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Stimulus-evoked synchronization in neuronal models

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Abstract

It is known that stimulus-evoked synchronization among neurons occurs in widely separated cortical regions. In this paper we test that how common, *random but not deterministic* inputs can synchronize groups of neurons with their parameters inside physiologically plausible regions. When a common, random input is presented, we find that a group of neurons—of integrate-and-fire or Hodgkin–Huxley models—are capable of rapidly synchronizing their firing. Interestingly the optimal average synchronization time occurs when the efferent spikes have a high coefficient of variation of interspike intervals (greater than 0.5).
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1. Introduction

How do neurons couple with each other to fire *synchronously* is an important issue both theoretically and experimentally [2,4]. It has been widely accepted that information is encoded by neurons via a variety of schemes: from the classical view of rate coding to the modern view involving time coding. A typical example of time coding is brain waves, oscillating at about 40 Hz for a group of neurons—the so-called gamma rhythm, which appear to be involved in higher mental activity and therefore are considered to be essential for processing information by the brain. However, how neurons respond to external stimuli to organize locally or over a wider range to fire together remains elusive, with a few mechanisms such as recurrent inhibition, mutual excitation, intrinsic oscillators and mutual inhibition having been put forward.

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For the purpose of elucidating mechanisms of synchronization, networks of model neurons have been extensively studied in the literature. We mention but a few studies. In [3] the authors proved the existence of the Lyapunov function for a specific case of interactions.

These studies above are confined to the case of deterministic inputs, but there is a consensus that the inputs and outputs of single neuron are frequently stochastic. Furthermore and most importantly, experimental results tell us that neurons in widely separated areas—which implies there is no local interaction among them—are capable of synchronizing with zero time lag. This possibly indicates that inputs play a vital role in the synchronization, at least for neurons in widely separated areas (see further discussion below on local interactions). The purpose of the paper is to reveal when and how efficiently common inputs ensure neurons to fire synchronously.

We first consider two identical neurons with different initial states but subjected to common, stochastic inputs propagating along excitatory and inhibitory synapses. The two neurons can be viewed as located either in separate areas (with no local interaction), or they receive inputs from many other neurons and therefore the contribution of each to the process of synchronization is much smaller than surrounding inputs, or that local interactions ensure them to receive common inputs. Under *different* (*independent*) stimuli the synchronization is not observable, but under *same* stimuli the neuronal activities quickly cohere with each other. This suggests that one of the most important factors which synchronizes neuronal activities is their *common* inputs. Let us call the time at which two neurons synchronize the *synchronization time*. Interestingly, the shortest synchronization time averaging over different initial states is attained when the coefficient of variation (CV) of efferent spike trains of individual neurons is greater than 0.5, i.e. inside a high CV region. Our results further reveal one of the functional roles of balanced inputs which are automatically maintained with the spike-timing-dependent synaptic plasticity rule. Further numerical examples and theoretical results on the synchronization of a large group of neurons are also included and properties of synchronization time are discussed. Finally, we also show that spiking reliability observed in [5] is simply a consequence of stochastic inputs.

2. The models

We consider a group of leaky integrate-and-fire neurons, with or without reversal potentials, subjected to inputs which are conventionally assumed to be Poisson processes. For $i = 1, 2, \ldots, m$ let $N_i^{\mathrm{E}}(t)$ and $N_i^{\mathrm{I}}(t)$ be total excitatory and inhibitory inputs of the ith neurons with rate $N_{\mathrm{E}} \times \lambda_{\mathrm{E}}$ and $N_{\mathrm{I}} \times \lambda_{\mathrm{I}}$, where $N_{\mathrm{E}}(N_{\mathrm{I}})$ is the number of total active excitatory (inhibitory) synapses and λ_{E} (λ_{I}) is the firing rate of EPSPs (IPSPs) of each excitatory (inhibitory) synapse. Suppose that $x_i(t)$ is the membrane potential of the ith neuron at time t then $x_i(t)$ are governed by the following dynamics with initial state x_i

$$dx_i(t) = -\frac{1}{\gamma}\,(x_i(t) - V_{\mathrm{rest}})\,\mathrm{d}t + I_{\mathrm{syn},i}(t), \tag{1}$$

where synaptic inputs $I_{\mathrm{syn},i}(t)=aN_i^{\mathrm{E}}(t)-bN_i^{\mathrm{I}}(t)$, $1/\gamma$ is the decay rate, $a>0$ and $b>0$ are the magnitude of each excitatory and inhibitory input. As soon as $x_i(t)$ reaches a prefixed value V_{thre}, the threshold, $x_i(t)$ is reset to V_{rest}, the resting potential. The model defined by Eq. (1) is usually called the integrate-and-fire (IF) model and it has been intensively studied for exploring properties of biological neurons.

The classical Hodgkin–Huxley (HH) model is also taken into account, denoting $V_i(t)$ as the membrane potential of ith cell at time t. We refer the reader to [1] for details of all parameters and notation. Synaptic inputs $I_{\mathrm{syn},i}(t)$ are defined as above.

For a given neuron i let us denote $T_n^{(i)}$ as the occurrence time of the nth spike. For two neurons i,j the time

$$T(i,j)=\inf\{T_n^{(i)}: T_{n+p}^{(i)}=T_{k+p}^{(j)}, \quad n,k=1,2,\ldots, \quad p=0,1,2,\ldots,\}$$

is their synchronization time.

Due to the space limit, we only report our results on the HH model and refer the reader to [1] for details.

For the convenience of discussion we have fixed a few parameters in our numerical simulations $N_{\mathrm{E}}=100$, $\lambda_{\mathrm{E}}=\lambda_{\mathrm{I}}=100$ Hz and $\gamma=20.2\pm14.6$ ms. We have used the same set of parameters elsewhere [1]. Note that the intensity of incoming signals is $10,000\ \mathrm{Hz}=N_{\mathrm{E}}\times\lambda_{\mathrm{E}}$ which is also equivalent to $N_{\mathrm{E}}=300$, $\lambda_{\mathrm{E}}\sim33$ Hz. We use step size of 0.01 msec in the simulations of the IF model (Euler scheme), and the Runge–Kutta method with variable step size in the HH model.

We generalize the theory in [1] to the Hodgkin–Huxley model, based upon numerical simulations. However, the following arguments provide us with rational reasons to explain that why similar phenomena are true for the HH models. Denote V_{k}, V_{Na}, V_{L} as the reversal potentials of K, Na channels and leakage, $g_{\mathrm{L}},g_{\mathrm{Na}},g_{\mathrm{k}}$ as the conductance of K, Na channels and leakage, and V_{re} as the resting potentials. We rewrite the HH model in the following way:

$$CdV=-\bar{g}_{\mathrm{L}}(V-V_{\mathrm{re}})\,\mathrm{d}t+\bar{g}_{\mathrm{Na}}m^3h\,\mathrm{d}t-\bar{g}_{\mathrm{k}}n^4\,\mathrm{d}t+d\bar{I}_{\mathrm{syn}}(t), \tag{2}$$

where $\bar{g}_{\mathrm{L}}=g_{\mathrm{Na}}\ \mathrm{m}^3\mathrm{h}+g_{\mathrm{k}}\mathrm{n}^4+g_{\mathrm{L}}\geqslant0$ is the actual leakage of the model (depending on time), $\bar{g}_{\mathrm{Na}}=g_{\mathrm{Na}}(V_{\mathrm{Na}}-V_{\mathrm{re}})>0$, $\bar{g}_{\mathrm{k}}=g_{\mathrm{k}}(V_{\mathrm{re}}-V_{\mathrm{k}})>0$, and $\bar{I}_{\mathrm{syn}}(t)=I_{\mathrm{syn}}(t)+g_{\mathrm{L}}(V_{\mathrm{L}}-V_{\mathrm{re}})t$.

Eq. (2) has advantage over the original form of the HH model: firstly, each term gives us a clear physical meaning as we mentioned above; secondly, it is analogous with the IF model, and so we can generalize results from the IF model to the HH model; thirdly, since all ionic channels have their reversal potentials and so Eq. (2) is universal for all biophysical models in the following sense: for a given biophysical model we can rewrite the model in a way similar to Eq. (2). According to our theory on the IF model, we know that the decay term and random inputs ensure the synchronization and thus it is natural to expect that our theory will be true for all biophysical models, although here we confine ourselves to the HH model.

In Fig. 1 (left) a common input is turned on at time = 500 ms (turned off at time = 700 ms) and 1500 ms (turn off at 1800 ms), namely the synaptic input of the ith neuron

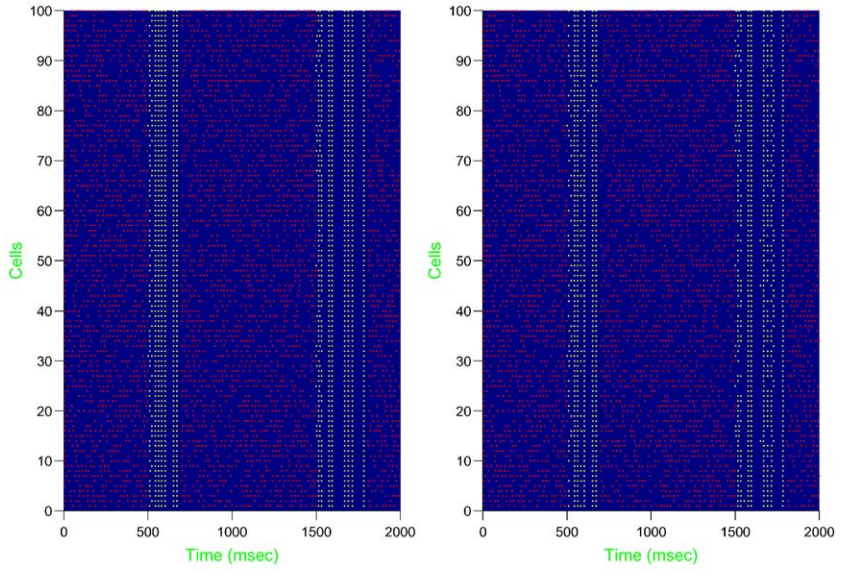

Fig. 1. One hundred HH neurons easily synchronize when a common stimulus is turned on (see text for further explanation). (Left) All inputs are common. (Right) 90% inputs are common and 10% are i.i.d.

now takes the form

$$[\delta(aN_1^{\mathrm{E}}(t) - bN_1^{\mathrm{I}}(t)) + (1-\delta)(aN_i^{\mathrm{E}}(t) - bN_i^{\mathrm{I}}(t))]I_{\{t\in T\}} + [aN_i^{\mathrm{E}}(t) - bN_i^{\mathrm{I}}(t)]I_{\{t\notin T\}}, \tag{3}$$

where $T = [500, 700] \cup [1500, 1800]$, $\delta = 1$ for 100% common inputs, $\delta = 0.9$ for 90% common inputs, and I_T is the indicator function for the set T. It is easily seen that the group of neurons synchronize after a few spikes (one or two). In Fig. 1 (right) 90% inputs are common and 10% are i.i.d., synchronization is still extensive and rapid.

For the synchronization time of the HH model we have conclusions as for the IF model, as shown in Fig. 2. The optimal synchronization time occurs neither the neuron receives pure EPSP inputs ($r = 0$) nor balanced EPSP and IPSP inputs ($r = 1$).

3. Discussion

We have shown and proved that the IF model is capable of synchronizing within a finite time and numerically that the time for them to synchronize is almost instantaneous. The conclusions are then generalized to the HH model, based upon numerical simulations. Our results might provide a possible mechanism for the phenomena of stimulus-evoked synchronization in widely separate cortex areas which has been observed in experiments [6]. On the other hand our results also lay a foundation for further investigation, in particular on the role of local interactions. According to our

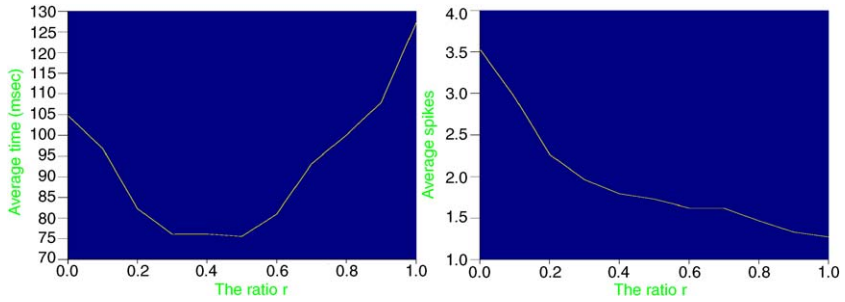

Fig. 2. Average synchronization time and spike of two Hodgkin–Huxley neurons with different initial states. The average synchronization time and spike number are obtained for $V_2(0) = -80, -79.85, \ldots, -65$ mV and $V_1(0) = V_{\mathrm{re}}$.

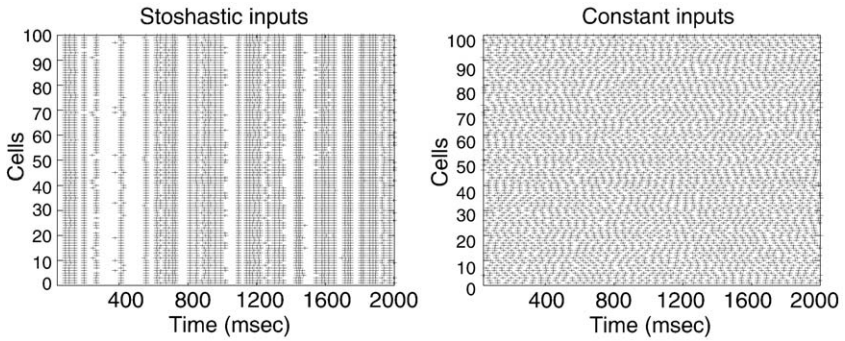

Fig. 3. Stochastic inputs and deterministic inputs $([\delta 10/9dt + (1-\delta)(aN_i^{\mathrm{E}}(t) - bN_i^{\mathrm{I}}(t))]I_{\{t\in T\}})$ with $\delta = 0.9$ and random initial states of the membrane potentials, $T = [0, 2000]$ ms. At $t = 0$ membranes of cells with deterministic inputs are equal to that with stochastic inputs. For the stochastic input case, the output firing rate is around 23.5 Hz, for the deterministic case, it is around 21.5 Hz. Other parameters are as before [1]. It is easily seen that neurons with stochastic inputs synchronize their activity, but not with deterministic inputs.

results presented here we also want to point out another possible role played by *local connections*, except for its role of speeding up or slowing down the synchronization time: the brain is wired in such a way the synchronous neurons to receive common inputs. With balanced inputs, the synchronization time attains its minimum.

To further show the implications of our results in the present paper, we simulate 100 neurons with stochastic and deterministic inputs with small random perturbations, as shown in Fig. 3. It is easily seen that random, rather than deterministic inputs synchronize neuronal activities. We already know from the results above that neurons with stochastic, common inputs are easily to synchronize. Let us now look at the case of deterministic inputs. Suppose that two neuron are driven by a constant input and the first neuron fires before the second neuron with a time difference of t, then this difference will be always present and so it is difficult for them to synchronize, as shown in Fig. 3.

This explains the phenomenon observed in [5] if we think of cells in Fig. 2 as trails in Fig. 1 in [5], provided that $(1-\delta)100\%$ inputs (i.i.d. part, it is 10% in Fig. 3) are feedback from other neurons. To understand the reliability of spiking time, many recent research activity has been devoted to the similar phenomenon and this is also the focus of recent years discussions on whether neuron interspike intervals are reliable or not.

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