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Neuronal avalanches and criticality: A dynamical model for homeostasis

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Abstract

The dynamics of microelectrode local field potentials from cortical slice cultures shows critical behavior. A desirable feature of criticality is that information transmission is optimal in this state. We explore a biologically plausible neural net model that can dynamically converge on criticality and that can return to criticality if perturbed away from it. Our model assumes the presence of a preferred target firing rate, with dynamical adjustments of internodal connection strengths to approach this firing rate. We suggest that mechanisms for maintaining firing rate homeostasis may also maintain a neural system at criticality.

Keywords: Firing rate homeostasis; Criticality; Synaptic scaling

1. Introduction

The dynamics of local field potentials (LFPs) from cortical slice cultures has been shown to demonstrate critical behavior [2]. When cortical slices are cultured on 60 channel microelectrode arrays, activity consists of periods of quiescence broken by bursts of activity of any number of electrodes, which occur in clusters ("avalanches") of all possible sizes. A branching ratio, σ, can be defined as the number of electrodes that are excited after any other single electrode is excited, averaged over time and over all electrodes. In unperturbed cortical slice cultures, this ratio is nearly unity for hours at a time [3]. The condition $\sigma = 1$ represents the critical point.

The critical point so defined has a number of interesting properties. First, the number of electrodes involved in each avalanche (or cluster of excitations) is distributed according to a power law [2], similar to that seen in sand pile avalanches, nuclear chain reactions and many other natural phenomena [1]. Second, the Lyapunov exponent is nearly zero at the critical point, signifying stable neutral dynamics that is capable of exploring all possible LFP configurations (no local phase space attractors), and yet is not random [5]. Thirdly, an analysis of information transmission also shows that critical branching optimizes information throughput [2]. These properties all suggest that a neural network at critical branching is ideal for information processing. Larger branching ratios lead to excessive spread of input excitation. Information is then lost due to the intrinsic instability of the system. Smaller branching ratios, on the other hand, result in rapid dampening of excitation. Information is again lost, in this case by quenching of the input signal. Haldeman and Beggs were able to construct a highly parsimonious neural net model that captures these basic features [5].

Given the potential utility of remaining near the critical point, how do local cortical networks converge on this state? Evidence suggests that cortical networks become critical through self-organization even in vivo. During weeks of incubation, neurons in cortical slice cultures send out axons and form new synapses, rewiring the network. The fact that such networks arrive at the critical point without an external instructive signal indicates that local rules are sufficient to achieve criticality. In addition, acute cortical slices show power law distributions of avalanche sizes [2], suggesting that networks from the intact brain are also critical.

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Current thinking on long-term synaptic plasticity center on Hebbian mechanisms of synaptic potentiation or depression, which strengthen or weaken individual synapses [4]. These changes are rapidly induced (minutes to hours) and tend to destabilize neural circuits because they tend to make strong synapses stronger and weak synapses weaker [6]. A second mechanism involving post-synaptic rescaling of firing rate operates over a much longer time scale (hours to days) and tends to stabilize neural circuits by moving all neurons toward a target firing rate [8]. The first mechanism allows neural circuits to learn and store information. The second mechanism prevents the first mechanism from causing either runaway excitations or "freezing" of all neural activity in the system. The second mechanism represents a type of *firing rate homeostasis*.

In this paper we describe a network model of post-synaptic firing rate homeostasis that not only is dynamically stable, but that can be tuned to remain near the critical point. A system that can return to the critical point when perturbed away from it is doubly advantageous, not only in being stable, but in being stable around a state that is optimal for information processing.

2. Methods

If an electrode (node) j fires a burst, it sends an excitation to node i. Let the probability that node i will fire be given by $P(i,j)$. If there are $N = 60$ nodes, the branching ratio is then given by

$$\sigma = \frac{1}{N}\sum_{i=1}^{N}\sum_{j=1}^{N} P(i,j). \tag{1}$$

Let $A(i;t)$ be the activation level of the local field potential (LFP) at electrode i at time t. It gives the probability that neurons in the immediate neighborhood discharge simultaneously. Define a firing function $F(i;t)$ such that $F(i;t) = 1$ means that node i fires a burst at time t, and $F(i;t) = 0$ means that it is quiescent. The time evolution of $A(i;t)$ is given by

$$A(i;t+\delta t) = S(i;t) + \sum_{j=1}^{N} P(i,j;t)F(j;t), \tag{2}$$

where δt is the incremental time step, and $S(i;t)$ is the spontaneous activity at node i. We find it necessary to introduce a spontaneous activity for each node, to prevent the overall system from freezing in a no-fire mode after moments of total quiescence.

Firing rate homeostasis is achieved by increasing or decreasing $S(i;t)$ and $P(i,j;t)$ if the firing rate is either too low or too high compared to a target firing rate, $F_o = 1/\tau_o$:

$$S(i;t+\delta t) = \exp[-k_S(\langle F(i;t)\rangle - F_o)\delta t]\, S(i;t), \tag{3}$$

$$P(i,j;t+\delta t) = \exp[-k_P(\langle F(i;t)\rangle - F_o)\delta t]\, P(i,j;t). \tag{4}$$

Here $\langle\cdots\rangle$ represents a time average over a preceding time interval τ_{mem}, and k_S and k_P are dimensionless rate constants that control how quickly the $S(i;t)$'s and $P(i,j;t)$'s adjust to deviations away from the target firing rate F_o.

Every node is allowed to be connected to every other. The total number of nodes is taken to be $N = 60$. The timestep is taken to be $\delta t = 4$ ms. A refractory period of 20 ms is imposed after every firing at each node. The target single electrode firing period is $\tau_o = 6.25$ s. These parameters are chosen to be in agreement with experiment [2,3]. For lack of more detailed information, we take the initial $P(i,j;0)$ to be chosen from a flat, random distribution between 0 and 1. The results presented below are not sensitive to the firing rate averaging period τ_{mem}, as long as this period is longer than or equal to the target firing period. For simplicity, we take $\tau_{\text{mem}} = \tau_o$. The results below depend on the ratio k_S/k_P, and are not sensitive to the individual values of k_S and k_P, as long as k_S and k_P are both much less than 1. This condition guarantees that the relative time rate of change of $S(i;t)$ and $P(i,j;t)$ given by Eqs (3) and (4) are small. Convergence of each simulation is checked visually. Convergence takes on the order of 1–2 million timesteps. A total of 10–20 million timesteps is taken to assure the results are converged.

3. Results

Fig. 1 shows σ as a function of k_S/k_P for $k_P = 0.01$. Note that σ is nearly unity at $k_S/k_P = 0.5$ (i.e., this is the critical point). Note that σ increases monotonically as k_S/k_P increases, while the average spontaneous activity decreases. The standard deviation of σ about its mean is about 2% near the critical point.

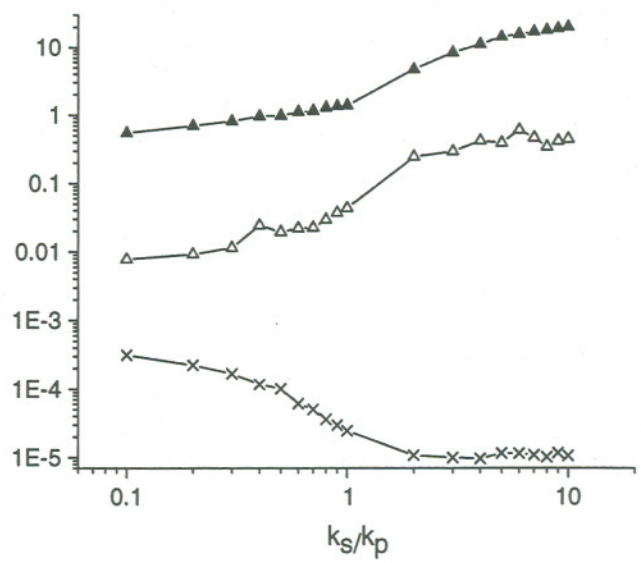

Fig. 1. Logarithm graphs. Solid triangles: σ, dimensionless units. Hollow triangles: standard deviation of σ. X's: the spontaneous rate $S(i)$ averaged over all nodes and over a time period of 625 s in units of probability of firing per timestep, where each timestep is 4 ms. The critical point is reached near $k_S/k_P = 0.5$.

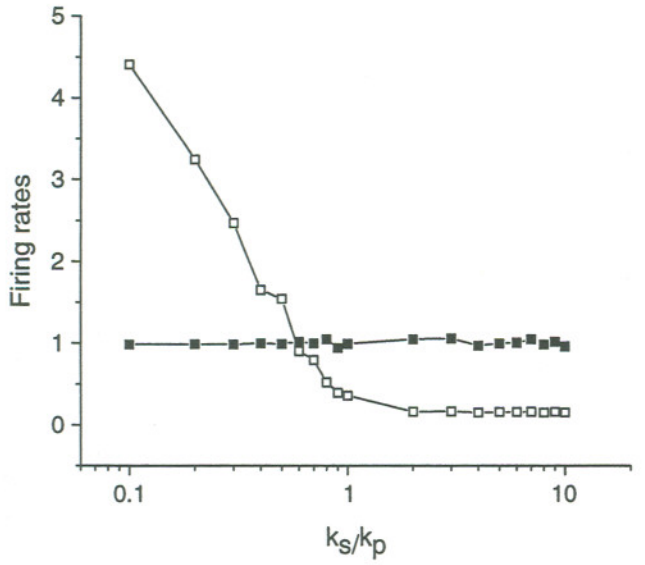

Fig. 2. Solid squares: the firing rate relative to the target firing rate, averaged over all nodes and over a time period of 625 s (dimensionless units). Hollow squares: the firing rate of the entire array, averaged over a time period of 625 s, in Hz. The critical point is reached near $k_S/k_P = 0.5$.

Fig. 2 shows the firing rate relative to the target firing rate, averaged over the prior 625 s ($100 \times \tau_o$). Homeostasis is maintained about the target firing rate. Fig. 2 also shows the firing rate of the entire array. Near the critical point ($k_S/k_P = 0.5$), the array firing rate is approximately 1.5 Hz, which is close to the experimental value of 1.3 Hz [2,3].

4. Discussion

We introduce a type of neural net dynamics based on continually adjusting spontaneous nodal activity and internodal connection strengths so that the firing frequency at each node always tries to approach a target frequency. Such a situation may plausibly arise from neuronal energy or kinetic constraints. We find that not only does the system converge on the target firing frequency, but that for certain values of our parameters k_S and k_P, a natural consequence of firing rate homeostasis is convergence to criticality. This property of critical homeostasis is of biological interest because criticality seems to be a general property of resting neocortical systems, and such a state is optimal for information transmission.

Our model rescales all the synapses of a given node either up or down by a multiplicative factor, in accordance with Turrigiano's criterion of synaptic scaling [6]. Our model does not adjust synapses within a single node individually. Thus, for a homogeneous population of neurons, the firing rate homeostasis mechanism does not alter the shape of the distribution of internodal connection strengths (the $P(i,j)$'s); it merely stretches or compresses this distribution so as to find a stable state. A Hebbian mechanism operating on a much faster timescale may possibly then be added to the firing rate homeostatic mechanism, without mutual interference [6,7]. The Hebbian mechanism would alter the distribution of internodal connection strengths, as part of a learning or information processing procedure. The firing rate homeostatic mechanism may then tune the new set of connectivities to return the system to a stable, optimal state.

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John Beggs received his B.S. in applied physics from Cornell, and later got his Ph.D. from Yale in neuroscience, where he did electrophysiological experiments in acute cortical slices under the guidance of Tom Brown. He went on to do a postdoctoral fellowship at the National Institutes of Health, studying spontaneous activity in cortical slice cultures using multielectrode arrays while working in the lab of Dietmar Plenz. He is currently an assistant professor of Physics at Indiana University in the Biocomplexity Institute, where he is interested in properties of local cortical networks.