Network activity in a Morris–Lecar population density model

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ABSTRACT

A population density approach is presented to simulate the network activity of Morris–Lecar (ML) neurons. The network is composed of identical excitatory and inhibitory ML neurons. Each neuron randomly receives excitatory and inhibitory connections from other neurons in the network and an excitatory external input which is described by an independent Poisson process from neurons outside the network. We solve the evolution equation for the population density approach numerically. The results were compared against conventional computation for groups of individual neurons in a few example networks. We found that when the neuronal network comprises a large number of identical excitatory ML neurons that are sparsely connected, the population density approach gives a closer approximation to the network activity. We also demonstrated that the population density approach using the ML neuron model can be used to simulate the activities of type I and type II neurons (integrators and resonators) in a network of sparsely connected inhibitory and excitatory neurons that was not possible using the integrate-and-fire neuron model.

1. Introduction

Previous studies of somatosensory, visual cortex [1,2] and pools of motor neurons [3] showed that in many areas of the brain, neurons are organized in populations of units with similar properties. Therefore it is convenient to describe the mean activity of the neuronal population rather than the spiking of individual neurons. The population density approach overcomes the limitation imposed by the large computation time required to compute simulations of a network of neurons when the number of neurons becomes very large. The computation time for population density approach is dependent on the number of interacting populations rather than the number of neurons [4].

The population density approach has been used to study the network behavior that is composed of a large number of identical integrate-and-fire neurons which have the similar biophysical properties. Most of the previous work focused on one-dimensional population density approach for the leaky integrate-and-fire (LIF) model [5] and the integrate-and-fire conductance based model [6–8]. These were extended to several studies of two-dimensional population density approach for the integrate-and-fire-or-burst (IFB) model [4,9–11]. In the limit of small voltage jump, the population density approach can be reduced to Fokker–Planck (diffusion) approximation to analyze the dynamics of the distribution of neuron potentials [12–18]. In order to describe the neuron dynamics precisely, a probability density approach that takes into consideration the effects of slow ionic currents was proposed by Chizhov et al. [19]. They simulated the activity of recurrent inhibitory neuron network to constant current step input.

The integrate-and-fire (IF) neuron model is well known as a simple and efficient spiking neuron model for simulating large-scale neuronal networks. However, due to its simplicity, it has poor biological plausibility and cannot produce many neuro-computational features of real neurons [20]. Conversely, if the neuronal behavior needs to be studied and investigated in detail, Hudgkin–Huxley (HH) model is the most important model that can exhibit many neuronal computational properties. Despite the HH model being biophysically meaningful, the computation is very time consuming.

The Morris–Lecar (ML) model is one of the biophysically meaningful models that can reproduce integrator or resonator (each is referred to as type I and type II neurons in the following paragraphs) depending on the parameter of voltage dependent potassium current [21] while the IF model is an integrator. Integrators exhibit saddle-node bifurcation when it transits between

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a rest state and repetitive firing state. In contrast, resonators exhibit Andronov–Hopf bifurcation [22]. Many cortical neurons are integrators while the resonator's behavior has been described in thalamic [23] and cortical region [24,25]. However, when simulating a huge number of interconnected spiking neurons, the ML model, being more complex than the IF neuron model, requires a longer computation time. Thus, having a method that is not only computationally efficient and but also biophysically meaningful is an important goal in computational neuroscience.

In the present paper, the population density approach is used to model the network that is comprised of a large number of identical Morris–Lecar (ML) neurons. With minimal biophysical characteristics of ML neurons, action potential events can be generated in response to synaptic perturbations [26–29]. Each identical ML neuron in the network is randomly connected and each synapse receives an excitatory external input which is described by the independent Poisson process [12–14,30,31]. Type I and type II neurons are also simulated in a network with sparsely connected excitatory and inhibitory neurons.

The paper is organized as follows. The conductance-based Morris–Lecar (ML) neuron model is described in Section 2. In Section 3, we introduce the population density approach for the ML model and derive the corresponding population density equations. The numerical algorithm for solving the population density equation is presented in Section 3.1. We demonstrate the results of a single uncoupled population of Type II neurons in Section 4. The effects of the number of connections on the network behavior is investigated and discussed in Section 5. The simulations of type I and type II neurons are presented in Section 6. In Section 7, we show the comparison of computation time between the population density approach and the direct simulation of a network of ML neurons. New implications and advances in the study of neural systems are stated in Section 8. Section 9 is the conclusion of this paper.

2. The conductance-based Morris–Lecar neuron model

A network of interconnected excitatory population and inhibitory population is shown in Fig. 1. There are N_{exc} and N_{inh} identical Morris–Lecar (ML) neurons in the excitatory and the inhibitory populations respectively. Each population randomly receives q_{exc} excitatory connections and q_{inh} inhibitory connections from other neurons inside the network. It also receives q_{ext} external excitatory inputs with rate ν_{ext} from neurons outside the network. The total effect of the external network is denoted as an external Poisson input. External spikes are statistically independent and can be well approximated by a Poisson distribution [12,13].

The set of differential equations that governs the dynamics of the membrane potential for neuron i ($i = 1, 2, 3, …, N_{exc} + N_{inh}$) are written as follows [26]:

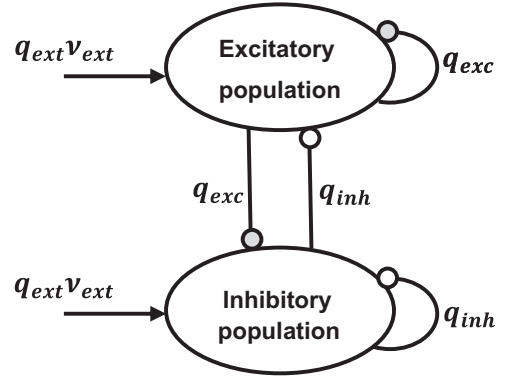$$C \frac{dV_i}{dt} = -I_{ion,i} + I_{syn,i} \tag{2.1}$$

$$\frac{dW_i}{dt} = \varphi \frac{(W_\infty(V_i) - W_i)}{\tau_R(V_i)} \tag{2.2}$$

where

$$I_{ion,i} = I_{Ca,i} + I_{K,i} + I_{L,i}$$
$$= g_{Ca} m_\infty(V_i)(V_i - E_{Ca}) + g_K W_i(V_i - E_K) + g_L(V_i - E_L) \tag{2.3}$$

$$m_\infty(V_i) = 0.5[1 + \tanh\{(V_i - V_1)/V_2\}] \tag{2.4}$$

$$W_\infty(V_i) = 0.5[1 + \tanh\{(V_i - V_3)/V_4\}] \tag{2.5}$$

Fig. 1. Schematic diagram of the network architecture. The network is composed of a population of excitatory neurons and a population of inhibitory neurons that interconnect with each other. Each population receives q_{ext} excitatory external input from neurons outside the network with rate ν_{ext}. q_{exc} and q_{inh} are the number of excitatory and inhibitory connections from neurons inside the network.

$$\tau_R(V_i) = \frac{1}{\cosh\{(V_i - V_3)/(2V_4)\}} \tag{2.6}$$

Here, V is the membrane potential measured in units of mV and W represents the slow recovery variable of the action of the potassium current. There are two kinds of source currents to each neuron, $I_{ion,i}$ and $I_{syn,i}$. $I_{ion,i}$ is the total ionic current that consists of a calcium current, $I_{Ca,i}$, a potassium current, $I_{k,i}$ and a leakage current, $I_{L,i}$. C is the capacitance of the membrane. The maximum conductance for the ion and the leakage channels are denoted by g_{Ca}, g_K and g_L whereas E_{Ca}, E_K and E_L represent the reversal potentials for the ion and the leakage channels. The gate variable W for the potassium channel tends to the saturation value $W_\infty(V_i)$ with relaxation time $(\tau_R(V_i)/\phi)$. Fast changes of the calcium current take the gate variable m_i as the saturation value $m_\infty(V_i)$.

When the pre-synaptic neuron j ($j = 1, 2, 3, …, q_{ext} + q_{exc} + q_{inh}$) emits a spike at time t, the potential of the postsynaptic neuron i is increased or decreased by postsynaptic potential (PSP) amplitude J_{ij}. For simplicity, we assume that $J_{ij} = J_{exc}$ for excitatory synapses and $J_{ij} = J_{inh}$ for inhibitory synapses. The synaptic current of the ith neuron is described as follows:

$$RI_{syn,i} = \tau_{syn} \sum_j J_{ij} \sum_k \delta(t - t_j^k) \tag{2.7}$$

where $\tau_{syn} = RC$ is the synaptic time constant and t_j^k is the emission time of the kth spike at neuron j. When V_i crosses the threshold value V_{th}, neuron i emits a spike.

3. The population density approach

A population density approach is introduced to represent the membrane behavior of a large number of identical ML neurons as described in the previous section [6,10,11],

$$\rho(v, w, t)\, dv\, dw = \Pr\{V(t) \in (v, v + dv) \text{ and } W(t) \in (w, w + dw)\} \tag{3.1}$$

for $v \in (V_{\min}, V_{\max})$ and $w \in (0, 1)$. The evolution equation for the probability of finding membrane potential of randomly chosen neuron in population $a = exc$, inh at v over all possible states at time t is based on conservation of probability:

$$\frac{\partial}{\partial t} \rho(v, w, t) = -\nabla \cdot \vec{J}(v, w, t) \tag{3.2}$$

where $\nabla = \hat{e}_v(\partial/\partial v) + \hat{e}_w(\partial/\partial w)$ and $\vec{J}(v, w, t)$ is the total probability flux cross v and w at time t. The total probability flux consists of