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Self-organized criticality in a cellular automaton model of pulse-coupled integrate-and-fire neurons

Dan-mei Chen†, S Wu‡, Aike Guo† and Z R Yang‡§

† Laboratory of Visual Information Processing, Institute of Biophysics, Academia Sinica, Beijing 100101, People's Republic of China

‡ Physics Department and Institute of Theoretical Physics, Beijing Normal University, Beijing 100875, People's Republic of China

§ CCAST (World laboratory), PO Box 8730, Beijing 100080, People's Republic of China

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Abstract. We have introduced a cellular automaton to investigate self-organized criticality in the activity of neural populations. The model is composed of pulse-coupled integrate-and-fire neurons and stimulated by continuous driving. Under an appropriate condition, the system is found to exhibit a robust self-organized critical behaviour accompanied with the large-scale synchronized activities among the units. It indicates the close relationship between self-organized criticality and synchronization.

1. Introduction

It has been found by Bak, Tang and Wiesenfield (BTW) that many large dynamical systems tend to self-organize themselves into a statistically stationary state which is critical in the sense that both the spatial and temporal correlations obey power-law behaviour [1]. In contrast to the situation in the phase transition, this critical state is achieved over a wide range of the parameters of the system and no fine tuning is needed. BTW called this phenomena self-organized criticality (SOC) and expected that SOC may explain the ubiquity of the fractal structure in nature. To illustrate this idea, BTW have introduced the well known sandpile model. It supposes a sandpile built up on a platform by adding sand randomly, a grain at a time. With the pile growing the slope will increase. Once the slope between two contiguous positions has reached a critical value, the pile will collapse and generate an avalanche. Eventually, the system evolves into a critical state where if any sand is added, there are avalanches at all length and time-scales which satisfy the power-law distribution. This dynamical attractor is the so-called SOC state. So far the concept of SOC has been applied successfully in many realms of science, such as earthquake, forest fire, ecological systems and so on [2–13].

What interests us is investigating SOC in the activity of neural populations. There are two motivations. Firstly, as one of the most complex systems, the human brain possesses more than 10^{14} neurons and should exhibit SOC under some suitable conditions [14, 15]. In fact, the strong analogies between the dynamics of the SOC model for earthquakes and that of neurobiology has been realized by Hopfield [15]. We hope that grasping the mechanics of SOC processes in the brain will be helpful in understanding the higher functions. The second motivation concerns the relationship between SOC and synchronization. Synchronized patterns of neural activity in the frequency range of 30–70 Hz have been found generally

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in the olfactory system, visual cortex and other brain areas [16–18]. They are supposed to play important computational roles, for example, it provides a mechanism for sensory segmentation [16, 19]. Since both SOC and synchronization are characterized by the large-scale spatiotemporal correlation, they may be closely related. We will reveal this relation in a general framework.

In this paper, we developed a cellular automaton which replicates the correlation structure of neural populations, that is, each cell behaves like an integrate-and-fire neuron and the coupling between them is the pulse-like interaction [15, 20–23]. Our model is shown to exhibit a robust SOC behaviour over a wide range of the parameters, and the SOC behaviour is accompanied by the large-scale synchronized pattern of the activity of the units.

2. The model and the result

According to the current neurodynamical picture of the brain, the essential features of its function are the following [24]. When the membrane potential of a neuron exceeds the threshold, the neuron sends out signals with the form of action potentials and then returns to the rest state (the neuron fires). The signal is transferred by the synapses to the other neurons, which has an excitatory or inhibitory influence on the membrane potential of the receiving cells according to whether the synapses are excitatory or inhibitory, respectively. The resulting potential, if it also exceeds the threshold, leads to the next step firing, and so on giving an avalanche. The non-firing neurons that are depolarized or hyperpolarized also relax to the rest state at a very slow rate compared to that in the firing case.

For comparisons between the biological knowledge and the artificial model, it is essential to model realistically. To grasp the mechanism and to do the simulation, however, it is necessary to disregarded superfluous details. Our model organization is a compromise between these considerations.

Let us consider a square lattice with nearest-neighbour coupling, which may represent a sheet of cells occurring in the neocortex [15]. With any cell i, we associate a continuous function V_i resembling the membrane potential. $V_i < 0$, $V_i = 0$ and $V_i > 0$ represent the neuron as hyperpolarized, in a rest state and depolarized, respectively. We choose the threshold $V_{th} = 1$. As to the property of the coupling (synapse), we randomly set them excitatory (or inhibitory) with weight P (or $1 - P$).

Each cell of the model behaves like an integrate-and-fire neuron. Suppose $V_i > 1$, the neuron i then fires and V_i decreases to zero. As a result of receiving the action potentials, the membrane potential of each of its nearest neighbours V_{nn} is changed. Without loss of generality, we assume that the change of V_{nn} is proportional to V_i, i.e. αV_i or $-\beta V_i$ depending on the coupling that is excitatory or inhibitory, respectively. Here α and β are the proportional constants.

Also considering the slow relaxation of the non-firing neurons to the rest state, we get the redistribution of the membrane potentials after the firing of neuron i as

$$V_i \to 0$$
$$V_{nn} \to \begin{cases} aV_{nn} + \alpha V_i & \text{(the coupling is excitatory)} \\ aV_{nn} - \beta V_i & \text{(the coupling is inhibitory)} \end{cases} \tag{1}$$

where a is a constant smaller than 1 denoting the remains of V_{nn} due to its slow relaxation after the firing.

In the recent study of SOC, there are generally two different driving rules that trigger the relaxation of the system. One is the local perturbation which is applied to the model

whose dynamics satisfies a conservation law [25, 26]. The other is the continuous driving (global perturbation) which is crucial to obtain SOC in those with non-conserving dynamics, such as the models of earthquake and forest fire [3, 5]. In the present model the dynamics is non-conserving owing to the slow relaxation and inhibition, so we choose the global perturbation to drive the model, that is, the membrane potentials of all neurons increase slowly and uniformly until one of them reaches the threshold and causes an avalanche; this process repeats again if the system quietens down. Note that the terminology 'non-conserving' refers to the redistribution of the membrane potential instead of the energy (see equation (1)). When applying continuous driving, we should distinguish two time-scales. One is defined by the duration between two successive avalanches. The other is the lifetime of the firing. By assuming the first time-scale to be much larger than the second, we separate them and regard the driving process as adiabatic: the system is allowed to quieten down before the global perturbation is used again. This adiabatic assumption has been generally used in many models including those of integrate-and-fire oscillators [3, 5, 20–23]. Then the continuous driving may be understood as the system is receiving a slow continuous signal from the external or other parts of the brain and, in this sense, continuous driving is also the natural choice of our model.

As for the boundary condition, we choose an open one, i.e. $V_i = 0$ on the boundary.

Finally the algorithm for simulating the above dynamical process is as follows:

(i) Initialize the membrane potential of each neuron below V_{th}, then randomly set each coupling excitatory or inhibitory with the weight P or $1 - P$, respectively.
(ii) Apply the global perturbation: finding out the maximum value V_{max} and adding $V_{th} - V_{max}$ to all neurons.
(iii) If there exits any $V_i \geqslant V_{th}$, redistribute the membrane potentials according to (1).
(iv) Repeat step (iii) until the system has quietened down.
(v) Apply step (ii) again.

Here, by using a random number generator, it is ensured that only one cell takes V_{max} in step (ii), which excludes the possibility of avalanche overlapping.

Evolving according to the above rule, the system will eventually arrive at a statistically stationary state which only depends on the parameters but not on the initial conditions.

Before showing the result, we would like to introduce another equivalent parameter. In the above, the couplings are differentiated as being excitatory and inhibitory with definite weights. Without causing any change in the statistical results, we can also regard all the couplings as equal by combining the excitatory and inhibitory effects together (this has been checked by our numerical calculation). Thus the change of V_{nn} after the firing of neuron i is

$$V_{nn} \to aV_{nn} + bV_i \tag{2}$$

where $b = P\alpha - (1 - P)\beta$ includes both the excitatory and inhibitory contributions. Afterwards we use b as the 'effective' parameter.

We note that when $a = 1$, expression (2) is just the dynamical rule of the cellular automaton of an earthquake [3].

To prove the SOC of our system, we measured the probability distribution of the size of avalanches, where the size is the number of neurons fired once during the avalanche. Our results are obtained after 10^6 drivings. Two different ways of varying the parameters have been investigated. One is changing b and setting $a = 0.98$ fixed, as shown in figure 1. When $b = 0.1$, the probability density decays exponentially with the size of the avalanches, which means there are only localized behaviours. As b is increased (by increasing the

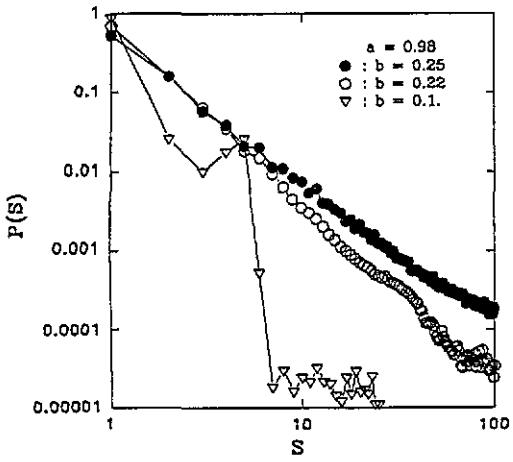

Figure 1. The probability distribution of the size of avalanches for a square 35 × 35 system is shown. The parameter a is fixed. When $b = 0.1$, the probability density decays exponentially. As b increases, there is a transition from localized to SOC behaviour. The critical exponents for $b = 0.22$ and 0.25 are obtained as 2.2 and 1.97, respectively.

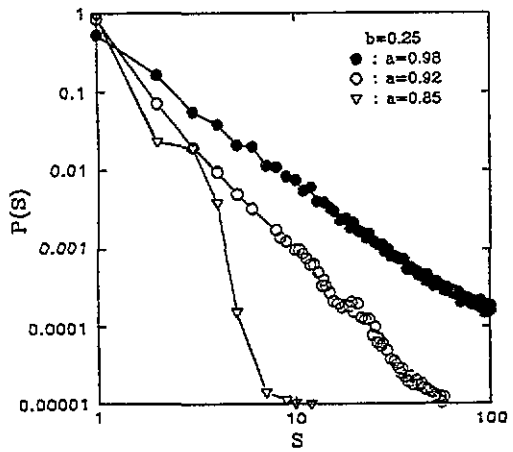

Figure 2. The similar result to figure 1 in the case of $b = 0.25$ and $a = 0.98$, 0.92 and 0.85. The critical exponents are 1.97 and 2.91 for $a = 0.98$ and 0.92, respectively.

weight P), the transition from localized to SOC behaviour occurs and the distribution of the size of the avalanches (S) satisfies the power law

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

where r is the critical exponent depending only on a and b and is scaling invariant. r will decrease if b is increased further. In figure 1, it is shown that $r = 2.2$ and 1.97 when $b = 0.22$ and 0.25, respectively.

The other case is varying a and letting $b = 0.25$ instead, as shown in figure 2. We also obtained the transition from localized to SOC behaviour when a is increased, and the critical exponent r will decrease when a is increased further.

To observe synchronous activity, we calculated the distribution of the membrane potential after an avalanche with the system separately in the SOC state and the state having localized behaviour, as shown in figure 3. It shows that the distributions both concentrate around a peak, which is different from the situation in the SOC models with local perturbation where there are many peaks and the number seems to be $2d$ [27]. This result agrees with the work of Grassberger on the distribution of stress in the SOC model of an earthquake [12]. It is very clear that the peak in the SOC state is much higher than that in the state with only localized behaviour, which indicates that in the SOC state there are many more units at

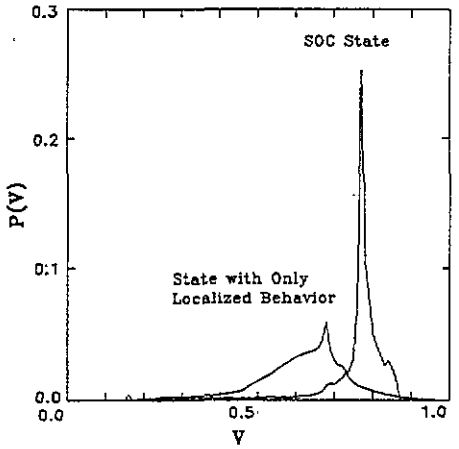

Figure 3. The distribution of membrane potential after an avalanche is shown, when the system is separately in SOC state ($a = 0.98$, $b = 0.25$) and the state having only localized behaviours ($a = 0.85$, $b = 0.25$). These distributions concentrate around a peak.

the same active level after an avalanche. We call the activities of neurons synchronized if their differences in membrane potential are smaller than 0.02. In figure 3, there are about 57% units in the SOC state whose activities are synchronized. From this point of view, the SOC process has been accompanied with the large-scale synchronization among the units. This close relationship between SOC and sychrony has also been found in the other systems [13, 23].

3. Conclusion and discussion

In conclusion we have introduced a cellar automaton which replicates the correlation structure of neural populations. The behaviour of the system is shown to depend on two parameters that describe the dynamics of the units. With the parameters increasing, the phase transition from localized to SOC behaviour takes place, and the SOC process is associated with the large-scale synchronization occurring among the elements.

Since synchronized activity patterns exist widely in neurobiological and other biological systems, much theoretical work has been done to grasp its mechanism [17–20, 25]. In our work it is interesting to find that the large-scale synchronization seems to be a coproduct of the SOC process. As the system self-organizes itself into the SOC state, its elements are also synchronized on a large scale.

The present model is an immensely oversimplified one with many details of neurobiology ignored. Also the notion of synchronization in the model does not match the real situation of neural activity well. Our work just indicates the close relation between SOC and synchronization in a rather artificial framework. This is far from the final answer. Further work taking into account more details is needed.

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