Shadowing and Blocking as Learning Interference Models

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Abstract

The link between reward and reward-eliciting actions may be impaired by increased sensitivity to background noise during the waiting period (Shadowing) and may also be interfered simultaneous presentation of two redundant inputs. Clinical studies have shown acute Schizophrenia patients lack the ability to blocking (learning interference). The model presented in this study aims to illustrate the effects of these interferences on the reinforced learning model and to identify the parameters that contribute to this perturbation. Based on our model, blocking may result due to decreased in response to coincidental firing or depletion of eligibility tracer with time while shadowing may result due to increased in sensitivity to background noise. Understanding the model of learning interference may provide insight into diagnosis of acute Schizophrenia.

1 Introduction

Classical Pavolian conditioning large-scale behaviour as well as neural networks relies upon the reward consistently coming seconds after the reward triggering actions. The distal-reward problems is the problem encountered when trying to link the reward to the reward-eliciting action as it comes second after, when the pattern of activity has changed. Various models have been hypothesised, but the most effective is the coincident-activity detector reliant upon a slow decaying synaptic tag that occurs when post-synaptic activity succeeds pre-synaptic activity (Izhikevich 2007).

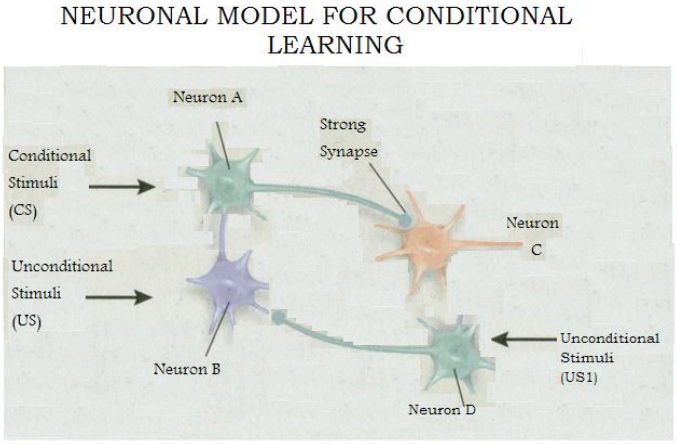

Figure 1: Neuron B however has synaptic connections onto Neuron A and Neuron D has connections onto B. As a result if the activity of Neuron B consistently precedes that of Neuron A then a direction connection forms between B and C, bypassing C; similar case occurs with Neuron D as well.

Classical conditioning relies upon the neuronal system being able to consistently update its associations, allowing the first event (firing) in the resulting cascade to be directly associated with the final outcome.

Blocking on the other hand is impaired learning of an association between a conditioned stimulus and unconditioned stimulus (Jones, Gray et al. 1990). This occurs due to simultaneous activity of another condition a stimulus that is already associated with the unconditioned stimuli. The blocking effect and lack thereof through dopamine super-sensitisation has been extensively demonstrated and studied in rate models (Crider and Blockel et al. 1986). It has essentially been found that the blocking effect is not as significant during incidental learning; as more attention is paid to non-salient aspects of the information.

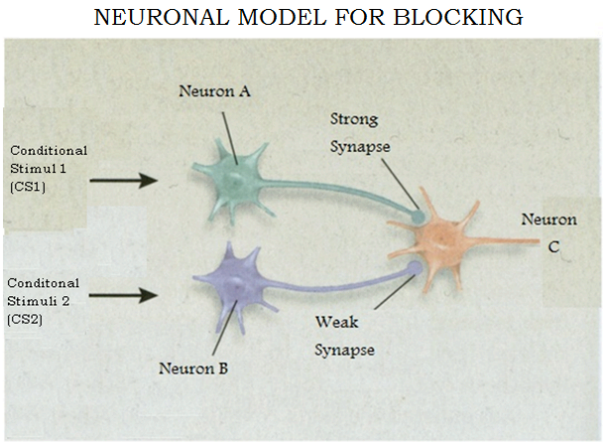

Figure 2: Neuronal Model for Blocking, impaired association between B and C. Neuron A and C are already synaptically connected, therefore when B and A fire simultaneously before C, only A is strengthened. Neuron B is instead blocked, as it is ignored as redundant information from a parallel channel.

Clinical studies with acute Schizophrenia patients revealed an absence of the blocking effect (Hemsley & Richardson, 1980). This is due to the fact that patients are more aware of and distracted by non-salient aspects of the information, making it hard for them to distinguish between relevant and redundant information (Jones, Gray et al. 1992).

Shadowing however is a reduction in the significance of the reward eliciting activity due to an increase in the sensitivity to background noise. Parkinson (1972) showed that there is impaired short-term memory during multi-tasking activity. The increase in sensitivity leads to a greater proportion of the synapses being potentiated and increasing in synaptic strength. As a result, it is difficult to discern the actual stimuli (reward-eliciting behaviour) among all the noise. Figure 3 illustrates this concept; since both neuron A and B synaptic connection to neuron C result increase synaptic strength during high dopamine level, then neuron C can not determine which connection results in dopamine increase.

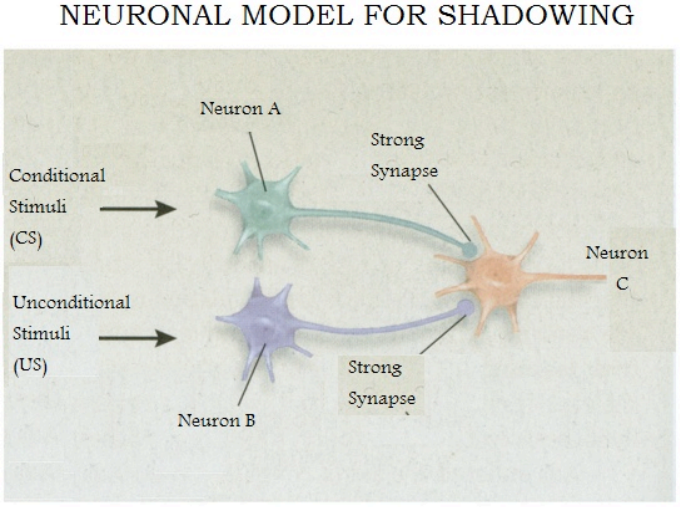

Figure 3: Neuronal model for Shadowing. Increases in the sensitivity to background noise leads to both Neuron A and B synaptic connections increasing in their synaptic connections with Neuron C. As a result it becomes significantly more difficult to discern the stimuli from Neuron A, when B is potentiated as well.

Aims

There was two distinct parts to this investigation, each aimed at investigating various aspects of learning interference in the presence of background noise. The first part involved modelling blocking of redundant information from a parallel stream, as a means to generate more efficient transmission.

The other section involved modelling shadowing of the reward-eliciting stimuli by introducing jitter into the system and making it more sensitive to background noise.

Therefore the overall aims of this investigation were to explore various modes of learning interference.

2 Method

In order to model blocking and shadowing, a model described by Izhikevich (2006) was implemented. All neurons are modeled as spiking neuron with one gating variable u and one voltage variable v, in the equations below.

$$\dot{v} = 0.04v^2 + 5v + 140 - u + I_{current}$$

$$\dot{u} = a(bv - u)$$

$$if \quad v = 30mv$$

$$then \ neuron \ \ fires$$

The synaptic connection between neurons and the modulation of synaptic strength is modeled using the phenomenological model of Dopamine (DA)

modulation of Spike Timing Plasticity (STDP). The state of each blocked synapse can be described by its synaptic strength (s), and eligibility trace (c) in the following equation:

$$\dot{c} = -\frac{c}{\tau_c} + STDP(\tau)\delta(t - t_{pre/post})$$

$$\dot{s} = c(d - d_0) * be^{-at}$$

$$\dot{d} = -\frac{d}{\tau_d} + DA(t)$$

$$\tau = t_{post} - t_{pre}$$

Where d(t) describes the extracellular concentration of dopamine, d_0 is the tonic extracellular concentration of dopamine and the parameters a and b are constants that were used to decay the blocked synapses. In the simulation a= 0.01, b=1.5 and d_0= 0.01. For non-blocked neurons

$\dot{s} = c(d - d_0)$

As described in Izhikevich (2007), the Dirac function provides the step-increase in variable c(t) when pre proceeds post synaptic neuron firing. c(t) changes by the amount specified by STDP within the time interval. When pre-proceed postsynaptic neuron firing then STDP results in potentiation of the postsynaptic neuron and decreased synaptic strength in the blocked neurons or increase synaptic strength in the non-blocked neuron.

To model shadowing, the time constant for the eligibility trace was increased to increase the probability both STDP occurring during high level of dopamine concentration extracellular. This will increase the random strengthening of the synapse in the neurons that fire randomly.

$$\dot{c} = -\frac{c}{\tau_{shadowing}} + STDP(\tau)\delta(t - t_{pre/post})$$

$$\tau_{shadowing} > \tau_c$$

This simulation of this model utilizes a network of 100 spiking neurons with 10 sypnases per neuron as described by Izhikevich (2006). 80% of the neurons were excitatory and 20% per inhibitory neurons with random poison-like spiking behavior. The solutions to the differential equations of each neuron when synaptic strength is increased are shown in figure 1 of Izhikevich (2007).

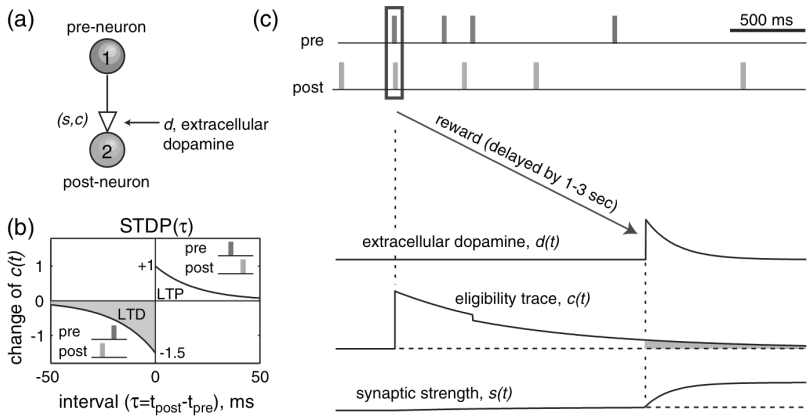

Figure 4: Solutions to the differential equation of the neuro network.

3 Results

From the 100 neuron simulated for 1500 sec using Izhikevich (2007) reinforcement model as the control group, we see that when pre synaptic neuron fires before the post synaptic neuron, STDP occurs and eligibility tracer concentration pulsed as shown in Figure 5 as the green curve. If dopamine concentration is high during a high eligibility concentration as indicated by the red x in figure 5, the synaptic strength increase in a multiplicative effect of eligibility tracer and dopamine concentration as indicated by the blue curve in figure 5. The first simulation was to compare the blocking network model with the control model. As shown in Figure 5, the synaptic strength of the control increases with every coincidental firing with dopamine and eligibility tracer on, while in the blocking network the designated blocked neurons show a decreased response in the synaptic strength increase and a very small time constant.

There is a decrease in synaptic strength response to coincidental reward driven by presence of dopamine and eligibility tracer with time in the network with blocking as indicated by Figure 5.

For shadowing, the histogram was used to assess the decrease of shadowing with time. In the control at time zero, Figure 6A, the histogram of classical reinforcement learning begins with majority of one synaptic strength and some Gaussian distribution during the waiting period for reward. However with time, this histogram is transformed as the synaptic strength of the coincidental firing neurons are increased reflecting the increase in eligibility trace during high dopamine concentration, Figure 6B. The variance of the Gaussian distribution increase within the network as the network increases in sampling.

In the shadowing simulation, there is a dramatic difference in the synaptic strength histogram. As expected there the distribution moves from a Gaussian

distribution of random noise with one neuron at the maximum synaptic strength to a uniform distribution where most neurons have high synaptic strength as indicated by Figure 7.

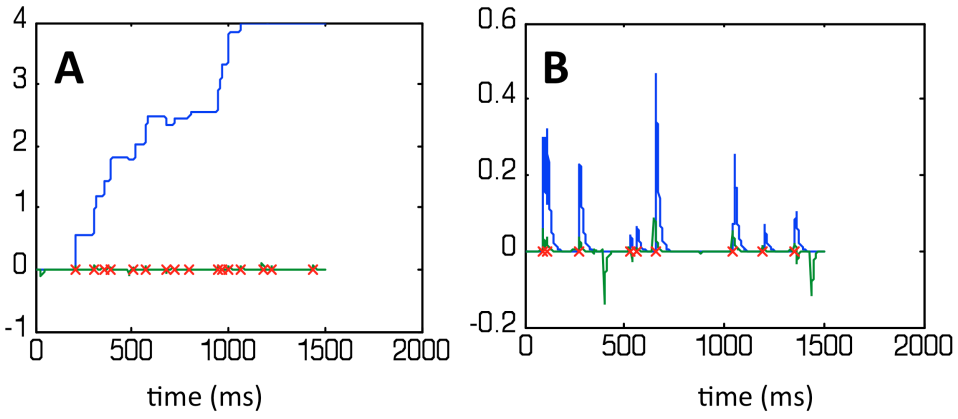

Figure 5: A. Synaptic plasticity or increase in synaptic strength **B Blocking.** (Neuron blocking, or decreased in synaptic strength with time. Synaptic strength of neurons is indicated by blue, red indicates the dopamine injection and green indicate the eligibility tracer, which may be negative or positive.

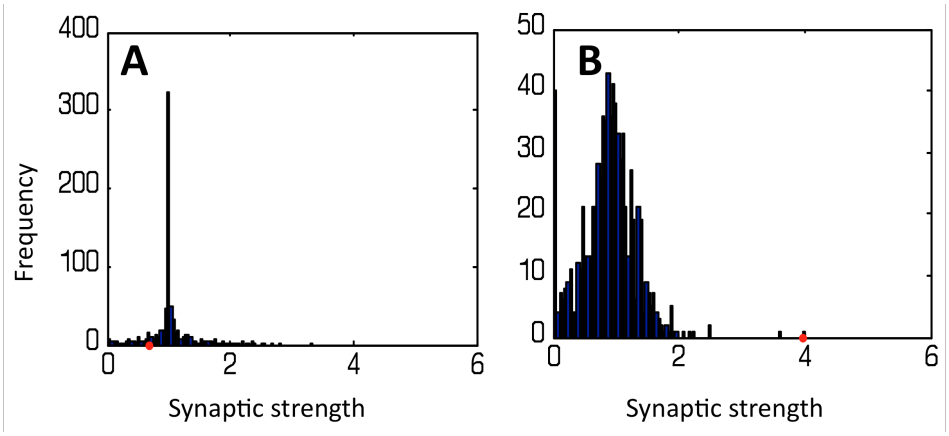

Figure 6: A. Random (Bolzmann distribution) firing at time zero**. B Classical.** With increased sampling rate, only one neuron exhibit synaptic plasticity with maximum synaptic strength. Red dot indicates the neuron of interest.

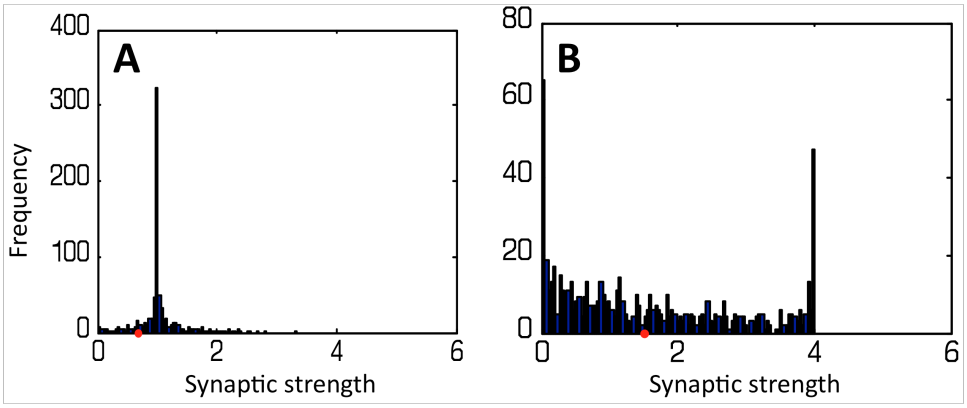

Figure 6: A. Random (Bolzmann distribution) at time zero**. B.Shadowing** With increased sampling (after 1.5 sec simulation) the Bolzmann distribution evolved close to a uniform distribution with more than 60 percent of the neuron exhibits synaptic plasticity with maximum synaptic strength.

5 Discussion

Blocking and shadowing were both successfully modelled in a 100 neuronal network in Matlab, as various forms of learning interference. Blocking was found to cause redundant information in the form of coincidental firing of a secondary neuron to be suppressed. Shadowing on the other hand caused the original signal to be masked, making it harder to discern in the presence of a great number of synapses all increasing in synaptic strength. Therefore blocking and shadowing are opposite forms of learning interference and both were successfully modelled within this investigation.

Blocking of a secondary neuron with coincidental firing was modelled in this investigation through the introduction of a secondary type of synapse that decayed in synaptic strength. This is primarily due to the introduction of an exponential decay factor.. The modification of the synaptic strengths and currents for the specific neuron led to a consistent decrease in the synaptic strength over a period of time as expected. Initially the synapse responds to the activity to the same extent as the non-blocked one, however over a period of time there is desensitisation and this was clearly seen with the low synaptic strengths. Physiologically, the decrease in the response to incidental firing may be due to the depletion of eligibility tracer with time or impaired assembly of eligibility tracer machineries.

Shadowing however was modelled by introducing a greater extent of jitter and degree of variability within the system, by increasing the coincidence interval by an order of magnitude. A greater number of random firings as a result had the required configuration of pre-synaptic before post-synaptic leading to them being potentiated and increasing in synaptic strength in the presence of dopamine injection. The overall effect was a greater number of synapses

being potentiated, making the original stimulus and resulting synapse connection harder to discern; effectively shadowing the actual learning.

The validity of this computational model for the learning interference conditions in animal models is still unverified. Therefore experimental studies on rat hippocampus region with stimuli paradigms aimed to elicit blocking and shadowing through coincidental activity would be a great future direction. This would test whether the simplification of the biological processed underlying synaptic plasticity at both the pre-synaptic and post-synaptic terminal, as a simple synaptic tag is valid.

This is essential as many neurobiology studies have demonstrated that synaptic plasticity has many different forms that relies upon very distinct molecular mechanism that have unique time courses as well (Zurcker and Regehr 2002). Understanding the model of learning interference may provide insight into diagnosis of acute Schizophrenia and also understanding the mechanism of learning.

The next step would be to actually introduce a larger number of gating variables within the neuronal network and analyse the resulting membrane voltage changes rather than just the synaptic strength. This would allow an effective dynamic analysis of how the membrane currents and the firing rates of the population of neuron change within these learning interference models.

Overall however this investigation has been relatively successful in modelling both blocking and shadowing and their resulting effects on synaptic strength changes for a population of 100 neurons.

6 References

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