+ = 0.01, A_- = 0.006, \tau_+ = 1.0, \) and \( \tau_- = 2.0 \). \( G_{ij} \) lies in \( 0 < G_{ij} \leq G_{max} \) and if \( G_{ij} \) increases over the maximal value, \( G_{ij} \) is set to \( G_{max} \). Other parameters are set to \( a = 0.5, b = 0.12, \epsilon = 0.005, V = 0.7, V = 0.0, \) and \( \tau_{\text{syn}} = \bar{\tau}_{\text{syn}} = 0.2 \).

We start from the globally coupled network of \( N = 1,000 \) neurons with random initial coupling strengths of \( G_{ij} \), \( 0 \leq G_{ij} \leq G_{max} \), and investigate how the functional structure develops spontaneously in time. Given the external dc-current, \( I_{\text{ext}} = 0.2 \), which is supra-threshold stimulus for spontaneous generation of action potentials, after a period of relaxation by STDP, some population of synapses are strengthened to the maximum conductance value, \( G_{max} \), while the other population of synapses are weakened to near zero and, therefore, become silent to their postsynaptic neurons. This is similar to the bimodal distribution in the case of the balanced excitation of synapses from many input neurons to a single neuron \cite{14}. As a result, even if the neurons in the network are morphologically connected all-to-all by synapses, the functional structure can be re-organized by STDP in which each neuron is functionally connected to only a small population of neurons.

Each synapse is regarded as functionally connected if the synaptic conductance is larger than a critical value, \( G_{ij} > G_c \), and functionally not connected otherwise. In Fig. 1(a), the average connection probability, the ratio of the number of strengthened synapses to the total number...
of synapses, \((k/N)\), is shown in the parameter space of \(G_{\text{max}}\) and \(G_{\text{inh}}\). 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. In accordance with the diagram, three distinct classes of the network states can be identified: synchronized, clustering, and dispersed network states. To characterize the dynamical properties of the functional network organized by STDP, we define the phase of a neuron at time \(t\) between each firing time piece-wise linearly:

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

where \(t_n^*\) is the \(n\)-th firing time of the neuron. The phase coherence, \(\Gamma\), of neurons in the network is defined as:

\[
\Gamma = \max \left\{ \left| \lim_{T \to \infty} \frac{1}{T} \int_0^T \frac{1}{N} \sum_{j \neq k} e^{in\Delta_{jk}(t)} dt \right|_{n} \right\},
\]

where \(\Delta_{jk}(t)\) is the difference of the instantaneous phases of \(j\)-th and \(k\)-th neurons at time \(t\). Note that \(\Gamma\) saturates to 1 if the firing times of all neurons are coherent with \(n\)-clusters and 0 if they are random. The dependence of the phase coherence, \(\Gamma\), in Fig. 1(b) is similar to the one for 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 fully synchronized. On the other hand, in the case that the inhibitory input dominates the excitatory input, the clustering state is formed where the neurons are partially synchronized and each synchronized group fires asynchronously. In the diagonal region, the excitatory and inhibitory inputs are balanced, and the firing pattern of the network is dispersed but not entirely random.

To characterize the structural properties of the complex functional network organized by STDP, we calculate the clustering coefficient, \(C\), the fraction of connections that actually exist between neighbors of each neuron with respect to all allowable 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, \(C/C_{\text{rand}}\), 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 \((k/N) \approx 0.03\), but the clustering coefficient for our network is large with \(C \approx 0.23\), whereas the clustering coefficient of the 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 between the excitatory and the inhibitory coupling has typical small-world characteristics: the clustering coefficient of the network is much larger than that of 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 structure of the neural network in the case of the balanced input are scale-free. Fig. 2 shows that the degree distributions follow a power-law decay with a cutoff at large \(k\): \(P_{\text{in}}(k) \sim k^{-\gamma_{\text{in}}}\), \(P_{\text{out}}(k) \sim k^{-\gamma_{\text{out}}}\), and \(P_{\text{tot}}(k) \sim k^{-\gamma_{\text{tot}}}\), where \(P_{\text{in}}, P_{\text{out}}, \) and \(P_{\text{tot}}\) are the frequency of nodes with the same number of in-coming, out-going and total synaptic connections independent of directions, respectively. In the middle of the diagonal region in Fig. 2 the scaling exponents are measured to be \(\gamma_{\text{in}} \approx 1.7\), \(\gamma_{\text{out}} \approx 1.4\) and \(\gamma_{\text{tot}} \approx 1.5\). The estimated values of \(\gamma\) do not depend much on the details of synaptic parameters, \(G_{\text{max}}\) and \(G_{\text{inh}}\), around the diagonal region.

After a period of relaxation, the average network properties of the functional network remain constant, but the synaptic coupling strengths continue to fluctuate as the neurons under the supra-threshold stimulus generate action potentials spontaneously. Fig. 2 shows that the distribution of the sizes of the change of the total synaptic coupling strength per unit time, \(D(s)\), and the
The fluctuation of the synaptic strengths (b). Both follows synaptic coupling strengths (a) and the power spectrum of the fluctuation of the synaptic strengths (b). Both follows a power-law decay with exponents, $\gamma_\alpha \approx 2.5$ and $\gamma_f \approx 2.0$. The power spectrum has peaks at the natural frequency of the FHN neuron and its harmonics.

The power spectrum of the fluctuation, $P(f)$, power-law decays, $D(s) \sim s^{-\gamma_\alpha}$ and $P(f) \sim 1/f^{\gamma_f}$, with scaling exponents $\gamma_\alpha \approx 2.5$ and $\gamma_f \approx 2.0$. These facts suggest that the firing dynamics of the neurons on the small-world scale-free functional network lies in a self-organized critical state, and the fluctuation is random with no time correlation. In this critical state, a slight change in synapses may bring a significant change in dynamical firing patterns, which can in turn induce a larger change in synapses in an avalanche-like manner as in the case of the sandpile models.

Our results show that by STDP the small-world and scale-free functional structure can be spontaneously organized in the neural network under common external input stimulus, in the form of the self-organized critical state. The balance between excitation and inhibition in the network dynamics is critical to the formation of the nontrivial network structure. The experimental studies using fMRI and MEG in human brain sites also show that the functional networks in the brain are in fact scale-free small-world networks. 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, and the scale-free network is robust against the random failure of nodes. The functional structure by STDP also shows both fast synchronization and high coherence which is dynamically effective and structurally robust.

In the case of conventional Hebbian networks, if the common external stimulus is given to a part of a neural network, the synaptic connection strengths between the neurons under the stimulus increase whereas the other synapses are weakened. However, our results suggest that even the neurons under common stimulus need not be functionally connected, but only a small portion of the synapses between neurons can be strengthened to make the network sparse but small-world and scale-free. We expect that our work would provide insights on the studies of the formation of complex networks and the developmental process of neural circuits in the brain, as in the learning and memory models. We also expect that this neural mechanism could be utilized in controlling the neural network efficiently and enlarging the memory capacity.

[1] M. E. J. Newman, 2003, SIAM Review, 45 167.
[2] R. Albert and A.-L. Barabási, 2002, Rev. Mod. Phys., 74 47.
[3] S. N. Dorogovtsev and J.F.F. Mendes, 2002, Adv. Phys., 51 1079.
[4] L. A. N. Amaral and J.M. Ottino, 2004, Eur. Phys. J. B, 38 147.
[5] J. G. White, E. Southgate, J. N. Thompson and S. Brenner, 1986. Philosophical Transactions of the Royal Society of London, Series B 314 1.
[6] H. Markram, J. Kübke, M. Frotscher, and B. Sakmann, 1997. Science 275 213.
[7] D. Debanne, B. H. Gähwiler, and S. M. Thompson, 1998, J. Physiol. 507 237.
[8] G.-Q. Bi and M.-M. Poo, 1998, J. Neurosci., 18 10464.
[9] C. C. Bell, V. Z. Han, Y. Sugawara and K. Grand, 1997, Nature 387 278.
[10] R. FitzHugh, 1961, Biophys. J., 1 445.
[11] M. C. W. van Rossum, G. Q. Bi and G. G. Turrigiano, J. Neurosci., 20 8812
[12] J. Rubin, D. D. Lee and H. Sompolinsky, 2001, Phys Rev Lett., 86 364.
[13] G.-Q. Bi, M.-M. Poo, 2001, Annu. Rev. Neurosci. 24 139.
[14] S. Song, K. D. Miller and L. F. Abbott, 2000, Nature Neurosci., 3 919.
[15] A. S. Pikovsky, M. G. Rosenblum, G. V. Osipov and J. Kurths, 1997, Physica D, 104 219.
[16] Donald L. Turcotte, 1999, Rep. Prog. Phys. 62 1377.
[17] V. M. Eguíluz, D. R. Chialvo, G. Cecchi, M. Baliki, and A. V. Apkarian, 2003, arXiv:cond-mat/0309002.
[18] C.J. Stam, 2004, Neurosci. Lett., 355 25.
[19] L. F. Lago-Fernández, R. Huerta, F. Corbacho, and J. A. Sigüenza, 2000, Phys Rev. Lett., 84 2758.
[20] H. Hong, M. Y. Choi, and B. J. Kim, 2002, Phys Rev. E 65 026139.
[21] N. Maduda and K. Aihara, 2004, Biol. Cybern., 90 302.