AN ABSTRACT MODEL OF A CORTICAL HYPERCOLUMN

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ABSTRACT

An abstract model of a cortical hypercolumn is presented. This model could replicate experimental findings relating to the orientation tuning mechanism in the primary visual cortex. Properties of the orientation selective cells in the primary visual cortex like, contrast-invariance and response saturation were demonstrated in simulations. We hypothesize that broadly tuned inhibition and local excitatory connections are sufficient for achieving this behavior. We have shown that the local intracortical connectivity of the model is to some extent biologically plausible.

1. INTRODUCTION

Most neurons in the primary visual cortex (V1) respond to specific orientations even though relay cells in the lateral geniculate nucleus (LGN), that carries the information from retina to V1, does not show evidence of orientation selectivity. It is not known in detail how orientation selectivity of the cells in V1 emerges and the issue is hotly debated (for a recent review see Ferster et al., [9]). Hubel and Wiesel [10] proposed that orientation selectivity of simple cells in V1 was a consequence of synaptic input from LGN. Still today the Hubel and Wiesel feedforward model serves as a model of thalamic input to cortex. However many of the properties of orientation selective cells in V1 cannot be predicted by such a feedforward model. Contrast-invariance of orientation tuning seen by simple and complex cells is perhaps the most striking example. As contrast increases the height of the response curve increases while the width remains almost constant [11,12,13].

It was also shown that response to contrast stimulus increases over approximately 50-60% of the response range and this behavior is followed by a rapid saturation and normalization of the cells activity [13]. The saturation level seems to be determined by stimulus property (orientation, spatial frequency) and not by electrical properties of the cells. Maximum response to non-preferred stimulus was reported to be lower than to preferred stimulus.

According to the findings by Hubel and Wiesel [16] the primary visual cortex has a modular structure. It is composed of orientation minicolumns each one comprising some hundreds of pyramidal cells and a smaller number of inhibitory interneurons of different kinds. Contrast edge orientation is coded such that the cells in each orientation minicolumn respond selectively to a quite broad interval of orientations. Further, the orientation hypercolumn contains orientation minicolumns with response properties distributed over all angles, and thus represents the local edge orientation pertinent to a given point in visual space. A similar modular arrangement is found in many other cortical areas, e.g. rodent whisker barrels [15].

The Bayesian Confidence Propagation Neural Network model (BCPNN) has been developed in analogy with this possibly generic cortical structure [14]. This is an abstract neural network model in which each unit corresponds to a cortical minicolumn. The network is partitioned into hypercolumn-like modules and the summed activity within each hypercolumn is normalized to one.

The above network model relates to the so-called normalization models of V1 proposed by Albrecht et al. [20] and Heeger [21] that address properties of simple cells mentioned above. These assume that input from the LGN grows linearly with contrast stimulus. This input is divided by a linearly growing inhibitory input. The effect is division of the input from the LGN and that the summed activity of the cells in a hypercolumn is normalized. This would correspond to saturation of a cells activity. Later Carandini et al. [22,23] proposed that a pool of cells with different preferred orientations and spatial frequencies drives the shunting inhibition.

Cross-orientation inhibition is yet another feature of cortical simple and complex cells. Response to superposition of two gratings is less than sum of each response alone [8,25]. Morrone et al. [8] suggested that this inhibition arises from a pool of cells with different orientations. The cross-inhibition effect could be

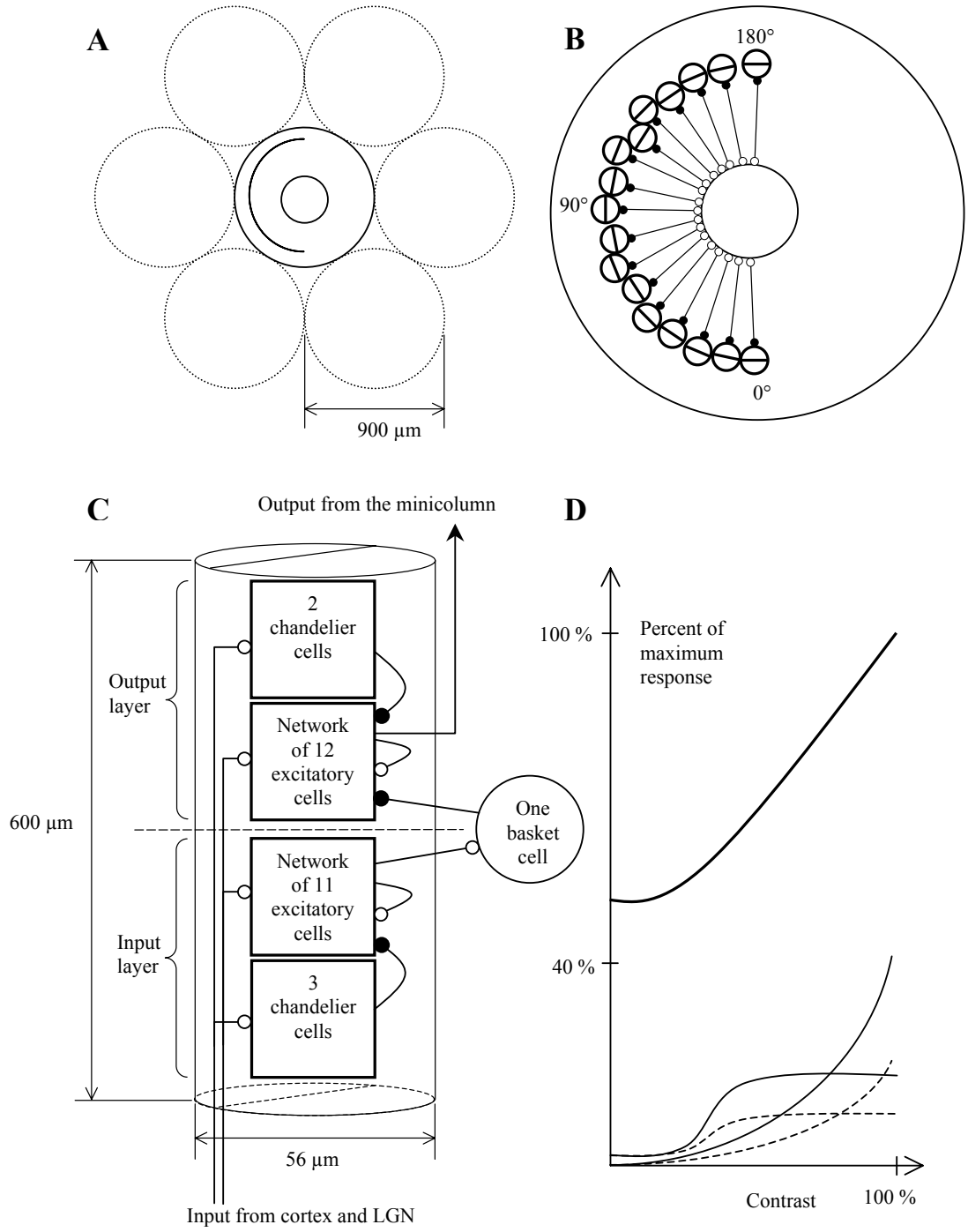

Figure 1. *A*, The hypothesized repetitive layout of the cat V1 demonstrated by 7 hypercolumns. *B,* The partial hypercolumn model used during the simulations consisted of 17 minicolumns. *C*, A scheme showing one of the subsampled orientation minicolumns and the basket cell representing the pool of inhibitory cells. Excitatory cells in the input layer are connected to the basket cell, and the basket cell inhibits the excitatory cells in the output layer. *D*, A scheme showing the activity of the excitatory cells in two minicolumns with preferred orientation (bottom solid lines) and non-preferred orientation (bottom dashed lines). Input layer excitatory cells (exponential curves) are driving the basket cell (thick top curve). The output layer excitatory cells (sigmoidal curves) are normalized when the basket cell's activity increases linearly.

explained by a shunting inhibition proposed by the normalization models [22,23].

Here we present an abstract model of a cortical hypercolumn derived from the above-mentioned BCPNN architecture. Our main intention has been to address response saturation and contrast-invariance of orientation tuning behaviors of cortical cells. Initial tests were showing that cross-orientation inhibition was also prominent. All these behaviors could be achieved by a very simple network architecture (Fig. 1).

2. NETWORK MODEL

The foundation of our network model is the columnar organization seen in V1 and elsewhere in the cortex [16]. We assume that V1 is composed of repetitive structures, so-called minicolumns, and that these minicolumns rotate around hypothetical centers [18], and form hypercolumns (Fig. 1a). In our model a hypercolumn is represented by a finite number of minicolumns, each representing a particular orientation (Fig. 1b). For sake of simplicity we decided to use a partial hypercolumn model composed of 17 subsampled orientation minicolumns, ranging from 0° to 180°, with the angular distance of 11.25° between two successive ones (Fig. 1b). The diameter of the cylinder shaped minicolumns was 56 μm, as a consequence of the study done by Peters et al. [17] on cat V1. In that study, it was reported that apical dendrites of layer V pyramid cells formed clusters with a center-to-center spacing of about 56 μm, which provides an estimate of the mean distance between two successive minicolumns. The circular arrangement of the minicolumns gives the biologically plausible hypercolumn diameter of 900 μm for cat V1. The minicolumns has a height of 600 μm, and are abstractions of the layer II-IV of cat V1 [17]. Each of the subsampled minicolumns is composed of 28 neurons (Fig. 1c) and the neuron population is heterogeneous with all values sampled from a uniform distribution with a standard deviation of 10%.

The hypercolumn model consists of two separate layers with two different tasks in order to achieve normalization behavior proposed by the BCPNN model (Fig. 1c). It will be shown later that the excitatory neuron in the output layer replicates experimental findings relating to the orientation tuning mechanism in V1.

There were no connections between these two layers, and thus the interaction between them was through one basket cell, representing a pool of inhibitory cells (Fig. 1b and 1c). This layout resulted in feedforward connectivity inside the hypercolumn model. Every input layer excitatory cell was connected to the basket cell, and thus could drive it. The basket cell was connected to every excitatory cell in the output layer. There were no connections between excitatory cells situated in different orientation minicolumns. Connection probability between two excitatory neurons inside a minicolumn layer was a function of the distance between them [6].

The implication of this scheme for the output layer is that, the distribution (in terms of orientation) of the inhibitory input to an excitatory cell is broader than the excitatory input. This is because the basket cell represents a pool of orientation specific neurons inhibiting a pool of excitatory neurons with all possible preferred orientation. Even though the connectivity pattern seen in the output layer is very simple, it is still biologically plausible. Kisvárday et al. [27] reported that, in an area of the size of cat V1 hypercolumn, 56 % of the excitatory and 47 % of the inhibitory connections were at iso-orientation, while cross-inhibition was shown by 14 % of excitatory and 20 % of inhibitory connections respectively. This indicates that, the inhibitory network is less orientation specific than the excitatory network. A study based on ferret prefrontal microcircuits is also pointing in the direction of the basket cells as responsible for gain control of the local cortical network [26].

Besides the basket cell, there were other inhibitory cells in the model. These cells were the local inhibitory chandelier cells [28] located inside the minicolumns, and hence inhibiting excitatory cells with the same orientation preference. In the input layer three chandelier cells inhibited an excitatory cell, while two chandelier cells inhibited the excitatory cells in the output layer.

Cortical neurons are known for their irregular spiking activity [3,4], and were thus modeled as Poisson processes. As the kernel of the Poisson process we used a leaky integrate-and-fire model [1,2]. The role of the leaky integrate-and-fire model was to sum the presynaptic inputs to generate the membrane potential of the cells. Maximum frequency of the excitatory and the inhibitory neurons were 100 Hz and 300 Hz respectively. Half-height of the IPSPs were 10 ms, and 15 ms for the EPSPs. Mean amplitudes of the EPSPs inside the minicolumns were 0.94 mV for the input layer, and 9.4 mV for the output layer. The strength of the synaptic connection between the input layer excitatory cells and the basket cell was set to give an EPSP of 3.5 mV. IPSPs generated by the chandelier cells had a mean of -3.2 mV, and that of the basket cell was -55.1 mV. Observe that the values are exaggerated, specially the IPSP generated by the basket cell, for compensating the small number of cells used in the network model. It is assumed that in cortex some 20 % of the cells are various inhibitory cells [31]. The PSP values and number of connections, especially inhibitory ones, were calculated to preserve this ratio between the inhibitory and the excitatory populations in V1. The PSP values were sampled from a uniform distribution with a standard deviation of 10%.

An axonal diameter of 0.3 μm [29] resulted in a spike propagation velocity of 0.85 m/s [30].

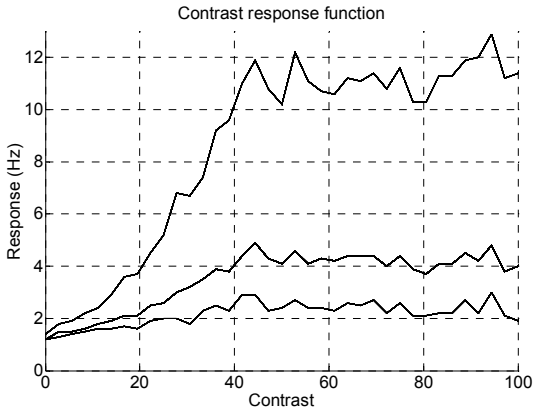

Figure 2. Contrast response function curves corresponding to mean activity of excitatory cells situated in the output layer. Top curve corresponds to cells situated in the minicolumn having the same orientation preference (90°) as the input to the hypercolumn. The thick curve in the middle corresponds to mean activity of cells in all 17 minicolumns. The bottom curve corresponds to activity of cells having a 45° orientation preference.

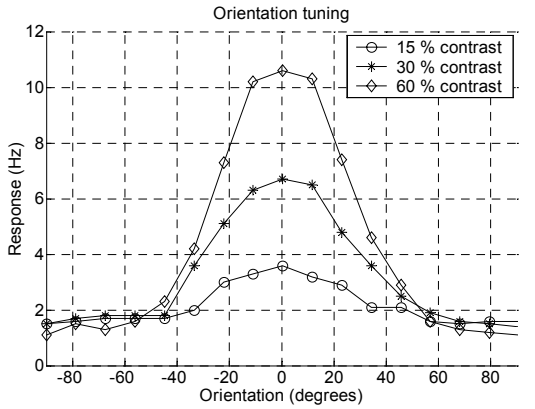

Figure 3. Contrast-invariance demonstrated by the network. Selectivity remains constant while the peak increases as a function of increasing contrast.

3. SIMULATION RESULTS

One important assumption made was the linear response of the LGN cells to the contrast stimulus increase. This assumption was in line with the normalization models mentioned above. Results by Movshon et al. [24] indicate that the majority of LGN cells (namely P cells) have linear response functions and shows very little or no sign of saturation as a function of contrast stimulus increase. Our model does not have a LGN component, hence the LGN input is modeled as a constant current. During the simulations we defined the input to the modeled cells in the following way. The simulated LGN had two components, both constant currents. The first was a function of the contrast stimulus increase, and the second was, besides of contrast, also a function of the orientation of the postsynaptic cell. Tuning of this second component was 40° half-width at half-height [19] both for the excitatory and the chandelier cells. Observe that the basket cell did not receive any input from the LGN. The orientation dependent LGN input at 100 % contrast, for cells with the preferred orientation was 2.7 nA for excitatory cells, and 0.9 nA for chandelier cells. Orientation independent part of the LGN input was 0.9 nA for all excitatory cells, and 0.32 nA for all chandelier cells. Observe that input to cells having non-preferred orientation during high contrast might exceed input to cells having preferred orientation during low contrast as a results of the orientation independent part of the LGN input. As the background activity all excitatory cells received additional current input. Excitatory cells in the output layer received 2 nA, while input layer excitatory cells received 1 nA. To guarantee that the inhibitory cells were active in their logarithmic range these cells received 3.5 nA throughout the simulation. The current values were sampled from a uniform distribution with a standard deviation of 10%.

Experimental findings related to the orientation tuning mechanism in V1, and thus normalization in the BCPNN framework [14] is possible to achieve by assuming that excitatory and inhibitory cells are active in specific regions of their gain functions, and that these regions define their ranges. The sigmoidal gain function of the Poisson neuron could be divided roughly into two regions; the low activity region (<50 %) would correspond to the exponential function, and the high activity region (≥ 50%) to the logarithmic function. We assume here that the excitatory cells are in their low activity region and the inhibitory cells are in their high activity region.

In order to analyze the network behavior we start with the input layer, and later continue with the output layer. Excitatory cells in the input layer of the hypercolumn model behaved like cells in a Hubel and Wiesel feedforward model [10]. This was not a surprise because orientation tuning of these cells was a function of the LGN input. Remember that the excitatory cells approximated the exponential function, and hence amplified their input. This resulted in the narrowing of the orientation tuning. Carandini et al. [19] reported that the half-width at half-height of the tuning of the spike responses was approx. 23° while membrane responses were approx. 38°. Their finding could motivate the narrowing of the half-width at half-height of the orientation tuning. At the same time, activity of the cells having non-preferred orientation was increased above resting activity levels as an effect of the increased

contrast. This resulted in widening of the orientation tuning curve.

However, activity shown by the excitatory cells in the output layer (Fig. 2) corresponded well to the reported results of the nonlinear behavior of simple and complex cells [13]. The first region corresponded to the dynamic response range of the cortical cells. During this phase the activity of the cells increased monotonically as a function of increased contrast. This phase was followed by a rapid saturation. During the last phase the cells were normalized i.e. their activity was constant even though the contrast was increasing. It should be stressed that saturation of activity was evident in all cells independent of their orientation preferences, and that, this level was not a function of the cells electrical properties as reported by [13].

It was shown by Albrecht et al. [13] that the contrast response function could be approximated by a hyperbolic function

$$\text{Response}(C) = R_{max} \cdot (C^n / (C^n + C_{50}^n))$$

where C_{50}^n defined contrast value that was required to produce 50% of the cell's maximum response. R_{max} was the cell's maximum response rate. It was also reported in that study that contrast response curves were shifted vertically downward as the stimulus orientation diverged from the preferred orientation. This would mean that R_{max} changed while C_{50} and n remained relatively constant. This behavior was believed to be important for preserving the relative frequency response function independent of the contrast [12,13]. The excitatory cells in the output layer of the model hypercolumn had all these properties (Fig. 2). The R_{max} levels, <12 Hz, were below maximum frequency levels (≈100 Hz) governed by the electrical properties of the cells (Fig. 2). C_{50} levels (approx. 24%) of modeled cells were biologically plausible (Fig. 2).

Contrast dependent inhibition was reported by Sclar et al. [11]. According to their results, as the contrast increased activity of the cells having orientation preference that differed significantly from the stimulus orientation decreased below their spontaneous activity levels. This behavior was also demonstrated in our simulation, as seen when comparing the low and high contrast curves (Fig. 3). Cells having orientation preference that differed more than approximately 50° from the stimulus orientation were inhibited below their spontaneous activity levels. Contrast-invariance of orientation tuning in simple and complex cells could be seen as an effect of this contrast dependent inhibition.

Both contrast response function and contrast-invariance of orientation tuning could be explained by our network architecture. In order to explain interactions in detail we would like to focus on the connections from the excitatory cells in the input layer to the basket cell, and from the basket cell to the excitatory cells in the output layer. Remember that linear increase of the contrast results in exponentially increased activity of the input layer excitatory cells, and that these cells are driving the basket cell. The basket cell linearizes the input from these cells, because the response function of the basket cell is logarithmic. As a result, the basket cell responds to contrast in a linear fashion. Output from the basket cell is then fed into the output layer excitatory cells. The main part of the input received by these excitatory cells is from the LGN input and this intracortical inhibition. Observe that, in theory, both these inputs increase linearly with contrast. This means that these two inputs have constant and positive slopes. Consequently, the relative difference between them corresponds to a constant value, and hence defines a cells activity during the normalized phase.

Within the dynamic response range (contrast < 50%), net input to excitatory cells is increasing. The reason for this is that the excitatory cells in the input layer cannot drive the basket cell. When a certain threshold (contrast ≈ 40 %) is reached the input to the basket cell is strong enough (Fig. 2) to drive it.

The cross-inhibition effect was also tested during the simulations (not shown here). In the presence of one additional line stimulus the basket cell's activity increased resulting in a stronger inhibition of the excitatory cells than in case with a single line stimulus. It is also straightforward to see that activity of the basket cell is dependent of the contrast of the additional line stimulus.

4. DISCUSSION

We have presented an abstract model of a cortical hypercolumn derived from the BCPNN architecture. This model could replicate important experimental findings relating to the orientation tuning mechanism in the primary visual cortex. Properties of the orientation selective cells in the primary visual cortex like, contrast-invariance and response saturation were demonstrated. One important assumption made was the linear response of the LGN cells to the contrast stimulus increase. As a result of this assumption, we showed that the normalization of the cells in the output layer could be explained by the local connections inside the hypercolumn.

Narrowing of the orientation tuning was possible through the reinforcement of the LGN input by the excitatory cells. As a side effect, cells having non-preferred orientation were excited above their resting activity levels, and this affected their orientation tuning negatively. The divisive inhibition of the excitatory cells in the output layer by the basket cell resulted in sharpening of the orientation tuning curves and normalization of their activity. The basket cell represented a pool of inhibitory cells with a mixture of preferred

orientations. The activity of the basket cell was a function of the excitatory cells in the input layer, and thus represented the total activity inside the hypercolumn. This network configuration is supported by studies made on cat V1.

It is well known that the long-range horizontal intracortical connections play an important role in V1. The impact of such connections to the orientation tuning mechanism of the cortical cells will be addressed in the near future. One experiment will be to simulate cortical plasticity in the framework of the BCPNN incremental learning algorithm. In these experiments, stimuli defined as lines with random orientations moving across the model cortex will provide the activity patterns required for the learning algorithm to form assemblies of connected minicolumns. Our intention is to look into how well these resemble cortical connectivity patterns seen in V1 and how they influence the response dynamics of the network.

5. REFERENCES

[1] W. Gerstner, *Pulsed Neural Networks*, The MIT Press, Chapter 1, 1998.

[2] W. Kistler, W. Gerstner, and J.L. van Hemmen, "Reduction of Hodgkin-Huxley equations to a threshold model", *Neural Comput.*, 9:1069-1100, 1997.

[3] C. Koch, *Biophysics of Computation: Information Processing in Single Neurons*, Oxford University Press, Chapter 15, 1999.

[4] M.N. Shadlen, W.T. Newsome, "The Variable Discharge of Cortical Neurons: Implications for Connectivity, Computation, and Information Coding", *J. of Neurosci.*, 18(10):3870–3896, 1998.

[5] V. Braitenberg, A. Schüz, *CORTEX: Statistics and Geometry of Neuronal Connectivity*, Chapter 36, Springer, 1998.

[6] B. Hellwig, "A quantitative analysis of the local connectivity between pyramidal neurons in layer 2/3 of the rat visual cortex", *Biol. Cybern*., 82:111-121, 2000.

[7] T.W. Troyer, A.E. Krukowski, N.J. Priebe, and K.D. Miller, "Contrast-invariant orientation tuning in cat visual cortex: thalamocortical input tuning and correlation-based intracortical connectivity", *J. of Neurosci*., 18:5908–27, 1998.

[8] M.C. Morrone, D.C. Burr, and L Maffei, "Functional implications of cross-orientation inhibition of cortical visual cells. I. Neurophysiological evidence", *Proc. R. Soc London Ser. B* 216:335–54, 1982.

[9] D. Ferster, K.D. Miller, "Neural Mechanisms of Orientation Selectivity in the Visual Cortex", *Annual Reviews of Neuroscience*, 23:441-471, 2000.

[10] D.H. Hubel, T.N. Wiesel, "Receptive fields, binocular interaction and functional architecture in the cat's visual cortex", *J. Physiol*., 160:106-154, 1962.

[11] G. Sclar, R.D. Freeman, "Orientation selectivity in the cat's striate cortex is invariant with stimulus contrast", *Exp. Brain Res*., 46:457–61, 1982.

[12] B.C. Skottun, A. Bradley, G. Sclar, I. Ohzawa, and R. Freeman, "The effects of contrast on visual orientation and spatial frequency discrimination: a comparison of single cells and behaviour", *J. of Neurophysiology*, 57:773–86, 1987.

[13] D.G. Albrecht, D.B. Hamilton, "Striate cortex of monkey and cat: contrast response function", *J. of Neurophysiology,* 48:217–37, 1982.

[14] A. Sandberg, A. Lansner, F.M. Petersson, and Ö. Ekeberg, "A Bayesian attractor network with incremental learning" *Network: Computing in Neural Systems, in press,* 2002.

[15] D. Purves, D.R. Riddle, A-S LaMantia, "Iterated patterns of brain circuitry (or how the cortex gets its spots)", *TINS,* 15 362–8, 1992.

[16] D. Hubel, T.N. Wiesel, "The functional architecture of the macaque visual cortex. *The Ferrier lecture." Proc. Royal. Soc. B* 198: 1-59, 1977.

[17] A. Peters, E. Yilmaz, "Neuronal organization of area 17 of cat cortex", *Cerebral Cortex*, 3:49-68, 1993.

[18] V. Braitenberg, C. Braitenberg, "Geometry of the orientation columns in the visual cortex", *Biol. Cyber.*, 33:179-186, 1979.

[19] M. Carandini, D. Ferster, "Membrane potential and firing rate in cat primary visual cortex", *J. of Neurosci.*, 20(1):470-484, 2000.

[20] D.G. Albrecht, W.S. Geisler, "Motion selectivity and the contrast-response function of simple cells in the visual cortex", *Visual Neuroscience*, 7:531–46, 1991.

[21] D.J. Heeger, "Normalization of cell responses in cat striate cortex", *Visual Neuroscience*, 9:181–97, 1992.

[22] M. Carandini, D.J. Heeger, "Summation and division by neurons in primate visual cortex", *Science,* 264:1333–36, 1994.

[23] M. Carandini, D.J. Heeger, and J.A. Movshon, "Linearity and normalization in simple cells of the macaque primary visual cortex", *J. of Neurosci.*, 17:8621–44, 1997.

[24] J.A. Movshon, M.J. Hawken, L. Kiorpes, A.M. Skoczenski, C. Tang, and L.P. O'Keefe, "Visual noise masking in macaque LGN neurons", *Invest. Ophthalmol. Vis. Sci.* [Suppl] 35:1662, 1994.

[25] A.B. Bonds, "The role of inhibition in the specification of orientation selectivity of cells in the cat striate cortex", *Visual Neuroscience*, 2:41–55, 1989.

[26] L.S. Krimer, P.S. Goldman-Rakic, "Prefrontal Microcircuits: Membrane properties and excitatory input of local, medium, wide arbour interneurons", *J. of Neurosci.*, 21(11):3788-3796, 2001.

[27] Z.F. Kisvárday, É. Tóth, M. Rausch, and U.T. Eysel, "Orientation-specific relationship between population of excitatory and inhibitory lateral connections in the visual cortex of the cat", *Cerebral Cortex*, 7:605-618, 1997.

[28] I. Farinas, J. DeFelipe, "Patterns of synaptic input on corticortical and cortithalamic cells in the cat visual cortex. II The axon initial segment", *J. Comp. Neurol.*, 304, 70-77, 1991.

[29] V. Braitenberg, A. Schüz, *CORTEX: Statistics and Geometry of Neuronal Connectivity*, Springer, 1998.

[30] C. Koch, Ö. Bernander, Axonal Modeling, in: M.A. Arbib (Ed.), *The Handbook of Brain Theory and Neural Networks*, 129-134, The MIT Press, 1998.

[31] C.D. Gilbert, J.A. Hirsch, and T.N. Wiesel, "Lateral interactions in cat visual cortex *Cold Spring Harbor Symp.*

On Quantitative Biology vol LV", *Cold Spring Harbor Press*, 663-76, 1990.