Sharpening of orientation selective receptive fields in the mammalian visual cortex by long-range interactions

Ernst Niebur*and Florentin Wörgötter†

April 9, 2013

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

Abstract

Lateral intracortical interactions are believed to be responsible for the sharpening of the receptive field profiles of visual cortical cells. This study demonstrates a structurally imposed limitation of long range interactions on the frequently invoked cross orientation inhibition scheme: it leads to inhomogeneous input for different cell populations which is experimentally not observed. We propose a novel connection scheme called "circular inhibition" which circumvents this problem. The scheme is analyzed by computer simulation of the early visual system of the cat, and by studying a simple analytically solvable model. Our model yields results consistent with the experimentally determined structure of the orientational hypercolumns in area 18 of the cat.

1 Introduction

Computer Vision still has a long way to go! There is no doubt that considerable progress has been made in this field, and that the performance on specific tasks is sometimes impressing. But overall, machine vision must be considered as a science in its infancy if it is compared to the visual system of many animals. Even small mammals or birds with a brain of a few grams outperform the most sophisticated man-made vision systems by far. It is thus a natural approach to study biological neural networks who solve the hard problems of vision apparently effortless. That this is a promising strategy is shown by the work of Mead and Mahowald who re-built the first -and best understood- element of the visual system, the retina, in analog VLSI-technology (Mead and Mahowald, 1988; Mead, 1989). While it is certainly a long way from their "Silicon Retina" to the construction of a "Silicon Cortex", it is clear that knowledge of the principles used by biological visual

*To whom correspondence should be addressed.

†Present address: Institut für Neurophysiologie, Ruhr-Universität Bochum, D-4630 Bochum, West Germany

systems can help us to build better artificial systems. In this paper, we want to make a contribution to the understanding of these principles.

The mammalian visual cortex shows a high degree of functional specificity, and for distances above 0.2° a topographical mapping from the visual field onto the cortex exists (Albus 1975 a,b). For shorter distances, the topography is lost in the random scatter of the receptive fields (RFs). Long range connections extend over distances on the cortical surface which are larger than the equivalent of 0.2°. The question arises if a detailed connection scheme between all neurons is necessary for the functioning of the cortex, even over such long distances, or if less specific connection schemes are sufficent.

We will show that low-specific long-range interactions between neurons can perform even better than highly specific connections for the task we are studying, namely the sharpening of the orientation selectivity of simple cell in primary cortex. 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 of the cat. We find that a certain type of specific 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 non-physiological asymmetries. In Section 3, we discuss a simple, analytically solvable model which provides insight into the underlying mechanism. In Section 4, we apply the model to the experimentally determined structure of the visual cortex.

2 Cross-Orientation Inhibition *vs.* Circular Inhibition

Processing of visual information starts in the retina. Although many details remain to be understood, we probably have now a fairly accurate idea of the basic principles of operation of this part of the central nervous system (see Dowling, 1987 for a review). The next stage of the major visual pathway of mammals is the Lateral Geniculate Nucleus (LGN), a layered structure in the Thalamus. All axons from retinal ganglion cells make synapses on LGN cells which project on their turn to Layer 4 of the primary visual cortex (area 17). While the receptive fields of retinal ganglion cells and of LGN cells are all circular, many neurons in V1 are characterized by 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 thalamic synaptic input (from the LGN) to the cortical cells. More recent work showed that this picture is probably too simple and that intracortical interactions contribute to the shaping of the receptive fields of cortical neurons (for a review see Sillito 1984).

Cells in visual cortex are arranged such that cells with similar orientation preference are grouped together. A set of cells with similar orientation preference is called an "orientation column", and a 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 from the fovea) of the cat is about 1 mm (Albus 1975b, see also Section 4).

Orientation hypercolumns form a rather complex pattern on the cortical surface (Brait-

Fig. 1: (A) Parallel orientation column structure. Black bars represent the preferred orientation of cortical cells. (B) Light bar (stippled rectangle) acting on RFs of a cortical neuron (black) and of two other inhibitory cortical neurons (shaded). The latter provide inhibitory input (arrows) to the center cell. (C) As in (B), but with preferred orientation of the center cell and stimulus orientation orthogonal to that in (A).

enberg, 1984). The details of this structure are not well understood, although some insight has been obtained recently (Durbin and Mitchison, 1990) in terms of dimension reducing mappings, from the many-dimensional parameter space of visual input (comprising ocular dominance, location, orientation,...) to the 2-dimensional cortical sheet. We assume in this section and in Section 3 that the hypercolumns are arranged in parallel rows (see Fig. 1a). This is a rather drastical simplification, 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.

A possible mechanism to obtain orientation tuning is 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 (Wörgötter, Niebur and Koch, 1990). 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 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 l/w 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 l/w is small and essentially identical for both cells when the RFs are generated exclusively by LGN

Fig. 2: Ratio length/width of Rfs for different intracortical circuitry schemes. Left is shown a cell which receives only excitatory input from the LGN. The other three other data sets show this ratio for cells which receive also intracortical inhibition from a sector orthogonal to their preferred orientation. These sectors are different for the three cells, as shown on top of the figure. Squares: preferred orientation of the center cell is parallel to the hypercolumns, Circles: preferred orientation is orthogonal to the hypercolumns. The lines are merely a guide for the eye. The inset shows a typical receptive field, as used in the simulation (see Wörgötter*et al.*, 1990 for details).

convergence (leftmost data points). In the classical cross-orientation inhibition scheme, only cells within a small angle (here $\pm 22.5°$, rightmost data points) are connected to the center cell, because only those cells do have the correct orientation preference (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 l/w 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 central sets of data points in Fig. 2. Increasing the angle within which the cells get their inhibition reduces l/w 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, l/w 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.

Fig. 3: Left: Neuron with 6 presynaptic partners (1-6) in equal distance. The shape of the RFs is shown by closed curves. The input to the center cell (white on black ground), shown for three stimulus bars (thin straight lines) is proportional to the overlap with the RFs (thick black lines). Right: Polar diagram of $\overline{I(\lambda/2,\gamma)}$, cf. eq. (4).

3 A Simplified Model for Circular Inhibition

As in the previous sections, we assume parallel hypercolumns and a one-to-one projection from the visual field to the cortex. 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, 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.

We obtain an analytically tractable model by assuming furthermore that 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. 3).

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 as,

$$I(x, y) = 2\mathcal{A}(\gamma - \phi(x - r\cos\gamma)) + 2\mathcal{A}(\gamma - \phi(x + r\cos\gamma)), \tag{3}$$

which yields

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

[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.

Fig. 4: Left: Orientation column structure in cat area 18 (modified from Swindale *et al.*, 1987). Right: Average tuning of circular inhibition ($r = 0.62$ mm) for the cortex in (A).

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

$$\overline{I(r,\gamma)} = \frac{1}{\lambda}\int_0^\lambda I(x)\,dx = 4A_0 - 2A_2 J_0(2\pi r/\lambda)\cos(2\gamma), \tag{5}$$

where J_0 is the Bessel function of order zero.

Figure 3 shows $\overline{I(r,\gamma)}$, for $r = \lambda/2$, in a polar diagram as a function of γ. The reason why we have chosen this radius of the circle of inhibition is that inhibitory connections are predominately found having a length of half a hypercolumn (i.e. $\lambda/2$, Wörgötter *et al.* 1990; Hata *et al.* 1988). 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. (5) that iso-orientation inhibition is obtained for small r. If both local and non-local interactions 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 Circular Inhibition in a Real Cortex

In this section, we will no longer make the assumption of parallel hypercolumns. Instead, we will study circular inhibition in a real cortex. Figure 4 shows 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

Fig. 5: Ratio of inhibitory input orthogonal and along the preferred orientation $I_{90}(r)/I_0(r)$ of cells in the real cortex, shown in Fig. 4 (curve S), compared to the model in Section 3, eq. (4) (curve C).

half a hypercolumn ($r = \lambda/2$, Fig. 4). The efficiency of circular inhibition, measured as the ratio of inhibitory input orthogonal to the preferred orientation relative to this input parallel to the preferred orientation is shown in Fig. 5 as a function of r. Values above unity indicate a net cross-orientation effect; values below show iso-orientation interactions. The qualitative features of the theoretically expected curve (curve C) agree with those of the curve obtained from the experimental data (curve S). For small radii, iso-orientation interactions are obtained, as expected from eq. (5). 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 unity. 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.

5 Discussion and Conclusion

The detailed nature of the mechanisms leading to the sharpening of the orientation tuning is still a matter of discussion. Several models have been proposed and, indeed, there is some experimental evidence for most of these. The simplest solution for this apparent contradiction is an "ecclectic model" (Ferster and Koch, 1987), i. e. the assumption that "all" mechanisms exist. Each mechanism is assumed to be relatively unspecific and it is their combination which leads to the observed tuning of inhibition.

There is substantial experimental evidence that the shaping of receptive fields by inhibitory long range interactions is one (if not *the*) mechanism for enhancing the orientation selectivity of cortical neurons (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 it is conceptually simpler than cross-orientation inhibition. This mechanism is thus ideally suited to work in concert with other, relatively unspecific mechanisms, in the spirit of the ecclectic model. Another advantage of circular inhibition is the simplicity of the synaptic circuitry. Growing of the synaptic connections during development following this scheme is simpler than it is necessary for cross-orientation inhibition, since each cell has only to "know" at which distance it has to make synaptic connections. In the classical cross-orientation scheme, the cell would have to grow synapses specifically towards cells whose preferred orientation is (or will be later) orthogonal to its own.

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 Christof Koch.

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