Circular Inhibition: A New Concept in Long-Range Interactions in the Mammalian Visual Cortex

Ernst Niebur and Florentin Wörgötter

January 31, 1990

California Institute of Technology, Computation and Neural Systems Program
Pasadena, CA 91125, USA

Abstract

Horizontal long range interactions are strongly involved in the generation of the receptive fields of visual cortical cells. This study demonstrates structurally imposed limitations of long range interactions. In particular, it is shown that the long discussed cross orientation inhibition scheme leads to inhomogeneous input for different cell populations which is experimentally not observed. This is not the case for circular inhibition, a new connection scheme we propose for long-range interaction. We show this by computer simulation of the early visual system of the cat and by a simpler but analytically solvable model. The results are confirmed by applying our methods to the experimentally determined structure of the orientational hypercolumns in area 18 of the cat.

1 Introduction

The importance of inhibitory interactions among the constituent elements of artificial and biological neural nets has long been recognized. Most work is focused on local or short range interactions between neurons. There is also, however, substantial evidence for inhibitory and excitatory long range connections (for a review see Martin 1988).

The visual cortex shows a high degree of functional specificity (e.g. orientation columns), and for distances above 0.2° a well-defined mapping from the visual field onto the cortex exists (Albus 1975a,b). For shorter distances the topography is lost in the random scatter of the receptive fields (RFs). In other words, the visual cortex is (at least) functionally highly anisotropical. Long range connections extend over distances on the cortical surface which are larger than the equivalent of 0.2°. The most obvious assumption is that the functional anisotropy over such long distances is achieved by highly specific wiring between cells, i.e. that cells are not wired onto their targets by chance.

This assumption, however, is not necessarily true and in this paper we will show that low-specific long-range interactions between neurons can perform better than highly specific connections. This will be demonstrated by the example of cortical orientation selectivity.

In Section 2 we study the tuning of the orientation selective neurons by intracortical long-range interactions, using a detailed computer simulation of the early visual system. We find that a certain type of intracortical long-range inhibition (cross-orientation inhibition, Sillito 1979) leads to unexpected and experimentally not observed asymmetries in the behavior of different populations of cortical cells. Instead of cross-orientation inhibition we introduce a new mechanism (*circular inhibition*) which is considerably less specific and we will show that it eliminates unwanted asymmetries. In Section 3, we discuss a simple, analytically solvable model which provides more insight into the underlying mechanism than detailed simulations. In Section 4, we apply the model to the experimentally determined structure of the visual cortex.

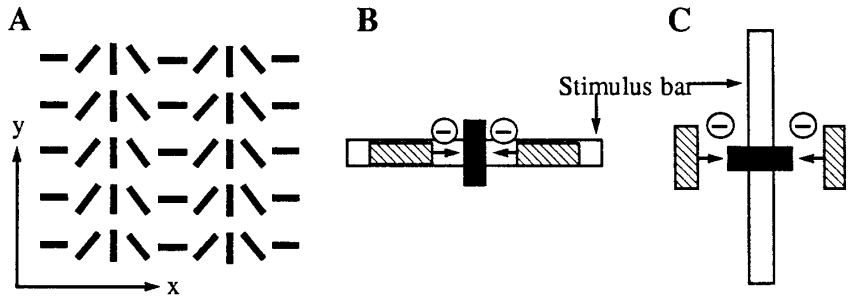

Figure 1: A) Orientation column structure. B) Light bar (stippled rectangle) acting on RFs of a cortical neuron (black) and of two other inhibitory cortical neurons (shaded) which provide input (arrows) to the center cell. C) As in B, but with preferred orientation of the center cell and stimulus orientation orthogonal to that from (A).

2 Circular Inhibition

One of the most elementary properties of cells in the visual cortex of mammals is their preference for elongated bar stimuli of a certain orientation. The first model (Hubel and Wiesel, 1962) postulated that this so-called "orientation selectivity" arises from an appropriate alignment of sub-cortical synaptic input to the cortical cells. More recent work showed that intracortical interactions contribute considerably to the shaping of the receptive fields of cortical neurons (for a review see Sillito 1984).

Cells in visual cortex are organized in orientation columns, such that cells with similar orientation preference are grouped together. One complete set of orientation columns, spanning all orientations, is called a "hypercolumn" (Hubel and Wiesel 1963). The width of a hypercolumn on the cortical surface (at about 5° eccentricity) of the cat is about 1 mm (Albus 1975b, see also Section 4).

The geometrical arrangement of hypercolumns is not known exactly (for a discussion of the underlying problems see Braitenberg 1984). We assume in this section and in Section 3 that the hypercolumns are arranged in parallel rows (see Fig. 1a). This is certainly a highly simplified model, but comparison of the results obtained in Sections 2 and 3 with this model with the physiological data used in Section 4 shows that the model seems to contain the essential features of the problem we are studying.

In principle, orientation tuning can be sharpened by cross-orientation inhibition. Figure 1B,C illustrates how cross-orientation inhibition acts on cells in a model cortex. In the classical cross-orientation inhibition scheme it is assumed that a given cell receives inhibitory synaptic input from neurons which have a preferred orientation orthogonal to the preferred orientation of the target cell. Although, at first sight, this mechanism seems to be very efficient for sharpening the orientation tuning, it is seen from Fig. 1 that this is only true for a subset of cells. This is due to the fact that a horizontal stimulus in Fig. 1B optimally covers the horizontally oriented RFs of the inhibitory cells, whereas the vertical bar does *not* cover the vertical RFs in Fig. 1c. Thus, cells with vertical and horizontal preferred orientation receive unequal amounts of inhibition when stimulated with their non-optimal stimuli.

This simplified scheme neglects the receptive field scatter and overlap, but the relevance of the above observation can be tested in a more realistic situation which includes these features. The same parallel structure for the orientation columns in the cortex is used. Individual pre-shaped RFs are generated by a superposition of *differences of Gaussians* according to their input from the Lateral Geniculate Nucleus (LGN). The RF of the cortical cell in the center is simulated by a spatial superposition (with appropriate weights) of all preshaped RFs that converge onto this cell. An example of a receptive field which is generated in this way is shown in the top left corner of Fig. 2. This figure shows the ratio R between length and width (determined at half of the peak height) of RFs for two cells, one with vertical and one with horizontal preferred orientation. Both cells receive a highly similar connection pattern from the LGN. The ratio R is small and essentially identical for both cells when the RFs are generated exclusively by LGN convergence (leftmost data points). In the classical cross-orientation inhibition scheme, only cells

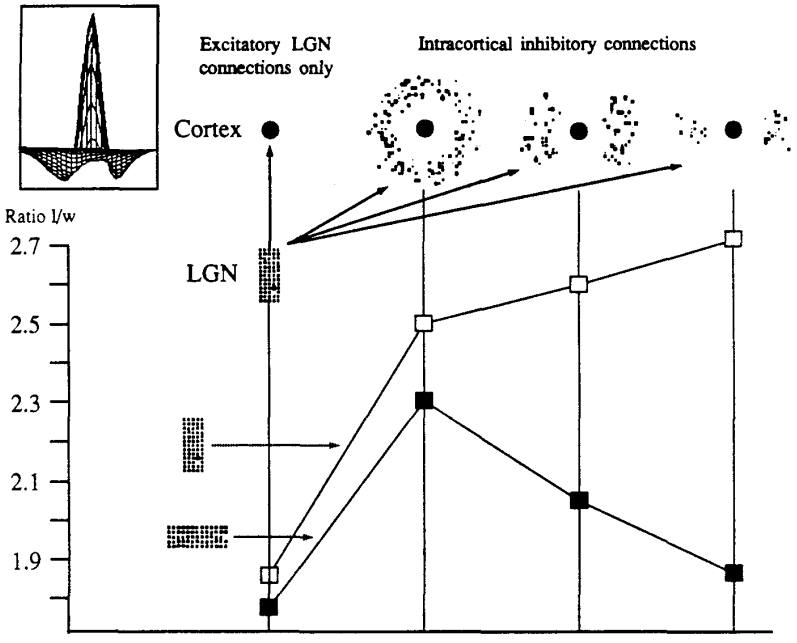

Figure 2: Ratio RF length/width for different circuitries. All cells receive excitatory input from LGN (left). Three other data sets show the ratio as function of the angle from which intracortical inhibition is received by center cell (top). White (black) squares: Preferred orientation parallel (orthogonal) to the hypercolumns.

within a small angle (here $\pm 22.5°$, rightmost data points) are connected to the center cell. (The center cell is shown by a black filled circle in the upper part of Fig. 2, and its presynaptic partners are represented by smaller black squares.) The ratio R is large for the cell with vertical preferred orientation. It stays, at a low value, however, for the other cell. This difference exists not only between single cells but between the complete populations of vertically and horizontally oriented cells. Although differences in tuning strength of individual cortical cells are abundant, differences in the tuning between whole cell populations have never been observed experimentally.

How can the tuning of inhibition be made more homogeneous? The answer is shown in the middle data points of Fig. 2. Increasing the angle within which the cells get their inhibition reduces R for the vertical cell and increases it for the horizontal cell. When the input comes from all cells on a full circle around the center cell, R is very similar for both cells. We call this circuitry scheme *circular inhibition* and it is seen that it reduces the asymmetry between the tuning strength of the cell populations with vertical and horizontal preferred orientation substantially.

3 Model Calculations

To gain better insight in the underlying mechanisms that lead to the results described above we study the simple model presented in the beginning of Section 2 by analytical calculations.

The first assumption in the sections above was that hypercolumns are parallel. Additionally we assume a one-to-one projection from the visual field to the cortex, and we choose a coordinate system on the cortex in which the y-axis is parallel to the hypercolumns and the x-axis orthogonal to them. As a consequence,

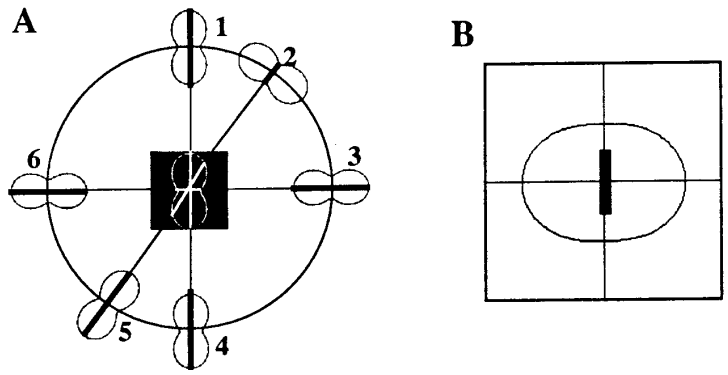

Figure 3: A) Neuron with 6 presynaptic partners (1-6) in equal distance. Shape of RFs is shown by closed curves. Input to center cell, shown for three stimulus bars (thin straight lines) is proportional to overlap with the RFs (thick black lines). B) Polar diagram of $\overline{I(\lambda/2,\gamma)}$, cf. eq. (4)

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.

In our 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,[1]

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

A stimulus bar (δ-function bar) which is centered on a cell with a preferred orientation of ϕ will then elicit a response 2 $\mathcal{A}(\gamma - \phi)$ (see Fig. 3A).

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. For the columnar structure defined in eq. (1), the input to the cell at the point (x, y) is found to be:

$$I(x, y) = 4[A_0 + A_2 \cos(2\gamma) \cos(2\pi/\lambda r \sin(\gamma + 2\pi/\lambda x))] \tag{3}$$

The tuning that arises within the whole population of cells along any stimulus angle γ and for a radius r of the circular inhibition is then calculated as the average

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

where J_0 is the Bessel function of order zero.

There are indications that connections contributing to cross-orientation inhibition are predominately found at the distance of half a hypercolumn (i.e. $\lambda/2$, Wörgötter *et al.* 1990; Hata *et al.* 1988). Figure 3 shows $\overline{I(\lambda/2,\gamma)}$ in a polar diagram as a function of γ. The preferred orientation is indicated by the black bar in the center. It is seen that inhibition is considerably more efficient perpendicularly to the preferred orientation than along the preferred orientation, which shows that circular inhibition will result in a net cross-orientation inhibition effect. We would like to emphasize that the non-locality of the intracortical connections is essential for obtaining cross-orientation inhibition. It can be seen from eq. (4) (see also Fig. 4B, inset 1) that iso-orientation effects are obtained for small r. If both local and non-local interactions

[1] For a theoretically more rigorous treatment of this model see Batschelet 1981; Thisbos and Levick 1985; Swindale et al. 1987; Wörgötter and Eysel 1987.

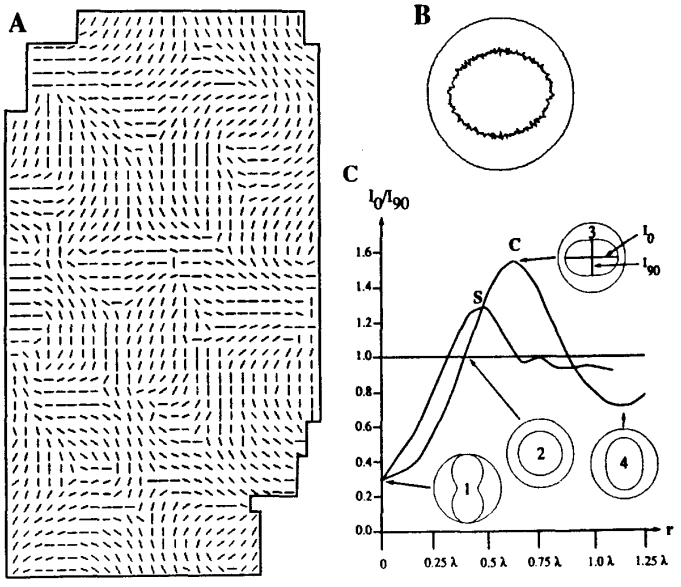

Figure 4: A) Orientation column structure (Swindale *et al.*, 1987). B) Average tuning of circular inhibition (r = 0.62 mm) for the cortex in (A). C) $I_{90}/I_0(r)$. Curve S obtained from cortex in (A), curve C from eq. (4). Insets: Polar plots of the tuning obtained from eq. (4).

are taken into account by adding inhibitory inputs from within a *disc* (and not from a circle), no appreciable cross-orientation inhibition is obtained (data not shown).

4 Application to a Real Cortex

So far our results were obtained with artificial column structures. Fig. 4A shows[2] a part of the column structure of area 18 in a cat, measured and analyzed by Swindale *et al.* (1987). They find 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. Averaging over all cells whose distance from all borders of the cortex is at least r, we found that the most efficient circular inhibition is obtained at a radius of about half a hypercolumn ($r = \lambda/2$, Fig. 4B). Fig. 4C shows the ratio of I_{90}/I_0 as a function of r. Values above 1 indicate a net cross-orientation effect; values below show iso-orientation interactions (see also insets). The comparison of the theoretically expected curve (curve C) with the curve obtained from the experimental data (curve S) shows high similarity. The shift of the maximum of the theoretical curve C is due to the artificial column structure (see Fig. 1A). This can be demonstrated conclusively, but would go beyond the limits of this paper. The tuning curve at the maximum (Fig. 4C, inset 3) is also essentially identical to the tuning observed in the real cortex (Fig. 4B). For small radii, iso-orientation interactions are obtained (inset 1 in Fig. 4C), as expected from eq. (4). For larger r, the calculation in the previous section yielded a minimum of the tuning curve; i.e. another iso-orientation effect, with values below 1 (inset 4 in Fig. 4B). In the real cortex (curve S), no clear tuning is observed for distances $r > 0.75\lambda$. This, however, could be due to the rather small number of cells (n=59 for $r = \lambda$) over which we could average to avoid border effects at this large radius. The results from a real cortex confirm the observations we made for our artificial cortices.

[2]Modified from Swindale *et al.* (1987).

5 Discussion and Conclusion

There is substantial experimental evidence that shaping of receptive fields by inhibitory long range interactions enhances the orientation selectivity of cortical neurons (for a review see Sillito 1984). We show that the classical cross-orientation inhibition connection pattern leads to inhomogeneous inhibitory input for different cell populations, and that this is not the case if the connections are arranged circularly symmetrically around the target cell. We call this connection pattern *circular inhibition*. It has both the advantage of providing consistent input to all cells, and is conceptually simpler. Furthermore, growing of synaptic connections during development following this scheme is largely simplified, since each cell has only to "know" at which distance it has to make synaptic connections. In the classical scheme, the cell would have to grow synapses specifically towards cells whose preferred orientation is (or will be later) orthogonal to its own. In general, this study presents the intriguing finding that despite the strong functional anisotropy in the cortex low-specificity connection schemes can outperform mechanisms with higher specificity.

ACKNOWLEDGEMENTS
EN is supported by the Swiss National Science Foundation through Grant No. 8220-25941 and FW by the Deutsche Forschungsgemeinschaft through Grant No. Wo 388-1. The authors gratefully acknowledge the support from the Air Force Office of Scientific Research and of a NFS Presidential Young Investigator Award and from the James S. McDonnell Foundation to C. Koch.

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