

A Self-Organizing Model for the Development of Ocular Dominance and Orientation Columns in the Visual Cortex



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Abstract. A binocular model describing the ontogenetic development in the visual nervous system is presented. It consists of a set of deterministic differential equations which have been derived from an statistical approach. The evolution of the solution is led by the spontaneous generation of input activity, characterized in this model by its spatial and temporal decorrelation. The development of a connection depends on the output activity of both connected neurons; for this purpose, Hebbian and anti-Hebbian learning have been used. The model can explain some properties observed in natural brains such as the appearance of ocular domains and orientation selectivity in the V1 visual cortex development.

Keywords: Neural Networks, Self-organization, Hebbian and Anti-Hebbian Learning, Spontaneous Activity, Orientation Columns, Ocular Domains.

1 Introduction

The first model for the development of orientation receptive fields based in the incredible synaptic plasticity of the brain appeared 25 years ago [15]. Since then, different kind of models have been proposed motivated by the idea that the understanding of the different processes, inner to the visual cortex, will develop new theories of learning and memory. These models show different properties which have been experimentally observed. They also imply new results which must be verified. Among those properties are the appearance of ocular domains [17, 16, 4, 10, 1, 13, 12] and the orientation selectivity columns [15, 8, 9, 2, 13, 12] (see [14] for more information).

The genome of a mammal is constituted by the order of 10^5 genes and the 60% of them are related to the nervous system; only the number of connections between the neurons of a mammal is on the order of 10^{15} . Thus, apparently,

the genome is not able to store all the information necessary for the ontogenetic development in a direct codification. Therefore, simple local rules which are able to be easily codified, constitute the base of these self-organizing processes [15, 7, 5, 6, 8]. On the other hand, it is not well known what kind of activity governs these processes in natural systems such as the spontaneous activity of the prenatal development or the visual driven experience. Most researchers think that each one has its own function in the development. A recent work states that some kind of correlated spontaneous activity in the prenatal development diminishes orientation selectivity [19].

This paper is an extension of a previous work [2] where a monocular model was able to explain the origin of orientation columns. Now, the idea of binocularity has been added (following [1] where ocular domains were simulated), which allows to explain simultaneously the origin of both ocular domains and orientation columns.

2 Model

A three layer architecture extended from that in [2] is used (see fig.1). In the following description, one-dimensional layers are used for simplicity. The input layers describe the activity of the magnocellular layers of the LGN (lateral geniculate nucleus) which receive information from the different retinas (ipsilateral and contralateral). In order to simplify, the pathway between retinas and the LGN, due to the retinotopic organization among them, has not been taken into account [18, 20]. The output layer represents the layer IV in the V1 visual cortex.

Both input layers are constituted by n neurons; m neurons form the output layer. N_i^a ($i = 1, \dots, n$) represents the i th neuron from the input layer (the same kind of representation has been used for the rest of the elements in the model). Unidirectional weighted connections join different neurons: afferent excitatory connections (\mathcal{W}_{iq}^a and \mathcal{W}_{kq}^b) join the input layers with the output layer, intra-layer lateral connections (\mathcal{Q}_{pq}) are placed in the output layer with inhibitory properties in order to simulate the local action of the interneurons. Excitatory and inhibiting properties can not be interchanged between different kind of connections.

$$\mathcal{W}_{iq}^a(t) \geq 0 \quad (1)$$

$$\mathcal{W}_{kq}^b(t) \geq 0 \quad (2)$$

$$\mathcal{Q}_{pq}(t) \geq 0 \quad (3)$$

Afferent and lateral connections change dynamically and stepwise depending on the output activities of the neurons they connect [2] as follows

$$\mathcal{W}_{iq}^a(t+1) = \mathcal{W}_{iq}^a(t) + \beta \mathcal{W}_{iq}^a(t) \left(F_{iq}^{w^a}(t) - R_{iq}^{w^a}(t) \right) + \alpha \quad (4)$$

$$\mathcal{W}_{kq}^b(t+1) = \mathcal{W}_{kq}^b(t) + \beta \mathcal{W}_{kq}^b(t) \left(F_{kq}^{w^b}(t) - R_{kq}^{w^b}(t) \right) + \alpha \quad (5)$$

$$\mathcal{Q}_{pq}(t+1) = \mathcal{Q}_{pq}(t) + \beta \mathcal{Q}_{pq}(t) \left(F_{pq}^q(t) - R_{pq}^q(t) \right) + \alpha \quad (6)$$

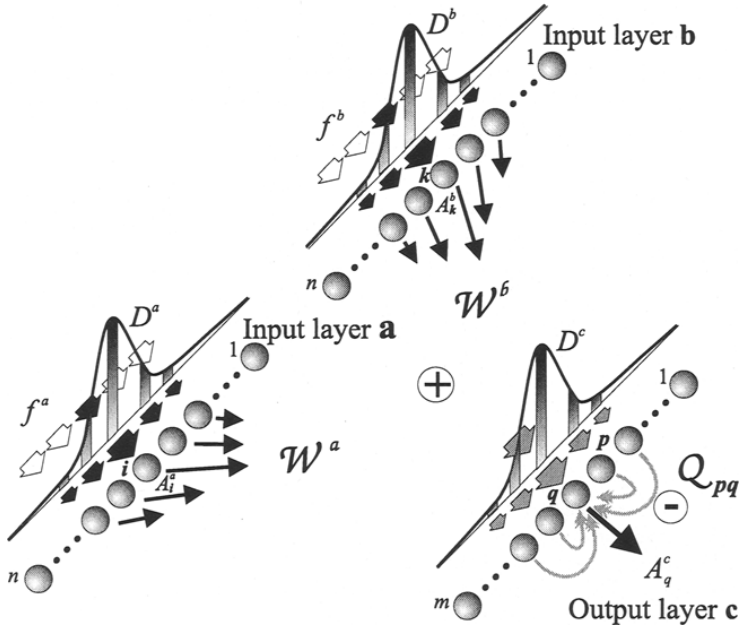


Fig. 1. Schematic representation of the architecture of the model. Both Input layers (layer a and layer b) represent LGN (Lateral Geniculate Nucleus) layers. The output layer (layer c) represents the layer IV of the V1 visual cortex. Dendro-dendritic diffusion functions D modulate the activity in all the layers. On the other hand, afferent connections are placed between input and output layers W , and intra-layer inhibiting lateral connections Q are in the output layer c

Their development will be a balance between increasing and decreasing factors. $F_{iq}^{w^a}(t)$, $F_{kq}^{w^b}(t)$ and $F_{pq}^q(t)$ are the growing synaptic factors of both afferent and lateral connections. The development of a connection can not increase continuously. To avoid it, restriction synaptic factors are used ($R_{iq}^{w^a}(t)$, $R_{kq}^{w^b}(t)$ for the excitatory connections, and $R_{pq}^q(t)$ for the inhibitory connections) instead of the classical mathematical normalization [11] which is more artificial. Synaptic plasticity speed is controlled by the parameter β . Finally, the parameter α maintains both the afferent and lateral connection properties avoiding the possibility of a property interchange according to (1-3)

The growing synaptic factor is a direct interpretation of the classic Hebb law [3]. The growth of a connection depends on the output activity correlation between both connected neurons. Therefore, the excitatory connections use

Hebbian learning while anti-Hebbian learning is used by the inhibiting ones [2]

$$F_{iq}^{w^a}(t) = \langle A_i^a(t), A_q^c(t) \rangle_t \quad (7)$$

$$F_{kq}^{w^b}(t) = \langle A_k^b(t), A_q^c(t) \rangle_t \quad (8)$$

$$F_{pq}^q(t) = \langle A_p^c(t), A_q^c(t) \rangle_t \quad (9)$$

$A_i^a(t)$, $A_k^b(t)$, $A_p^c(t)$ and $A_q^c(t)$ are the output activities of N_i^a , N_k^b , N_p^c and N_q^c respectively. On the other hand, the restriction factor of a connection depends on its own value [2], so that

$$R_{iq}^{w^a}(t) = \gamma_{w^a} (\mathcal{W}_{iq}^a(t))^2 \quad (10)$$

$$R_{kq}^{w^b}(t) = \gamma_{w^b} (\mathcal{W}_{kq}^b(t))^2 \quad (11)$$

$$R_{pq}^q(t) = \gamma_q (\mathcal{Q}_{pq}(t))^2 \quad (12)$$

where, γ_{w^a} , γ_{w^b} and γ_q are the parameters which control the amount of synaptic material available for the neurons.

The dynamics of the system is as follows: at each time t a spatial and temporal uncorrelated input activity pattern (f) is generated as a result of simulating the spontaneous activity in the prenatal period. No correlation has been introduced between different input layers [13]. Hence

$$\langle f_i^a(t), f_k^a(t) \rangle_t = \delta_{ik} \quad (13)$$

$$\langle f_{i'}^b(t), f_{k'}^b(t) \rangle_t = \delta_{i'k'} \quad (14)$$

$$\langle f_i^a(t), f_k^a(t + \Delta t) \rangle_t = 0 \quad (15)$$

$$\langle f_k^b(t), f_{k'}^b(t + \Delta t) \rangle_t = 0 \quad (16)$$

$$\langle f_i^a(t), f_{i'}^b(t + \Delta t) \rangle_t = 0 \quad (17)$$

where, δ_{ik} is the Kronecker delta.

The output activity of an input layer neuron (see fig.1) is the result of dendro-dendritic interactions adding the diffused activity from neighbor neurons according to

$$A_i^a(t) = \sum_{h=1}^n f_h^a(t) D_{hi}^a \quad (18)$$

$$A_k^b(t) = \sum_{g=1}^n f_g^b(t) D_{gk}^b \quad (19)$$

where f represents the spontaneous uncorrelated activity in the input layers and D the diffusion of activity between two neurons of the same layer (modelled with a Gaussian function). The activity is propagated through the afferent connections towards the output layer

$$I_o^c(t) = \sum_{i=1}^n A_i^a(t) \mathcal{W}_{io}^a(t) + \sum_{k=1}^n A_k^b(t) \mathcal{W}_{ko}^b(t) \quad (20)$$

where $I_o^c(t)$ is the input which reaches the $N_o^c(t)$ cortical neuron. The same kind of dendro-dendritic interactions of the input layer takes place in the output layer

$$S_r^c(t) = \sum_{o=1}^m I_o^c(t) D_{or}^c \quad (21)$$

The response of an output neuron is the result of all the activity which arrives to the neuron less the action of the interneurons

$$A_q^c(t) = S_q^c(t) - \sum_{r=1}^m S_r^c(t) Q_{rq}(t) \quad (22)$$

On the other hand, a receptive field matrix is used, where each $E_{hq}^a(t)$ element represents the influence of an N_h^a spontaneous activation over the output activity of N_q^c

$$E_{hq}^a(t) = \sum_{i=1}^n D_{hi}^a \sum_{o=1}^m W_{io}^a(t) \left(D_{oq}^c - \sum_{r=1}^m D_{or}^c Q_{rq}(t) \right) \quad (23)$$

It can be shown that (see equations 18–23):

$$A_q^c(t) = \sum_{h=1}^n f_h^a(t) E_{hq}^a(t) + \sum_{g=1}^n f_g^b(t) E_{gq}^b(t) \quad (24)$$

As a result of what is described above, and using an statistical approach which eliminates the explicit dependence of the formulation on the uncorrelated activity values in the input layers (f), it can be shown [2], after some lengthly straightforward algebra, that

$$\langle A_i^a(t), A_q^c(t) \rangle_t = \sum_{h=1}^n D_{hi}^a E_{hq}^b \quad (25)$$

$$\langle A_k^b(t), A_q^c(t) \rangle_t = \sum_{g=1}^n D_{gk}^b E_{gq}^b \quad (26)$$

$$\langle A_p^c(t), A_q^c(t) \rangle_t = \sum_{h=1}^n E_{hp}^a E_{hq}^a + \sum_{g=1}^n E_{gp}^b E_{gq}^b \quad (27)$$

Therefore, it can be concluded that

$$\Delta \mathcal{W}_{iq}^a(t) = \beta \mathcal{W}_{iq}^a(t) \left(\sum_{h=1}^n D_{hi}^a E_{hq}^a - \gamma_{w^a} (\mathcal{W}_{iq}^a(t))^2 \right) + \alpha \quad (28)$$

$$\Delta \mathcal{W}_{kq}^b(t) = \beta \mathcal{W}_{kq}^b(t) \left(\sum_{g=1}^n D_{gk}^b E_{gq}^b - \gamma_{w^b} (\mathcal{W}_{kq}^b(t))^2 \right) + \alpha \quad (29)$$

$$\Delta \mathcal{Q}_{pq}(t) = \beta \mathcal{Q}_{pq}(t) \left(\sum_{h=1}^n E_{hp}^a E_{hq}^a + \sum_{g=1}^n E_{gp}^b E_{gq}^b - \gamma_q (\mathcal{Q}_{pq}(t))^2 \right) + \alpha \quad (30)$$

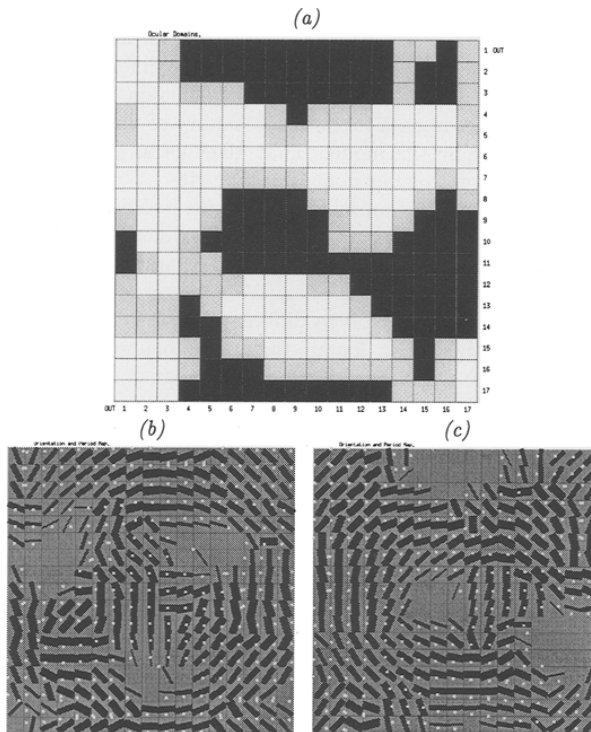


Fig. 2. (a) ocular domains (b) and (c) maximum orientation response in the output layer to both eye oriented input stimuli. The topography is also represented (little squares).

3 Results

Simulations have been worked out using two-dimensional input and output layers. Each one has a total amount of 17x17 neurons. This system is composed by more than 250,000 coupled ordinary differential equations (eqs.28–30) which evolve in a deterministic way. The weights have been initialized with a very low value and a 0.8 retinotopic degree [1, 2]. In order to avoid boundary effects and the problems that arise from the low number of cells, layers with continuous boundary conditions (toroidal geometry) have been used.

We are going to analyze the state reached at $t = 2000$, where the properties we are looking for have been successfully obtained. The ocular domain distribution can be clearly seen in fig.2a: patches of cells which respond better to the input layer a (white color), patches which respond better to stimuli in the input

layer b (black color) and other neurons which receive approximately the same influence from both eyes (grey color). According to other models, binocular neuron responses are placed in the boundaries between different ocular domains. The geometry of the ocular domains reached depends on different factors such as the number of neurons in each layer, the geometry of the layer, and the value of the parameters used by the model. That gives us an idea of why different mammals have different ocular domain geometries.

On the other hand, orientation selective columns (see figs.2b,2c) appear with the typical slight orientation variation in the nearest neurons. Each orientation represents the best response of an orientation column because we are dealing with 2-D layers and not with 3-D ones. We have represented the maximum response of an output layer neuron to different oriented input stimuli (in one eye), independently of the ocular domain the neuron belongs to. In the course of the system evolution we have observed that the ocular domains are established faster than the orientation receptive fields of the cortex neurons.

The little points (see figs.2b,2c) represent the center of the maximum response to symmetrical stimuli. In order to observe properly the retinotopic degree of the projections, continuous boundary conditions have to be considered. Note that, as for orientation selectivity, close neurons have receptive fields centered at close retinal positions, i.e., a retinotopical projection of the visual space is obtained in the output layer. Moreover, we have observed in other simulations, in which the weights were initialized in a completely random way, that a retinotopic topography has also been reached.

4 Conclusions

A new binocular model for self-organizing development in the visual nervous system is presented which evolves with a low amount of provided information. The evolution only depends on the dynamic change of both its afferent and its lateral connections, which evolve following the output activity of both connected neurons using Hebbian and anti-Hebbian learning. Although the system is very complex from a computational point of view, an statistical approach is used in order to simplify it. As a result, quite simple deterministic differential equations have been obtained.

The model shows the origin of both ocular domains and orientation selective columns. We have shown that the development of orientation receptive fields is posterior to the development of ocularity domains and, therefore, it is extremely dependent on it. The fidelity to real biological data of the simulated maps of cortical orientation selectivity requires its combined development with that of ocular domains. The integration of two different developmental models [1, 2] is a step in this direction. Moreover, the appearance of different ocular domain geometries in different mammals can be explained based on slight physiological variations from organism to organism which correspond to different sets of parameters in our model. At this point, the results have to be further analyzed in order to develop more assumptions about the development of the cortex.

We have shown that it is possible to reach retinotopic projection from noisily initialized weights. Instead of the strabismus used here, correlation between the eyes can be simulated and, in that case, narrow ocular domains should be expected. On the other hand, future works based on the present article are expected to describe more complicated properties such as directional selectivity.

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