Models of receptive field dynamics in visual cortex

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Abstract

The position, size, and shape of the receptive field (RF) of some cortical neurons change dynamically, in response to artificial scotoma conditioning (Pettet & Gilbert, 1992) and to retinal lesions (Chino et al., 1992; Darian-Smith & Gilbert, 1995) in adult animals. The RF dynamics are of interest because they show how visual systems may adaptively overcome damage (from lesions, scotomas, or other failures), may enhance processing efficiency by altering RF coverage in response to visual demand, and may perform perceptual learning.

This paper presents an afferent excitatory synaptic plasticity rule and a lateral inhibitory synaptic plasticity rule – the EXIN rules (Marshall, 1995a) – to model persistent RF changes after artificial scotoma conditioning and retinal lesions. The EXIN model is compared to the LISSOM model (Sirosh et al., 1996) and to a neuronal adaptation model (Xing & Gerstein, 1994). The rules within each model are isolated and are analyzed independently, to elucidate their roles in adult cortical RF dynamics. Based on computer simulations, the EXIN lateral inhibitory synaptic plasticity rule and the LISSOM lateral excitatory synaptic plasticity rule produced the best fit with current neurophysiological data on visual cortical plasticity in adult animals (Chino et al., 1992, Darian-Smith & Gilbert, 1995; Pettet & Gilbert, 1992) including (1) the retinal position of the expanding RFs, (2) the amount of change in spontaneous activation in the absence of any visual stimulation, (3) the corticotopic direction in which responsiveness returns to lesioned cortex, (4) the direction of RF shifts, (5) the amount of change in response to blank stimuli, and (6) the lack of dynamic RF changes during conditioning with a retinal lesion in one eye and the unlesioned eye kept open, in adult animals. The effects of the LISSOM lateral inhibitory synaptic plasticity rule during artificial scotoma conditioning are in conflict with those of the other two LISSOM synaptic plasticity rules. A novel “complementary scotoma” conditioning experiment, in which stimulation of two complementary regions of visual space alternates repeatedly, is proposed to differentiate the predictions of the EXIN and LISSOM rules.

Keywords: Artificial scotoma and retinal lesions, cortical plasticity, self-organization, neural

adaptation, EXIN (excitatory + inhibitory) learning.

1 Introduction

In experiments using artificial scotoma conditioning (Pettet & Gilbert, 1992) and retinal lesions (Chino et al., 1992; Darian-Smith & Gilbert, 1995), neurons in primary visual cortex corresponding to a particular region of visual space were deprived of visual stimulation, while 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 of the neurons. For example, after 15 minutes of artificial scotoma conditioning, the RF area of some cortical neurons whose RF was located inside the scotoma expanded by a factor of five; after 15 minutes of subsequent normal stimulation, the RF returned to its original size (Pettet & Gilbert, 1992). The dynamic RF expansion following artificial scotoma conditioning in one eye also transfers to the other eye (Volchan & Gilbert, 1994).

Pettet and Gilbert (1992) simulated a retinal lesion experiment by presenting a pattern of moving lines in the visual field while masking out an artificial “scotoma” region covering the original RF of the recorded neuron. After 10–15 minutes of stimulation, a five-fold average expansion in RF area was found. In the second phase of the experiment, the scotoma region was unmasked, and moving lines were presented in the whole field. After several minutes of stimulation, the RF shrank back to its original extent. The RF expansion and contraction was repeatable. A key observation was that stimulation exclusively in the surrounding region was necessary for the RF expansion to occur. Exposure to a blank screen for as long as 20 minutes had little effect on the RF size.

Pettet and Gilbert (1992) tested the effect of orientation of the conditioning stimuli on RF expansion, during artificial scotoma conditioning. For a few neurons (3 out of 15), they found an expansion with iso-orientation conditioning stimuli and did not find an expansion with the orthogonal pattern. In these cases, the orthogonal pattern actually reduced the RF size and responsiveness of the neuron.

Darian-Smith and Gilbert (1995) studied topographic reorganization in the striate cortex of the adult cat and monkey after binocular retinal lesions, using physiological and anatomical techniques. They found that immediately (between 5 minutes and up to 1 hour) after making

corresponding retinal lesions of 3.5° – 14° in diameter, there was a cortical scotoma region containing neurons whose RF was located more than 0.5–1.0 mm inside the initial scotoma boundary. However, cortical neurons located close to or just inside the cortical scotoma boundary showed an increase in RF size. The greatest expansion occurred for neurons whose RF was located closest to the scotoma boundary. In addition, the expanded RFs shifted centrifugally toward the outside of the scotoma. Neurons that acquired responsiveness to locations outside the scotoma, i.e., neurons in the recovered region of the original cortical scotoma, were less responsive, more sluggish in their response, and more easily fatigued compared to those in normal cortex or in cortex located more than 1 mm outside the cortical scotoma boundary. In spite of the changes in their RF size and position, these neurons retained some of their original RF properties, such as directionality, orientation specificity, and binocularity.

Measurements within the same cortex 2–12 months after the lesions showed that cortical neurons located several millimeters inside the original boundary of the cortical scotoma became responsive to stimulation of perilesion retina (Darian-Smith & Gilbert, 1995). Over time, function returned to the cortex in a roughly concentric inward direction. The cortical reorganization was accompanied by RF shifts. In spite of distortions in representation, topographic order was maintained.

In contrast to the cortical reorganization, the LGN scotoma and thalamocortical afferents did not undergo any change, as reflected in electrophysiological recordings and anatomical studies. This led Darian-Smith and Gilbert to conclude that the reorganization of cortical topography following retinal lesions originates in the cortex and is likely to be mediated, at least in part, by the long-range collaterals of cortical neurons rather than by thalamocortical afferents.

1.1 Significance of RF dynamics

The dynamics of RFs 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 (Das, 1997; Gilbert, 1998). Dynamic visual RFs might also be related to the dynamic response properties found in other cortical areas (Das, 1997), such as the tactile RF expansion/contraction found in adult somatosensory cortex in

response to intracortical microstimulation (Recanzone, Merzenich, & Dinse, 1992) and localized peripheral stimulation (Recanzone, Merzenich, Jenkins, Grajski, & Dinse, 1992).

Artificial scotoma conditioning can elucidate the neural basis of *perceptual learning*. In perceptual learning, human observers improve their performance in perceptual tasks such as orientation perception (Fiorentini & Berardi, 1980), vernier acuity (Fahle & Edelman, 1993), and discrimination of texture (Karni & Sagi, 1991) after training (repeated performance of a perceptual task). Perceptual learning is stable: it does not wear off after periods without visual stimulation. Furthermore, in these studies perceptual learning was not simply a matter of becoming accustomed to the perceptual task. Perceptual learning was specific for features of the training stimuli (Crist et al., 1997; Fahle, 1997); it was usually confined to the portion of the retina that was stimulated during training, or the improvement was restricted to the orientation of the training stimuli. Since neurons in the visual cortex are selective for specific stimulus features, repeated presentation of training stimuli repeatedly activates a small group of neurons. Thus, perceptual learning may be realized by cortical plasticity that depends on repeated activation of a group of neurons. In artificial scotoma conditioning, visual cortical neurons selective for a particular region of visual space are deprived of visual stimulation while neurons selective for surrounding visual space receive stimulation. Thus, artificial scotoma conditioning provides a systematic procedure to control the activation of specific groups of neurons and to study the neural basis of perceptual learning.

1.2 Modeling of RF dynamics

Figure 1 shows the input pathways to primary visual cortical neurons. The RF of a cortical neuron can be affected by changes in the inputs to the neuron. The long-range horizontal excitatory or inhibitory pathways in visual cortex have been regarded as the substrate for RF dynamics (Darian-Smith & Gilbert, 1995; Das & Gilbert, 1995ab; Gilbert et al., 1996; Pettet & Gilbert, 1992; Somogyi & Martin, 1985; Volchan & Gilbert, 1994). It has been shown that dynamic RF changes result from changes in the amount of excitation and/or inhibition received by the neurons (Chapman & Stone, 1996; Petersen & Taylor, 1997). Changes in the amount of excitation and/or inhibition to neurons can result from neuronal adaptation

(DeAngelis et al., 1995; Xing & Gerstein, 1994), short-term inhibitory synaptic adaptation (Todorov et al., 1997), long-term synaptic modifications in long-range horizontal pathways (Darian-Smith & Gilbert, 1994, 1995; Das & Gilbert, 1995ab; Gilbert et al., 1996; Pettet & Gilbert, 1992), long-term synaptic plasticity in lateral inhibitory pathways (Marshall & Kalarickal, 1997), or long-term synaptic plasticity in afferent excitatory pathways (Marshall, 1995a; Sirosh et al., 1996). Reafferent feedback pathways between cortical layers may also be involved in producing cortical plasticity (Gilbert, 1996).

Figure 1 near here.

A drawback of adaptation-based models (Todorov et al., 1997; Xing & Gerstein, 1994) is that they cannot sustain the RF expansions during periods with no visual stimulation in artificial scotoma conditioning as reported by Pettet and Gilbert (1992). Sirosh et al. (1996) attributed RF expansion after artificial scotoma conditioning to afferent excitatory plasticity in their LISSOM model. Their model also had plasticity in lateral excitatory and lateral inhibitory pathways, but the role of these forms of plasticity in RF changes after artificial scotoma conditioning was not analyzed.

This paper compares the properties of RF changes produced by several models of cortical plasticity to the neurobiological data on RF changes after artificial scotoma conditioning and after retinal lesions in adult animals. In particular, RF changes after artificial scotoma conditioning and retinal lesions produced by the EXIN rules (Marshall, 1990a, 1995a), the LISSOM rules (Sirosh & Miikkulainen, 1994ab, 1995, 1997; Sirosh et al., 1996), and an adaptation rule (Xing & Gerstein, 1994) are analyzed.

1.2.1 EXIN plasticity rules

The EXIN model uses an an instar Hebbian afferent excitatory synaptic plasticity rule and an outstar anti-Hebbian lateral inhibitory synaptic plasticity rule (Marshall, 1990a, 1995a). An instar rule is enabled when the postsynaptic neuron is activated, and excitatory pathways *into* the neuron undergo synaptic plasticity (Grossberg, 1972, 1976ab), whereas an outstar rule is enabled when the presynaptic neuron or presynaptic element is activated, and excitatory

pathways *out* of the neuron or the presynaptic element undergo synaptic plasticity (Grossberg, 1976c). This subtle distinction makes dramatic difference in the plasticity and behavior of the neural circuits (Grossberg, 1976abc; Marshall, 1995a).

In EXIN networks, the instar excitatory synaptic plasticity rule modifies the weights of afferent excitatory pathways to active neurons so that the active neurons become more responsive to the currently presented input pattern. The instar excitatory synaptic plasticity rule is responsible for the development of the broad excitatory RF of the neurons. The development of weights of lateral inhibitory pathways according to the outstar lateral inhibitory synaptic plasticity rule ensures that different neurons become selective to different input patterns. Yet, if the input environment contains several similar patterns, the outstar lateral inhibitory synaptic plasticity rule causes strong lateral inhibitory pathways to develop between neurons selective for the similar input patterns, thereby producing high discrimination. In EXIN networks, lateral inhibitory pathways from often-activated neurons to unresponsive neurons weaken, thereby making the unresponsive neurons more likely to respond to some input. The outstar lateral inhibitory synaptic plasticity rule is responsible for dispersion of neuronal selectivity and sharpening of the RF of the neurons. The EXIN rules develop efficient representation of input patterns according to their distribution in an input environment. The EXIN rules self-organize networks capable of representing multiple superimposed patterns, ambiguous patterns, overlapping patterns at different scales, and contextually constrained patterns starting from completely nonspecific afferent excitatory and lateral inhibitory pathway weights (Marshall, 1995a).

1.2.2 LISSOM plasticity rules

The LISSOM model (Sirosh & Miikkulainen, 1994b) uses instar Hebbian afferent excitatory and lateral excitatory synaptic plasticity rules, and an instar anti-Hebbian lateral inhibitory synaptic plasticity rule. Unlike the EXIN model, the LISSOM model has modifiable lateral excitatory pathways and uses an instar lateral inhibitory synaptic plasticity rule. However, like the EXIN rules, the LISSOM rules produce a sparse, distributed coding that reduces redundancies (Marshall, 1995a; Marshall & Gupta, 1998; Sirosh et al., 1996). Lateral excitatory pathways in

the LISSOM model help the development of a topographic RF arrangement. The LISSOM lateral excitatory and inhibitory synaptic plasticity rules cause highly specific lateral pathway connectivity to develop between neurons that have similar RF properties (Sirosh & Miikkulainen, 1997).

1.2.3 Adaptation rules

In adaptation models (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.

1.3 Significance and contributions of this paper

This paper analyzes the role of each rule individually. The EXIN rules and the LISSOM rules have been used to model development of cortical properties and functions in young animals. Studies on young animals in their critical periods show that the afferent and lateral pathway connectivity in the primary visual cortex are modified by changes in the visual environment (Hubel & Wiesel, 1965, 1970; Hubel et al., 1977; Katz & Callaway, 1992). However, the neural basis of adult cortical plasticity, e.g., RF changes after artificial scotoma conditioning and retinal lesions in adult animals, is not known. Thus, the rules in the EXIN model, the LISSOM model, and the adaptation models are studied individually to determine whether they produce effects consistent with the experimentally observed RF changes after artificial scotoma conditioning and retinal lesions in adult animals. The possible effects of the full EXIN and the full LISSOM model are also discussed. The simulation results based on individual rules serve as predictions for the effects of artificial scotoma conditioning and retinal lesions in adult animals in the presence of pharmacological agents that block plasticity in specific pathways, e.g., NMDA receptor antagonists which block plasticity in excitatory pathways (Kirkwood et al., 1993). The simulations show differences in two plausible rules for plasticity in lateral inhibitory pathways (the outstar EXIN lateral inhibitory rule and the instar LISSOM lateral inhibitory rule) in the context of artificial scotoma conditioning and retinal lesions. A novel experiment is suggested to further differentiate between the rules.

In this paper, experimental data on cortical effects of artificial scotoma conditioning and retinal lesions are used to constrain plausible rules for dynamic RF changes. In particular,

1. the paper analyzes the effects of an *instar* and an *outstar* lateral inhibitory synaptic plasticity rule during scotoma conditioning;
2. the effects of different plausible ways of modifying *effective* inhibition to neurons during scotoma conditioning, e.g. due to changes in lateral excitatory or afferent excitatory pathway strength, or neuronal adaptation, are studied;
3. some of the possible rules that *could* produce dynamic RF changes are eliminated based on comparison with experimental data;
4. the paper shows that the EXIN outstar lateral inhibitory synaptic plasticity rule and the LISSOM instar lateral excitatory synaptic plasticity rule are sufficient to account for most of the data on artificial scotoma conditioning and on the short-term effects of retinal lesions (the effects of orientation selectivity on RF dynamics during artificial scotoma were not simulated);
5. the sufficiency of the EXIN lateral inhibitory synaptic plasticity rule in producing RF changes after artificial scotoma conditioning and after retinal lesions in adult animals provides indirect evidence for the existence of plasticity in lateral inhibitory pathways and predicts characteristics of inhibitory synaptic plasticity in cortex; and
6. a novel experiment, *complementary scotoma conditioning*, is proposed to distinguish the effects caused by neuronal adaptation from those caused by synaptic plasticity.

2 Methods

2.1 Network simulation organization

The architecture used for the simulations is a two-layered neural network with afferent and lateral connections, corresponding to parts of subcortex and primary visual cortex. A patch of neurons in the primary visual cortex, 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 the computer simulations, Layer 1 (corresponding to LGN processing) and Layer 2 (corresponding to early laminae of primary visual cortex) each had a 30×30 array of neurons. For ease of simulation, the initial afferent pathway weight and lateral pathway weight distributions in the simulations are chosen to be spatially isotropic. Furthermore, the input feature to these networks is an isotropic Gaussian blob (see Section 2.2), which when used to train the networks produces spatially isotropic receptive fields. However, isotropic RFs are not essential for these networks to produce changes in RF properties during the various forms of input conditioning. The EXIN and LISSOM learning rules are competitive learning rules and produce orientation selective neurons if the input features are oriented (Marshall, 1990d; Sirosh et al., 1996); scotoma conditioning using oriented features would affect the networks as described in Sections 2.5.4 and 2.6.3. The adaptation networks are based on neuronal adaptation, without synaptic plasticity. The weights in the adaptation networks can be assigned to produce orientation selective neurons. The adaptation of the orientation selective neurons during artificial scotoma conditioning will produce RF expansions because of differences in adaptation levels as explained in Section 2.7.2.

Following Xing and Gerstein (1994), orientation selectivity is not built into the simulations. This is a gross simplification, as it discounts the effects of neurons selective to other orientations on dynamic RF changes. The simplification of representing only iso-orientation selective neurons in Layer 2 is partially justified by observing that the RF expansion was more pronounced and robust during conditioning with iso-orientation patterns than during conditioning with ortho-orientation patterns (Pettet & Gilbert, 1992). In the discussion section, a mechanism for orientation selectivity is described which may model the influence of neurons with other orientation selectivity on dynamic RF properties.

The following symbols are used to refer to the various entities of the network. The indices (i, j) and (k, l) are used to refer to Layer 1 neurons, and (p, q) , (r, s) , and (u, v) refer to Layer 2 neurons, where $i, j, k, l, p, q, r, s, u, v \in \{0, \dots, 29\}$. These indices also represent the retinotopic coordinates of the neurons’ RF. The weight of the afferent excitatory connection pathway from a

Layer 1 neuron (i, j) to a Layer 2 neuron (p, q) is denoted by $Z_{ij,pq}^+(t)$. The weight of the lateral inhibitory connection pathway and the weight of the lateral excitatory connection pathway from Layer 2 neuron (p, q) to Layer 2 neuron (r, s) are represented by $Z_{pq,rs}^-(t)$ and $Z_{pq,rs}^+(t)$, respectively. These pathway weight values may represent the effect of a monosynaptic connection, the total effect of a polysynaptic chain of connections (see Section 5.7), or the population effect of multiple direct synapses. The activation levels (mean spike rate) over time of Layer 1 neuron (i, j) and Layer 2 neuron (p, q) are represented by $x_{ij}(t)$ and $x_{pq}(t)$, respectively.

2.2 The inputs

The inputs to Layer 1 were obtained as follows. First, two-dimensional 30×30 images were convolved with a Gaussian kernel, K , with toroidal wraparound. The input stimulus at each position in the images could be 0 or 1. The input at each position took value 1 with probability Φ during a given simulation step. After convolution, the resultant image was normalized by the maximum intensity value in the image. In the simulations, the mean of the normalization factor was 1.68, with a standard deviation of 0.34, over 10,000 inputs. The resultant images from the normalization stage were the inputs to Layer 1. These inputs to Layer 1 are called normal stimuli.

In artificial scotoma conditioning, random stimulation in the whole visual field except in a masked region was followed by whole-field random stimulation. To simulate inputs with a scotoma, images with a scotoma were convolved with the kernel K and then normalized. Input stimuli at positions outside the scotoma region had probability Φ of being assigned value 1, and input stimuli inside the scotoma region had value 0. These inputs are called scotoma stimuli. The term “cortical scotoma” refers to the silenced region in Layer 2 as a result of using retinal scotoma stimuli (Xing & Gerstein, 1994; Darian-Smith & Gilbert, 1995).

2.3 Simulation procedure

In the simulations, the experimental paradigm of Pettet and Gilbert (1992) was followed. The original RF was determined after a period of random whole-field stimulation. In all the simulations, the initial whole-field stimulation was continued until the sum of the magnitude of individual weight changes after 100 training steps had reached an asymptote. Then the RF was

again measured after conditioning with the artificial scotoma. To determine reversibility of RF changes, the RF was measured again after whole-field stimulation.

2.4 RF measurements

The RF was mapped using single-point stimulation, blurred with the Gaussian kernel K , at all input positions (i, j) . The RF of a Layer 2 neuron (p, q) is defined as the collection of positions (i, j) at which the test input causes the activation level x_{pq} to exceed a threshold Θ .

2.5 The EXIN model

The EXIN model (Figure 2) combines an instar afferent EXcitatory synaptic plasticity rule and an outstar lateral INhibitory synaptic plasticity rule. The EXIN synaptic plasticity rules change the weights as a function of the input stimuli so that different neurons become selective for different input patterns and every input pattern is represented by a sparse output pattern (Marshall, 1995a). In scotoma conditioning, a subset of the visual input is removed, and therefore neurons previously selective for these inputs are not stimulated. The EXIN rules change the weights so that the unstimulated neurons become responsive to different input patterns, resulting in changes in their RFs.

Figure 2 near here.

2.5.1 The EXIN lateral inhibitory synaptic plasticity rule

The lateral inhibitory weights, $Z_{pq,rs}^-$, are modified according to the anti-Hebbian rule

$$\frac{d}{dt}Z_{pq,rs}^- = \delta \mathcal{G}(x_{pq}) \left(-Z_{pq,rs}^- + \mathcal{Q}(x_{rs}) \right) \quad (1)$$

(Marshall, 1995a), where $\delta > 0$ is a small learning rate constant and $\mathcal{G}$ and $\mathcal{Q}$ are half-rectified non-decreasing functions. Thus, *whenever a neuron is active, its output inhibitory connections to other active neurons tend to become slightly stronger (i.e., more inhibitory), while its output inhibitory connections to inactive neurons tend to become slightly weaker*. Neuron activations remain within $[-C, B]$ according to a shunting equation (Section 2.5.5) based on the Hodgkin model (Hodgkin, 1964); this causes the weight values to remain bounded as well, because

according to Equation 1, $Z_{pq,rs}^-(t) \in [0, Q(B)]$ for $t \geq 0$, if $Z_{pq,rs}^-(0) \in [0, Q(B)]$ (Grossberg, 1982). The weight change in Equation 1 approaches zero as $Z_{pq,rs}^-$ approaches $Q(x_{rs})$, the weight change is positive when $Z_{pq,rs}^- < Q(x_{rs})$, and the weight change is negative when $Z_{pq,rs}^- > Q(x_{rs})$. The weight change approaches zero as $Z_{pq,rs}$ approaches $Q(x_{rs})$.

In an outstar synaptic plasticity rule (Grossberg, 1972), *presynaptic* activity “enables” the plasticity at a synapse; when the plasticity is enabled, the weight tends to become proportional to the postsynaptic activity. In an instar synaptic plasticity rule, *postsynaptic* activity enables the plasticity; when the plasticity is enabled, the weight tends to become proportional to the presynaptic activity. Thus, to make Equation 1 into an instar rule, x_{pq} and x_{rs} would be interchanged.

An effect of the EXIN inhibitory synaptic plasticity rule is that if two neurons are frequently coactivated, then the lateral inhibitory weights between them become strong. If two neurons are only rarely coactivated then their reciprocal lateral inhibitory weights become weak. Strong lateral inhibition between two neurons tends to make them less likely to be coactivated, causing the two to become selective to different inputs according to the excitatory synaptic plasticity rule (Section 2.5.2). Thus, when the network is exposed to normal stimuli, the lateral inhibitory weights and the excitatory afferent weights are modified so that each neuron becomes selective to different inputs and the RFs of all Layer 2 neurons cover the input space (Marshall, 1995a; Marshall & Gupta, 1998). This leads to improved discrimination and sparse coding (Marshall, 1995a).

2.5.2 The EXIN afferent excitatory synaptic plasticity rule

The afferent excitatory weight changes are governed by a variant of a Hebbian learning rule. The rule can be expressed (Grossberg, 1982; Marshall, 1995a) as

$$\frac{d}{dt}Z_{ij,pq}^+ = \epsilon \mathcal{F}(x_{pq}) \left(-Z_{ij,pq}^+ + \mathcal{H}(x_{ij}) \right), \quad (2)$$

where $\epsilon > 0$ is a small learning rate constant, and $\mathcal{F}$ and $\mathcal{H}$ are half-rectified non-decreasing functions.

Thus, *whenever a neuron is active, its input excitatory connections from active neurons tend to become slightly stronger, while its input excitatory connections from inactive neurons tend to*

become slightly weaker. As discussed for Equation 1, the weight values in Equation 2 remain bounded.

The EXIN excitatory synaptic plasticity rule is an instar competitive learning rule. When used in conjunction with strong lateral inhibition, it causes model cortical neurons to become selective for a specific pattern of input activations (Marshall, 1995a).

2.5.3 Stability of EXIN networks

Like other competitive learning rules, the EXIN rules do not produce absolutely stable synaptic weights. The stability of the network depends on the input environment. If the input distribution changes for a sufficiently long time, the weights change to encode the new statistics. Such instability, reflecting the statistics of the input environment is advantageous at lower-levels of cortical processing; e.g., the cortex can reorganize after cortical or peripheral damage.

The learning rates in competitive learning networks must be kept small enough to allow approximate stability in a statistically stationary input environment, yet large enough to allow plasticity in response to the statistical changes posed by perturbations such as scotomas. Stability in competitive learning networks and the various learning parameters are discussed in the Appendix (Section 6.1.2).

2.5.4 Explanation of dynamic RF changes based on the EXIN rules

The maximum extent of a Layer 2 neuron's RF is limited by the axonal arborization spread of the Layer 1 neurons from which it receives afferent excitation. In addition, a Layer 2 neuron receives lateral inhibition from neurons with which it is frequently co-excited. Neurons in Layer 2 can be consistently co-excited if they share inputs from common Layer 1 neurons. Because of inhibition, it is possible that a Layer 2 neuron (p, q) does not become active in response to some active Layer 1 neuron (i, j) , even though $Z_{ij,pq}^+ > 0$.

The role of the EXIN afferent excitatory and lateral inhibitory synaptic plasticity rules in producing RF changes during scotoma conditioning are studied independently.

Role of EXIN lateral inhibitory synaptic plasticity. Consider the EXIN network with the afferent excitatory synaptic plasticity rule disabled and the lateral inhibitory synaptic plasticity

rule enabled. When the network is exposed to scotoma stimuli with a sufficiently large scotoma, there exists a cortical scotoma region in Layer 2. During scotoma conditioning, neurons outside the cortical scotoma region are active and those inside the cortical scotoma region are not. Let neuron (p, q) be outside and neuron (r, s) be inside the initial cortical scotoma region (Figure 3a). Assume that after conditioning with normal stimuli, $Z_{pq,rs}^-$ and $Z_{rs,pq}^-$ are not zero, because neurons (p, q) and (r, s) share common afferent inputs. If for a given stimulus, neuron (p, q) is active, then according to the EXIN inhibitory synaptic plasticity rule, $Z_{pq,rs}^-$ weakens (Figure 3b). However, $Z_{rs,pq}^-$ is unchanged, since x_{rs} is zero. Thus, lateral inhibitory weights to neuron (r, s) from active neurons outside the cortical scotoma region weaken, but lateral inhibitory weights from neuron (r, s) are unaffected. The net effect, from the point of view of neuron (r, s) , is that its afferent excitatory input weights remain unchanged and its input lateral inhibitory weights from neurons outside the cortical scotoma region weaken (if nonzero before the conditioning). If the RF of neuron (r, s) is measured then, it will be more responsive to positions in its old RF and will be responsive to some new positions too (Figure 3b), thus producing RF expansion. The simulation results are presented in Section 3.1.

Figure 3 near here.

When the network is again conditioned with normal stimuli, the asymmetric lateral inhibitory weights between the neurons inside and outside the cortical scotoma region regain symmetry. Thus, the RFs of neurons in the cortical scotoma are restored.

Role of EXIN afferent excitatory synaptic plasticity. Now consider the EXIN network with the lateral inhibitory synaptic plasticity rule disabled and the afferent excitatory synaptic plasticity rule enabled. Neurons close to the edge of the cortical scotoma but outside it show some interesting changes (e.g., neuron (p, q) in Figure 3c). Because these neurons are near the cortical scotoma edge, the region of their RF inside the scotoma is not stimulated. Thus, because of the EXIN excitatory synaptic plasticity rule, afferent excitatory connections from Layer 1 neurons in the scotoma region to this neuron become weaker. Hence its RF shrinks, and the center of its RF shifts outward. During RF measurement after scotoma conditioning, these neurons (e.g., (p, q)) respond only weakly to stimuli in the scotoma region and hence exert less inhibition on neurons

that were inactive during conditioning (e.g., (r, s)). Thus, the neurons that were inactive during the conditioning show increased responsiveness and RF expansion. This explanation for RF expansion during scotoma conditioning was proposed by Sirosh et al. (1996). The RF expansion causes the RF centers to shift away from the scotoma center (Section 3.2).

When the EXIN network with only afferent excitatory synaptic plasticity enabled is conditioned again using normal stimuli, the neurons do not recover their original RFs. The neurons whose afferent excitatory pathways from the scotoma region were weakened are weakly activated by stimulation of the region that was occluded during artificial scotoma conditioning, during the subsequent whole-field stimulation. On the other hand, the neurons whose initial RF was inside the scotoma region during artificial scotoma conditioning are more responsive to positions inside the scotoma during whole-field stimulation, following artificial scotoma conditioning. Thus, during whole-field stimulation following artificial scotoma conditioning, the neurons whose initial RF was inside the scotoma region strengthen afferent excitatory pathways from positions inside the scotoma region to which they are more responsive and exert greater inhibition on neurons whose initial RF straddled the scotoma boundary. The neurons whose RF straddled the scotoma boundary further weaken afferent excitatory pathways from positions inside the scotoma region to which they have become less responsive after artificial scotoma conditioning, as their responsiveness to positions inside the scotoma region is further suppressed. With strong fixed lateral inhibitory pathways weights, the lateral inhibition between neurons suppresses the activation of the neurons whose RF size was decreased (because of weakening of pathways from the scotoma during scotoma conditioning) when inputs are presented at positions inside the scotoma. According to the EXIN afferent excitatory synaptic plasticity rule, afferent pathway weights change only when postsynaptic neurons are active. Thus, the weak afferent connections from the scotoma region to the neurons whose RF size was decreased are not strengthened, and the RFs of these neurons do not shift back to their original positions (Section 3.2).

In the EXIN network (with either lateral inhibitory or afferent excitatory synaptic plasticity alone), in response to scotoma stimuli, all neurons that show RF expansion belong to the set of neurons that were inside the initial cortical scotoma. Thus, after scotoma conditioning, the

EXIN network with both lateral inhibitory and afferent excitatory synaptic plasticity will produce RF expansion in neurons that were inside the initial cortical scotoma.

2.5.5 The activation equation

The activation level x_{pq} of each Layer 2 neuron is governed by a shunting equation (Grossberg, 1972) based on the Hodgkin (Hodgkin, 1964) model:

$$\frac{d}{dt}x_{pq} = -Ax_{pq} + \beta(B - x_{pq})E_{pq} - \gamma(C + x_{pq})I_{pq}, \quad (3)$$

where A, B, C, β , and γ are constants, and E_{pq} and I_{pq} represent respectively the neuron's total afferent excitatory and lateral inhibitory input signals. Because Equation 3 is a shunting equation, if $x_{pq}(0) \in [-C, B]$ then $x_{pq}(t) \in [-C, B]$ for all time $t \geq 0$ (Cohen & Grossberg, 1983). Thus, activation levels are forced to remain within a bounded range, between $-C$ and B . The total input excitation E_{pq} is defined as

$$E_{pq} = \left(\sum_{ij} [x_{ij}] Z_{ij,pq}^+ \right)^2, \quad (4)$$

and the total input inhibition I_{pq} is given by

$$I_{pq} = \sum_{rs} [x_{rs}] Z_{rs,pq}^-, \quad (5)$$

where $[a] \equiv \max(a, 0)$. Parameters β and γ , respectively, control the effectiveness of the excitation and inhibition received by a Layer 2 neuron. The squaring in Equation 4 sharpens the RF profile of the Layer 2 neurons; squaring enhances excitation to Layer 2 neurons when $\sum_{rs} [x_{rs}] Z_{rs,pq}^- > 1$ and suppresses excitation to Layer 2 neurons when $\sum_{rs} [x_{rs}] Z_{rs,pq}^- < 1$.

2.5.6 Stability of the shunting equation: Cohen-Grossberg theorem

The shunting equation (Equation 3) with $Z_{rs,pq}^- = Z_{pq,rs}^- \geq 0$, belongs to a class of competitive dynamical systems that are absolutely stable; i.e., the system has fixed points for any choice of parameters (Cohen & Grossberg, 1983). The neuronal activations in such a system are guaranteed to reach stable equilibrium values for all synaptic weight values with the restriction.

However, it is not known whether the shunting equation remains absolutely stable even when $Z_{rs,pq}^- \neq Z_{pq,rs}^- \geq 0$ for some pairs of neurons. The symmetry of reciprocal pairs of lateral

inhibitory weights is not guaranteed by the EXIN lateral inhibitory synaptic plasticity rule. During normal stimulation, the lateral inhibitory weights are approximately symmetric (Marshall, 1995a). They become asymmetric between neurons across the scotoma boundary during scotoma conditioning. Nevertheless, simulations empirically show the stability of the EXIN network (Section 6.1.1).

2.5.7 The initial weights

In the EXIN simulations, the initial afferent excitatory weight from Layer 1 neuron (i, j) to Layer 2 neuron (p, q) is given by

$$Z_{ij,pq}^+(0) = \left[\Psi \times \exp \left(\frac{-(x^2 + y^2)}{\sigma_{\mathbf{ff}}^2} \right), \Gamma_{\mathbf{ff}} \right], \quad (6)$$

where

$$[a, b] \equiv \begin{cases} a & \text{if } a > b, \\ 0 & \text{otherwise,} \end{cases} \quad (7)$$

$x, y \in \{-15, \dots, 14\}$, $p = ((i + x \bmod 30))$, and $q = ((j + y) \bmod 30)$. The indices i, j, p , and q are in $\{0, \dots, 29\}$. The indices i, j, p , and q and the distances x and y are related by the above equations because the model cortical and thalamic neurons are arranged in a wrapped-around two-dimensional grid. The parameters Ψ , $\sigma_{\mathbf{ff}}$, and $\Gamma_{\mathbf{ff}}$ are positive constants.

The initial lateral inhibitory weights between Layer 2 neurons (p, q) and (r, s) , where $p \neq r$ or $q \neq s$, are set as follows. Let

$$X_{pq,rs} = \sum_{ij} \min(Z_{ij,pq}^+, Z_{ij,rs}^+) \quad (8)$$

and

$$W_{pq,rs}^-(0) = W_{rs,pq}^-(0) = [X_{pq,rs}, \Gamma_{\mathbf{i}}], \quad (9)$$

where $\Gamma_{\mathbf{i}}$ is a constant. Then

$$Z_{pq,rs}^-(0) = Z_{rs,pq}^-(0) = \frac{\Omega \times W_{ps,rs}^-}{\max_{ab,cd \in \text{Layer 2}} W_{ab,cd}^-}, \quad (10)$$

where Ω is a constant. Equation 10 assigns inhibitory weights between two distinct Layer 2 neurons in proportion to the amount of overlap in the RFs of the two neurons.

The initial weight values of the pathways were chosen according to Equations 6–10, instead of completely random weights, to speed the convergence of weight values during subsequent whole field stimulation and to ensure RF topography, thereby avoiding RF shifts and RF size changes caused by RF scatter that may be present when the initial weights are chosen randomly. The networks produced after the whole field stimulation were used for scotoma conditioning simulations.

Neurons do not directly inhibit themselves in the EXIN network; that is, $Z_{pq,pq}^-$ is zero or nonexistent, and $\frac{d}{dt}Z_{pq,pq}^- = 0$.

Lateral excitatory pathways are omitted in this model; all $Z_{pq,rs}^+$ are fixed at zero. This is a simplification based on the assumption that the net effect of the lateral excitatory and inhibitory pathways on excitatory neurons is inhibitory. Partial support for setting $Z_{pq,rs}^+$ to zero comes from the lack of disynaptic excitatory postsynaptic potential due to stimulation of thalamocortical afferents during intracellular recordings in simple neurons of the cat visual cortex (Ferster, 1989) and in layer 5 neurons of adult mice (Gil & Amitai, 1996). Although lateral excitatory pathways exist in the cortex, Weliky et al. (1996) and Gil and Amitai (1996) showed that at high stimulation strengths the long-range horizontal pathways exert overall inhibition on pyramidal neurons. This issue is discussed further in Section 5.7.

2.6 The LISSOM model

The LISSOM model (Sirosh & Miikkulainen, 1994ab, 1995, 1997; Sirosh et al., 1996) uses afferent excitatory, lateral excitatory, and lateral inhibitory synaptic plasticity rules. All three rules are instar rules based on weight normalization. The LISSOM rules produce use-dependent weight changes and thus produce changes in RF properties during artificial scotoma conditioning and lesions.

2.6.1 The LISSOM synaptic plasticity rules

The most significant differences between the LISSOM model and the EXIN model are that LISSOM uses an instar, rather than outstar, inhibitory synaptic plasticity rule, and that LISSOM has used lateral excitatory pathways, in addition to lateral inhibitory and afferent excitatory pathways.

In the LISSOM model (Figure 4), 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 Hebbian synaptic plasticity rule. This rule keeps the sum of the squares of the synaptic weights of the excitatory connections constant, and likewise for the inhibitory connections. After the activations of Layer 2 neurons have stabilized, the weights are modified according to

$$Z_{ab,cd}(t+1) = \frac{Z_{ab,cd}(t) + \xi x_{ab}x_{cd}}{\left(\sum_{ef} (Z_{ef,cd}(t) + \xi x_{ef}x_{cd})^2\right)^{\frac{1}{2}}} \quad (11)$$

(Sirosh & Miikkulainen, 1994b), where the constant ξ controls the rate of learning. The LISSOM rules are “instar” (Grossberg, 1972) rules because weight change in pathways to a target neuron is enabled only if the target neuron is active.

Figure 4 near here.

Sirosh et al. (1996) used Equation 11 for the afferent excitatory synaptic plasticity. They used sum normalization $\left(\sum_{ef} (Z_{ef,cd}(t) + \xi x_{ef}x_{cd})\right)$ for the lateral excitatory and lateral inhibitory synaptic plasticity rules, instead of length normalization as in this paper. Qualitatively, both length and sum normalization have the same effects. Length normalization causes the lateral excitatory and lateral inhibitory weight values to be larger than does sum normalization. In the simulations, length normalization produced larger RF size changes than did sum normalization.

For the afferent excitatory synaptic plasticity rule, ab and ef refer to Layer 1 neurons, and cd refers to Layer 2 neurons, $\xi = \xi_{\mathbf{ff}}$. For the lateral excitatory and lateral inhibitory synaptic plasticity rules, ab , cd , and ef refer to Layer 2 neurons. The parameter ξ is set to ξ_e and ξ_i for the lateral excitatory and lateral inhibitory synaptic plasticity rules, respectively. The weights remain bounded because of the weight normalizations.

In response to normal stimuli, LISSOM’s learning rules cause the lateral inhibitory and lateral excitatory weights between pairs of Layer 2 neurons to remain approximately symmetric. In addition, the lateral inhibitory and excitatory weights become approximately proportional to the amount of coactivation between neuron pairs. The learning rules maintain the initial topographically arranged RFs (Sirosh & Miikkulainen, 1997).

2.6.2 Stability of LISSOM networks

The LISSOM learning rules are competitive learning rules. Therefore, the stability of LISSOM networks depends on the input environment and the learning rates (Sirosh & Miikkulainen, 1994b). This issues of network stability and choice of the various parameters in the current simulations are discussed in the Appendix, Section 6.2.

2.6.3 Explanation of dynamic RF changes based on LISSOM rules

The effect of each of the three LISSOM synaptic plasticity rules is considered separately. The overall behavior of the LISSOM model depends on the relative learning rates of the three rules.

Role of LISSOM lateral inhibitory synaptic plasticity. Consider the LISSOM network with only the lateral inhibitory synaptic plasticity rule enabled. Let neuron (p, q) be outside and neuron (r, s) be inside the cortical scotoma region. Assume that after conditioning with normal stimuli, $Z_{pq,rs}^-$ and $Z_{rs,pq}^-$ are not zero, because neurons (p, q) and (r, s) share some common afferent inputs, and that they are approximately equal. According to the LISSOM lateral inhibitory synaptic plasticity rule, $Z_{pq,rs}^-$ does not change, because x_{rs} is 0. However, $Z_{rs,pq}^-$ decreases if some other neuron (u, v) is active, because the weights are normalized. In addition, the normalization causes the lateral inhibitory weights from active neurons (e.g, neurons (u, v) and (p, q)) to neuron (p, q) to become slightly stronger. In the LISSOM model, Layer 2 neurons send lateral excitatory and inhibitory pathways to themselves. Thus, because of the lateral inhibitory synaptic plasticity, neurons outside the cortical scotoma region receive a reduced overall amount of inhibition from neurons within the cortical scotoma. This causes neurons like (p, q) that are outside the cortical scotoma region to exhibit increased responsiveness, a RF expansion, and a slight inward RF shift toward the scotoma edge. The RF expansion of neuron (p, q) is asymmetric because it receives reduced inhibition only when the input is in the scotoma region. For neurons in the overall excitatory zone of neuron (p, q) (e.g., (u, v) and (r, s)), the increased responsiveness of neuron (p, q) may result in increased responsiveness, a RF expansion, and a slight inward RF shift toward the scotoma edge. The increased responsiveness of (p, q) results in increased inhibition to neurons in the overall inhibitory zone of neuron (p, q) (e.g., (c, d) and (a, b)) (Figure 5b), when input locations inside the scotoma are

stimulated. Because of the asymmetric RF profile, neurons whose initial RF center is inside the scotoma and in the overall inhibitory zone of neuron (p, q) (e.g., (a, b)) would show decreased responsiveness, RF contraction, and a small inward RF shift away from the scotoma edge (Figure 5b). The simulation results are in Section 3.1.

Figure 5 near here.

Role of LISSOM lateral excitatory synaptic plasticity. When only the LISSOM lateral excitatory synaptic plasticity rule is enabled, $Z_{pq,rs}^+$ does not change, $Z_{rs,pq}^+$ decreases, and the lateral excitatory weights from active neurons (e.g, neurons (u, v) and (p, q)) to neuron (p, q) become slightly stronger (in the LISSOM network neurons receive lateral excitatory pathways from itself), for the same reasons described in the previous paragraph (Figure 5c). Thus, neurons outside the cortical scotoma region receive a reduced amount of lateral excitation from neurons in the cortical scotoma. This results in reduced responsiveness, decreased RF size, and an outward RF shift, away from the scotoma edge, for neurons like (p, q) that are outside the cortical scotoma region. For neurons in the overall excitatory zone of neuron (p, q) (e.g., (u, v) and (r, s)), the decreased responsiveness of neuron (p, q) may result in decreased responsiveness, a RF contraction, and a slight outward RF shift away from the scotoma edge. For neurons in the overall inhibitory zone of neuron (p, q) (e.g., (c, d) and (a, b)), the decreased responsiveness of (p, q) results in reduced inhibition. Active neurons during scotoma conditioning receive weakened lateral excitatory signals from neurons inside the cortical scotoma. These asymmetric lateral excitatory weight changes lead to a decrease in RF size and an outward RF shift away from the scotoma in neurons whose initial RF center is close to the scotoma boundary (e.g., (p, q)). They also lead to an increase in RF size and an outward RF shift away from the scotoma center in neurons (e.g., (a, b)) whose initial RF is inside and close to the scotoma boundary (Figure 5c). The simulation results are shown in Section 3.3.

Role of LISSOM afferent excitatory synaptic plasticity. In the LISSOM network with only afferent excitatory synaptic plasticity enabled, the synaptic plasticity causes changes in the afferent connectivity of neurons close to the edge of the cortical scotoma region (Figure 5d). These neurons (e.g., (u, v) and (p, q)) have part of their input in Layer 1 within the scotoma.

Thus, according to the afferent excitatory synaptic plasticity rule, when these neurons are activated, the weights from neurons within the scotoma become weaker, and weights from Layer 1 neurons outside the scotoma become stronger. This causes the RFs of Layer 2 neurons (e.g., (u, v) and (p, q)) close to the initial cortical scotoma edge to shrink and shift away from the scotoma center. In addition, these neurons respond weakly to stimulation at positions in the scotoma region and exert less inhibition on neurons (e.g., (a, b)) that were inactive during conditioning. Thus, the neurons that were inactive during the conditioning show increased responsiveness and RF expansion away from the scotoma center (Sirosh et al., 1996). The RF expansion causes the RF centers to shift away from the scotoma center (Figure 5d). The simulation results are in Section 3.2.

The overall effect on responsiveness, RF size, and RF position depends on the relative strengths and sizes of afferent excitation, lateral excitation, and lateral inhibition. It also depends on the learning rates of the three types of connections. In the LISSOM model, the effects of inhibitory synaptic plasticity are in conflict with the effects of afferent and lateral excitatory synaptic plasticity. The RF expansion occurs in neurons *outside the cortical scotoma* if the relative strength and learning rate of lateral inhibitory weights are greater than those of the lateral and afferent excitatory weights. The RF expansion occurs in neurons *inside the cortical scotoma* if the relative strength and learning rate of lateral excitatory or afferent excitatory weights are greater than those of the lateral inhibitory weights. After artificial scotoma conditioning, the LISSOM rules produce RF expansion on one side of the scotoma boundary and RF contraction on the other side.

When the LISSOM networks (with plasticity in only one of the three types of pathways) are again conditioned with normal stimuli, the RFs of all affected neurons are restored.

2.6.4 The activation equation

In the LISSOM simulations, the Layer 2 neuron activations are determined iteratively by

$$x_{pq}(t+1) = S \left(\gamma_{\mathbf{f}} \sum_{ij} Z_{ij,pq}^+ x_{ij}(t) + \gamma_{\mathbf{e}} \sum_{rs} Z_{rs,pq}^+ x_{rs}(t) - \gamma_{\mathbf{i}} \sum_{rs} Z_{rs,pq}^- x_{rs}(t) \right) \quad (12)$$

where

$$S(a) = \frac{1}{1 + \exp(-[a])} - 0.5 \quad (13)$$

(Sirosh & Miikkulainen, 1994b), and $[x] = \max(0, x)$. The constants $\gamma_{\mathbf{ff}}$, $\gamma_{\mathbf{e}}$ and $\gamma_{\mathbf{i}}$ are scaling factors on the excitatory and inhibitory weights and determine the strength of afferent and lateral interactions. The activations are bounded because of the sigmoid function S .

2.6.5 Stability of LISSOM activation equation

The LISSOM activation equation can quickly equilibrate (Sirosh & Miikkulainen, 1994b). Equation 12 approached a fixed point during the various input conditioning regimes for the parameters that are used in this paper (Appendix, Section 6.2.1).

2.6.6 The initial weights

In simulations using the LISSOM model, the connection weights were computed as follows. Let

$$Y_{ab,cd} = \left[\exp \left(\frac{-(m^2 + n^2)}{\sigma^2} \right), \Gamma \right], \quad (14)$$

where $a, b, c, d \in \{0, \dots, 29\}$, $m, n \in \{-15, \dots, 14\}$, $c = ((a + m) \bmod 30)$, and $d = ((b + n) \bmod 30)$. The relationship between the indices a, b, c , and d and the distances m and n is such that the model cortical and thalamic neurons are arranged in a wrapped-around two-dimensional grid. The parameters σ and Γ are positive constants, and the notation $[.,.]$ is defined by Equation 7. Then

$$Z_{ab,cd}(0) = \frac{Y_{ab,cd}}{\left(\sum_{ef} Y_{ef,cd}^2 \right)^{\frac{1}{2}}} \quad (15)$$

is the initial weight of the connection pathway from neuron (a, b) to neuron (c, d) .

For afferent weights, ab and ef refer to Layer 1 neurons, cd refers to Layer 2 neurons, $\sigma = \sigma_{\mathbf{ff}}$, and $\Gamma = \Gamma_{\mathbf{ff}}$. For lateral excitatory and lateral inhibitory weights, ab , cd , and ef refer to Layer 2 neurons. For lateral excitatory weights, $\sigma = \sigma_{\mathbf{e}}$ and $\Gamma = \Gamma_{\mathbf{e}}$, and for inhibitory weights, $\sigma = \sigma_{\mathbf{i}}$ and $\Gamma = \Gamma_{\mathbf{i}}$.

The initial weights were chosen to speed the simulations; in all simulations, these weight values were overridden by new values during an initial phase of whole-field stimulation.

2.7 The inhibition-dominant adaptation model

Xing and Gerstein (1994) described four models of dynamic RFs and argued in favor of an inhibition-dominant network with neural adaptation, or habituation. A neuron's ability to fire decreases/increases after a period of activity/inactivity, without any synaptic changes. In an inhibition-dominant adaptation network, the strength of the lateral inhibitory connections is greater than that of lateral excitatory connections, and all the weights are fixed.

Xing and Gerstein used a spiking neuron model. They modeled adaptation by modifying the action potential threshold, which depended on the number of spikes of a neuron in the recent past. Thus, if a neuron spikes frequently, its action potential threshold increases, thereby making it spike less vigorously, even though the same input is present. On the other hand, if a neuron has not been activated for a long time, then it becomes highly responsive to inputs in its RF.

2.7.1 The adaptation equation

In the present simulations the spiking neuron model is not used. Instead, the output of a Layer 2 neuron (p, q) is modeled as $[x_{pq} - T_{pq}]$, where x_{pq} is controlled by a shunting equation (Grossberg, 1972):

$$\frac{d}{dt}x_{pq} = -Ax_{pq} + (B - x_{pq})E_{pq} - \gamma(C + x_{pq})I_{pq}, \quad (16)$$

with

$$E_{pq} = \beta_{\mathbf{f}} \sum_{i,j \in \text{Layer 1}} [x_{ij}]Z_{ij,pq}^+ + \beta_{\mathbf{e}} \sum_{r,s \in \text{Layer 2}} [x_{rs} - T_{rs}]Z_{rs,pq}^+, \quad (17)$$

$$I_{pq} = \sum_{(r,s) \in \text{Layer 2}} [x_{rs} - T_{rs}]Z_{rs,pq}^-, \quad (18)$$

and $[a] \equiv \max(a, 0)$. The positive constants $\beta_{\mathbf{f}}$ and $\beta_{\mathbf{e}}$ control the effectiveness of afferent excitation and lateral excitation, respectively. The variable T_{pq} represents the adaptive firing threshold of neuron (p, q) . After the activation of Layer 2 neurons has reached equilibrium, the adaptation parameter T_{pq} is modified according to

$$\frac{d}{dt}T_{pq} = \rho(-\eta T_{pq} + (\tau - T_{pq})[x_{pq} - T_{pq}]), \quad (19)$$

where ρ , η , τ are positive constants. The constant ρ controls the rate of change in T_{pq} . The constants η and τ determine the maximum value of T_{pq} , and the relative rates of increase and decrease in T_{pq} , respectively. As η increases the maximum value of T_{pq} decreases and the rate of decrease in T_{pq} when $[x_{rs} - T_{rs}] = 0$ increases, and as τ increases the maximum value of T_{pq} increases and the rate of increase in T_{pq} increases. According to Equation 19, if $T_{pq}(0) \geq 0$, then $T_{pq}(t) \geq 0$ for all $t \geq 0$. The threshold T_{pq} increases if neuron (p, q) was active in its recent past and decreases if (p, q) was not very active.

The spiking model was not implemented here because Xing and Gerstein (1994) did not provide complete implementation details of their model. Even though the model described by Equations 16–19 differs from the spiking model used by Xing and Gerstein in their simulations, it captures the essential characteristics of their inhibition-dominant adaptation model: the simulation results based on Equations 16–19 replicate their results.

In the inhibition-dominant adaptation network, the lateral inhibitory and lateral excitatory pathway weights are symmetric. Extensive simulations show that the system defined by Equation 16 may be absolutely stable (Cohen & Grossberg, 1983). Simulations presented in this paper show that the network equilibrates. The adaptation levels of the neurons reach stable fixed points with sufficient training. The initial pathway weights are set according to Equations 14 and 15.

2.7.2 Explanation of dynamic RF changes based on adaptation

After the network receives normal stimulation for a sufficiently long time, every Layer 2 neuron becomes adapted by approximately the same amount. In response to the scotoma stimuli, the neurons outside the cortical scotoma region are activated, thereby keeping them habituated. However, the inactive neurons whose RF is inside the scotoma become dishabituated. Dishabituation of neurons with RF inside the scotoma produces increased responsiveness and RF expansion. The increased responsiveness increases inhibition to neurons whose RF is outside the scotoma. Thus, the RF size of the neurons whose RF is occluded by the scotoma region increases, and the RF size of some neurons whose RF is just outside the scotoma decreases. As scotoma conditioning proceeds, some neurons in the initial cortical scotoma may recover

functionality. As in the EXIN model, all neurons that show RF expansion are those whose RF lies within the scotoma (Figure 6).

Figure 6 near here.

If the network is exposed to normal stimuli again, the RF of the neurons that were in the cortical scotoma region is restored. The simulation results are in Section 3.1.

The above explanation is different from the one given by Xing and Gerstein (1994). In their simulations, they measured the initial RFs *before* any conditioning by the normal stimuli. Then they conditioned the network using scotoma stimuli. In this case, the neurons in the cortical scotoma region are not affected, and those outside the cortical scotoma adapt. The adaptation of the neurons outside the cortical scotoma causes them to be less responsive, and hence they exert less inhibition on neurons in the cortical scotoma. As a result, the size of the RF of the neurons in the scotoma region increases (see Section 3.1). The conditioning procedure used by Xing and Gerstein differs from that used by Pettet and Gilbert (1992). In the experiments of Pettet and Gilbert, the original RF was determined after a period of random stimulation within and outside the field.

In the inhibition-dominant adaptation model used in simulations presented in this paper, if the T_{pq} values had not equilibrated during normal stimuli presentations, then they would increase for neurons outside the cortical scotoma and would decrease for those in the cortical scotoma. This situation would also produce expansion of the RF of neurons in the cortical scotoma.

2.8 The adaptation model with no lateral interaction

In this variant of the adaptation model, there are no lateral excitatory or lateral inhibitory pathways. The neurons adapt according to Equation 19. In the simulations, the activation level of Layer 2 neuron (p, q) is $[x_{pq} - T_{pq}]$, where x_{pq} is controlled by Equation 16. With no lateral interactions, Equation 16 can be solved analytically (Appendix, Section 6.3.1). The afferent excitatory weights were the same as for the inhibition-dominant network.

2.8.1 Explanation of dynamic RF changes based on adaptation

In the adaptation network with no lateral interactions, all the neurons are adapted equally after normal stimulation. During scotoma conditioning, neurons with initial RFs in the scotoma region dishabituate due to inactivity. As the adaptation level in neurons within the cortical scotoma decreases, the effective RF size of these neurons increases. Neurons outside the cortical scotoma remain adapted because of activation by the input stimuli. During normal stimulation following scotoma conditioning, the previously inactive neurons are activated; hence they become adapted, and their RF size contracts. In the absence of lateral interactions, the RFs remain symmetric, and there are no shifts in RF position during scotoma conditioning. Simulation results are in Section 3.1.7.

2.9 The excitation-dominant adaptation model

In an excitation-dominant adaptation network, the strength of the lateral excitatory connections is greater than that of lateral inhibitory connections. In this model, the neurons adapt according to Equation 19. The output of Layer 2 neuron (p, q) is $[x_{pq} - T_{pq}]$, where x_{pq} is controlled by Equation 16. The initial connection weights are set according to Equation 15. In the simulations, the activation equation equilibrated (Appendix, Section 6.3.1).

2.9.1 Explanation of dynamic RF changes based on adaptation

In the excitation-dominant adaptation network, all the neurons are adapted equally after normal stimulation. During scotoma conditioning, neurons with initial RF in the scotoma region become less adapted, because of inactivity. The neurons close to the center of the cortical scotoma become least adapted and hence most responsive. In addition, because of lateral excitation, the neighboring neurons excite one another. Thus, neurons within the cortical scotoma show RF expansion. Since neurons receive more lateral excitation from neurons within the cortical scotoma, the RFs of the neurons shift toward the center of the scotoma. Neurons outside the cortical scotoma remain adapted because of activation by the input stimuli. During normal stimulation following scotoma conditioning, the previously inactive neurons are activated; hence they become adapted, and their RF size contracts. Simulation results are in Section 3.1.7.

3 Simulation results: Scotoma stimuli

The simulation results are organized to emphasize the effects of the different rules for RF changes after artificial scotoma conditioning and retinal lesions. The different synaptic plasticity rules in the EXIN and the LISSOM model serve different purposes during self-organization of various cortical properties. The analyses of the effects of each rule individually during artificial scotoma conditioning and retinal lesions elucidates the unique properties of the rules. Furthermore, the dynamic RF changes produced by the full EXIN and the full LISSOM model during artificial scotoma conditioning depend on the relative learning rates of the different synaptic plasticity rules, and thus the effects produced by one rule can mask the effects produced by the others.

In Section 3.1, the EXIN network with *only* lateral inhibitory synaptic plasticity enabled, the LISSOM network with *only* lateral inhibitory synaptic plasticity enabled, the inhibition-dominant adaptation network, the adaptation network with no lateral interaction, the excitation-dominant adaptation network, and the inhibition-dominant adaptation network with no prior normal stimulation are simulated during scotoma conditioning. These results are compared to experimental data. In Section 3.2, the effects of synaptic plasticity in afferent excitatory pathways alone in the EXIN and LISSOM networks during scotoma conditioning are presented, and the effects of lateral excitatory synaptic plasticity alone during scotoma conditioning are presented in Section 3.3 using the LISSOM network. To further distinguish the effects of the various rules for cortical plasticity, a novel experiment is presented in Section 4.

3.1 Comparison of outstar/instar lateral inhibitory synaptic plasticity rules and neuronal adaptation

The following simulations highlight the opposite effects of instar and outstar lateral inhibitory synaptic plasticity rules.

3.1.1 Dynamic RF expansion and contraction

To induce robust expansion and contraction of RF size, Pettet and Gilbert (1992) presented the artificial scotoma conditioning stimuli for several minutes. Because quantitative mapping took several minutes, the exact time course of the observed changes was not determined. To minimize

the effect of the RF measurement process on the RF size, Pettet and Gilbert (1992) alternated conditioning stimuli and RF mapping. Pettet and Gilbert (1992) reported that the RF expansion after artificial scotoma conditioning was always accompanied by an increased responsiveness from the region of the original RF. However, the spontaneous firing of the neuron in the absence of visual stimuli did not change.

In the simulations, the synaptic plasticity rules and the adaptation rules were turned-off so that the RF sizes are not affected by RF measurements. Figure 7 compares the RF sizes of Layer 2 neurons that show maximal RF expansion after scotoma conditioning. Neurons with maximal RF expansion are shown to emphasize the asymmetry in the RF profile after artificial scotoma conditioning in the models. The RF expands in the EXIN network with only lateral inhibitory synaptic plasticity enabled (Figure 7d) and in the inhibition-dominant adaptation network (Figure 7f) after scotoma conditioning, in neurons whose initial RF is inside the scotoma. In the LISSOM network with only lateral inhibitory synaptic plasticity enabled, the initial RF of the neuron that showed maximal RF expansion after scotoma conditioning was located outside the scotoma (Figure 7e). Re-conditioning with normal stimuli resulted in RF restoration in all the three models, as shown in Figures 7g-i. The simulations illustrate the qualitative behavior of the models. The absolute RF size and the absolute RF size change in the three networks are parameter dependent, and can be matched better with some parameter adjustments. Therefore, the differences in the RF size and the absolute RF size change of model neurons in the models are not significant.

Figure 7 near here.

Pettet and Gilbert (1992) claimed that the expansion elicited by the artificial scotoma never exceeded the boundaries of the scotoma. However, the neurons that they studied had initial RFs in the center of the artificial scotoma, and the size of the scotoma was about three times the diameter of the initial RF. Darian-Smith and Gilbert (1995) showed rapid recovery of responsiveness in neurons whose RF was inside the cortical scotoma, following bilateral retinal lesions. This indicates that the RF of some neurons in the original cortical scotoma crossed the retinal scotoma boundary. The EXIN network with lateral inhibitory synaptic plasticity alone,

the LISSOM network with only lateral inhibitory synaptic plasticity enabled, and the inhibition-dominant adaptation model produced RF expansions that cross the scotoma boundaries (Figure 7).

The RF expansions in the three simulations are accompanied by increased responsiveness of the corresponding Layer 2 neurons. Figures 8a–c compare the responsiveness of the neurons whose RFs are shown in Figure 7, before and after conditioning with scotoma stimuli in the three models. Note that after scotoma conditioning, the RF profile of some neurons in these models is asymmetric. The asymmetric RF shape is caused by asymmetric changes of the weights in the EXIN and LISSOM simulations and by asymmetric changes in adaptation level of the neurons in the inhibition-dominant adaptation network. The asymmetry in the RF profile produced by the EXIN and the inhibition-dominant adaptation networks is consistent with observations made by Das and Gilbert (1995b), that neurons were more responsive to locations outside the scotoma than to those inside, following artificial scotoma conditioning. DeAngelis et al. (1995) did not observe asymmetry in the RF of recorded neurons whose RF was inside the scotoma during scotoma conditioning; this may have happened because the RF of the recorded neurons in their experiments was in the center of the scotoma. In the simulations, asymmetric RFs were observed in neurons close to the scotoma boundary (Figure 8) but not in neurons at the scotoma center.

Figure 8 near here.

Spontaneous cortical activations were not incorporated in the EXIN and LISSOM simulations. However, synaptic plasticity during scotoma conditioning does not affect spontaneous activations in the absence of visual stimulation. Cortical spontaneous activity in the absence of any visual stimuli is close to zero (Movshon et al., 1978). This weak activity would have negligible effect on the spontaneous activation of other neurons, even if synapses between them were modified. Thus, spontaneous activity in the absence of visual stimuli would be almost unchanged if scotoma conditioning resulted in synaptic plasticity. Xing and Gerstein (1994) did not simulate the effect of adaptation on the spontaneous activation levels of Layer 2 neurons. However, they assumed that the spontaneous activation level is independent of adaptation.

3.1.2 RF size as a function of position

Pettet and Gilbert (1992) showed RF expansion of cortical neurons whose initial RF was within the artificial scotoma, after conditioning with artificial scotoma stimuli. Darian-Smith and Gilbert (1995) reported that between five minutes and one hour after bilateral retinal lesions, cortical neurons located close to or just inside the cortical scotoma boundary showed a striking increase in RF size.

Figures 9a–c show the RF size before and after scotoma stimuli conditioning as a function of the position of the initial RF center, for the three models. For the EXIN network with only lateral inhibitory synaptic plasticity enabled, Figure 9a, and the inhibition-dominant adaptation model, Figure 9c, the most prominent RF expansions occur for Layer 2 neurons with initial RF centers close to and inside the scotoma edge. However, for the LISSOM network with only lateral inhibitory synaptic plasticity enabled, Figure 9b, the most prominent RF expansions occur for Layer 2 neurons with initial RF centers close to and *outside* the scotoma edge.

Figure 9 near here.

3.1.3 RF size profile as a function of scotoma size

In the three simulations, neurons whose initial RF was close to the scotoma boundary showed the maximal expansion. This is clearly visible in the bimodal peaks in Figure 9. In the EXIN network with lateral inhibitory synaptic plasticity alone and the inhibition-dominant adaptation model, the peaks occur for neurons with initial RF inside the scotoma region; in the LISSOM network with only lateral inhibitory synaptic plasticity enabled, the peaks occur for neurons with initial RF outside the scotoma region.

The three simulations suggest the prediction that as the scotoma size is reduced, the peaks will move closer. This prediction is illustrated by results shown in Figure 10. Figure 10 shows the RF size of a cross section of Layer 2 neurons after scotoma conditioning with a scotoma of size 9×9 . The EXIN model with only lateral inhibitory synaptic plasticity enabled produced a unimodal function (Figure 10a). In the LISSOM model with only lateral inhibitory synaptic plasticity enabled and the inhibition-dominant adaptation model, the peaks are closer in Figures 10b–c than in Figures 9b–c.

Figure 10 near here.

3.1.4 Recovery of neurons in the cortical scotoma

After bilateral retinal lesions, function returned over time to the cortex in a roughly concentric inward direction (Darian-Smith & Gilbert, 1995).

The EXIN network with only lateral inhibitory synaptic plasticity enabled exhibited this property during scotoma conditioning. According to the EXIN inhibitory synaptic plasticity rule, lateral inhibitory pathways to inactive or weakly active Layer 2 neurons with RF centers inside the scotoma weaken. The neurons closest to the edge of the cortical scotoma have relatively strong afferent inputs from locations outside the scotoma. Thus, these neurons respond first to inputs outside the scotoma. These newly responsive neurons in turn weaken inhibition to neurons farther inside the cortical scotoma. This behavior is illustrated in Figure 11a.

Figure 11 near here.

The LISSOM lateral inhibitory synaptic plasticity rule alone did not produce recovery in Layer 2 neurons in a concentric inward direction during scotoma conditioning. Contrary to the experimental data, the LISSOM lateral inhibitory rule caused loss of functionality in a concentric inward direction. According to the LISSOM lateral inhibitory synaptic plasticity rule, during scotoma conditioning Layer 2 neurons outside and close to the cortical scotoma edge show an increase in their responsiveness and RF size because of a decrease in the inhibitory weights from neurons inside the cortical scotoma. This increase in responsiveness results in increased inhibition to functional Layer 2 neurons very close to the scotoma boundary, and consequently, these lose responsiveness to locations outside the scotoma (Figure 11b).

In the inhibition-dominant adaptation model, during scotoma conditioning, adaptation of Layer 2 neurons in the cortical scotoma decreases, which lets neurons closest to the cortical scotoma edge recover responsiveness first (Figure 11c).

3.1.5 RF shifts

Figures 12a–c display the shift in RF center after conditioning with scotoma stimuli as a function of the position of the initial RF center of each Layer 2 neuron. The EXIN network with only

lateral inhibitory synaptic plasticity enabled, Figure 12a, and the inhibition-dominant adaptation model, Figure 12c, exhibit consistent outward shifts in the RF center of Layer 2 neurons, and the maximal shifts occur for neurons with RF center close to the scotoma edge, consistent with the centrifugal RF displacements found within 1 hour after retinal lesions (Darian-Smith & Gilbert, 1995). In contrast, the LISSOM network with only lateral inhibitory synaptic plasticity enabled, Figure 12b, displays consistent shifts toward the scotoma center.

Figure 12 near here.

3.1.6 Effect of blank stimuli on RF

In the simulations with only lateral inhibitory synaptic plasticity (EXIN or LISSOM lateral inhibitory rule), the changes in RFs are the consequence of synaptic strength modifications that depend on neuronal activation. When the network is presented with a blank display, no Layer 2 neuron is activated, and hence no RF change occurs, consistent with the absence of changes observed in RFs during periods of no visual stimulation (Pettet & Gilbert, 1992).

In the inhibition-dominant adaptation model, the RF changes are due to differences in the adaptation thresholds of Layer 2 neurons whose initial RF is within and outside the scotoma region. In the inhibition-dominant adaptation model the adaptation threshold is a function of neuronal activation within a time interval. Blank stimuli do not activate Layer 2 neurons, and this causes the adaptation thresholds of all Layer 2 neurons to become approximately equal. The neurons whose RF is outside the scotoma region show RF expansion. The RF size increases because of the decrease in the threshold (Figure 13). In Figure 13a, the neurons inactive during scotoma conditioning are not fully dishabituated; therefore, during presentation of blank stimuli, these neurons too show RF expansion.

In Figure 13b, after a longer period of scotoma conditioning, some of the neurons are almost fully dishabituated, especially the neuron with initial RF center at position 0. During presentation of blank stimuli, changes in inhibition caused by dishabituation of the neurons whose initial RF center is outside the scotoma become dominant. Thus, during presentation of blank stimuli, as neurons whose RF is outside the scotoma rapidly become more responsive, and they

exert more inhibition on neurons whose RF is inside the scotoma. This results in a slight RF contraction for neurons whose RF is inside the scotoma (Figure 13b).

Figure 13 near here.

3.1.7 Other adaptation models

Xing and Gerstein (1994) measured the initial RFs *before* conditioning with normal stimuli. Then they conditioned the network using scotoma stimuli. In this case, the neurons in the cortical scotoma region were not affected, and those outside the cortical scotoma adapted. Adaptation of the neurons outside the cortical scotoma decreases their responsiveness; hence they exert less inhibition on neurons in the cortical scotoma. As a result, the RFs of the neurons in the scotoma region increased in size and showed other changes consistent with experimental data (Figures 14–17a). However, presenting blank stimuli to the model would result in a decrease in adaptation of all neurons. Thus, the RFs would be restored to their pre-scotoma sizes.

Figure 14 near here.

Figures 14b–c show changes in RF size following artificial scotoma conditioning in an adaptation network with no lateral interaction and in an adaptation network with dominant lateral excitation, respectively. The two networks produce RF expansion in inactive neurons after scotoma conditioning, and they produce RF contraction during normal stimulation following scotoma conditioning.

However, in the adaptation network with no lateral interactions, RF profiles after scotoma conditioning are symmetric (Figure 15b), and the RF position of the neurons do not change (Figure 16b). In the adaptation network with dominant lateral excitation, the RF profile shows asymmetry, but the neurons are more responsive to locations within the occluded region during scotoma conditioning (Figure 15c); this manifests itself as RF shifts toward the center of the scotoma (Figure 16c). These effects are inconsistent with the observations of Darian-Smith and Gilbert (1995) and Das and Gilbert (1995b).

Figure 15 near here.

Figure 16 near here.

Figures 17b–c show that in these adaptation networks, responsiveness returns to neurons within the cortical scotoma in a concentric inward direction.

Figure 17 near here.

Because the adaptation level in these adaptation models depends on neuronal activation, the RF size of neurons in these models changes in the absence of input stimulation.

3.1.8 Conclusions

From the simulations presented in this section, it is clear that the LISSOM instar lateral inhibitory synaptic plasticity rule is insufficient to model the effects of artificial scotoma conditioning and retinal lesions. However, the EXIN outstar lateral inhibitory synaptic plasticity rule is sufficient to model these effects.

Xing and Gerstein’s (1994) inhibition-dominant adaptation network failed only in modeling the effects of presentation of blank stimuli after scotoma conditioning. In Section 4, a novel experiment is suggested to determine the role of neuronal adaptation in producing dynamic RF changes that persist over a long periods (about 15 minutes). Cortical neurons are maximally adapted within tens of seconds, and neurons recover their responsiveness over a period of tens of seconds (Hammond et al., 1986, 1989). Psychophysical experiments on humans using artificial scotoma conditioning produces shifts in position judgments consistent with the RF expansion hypothesis (Kapadia et al., 1994). These changes occurred over a period of 1-2 seconds, however, and would be consistent with the inhibition-dominant adaptation model.

Thus, we conclude that during scotoma conditioning, effects of neuronal adaptation occur over a period of tens of seconds; however, the persistent effects produced over a period of 15 minutes to hours may be caused by a long-term process, e.g., long-term synaptic plasticity. The RF changes observed over a period of months, however, may involve sprouting and establishment of new connections (Darian-Smith & Gilbert, 1994). The issue of time-scales is further discussed in Section 5.4.

The adaptation models without lateral interaction and with dominant lateral excitation

produced RF expansion in neurons whose RFs were within the scotoma region. However, they produced RF shifts inconsistent with experimental data.

Sirosh et al. (1996) explained RF changes during scotoma conditioning using the LISSOM afferent excitatory synaptic plasticity. However, in their simulations the LISSOM lateral excitatory and lateral inhibitory synaptic plasticity rules were also enabled. To study the effects of afferent excitatory synaptic plasticity alone during scotoma conditioning, the next section (Section 3.2), presents simulations on the EXIN and LISSOM networks with their respective afferent excitatory synaptic plasticity rules.

3.2 Role of afferent excitatory synaptic plasticity

In this section, the effects of scotoma conditioning on the EXIN and LISSOM networks with only afferent excitatory synaptic plasticity enabled are presented. Both the EXIN and LISSOM afferent excitatory synaptic plasticity rules are instar rules. One difference between the two networks is the presence of short-range lateral excitatory connections in the LISSOM network.

3.2.1 RF size as a function of position

Figures 18a–b show the RF size before and after scotoma conditioning as a function of the position of the initial RF center, for the EXIN and the LISSOM networks with only afferent excitatory synaptic plasticity enabled. For the EXIN network with only afferent excitatory synaptic plasticity enabled, small RF expansions occurred for Layer 2 neurons whose initial RF center is close to and inside the scotoma edge, and Layer 2 neurons whose initial RF center is close to and outside the scotoma boundary showed larger RF contraction (Figure 18a). Similarly, the LISSOM network with only afferent excitatory synaptic plasticity enabled produced RF expansion in neurons whose initial RF is inside the scotoma and produced RF contraction in neurons close to the scotoma boundary (Figure 18b).

Figure 18 near here.

The EXIN and the LISSOM networks with only afferent excitatory synaptic plasticity enabled produced RF expansion in neurons whose initial RF center is inside the scotoma, consistent with the results of Pettet and Gilbert (1992).

The LISSOM network with afferent excitatory synaptic plasticity alone showed larger RF expansion than the EXIN network with only afferent excitatory synaptic plasticity enabled because of the short-range lateral excitatory connections in the LISSOM network. In the LISSOM network, the neurons whose responsiveness increased (because of decreased inhibition through weakening of afferent pathways converging on other neurons) elevated the responsiveness of their neighbors via lateral excitatory connections, thereby producing large RF expansions. In Figure 18b, the curve showing RF size after scotoma conditioning is unimodal. The curve would become bimodal when the scotoma size is increased. In the LISSOM network, the weight of the lateral excitatory and inhibitory pathway decreases as the distance between the source and target neurons increases. Thus, as the scotoma size is increased, the effects of lateral pathways between neurons whose RFs are close to the scotoma boundary and neurons whose RFs are close to the center of the scotoma decrease, and therefore the amount of RF expansion in neurons whose RFs are close to the scotoma center will be less than the amount of RF expansion in neurons whose RFs are inside the scotoma, but closer to the scotoma boundary than the scotoma center. Thus, the RF size curve after scotoma conditioning becomes bimodal.

Figure 19 shows the RF profile of a neuron that exhibited RF expansion and another that exhibited RF contraction in the two simulations. After scotoma conditioning, the RF profile of some neurons in these networks was asymmetric, consistent with observations of Das and Gilbert (1995b). The asymmetric RF shape is caused by asymmetric changes in weights in the two networks.

Figure 19 near here.

In Figure 18a, neurons far away from the scotoma showed RF expansion. In the EXIN network with only afferent excitatory synaptic plasticity enabled, during scotoma conditioning neurons initially in the cortical scotoma strengthened their afferent connections from locations outside the scotoma. Thus, neurons surrounding the cortical scotoma received more inhibition when locations outside the scotoma were stimulated, and suppression of responsiveness of these neurons resulted in a slight increase in responsiveness of neurons farther away from the scotoma (Figure 20).

Figure 20 near here.

3.2.2 Recovery of neurons in the cortical scotoma

The EXIN and the LISSOM networks with only afferent excitatory synaptic plasticity enabled exhibited new responsiveness to stimuli outside the scotoma in neurons whose initial RF is inside the scotoma, during scotoma conditioning (Figure 21). This is consistent with the finding that after bilateral retinal lesions, function returned over time to the cortex in a roughly concentric inward direction (Darian-Smith & Gilbert, 1995).

Figure 21 near here.

3.2.3 RF shifts

Figures 22a–b display the shift in RF center after conditioning with scotoma stimuli as a function of the position of the initial RF center of each Layer 2 neuron. The EXIN and LISSOM networks with only afferent excitatory synaptic plasticity enabled, Figures 22a–b, exhibited consistent outward shifts in the RF center of Layer 2 neurons. The maximal shifts occurred for neurons whose RF center was close to the scotoma edge. These results are consistent with the consistent small centrifugal RF shift away from the lesion center, between five minutes and one hour after the retinal lesions, observed by Darian-Smith and Gilbert (1995). In the two networks, neurons whose initial RF center was inside the scotoma showed outward shift because of the asymmetric RF expansion (Figure 19), and neurons whose initial RF overlapped the scotoma boundary showed an outward shift because of RF contraction (Figure 19).

Figure 22 near here.

During normal stimulation following scotoma conditioning, the EXIN network with only afferent excitatory synaptic plasticity enabled did not recover (Figure 22a). This effect was caused by strong lateral inhibition, which prevented the neurons whose RF size decreased (because of weakening of pathways from the scotoma during scotoma conditioning) from becoming strongly active to inputs at positions inside the scotoma. The weak afferent connections were thus prevented from becoming strong. In the LISSOM network with afferent excitatory

synaptic plasticity alone, the short-range lateral excitatory connections helped neurons with weak afferent connections from the scotoma region to become more active, and hence to strengthen the weak afferent pathways (Figure 22b).

To ensure that the lack of recovery in the EXIN simulation was not caused by insufficient training, the simulation was run for much longer time (dotted line in Figure 22a). In fact, additional training shifted the RFs of neurons farther away from their original positions. The dotted line in Figure 22a is jagged, implying that the RF positions of some neurons shifted toward the scotoma center and the RF positions of neighboring neurons shifted away from the scotoma center. This behavior is the consequence of the strong lateral inhibitory interactions between the neurons. Because of strong lateral inhibition, when a neuron’s responsiveness increases it suppresses the activation of neighboring neurons, and thus neurons farther away from the neighbor neurons receive less inhibition. This alternation of increased inhibition and decreased inhibition causes RF shifts in opposite directions in neighboring neurons.

However, with both the afferent excitatory and the lateral inhibitory synaptic plasticity enabled, the neurons in the EXIN network tended to recover their original RF properties (size, position, shape, and responsiveness) during normal stimulation following scotoma conditioning. During normal stimulation following scotoma conditioning, lateral inhibitory connection weights to the weakly active neurons decreased, and lateral inhibitory connection weights to strongly neurons increased. This led to an attraction effect on neurons with weakened afferent connections from the scotoma region and a repulsive effect on neurons with strengthened afferent connections from outside the scotoma region, resulting in a shift in RF centers toward their original locations (Figure 22c) and restoration of the original RF sizes (Figure 18c). During the normal stimulation following scotoma conditioning, the EXIN network with only lateral inhibitory synaptic plasticity rule completely recovered the original RF properties, but the EXIN network with only afferent excitatory synaptic plasticity enabled did not recover the original RF properties. Thus, in the full EXIN network, the amount of recovery of RF properties during normal stimulation following scotoma conditioning will depend on which rule produces the dominant effects.

3.2.4 Effect of blank stimuli on RFs

In the EXIN and LISSOM networks with afferent excitatory synaptic plasticity alone, a blank input display causes the Layer 2 neurons to be inactive, and therefore afferent excitatory pathway weights and cortical RFs do not change.

3.2.5 Conclusions

The EXIN and the LISSOM networks with only afferent excitatory synaptic plasticity enabled produced RF expansion consistent with experimental effects of artificial scotoma conditioning and bilateral retinal lesions. The EXIN network with only afferent excitatory synaptic plasticity enabled did not recover during normal stimulation following scotoma conditioning. However, the full EXIN network with both afferent excitatory and lateral inhibitory synaptic plasticity enabled, did recover. In both the EXIN and the LISSOM networks with only afferent excitatory synaptic plasticity enabled, RF contraction in neurons whose initial RF overlapped the scotoma edge was observed during scotoma conditioning.

3.3 Role of lateral excitatory synaptic plasticity

In this section, the effects of the LISSOM instar lateral excitatory synaptic plasticity rule alone during scotoma conditioning are presented.

These simulations demonstrate that in a network with short-range lateral dominant excitatory connections and long-range lateral dominant inhibitory connections, an instar rule to modify the short-range lateral excitatory connections during scotoma conditioning produces RF expansion in neurons whose initial RF center is inside the scotoma.

3.3.1 RF size as a function of position

Figure 23a shows the RF size before and after scotoma stimuli conditioning as a function of the position of the initial RF center. In the LISSOM network with only instar lateral excitatory synaptic plasticity enabled, Figure 23a, RF expansions occurred for Layer 2 neurons whose initial RF center is inside the scotoma edge. In addition, neurons whose initial RF center is close to and outside the scotoma boundary showed RF contraction. These effects are similar to those observed

in the EXIN and the LISSOM networks with only afferent excitatory synaptic plasticity enabled (Section 3.2).

Figure 23 near here.

In the LISSOM network with instar lateral excitatory synaptic plasticity alone, during scotoma conditioning the lateral excitatory pathways from the neurons just inside the cortical scotoma to those just outside weaken. Thus, in the LISSOM network with only instar lateral excitatory synaptic plasticity enabled, neurons whose RF is close to and outside the scotoma become less responsive to stimulation within and close to the scotoma boundary. This causes RF contraction in neurons whose initial RF is close to and outside the scotoma. Furthermore, because of the decreased responsiveness of these neurons to stimulation within and close to the scotoma boundary, they exert less inhibition on neurons whose initial RF is inside and away from the scotoma boundary, resulting in RF expansion.

In Figure 23b, the neuron became more responsive to locations away from the scotoma center, consistent with the results of Das and Gilbert (1995b). Normal stimulation following scotoma conditioning restored the RF size in the LISSOM network with instar excitatory synaptic plasticity alone (Figure 23).

3.3.2 Recovery of neurons in the cortical scotoma

The LISSOM network with instar lateral excitatory synaptic plasticity alone exhibited new responsiveness to stimuli outside the scotoma in neurons whose initial RF is inside the scotoma, during scotoma conditioning (Figure 23c).

3.3.3 RF shifts

Figure 23d displays the shift in RF center after conditioning with scotoma stimuli as a function of the initial RF center position of Layer 2 neurons. The LISSOM network with only instar lateral excitatory synaptic plasticity enabled exhibits consistent outward shifts (Figure 23d).

In the LISSOM network with only instar lateral excitatory synaptic plasticity enabled, neurons whose initial RF center is inside the scotoma showed outward shift because of

asymmetric RF expansion (Figure 23b), and neurons whose initial RF overlapped the scotoma boundary showed outward shift because of RF contraction.

3.3.4 Effect of blank stimuli on RF

In the LISSOM network with only lateral excitatory synaptic plasticity enabled, when no input stimulation is present, Layer 2 neurons are inactive, and therefore lateral excitatory pathway weights and cortical RFs do not change.

3.3.5 Conclusions

The LISSOM network with instar lateral excitatory synaptic plasticity alone produces many effects consistent with the experimental effects of artificial scotoma conditioning and bilateral retinal lesions. The LISSOM network with only instar lateral excitatory synaptic plasticity enabled produces RF contraction in neurons whose initial RF is just outside the scotoma during scotoma conditioning. Instar lateral excitatory synaptic plasticity in a network with short-range lateral excitatory connections and long-range inhibitory connections thus models dynamic RF changes during artificial scotoma conditioning.

In the LISSOM network, if the outstar lateral excitatory synaptic plasticity rule is used instead of the LISSOM instar lateral excitatory synaptic plasticity rule, then after scotoma conditioning the network may produce RF expansion in neurons outside the cortical scotoma and may produce RF shifts toward the scotoma center. These may happen during scotoma conditioning because an outstar lateral excitatory synaptic plasticity rule weakens lateral excitatory pathways *from active neurons to inactive neurons*, whereas an instar lateral excitatory synaptic plasticity rule weakens lateral excitatory pathways *from inactive neurons to active neurons*.

4 Simulation results: Complementary scotoma stimuli

The EXIN network (with afferent excitatory synaptic plasticity or lateral inhibitory synaptic plasticity alone), the LISSOM network (with afferent excitatory synaptic plasticity or instar lateral excitatory synaptic plasticity alone), and the inhibition-dominant adaptation model

explain many important features of the artificial scotoma experiment (Pettet & Gilbert, 1992) and the experiment on the short-term effects of the bilateral retinal lesion (Darian-Smith & Gilbert, 1995). However, the inhibition-dominant adaptation model differs from other models in its behavior after blank screen conditioning (Section 3.1.6). The EXIN network with instar afferent excitatory synaptic plasticity alone and the LISSOM network with instar afferent or instar lateral excitatory synaptic plasticity alone produce RF contraction during scotoma conditioning, in neurons whose initial RF overlaps the scotoma boundary.

To further distinguish between the EXIN synaptic plasticity rules, the LISSOM synaptic plasticity rules, and the adaptation rule, a “complementary scotoma” stimulation experiment is proposed. In this conditioning paradigm, after the initial conditioning with normal stimuli, the network is presented with stimuli that have two alternating, complementary scotoma regions. That is, for any stimulus, the scotoma is a hemifield or its complementary hemifield. The duration of presentation, inter-trial interval, and duration before testing after conditioning should be varied to control for the ubiquitous neuronal adaptation in the cortex. The duration of presentation of each hemifield should short (e.g., 1–10 seconds).

4.1 RF changes because of synaptic plasticity

In the EXIN network with only lateral inhibitory synaptic plasticity enabled, neurons whose RF is near the common boundary of the complementary scotoma regions show maximal increase in RF size. The expansion is due to the decrease in the lateral inhibitory weights between layer 2 neurons whose RFs are inside and outside the common boundary of the complementary scotoma regions. The decrease occurs because these neurons are never coactivated during complementary scotoma conditioning. Figure 24a shows the results of a complementary scotoma simulation, plotting Layer 2 RF size as a function of position of the initial RF center. The RF expansion was accompanied by an increase in responsiveness of the corresponding Layer 2 neurons.

Figure 24 near here.

Figure 24b shows Layer 2 RF size as a function of position of the initial RF center, after the LISSOM network with only lateral inhibitory synaptic plasticity enabled was presented with

complementary scotoma stimuli. The behavior of the LISSOM network with only lateral inhibitory synaptic plasticity enabled was similar to that of the EXIN network with only lateral inhibitory synaptic plasticity enabled for this conditioning. Layer 2 neurons in the LISSOM network, with RFs inside and outside the common boundary of the complementary scotoma regions, were never coactivated. This resulted in a decrease in the strength of lateral inhibitory connections between these neurons. Thus, RFs near the boundary increased in size.

Figure 25 near here.

Figure 25a shows Layer 2 RF size as a function of position of the initial RF center after the EXIN network with only afferent excitatory synaptic plasticity enabled was presented with complementary scotoma stimuli. The neurons whose initial RF overlapped the complementary scotoma boundary showed RF contraction, as these neurons received weakened afferent pathways from positions close to the complementary scotoma boundary. Complementary scotoma conditioning of the LISSOM network with only afferent excitatory synaptic plasticity enabled produced the same effects (Figure 25b).

The LISSOM network with instar lateral excitatory synaptic plasticity alone produced RF contraction in neurons whose initial RF straddled the complementary scotoma boundary. This happens because anti-correlated activity in neurons whose initial RF center lay on opposite sides of the complementary scotoma boundary results in weakening of mutual lateral excitatory connections, leading to RF contraction (Figure 25c).

In the EXIN and the LISSOM networks with only lateral inhibitory synaptic plasticity enabled, the RFs of neurons across the complementary scotoma boundary shifted *toward* each other (Figure 26). However, in the EXIN network with only afferent excitatory plasticity enabled and in the LISSOM network with either afferent excitatory or lateral excitatory plasticity enabled, the RFs of neurons across the complementary scotoma boundary shifted *away* from each other (Figure 27). In the LISSOM network with lateral excitatory plasticity alone, smaller RF shifts in neurons far from the cortical complementary scotoma boundary were due to reduced inhibition to these neurons from neurons close to the cortical complementary scotoma boundary.

Figure 26 near here.

Figure 27 near here.

4.2 RF changes because of neuronal adaptation

During complementary scotoma conditioning, Layer 2 neurons are activated alternately because of the alternate complementary input stimulations. Thus, Layer 2 neurons become less adapted compared to the adaptation level after the initial whole field stimulation.

In the computer simulations of complementary scotoma conditioning of the inhibition-dominant network, Layer 2 neurons whose initial RF straddles the complementary scotoma boundary were more adapted than neurons whose initial RF is away from the complementary scotoma boundary. But the Layer 2 RF size as a function of the initial RF center position was almost flat (Figure 24c). Note that the RF size of a Layer 2 neuron was defined as the number of input positions at which stimulation drives the neuron's activation above a threshold. The difference in adaptation level among the neurons was not large enough to produce significant change in RF size. The small differences in the adaptation level of Layer 2 neurons after complementary scotoma conditioning was manifested as small RF shifts (Figure 26c). Because neurons whose RF center was close to the complementary scotoma boundary were more adapted than the other neurons, they exerted less lateral inhibition on neurons whose initial RF center was away from the complementary scotoma boundary, and the RF center of neurons whose RF center was on either side of the complementary scotoma boundary shifted toward each other. If neurons whose RF center was close to the complementary scotoma boundary were less adapted than the other neurons, then they would have maximal RF size, and they would exert more lateral inhibition on neurons whose initial RF center was away from the complementary scotoma boundary; thus the RF centers of neurons whose RF centers were on either side of the complementary scotoma boundary would shift away from each other.

In the simulation of complementary scotoma conditioning of the adaptation network with no lateral interaction, Layer 2 neurons whose RF center was close to the complementary scotoma boundary were more adapted than other neurons. Thus, they may have a smaller RF than neurons whose initial RF center was away from the complementary scotoma boundary. As in the simulation of the inhibition-dominant adaptation model, the RF sizes of Layer 2 neurons were

almost the same (Figure 28a). No RF shifts occurred because there are no lateral interactions (Figure 29a). If Layer 2 neurons whose RF center was close to the complementary scotoma were less adapted than other neurons, they would have larger RFs than other neurons, and again there would not be any RF shifts.

Figure 28 near here.

Figure 29 near here.

In the simulation of complementary scotoma conditioning of the excitation-dominant adaptation, Layer 2 neurons whose RF center was close to the complementary scotoma boundary were less adapted than other neurons. Thus, they had larger RFs than neurons whose initial RF center was away from the complementary scotoma boundary (Figure 28b). Because neurons whose RF center was close to the complementary scotoma boundary were less adapted than the other neurons, they exerted more lateral excitation on neurons whose initial RF center was away from the complementary scotoma boundary, and the RF centers of neurons whose RF centers were on either side of the complementary scotoma boundary shifted toward each other (Figure 29b). If neurons whose RF center was close to the complementary scotoma boundary were more adapted than the other neurons, then they would have smaller RFs than other neurons, and they would exert less lateral excitation on neurons whose initial RF center was away from the complementary scotoma boundary; thus, the RF centers of neurons whose RF centers were on either side of the complementary scotoma boundary would shift away from each other.

4.2.1 Effects of network interactions on neuronal adaptation

In the adaptation networks, the complementary scotoma stimulation activates Layer 2 neurons with close to equal probability. The adaptation levels of Layer 2 neurons in the adaptation networks are not necessarily equal for the following reasons. (1) Neurons whose initial RF straddles the complementary scotoma boundary are activated slightly more often (although possibly at a lower intensity), and therefore these neurons may become slightly more adapted than other neurons. (2) Neurons whose RF center is close to the center of a hemifield have a large number of their afferent excitatory pathways excited when that hemifield is stimulated, whereas

neurons whose RF center is close to the complementary scotoma boundary have only a small number of their afferent excitatory pathways excited. Thus, neurons whose RF center is close to the center of a hemifield are likely to be more strongly activated and therefore more adapted than neurons whose RF center is close to the complementary scotoma boundary. (3) The difference in the amount of afferent excitation received by the Layer 2 neurons is further accentuated by the adaptation threshold. If Layer 2 neurons have large thresholds, neurons receiving larger afferent excitation are more likely to be activated and hence be more adapted. (4) In the inhibition-dominant adaptation network, neurons whose RF center is close to the complementary scotoma boundary receive less lateral inhibition than neurons whose RF center is close to the center of a hemifield, thus making neurons whose RF center is close to the complementary scotoma boundary likely to respond strongly and become more adapted. On the other hand, in the excitation-dominant adaptation network, neurons whose RF center is close to the complementary scotoma boundary receive less lateral excitation than neurons whose RF center is close to the center of a hemifield, thus making them respond weakly and become less adapted. The eventual distribution of adaptation levels will be determined by the more dominant factors.

4.3 Recovery of RF properties

Normal stimulation following complementary scotoma conditioning caused restoration of the original RF size and position in the EXIN and LISSOM networks with only lateral inhibitory synaptic plasticity enabled, in all the adaptation networks, and in the LISSOM network with only afferent synaptic plasticity enabled (Figures 24–29).

The EXIN network with only afferent excitatory synaptic plasticity enabled (Figures 25a and 27a) did not recover its original RF properties. The LISSOM network with only lateral excitatory synaptic plasticity enabled (Figures 25c and 27c) also did not recover some of its original RF properties, because as the lateral excitatory pathways weaken, effective inhibition between neurons increases, and increased lateral inhibition makes Layer 2 neurons less coactive.

4.4 Conclusions

Complementary scotoma conditioning revealed differences in the behaviors of the various models. Thus, a neurobiological experiment using complementary scotoma conditioning could discriminate

between the different models based on their predictions. The predictions of the different models are described below.

After complementary scotoma conditioning, the EXIN and the LISSOM network, with only their respective lateral inhibitory synaptic plasticity rules enabled, predict that cortical neurons whose initial RF centers were close to the complementary scotoma boundary would have larger RFs than neurons whose initial RF centers were far from the complementary scotoma boundary, and the initial RFs on opposite sides of the complementary scotoma boundary would shift toward each other, whereas the LISSOM network with only lateral excitatory synaptic plasticity and the EXIN and the LISSOM network with only afferent excitatory synaptic plasticity predict that neurons whose initial RF centers were close to the complementary scotoma boundary would have smaller RFs than neurons whose initial RF centers were far from the complementary scotoma boundary, and the initial RFs on opposite sides of the complementary scotoma boundary would shift away from each other.

After complementary scotoma conditioning, the adaptation network with no lateral interaction predicts that RFs of neurons whose initial RF centers were close to the complementary scotoma boundary would be smaller than, equal to, or greater than the RFs of other neurons, but no RF shifts would occur because the afferent weights were symmetric.

The inhibition-dominant adaptation network network predict that, complementary scotoma conditioning would (1) cause the RFs of neurons whose initial RF centers were close to the scotoma boundary to be smaller than the RFs of other neurons and cause the RFs on opposite sides of the complementary scotoma boundary to shift toward each other, or (2) cause the RFs of neurons whose initial RF centers were close to the scotoma boundary to be larger than the RFs of other neurons, and cause the RFs on opposite sides of the complementary scotoma boundary to shift away from each other. In the excitation-dominant network, however, when the RFs of neurons whose initial RF centers were close to the scotoma boundary are smaller (larger) than the RFs of other neurons, the RFs across the complementary scotoma boundary shift away from (shift toward) each other. The specific effects will depend on the conditions discussed in Section 4.2.1.

It is possible that the rates of change in the effective lateral excitation and the effective lateral inhibition may differ at short and long ranges. This can be probed by scotoma and

complementary scotoma conditioning. For example, if at short ranges effective lateral excitation decreases faster than lateral inhibition, then scotoma conditioning should produce RF contraction in neurons whose initial RF center is close to the scotoma boundary and RF expansion in neurons whose initial RF is inside and far from the scotoma boundary.

5 Discussion

The effects of the EXIN rules, the LISSOM rules, and the adaptation rule in response to artificial scotoma stimuli have been compared. The experimental data and the simulation predictions are summarized in Table 1. The entries in boldface indicate where the models are in agreement with experimental data.

Table 1 near here.

The EXIN network with outstar lateral inhibitory synaptic plasticity alone produced the following effects, corresponding closely to the reported neurophysiology. During scotoma/normal conditioning, the EXIN model with only outstar lateral inhibitory synaptic plasticity enabled produced

- centrifugal expansion of RFs that were initially *inside* the scotoma region;
- the greatest expansion for RFs closest to the scotoma boundary;
- RF expansion that exceeded the boundaries of the scotoma for RFs close to the scotoma boundary;
- increased response from the area of the initial RF (DeAngelis et al., 1994, 1995), without changes in spontaneous activation in the absence of visual stimulation (Pettet & Gilbert, 1992);
- asymmetric RF profiles in neurons with initial RF close to the scotoma boundary (Das & Gilbert, 1995b);
- RF contraction to original size during subsequent normal stimulation; and

- no RF changes in the absence of stimulation.

During complementary scotoma conditioning the EXIN network with only outstar lateral inhibitory synaptic plasticity enabled showed

- maximal RF size for neurons whose initial RF was on *either* side of the scotoma boundary; and
- a shift in RF of neurons whose initial RFs were close the scotoma boundary *toward* each other.

The EXIN model with only afferent excitatory synaptic plasticity enabled produced the following effects, different from those produced by the EXIN model with only lateral inhibitory synaptic plasticity enabled:

- neurons affected by scotoma conditioning *did not* recover their original RF size and RF position; and
- complementary scotoma conditioning caused RF *contraction* and minimal RF size in neurons whose initial RF was on either side of the scotoma boundary; and
- complementary scotoma conditioning shifted the RFs of neurons whose initial RFs were close to the scotoma boundary *away* from each other.

The other effects were the same as those produced by the EXIN model with only lateral inhibitory synaptic plasticity enabled (see Table 1).

The effects of scotoma conditioning and complementary scotoma conditioning the full EXIN network (with the afferent excitatory and the lateral inhibitory synaptic plasticity rules enabled) will depend on the relative rate of learning in the two rules. The effects obtained by disabling one of the two rules are at the ends of a continuum of effects produced by the EXIN rules. Because the two EXIN rules produce many common effects after scotoma conditioning (see Table 1), the full EXIN network too produces these effects. Unlike the EXIN network with only afferent excitatory synaptic plasticity, the full network can recover the

original RFs during normal conditioning following scotoma conditioning. Thus, the full EXIN network produces effects consistent with neurophysiological data on artificial scotoma conditioning. However, during complementary scotoma conditioning the two EXIN rules produce opposite effects (Table 1), and thus the overall effects produced by the full EXIN network will depend on the relative magnitudes of changes produced by the two rules.

The LISSOM network with only instar lateral inhibitory synaptic plasticity enabled produced the following effect inconsistent with neurophysiological data. In response to normal/scotoma conditioning, the network showed

- expansion of RFs that were initially *outside* the scotoma and close to the scotoma boundary.

The LISSOM network with afferent excitatory synaptic plasticity alone or instar lateral excitatory synaptic plasticity alone produced RF expansion in neurons whose initial RF was inside the scotoma during scotoma conditioning and produced effects similar to those produced by the EXIN network with only outstar lateral inhibitory synaptic plasticity enabled during scotoma conditioning. However, during complementary scotoma conditioning, the LISSOM network with instar afferent excitatory synaptic plasticity alone or instar lateral excitatory synaptic plasticity alone produced

- RF *contraction* and minimal RF size in neurons whose initial RF was on either side of the scotoma boundary; and
- shifts in RF of neurons whose initial RFs were close the scotoma boundary, *away* each other.

As in the full EXIN network, the effects produced by the full LISSOM network after scotoma conditioning and complementary scotoma conditioning will depend on the relative magnitudes of changes produced by the three rules in the LISSOM model. The important observation is that, after scotoma or complementary conditioning, many of the effects produced by the LISSOM lateral inhibitory synaptic plasticity rule are in conflict with those produced by the LISSOM afferent and lateral excitatory synaptic plasticity rules (see Table 1).

The inhibition-dominant adaptation model produced several effects (Xing & Gerstein, 1994) consistent with neurophysiological data. However, the inhibition-dominant adaptation network produced the following effect inconsistent with neurophysiological data:

- changes in RF size in the absence of stimulation.

The adaptation network with no lateral interaction and the excitation-dominant adaptation network produced RF expansion in neurons in the cortical scotoma. However, the RF size in these networks changed in the absence of stimulation, inconsistent with experimental data. In the adaptation network with no lateral interaction, the RFs remained symmetric, and RF positions did not change following scotoma conditioning. In the excitation-dominant adaptation network, the RFs shifted toward the scotoma center. These results are inconsistent with neurophysiological data.

After complementary scotoma conditioning the adaptation networks, the RFs of neurons whose initial RF centers were close to the complementary scotoma boundary may be smaller or larger than the RFs of other neurons; the RF size is parameter dependent. In the adaptation network with no lateral interaction no RF shifts occurred. In the inhibition-dominant adaptation network, when the RFs of neurons whose initial RF centers were close to the complementary scotoma boundary were larger than the RFs of other neurons, then the RFs on opposite sides of the boundary shifted away from each other, but when the RFs of neurons whose initial RF centers were close to the complementary scotoma boundary were smaller than the RFs of other neurons, then the RFs on opposite sides of the boundary shifted toward each other. In the excitation-dominant adaptation network, the relationship between the relative RF size and RF shift are opposite to that in the inhibition-dominant adaptation network.

The role of afferent excitatory synaptic plasticity rule in producing fast (on the order of minutes or hours) RF changes in adult animals may be very limited or non-existent. Restricted retinal lesion in cats produced RF changes in neurons in layers 3 and 4 of area 17 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 only a fast *instar* afferent excitatory synaptic plasticity rule (e.g., the EXIN and the LISSOM afferent excitatory synaptic plasticity rules), because active neurons would weaken their connections from the lesioned region, regardless of whether the other eye is open or closed, to produce changes in RF properties. Furthermore, changes in ocular dominance, which are presumed to be caused by changes in thalamocortical afferents (Clothiaux et al., 1991;

Miller et al., 1989), cannot be induced in adult animals by visual deprivation. However, dynamic RF changes because of afferent excitatory plasticity would be consistent with the results of Chino et al. (1992) *if the afferent excitatory plasticity occurs in pathways originating from a binocular layer*. With the unlesioned eye open, there will be no scotoma in the binocular layer, and hence afferent excitatory pathways from the binocular layer will not change. But, Chino et al. (1992) observed no change in RF of neurons in layer 4 of cat area 17 (which may receive monocular thalamocortical inputs) when the unlesioned eye was open. In the EXIN network with outstar lateral inhibitory synaptic plasticity alone, the LISSOM networks with instar lateral excitatory or instar lateral inhibitory synaptic plasticity alone, and the adaptation networks, with the unlesioned eye open there is no cortical scotoma; all binocular neurons in the networks are active during the conditioning and therefore the lateral weights in the EXIN or the LISSOM networks and the adaptation level in the inhibition-dominant adaptation network would remain almost unchanged. However, there might be some small changes in RF properties because some cortical neurons are binocularly activated while others are monocularly activated by the unlesioned eye.

Instar lateral excitatory synaptic plasticity alone in the LISSOM network decreases lateral excitatory connection weights from neurons inside the cortical scotoma to those outside the cortical scotoma. This directly reduced the RF size of neurons whose initial RF center is close to and outside the scotoma (Figure 23) and indirectly leads to RF expansion in neurons whose initial RF is inside the scotoma (Figure 23). In the EXIN model with lateral inhibitory synaptic plasticity alone, scotoma conditioning leads to weakening of the lateral inhibitory connections from neurons outside the cortical scotoma to those inside. This directly leads to RF expansion in neurons inside the cortical scotoma. The increased responsiveness of neurons inside the cortical scotoma then lets those neurons exert more inhibition on neurons outside the cortical scotoma, leading indirectly to RF contraction of neurons outside the cortical scotoma (Figures 7 and 10). Thus, these two models produce qualitatively similar results during scotoma conditioning; the results are closest to the experimental data on artificial scotoma conditioning and retinal lesions. However, as shown in Section 4, they exhibit different RF changes during complementary scotoma conditioning.

Grajski and Merzenich (1990) proposed a model with plasticity in afferent excitatory, lateral excitatory, and lateral inhibitory pathways, for RF changes following repetitive peripheral stimulation of a restricted skin region. Their plasticity rule is a covariance rule with normalization; pathway strength is weakened if either the source or the target neuron is inactive. Such a rule would produce roughly symmetric RF changes across the cortical scotoma boundary even during scotoma conditioning, contrary to the experimental data and contrary to the EXIN and LISSOM rules.

5.1 Models based on the EXIN and the LISSOM rules

Several visual functions and visual cortical properties have been modeled by the EXIN and the LISSOM rules.

The EXIN afferent excitatory and lateral inhibitory synaptic plasticity rules together have been used to model visual disparity selectivity (Marshall, 1990c), visual motion selectivity and grouping (Marshall, 1990a), visual inertia (Hubbard & Marshall, 1994), visual motion integration in the aperture problem (Marshall, 1990a), visual length selectivity and end-stopping (Marshall, 1990b), visual depth perception from occlusion events (Marshall & Alley, 1993; Marshall et al., 1996a), visual depth from motion parallax (Marshall, 1989), visual motion smearing (Martin & Marshall, 1993), visual orientation selectivity (Marshall, 1990d), and visual stereomatching (Marshall et al., 1996b).

The LISSOM rules have been used to model development of topographic RFs (Sirosh & Miikkulainen, 1994b, 1997), visual orientation tuning and orientation columns (Sirosh et al., 1996), ocular dominance columns (Sirosh & Miikkulainen, 1995, Sirosh et al., 1996), RF changes after cortical lesions (Sirosh & Miikkulainen, 1994a), and tilt aftereffects (Bednar & Miikkulainen, 1997).

Although, the EXIN and the LISSOM rules model some visual functions and cortical properties, they produce different effects after artificial scotoma conditioning. Thus, the analyses of the effects of the various synaptic plasticity rules during artificial scotoma conditioning provides a basis for determining the rules for cortical plasticity.

5.2 Transient response bias in RF measurements

The changes in RFs produced by the EXIN and the LISSOM rules persist after cessation of the conditioning stimuli. However, several results show that some dynamic RF changes produced by artificial scotoma conditioning are transient. For example, some RF changes occurred within seconds of the conditioning, and recovery time was also on the order of seconds, in the absence of stimulation (DeAngelis et al., 1995; Kapadia et al., 1994). These transient changes in RF can be modeled by the Xing and Gerstein (1994) inhibition-dominant adaptation model, operating at a fast time-scale (1–10 seconds). Adaptation could also be added to the EXIN and the LISSOM models to describe the transient RF changes. The transient RF effects appear to be a separate phenomenon from the RF dynamics that operate at a slower time scale (5–15 minutes).

5.3 Effect of orientation on RF dynamics

Pettet and Gilbert (1992) studied the effects of conditioning a neuron by presenting the artificial scotoma against a background of moving bars oriented orthogonally to the preferred orientation of the neuron (cross-orientation artificial scotoma). For a few neurons (3 out of 15), they found an expansion with iso-orientation conditioning stimuli and did not find an expansion with the orthogonal pattern. In these cases, the orthogonal pattern actually reduced the RF size and responsiveness of the neuron.

The EXIN rules predict that cross-orientation artificial scotoma conditioning of a neuron would produce less expansion than would iso-orientation artificial scotoma conditioning. Lateral inhibition between neurons becomes roughly proportional to the amount of overlap in their RFs, using the EXIN rules (Marshall, 1990bcd; 1992ab; 1995a). The EXIN model predicts strong iso-orientation inhibition and weak ortho-orientation inhibition (Marshall, 1990d) consistent with the results of Ferster (1989). During cross-orientation artificial scotoma conditioning, the decrease in lateral inhibition to the test neuron will be small because according to the EXIN rules, the ortho-orientation inhibition is small. In addition, the EXIN rules can model the shrinkage after cross-orientation artificial scotoma conditioning observed by Pettet and Gilbert (1992). The cross-orientation scotoma conditioning causes neurons with the near-orthogonal preferred

orientation within the cortical scotoma to become more responsive and to exert more inhibition on the recorded neuron. Further simulations are needed to demonstrate this prediction.

Gilbert and Wiesel (1990) found short term modifications in the orientation specificity of neurons, in response to contextual stimuli placed outside a neuron's RF. Presentation of differently oriented bars in the surround of a neuron's RF caused the neuron's tuning curve to shift. In some cases, the change persisted even after the removal of the surround stimuli. The most effective way of restoring the neuron's original orientation tuning was to stimulate the surround with lines of varying orientation, for a period of about 10 mins. This persistent effect may be produced by changes in the lateral inhibitory weights between neurons with different orientation specificity. An explanation of this effect based on synaptic changes may explain development of connection patterns that can produce the various kinds of contextual effects (Badcock & Westheimer, 1985; Butler & Westheimer, 1978; Westheimer, 1986, 1989; Westheimer & McKee, 1977; Westheimer et al., 1976).

5.4 Long-term effects of retinal lesions on RF properties

Darian-Smith and Gilbert (1995) found that about 5 minutes after bilateral retinal lesions neurons in the cortical scotoma became responsive to visual stimuli outside the lesioned regions. However, the neurons that acquired responsiveness to locations outside the lesioned retina were less responsive and more sluggish in their response compared to neurons in the normal cortex. The sluggish response of neurons in the recovered area of the cortical scotoma in the primary visual cortex may arise because these neurons receive afferent excitation via weak connections, which can activate these neurons because of the reduced lateral inhibition they receive, as a consequence of the EXIN lateral inhibitory synaptic plasticity rule, the EXIN or the LISSOM afferent excitatory synaptic plasticity rule, or the LISSOM lateral excitatory synaptic plasticity rule. It is also possible that the cortical recovery is aided by long-range lateral excitation or by feedback connections from other cortical layers (Darian-Smith & Gilbert, 1994, 1995), in addition to the changes in lateral inhibition in the primary visual cortex. Cortical scotoma in deeper layers can modify RF properties of neurons in the deeper layers, and these changes can affect RF properties in lower cortical layers via feedback pathways. Within the EXIN learning

framework, feedback connections have been used in the representation of oblique and transparent surfaces defined by stereo disparity (Marshall & Kalarickal, 1995; Marshall et al., 1996b) and in motion grouping (Schmitt & Marshall, 1995, 1996).

The cortical reorganization occurring over a period of weeks and months following retinal lesions may also involve the sprouting and establishment of new connections – either synaptogenesis along existing fibers or the physical extension of axonal/dendritic terminals in addition to synaptogenesis (Darian-Smith & Gilbert, 1994).

Even after several months after the retinal lesions, a small region of the cortex remained unresponsive to visual stimulation in the unlesioned retinal regions. Darian-Smith and Gilbert (1995) invoke physical limits on the extent of changes in the horizontal connections to explain the existence of the persistent deprived cortical region.

5.5 Role of lateral excitatory pathways in RF properties

The LISSOM network has been used to model self-organization of topographic RF organization and ocular dominance columns and the effects of cortical lesions (Sirosh & Miikkulainen, 1994ab, 1995, 1997). In the LISSOM network, topographically ordered RFs develop if the initial afferent connections are ordered in overlapping patches and the synaptic weights are random (Sirosh & Miikkulainen, 1997). This possibility suggests that undeveloped cortex with input afferents ordered in overlapping connections but with random synaptic weights can develop into topographically organized cortex.

The EXIN network described in this paper does not have lateral excitatory connections. Lateral excitatory connections with signal transmission latencies have been used in conjunction with the EXIN rules to model several aspects of visual motion perception (Hubbard & Marshall, 1994; Marshall, 1989, 1990a, 1991, 1995b; Marshall & Alley, 1993; Martin & Marshall, 1993). The EXIN lateral connectivity pattern can be viewed of as a limiting case of the LISSOM connectivity pattern, when the lateral excitatory zone of a Layer 2 neuron contains only itself. Smaller lateral excitatory zones lead to smaller regions of topographic ordering. Like the LISSOM network, the EXIN network could show local topographic ordering if it had lateral excitatory connection pathways.

5.6 Significance of the EXIN lateral inhibitory plasticity rule

In the EXIN model, strong lateral inhibitory pathways develop between neurons that are consistently coactivated. Neurons can be consistently coactivated if they receive excitatory afferents from many common input neurons. Thus, in the EXIN model, cortical neurons that share inputs have strong lateral inhibitory pathways between them. This is consistent with experimental results suggesting that a neuron receives the strongest inhibition when its stimuli are most similar to the preferred stimuli of the neuron (Blakemore & Tobin, 1972; DeAngelis et al., 1992; Ferster, 1989).

The EXIN lateral inhibitory plasticity rule has several desirable functional properties. The inhibitory synaptic plasticity rule leads to improved stimulus discrimination, sparse and distributed coding, and exclusive allocation (Marshall, 1995a; Marshall & Gupta, 1997). The EXIN synaptic plasticity rules have been used to model the development of disparity selectivity (Marshall, 1990c), motion selectivity and grouping (Marshall, 1990a, 1995b; Schmitt & Marshall, 1995), orientation selectivity (Marshall, 1990d), and length selectivity and end-stopping (Marshall, 1990b).

Note that the EXIN lateral inhibitory plasticity rule (Equation 1) is an asymmetric rule; lateral inhibitory pathways from active neurons to inactive weaken, however, lateral inhibitory pathways from inactive neurons to other neurons *do not* change. This asymmetry makes it possible to produce RF expansion in the inactive neurons in response to artificial scotoma conditioning and retinal lesions, without necessarily producing RF expansion in neurons that are activated. The EXIN lateral inhibitory synaptic plasticity rule directly reduces inhibition to neurons inactivated by peripheral scotomas or lesions, thus making them more likely to respond to some visual stimuli. The EXIN lateral inhibitory synaptic plasticity rule enhances the efficiency of a neural network's representation of perceptual patterns, by recruiting unused and under-used neurons to represent input patterns (Marshall, 1995a; Marshall & Gupta, 1998). In comparison, the LISSOM lateral inhibitory synaptic plasticity rule weakens lateral inhibitory pathways *from inactive neurons to active neurons*, thereby tending to make the active neurons more strongly active and to suppress the inactive neurons more strongly. In the LISSOM model,

neurons that are inactive or very weakly active because of a peripheral scotoma or because of weak afferent excitatory pathways can become more responsive to some specific visual feature only *indirectly* via weakening of either afferent excitatory pathways to other active neurons or lateral excitatory pathways to other active neurons.

Thalamocortical afferent arbors can spread over a large cortical area; thalamocortical afferents from the lateral geniculate nucleus can extend over a region up to 3 mm in cat primary visual cortex (Humphrey et al., 1985). Gilbert & Wiesel (1983) observed thalamocortical arbors that extended 2 mm in layer 4 of primary visual cortex of cats. Interlaminar excitatory pathways in the primary visual cortex of cats spread over a few millimeters (Gilbert & Wiesel, 1983). Thus, large overlap in the afferent excitatory inputs to model neurons in the simulations is reasonable.

In animal cortex, lateral pathways spread over large distances. Axonal arbors of GABAergic large basket neurons extend up to 1.5 mm in cortex and terminate on the soma of pyramidal neurons in small patches of cortex (Somogyi et al., 1983; Somogyi & Martin, 1985). Based on the anatomical structure of the axonal arbors of basket neurons, these neurons appear to have the greatest effect on neurons with orientation selectivity similar to their own; however, they may affect neurons with other orientations and other RF positions (Martin, 1988). Long-range inhibitory influences in cortex may also be subserved by the long-range horizontal pathways that extend 2–8 mm in primary visual cortex of cat (Gilbert & Wiesel, 1983, 1989). The long-range horizontal pathways have an excitatory effect at low stimulation strength and have an inhibitory effect at high stimulation strength (Gil & Amitai, 1996; Weliky et al., 1995). Furthermore, the excitatory and inhibitory effects of the long-range horizontal connections are concentrated on neurons with similar orientation selectivity to that of the source neuron (Weliky et al., 1995). Combined measurement of spiking point-spread using extracellular recording and optical point-spread in cat primary visual cortex showed that the spiking point-spread accounts for only 5% of the optical point-spread; the remainder of the optical point-spread was largely caused by inhibition (Das & Gilbert, 1995a). The optical point-spread had a diameter between 3.2 and 5.2 mm and showed greatest magnitude for cortical neurons with similar stimulus orientation preference to that of the spiking neurons.

These data are consistent with the suggestion that cortical neurons with common inputs, and

hence similar properties, should have relatively strong lateral inhibitory pathways between them, for improved stimulus discrimination (e.g., orientation selectivity, disparity selectivity, length selectivity, spatial frequency selectivity, motion direction selectivity) and sparse distributed coding. Thus, lateral inhibitory plasticity may play an active and important role in the development of cortical function. An alternative is to hardwire lateral inhibitory pathway weights as a function of cortical distance. However, the strength of lateral inhibitory pathways in primary visual cortex is not uniform, but depends on topographical organization of RF properties such as orientation selectivity (Weliky et al., 1995).

5.7 Neurophysiological realization of the EXIN lateral inhibitory plasticity rule

The EXIN model is a functional model that describes modifications in the effective synaptic weights, including modifications in effective lateral inhibitory weights. *In vivo*, intracortical inhibition is mediated by inhibitory interneurons, which receive excitation from excitatory neurons in addition to afferent input (Douglas & Martin, 1991; Somogyi, 1989).

Neurophysiologically, the EXIN lateral inhibitory synaptic plasticity rule could be realized in a disynaptic circuit containing a lateral excitatory horizontal connection (either short- or long-range) and an inhibitory interneuron, either by modifying the excitatory weights from the excitatory neuron or by changing the inhibitory weight from the inhibitory neuron (Darian-Smith & Gilbert, 1994, 1995; Das & Gilbert, 1995ab; Gilbert et al., 1996; Hirsch & Gilbert, 1993). The axonal arbors of many inhibitory neurons (e.g., clutch, basket, chandelier) terminate mainly on excitatory neurons (Somogyi, 1989; Somogyi & Martin, 1985), and axonal arbors of most excitatory neurons terminate on other excitatory neurons (McGuire et al., 1991; Somogyi, 1989; Somogyi & Martin, 1985). During development, lateral pathways in the primary visual cortex are initially widespread, and then develop into clustered patches (Katz & Callaway, 1992; Dalva & Katz, 1994). The development of the lateral connectivity depends on external input (Katz & Callaway, 1992). The axonal arbors of inhibitory large basket neurons are also clustered (Somogyi & Martin, 1985) and may develop from initially widespread pathways during development, suggesting that there is synaptic plasticity in these connections. Stimulation

of the long-range horizontal excitatory pathways produce excitatory and inhibitory effects on excitatory neurons (Gil & Amitai, 1996; Weliky et al., 1995). Thus, changing the efficacy of lateral inhibitory pathways or the lateral excitatory pathways to inhibitory neurons will change effective inhibition to cortical neurons. Hirsch & Gilbert (1993) have suggested that long-term depression could be a decrease in the strength of excitatory connections or an increase in the strength of inhibitory connections. If the synapses of the long-range excitatory connections to both excitatory and inhibitory neurons change, then to be consistent with the EXIN inhibitory rule, the synapses onto inhibitory neurons should change faster than the synapses onto excitatory neurons, so that the overall effect is a change in lateral inhibition.

Plasticity in inhibitory synapses would be more likely to produce large RF changes than plasticity in excitatory synapses on inhibitory interneurons. The axonal arbors of many inhibitory neurons (e.g., clutch, basket, chandelier) terminate mainly on excitatory neurons (Somogyi, 1989; Somogyi & Martin, 1985), and axonal arbors of most excitatory neurons terminate on other excitatory neurons (McGuire et al., 1991; Somogyi, 1989; Somogyi & Martin, 1985).

Thalamocortical stimulation produces a monosynaptic EPSP and a disynaptic IPSP in primary visual cortical neurons, but disynaptic EPSPs are rarely produced (Gil & Amitai, 1996; Ferster, 1989). Neurons receive disynaptic IPSPs because of thalamocortical excitation at all stimulation intensities that evoke early EPSPs (Gil & Amitai, 1996). Weak stimulation of the long-range horizontal excitatory pathways produces excitatory effects on excitatory neurons, but strong stimulation leads to inhibition of excitatory neurons (Gil & Amitai, 1996; Weliky et al., 1995); this implies that inhibitory interneurons are inactive or very weakly active during weak stimulation of the long-range horizontal excitatory pathways. The above data suggest that the inhibition received by excitatory neurons from inhibitory interneurons tends to be stronger than the lateral excitation received by the excitatory neurons. Thus, changing the efficacy of lateral inhibitory pathways directly (using the EXIN lateral inhibitory synaptic plasticity rule) may affect RF properties more drastically than changing lateral excitatory pathways to inhibitory neurons or lateral excitatory pathways to excitatory neurons (e.g., using the LISSOM lateral excitatory synaptic plasticity rule).

5.8 Conclusions

The major conclusions of this paper are:

1. the subtle distinction between instar and outstar rules produces a dramatic difference in neural behavior and plasticity;
2. the outstar EXIN lateral inhibitory and the instar LISSOM lateral excitatory synaptic plasticity rules are sufficient to produce effects consistent with neurophysiological data on RF changes after artificial scotoma conditioning and retinal lesions in adult animals;
3. the instar LISSOM lateral inhibitory synaptic plasticity rule produces effects contrary to experimental data;
4. the adaptation networks do not produce stable RF changes after scotoma conditioning; and
5. synaptic plasticity in afferent excitatory pathways does not contribute to RF changes after artificial scotoma conditioning and retinal lesions.

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References

- [1] Badcock, D.R. & Westheimer, G. (1985). Spatial location and hyperacuity: The center-surround localization function has two substrates. *Vision Research*, **25**, 1259–1269.
- [2] Bednar, J.A. & Miikkulainen, R. (1997). A neural network model of visual tilt aftereffect. In *Proceedings of the 19th Annual Conference of the Cognitive Science Society*, pp. 37–42. Hillsdale, NJ: Erlbaum.
- [3] Blakemore, C. & Tobin, E.A. (1972). Lateral inhibition between orientation detectors in the cat’s visual cortex. *Experimental Brain Research*, **15**, 439–440.
- [4] Butler, T. & Westheimer, G. (1978). Interference with stereoscopic acuity: Spatial, temporal, and disparity tuning. *Vision Research*, **18**, 1387–1392.
- [5] Chapman, B. & Stone, L.S. (1996). Turning a blind eye to cortical receptive fields. *Neuron*, **16**, 9–12.
- [6] Chino, Y.M., Kaas, J.H., Smith III, E.L., Langston, A.L., & Cheng, H. (1992). Rapid reorganization of cortical maps in adult cats following restricted deafferentation in retina. *Vision Research*, **32**(5), 789–796.
- [7] Clothiaux, E.C., Bear, M.F., & Cooper, L.N. (1991). Synaptic plasticity in visual cortex: Comparison of theory with experiment. *Journal of Physiology*, **66**, 1785–1804.
- [8] Cohen, M.A. & Grossberg, S. (1983). Absolute stability of global pattern formation and parallel memory storage by competitive neural networks. *IEEE Transactions on Systems, Man, and Cybernetics*, **13**(5), 815–826.
- [9] Crist, R.E., Kapadia, M.K., Westheimer, G., & Gilbert, C.D. (1997). Perceptual learning of spatial localization: Specificity for orientation, position, and context. *Journal of Neurophysiology*, **78**(6), 2889–2894.
- [10] Dalva, M.B. & Katz, L.C. (1994). Rearrangements of synaptic connections in visual cortex revealed by laser photostimulation *Science*, **265**, 255–258.

- [11] Darian-Smith, C. & Gilbert, C.D. (1994). Axonal sprouting accompanies functional reorganization in adult cat striate cortex. *Nature*, **368**, 737–740.
- [12] Darian-Smith, C. & Gilbert, C.D. (1995). Topographic reorganization in the striate cortex of the adult cat and monkey is cortically mediated. *The Journal of Neuroscience*, **15**(3), 1631–1647.
- [13] Das, A. (1997). Plasticity in adult sensory cortex: A review. *Network: Computation in Neural Systems*, **8**, R33–R76.
- [14] Das, A. & Gilbert, C.D. (1995a). Long-range horizontal connections and their role in cortical reorganization revealed by optical recording of cat primary visual cortex. *Nature*, **375**, 780–784.
- [15] Das, A. & Gilbert, C.D. (1995b). Receptive field expansion in adult visual cortex is linked to dynamic changes in strength of cortical connections. *Journal of Neurophysiology*, **74**(2), 779–792.
- [16] DeAngelis, G.C., Anzai, A., Ohzawa, I., & Freeman, R.D. (1992). Organization of suppression in receptive fields of neurons in cat visual cortex. *Journal of Neurophysiology*, **68**(1), 144–163.
- [17] DeAngelis, G.C., Anzai, A., Ohzawa, I., & Freeman, R.D. (1995). Receptive field structure in the visual cortex: Does selective stimulation induce plasticity? *Proceedings of the National Academy of Sciences of the USA*, **92**, 9682–9686.
- [18] DeAngelis, G.C., Ohzawa, I., Anzai, A., & Freeman, R.D. (1994). Does an artificial scotoma induce plasticity of receptive field structure for neurons in the visual cortex? *Investigative Ophthalmology & Visual Science*, **35**(4), 1468.
- [19] Douglas, R.J. & Martin, K.A.C. (1991). A functional microcircuit for cat visual cortex. *Journal of Physiology*, **440**, 735–769.
- [20] Fahle, M. (1997). Specificity of learning curvature, orientation, and vernier discriminations. *Vision Research*, **37**(14), 1885–1895.

- [21] Fahle, M. & Edelman, S. (1993). Long-term learning in vernier acuity: Effects of stimulus orientation, range and of feedback. *Vision Research*, **33**, 397–412.
- [22] Ferster, D.L. (1989). The synaptic inputs to simple cells of the cat visual cortex. In *Proceedings of the Retina Research Foundation, Vol. 2: Neural Mechanisms of Visual Perception*, ed. Lam, D.M. & Gilbert, C.D., pp. 63–85. Woodlands, TX: Portfolio.
- [23] Fiorentini, A. & Berardi, N. (1980). Perceptual learning is specific for orientation and spatial frequency. *Nature*, **287**, 43–44.
- [24] Gil, Z. & Amitai, Y. (1996). Spatial integration and cortical dynamics. *The Journal of Neuroscience*, **16**(20), 6567–6578.
- [25] Gilbert, C.D. (1996). Plasticity in visual perception and physiology. *Current Opinion in Neurobiology*, **6**(2), 269–274.
- [26] Gilbert, C.D. (1998). Adult cortical dynamics. *Physiological Review*, **78**(2), 467–485.
- [27] Gilbert, C.D., Das, A., Ito, M., Kapadia, M., & Westheimer, G. (1996). Spatial integration and cortical dynamics. *Proceedings of the National Academy of Sciences of the USA*, **93**, 615–622.
- [28] Gilbert, C.D. & Wiesel, T.N. (1983). Clustered intrinsic connections in cat visual cortex. *The Journal of Neuroscience*, **3**(5), 1116–1133.
- [29] Gilbert, C.D. & Wiesel, T.N. (1989). Columnar specificity of intrinsic horizontal and corticocortical connections in cat visual cortex. *The Journal of Neuroscience*, **9**(7), 2432–2442.
- [30] Gilbert, C.D. & Wiesel, T.N. (1990). The influence of contextual stimuli on the orientation selectivity of cells in primary visual cortex of the cat. *Vision Research*, **30**, 11, 1689–1701.
- [31] Grossberg, S. (1972). Neural expectation: Cerebellar and retinal analogs of cells fired by learnable or unlearned pattern classes. *Kybernetik*, **10**, 49–57.

- [32] Grossberg, S. (1982). *Studies of Mind and Brain*. Boston: Reidel Press.
- [33] Hammond, P., Mouat, G.S., & Smith, A.T. (1986). Motion after-effects in cat striate cortex elicited by moving textures. *Vision Research*, **26**(7), 1055–1060.
- [34] Hammond, P., Pomfrett, C.J.D., & Ahmed, B. (1989). Neural motion after-effects in the cat's striate cortex: Orientation selectivity. *Vision Research*, **29**(12), 1671–1683.
- [35] Hirsch, J.A. & Gilbert, C.D. (1993). Long-term changes in synaptic strength along specific intrinsic pathways in the cat visual cortex. *Journal of Physiology*, **461**, 247–262.
- [36] Hodgkin, A.L. (1964). *The Conduction of the Nervous Impulse*. Liverpool, England: Liverpool University Press.
- [37] Hubbard, R.S. & Marshall, J.A. (1994). Self-organizing neural network model of the visual inertia phenomenon in motion perception. Technical Report 94-001, University of North Carolina at Chapel Hill, Department of Computer Science, 26 pp.
- [38] Hubel, D.H. & Wiesel, T.N. (1965). Binocular interaction in striate cortex of kitten reared with artificial squint. *Journal of Neurophysiology*, **28**, 1041–1059.
- [39] Hubel, D.H. & Wiesel, T.N. (1970). The period of susceptibility to the physiological effects of unilateral eye closure in kittens. *Journal of Physiology*, **206**, 419–436.
- [40] Hubel, D.H., Wiesel, T.N., & LeVay, S. (1977). Plasticity of ocular dominance columns in monkey striate cortex. *Philosophical Transactions of the Royal Society* (London), **278**, 377–409.
- [41] Humphrey, A.L., Sur, M., Ulrich, D.J., & Sherman, S.M. (1985). Receptive fields, binocular interaction, and functional architecture in the cat's visual cortex. *The Journal of Comparative Neurology*, **233**, 159–189.
- [42] Kalarickal, G.J. & Marshall, J.A. (1996a). Dynamic receptive fields in visual cortex: Adaptive neural model and predictions. *Investigative Ophthalmology & Visual Science*, **37**(3), 483.

- [43] Kalarickal, G.J. & Marshall, J.A. (1996b). Dynamic cortical receptive field changes: Neuronal adaptation or connection plasticity? *Society for Neuroscience Abstracts*, **22**, 1613.
- [44] Kapadia, M.K., Gilbert, C.D., & Westheimer, G. (1994). A quantitative measure for short-term cortical plasticity in human vision. *The Journal of Neuroscience*, **14**(1), 451–457.
- [45] Karni, A. Sagi, D. (1991). Where practice makes perfect in texture discrimination: Evidence for primary visual cortex plasticity. *Proceedings of the National Academy of Sciences of the USA*, **88**, 4966–4970.
- [46] Katz, L.C. & Callaway, E.M. (1992). Development of local circuits in mammalian visual cortex. *Annual Review of Neuroscience*, **15**, 31–56.
- [47] Kirkwood, A., Dudek, S.M., Gold, J.T., Aizenman, C.D., & Bear, M.F. (1993). Common forms of synaptic plasticity in the hippocampus and neocortex *in vitro*. *Science*, **260**, 1518–1521.
- [48] Marshall, J.A. (1989). Self-organizing neural network architectures for computing visual depth from motion parallax. In *Proceedings of the International Joint Conference on Neural Networks*, Washington DC, **II**, pp. 227–234.
- [49] Marshall, J.A. (1990a). Self-organizing neural networks for perception of visual motion. *Neural Networks*, **3**, 45–74.
- [50] Marshall, J.A. (1990b). Development of length-selectivity in hypercomplex-type cells. *Investigative Ophthalmology & Visual Science*, **31**(4), 397.
- [51] Marshall, J.A. (1990c). A self-organizing neural network for computing stereo disparity and transparency. *Technical Digest, Optical Society of America Annual Meeting*, 268.
- [52] Marshall, J.A. (1990d). Adaptive neural methods for multiplexing oriented edges. In *Intelligent Robots and Computer Vision IX: Neural, Biological, and 3-D Methods*, ed. Casasent, D.P., Proceedings of the SPIE, **1382**, Boston, MA, pp. 282–291.

- [53] Marshall, J.A. (1991). Challenges of vision theory: Self-organization of neural mechanisms for stable steering of object-grouping data in visual motion perception. In *Stochastic and Neural Methods in Signal Processing, Image Processing, and Computer Vision*, ed. Chen, S.-S., Proceedings of the SPIE, **1569**, San Diego, CA, pp. 200–215.
- [54] Marshall, J.A. (1992a). Development of perceptual context-sensitivity in unsupervised neural networks: Parsing, grouping, and segmentation. In *Proceedings of the International Joint Conference on Neural Networks*, **III**, Baltimore, MD, pp. 315–320.
- [55] Marshall, J.A. (1992b). Unsupervised learning of contextual constraints in neural networks for simultaneous visual processing of multiple objects. In *Neural and Stochastic Methods in Image and Signal Processing*, ed. Chen, S.-S., Proceedings of the SPIE, **1766**, San Diego, CA, pp. 84–93.
- [56] Marshall, J.A. (1995a). Adaptive perceptual pattern recognition by self-organizing neural networks: Context, uncertainty, multiplicity, and scale. *Neural Networks*, **8**, 335–362.
- [57] Marshall, J.A. (1995b). Visual motion perception: Self-organization. In *The Handbook of Brain Theory and Neural Networks*, ed. Arbib, M., pp. 589–591. Cambridge, MA: MIT Press.
- [58] Marshall, J.A. & Alley, R.K. (1993). A self-organizing neural network that learns to detect and represent visual depth from occlusion events. In *Proceedings of the AAAI Fall Symposium on Machine Learning and Computer Vision*, ed. Bowyer, K. & Hall, L., pp. 70–74.
- [59] Marshall, J.A., Alley, R.K., & Hubbard, R.S. (1996a) Learning to predict visibility and invisibility from occlusion events. In *Advances in Neural Information Processing Systems*, **8**, ed. Touretzky, D.S., Mozer, M.C., & Hasselmo, M.E., pp. 816–822. Cambridge, MA: MIT Press.
- [60] Marshall, J.A. & Gupta, V.S. (1998). Generalization and exclusive allocation of credit in unsupervised category learning. *Network: Computation in Neural Systems*, **9**, 279–302.
- [61] Marshall, J.A. & Kalarickal, G.J. (1995). A neural circuit for stereomatching of transparent and oblique surfaces. *Investigative Ophthalmology & Visual Science*, **36**(4), 366.

- [62] Marshall, J.A. & Kalarickal, G.J. (1997). Modeling dynamic receptive field changes in primary visual cortex using inhibitory learning. In *Computational Neuroscience: Trends in Research, 1997*, ed. Bower, J.M., pp. 395–401. New York: Plenum Press.
- [63] Marshall, J.A., Kalarickal, G.J., & Graves, E.B. (1996b). Neural model of visual stereomatching: Slant, transparency, and clouds. *Network: Computation in Neural Systems*, **7**, 635–669.
- [64] Martin, K.E. & Marshall, J.A. (1993). Unsmearing visual motion: Development of long-range horizontal intrinsic connections. In *Advances in Neural Information Processing Systems Networks*, **5**, ed. Hanson, S.J., Cowan, J.D., & Giles, C.L., pp. 417–424. San Mateo, CA: Morgan Kaufmann Publishers.
- [65] McGuire, B.A., Gilbert, C.D., Rivlin, P.K., & Wiesel, T.N. (1991). Targets of horizontal connections in macaque primary visual cortex. *The Journal of Comparative Neurology*, **305**, 370–392.
- [66] Miller, K.D., Keller, J.B., & Stryker, M.P. (1989). Ocular dominance column development: Analysis and simulation. *Science*, **245**, 605–615.
- [67] Movshon, J.A., Thompson, I.D., & Tolhurst, D.J. (1978). Spatial summation in the receptive fields of simple cells in the cat’s striate cortex. *Journal of Physiology*, **283**, 53–77.
- [68] Petersen, R. & Taylor, J. (1997). Short-term cortical reorganisation in a lateral inhibitory neural network. In *Computational Neuroscience: Trends in Research, 1997*, ed. Bower, J.M., pp. 449–453. New York: Plenum Press.
- [69] Pettet, M.W. & Gilbert, C.D. (1992). Dynamic changes in receptive-field size in cat primary visual cortex. *Proceedings of the National Academy of Sciences of the USA*, **89**(7), 8366–8370.
- [70] Recanzone, G.H., Merzenich, M.M., & Dinse, H.R. (1992). Expansion of the cortical representation of a specific skin field in primary somatosensory cortex by intracortical microstimulation. *Cerebral Cortex*, **2**, 181–196.

- [71] Recanzone, G.H., Merzenich, M.M., Jenkins, W.M., Grajski, K.A., & Dinse, H.R. (1992). Topographic reorganization of the hand representation in cortical area 3b of owl monkeys trained in a frequency-discrimination task. *Journal of Neurophysiology*, **67**(5), 1031–1056.
- [72] Schmitt, C.P. & Marshall, J.A. (1995). Development of a neural circuit for motion grouping and disambiguation. *Investigative Ophthalmology & Visual Science*, **36**(4), 377.
- [73] Schmitt, C.P. & Marshall, J.A. (1996). Effects of coherence and context on visual motion grouping: A self-organizing neural model. *Investigative Ophthalmology & Visual Science*, **37**(4), 483.
- [74] Sirosh, J. & Miikkulainen, R. (1994a). Modeling cortical plasticity based on adapting lateral interaction. In *The Neurobiology of Computation: Proceedings of the Third Annual Computational and Neural Systems Conference*, ed. Bower, J.M., pp. 305–310. Boston, MA: Kluwer Academic Publishers.
- [75] Sirosh, J. & Miikkulainen, R. (1994b). Cooperative self-organization of afferent and lateral connections in cortical maps. *Biological Cybernetics*, **71**, 66–78.
- [76] Sirosh, J. & Miikkulainen, R. (1995). Ocular dominance and patterned lateral connections in a self-organizing model of the primary visual cortex. In *Advances in Neural Information Processing Systems*, **7**, ed. Tesauro, G., Touretzky, D.S., & Leen, T.K., pp. 109–116. Cambridge, MA: MIT Press.
- [77] Sirosh, J., Miikkulainen, R., & Bednar, J.A. (1996). Self-organization of orientation maps, lateral connections, and dynamic receptive fields in the primary visual cortex. In *Lateral Interactions in the Cortex: Structure and Function*, ed. Sirosh, J., Miikkulainen, R., & Choe, Y., UTCS Neural Networks Research Group, Austin, TX, 1996. Electronic book, ISBN 0-9647060-0-8, <http://www.cs.utexas.edu/users/nn/web-pubs/htmlbook96>.
- [78] Sirosh, J. & Miikkulainen, R. (1997). Topographic receptive fields and patterned lateral interaction in a self-organizing model of the primary visual cortex. *Neural Computation*, **9**, 577–594.

- [79] Somogyi, P. (1989). Synaptic organization of GABAergic neurons and GABA_A receptors in the lateral geniculate nucleus and visual cortex. In *Proceedings of the Retina Research Foundation, Vol. 2: Neural Mechanisms of Visual Perception*, ed. Lam, D.M. & Gilbert, C.D., pp. 35–62. Woodlands, TX: Portfolio.
- [80] Somogyi, P., Kisvárdy, Z.F., Martin, K.A.C., & Whitteridge, D. (1983). Synaptic connections of morphologically identified and physiologically characterized large basket cells in the striate cortex of cat. *Neuroscience*, **10**, 261–294.
- [81] Somogyi, P. & Martin, K.A.C. (1985). Cortical circuitry underlying inhibitory processes in cat area 17. In *Models of the Visual Cortex*, ed. Rose, D. & Dobson, V.G., pp. 514–523. New York: John Wiley & Sons.
- [82] Todorov, E.V., Siapas, A.G., Somers, D.C., & Nelson, S.B. (1997). Modeling visual cortical contrast adaptation effects. In *Computational Neuroscience: Trends in Research, 1997*, ed. Bower, J.M., pp. 525–531. New York: Plenum Press.
- [83] Volchan, E. & Gilbert, C.D. (1995). Interocular transfer of receptive field expansion in cat visual cortex. *Vision Research*, **35**, 1–6.
- [84] Weliky, M., Kandler, K., Fitzpatrick, D., & Katz, L.C. (1995). Patterns of excitation and inhibition evoked by horizontal connections in visual cortex share a common relationship to orientation columns. *Neuron*, **15**, 541–552.
- [85] Westheimer, G. (1986). Spatial interaction in the domain of disparity signals in human stereoscopic vision. *Journal of Physiology*, **370**, 619–629.
- [86] Westheimer, G. (1989). Line orientation considered as a separate psychophysical domain. In *Proceedings of the Retina Research Foundation, Vol. 2: Neural Mechanisms of Visual Perception*, ed. Lam, D.M. & Gilbert, C.D., pp. 237–246. Woodlands, TX: Portfolio.
- [87] Westheimer, G. & McKee, S.P. (1977). Spatial configurations for visual hyperacuity. *Vision Research*, **17**, 941–949.

- [88] Westheimer, G., Shimamura, K., & McKee, S.P. (1976). Interference with line orientation sensitivity. *Journal of the Optical Society of America A*, **66**, 332–338.
- [89] Xing, J. & Gerstein, G.L. (1994). Simulation of dynamic receptive fields in primary visual cortex. *Vision Research*, **34**, 1901–1911.

6 Appendix

The parameter values used to simulate the three models were chosen as follows. The notation $[a]$ is defined to mean $\max(0, a)$.

6.1 Parameters for the EXIN model simulations

The following parameters were used in all the EXIN simulations. To compute the initial weights, $\sigma_{\mathbf{ff}} = 1.41$, $\Psi = 0.2$, $\Gamma_{\mathbf{ff}} = 0.01$, $\Omega = 0.45$, and $\Gamma_{\mathbf{i}} = 0$ were used.

The parameters used for computing the activations were $A = 0.2$, $B = 2$, $C = 0.3$, $\beta = 0.1$, and $\gamma = 0.2$. The activation equation for Layer 2 neurons were numerically integrated using the Euler method with a time step of 0.2. The initial activation level of all the neurons was zero. The simulations were stopped at time = 110. At time = 110, the network was close to an equilibrium state by time = 110; the maximal change in Layer 2 neuronal activation at that point was less than 10^{-5} .

In the simulation, the weights were modified after the Layer 2 activations reached equilibrium on each input presentation. To compute the lateral inhibitory weight changes, parameters $\delta = 0.2$, $\mathcal{G}(a) = [a]$, and $\mathcal{Q}(a) = 3[a]$ were used. To compute the afferent excitatory weight changes, $\epsilon = 0.0016$, $\mathcal{F}(a) = [a]$, and $\mathcal{H}(a) = 0.4[a]$ were used. When the afferent excitatory synaptic plasticity was enabled, the initially zero afferent weights are not changed. This was done to speedup convergence of the afferent weights.

6.1.1 Parameters for the activation equation

The parameters of the activation equation were chosen so that the network gave a distributed activation response to an input, instead of a winner-take-all response.

At equilibrium, $\frac{d}{dt}x_{pq} = 0$ and the activation levels of Layer 2 neurons are given by

$$x_{pq} = \frac{\beta B E_{pq} - \gamma C I_{pq}}{A + \beta E_{pq} + \gamma I_{pq}} \quad (20)$$

Because Equation 3 is a shunting equation, $x_{pq}(t) \in [-C, B]$ if $x_{pq}(0) \in [-C, B]$, for all $t \geq 0$ (Cohen & Grossberg, 1983). Thus, B is the maximum activation level and $-C$ is the minimum activation level of Layer 2 neurons. The constant A determines the passive decay rate.

Increasing β is equivalent to increasing the input strength. As β is increased, the activation levels of neurons receiving large excitation increase and the activation levels of neurons receiving zero or very weak excitation are suppressed. This property of the shunting equation is shown in Figure 30a. Note that as β is increased (by a factor of 21), the Layer 2 activation profile expands very little. The activation level of neurons receiving strong excitation increases, and the activation level of neurons receiving very weak or zero excitation is further suppressed. As γ is increased, the activation levels of neurons first decrease and the response profile becomes narrower. At very large values of γ , the network behaves in a winner-take-all manner (Figure 30b).

Figure 30 near here.

Increasing B is similar to increasing β . This property of the shunting equation is shown in Figure 30c. Increasing C causes the Layer 2 activation profile to shift downwards. This property of the shunting equation is shown in Figure 30d. At very large values of C , the network behaves in a winner-take-all manner.

The parameters were chosen so that none of the terms in the shunting equation (Equation 3) dominates the others. With such a choice, changes in the weights resulted in significant changes in RF size, shape, and responsiveness. The desirable dynamic RF changes can be obtained for a wide range of choices for the parameters in the shunting equation, as long as no term dominates the others.

The stability of the activation equation was established empirically. The activation equation was computed using the Euler method. Care was taken to ensure that the step size and the number of steps resulted in convergence and that there were no oscillations. Figure 31 shows the activation as a function of time of 30 Layer 2 neurons in response to input at (15, 15).

Figure 31 near here.

Figures 32b–c show that the activation equation converged during the training phase. During the training phase, the sum over all the Layer 2 neurons of the absolute change in activation level, at each step of the Euler method, is averaged over training input presentation at intervals of 100 input presentations.

Figure 32 near here.

Figures 32b–c demonstrate the convergence of the shunting equation during the training phase using normal stimuli and scotoma stimuli, respectively, in the EXIN network with only lateral inhibitory synaptic plasticity enabled. The activation equation also converged to a fixed point during the other types of conditioning; the maximal change in Layer 2 neuronal activation was less than 10^{-5} .

6.1.2 Parameters for the learning equations

The rates of weight change in the EXIN synaptic plasticity rules were chosen so that spurious correlations do not produce large changes in the connection weights; only consistent correlations over several input presentations produce significant changes in connection weights.

Figure 32a plots the sum of the absolute weight change in all lateral inhibitory pathways after every 100 training input presentations, in the EXIN network with only lateral inhibitory synaptic plasticity enabled during normal conditioning.

During the normal conditioning phase, the EXIN network simulations with lateral inhibitory plasticity alone, with afferent excitatory plasticity alone, and with both lateral inhibitory and afferent excitatory plasticity were trained until the sum of the absolute weight change in plastic pathways after every 100 input presentations reached an asymptote.

The Euclidean distance between the network weight vector before and after an additional 10,000 training steps (D), was compared with the average Euclidean distance between the network weight vectors at successive intervals of 100 training steps (d). With lateral inhibitory synaptic plasticity alone $D = 24.599667$, $d = 15.691748 \pm 0.251141$ ($D/d = 1.568$), with afferent excitatory synaptic plasticity alone $D = 0.451494$ and $d = 0.050187 \pm 0.000005$ ($D/d = 8.996$), and with both synaptic plasticity rules $D = 0.699971$ and $d = 0.052240 \pm 0.000005$ ($D/d = 13.399$) for the afferent excitatory weights and $D = 19.516132$ and $d = 12.193612 \pm 0.194959$ ($D/d = 1.601$) for the lateral inhibitory weights. In all cases $D/d \ll 10,000/100 = 100$, suggesting that the networks are close to an equilibrium point.

6.2 Parameters for the LISSOM simulations

To compute the initial weights in the LISSOM simulations $\sigma_{\mathbf{ff}} = 1$, $\sigma_{\mathbf{e}} = 1.41$, $\sigma_{\mathbf{i}} = 2.36$, $\Gamma_{\mathbf{ff}} = 0.01$, $\Gamma_{\mathbf{e}} = 0.1$, and $\Gamma_{\mathbf{i}} = 0.001$ were used.

The parameters used for computing the activations were $\gamma_{\mathbf{ff}} = 0.8$, $\gamma_{\mathbf{e}} = 1.8$, and $\gamma_{\mathbf{i}} = 1.3$. During training and testing LISSOM with only afferent excitatory, lateral excitatory, or lateral inhibitory synaptic plasticity enabled, the activation equation was close to an equilibrium state by 30 iterations. The initial value of the activation level was zero for all neurons.

The weights were modified after the Layer 2 activations reached equilibrium on each input presentation. To compute the weight changes, parameters $\xi_{\mathbf{ff}} = 0.01$, $\xi_{\mathbf{e}} = 0.0005$, and $\xi_{\mathbf{i}} = 0.01$ were used. The weights, which were initially set to zero, were not changed. Without this restriction, normalization would cause all the weights to become very small.

6.2.1 Parameters for the activation equation

The parameters of the activation equation were chosen so that the network gave a distributed activation response to its inputs. The LISSOM activation equation rapidly converges (Sirosh & Mikkulainen, 1994b). The parameters were chosen so that none of the terms in the LISSOM activation equation (Equation 14) dominated the others. In all the LISSOM simulations, the maximal change in Layer 2 neuronal activation was less than 10^{-5} when the activation equation computation was stopped.

6.2.2 Parameters for the learning equations

The rates of weight change in the LISSOM synaptic plasticity rules were chosen so that changes in the connection weights because of spurious correlations gets averaged over a large number of input presentations.

In the initial normal conditioning phase, the networks were trained until the amount of weight change reached an asymptote (the criterion is the same as for the EXIN simulations).

With an additional 10,000 normal training inputs, $D = 0.219775$, $d = 0.017082 \pm 0.000001$ ($D/d = 12.866$) with only lateral inhibitory synaptic plasticity enabled, $D = 2.256291$ and $d = 0.620959 \pm 0.000422$ ($D/d = 3.634$) with only lateral excitatory synaptic plasticity enabled,

and $D = 3.065377$ and $d = 0.795790 \pm 0.000873$ ($D/d = 3.852$) with only afferent excitatory synaptic plasticity enabled. $D/d \ll 100$, suggesting that the networks are close to an equilibrium state.

6.3 Parameters for the adaptation model simulations

To compute the initial weights in the inhibition-dominant adaptation model, parameters $\sigma_{\mathbf{ff}} = 2.13$, $\sigma_{\mathbf{e}} = 1.19$, $\sigma_{\mathbf{i}} = 3.53$, $\Gamma_{\mathbf{ff}} = 0.01$, $\Gamma_{\mathbf{e}} = 0.1$, and $\Gamma_{\mathbf{i}} = 0.001$ were used.

In the adaptation model with no lateral interaction, the lateral excitatory and lateral inhibitory weights were set to zero. The excitation-dominant adaptation model had the same weights as the inhibition-dominant adaptation model. The initial adaptation level was 0.

In the inhibition-dominant adaptation model, the parameters used for computing the activations were $A = 0.2$, $B = 2$, $C = 0.3$, $\beta_{\mathbf{ff}} = 1$, $\beta_{\mathbf{e}} = 0.1$, and $\gamma = 8$. The activation equations for Layer 2 neurons were numerically integrated using the Euler method with a time step of $1/75$. The network was very close to an equilibrium state by time = 15; the maximal change in Layer 2 neuronal activation was less than 10^{-5} .

In the adaptation model with no lateral interaction, the activation equation was solved analytically. With no lateral interactions, the activation equation equilibrates when

$$x_{pq} = \frac{BE_{pq}}{A + E_{pq}}, \quad (21)$$

where $E_{pq} = \beta_{\mathbf{ff}} \sum_{(i,j) \in \text{Layer 1}} [x_{ij}] Z_{ij,pq}^+$, $A = 0.2$, $B = 2$, and $\beta_{\mathbf{ff}} = 1$.

In the excitation-dominant adaptation model, $A = 0.2$, $B = 2$, $C = 0.3$, $\beta_{\mathbf{ff}} = 0.1$, $\beta_{\mathbf{e}} = 0.01$, and $\gamma = 0$. The activation equations for Layer 2 neurons were numerically integrated using the Euler method with a time step of $1/75$. The network reached an equilibrium state by time = 15.

In all three adaptation models, the initial activation level of all neurons was zero. The adaptation threshold parameters were modified after the Layer 2 activations reached equilibrium on each input presentation. To compute the changes in the adaptation threshold, $\rho = 0.0004$, $\eta = 0.3$, and $\tau = 15$ were used in the inhibition-dominant adaptation model. In the adaptation model without lateral interaction and the excitation-dominant adaptation model, $\rho = 0.0004$, $\eta = 0.3$, and $\tau = 2$ were used.

6.3.1 Parameters for the activation equation

The shunting equation (Equation 16) was used in the adaptation models. However, the activation equation in the adaptation models differs from the activation equation in the EXIN model in two respects: (1) the presence of lateral excitation in the adaptation model; (2) the threshold in computing lateral excitation and lateral inhibition. The parameters of the activation equation were chosen so that the network gave a distributed response to its inputs.

The activation equation in the adaptation model behaved similarly to the activation equation in the EXIN model. To make the model inhibition-dominant (excitation-dominant) the constant β_e was chosen to be much smaller (larger) than the constant γ . As neurons are habituated they propagate less lateral excitation and lateral inhibition.

In all the adaptation model simulations, the maximal change in Layer 2 neuronal activation was less than 10^{-6} when the activation equation computation was stopped. The adaptation model with no lateral interaction had a unique fixed point (Equation 21).

6.3.2 Parameters for the adaptation equation

The rate of the adaptation equation was chosen so that the adaptation level of neurons depended on neuronal activation over a large number of input presentations. In the initial normal conditioning phase, the networks were trained until the amount of weight change reached an asymptote (the criterion is the same as for the EXIN simulations).

With an additional 10,000 normal training inputs, $D = 0.229360$, $d = 0.088466 \pm 0.000293$ ($D/d = 2.593$) in the network with no lateral interaction, $D = 0.113066$ and $d = 0.032176 \pm 0.000004$ ($D/d = 3.514$) in the inhibition dominant network, and $D = 0.131278$ and $d = 0.026841 \pm 0.000017$ ($D/d = 4.891$) in the excitation dominant network. In all these networks $D/d \ll 100$, suggesting that the networks are close to an equilibrium point.

6.4 Parameters for generating the inputs for the simulations

In generating the input patterns for training, the kernel K was

$$K = \begin{bmatrix} 0.55 & 0.74 & 0.55 \\ 0.74 & 1.00 & 0.74 \\ 0.55 & 0.74 & 0.55 \end{bmatrix}.$$

The probability, Φ , that the input at each position is 1 was 0.02.

6.5 Parameters for RF measurements

The threshold Θ used for RF measurements was chosen as follows. The activation level of Layer 2 neurons was scaled relative to 1.25 times the maximal response of Layer 2 neuron to test stimuli, in the network obtained after the initial whole-field stimulation. In all the simulations, $\Theta = 0.01$.

6.6 Conditioning procedure

After setting the initial weights in the models, the equilibrium state of the weights or the adaptation levels with respect to the inputs used in the simulations was obtained by training the networks with 25,000 presentations of normal stimuli, except for the EXIN network with afferent synaptic plasticity, and the EXIN network with afferent and lateral inhibitory synaptic plasticity, which were trained with 50,000 presentations of normal stimuli, and the LISSOM network with only lateral excitatory synaptic plasticity, which was trained with 75,000 presentations of normal stimuli. For scotoma and complementary scotoma conditioning and for reversing the effects of scotoma and complementary scotoma conditioning, 5000 presentations of the appropriate stimulus were used, except for the EXIN network with afferent synaptic plasticity and the EXIN network with afferent and lateral inhibitory synaptic plasticity, which were trained with 50,000 input presentations, and the LISSOM network with only lateral excitatory synaptic plasticity, which was trained with 25,000 input presentations. In complementary scotoma conditioning, the complementary scotoma stimuli were alternated.

Measurement	Exp'tal Data	EXIN Outstar Lateral Inh. Learning alone	EXIN Instar Afferent Exc. Learning alone	LISSOM Instar Lateral Inh. Learning alone	LISSOM Instar Lateral Exc. Learning alone	LISSOM Instar Afferent Exc. Learning alone	AN with no lateral interactions	AN Exc. Dom.	AN Inh. Dom.
RF expansion inside the scotoma	Yes	Yes	Yes	No	Yes	Yes	Yes	Yes	Yes
Asymmetric RF profile after scotoma conditioning	Yes	Yes	Yes	Yes	Yes	Yes	No	Yes	Yes
Asymmetric RF more responsive to locations away from scotoma center	Yes	Yes	Yes	No	Yes	Yes	No	No	Yes
Maximal RF expansion just inside the cortical scotoma boundary	Yes	Yes	Yes	No	Yes	Yes	No	No	Yes
Function returns in concentric inward direction	Yes	Yes	Yes	No	Yes	Yes	Yes	Yes	Yes
Retinal lesion produces small centrifugal RF shifts	Yes	Yes	Yes	No	Yes	Yes	No	No	Yes
Blank display changes RF size	No	No	No	No	No	No	Yes	Yes	Yes
Complem. scotoma cond. produces peak expansion near scotoma boundary	??	Yes	No	Yes	No	No	No/Yes	No/Yes	No/Yes
Complem. scotoma cond. shifts RFs across and close to the scotoma boundary toward each other	??	Yes	No	Yes	No	No	No/No	No/Yes	Yes/No

Table 1

Figure 1: **Site of RF changes.** The RF of cortical neurons changes when input excitation and inhibition to the neurons change. Cortical neurons receive afferent, lateral, and feedback excitation. Cortical neurons receive lateral inhibition from inhibitory neurons (shown by the shaded ellipse in layer 2). The excitatory cortical neurons (represented by the unfilled ellipses) send lateral excitation to excitatory and inhibitory neurons. Changes in input excitation and inhibition can occur because of synaptic plasticity in excitatory and inhibitory synapses, respectively, or because of adaptation cortical neurons.

Figure 2: **Network architecture for the EXIN model.** The afferent pathways from Layer 1 to Layer 2 are excitatory. The lateral pathways within Layer 2 are inhibitory. The unfilled ellipses represent the afferent connectivity pattern from Layer 1 to Layer 2 neurons. The shaded ellipses represent the RFs of Layer 2 neurons. The strength of lateral inhibitory pathways is a function of the amount of overlap in the afferent connectivity patterns to the Layer 2 neurons. The strength of lateral inhibitory pathways is indicated by the thickness of the arrows.

Figure 3: **The effects of scotoma conditioning on the EXIN model.** The unfilled ellipses represent the afferent connectivity pattern from Layer 1 to Layer 2 neurons. The shaded ellipses represent the RFs of Layer 2 neurons. The parallelogram within Layer 1 represents the scotoma region, and the parallelogram within Layer 2 represents the cortical scotoma region before scotoma conditioning. (a) The network state before scotoma conditioning. Neuron (r, s) is in the cortical scotoma region because its RF is within the scotoma region; neuron (p, q) is outside the cortical scotoma region. (b) The network state after scotoma conditioning with only lateral inhibitory synaptic plasticity enabled. After scotoma conditioning, lateral inhibitory connections between neurons in the cortical scotoma and lateral inhibitory connections between neurons outside the cortical scotoma do not change. However, lateral inhibitory connections from neurons outside the cortical scotoma (e.g., (p, q)) to neurons inside the cortical scotoma (e.g., (r, s)) weaken (dashed line). The decrease in inhibition received by neuron (r, s) results in expansion of its RF. (c) The network state after scotoma conditioning with only afferent excitatory synaptic plasticity enabled. After scotoma conditioning, afferent connections to neurons in the cortical scotoma do not change. However, afferent connections from locations inside the scotoma to neurons outside the cortical scotoma (e.g., (p, q)) weaken (dashed line). Neurons inside the cortical scotoma (e.g., (r, s)) receive less inhibition from neuron (p, q) when locations inside the scotoma are stimulated. Thus, neuron (r, s) shows new responsiveness to these locations, and its RF size thus increases.

Figure 4: **Network architecture for the LISSOM model and the inhibition-dominant adaptation model.** The connections from Layer 1 to Layer 2 are excitatory. There are lateral inhibitory and excitatory connections between Layer 2 neuron (p, q) and Layer 2 neurons within the inner ellipse (e.g., (r, s)). Neuron (p, q) also sends both inhibitory and excitatory connections to itself. The lateral connections between neuron (p, q) and Layer 2 neurons (e.g., (u, v)) outside the inner ellipse and inside the outer ellipse are inhibitory. The unfilled ellipses in Layer 1 represent the afferent connectivity pattern from Layer 1 to Layer 2 neurons. The shaded ellipses represent the RFs of Layer 2 neurons.

Figure 5: **The effects of scotoma conditioning on the LISSOM model.** The unfilled ellipses in Layer 1 represent the afferent connectivity pattern from Layer 1 to Layer 2 neurons. The shaded ellipses represent the RFs of Layer 2 neurons. The parallelogram within Layer 1 represents the scotoma region, and the parallelogram within Layer 2 represents the cortical scotoma region before scotoma conditioning. (a) The network state before scotoma conditioning. Neurons (r, s) and (a, b) are inside the cortical scotoma region because their RFs are within the scotoma region. Neurons (p, q) , (u, v) , and (c, d) are outside the cortical scotoma region. There are excitatory and inhibitory connections between neuron (p, q) and neurons in the small ellipse (e.g., (u, v) and (r, s)). The connections between neuron (p, q) and the neurons outside the small ellipse and inside the large ellipse are inhibitory. (b) The network state after scotoma conditioning with only instar lateral inhibitory synaptic plasticity enabled. The dashed lines represent a decrease in the connection weights and the thick lines represent an increase in the connection weights. (c) The network state after scotoma conditioning with only instar lateral excitatory synaptic plasticity enabled. (d) The network state after scotoma conditioning with only afferent excitatory synaptic plasticity enabled.

Figure 6: **The effects of scotoma conditioning on the inhibition-dominant adaptation model.** The unfilled ellipses represent the afferent connectivity pattern from Layer 1 to Layer 2 neurons. The shaded ellipses represent the RFs of Layer 2 neurons. The parallelogram within Layer 1 represents the scotoma region, and the parallelogram within Layer 2 represents the cortical scotoma region before scotoma conditioning. (a) The network state before scotoma conditioning. All Layer 2 neurons have almost the same adaptation level (shaded circles). Thus, all Layer 2 neurons have almost the same RF size. (b) The network state after scotoma conditioning. The neurons inside the cortical scotoma are dishabituated (white circles) and hence are highly responsive to input stimulation. The neurons inside the cortical scotoma inhibit neurons outside the cortical scotoma and in turn receive weaker inhibition. Thus, neurons within the cortical scotoma show RF expansion.

Figure 7: Simulation results: RF expansion and contraction. The RFs of Layer 2 neurons that showed maximal expansion, in the EXIN network with only lateral inhibitory synaptic plasticity enabled (a,d,g), the LISSOM network with only lateral inhibitory synaptic plasticity enabled (b,e,h), and the inhibition-dominant adaptation network (c,f,i) are shown. The inner square indicates the extent of the scotoma. The responsiveness of the neurons to the test stimuli is proportional to the gray level. Panels (a,b,c) show the initial RF, (d,e,f) show the RF after scotoma conditioning using a 13×13 square scotoma centered at (15, 15), and (g,h,i) show the RF after re-conditioning using normal stimuli. Panels (d,e,f) show expansion, and (g,h,i) show that the RF is restored by re-conditioning with normal stimuli. In the EXIN network with only lateral inhibitory synaptic plasticity enabled and in the inhibition-dominant adaptation network, the center of the RF is within the scotoma (d,f). In contrast, in the LISSOM network with only lateral inhibitory synaptic plasticity enabled the center of the RF is outside the scotoma (e). Furthermore, the RFs shown in panels (a,c), which are within the scotoma, cross the scotoma boundary after artificial scotoma conditioning, as shown in panels (d,f). The RF in panel (b), which is outside the scotoma, crosses the scotoma boundary after artificial scotoma conditioning, as shown in panel (e).

Figure 8: Simulation results: The iceberg effect. Layer 2 neuron responsiveness before scotoma conditioning (solid line), after scotoma conditioning (dashed line), and after re-conditioning with normal stimuli (dotted line) are shown. Neuron activations in the EXIN network with only lateral inhibitory synaptic plasticity enabled (a), the LISSOM network with only lateral inhibitory synaptic plasticity enabled (b), and the inhibition-dominant adaptation network (c), are shown as a function of one-dimensional input positions across Layer 1 passing through the scotoma center. The responsiveness is for the neurons in Figure 7. The responsiveness is computed by summing the response of the Layer 2 neuron to test stimuli along the y axis at each x axis position. The neuron position is represented relative to the x coordinate of the scotoma center. The scotoma is a square of size 13×13 . The thick line segment on the abscissa represents the scotoma region.

Figure 9: **Simulation results: 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 with only lateral inhibitory synaptic plasticity enabled (a), the LISSOM network with only lateral inhibitory synaptic plasticity enabled (b), and the inhibition-dominant adaptation network (c), are shown as a function of the position of the initial RF center of Layer 2 neurons relative to the scotoma center (15,15). The RF area shown is for a one-dimensional cross-section through Layer 2: neurons (15,0)–(15,29). In panel (c) the dotted curve overlaps with the solid curve. 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. See Figure 8 for simulation details and conventions.

Figure 10: **Simulation results: Changes in RF after scotoma conditioning with a smaller scotoma.** 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 with only lateral inhibitory synaptic plasticity enabled (a), the LISSOM network with only lateral inhibitory synaptic plasticity enabled (b), and the inhibition-dominant adaptation network (c), are shown as a function of the position of the initial RF center of Layer 2 neurons relative to the scotoma center (15,15). The scotoma is a square of size 9×9 centered at (15,15). In panel (c) the dotted curve overlaps with the solid curve. See Figure 9 for the definition of RF size. The RF area shown is for a one-dimensional cross-section through Layer 2: neurons (15,0)–(15,29). The thick line segment on the abscissa represents the scotoma region.

Figure 11: **Simulation results: Recovery of responsiveness of Layer 2 neurons.** The figures show the activation pattern in Layer 2 in response to input outside the scotoma before scotoma conditioning (solid line), after 2500 steps of scotoma conditioning (dashed line), and after 5000 steps of scotoma conditioning (dotted line) in the EXIN network with only lateral inhibitory synaptic plasticity enabled (a), the LISSOM network with only lateral inhibitory synaptic plasticity enabled (b), and the inhibition-dominant adaptation network (c). The abscissa represents the distance of initial RF center of Layer 2 neurons from the scotoma center. The input is a test stimulus centered at Layer 1 neuron (15,6). Panels (a) and (c) show recovery of neurons in a concentric inward direction. In panel (b) responsiveness is lost in a concentric inward direction. See Figure 8 for simulation details and conventions.

Figure 12: **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 the initial RF center of Layer 2 neurons from the scotoma center for the EXIN network with only lateral inhibitory synaptic plasticity enabled (a), the LISSOM network with only lateral inhibitory synaptic plasticity enabled (b), and the inhibition-dominant adaptation network (c). 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. See Figure 8 for simulation details and conventions.

Figure 13: **Simulation results: Blank screen causes RF changes in the inhibition-dominant adaptation model.** The RF area after scotoma conditioning (solid line), after 2500 steps of conditioning with blank stimuli (dashed line), and after 5000 steps of conditioning with blank stimuli (dotted line) in the inhibition-dominant adaptation network after 5000 steps of scotoma conditioning (a), and after 15000 steps of scotoma conditioning (b), are shown as a function of the position of the initial RF center of Layer 2 neurons relative to the scotoma center (15, 15). In (a) the neurons in the cortical scotoma were not fully dishabituated and therefore show RF expansion during no visual stimulation. In (b) prolonged inactivity in neurons in the cortical scotoma causes them to become almost fully dishabituated. With no visual stimulation, the neurons outside the cortical scotoma are dishabituated and they exert stronger inhibition on neurons inside the cortical scotoma. Thus, neurons outside the cortical scotoma show RF expansion, and neurons inside the cortical scotoma, which were highly dishabituated, show RF contraction. See Figure 10 for simulation details and conventions.

Figure 14: **Simulation results: RF size changes in other adaptation models.** The RF area before scotoma conditioning (solid line), after scotoma conditioning (dashed line), and after re-conditioning with normal stimuli (dotted line) in the inhibition-dominant adaptation network without prior normal stimulation (a), the adaptation network without lateral connections (b), and the excitation-dominant adaptation network (c), are shown as a function of the position of the initial RF center of Layer 2 neurons relative to the scotoma center (15, 15). In panel (b) the dotted curve overlaps with the solid curve. See Figure 9 for simulation details and conventions.

Figure 15: **Simulation results: The iceberg effect in other adaptation models.** Layer 2 neuron responsiveness before scotoma conditioning (solid line), after scotoma conditioning (dashed line), and after re-conditioning with normal stimuli (dotted line) in the inhibition-dominant adaptation network without prior normal stimulation (a), the adaptation network without lateral connections (b), and the excitation-dominant adaptation network (c), are shown as a function of one-dimensional input positions across Layer 1 passing through the scotoma center. See Figure 8 for simulation details and conventions.

Figure 16: **Simulation results: RF shifts in other adaptation models.** 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 the initial RF center of Layer 2 neurons from the scotoma center for the inhibition-dominant adaptation network without prior normal stimulation (a), the adaptation network without lateral connections (b), and the excitation-dominant adaptation network (c). In panel (b) the solid and the dashed curves overlap with the abscissa. See Figure 12 for simulation details and conventions.

Figure 17: **Simulation results: Recovery of responsiveness in other adaptation models.** The figures show the activation pattern in Layer 2 in response to input outside the scotoma before scotoma conditioning (solid line), after 2500 steps of scotoma conditioning (dashed line), and after 5000 steps of scotoma conditioning (dotted line) in the inhibition-dominant adaptation network without prior normal stimulation (a), the adaptation network without lateral connections (b), and the excitation-dominant adaptation network (c). The input is a test stimulus centered at Layer 1 neuron (15,6). See Figure 11 for simulation details and conventions.

Figure 18: **Simulation results: RF size changes caused by afferent excitatory plasticity.** 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 with only afferent excitatory synaptic plasticity enabled (a), the LISSOM network with only afferent excitatory synaptic plasticity enabled (b), and the EXIN network with afferent excitatory and lateral inhibitory synaptic plasticity (c), are shown as a function of the position of the RF center of Layer 2 neurons relative to the scotoma center (15, 15). See Figure 9 for conventions and simulation details.

Figure 19: **Simulation results: RF profiles of neurons that show expansion or contraction.** Layer 2 neuron RF profiles before scotoma conditioning (solid line), after scotoma conditioning (dashed line), and after re-conditioning with normal stimuli (dotted line) in the EXIN network with only afferent excitatory synaptic plasticity enabled (a,b), and the LISSOM network with only afferent excitatory synaptic plasticity enabled (c,d), as a function of a one-dimensional input positions across Layer 1 passing through the scotoma center. In (a) and (c), the neurons show RF expansion, and in (b) and (d), the neurons show RF contraction. See Figure 8 for simulation details and conventions. The thick line on the abscissa represents the scotoma region. In (a) and (c), the neurons showed RF expansion, and in (b) and (d) the neurons showed RF contraction.

Figure 20: **Simulation results: Activation profiles in response to inputs at locations away from the scotoma.** The figure shows activation of a one-dimensional cross-section of Layer 2 neurons: (15, 0)–(15, 29), in response to input stimulation at (15, 26) (a), (15, 27) (b), (15, 28) (c), and (15, 29) (d) in the EXIN network with only afferent excitatory synaptic plasticity enabled. The activation level of the neurons was scaled by a factor of 20. See Figure 11 for conventions and simulation details.

Figure 21: **Simulation results: Recovery of responsiveness of Layer 2 neurons caused by afferent excitatory plasticity.** The figures show the activation pattern in Layer 2 in response to input outside the scotoma before scotoma conditioning (solid line), after 2500 steps of scotoma conditioning (dashed line), and after 5000 steps of scotoma conditioning (dotted line) in the EXIN network with only afferent excitatory synaptic plasticity enabled (a) and the LISSOM network with only afferent excitatory synaptic plasticity enabled (b). See Figure 11 for conventions and simulation details.

Figure 22: **Simulation results: RF shifts caused by afferent excitatory plasticity.** Shift in RF center after scotoma conditioning (solid line), after re-conditioning with normal stimuli for 50,000 steps (dashed line), and after re-conditioning with normal stimuli for 100,000 steps (dotted line) with respect to the initial RF centers is shown as a function of distance of the initial RF center of Layer 2 neurons from the scotoma center for the EXIN network with only afferent excitatory synaptic plasticity enabled (a), the LISSOM network with only afferent excitatory synaptic plasticity enabled (b), and the EXIN network with both afferent excitatory and lateral inhibitory synaptic plasticity (c). See Figure 12 for conventions and simulation details.

Figure 23: **Simulation results: RF changes in the LISSOM network with only lateral excitatory plasticity enabled.** (a) The RF area before scotoma conditioning (solid line), after scotoma conditioning (dashed line), and after re-conditioning with normal stimuli (dotted line), is shown as a function of the position of the initial RF center of Layer 2 neurons relative to the scotoma center (15,15). See Figure 9 for conventions and simulation details. (b) Layer 2 neuron responsiveness before scotoma conditioning (solid line), after scotoma conditioning (dashed line), and after re-conditioning with normal stimuli (dotted line), are shown as a function of one-dimensional input positions across Layer 1 passing through the scotoma center. See Figure 8 for conventions and simulation details. (c) The activation pattern in Layer 2 in response to input outside the scotoma before scotoma conditioning (solid line), after 2,500 steps of scotoma conditioning (dashed line), and after 5,000 steps of scotoma conditioning (dotted line). The input is a test stimulus centered at Layer 1 neuron (15,6). (d) 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 the scotoma center. See Figure 12 for conventions and simulation details.

Figure 24: **Simulation results: Average RF size changes after complementary scotoma conditioning.** The average RF area before complementary scotoma conditioning (solid line), after complementary scotoma conditioning (dashed line), and after re-conditioning with normal stimuli (dotted line) in the EXIN network with only lateral inhibitory synaptic plasticity enabled (a), the LISSOM network with only lateral inhibitory synaptic plasticity enabled (b), and the inhibition-dominant adaptation network (c), are shown as a function of the position of the initial RF center of Layer 2 neurons relative to the scotoma center. The scotoma is a hemifield of size 15×30 . The average RF area shown at each position is the mean over the RF area of neurons with same x coordinate. The RF area of a Layer 2 neuron is defined as the number of locations at which the test stimulus evokes a response in the Layer 2 neuron. In panel (c) the dotted curve overlaps with the solid curve. The thick line segment on the abscissa represents the scotoma region.

Figure 25: **Simulation results: Average RF size changes after complementary scotoma conditioning.** The average RF area before complementary scotoma conditioning (solid line), after complementary scotoma conditioning (dashed line), and after re-conditioning with normal stimuli (dotted line) in the EXIN network with only afferent excitatory synaptic plasticity enabled (a), the LISSOM network with only afferent excitatory synaptic plasticity enabled (b), and the LISSOM network with only instar lateral excitatory synaptic plasticity enabled (c), are shown as a function of the position of the initial RF center of Layer 2 neurons relative to the scotoma center. See Figure 24 for conventions and simulation details.

Figure 26: **Simulation results: Average RF shifts after complementary scotoma conditioning.** The average shift in the RF center after complementary 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 the initial RF center of Layer 2 neurons from the scotoma center for the EXIN network with only lateral inhibitory synaptic plasticity enabled (a), the LISSOM network with only lateral excitatory synaptic plasticity enabled (b), and the adaptation network (c). The average RF shift shown at each position is the mean over the shift in RF center of neurons with same x coordinate. 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. See Figure 24 for simulation details and conventions.

Figure 27: **Simulation results: Average RF shifts after complementary scotoma conditioning.** The average shift in the RF center after complementary 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 the initial RF center of Layer 2 neurons from the scotoma center for the EXIN network with only afferent excitatory synaptic plasticity enabled (a), the LISSOM network with only afferent excitatory synaptic plasticity enabled (b), and the LISSOM network with only instar lateral excitatory synaptic plasticity enabled (c). See Figure 26 for conventions.

Figure 28: **Simulation results: Average RF size changes after complementary scotoma conditioning.** The average RF area before complementary scotoma conditioning (solid line), after complementary scotoma conditioning (dashed line), and after re-conditioning with normal stimuli (dotted line) in the adaptation network with no lateral interaction (a), and in the excitation-dominant adaptation network (b), are shown as a function of the position of the initial RF center of Layer 2 neurons relative to the scotoma center. In panels (a,b) the dotted curve overlaps with the solid curve. See Figure 24 for conventions and simulation details.

Figure 29: **Simulation results: Average RF shifts after complementary scotoma conditioning.** The average shift in the RF center after complementary 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 the initial RF center of Layer 2 neurons from the scotoma center for the adaptation network with no lateral interaction (a) and for the excitation-dominant adaptation network (b). In panel (a) the solid and dashed curves overlap with the abscissa because there were no RF shifts. See Figure 26 for conventions.

Figure 30: **Simulation results: Behavior of the EXIN network as a function of activation equation parameters.** The equilibrium activation level of a one-dimensional cross-section of Layer 2: neurons (15,0)–(15,29), in response to input at (15,15). The EXIN network used was obtained after training with 25,000 normal inputs with only lateral inhibitory synaptic plasticity enabled. The network reached a stable state by time 110. (a) The parameter β in Equation 3 is varied by a factor of 21, yet the width of the response curve increases by only a small amount. (b) When the parameter γ in Equation 3 is increased from 0.2 to 4.2, the network exhibits winner-take-all behavior. (c) The parameter B in Equation 3 is varied by a factor of 16, yet the width of the response curve increases by only a small amount. (d) When the parameter C in Equation 3 is set to 4, the network exhibits winner-take-all behavior. When C is zero, the neurons have non-negative activation levels.

Figure 31: **Simulation results: Activation curves in the EXIN network.** The activation level of a one-dimensional cross-section of Layer 2: neurons (15,0)–(15,29), in response to input at (15, 15) in the EXIN network after training with 25,000 normal inputs with only lateral inhibitory synaptic plasticity enabled. The network equilibrates by time 110. Note that some neurons that were active during the initial stages are eventually suppressed.

Figure 32: **Simulation results: Stability of the EXIN network with lateral inhibitory synaptic plasticity alone.** The jagged line in panel (a) shows the total change in the lateral inhibitory pathway weights in the EXIN network, after 100 presentations of normal stimuli, over the course of 25,000 normal input presentations. The total change is obtained by summing the magnitude of the weight changes at intervals of 100 input presentations, over all the lateral inhibitory pathways. The smooth line in panel (a) is the nonlinear least squares fit of the data using the Marquardt-Levenberg algorithm. The non-linear function is $a \times \exp(-b \times \text{iteration number}) + c$. The best fit parameters are $a = 53864.9 \pm 5665.23$, $b = 1.490 \pm 0.098$, and $c = 10648.6 \pm 18.26$. The sum over all Layer 2 neurons, of the magnitude of change in the activation level at each step in the Euler method for solving the activation equation (Equation 3), is averaged over input presentations at intervals of 100 during normal conditioning (b), and during scotoma conditioning (c). The vertical lines in (b,c) represent the standard deviation.

Figure 1

Figure 2

Figure 3a

Figure 3b

Figure 3c

Lateral excitatory and inhibitory connections

Figure 4

Figure 5a

Figure 5b

Figure 5c

Figure 5d

(a)

Figure 6a

(b)

Figure 6b

Figure 7

Distance from scotoma center

Figure 8

Distance from scotoma center

Figure 9

Distance from scotoma center

Figure 10

Distance from scotoma center

Figure 11

Distance from scotoma center

Figure 12

Distance from scotoma center

Figure 13

Distance from scotoma center

Figure 14

Distance from scotoma center

Figure 15

Distance from scotoma center

Figure 16

Distance from scotoma center

Figure 17

Distance from scotoma center

Figure 18

Figure 19

Figure 20

Distance from scotoma center

Figure 21

Distance from scotoma center

Figure 22

Figure 23

Distance from scotoma center

Figure 24

Figure 25

Distance from scotoma center

Figure 26

Figure 27

Distance from scotoma center

Figure 28

Distance from scotoma center

Figure 29

Figure 30a

Figure 30b

Figure 30c

Figure 30d

Figure 31

Figure 32