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 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.

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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 (IF) 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-on-hurt (IF) model [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, Hodgkin–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
a resting state and repetitive firing state. In contrast, resonators exhibit Andronov–Hopf bifurcation [42]. 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 large 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 [12–16,30,31].

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 \( \nu_{ext} \) 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 \( i \) (\( i = 1, 2, \ldots, N_{exc} + N_{inh} \)) are written as follows [26]:

\[
\frac{dV_i}{dt} = -I_{ion,i} - I_{syn,i} \tag{2.1}
\]

\[
\frac{dW_i}{dt} = \frac{(O_{max}(V_i) - W_i)}{\tau_s(V_i)} \tag{2.2}
\]

where

\[
I_{ion,i} = I_{Na,i} + I_{K,i} + I_{leak,i} \tag{2.3}
\]

\[
= g_{Na} m_{Na}(V_i - E_{Na}) + g_{K} W_i (V_i - E_{K}) + g_{leak}(V_i - E_{Na}) \tag{2.4}
\]

\[
W_{max}(V_i) = 0.5 \left( 1 + \tanh \left( \frac{V_i - V_{th}}{\Delta V} \right) \right) \tag{2.5}
\]

\[
g_{Na} m_{Na} = \frac{1}{\cosh \left( \frac{V_i - V_{th}}{\Delta V} \right)} \tag{2.6}
\]

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 \( I_{syn,i} \), \( I_{syn,i} \) is the total ionic current that consists of a calcium current, \( I_{Ca,i} \), a potassium current, \( I_{K,i} \), and a leakage current, \( I_{leak,i} \). \( \tau_s \) is the time constant of the calcium current. The maximum conductance for the ion and the leakage channels are denoted by \( g_{Ca} \), \( g_{K} \) and \( g_{leak} \). \( E_{Ca} \) and \( E_{K} \) represent the reversal potentials for the ion and the leakage channels. The gate variable \( W \) for the potassium channel tends to the saturation value \( W_{max}(V_i) \) with relaxation time \( \tau_s(V_i) \). Fast changes of the calcium current take the gate variable \( m \) as the saturation value \( m_{Na}(V_i) \).

When the pre-synaptic neuron \( j \) fires, \( j = 1, 2, \ldots, N_{exc} + N_{inh} \), a spike at time \( t \), the potential of the postsynaptic neuron \( i \) is increased or decreased by postsynaptic potential (PSP) amplitude \( f_{ij} \). For simplicity, we assume that \( I_{syn,i} = I_{exc,i} \) for excitatory synapses and \( I_{syn,i} = I_{inh,i} \) for inhibitory synapses. The synaptic current of the \( i \)th neuron is described as follows:

\[
W_{syn,i} = \sum_{j=1}^{N} \left( I_{exc,i} + I_{inh,i} \right) f_{ij} \tag{2.7}
\]

where \( \tau_{syn} = RC \) is the synaptic time constant and \( f_{ij} \) is the emission time of the \( j \)th 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].

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

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 \( a = \text{exc, inh} \) in \( v \) at over all possible states at time \( t \) is based on conservation of probability:

\[
\frac{d}{dt} \rho(v, w, t) = -\varphi V (v, w, t) \tag{3.2}
\]

where \( \varphi = \varphi_{V}(v, w, t) + \varphi_{W}(v, w, t) \) and \( \varphi (v, w, t) \) is the total probability flow across \( v \) and \( w \) at time \( t \). The total probability flow consists of:

\[
\varphi = -\varphi_{V}(v, w, t) = -\epsilon \hat{V} (v, w, t) \tag{3.3}
\]

\[
\varphi_{W}(v, w, t) = \epsilon_{Na} \hat{m} (v, w, t) + \epsilon_{K} \hat{W} (v, w, t) + \epsilon_{leak} \hat{W} (v, w, t) \tag{3.4}
\]
two components:
\[ \tilde{f}(v, w, t) = \tilde{f}_\text{across}(v, w, t) + \tilde{f}_\text{intrinsic}(v, w, t) \]  
(3.3)
\[ \tilde{f}_\text{across}(v, w, t) \] is the flux due to the intrinsic membrane dynamics:
\[ \tilde{f}_\text{across}(v, w, t) = F_a(v, w) \rho_a + F_w(v, w) \rho_w \]  
(3.4)
where
\[ F_a(v, w) = -\frac{v}{C} \]  
(3.5)
\[ F_w(v, w) = -\frac{w - w_m}{\tau_w} \]  
(3.6)
The flux, \( \tilde{f}_\text{across}(v, w, t) \) due to the synaptic input from external network and the connected neurons in the network is written in the form
\[ \tilde{f}_\text{across}(v, w, t) = \tilde{f}_\text{exc}(v, w, t) + \tilde{f}_\text{inh}(v, w, t) \]  
(3.7)
When a neuron with voltage \( v = v \) receives \( \eta_{\text{exc}} \) excitatory external input rate of \( \eta_{\text{exc}} \), it could cross \( v \) to higher voltages from any voltage \( v' \in (v - \eta_{\text{inh}}, v) \) and generating a positive excitation flux:
\[ \tilde{f}_\text{exc}(v, w, t) = \eta_{\text{exc}} \nu(v, t) \rho(v, w, t) \]  
(3.8)
A neuron with voltage \( v' \in (v - \eta_{\text{inh}}, v) \) could generate another positive excitation flux cross voltage \( v \) upon the arrival of \( \eta_{\text{inh}} \) excitatory external input rate of \( \eta_{\text{inh}} \) from the connected neurons within the network:
\[ \tilde{f}_\text{exc}(v, w, t) = \eta_{\text{inh}} \nu(v, t) \rho(v, w, t) \]  
(3.9)
Conversely, if a neuron with voltage \( v' \in (v - \eta_{\text{inh}}, v) \) receives \( \eta_{\text{inh}} \) inhibitory internal input rate of \( \eta_{\text{inh}} \) and cross \( v \) to lower voltages, it could create a negative inhibition flux:
\[ \tilde{f}_\text{inh}(v, w, t) = \eta_{\text{inh}} \nu(v, t) \rho(v, w, t) \]  
(3.10)
When the upward movement of total probability flux, \( \tilde{f}_\text{exc} \) cross \( v = V_m \), it corresponds to the fraction of neurons firing per unit time. Thus, the population firing rate that describes the average firing rate across all neurons in the population is obtained by integrating over all slow recovery variable of the action of the potassium current, \( v \):
\[ \nu(t) = \int \tilde{f}_\text{exc}(v, w, t) \]  
(3.11)
where \( \tilde{f}_\text{exc}(v, w, t) \) is the flux when there are positive fluxes of \( \tilde{f}_\text{across}(v = V_m, w, t) \) and \( \tilde{f}_\text{across}(v = V_m, w, t) \). Assuming that there is no probability flux across the domain boundaries located at \( v = V_m, w = w_m = 0 \) and \( w = 1 \), leads to the following boundary conditions associated with Eq. (3.2).

\[ p(v = V_m, w, t) = 0 \]  
(3.12)
\[ p(v = V_m, w = 0, t) = 0 \]  
(3.13)
\[ p(v = V_m, w = 1, t) = 0 \]  
(3.14)
At any time \( t \), the population density functions in Eq. (3.2) with the boundary conditions above must satisfy the conservation of probability law,
\[ \nu(t) = \int p(v, w, t) dv \]  
(3.15)

### 3.1 Numerical algorithm

To solve the model equations in the population density approach, we discretize \( \frac{\partial f}{\partial v} \) and \( \frac{\partial f}{\partial w} \) and solve the resulting set of ODEs using Runge-Kutta 4th order. For \( k = 1, 2, \ldots, N_v \), the membrane voltage is discretized as
\[ v_k = k \frac{V_{\text{max}} - V_{\text{min}}}{N_v} \]  
(3.16)
Whereas for \( n = 1, 2, \ldots, N_w \), the discretization of gating variable for potassium is shown as
\[ w_n = \frac{1}{N_w} \]  
(3.17)
The spatial derivatives of \( \tilde{f}_\text{across}(v, w, t) \) at the grid points is given as
\[ \frac{\partial \tilde{f}_\text{across}}{\partial v} = \frac{v_{k+1} - v_k}{w_{n+1} - w_n} \nu(v, t) \rho(v, w, t) \]  
(3.18)
\[ \frac{\partial \tilde{f}_\text{across}}{\partial w} = \frac{w_{n+1} - w_n}{w_{n+1} - w_n} \nu(v, t) \rho(v, w, t) \]  
(3.19)
The spatial derivatives of \( \tilde{f}_\text{across}(v, w, t) \) at the grid points is given as
\[ \frac{\partial \tilde{f}_\text{across}}{\partial w} = \frac{w_{k+1} - w_k}{w_{k+1} - w_k} \nu(v, t) \rho(v, w, t) \]  
(3.20)
\[ \frac{\partial \tilde{f}_\text{across}}{\partial w} = \frac{w_{k+1} - w_k}{w_{k+1} - w_k} \nu(v, t) \rho(v, w, t) \]  
(3.21)
The discretization of the flux, \( \frac{\partial f}{\partial v} \), is denoted by
\[ \frac{\partial f}{\partial v} = \frac{f(v + 1, w, t) + f(v - 1, w, t)}{2} \]  
(3.22)
For \( v = V_{\text{max}} \), a downward first order approximation is used:
\[ \frac{\partial f}{\partial v} = \frac{f(v + 1, w, t) + f(v - 1, w, t)}{2} \]  
(3.23)
4. Single uncoupled population of neurons results

The root-mean-square error (RMSE) was computed to measure the difference between the individual neuron histograms and the corresponding areas under the population density curve. Let \( y(V) \) denote the population of individual neuron results and \( \tilde{y}(V) \) the population density model, then the RMSE at time \( t \) is given as

\[
\text{RMSE}(\tilde{y}, y) = \frac{1}{n} \sum_{i=1}^{n} (\tilde{y}(V_i) - y(V_i))^2
\]

where \( n \) is the size of voltage data.

The response of a single excitatory population of ML neurons to the external Poisson input is simulated. Each neuron receives an excitatory external input, \( q_{ext} = 1 \) from neurons outside the network with mean firing rate, \( \lambda_{int} = 200 \text{ spikes/ms} \) and external postsynaptic potential, \( E_{syn} = 0.1 \text{ mV} \). The other network parameters of type I neurons are shown in Table 1 [32].

The population density model is compared with the individual neuron populations of 100, 1000 and 10,000 neurons in Fig. 2. Fig. 2(a-e) compares the firing rates during 50 ms time period while Fig. 2(d-f) compares the snapshots of the probability density across membrane potential at time, \( t = 25 \text{ ms} \). For the results of

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**Table 1** Parameters for conductance based ML neuron model.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Description</th>
<th>Value for type I (100 neurons)</th>
</tr>
</thead>
<tbody>
<tr>
<td>( V_1 )</td>
<td>Threshold value for ( m_m )</td>
<td>1.2 mV</td>
</tr>
<tr>
<td>( V_2 )</td>
<td>Steepness parameter for ( m_m )</td>
<td>18 mV</td>
</tr>
<tr>
<td>( V_3 )</td>
<td>Threshold value for ( W_{K} )</td>
<td>18 mV</td>
</tr>
<tr>
<td>( V_4 )</td>
<td>Steepness parameter for ( W_{K} )</td>
<td>18 mV</td>
</tr>
<tr>
<td>( E_{a} )</td>
<td>Reversal potential for ( Ca^{2+} ) channels</td>
<td>120 mV</td>
</tr>
<tr>
<td>( E_{K} )</td>
<td>Reversal potential of leakage channels</td>
<td>80 mV</td>
</tr>
<tr>
<td>( E_{K} )</td>
<td>Reversal potential of ( K^{+} ) channels</td>
<td>80 mV</td>
</tr>
<tr>
<td>( C )</td>
<td>Capacitance of membrane</td>
<td>5 \text{ pF/cm}^2</td>
</tr>
<tr>
<td>( \tau )</td>
<td>Temperature time scale factor</td>
<td>0.914</td>
</tr>
<tr>
<td>( \alpha )</td>
<td>Maximum conductance for ( Ca^{2+} ) channels</td>
<td>4.4 \text{ mS/cm}^2</td>
</tr>
<tr>
<td>( \beta )</td>
<td>Maximum conductance for ( K^{+} ) channels</td>
<td>8 \text{ mS/cm}^2</td>
</tr>
<tr>
<td>( \gamma )</td>
<td>Maximum conductance for leakage channels</td>
<td>2 \text{ mS/cm}^2</td>
</tr>
<tr>
<td>( \tau_{syn} )</td>
<td>Synaptic time constant</td>
<td>1 \text{ ms}</td>
</tr>
<tr>
<td>( V_m )</td>
<td>Threshold value for spiking state</td>
<td>0 mV</td>
</tr>
</tbody>
</table>

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![Fig. 2](image-url) **Fig. 2**: Comparison of the population density model with populations of individual neurons with three different population sizes (a, d) 100 neurons, (b, e) 1000 neurons and (c, f) 10,000 neurons. The firing rates are shown in figures (a-c) and snapshots of probability density across membrane potential at time \( t = 25 \text{ ms} \) are shown in figures (d-f). The firing rates of the population density results are shown by the solid lines whereas the individual neuron results are shown by histograms.
populations of individual neurons, histograms of fixed bin size, 0.6 mV and 0.02 ms are used for the membrane potential distribution and firing rate respectively. Both the probability density across membrane potential and firing rates show that the error of the population density approach decreases when the network comprises a large number of identical neurons. For individual neuron population of 100 neurons, the membrane potential distribution and firing rate are sparse to compare to the population density model with the average RMSE = 0.0056 and 0.0486 respectively. Similar results have been shown for network size of 1000 neurons with the average RMSE = 0.0031 and 0.0464. The membrane potential distribution and firing rate for 10,000 neurons compares well with that obtained from the population density model with the average RMSE = 0.0023 and 0.0278.

**Fig. 3.** Comparison of the firing rate for the population density model with populations of individual neurons for two different connectivities: (a) $q_{\text{syn}} = 2500$ and (b) $q_{\text{syn}} = 500$. The firing rates of the population density results are shown by the solid line whereas the individual neuron results are shown by histograms.

**Fig. 4.** Comparison of the firing rate for the population density model with populations of individual neurons for networks of (a) type I and (b) type II neurons. The firing rates of the population density results are shown by the solid lines whereas the individual neuron results are shown by histograms.
5. Single coupled population of excitatory neurons

5.1. Effect of number of connections

The population density approach is improved when the network comprises a large number of sparsely connected identical neurons [6,8,32]. We compare the population density with individual neuron populations of two different numbers of connectivity, \(N_{\text{arr}} = 25000\) and \(500\) in Fig. 1. Parameters used in the simulations were \(N_{\text{arr}} = 10000\), \(J_{\text{arr}} = 0.01\ \text{mV}\), \(J_{\text{ff}} = 0.15\ \text{mV}\), \(q_{\text{arr}} = 1\) and \(q_{\text{ff}} = 120\) arrivals/\(\text{ms}\). Other parameters are given in Table 1. In our simulation results, the smallest RMSE was obtained (0.0012 for RMSE of firing rate) for the low connectivity of \(q_{\text{arr}} = 500\) (Fig. 3[b]). The population results appear closer to the results from individual neuron's networks when the number of connections is small. When the number of connections has increased to 2500, the results of individual neuron's networks begin to diverge from the population density simulation (Fig. 3[a]) with RMSE=0.0104. The sparse coupling lowers the probability of neurons sharing the common inputs. This is justified in the assumption of population density approach in which the input spike trains to each neuron are independent. Therefore, a large error is obtained for dense connected network.

6. Network of excitatory and inhibitory neurons

In this section, the performance of the population density approach is tested for simulating type I and type II neurons. Parameters used in the following simulations were \(J_{\text{arr}}=J_{\text{ff}}=0.01\ \text{mV}\), \(J_{\text{ff}}=0.1\ \text{mV}\), \(N_{\text{arr}}=8000\), \(N_{\text{ff}}=2000\), \(q_{\text{arr}}=1\), \(q_{\text{ff}}=500\), and \(q_{\text{ff}}=200\). Other network parameters are specified in Table 1 [32]. By changing the potassium activation curve, the ML model can reproduce type I and type II neurons. The comparison of the firing rate for the population density model with direct simulation for network of type I and II neurons is illustrated in Fig. 4. Type I and II neurons are driven by external synaptic input with \(J_{\text{arr}}=120\ \text{arrivals/\(\text{ms}\)}\) and 180 arrivals/\(\text{ms}\) (Fig. 4[a]) and 180 arrivals/\(\text{ms}\) (Fig. 4[b]) respectively. Both showing good comparison for population density approach and individual neuron's network with average RMSE=0.0008 and 0.0019.

7. Comparison of computation time

The comparison of computation time for the population density model with direct simulation of a network of ML neurons is shown in Fig. 5. The network consists of \(M\) subnetworks. Each subnetwork has 8000 and 2000 ML neurons in excitatory and inhibitory populations respectively. Each neuron in the population receives synaptic input from randomly chosen neurons inside the subnetwork with probability 0.1 and an external Poisson synaptic input. The computation time shown in the \(y\)-axes is relative to the computation time of single subnetwork of direct simulation with time step of 0.01 \(\text{ms}\). All the neural network simulations are computed by a quad 2.8 \(\text{GHz}\) processor workstation. The population density approach outperforms direct simulation especially when the number of subnetworks is increased.

8. New implications and advances in the study of neural systems

The present approach can be applied to study more realistic models that consist of complex combinations of subnetworks such as the neocortex of human brain. The neocortex can contain up to \(28 \times 10^6\) neurons and a huge number of synapses of the order of \(10^{11}\). The cortical neurons are organized vertically into cortical columns. Each cortical column contains approximately 60,000 neurons which have certain sets of common static and physiological dynamic properties [34]. The present population density approach is suitable for simulating the mean activity of each column using the interactions with adjacent columns as external inputs to the column. Also with the participation of a large network of biologically plausible type I and type II neurons (integrators and resonators respectively), the present population density approach can be applied to study complex phenomena such as resonance and oscillations observed in many biological neurons in thalamic and cortical regions. Resonator neurons with low and high-frequency resonances support the thalamocortical delta-wave oscillations during deep sleep and high frequency rhythm during cognition [35]. Furthermore resonator neurons also exhibit damped subthreshold oscillations of membrane potential that are significant in sustaining synchronized rhythmic activity [36].

9. Conclusion

We have introduced a population density approach for modeling a network of Morris-Lecar (ML) neurons. In order to assess the accuracy, the simulations of the population density method and the direct simulation of individual neurons were compared based on the population firing rates and distributions of neurons across the network. Given that the neuronal network computational speed increases with the number of subnetworks.
a large number of identical neurons that are sparsely connected, the population density approach provides a good approximation to the behavior of networks. For large populations/subnetworks, the computational efficiency of the population density approach is better than direct simulation of individual neurons.

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Appendix A. Supplementary materials

Supplementary data associated with this article can be found in the online version at: http://dx.doi.org/10.1016/j.neucom.2014.02.002.

References


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