The Role of Postsynaptic Potential Decay Rate in Neural Synchrony [1]

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Abstract

Integrate-and-fire neurons have been used widely to model large-scale networks with temporal dynamics. Previous work has focused on temporal *delays* in modulating synchronization behavior; not much attention has been given to the postsynaptic potential (PSP) *decay rate*. In this paper, we show that varying the PSP decay rate has the same effect as adjusting the axonal conduction delay. The decay rate can be adjusted independently at different locations in the neuron, allowing precise fine tuning of synchronization behavior. Also, because the adjustments can be local and small, this process can be more efficient than adjusting the larger-scale axonal delays.

Key words: Integrate-and-Fire Neurons, Decay Adaptation, Synchronization.

1 Introduction

Experimental evidence suggests that temporally correlated activity contributes to binding and segmenting features in sensory input [4, 10]. Thus, temporal information may be very important for neural function. Integrate-and-fire neurons have been studied extensively in this role because their simplicity makes it possible to study synchronization behavior in large-scale networks [2, 8, 11, 13].

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Some of this work assumes that spikes are transmitted instantaneously [2], but others take into account axonal conduction delays [11, 13]. It turns out that the delay characteristic of different types of connections (e.g. excitatory and inhibitory) determine the synchronization behavior in a highly interconnected network of neurons. The main results are: (1) excitatory connections with no delay cause synchrony [2, 9, 11], (2) excitatory connections with delay cause desynchrony [13], (3) inhibitory connections without delay cause desynchrony [11, 13], and (4) inhibitory connections with delay cause synchrony [11, 12, 13, 17]. Previous models of spiking neurons have either adapted or selected the axonal delays to regulate synchronization behavior [7, 16]. Although delay can be adapted by changing the axonal morphology (length, thickness, and myelination; [6]), the fine degree of delay tuning needed in the above models may not be easy to achieve in a macro structure as an axon in biological neurons.

An alternative to delay adaptation is to change the decay rate of the postsynaptic potential. Decay may be easier to alter in biological neurons since ion channels can be added or removed to tune the leakage of currents through the cell membrane. The number and distribution of ion channels can change through various mechanisms including activity-dependent gene expression and activity-dependent modulation of assembled ion channels (see [1] for a comprehensive review). Nowak and Bullier [14] studied various mechanisms of decay (or integration time), and further investigations of these mechanisms may well reveal how decay rate can be controlled. Some models already utilize synaptic decay [5, 15], but the influence of different levels of decay on synchronization has not been fully tested.

Can decay rate affect synchronization behavior just as delay does? This paper shows that different decay rates can indeed cause greatly different synchronization behavior even for the same type of connections (i.e. excitatory or inhibitory). The results suggest that PSP decay rates should be studied in more detail in order to understand how synchronization is modulated in biological neural networks.

2 Model

The neuron model used in this paper is a generalization of the popular integrate-and-fire neuron. Instead of a single integration mechanism per neuron, each individual postsynaptic membrane has a separate integration term with independent PSP decay rates (figure 1).

Each connection between neurons is a leaky integrator that continuously calculates an exponentially decayed sum of incoming spikes: [2]

$$s(t) = x(t) + s(t-1)e^{-\lambda}, \tag{1}$$

[2] This equation has the same form as the one derived from convolution equations in [5].

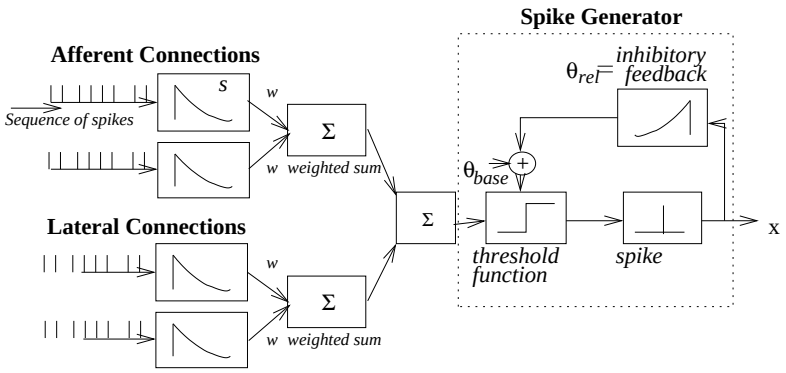


Fig. 1. **The Neuron Model.** Leaky integrators at each synapse perform decayed summation of incoming spikes, and the outgoing spikes are generated by comparing the sum of weighted sums to the dynamic spiking threshold. The dynamic threshold consists of the base threshold θ_{base} and the θ_{rel} that represents the refractory period.

where $s(t)$ and $s(t-1)$ are the current and the previous decayed sum (i.e. at time step t and $t-1$), $x(t)$ is the input spike (either 0 or 1), and λ is the decay rate. The excitatory connections and inhibitory connections have separate decay rates λ_{e} and λ_{i}. Such a leaky integrator models the Post-Synaptic Potential (PSP) that decays exponentially over time in biological neurons. The formulation is a finite difference approximation of the leaky integrate-and-fire neuron when λ is the same for all synapses.

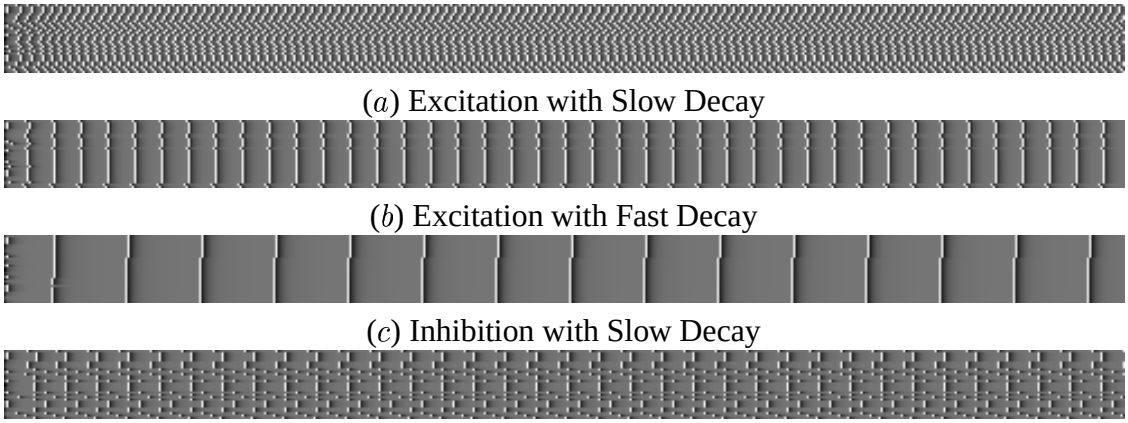
Each cortical neuron in the model is connected to one input neuron and to all other cortical neurons through excitatory or inhibitory lateral connections. The weighted sum of the afferent and lateral leaky synapses are calculated and passed through a squashing function: 0 if sum $< \delta$, 1 if sum $> \beta$, and $\frac{\text{sum}-\delta}{\beta-\delta}$ otherwise. The resulting activation $\sigma(t)$ is compared to the dynamic threshold:

$$\theta(t) = \theta_{\text{base}} + \tau\theta_{\text{rel}}(t), \tag{2}$$

where θ_{base} is the base threshold, $\theta_{\text{rel}}(t)$ represents the refractory period, and τ is a scaling constant. The term $\theta_{\text{rel}}(t)$ is also a leaky integrator as in equation 1, but with a different decay rate λ_{rel}. If $\sigma(t)$ is greater than $\theta(t)$, an output spike is generated.

3 Experiments and Results

Thirty fully connected neurons were simulated for 500 iterations. Each neuron received a constant input of value 1.0 throughout the simulation, and the connection weights were uniform with a value of $1/30$. Afferent input was scaled by 0.8 and lateral input by 0.01 prior to passing through the squashing function to make the total afferent vs. lateral contributions to have a similar range. The squashing function parameters were $\delta = 0.0$ and $\beta = 3.0$. The threshold parameters were $\theta_{\text{base}} = 0.1$,



(a) Excitation with Slow Decay

(b) Excitation with Fast Decay

(c) Inhibition with Slow Decay

(d) Inhibition with Fast Decay

Fig. 2. **Effect of Connection Type and Decay Rate on Synchronization.** Thirty neurons with full lateral connections were simulated for 500 iterations. Four separate experiments were conducted to determine how decay in inhibitory and excitatory lateral connections affects synchronization. The x-axis represents the simulation iteration, and y-axis the neuron index. The membrane voltage is plotted in grayscale, from low to high (black to white). (a) Excitatory connections with slow decay result in desynchronized activity. (b) Excitatory connections with fast decay result in synchronized activity. (c) Inhibitory connections with slow decay result in synchronized activity. (d) Inhibitory connections with fast decay result in desynchronized activity. The results show that synchronization behavior can vary greatly even for the same connection type if the decay rate differs.

$\tau = 0.65$, and $\lambda_{\text{rel}} = 0.05$. Membrane voltage of each neuron was randomly initialized.

Four separate experiments were conducted to test the effect of altered decay rate on synchronization behavior: (1) excitatory lateral connections alone with slow decay ($\lambda_{\text{e}} = 0.1$), (2) inhibitory lateral connections alone with slow decay ($\lambda_{\text{i}} = 0.1$), (3) excitatory lateral connections alone with fast decay ($\lambda_{\text{e}} = 1.0$), and (4) inhibitory lateral connections alone with fast decay ($\lambda_{\text{i}} = 1.0$). The results are summarized in figure 2a–d, respectively.

Two conditions, excitatory connections with fast decay and inhibitory connections with slow decay, resulted in synchrony. In contrast, excitatory connections with slow decay and inhibitory connections with fast decay resulted in desynchrony. These results are similar to the results with conduction delays as reported in [11, 13]. Since decay rate may be easier for neurons to adjust than conduction delay, neurons may indeed employ this process to fine-tune their synchronization behavior.

4 Discussion

Besides PSP decay, several other factors affect synchronization behavior in a network of integrate-and-fire neurons. For example, (1) noisy initialization of membrane voltage, or on-going addition of noise helps desynchronization between different populations [3, 11, 18], (2) higher levels of excitation can overcome moderate levels of noise to synchronize activity, and (3) longer absolute refractory period can help overcome noise and help synchronize neurons. All of these factors were systematically tested with the same model presented in this paper, but because of limited space the results are not presented here (see [3] for the full data).

An interesting future direction is to see whether adjusting the decay rate can counter the effects of conduction delays. A combined model with both adjustable PSP decay rate and adjustable conduction delay can be developed and the interaction of the two processes can be studied. It is possible that adjusting the decay rate can help overcome various side-effects such as unintended desynchronization introduced by conduction delay by resynchronizing the neurons.

Biological experiments can be done to verify whether the dendritic membrane potential can decay at different rates at different locations, and also whether there is such a difference between different types of synapses (e.g. glutaminergic vs. GABA-ergic synapses). If there is a difference, that data can be compared to the results presented in this paper, and it can help us understand the role of these different kinds of connections in modulating synchrony.

5 Conclusion

In this paper, we have shown that synchronization behavior can be modulated by adjusting the PSP decay rate for different types of synapses. Adjusting the decay rate at a local scale can allow a finer degree of synchronization behavior tuning , and it may be a more efficient process than adjusting axonal conduction delays which may require a macro-level change. The computational results presented in this paper calls for further investigations into the role of PSP decay rate in modulating neural behavior.

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