MODELING VISUAL CORTEX: HIDDEN ANISOTROPIES IN AN ISOTROPIC INHIBITORY CONNECTION SCHEME

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A detailed cortex model (15,000 cells) of the adult cat is presented and it is shown that a combination of unspecific inhibitory mechanisms together with input of aligned receptive fields from the LGN with little elongation (1:1.5) is able to reproduce cortical orientation selectivity and other features of cortical cell behavior. We introduce a novel isotropic intracortical connection scheme (*"circular inhibition"*) and demonstrate analytically that this mechanism results in two anisotropies: orientation tuning and a directional bias. Thus, our network shows that structurally unspecific isotropic connections can result in functionally specific behavior. Directional anisotropy introduced in this way could be the starting point for the development of the true direction specificity found in cortical cells.

1. Introduction

Two features are outstanding in cortical architecture: (1) a high degree of uniformity of the connections between cortical cells and (2) a very high sub- and intracortical convergence onto each cortical cell (Martin 1988). Although little anatomical order seems to exist, functional elements (in the visual cortex: orientation columns, ocular dominance columns, etc.) are clearly defined, and cell response characteristics show a high degree of stimulus specificity. Low structural and high functional specificity, however, seem to contradict each other and major efforts have been undertaken to resolve this problem and to establish a link between structure and function (Braitenberg 1985, Martin 1988). Probably the best studied cell property in the visual cortex is the so called *orientation specificity* or *orientation tuning* (Hubel and Wiesel 1962). Cortical cells fire most strongly when they are stimulated with an elongated contrast step (e.g. light bar) within a restricted range of orientations. Many models have been proposed (aligned convergence from the LGN, cross-orientation inhibition, iso-orientation inhibition or excitation, etc.). In fact, there is experimental evidence for all of them. The simplest solution which would resolve the apparent conflict between low structural and high functional specificity and provide an explanation for the different experimental data is to assume that "all" mechanisms exist (Ferster and Koch 1987), that they are rather unspecific, but that the combination of all of them results in the observed functional specificity.

2.1. A Detailed Model of the Visual Cortex - Methods

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We model a 5×5 degree patch of the primary visual pathway of cat at a retinal eccentricity of about 4°, including retina, lateral geniculate nucleus (LGN) and layer IV of area 17.

Figure 1: Detailed cortex model.
(A-D) Sub- and intracortical connection patterns. (E-I) Response of the cell population in one hypercolumn. (J-N) Average orientation tuning curves; the inset (unpublished data from Wörgötter and Eysel) shows the average tuning of real cortical simple cells.

Neurons are implemented as improved integrate-and-fire units modeling soma and axon but excluding the dendrites. The model retina is stimulated by light intensity functions that correspond to moving oriented bars. Retinal ON- and OFF-center beta-ganglion cells (n=2048) are implemented on a noisy hexagonal grid. They project with a one-to-four divergence, and preserving the topography, onto 8192 ON- and OFF-center LGN cells. In the cortex, only inhibitory cells were modeled. We implemented 4096 neurons which corresponds to approximately one quarter of all inhibitory cells in the central $2.5° \times 2.5°$ part of the patch. The projection from the LGN to the cortex is approximately linear at the chosen eccentricity and the cortical magnification factor is about 1. Thus, 2.5° roughly correspond to 2.5 mm on the cortical surface, which in turn contain about 2.5 hypercolumns (Albus 1975b). According to the model of Hubel and Wiesel (1962), each cortical cell receives input from LGN cells with receptive fields (RFs) that lie in a rectangle (Fig. 1A). On average, 5x13 LGN cells converge onto one cortical cell which corresponds to an *elongation* of about 1.5. This small elongation results in a weak orientation tuning. The angle of orientation preference continuously changes along the x-axis and was held constant along the y-axis. Thus, orientation columns resemble vertical stripes. The model neurons were implemented with realistic receptive field scatter and jitter in the orientation preferences (Albus 1975). Several inhibitory intracortical mechanisms were implemented, some of which are shown in Fig. 1B-D. The most unspecific

inhibitory connections are defined by random wiring (Fig. 1B). In this case, the probability of making a connection with the cell in the center is the same for all cells within a distance of half a hypercolumn, which results in a disk-like structure of connected cells. *Circular inhibition* (Fig. 1C), which we suggest as possible intracortical connection scheme, is more specific. Only cells whose distance from the center cell is about half a hypercolumn are connected with high probability to the center cell. The resulting structure resembles an annulus. A third scheme is shown in Fig. 1D: local inhibition can be achieved by wiring all cells with similar orientation tuning, but whose receptive fields are displaced laterally, onto the center cell (Heggelund 1981). In this case, the probability of a connection is low very close to the cell in the center and at large distances. Furthermore, this probability is lowered along the long axis of the receptive field. Our model has been implemented on SUN4 workstations; for details of the implementation see Wehmeier *et al.* (1989) and Wörgötter and Koch (1990).

2.2. A Detailed Model of the Visual Cortex - Results

The center row of Fig. 1 (E-I) shows the accumulated activity of all cells within one hypercolumn after stimulation with a moving vertical bar. Little black squares represent single neurons. The size of the squares scales with the total number of spikes elicited during one stimulus sweep. Only for the rightmost panel (Fig. 1I), a realistic separation of the columns containing cells with more horizontal (left side of the panel) or more vertical orientation preference (right) can be seen.

The bottom row (Fig. 1J-N) shows the average orientation tuning achieved using the different mechanisms. Fifty-five cells were averaged after rotating their tuning curves to 0° preferred direction; plotted is the maximal impulse rate against the direction of motion of the stimulus in a polar diagram. The narrower a polar plot, the stronger is the orientation tuning; an effect which is seen to increase with increasing specificity of the mechanism (i.e. from left to right). Only the combination of all mechanisms, however, results in a realistic orientation tuning (Fig. 1N) as compared to the tuning obtained in real cells (inset between Fig. 1M,N). Note that even for the realistical orientation tuning (Fig. 1N) the column structure (Fig. 1I) remains rather fuzzy. This reflects reality. Clearer pictures of orientation columns in the cortex can only be obtained with methods that strongly amplify the columnar organization (e.g. desoxyglucose uptake). Random inhibition served as a control situation to show if a sharpening of orientation tuning can be achieved by a completely unspecific connection scheme. With local inhibition (Fig. 1D), the average orientation tuning is also strong (Fig. 1N). However, as can be seen in the population response (Fig. 1H), the columnar structure is destroyed: quite often cells which originally (i.e. with Hubel and Wiesel convergence alone) had a horizontal orientation preference will turn and now respond best to vertical stimuli. An additional observation is that all studied intracortical mechanisms yield a directional bias which is strongest in circular- and least strong in random inhibition. This effect can easily be explained for local inhibition. Due to the small number of connected cells (≈ 20) it is likely that inhibition from one side exceeds that from the other side, thus resulting in a directional bias. This explanation, however, does not hold for circular inhibition (and random inhibition) because with over 100 connected cells random asymmetries average out.

In the first part of this paper we have demonstrated that a combination of intracortical mechanisms, all of which have only very little connection specificity, results in a realistic average orientation tuning. Two intriguing findings were made:

1) Inhibition which arises isotropically around the target cell (circular inhibition) sharpens the orientation tuning.

2) An unexpected directional bias is obtained with circular inhibition.

The question arises if these effects are specifically generated by the structure of the connections in circular inhibition. The detailed cortex simulation is much too complex to allow a straight-forward analytical description. Therefore, in the next section we will introduce a simpler model that captures all essential features and that yields a better understanding of the results of the simulation.

Figure 2: Structurally confined model for *circular inhibition*
(A) Idealized cortical column structure. (B) Computation of the tuning of inhibition. The orientation selective response of the cells (1-6) is given by eq. 2. (C) Average tuning of inhibition. Open bars represent the preferred (PO) and the non-preferred (NPO) stimuli.

3.1. A structurally confined model

We define a coordinate system on the cortical surface such that the y-axis is parallel to the orientation columns (vertical in Fig. 2A). As a consequence, the preferred orientation ϕ of a cell which is located at the point (x, y) is only dependent on x, $\phi = \phi(x)$, with

$$\phi(x) = \frac{\pi}{\lambda} x, \tag{1}$$

where λ is the width of one hypercolumn (Fig. 2A).

Fig. 2B shows the orientation selective response of individual cells (1-6 and center) in the simplified model. The cell activity $\mathcal{A}$ is described as a function of the stimulus angle γ relative to the preferred orientation ϕ of the cell,

$$\mathcal{A}(\gamma - \phi) = A_0 + A_2 \cos(2\gamma - 2\phi). \tag{2}$$

A stimulus bar (δ-function bar) with orientation γ (Fig. 2B) will stimulate two cells on the circle (2 and 5) and elicit responses $\mathcal{A}$ from them as depicted by the thick lines.

In an idealized circular inhibition scheme, a cell receives input from all cells that are located on the circle with radius r around the cell in the center (e.g. cells 1-6 in Fig. 2B). For the columnar structure defined in eq. (1), the total inhibitory input to the cell at the point (x, y) is then:

$$I(x,y) = 2A_0 + A_2 \cos(2\gamma) \cos(2\pi/\lambda r \cos(\gamma + 2\pi/\lambda x)) \tag{3}$$

We were concerned with the question if circular inhibition can specifically sharpen the orientation tuning. The answer is given by the average tuning of inhibition that arises throughout the whole cell population. It is calculated as (J_0 is the Bessel function of order zero):

$$\overline{I(r,\gamma)} = \frac{1}{\lambda} \int_0^{\lambda} I(x)\, dx = 2A_0 - A_2 J_0(2\pi r/\lambda) \cos(2\gamma), \tag{4}$$

The shape of the average inhibition tuning curve, plotted relative to a horizontal preferred orientation (black RF), is shown in Fig. 2C for $r = \lambda/2$. It is seen that inhibition is considerably more efficient perpendicularly to the preferred orientation (NPO) than along the preferred orientation (PO). This shows that *circular inhibition* does not act unspecifically but *results in a net cross-orientation inhibition effect*. This finding corresponds to experimental evidence indicating that connections contributing to cross-orientation inhibition are predominantly found at a distance of half a hypercolumn (i.e. $\lambda/2$, Matsubara *et al.* 1985).

It must be emphasized that the non-locality of the intracortical connections is essential for obtaining a net cross-orientation inhibition effect. It can be seen from eq. (4) that iso-orientation effects are obtained for $r \to 0$. Consequentially, no appreciable cross-orientation inhibition is obtained if both local and non-local interactions are taken into account by adding inhibitory inputs from within a *disc* (i.e. "random inhibition", Fig. 1B).

Figure 3: The effect of circular inhibition in a real cortical column structure
(A) Column structure of cat area 18 (mod. from Swindale *et al.* (1987)). (B) Average tuning of circular inhibition for $r = \lambda/2$. (C) Ratio of the two major axes of the tuning curves for different radii r. The insets show examples of tuning curves. Curve C and the dashed curve were obtained from the artificial cortices; curve S was determined from the real cortex in (A).

3.2. Circular inhibition in a real cortex

The analytical results obtained in an artificial vertical column structure are supported by results from real cortical orientation columns. Swindale *et al.* (1987) measured and analyzed a part of the column structure of area 18 in cat (Fig. 3A) and found a mean distance between hypercolumns of $\lambda = 1.25$ mm. For this cortex structure, we have determined the inhibitory input produced by circular inhibition as a function of the circle radius r,by averaging over all cells whose distance from all borders of the cortex is at least r. Most efficient circular inhibition is obtained at a radius of half a hypercolumn ($r = \lambda/2$, Fig. 3B).

Depending on the radius r of circular inhibition, tuning curves can have a vertical or horizontal elongation (insets in Fig 3C). The ratio of I_{90}/I_0 (inset 3) as a function of r gives an indication of the actual shape. Values above 1 indicate vertical elongation of the tuning curve and, thus, a net cross-orientation effect (inset 3); values below 1 show horizontal elongation, i.e. iso-orientation interactions (insets 1 and 4). The curve obtained by the analytical calculations in the previous section (curve C) reproduces most features of the curve obtained from the experimental data (curve S), except for a shift of its maximum. This shift is due to the parallel and straight column structure (see Fig. 2A) which was used for the model calculations. Implementing more realistically curved columns (see Wörgötter *et al.*, 1990) will result in identical locations for the maxima of both curves (dashed curve). This can only be demonstrated numerically, because we were not able to find an analytical expression for the integral (analog to eq. 4) for the average tuning. The tuning curve at the maximum (Fig. 3C, inset 3) is essentially identical to the tuning observed in the real cortex (Fig. 3B). Iso-orientation interactions are obtained for small radii (inset 1), as expected from eq. (4). At the distance of about a full hypercolumn ($r \approx \lambda$), a second minimum is obtained; i.e. another iso-orientation effect occurs (inset 4). In the real cortex (curve S), no clear tuning is observed for distances $r > 0.75\lambda$. This, however, is probably due to the small number of cells (n=59 for $r = \lambda$) over which averaging could be performed to avoid border effects at this large radius. The results from a real cortex confirm the observations we made for our artificial cortices.

4. Directional bias

So far we discussed the effect elicited by a δ-function flashing bar. To demonstrate the effects elicited by motion we discuss the tangential bar position shown in Fig. 4A. The corresponding tuning curve for this individual cell is plotted in Fig. 4B. Identical inhibition is obtained for movement from bottom to top and from top to bottom, provided the movement follows exactly this axis (Fig. 4A; 1,5). This, however, is an unstable equilibrium point and any deviation from this axis results in growing asymmetries. In real cortical cells the preferred orientation of a cell varies from trial to trial within about $\pm 5^\circ$ and, in addition, orientation columns contain a jitter of $\pm 10^\circ$. Both effects result in a breaking of the symmetry along this axis. This can be simulated by assuming that the actual inhibition along any axis of stimulus motion is not obtained from a single point but rather as the average of all inhibition-values within an angle around the axis of motion. Assuming an angle of $\pm 15^\circ$, the average inhibition for the whole cell population results in a tuning curve as shown in Fig. 4C. Note that the amount of inhibition is plotted against the *direction* of motion of the bar which is orthogonal to its orientation. Thus, the tuning curve is rotated by 90° as compared to the tuning curve in Fig. 2C and still represents the same preferred and non-preferred orientations (PO,

NPO). The average asymmetry between the preferred and non-preferred *direction* (PD,NPD) is significant and would result in a direction index (for a definition see Orban 1984) of 24% which is similar to direction indices observed in the detailed cortex simulation.

Figure 4: Generation of a directional bias.
(A) Snap-shots of a bar moving across the cortical column structure. (B) Computation of direction tuning. (C) Tuning curve for an individual cell. (D) Average tuning of inhibition for the whole cell population.

5. Discussion

Two major points have been shown in this paper.

1) A combination of very low specificity mechanism can generate cortical orientation tuning.

2) Isotropically arranged connections result in anisotropic behavior.

(1) There is substantial (but controversial) experimental evidence for a multitude of intracortical mechanisms. It is, therefore, plausible to conclude that *all* those mechanisms do in fact exist. In addition, many experimentally observed effects which support any of the discussed mechanisms are rather weak or cannot be observed in all cells. This favors the view that none of the mechanisms is highly specific. This argument is supported by the low anatomical order in the cortex that can be consistent with low-specificity connection schemes. For these reasons, the combination of low specificity mechanisms in our model might in fact reflect reality. At least, our model shows that it is possible to obtain high functional specificity with low structural specificity.

The model itself, although already quite detailed, is certainly oversimplifying the actual cortical layout. Many important features are missing (e.g. all intracortical excitatory connections), but we believe that this stage represents a valid step with interesting results in the ongoing "evolution" of this model.
(2) Circular inhibition is advantageous from a developmental point of view because only distance information is required for the connections. In addition, we expected that some kind of anisotropic behavior would be generated by this mechanism because of the periodic orientation column structure in the cortex. Consequently, we found that a net cross-orientation

inhibition effect was obtained for a radius of about half a hypercolumn. The average effect is similar to that observed experimentally (Bonds 1989). The structurally confined model proved that these results are generic for circular inhibition. The change in the net effect (i.e. cross- vs. iso-) of circular connections with changing radius (Fig. 3C) is reflected in the discussions among experimental investigators about the importance of iso- vs. cross-orientation inhibition (Blakemore and Tobin 1972, Benevento *et al.* 1972, for a review see Martin 1988). Our model suggests that both effects might be mediated by the same mechanism.

The most intriguing result was the generation of a directional bias with circular inhibition. The structural model indicated that this effect is due to the different degrees of inhibition elicited along different axes of motion. Stochastic processes (e.g. jitter in the preferred orientation) will break the unstable symmetry along the preferred axis of motion and result in a directional bias.

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