Development of *on-off* and *off-on* Receptive Fields Using a Semistochastic Model

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Abstract. A model for ontogenetic development of receptive fields in the visual nervous system is presented. The model uses a semistochastic approach where random uncorrelated activity is generated in the input layer and propagated through the network. The evolution of the synaptic connections between two neurons are assumed to be a function of their activity, with two interpretations of the Hebb's rule: (a) the synaptic weight is modified proportional to the product of the activity of the two connected neurons; and (b) proportional to the statistical correlation of their activity. Both models explain the origin of either *on-off* and *off-on* receptive fields with symetric and non symetric forms. These results agree with previous models based on deterministic equations. The approach presented here has two main advantages. Firstly the lower computer time that allows the study of more complex architectures. And secondly, the possibility of the extension of this model to cover more complex behavior, for instance, the inclusion of time delay in the transmition of the activity between layers.

keywords: neural networks, self-organization, receptive fields, unsupervised learning, semistochastic models.

1 Introduction

The nervous visual system of most higher mammals is formed and organized during the prenatal period in a process dependent of the spontaneous activity inner to the visual system [3]. This process gives raise to a highly structured distribution of neurons and connections, needed for the complex selective properties that appear in the adult visual system. One of the characteristics of the primary visual cortex is the organization in columns selective to the orientation of the stimulus. The origin of this selectivity is based on the formation of receptive fields with different shapes and orientation [9].

Apparently, the information contained in the genome is not sufficcient to establish the adult functional state of the system. However, several neurophysiological models using neural networks have shown that this ontogenetic development is based on self-organization processes driven by quite simple rules, that can be equally codified in the individuals of the system (the neurons) [11], [6], [7], [8] and [10].

In previous works [1] and [2] we have developed a neural network model that account for the ontogenetic development of occularity domains and variable-sized receptive fields. These models are based on the principles of cortical self-organization early proposed by von der Malsburg [4]. The final or mature state of the connectivity between two neural layers in the visual system is calculated as the solution of a set of differential equations for given initial conditions. Thus, starting from a desordered state, given by connection (weight) matrixes with random values, the system evolves *deterministically* towards the final ordered solution that accounts for a given functional state.

In this paper a modification of our previous model is presented. Using the same approach in the calculation of the evolution of the connecting weights, we have introduced here a semistochastic approach. The input activity is randomly generated and propagated through the network. In this way the activity of any couple of connected neurons can be calculated numerically. The modification of the corresponding weight is then performed according to two interpretations of the Hebb's rule: (a) proportional to the product of the activities; and (b) proportional to the correlation. As described elsewere [2], hebbian and anti-hebbian learning is used for activating and inhibiting connections, respectively. The cycle of generation and propagation of activity and modification of the weights (unsupervised learning) are repeated until a convergence in the weight distribution is encountered. Then, the final connectivity structure is analized. As a first approximation we have shown that the application of either of the Hebb's rule interpretations leads to similar results.

Finally, the extension of this model to account for some other characteristics or global properties of the visual system is discussed.

2 Model

In this work we use a two layer architecture [2], which attempts to reproduce the global property evolution of a locally ruled system as observed in the visual nervous system. The input layer is constituted by n neurons, (see fig.1) and the output layer by m. N_i^a $(i = 1, \ldots, n)$ represents the i input layer neuron and N_j^b $(j = 1, \ldots, m)$ the j output layer neuron. We have used the same descriptive criterium in the rest of elements of our model. The two layers are connected using different unidirectional weighted connections (synaptic weights): 1) action activators ($\mathcal{W}$) between both layers and 2) lateral inhibitors in the output layer ($\mathcal{Q}$). Both connectors control the level of activity interchanged among different neurons. Inhibitors model the inhibiting action of the visual cortex interneurons. Since the activating/inhibiting properties of connectors cannot be interchanged,

a neuron in the input layer can directly inhibit no neuron in the output layer. The strength of these unidirectional connections has been artificially represented by two matrixes $\mathcal{W}(t)$ and $\mathcal{Q}(t)$ with real positive weights:

$$\mathcal{W}_{ij}(t) > 0 \tag{1}$$

$$\mathcal{Q}_{kl}(t) > 0 \tag{2}$$

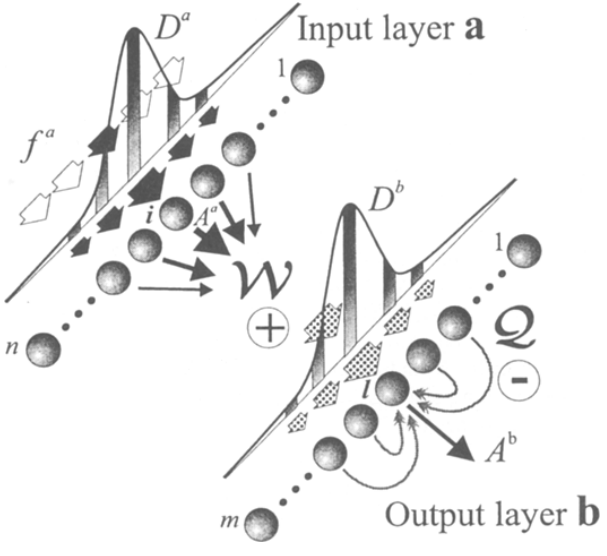

Fig. 1. Schematic representation of the architecture of the model.

The dynamics of the system can be described as follows. A random input activity pattern $f_h^a(t)$ $h \in [1, \ldots, n]$, both spatially and temporally uncorrelated, is introduced to the input layer. This activity is laterally propagated using a decreasing diffusion gaussian distribution D, establishing simple-neighbour relationships. The activity generated at each neuron is transmitted to the output layer through $\mathcal{W}(t)$, the matrix of activating connections, and diffused using another gaussian function. This activity is also received by the inhibiting interneurons. The output activity of each neuron in this layer is the balance between the input activity and the inhibiting action representing the interneuron dynamics. At a higher level, cooperative and competitive processes can be obtained through a self-organization process led by local rules, fixed diffusion functions and synaptic plasticity.

As a result of synaptic plasticity, the above referred weights are assumed to change stepwise and dynamically through the following evolution equations:

$$\mathcal{W}_{ij}(t+1) = \mathcal{W}_{ij}(t) + \Delta\mathcal{W}_{ij}(t) \tag{3}$$

$$\mathcal{Q}_{kl}(t+1) = \mathcal{Q}_{kl}(t) + \Delta\mathcal{Q}_{kl}(t) \tag{4}$$

According with a previous model described by [2] the increments can be calculated as:

$$\Delta\mathcal{W}_{ij}(t) = \beta\mathcal{W}_{ij}(t)\left(\left(F^{w}_{ij}(t) - R^{w}_{ij}(t)\right)\right) + \alpha \tag{5}$$

$$\Delta\mathcal{Q}_{kl}(t) = \beta\mathcal{Q}_{kl}(t)\left(\left(F^{q}_{kl}(t) - R^{q}_{kl}(t)\right)\right) + \alpha \tag{6}$$

where $F^{w}_{ij}(t)$ and $F^{q}_{kl}(t)$ respectively represent the activating and inhibiting growing synaptic factors, which control the rate of weigth amplification. These factors model the self-amplification properties observed when synaptic weights are modified [12]. Both equations are coupled through the implicit or explicit dependence of the growing factors with $\mathcal{W}_{ij}(t)$ and $\mathcal{Q}_{kl}(t)$.

The model restricts the growing behaviour of the solutions by limiting the synaptic resources available to the neurons, which are given by $\mathcal{W}_{ij}(t)R^{w}_{ij}(t)$ (for activating weights) and $\mathcal{Q}_{kl}(t)R^{q}_{kl}(t)$ (for inhibiting weights). Synaptic plasticity speed is controlled by the parameter β. Finally, the parameter α is a consequence of (1) and (2) in order to maintain both the activating and inhibiting connection properties avoiding the possibility of a property interchange. Following [2], restriction factors are set to:

$$R^{w}_{ij}(t)) = \gamma_w\mathcal{W}^2_{ij}(t) \tag{7}$$

$$R^{q}_{kl}(t)) = \gamma_q\mathcal{Q}^2_{kl}(t) \tag{8}$$

where γ_w and γ_q are the parameters controlling the resources available to neurons.

The process carried out by each neuron or processor depends on the kind of input activity pattern the system is fed with. Random positive real input patterns have been used to simulate spontaneous photoreceptors activities in the ontogenetic development of the visual nervous system. On the other hand, the development of self-organized connections depends on the way in which weights are initialized. We have set them to:

$$(\mathcal{W},\mathcal{Q})_{ij}(0) = m\left((1-b)r + \tfrac{b}{1+d_{ij}}\right) \tag{9}$$

where r is a random real number in $[0,1]$, m is the maximum value any weight can take, b is a real number in $[0,1]$ representing the retinotopic degree, and d_{ij} is the distance between the two neurons connected by $(\mathcal{W},\mathcal{Q})_{ij}(t)$.

In this work, two different kinds of dynamically changing growing factors are presented, both of them calculated using different implementations of hebbian [5] and anti-hebbian plasticity.

2.1 Activity-product learning model

As a first step we have used the usual implementation of Hebb's rule based on the product of the output activities of the neurons joined by the respective connection. In this way, at any time, the growing synaptic factors $F_{ij}^{w}(t)$ and $F_{il}^{q}(t)$ are calculated as:

$$F_{ij}^{w}(t) = \delta_w A_i^a(t) A_j^b(t) \tag{10}$$

$$F_{kl}^{q}(t) = \delta_q A_k^b(t) A_l^b(t) \tag{11}$$

where δ_w and δ_q are learning rates and $A_i^a(t)$ and $A_j^b(t)$ represent respectively N_i^a and N_j^b output activities.

At time t and in each first layer neuron N_h^a we set $f_h^a(t)$ equal to a randomly generated input in the interval $[0, 1]$. The output activity of a first layer neuron is then calculated as the contribution of its own activity and those of the other neurons after being modulated (in a neighbouring sense) using a fixed diffusion function D^a, which decreases with the distance between neurons.

$$A_i^a(t) = \sum_{h=1}^{n} f_h^a(t) D_{hi}^a \tag{12}$$

The activity outputs produced by the first layer neurons are propagated through activating connections:

$$I_j^b(t) = \sum_{i=1}^{n} A_i^a(t) \mathcal{W}_{ij}^a(t) \tag{13}$$

The resulting total input activity of any second-layer neuron is then calculated as the result of its own activity and the neighbours activities affected by another fixed lateral-diffusion function.

$$S_k^b(t) = \sum_{j=1}^{m} I_j^a(t) D_{jk}^b \tag{14}$$

Taking into account the second layer inhibiting interneuron connections, the output neuron activity is:

$$A_l^b(t) = S_l^b(t) - \sum_{k=1}^{m} S_k^b(t) \mathcal{Q}_{kl}(t) \tag{15}$$

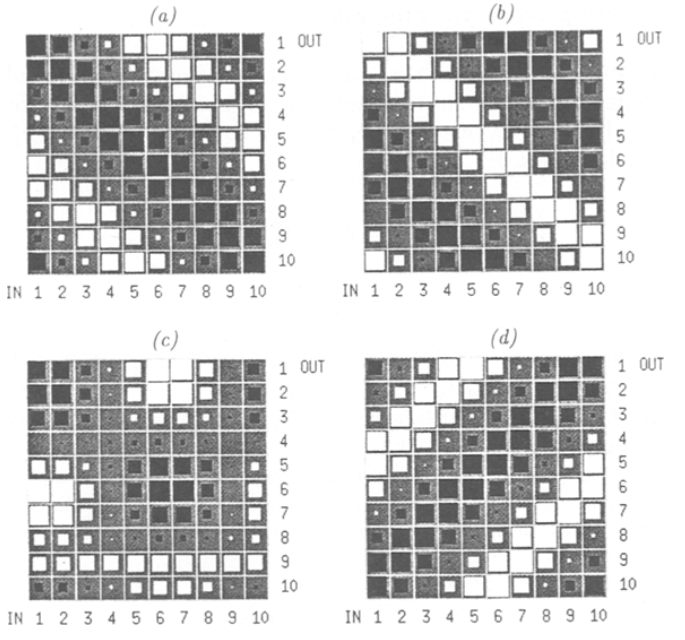

Fig. 2. Receptive fields obtained with the activity-product learning model. *(a)On-off* homogeneous receptive fields. *(b)Off-on* homogeneous receptive fields. *(c)On-off* non-homogeneous receptive fields. *(d)On-off* antiretinotopic receptive fields.

Results of the activity-product learning model. In order to analize the self-organization process results, an array E has been used with the element $E_{il}(t)$ representing the influence of the activation of the input activity $f_i^a(t)$ of neuron N_i^a on the output activity $A_l^b(t)$ of the second layer neuron N_l^b. If $E_{il}(t) > 0$, the first layer neuron (N_i^a) belongs to the *on* part of the receptive field of N_l^b neuron, and if $E_{il}(t) < 0$ it belongs to the *off* part of its receptive field. Therefore, $E_{il}(t)$ constitutes the N_l^b receptive field. After some lengthy straight-forward algebra, it is easily deduced that:

$$E_{il}(t) = \sum_{j=1}^{n} D_{ij}^a \sum_{k=1}^{m} \mathcal{W}_{jk}(t) \left(D_{kl}^b - \sum_{o=1}^{m} D_{ko}^b \mathcal{Q}_{ol}(t) \right) \tag{16}$$

As a first approach, we have used one-dimensional input and output layers with 10 neurons. This simple topology reduces the complexity (number of connections, computing time, ...) of the problem. In order to avoid border effects, the low number of neurons is compensated by toroidal boundary conditions in any of the layer geometries.

Results are presented using Hinton's diagrams (see fig.2), where black and white colours are respectively related to the on and off parts of the receptive fields.

In fig.2, homogeneous *on-off* receptive fields have been obtained after 3.10^5 time cycles (a). In this case high retinotopic initiation bias has been used. If low retinopic initiation bias is used then *off-on* receptive fields are found (b). For some middle initial conditions an antiretinotopic homogeneous solutions are obtained (d). When the synaptic growth restriction is decreased some non symetric solutions can be obtained (c). This indicates the existence of different attractors depending on the initial conditions and controlling parameters. It can be deduced that the atraction basin of the symetric retinotopic solution diminishes when the competence term decreases. Then, for some initial conditions the system lay on other non symetric solutions. For long term simulations, most of the solutions evolve towards symetric solutions, either *on-off*, *off-on*, retinotopic, or anti-retinotopic.

2.2 Activity-correlation learning model

At time t_p, the synaptic growth factor is taken proportional to the statistical correlation between the different output activities of those neurons joint until t_p by the connections we are interested in.

$$F_{ij}^{w}(t_p) = \delta_w \, \rho \left(\mathcal{A}_i^a(t_p), \mathcal{A}_j^b(t_p) \right) \tag{17}$$

$$F_{kl}^{q}(t_p) = \delta_q \, \rho \left(\mathcal{A}_k^a(t_p), \mathcal{A}_l^b(t_p) \right) \tag{18}$$

where ρ is the correlation given by:

$$\rho(x,y) = \frac{cov(x,y)}{\sigma_x \sigma_y} = \frac{\overline{xy} - \overline{x}\,\overline{y}}{\sigma_x \sigma_y} \tag{19}$$

where x and y are data sets and σ_x and σ_y their respective standard deviation. Therefore

$$\mathcal{A}_i^a(t_p) = [A_i^a(t_0), A_i^a(t_0 + \Delta t), \ldots, A_i^a(t_0 + p\Delta t)] \tag{20}$$

$$\mathcal{A}_j^b(t_p) = [A_j^b(t_0), A_j^b(t_0 + \Delta t), \ldots, A_j^b(t_0 + p\Delta t)] \tag{21}$$

Results of the activity-correlation learning model. Using similar strategies to those used in the activity-product growing model experiments, the results plotted in fig.3 have been obtained.

The different receptive fields shown in fig.3, have been obtained after the same number of time cycles, and similar solutions are found. Homogeneous receptive fields are obtained using a high retinotopic initiaton bias (a) and can be shifted in order to find antiretinotopic fiels decreasing the retinotopic initiation degree

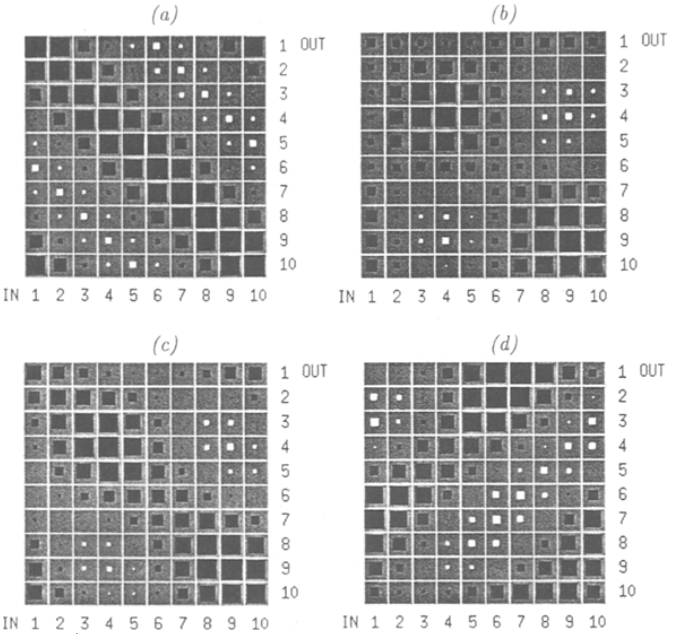

Fig. 3. Receptive fields obtained with the activity-correlation learning model. *(a) On-off* homogeneous receptive fields. *(b) On-off* non homogeneous receptive fields. *(c) On-off* non homogeneous receptive fields. *(d) Off-on* antiretinotopic receptive fields.

(d). As before, inhomogeneous receptive fields can also be found (b) and (c). If this inhomogenity could be found in a two or tree dimensional layers system, each receptive field would then be able to detect other important global properties such as orientation.

3 Conclusions

The results obtained (fig.2 and fig.3) agree with those found using the deterministic model proposed in earlier works [2]. In that case, statistic was used to correlate the input patterns, and as a result of this correlating process, deterministic differential equations were obtained. The two different self-organized models proposed here are semistochastic. Their development depends on a random input pattern generated at real time, in the same way that in natural systems. This activity patterns are then propagated through the network and the activity of the different neurons is calculated each time cycle. Since lower computational time is needed in these new models, future works are expected to be able to

describe the self-organized dynamics of more complex global properties that can be detected by neurons of the visual cortex.

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