

Spike-timing-dependent plasticity with axonal delay tunes networks of Izhikevich neurons to the edge of synchronization transition with scale-free avalanches

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Critical brain hypothesis has been intensively studied both in experimental and theoretical neuroscience over the past two decades. However, some important questions still remain: (i) What is the critical point the brain operates at? (ii) What is the regulatory mechanism that brings about and maintains such a critical state? (iii) The critical state is characterized by scale-invariant behavior which is seemingly at odds with definitive brain oscillations? In this work we consider a biologically motivated model of Izhikevich neuronal network with chemical synapses interacting via spike-timing-dependent plasticity (STDP) as well as axonal time delay. Under generic and physiologically relevant conditions we show that the system is organized and maintained around a synchronization transition point as opposed to an activity transition point associated with an absorbing state phase transition. However, such a state exhibits experimentally relevant signs of critical dynamics including scale-free avalanches with finite-size scaling as well as branching ratios. While the system displays stochastic oscillations with highly correlated fluctuations, it also displays dominant frequency modes seen as sharp peaks in the power spectrum. The role of STDP as well as time delay is crucial in achieving and maintaining such critical dynamics, while the role of inhibition is not as crucial. In this way we provide definitive answers to all three questions posed above. We also show that one can achieve supercritical or subcritical dynamics if one changes the average time delay associated with axonal conduction.

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INTRODUCTION

Since its inception nearly two decades ago, the critical brain hypothesis has gained a considerable amount of attention in the literature [1–3]. Although it has encountered some skepticism at times [4], it has now grown to a relatively mature field with substantial body of theoretical and experimental evidence to support it [4–11]. Brain criticality is thought to underlie many of its fundamental properties such as optimal response, learning, information storage, as well as transfer [12]. The original ideas of brain criticality came out of studies of self-organized criticality, where a threshold dynamics leads to a balance between slow drive and fast dissipation in open nonequilibrium systems and thus observation of critical dynamics [13]. It is now generally believed that long-term evolution has led to a balance between excitatory as well as inhibitory tendencies which place the brain “on the edge”, i.e. a critical point. However, this does not necessarily answer the problem of stability of the critical state, as some neurophysiological mechanism is needed to maintain the system near the critical point against many possible perturbative effects.

It seems like there are some important theoretical issues which have remained open in regards to brain criticality: (i) What exactly is the phase transition which determines the critical point? Traditionally, this has been assumed to be the absorbing-state phase transition motivated by the studies of self-organized criticality [14, 15]. However, in some recent studies, it has been indicated that the brain is maintained near a synchronization tran-

sition [10, 16]. We note that some authors have also argued for the existence of the extended critical region similar to that of “Griffiths phase” [17–20]. However, such critical regions also typically occur near the absorbing phase transition where the system transitions from an inactive phase to an active phase. (ii) What is the self-organizing mechanism which leads to, and maintains the system in a critical state? As mentioned above the balance between excitatory and inhibitory tendencies are thought to be the long time solution to this question. However, physiological mechanism such as synaptic plasticity are also considered to be important mechanism to maintain the nervous system in a balanced state on shorter time scales. Clearly, extended criticality can also alleviate such a problem to a certain extent as criticality is observed for a range of parameter instead of a particular point. (iii) If the brain is in the critical state with its associated scale-invariant behavior, how could it also display definitive rhythmic behavior via brain oscillations?

Brain plasticity is increasingly being recognized as an important and fundamental property of a healthy nervous system. In particular, spike-timing-dependent-plasticity (STDP) is an important mechanism which can modify synaptic weights on very short time scales. Therefore, it seems reasonable to invoke STDP as a self-organizing mechanism. In a STDP protocol, the strength of a synapse is modified based on the relative spike-timing of its corresponding pre- and post-synaptic neurons, i.e., STDP incorporates the causality of pre- and post-synaptic spikes into the synaptic strength modifications. If the pre-synaptic neuron spikes first and leads to

the post-synaptic neuron to spike shortly afterward, then the synapse is potentiated. Reversely, if the pre-synaptic spike follows the post-synaptic spike the synapse will be depressed [21–24]. The competition between coupling and decoupling forces arising from successive potentiation and depression of synapses tunes the neural network into a balanced dynamical state.

Our work in this paper is motivated by the above considerations. In particular, we propose to study a biologically plausible model of cortical networks, i.e. Izhikevich neurons, along with neurophysiological regulatory mechanism such as STDP with suitable axonal conduction delays in order to answer some of the above posed questions. Interestingly, we find that our regulatory system self-organizes the neuronal network to the “edge of synchronization” in physiologically meaningful parameter regime. We first establish some of the characteristics of such a steady state. More importantly, we look for characteristics of critical dynamics in such a minimally synchronized steady state. Motivated by various experiments, we look for neuronal avalanches, branching ratios, as well as power spectrum of activity time-series. We find that such a system on the edge of synchronization exhibits significant indications of critical dynamics including scale-invariant avalanches with finite-size scaling. Our results provide definitive answers to the above questions in a biologically plausible model of neuronal networks.

In the following section, we describe the model we use for our study. Results of our numerical study is presented in section III, and we close the paper with some concluding remarks in section IV.

MODEL AND METHODS

The studied cortical networks consist of N spiking Izhikevich neurons which interact by transition of chemical synaptic currents with axonal conduction delays. The dynamics of each neuron is described by a set of two differential equations [25]:

$$\frac{dv_i}{dt} = 0.04v_i^2 + 5v_i + 140 - u_i + I_i^{DC} + I_i^{syn} \quad (1)$$

$$\frac{du_i}{dt} = a(bv_i - u_i) \quad (2)$$

with the auxiliary after-spike reset:

$$\text{if } v_i \geq 30, \text{ then } v_i \rightarrow c \text{ and } u_i \rightarrow u_i + d \quad (3)$$

for $i = 1, 2, \dots, N$. Here v_i is the membrane potential and u_i is the membrane recovery variable. When v_i reaches its apex ($v_{max} = 30$ mV), voltage and recovery variable

are reset according to Eq.(4). a , b , c and d are four adjustable parameters in this model. Tuning these parameters, Izhikevich neuron is capable of reproducing different intrinsic firing patterns observed in real excitatory and inhibitory neurons [25]. We set these parameters so that excitatory neurons spike regularly and inhibitory neurons produce fast spiking pattern [25–27].

The term I_i^{DC} is an external current which determines intrinsic firing rate of uncoupled neurons. Regularly spiking Izhikevich neurons exhibits a Hopf bifurcation at $I^{DC} = 3.78$ [28]. We choose values of I_i^{DC} randomly from a Poisson distribution with the mean value 10. The term I_i^{syn} represents the chemical synaptic current delivered to each post-synaptic neuron i [29]:

$$I_i^{syn} = \frac{V_0 - v_i}{D_i} \sum_j g_{ji} \frac{\exp(-\frac{t-(t_j+\tau_{ji})}{\tau_s}) - \exp(-\frac{t-(t_j+\tau_{ji})}{\tau_f})}{\tau_s - \tau_f} \quad (4)$$

Here D_i is the in-degree of node i , t_j is the instance of last spike of pre-synaptic neuron j , and τ_{ji} is the axonal conduction delay from pre-synaptic neuron j to post-synaptic neuron i . If axonal delays are not taken into account, then $\tau_{ji} = 0$ for all $j \neq i$. Axonal delay values of τ_{ji} are chosen randomly from a Poisson distribution with mean value $\tau = \langle \tau_{ji} \rangle$. τ_f and τ_s are the synaptic fast and slow time constants and V_0 is the reversal potential of the synapse. If inhibition is included, then motivated by the properties of cortical networks [30], we set population density of inhibitory neurons to twenty percent, i.e. $\alpha = 0.2$ while the initial strength of inhibitory synapses are chosen four times the strength of excitatory synapses. Therefore, the excitation-inhibition ratio is balanced. $\alpha = 0$ indicates that we are only considering a network of excitatory neurons. g_{ji} is the corresponding element of the adjacency matrix of the network which denotes the strength of synapse from pre-synaptic neuron j to post-synaptic neuron i . Each type of synapses are initially static and have equal strength. $g_{ji} = g_s$ if neurons j and i are connected and the synapse is excitatory, $g_{ji} = 4g_s$ if neurons j and i are connected and the synapse is inhibitory, and $g_{ji} = 0$ otherwise. When we turn the STDP on, strength of *excitatory* synapses are modified according to a soft-bound STDP rule [21–24], while the strength of inhibitory synapses are fixed. If pre-synaptic neuron j fires a spike at time $t = t_{pre}$, then the strength of synapse is modified to $g_{ji} \rightarrow g_{ji} + \Delta g_{ji}$, where:

$$\Delta g_{ji} = \begin{cases} A_+(g_{max} - g_{ji})e^{-\frac{\Delta t - \tau_{ji}}{\tau_+}} & \text{if } \Delta t > \tau_{ji} \\ -A_-(g_{ji} - g_{min})e^{-\frac{\Delta t - \tau_{ji}}{\tau_-}} & \text{if } \Delta t \leq \tau_{ji} \end{cases} \quad (5)$$

Here, $\Delta t = t_{post} - t_{pre}$ is the time difference of last post- and pre-synaptic spikes, A_+ and A_- determine the maximum synaptic potentiation and depression, τ_+ and τ_-

TABLE I: Values of constant parameters used in this study.

| | | | | | | | | |
|-------------------|-----------------|----------------|----------------|------------------|----------------|-----------------|----------------|--------------|
| Izhikevich neuron | $a_{ex} = 0.02$ | $b_{ex} = 0.2$ | $c_{ex} = -65$ | $d_{ex} = 8$ | $a_{in} = 0.1$ | $b_{in} = 0.2$ | $c_{in} = -65$ | $d_{in} = 2$ |
| Synaptic current | $\tau_f = 0.2$ | $\tau_s = 1.7$ | $V_{0,ex} = 0$ | $V_{0,in} = -75$ | | | | |
| STDP rule | $A_+ = 0.05$ | $A_- = 0.05$ | $\tau_+ = 20$ | $\tau_- = 20$ | $g_{min} = 0$ | $g_{max} = 0.6$ | | |

determine the temporal extent of the STDP window for potentiation and depression, and g_{min} and g_{max} are the lower and upper bounds of synaptic strength. The values of all parameters for Izhikevich neuron, synaptic current and STDP rule are listed in Table I.

We consider a temporally shifted STDP window for which the boundary separating potentiation and depression does not occur for simultaneous pre- and post-synaptic spikes, but rather for spikes separated by a small time interval [31]. We set the value of this shift equal with the actual axonal delay for each synapse. This rule retrieves the conventional STDP rule when no time-delay is considered, $\tau_{ji} = 0$. We have plotted the STDP temporal window function $\Delta g = f(\Delta t)$ and its shift in Fig.1. This temporal shift causes synchronous or nearly-synchronous pre- and post-synaptic spikes to induce long-term depression.

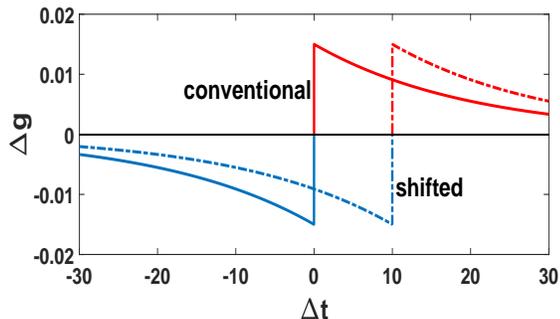


FIG. 1: Conventional (solid line) and shifted (dashed line) STDP temporal window function $\Delta g = f(\Delta t)$. Blue parts denote depression and red parts denote potentiation. Units of Δt is ms.

We integrate the dynamical equations using fourth-order Runge-Kutta method with a time step $h = 0.01$ ms and obtain $v_i(t)$. We typically evolve the entire system for a long time and make sure that the system has reached a stationary state. We then perform our measurements and calculations. We obtain the instants of firings of all neurons and then assign a phase to each neuron between each pairs of successive spikes [32]:

$$\phi_i(t) = 2\pi \frac{t - t_i^m}{t_i^{m+1} - t_i^m} \quad (6)$$

while t_i^m is the time that neuron i emits its m^{th} spike.

We define a time-dependent order parameter:

$$S(t) = \frac{2}{N(N-1)} \sum_{i \neq j} \cos^2\left(\frac{\phi_i(t) - \phi_j(t)}{2}\right) \quad (7)$$

This order parameter measures the collective phase synchronization at time t . $S(t)$ is bounded between 0.5 and 1. If neurons spike out-of-phase, then $S(t) \simeq 0.5$, when they spike completely in-phase $S(t) \simeq 1$ and for states with partial synchrony $0.5 < S(t) < 1$. The global order parameter S^* is the long-time-average of $S(t)$ at the stationary state after the influence of STDP ($S^* = \langle S(t) \rangle_t$). We note that the intricate details of the model along with the need to obtain long-time dynamics of the system, limit our computational abilities. We have therefore performed simulations for $100 < N < 1000$. We find that our general results and conclusions are independent of the system size used and therefore report most of our results for $N \approx 500$. In the next section we will present a systematic study of the system above, paying particular attention to the effect of STDP, time delay, and inhibition.

RESULTS

Spiking Izhikevich neurons with static chemical synapses exhibit a continuous transition to phase synchronization upon increasing synaptic strength, i.e. the amount of global synchrony depends on the average synaptic strength [28]. Now, consider the simple case of an all-to-all network of excitatory neurons without axonal delays. STDP is off initially. $S(t)$ timeseries for different values of g_s are illustrated in Fig.2(a). It is observed that $S(t)$ depends on g_s as is expected. Next, we turn on the STDP at $t = 5$ s. Interestingly, it is seen that $S(t)$ timeseries evolve to a common state regardless of their initial values. Thus, as STDP modifies the synaptic strengths, neural network organizes into a final state with a specific global phase synchronization S^* independent of the initial synaptic strengths. Our investigations reveal that this is a generic condition emerging in neural networks with different underlying structures. We also find that the amount of S^* is independent of many parameters including the amplitudes and time extents of STDP rule, and intrinsic firing rate of neurons. However S^* depends drastically on the average value of axonal conduction delays. Fig.2(b) shows that increasing τ leads to a phase

transition from strongly synchronized states with $S^* \simeq 1$ to asynchronous states with $S^* \simeq 0.5$, for neural networks with $\alpha = 0$ and $\alpha = 0.2$. Fig.2(b) also shows that inhibition has a secondary role in the amount of steady state synchronization, S^* , as compared to axonal delay, τ . Important to our purposes, it shows that for $\tau = 10$ ms the systems stand at the boundary of phase synchronization for both α values. Note the importance of time delay as it causes STDP to depress (weaken) the synchronous neurons, thus reducing the amount of S^* in the system.

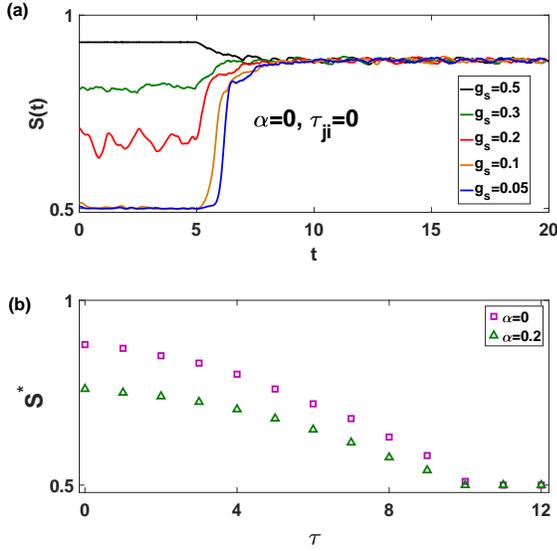


FIG. 2: (a) Effect of STDP on the time evolution of $S(t)$ for all-to-all networks of excitatory spiking neurons with different g_s . The unit of time axis is in seconds. (b) Dependence of S^* on τ for $\alpha = 0$ and $\alpha = 0.2$.

In order to further investigate the properties of Izhikevich neuronal networks, we consider four different networks of $N = 500$: (1) a network of purely excitatory neurons without time-delay ($\alpha = 0, \tau_{ji} = 0$), (2) a network of purely excitatory neurons with axonal conduction delays ($\alpha = 0, \tau = 10$ ms), (3) a network of excitatory and inhibitory neurons without time-delay ($\alpha = 0.2, \tau_{ji} = 0$), and (4) a network of excitatory and inhibitory neurons with axonal conduction delays ($\alpha = 0.2, \tau = 10$ ms). We have studied networks with different τ values, but we display mostly the results in cases for which all delays are zero ($\tau_{ji} = 0$) and $S^* \gg 0.5$ as well as those with $\tau = 10$ ms for which $S^* \rightarrow 0.5^+$. We note that while our results (Fig.2) show that $\tau = 10$ ms is an interesting case of transition point, such an actual value for axonal delay is experimentally meaningful [33]. We turn on STDP at $t = 5$ s in a complete network and monitor its influence on different features of each system.

Synchronization and average synaptic weights

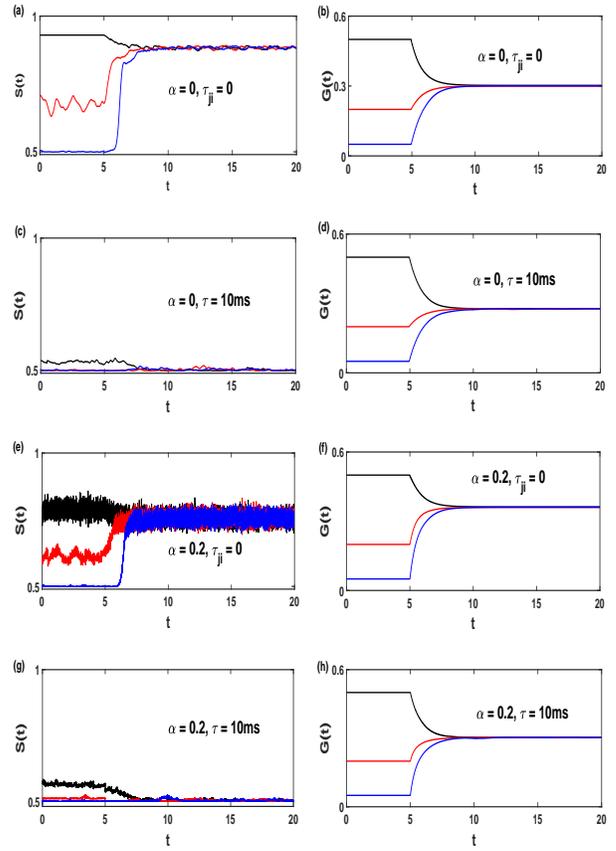


FIG. 3: Timeseries of $S(t)$ and $G(t)$ and the influence of STDP on them. The unit of time axis is in seconds. STDP is turned on at $t = 5$ s. In each panel different line colors demonstrate different static synaptic strengths, $g_s = 0.5$ (black), $g_s = 0.2$ (red) and $g_s = 0.05$ (blue).

The influence of STDP on the timeseries $S(t)$ in different conditions is illustrated in the left column of Fig.3. Each panel contains three plots with different values of g_s , i.e. the initial synaptic weights. When STDP is off, $S(t)$ depends on g_s . Turning STDP on, each system reaches a final state with a specific amount of synchronization S^* , regardless of initial level of order (regardless of g_s). However, S^* depends on τ and α . Systems (1) and (3) reach a strongly synchronized states with $S^* \simeq 0.88$ and $S^* \simeq 0.75$, respectively. Implementation of conduction delays drive the dynamics toward lower levels of order. Systems (2) and (4) with $\tau = 10$ ms lead to states at the edge of transition with $S^* \simeq 0.509$ and $S^* \simeq 0.503$, respectively. The right column of Fig.3 represents the timeseries of the average strength of excitatory synapses, for the corresponding system in the left column represented by, $G(t) = \frac{1}{N_L} \sum_{j \neq i} g_{ji,ex}(t)$, where N_L is the number of existing excitatory links. It is observed that

at the final states $G(t) \simeq 0.3$ for all the systems. It is interesting that the final average value of synaptic weight is independent of the amount of inhibition and and/or axonal delay, as well as initial distribution. However, the main point here is that the amount of synchronization in the system is not solely determined by average synaptic strength but crucially depends on axonal conduction delay, and to a lesser degree on inhibition.

Synaptic distributions

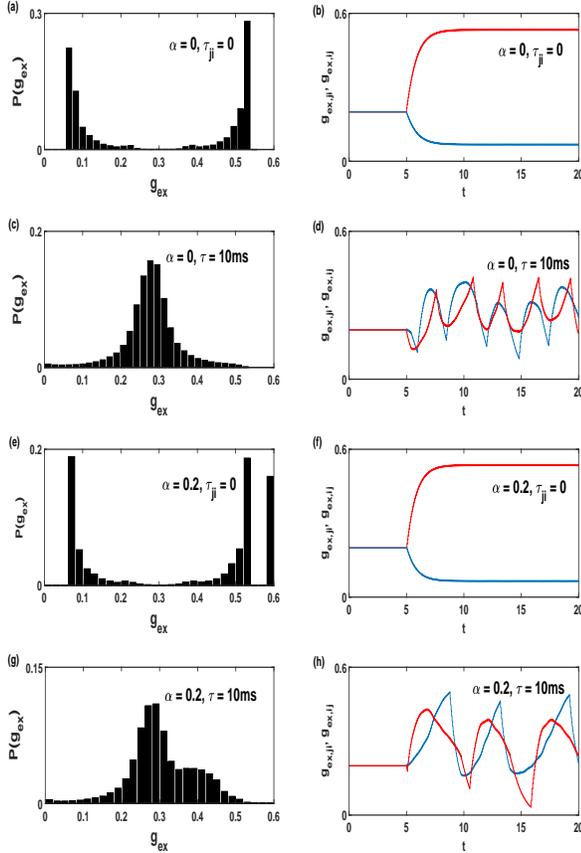


FIG. 4: Distribution of the excitatory synaptic strength at the stationary state of the systems after the influence of STDP (left) and time evolution of a pair of reciprocal synapses (right). The unit of time axis is in seconds.

It is somewhat unexpected that similar average synaptic weights would lead to decidedly different synchronization patterns. The answer is in the form of the actual distributions of the weights. In one scenario the average is the most likely value (unimodal distribution) and in the other case is the least likely value (bimodal distribution). The probability distribution function of excitatory synaptic strengths $P(g_{ex})$ (in the steady state) for each system is shown in the left column of Fig.4. Also, the

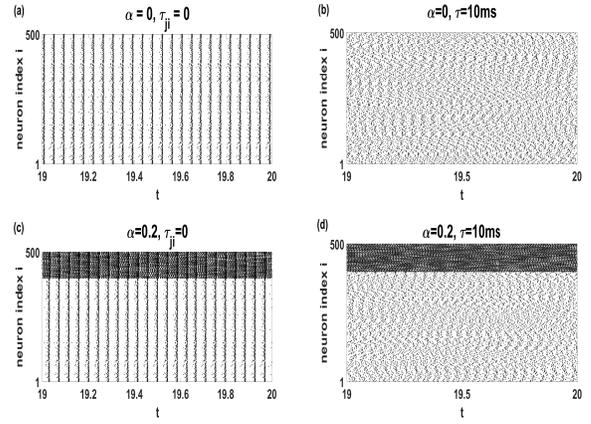


FIG. 5: Raster plots of the neural networks with different values of τ and α at the stationary states after influence of STDP. The unit of time axis is in seconds.

right column of this figure illustrates time evolution of strength of a pair of reciprocal synapses. At the absence of axonal delays, STDP produces a bimodal distribution of synaptic strengths (Figs.4(a) and 4(e)) which is incompatible with the experimentally observed distributions of synaptic strength. However, addition of time-delays to the neural network modifies this condition. Simultaneous presence of STDP and time-delays produce a unimodal distribution of synaptic strengths (Figs.4(c) and 4(g)) resembling those measured in cultured and cortical networks [34, 35].

Emergence of these different distributions of synaptic strengths is associated with the amount of phase synchronization in the networks. When neurons interact without time-delay, the final state of the system is strongly synchronized. Therefore, for each pair of symmetric links, STDP depresses the link in one direction and potentiates the link in the opposite direction as in Figs.4(b) and 4(f). Thus all symmetric connection would be broken into unidirectional connections after a while in this case. This leads to a bimodal distribution of synaptic strengths whether the network consists of purely excitatory neurons or a mixture of excitatory and inhibitory neurons. With the inclusion of time-delay in the system the level of order declines as it also causes to preserve symmetric connections between each pair of neurons [36]. Although the strength of synapses fluctuates over time (Figs.4(d) and 4(h)), both links in opposite directions remain active. This leads to a unimodal distribution of synaptic strengths.

Indicators of criticality

So far we have seen that STDP along with reasonable time delay (and inhibition) will lead the system on the

edge of synchronization. However, being on the edge of synchronization could be caused by vastly different spiking patterns [28]. More importantly for the purpose of the present study, we would like to know whether such a state of minimal synchronization has any experimentally relevant indications of criticality. In this subsection we will address such issues.

Raster plots of neural networks with different values of α and τ (in the steady state) are displayed in Fig.5. When time-delay is ignored, neuronal spikes are highly ordered (Figs.5(a) and 5(c)). This is not the real state of a healthy nervous system. However, addition of axonal conduction delay modifies the amount of global order in the networks. Simultaneous effect of STDP and a suitable axonal conduction delays decrease global coherence in neural oscillations (See Figs.5(b) and 5(d)). In Figs.5(c) and 5(d), inhibitory neurons indexed as 401 – 500, spike with a higher rate as compared to excitatory neurons 1 – 400.

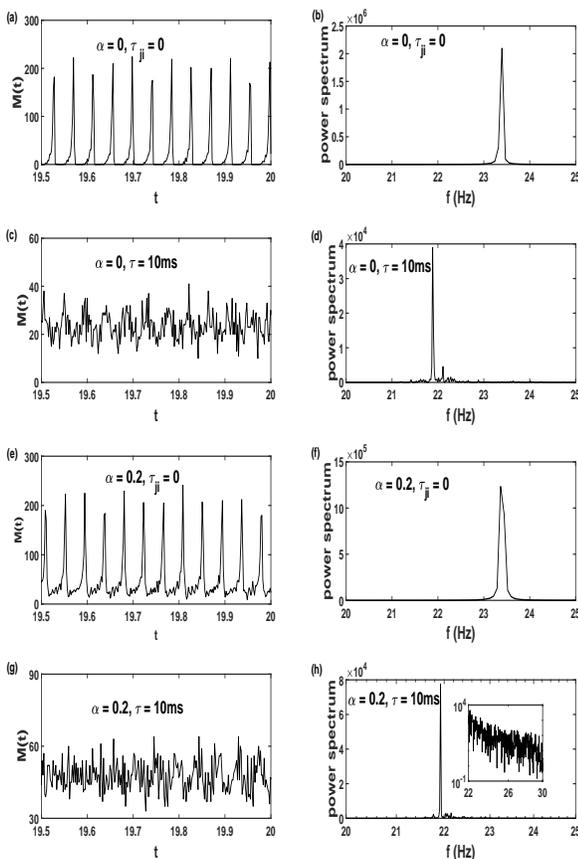


FIG. 6: Timeseries of network activity $M(t)$ in the stationary state after the influence of STDP (left) and their power spectrum (right). The unit of time axis is in seconds. The inset of (h) shows the same on a log-log scale.

The amount of order parameter S^* and the raster plots are reasonable evidences indicating the system with

$\tau = 10\text{ms}$ organizes to the edge of synchronization transition with minimal value of S^* . We now present experimentally relevant results which indicate that such a system is in a critical state. We first consider the network activity timeseries $M(t)$ which is defined as the number of neuronal spikes at time t , as well as its power spectrum. These plots are illustrated in Fig.6. The network activity oscillates regularly in systems without time-delay for which phase synchronization is strong (Figs.6(a) and 6(e)). Therefore the power spectrum of these systems exhibit a sharp peak at $f \simeq 23.5\text{Hz}$ (Figs.6(b) and 6(f)). While neurons are delay-coupled the oscillations of $M(t)$ are irregular (Figs.6(c) and 6(g)). Despite this deceptive irregularity, the power spectrum exhibits a large peak at frequency $f \simeq 21.5\text{Hz}$ (Figs.6(d) and 6(h)) along with a range of other frequencies. This dominant peak reveals that rhythmic oscillations are still robust at these neural networks. The inset of Fig.6(h) shows a log-log plot which indicates that the spectrum has a decaying tail in the system for which $\alpha = 0.2$ and $\tau = 10\text{ms}$. Note that the amplitude of oscillations of $M(t)$ depends on the level of phase synchronization. The stronger the neurons are synchronized, the larger is the amplitude of $M(t)$ oscillations, i.e. note the scale of the power spectrum on the y-axis.

Scale-invariant statistics of neural avalanches is thought to be the most important indicator of critical brain dynamics. Hence, the network displays spontaneous activity of various sizes s , known as neural avalanches, which exhibit scale-free distribution, i.e. $P(s) \sim s^{-\beta}$ [1]. By monitoring the spiking activity of our systems, we can identify outbursts of spikes the number of which is associated with the size of avalanches. An avalanche begins when the network activity exceeds a threshold M_{th} and ends when it turns back below that threshold. Here, we set the threshold to be equal with the mean value of activity in the system. s is defined as the total number of spikes during this avalanche. Criticality is supposed to be indicated by a power-law behavior $P(s) \sim s^{-\beta}$ and a finite-size cut-off which diverges as system size diverges ($N \rightarrow \infty$).

We consider neural networks with $\alpha = 0.2$ and three different τ values, i.e. $\tau = 14\text{ms}$, $\tau = 10\text{ms}$ and $\tau = 8\text{ms}$. From the synchronization point of view, Fig. 2(b), these systems would be subcritical, critical, and supercritical. Each network is also simulated with different network sizes N . For any given set of parameters the network is simulated for a considerably long time, producing a large number of avalanches. Probability distribution functions of avalanche sizes for such networks is illustrated in the left column of Fig.7. For neural networks with $\tau = 14\text{ms}$, $P(s)$ decays with a characteristic scale which is an indicator of subcritical behavior (Fig.7(a)). Note how this scale saturates as system size increases. For networks with $\tau = 8\text{ms}$, $P(s)$ exhibits a bump for large s which is an evidence of supercritical behavior (Fig.7(e)). Here, large

avalanches are more likely to occur than intermediate size avalanches. Interestingly, in networks with $\tau = 10\text{ms}$ $P(s)$ exhibits a power-law behavior $P(s) \sim s^{-\beta}$ and a finite-size cut-off which increases relative to the system size (Fig.7(c)).

Emergence of power-law behavior in a finite system does not necessarily prove criticality of the system. To verify criticality, we perform a finite-size scaling of our data for different network sizes N (inset of Fig.7(c)). We observe that indeed we obtain a good collapse for the system sizes considered in this study. Incidentally, our finite-size scaling collapse allows us to calculate the β value more reliably where we obtain the critical exponent $\beta = 1.4$ which is close to the accepted experimental value $\beta \approx 1.5$ obtained by various studies including the original neuronal avalanche study of [5].

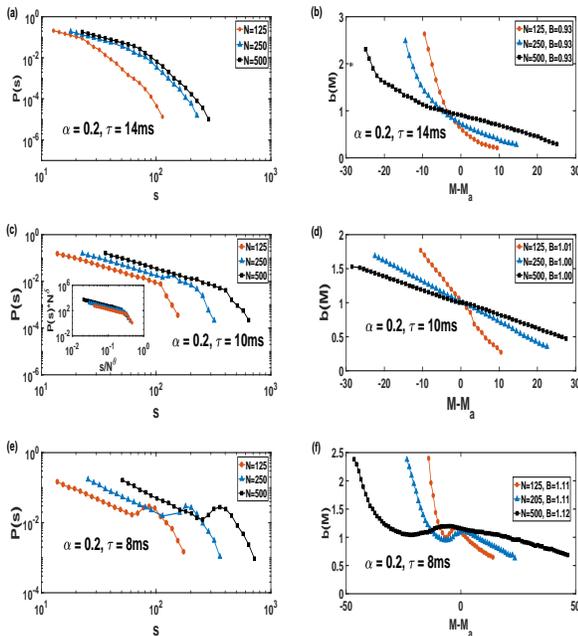


FIG. 7: Distribution function of size of avalanches, and activity-dependent branching-ratio $b(M)$ vs $M - M_a$, for various network sizes N : (a) and (b) subcritical, (c) and (d) critical, (e) and (f) supercritical. Inset of (c) shows the finite-size-scaling collapse of size of avalanches in the critical system for $\theta = 1.20$ and $\delta = 1.68$. Thus the critical exponent is $\beta = \frac{\delta}{\theta} = 1.4$. On the right column, the average branching-ratio B for each network with size N is reported in the legend.

Another important quantity to characterize critical dynamics is *activity-dependent* branching ratio [37]. Essentially, this function gives the (relative) expectation value of the timeseries in the next time step for a given amount of activity at the present time step. More precisely, it is defined as, $b(M) = E\{\xi_M/M\}$. The variable ξ_M is the value of the next signal given that the present one is equal to M , so $\xi_M = \{M(t+dt)|M(t) = M\}$ [37]. Since a critical system is on the edge and is inherently

unpredictable, $b(M) \approx 1, \forall M$. For a finite system one expects a similar result with the additional consideration that, with increasing system size, the range of activity M should increase and that $b(M)$ should asymptotically approach 1. Therefore, one expects $b(M) < 1$ to generally indicate subcritical behavior, while $b(M) > 1$ to indicate supercritical behavior. In fact, $b(M)$ has been used to ascertain criticality in a wide range of systems including sandpile models of SOC or solar flares [37] as well as neural networks [20, 38].

We obtain the activity-dependent branching-ratio $b(M)$ using timeseries $M(t)$. The right column of Fig.7 displays $b(M)$ plots for each one of subcritical, critical and supercritical systems for different system sizes N (Figs.7(b), 7(d) and 7(f)). Note that the plots are centered around their respective average activity M_a . Only in the critical case (Fig.7(d)) do we observe $B(M_a) = 1$. However, more importantly, we see $b(M)$ increases its range and decreases its slope (towards zero) with increasing system size, consistent with critical dynamics of the network. In the two other cases, no such behavior is observed. For a more common branching ratio, one calculates the average value of $B(M)$, i.e. $B = 1/(M_{max} - M_{min}) \int_{M_{min}}^{M_{max}} b(M)dM$. We find $B \simeq 1$ ($\tau = 10\text{ms}$), $B \simeq 0.93$ ($\tau = 14\text{ms}$), and $B \simeq 1.1$ ($\tau = 8\text{ms}$) again indicating critical, subcritical, and supercritical dynamics accordingly. The average branching ratios are reported in the legend of the corresponding plots in Fig.7.

We have therefore shown how the system with STDP and physiologically relevant inhibition and axonal delays will evolve to a unimodal distribution of synaptic weights starting from a complete uniform network. The resulting state is a state on the edge of synchronization transition (not an activity transition) which nevertheless shows experimentally relevant indicators of critical dynamics including power-law avalanches with finite-size scaling as well as branching ratios. We also show how such indicators of criticality disappear as one moves away from the edge of synchronization transition via change in average delay times.

CONCLUDING REMARKS

In this paper we showed that invoking neurophysiological regulatory mechanisms such as temporally shifted STDP and specific amounts of axonal conduction delays ($\tau = 10\text{ms}$) in a biologically plausible model of cortical networks put the system in a critical state at the neighborhood of synchronization transition point. In this state the system exhibits robust rhythmic behavior along with power-law distributions of avalanche sizes. Furthermore, the behavior of activity-dependent branching-ratio confirms the criticality of system in this state as well. However for smaller or larger values of axonal conduc-

tion delays neural networks self-organize into supercritical or subcritical states, respectively. While the state of the network is off-critical, neither the statistics of sizes of avalanches nor branching-ratio exhibit the signs of criticality.

Coexistence of rhythmic oscillations and scale-invariant avalanches is important for development of cortical layers [39]. Evidence for this coexistence has been found in experimental investigations [39, 40]. Also in theoretical studies, this phenomenon has been reported to occur as a result of balance between inhibition and excitation [41], as well as in a periodically driven SOC model [42]. The neurophysiological mechanisms leading to this intricate dynamics in the cortex is of fundamental importance in neuroscience. Here, we revealed that such intricate dynamics emerges as a result of intrinsic regulatory mechanisms like STDP and axonal conduction delays. More strictly, we obtained self-regulated criticality along with coexistence of rhythmic oscillations and scale invariant activity in a biologically relevant model.

We began this paper by posing three open questions regarding the critical brain hypothesis. Our results have provided interesting answers to all three questions. (i) The critical point and corresponding phase transition that the brain organizes itself into is not the usual activity and/or absorbing phase transition, but the synchronization phase transition. (ii) The self-organizing mechanism which tunes and maintains the system around such critical point is a standard neurophysiological regulatory mechanism of a temporally shifted STDP. (iii) The existence of individual neuronal oscillations which self-organize to a highly correlated but weakly synchronized collective state is responsible for a dominate oscillatory mode in addition to scale-free fluctuations.

We have studied neural networks with different topologies, various initial conditions, as well as various choices of STDP parameters and observed that our results are generally the same upon all such changes. We have also examined that hard-bound STDP leads to similar results, except for the distribution function of synaptic strengths that would be bimodal regardless of all conditions implemented in the neural network.

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