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Predicting spike-timing of a thalamic neuron using a stochastic synaptic model

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Abstract. A twofold spike-timing dependent stochastic synaptic model is used along with a leaky Integrate-and-Fire neuronal model to predict the spike timing of a single post-synaptic neuron in the lateral geniculate nucleus, knowing the spike train on the pre-synaptic side (i.e. in a retinal ganglion cell). In this synaptic model, spike-timing dependency is introduced for both the magnitude and relaxation of the dynamics representing the synaptic action. The results show that the used model is able to reliably predict the exact timing of spikes. These results and the model are the winner of a recent international competition.

1 Introduction

The computational model by Hodgkin-Huxley [1] is considered to be the most influential work in computational neuroscience. This mathematical description of action potential generation and the involved dynamics of ion channels has led to a series of simplified neuronal and a number of synaptic models. These models try in a simplified quantitative way to describe the underlying dynamics of both neuronal and synaptic activities within the neural systems, see e.g. [2, 3]. However, the precise description of neural activity involves a larger number of synergetic and cooperative variables, that may prevent the understanding of the whole underlying dynamics [4]. Moreover, it is not clear if these simplified models are sufficient to realize the essence of combined neuronal and synaptic dynamics. The work in the field of predicting the exact spike-timing of certain neuronal activities comprises either the development of new models [5] or designing algorithms for automatic parameter fitting [6]. It was shown that some of the *neuronal* models are able to yield good and reliable predictions when compared to biological data, see [6] for a short review.

In a recent study, we have reported that adopting certain features into the representation, even with a relatively simple neuronal model, allows to realize a more reliable simulation of the activities observed in the biological neural systems [7]. These features are specifically meant to be the stochastic nature of synaptic release and the explicit representation of synaptic resources. The synaptic resources are the calcium ions and the Neurotransmitter (Nt).

Thus, we present both our model and results in predicting the exact spike-timing from a single post-synaptic neuron in the lateral geniculate nucleus (LGN) knowing the spike train on the pre-synaptic side (i.e. in a retinal ganglion cell (RGC)). Both the model and results were the winning submission of a recent

international challenging competition [8, 9] for predicting spiking times from biological neurons [1]. This competition was based on the work of M. Carandini et al. in [10]. The experimental setup and acquisition of neuronal activity were extensively described in [10] for all details. In short, extracellular recordings were performed in-vivo in rhesus monkeys. Retinal postsynaptic potentials from RGC and LGN action potentials were extracted by off-line waveform templating. Visual stimuli as the light intensity of a LED illuminating only the field center varied continuously, with a temporal frequency power spectrum between 0.2 and 80 Hz. The visual stimulus was 10 s long and was repeated 76 times, see upper panel in Fig. 1. In [10] they tried to predict the spike-timing of the postsynaptic neuron. They used an Integrate-and-Fire (IAF) neuronal model along with a simple synaptic parameterization of the excitatory postsynaptic potential. Their predictions were considered the benchmark for the this experiment. The approach that is reported here is based on the modified stochastic

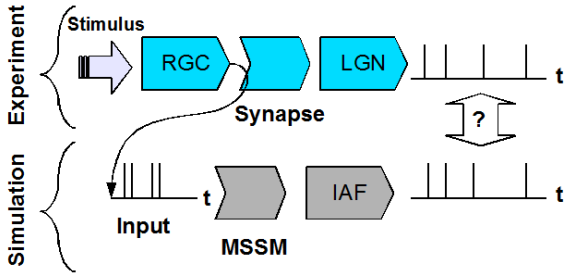


Fig. 1: Schematic of experimental setup (Upper panel) and simulation setup (Lower panel)

synaptic model (MSSM) that we introduced in [7]. The results show that using the introduced framework, the spike times can be reliably predicted. Moreover, a new benchmark was defined as well.

2 The Model

The post synaptic neuron is modeled as leaky-IAF neuron [2]. It is described by its voltage membrane potential u:

$$\tau_u \frac{du}{dt} = -u + E_{psp} - E_{\text{ref}}, \tag{1}$$

where τ_u is the membrane time constant, and E_{psp} is the total observed excitatory postsynaptic potential from all pre-synaptic terminals. When $u(t) \geq u_{\text{th}}$ ($u_{th} = -60\text{mV}$), a spike is generated and $u(t^+) := u_{\text{rest}}$ where $u_{\text{rest}} = -70\text{mV}$. τ_u is the membrane time constant set at 20 mV. E_{ref} replaces a fixed absolute refractory period; it represents the reversal potential for spike rate adaptation

[1] http://www.incf.org/community/competitions/spike-time-prediction/2009/challenge-d

(SRA) and a refractory current. Hence, $E_{\text{ref}} = r_m * (g_{sra} + g_{ref}) * (u - E_k)$; where E_k is set to -65 mV; g_{sra} and g_{ref} are the SRA and refractory conductances respectively: $\dot{g_{sra}} = -g_{sra}/\tau_{sra}$ and $\dot{g_{ref}} = -g_{ref}/\tau_{ref}$; τ_{sra} and τ_{ref} are set to 200 msec and 2 msec respectively. r_m is the neuronal resistance set arbitrarily at 90 MΩ.

The synapse is modeled as a novel twofold stochastic activity-dependent synaptic model using our modified synaptic stochastic model (MSSM) [7]. In general, the synapse is modeled to be stochastically activity-dependent. This model estimates the transmission probability of an arriving spike from a presynaptic neuron via a synapse to a postsynaptic neuron. The probability-of-release involved is governed by two counteracting mechanisms: facilitation and depression. Facilitation reflects the Ca^{2+} concentration in the presynaptic neuron, while depression represents the effect of the concentration of ready-to-release vesicles in the pre-synaptic neuron. The probability that a spike in the spike train triggers the release of a vesicle at a time instant n at a given synapse is given by $P(n) = 1 - e^{(-C(n)\ V(n))}$, where $C(n)$ and $V(n)$ represent the facilitation and depression mechanisms respectively [7, 11]; as discrete time difference equations, they read:

$$C(n) = \alpha\ \theta(n-1) + k_C\ (C(n-1) - C_o) + C_o, \tag{2}$$

$$V(n) = -P(n-1)\ \theta(n-1) + k_V\ (V(n-1) - V_o) + V_o, \tag{3}$$

In eq. 2, k_C corresponds to the decay time constant, τ_C, of the response to a single incoming spike. α represents the magnitude of the response while C_o represents the initial concentration of Ca^{2+} in the pre-synaptic terminal. In eq. 3, $V(n)$ is the expected number of vesicles of neurotransmitter (Nt) molecules in the ready-for-release pool at time instant n. V_o is the max. number of vesicles that can be stored in the pool. k_V corresponds to the time constant, τ_V, for refilling the vesicles. $\theta(n)$ represents the instantaneous input firing rate observed at the synapse at time instant n; it equals then Δ_{isi}^{-1}, where Δ_{isi} is the last observed inter-spike-interval (ISI). As the binding process of Nt in the postsynaptic membrane induces E_{psp}, the equation governing its generation is given by [7, 12]:

$$\tau_{epsp}\ \frac{dE_{\text{psp}}}{dt} = -E_{\text{psp}} + k_{\text{epsp}}\ N_t, \tag{4}$$

where τ_{epsp} is a decay time-constant and k_{epsp} is a scaling factor. N_t is the concentration of the released Nt in the synaptic cleft [7]. This concentration can be estimated by tracing the amount of vesicles[2] of Nt that remains in the presynaptic neuron, $V(n)$, over time. Thus, $N_t(n)$ is:

$$N_t(n) = max(0,\ V(n) - V(n-1)) + N_t(n-1)e^{-\Delta_{\text{isi}}/\tau_N} \tag{5}$$

In eq. 5, $N_t(n)$ is the summation of: a) the estimated amount of Nt added with each release at any time step n (or the decrease in $V(n)$ over the last time step);

[2]Each quantum of Nt is stored in one synaptic vesicle. Thus, the concentration of Nt in the synaptic cleft is meant to be its corresponding concentration of quanta of Nt [3].

where the $max(\ldots)$ avoids negatives plus b) the amount of Nt that remains in the cleft from previous releases. The decay with τ_N reflects the biological cleaning action, or the removal of the Nt from the cleft. Equations 2, 3 and 5 implement the spike-timing dependence as being function of Δ_{isi}. This affects the speed of either the decay or build of any of the above quantities in accordance to the instantaneous timing of invading spikes [7]. The second fold of this dependence which we propose here is implemented for the response constants themselves, e.g. α and C_o. Thus, the magnitudes of the response itself are tuned according to Δ_{isi}. Hence, let

$$V_o = V_o^\star e^{-\Delta_{isi}/\tau_V} \tag{6}$$

$$C_o = C_o^\star e^{-\Delta_{isi}/\tau_C} \tag{7}$$

$$\alpha = \alpha^\star e^{-\Delta_{isi}/\tau_C} \tag{8}$$

$$k_{epsp} = k_{epsp}^\star e^{-\Delta_{isi}/\tau_{epsp}} \tag{9}$$

The parameters $V_o^\star$, $C_o^\star$, $\alpha^\star$ and $k_{epsp}^\star$ are the starting values of the corresponding variables. The starting values for all the simulation parameters are adopted from [13].

3 Training and Benchmark Test

The data set in general comprises 76 pair of files, as a pair per repetition. For each pair, one file contains the input spike times while the second holds output spike times. The odd repetitions out of the 76 ones are used as the training set, while the even ones are the test set. Training is implemented by applying the Hebbian rules to the starting values of the response constants only. Thus, all the timing decay constants are set to biologically plausible values similar to those reported in [13]. The training process involves 200 training runs. In each run, a repetition is randomly selected from the training set and its input is fed to the model; the parameters are tuned accordingly using its Carandini output as the reference signal (briefly explained in next paragraph). Each repetition is not allowed to appear more than 6 times across the whole training process to ensure the introduction of the available 38 repetitions. The training algorithm can be summarized as follows: each constant, m_i, can contribute to either excitatory or inhibitory regimes in the synaptic action; and according to the pre- and postsynaptic activity, its value is either increased or decreased following the Hebbian approach [12]. The value of the excitatory variable is increased while it is decreased for the inhibitory one when a spike at the pre-synaptic neuron induce a correct timed spike at the post-synaptic neuron, and vice versa. The update of the parameter values, then, can be read as $m_i^{\text{new}} = (1 \pm r_l) m_i^{\text{current}}$ [7], where r_l is the learning rate. m_i is each constant of the tunable parameters: $V_o^\star$, $C_o^\star$, $\alpha^\star$ and $k_{epsp}^\star$. By training, they are set to 3.7, 0.05, 0.09 and 6 respectively.

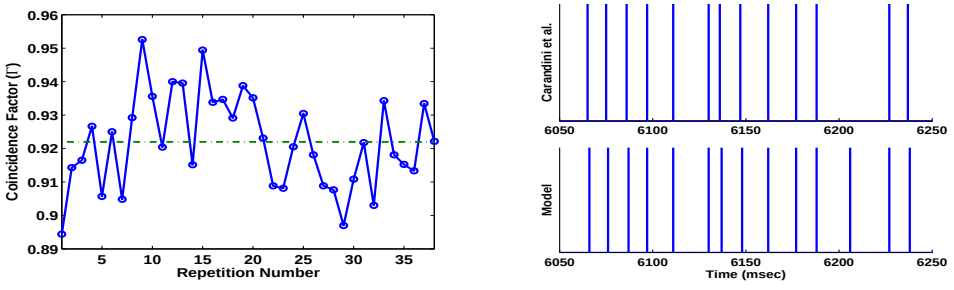
The benchmark test was originally introduced in [4]. This test evaluates quantitatively the predictions between two spike trains as a coincidence factor Γ. To evaluate this quantity, the number of coincidences $\mathcal{N}_{coinc}$ is calculated

between the spikes in the Carandini spike train from one repetition (as target) and the spike train from simulation. This number is calculated by counting the number of target spikes for which we can find at least one simulated spike within $\pm$ 4 ms. Then, the expected number of coincidences $\langle \mathcal{N}_{coinc} \rangle$ that a Poisson spike train with the same average frequency would give is calculated. The factor Γ, thus, reads [4]:

$$\Gamma = \frac{\mathcal{N}_{coinc} - \langle \mathcal{N}_{coinc} \rangle}{0.5(\mathcal{N}_{Crnd} + \mathcal{N}_{Mdl})} \frac{1}{\nu} \tag{10}$$

where $\mathcal{N}_{Crnd}$ and $\mathcal{N}_{Mdl}$ denote the number of spikes from the Carandini data set and simulated (the model) spike trains respectively; ν is a factor that normalizes the coincidence factor Γ to a maximum of 1. $\Gamma = 0$ implies that the prediction is not better than chance level. $\Gamma = 1$ implies that the prediction by the model is optimal, for more details please review [4, 6]. The predictions from Carandini et al. [10] yield a coincidence factor of $\Gamma = 79.1\% \pm 0.6$ for the test set.

4 Results and Discussion



(a) (b)

Fig. 2: a) Mean Γ of the model per repetition; Dashed line: the mean value across repetitions. b) Sample from the 9th repetition. Upper Panel: The target output from the LGN neuron from [10]. Lower Panel: Spikes from the model.

The experimentally recorded spike times from RGC neurons are the input data while those from the LGN neurons are the output target ones, see lower panel in Fig. 1. The Carandini input train of spikes from each repetition is processed through the synaptic model and the neuronal model as described above. The simulated output spikes are then compared to the recorded data set for each repetition, and the spike-timing coincidence factor Γ is calculated consequently. Figure 2.a illustrates the value of this factor for all the 38 training odd repetition. The mean Γ factor across them is $92.2\% \pm 1.3$. The output data set for the even repetitions (test set) is not yet available from the organizers of the competition but are used to calculate the challenge performance and thus were unknown to the competitors. The results of the challenge has been announced

that the mean Γ factor is $90.6\% \pm 0.3$. Figure 2.b shows sample of simulated and target Carandini spikes. The results illustrate a high precision in predicting the spike-timing within a time window of 2 msec.

Hence, the framework presented here is able to reliably capture the transformation between the incoming and outgoing spike trains for a single defined neuron-synapse-neuron circuitry in the visual system. These results with the above mentioned coincidence factor (90.6%) has been considered the new benchmark for this predictions. This study ensures the role of adopting a wealthy synaptic dynamics in order to capture more realistic features from the biological nervous system. It remains to be seen if a simplified synaptic parameterization, e.g. the synaptic model from Markram et al. reviewed in [3], can accomplish a comparable performance. Considering the neural prosthesis and rehabilitation options, the hardware implementation of such model represents a highly challenging but yet a promising task as well.

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