Implementation of a Spiking Neural Network with Dopamine

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December 19, 2009

Abstract

Spiking Neural Networks are an interesting implementation of a biologically plausible mechanism for computation. They are significantly different from Artificial Neural Networks, in that they do not all fire at each time step. There are a number of factors that influence neural networks, such as Axonal conduction delay, that are not possible within the realm of Perceptron-based ANN. It has been shown that SNN can mimic the properties of ANN. However it is possible that the capabilities of the SNN greatly outpace those of the ANN, especially when the SNN operates both spatial and temporal mechanisms for computation. The most well known example of a SNN in practice is the human brain, capable of untold high dimensional processing. In fact the human brain is the currently most powerful processing device known to man. Recreating it's characteristics and mechanisms within a computational model is important, and Spiking Neural Networks are at the heart of that effort. In this paper we present an initial implementation of a SNN based on the Izhikevich Model.

1 Introduction

Spiking Neural Networks are an active area of research within Neural Computation. Since the integrate & fire neuron model, a great deal of models have been developed that can be considered Spiking. These differ from the Boolean neuronal logic [8], the perceptron [9], and the derivations of these concepts over the years because the Spiking Model does not explicitly calculate a solution manifold. However these models are plagued by the inability to scale up to large or highly dimensional problems, and therefore cannot achieve the level of general intelligence that is found within humans. However human intelligence does not exist within virtual grid worlds, but instead exists with time and space that is far richer in detail and dimension than we can perceive. For that reason the mammalian brain evolved to operate utilizing the mechanisms of time and space in order to generate intelligent behavior, giving it an evolutionary advantage. For this reason Artificial Neural networks such as the Perceptron or it's more advanced predecessors will never successfully emulate general intelligence. The consideration of how the brain operates may be a higher level of discussion than necessary, however it is important to analyze. Consciousness integrates experiential data that is generated from a vast number of spatial and temporal processes [12]. We can only assume that the mechanisms that arose to operate within this conditions utilize both space and time in a computational framework, and any artificial mechanism we hope to operate intelligently within our environment must do the same. This leads us to the key difference in Spiking Neural networks and Artificial Neural Networks. Spiking Neural networks utilize temporal mechanisms such as Axonal conduction delay in order to modulate the "signal" time from one neuron to another. Neurons operate utilizing Spike Timing Dependent plasticity, in which the strength/weight of a synapse is modulated based on it's post-synaptic and pre-synaptic firing times. Dopamine plays an important role in the modulation of synapses, allowing the synapses to adapt to rewarded behavior that may be delayed by some period of time. This system allows the brain to correlate predictions in action with future reward. And neurons are situated within geometrical configurations with 3 dimensional space that must play some role in computation.

Of course computer scientists working with Artificial Neural Networks and in the field of Awe have not ignored the fact that intelligent behavior requires the actor to both be situated in space and time. Q Learning and TD(λ) have been developed to associate time and space dependent rewards on actions that occurred prior to the reward. These have shown great success, although the algorithms are limited and simply are not computationally feasible outside of toy problems and grid worlds. In an effort to develop algorithms that can compete or emulate general intelligence, these are likely dead ends for research and development. However they are well suited to a number of optimization and other interesting problems in academia and industry. Efforts have been made to extend the Artificial neural network to include spatio-temporal computation. Recurrent networks have also been implemented to allow for time-series prediction within ANNs. But these extensions have not received enough focus within the research community and although they improve the capabilities of the ANN they have not made significant progress in eliminating the scale up problem.

Given that Spiking Neural Networks are a somewhat accurate representation of the brain and relatively unexplored in terms of Machine Learning, we chose to implement a Spiking Neural Network utilizing the Izhlkevich model. we will implement Spike Timing Dependent Plasticity as well as Extracellular dopamine in an attempt to solve to train the network to respond to inputs from a Machine Learning dataset. Although Machine Learning algorithms have been devised that are optimal for this data, it is important to identify the advantages and disadvantages of SNN by comparing it's performance to that of existing algorithms. Unfortunately the implementation described next is Serial, and therefore highly inefficient. It would be better to implement a fully parallel approach, however given the time available we was unable to complete a parallel implementation. Further the implementation of dopamine requires timescales of training that are simply infeasible due to this computational complexity. For this reason training of the ML data does not produce accurate results. Further exploration of the issues, especially that of input and output mechanisms, is necessary in order to reproduce the results received from optimal ML algorithms. However there has not been significant exploration of traditionally ML data being evaluated within the SNN literature, and these are relatively uncharted waters

The SNN we developed is largely based on the implementation described by Izhikevich in his attempt to solve the distal reward problem utilizing an abstract representation of dopamine [10]. Some of the problems with learning mechanisms such as Hebbian learning is that it is not capable of adjusting to time-delayed changes in synaptic plasticity. Hebbian learning is only useful when the correlation between Input and Output occurs simultaneously. Rewards often occur many seconds after the initial stimulatory input and motor output, and since synapses operate on a time scale of microseconds Hebbian learning is not sufficient to train Spiking Neural Networks. To create a computational solution we look towards Extracellular dopamine.

2 Biological Perspective on Dopamine

Dopamine is one of the monoaminergic neurotransmitters active in the central nervous system. Dopaminergic neuronal cell bodies, relevant to cognitive activities, reside on the substantia nigra and the ventral tegmental area of the ventroanterior midbrain. The connecting axon terminals typically extend towards sections of the striatum, ventral striatum, and the neo cortex [6]. Dopaminergic neurons have been suggested to play a significant role in the control of motor behavior and regulate motivational and mood behaviors trained via reward/punishment modules. Respectively, these are brought upon by the nigrostriatal dopaminergic pathway and dopaminergic mesolimbic and mesocortical pathway. The nigrostriatal pathway originates in the zona compacta of the substantia nigra projecting towards the caudate putamen and the mesocortical pathway from the ventral tegmental area projecting primarily to the nucleus accumbens specifying to the prefrontal [13]. Levels of dopamine increase in the nucleus accumbens of the ventral striatum, when rewarding stimulwe are encountered. Lesions to the nucleus accumbens, in rats, result in a negative impact on the stimulus reward association. [11] Variation in dopamine levels lead to dis-

ease: an excess in concentration of dopamine correlates in schizophrenia, while a deficiency in Parkinsons disease [7].

Dopamine is released via bursting innervations of striatal varicosities and is received by dopamine receptors, the most relevant: the excitatory D1 receptor and the inhibitory D2 receptor. Occurrence of saturation of dopamine at receptor sites allows for further synapse enhancement. Activation of either receptor can result in the termination or progression of the pathway being as the function of the dopamine varies itself from target to target. Though dopamine release is such a great magnitude to being global and target multiple synapses the transmissions are typically regional [6]. The diffusion of dopamine to be processed occurs within 75 ms after its initial release to a radius of 7-12 micrometers. Levels are brought back to baseline numbers through high-affinity extrasynaptic dopamine reuptake transporter in quick motion making the activation and effect timeframe relatively short and keeping general extracellular dopamine levels low [7].

3 Dopamine In Learning

Dopaminergic neurons have been seen to have a significant influence on learning. The main influence stems from their manipulations of values of incentive motivation by establishing conditioned incentive stimuli. Empirical evidence shows that midbrain neurons have been seen to act in parallel with actual learning behaviors. In response to rewarding stimulwe dopamine activation levels have been shown to be of a higher order. In response to aversive stimuli, dopamine activation was seen to either be inhibited or of a lower order with a delay in response. Salient, distal stimuli, actions in association with either response, such as physical movement, typically acted in relation to the response [6]. Stimuli, however, can be induced to be ignored by the dopaminergic neurons through the use of dopamine antagonistic substances blocking dopamine transmission and reception [13].

In the learning process, the role of prediction is pertinent. Prediction is based off of the ideal of whether or not the received reward will act to be better than expected, the same as expected, or lesser/worse than expected. During learning, activation responses are only elicited by unpredicted rewards or sensory events relating to the reward, while predicted rewards elicited either minimally no response. To further this, previous work shows that if a stimulwe repeatedly occurring in a predictable mannerconstantly at exactly the precise time intervalminimal to no activation of the neurons occur. A delay in reward reception (on the millisecond scale) causes a depression in neuronal activity. Only when the In this manner, timing acts as an important variable in dopamine modulation. Delay and absence of a reward constitutes as lesser than expected, and results in a depression in neuronal activation, which motivates an extinction of behavior [6]. In support, some ventral tegmental area neurons as well as prefrontal deep layer neurons in rats were found to increase their activity in correlation to the behavioral phases in rewarding operant task [14].

During training, it has been shown that there exists activity in primate prefrontal cortex neurons and in rat medial frontal cortex neurons. Dopamine transmission occurring to these two distinct areas have been seen to play a significant role in long-term depression and long-term potentiation of glutamatergic synapses, and ultimately memory. Observed release of dopamine coincides with the activation of the areas of the brain significant in memory building and motor control. In rats, cases of LTD have occurred with dopamine activations of MAP-K, which has been suggested to be linked in modifying dendritic spine shape and formatting it towards better functioning [14].

4 Implementation

The neuron model used in this project is based off the Izlkhevich model in Project 1 [1]. It is an efficient spiking neuron that has many biologically plausible properties, and therefore is a good basis for the extension of a dopaminergic property. we implemented Spike Timing

Dependent plasticity given by the following python code. *syn* is the synaptic weight of the synapse being updated, and $tao = PostSynaptic_t - PreSynaptic_t$. n_plus and n_minus allow us to modulate the areas governing the increase of LTD and LTP. It took quite a bit of tweaking to achieve satisfactory values for these variables.

```
def STDP(tao, syn):
        n_plus = .3
        n_minus = 3.0
        if tao > 0:
                A = (4 - syn)*n_plus
                return A*exp(-tao/10.0)
        elif tao < 0:
                A = (syn) * n_minus
                return -A*exp(tao/10.0)
        else: return 0.0
```

This successfully caps the synaptic strength at end points of 0 and 4 mV. It is equivalent to the experimental data collected by Bwe and Poo (1998) as shown. There is a slight discrepency because in the case of the experimental data Bwe and Poo consider the presynaptic time subtracted by the postsynaptic time. The STDP update we use is equivalent but inverse. Synaptic update is not guided entirely by the STDP rule however. Instead the synaptic update rule is given in Equation 2. The variable c is gated by the dopamine reward d. c represents a synaptic tag such as the oxidation of PKC or PKA, or some other 2nd messenger process which modulate learning and memory [10].

$$c' = -c/tau_c + STDP(\tau)\delta(t_{post} - t_{pre}) \tag{1}$$

Here the STDP value is gated by the Delta dirac function. This function steps the STDP when the postsynaptic and presynaptic times are within 10 ms. By using this unit step, we can allow c to decay over time. This decay serves a similar purpose as an eligibility trace used in Temporal Difference learning.

$$s' = cd \tag{2}$$

$$d' = d = \tau_d + DA(t == t_{reward}) \tag{3}$$

The dopamine update equation DA(t) provide a 0.5 injection of dopamine when the reward time has been reached. Dopamine is also decayed by a similar time constant to that of c. However we set $\tau_d = 0.2s$ so that the decay of dopamine operates on a faster timescale than that of c.

We implemented a SNN consisting of 500 nuerons. The network consists of 80% excitatory neurons of the regular spiking type and 20% inhibitory neurons of the fast spiking type (Connors and Gutnick 1990). This gives our network the characteristics of layer 2/3 part of the cortical minicolumn. The network is initialized with a 10% probability of connection and has on average 100,000 synapses. Excitatory-to-excitatory and excitatory-to-inhibitory synapses are updated with the Synaptic update rule. Inhibitory synapses are not updated. The following graph show c over time in a simulation, following a single synapse.

As you can see c is increased with a step input, then decays over time. The synaptic strength is increased when the dopamine is released. By this time c has decayed, but is still robust enough to allow a change in the synaptic weight. The noise in the system, approximately 1 Hz of Poisson spike trains, ensure that changes in c caused by the random nature of the system will not affect it's mean performance, due to the central limit theorem. In this way the connections that allowed for the network to link Stimulation and Motor Output can be made.

Figure 1: STDP From Pseudo Code with $syn = .5$

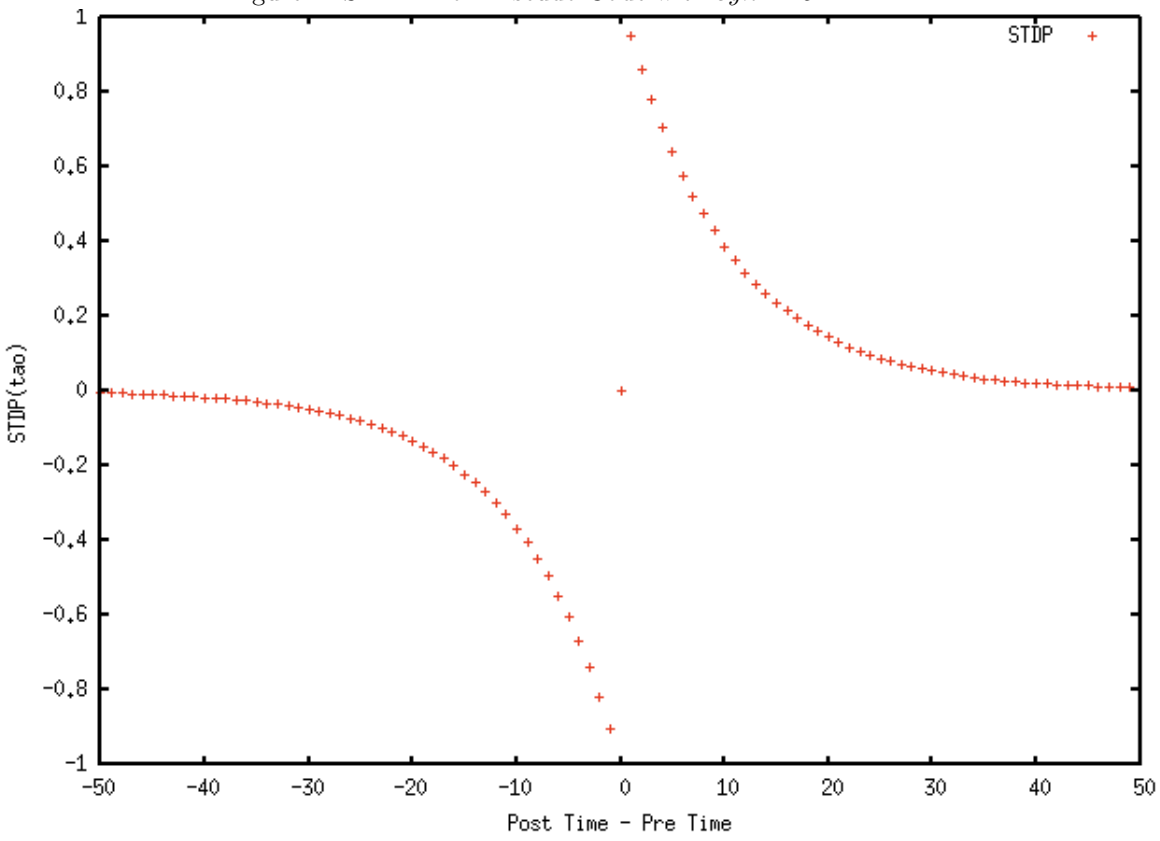
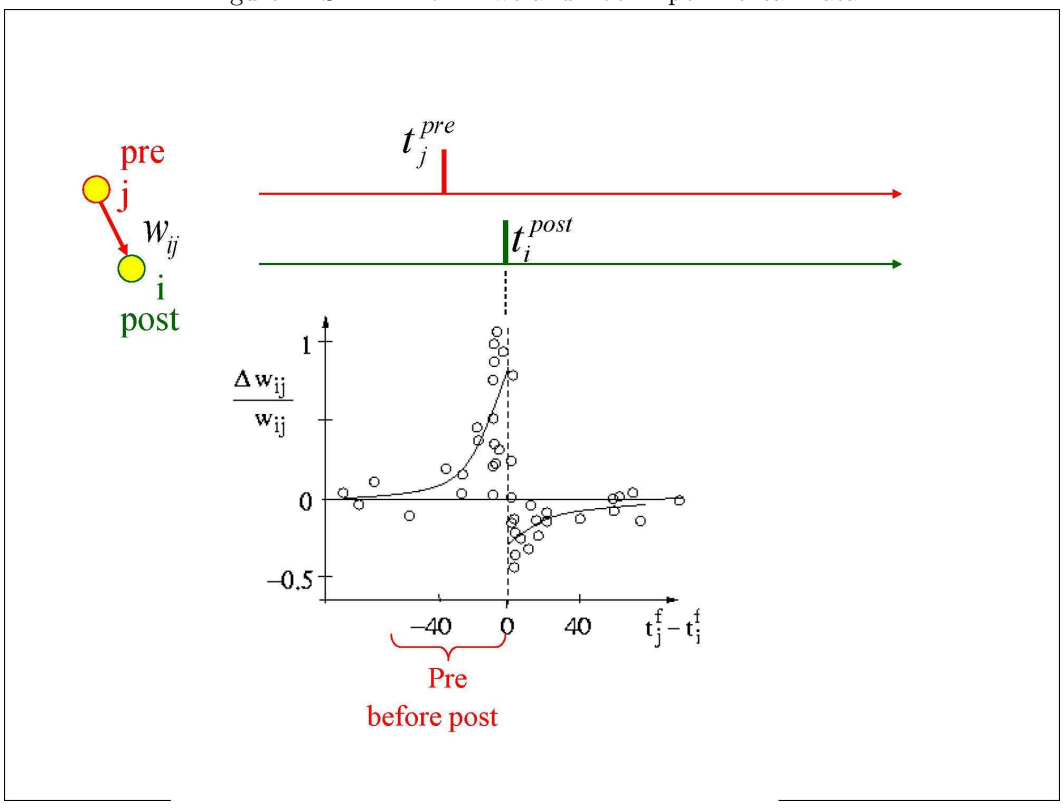


Figure 2: STDP From Bwe and Poo Experimental Data



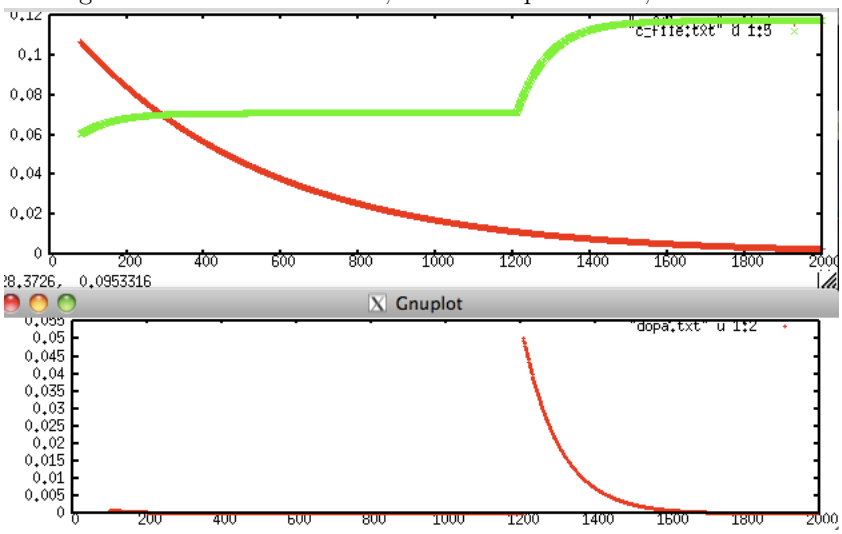
5 Testing

In order to test the effectiveness of the update equation we created three groups of 50 neurons, S, 0, and 1. S is the stimulus group, 0 and 1 are the response groups. This groups could be nonantagonist motor neuron groups, competing for the use of a muscle. we wanted to test if dopamine that is delivered with a delay will improve the probability of the output correlating to the reward signal. In order to sufficiently explore this problem we had to make some tweaks to the time constants, dividing both τ_c and τ_d in half. A 1ms Pulse stimulus to the S group was employed at approximately every 500 ms. After the stimulus reward is delivered at a random time up to 500 ms after the initial stimulus. There are no stimulwe active before the reward is distributed, and only the noise from the Poisson spike trains is preset. To determine if the dopamine reward should be activated time t_{reward} we compare the number of neuron firings in group 0 and 1, over a 20ms period following the initial stimulus. The group with more neurons is considered the dominant. In my experiments, if during the observation period there were more reported firings of group 0, the reward is distributed. we ran a series of trials in this nature until the time limit is reached. However a 20s simulation takes a considerable amount of computational time.

This configuration is a seemingly good approach to the problems encountered when we attempted to use the parameters available in the paper this effort is derived from [10]. However the timescales necessary to reproduce the trials as cited is impossible with my computing resources and the programming platform we have chosen. In order to truly take advantage of the capabilities of this model it is necessary to have a highly parallel hardware and software implementation. With the serial approach it is very difficult to test the learning capabilities of the model because the synaptic updates create a massive computational expense and they lie at the heart of the learning mechanism. The implementation in the parameters from literature require a trial once every 10s, and run for a total 0f 800 trials. With my current constraints running this length of simulation would take a considerable amount of computational time. For that reason we pared down the parameters, but this will be a detriment to learning and the results we achieved may not be indicative of the algorithms overall usefulness in learning.

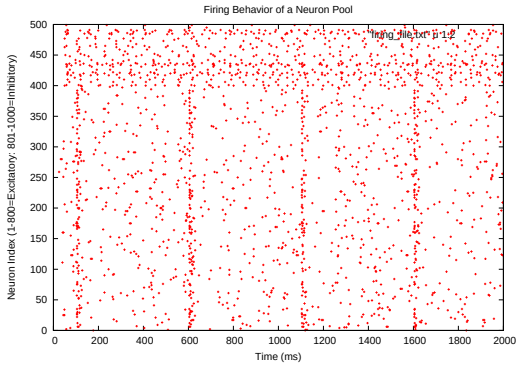
With that in mind we performed a series of tests in an attempt to better understand the learning mechanism and the graded uptake/release of c and d. These experiments where performed with the stimulus and response groups. we gathered data concerning the c value of a single neuron, the global dopamine level d, the distribution of the synapse strength s, and the probability of choosing a neuron group as output.

Figure 3: s is the Green line, c is the Top Red line, and d is the Bottom Red Line



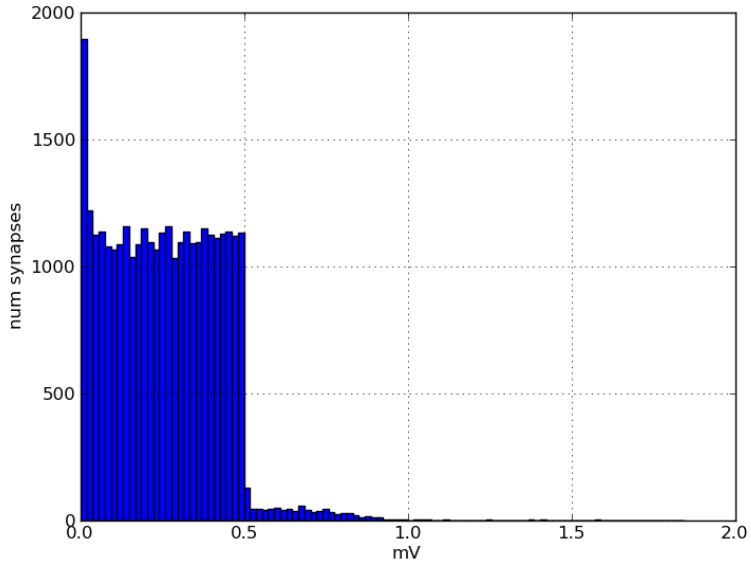
This graph shows the relationship between s, c, and d. As you can see correlated timings increases the activation of the synaptic tag, which then decays slowly over a period of time. Before that decay is complete, dopamine is injected as a reward, gating c and allowing the synapse weight s to increase. This is the basis from which this method of learning is made useful.

Figure 4: Average Trial



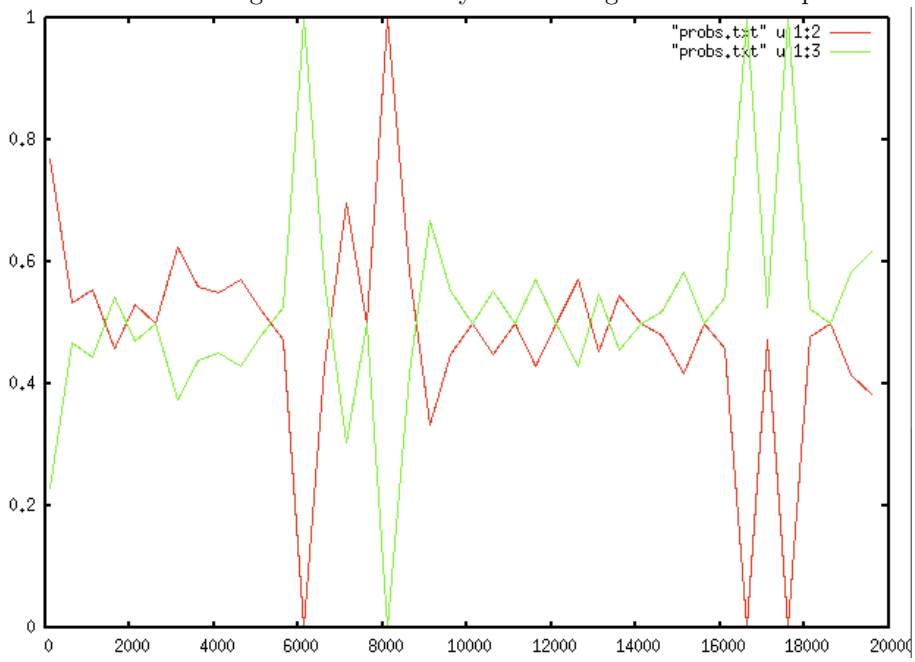
This graph shows behavior of the entire neuron firing pool. You can see regular intervals of stimulation by the somewhat thicker lines, around x,y,z. The network is able to distinguish these inputs from the noise, and 0 group of neurons are fired the pool will reinforce connections between S and 0.

Figure 5: Synapse Distribution



This graph shows the distribution of the synapse weights after 20s of simulation. Although the distribution is still heavily biased from the initial uniform random initialization, you can see it is beginning to skew towards the power law distribution. This is an important experimentally verified biological characteristic of a synapse pool.

Figure 6: Probability of Choosing Neuronal Groups



Finally, we show the probability of choosing group 1 versus 0, respectively green and red in the graph. As you can see the pool of neurons is unable to learn the necessary firing sequence in order to ensure it receives the reward. This is due to the small timescale of the trials as compared to the time constants. Increasing the learning rate also increases the noise in the system, and does not guarantee convergence to the solution. Unfortunately we cannot use the configurations as specified, because the computational time is too long under my current constraints. However in the future I wish to implement a parallel approach that can run significantly closer to real time.

6 Discusion

we present the implementation and preliminary testing of a dopamine enhanced spiking neural network. While spiking neural networks are interesting and an important domain in the field of AI, they are not as mature as existing ANNs or other methods for Machine Learning. we attempted to use the SPECT ML dataset, however since the network was unable to learn simple motor reflexes as related to reward we doubted it would be able to distinguish 22 different boolean properties over two classes, especially in the timescale that we was running the simulations on. However we believe that SNN are promising from this experience. There are 10^{164} different combinations of 2 groups of 50 neurons in a pool of 1000. This is a combinatorial explosion, allowing for many different types of configurations and inputs. However under practical limitations it is difficult to implement SNN, because they offer little advantage over ANN for small problems. It would be interesting to scale the SNN up to more neurons in a massively parallel implementation to determine the performance as the

number of dimensions scales up. One thing that we can simple observe from a conscious analysis of SNN versus ANN is the ability to scale SNN up with as many cores as you need. Since each neuron in the SNN acts and functions independently, the computing resources necessary probably scales fairly linearly with the problem size. However in terms of ANN, each layer must be computed, before the computation of the output of the next layer. As the number of neurons grows, the calculations necessary increases exponentially. From this fairly basic analysis, we believe that SNN are likely more capable of ANN, although they are better suited to high dimensional and hard problems.

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