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Modeling dynamic receptive field changes in primary visual cortex using inhibitory learning

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Abstract

The position, size, and shape of the visual receptive field (RF) of some primary visual cortical neurons change dynamically, in response to artificial scotoma conditioning in cats (Pettet & Gilbert, 1992) and to retinal lesions in cats and monkeys (Darian-Smith & Gilbert, 1995). The "EXIN" learning rules (Marshall, 1995) are used to model dynamic RF changes. The EXIN model is compared with an adaptation model (Xing & Gerstein, 1994) and the LISSOM model (Sirosh & Miikkulainen, 1994; Sirosh et al., 1996). To emphasize the role of the lateral inhibitory learning rules, the EXIN and the LISSOM simulations were done with *only* lateral inhibitory learning. During scotoma conditioning, the EXIN model without feedforward learning produces centrifugal expansion of RFs initially inside the scotoma region, accompanied by increased responsiveness, without changes in spontaneous activation. The EXIN model without feedforward learning is more consistent with the neurophysiological data than are the adaptation model and the LISSOM model. The comparison between the EXIN and the LISSOM models suggests experiments to determine the role of feedforward excitatory and lateral inhibitory learning in producing dynamic RF changes during scotoma conditioning.

1 Introduction

1.1 Dynamic receptive fields

In experiments using artificial scotoma conditioning (Pettet & Gilbert, 1992) and retinal lesions (Darian-Smith & Gilbert, 1995), primary visual cortical neurons corresponding to a particular region of visual space were deprived of visual stimulation, while primary visual cortical neurons corresponding to a surrounding region received visual stimulation. In response to these manipulations, a variety of dynamic changes occurred in the position, size, and shape of the receptive field (RF) of some primary visual cortical neurons. For example, after 15 minutes of artificial scotoma conditioning, the RF size of some primary visual cortical neurons corresponding to the inside of the scotoma expanded by a factor of five in area; subsequently, after 15 minutes of normal stimulation, the RFs returned to their original size (Pettet & Gilbert, 1992).

Dynamic receptive fields are of interest for several reasons. They reveal some of the ways in which visual systems may adaptively overcome damage from lesions or scotomas. In addition, they reveal some of the functional organization of visual cortex. Dynamic visual receptive fields might also be related to the dynamic response properties found in other cortical areas, such as the tactile RF expansion/contraction found in somatosensory cortex in response to intracortical microstimulation (Recanzone et al., 1992).

1.2 Three models of dynamic RFs

A neural network model that exhibits similar dynamic RF changes was formulated and tested. The EXIN model (Marshall, 1995) is compared with the "LISSOM" model (Sirosh & Miikkulainen, 1994; Sirosh et al., 1996) and an "adaptation" model (Xing & Gerstein, 1994). The EXIN model (Marshall, 1995) uses an instar feedforward excitatory learning rule (Grossberg, 1972) and an outstar lateral inhibitory learning rule (Marshall, 1995) (see Section 2.1).

The LISSOM model (Sirosh & Miikkulainen, 1994; Sirosh et al., 1996) uses synaptic modifications to account for changes in RFs. The LISSOM model uses feedforward excitatory, lateral excitatory, and lateral inhibitory learning rules with weight normalization. All of the LISSOM learning rules are instar rules (Grossberg, 1972) (see Section 2.2).

In the adaptation model (Xing & Gerstein, 1994), the RF changes occur as a result of adaptive modifications in the sensitivity of single neurons, rather than as a result of modifications in the synaptic weights between pairs of neurons (see Section 2.3).

The three models were simulated, and their performance was compared with the experimental neurophysiological data (Darian-Smith & Gilbert, 1995; Pettet & Gilbert, 1992). To emphasize the role of lateral inhibitory learning, the EXIN model was simulated without feedforward learning, and the LISSOM model was simulated without feedforward and lateral excitatory learning.

2 Method

A patch of primary visual cortical neurons, arranged in a 30×30 grid of spatial positions, was simulated. The position of each neuron's RF corresponded to the neuron's position in the grid. Adjacent RFs initially had more than 50% spatial overlap.

In *scotoma/normal* conditioning, random stimulation in the whole visual field except in an "artificial scotoma" region was followed by whole-field stimulation.

Three sets of learning/adaptation rules, described below, were used. The critical difference between the three sets of rules is how they model the changes in lateral interactions within the cortex.

2.1 EXIN network simulation of dynamic RFs

In the EXIN model (Marshall, 1995) of dynamic RFs, cortical interactions are mediated by lateral inhibitory pathways. The weights Z_{ij}^{-} of lateral inhibitory pathways from neuron i to neuron j vary according to an asymmetric anti-Hebbian outstar learning rule:

$$\frac{d}{dt} Z_{ij}^{-} \;=\; \delta g(x_i) \left(-Z_{ij}^{-} + q(x_j) \right), \tag{1}$$

where $\delta > 0$ is a small learning rate constant and g and q are half-rectified nondecreasing functions. (The EXIN model can also accommodate lateral excitatory pathways and has been used in models of other visual cortical functions with lateral excitation; however, the lateral excitatory pathways are not strictly necessary in the EXIN model of dynamic RFs.)

In an outstar learning rule (Grossberg, 1972), *pre*synaptic activity "enables" the learning at a synapse; when the learning is enabled, the weight tends to become proportional to the *post*synaptic activity. In an instar learning rule, postsynaptic activity enables the learning; when the learning is enabled, the weight tends to become proportional to the presynaptic activity.

2.2 Comparison with LISSOM model

In the LISSOM model (Sirosh & Miikkulainen, 1994; Sirosh et al., 1996) intracortical interactions are mediated by both lateral excitatory and lateral inhibitory pathways. The weights of both

lateral excitatory and lateral inhibitory pathways change according to an instar learning rule:

$$Z_{ij}(t+1) \;=\; \frac{Z_{ij}(t) + \xi x_i x_j}{\left(\sum_k \left[Z_{kj}(t) + \xi x_k x_j\right]^2\right)^{\frac{1}{2}}} \tag{2}$$

where Z_{ij} is the pathway weight and ξ is the learning rate for each type of pathway. The LISSOM rules also incorporate a spatial distance function, so that the net effect of learning between two nearby neurons is Hebbian, while the net effect of learning between two distant neurons is anti-Hebbian.

Sirosh et al. (1996) modeled RF changes during scotoma conditioning using the LISSOM model. During scotoma conditioning, neurons with RFs straddling the scotoma boundary are active, and the LISSOM feedforward instar learning causes the feedforward connection weights from the scotoma region to these active neurons to weaken. During RF measurement after scotoma conditioning, these neurons are insensitive to the previously occluded regions and hence exert less inhibition on neurons that were inactive during conditioning. Thus, the neurons that were inactive during the conditioning show increased responsiveness and RF expansion. Changes in the feedforward connection weights are sufficient to produce RF expansion during scotoma conditioning.

To compare the consequences of an instar and an outstar inhibitory learning rule, the EXIN and the LISSOM models are simulated with *only* the lateral inhibitory learning.

2.3 Comparison with adaptation model

In the adaptation model, intracortical interactions are mediated by fixed (nonadapting) lateral inhibitory pathways. A neuron's ability to fire decreases/increases after a period of activity/inactivity, without any synaptic changes. Neuronal adaptation is modeled by the value value T_i, for neuron i, which varies according to

$$\frac{d}{dt}T_i \;=\; -\eta T_i + (\tau - T_i)\, x_i, \tag{3}$$

where η and τ are positive constants and T_i is bounded within $[0, \tau]$. Neuronal activation is inversely proportional to the adaptation parameter. Thus, periods of neuronal activation/inactivation cause the adaptation parameter to increase/decrease, thereby decreasing/increasing the neuron's responsiveness.

3 Results

3.1 RF expansion during scotoma conditioning

During scotoma conditioning, the EXIN inhibitory learning rule decreases the weights of inhibitory pathways from neurons with RFs outside the scotoma to those with RFs inside the scotoma. This decrease in strength of inhibitory pathways results in a centrifugal expansion of RFs that were originally inside the scotoma (Figures 1(a) and 2(a)).

The LISSOM inhibitory learning rule decreases the strength of lateral inhibitory pathways from neurons with RFs within the scotoma region to those with RFs outside the scotoma region, during scotoma conditioning. This becomes manifested as centripetal expansion of the RF of neurons whose RF was initially outside the scotoma region (Figures 1(b) and 2(b)).

Scotoma conditioning, in the adaptation model, causes neurons whose RF is in the scotoma region to increase their responsiveness and those whose RF is outside the scotoma region to decrease their responsiveness. Thus, the neurons whose RF is outside the scotoma region exert less inhibition on those whose RF is inside the scotoma region. This results in expansion of the RF of neurons whose RF is initially within the scotoma region (Figures 1(c) and 2(c)).

In the absence of any visual stimulation, all neurons in the adaptation model tend to become equally responsive. Thus, the RF expansion following scotoma conditioning is diminished by a period of no visual stimulation (Figure 3). However, Pettet & Gilbert (1992) reported that RF expansion in cat primary visual cortex persisted in the absence of visual stimulation.

3.2 Comparison of models with neurophysiological data

During scotoma/normal conditioning, a computational simulation of the EXIN model produced the following effects, corresponding closely to the experimental neurophysiological results reported by Pettet & Gilbert (1992) and Darian-Smith & Gilbert (1995):

1. centrifugal expansion of RFs that were initially *inside* the scotoma region;
2. the greatest expansion for RFs closest to the scotoma boundary and inside it;
3. increased response from the area of the initial RF (DeAngelis et al., 1994) without changes in spontaneous activity;
4. RF contraction to original size during normal stimulation; and
5. negligible RF contraction in the absence of stimulation.

The adaptation model (Xing & Gerstein, 1994) produced several effects similar to those produced by the EXIN model (compare Figure 1(a) with 1(c) and Figure 2(a) with 2(c)). However, the adaptation model produced the following effects inconsistent with the EXIN model and neurophysiological data: (1) changes in spontaneous activity because of adaptation; and (2) RF changes in the absence of stimulation.

In response to scotoma/normal conditioning, the LISSOM model (Sirosh & Miikkulainen, 1994; Sirosh et al., 1996) with only lateral inhibitory learning produced the following effects inconsistent with neurophysiological data: (1) centripetal expansion of RFs that were initially *outside* the scotoma region; and (2) the greatest expansion for RFs closest to the scotoma boundary and outside it.

4 Summary and conclusions

In scotoma/normal conditioning, the three models reproduce the neurophysiological experimental data to varying degrees; the EXIN model is closest to experimental data, followed by the adaptation model and then the LISSOM model.

The adaptation model differs from the EXIN model in its responses to blank stimuli and change in spontaneous neural activation after artificial scotoma conditioning. The LISSOM model with only lateral inhibitory learning and the EXIN model with only lateral inhibitory learning differ in the locus and direction of the effects of artificial scotoma conditioning.

Simulating the EXIN model and the LISSOM model with only lateral inhibitory learning shows that the EXIN *outstar* inhibitory learning rule is *sufficient* and the LISSOM *instar* inhibitory learning rule is *insufficient* to model RF expansion during scotoma conditioning. Thus, in the LISSOM model, other learning, such as feedforward learning, is necessary and should be faster than the inhibitory learning to overcome the opposite effects of the inhibitory learning.

The role of feedforward excitatory learning rule in producing fast (on the order of minutes or hours) RF changes in adult animals may be very limited. Restricted retinal lesion in cats produced RF changes in area 17 neurons within hours *only* if the non-lesioned eye was *closed* (Chino et al., 1992). This result is contrary to the prediction of a model with fast *instar* feedforward excitatory learning rule, because active neurons would weaken their connections from the lesioned region, regardless of whether the other eye is open or closed, to produce RF shifts. Furthermore, fast *instar* feedforward excitatory learning rule predicts that the active neurons would weaken their connections from the closed eye.

The validity of the EXIN and LISSOM lateral inhibitory rules can be tested by intracellularly inactivating a test neuron during whole field stimulation with bars. In this situation, the feedforward weights would remain close to their equilibrium values in both models. The EXIN outstar inhibitory rule predicts that after conditioning the test neuron would show increased responsiveness and an RF expansion, because the inhibitory weights from the active neurons to the test neuron decrease. On the other, hand the LISSOM inhibitory rule would cause the lateral

inhibition from the test neuron to the active neurons to decrease, resulting in an decrease in the responsiveness and RF size of the test neuron. A similar conditioning experiment in *Aplysia* showed increased responsiveness of the test neuron (Carew et al., 1984).

These modeling results reveal some aspects of the functional organization and the plausible rules underlying cortical dynamic RFs and point to new experiments to validate the proposed rules.

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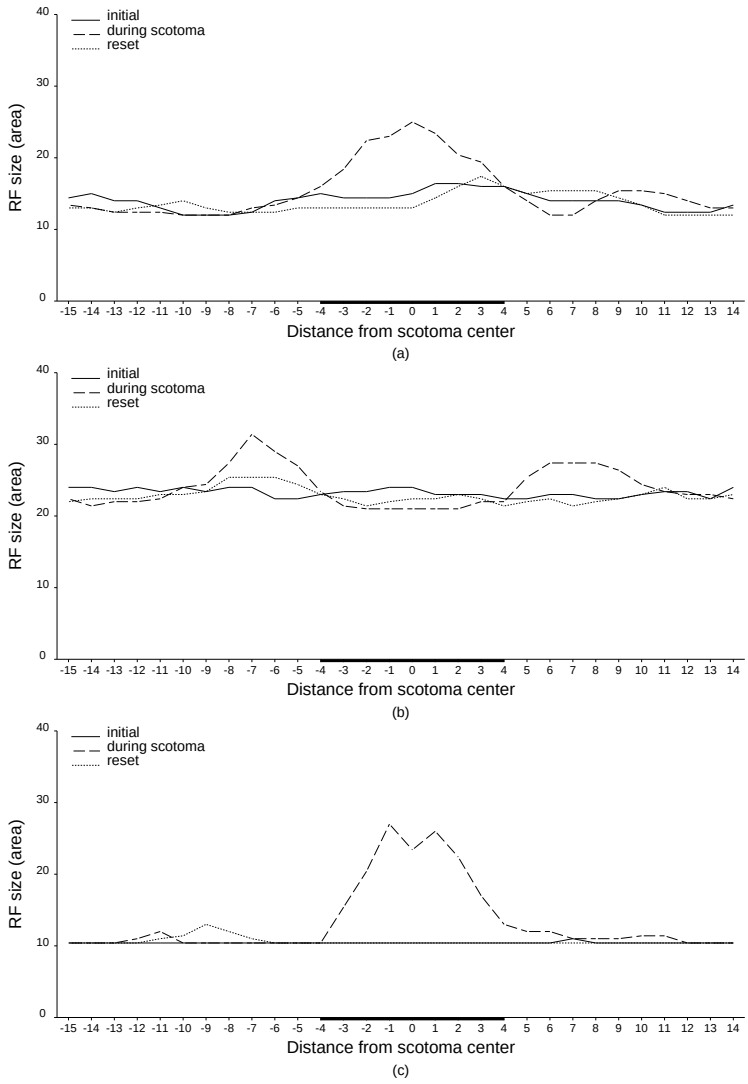

Figure 1: **RF size as a function of position.** The RF area before scotoma conditioning (solid line), after scotoma conditioning (dashed line), and after re-conditioning with normal stimuli (dotted line) in the EXIN network (a), the LISSOM network (b), and the adaptation network (c), are shown as a function of the position of Layer 2 neurons relative to the scotoma center $(15, 15)$. The scotoma is a square of size 9×9. In the graph, the scotoma edges are at 4 and -4. The RF area shown is for a one-dimensional cross-section through Layer 2: neurons $(15, 0)$–$(15, 29)$. The RF area of a Layer 2 neuron is the number of locations at which the test stimulus evokes a response in the Layer 2 neuron. The thick line on the abscissa represents the scotoma region.

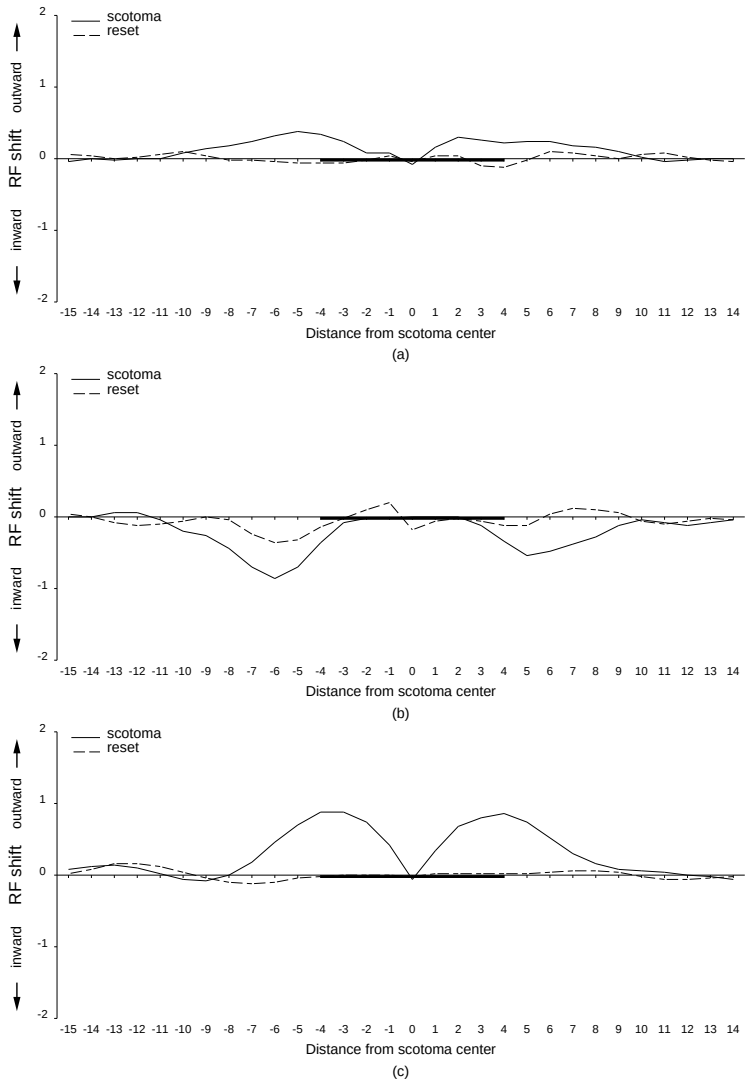

Figure 2: **RF shift as function of position.** Shift in RF center after scotoma conditioning (solid line) and after re-conditioning with normal stimuli (dashed line) with respect to the initial RF centers is shown as a function of distance of Layer 2 neurons from scotoma center for the EXIN network (a), the LISSOM network (b), and the adaptation network (c) as in Figure 1. The RF shift shown is for a one-dimensional cross-section through Layer 2: neurons $(15, 0)$–$(15, 29)$. Positive and negative shifts represent a shift away from and toward the center of the scotoma, respectively. The RF center of a Layer 2 neuron is the center of moment of the neuron's responsiveness to input at different positions within its RF.

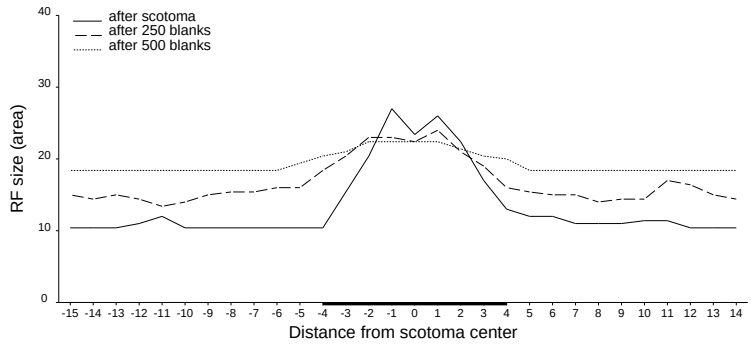

Figure 3: **Blank screen causes RF changes in the adaptation model.** The RF area after scotoma conditioning (solid line), after 250 steps of conditioning with blank stimuli (dashed line), and after 500 steps of conditioning with blank stimuli (dotted line) in the adaptation network, are shown as a function of the position of Layer 2 neurons relative to the scotoma center $(15, 15)$. See Figure 1 for conventions.