Examining the Effects of Noise Contamination during Epilepsy using Low Complexity Coupled Rulkov Maps and Spike Timing Dependent Plasticity

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Abstract—Epilepsy is a neurological disorder provoked from an imbalance between long-term potentiation (LTP) and long-term depression (LTD) of the synapses. This results in an abnormal synchrony of the neurons in the brain, visible in the electroencephalogram (EEG). In certain types of epilepsy such as tonic-clonic (grand mal) seizures synaptic disturbance occurs. In this work, we explore the effect of how noise contamination in a coupled lattice of neurons alters the synchrony in such circuits. We build a low complexity model using Rulkov maps to model the spiking activity and Spike Timing Dependent Plasticity (STDP) to model the LTP and LTD. From the simulation results, we observed that the chaotic noise STDP model has resistance characteristic of diverging of membrane potential.

1. Introduction

Epilepsy is a neurological disorder effecting 1 in 100 people worldwide [1]. During epilepsy, the excitation and inhibition of neurons becomes unbalanced leading to supra-normal excitability which leads to seizure. When epilepsy occurs the neurons are said to undergo an “abnormal synchrony” which is prevalent in the electroencephalogram (EEG). There are approximately 40 different types of epilepsy each having their own very different EEG signature. In certain types of epilepsy such as tonic-clonic (grand mal) seizures synaptic disturbance occurs. Moreover, we consider some noises which occur in our daily lives affect the development of epilepsy. In this work, we explore the effect of how noise contamination in a coupled lattice of neurons alters the synchrony in such circuits.

In this study, we construct a low complexity, grid lattice model of neurons using Rulkov maps to produce a two-dimensional spike-bursting behavior of similar real biological neurons [5],[6]. In addition, we have defined both excitatory neurons and inhibitory neurons to exist in the lattice which express long-term potentiation (LTP) and long-term depression (LTD) [2] respectively, using Spike Timing Dependent Plasticity (STDP) [3],[4]. Thus, incorporating noise into the original STDP, we can explore the synchronous behavior of biological spiking activity.

2. Spike Timing Dependent Plasticity

Spike Timing Dependent Plasticity (STDP) [3],[4] is a temporally asymmetric form of Hebbian learning induced by tight temporal correlations between the spikes of pre-synaptic and post-synaptic neurons. STDP provokes the LTP of the synapses, if the pre-synaptic spike arrival occurs a few milliseconds before post-synaptic spikes. Whereas, STDP provokes the LTD of the same synapse, if pre-synaptic spike arrival after post-synaptic spikes. (Fig. 1).

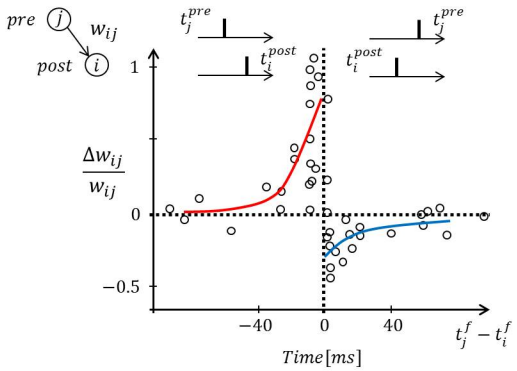

Figure 1: The STDP function of changing synaptic connection.

The weight change Δw_{ij} depends on the relative timing between pre-synaptic spike arrivals and post-synaptic spikes. The total weight change Δw_{ij} induced by a simulation protocol with pairs of pre-synaptic and post-synaptic spikes is described as following.

$$\Delta w_{ij} = \sum_{f=1}^{N} W(t_j^f - t_i^f) \tag{1}$$

where $W(x)$ denotes one of the STDP functions in Fig. 1. The method to choose for the STDP function $W(x)$ is shown as following.

$$W(x) = \begin{cases} A_+ exp(-x/\tau_+) + z(x) & (x < 0) \\ -A_- exp(x/\tau_-) + z(x) & (x > 0) \end{cases} \tag{2}$$

which is in alignment to experimental models. The parameters A_+ and A_- depend on the current value of the synaptic weight w_{ij}, and $z(x)$ is the time-series of noise. Additionally, the parameters A_+ and A_- are fixed to the value of 0.05 and the time constants are $\tau_+ = \tau_- 10ms$ which parameters are determined by reference to previous study [9].

In this study, we consider the 3 kinds of STDP function; Basic STDP (i.e., $z(x) = 0$), STDP with random noise (uniform random numbers between -0.004 and 0.004) and STDP with chaotic noise (Eqs. (3), (4) and Fig. 2). The chaotic noise which is generated by logistic map is given as following equation.

$$\hat{z}(x+1) = \alpha\hat{z}(x)(1-\hat{z}(x)), \tag{3}$$

it is known that the map produces intermittent bursts just before periodic-windows appear. We apply the intermittency chaos time series after the following normalization to STDP function $W(x)$.

$$z(x+1) = \frac{\hat{z}(x) - \overline{z}}{\sigma_z}, \tag{4}$$

where $\overline{z}$ and σ_z are the average and the standard division of $\hat{z}(x)$, respectively. Figure 2 shows the intermittency chaos near the three-periodic window obtained from Eq. (4) for $\alpha = 3.828$.

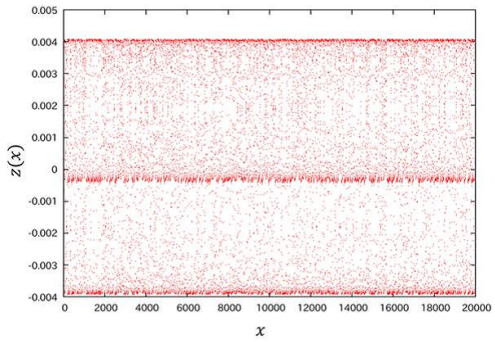

Figure 2: Time series obtained from logistic map ($\alpha = 3.828$).

3. Coupled Rulkov Maps

In recent years, a simple model which replicates the dynamics of spiking and spiking-bursting activity of real biological neurons has proposed by N. F. Rulkov [5],[6]. The model is a two-dimensional map that produces chaotic spiking-bursting neural behavior. It is demonstrated that the results of this model are in agreement with the synchronization of chaotic spiking-bursting behavior experimentally found in real biological neurons [8]. The expressions of the Rulkov map are shown in equations (5-7) below.

$$\begin{aligned} x_{n+1} &= f(x_n, y_n), \\ y_{n+1} &= y_n - \mu(x_n + 1) + \mu\sigma, \end{aligned} \tag{5}$$

Where, x represents the fast and y is the slow dynamical variables. The nonlinear function $f(x, y)$ is described in equation (6) as follows:

$$f(x,y) = \begin{cases} \alpha/(1-x) + y, & (x \le 0) \\ \alpha + y, & (0 < x < \alpha + y) \\ -1, & (x \ge \alpha + y) \end{cases} \tag{6}$$

The equation of the coupled grid lattice of Rulkov maps was then derived to be as in equation (7).

$$\begin{aligned} x_{m,n+1} &= f(x_{m,n}, y_{m,n}) + \frac{1}{2}w_{ij}(x_{m+1,n} - 2x_{m,n} + x_{m-1,n}), \\ y_{m,n+1} &= y_{m,n} - \mu(x_{m,n}+1) + \mu\sigma + \frac{1}{2}w_{ij}(x_{m+1,n} - 2x_{m,n} + x_{m-1,n}), \end{aligned} \tag{7}$$

Where, parameter m is the number of neurons, n is the number of iterations, and w_{ij} shows the coupling weight of the connection between the maps. The coupling weights w_{ij} are then updated by the STDP function. When the STDP provokes LTP, the coupling weights are updated as positive. Whereas, if STDP provokes LTD, the coupling weights are updated in a negative manner (Fig. 1). In the following section, we demonstrate how Rulkov maps demonstrate various synchronization phenomena due to coupling weight.

4. Network Model and Simulations

In this work, we consider the 5 coupled maps which is constructed by excitatory neurons and the inhibitory neurons as shown in Fig. 3. The network of excitatory neurons and the inhibitory neurons exhibit spike/bursting activity. The excitatory neurons exhibit the bursting activity which amplitude is around $60mV$ and 5-10 spikes burst all at once in a bursting wave, and the inhibitory neurons exhibit the spike activity which spike amplitude is 60-70mV and spike interval is $10ms$ in the biological neurons [10]. Therefore, the parameter α, σ and μ of the Rulkov Maps were set to 50, 0.1 and 0.04-0.06 respectively for the excitatory neurons and the parameter α, σ and μ were set to 40, 0.33 and 0.6 respectively for the inhibitory neurons in the network. In this study, the parameters of coupled Rulkov Maps are fixed as Tab. 1.

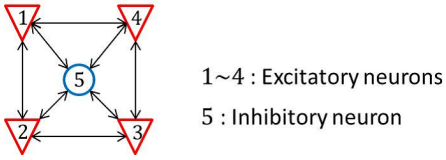

Figure 3: Connective arrangement of 5 coupled maps.

Table 1: The Parameters of Coupled Rulkov Maps

Neuron #	α	σ	μ
1	50	0.1	0.04
2	50	0.1	0.05
3	50	0.1	0.06
4	50	0.1	0.05
5	40	0.33	0.6

Figure 4-(a) shows the coupling weights (0-50000ms) and the spiking activities (20000-20500ms, 25000-25500ms, 30000-30500ms) on 5 coupled Rulkov maps with STDP. The color bar and the number on the bottom-left of Fig. 4 describe the number of neurons and neuronal connection on the result of coupling weight, and the red/blue waveforms show the spiking activity of excitatory/inhibitory neuron each. The reason why the coupling weights are not updated from around 40000ms is the coupled Rulkov maps are convergence and do not exhibit the bursting activity. The magnitude relation of coupling weight are fixed relatively early compared to another models. In addition, we consider that the synchronization accuracy between the excitatory neurons has an effect on diverging of membrane potentials from what the coupling weights between the excitatory neurons increase sharply just before the membrane potentials diverge. The spiking activity of neuron 5 (inhibitory neuron) has already exhibited recognizable bursting activity around 20000ms, and it becomes involved in high accuracy in-phase synchronization of neuron 1, 2 and 4 around 30000ms. In basic STDP model, the synchronization accuracy has been increased with time, since the membrane potentials will increase at an accelerating pace and diverge (i.e., lead to seizure) from around 40000ms.

Figure 4-(b) shows the coupling weights (0-50000ms) and the spiking activities (20000-20500ms, 30000-30500ms, 40000-40500ms, 49500-50000ms) on 5 coupled Rulkov maps with random noise STDP. The magnitude relation of coupling weight are hard to converge compared to another models. Moreover, the excitatory neuron 1, 3 and 4 show high accuracy in-phase synchronous firing, and the spiking activity of excitatory neuron 2 shows anti-phase synchronization with another excitatory neurons. The spiking activity of neuron 5 (inhibitory neuron) has exhibited recognizable bursting activity and the diverging of membrane potentials around 40000ms. Also in random noise STDP model, the synchronization accuracy has been increased with time, since the membrane potentials will increase at an accelerating pace and diverge (i.e., lead to seizure) relatively late compared to basic STDP model.

Figure 4-(c) also shows the coupling weights (0-50000ms) and the spiking activities (20000-20500ms, 30000-30500ms, 40000-40500ms, 49500-50000ms) on 5 coupled Rulkov maps with chaotic noise STDP. The magnitude relation of coupling weight are stable compared with random noise STDP model. Each excitatory neuron shows high accuracy in-phase synchronous firing around 50000ms, whereas the neuron 5 (inhibitory neuron) does not get involved in synchronous firing of excitatory neurons (i.e., it exhibits spike activity without being bursting activity). The membrane potentials do not increase and diverge for the above reason. By the way, the reason why the chaotic noise STDP model shows different spiking behavior compared to the random noise STDP model is that the logistic map represents the value close to extreme.

5. Conclusions

In this work, we built a low complexity model using Rulkov maps to model the spiking activity and Spike Timing Dependent Plasticity (STDP) to model the LTP and LTD. Using the aforementioned models, we then proceed to explore the effect of noise contamination on the spiking activity and the synchronous provoked. From the simulation results, we observed that the membrane potentials increase at an acceleration pace and diverge (i.e., lead to seizure) , if the inhibitory neuron obtains involved in synchronous firing of excitatory neurons. Additionally, the chaotic noise STDP model has resistance characteristic of diverging of membrane potential. Basically, certain kind of noise prevents development of epilepsy.

Acknowledgments

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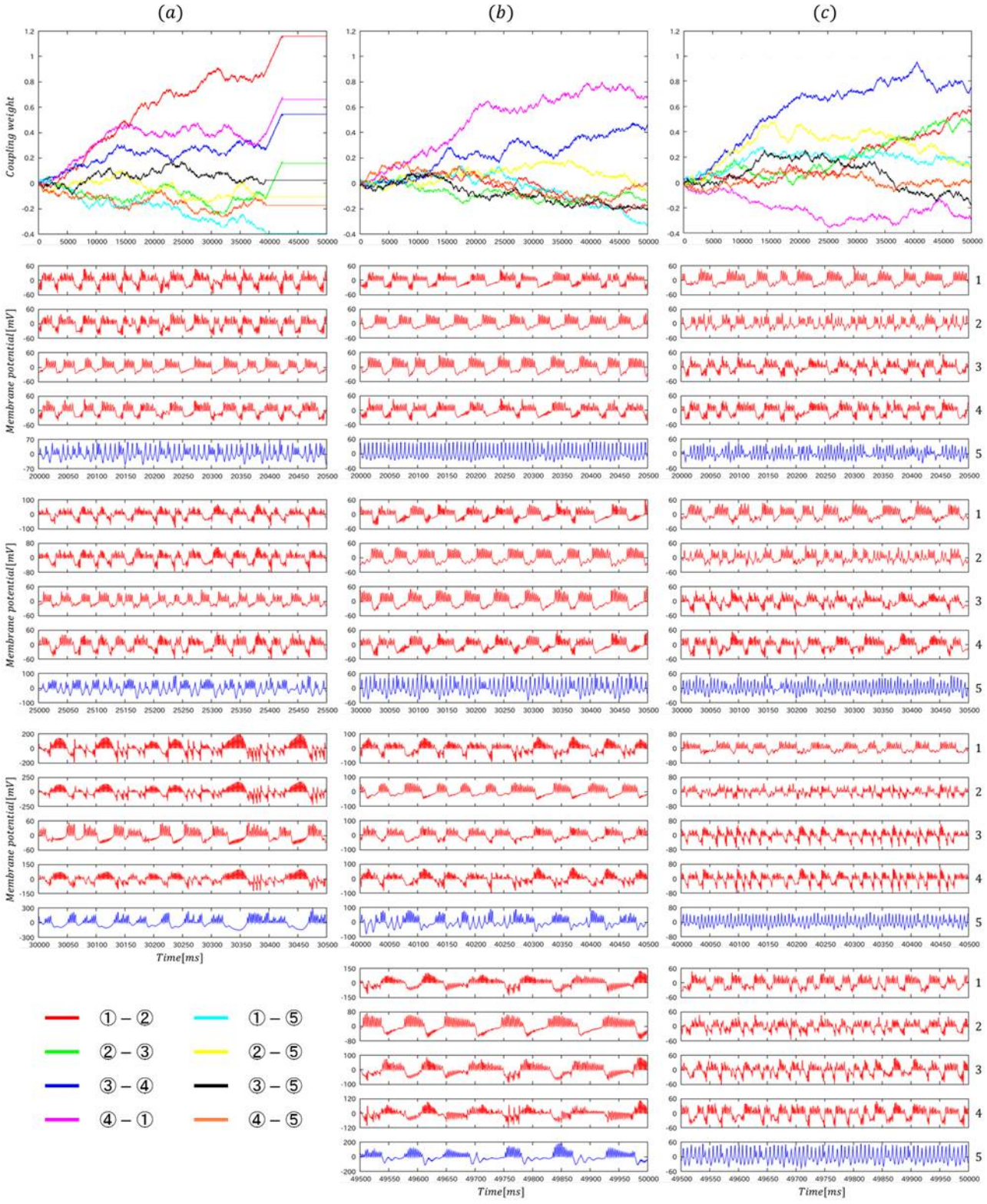

Figure 4: Spiking activity and coupling weight of 5 coupled maps with: (a) Basic STDP (b) Random noise STDP and (c) Chaotic noise STDP.