

Development of Receptive Field Structure of Simple Cell using Spike Timing Dependent Plasticity (STDP)

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ABSTRACT

Simple cells found in primary visual cortex are orientation selective. It has been experimentally found that they acquire this property with time i.e. learning of orientation selectivity takes place. Many computational models have been proposed for the development of orientation selectivity. Most of the models proposed so far are either abstract in nature or are very simplified version of actual learning mechanism. In this work we propose a model for development of orientation selectivity based on spike timing dependent plasticity (STDP), which till now is considered to be the actual learning mechanism adopted by neural circuits. We could obtain elongated segregated receptive field structure thus giving simple cells the property of orientation selectivity. We also observe that input activity plays a major role in the development of orientation selectivity, too much or too less a correlation between the inputs activities do not result in the formation of segregated ON and OFF regions in the RF structure [1]. There is also a need of normalization for the development of orientation selectivity.

Keywords

Simple cells, visual cortex, Hebbian model, STDP, BCM learning.

1. INTRODUCTION

The brain is complex network of neurons with interconnections. The visual cortex of the brain is that part of the cerebral cortex responsible for processing visual information. The average number of neurons in the adult human primary visual cortex, in each hemisphere, has been estimated at around 140 million [2]. The scientists are trying to simulate and understanding of the brain functions with the computation of different theories i.e. Hebbian[3], STDP[4] and BCM[5] learning rules.

The image captured by each eye is transmitted to the brain by optic nerves. These nerves terminate on the cells of the Lateral Geniculate Nucleus (LGN), the first relay in brain visual pathway. The cells of LGN then project to the primary visual cortex. It is in the primary visual cortex that brain begins to construct the image.

The scientists has been invented some computational learning method to develop the receptive fields to simulate primary visual cortex. In this paper, we take STDP method to develop the receptive field in primary visual cortex[6]. We also extend the effect of normalization for STDP to the analysis of ON-center and OFF-center LGN cells, and investigate whether normalization effects for the segregation of the ON/OFF subfields[1]. The effect of correlation factor is also analyzed for the input activity. The activities at time t of the ON type and OFF type LGN cells at location i with appropriate correlations were generated as given in Goodhill 1993[7].

1.1 Synaptic Plasticity

Synaptic plasticity [5,8] is the ability of the connection, or synapse between two neurons to change in strength in response to either use or disuse of transmission over synaptic pathways. Plastic change also results from the alteration of the number of receptors located on a synapse. There are several underlying mechanisms that cooperate to achieve synaptic plasticity, including changes in the quantity of neurotransmitters released into a synapse and changes in how effectively cells respond to those neurotransmitters. Synaptic plasticity[9] in both excitatory and inhibitory synapses has been found to be dependent upon calcium. Since memories are postulated to be represented by vastly interconnected networks of synapses in the brain, synaptic plasticity is one of the important neurochemical foundations of learning and memory.

1.2 Synaptic Strength

The strength of a synapse is defined by the change in transmembrane potential resulting from activation of the postsynaptic neurotransmitter receptors. This change in voltage is known as a postsynaptic potential, and is a direct result of ionic currents flowing through the postsynaptic ion channels. Changes in synaptic strength[10] can be short-term and without permanent structural changes in the neurons themselves, lasting seconds to minutes or long-term (long-term potentiating, or LTP), in which repeated or continuous synaptic activation can result in second messenger molecules initiating protein synthesis, resulting in alteration of the structure of the synapse itself. Learning and memory are believed to result from long-term changes in synaptic strength, via a mechanism known as synaptic plasticity.

1.3 SPIKE TIME DEPENDENT PLASTICITY (STDP)

Spike Timing Dependent Plasticity (STDP)[4,11] is a biological process that adjusts the strength of connections between neurons in the brain. The process adjusts the connection strengths based on the relative timing of a particular neuron's output and input action potentials (or spikes). The STDP process is a tentative candidate for a hypothesis that fully explains the development of an individual's brain.

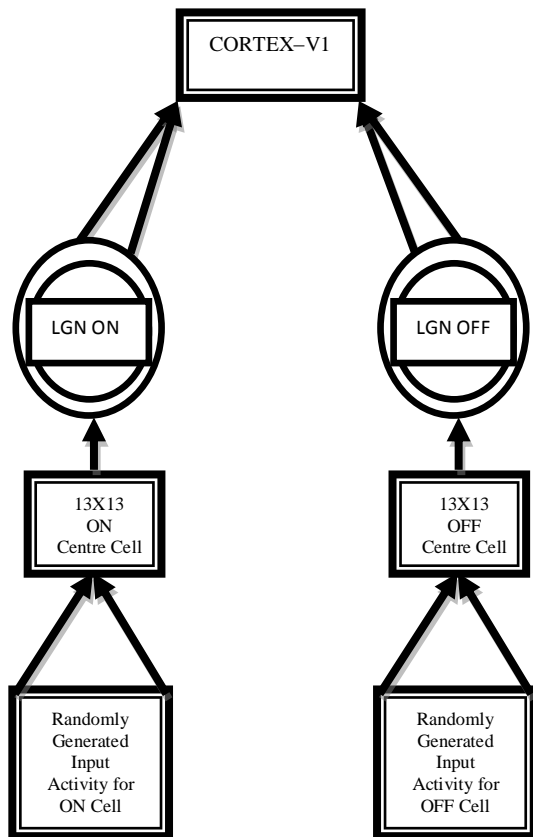


Figure 1: The pathway of ON/OFF channel, different randomly generated input activity to the 13X13 ON/OFF centre cell

Under the STDP process, if an input spike to a neuron tends, on average, to occur immediately before that neuron's output spike, then that particular input is made somewhat stronger. If an input spike tends, on average, to occur immediately after an output spike, then that particular input is made somewhat weaker hence it is: "Spike-Timing-Dependent Plasticity". Thus, inputs that might be causing the spiking of the neuron are made even more likely to contribute in the future, while inputs that are not causing the neuron to spike are made less likely to contribute in the future. The process continues until subsets of the initial set of connections remain, while the influence of all others is reduced to 0. Since a neuron produces an output spike when many of its inputs occur within a brief period the subset of inputs that remain are those that tended to be correlated in time. In addition, since the inputs that occur before the output are strengthened, the inputs that provide the earliest indication of correlation will eventually become the final input to the neuron. Although the process occurs throughout the brain, its implementation is achieved at the level of individual neurons, and there is no need for any central oversight.

1.4 Weight Normalization

Weight normalization refers to a procedure whereby some measure of the total synaptic weight onto the recipient neuron is used to limit the growth of the synaptic weights. There are two ways to normalize the synaptic weights: (i) subtractive normalization and (ii) multiplicative normalization[12]. The implementation of the weight normalization is explained in method and material.

Hebb's theoretical considerations[3] and neurocomputational models proposed the idea that memories could be encoded in neural networks by changes in synaptic strength. At present, there are robust connectionist models that support this idea[10]. Exploring neurobiological data corresponding to this hypothesis started when Bliss and Lomo (1973) discovered long-term potentiating (long-lasting form of synaptic plasticity) in hippocampus[13,14].

2. MATERIAL AND METHODS

We take simple neuron in the primary visual cortex receiving input from the 13x13 ON/OFF LGN[1]. Both ON/OFF activity are generated by the random numbers. Various theoretical ideas have been proposed to account for the manner in which this subfield segregation develops. In 1962, Hubel and Wiesel had suggested that simple cells in primary visual cortex acquire the property of orientation selectivity due to the structure of their receptive field (RF)[6]. The RF structure of these cells is composed of segregated elongated ON/OFF subfields. They suggested that the ON subfield of a RF is formed due to the convergence of inputs from several ON center relay cells all having their RF centers lying along the axis of the subfield. Similarly, an OFF subfield is formed due to the convergence of inputs from several OFF center cells arranged in a similar fashion at an adjacent location [15,16].

In this paper we have chosen to model the spatial properties of the retinal preprocessing by convolving the input activities with Difference of Gaussian (DOG) filter. The nature uses the Difference of Gaussian as the basis for the architecture of the retina's visual receptive field. The retina actually implements DOG band pass filters at several spatial frequencies. First, we stored the input activity in form of an array and this activity is used for STDP method.

3. ARCHITECTURE OF MODEL

In this subsection, the basic architecture of the model is described. In our simulation of the receptive field development of simple cells in visual cortex, the neuron receives inputs from two channels, one corresponds to ON-center lateral geniculate cells, and the other to OFF-center cells. The two pathways that are ON and OFF cells do not interact at the level of LGN but converge in the cortex. .

For the development of thalamocortical connections we assume a two layer structure as shown in the figure 1. The output layer composed of a simple cortical cell, which represent cell of layer IV C of cat primary visual cortex [16, 17]. The input layer, which represents the corresponding LGN layer, is subdivided into two dimensional sheets. One sheet labeled "ON" consisting of ON-type LGN cells and other sheet labeled "OFF" consisting of OFF type-LGN cells.

Each LGN cell is constructed to always arborize over a fixed, topographically appropriate circular patch of cortical cell with diameter of 13 grid unit. Also, in the beginning the cortical cell is connected to both types of all the LGN cells lying in topographically appropriate circular region in the LGN layer. For computational convenience all the synaptic contacts between one LGN cell (ON or OFF) and cortical cell are lumped together and are represented by a single quantity i.e. synaptic strength. The value of the synaptic strength at time t between LGN cell i in sheet labeled "ON" are cortical cell is described by its peak synaptic conductance $g_i^{ON}(t)$. Similarly the synaptic strength between LGN cell i in sheet labeled "OFF" and cortical cell is given

by $g_i^{OFF}(t)$. The synaptic strength of the feed forward connection between LGN cell and cortical cell is considered to be modifiable. An integrate-and-fire neuron model describes each of the cortical cells. The membrane potential of the integrate-and-fire model neuron of the cortical cell then changes according to

$$\tau_m \frac{dV}{dt} = V_{rest} - V(t) + G_{ex}(t)(E_{ex} - V(t)) + G_{in}(E_{in} - V)$$

With $\tau_m = 20ms$, $V_{rest} = -70mV$ and

$E_{in} = -70mV$. E_{ex} is the reverse potential for the excitatory synapses, E_{in} is reverse potential for inhibitory synapses and $V(t)$ is the membrane potential at time step t of the cortical cell in the cortical sheet. When the membrane potential of the neuron reaches the threshold value of $-54mV$, the neuron fires an action potential and subsequently membrane potential is reset to $-60mV$.

Whenever a particular ON (and OFF) type of LGN cell fires, the corresponding peak synaptic conductance contributes towards the value of excitatory synaptic conductance (G_{ex}):

$$G_{ex}(t+1) = G_{ex}(t) + \sum_{i=1}^M g_i^{ON}(t).A_i^{ON}(t) + \sum_{i=1}^M g_i^{OFF}(t).A_i^{OFF}(t)$$

Here M is the total number of ON (and OFF) type LGN cells connected to a particular cortical cell. Looking onto the above equation we can see that only activate presynaptic cell are contributing toward the increase of the value of excitatory synaptic conductance. During the time dt when there is no presynaptic activity this synaptic conductance decays exponentially.

$$\tau_{ex} \frac{dG_{ex}}{dt} = -G_{ex} \text{ Here } \tau_{ex} = 5m$$

$A_i^{ON}(t)$ and $A_i^{OFF}(t)$ are defines as the defined as the activity at time t of the ON type and OFF type LGN cells at location i respectively.

The model is based on a Spike-Timing Dependent Plasticity rule in which a function $F(\Delta t)$ determines the amount of synaptic modification arising from a single pair of pre- and post synaptic spikes separated by a time Δt . The function shown in figure 2:

$$F(\Delta t) = \begin{cases} A_+ \exp(\Delta t / \tau_+) & \text{if } \Delta t < 0 \\ -A_- \exp(\Delta t / \tau_-) & \text{if } \Delta t > 0 \end{cases}$$

All value used for model are listed in the table 1.

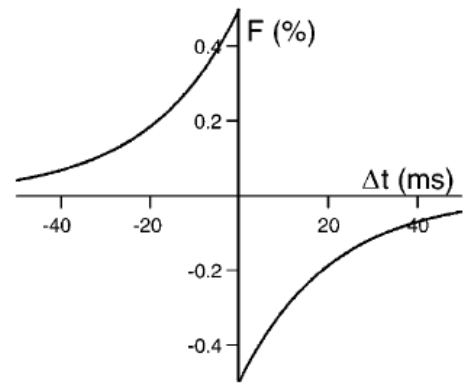


Figure 2: The STDP modification function. The change of the peak conductance at a synapse due to a single pre- and postsynaptic action potential pair is $F(\Delta t)$ times the maximum value \bar{g}_{max} with Δt the time of the presynaptic spike minus the time of the postsynaptic spike. In this figure, F is expressed as a percentage.

Table 1: Standard Parameters for STDP

S.No	PARAMETER	SYMBOL	OPT. VALUE
1	Number of iterations	T	2700000
2	Number of input synapses		
	Excitatory synapses(for ON and OFF center)	nSe	169*2
	Inhibitory synapses	nSi	0
3	Spread	rho	1.54
4	Conductance -Excitatory	g_{ampa}	0.015
5	Time constants		
	For total excitatory synaptic	t_{ampa}	5ms
	For membrane potential	t_m	20ms
	For parameter Pa	t_{ap}	20ms
	For parameter M	t_{am}	20ms
6	Correlation Factor	h	0.22
7	Incremental Factor on arrival of action potential at a synapse (For Pa)	A+ (Ap)	0.005
8	Detrimental factor (for M), every time post synaptic neuron fires an action	A- (Am)	1.05*Ap
9	Potentials		
	Threshold Potential	Xth	-54mV
	Inhibitory synaptic potential	$E_{cl}(E_{in})$	-70mV
	Resting Potential	$E_1(V_{rest})$	-70mV
	Excitatory synaptic potential	E_{ampa}	0mV
	Membrane reset potential	Xrest	-60mV

4. WEIGHT NORMALIZATION

The implementation of the weight normalization is explained below:

4.1 Subtractive normalization

At the starting of iteration we calculate the sum of all synaptic weights for ON and OFF LGNs and after updating of synaptic weight we also calculate the sum of all synaptic weights for ON and OFF LGNs. Subtractive normalization factor (sn) is calculated as

$$sn = \frac{\sum_{j=1}^n w_j - \sum_{j=1}^n w_{j-1}}{n}$$

Where i is ith iteration, w_j is the weight of jth connection and n is total number of ON and OFF connection synaptic weight. All ON and OFF synaptic weights are normalized (by subtracting sn from all synaptic weights) as:

$$w_j = w_j - sn$$

Subtractive normalization is combined with hard bounds $0 \leq w_j \leq w_{max}$ in order to avoid runaway of individual weights.

4.2 Multiplicative Normalization

For the implementation of multiplicative normalization, we calculate a multiplication normalization factor as

$$mn = \frac{\sum_{j=1}^n w_{j-1}}{\sum_{j=1}^n w_j}$$

Now all the weights are re-scaled as:

$$w_j = w_j * mn$$

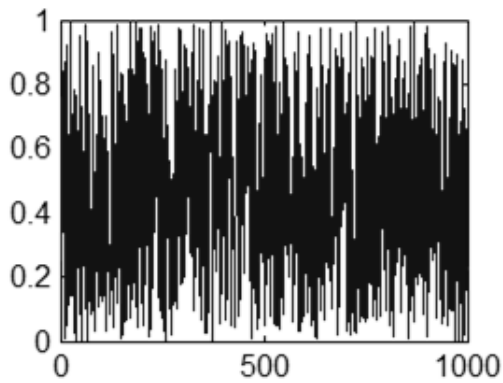


Figure 3: Random numbers for 270000 iterations of input activity and range from 0 to 1. Only 1000 random numbers are shown in the figure.

5. RESULTS AND ANALYSIS

The random numbers are stored in a separate data file. This file is using to generate the input activity for the ON and OFF centre cell. All random numbers are range from 0 to 1 as shown in figure 3.

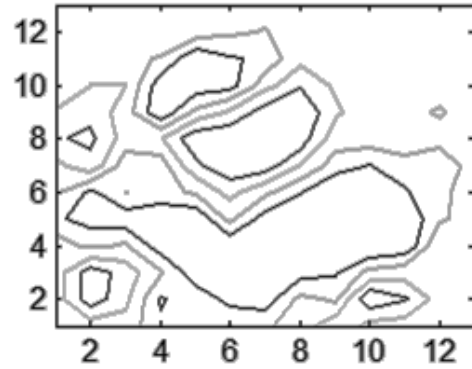


Figure 4(a): Segregation of Receptive Field using STDP method without normalization, the value of correlation



Figure 4(b): Segregation of Receptive Field using STDP method with normalization, the value of correlation factor (h=0.22)

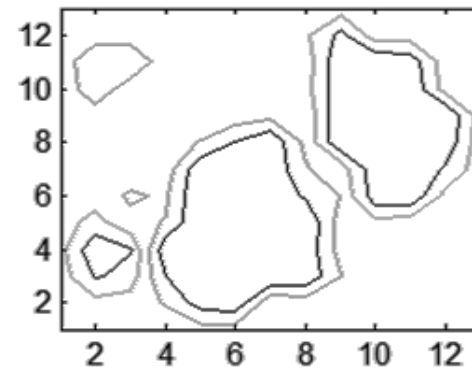


Figure 5: Receptive Field is Segregated for the different input activity, using STDP method with normalization, the value of correlation factor (h=0.22)

Figure 4(a) shows the segregation of RF for STDP method without normalization. Though we obtained the segregation of RF but it is clear that they are not in good shape as per the previous research papers.

When we apply normalization technique (Subtractive and Multiplicative) then we obtained the segregation of RF with good shape as shown in figure 4(b). We also developed the receptive field for different input activity.

In the figure 4 and figure 5, the receptive field developed for different input activities. We also vary correlations factor (h) from 0.16 to 0.5. For correlations factor ($h=0.25$), segregated receptive field is developed in more than two part as shown in figure 6 (taken same input activity as in figure 4). For correlations factor ($h=0.27$), distributed segregated receptive field is developed as shown in figure 7 (taken same input activity as in figure 4). This is analysis that the receptive field developed with the normalization in appropriate shape.

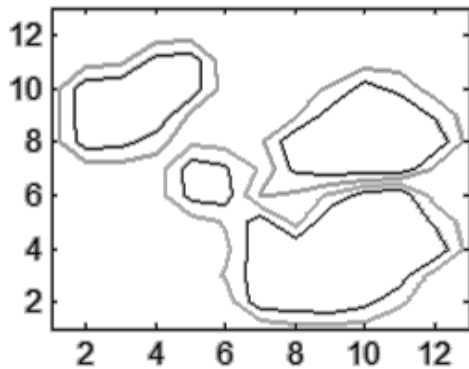


Figure 6: Segregation of Receptive Field using STDP method with normalization, the value of correlation factor ($h=0.25$), taken same input activity as in the figure 4.

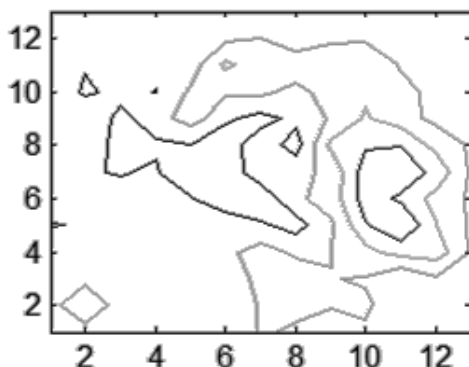


Figure 7: Segregation of Receptive Field using STDP method with normalization, the value of correlation factor ($h=0.27$), taken same input activity as in the figure 4.

6. DISCUSSION

However, there is no evidence present for normalization of synaptic weight in the biological system, even though we used normalization technique in STDP model. STDP model is more realistic as compare to BCM and other models because STDP model is more near to biological system where as BCM model is basically a mathematical model.

The correlation factor (h) varies from 0.1 to 0.5 and it is analyzed that there is no segregation of RF, if the value of h is greater than 0.27 and less than 0.1. The same size of two receptive field is developed with $h=0.22$.

The model executed for 10 different input activities and this is found that after the segregation of receptive field, more than 30 connections are available for ON and OFF centers.

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