The Role of Constraints in the Development of Receptive Field Structure of Simple Cells using BCM Learning

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ABSTRACT
Experimentally studies have shown that visual cortical neurons apply BCM (Bienenstock Cooper and Munro) learning rule for modifications in synaptic strength. BCM rule uses adaptive threshold and in this both long term potentiation (LTP) and long term depression (LTD) is automatically taken care of. This overcomes the major disadvantage of Hebbian learning in which there is a mechanism only for LTP and no mechanism for LTD. Based on the above-mentioned experimental findings we apply BCM learning rule for the development of orientation selectivity by simple cells. We find that BCM learning rule is sufficient for segregation of ON and OFF regions in developed receptive field (RF) structure of simple cells. Starting from unsegregated ON - OFF regions we obtain elongated segregated ON and OFF regions in the RF structure very similar to actual RF structure of simple cells. The orientation selectivity thus developed is also very similar to what is found in actual simple cells.

Keywords
Simple cells, orientation maps, LGN spontaneous activity, visual cortex, BCM Neuron, BCM learning.

1. INTRODUCTION
A cubic millimeter of primary visual cortex contains about 100,000 neurons that are heavily interconnected by intrinsic and extrinsic afferents. The effort of many neuroanatomists over the past has revealed the general outline of these connections; however, their function remains a mystery. Recently, combined physiological and anatomical approaches are beginning to reveal the role of these connections in the generation of cortical receptive fields (RF). A common theme emerges from all these studies: cortical connections are remarkably specific and this specificity is determined in great extent by the type of connection and the neuronal response properties [1].

In this paper, we extend the BCM theory [2] to the analysis of ON-center and OFF-center retinal and LGN cells, and investigate whether BCM synaptic modification can account for the segregation of the ON/OFF subfields. There is evidence that the adjacent excitatory and inhibitory subfields of orientation selective cells in visual cortex are projections from ON-center and OFF-center retinal and LGN cells respectively and that the proper development of the cortical receptive fields requires activity. We explore the segregation of the ON- and OFF-center LGN afferents, by using a model of a single cortical cell with inputs from ON and OFF LGN cells. Our results indicate that there is a relation between the organization of simple receptive fields and the input activity pattern.

2. SIMPLE AND COMPLEX CELL
Neurons in the primary visual cortex have been traditionally classified as “simple” and “complex” based on their receptive field properties (Hubel & Wiesel, 1962, 1968) [9] classified a cell as “simple” based on four different criteria.

1. The receptive field was spatially subdivided into distinct sub regions that responded to either light on (on-subregion) or light off (off-subregion).
2. There was spatial summation within each subregion.
3. There was spatial antagonism between on- and off-subregions.
4. The visual responses to stationary or moving spots could be predicted from the spatial organization of the subregions [3].

Cells that did not fulfill these four criteria were classified as complex cells. The classification approach proposed by Hubel and Wiesel correlates well with laminar position and synaptic connectivity (Ferster & Lindstrom, 1983; Gilbert, 1977; Hirsch et al., 2002);

3. RECEPTIVE FIELD [1]
That part of the retina whose photoreceptors (rods and cones) pertain to a single optic nerve fibre. The response of a neuron to stimulation of its receptive field depends on the type of neuron and the part of the field that is illuminated; an on-centre neuron is stimulated by light falling at the centre of its receptive field and inhibited by light falling at the periphery; an off-centre neuron reacts in exactly the opposite fashion; that is, it is inhibited by light falling at the centre of its receptive field. In either case, the net response depends on a complex switching action in the retina. When an entire receptive field is equally illuminated, the response of receptors at the centre of the field predominates.

4. SYNAPTIC PLASTICITY
Synaptic plasticity [1] is a process in which synapses change their efficacy as a consequence of their previous activity. Synaptic efficacy (synaptic weight, synaptic strength) can be defined as an amplitude of the transmembrane voltage on the membrane of the postsynaptic neurons soma which arises as a consequence of defined unit stimulation of the presynaptic terminal of the synapse (Benuskova, 1988). Synaptic efficacy is a measure of the synapses contribution to the summary somatic postsynaptic potential which determines the time and frequency of the spike train generated after exceeding the excitation threshold of the neuron.

Thus, it is directly proportional to the amplitude and duration of the postsynaptic potential (PSP) at the synapse. Synaptic weight (excitatory PSP after unit stimulation of the synapse) depends on two groups of factors (Benuskova, 1988; Kral, 1997):

A. Presynaptic factors: released amount of the transmitter
B. Postsynaptic factors: number of the receptors types and properties of the receptors input electric impedance (depends on the morphology of the dendritic spine and its electric properties)
Change of these synaptic properties leads to the change of synaptic strength. This change can be short or long-lasting and negative or positive.

5. 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 can be short-term and without permanent structural changes in the neurons themselves, lasting seconds to minutes — or long-term (long-term potentiation, 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.

6. BCM THEORY AND BCM NEURON [2, 4]
Experimental data from the developing visual cortex have led to the formulation of a synaptic modification rule, known as Bienstock-Cooper-Munro (BCM) rule (Bienstock et al., 1982). The model has two main features: First, it postulates that a neuron possesses a synaptic modification threshold (LTP/LTD threshold or \( \Theta_M \)), which dictates whether the neuron's activity at any given instant will lead to strengthening or weakening of its input synapses. Thus, the modification threshold, \( \Theta_M \), determines the direction of synaptic efficacy change. Synaptic modification varies as a nonlinear (parabolic) function \( \Phi(y) \) of postsynaptic activity \( y \) which is defined as the product between presynaptic activity \( A \) and synaptic efficacy \( S \). Although the firing rate of a neuron \( T(t) \) depends in a nonlinear fashion on the postsynaptic potentials, BCM theory considers that the region between the excitation threshold and saturation may be reasonably approximated by a linear input-output relationship of the model neuron (Benuskova, 2001). The function \( \Phi(y) \) changes sign at a particular value of \( y \), that is the modification threshold \( \Theta_M \). \( \Theta_M \) is the point of crossover from LTD to LTP. If postsynaptic activity is below \( \Theta_M(y<\Theta_M) \), but above baseline, \( \Phi(y) \) is negative and synaptic efficacies are weakened. Conversely, if \( y \) exceeds \( \Theta_M \), active synapses \( \Phi(y) \) becomes positive and active synapses potentiate.

For any value of \( y<\Theta_M \), synaptic strength decays until it reaches 0.

\[
\Phi(y(t),\Theta_M(t)) = y(t)A(t) - \delta M(t);
\]
\[
T(t) = \sum S(t)A(t);
\]
\[
dS = \delta A \delta \Theta \quad (\delta \text{ is the modification rate})
\]

Synaptic weight \( S \) changes according to Hebb's learning rule which requires correlate pre- and postsynaptic activity at the synapse.

![Figure 1: The BCM Synaptic Modification Rule denotes the y output activity of the neuron.]()

7. MATERIAL AND METHODS
Receptive fields of orientation selective cells in visual cortex are composed of excitatory and inhibitory subfields connected to ON and OFF center retinal and LGN cells, respectively. 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). 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 [5, 6, and 7].

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.

8. ASSUMPTIONS FOR SIMPLIFICATION
For mathematical simplification, we take the following assumptions:

1. Some retinal Ganglion cells are highly transient while others have a more stationary response. In our work we have assumed stationary temporal filter. This assumption is appropriate because a BCM model is a rate based model of synaptic plasticity which is not very sensitive to the temporal properties of their input.

2. The reflected light from the object is focused by the lens in our eyes onto the retina. It is then sampled and transduced by the receptor in the retina where the retinal circuitry transforms these signals. The Ganglion cells which are the output neurons of the retina transmit this information through the optic nerve via optic chiasm. In this, input from both the eyes cross into the Lateral Geniculate Nucleus (LGN). From the LGN, the signal is projected to the visual cortex.

3. The LGN has a complex system, including a massive feedback from the cortex. Recently it was shown by Siellito et al. in 1994, that this back projection has a significant impact on the temporal correlation in LGN. Welicky and Katz in 1994, has demonstrated that it increases the correlation between LGN layers from

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4. For simplicity we have chosen, to model the LGN as simple relay station which does not alter the response properties of the retinal ganglion cells.

9. ARCHITECTURE OF MODEL

In this subsection, the basic architecture of the model is described. In our simulation of the receptive field development of single 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 2. The output layer composed of a single cortical cell, which represent cell of layer IV C of cat primary visual cortex [7]. 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.

We also assume that cells of each center type are present at all locations in both the sheets of the LGN layer. Each LGN cell is constrained to always arborize over a fixed, topographically appropriate circular patch of cortical cells with diameter of 13 grid units. 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 one cortical cell are lumped together and are represented by a single quantity that is synaptic strength. The value of the synaptic strength at time t between LGN cell in sheet labeled “ON” and cortical cell is given by “SON”. Similarly the synaptic strength between LGN cell in sheet labeled “OFF” and cortical cell is given by “SOFF”. The ON and OFF Cell has 13*13 connections (169 each). The input of “ON” cell is given by “AON” where, AON represents the activity of ON cell. Its weight is randomly taken as from 0.8 to 1.2 (0.8 + 0.4*rand) by generating a random number between 0-1. Initially, we take threshold as 0.8. It means that it will give output as 1 when its value is more than 0.8 otherwise it will give zero. The same treatment is applicable for OFF type cell. The synaptic weight for ON and OFF type cell are represented as SON and SOFF respectively. The total input of ON cell and OFF cell to the cortical cell is given by equation 3.

\[ TON = \sum_{i=1}^{169} SON_i, AON_i \cdots \cdots \cdots \cdots \cdots \cdots (1) \]

Similarly for “OFF” cells

\[ TOFF = \sum_{i=1}^{169} SOFF_i, AOFF_i \cdots \cdots \cdots \cdots \cdots \cdots (2) \]

We define a nonlinear function \( \phi \) as

\[ \phi = y \ast (y-av) \cdots \cdots \cdots \cdots \cdots \cdots (7) \]

The sigmoid function sets the maximum and minimum values of the postsynaptic response relative to spontaneous cortical activity. Thus we can make the following relation

\[ y = (1/(1+exp(-y))) \cdots \cdots \cdots \cdots \cdots \cdots (4) \]

The threshold is made variable depending on the postsynaptic response. Therefore we have calculated average of total post synaptic response(y). This is given as

\[ sumy = sumy+y \cdots \cdots \cdots \cdots \cdots \cdots (5) \]

\[ avy = sumy/t1 \cdots \cdots \cdots \cdots \cdots \cdots (6) \]

The total post synaptic response to the cortical cell is given by

\[ y = TON + TOFF \cdots \cdots \cdots \cdots \cdots \cdots (3) \]

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\[ y = TON + TOFF \cdots \cdots \cdots \cdots \cdots \cdots (3) \]

\[ \phi = y \ast (y-av) \cdots \cdots \cdots \cdots \cdots \cdots (7) \]

Therefore change in synaptic weight is given as follows

\[ dSON_i = \delta \ast \phi \ast AON_i, \cdots \cdots \cdots \cdots \cdots \cdots (8) \]

Where, dSONi is the change in the synaptic weight of ith ON cells, AON is the input to the ON cells and delta is a leaning rate.

Similarly for OFF cells,

\[ dSOFF_i = \delta \ast \phi \ast AOFF_i, \cdots \cdots \cdots \cdots \cdots \cdots (9) \]

Where, dSOFFi is the change in the synaptic weight of ith OFF cells and AOFF is the input to OFF cells.

10. WEIGHT NORMALIZATION

Weight normalization is an essential feature in the development of receptive field. It is a procedure whereby some measure of the total synaptic weight onto the recipient neuron is used to limit the growth of the synaptic weights. Normally there are two ways to normalize the synaptic weights.
weights that are multiplicative normalization and subtractive normalization [8].

The implementation of the weight normalization is explained below

11. SUBTRACTIONAL NORMALIZATION
In the beginning OST (Original Sum Total) is calculated as

\[ \text{OST} = \text{sum}(\text{sum}(	ext{SON}))+\text{sum}(\text{sum}(	ext{SOFF})). \] (10)

Subtractive normalization factor K is calculated as

\[ K = \frac{\text{NST} - \text{OST}}{\text{TC}} \] (11)

Where NST is New Sum Total and calculated as OST in the loop and TC is the total connections. Finally, the subtractive normalization is performed as

\[ \text{SON} = \text{SON} - K \] (12)

and

\[ \text{SOFF} = \text{SOFF} - K \] (13)

After this normalization SON and SOFF less than zero are set to zero and SON and SOFF greater than SMAX are set to SMAX.

12. MULTIPLICATIVE NORMALIZATION
For the implementation of multiplicative normalization, we calculate a factor

\[ K_1 = \frac{\text{OST}}{\text{NST}} \] (14)

Now the weights are re-scaled as:

\[ \text{SON} = \text{SON} \times K_1 \] (15)

And

\[ \text{SOFF} = \text{SOFF} \times K_1 \] (16)

13. ACTIVITIES OF LGN CELLS
The activities at time t of the ON type and OFF type LGN cells at location i with appropriate correlations (h=0.2) were generated as given in Goodhill 1993.

The standard parameters used in the model, are shown in the Table 1.

<table>
<thead>
<tr>
<th>Sl.</th>
<th>Parameter</th>
<th>Symbol</th>
<th>Optimal Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Number of Iterations</td>
<td>t1</td>
<td>35000</td>
</tr>
<tr>
<td>2</td>
<td>Number of Input Activities</td>
<td>AON</td>
<td>169</td>
</tr>
<tr>
<td>3</td>
<td>Synaptic Weight (Initial)</td>
<td>SON SOFF</td>
<td>From 0.8 to 1.2</td>
</tr>
<tr>
<td>4</td>
<td>Learning rate</td>
<td>Δ</td>
<td>0.1</td>
</tr>
<tr>
<td>5</td>
<td>Maximum Synaptic strength</td>
<td>SMAX</td>
<td>6</td>
</tr>
<tr>
<td>6</td>
<td>Time Difference</td>
<td>Dt</td>
<td>0.5</td>
</tr>
</tbody>
</table>

14. RESULTS AND ANALYSIS:
In our model, we stored random numbers in a 35000x169 array. This array used for generation of input activity. We run this model with optimal constraints as given in table 1, for nine times and found same result (segregated RF) as shown in figure 3.

14.1 Effect of Correlation Factor 'h' on Development of Simple Cell RF Structure
The correlation factor ‘h’ determines how correlated or uncorrelated the activities of ON and OFF type of LGN cells are? In this model, we vary ‘h’ from 0 to 0.5 in the step of 0.1. h=0, means that ON and OFF cell activities are anticorrelated, h=0.5 means that the activities are perfectly correlated. As can be seen from figure 4 that for h=0 the activities of ON and OFF type cells are not same at any spatial location whereas for h=0.5 the activities of ON and OFF type cells are exactly similar at all the spatial locations. The development of receptive field is also dependent on amount of correlation between the ON and OFF type of LGN cells. We varied the value of ‘h’ and kept all the other variables constant as mentioned in the table 1.

Figure 3: Development of RF for same input activity

Figure 4: The effect of correlation factor (h) on the development of RF for same input activity. 'h' vary from 0 to 0.5 with the step of 0.1
The BCM model is an important theoretical treatment of plasticity in the developing visual cortex that appears applicable to many other brain regions [2]. As we have seen that when the correlation factor is varied, the segregation gets disturbed. After the particular value of h, the segregation does not occur.

We found that optimal value of h is 0.2. The reason is that as the correlation between ON and OFF type cell increases the synaptic strength of connection between ON/OFF and simple cell increase/decrease in similar fashion from similar spatial locations.

It is obvious that if we decrease the learning rate then we have to increase the number of iterations.

16. REFERENCE