

Functional Resonant Synaptic Clusters for Decoding Time-Structured Spike Trains

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Abstract.

Biological neurons communicate with each other using two broad categories of spike event coding: rate-based and temporal. Rate-based coding communicates analog information on a continuous scale through the intensity of bursts of spikes while temporal coding relies on the timing of spike events. It has been shown that temporal coding has higher information capacity than rate based coding, but is much more challenging to model due to difficulties estimating spike-time statistics. In this paper we demonstrate how history dependent NMDA-modulated ‘resonant’ synapses organised in ‘functional synaptic clusters’ provide a robust mechanism for decoding temporally structured spike trains.

1 Introduction

Spiking neural networks (SNNs) are biologically inspired event driven models that are gaining much attention in the Machine Learning (ML) community, largely due to the computational efficiencies they afford compared to conventional artificial neural networks (ANNs) [1, 2]. Numerous SNN models proposed and studied in the literature use rate-based coding, despite the fact that temporal coding is capable of representing a wider spectrum of information types such as categorical information, which can be represented by the relative order of specific sequences of inter-spike intervals (ISIs) that make up the sequence [3].

Decoding time structured spike trains, however, remains challenging, particularly with respect to the problem of overlapping spikes carrying different timings arriving at the same neuron. Current state-of-the-art in synaptic adaptation requires that spikes are synchronous (i.e. phase-locked to a ‘reference’ spike train of constant period) otherwise there can be no way of preventing a fortuitous timing overlap corresponding to an encoded pattern. This paper presents a new asynchronous learning mechanism based on the statistics of ISI timing sensitivities in synapses to address the decoding challenge.

Recent experimental evidence strongly suggests that certain classes of biological synapses are (short-term) history dependent [4], responding to the short-term temporal features of incoming presynaptic spike trains. A growing body of research [5, 6] indicates the potential importance of N-methyl D-aspartate (NMDA) synaptic receptors in the mediation of timing-dependent learning. Our methodology aims to simulate these NMDA mechanisms at a functional level (as opposed to an accurate biophysical level) in order to create synapses that differentially adapt to particular ISIs.

Using such ‘resonant’ synapses to decode temporal sequences of ISIs requires local coordination between each synapse responding to a particular ISI incident on a given neuron. If a given sequence is to be decoded, multiple resonant synapses must transmit in a specific order to induce a postsynaptic action potential that signifies the successful decoding of the presynaptic spike train (Figure 1B). Further recent experimental evidence indicates the existence of functional synaptic clusters where nearby dendritic synapses are recruited into self-organised groups capable of collectively generating specific transmission responses [7]. Our model will make use of this to enable the emergence of clusters of synapses that align their axonal and dendritic time delays to respond jointly to the specific sequences of ISIs of a given spike train (Figure 1C).

2 Methodology

We take as a starting point the NMDA synapse. NMDA kinetics, acting on timescales on the order of tens of milliseconds, involve a magnesium block [8] that must be ‘unblocked’ by an initial spike (action potential) to trigger eligibility for synaptic modification mediated by Ca^{2+} influx, which can then be induced by a second spike arriving during the window of the Mg^{2+} unblock.

We seek a reasonable computational approximation for the NMDA channel kinetics that can be modified under the action of spike pairs to produce responses tuned for specific ISIs, without necessarily having to model detailed biophysical kinetics. If the hypothesis of Shouval, et al. [5], that a slow depolarising tail in the NMDA kinetics leads to favourable conditions for backpropagating action potentials (BPAPs) in the dendrites, and following [8] this response is in turn a consequence of asymmetric unblocking of individual channels (localised receptor sites), then we may consider the likelihood of unblock of a single channel as a Bernoulli trial (random event with binary outcome) with some probability in the range $[0,1]$. We aim to model the open channel probability, $P(o)$, of the synapse.

Assuming that there are many such individual channels at a synapse and that the overall kinetic is governed by the number of channels unblocked, the posterior distribution $P(c|o)$ where c is the number of open channels, will be a binomial distribution, whose conjugate prior is the beta distribution $P(o) = \frac{o^{a-1}(1-o)^{b-1}}{B(a,b)}$ where $B(a,b)$ is the Beta function $B(a,b) = \int_0^1 u^{a-1}(1-u)^{b-1} du; a, b \in (0, \infty)$. It therefore seems plausible to model the channel kinetics using a beta distribution. The mean of the distribution would represent the optimum delay in the arrival of successive presynaptic spikes, that will facilitate synaptic transmission. If we further postulate that the distributions two hyper-parameters α and β can be modulated during induction of synaptic plasticity, we can introduce a mechanism for learning of specific ISI delays.

The Beta distribution is defined over the range $[0,1]$. This is suitable for defining open channel probability; however, it is convenient to use time as a proxy variable for the open channel probability, and in this case, we need a distribution defined over the interval (t_{min}, t_{max}) where the time bounds are a further pair of hyper-parameters defining the timescale of the NMDA response

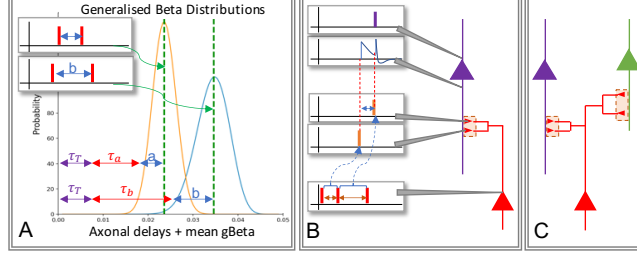


Fig. 1: The resonant synaptic concept

kinetic. For this purpose, we employed the generalised beta distribution [9]:

$$gBeta(x; a, b, t_{min}, t_{max}) = \frac{(x - t_{min})^{a-1} (t_{max} - x)^{b-1}}{B(a, b) (t_{max} - t_{min})^{a+b-1}} \quad (1)$$

over the open interval specified by t_{min} and t_{max} , where $B(a, b)$ is a beta distribution with shape parameters a and b . The mean and variance of the generalised Beta distribution (Equation 1) are given by $\mu = t_{min} + \frac{a(t_{max} - t_{min})}{a+b}$ and $\sigma = \frac{ab(t_{max} - t_{min})^2}{(a+b)^2(a+b+1)}$ respectively. Solving a system of two equations (μ, σ) with two unknowns (a, b) gives the values of a and b as:

$$a = \frac{l(t_{max} - t_{min})^2 - \sigma(l+1)^2}{\sigma(1+3l+3l^2+l^3)}; \quad b = al, \quad \text{where } l = \frac{t_{max} - \mu}{\mu - t_{min}} \quad (2)$$

The values of min and max were treated as known ($min=0, max=0.05$).

We used Brian2 (<https://briansimulator.org>) to create a model for the NMDA synapse and integrated it with a standard leaky-integrate-and-fire (LIF) model evaluated using closed-form integration to build a very simple series of networks that can learn to decode temporally coded spike trains between neuron pairs. The details of the model are as follows. Neurons, as noted, use the Leaky Integrate-and-Fire model:

$$\tau_m \frac{dV}{dt} = V_r - V + \frac{\tau_m (I_{syn} + I_{os})}{C_m}; \quad \text{If } (V \geq V_t), V = V_s \quad (3)$$

Synapses are modelled using the unimodal Generalised Beta Distribution (GBD) indicated in equation 1, which approximates the experimentally observed NMDA kinetics. Parameters of the GBD were initialised randomly per synapse. Synaptic output was generated by Bernoulli sampling with probability equal to the value of the GBD corresponding to the ISI since the previous spike, with fixed current injection of 0.075 nA for a membrane capacitance of 1 nF.

Each synapse has an independent temporal delay in the range 10-20 ms for spike arrival from the pre-synaptic neuron. These temporal delays were adapted in batch mode to enable the transmission of multiple synapses in short-term temporal sequences (e.g. $t_1, t_2 \dots t_n$), preserving the order of the inter-spike

intervals of the received spike train ($ISI_1, ISI_2 \dots ISI_n$). The delays between all transmissions apart from the last one were calculated to result in an excitatory post-synaptic potential (EPSP) below threshold but above rest for neuron (v_b), whilst the delay of the last transmission of the sequence was calculated to achieve above-threshold EPSP for neuron (v_a). Time delays were calculated using $t_T = -\tau_m \log((v_T - I_s)/v_s)$, where t_T is the target time delay between successive transmissions, τ_m is the decay constant in Equation 2, v_T is the target potential (either v_b or v_a), and v_s is the anticipated pre-transmission potential which will be either 0 if the spike is the first transmission of the sequence, or v_b following a previous sub-threshold transmission. The initial delays were updated by a delta-rule-like function which moves the delays a fractional step towards the target delays. Synaptic plasticity was modelled by adapting the mean and standard deviation of the GBD according to the statistics of the corresponding ISIs.¹

3 Results

Two experiments were carried out using our model. Experiment 1 tests the capacity of a two neuron model to selectively decode a specific time structured spike train amongst a series of similar spike trains. Experiment 2 explores the precision and recall of a three neuron model with jittered versions of two different time structured spike trains.

In Experiment 1 the architecture shown in Figure 1B was used to study the functional resonant synapse cluster's capacity to correctly decode a specific three spike input. The input consisted of a series of 3-spike sequences in which the timing of the first and the third spike relative to each other were at a fixed interval, but the timing of the second spike was varied to sweep a range of values between the first and third spikes. One of these sequences was arbitrarily selected as the target sequence to decode and the delays and the gBeta parameters of the two resonant synapses were manually set to decode the target sequence.

Figure 2 presents the results of one pass through the full input sequence. As can be seen, the model successfully decodes the target sequence (indicated with '*'), including slight variations thereof. Note also that later in the input there are sequences which have ISIs that cause the synapses to transmit, but because these intervals are not in required order, the transmissions do not result in the erroneous firing of the output neuron.

Experiment 2 sought to evaluate the precision and recall capacity of the synaptic adaptation mechanism of this model using a 3-neuron network, as shown in Figure 1C. The input neuron emits noisy versions of two distinct time-structured spike trains labelled 'I1' and 'I2'. Each output neuron is connected to the input neuron via a cluster of two resonant synapses. After learning, each neuron should fire only and uniquely for variants of one of the patterns.

A 'training signal' in the form of an above-threshold input to the corresponding output neuron, is used to indicate to the synaptic clusters which of the two

¹Full model details are given in the supplementary material at <https://github.com/ntcrook/ESANN2023>

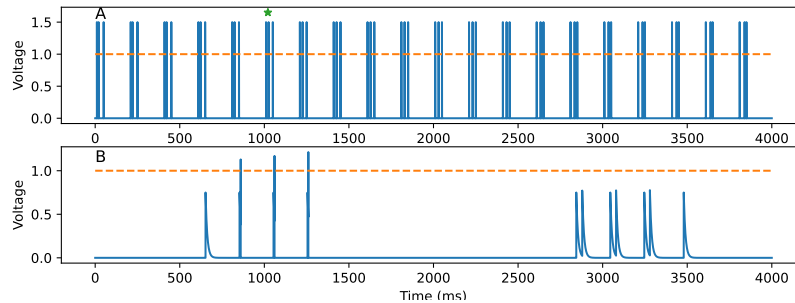


Fig. 2: Time series of the neurons for Experiment 1. Dashed line indicates firing threshold. Top plot is presynaptic, bottom postsynaptic. ‘*’ = target sequence

output neurons should fire in response to a given input sequence. When a cluster adapts to a given input sequence, it will assign each incoming ISI to the corresponding resonant synapse deterministically (i.e. first ISI to synapse 1, second to synapse 2 etc). The activated cluster will adjust the mean and variance of its synapses’ GBD and their relative axonal delays to decode the input sequence.

The results from fifty experimental runs were collected using this network. Each run consisted of alternating jittered versions of spike trains I1 and I2 totalling 700 spike train sequences. Adaptation was not activated during the first and last 100 sequences, during which precision and recall data was collected and averaged, but was otherwise enabled. Delays and GBD parameters of each synapse were uniformly randomised at the start of each experimental run.

	Precision	Recall
Pre-training	0.481	0.183
Post-training	0.856	0.919

Table 1: Precision and Recall results from Experiment 2

The precision and recall data before and after adaptation gives a measure of how successful the adaptation mechanisms have been in facilitating the decoding of the two training patterns. Table 1 shows precision and recall values averaged over the 50 experimental runs, using jittered versions of I1 and I2. These values were calculated using counts of true positives, false positives and false negatives of the spikes generated by each output neuron. Results demonstrate that the proposed model for adaptive functional synaptic clusters is capable of learning to successfully decode jittered versions of target time-structured spike trains.

4 Conclusion

Our new ‘resonant’ synapse model introduces a simple computational method demonstrating the viability of a timing-based approach to spiking neural computation. The model is loosely based upon biology, but uses a series of approximations to achieve a tractable form for large-scale networks. Our aim is to use the learning rule integrated into the model to generate efficient networks for real-time computing applications. It should be emphasised that this particular model is an *empirical approximation* - it is not designed either to replicate exact biophysical kinetics or to represent a causal model for how those kinetics might arise. However, there may be useful implications for biology arising from the parametric properties of the model. There are also interesting future theoretical problems in information theory: our experiments revealed potential bounds on the ISI width that corresponds to the same ‘symbol’ in the input space, and we are interested in developing formal expressions for the representational capacity of the overall network and information-per-spike of any given input sequence. Although our results to date are simply a preliminary demonstration of concept, they set the stage for a new generation of efficient, spike-based neural models.

References

- [1] Yamazaki K, Vo-Ho VK, Bulsara D, Le N. Spiking neural networks and their applications: A Review. *Brain Sciences*. 2022;12(7):863.
- [2] Greengard S. Neuromorphic chips take shape. *Commun ACM*. 2020;63(8).
- [3] Gütig R, Sompolinsky H. The tempotron: a neuron that learns spike timing-based decisions. *Nature Neurosci*. 2006 Mar;9(3):420-4.
- [4] Bird AD, Richardson MJ. Transmission of temporally correlated spike trains through synapses with short-term depression. *PLOS Comp Biol*. 2018;14(6).
- [5] Shouval HZ, Bear MF, Cooper LN. A unified model of NMDA receptor-dependent bidirectional synaptic plasticity. *P Natl Acad Sci USA*. 2002 Aug;99(16):10831-6.
- [6] Harnett MT, Bernier BE, Ahn KC, Morikawa H. Burst-timing-dependent plasticity of NMDA receptor-mediated transmission in midbrain dopamine neurons. *Neuron*. 2009;62(6):826-38.
- [7] Hedrick ea Nathan G. Learning binds new inputs into functional synaptic clusters via spinogenesis. *Nature Neurosci*. 2022;25(6):726-37.
- [8] Vargas-Caballero M, Robinson HPC. Fast and Slow Voltage-Dependent Dynamics of Magnesium Block in the NMDA Receptor: The Asymmetric Trapping Block Model. *J Neurosci*. 2004 Jul;24(27):6171-80.
- [9] McDonald JB, Xu YJ. A generalization of the beta distribution with applications. *J Econometrics*. 1995 Mar;66(1-2):133-52.