Neurobiological Determinants of Self-Organized Criticality in Neuronal Avalanches

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Abstract—**Over the last two decades, self-organized criticality in brain activity has been widely discussed. The scale-free activation patterns, called neuronal avalanches, provide evidence for the postulation that the brain is operating in a critical state. Several neurobiological properties found in experiments are indicated to be important parameters in neuronal avalanches. Here, we developed a dynamic neural network model including these neurobiological properties, such as the balance between excitation and inhibition, the synaptic delay times, and the spatial connection structure, to theoretically test and verify the possibility of these neurobiological properties as the control parameters in tuning the network through criticality in neuronal avalanches.**

Keywords-self-organized criticality; neuronal avalanches; neurobiological determinant; order parameter

I. INTRODUCTION

More and more experiments in vitro and in vivo support the idea that cortical networks in brain operates at a critical point[1-6]. This phenomenon was called neuronal avalanches whose sizes follow a power law distribution. Neuronal avalanches have been observed in several animals' neural systems, such as awake monkeys[7], anesthetized rats[8], isolated leech ganglion[9], and in human brain oscillations[10].

Brain criticality is important to understand the cortical neural activity, especially in the process of brain formation, neural network formation, synaptic formation, and sleep[11, 12]. Studies showed that neuronal activities in the cortex might be critical arising from the premise that a critical brain can show the fastest and most flexible adaptation to a rather unpredictable environment[13,14]. In clinical application research, avalanches activity patterns provide important information to us to understand the mechanism of epilepsy[15]. Studies have shown that neurons in the normal brain can regulate neural activity to a critical state, while neurons in epileptic patients lose their regulatory function, which makes the spontaneous activity of cortex not have power-law distribution characteristics and is in supercritical state[16,17]

Many theoretical models of neuronal avalanches have been developed, which indicated that neural system would be optimized in computational power, information storage, long distance communication, dynamic range, and learning at the critical point[18, 19]. However, these models did not discuss the neurobiological determinants of neuronal avalanches. Actually, the dynamics in neural networks, such as the dynamic of neurons and the spike-timing-dependent plasticity, have great effect on the critical behavior of networks[20]. Recently, some models based on the neural dynamics and synaptic dynamics were proposed to discuss the neurobiological determinants of neuronal avalanches. These researches found that in a network with modular connectivity, the state of the network was sensitive to synaptic plasticity[21-23]. However, the neurobiological determinants of neuronal avalanches are far from clear.

Several neurobiological properties found in experiments, such as the balance between excitation and inhibition, the synaptic delay times, and the connection structures, are indicated to be important parameters in neuronal avalanches[13, 24]. In this paper, we developed a dynamic neural network model including these neurobiological properties to theoretically test the possibility of these neurobiological properties as the control parameters in tuning the network through criticality in neuronal avalanches.

II. MODEL

A. Neurons

We model a neural network of 1000 dynamic neurons, including 800 excitatory neurons and 200 inhibitory neurons. Each neuron is described by the spiking model[25]:

$$\begin{aligned} \frac{dv}{dt} &= 0.04v^2 + 5v + 140 - u + I \\ \frac{du}{dt} &= a(bv - u) \end{aligned} \tag{1}$$

where v denotes the membrane voltage of the neuron; u represents a membrane recovery variable, which accounts for the activity of Na^+ and K^+. If $v = 30mV$, then $v = c$, $u = u + d$. For all the neurons, $(b, c) = (0.2, -65)$. For excitatory neurons, $(a, d) = (0.02, 8)$ corresponding to cortical pyramidal neurons with the regular spiking pattern. For inhibitory ones, $(a, d) = (0.1, 2)$ corresponding to cortical inter-neurons exhibiting fast spiking firing patterns.

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B. The structure of the neural network

The input signal *I* of each neuron is composed by two parts. One part is the noise input that a random chosen neuron will receive an input current at each time step. The other part is the synaptic input from the other neurons. The coupled structure of the neurons is based on the neural model created by Izhikevich[25]. Each neuron has *M* synapses connecting to other neurons. Each synapse randomly connects to a neuron, no matter whether the postsynaptic neuron is excitatory or inhibitory. The synaptic weight of inhibitory neurons is w_i, and that of excitatory ones is w_e. Particularly, the signal conduction delay time is considered in the neural network, which makes the neural network not only has a spatial structure but also has a temporal structure. In the simulation, the delay time of the inhibitory synapse is fixed to *1 ms* and the delay time of each excitatory synapse is randomly chosen between *1 ms* and $\tau_{\max}$.

C. Spike-timing-dependent plasticity

The synaptic connection in the network is basically modified according to the spike-timing-dependent plasticity (STDP) rule[26]. If a spike from an excitatory pre-synaptic neuron arrives at a postsynaptic neuron before the postsynaptic neuron fired, then this synapse is potentiated (strengthened). On the contrary, if the spike arrives after the postsynaptic neuron fired, the synapse is depressed. The magnitude of potentiation or depression relies on the time interval between the spikes with

$$\Delta w = \begin{cases} A_+ e^{-\Delta t/\tau_+}, & \Delta t > 0 \\ A_- e^{\Delta t/\tau_-}, & \Delta t \le 0 \end{cases} \tag{2}$$

in which $A_+ = 0.1, A_- = 0.12,\ \tau_+ = \tau_- = 20ms,$ When a neuron fires, the Δw is reset to 0.1. Every millisecond (one time step is one millisecond), Δw decreases by $0.95\Delta w$, so that it decays to zero as $0.1e^{-t/20}$.

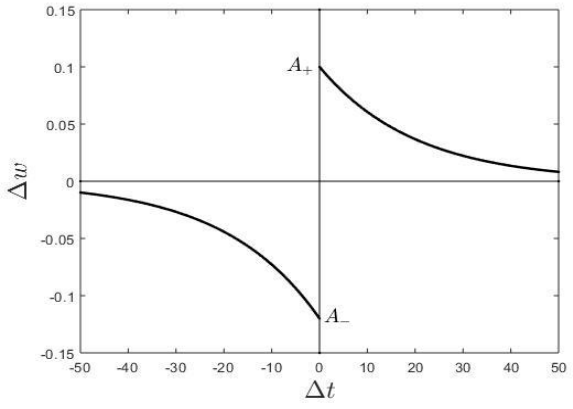


Figure 1. The synaptic connection in the network is modified according to the spike-timing-dependent plasticity (STDP) rule.

III. RESULTS

A. The Firing Pattern

In the resting state, there are no outside input signals, only noise signal input. When the input to a neuron accumulated to the threshold in a period, the post synaptic neuron fires a spike. So, the different spatial and temporal firing patterns of neurons in the former time steps will excite different numbers of neurons in the following time steps. If many neurons fire in a short period, the large firings of inhibitory neurons will inhibit the network into a quiet period. After this quiet period, the network experiences another firing period.

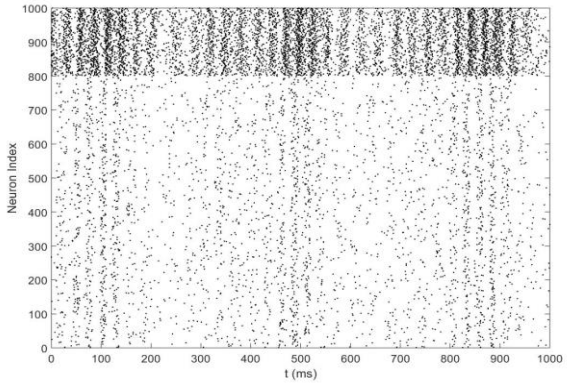


Figure 2. The spatial and temporal firing patterns of neurons.

B. Critical Size and Duration

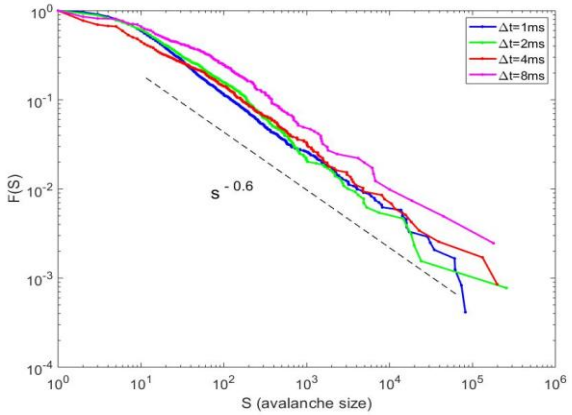
The definition of a neuronal avalanche in this model is as same as that in the experiments of Plenz[1]. The simulation data are binned at width $\Delta t = 4ms$. The spatial pattern of firing activity during one time bin Δt is called a frame. A sequence of consecutively active frames that is preceded by a bland frame and ended by a blank frame is called an avalanche. Here, we also tried the other time bins from 1ms to 8ms, and the result shows that the width of the time bin will not impact the power law distribution.

The size of a neuronal avalanche is defined as the total number of fired neurons in a neuronal avalanche, and the duration of a neuronal avalanche is defined as whole time steps (each time step means one millisecond) during a neuronal avalanche. Each neuron in the network has *M* synapses connecting to other neurons. STDP rule works during all the 10^5 time steps.

As shown in Figure 3, the results indicated that the cumulative probability distribution of the neuronal avalanches size was almost a line with characteristic slope of -0.6 (as shown in Fig 3A). Using KS statistics tests, we found that avalanche size distribution was well fitted by $p(S) \sim S^{-0.6}$. This means the probability density function of the neuronal avalanche size was $P(S) \sim S^{-1.6}$, which was quite similar with the experiment results [1]. Under the same simulating parameters, the cumulative probability distribution of the

neuronal avalanches duration was well fitted by $p(T) \sim T^{-0.9}$ (as shown in Fig 3B). This means the probability density function of the neuronal avalanche duration was $P(T) \sim T^{-1.9}$, which was similar with the experiment results of neuronal avalanches [1] .

A.



B.

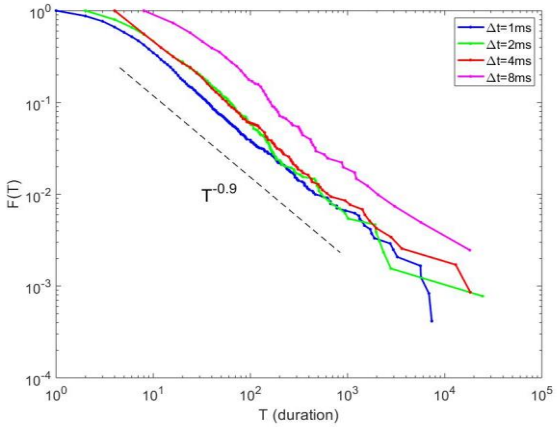


Figure 3. The cumulative probability distributions of neuronal avalanches size and duration. A.the exponent of the cumulative probability distribution of neuronal avalanches size is -0.6. B.the exponent of the cumulative probability distribution of neuronal avalanches duration is -0.9.

IV. TUNING THE NETWORK THROUGH CRITICALITY

An avalanche is described by its size S and its duration T. Critical theory predicts the probability density distribution function forms of these two variables when the system is near criticality,

$$P(S) \sim S^{-\tau} \tag{3}$$

$$P(T) \sim T^{-\alpha} \tag{4}$$

τ and α are critical exponents of the system, which are expected to be independent of the details of the system. For all the critical datasets in the simulations, the exponents were $\tau = 1.6 \pm 0.2$, $\alpha = 1.9 \pm 0.2$. This means that the system is near a critical point.

Criticality theory also predicts that the average avalanche size of a certain duration T is given by the scaling relation

$$\langle S \rangle(T) \sim T^{k} \tag{5}$$

Furthermore, critical theory predicts the exponent relations that the above three exponents must obey the relation,

$$k = \frac{\alpha\text{-}1}{\tau\text{-}1} \tag{6}$$

For the critical datasets in the simulation, $k = 1.3 \pm 0.2$ ($p<0.001$, two-sample t test, as shown in Figure 4). So, the exponent values in the critical samples are consistent with this relation. The value for k in the simulation was also consistent with that obtained in experiments[27].

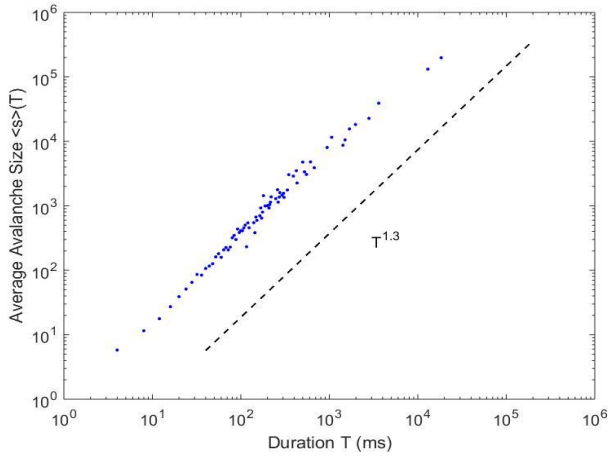


Figure 4. The average avalanche size of duration follows the scaling relation.

V. NEUROBIOLOGICAL DETERMINANTS AS CONTROL PARAMETERS

A. *The Balance between Excitation and Inhibition as a Control Parameter*

For a system operating near the critical point, there is a control parameter that can tune the system among the subcritical state, the critical state and the supercritical state. In the in-vitro experiments, the balance between excitation and inhibition is indicated as a control parameter[28]. The neuronal avalanches predominantly depend on the $GABA_A$ and glutamatergic NMDA receptor. When blocking excitatory synaptic transmission, the system leads to the subcritical state. Conversely, by blocking inhibitory synaptic transmission, the system leads to the supercritical state. A balance between excitation and inhibition will lead the system accesses the critical state.

To discuss the mechanism of control parameters found in the in-vitro experiments, we examined the balance of excitation and inhibition as a control parameter in our model. We simulated the neural network in different values of excitatory and inhibitory synaptic weights. The simulation results showed that when maximum excitatory synaptic weight was 10 and the inhibitory synaptic weight was 5, the size distribution of the neuronal avalanches followed power law, which meant the system was at the critical state. When maximum excitatory synaptic weight was 6 and the inhibitory synaptic weight was 5, the firing pattern of the whole network showed to be at the sub-critical state. On the other hand, when maximum excitatory synaptic weight was 10 and the inhibitory synaptic weight was 3, the firing pattern achieved the super-critical state instead (as shown in Figure 5).

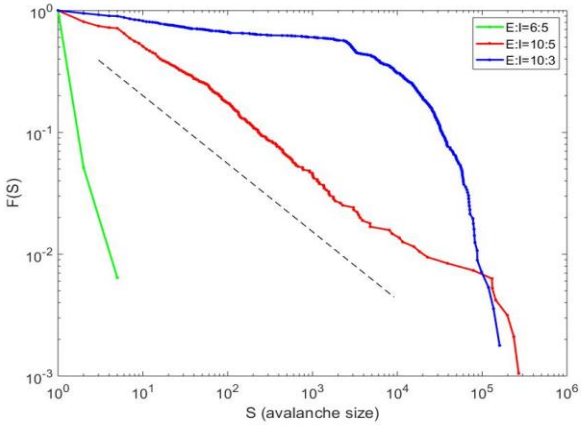


Figure 5. The cumulative probability distributions of neuronal avalanches size in different values of excitatory and inhibitory synaptic weights.

B. The temporal connection structure as a control parameter

The temporal connection structure in this neural network model was composed by the different delay times of the excitatory and inhibitory synapses. Our results showed that the synaptic delay time also acted as an important control parameter for the system to be tunable among the subcritical state, the critical state, and the supercritical state.

We tested the neural network in different synaptic delay times. We fixed the delay time of the inhibitory synapses to *1 ms*. The delay time of the excitatory synapses was randomly chosen between *1 ms* and $\tau_{\max}$, in which $\tau_{\max}$ was the upper limit delay time of the excitatory synapse. $\tau_{\max}$ was tunable between *5ms* and *30ms*. The simulation result showed that the critical state of the system changed according to $\tau_{\max}$. When $\tau_{\max}$=20*ms*, the size distribution of the neuronal avalanches followed power law, which meant the system was at the critical state. When $\tau_{\max}$ decreased to *5ms*, the firing pattern of the whole network showed to be at the sub-critical state. On the other hand, when $\tau_{\max}$ increased to *30ms*, the firing pattern achieved the super-critical state (as shown in Figure 6).

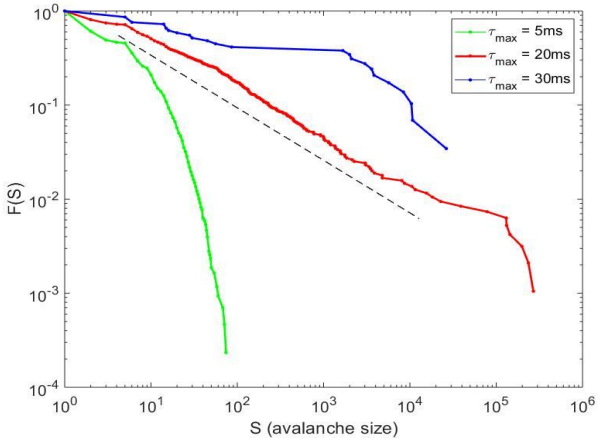


Figure 6. The cumulative probability distributions of neuronal avalanches size under different synaptic delay times.

C. The spatial connection structure as a control parameter

The spatial connection structure was indicated as a control parameter in the in-vitro experiments. In our simulation, the network is randomly connected and the spatial connection structure was composed by the numbers of the synapses that each neuron connects to other neurons. We tested the neural network in different numbers of synapses per neuron *M*. The results showed that the spatial connection structure was one of the key control parameters for the system to be tunable among the subcritical state, critical state and supercritical state. When *M*=100, the size distribution of the neuronal avalanches was power law, which meant the system was at the critical state. When *M*=40, the firing pattern of the whole network showed to be at the sub-critical state. On the other hand, when *M*=160, the firing pattern achieved the super-critical state instead (as shown in Figure 7).

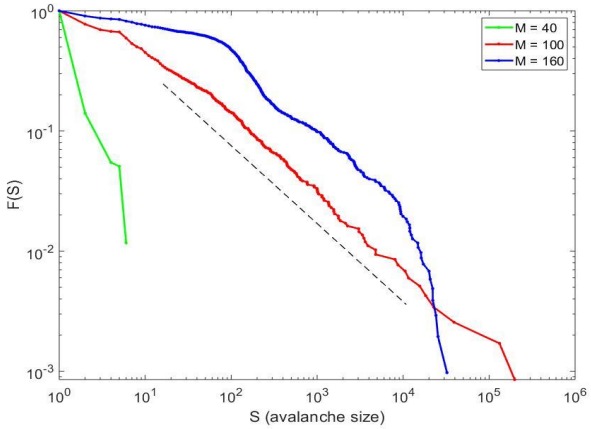


Figure 7. The cumulative probability distributions of neuronal avalanches size under different numbers of synapses per neuron *M*.

VI. DISCUSSION

Over the last two decades, self-organized criticality in brain activity has been widely discussed. The scale-free activation patterns, called neuronal avalanches, provide evidence for the postulation that the brain is operating in a critical state. For a system that operates in a critical state, there should be a control parameter that can tune the system to the critical state. Experiments results show that some neurobiological properties are critical parameters in neuronal avalanches, such as the balance between excitation and inhibition, the synaptic delay times, and the connection structures. Whether these properties act as the control parameters in the system needs to be tested and verified.

In this paper, based on a dynamic neural network model, we theoretically tested and verified the possibility of the balance between excitation and inhibition, the temporal connection structure, and the spatial connection structure as the control parameters in tuning the network through criticality in neuronal avalanches. Here, we want to make further analysis on the mechanism that how do these properties act as the control parameters.

The first property is the balance between the excitatory and inhibitory neurons. The system experienced a transition among the sub-critical state, the critical state and the super-critical state according to different ratio of excitatory and inhibitory synaptic weights. The main reason is that the response time of the excitatory neurons is longer than the inhibitory neurons. Therefore, the increase in the firing activity of the excitatory neurons will induce more activity of the inhibitory neurons. Thus, we conclude that the transition among the three critical states depends on the response time of neurons and it also indicates that the balance of the excitatory and inhibitory neurons is one of the important mechanisms in neuronal avalanches.

The second one is the temporal connection structure due to the delay time of synapses. When the upper limit delay time of the excitatory synapse was tuned from 5ms to 30ms, the system experienced a transition from the sub-critical state to the critical state and then to the super-critical state. The shorter the delay times of excitatory synapses, the more inhibitory neurons are excited. The large firing of inhibitory neurons will quickly inhibit the network into a quiet period, so the system will be at a sub-critical state. On the other hand, when the delay times of excitatory synapse are longer, less inhibitory neurons are excited in the same period, which makes the system to be at a super-critical state.

The third one is the spatial connection structure composed by the numbers of the synapses. The result showed that the sparsely connected network was at a sub-critical state, and the densely connected network was at a super-critical state. Only the network of a moderate density of connection was at the critical state.

These results demonstrated that the order parameters of self-organized criticality in brain might be neurobiological properties, which are quite different from the mechanisms of self-organized criticality in other systems. Furthermore, there are several other neurobiological properties playing key roles in the self-organized criticality in brain, such as the dynamics in neurons and synapses, connection strengths, and pattern of connections. Whether these properties are the control parameters in neuronal avalanches is an important question that needs further exploring.

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