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Simulation of visual cortex development under lid-suture conditions: Enhancement of response specificity by a reverse-Hebb rule in the absence of spatially patterned input

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Abstract. In this report, I show that a reverse-Hebb synaptic modification rule leads to the enhancement of response specificity of simulated visual cortex neurons in the absence of spatial patterning of the afferent activity. Although it is clear that receptive fields in the visual cortex can be modified by experience, many studies have shown a substantial increase of response specificity in cats deprived of pattern vision by lid suture, leading some to conclude that receptive field properties are essentially hard-wired. The hard-wired *vs.* experience-dependent controversy can be resolved by assuming that while Hebb-type plasticity is responsible for developmental synaptic changes, the organization of presynaptic activity which exists under conditions of visual deprivation is sufficient to drive the neurons towards greater specificity (Linsker 1986a-c; Miller 1989, 1992; Miller et al. 1989). As a reverse-Hebb rule enhances response specificity by balancing the push-pull system of ON- and OFF-center afferents, the sufficient condition is that the activity of ON- and OFF-center retinal ganglion cells be negatively correlated, a condition which will be met by diffuse illumination as seen through sutured eyelids. Unlike the models of Linsker and Miller and colleagues, which are based on a standard-Hebb rule, the model presented here does not require the presence of a "Mexican hat" spatial patterning of the afferent correlations, which has not been observed experimentally.

1 Introduction

The objective of this study was to compare the effects of patterned vs. unpatterned visual experience on the development of simulated striate cortex simple cells. The intent was to mimic both the normal developmental process and development under lid-suture conditions, an experimental paradigm used to deprive animals of patterned vision (Wiesel and Hubel 1965). The synaptic plasticity rule, which modified the inputs to the simulated simple cells, was the same reverse-Hebb rule shown in previous simulations to lead to the enhancement of stimulus specificity in a patterned visual environment (Soodak 1991). I report here that this rule also results in a significant enhancement of stimulus specificity in an unpatterned visual environment.

Although it is clear that receptive fields in the visual cortex can be modified by experience (Wiesel and Hubel 1963; Hubel and Wiesel 1965; Blakemore and Cooper 1970; Hirsch and Spinelli 1970; Movshon 1976; Stryker et al. 1978), the role, and necessity, of visual experience in the development of response specificity has been the subject of considerable debate. One view on this issue is that cortical receptive fields are for the most part hard wired, that is, determined by intrinsic (genetic) developmental rules, and the role of the environment is simply a permissive one. The opposing view holds that the development of receptive fields is an instructional process, in which the visual environment, acting through an activity dependent rule, determines the course of development and the adult response properties ultimately attained (see Movshon and Van Sluyters 1981; Frégnac and Imbert 1984; Singer 1985; for reviews and discussion).

The lid-suture paradigm has been frequently used to determine the extent to which receptive field properties are innately determined vs. experience dependent. This procedure deprives the animal of high spatial frequency patterned vision due to dispersion of light by the closed lids, but as the lids are not completely opaque, the retina is exposed to diffuse illumination. [The closed lid of pigmented cats transmits about 5% of the incident light (Crawford and Marc 1976), and cats with sutured lids can be trained to perform brightness, but not orientation discrimination (Loop and Sherman 1977)]. While the failure of normal development under lid-suture conditions can be interpreted as the absence of either an instructional or permissive factor, the observation of normal development in the absence of patterned vision argues in favor of the innate determination of receptive field properties. Experimental results regarding the development of orientation specificity in the absence of patterned vision have differed considerably. While some studies reported a failure to show any increase in selectivity under lid-suture conditions (Barlow and Pettigrew 1971; Pettigrew 1974), most studies have concluded that at least some proportion of neurons in the striate cortex develop normal selectivity to stimulus orientation in the absence of spatially patterned vision (Blakemore and Van Sluyters 1975; Kratz and Spear 1976; Sherk and Stryker 1976; Leventhal and Hirsch 1977; Watkins et al. 1978).

Although the most straightforward interpretation of these results is that the development of orientation tuning can occur without the involvement of an activity-dependent instructional process, the models proposed by Linsker (1986a-c) and Miller and colleagues (Miller 1989, 1992; Miller et al. 1989) are based on a different assessment. The latter involve the premise that the instructional and intrinsic viewpoints are not exclusive of one another. While these models assume that synaptic modification is driven by an activity dependent Hebb-type rule (Hebb 1949), they propose that the intrinsic spatial patterning of the correlation among cortical afferents (Mastronarde 1983) is sufficient to drive the postsynaptic cortical neurons towards higher response specificity in the absence of visual input. Specifically, these models assume that the spontaneous activity of the afferent input neurons are correlated with a “Mexican hat” spatial structure, i.e., at less than a certain distance of separation, spontaneous activities of like-center type (ON vs ON and OFF vs OFF) are positively correlated, and for some distances greater than this, the spontaneous activities of like-center type are negatively correlated. The opposite relation is assumed to hold for opposite-center types (ON vs OFF).

The model presented here differs from those of Linsker and Miller and colleagues in two major respects. First, the synaptic modification rule employed here is of the reverse-Hebb-type rather than the standard-Hebb type used by Linsker and Miller and coworkers. Second, as a consequence of the reverse-Hebb rule, response specificity can evolve in the absence of any spatial patterning of input activity; the only requirement is that ON- and OFF-center afferents show a negative correlation with each other. That is, the "Mexican hat" correlation pattern, which has not been experimentally observed, is not a requirement of this model. As a reverse-Hebb rule enhances response specificity by balancing the push-pull system of ON- and OFF-center afferents, the sufficient condition is that the activity of ON- and OFF-center retinal ganglion cells be negatively correlated, a condition which will be met by diffuse illumination as seen through sutured eyelids.

2 Methods

2.1 Structural parameters

The simulation comprised 12 or 24 simple cells within a 0.6 by 1 mm area of primary visual cortex. The structural parameters of the simulation were derived from published anatomical and physiological data from the cat. The retinal coordinates of ON- and OFF-center X-type ganglion cells were obtained from flat-mount drawings of Wässle et al. (1981). The total number of ON-center ganglion cells was 65 and the total number of OFF-center ganglion cells was 69. The mean distance to the nearest neighbor was 0.59 deg for the ON-cell mosaic and 0.56 deg for the OFF-cell mosaic. The lateral geniculate nucleus was assumed to function as a one-to-one relay; thus the ganglion cell mosaic was mapped directly onto the cortex. The scale of this mapping was determined by the cortical magnification factor given in Fig. 12 of Tusa et al. (1978), at the eccentricity of 33 deg (corresponding to the eccentricity of the sample of Wässle et al.). The value used was 0.1 mm^2 cortex per deg^2 visual field. Prior to visual experience, the strength of connection from a given retinal ganglion cell (RGC) to a given simple cell was determined by a retinotopic rule, that is, the strength of connection was simply a function of the retinotopic distance between them. A Gaussian function was used, its width determined by the spread of geniculate afferents tangential to the cortical surface, from data of Ferster and LeVay (1978) and Humphrey et al. (1985). The Gaussian function fell to 25% of its peak value at 0.5 mm and was truncated at 1% of its peak value at a diameter of 0.9 mm. RGCs were assumed to sum retinal illumination linearly, and the simple cell outputs were a linear summation of their afferent inputs weighted by the respective synaptic strengths. These parameters are summarized in Table 1. For further details, see Soodak (1986, 1987).

2.2 Ganglion cell receptive fields

The RGC receptive fields were modeled as a radially symmetric difference of Gaussians (Rodieck 1965; Enroth-Cugell and Robson 1966). The width of the center was 2 deg, and the width of the surround was 10 deg. The integrated center strength was twice that of the surround. These parameters yielded spatial frequency tuning curves similar to those shown for kitten cortical neurons by Derrington and Fuchs (1981).

2.3 Visual environment

For the simulation of normal development, the visual environment was made up of sinusoidal drifting gratings. A total of 64 stimuli were used, eight equally spaced (22.5 deg) orientations at each of eight different spatial frequencies (0.025, 0.035, 0.050, 0.071, 0.10, 0.14, 0.20, and 0.28 cycles/deg). For simulation of development under lid-suture conditions, a single stimulus was used, i.e., full-field sinusoidal temporal modulation. (While some very low spatial frequency information may pass through sutured lids, it was the intent of this study to show the behavior of the simulation in the absence of patterned stimuli; simulating lid-suture with only very low spatial frequencies yielded similar results.) With drifting sinusoidal gratings, all RGCs will respond with equal amplitudes but with phases which depend upon their position on the retina and the orientation and spatial frequency of the stimulus. With full-field temporal modulation, all RGCs will respond with identical amplitudes and phases, except for a sign difference between ON- and OFF-center cells. Thus, under lid-suture conditions, the course of development does not depend on the structural parameters of the ganglion cell receptive fields.

Table 1. Model Parameters

Parameter	Value	Reference
Ganglion cell number and coordinates	65 ON-center RGCs 69 OFF-center RGCs (at 33^o eccentricity)	Coordinates measured from flat-mount drawings of Wässle et al. (1981)
Ganglion cell receptive fields	Difference of Gaussians: Center width: 2^o Surround width: 10^o Center/surround strength ratio: 2.0	Scaled to approximate spatial frequency tuning of kitten cortical neurons from Derrington and Fuchs (1981)
Cortical magnification factor	0.1 mm^2 cortex per degree^2 visual field	Measured at 33^o eccentricity from Fig. 12 of Tusa et al. (1978)
Initial RGC to simple cell connectivity	Gaussian function of relative retinotopic positions Fell to 25% peak at 0.5 mm diameter; truncated at 1% of peak at 0.9 mm diameter	Based on LGN afferent spread from Ferster and LeVay (1978) Humphrey et al. (1985)

RGC, retinal ganglion cell; LGN, lateral geniculate neuron

2.4 Reverse-Hebb Rule

In the general form of the synaptic modification rule, changes in synaptic strength are defined as a weighting of the input-output cross-correlation, $\chi_{io}(\tau)$, with a function $H(\tau)$, where τ is the time interval between input and output (Soodak 1991). The fractional change in synaptic weight as a result of a stimulus presentation, dW/W, is given by

$$\frac{dW}{W} = \int_{-\infty}^{\infty} \chi_{io}(\tau)\, H(\tau)\, d\tau$$

which for the case of sinusoidal stimuli, as employed in these simulations, reduces to

$$\frac{dW}{W} = HA_i A_o \cos(\phi_i - \phi_o - \phi_H)$$

where A_i and A_o represent the amplitudes of the input (RGC) and output (simple cell) responses, respectively, and ϕ_i and ϕ_o the corresponding phases. The parameter H defines the gain of synaptic plasticity, and the phase parameter, ϕ_H, determines the relative phase of pre- and post-synaptic activities that will effect an increase in synaptic weight. When $\phi_H = 180$ deg, the synaptic weight will increase when the pre- and post-synaptic activities are out of phase, or negatively correlated. This is the reverse-Hebb condition. The parameters H and ϕ_H are derived from the Fourier transform of $H(\tau)$ as described in Soodak (1991). For all simulations presented here, the ϕ_H parameter was fixed at 180 deg.

3 Results

Figure 1 (top) shows the orientation and spatial frequency tuning of 12 simple cells in a “visually inexperienced” simulation, i.e., synaptic connections were determined by the retinotopic rule alone. On the left are shown the spatial frequency tuning curves of all 12 neurons in the simulation, and on the right, the corresponding orientation tuning curves. The spatial frequency tuning curves are shown at the preferred orientation and normalized to a peak response of unity. The orientation tuning curves are shown at a spatial frequency value of 0.1 cycles/deg. The simulated neurons show some degree of orientation tuning, though it is typically quite low. The spatial frequency tuning of the simulated simple cells prior to visual experience is for the most part a reflection of the tuning of their afferents, i.e., the spatial frequency tuning of the RGCs.

To simulate experience-dependent development, the synaptic connection weights determined by the retinotopic rule served as the starting values to be modified by the reverse-Hebb rule. Patterned visual experience consisted of drifting sinusoidal gratings at eight equally spaced orientations at each of eight different spatial frequencies. The reverse-Hebb rule strengthened those RGC inputs that responded out of phase with the summed postsynaptic response over those RGC inputs that responded in phase with the simple cell, as detailed in Methods. The orientation and spatial frequency tuning of the 12 simulated simple cells after 100 stages of patterned visual experience with an H parameter of 0.005 are shown in Fig. 1 (center). As in normally reared cats, both the orientation (Albus and Wolf 1984), and spatial frequency (Derrington and Fuchs 1981) tuning show a substantial increase, although the levels attained by the simulated neurons remain well below that observed experimentally. The absence of inhibitory interactions in the simulation may account for the difference.

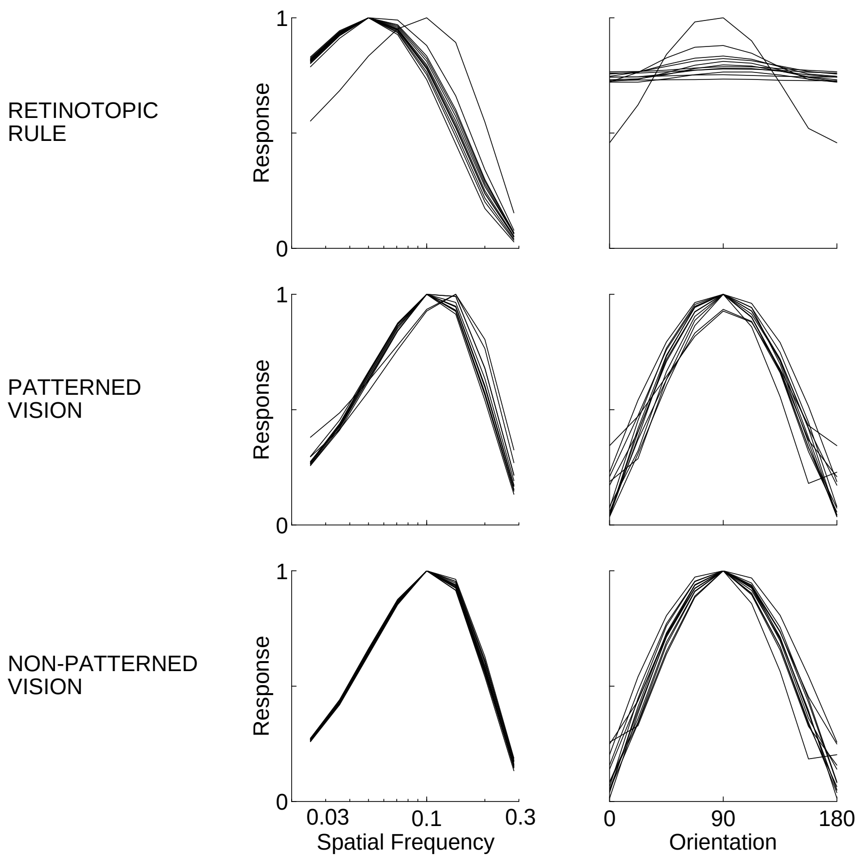

Fig. 1. Simulation of experience-dependent development of orientation and spatial frequency tuning of 12 simple cells with a reverse-Hebb rule. For both the "patterned vision" and "non-patterned vision" conditions, the *H* parameter was 0.005 and the number of learning stages was 100. Responses are normalized to the peak response over the eight orientations and eight spatial frequencies. Spatial frequency tuning curves are log-linear. Orientation tuning curves are at a spatial frequency of 0.1

Nonpatterned visual experience was simulated as the sinusoidal temporal modulation of the entire visual field (a spatial frequency of zero). The results of such a simulation are shown in Fig. 1 (bottom), again for all 12 simple cells for 100 stages with an *H* parameter of 0.005. The composite orientation tuning is virtually indistinguishable from the case of spatially patterned visual experience. While the spatial frequency tuning that results from nonpatterned visual experience shows less neuron to neuron variability than that which results from patterned vision, the peak frequencies and bandwidths are comparable in the two cases.

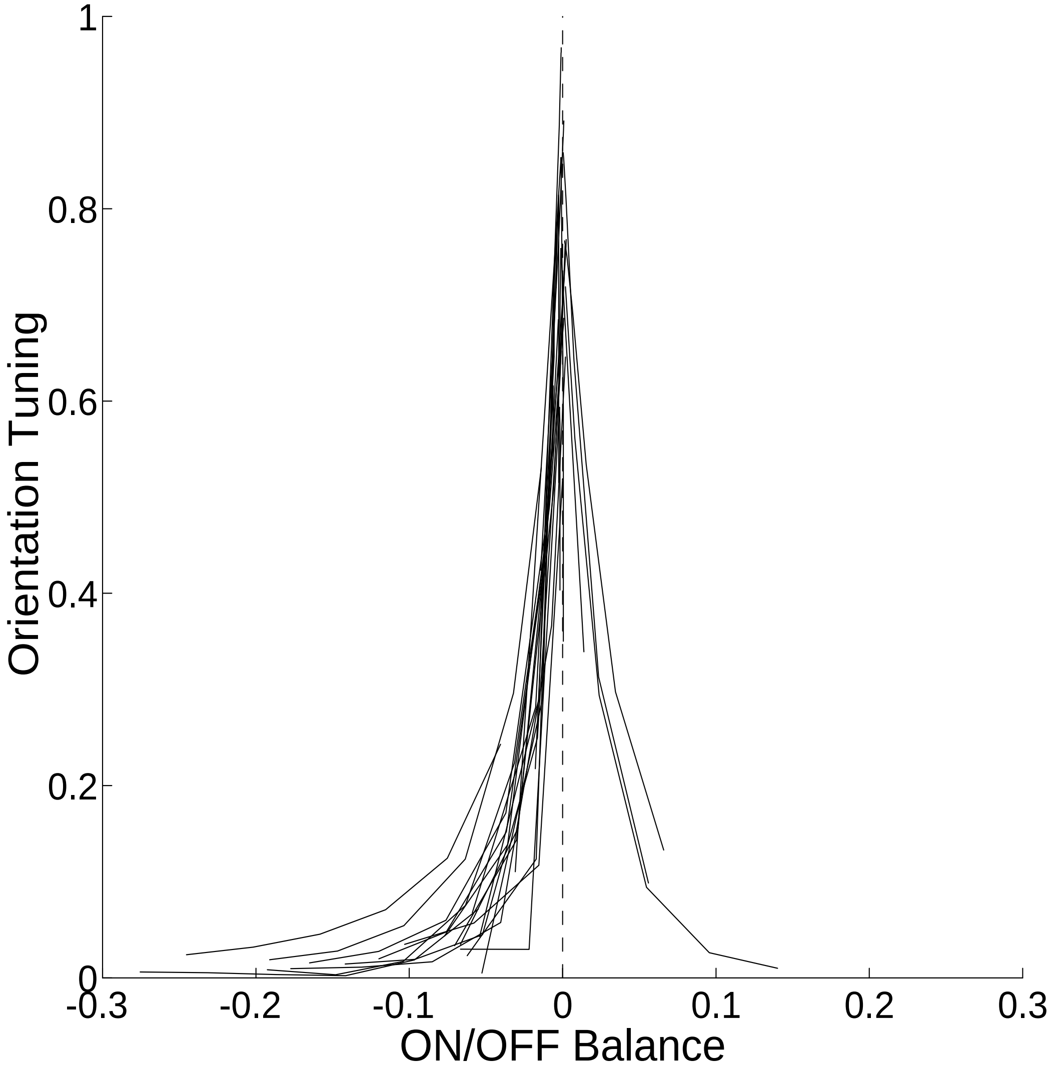

Fig. 2. Relationship between ON/OFF balance and orientation tuning for simulated development under lid-suture conditions. The *H* parameter was 0.005, and the number of learning stages was 0, 10, 20, ... 50

Figure 2 illustrates the relationship between ON/OFF balance and orientation specificity for a simulation containing 24 simple cells. The simulation was exposed to full-field temporal modulation (lid-suture condition) with an H parameter of 0.005. In this figure, orientation tuning is taken as a ratio of responses, $(R_{\max} - R_{\min}) / (R_{\max} + R_{\min})$, where $R_{\max}$ and $R_{\min}$ are the maximum and minimum responses, respectively, as a function of orientation, at a spatial frequency of 0.1 cycles/deg. This measure will range from zero (no dependence of responses on orientations) to a peak value of 1. The ON/OFF balance is represented by a similar measure $(\Sigma_{ON} - \Sigma_{OFF}) / (\Sigma_{ON} + \Sigma_{OFF})$ where Σ_{ON} and Σ_{OFF} represent the summed synaptic weight of all ON- and OFF-center afferents, respectively, onto a given simple cell. A value of zero corresponds to equal total input from ON- and OFF-center afferents, a value of +1 to pure ON-center input and a value of -1 to pure OFF-center input.

Each line in the figure represents the trajectory of one simulated simple cell, with data plotted before development and then after 10, 20, 30 ... 50 stages of learning. The line connects all these "time" points for a given cell. Two things are apparent from this figure. First, during development the input from ON- and OFF-center afferents tends to balance; this is seen as the trajectories moving towards the central line, where the ON/OFF balance is zero (although the direction of movement is not indicated in the figure, all trajectories move towards the midline). The second fact evident in Fig. 2 is the strong dependence of orientation tuning on the balance between the ON- and OFF-center afferents. As the trajectories approach the midline from either side, which corresponds to equal summed weights from ON- and OFF-center inputs, the orientation tuning rises steeply.

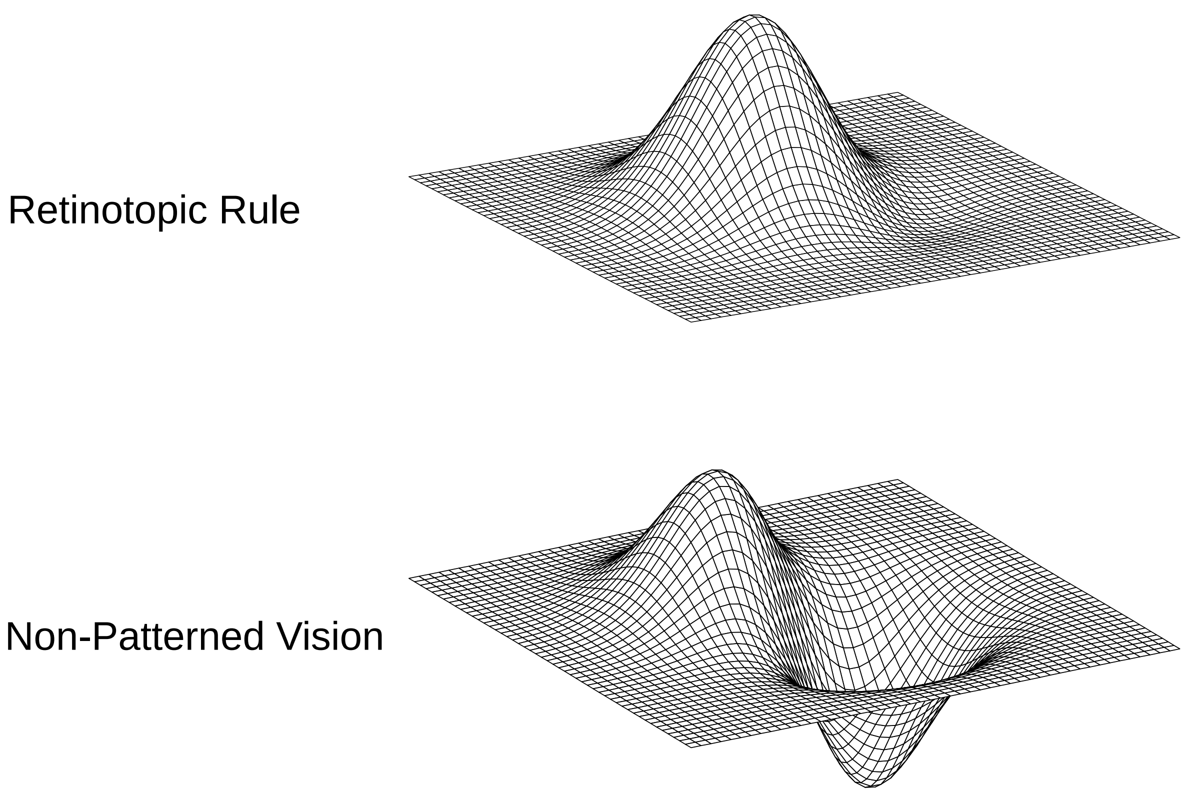

Fig. 3. Two-dimensional receptive field sensitivity profile of a simulated simple cell before and after development under lid-suture conditions. The *H* parameter was 0.005,and the number of learning stages was 100

Figures 3 and 4 illustrate the two-dimensional sensitivity profile of a simulated simple cell receptive field. Both figures show the same simple cell, Fig. 3 with sensitivity represented by the Z-axis coordinate and Fig. 4 with sensitivity represented by shading. Each figure shows the sensitivity profile before visual experience at the top, and after development under lid-suture conditions at the bottom. The area shown is 10.6 by 10.6 deg. and an ON response is represented by a positive Z-axis (upward deflection) and light shading in Figs. 3 and 4, respectively.

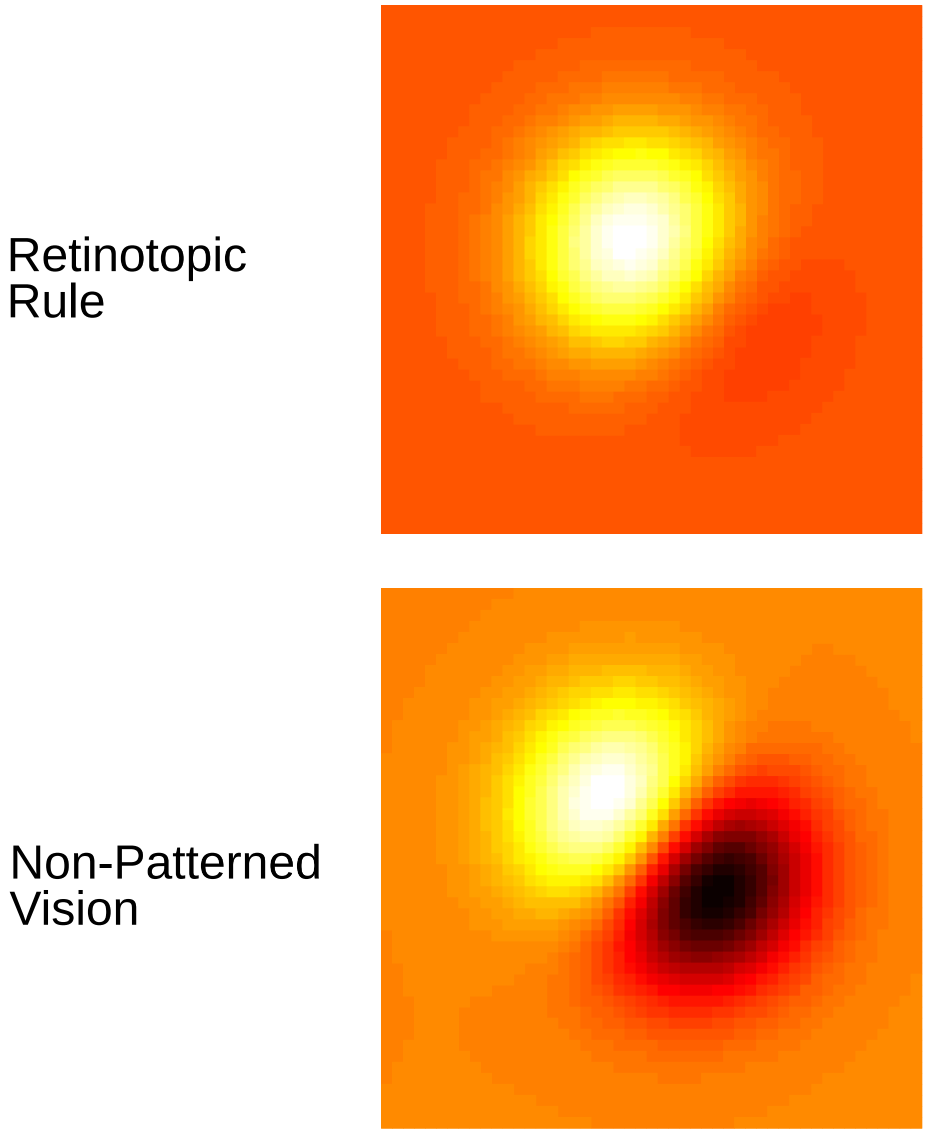

Fig. 4. Same data as in Fig. 3, with sensitivity represented by shading

4 Discussion

The adequacy of the nonpatterned visual experience to enhance the specificity of pattern vision is a consequence of the mode of action of a reverse-Hebb rule and the statistics of the RGC mosaic. A reverse-Hebb rule favors those inputs that are negatively correlated with the postsynaptic activity; thus, it will ensure a balance between ON- and OFF-center afferents under any condition in which the two types tend to fire in opposite phase. As the ON- and OFF-center RGCs are arrayed in regular but independent retinal mosaics (Wässle et al. 1981), the region of the retina which maps onto a given simple cell (i.e., the simple cell receptive field) will statistically show an offset between the "centroids" of the summed ON center and summed OFF center RGCs. Initially, however, when synaptic weights are determined only by the retinotopic rule, the ON and OFF inputs will typically not be in balance [except at chance locations on the cortical surface, giving rise to local clusters of sharply tuned neurons (Soodak 1987), as observed experimentally by Leventhal and Hirsch (1980)]. By balancing the spatially offset push-pull system, a reverse-Hebb rule will transform simple cell receptive fields into ON/OFF dipoles, resulting in a considerable degree of orientation specificity and substantial high-pass spatial frequency filtering.

Although the visual cortex of lid-sutured cats can show considerable response specificity, it cannot be considered normal. There are large numbers of unresponsive neurons (Wiesel and Hubel 1965; Sherk and Stryker 1976; Derrington 1984; Pettigrew 1974; Blakemore and Van Sluyters 1975; Singer and Tretter 1976), and some studies show far less orientation specificity than in cats reared with patterned vision (Pettigrew 1974; Blakemore and Van Sluyters 1975; Singer and Tretter 1976). There are two possible reasons why the simulations presented here fail to predict any substantial deficit from lid suture. First, the simulations assumed equivalent total retinal contrast under normal and lid-sutured conditions. As the eyelids not only absorb, but also diffuse incident illumination, total retinal contrast will be greatly reduced in lid-sutured animals. Second, the role of intracortical synaptic interactions is omitted from the simulations. The fact that the afferent drive can be structured into ON/OFF dipoles in the absence of patterned vision does not imply that the contacts between cortical neurons will become appropriately organized under such conditions.

For a cat to experience diffuse temporal luminance modulation through sutured lids, the environment must either contain a temporally modulated light source or, more realistically, a standing spatial contrast which is perceived as temporal modulation when the lid-sutured animal moves about. Differences in environmental contrast and mobility of the animals may thus be the basis of the diversity of results obtained from binocular suture experiments (Wiesel and Hubel 1965; Sherk and Stryker 1976; Derrington, 1984; Pettigrew 1974; Blakemore and Van Sluyters 1975; Singer and Tretter 1976).

While it is not clear that the response specificity in cats reared in total darkness (Frégnac and Imbert 1978; Bonds 1979; Leventhal and Hirsch 1980) is higher than would be expected from a retinotopic rule alone (Soodak 1987), the simulations of this paper suggest a possible mechanism for enhancement in the absence of all visual experience. The sufficient condition for specificity enhancement by a reverse-Hebb rule, that is, negative correlation between ON- and OFF-center RGCs, has been shown to exist in the spontaneous activity of cat ganglion cells (Mastronarde 1983). Linsker (1986a-c) and Miller and colleagues (Miller 1989, 1992, Miller et al. 1989) have presented models for the development of oriented receptive fields in the absence of visual experience, based on spontaneous correlations among afferents. However, these models assumed that spontaneous correlations show a biphasic or "Mexican hat" spatial patterning. Although the spatial dependence of ganglion cell correlations has been studied, such patterning was not observed (Mastronarde 1983). The model presented here does not require spatial patterning, of either driven or spontaneous activities, for the enhancement of response specificity.

One of the properties of a reverse-Hebb modification rule is that it is intrinsically stable with respect to retaining multiple synaptic inputs, while in contrast, a standard-Hebb rule, if employed without additional constraints, will lead to a winner-take-all outcome. The reverse-Hebb rule is intrinsically stable as it is essentially a negative-feedback process, whereas a standard-Hebb rule is unstable due to its positive-feedback properties. For this reason, a sign-inverted Hebb rule was proposed by Griffith (1966) as a mechanism which stabilized connections between peripheral nerves and their effectors, a process he termed "peripheral learning." Griffith also speculated that a sign-inverted Hebb rule acted to stabilize connections in the cerebral cortex, acting in concert with a standard-Hebb rule which was gated by a "reinforcing" input element. Hopfield et al. (1983) used a sign-inverted Hebb rule to stabilize learning which was brought about by a standard-Hebb rule, though in a very different sense. In this case, stabilization was not to prevent a winner-take-all outcome but to reduce the formation of "spurious memories" by increasing the values of minima in the energy function of the network (Hopfield 1982) which did not correspond to an input state. Recent experiments on the development of neuromuscular junctions have provided evidence in support of Griffith's hypothesis with regard to peripheral learning (Callaway et al. 1987, 1989).

Kohonen and coworkers (Kohonen et al. 1974; Kohonen and Oja 1976; Kohonen 1989) were the first to examine the behavior of a sign-inverted Hebb rule and to show that such a rule, when applied to either feedforward inputs or excitatory feedback connections, resulted in "habituation," i.e., the output was reduced in response to input patterns to which the system had been exposed. Models of adaptation in the visual cortex have been based on this habituation property, at the level of intracortical interactions (Wilson 1975; Barlow and Földiák 1989; Barlow 1990) or afferent inputs (Soodak 1991). In distinction to Kohonen and Oja's adaptive filter, in which habituation results from a decrease in the synaptic weight of excitatory feedback connections (strictly, a single variable representing the difference between excitatory and inhibitory connectivities) in response to a positive correlation of the input and output activities, in the models of Wilson and Barlow and Földiák, adaptation results from an increase in magnitude of inhibitory connections when the input and output are positively correlated.

In the theory of visual cortex development of Cooper and colleagues (Cooper et al. 1979, 1985; Bienenstock et al. 1982; Clothiaux et al. 1991), synaptic weights are modified by a rule containing a sliding threshold, which varies slowly as a function of the time-averaged value of the postsynaptic activity. When a stimulus evokes a response above this threshold value, synaptic weights are altered by a standard-Hebb rule. When a stimulus evokes a subthreshold response, the sign of weight changes are reversed. Simulations based on this theory under conditions of pattern deprivation were presented by Bienenstock et al. (1982) and Clothiaux et al. (1991). In these simulations, pattern deprivation was modeled as either a noise input vector that was uncorrelated among the afferents or as an arbitrary fixed input vector that was multiplied by a scalar noise term (correlated noise among the afferents). In the former condition, a stable relationship between inputs and output was not attained, and the average state was nonoriented. In the latter condition, a stable state was reached in which the tuning was determined by the arbitrary fixed stimulus vector present during development and the set of oriented stimuli used to assess the response properties. For the particular stimuli used in the simulations (as would be expected in general), the orientation tuning was substantially less than that which was attained in simulations of normal development.

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