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LAYOUT AND FUNCTION OF THE INTRACORTICAL CONNECTIONS WITHIN THE PRIMARY VISUAL CORTEX

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Abstract

This thesis describes a top-down view of the cat primary visual cortex (area 17), which is faithful to the relevant aspects of the known physiology and anatomy. The centerpiece of this work is an abstract hypercolumn model that is mapped to cat primary visual cortex. This model shows how the recurrent (intracortical) connections can be self-organized and that their presence can explain the emergence of several response properties of the striate cells, such as contrast-invariance of orientation tuning, narrowing of the orientation tuning, response saturation followed by normalization and response facilitation mediated by long-range horizontal connections.

The abstract hypercolumn model is in line with other recurrent models by assuming broadly tuned inhibition. It is believed that large basket cells mediate this inhibition. However, the model is unique in that, when mapped to layer 4 it incorporates an additional class of interneurons, which only inhibit excitatory simple cells in their close surroundings.

The layout and function of the intracortical connections within a patch of area 17 have been addressed using developmental network models. These models are developed during exposure to visual input using a Bayesian Confidence Propagation Neural Network (BCPNN) with an incremental learning rule. The connectivity pattern demonstrated by the correlation-based network model is similar to that of area 17. Excitatory local connections are dense, whereas excitatory long-range horizontal connections are sparse and elongated along the orientation axis. Furthermore, layer 4 excitatory local connections target mainly the iso-orientation domain, whereas the excitatory long-range horizontal connections are equally distributed between all orientation domains. However, both local and long-range horizontal connections of the layer 2/3 connections are biased towards the iso-orientation domains. It is hypothesizes that this patchiness is a consequence of excitatory long-range connections made by the pyramidal cells targeting mainly other pyramidal cells located in distal iso-orientation domains. The dissimilarity between the two layers' layout might indicate functional differences.

The function of the intracortical connections has been investigated by studying response facilitation of simulated striate cells. This phenomenon is manifested by improved visibility of a Gabor patch when it is elongated along the orientation axis. This phenomenon is probably due to the elongated shape of the long-range horizontal connections. Response facilitation requires robust communication between striate cells located several millimeters from each other. It is hypothesized that spike and burst synchronization might be responsible for this. Furthermore, the anisotropy of the long-range horizontal connections seems also to help narrowing the orientation tuning of the excitatory cells.

The main conclusion to be drawn is that it is possible to explain several response properties of the striate cells by an abstract hypercolumn model, which is faithful to the known anatomy and physiology of the neocortex. When simplicity is combined with biological plausibility the models of hypercolumns can give valuable insight into the structure and function of cortical circuitry.

Keywords: Primary Visual Cortex; Hypercolumns; Cortical Microcircuits; BCPNN; Intracortical Connections; Long-Range Horizontal Connections; Recurrent Artificial Neural Networks; Response Saturation; Normalization; Contrast-invariance of Orientation Tuning; Response Facilitation; Summation Pools

To Ceren

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I was actually fascinated by brain prosthesis and was puzzled by questions like 'what would happen if a brain is replaced bit by bit?' and 'can we simulate consciousness just like any other process?'. I'm still puzzled by these questions, probably just like most people.

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Contributions

[I] Çürüklü B, Lansner A (2001) Spike and burst synchronization in a detailed cortical network model with I-F neurons. In proceedings of the International Conference on Artificial Neural Networks (ICANN), pp. 1095–1102, Vienna, Springer-Verlag.

[II] Çürüklü B, Lansner A (2002) An abstract model of a cortical hypercolumn. In Proceedings of the 9th International Conference on Neural Information Processing (ICONIP), pp. 80–85, Singapore, IEEE.

[III] Çürüklü B, Lansner A (2003) Layout and Function of the Intracortical Connections within Layer 4 of Cat Area 17. Technical Report.

[IV] Çürüklü B, Lansner A (2003) Quantitative Assessment of the Local and Long-Range Horizontal Connections within the Striate Cortex. In proceedings of the special session on 'Biologically Inspired Computer Vision', at the 2nd Computational Intelligence, Robotics and Autonomous Systems (CIRAS), Singapore, IEEE.

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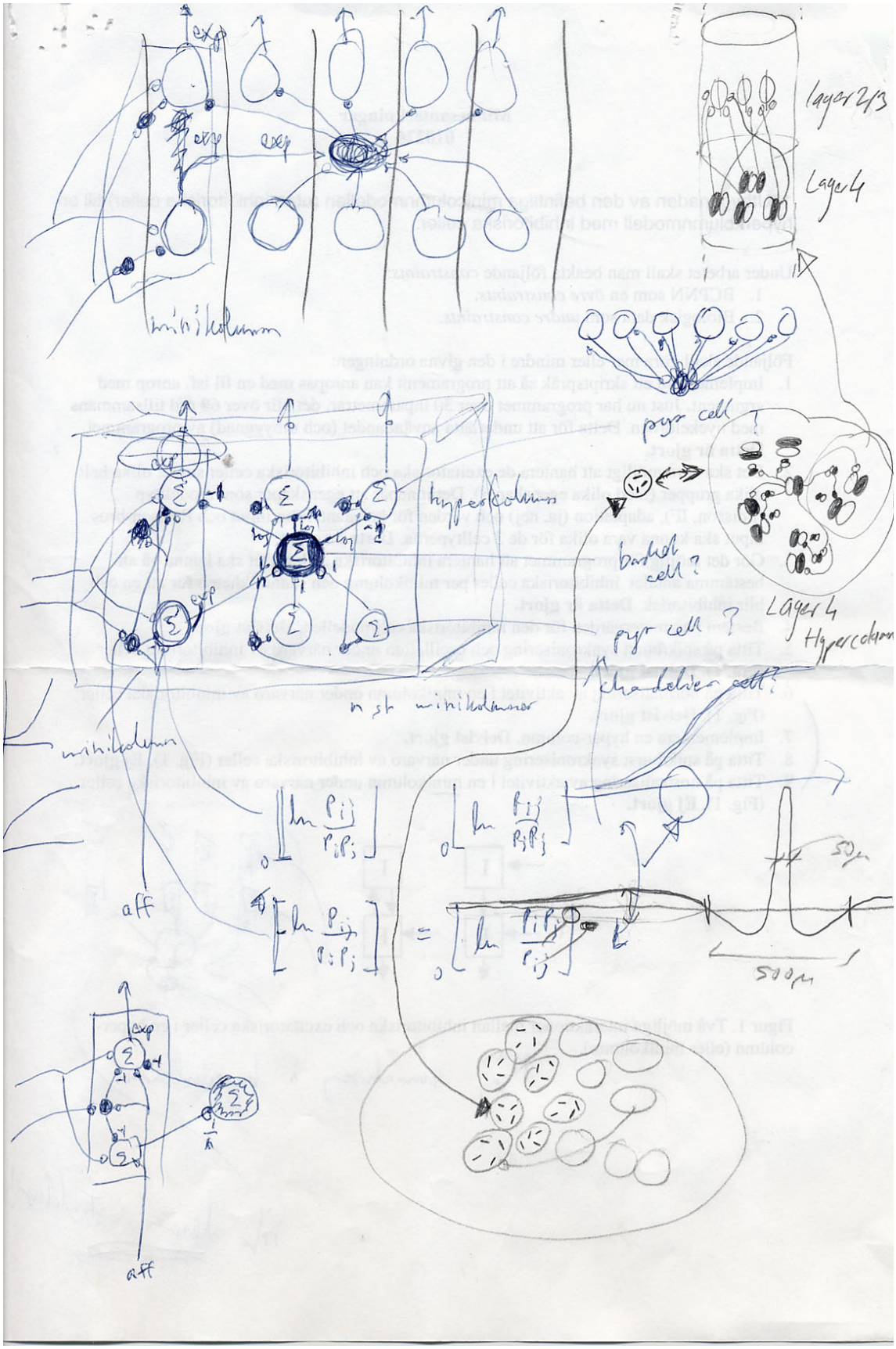
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"What chiefly distinguishes the cerebral cortex from other parts of the nervous system is the great diversity of its cell types and interconnections. It would be astonishing if such a structure did not profoundly modify the response of patterns of the fibers coming into it."
Hubel DH and Wiesel TN, (1962) Receptive fields, binocular interaction and functional architecture in the cat's visual cortex. J. Physiol., 160:106–154.

1 Introduction

1.1 Neocortical Microcircuits – Aim and Scope of the Thesis

This thesis presents a top-down view of the cat primary visual cortex (area 17) that can help to unveil the computations that are carried out within the neocortex. The primary visual cortex (striate cortex, Brodmann's area 17 of cat, area V1 of monkey) of cat and other species, such as monkey and rodents, is relatively well documented, and hence have been the principal system for studying the structure and function of cortical systems. The structural similarities between various cortical areas, which are primarily manifested by the modular and laminar organization of the neocortex, suggest that studies of the primary visual cortex might contribute to a general understanding of other cortical systems as well.

Microcircuits found in the primary visual cortex (hypercolumns) have been subject to intense study mainly as a result of the hypothesized modular organization of the neocortex. One of the aims has been to spatially relate various response properties of the striate cells (neurons populating the primary visual cortex) to each other. This leads to one of the ultimate goals of the neuroscience, which is to propose a blue print of the cortical microcircuits. Such a blue print could help to reveal the organization principles of the neocortex.

Emergence of response properties of the striate cells, such as orientation selectivity, contrast-invariance of orientation tuning, response saturation, normalization, response facilitation and cross-orientation inhibition are among the first tasks to be tackled by any model of the hypercolumns. The functional role of the thalamocortical circuitry in emergence of some of these response properties, such as orientation selectivity, is relatively well understood (Hubel and Wiesel, 1962). It is also generally accepted that the thalamocortical circuitry fails to explain other fundamental response properties, e.g. contrast-invariance of orientation tuning. However, the functional role of the cortical circuitry in the

emergence of these response properties is still unclear. This should not come as a surprise, since the layout of the intracortical connections is not fully understood. However, as it has been pointed out by Hubel and Wiesel (1962), the functional role of the cortical circuitry in emergence of orientation selectivity and other response properties of the striate cells should not be underestimated: 'what chiefly distinguishes the cerebral cortex from other parts of the nervous system is the great diversity of its cell types and interconnections. It would be astonishing if such a structure did not profoundly modify the response of patterns of the fibers coming into it'.

Based on the hypothesized modular organization of the neocortex, this thesis work intends to motivate the hypothesis saying that the response properties of the striate cells are *not solely* governed by the thalamocortical circuitry. In order to motivate this hypothesis, and hence demonstrate the functional role of the intracortical connections, in shaping the response properties of the striate cells, an abstract hypercolumn model has been presented (Papers II and III). This model reveals interactions within a small region that roughly corresponds to a cortical hypercolumn.

With this abstract hypercolumn model comes also the possibility of investigating a larger patch within area 17, since such a two-dimensional patch could be constructed by tiling the hypercolumn models next to each other. This has been done to investigate the functional role of the connections between orientation domains located several millimeters from each other (Papers I, III and IV). Especially the response saturation of striate cells and putative role of the summation pools in emergence of this behavior has been shown by simulations (Paper III).

The intracortical connections within the proposed models have also been analyzed quantitatively (Paper IV). This analysis helps to compare the presented models with area 17 (layers 4 and 2/3), and gives a plausible explanation of the differences in layout of the intracortical connections between these two layers. The hypothesis is that the pyramidal cells of layer 2/3 are mainly projecting to the other pyramidal cells in distal iso-orientation domains. This explains the patchy layout of the layer 2/3 connections.

By concluding this thesis work by an analysis of a cortical patch that contains tens of hypercolumns it is also underlined that the neocortex is a continuum. It is neither possible to reduce this complex structure to a single microcircuit nor *grasp* the microcircuits by ignoring the rest of the neocortex.

Hopefully, this thesis work will lead to a better understanding of the cortical microcircuits and clarify the challenges that lie ahead, towards more detailed models.

1.2 Thesis Structure

The thesis is structured in the following way. Chapter 2 reviews the biological background. The substance of chapter 2 is both essential and sufficient for understanding this thesis work. In sections 2.1 elementary topics related to early stages of vision is briefly reviewed. The intention is to introduce the readers, which are not familiar with neuroscience, to the topic and scope of this thesis work. The columnar organization of the neocortex is reviewed in section 2.2, whereas the following section is dedicated to the layout and function of the intracortical connections. Section 2.4 explains emergence of orientation selectivity. The putative functional role of the long-range horizontal connections in shaping the response properties of the striate cells is summarized in section 2.5.

Readers that are familiar with the topic of this thesis work will notice that some important parts, which are related to the cortical circuitry, such as projections from other (higher) cortical areas, a more detailed description of the sublayers of layer 4 and how they are related to the parvo- and magnocellular layers of the thalamus, layers 5 and 6, connections between different cortical laminea etc. are omitted. It has been considered that these parts would not contribute significantly to the understanding of this thesis work. On the contrary by their absence they help to define the scope of the work. Readers that are not familiar with the topic of this thesis work (and finds it interesting) should turn to university level textbooks in neuroscience for a more comprehensive coverage of the less accessible parts of this thesis work (Purves et al., 1997; Kandel et al., 2000; Connors et al., 2002). In fact, I believe that this thesis work is much more accessible if the basic topics related to vision are briefly covered beforehand.

Chapter 3 summarizes the BCPNN family of neural networks including the BCPNN incremental learning rule. Related work is covered in chapter 4. The first two sections in this chapter are classified according to the role of the thalamocortical circuitry in shaping the response properties of the striate cells, whereas section 4.3 reviews models that address various surround effects. The results are thoroughly described in chapter 5. Section 5.1 focuses on the findings related to the layout of the intracortical connections. Section 5.2 summarizes findings related to the emergence of response properties of the striate cells. The following section is dedicated to the response facilitation of striate cells, and the putative functional role of cortical summation pools in emergence of this behavior. Section 5.4 contains a summary of each of the four papers that are included in the thesis work. Chapter 6 sums up the thesis work by providing a top-down view of the results described in chapter 5. Chapters 7–10 contain the papers that are included in this thesis work.

2 Biological Background

2.1 Early Stages of Vision – The Retino-Geniculo-Cortical Pathway

Early stages of vision consist of three structures, i.e. the retina, the dorsal lateral geniculate nucleus (dLGN, or LGN), and the primary visual cortex (striate cortex, Brodmann's area 17 of cat, area V1 of monkey). In this section, a brief summary of the properties of the neurons populating these structures will be given. However, some of the properties, which are of special interest, will be explained more thoroughly in later sections (2.2–5). Nevertheless, this summary is limited to those properties, which are relevant to this thesis.

2.1.1 The Retina

The retina is the first processing step of the incoming information of the visual field, since the visual field is projected to the two retinae located in each eye. The retina consists of three granual layers, i.e. the photoreceptor layer, the bipolar layer (consists of bipolar, amacrine, and horizontal cells), and the retinal ganglion layer. Regions between these three layers are referred to as the plexiform layers. Synaptic connections between cells located in different granual layers occur within the plexiform layers.

The photoreceptors are divided into two types (rods and cones). These two types of cells are the only elements sensitive to light within the retina. Thus, they form the very first processing step of visual signals within the visual pathway. The specialization into rods and cones reflects different aspects of vision. The rods are extremely sensitive to light but have poor spatial resolution. These cells are specialized for sensitivity at the expense of resolution. In contrast, the cone system has very high spatial resolution. The cones are, however, less sensitive to light. The cone system is also responsible for color vision.

The bipolar cells receive exclusively input from rods or cones. Thus, the bipolar cells can be divided into rod- or cone-bipolar cells. Furthermore, these synaptic connections can be either excitatory or inhibitory. The lateral projections, which target the periphery of the bipolar cells' receptive fields, mediate the opposite to the direct path, which targets the center of the bipolar cells' receptive fields. The term 'receptive field' (RF) is often used when the response properties of the neurons are discussed. In terms of vision this term can be defined as: 'the receptive field of a neuron in the visual system is the region of the retina that influences the firing rate of the target neuron by increasing it (excitation) or decreasing it (inhibition)' (Palmer, 1999).

The connection pattern results in two types of bipolar cells, ON and OFF, with opposite response properties. The ON bipolar cells have ON-center (direct path) and OFF-surround (lateral path) receptive fields, whereas the OFF bipolar cells have OFF-center (direct path) and ON-surround (lateral path) receptive fields.

The bipolar cells make, however, only excitatory connections to the retinal ganglion cells. These connections are of two types, either ON bipolar cells to ON retinal ganglion cells or OFF bipolar cells to OFF retinal ganglion cells. As a consequence of this wiring pattern, the bipolar cells have similar response properties as the retinal ganglion cells, which are the last processing step of the visual field within the retina.

The physiological studies done by Kuffler (1953) have shown that the cat retinal ganglion cells react to small spots of light. This study indicates that each ganglion cell responds to stimulation of a small, circular patch of the retina. This patch defines the receptive field of a single retinal ganglion cell. Kuffler (1953) have discovered two different types of retinal ganglion cells (ON and OFF). When the center of an ON retinal ganglion cell is stimulated with a light spot, the cell reacts by generating a burst of spikes as a result of increased activity. When the light stimulus, which covers the center of the ON retinal ganglion cell, is moved to the periphery of the cell's receptive field, the cell is inhibited (decreased activity). The OFF retinal ganglion cells have opposite receptive field properties. These cells prefer dark spots in the center of their receptive fields, which is surrounded by a light region. The receptive fields of the bipolar and the retinal ganglion cells can be mathematically described with a so-called 'Mexican-hat' function.

Note however that the activity of the retinal ganglion cells depends on the contrast, i.e. the difference in the amount of light that falls on their receptive field centers and surrounds. Both types of retinal ganglion cells are equally numbered, and are distributed equally in the retina.

The retinal ganglion cells are not only divided into ON or OFF cells. There is also a morphological (based on form and structure) classification of these cells. According to this classification best known retinal ganglion cells are either α (5%) or β (55%) cells (Wässle et al., 1975). The remaining 40% are γ retinal ganglion cells (Boycott and Wässle, 1974). The γ retinal ganglion cells are further

subdivided into groups. Not much is know about their functional impacts on vision.

The α and β retinal ganglion cells show functional differences as well, and are physiologically classified as Y and X cells, respectively. The γ retinal ganglion cells are classified as W cells. The α retinal ganglion cells act as novelty detectors, they are fast and sensitive to motion, and they can operate during low contrast. On the contrary, the β retinal ganglion cells are specialized for a more detailed analysis of a stationary scene.

Distribution of the different types of photoreceptors (rods and cones) and retinal ganglion cells (α or β) reveals different aspects of vision. The cones are the only photoreceptors located in the fovea, whereas the rods dominate the periphery. (The fovea is a region located in the center of the retina, about 2º of visual angle in diameter. This region is responsible for highest spatial accuracy.) The convergence between photoreceptors and the retinal ganglion cells is very small within the fovea. Not surprisingly the β retinal ganglion cells dominate the fovea. The α retinal ganglion cells dominate the periphery of the retina. With increasing retinal eccentricity the density of photoreceptors and retinal ganglion cells decrease, and the sizes of receptive fields of all cells increase. Furthermore, the fraction of rods and α retinal ganglion cells, as well as convergence rate increase.

2.1.2 The LGN

The major target of the retinal ganglion cells is the LGN of the thalamus (Hubel and Wiesel, 1977). Neurons in the thalamus send their axons to the cerebral cortex. The retinotopic representation of the visual field, which emerges in the retina, is preserved throughout the retino-geniculo-cortical pathway. Hubel and Wiesel (1977) have shown that the fibers that start from the neighboring retinal ganglion cells within the retina converge to neighboring geniculate cells. The geniculate cells project in turn to neighboring regions within the primary visual cortex (Hubel and Wiesel 1977).

The LGN consists of six layers, four parvocellular (P-cell) and two magnocellular (M-cell). The ipsalateral eye drives two parvocellular and one of the magnocellular layers (one half of the layers). The contralateral eye drives remaining LGN layers. Axons from the α retinal ganglion cells converge to the M-layers, whereas the β retinal ganglion cells converge to the P-layers. Note however that the retinotopic maps within the LGN and the primary visual cortex are distorted, since the visual field located in the fovea needs higher resolution and more computing power than the periphery of the visual field.

The receptive field properties of the geniculate P and M cells are similar to those of β and α retinal ganglion cells, respectively. The LGN cells are also classified as ON and OFF cells. Note that the antagonistic center surround receptive field profile of the bipolar cells is preserved in the LGN. As a result the

LGN is often seen as a relay between the retina and the primary visual cortex. However, the LGN also receives input from two other sources than the retina. These two modulatory sources are, the primary visual cortex and the inhibitory cells located in the thalamic reticular nucleus. These inputs can affect the gain of the signal transmission (Coenen and Vendrik, 1972) and the mode of the firing of the geniculate cells (burst or continuous firing) (Steriade et al., 1993).

2.1.3 The Primary Visual Cortex

The cerebral cortex is a thin folded structure that covers the cerebral hemispheres (Purves et al., 1997). The neocortex is a six-layered structure, which takes up most of the cerebral cortex. The laminar division of the neocortex is based on the properties of the neurons (nerve cells) of different kinds populating the neocortex. These properties are size, shape and density. Some of the neocortical layers (laminea) are divided further into sublayers. (See the excellent book by Braitenberg and Schüz (1998) for a comprehensive review on the cortex) The neocortex of cat is ~1.3 mm thick (~0.08 mm in mouse, ~1.6 mm in monkey, ~3 mm in human). Approximately 80% of the neurons are pyramidal or spiny stellate cells. These cells are excitatory (non-GABAergic). Remaining cells are classified as GABAergic inhibitory cells. These cells are referred to as interneurons (they are also mentioned as sparsely spinous cells, local circuit neurons, stellate cells) (Gabbott and Somogyi, 1986). Each neuron makes and receives thousands of synaptic connections to neurons located nearby and neurons in other parts of the brain.

The rest of the cerebral cortex is made up of phylogenetically older structures (Purves et al., 1997). These structures are paleocortex, which has four layers, and archicortex (hippocampus), which has three layers.

The neocortex is composed of different areas, which are dedicated to different tasks such as vision, hearing, and reasoning etc. Based on their properties, these areas are distinguishable from each other. The primary visual cortex, which is a part of the visual cortex, is one such area. It has (i) distinct architecture, (ii) a unique connectional signature with other brain regions, (iii) characteristic functional maps, (iv) a unique inventory of receptive field properties, (v) a distinct catalog of contributions to visual processing and visually guided behaviors (Payne and Peters. 2002).

As mentioned above the primary cortex is composed of six layers. The most superficial layer is referred to as layer 1. This very thin layer is also known as the plexiform layer, since it contains few neurons. Neurons that populate this layer are mainly inhibitory (Gabbott and Somogyi, 1986).

The border between layer 1 and layer 2 is easy to define. Upper layer 2 is populated with pyramidal cells. All the way through layer 2 and layer 3 the pyramidal cells increase in size. There is no clear border between layer 2 and layer 3. Most often these two layers are referred to as layer 2/3. However, layer 2

pyramidal cell axons are short, whereas layer 3 pyramidal cell axons go through the deeper layers into the white matter and continuous to other cortical areas (O'Leary, 1941). It has been shown that besides the projections to other cortical areas, the axons of the pyramidal cells can extend horizontally for several millimeters (Rockland and Lund, 1982; 1983; Gilbert and Wiesel, 1983; 1989; Rockland, 1985; Amir, 1993; Malach et al., 1993; Fitzpatrick, 1996; Durack and Katz, 1996; Bosking et al., 1997; Kisvárday et al., 1997; Schmidt et al., 1997; Yousef et al., 1999; Chisum et al., 2003). These axons seem to form clusters of terminals, which mainly target dendritic spines (Kisvárday et al., 1986). The functional implication of these horizontal projections seems to be to spread the activity to other pyramidal cells, which have similar physiological properties (section 2.5).

Layer 4 is the thickest of the layers, and takes up one third of the cortex. It is mainly populated with small and closely packed spiny stellate cells, and some pyramidal cells. The spiny stellate cells are similar to the pyramidal cells. However, they lack the typical apical dendrite of the pyramidal cells, and hence make symmetrical synapses around the cell body. Layer 4 is further divided into two sublayers (layers 4A and 4B). Layer 4A borders to layer 3, whereas layer 4B borders to layer 5. Spiny stellate cells found in layer 4A are bigger and more evenly placed than those in layer 4B. Layer 4A spiny stellate cells project to layer 3 and deeper layers. Layer 4B spiny stellate cells on the other hand mainly project to layer 5 and layer 6.

One more fundamental difference between the two sublayers is the type of the thalamic afferents. Layer 4A is dominated by the Y-visual signals from the LGN, whereas layer 4B receive exclusively X-visual signals. Ahmet et al. (1994) propose a quantitative assessment of the excitatory inputs to a spiny stellate cell. They suggest that 45% comes from layer 6, 28% from other spiny stellate cells, and 6% from the thalamic afferents. The remaining 21% could not be classified. They suggest also that up to 84% of the inhibitory connections might come from the small basket cells (see below).

Layer 5 is divided into two sublayers. Layer 5A contains irregular discontinuous clusters of small and medium size pyramidal cells (O'Leary, 1941). These cells send their axons to the layer 2/3. The upper part of the layer 5B contains large pyramidal cells. The apical dendrites of these cells go through all the way to the layer 1. On their way the apical dendrites of these cells branch in layer 4 and later in layer 2/3. However, the small pyramidal cells dominate layer 5B. Their apical dendrites terminate in layer 1 without branching. Some of their axons terminate in layers 5 and 6, whereas some enter the white matter.

Layer 6A contains small and medium size pyramidal cells. These cells are arranged into vertical columns. According to Katz (1987) these cells can be divided into two groups. The claustrum-neurons, which have apical dendrites that reach layer 1 and have fine axonal collateral branches that arborize only in layer 6 and lower layer 5, and corticothalamic neurons, which have apical dendrites that

never reach higher than layer 3. Axons of these neurons send widespread collaterals into layer 4. Layer 6B borders to the white matter. Not much is known about the neurons that populate this sublayer.

In summary, the signals from the LGN that enter layer 4A are distributed within layer 4A, later to layer 2/3 and to the deeper layers. From layer 2/3 the signals are sent to layers 5B and 6. Signals entering layer 4B are projected to deeper layers, and back to layer 4B or to layer 2/3.

Only 20% of the cortical neurons are classified as interneurons (Gabbott and Somogyi, 1986). The distribution of the interneurons is not uniform across the cortical layers. Lowest density of interneurons is found in layer 5, followed by layer 2, upper parts of layer 3 and layer 6. Highest density of interneurons is found in layer 4 (note that this layer is the main recipient of the thalamic input) and lower parts of layer 3 (~50% both layers). Until now no morphological scheme has been generally accepted to classify these cells. Below the interneurons that are of especial interest are reviewed. These are chandelier cells, small and large basket cells.

Chandelier cells are multipolar cells (Tömböl, 1978). These cells are more common in layer 2/3, and to some extent in layer 5. These cells are characterized by their axons, which gives rise to short, vertically oriented string of boutons. These cells are also referred to as axoaxonic cells, since they synapse with the axons of the postsynaptic cells. Based on the strategic placement of the axon terminals, it is assumed that these cells are powerful inhibitors of the postsynaptic cells.

Large basket cells are common in all layers except layer 1 (O'Leary, 1941; Tömböl, 1978). These cells have vertically oriented dendrites. Their axons form up to five branches. These branches extend up to 1.5 mm, parallel to the pial surface (horizontal projections). Main postsynaptic targets of these cells are both excitatory cells and other basket cells within the layer where they are located. Inhibition of other basket cells can be used for indirect facilitation of excitatory cells. Kisvárday et al. (1987) suggest that the basket cells do also target cells found in other layers as well.

Small basket (multipolar) cells are found in layer 3 (Meyer, 1983) and layer 4 (Kisvárday et al., 1985). Results by Kisvárday et al. (1985) suggest that these cells inhibit excitatory cells found in layer 4. The targets of their axons are the dendrites of the postsynaptic cells.

In this section some of the properties of the primary visual cortex, which are fundamental and highly relevant to this thesis, are leaved out. In the later sections these properties will be review in detail. In section 2.2 the modular structure of the neocortex is explained. This hypothesis is founded on the vertical organization of the apical dendrites of the larger pyramidal cells within layer 5. Section 2.3 focuses on the lateral interactions within layer 4 and the superficial layers (layer 2/3). Sections 2.4–5 focus on the response properties of the cells populating the primary visual cortex.

2.2 Modular Structure of the Neocortex

2.2.1 Neocortical Mini- and Macrocolumns

The vertical organization of the neocortex is as evident as the laminar organization. The neocortex is composed of repetitive structures referred to as the minicolumns (Mouthcastle, 1957; 1978; 1997; Powell and Mouthcastle, 1959; Hubel and Wiesel, 1962; 1977; Buxhoeveden and Casanova, 2002). The minicolumns are positioned orthogonally to the cortical surface, and goes through all cortical layers, i.e. from the pial surface of layer 1 to layer 6, which borders to the white matter. A minicolumn contains roughly some 100 neurons. Minicolumns found within the primary visual cortex contain 2–3 times more neurons.

Mountcastle (1957) have reported the very first evidence of the columnar organization of the neocortex (cat's somatosensory cortex). It was also the first time the term 'column' was used. Later, Mountcastle and Powell (1959) have revealed that also the monkey's somatosensory cortex is based on the same organization principals. Vertical penetration through the cortical layers of cat somatic sensory cortex revealed that cells in each layer have similar properties of place and modality. However, penetrations oblique or parallel to the cortical surface showed that the properties change continuously.

Hubel and Wiesel (1962; 1977) have revealed the columnar organization of the primary visual cortex. They have discovered that neurons are reacting selectively to 'line orientations' (Hubel and Wiesel 1962). They found that the orientation preference of the neurons along the vertical axis, i.e. perpendicular to the cortical surface, is almost constant. Only exception is the layer 4 neurons of monkey, which are only 'mildly' orientation specific (Hubel and Wiesel, 1977). However, the parallel and the oblique penetrations revealed the systematic change of orientation preference of the neurons with distance. Their study reveals that these changes are either clockwise or counterclockwise with steps of $\leq 10^{o}$, roughly every 50 μm. During several penetrations, one full sequence of orientations, corresponding to 180^{o}, or more is completed. Hubel and Wiesel have named the region that corresponds to a full sequence the 'orientation hypercolumn'. The length of a full sequence is approximately 500–850 μm on cortical surface.

Columnar organization of other heterotypical areas, such as the auditory cortex (Woolsey and Waltz, 1942), and the motor cortex (Meyer, 1987) is also prominent. The homotypical cortical areas, i.e. the association cortex, do also show same organization principles. This finding is highly interesting since, the modalities of the neurons populating these areas are created within the neocortex through intrinsic connections, and are not directly linked to sensory input.

More recent evidence of minicolumn structures is presented by Peters and colleagues (Peters and Yilmaz, 1993; Peters and Sethares, 1996) based on the arrangement of the pyramidal cells of cat and monkey primary visual cortex. Their

analysis is based on the layout of the apical dendrites of larger layer 5 pyramidal cells of cat area 17. Peters and Yilmaz (1993) have shown that the apical dendrites of these cells form clusters, and are joined by the apical dendrites of layer 2/3 pyramidal cells, while they continue to pial surface. The result is an axis of dendrites going from layer 5 to layer 1. Layer 6A pyramidal cells do not participate in this arrangement. A similar pattern is also found within the monkey area V1 (Peters and Sethares, 1996). In monkey the center-to-center cluster distance is 23 μm, whereas in cat the distance between the clusters is 56 μm (these values are roughly constant). It seems that the minicolumns within the monkey's neocortex are smaller and more densely packed than those found in cat's neocortex.

It is also shown that cells within a small patch receive input mainly from one eye. Horizontal penetrations show that there is a transition between regions of different eye dominations (Hubel and Wiesel, 1977). However, penetrations perpendicular to the cortical surface shows that the same eye dominates all neurons within an orientation minicolumn. All layer 4 cells are, however, driven exclusively from one eye, and hence are classified as monocular cells, whereas cells in other layers are classified as binocular. These findings gave birth to the 'ocular dominance columns'. The ocular dominance columns run parallel to the cortical surface and alternate with each other (right eye and left eye). Later, Hubel and Wiesel have integrated orientation and ocular dominance columns into one general model. The result is the highly idealized 'ice cube' model (Hubel and Wiesel, 1972; Hubel, 1988). Inside the ice cube model, the two columnar systems (orientation and ocular dominance) are positioned orthogonally to each other. Ice cube modules are hypothesized to analyze small spots of the visual field. According to this scheme, adjacent ice cube modules analyze adjacent small spots of the visual field, with some overlap between them. Note however that the hypercolumns are not physical entities with well-defined input and outputs such as components in electrical circuits.

During the late seventies a new type of circular-shaped centric models have emerged (Braitenberg, 1979). Within this circular-shaped model, orientation minicolumns are organized around the middle points, corresponding to the orientation 'singularities'. The orientation singularities are regions where all orientations meet (Swindale, 1982). Around this middle point of the model, the orientation changes continuously. Braitenberg (1979) have proposed that during one lap around the middle point same orientation should be encountered twice. Thus, on the 'opposite side' of an orientation singularity cells having the same orientation preference are located. Blasdel and Salama (1986) have named this model the 'pinwheel model'. Later, the pinwheel model has received renewed attention after the discovery of the cytochrome oxidase blobs in cat (Wong-Riley, 1979) and in monkey primary visual cortex (Hendrickson et al., 1981; Horton and Hubel, 1981). Cells within the blobs are poorly tuned for orientation. Thus, it is tempting to speculate that blobs are located at the orientation singularities of the

pinwheel model. After the discovery of blobs several circular-shaped models have been proposed (Horton, 1984; Gotz, 1987; 1988; Baxter and Dow, 1989) (see the reviews by Erwin et al. (1995) and Dow (2002)). However, most of them have been rules out after the voltage-sensitive dye examinations of the monkey primary visual cortex.

Despite their ripe age, the ice cube and the pinwheel models are still very popular among the modelers. In the literature, the ice cube model, the pinwheel model, and others are referred to as cortical microcircuits, cortical columns, macrocolumns, cortical modules, hypercolumns (this term is reserved for microcircuits found in the visual cortex (Hubel and Wiesel, 1977)) and segregates (this term is reserved for microcircuits found in the somatosensory cortex (Favorov and Diamond, 1990)) etc.

2.2.2 The Orientation Map

A more careful analysis of the distribution of the orientation minicolumns and how they are related, in terms of geometry, to the ocular dominance columns and the singularities become possible first after the voltage-sensitive dye examinations of the monkey primary visual cortex (Blasdel and Salama, 1986; Blasdel, 1992; Obermaiyer and Blasdel, 1993). Through these studies, several features of the orientation map are revealed. The singularities, described by several independent groups, are apparently located in the center of the ocular dominance columns. During clockwise movement around a singularity point, the orientation rotates either clockwise (positive singularity) or counterclockwise (negative singularity). Furthermore, one lap around the singularity point corresponds to 180º, and not to 360º, as proposed by the original pinwheel model of Braintenberg (1979). Thus, cross-orientation domains are positioned on the opposite sides of a singularity point.

Besides the orientation singularities the orientation map has other features, such as ‘linear zones’, ‘saddle points’, and ‘fractures’. Linear zones are frequently located on the borders between the ocular dominance columns. Within these zones the iso-orientation lines are parallel to each other. Furthermore, these lines are roughly orthogonal to the ocular dominance columns. Saddle points are found within the ocular dominance columns. The saddle points are relatively large zones where the orientation selectivity remains almost constant. Most often these areas are found between two adjacent singularity points that are within the same ocular dominance column. Fractures are small local discontinuous patches where orientation selectivity changes irregularly (Hubel and Wiesel, 1977).

2.3 Layout of the Intracortical Connections within the Primary Visual Cortex

The two-dimensional orientation map is often used as a starting-point when the organization of the neocortex is analyzed. The orientation map reveals the orientation preference of the striate cells located in different regions on the cortical surface. It can also be used to link the visual field, covered by both eyes, to cortical dimensions. Furthermore, the orientation map is a perfect tool for the visualization of the layout of the intracortical connections. This is done by mapping the connections, within a specific cortical layer, on an orientation map, which holds information on the orientation preference of the regions exposed to projections from the injection site.

In this section, a brief review of the layout of the local and long-range horizontal connections within layer 17 of cat will be given. This review is based on the qualitative assessments. The focal point is the intracortical connections within layer 4 and the superficial layers (layer 2/3). Remember that layer 4 is the main recipient of the thalamic input. Furthermore, in cat area 17 orientation selectivity emerges in layer 4 (section 2.4).

The superficial layers are interesting for the analysis of the computations within the primary visual cortex. Furthermore, long-range horizontal connections within the superficial layers extend more than in any other layer of the neocortex. Neurons within layer 2/3 receive input from layer 4 neurons.

In the literature the local connections are defined as connections within a region, which roughly corresponds to a hypercolumn (‘Diameter’ of a hypercolumn is in the range of 0.5–1.2 mm depending on the area and species. Cat hypercolumns are larger than monkey, and area 17 hypercolumns are larger than area 18). As a consequence, an imaginary border isolates a hypercolumn from the rest of the neocortex. Connections crossing this imaginary border are defined as long-range horizontal connections. (These connections are also referred to as long-range intracortical (intrinsic, lateral) connections, distal projections etc.). Thus, it is hypothesized that those connections link cells, which are located in different hypercolumns, even though the distances covered by the axons are relatively small.

One important consequence of this division is to be able to distinguish the functional role of the cells located in the near surrounding of a cell, and cells located elsewhere within the neocortex, in altering the response properties. It is obvious that the term ‘local’ is highly subjective and depends on the intention of the author. In some cases local is defined as a region that is composed of up to four or five hypercolumns.

2.3.1 Patchy Layout of the Long-Range Horizontal Connections

During the early eighties, Rockland and Lund (1982) discovered the patchy layout of the long-range horizontal connections of the tree shrew visual cortex. These patches were located periodically several millimeters from the injection sites. Since their discovery, the long-range horizontal connections have received increased attention. It is now widely accepted that the long-range horizontal connections of the superficial layers are a prominent feature of the visual cortex. The long-range horizontal connections are also evident in other neocortical areas, such as auditory, motor, somatosensory, and prefrontal cortex (Schmidt and Löwel, 2002). Now it is widely believed that the pyramidal cells are responsible for these projections to remote regions.

The patchy, iso-orientation biased, layout of the long-range horizontal connections have been shown on a variety of species including cat (Gilbert and Wiesel, 1983; 1989; Kisvárday et al., 1997; Schmidt et al., 1997; Yousef et al., 1999), tree shrew (Rockland and Lund, 1982; Fitzpatrick, 1996; Bosking et al., 1997; Chisum et al., 2003), ferret (Rockland, 1985; Durack and Katz, 1996), and monkey (Rockland and Lund, 1983; Amir, 1993; Malach et al., 1993). These connections can extend up to 8 mm on cortex surface. However, most connections are within an area of ~2.5 mm from an injection site (Kisvárday et al., 1997).

2.3.2 Quantitative Assessment of the Intracortical Connections of the Layer 2/3

A qualitative assessment of the intracortical connections has been possible to do only recently. Excitatory pyramidal cells and inhibitory cells of different kinds such as the chandelier cells and the basket cells populate the superficial layers. Kisvárday et al. (1997) report that in layer 2/3, 56.2% of the local excitatory connections target the iso-orientation (±30º) domain. Oblique- (±30–60º) and cross-orientation (±60–90º) domains receive 28.4 and 15.3 per cent of the connections respectively. Long-range connections show a similar pattern. The distribution of excitatory connections shows that 40.0% are located at the distal iso-orientation domains, 36.9% at the distal oblique-, and 23.1% at the distal cross-orientation domains. Note that roughly only one half of the connections target the iso-orientation domains. It is clear that the projections targeting the iso-orientation domains do not dominate.

The inhibitory long-range horizontal connections extend only one third to one half of the excitatory network. Furthermore, the excitatory connections out number them. It is also assumed that large basket cells make these inhibitory long-range horizontal connections. Thus, the majority of the long-range connections are considered to be excitatory in both layer 4 and layer 2/3 (Kisvárday et al., 1997). Inhibitory local connections target mainly the iso-orientation domain, similar to

the excitatory local connections. Roughly 48.0% of them are found within the iso-orientation domain. 29.2% are located at the oblique-orientation domain, and 22.8% are located at the cross-orientation domain. Long-range projections are even less orientation specific, with only 47.7% targeting the distal iso-orientation domains. 21.7% are located at the oblique-, and 30.6% at the cross-orientation domains.

The pattern shown by the excitatory and inhibitory connections is that the inhibitory projections are less specific for orientation. Both networks are biased towards the iso-orientation domain, however, as pointed out by Kisvárday and colleagues (Kisvárday et al., 1997) this bias is moderate.

2.3.3 Quantitative Assessment of the Intracortical Connections of Layer 4

Surprisingly, the layout of the long-range horizontal connections found within layer 4 has drawn much less attention, when compared to those found within the layer 2/3. Layer 4 is the main recipient of the thalamic input, and in cat layer 4 cells are well tuned for orientation (Hubel and Wiesel, 1962; Reid and Alonso, 1995). Thus, the interplay between the orientation map and the layout of long-range connections is essential for understanding the origins of orientation selectivity.

The findings suggest that the layout of the layer 4 long-range horizontal connections is different from those found within the superficial layers (Chisum et al., 2003; Kisvárday et al., 1997; Yousef et al., 1999; Schmidt and Löwel, 2002). Their extent is only 50% of the connections of the superficial layers (Kisvárday et al., 1997). Furthermore, Yousef et al. (1999) found that layer 4 long-range connections (of area 18) are hardly biased towards the iso-orientation domains. The patches, which characterize the superficial layers, are absent. Furthermore, only 35.4% of the connections (>740 μm) target the distal iso-orientation domains. Oblique- and cross-orientation domains receive 33.7% and 30.9% of the connections respectively. The pattern shown by the local connections (<740 μm) is different. The local iso-orientation domain receives 60.0% of the connections, whereas 22.0% of the connections are targeting the oblique- and 18.0% the cross-orientation domains. Note that 40% of the local connections target other than the iso-orientation domain.

It seems that independent of the layer, the excitatory long-range horizontal connections are neither random nor restricted to the iso-orientation domains. Cross talk between different orientation domains is a prominent feature of both the layer 4 and the superficial layers.

2.4 Emergence of Orientation Selectivity within the Primary Visual Cortex

In this section, the functional role of the thalamocortical circuitry in the response properties of the striate cells is discussed. Furthermore, shortcomings of the thalamocortical circuitry in explanation of several response properties of the striate cells will be revealed. Thus, this section will lay the foundation for the hypothesis that the intracortical connections are essential parts in explaining several fundamental response properties of the striate cells.

As reported by Hubel and Wiesel (1962; 1977) the cells populating the primary visual cortex react selectively to line orientations (these cells are named 'simple', 'complex' and 'hypercomplex' depending on their response properties. See section 2.4.1). However, the relay cells within the LGN, which carries the visual information from the retina to the primary visual cortex, are not orientation selective. It is not known in detail how orientation selectivity of the cells within the primary visual cortex emerges (Das, 1996; Sompolinsky and Shapley, 1997; Ferster and Miller 2000). Hubel and Wiesel (1962) have proposed that orientation selectivity of the cat simple cells is a consequence of the arrangement of the thalamic afferents. According to this arrangement, the ON-center LGN cells converge to ON-subregions of the simple cells. The OFF-subregions of the simple cells are constructed in the same way by the OFF-center LGN cells. This is the first model of the thalamocortical circuitry. It is named the 'feedforward' model, since the intracortical connections do not play any prominent functional role in generating the orientation selectivity properties of the striate cells. The information flows in a pure feedforward fashion, along the retino-geniculo-cortical pathway. Note that the recurrent intracortical connections are not included in the model.

The feedforward model relates the receptive fields of the striate cells to the visual field. Later, Reid and Alonso (1995) have suggested that the feedforward model of Hubel and Wiesel can indeed explain the structure of the thalamocortical circuitry of cat. They have recorded simultaneously the simple cells and the LGN cells, which have overlapping receptive fields. They have detected that some simple and LGN cells have highly correlated activity if they have overlapping 'receptive fields'. Based on this finding they have discovered that ON-center LGN cells are connected monosynaptically to the ON-subregion of the simple cells. However, the results by Reid and Alonso (1995) cannot explain the high aspect ratios of the subregions required by the feedforward model as revealed by the study of Pei et al. (1994).

Note further that layer 4 cells found in other species than cat, i.e. monkey (Blasdel and Fitzpatrick, 1984; Hawken and Parker, 1984) tree shrew (Humprey and Norton, 1980), and ferret (Chapman and Stryker, 1993), are poorly tuned for

orientation. Thus, the feedforward model cannot alone explain emergence of orientation selectivity in primary visual cortex of these species.

2.4.1 Simple, Complex and Hypercomplex Cells

As mentioned above, Hubel and Wiesel (1962; 1977) have discovered and named three types of striate cells, which have fundamentally different response properties. The simple cells, which mainly populate layer 4 of cat, have elongated ON- and OFF-subregions that reflect the thalamic inputs. Hubel and Wiesel (1962; 1977) have discovered that these cells' responses to complex stimuli can be predicted from their responses to individual spots of lights. As a consequence, a simple cell's receptive field can be mapped based on its response to small light spots positioned on different locations on the retina. A light, which is positioned at the cells' ON-subregion, excites the cell, whereas a light spot positioned at the cells OFF-subregion inhibits the cell. The responses to 'dark' spots are opposite to light spots.

Complex cells are the single most common cell type in the primary visual cortex. Approximately 75% of the striate cells are classified as complex cells. It is assumed that projections originating from layer 4 simple cells form the receptive fields of these cells (Alonso and Martinez, 1998). Complex cells do not have the linear receptive fields of the simple cells. The nonlinear receptive field properties of the complex cells are much harder to predict. They do not respond to light spots at all. They respond to lines (bar or grating) located within their receptive fields. The location of the line within the receptive field seems to have less importance compared to the simple cells. Complex cells are highly sensitive to motion though. They have also larger receptive fields than the simple cells.

Hypercomplex cells (end-stop cells) are named after their peculiar responses to lines that are positioned in their receptive fields. Surprisingly, lines that normally excite the hypercomplex cells inhibit them if they extend beyond their receptive fields. Now it is widely believed that the hypercomplex cells are actually end-stopped simple or complex cells.

2.4.2 Spatial Frequency Selectivity

Moving and static sinusoidal gratings with different spatial and temporal configurations are often used to reveal response properties of the striate cells. The 'spatial frequency' of a sinusoidal grating is defined as the width of the light and dark bars, which a sinusoidal grating is composed of. If a sinusoidal grating has low frequency, then its light and dark bars are thick. High frequency implies that the sinusoidal grating consists of thin bars.

It has been shown that the striate cells respond selectively to spatial frequencies (Campbell et al., 1969; Maffei et al., 1973). Their responses to sinusoidal gratings, which have lower or higher spatial frequencies than the preferred ones are lower

(they behave like band-pass filters). Note that the striate cells respond similarly to orientations.

Tootell et al. (1981) suggest that spatial frequency is also organized in a columnar fashion, such as orientation and ocular dominance. It is not clear, however, how spatial frequency columns are related to orientation and ocular dominance columns. Shoham et al. (1997) have shown that striate cells preferring lower frequencies are positioned at cytochrome oxidase blobs, which coincide with the singular points (section 2.2). It is also shown that these regions are surrounded by striate cells preferring higher spatial frequencies (Shoham et al., 1997). Remember that the singular points are positioned in the middle of ocular dominance columns (section 2.2). One year earlier, the same group has also revealed that LGN Y-cells are projecting predominantly to the blobs, whereas interblob areas are receiving input mainly from LGN X-cells (Hübener et al., 1996). LGN X-cells are tuned to higher spatial frequencies than LGN Y-cells (Derrington et al., 1979).

Issa and colleagues (2000) suggest that striate cells preferring the extremes of the spatial frequency continuum (low and high) are located at the singular points. Issa et al. (2000) point out that Shoham and colleagues (1997) classify spatial frequencies as ‘low’ or ‘high’, whereas their approach is based on three classes of spatial frequencies (low, high and intermediate). Issa et al. (2000) remark also that they test a larger (and especially higher) range of spatial frequencies than Shoham et al. (1997). Everson et al. (1998) on the other hand proposes that the spatial frequencies are organized in a pinwheel fashion around singular points, in the same manner as orientation selectivity.

Somewhat related to the spatial frequency selectivity are the terms ‘absolute spatial phase’ and ‘relative spatial phase’. These two terms are practical when a receptive field is described in terms of its subfields. Relative spatial phase refers to the position of the ON- and OFF-subregions with respect to the center of a receptive field. Absolute spatial phase refers to the position of the ON- and OFF-subregions with respect to the visual field.

2.4.3 Degree of Invariance within the Orientation Map

The orientation minicolumns are not homogenous structures. There are variations in orientation preferences of the neurons populating the orientation minicolumns. As reported by Murphy and Sillito (1986) orientation preference inside an orientation minicolumn can vary 9–18º between the cells. Furthermore, each cell responds selectively to a broad range of orientation.

A more recent study by DeAngelis et al. (1999) reveal the degree of invariance of response variables, such as orientation, spatial frequency and absolute spatial phase. Highest degree of invariance is shown by the orientation, closely followed by spatial frequency. One response variable did not show any evidence of

clustering, namely, the relative spatial phase. Given the fact that orientation is the most clustered response variable (and the results by Murphy and Sillito (1989)) it is evident that orientation minicolumns of the neocortex are highly heterogeneous.

2.4.4 Strength of the Thalamocortical Synapses

The feedforward model describes in an excellent way individual connections within the thalamocortical circuitry. However, it does not reveal the strength of the geniculate input. Results by Reid and Alonso (1995) suggest that the input is strong and monosynaptic. Their result is based on cross-correlation analysis on the spikes generated by the cortical and thalamic cells. Ahmet et al. (1994) reveal that the thalamocortical synapses are larger than intracortical synapses, and hence contain more release sites. A study by Gil and colleagues (Gil et al., 1999) indicate that the thalamocortical synapses within the somatosensory system of rat is five times stronger than intracortical synapses.

Results from the intracellular recordings by Ferster and colleagues (Ferster et al., 1996; Chung and Ferster, 1998) indicate that the thalamocortical synapses are strong. Ferster et al. (1996) show that even when the cortex is cooled (~9ºC) the shape of the orientation tuning curve is intact. However, the amplitudes of the EPSPs are reduced dramatically. Furthermore, the latency of an EPSP increases to 5 ms. Chung and Ferster (1998) have shocked the cortex electrically in order to silence the activity of the cells in the layer 4 and the superficial layers of the primary visual cortex. After the inactivation the amplitudes of the EPSPs are down to 46%. These experiments indicate that roughly one third to one half of the spikes generated within the layer 4 can be tracked back to the thalamocortical synapses. However, Somers et al. (2002) remark that the orientation tuning curves of intracellular recordings in cortical silencing experiments by Ferster and colleagues (Chung and Ferster, 1998; Ferster et al., 1996) are much broader than the extracellular measurements of the tuning curves.

2.4.5 Contrast Gain Functions of the Geniculate Cells

It seems that the parvocellular cells do have roughly linear contrast gain functions, whereas the magnocellular cells have logarithmic contrast gain functions (Derrington and Lennie, 1984; Sherman et al., 1984; Movshon et al., 1994). Note further that 90% of the geniculate cells are found in the parvocellular layers of the LGN (Dreher et al., 1976). These cells dominate the input to the layer 4, and hence the hyperbolic contrast gain functions of the striate cells (Albrecht and Hamilton, 1982) cannot be predicted by the saturation of the thalamic cells.

2.4.6 Contrast Gain Functions of the Striate Cells

Several groups have been investigating the contrast gain functions of the striate cells (Maffei et al., 1973; Dean, 1981; Albrecht and Hamilton, 1982). Furthermore,

Albrecht and Hamilton (1982) have quantified the contrast gain functions of the striate cells found within both the cat's and the monkey's primary visual cortex. During the experiments, 247 simple and complex neurons from cat and macaque monkey are recorded. It is found that the contrast gain functions of the neurons have the form of a hyperbolic function. The first phase of this function is defined as a linearly increase of response to linear increased contrast. This phase is followed by a rapid saturation, and later by total saturation (normalization or division).

Albrecht and Hamilton (1982) have also showed that the saturation level seems to be determined by the properties of the stimulus, i.e. orientation and spatial frequency, and not by the electrical properties of the neurons. Furthermore, maximum response to non-preferred stimulus is reported to be lower than to preferred stimulus.

Note that the striate cells saturate more than the geniculate cells (Derrington and Lennie, 1984; Sherman et al., 1984; Movshon et al., 1994). This finding reveals the existence of the intracortical inhibition. Carandini and Heeger (1994) propose that normalization of the activity comes from a pool of inhibitory cells representing all possible orientations and spatial frequencies. This pool generates the shunting inhibition, which is required to divide the excitatory input from the thalamus. The effect is saturation of the responses of the striate cells. However, the shunting inhibition requires that the membrane time constant of the striate cells decreases ~60 ms. This is not possible, since the resting time constant of the most striate cells are ~20 ms. Nevertheless it seems that the intracortical inhibition is indeed responsible, in one form or another, for shaping the contrast gain functions of the striate cells.

2.4.7 Contrast-Invariance of Orientation Tuning

The feedforward model cannot solely predict many of the response properties of orientation selective cells (Ferster and Miller, 2000). Contrast-invariance of orientation tuning shown by striate cells is perhaps the most striking example. As contrast increases the height of the response curve increases. However, the half-width at half-height of the response curve remains almost constant (Sclar and Freeman, 1982; Skottun et al., 1987). Loosely speaking, the striate cells can detect a line and 'tell' its orientation, also in poor light conditions. The study carried out by Sclar and Freeman (1982) shows that orientation tuning of a striate cell is independent of the contrast of the stimulus. The stimuli, which are used during the experiment, are sine-wave gratings. Data obtained from 45 cells (19 simple and 26 complex) within the cat primary visual cortex shows that orientation tuning is indeed independent of stimulus contrast. Furthermore, saturation of activity when the contrast of the stimulus is increased is evident in all 45 cases.

2.4.8 Cross-Orientation Inhibition

One last interesting finding is related to responses of striate cells to superimposed stimuli. When two stimuli are super positioned, the response of a cell is less than to the sum of each stimulus alone. The cross-orientation inhibition experiments reveal this finding. During the experiment two gratings stimulate the cell. The first grating is of the preferred orientation, and the second grating is of the orthogonal orientation. It is shown that the second grating inhibits a cell, which is of orthogonal orientation (Morrone et al., 1982; Bonds, 1989). Morrone et al. (1982) suggest that this inhibition arises from a pool of cells with different orientations.

2.4.9 On the Interplay between the Thalamocortical Circuitry and the Intracortical Circuitry

There is growing evidence that the thalamocortical circuitry and the intracortical network collaborate in order to shape the response properties of the striate cells. In cat, the orientation selectivity of the striate cells emerges in layer 4. This layer is the main target of the thalamocortical circuitry. However, the layer 4 lateral network of cat extends more than layer 4 lateral networks in other species, such as monkey, ferret and tree shrew. Furthermore, elongated subfields required by the feedforward model are rare even in cat (Pei at el., 1994). It seems that the intracortical connections alter the initial input from the thalamus to create the known response properties of the striate cells.

2.5 Putative Functional Role of the Long-Range Horizontal Connections in Responses of the Striate Cells

Even though the extent and shape of the long-range horizontal connections are relatively well documented, their functional role in cortical processing is still not clear. It is believed that in the primary visual cortex these connections are responsible for a variety of surround effects and configuration of the summation pools. Depending on stimulus configuration, these effects can be facilitatory or suppressive (Kastner et al., 1997; Polat and Sagi, 1993; Polat and Norcia, 1996; 1998; Solomon et al., 1999; Solomon and Morgan, 2000; Yu et al., 2002; Chisum et al., 2003). These effects are exposed by psychophysical studies on humans and neurophysiological studies on other species. Neurophysiology is the study of the relation between the sensation and the stimuli that produce them.

It is also hypothesized that the long-range horizontal connections are responsible for spike and burst synchronization, which is believed to play an

important functional role in perceptual grouping (Gray and Singer, 1989; Gray et al., 1989; Singer, 1993; 1999).

2.5.1 Summation Pools and Contour Integration

According to Chisum et al. (2003) the summation pools of tree shrew primary visual cortex reflect the elongated long-range horizontal connections found in layer 2/3. One support for this belief is the length summation experiment showing the facilitatory effect (Chisum et al., 2003). Neurons in the layer 2/3 show clear evidence for length summation up to distances, which correspond to the extent of the layer 2/3 long-range horizontal connections. Polat and Norcia (1998) stress the possible impact of long-range horizontal connections on the elongated physiological summation pools within the visual cortex of humans. Their experiments show that, in humans, when the area of the stimulus (a Gabor patch) is increased the visibility is improved. Furthermore, collinear configuration enhances striate cells more than orthogonal configuration. Consequently, as indicated by the two studies described above, the elongation of the long-range horizontal connections along the orientation axis could be linked to summation pools.

A recent study carried out by Ernst and colleagues (Ernst et al., 2003) relate psychophysical investigations with macaque monkeys to information theoretical modeling of contour integration. More precisely, the intention of this study is to compare quantitatively the performance of monkeys on detection simple contours, which are composed of aligned Gabor patches, with an ideal observer defined by Williams and Thornber (2001). Ernst et al. (2003) reveal that monkeys perform close to what is defined as an ideal observer. Their results indicate that the mechanisms within the visual cortex of monkey are highly specialized for contour integration. Thus, monkeys perform equally well as humans on contour integration. Furthermore, Ernst et al. (2003) stress that the gap between a heuristical explanation of the Gestalt criteria and a solid mathematical framework of the Gestalt properties remains. However, according to Ernst et al. (2003) the work by Williams and Thornber (2001) helps to narrow this gap. Williams and Thornber (2001) have shown that a contour can be defined as the outcome of a stochastic process with a known probability distribution. When this relation is inverted the model can take an arbitrary collection of elements, e.g. Gabor patches, and calculate the relative probability of them to belong to a contour. The result is the performance of an optimal observer on contour detection. These calculations give an upper limit, which can be compared to real observers as humans or monkeys, and hence give an indication on the performance of various species and individuals within a species.

Solomon at al. (1999) have studied the facilitation from flanks in human visual cortex. In this experiment Solomon at al. (1999) have used a target and two flanks, which are Gabor patches. The target and the flanks are collinearly positioned.

Their results show that even tough the flanks have opposite sign, relative to the target, the effect of presence of flanks is facilitation. However, this facilitation is not as prominent as if the flanks have same sign as the target. Furthermore, Solomon and Morgan (2000) have reported that when the flanks extend to surround the target the facilitation is reduced dramatically. This finding is interesting, since non-collinear flanks by themselves do not mask the target prominently.

Yu et al. (2002) have recently studied modulatory effects of cross-orientation. In this study the stimulus that is positioned in the surround has orthogonal orientation relative to the target. They report that cross surround facilitation is a surround-contrast dependent effect. This effect is more evident at low surround contrasts. Thus, when the full-surround has low contrast threshold is lowered. Interestingly, collinear surround facilitation is unaffected by surround contrast (Polat and Sagi, 1993). Yet another interesting result of the study by Yu et al. (2002) is related to the tuning of spatial frequency and orientation. The results indicate that on the contrary to the orientation, surround facilitation is narrowly tuned to spatial frequency. Yu et al. (2002) hypothesize that the target cells receive surround input from a group of cells narrowly tuned to target spatial frequency but loosely tuned to cross-orientation, indicating signal pooling over a large range of orientations.

Kastner et al. (1997) have investigated the neuronal responses to static and moving texture in area 17 of anaesthetized and paralyzed cats. The motivation is to investigate the neural basis of visual pop-out of orientation and motion. In the orientation test the stimulus that is positioned in the classical receptive field (CRF) is either orthogonal or parallel (uniform pattern) to the other bars that surround it. Their results indicate that orthogonal configuration enhances pop-out, since during this configuration the striate cells response more strongly compared to the uniform pattern. In the motion test all bars have same orientation, however, center and surround elements move either in the same or opposite directions. The results indicate that when the center element moves in the opposite directions the striate cells respond stronger than if all patterns move in the same direction. As a result of the experiments Kastner et al. (1997) conclude that static and moving patterns positioned outside the CRF may alter response properties of the striate cells.

2.5.2 Synchronization and its Putative Functional Role in Perceptual Grouping

Neurons in the visual cortex of anesthetized and awake animals (cat and monkey) tend to synchronize with each other, with near-zero phase lag and in the millisecond range, when activated with a single contour (Gray and Singer, 1989; Gray et al., 1989; Singer, 1993; 1999; Singer and Gray, 1995; Kreiter and Singer, 1996; Engel et al., 1997; Usrey and Reid, 1999; Friedman-Hill et al., 1999; 2000; Haig et al., 2000; Maldonado et al., 2000). Furthermore, the synchronized neurons

oscillate in the γ frequency range, which is 30–50 Hz (Singer and Gray, 1995). However, this precise synchronization is absent if the stimulus is composed of different contours moving in different directions (Gray et al., 1989).

Neurons that are synchronized are found several millimeters from each other (and are even located in different hemispheres) it is therefore assumed that the reciprocal long-range horizontal connections play a prominent functional role in emergence of this behavior (Engel et al., 1991; Löwel and Singer, 1992; König et al., 1993). One consequence of synchronization is enhancement of the incoming signals. This has been shown in hippocampus (Stevens and Zador, 1998), along the thalamocortical (Alonso et al., 1996; Usrey and Reid, 1999) and the intracortical pathway (Alonso and Martinez, 1998).

According to the Gestalt physiologists the cognitive system of humans tends to relate objects (or events) to each other if they are connected in time and space (Köhler, 1930; Koffka, 1935). Events that happen simultaneously are more likely to be related to each than events that are separated in time. The same idea is also valid for objects. Contours (or contrast edges) that have a similar contrast or move with the same speed and direction are more likely to be part of the same object. It is assumed that synchronization in the early stages of vision is a result of internal dynamics. It is also believed that synchronization is context-dependent and probably reflects Gestalt criteria for perceptual grouping (Gray et al., 1989; Kreiter and Singer, 1996).

3 The Bayesian Confidence Propagation Neural Network

The environment where the animals and the humans live in is ever changing and highly complicated. The information, which is available in the environment, is often noisy and incomplete. Furthermore, several events occur simultaneously, and on different time scales. Biological learning systems seem to cope with these properties of the real world successfully. These systems are adapted to specific habitats and are formed by millions of years of evolution.

Sandberg et al. (2002) have recently developed an incremental learning rule derived from the Bayesian confidence propagation neural network (BCPNN) (Lansner and Ekeberg, 1987; 1989; Lansner and Holst, 1996; Holst, 1997). Sandberg et al. (2002) have also demonstrated that when the network is configured as a recurrent attractor network it has 'palimpsest' properties (see also Sandberg (2003)). Thus, recently learned patterns are more stable than older patterns to avoid catastrophic forgetting.

The recurrent network proposed by Sandberg et al. (2002), which consists of dense local and sparse long-range connections, fits well the known connectivity of the neocortex (section 2.3). In the same study it is shown that the BCPNN does not suffer from catastrophic forgetting. Furthermore, the network's capacity depends on the learning time constant, and it recalls newer patterns faster than the older ones (during the retrieval phase).

The BCPNN has been developed in analogy with the known columnar structure of the neocortex (Mouthcastle, 1957; 1978; 1997; Powell and Mouthcastle, 1959; Hubel and Wiesel, 1962; 1977). The network consists of units that correspond to cortical minicolumns. The units are grouped into hypercolumn-like modules and the summed activity within each hypercolumn module is normalized to one. The normalization is a way of controlling the total activity of the network. The normalization scheme is supported by the studies on the response saturation

properties of the striate cells (Maffei et al., 1973; Dean, 1981; Albrecht and Hamilton, 1982; section 2.4.6).

The BCPNN learning rule is based on a probabilistic view of learning, and is derived from Bayes' rule (Bayes, 1958). Units receive input from all other units in the network representing confidence of feature detection. Based on the input, the units calculate posterior probabilities of outcomes. The learning rule is based on Hebb's ideas on synaptic plasticity and emergence of cell assemblies (Hebb, 1949). Thus, correlated activity reinforces the connections between pairs of units. Anti-correlated activity results in weakening of a connection, and emergence of an inhibitory one. A brief derivation of the BCPNN architecture and the incremental learning rule can be found in the next section (see Sandberg et al. (2002) and Sandberg (2003) for a more detailed derivation).

3.1 Derivation of the Network Architecture and the Incremental Learning Rule

As mentioned above the starting point is Bayes' rule and the naive Bayesian classifier (Good, 1950). Using the naive Bayesian classifier (NBC), probabilities of attributes y_i, can be calculated given other attributes x_i. It is assumed that both these two sets of attributes are discrete. Furthermore, the variable x_i is assumed to be independent ($P(x_i,\ldots,x_n)=\prod_{i=1}^{n}P(x_i)$), and conditionally independent given y_i ($P(x_i,\ldots,x_n \mid y_j)=\prod_{i=1}^{n}P(x_i \mid y_j)$).

Based on the properties of x_i and y_i Bayes' rule gives

$$\pi_j = P(y_j \mid x) = P(y_j)\prod_{i=1}^{n}\frac{P(x_i \mid y_j)}{P(x_i)} = P(y_j)\prod_{i=1}^{n}\frac{P(x_i,y_j)}{P(y_j)P(x_i)}.$$

Based on the assumption that information on some attributes is missing, the formula above can be modified as

$$\pi_j = P(y_j \mid x_i, i\in A) = P(y_j)\prod_{i\in A}\frac{P(y_j,x_i)}{P(y_j)P(x_i)}.$$

Taking the logarithm of π_j eliminates the product

$$\log\pi_j = \log P(y_j)+\sum_{i\in A}\log\left[\frac{P(y_j,x_i)}{P(y_j)P(x_i)}\right] = \log P(y_j)+\sum_{i\in A}o_i\log\left[\frac{P(y_j,x_i)}{P(y_j)P(x_i)}\right]$$

where the indicator o_i represent whether there is information about a feature i or not

$$o_i = \begin{cases}1 & i\in A\\ 0 & i\notin A\end{cases} = I_A(i).$$

Equations above can be implemented as a one-layered feedforward neural network with the weights w_{ij}= log[P(y_j,x_i)/(P(y_j)P(x_i))] and the biases β_j = log P(y_j).

The input layer activations are o_i. The one-layered feedforward neural network calculates the posterior probabilities π_j using an exponential transfer function, given the input attributes.

The one-layered feedforward neural network becomes modular when the discrete attribute values are represented by the indicator variables. Continuous valued attributes can be interval coded. Note that this scheme is common within the neocortex (Mouthcastle, 1957; 1978; 1997; Powell and Mouthcastle, 1959; Hubel and Wiesel, 1962; 1977). Every orientation minicolumn within the primary visual cortex responds selectivity to an interval of orientation (Hubel and Wiesel, 1962; 1977). Furthermore, all orientations within a small patch of the visual field are represented by a collection of such orientation minicolumns, which 'populate' an orientation hypercolumn.

Assume now that the representation of the attributes is changed so that each attribute can take M_i different values. Furthermore, the observation of a given value of a given attribute as a new binary value is defined (marked with double indices)

$$\pi_{jj'} = P(y_{jj'}) \prod_{i \in K} \frac{P(y_{jj'}, x_{ik})}{P(y_{jj'})P(x_{ik})}$$

and similarly

$$\pi_{jj'} = P(y_{jj'}) \prod_{i=1}^{n} \sum_{i'=1}^{M_i} \frac{P(y_{jj'}, x_{ii'})}{P(y_{jj'})P(x_{ii'})} o_{ii'} .$$

with indicator $o_{ii'} = 1$ if $i' = k$ and zero otherwise

Consider now the attributes X_i as stochastic variables with values $\{x_{i1}, \ldots, x_{iM_i}\}$, which are explicitly represented in the network. Now $o_{X_i}(x_{ii'}) := o_{ii'}$ can be view as a degenerate probability $o_{X_i}(x_{ii'}) := \delta_{x_{ik}}(x_{ii'})$, which is zero for all $x_{ii'}$ except for the known value x_{ik}. When o_{X_i} is replaced with a general probability P_{X_i} we get

$$\hat{\pi}_{jj'} = P(y_{jj'}) \prod_{i=1}^{n} \sum_{i'=1}^{M_i} \frac{P(y_{jj'}, x_{ii'})}{P(y_{jj'})P(x_{ii'})} P_{X_i}(x_{ii'})$$

$$\log(\hat{\pi}_{jj'}) = \log P(y_{jj'}) + \sum_{i=1}^{n} \log \left[\sum_{i'=1}^{M_i} \frac{P(y_{jj'}, x_{ii'})}{P(y_{jj'})P(x_{ii'})} P_{X_i}(x_{ii'}) \right].$$

If the outcomes $x_{ii'}$ of different attributes are independent of each other when conditioned on X_i, $\hat{\pi}_{jj'}$ will be the expectation of $\pi_{jj'}$ given input X_i.

The resulting equations reveal that the network has a modular structure. The unit *ii'* represent explicitly values $x_{ii'}$ of X_i, which can be viewed as a hypercolumn. By definition the total activity within a hypercolumn *i* is normalized

$$\sum_{i'=1}^{M_i} P_{X_i}(x_{ii'}) = 1 .$$

The probability of $y_{jj'}$ can be calculated given the uncertain information related to $x_{ii'}$. Probability $P_{X_i}(x_{ii'})$ reflects the uncertainty. The network setting of these relations is

$$h_{jj'} = \beta_{jj'} + \sum_{i}^{N} \log\left(\sum_{i'}^{M_i} w_{ii'jj'} P_{X_i}(x_{ii'}) \right).$$

In this network setting $h_{jj'}$ is defined as the support of unit *jj'*. The output of a unit *jj'* is given by $\hat{\pi}_{jj'} = f(h_{jj'}) = e^{h_{jj'}}$. The biases of the units and the weights between the pairs of units are defined as

$$\beta_{jj'} = \log(P(y_{jj'}))$$

$$w_{ii'jj'} = \frac{P(x_{ii'}, y_{jj'})}{P(x_{ii'})P(y_{jj'})}$$

The independence assumption is too strong and can only be approximately fulfilled as we deal with approximations of probabilities. As a consequence the outputs within each hypercolumn is normalized

$$\hat{\pi}_{jj'} = f(h_{jj'}) = \frac{e^{h_{jj'}}}{\sum_{j'} e^{h_{ij'}}}.$$

Note that both the input and the output of the units are probabilities. It is now possible to configure the network as a fully recurrent network, which can work as an autoassociative memory. The procedure starts with the currently observed probability $P_{X_i}(x_{ii'})$, which is used as an initial approximation of the true probability $X_{ii'}$. From $P_{X_i}(x_{ii'})$ the posterior probability is calculated, which tends to be a better approximation. This probability value is later fed back to the process. This procedure is iterated until a stable state is reached. Sandberg et al. (2002) explain the procedure with: 'this represents a heuristically estimated probability closest to the observed data and consistent with the already acquired knowledge, that is prior information represented by the learning parameters $\beta_{jj'}$ and $w_{ii'jj'}$.'

The continuous time updating rule is defined as

$$\tau_c \frac{dh_{jj'}(t)}{dt} = \beta_{jj'} + \sum_{i}^{N} \log\left(\sum_{i'}^{M_i} w_{ii'jj'} f(h_{ii'}(t)) \right) - h_{jj'}(t)$$

where parameter τ_c is the 'membrane time constant' of each unit.

The BCPNN has two modes. During the learning mode the weights and the biases are modified, whereas during the retrieval mode these parameters are static and a pattern is being retrieved. At the start of the retrieval mode the units are initiated with probabilities. These values propagate through the network during the retrieval mode.

The original BCPNN learning rule (Lansner and Ekeberg, 1989; Lansner and Holst, 1996) uses counter for calculating the probabilities $P_{X_i}(x_{ii'})$. The counter associated with a certain unit is updated each time its unit is active. Later, when

the training is finished, the counters are divided with the total number of training examples. Probabilities $P_{X_i}(x_{ii'jj'})$ associated with co-activations of units are calculated in the same way. Whenever the units *ii'* and *jj'* are active (as a result of a training example) the counter associated with the weight between them is updated. Later this counter is divided with the total number of training examples. When the estimations $P_{X_i}(x_{ii'})$ and $P_{X_i}(x_{ii'jj'})$ are calculated the weights and the biases of the units can be calculated. Afterwards the network can be used in the retrieval mode.

A continuously operating learning system operates in a fundamentally different way. Such a system learns new patterns and updates the weights and the biases continuously. Estimations $P_{X_i}(x_{ii'})(t)$ and $P_{X_i}(x_{ii'jj'})(t)$ need to be estimated given the information that $\{x(t'), t' < t\}$. There are three requirements, which such a system must fulfill. Firstly, it should converge towards $P_{X_i}(x_{ii'})(t)$ and $P_{X_i}(x_{ii'jj'})(t)$ in a stationary environment. Later, more recently learned patterns must be more stable than older ones. The last requirement is that it should smooth or filter out noise and adapt to longer trends.

These requirements are achieved by the incremental BCPNN learning rule by approximating $P_{X_i}(x_{ii'})(t)$ and $P_{X_i}(x_{ii'jj'})(t)$ with the exponentially smoothed running averages $\Lambda_{ii'}(t)$ of the activity $\hat{\pi}_{ii'}$ and $\Lambda_{ii'jj'}(t)$ of coincident activity $\hat{\pi}_{ii'}\hat{\pi}_{jj'}$.

The continuous time version of the update and learning rule is defined as

$$\hat{\pi}_{ii'} = \frac{e^{h_{ii'}}}{\sum_j e^{h_{ij}}}$$

$$\frac{d\Lambda_{ii'}(t)}{dt} = \alpha([(1-\lambda_0)\hat{\pi}_{ii'}(t) + \lambda_0] - \Lambda_{ii'}(t))$$

$$\frac{d\Lambda_{ii'jj'}(t)}{dt} = \alpha([(1-\lambda_0^2)\hat{\pi}_{ii'}(t)\hat{\pi}_{jj'}(t) + \lambda_0^2] - \Lambda_{ii'jj'}(t))$$

$$\beta_{ii'}(t) = \log(\Lambda_{ii'}(t))$$

$$w_{ii'jj'}(t) = \frac{\Lambda_{ii'jj'}(t)}{\Lambda_{ii'}(t)\Lambda_{jj'}(t)}$$

The parameter α is defined as the inverse of the learning time constant τ_L. During the retrieval mode α is set to zero, and hence the weights and the biases are not updated.

The parameter λ_0, which corresponds to noisy background activity, is introduced to avoid logarithmic zero. In the absence of input the parameters $\Lambda_{ii'}(t)$ and $\Lambda_{jj'}(t)$ converge towards λ_0 and $\Lambda_{ii'jj'}(t)$ converges towards λ_0^2. As a consequence the weight between two units becomes $w_{ii'jj'}(t) = 1$.

4 Related Work

There are several types of network models that address vastly different response properties of the cells populating the primary visual cortex, such as response saturation followed by normalization (Albrecht and Geisler, 1991; Heeger, 1992; Carandini et al., 1997), contrast-invariance of orientation tuning (Somers et al., 1995; 1998; 2002; Troyer et al., 1998; Adorján et al., 1999), and various surround effects (Somers et al., 1998; 2002; Yen and Finkel, 1998; Li, 1998; Grossberg and Raizada, 2000) (See relatively new reviews by Ferster and Miller (2000) and Martin (2002)). Most of these models are of either cat (Hubel and Wiesel, 1962; Somers et al., 1995; 2002; Troyer et al., 1998; Grossberg and Raizada, 2000) or monkey (McLaughin et al., 2000; Wielaard et al., 2001) primary visual cortex. Some network models address the interactions within a very small region (Kayser and Miller, 2002), while others focus on interactions within patches that are several square millimeters on cortical surface (Somers et al., 1998; 2002; Yen and Finkel, 1998; Li, 1998; Grossberg and Raizada, 2000).

Most often these models are, however, classified after the importance of the thalamocortical circuitry (or the intracortical connections) in generating orientation selectivity and other response properties of the striate cells. The feedforward models rely heavily on the thalamocortical circuitry for achieving contrast-invariance of orientation tuning. The normalization models, which can be seen as a sub-class of the feedforward models, address mainly the contrast gain functions of the striate cells. The recurrent models, however, assume that the thalamic input is both weak and poorly tuned. They address mainly contrast-invariance of orientation tuning like the feedforward models.

4.1 Feedforward and Normalization Models of Orientation Selectivity

The models of the orientation hypercolumns, which rely heavily on the thalamocortical circuitry for achieving orientation selectivity, are classified as

feedforward models (Hubel and Wiesel, 1962; Troyer et al., 1998; Kayser and Miller, 2002).

Hubel and Wiesel (1962) have proposed the original feedforward model (section 2.4). LGN cells of the same type (ON-center or OFF-center) converge to form elongated regions, which becomes the subregions of the simple cells. This model explains the origins of orientation selectivity in an elegant way. However, it cannot explain contrast-invariance of orientation tuning. Nevertheless, this simple model is still used for studying the thalamocortical circuitry.

Miller and colleagues (Troyer et al., 1998; Kayser and Miller, 2002) have shown that contrast-invariance of orientation tuning of cat layer 4 simple cells can be achieved within a very small patch of the primary visual cortex. They assume that the input from the thalamus is strong and well tuned for orientation as proposed by Reid and Alonso (1995). In these models the intracortical recurrent connections, especially the long-range horizontal connections, play a minor functional role in shaping the response properties of the striate cells. Interactions inside the modeled small patch are based on the spatially opponent inhibition theory (Ferster, 1988). According to the study by Ferster (1988) inhibitory simple cells mediate local inhibition between excitatory simple cells, which have opposite absolute spatial phase. Miller and colleagues (Troyer et al., 1998; Kayser and Miller, 2002) assume that the cells participating in this assembly are located inside a very small region corresponding to an orientation minicolumn. As a consequence this model assumes that inhibition is local.

A sub-class of the feedforward models is the normalization models (Albrecht and Geisler, 1991; Heeger, 1992; Carandini and Heeger, 1994; Heeger et al., 1996; Carandini et al., 1997). The primary goal of these models is to address the contrast gain functions (response saturation) of the striate cells. Thus, these network models assume that input from the LGN grows linearly with contrast of the stimulus. The geniculate input is divided by a linearly growing inhibitory input, which is of cortical origin and depends of the contrast of the stimulus. The effect is division of the input from the LGN.

Carandini and Heeger (1994) proposed that the inhibition would take the form of a shunting inhibition. This inhibition would originate from a pool of striate cells with all possible orientation and spatial frequency preferences (the normalization pool). The shunting effect corresponds to the lowering of the time constants of the striate cells, and as a result the cells' integration times decreases. However, the shunting inhibition requires that the membrane time constants of the striate cells decreases ~60 ms. This is not possible, since the resting time constants of most striate cells are ~20 ms, and membrane time constants cannot decrease below zero. Nevertheless it seems that the intracortical inhibition is indeed responsible, in one form or another, for shaping the contrast gain functions of the striate cells, since most thalamic cells saturate only mildly (sections 2.4.5 and 2.4.6).

4.2 Recurrent Models of Orientation Selectivity

Recurrent models are fundamentally different in their approach on addressing response properties of the striate cells (Ben-Yishai et al., 1995; 1997; Somers et al., 1995; 1998; 2002; Hansel and Sompolinsky, 1996; Adorján et al., 1999; McLaughin et al., 2000; Wielaard et al., 2001). In these network models the intracortical recurrent connections play a prominent functional role in achieving orientation selectivity and contrast-invariance of orientation tuning. The geniculate input is assumed to be weak and poorly tuned for orientation (Pei et al., 1994). Intracortical connections are assumed to be responsible for enhancing the geniculate input.

Somers and colleagues (1995; 2002) assume that inhibition originates from a pool of cells with more spread in orientation selectivity than the excitatory input. This network topology helps to prevent the spread of a local excitatory activity. A prominent feature of the recurrent models is thus the emergence of the 'Mexican-hat' function (Somers et al., 1995; 2002; Hansel and Sompolinsky, 1996). The prominent feature of the Mexican-hat functions is that striate cells in close surroundings receive net excitatory input, whereas cells located further away receive net inhibitory input. Furthermore, the long-range excitatory connections are patchy and target the iso-orientation domains. As a consequence of this local and long-range connections pattern, the orientation tuning becomes a property of the recurrent cortical circuitry, and does not depend on the tuning of the geniculate input. Note also that the layout of excitatory and inhibitory connections alone define the tuning width of the cells.

The network model of macaque primary visual cortex (layer 4Cα) by Adorán et al. (1999) is based on the anisotropic intracortical excitatory connections within the primary visual cortex of the macaque monkey. These connections provide both the initial orientation bias and its subsequent amplification. Furthermore, the thalamic input from the M cells does not provide any orientation bias. It is assumed that the aspect ratios of the striate cells' receptive fields are not in line with sharply tuned simple cells (Pei et al, 1994).

McLaughlin and colleagues (McLaughin et al., 1999; Wielaard et al., 2001) presented yet another model of the layer 4Cα macaque primary visual cortex. The Mexican-hat connectivity, which near the orientation singularities provide the cells with broadly tuned local inhibition. The result is sharpening of the orientation tuning of the striate cells.

The hypercolumn model that have been proposed in this thesis work is based on the hypothesis that the recurrent excitatory and inhibitory intracortical connections can explain emergence of several response properties, such as response saturation (followed by normalization) and contrast-invariance of orientation tuning of the striate cells. Note that feedforward model proposed by Miller and colleagues (Troyer et al., 1998; Kayser and Miller, 2002) does also assume that the intracortical connections play an important functional role in forming the response

properties of the striate cells, however, their model assumes that connections are mainly feedforward and local (within a couple of orientation minicolumns).

4.3 Models that Address Various Surround Effects

Besides contrast invariance of orientation-tuning Somers et al. (1998; 2002) address several other phenomena, such as contrast-dependent length tuning (end-stop cells), supersaturation and facilitation suppression based on the local and long-range synaptic interactions. Contrast-dependent length tuning is the effect of increase in excitatory receptive field size of a cell with decreased contrast. Supersaturation, which is related to contrast-dependent length tuning, means that increased contrast of a stimulus can actually cause decreased activity of a cell. Facilitation suppression reveals the modulatory effects of the (uniform or orthogonal) surround grating to the center grating. When the center grating has low contrast surround grating has facilitatory effect independent of the configuration. However, during high contrast an orthogonal surround grating has more prominent facilitatory effect.

Several groups have addressed contour integration and perceptual grouping (Grossberg and Raizada, 1998; Yen and Finkel 1998; Li, 1998). Grossberg and Raizada (1998) present a laminar model of areas V1 and V2 that addresses contrast-sensitive perceptual grouping and attention. Their highly detailed model pools and sharpens raw contrast edge signals, which become closed boundaries that are ‘filled-in’ by neuronal activity representing color. In this model, above-threshold firing layer 2/3 complex cells are responsible for perceptual grouping. The model also demonstrates that long-range excitation and disynaptic inhibition is sufficient for explaining emergence of illusory contours.

Yen and Finkel (1998) address the extraction of salient contours within the primary visual cortex. Their model relies heavily on temporal synchronization, which is a result of strong facilitation of the modeled striate cells. This facilitation is mediated by the long-range horizontal connections. Furthermore, the level of temporal synchronization between modeled striate cells determines perceptual salience. Li (1998) address contour integration within the primary visual cortex. The initial activity is amplified so that edges that form smooth contours oscillate in synchrony. In this model, higher cortical areas, such as V2, that are involved in perceiving illusory contour are absent. However, the author notes that the model could easily be modified to include other (higher) cortical areas.

5 Results of the Thesis

5.1 Issues Related to the Layout of the Intracortical Connections

The centerpiece of this thesis work is the abstract hypercolumn model, which is presented in Paper II and later refined in Papers III and IV. The model addresses the interactions within an area that roughly corresponds to a cortical hypercolumn and can be mapped to both layer 4 and layer 2/3 of cat area 17 (Papers III and IV).

The abstract hypercolumn model, which is based on the relevant aspects of known physiology and anatomy of cat area 17, is derived from the BCPNN architecture (chapter 3). The BCPNN architecture requires that the activity within a modeled hypercolumn module is constant. This requirement is founded on the normalization mechanism of the striate cells (section 2.4.6). The inhibitory cell, which is positioned between the two layers of the hypercolumn model (not to mix with cortical layers), fulfils this requirement (section 5.2). This cell corresponds to a uniformly distributed population of large basket cells within a cortical layer. Note that large basket cells are know for sending out horizontally aligned axons up to 1.5 mm (Kisvárday et al., 1997), whereas other interneurons, such as chandelier cells (that populate mainly layer 2/3) and small basket cells target cells in their near surroundings.

The abstract hypercolumn model is based on the assumption that large basket cells can inhibit a region, which corresponds to a cortical hypercolumn. Note also that distribution of the connections from an injection site to local iso-, oblique- and cross-orientation domains also suggests that the inhibitory connections are less biased towards the local iso-orientation domain than the excitatory connections (Kisvárday et al., 1997). (Usually such an injections site is smaller than an iso-orientation domain.) It is further assumed that the excitatory cells within a region that correspond to a hypercolumn can drive these large basket cells. The outcome of this scheme is local inhibition that can control the total activity of a region,

which roughly corresponds to a hypercolumn. This scheme can occur both in layer 4 and layer 2/3 (independent from each other), since large basket cells are found in both these layers (O'Leary, 1941; Tömböl, 1978).

The correlation-based developmental models of layer 4 in Papers III and IV propose yet another local inhibitory network, which probably operates parallel to the inhibitory network of large basket cells. The connections within the correlation-based models are created using the BCPNN incremental learning rule. The stimuli consist of randomly positioned contrast edges. The weight matrix, which is the result of training, reflects the degree of correlation between units (recall that BCPNN units correspond to cortical minicolumns). The units in Paper III have layer 4 simple cells' receptive fields, and hence consist of two elongated subregion of opposite (ON/OFF) sign. One implication of the correlation-based activity is that developed connections between units do not solely depend on orientation preference. Relative spatial phase is as important. Thus, this correlation-base circuitry is also in line with the opponent inhibition theory by Ferster (1988), which suggests that inhibitory simple cells inhibit excitatory simple cells (in their close surroundings) if they have opposite absolute spatial phase. (Hirch and colleagues (Hirch et al., 2000) have recently reported inhibitory simple and complex cells. However, their functional roles are not fully understood.) The implication of the opponent inhibition theory is strong iso-orientation inhibition, which originates from inhibitory simple cells that have opposite absolute spatial phase, but same orientation preference. Excitatory simple cells can inhibit other excitatory cells through these inhibitory simple cells.

The estimations of the bouton distribution in layer 4 reveal that local projections are biased towards the iso-orientation domain, whereas long-range projections are equally distributed between the iso-, oblique- and cross-orientation domains (Yousef, 1999; Chisum, 2003). This finding is in contradiction with the patchy, iso-orientation domain biased, layout of the layer 2/3 (Bosking, 1997; Kisvárday, 1997; Schmidt, 1997; Chisum, 2003). In Paper IV the reasons behind these differences have been addressed. It is assumed that the patchy layout of the layer 2/3 is due to excitatory cells targeting mainly other excitatory cells located in distal iso-orientation domains. These excitatory cells are probably the pyramidal cells.

The correlation-based networks of layer 4 and layer 2/3 are similar to area 17 in that local connections are dense, whereas distal connections are sparse. Furthermore, the long-range horizontal connections of both layers are elongated along the orientation axis.

5.2 Local Connections Account for Response Saturation and Contrast-Invariance of Orientation Tuning

In cat, orientation selectivity emergences within layer 4, which is also the main recipient of the thalamic input (Hubel and Wiesel, 1962). Compared to other species (monkey, tree shrew and mouse) connections within layer 4 of cat extend longer. It is also striking that the fraction of the inhibitory cells is higher in layer 4 than in any other cortical layer. Thus, it is tempting to speculate on the functional role of the intracortical connections and different cell types in the emergence of various response properties of the striate cells.

Note however that when considering the intracortical connections it is also important to distinguish between those that functionally belong to the feedforward network (the thalamocortical circuitry), and those that are recurrent connections. The intention of the abstract hypercolumn model is to demonstrate that the recurrent connections, which originate from an area that roughly corresponds to a cortical hypercolumn, can explain emergence of several response properties of the striate cells.

It is shown that the striate cells saturate with stimulus contrast increase (Maffei et al., 1973; Dean, 1981; Albrecht and Hamilton, 1982; section 2.4.6). For most striate cells this saturation, which is very rapid, starts at 50-60% of their response ranges. As a result of this rapid saturation, the contrast gain functions take the form of a hyperbolic function (Albrecht, 1982). However, results by Movshon et al. (1994) indicate that the majority of LGN cells, namely the P cells, have practically linear response functions and shows very little sign of saturation as a result of contrast stimulus increase (section 2.4.5). On the contrary, the M cells do saturate more prominently, and hence have logarithmic gain functions. It is also known that most striate cells (especially those found in lower parts of layer 3, layer 4A and layer 6A) receive a mixture of P and M signals. Thus, saturation of the input from the LGN cannot solely explain the hyperbolic contrast gain functions of the striate cells. It is thus hypothesized that the cortical inhibition plays an important functional in emergence of this behavior.

In Paper II, a possible mechanism behind response saturation followed by normalization is demonstrated. The large basket cell, which is positioned in between the layers of the hypercolumn model (section 5.2), integrates the total activity of the excitatory cells located in the input layer. This cell can estimate the total input from the LGN and cortex to a small patch, which corresponds to a hypercolumn. The strength of this thalamic input is primarily a function of the stimulus contrast, since the large basket cell receives input from all orientation domains. In the next step, the large basket cell inhibits the excitatory cells in the output layer of the model. The result is the control of the activity of the excitatory cells located in the output layer. This circuitry, which is limited to a hypercolumn,

is sufficient to explain the machinery needed to control the total activity within a modeled hypercolumn, and hence response saturation followed by normalization.

Contrast-invariance of orientation tuning is also explained by the same local circuitry (Paper II). As an effect of stimulus contrast increase the large basket cell increases its activity, and hence inhibits excitatory cells found in the output layer more strongly. For those output layer excitatory cells, whose orientation preferences differ more than ~45º relative to the stimulus, the inhibition increases more rapidly than the feedforward excitation from the LGN (and recurrent excitation from the cortex). Paradoxically, stimulus contrast increase causes these cells to decrease their activities. It is possible to control the balance between inhibition and excitation by adjusting the excitatory connections between modeled cells located in the same orientation minicolumn module and layer.

From this follows also narrowing of the orientation tuning (Paper II). However, this narrowing is only marginal, since the model does not assume unrealistically strong inhibitory synapses (see (Martin, 2002) for a critical review of the existing feedforward and recurrent models of hypercolumns). Instead, the orientation tuning is mainly by the inhibitory simple cells (Hirch et al., 2000), which inhibit excitatory cells that have opposite absolute spatial phases (Paper III).

5.3 Findings Related to the Function of the Long-Range Horizontal Connections

5.3.1 Long-Range Horizontal Connections Account for Spike and Burst Synchronization

Simulations in Paper I are intended to show that spike and burst synchronization is possible to achieve between striate cells positioned several millimeters from each other. This study reveals also the facilitatory effect of synchronization on different levels (section 2.5.2).

Synchronized activity is demonstrated with a biologically plausible network model of layer 2/3 of tree shrew primary visual cortex. The network consists of six sub-sampled orientation minicolumns that are composed of integrate-and-fire neuron models, all having similar orientation preference. The orientation minicolumns are co-linearly positioned in adjacent hypercolumns. The intention with the layout of this network is to mimic the elongated shape of the summation pools (sections 2.5.1 and 5.3.2; Papers III and IV). Connections between modeled cells inside the orientation minicolumns are dense, whereas connections between cells located in different orientation minicolumns are sparse. On average, every modeled cell receives six inputs from the other cells that are positioned in the

same orientation minicolumn, whereas on average two connections are from cells positioned in the other five orientation minicolumns.

This study demonstrates that burst and zero-lag (γ frequency) spike synchronization is possible to achieve in a biologically detailed network model. Spike synchronization is however fragile, since it could be destroyed momentary by repetitive firing shown by some of the modeled cells. Spike synchronization is only slightly tighter between neurons spatially closer to each other, i.e. neurons positioned inside an orientation minicolumn. The correlation between distance and degree of spike synchronization is noticeable when much larger models are simulated (in these model the distance between the orientation minicolumns on the extremes is 8 mm). Nevertheless, high precision zero-lag spike synchronization is possible to achieve between cells located within the orientation minicolumns on the extremes of the modeled network. These two orientation minicolumns are located 2.5 mm from each other. Note that the spike propagation delay between them is ~4 ms, and that there are at most two direct connections between cells located within these two orientation minicolumns.

It is evident that synchronization on different levels facilitates the modeled cells. It is thus hypothesized that synchronization seen in visual cortex (section 2.5.2) plays a similar role, e.g. during low-contrast conditions to facilitate the thalamic signals.

5.3.2 Findings Related to the Summation Pools and Contour Integration in Visual Cortex

Several groups have suggested that long-range horizontal connections might play an important functional role in response facilitation of the striate cells of humans and other species (Polat and Sagi, 1993; Polat and Norcia, 1996; 1998; Yu et al., 2002; Chisum et al., 2003; sections 2.5.1 and 4.3). These studies relate detection or increased visibility of abstract figures, such as contours or Gabor patches, to response facilitation of the striate cells. Note that the intention of these studies is somewhat different from those that focus on synchronization (section 2.5.2).

In Paper III (and partially in Paper IV) the functional role of the long-range horizontal connections in response facilitation of the striate cells is investigated. These simulations are primarily influenced by the experiments of Polat and colleagues (Polat and Sagi, 1993; Polat and Norcia, 1996; 1998). They suggest that elongated physiological summation pools can explain response facilitation in visual cortex (see section 2.5.1 for the details).

Two assumptions are made in Papers III and IV. Firstly, total activity of the units inside a hypercolumns is constant, as required by the BCPNN incremental learning rule. The abstract hypercolumn model (Paper II) addresses this requirement. Furthermore, during the simulations activities of the units are calculated in every time step without taking the delays between units into account.

However, Paper I shows that synchronization on different levels is possible over several millimeter on cortex surface.

Recall that the correlation-based model of the cat area 17 in Paper III represents layer 4 circuitry, and hence the units' receptive fields are defined as contrast edge detectors. The stimuli used during training are randomly positioned contrast edges. The end result is overlapped circuitries that comprise units located in several hypercolumns and operate as contour integrators. This network can efficiently reinforce the relatively weak and noisy input from the LGN. Units along a contrast edge are facilitated or inhibited by their neighbors as a result of their orientation preferences and spatial phases (section 5.1). Yet another effect of response facilitation, which is obviously selective, is to sharpen the orientation tuning of the units.

5.4 Summary of the Papers

Professor Anders Lansner at the 'Studies of Artificial Nervous Systems' (SANS, NADA, KTH) has supervised this thesis work[1].

5.4.1 Paper I

In this paper it has been shown that phenomena like spike and burst synchronization are possible to simulate with a biologically detailed network of integrate-and-fire neurons. One of the most important conclusions of this paper is that high precision spike synchronization (<10 ms) is possible to achieve with a constant current input. However, then the strength of the current input is above a certain level neurons display repetitive bursting. This behavior enhances population oscillation but destroys high precision spike synchronization.

The network behavior is rather independent of the input type. It is further assumed that more pronounced spike synchronization is possible achieved for the one bar stimulus simulation, if the stimulus configuration is different.

The long-range horizontal connections play an important functional role for synchronization. Even with very few connections it is possible to spike synchronize neurons situated in minicolumns 2.5 mm from each other. During the simulations there has been at most two connections between neurons in orientation minicolumn one and six. These two orientation minicolumns are positioned on the extremes of the network.

Spike synchronization is slightly tighter between neurons spatially closer to each other and decreased with distance. This finding is detectable when much

[1] Prof. Lansner has generated most of the ideas, especially at the beginning of the thesis work. I have detailed the connections to the anatomy and physiology of the visual cortex, done the implementations of the models, the simulations and most of the writing.

larger networks are simulated (up to 8 mm between orientation minicolumns on the extremes).

This paper is presented at the ICANN – International Conference on Artificial Neural Conference in Vienna, August 2001.

5.4.2 Paper II

In this paper we derived and presented a model of a cortical hypercolumn from the BCPNN architecture. This model replicates 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 of orientation tuning and response saturation (followed by narmalization) are demonstrated.

One important assumption made is on the linear responses of the LGN cells to the contrast stimulus increase (The LGN input is mainly from the P-cells, see section 2.4.5). Based on this assumption, it is shown that the response saturation (followed by normalization) behavior of the modeled striate cells in the output layer is possible to explain by the local recurrent connections inside the abstract hypercolumn model.

Narrowing of the orientation tuning is possible through the reinforcement of the LGN input by the inputs from excitatory cells that have similar orientation preferences. However, as a side effect, cells having non-preferred orientations are also excited above their resting activity levels, and this affects their orientation tuning negatively.

The divisive inhibition of the excitatory cells in the output layer (by the large basket cell) results in sharpening of the orientation tuning curves and normalization of their activity. The large basket cell represents a pool of large basket cells with a mixture of preferred orientations. It is assumed that these inhibitory cells are distributed homogenously within the layers of the area 17. The activity of the large basket cell is a function of the excitatory cells in the input layer and the LGN input. Thus, it samples the total activity inside the hypercolumn. Studies made on cat area 17 supports this network configuration.

This paper is presented at the 9th ICONIP – International Conference on Neural Information Systems in Singapore, November 2002.

5.4.3 Paper III

A patch of layer 4 of cat area 17 has been modeled. This developmental network model is based on the modular structure of the neocortex. The BCPNN incremental learning rule develops the connections between the units. The correlation-based network captures some of the known properties of area 17 of cats, such as dense local and sparse distal connectivity.

The network has two different types of interneurons. Large basket cells are responsible for keeping the total activity within a hypercolumn constant. The second group of interneurons, the inhibitory simple cells, mediate local and distal inhibition through targeting excitatory cells that are located in their close surroundings and have opposite absolute and relative spatial phase (relative to the interneuron).

Excitatory local connections seem to be biased towards the iso-orientation domain. However, excitatory long-range horizontal connections target all orientation domains in a balanced manner, thus there is a strong crosstalk between all orientation domains. Note however that some of the targets of the long-range horizontal connections are excitatory cells, whereas some are inhibitory simple cells, since the network is correlation-based.

The excitatory long-range horizontal connections are mildly elongated along the orientation axis, most likely as a result of the stimulus configuration. During the learning phase the stimuli are contrast edges. Note that contrary to in layer 2/3, there has not been any report on axial specificity of the layer 4 long-range horizontal connections.

Nevertheless, we believe that the network behavior supports the existence of elongated summation pools in visual cortex, and gives a simple explanation for how it might be carried out within area 17.

This paper is a technical report.

5.4.4 Paper IV

A quantitative assessment of the layer 4 and layer 2/3 local and long-range horizontal connections based on two separate models is presented. The results show that layer 4 long-range horizontal connections target all orientation domains in a balanced manner, whereas layer 4 local connections are biased towards the iso-orientation domain.

The layer 2/3 network is significantly different. Both local and long-range horizontal connections of the layer 2/3 network are biased towards the iso-orientation domains. We hypothesize that the patchy layout of the long-range connections is a consequence of excitatory long-range connections targeting mainly other excitatory cells located in distal iso-orientation domains.

Furthermore, the fall-off with distance results in dense local and sparse distal connectivity for both networks. Preliminary results indicate that the layer 2/3 network, like the layer 4 network, can detect low contrast stimulus.

This paper was presented at the special session on ‘Biologically Inspired Computer Vision’, at 2nd CIRAS – Computational Intelligence, Robotics and Autonomous Systems in Singapore, December 2003.

6 General Conclusions

The motivation of this thesis work is to present a top-down view of the cat primary visual cortex (area 17) that can help to unveil the computations that are carried out within the neocortex. The primary visual cortex of cat and other species, such as monkey and rodents, are relatively well documented, and hence has been the principal system for studying the structure and function of cortical systems. The structural similarities between various cortical areas, which are primarily manifested by the modular and laminar organization of the neocortex, suggest that studies of the primary visual cortex might contribute to a general understanding of other cortical systems as well.

The top-down view helps to organize the enormous material that is available on the primary visual cortex. However, such an approach does not necessarily imply that the developed models are biologically plausible. Since the intention of this thesis work is to unveil the computations that are carried out within the neocortex, the developed models are faithful to the relevant aspects of known physiology and anatomy. However, a bottom-up manner of implementing the models with the top-down view has helped to construct biologically plausible models, which are also simple enough to allow reasoning.

The main contribution of this thesis work is the mapping of an abstract hypercolumn model to cat primary visual cortex (Papers II, III and IV). This highly general model could be mapped to both layer 4 and layer 2/3 local circuitry. The hypercolumn model shows that the recurrent (intracortical) connections can explain the emergence of several response properties of the striate cells. The uniqueness of this model is that it demonstrates response saturation (followed by normalization) together with contrast-invariance of orientation tuning. Narrowing of the orientation tuning is also demonstrated.

The layer 4 abstract hypercolumn model is based on the assumption that there exist two parallel inhibitory networks, which originate from two different classes of interneurons. (To our knowledge, existing models of layer 4 include only one class of inhibitory cells (Ferster and Miller, 2000; Martin, 2002).) These two inhibitory networks have different functional implications. In the first network the inhibition arises from a pool of interneurons, the connections of which are

uniformly distributed within an area that corresponds roughly to a hypercolumn. It is assumed that large basket cells are responsible for these inhibitory local connections, which are less specific for orientation than the excitatory local connections (Kisvárday et al., 1997; Yousef et al., 1999). Such an inhibitory network can efficiently control the total activity within a small patch, e.g. a hypercolumn, of the neocortex and thus explain response saturation followed by normalization, and also contrast-invariance of orientation tuning.

Narrowing of the orientation tuning is also demonstrated through the moderate reinforcement of the thalamic input by the excitatory local connections. However, this narrowing is only marginal, since the model does not assume unrealistically strong inhibitory synapses, such as many other recurrent models (Martin, 2002). Instead, the inhibitory simple cells (Hirch et al., 2000) achieve narrowing of the orientation tuning. It is assumed that these neurons target excitatory cells located in their close surroundings (Ferster, 1988). The inhibitory simple cells and their postsynaptic excitatory simple cell targets have opposite absolute (and relative) spatial phases relative to each other. Thus, anticorrelated excitatory cells can inhibit each other by targeting these inhibitory simple cells. The result is a correlation-based network, which narrows orientation tuning (Paper III). However, the layer 2/3 model lacks inhibitory simple cells. Otherwise the behavior of layer 2/3 model is similar to that of layer 4.

Furthermore, the layout and function of the intracortical connections within a patch of area 17 have been addressed using developmental network models (Papers III and IV). Such a cortical patch contains some tens of hypercolumns. More specifically, this study is based on the correlation-based models, which are generated by the BCPNN incremental learning rule using visual input.

A quantitative assessment of the excitatory intracortical connections within layer 4 and layer 2/3 developmental network models has been presented (Paper IV). This study reveals that the local and long-range connections of the developmental network models are similar to the known connectivity of the primary visual cortex (Kisvárday et al., 1997; Yousef et al., 1999).

Local connections are dense, whereas long-range horizontal connections are sparse. Furthermore, long-range horizontal connections are elongated along the orientation axis. Layer 4 long-range horizontal connections target all orientation domains in a balanced manner, whereas local connections of this layer are biased towards the iso-orientation domain. However, both local and long-range horizontal connections of layer 2/3 are biased towards the iso-orientation domains. It is hypothesized that the patchy layout of the long-range connections in layer 2/3 is a consequence of the excitatory long-range connections made by the pyramidal cells targeting mainly other pyramidal cells located in distal iso-orientation domains. As propose by Yousef et al. (1999) and others the differences between the circuitry of layers 4 and 2/3 might be an effect of their different functional roles.

The function of the intracortical connections has been investigated by studying response facilitation of the cortical cells (Paper III). This phenomenon is

manifested by improved visibility of a Gabor patch when it is elongated along the orientation axis (Polat and Norcia, 1998). Similar results have been reported in tree shrew primary visual cortex based on neurophysiological experiments (Chisum et al., 2003). As a result, elongated summation pools, which are hypothesized to be functionally linked to the long-range horizontal connections, have been proposed (Polat and Norcia, 1998; Chisum et al., 2003). The elongated shape of the long-range horizontal connections along the orientation axis (Papers III and IV) supports this belief.

Response facilitation requires robust communication between striate cells located several millimeters from each other. Spike and burst synchronization might be responsible for this. In Paper I it is shown that groups of striate cells (organized into orientation minicolumns) could indeed facilitate each other, through spike and burst synchronization, even tough they are located several millimeters from each other. The simulations do also show that response facilitation helps to narrow the orientation tuning of the striate cells. This might have a functional implication especially during low-contrast conditions. However, this hypothesis must be tested carefully before any conclusions can be drawn.

The main conclusion here is that several fundamental response properties of the striate cells are possible to explain by an abstract hypercolumn model, which is faithful to the known anatomy and physiology of the neocortex. When simplicity is combined with biological plausibility the models of hypercolumns can give valuable insight into the structure and function of cortical circuitry.

During this thesis work several additional questions have emerged that are closely related to this subject. In the near future the intention is to revisit the abstract hypercolumn model and address these questions. Firstly, a more careful study must be done on the different classes of inhibitory cells and their roles in emergence of various response properties. Furthermore, it is planned to present a quantitative assessment of the inhibitory connections, similar to the one on their excitatory counterparts. Finally, the behavior of the summation pools needs to be studied more thoroughly by simulation of activity during low-contrast conditions.

Bibliography

Adorján P, Levitt JB, Lund JS, Obermayer K (1999) A model for the intracorticla origin of orientation preference and tuning in macaque striate cortex. Vis. Neurosci. **16**:303–318.

Ahmet B, Anderson JC, Douglas RJ, Martin KA, Nelson JC (1994) Polyneural innervation of spiny stellate neurons in cat visual cortex. J. Comp. Neurol. **341**:39–49.

Albrecht DG, Hamilton DB (1982) Striate cortex of monkey and cat: contrast response function. J. Neurophysiol. **48**:217–237.

Albrecht DG, Geisler WS (1991) Motion selectivity and the contrast-response function of simple cells in the visual cortex. Visual Neurosci. **7**:531–546.

Albrecht DG, Geisler WS, Frazor RA, Crane AM (2001) Visual cortex neurons of monkeys and cats: temporal dynamics of the contrast response function. J. Neurophysiol. **88**:888–913.

Alonso J-, Martinez LM (1998) Functional connectivity between simple cells and complex cells in cat striate cortex. Nat. Neurosci. **1**:395–403.

Alonso J-M, Usrey WM, Reid RC (1996) Precisely correlated firing in cells of the lateral geniculate nucleus. Nature **383**:815–819.

Amir Y, Harel M, Malach R (1993) Cortical hierarchy reflected in the organization of intrinsic connections in macaque monkey visual cortex. J. Comp. Neurol. **334**:19–46.

Baxter WT, Dow BM (1989) Horizontal organization of orientation-sensitive cells in primate visual cortex. Biol. Cybern. **61**:171–182.

Bayes T (1958) An essay towards solving a problem in the doctrine of changes. Biometrica **45**:296–315 (reprint of original article in Phil. Trans. R. Soc. **53**:370–418, 1763).

Ben-Yishai R, Bar-Or RL, Sompolinsky H (1995) Theory of orientation tuning in visual cortex. Proc. Natl. Acad. Sci. USA **92**:3844–3848.

Ben-Yishai R, Hansel D, Sompolinsky H (1997) Traveling waves and the processing of weakly tuned inputs in a cortical network module. J. Comput. Neurosci. **4**: 57–77.

Blasdel GG (1992) Orientation selectivity, preference, and continuity in monkey striate cortex. J. Neurosci. **12**:3139–3161.

Blasdel GG, Fitzpatrick D. 1984. Physiological organization of layer 4 in macaque striate cortex. J. Neurosci. **4**:880–95.

Blasdel GG, Salama G (1986) Voltage-sensitive dye reveal a modular organization in monkey striate cortex. Nature **321**:579–585.

Bonds AB (1989) The role of inhibition in the specification of orientation selectivity of cells in the cat striate cortex. Visual Neurosci. **2**:41–55.

Bosking WH, Zhang Y, Schofield B, Fitzpatrick D (1997) Orientation selectivity and the arrangement of horizontal connections in tree shrew striate cortex. J. Neurosci. **17**:2112–2127.

Boycott BB, Wässle H (1974) The morphological types of ganglion cells of the domestic cat's retina. J. Physiol. **240**: 397–419.

Braitenberg V, Braitenberg C (1979) Geometry of orientation columns in the visual cortex. Biol. Cybern. **33**:179–186.

Braitenberg V, Schüz A (1998) Cortex: statistics and geometry of neuronal connectivity. Springer.

Buxhoeveden DP, Casanova MF (2002) The minicolumn hypothesis in neuroscience. Brain **125**:935–951.

Campbell FW, Cooper GF, Enroth-Cyrell C (1969) The spatial selectivity of the visual cells of the cat J. Physiol. **203**:223–235.

Carandini M, Heeger DJ (1994) Summation and division by neurons in primate visual cortex. Science **264**:1333–1336.

Carandini M, Heeger DJ, Movshon JA (1997) Linearity and normalization in simple cells of the macaque primary visual cortex. J. Neurosci. **17**:8621–8644.

Chapman B, Stryker MP (1993) Development of orientation selectivity in ferret visual cortex and effects of deprivation. J. Neurosci. **13**:5251–62.

Chisum HJ, Mooser F, Fitzpatrick D (2003) Emergent properties of layer 2/3 neurons reflect the collinear arrangement of horizontal connections in tree shrew visual cortex. J. Neurosci. **23**:2947–2960.

Chung S, Ferster D (1998) Strength and orientation tuning of the thalamic input to simple cells revealed by electrically evoked cortical suppression. Neuron **20**:1177–1189.

Coenen AML, Vendrik AJH (1972) Determination of the transfer ratio of cat's geniculate neurons through quasi-intracellular recordings and the relation with the level of alertness. Exp. Brain Res. **14**:227–242.

Connors BW, Bear MF, Paradiso MA (2002) Neuroscience: exploring the brain. Lippincott Williams and Wilkins Publishers.

Das A (1996) Orientation in visual cortex: a simple mechanism emerges. Neuron **16**:447–480.

Dean AF (1981) The relationship between response amplitude and contrast for cat striate cortical neurons. J. Physiol. **318**:413–27.

DeAngelis GC, Geoffrey MG, Ohwaza I, Freeman RD (1999) Functional micro-organization of primary visual cortex: receptive field analysis of nearby neurons. J. Neurosci. **19**:4046–4064.

Derrington AM, Fuchs AF (1979) Spatial and temporal properties of X and Y cells in the cat lateral geniculate nucleus. J. Physiol. **293**:347–364.

Derrington AM, Lennie P (1984) Spatial and temporal contrast sensivities of neurons in lateral geniculate nucleus of macaque. J. Physiol. (Lond) **357**:219–240.

Dow BM (2002) Orientation and color columns in monkey visual cortex. Cerebral Cortex **12**:1005–1015.

Dreher B, Fukada Y, Rodieck RW (1976) Identification, classification and anatomical segregation of cells with X-like and Y-like properties in the lateral geniculate nucleus of old-world primates. J. Physiol. (Lond) **258**:433–452.

Durack JC, Katz LC (1996) Development of horizontal projections in layer 2/3 of ferret visual cortex. Cerebral Cortex **6**:178–183.

Engel AK, König P, Kreiter AK, Singer W (1991) Inter-hemispheric synchronization of oscillatory neuronal responses in cat visual cortex. Science **252**:1177–1179.

Engel AK, Roelfsema PR, Fries P, Brecht M, Singer W (1997) Role of the temporal domain for response selection and perceptual binding. Cerebral Cortex **7**:571–582.

Ernst UA, Mandon S, Pawelzik KR, Kreiter AK (2001) How ideal do macaque monkeys integrate contours? Submitted to Elsevier Science.

Erwin E, Obermayer K, Schulten K (1995) Models of orientation and ocular dominance columns in the visual cortex: a critical comparison. Neural Comput. **7**:425–468.

Everson RM, Prashanth AK, Gabbay M, Knight BW, Sirovich L, Kaplan E (1998) Representation of spatial frequency and orientation in the visual cortex. Vision Res. **11**:251–259.

Favorov OV, Diamond ME (1990) Demonstration of discrete place―defined columns-segregates-in the cat SI. J. Comp. Neurol. **298**:97–112.

Ferster D (1988) Spatially opponent excitation and inhibition in simple cells of the cat visual cortex. J. Neurosci. **8**:1172–1180.

Ferster D, Chung S, Wheat H (1996) Orientation selectivity of thalamic input to simple cells of cat visual cortex. Nature **380**:249–252.

Ferster D, Miller KD (2000) Neural mechanisms of orientation selectivity in the visual cortex. Annual Reviews of Neurosci. **23**:441–471.

Fitzpatrick D (1996) The functional organization of local circuits in visual cortex: insights from the study of tree shrew striate cortex. Cerebral Cortex **6**:329–341.

Friedman-Hill S, Maldonado PE, Gray CM (2000) Dynamics of striate cortical activity in the alert macaque: I. incidence and stimulus-dependence of gamma-band neuronal oscillations. Cerebral Cortex **10**:1105–1116.

Gabbott PLA, Somogyi P (1986) Quantitative distribution of GABA-immunoreactive neurons in the visual cortex (area 17) of the cat. Exp. Brain Res. **61**:323–331.

Gil Z, Connors BW, Amitai Y (1999) Efficacy of thalamocortical and intracortical synaptic connections: quanta, innervation, and reliability. Neuron **23**:385–397.

Gilbert CD, Wiesel TN (1983) Clustered intrinsic connections in cat visual cortex. J. Neurosci. **3**:1116–1133.

Gilbert CD, Wiesel TN (1989) Columnar specificity of intrinsic horizontal connections and corticocortical connections in cat visual cortex. J. Neurosci. **9**:2432–2442.

Good IJ (1950) Probability and the weighing of evidence. London: Charles Griffin.

Gotz KG (1987) Do “d-blob” and “l-blob” hypercolumns tessellate the monkey visual cortex? Biol. Cybern. **56**:107–109.

Gotz KG (1988) Cortical templates for the self-organization of orientation-specific d- and l-hypercolumns in monkey and cats. Biol. Cybern. **58**:213–223.

Gray CM, Koenig P, Engel AK, Singer W (1989) Oscillatory responses in cat visual cortex exhibit inter-columnar synchronization which reflects global stimulus properties. Nature **338**:334–337.

Gray CM, Singer W (1989) Stimulus-specific neuronal oscillations in orientation columns of cat visual cortex. **86**:1698–1702.

Grossberg S, Raizada RDS (2000) Contrast-sensitive perceptual grouping and object-based attention in the laminar circuits of primary visual cortex. Vision Res. **40**:1413–1432.

Haig AR, Gordon E, Wright JJ, Meares RA, Bahramali H (2000) Synchronous cortical gamma-band activity in task-relevant cognition. Neuroreport **11**:669–675.

Hansel D, Sompolinsky H (1996) Chaos and synchrony in a model of a hypercolumn in visual cortex. J. Comput. Neurosci. **3**:7–34.

Hawken M, Parker AJ (1984) Contrast sensitivity and orientation selectivity in lamina IV of the striate cortex of old world monkeys. Exp. Brain Res. **54**:367–72.

Hebb DO (1949) The organization of behavior. New York: Wiley.

Heeger DJ (1992) Normalization of cell responses in cat striate cortex. Visual Neuroscience **9**:181–197.

Heeger DJ, Simoncelli EP, Movshon JA (1996) Computational models of cortical visual processing. Proc. Natl. Acad. Sci. USA **93**:623–627.

Hellwig B (2000) A quantitative analysis of the local connectivity between pyramidal neurons in layer 2/3 of the rat visual cortex. Biol. Cybern. **82**:111–121.

Hendrickson AE, Hunt SP, Wu J-Y (1981) Immunocytochemical localization of glutamic acid decarboxylase in monkey striate cortex. Nature **292**:605–607.

Hirch JA, Alonso J-M, Pillai C, Pierre C (2000) Simple and complex inhibitory cells in layer 4 of cat visual cortex. Soc. Neurosci. Abstr. **26**:1083.

Holst A (1997) The use of a Bayesian neural network model for classification tasks. PhD Thesis Department of Numerical Analysis and Computing Science, Royal Institute of Science, Stockholm, Sweden, TRITA-NA-P9708.

Horton JC (1984) Cytochrome oxidase patches: a new cytoarchitectonic feature of monkey visual cortex. Phil. Trans. R. Soc. Lond. B **304**:199–253.

Horton JC, Hubel DH (1981) Regular patchy distribution of cytochrome oxidase staining in primary visual cortex of macaque monkey. Nature **292**:762–764.

Hubel DH (1988) Eye, brain, and vision. New York: WH Freeman.

Hubel DH, Wiesel TN (1962) Receptive fields, binocular interaction and functional architecture in the cat's visual cortex. J. Physiol. **160**:106–154.

Hubel DH, Wiesel TN (1972) Laminar and columnar distribution of geniculo-cortical fibers in the macaque monkey. J. Comp. Neurol. **146**:421–450.

Hubel DH, Wiesel TN (1977) The functional architecture of the macaque visual cortex. The Ferrier lecture. Proc. Royal. Soc. B **198**:1–59.

Hübener M, Schulze S, Bonhoeffer T (1996) Cytochrome-oxidase blobs in cat visual cortex coincide with low spatial frequncy columns. Soc. Neurosci. Abstr. **22**:951.

Humphrey AL, Norton TT (1980) Topographic organization of the orientation column system in the striate cortex of the tree shrew (Tupaia glis). I. Microelectrode recording. J. Comp. Neurol. **192**:531–47.

Issa NP, Trepel C, Stryker MP (2000) Spatial frequency maps in cat visual cortex. J. Neurosci. **20**:8504–8514.

Kandel ER, Schwartz JH, Jessell TM (eds.) (2000) Principals of neural science. Graham-Hill.

Kastner S, Nothdurft H-C, Pigarev IN (1997) Neuronal correlates of pop-out in cat striate cortex. Vision Res. **37**:371–376.

Katz LC (1987) Local circuitry of identified projection neurons in cat visual cortex. J. Neurosci. **7**: 1223–1249.

Kayser AS, Miller KD (2002) Opponent inhibition: a development model of layer 4 of the neocotical circuit. Neuron **33**:131–142.

Kisvárday ZF, Martin KAC, Freund TF, Magloczky Z, Whitteridge D, Somogyi P (1986) Synaptic targets of HRP-filled layer III pyramidal cells in the cat striate cortex. Exp. Brain Res. **64**: 541–552.

Kisvárday ZF, Martin KAC, Friedlander MJ, Somogyi P (1987) Evidence for interlaminar inhibitory circuits in the striate cortex of cat J. Comp. Neurol. **260**:1–19.

Kisvárday ZF, Tóth E, Rausch M, Eysel UT (1997) Orientation-specific relationship between populations of excitatory and inhibitory lateral connections in the visual cortex of the cat. Cerebral Cortex **7**:605–618.

Koffka K (1935) Principals of Gestalt psychology. New York: Harcourt, Brace and World.

Köhler W (1930) Gestalt psychology. London: Bell and Sons.

König P, Engel AK, Löwel S, Singer W (1993) Squint affects synchronization of oscillatory responses in cat visual cortex. Eur. J. Neurosci. **5**:501–508.

Kreiter AK, Singer W (1996) Stimulus-dependent synchronization of beuronal responses in the visual cortex of awake macaque monkey. J. Neurosci. **16**:2381–2396.

Krimer LS, Goldman-Rakic PS (2001) Prefrontal mirocircuits: membrane properties and excitatory input of local, medium, wide arbour interneurons. J. Neurosci. **21**:3788–3796.

Kuffler SW (1953) Discharge patterns and functional organization of mammalian retina. J. Neurophysiol **16**:37–68.

Lansner A, Ekeberg Ö (1987) An associative network solving the "4-bit adder problem". In: Caudill M, Butler C (editors). IEEE First International Conference on Neural Networks. Pp II–549.

Lansner A, Ekeberg Ö (1989) A one-layer feedback artificial neural network with a bayesian learning rule. Int. J. Neural Syst. **1**:77–78.

Lansner A, Holst A (1996) A higher order bayesian neural network with spiking units. Int. J. Neural Syst. **7**:115–128

Li Z (1998) A neural model of contour integration in the primary visual cortex. Neural Computation. **10**:903–940.

Löwel S, Singer W (1992) Selection of intrinsic horizontal connections in the visual cortex by correlated neuronal activity. Science **255**:209–212.

McLaughin D, Shapley R, Shelley M, Wielaard DJ (2000) A neuronal network model of macaque primary visual cortex (V1): orientation selectivity and dynamics in the input layer 4Cα. Proc. Natl. Acad. Sci. USA **97**:8087–8092.

Maffei L, Fiorentini A (1973) The visual cortex as a spatial frequency analyser. Vis. Res. **13**:1255–1267.

Maffei L, Fiorentini A, Bisti S (1973) Neural correlate of perceptual adaptation to gratings. Science **182**:1036–38.

Malach R, Amir Y, Harel M, Grinvald A (1993) Relationship between intrinsic connections and functional architecture revealed by optical imagining and in vivo targeted biocytin injections in primate striate cortex. Proc. Natl. Acad. Sci. USA **90**:10496–10473.

Maldonado PE, Friedman-Hill S, Gray CM (2000) Dynamics of striate cortical activity in the alert macaque: II. Fast time scale synchronization. Cerebral Cortex **10**:1117–1131.

Martin KAC (2002) Microcircuits in visual cortex. Current Opinion in Neurobiology **12**:418–425.

Meyer G (1983) Axonal patterns and topography of short-axon neurons in visual areas 17, 18 and 19 of the cat . J. Comp. Neurol. **220**:405–438.

Meyer G (1987) Forms and spatial arrangement of neurons in the primary motor cortex of man. J. Comp. Neurosci. **262**:402–428.

Morrone MC, Burr DC, Maffei L (1982) Functional implications of cross-orientation inhibition of cortical visual cells. I. Neurophysiological evidence. Proc. R. Soc London Ser. B **216**:335–354.

Mountcastle VB (1957) Modality and topographic properties of single neurons of cat's somatic sensory cortex. J. Neurophysiol. **20**:408–438.

Mountcastle VB (1978) An organizing principle for cerebral function. In: Edelman GM, Mountcastle VB (editors). The mindful brain. MIT Press 7–50.

Mountcastle VB (1997) The columnar organization of the neocortex. Brain **120**:701–722.

Movshon JA, Hawken MJ, Kiorpes L, Skoczenski AM, Tang C, O'Keefe LP (1994) Visual noise masking in macaque LGN neurons. Invest. Ophthalmol. Vis. Sci. [Suppl] **35**:1662.

Murphy PC, Sillito AM (1986) Continuity of orientation columns between superficial and deep laminea of cat primary visual cortex. J. Physiol. **381**:95–110.

Obermayer K, Blasdel GG (1993) Geometry of orientation and ocular dominance columns in monkey striate cortex. J. Neurosci. **13**:4114–4129.

O'Leary JL (1941) The structure of area striata of cat. J. Comp. Neurol. **75**:131–164.

Palmer SE (1999) Vision science. MIT Press. pp 726.

Payne BR, Peters A (2002) The concept of cat primary visual cortex. in: BR Payne, A Peters (ed.). the cat primary visual cortex. Academic Press.

Pei X, Vidyasagar TR, Volgushev M, Creutzfeldt OD (1994) Receptive field analysis and orientation selectivity of postsynaptic potentials of simple cells in cat visual cortex. J. Neurosci. **14**:7130–40.

Peters A, Sethares C (1996) Myelinated axons and the pyramidal cell modules in monkey visual cortex. J. Comp. Neurol. **365**:232–255.

Peters A, Yilmaz E (1993) Neuronal organization of area 17 of cat visual cortex. Cerebral Cortex **3**:49–68.

Polat U, Norcia AM (1996) Neurophysiological evidence for contrast dependent long-range facilitation and suppression in the human visual cortex. Vision Res. **36**: 2099–2109.

Polat U, Norcia AM (1998) Elongated physiological summation pools in the human visual cortex. Vision Research **38**:3735–3741.

Polat U, Sagi D (1993) Lateral interactions between spatial channels: Suppression and facilitation revealed by masking experiments. Vision Res. **33**:993–999.

Powell TPS, Mountcastle VB (1959) Some aspects of the functional organization of the cortex of the postcentral gyrus of the monkey: a correlation of findings obtained in a single unit analysis with cytoarchitecture. Bull Johns Hopkins Hosp. **105**:133–162.

Purves D, Augustine GJ, Fitzpatrick D, Katz LC, LaMantia A-S, McNamara JO (eds.) (1997) Neuroscience. Sunauer Associates Inc. pp 1–34.

Reid RC, Alonso JM (1995) Specificity of monosynaptic connections from thalamus to visual cortex. Nature **378**:281–284.

Rockland KS (1985) Anatomical organization of primary visual cortex (area 17) in the ferret. J. Comp. Neurol. **241**:225–236.

Rockland KS, Lund JS (1982) Widespread periodic intrinsic connections in the tree shrew visual cortex. Science **215**:1532–1534.

Rockland KS, Lund JS (1983) Intrinsic laminar lattice connections in primate visual cortex. J. Comp. Neurol. **216**:303–318.

Sandberg A (2003) Bayesian attractor neural networks of memory. PhD Thesis Department of Numerical Analysis and Computing Science, Royal Institute of Science, Stockholm, Sweden, TRITA-NA-0310.

Sandberg A, Lansner A, Petersson FM, Ekeberg Ö (2002) A Bayesian attractor network with incremental learning. Network: Computing in Neural Systems. **13**:179–194.

Schmidt KE, Kim D-S, Singer W, Bonhoeffer T, Löwel S (1997) Functional specificity of long-range intrinsic and interhemisphic connections in the visual cortex of strabismic Cats. J. Neurosci. **17**:5480–5492.

Schmidt KE, Löwel S (2002) Long-range intristic connections in cat primary visual cortex. in: Payne BR, Peters A (ed.). The cat primary visual cortex. Academic Press.

Sclar G, Freeman RD (1982) Orientation selectivity in the cat's striate cortex is invariant with stimulus contrast. Exp. Brain Res. **46**:457–461.

Shandlen MN, Movshon JA (1999) Synchrony unbound: a critical evaluation of the temporal binding hypothesis. Neuron **24**:67–77.

Sherman SM, Schumer RA, Movshon JA (1984) Functional cell classes in the macaque's LGN. Soc. Neurosci. Abstr. **10**:296.

Shoham D, Hübener M, Schulze S, Grinvald A, Bonhoeffer T (1997) Spatio-temporal frequency domains and their relation to cytochrome oxidase staining in cat visual cortex. Nature 385: 529–533.

Singer W (1993) Synchronization of cortical activity and its putative role in information processing and learning. Annu. Rev. Physiol. **55:**349–374.

Singer W (1999) Neuronal synchrony: a versatile code for the definition of relations? Neuron **24**:49–65.

Singer W, Gray CM (1995) Visual feature integration and the temporal correlation hypothesis. Annual Review of Neurosci. **18**:555–586.

Skottun BC, Bradley A, Sclar G, Ohzawa I, Freeman RD (1987) The effects of contrast on visual orientation and spatial frequency discrimination: a comparison of single cells and behaviour. J. Neurophysiology **57**:773–86.

Solomon JA, Watson AB, Morgan MJ (1999) Transducer model produces facilitation from opposite-sign flanks. Vision Res. **39**: 987–992.

Solomon JA, Morgan MJ (2000) Facilitation from collinear flanks is cancelled by non-collinear flanks. Vision Res. **40**: 279–286.

Somers D, Dragoi V, Sur M (2002) Orientation selectivity and its modulation by local and long-range connections in visual cortex. in: Payne BR, Peters A (ed.). The cat primary visual cortex. Academic Press.

Somers D, Nelson SB, Sur M (1995) An emergent model of orientation selectivity in cat visual cortical simple cells. J. Neurosci. **15**:5448–5465.

Somers D, Todorov EV, Siapas AG, Toth LJ, Kim D-S, Sru M (1998) A local circuit approach to understand integration of long-range inputs in primary visual cortex. Cerebral Cortex **8**:204–217.

Sompolinsky H, Shapley R (1997) New percpectives on the mechanisms for orientation selectivity. Current Opinion in Neurobiology **7**:514–522.

Steriade M, McCormic DA, Sejnowski TJ (1993) Thalamocortical oscilation in the sleeping and aroused brain. Science **262**:679–685.

Stevens CF, Zador AM (1998) Input synchrony and the irregular firing of cortical neurons. Nat. Neurosci. **1**:210–217.

Swindale NV (1982) A model for the formation of orientation columns. Proc R Soc Lond B **215**:211–230.

Tootell RBH, Silverman MS, De Valois RL (1981) Spatial frequency columns in primary visual cortex. Science **214**:813–815.

Troyer TW, Krukowski AE, Miller KD (1998) Contrast-invariant orientation tuning in cat visual cortex: thalamocortical input tuning and correlation-based intracortical connectivity. J. Neurosci. **18**:5908–27.

Troyer TW, Krukowski AE, Miller KD (2002) LGN input to simple cells and contrast-invariant orientation tuning: an analysis. J. Neurophysiol. **87**:2741–2752.

Tömböl T (1978) Comparative data on the Golgi architecture of interneurons of different cortical areas in cat and rabbit. in: Brazier MAB, Petsche H (eds.) Architectonics of the Cerebral Cortex. pp. 59–76. New York. Raven Press.

Usrey WM, Reid RC (1999) Synchronous activity in the visual system. Annu. Rev. Physiol. **61**:435–456.

Wielaard DJ, Shelley M, McLaughin D, Shapley R (2001) How simple cells are made in a nonlinear network model of the visual cortex. J. Neurosci. **21**:5203–5211.

Williams LR, Thornber KK (2001) Orientation, scale, and discontinuity as emergent properties of illusory contour shape. Neural Computing. **13**:1683–1711.

Wong-Riley MTT (1979) Changes in the visual system of monocularity sutured or enucleated kittens demonstrable with cytochrome oxidase histochemistry. Brain Res. **171**:11–28.

Wässle H, Levick WR, Cleland BG (1975) The distribution of the alpha type of ganglion cells in the cat's retina. J Comp. Neurol. **159**:419–438.

Woolsey CN, Waltz EM (1942) Topical projection of nerve fibres from local regions of the cochlea to the cerebral cortex of the cat. Bull Johns Hopkins Hosp. **71**:315–344.

Yen S-C, Finkel LH (1998) Extraction of perceptually salient contours by striate cortical networks. Vision Res. **38**:719–741.

Yousef T, Bonhoeffer T, Kim D-S, Eysel UT, Tóth E, Kisvárday ZF (1999) Orientation topography of layer 4 lateral networks revealed by optical imagining in cat visual cortex (area 18). E. J. Neurosci. **11**:4291–4308.

Yu C, Klein SA, Levi DM (2002) Facilitation of contrast detection by cross-oriented surround stimuli and its psychophysical mechanisms. J. Vision. **2**:243–255.

7 Paper I – Spike and Burst Synchronization in a Detailed Cortical Network Model with I-F Neurons

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Abstract

Previous studies have suggested that synchronized firing is a prominent feature of cortical processing. Simplified network models have replicated such phenomena. Here we study to what extent these results are robust when more biological detail is introduced. A biologically plausible network model of layer II/III of tree shrew primary visual cortex with a columnar architecture and realistic values on unit adaptation, connectivity patterns, axonal delays and synaptic strengths was investigated. A drifting grating stimulus provided afferent noisy input. It is demonstrated that under certain conditions, spike and burst synchronized activity between neurons, situated in different minicolumns, may occur.

7.1 Introduction

The synchronized activity of the neurons in visual cortex is believed to contribute to perceptual grouping [14,20,21,22,24,28], see also [23]. It is also assumed that neurons in primary visual cortex have small and overlapping receptive field [8]. We assume also that local cortical connectivity (<300 μm) is dense [7]. Dense local connectivity should help neurons to synchronize their activities. This should mean that neurons with same or contiguous receptive fields are active in presence of stimulus.

Studies have shown evidence for long-range horizontal connections in primary visual cortex [10,13]. Recently Bosking et al. also showed evidence for modular and axial specificity of long-range horizontal connections in layer II/III, and suggested that these connections could help neurons respond to a stimulus, in part because they receive input from other layer II/III neurons [11].

We suggest that long-range horizontal connections that exist in layer II/III together with local connections can be used by the neurons for synchronization of their activities over distances of several millimeters on cortex surface.

This follows the same ideas as earlier network model simulations where horizontal connections [16,17,19,25, 27] and synchronization [15,18,26] play an important role.

7.2 Network Model

We have built a biologically plausible, but sub-sampled, network model. The network consists of neurons situated in six cortical minicolumns (orientation columns), having the same orientation preference. The minicolumns were lined up with a distance of 0.5 mm between two successive ones. We assume that minicolumns are co-linearly positioned in adjacent hyper-columns [8]. The cylinder shaped minicolumns had a height of 300 μm [7] and diameter of 50 μm [8].

Each of the six minicolumns was composed of 12 layer II/III pyramid cells. The neurons were positioned stochastically to fill up the volume of a minicolumn. Connection probability between two neurons was a function of the distance between them [7]. This resulted in a very spread connection probability of 15-80% for neuron pairs, and led to a connectivity of 50-60% between neurons inside a minicolumn [8].

Long-range horizontal connections were defined as connection between two neurons situated in different minicolumns. We computed the connection probability between such pairs of neurons by extrapolating the reported connection probabilities (<500 μm) by Hellwig [7] so that they fitted the findings by Bosking et al. [11]. This resulted in a smooth transition between local and long-range connection probabilities. In average a neuron received 6.2 intra-columnar inputs and 2.1 inter-columnar inputs.

We implemented a leaky Integrate-and-Fire model neuron with noise [1,2]. It was modified to allow adaptation of the membrane time constant [3]. Adaptation is very crucial for the dynamics because of the fact that our network, as many others, does not have inhibitory interneurons. The neuron population was heterogeneous with all values sampled from a uniform distribution with a deviation of 10%.

An axonal diameter of 0.3 μm [8] resulted in a spike propagation velocity of 0.85 m/s [5]. This value together with distances between neurons, and the synaptic delay, resulted in maximum delay inside a minicolumn of approximately 1.36 ms. Maximum delay between neurons situated in two minicolumns was approximately 3.96 ms. This delay corresponds to a distance of approximately 2.52 mm. Maximum values of EPSP:s were in the range of 0.5-2.2 mV for intra-columnar connections and three times those values for inter-columnar connections [4]. The simulation time step was 0.5 ms.

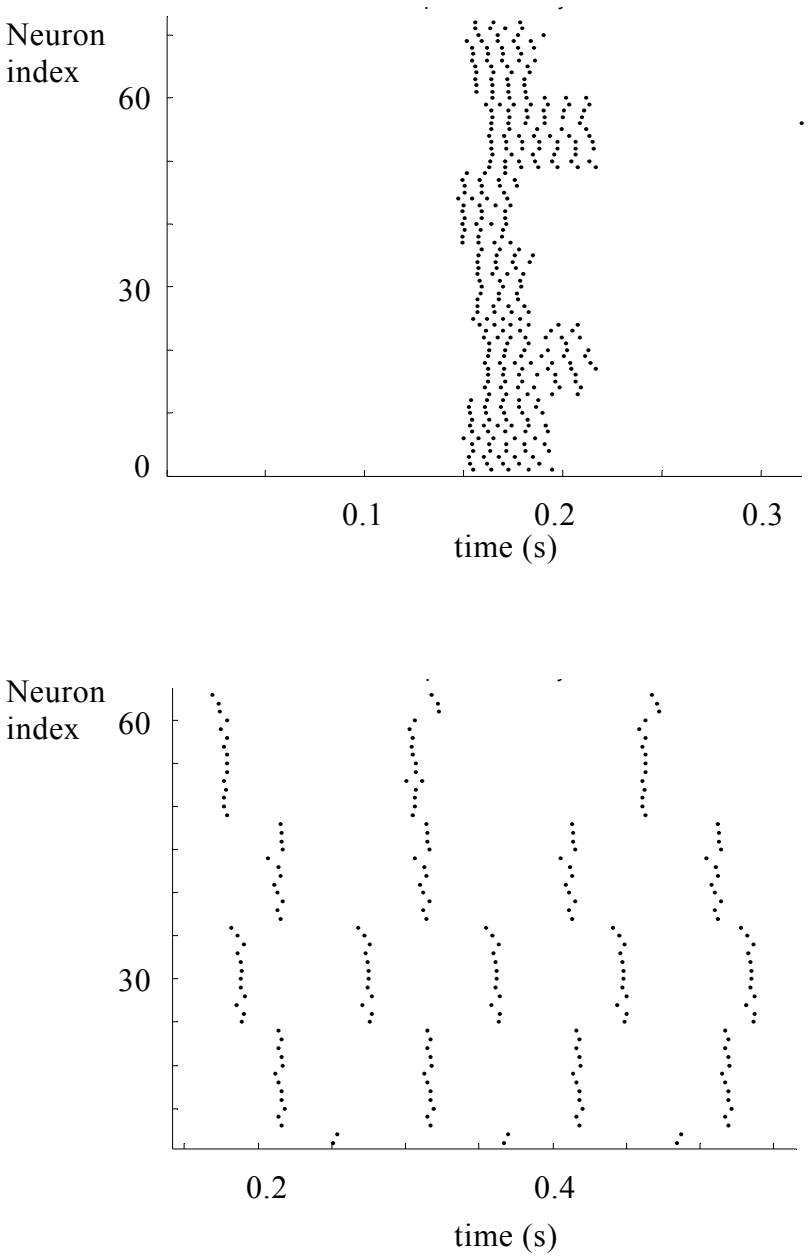

Figure 7.1. Population activity in the absent of long-range horizontal connections. Poisson spike train input (*above*), Constant current input (*below*).

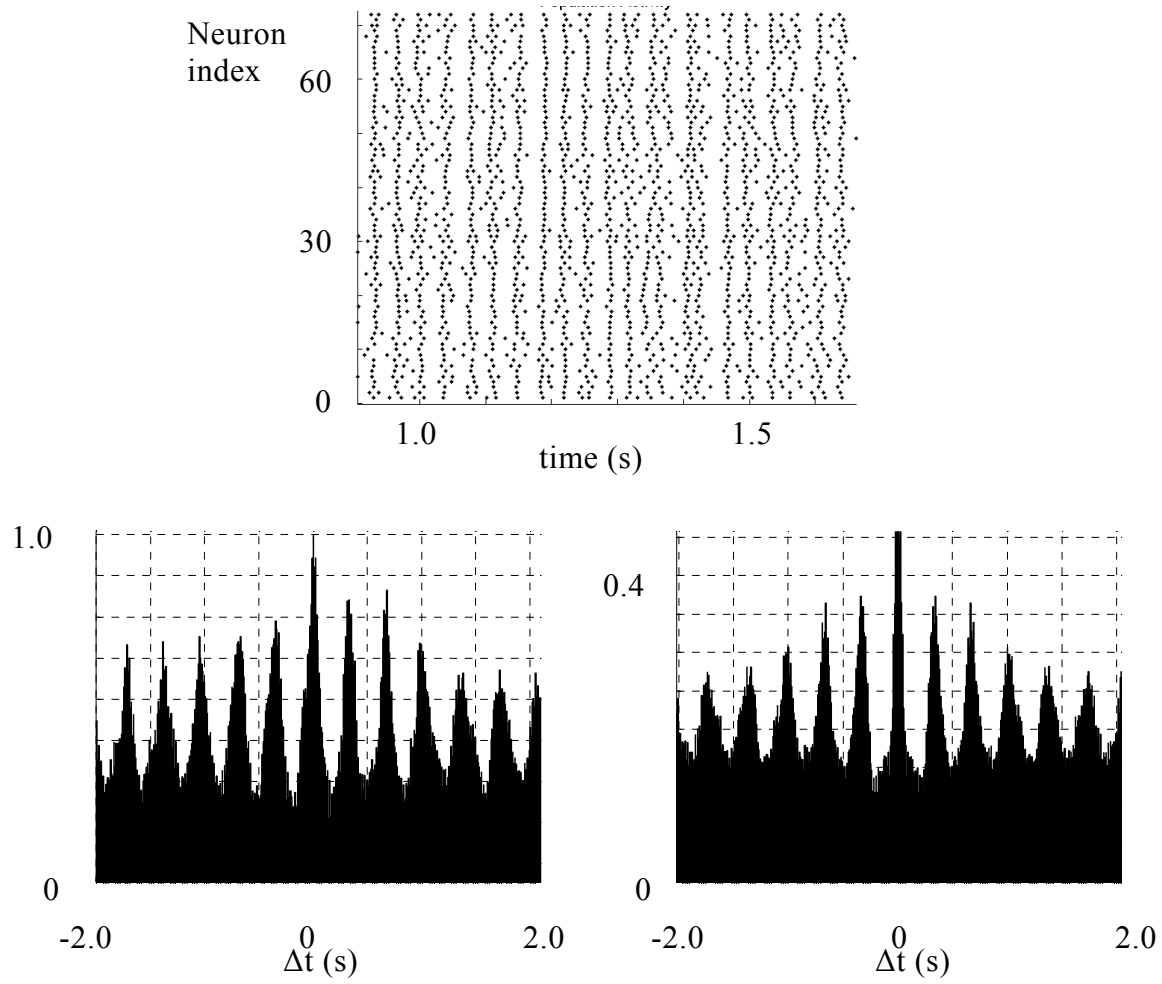

Figure 7.2. Population activity (*top*), and cross-correlation (*bottom left*) between minicolumn one (neurons 1-12) and six (neurons 61-72). Auto-correlation on minicolumn six (*bottom right*).

7.3 Simulation Results

We did three simulations series to demonstrate the influence of long-range horizontal connections on spike and burst synchronization. The initial simulation was done to show that, for our network, synchronization between neurons situated in different minicolumns is not possible, in the absent of the long-range horizontal connections. The network was tested on two different types of inputs. A drifting grating stimulus, modeled as a constant current [6], and a moving bar stimulus, modeled as a Poisson spike train with maximum frequency defined in a Gaussian

manner [6]. We saw that in the absence of long-range horizontal connections, the neurons in different minicolumns were not synchronized with each other (Fig. 7.1). Although the response of the network to the two inputs was different, spike synchronization between neurons inside a minicolumn was present in both cases, especially for constant current input.

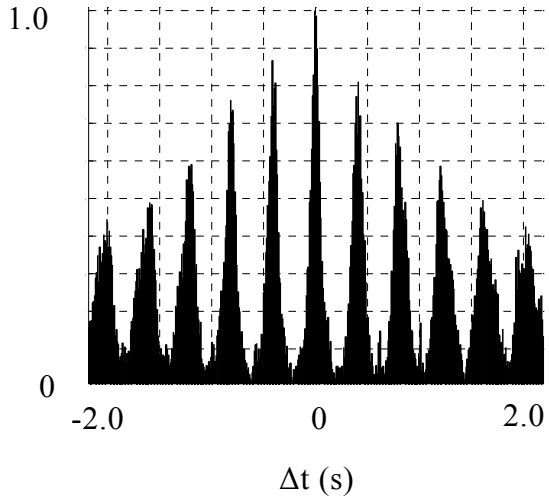

Figure 7.3. Cross-correlation between minicolumn one and six. Neurons in the two middle minicolumns were not receiving current input, and were not spiking throughout the simulation. Oscillation frequency is 25 Hz.

The following two simulations were done with the same two input types as described above, and in the presence of long-range horizontal connections. A drifting grating stimulus (constant current) resulted in both spike and burst synchronization for different values on input currents. In the first part of this simulation all the neurons received constant current. Some of the cells displayed repetitive bursting, for higher values of input current, synaptic weights or more dense connections. Spike synchronization as well as burst synchronization could be seen (Fig. 7.2). It is assume that the repetitive bursting behavior contributes to synchronous oscillation of the population [12]. But this behavior destroyed high precision spike synchronization (Fig. 7.2). In the second part of this simulation we fed only neurons in minicolumns one, two, five and six with constant current to see if neurons in minicolumns one and six still were correlated with each other. Observe that there were no direct connections between minicolumns one and six during this particular simulation, and neurons in the two middle minicolumns were not spiking (not shown here). Spike synchronization between neurons in minicolumn one and six was however present (Fig. 7.3). There was a tendency for a lag of a couple of milliseconds if synaptic weights were low or connections were sparse (not shown here). Observe that the shortest possible delay between two

neurons situated in minicolumn one and six was 2.75 ms. Gamma-band oscillation (20-70Hz) could be generated with different values on current input, and we assumed that oscillation was a property of the network.

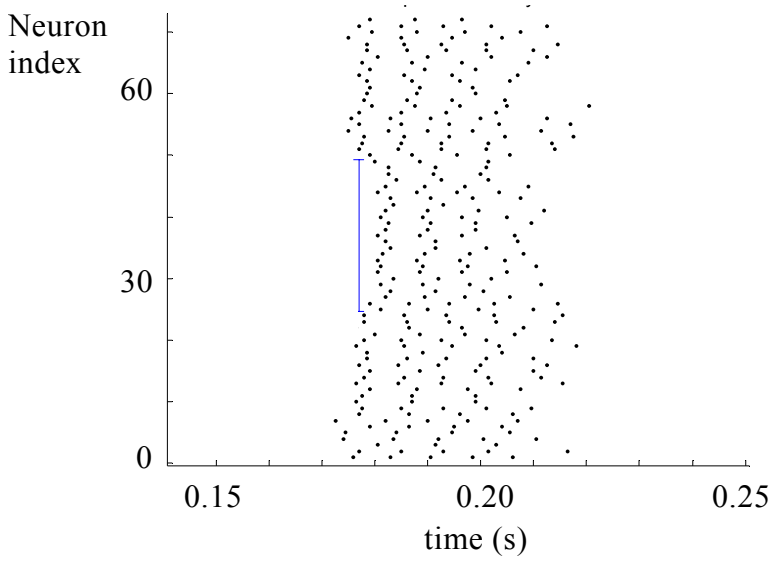

Figure 7.4. Population activity. Notice how minicolumns three and four lagged behind.

The intention of the moving *one* bar-stimulus simulation was to show that, it is possible for neurons to respond to a stimulus that they are not receiving directly. In this simulation all neurons received constant current input. However this input was not sufficient for generating a spike, and corresponded to lowering of the thresholds. Neurons in minicolumns one, two, five and six received uncorrelated Poisson spike trains for a short period of time (80-100 ms) as described above. Poisson spike EPSP:s were in the range of 0.05mV. On average neurons received 65 of these spikes. We saw that neurons in minicolumns three and four were activated by other neurons, with a lag of approximately 4 ms (Fig. 7.4 and 7.5).

High precision spike synchronization was present only during the first two spikes (Fig. 7.4). With lower values on Poisson spike EPSP:s or larger deviation for the Gaussian distribution high precision spike synchronization could be achieved for longer durations (not shown here). However we could see that burst synchronization and oscillation was present (Fig. 7.6).

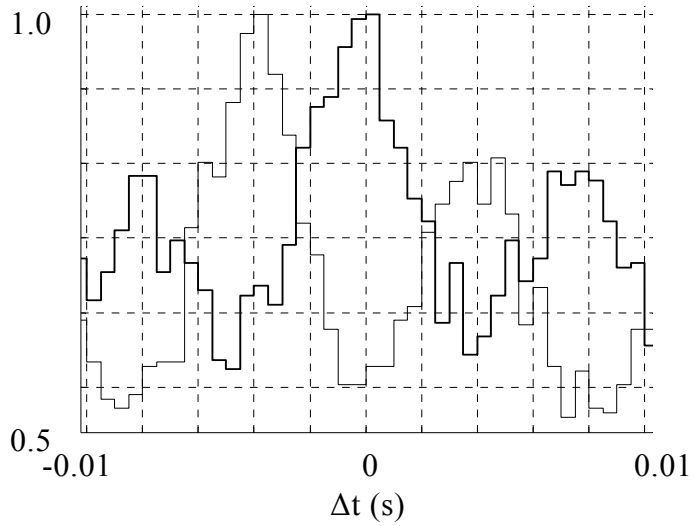

Figure 7.5. Cross-correlation between neurons 1-24 and 49-72 (*thick line*). Cross-correlation between neurons 1-24 and 25-48 (*thin line*) for the trial shown in Fig. 7.3.

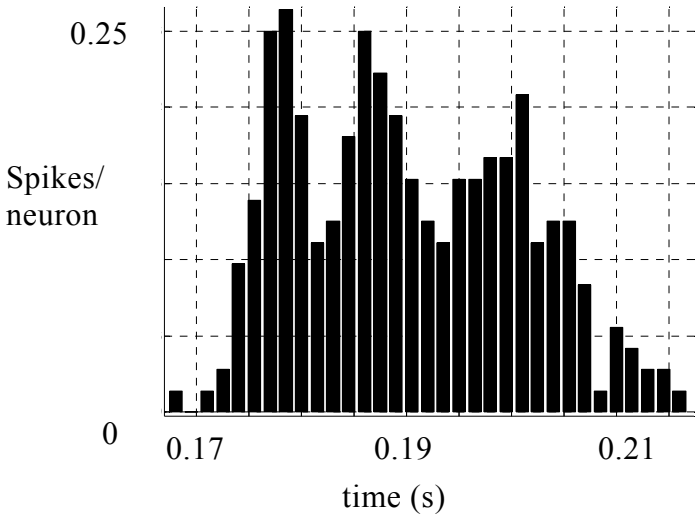

Figure 7.6. Average PSTH of six trials.

7.4 Conclusion

We have shown that phenomena like spike and burst synchronization is possible to simulate with a biologically detailed network of I-F neurons. High precision spike synchronization (<10 ms) was possible to achieve with a constant current input. With increased input some of the neurons displayed repetitive bursting, which helped population oscillation but destroyed high precision spike synchronization.

We saw also that the network behavior was rather independent of the input type. We assume that more pronounced spike synchronization could be achieved for the one bar stimulus simulation, if the stimulus configuration was different, as stated before.

The long-range horizontal connections played an important role for synchronization. Even with very few connections it was possible to spike synchronize neurons situated in minicolumns 2.5 mm from each other. We would like to stress the fact that, on average, there were at maximum two connections between neurons in minicolumn one and six during the simulations. Spike synchronization was tighter between neurons spatially closer to each other and decreased with distance.

Direction of our future research is to make the network model even more realistic. We are currently testing the network model with Poisson neurons. Cortical neurons are known for their irregularity of the interspike interval [3,9]. Preliminary results have shown that oscillation is possible to achieve with our network using Poisson neurons. Our intention is to expand the model with inhibitory neurons as well. We believe that inhibition will contribute to synchronized activity [4].

7.5 References

[1] W. Gerstner, Spiking Neurons, in: W. Maass, C. M. Bishop, Pulsed Neural Networks, The MIT Press, 1998.

[2] W. Kistler, W. Gerstner, J.L. van Hemmen, Reduction of Hodgkin-Huxley equations to a threshold model, Neural Comp. 9 (1997) 1069-1100.

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

[4] E. Fransén, A. Lansner, A model of cortical associative memory based on a horizontal network of connected columns, Network: Comput. Neural Syst. 9 (1998) 235-264.

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

[6] R.L. De Valois, N.P. Cottaris, L.E. Mahon, S.D. Elfar, J.A. Wilson, Spatial and temporal fields of geniculate and cortical cells and directional selectivity, Vision Research, 40 (2000) 3685-3702.

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

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

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

[10] C. Lyon, N. Jain, J.H. Kaas, Cortical Connections of Striate and Extrastriate Visual Areas in Tree Shrews, J of Comparative Neurology 401 (1998) 109-128.

[11] H. Bosking, Y. Zhang, B. Schofield, D.Fitzpatrick, Orientation Selectivity and the Arrangement of Horizontal Connections in Tree Shrew Striate Cortex, J of Neuroscience, 17(6):2112-2127, 1997.

[12] C.M. Gray D.A. McCormick, Chattering cells: superficial pyramidal neurons contributing to the generation of synchronous oscillations in the visual cortex. Science 274 (1996) 109-113.

[13] M. Stetter, K. Obermayer, Biology and theory of early vision in mammals, in: H. H. Szu (Ed.), Brains and Biological Networks, INNS Press, 2000.

[14] W. Singer, C.M. Gray, Visual feature integration and the temporal correlation hypothesis, Annual Review of Neuroscience, 18 (1995) 555-586.

[15] K.E. Martin, J.A. Marshall, Unsmearing Visual Motion: Development of Long-Range Horizontal Intristic Connections, in: S.J. Hanson, J.D. Cowan, C.L. Giles (Eds.) Adv. in Neural Inf. Pro. Sys. 5 (1993) 417-424.

[16] S.C. Yen, L.H. Finkel, Extraction of Perceptually Salient Contours by Striate Cortical Networks, Vision Research 38(5):719-741, 1998.

[17] S.C. Yen, E.D. Menschik, L.H. Finkel, Perceptual grouping in striate cortical networks mediated by synchronization and desynchronization, Neurocomp. 26-27 (1999) 609-616.

[18] J.J. Wright, P.D. Bourke, C.L. Chapman, Synchronous oscillation in the cerebral cortex and object coherence: simulation of basic electrophysiological findings. Bio. Cyber. 83 (2000) 341-353.

[19] S.C.Yen, E.D. Menschik, L.H. Finkel, Cortical Synchronization and Perceptual Salience, Neurocomp. 125-130, 1998.

[20] S. Friedman-Hill, P.E. Maldonado, C.M. Gray, Dynamics of Striate Cortical Activity in the Alert Macaque: I. Incidence and Stimulus-dependence of Gamma-band Neuronal Oscillations, Cerebral Cortex, 10 (2000) 1105-1116.

[21] P.E. Maldonado, S. Friedman-Hill, C.M. Gray, Dynamics of Striate Cortical Activity in the Alert Macaque: II. Fast Time Scale Synchronization, Cerebral Cortex, 10 (2000) 1117-1131.

[22] A.R. Haig, E. Gordon, J.J. Wright, R.A. Meares, H Bahramali, Synchronous cortical gamma-band activity in task-relevant cognition, 11 (2000) 669-675.

[23] M.N. Shandlen, J.A. Movshon, Synchrony Unbound: A Critical Evaluation of the Temporal Binding Hypothesis, Neuron, 24 (1999) 67-77.

[24] W.M. Usrey, R.C. Reid, Synchronous Activity in the Visual System, Annu. Rev. Physiol. 61 (1999) 435–56.

[25] S. Grossberg, R. Williamson, A Neural Model of how Horizontal and Interlaminal Connections of Visual Cortex Develop into Adult Circuits that Carry Out Perceptual Grouping and Learning, Cerebral Cortex, 11 (2001) 37-58.

[26] T. Wennekers, G. Palm. How imprecise is neuronal synchorization?, Neurocomp. 26-27 (1999) 579-585.

[27] T. Hansen, H. Neumann, A model of V1 visual contrast processing utilizing long-range connections and recurrent interactions, ICANN, 61-66, 1999.

[28] A.K. Engel, P.R. Roelfsema, P. Fries, M. Brecht, W. Singer, Role of the Temporal Domain for Response Selection and Perceptual Binding, Cerebral Cortex, 7 (1997) 571-582.

8 Paper II – 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.

8.1 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 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. 8.1–3).

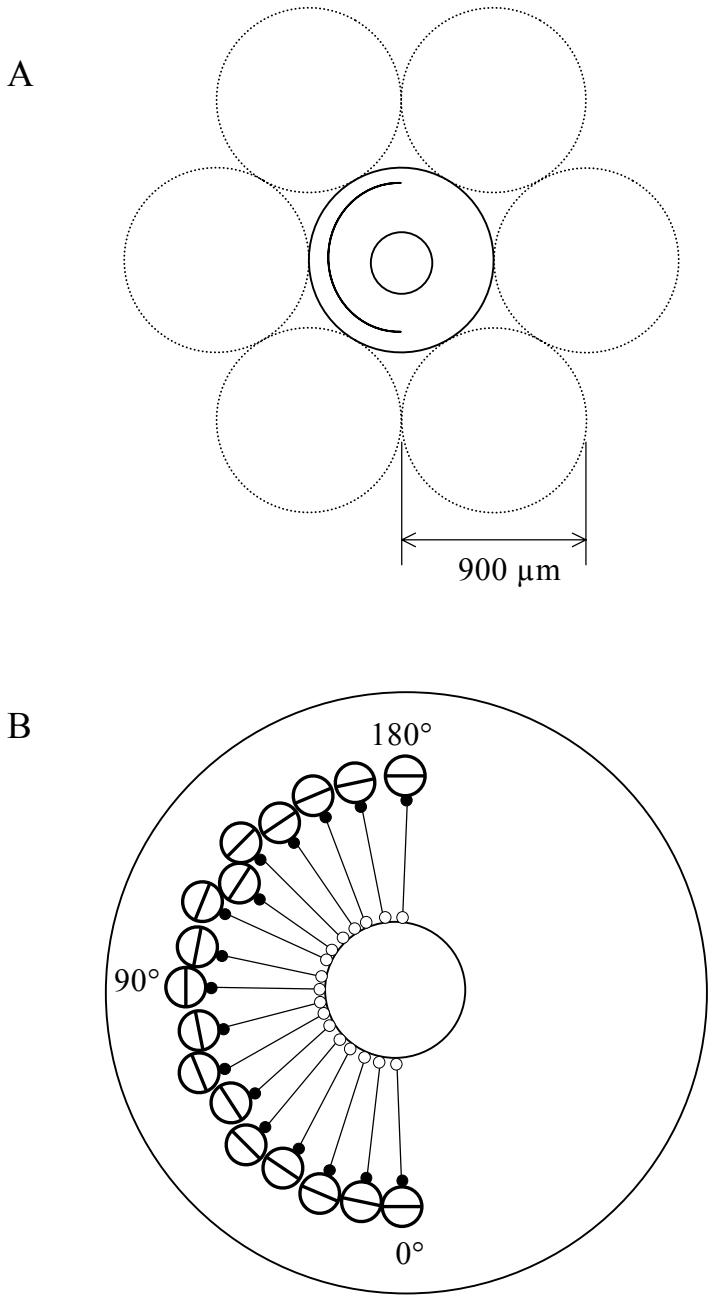

Figure 8.1. *A*, The hypothesized repetitive layout of the cat V1 demonstrated by 7 hypercolumns. *B,* The partial hypercolumn model used during the simulations consisted of 17 minicolumns.

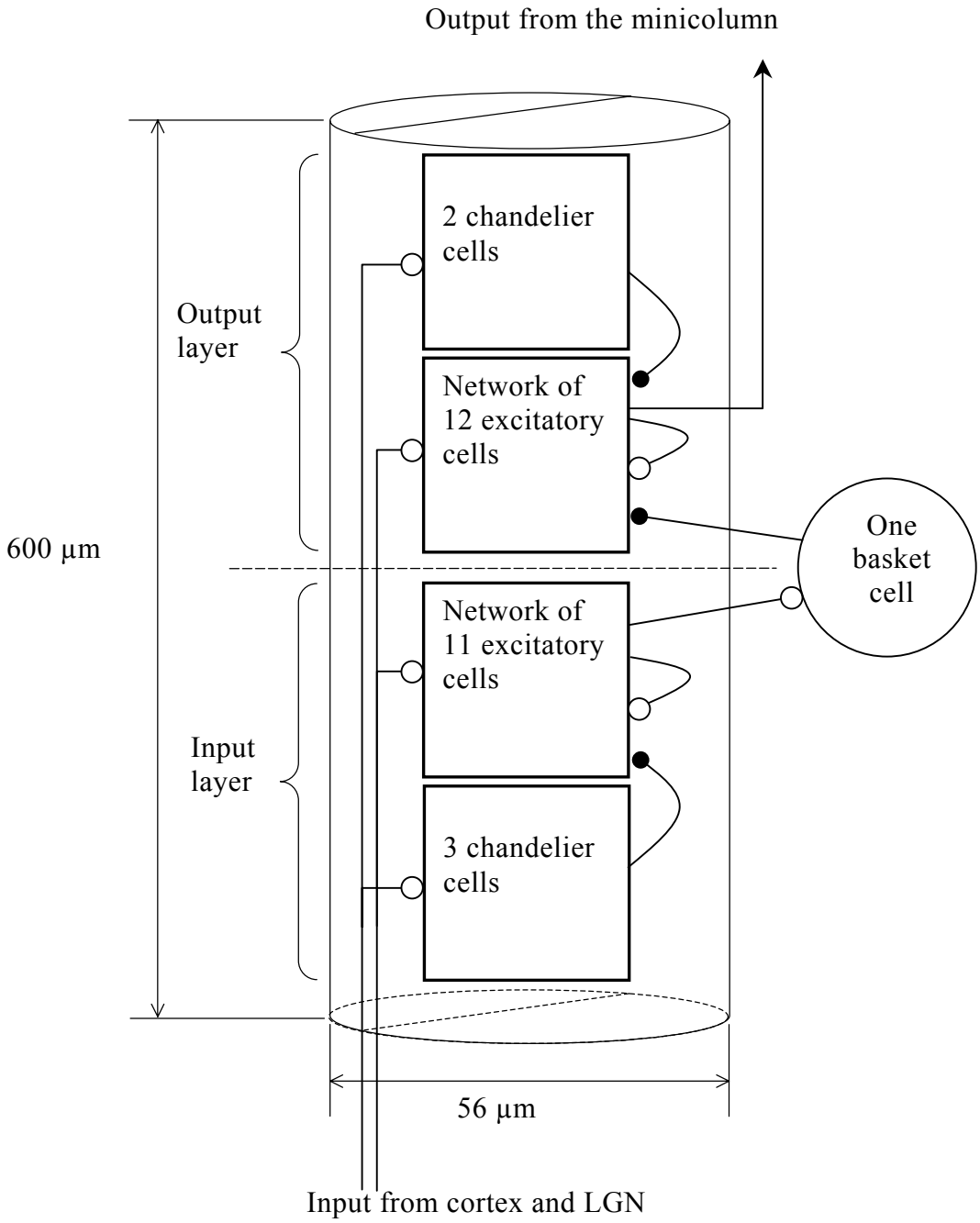

Figure 8.2. A scheme showing one of the subsampled orientation minicolumns and the basket cell representing the pool of inhibitory cells. Excitatory cells in the input layer are connected to the basket cell, and the basket cell inhibits the excitatory cells in the output layer.

8.2 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. 8.1*A*). In our model a hypercolumn is represented by a finite number of minicolumns, each representing a particular orientation (Fig. 8.1*B*). 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. 8.1*B*). 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. 8.2) 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. 8.2). It will be shown later that the excitatory neurons in the output layer replicate 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. 8.1*B* and Fig. 8.2). 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].

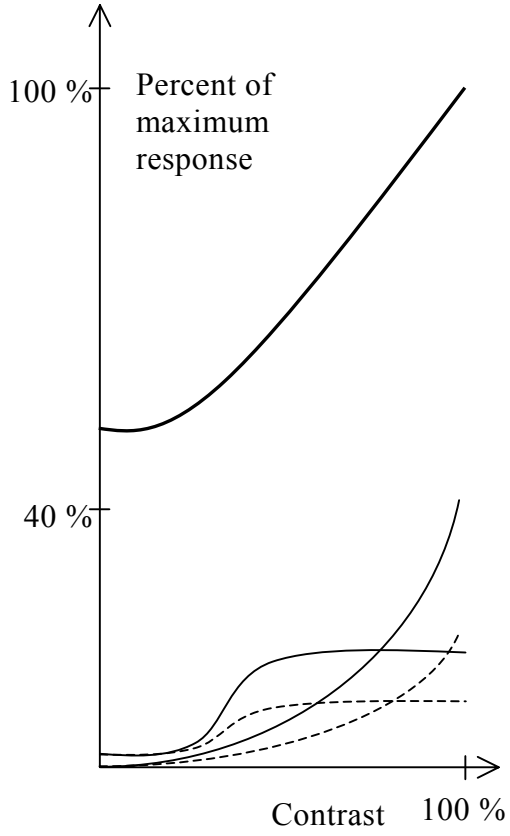

Figure 8.3. A scheme showing the activity of the excitatory cells in two minicolumns with preferred orientation (bottom solid lines) and non-preferred orientation (bottom dashed lines). Input layer excitatory cells (exponential curves) are driving the basket cell (thick top curve). The output layer excitatory cells (sigmoidal curves) are normalized when the basket cell's activity increases linearly.

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].

8.3 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%.

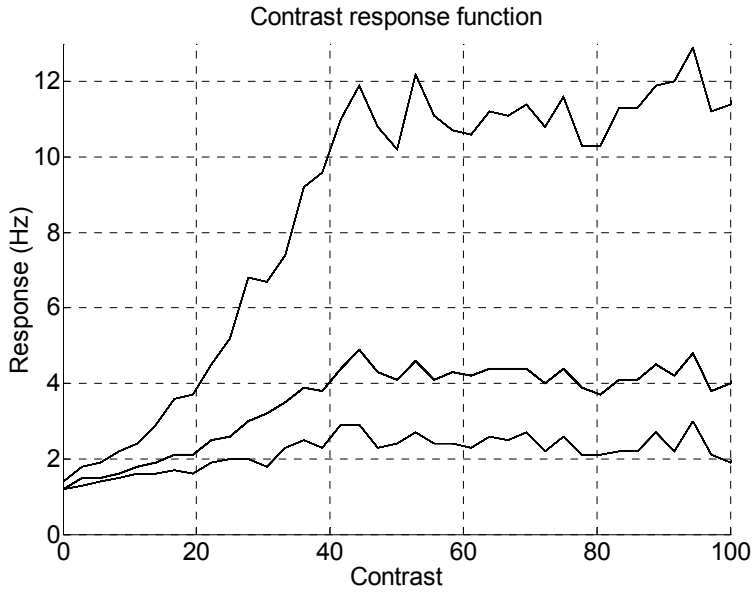

Figure 8.4. Contrast response function curves corresponding to mean activity of excitatory cells situated in the output layer. Top curve corresponds to cells situated in the minicolumn having the same orientation preference (90°) as the input to the hypercolumn. The thick curve in the middle corresponds to mean activity of cells in all 17 minicolumns. The bottom curve corresponds to activity of cells having a 45° orientation preference.

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.

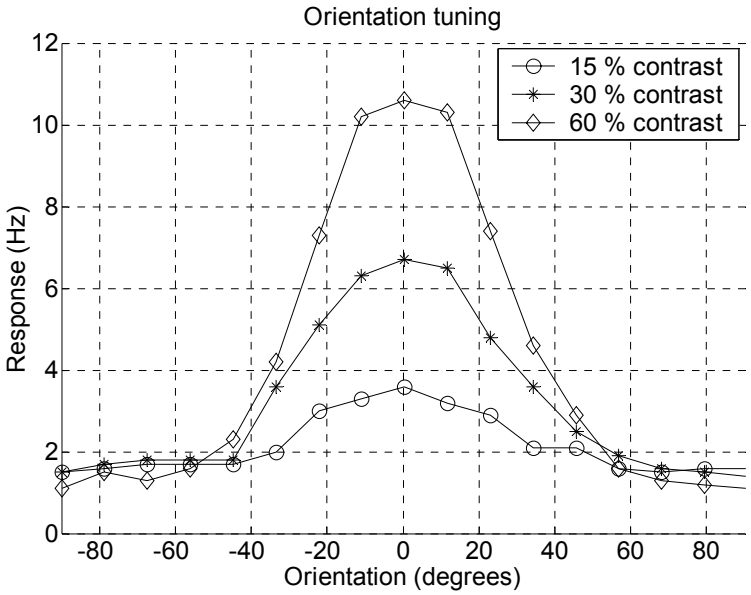

Figure 8.5. Contrast-invariance demonstrated by the network. Selectivity remains constant while the peak increases as a function of increasing contrast.

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. 8.4) 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. 8.4). The R_{max} levels, <12 Hz, were below maximum frequency levels (≈100 Hz) governed by the electrical properties of the cells (Fig. 8.4). C_{50} levels (approx. 24%) of modeled cells were biologically plausible (Fig. 8.4).

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. 8.5). 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. 8.4) 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.

8.4 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 orientations 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.

8.5 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.

9 Paper III – Layout and Function of the Intracortical Connections within Layer 4 of Cat Area 17

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Abstract

A patch of layer 4 of cat area 17 has been modeled. The developmental network model is based on the modular structure of the neocortex. Connections between the orientation minicolumns, building the network model, are developed during exposure to visual input. The network model captures some of the known properties of the layer 4 of cat area 17. Local connections are dense, whereas distal connections are sparse. Both local and distal inhibition is mediated by inhibitory simple cells, which target excitatory cells that are located in their close surroundings and have opposite absolute and relative spatial phase. Excitatory local connections seem to be biased towards the iso-orientation domain. However, there is a strong crosstalk between all orientation domains made by the excitatory long-range horizontal connections. Furthermore, the excitatory long-range horizontal connections are mildly elongated along the orientation axis. We hypothesize that this network layout can give a simple explanation to the psychophysical experiments demonstrating response facilitation as a result of elongation of a Gabor patch along the orientation axis.

9.1 Introduction

Orientation selectivity of the striate cells populating the primary visual cortex (area 17 of cat, area V1 of monkey) is one of the most investigated issues in visual neuroscience [3,21]. The feedforward model, proposed by Hubel and Wiesel [1], was the first attempt to explain the origin of the orientation selectivity of the striate cells. It was proposed that orientation selectivity of neurons classified as simple cells was a consequence of the synaptic input from the LGN. According to this arrangement, the ON-center LGN cells project to the ON-subregions of a simple cell's receptive fields (RF). The OFF-subregions of a simple cell were constructed in the same way by the OFF-center LGN cells (see also [2]). Still today the Hubel and Wiesel feedforward model serves as a model of thalamic input to neocortex.

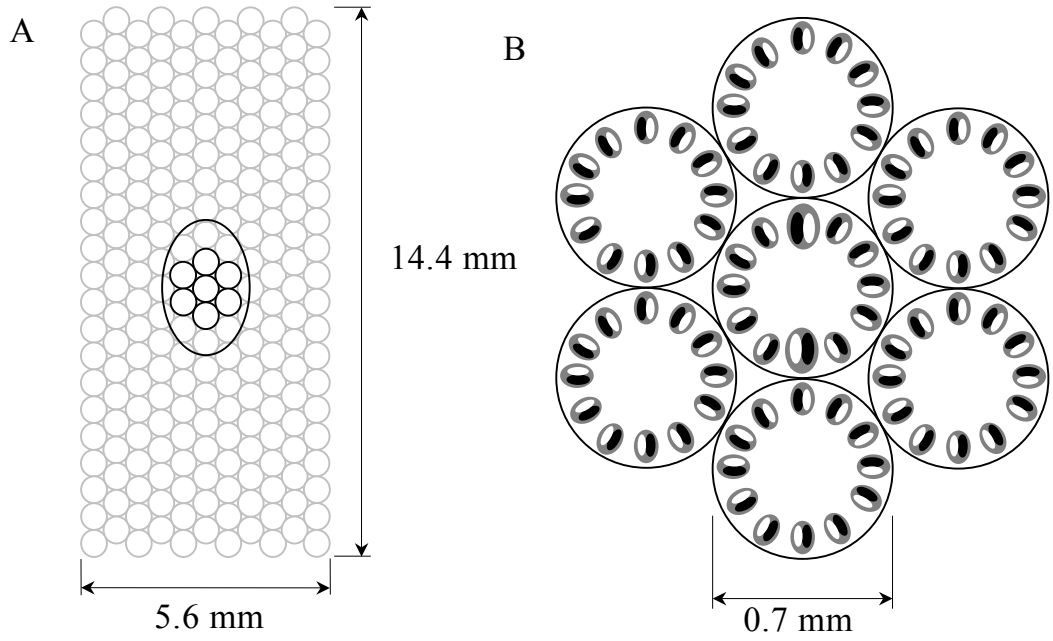

Figure 9.1. *A*, The network model consists of an 11x20 hexagonal array of hypercolumn modules. The oval illustrates the RF of the magnified units, positioned in the hypercolumn in the center in Fig. 9.1*B*. These units detect vertical lines but have opposite absolute and relative spatial phases. We see that these units' RF overlap with many other hypercolumns, and hence indicate strong overlap between RF of units situated in neighboring hypercolumns. *B*, *A* sub-network consisting of seven hypercolumns. Inside each hypercolumn 12 units are placed. Every hypercolumn figure is also a polar plot (see polar-plots in Fig. 9.2). The units indicate the positions in the polar-plot.

However, some of the properties related to the orientation selectivity are not possible to predict by a pure feedforward model [3–8]. Especially the psychophysical studies [4–8] examining the long-range spatial interactions in visual cortex clearly demonstrate that the cortical circuit plays a major role in altering the responses of the striate cells. Polat and Norcia [5] demonstrated that elongation of a Gabor patch along the orientation axis results in facilitation of the responses of the striate cells. As a result, Polat and Norcia [5] propose elongated summation pools.

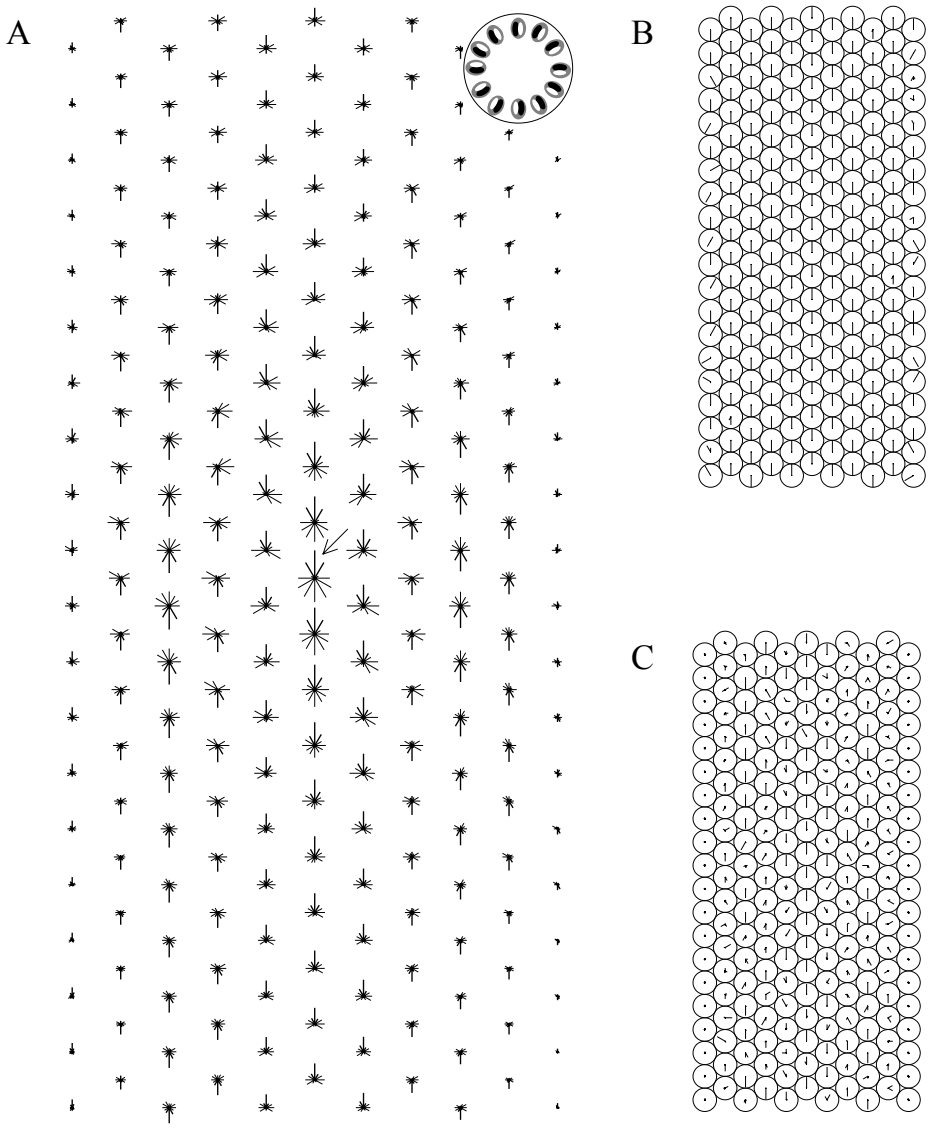

Figure 9.2. Polar-plots organized as the network model, each one representing a hypercolumn module. The legend in *A* (top right) shows the orientation and the relative spatial phase of the units in each polar-plot representing a hypercolumn module. *A*, Projections of the 'reference unit', which is positioned in the middle hypercolumn (marked with an arrow). Thick lines are excitatory connections and thin lines correspond to inhibitory connections. Distance from the origin is proportional to the strength of the connection. *B*, Polar-plots showing normalized activities of the units in hypercolumn modules after 50 ms. Distance from the origin is proportional to the activity level. During this simulation the units receive both thalamic and cortical input. *C*, Same as *B*, but the units receive only thalamic input. Note that only hypercolumns receiving strongest input, those in the middle column, do converge.

The excitatory long-range horizontal connections are a prominent feature of the visual cortex [9–14]. These connections can extend for several millimeters on cortex surface. Local connections are defined as connections between neurons located inside an area as big as a hypercolumn (<500 μm). Especially the layer 2/3 excitatory long-range horizontal connections have been subject to intense study since the discovery of their patchy layout [13]. More recent studies have confirmed the patchy, iso-orientation biased, layout of these connections [10,12]. The patches do, however, vary in size. Kisvárday et al. [10] report that the patch size can vary between 200 and 1000 μm in area 17 of cat. Observe that an iso-orientation domain is roughly 400–600 μm in diameter. These values indicate that the patches are heterogeneous. Differences in orientation preferences between striate cells up to 90º inside such a patch are not unusual. Furthermore, according to Kisvárday et al. [10], also the local connections prefer the iso-orientation domain, i.e. the local connections show same connectivity patterns as the long-range horizontal connections. Schmidt et al. [12] do also report axial specificity of the excitatory long-range horizontal connections. Later we will see that this anisotropy, also shown by our network model, can explain some of the observations described above [4–8].

On the contrary, the excitatory long-range horizontal connections found in layer 4 have drawn less attention [11,14]. However, the study by Yousef et al. [11] reveals some of the layer 4 excitatory long-range horizontal connections' properties. This study indicate that these connections are not, or very little, biased towards iso-orientation domains. The excitatory long-range horizontal connections to iso-, oblique- and cross-orientation domains are almost equal in distribution. However, local connections are still biased towards the iso-orientation domain. It seems that independent of the layer, the excitatory long-range connections are neither random nor restricted to iso-orientation domains. We can therefore assume that crosstalk between different orientation domains is a prominent feature of both layer 4 and layer 2/3. This belief is also supported by the experiments described above [4–8].

According to the findings by Hubel and Wiesel [15] the primary visual cortex has a modular structure (for a recent review see [16]). It is composed of orientation minicolumns each one comprising some hundreds of excitatory cells and a smaller number of inhibitory interneurons of different kinds. Contrast edge orientation is coded such that the striate cells in each orientation minicolumn respond selectively to a broad range of orientations. A recent study by DeAngelis et al. [17] reveal the degree of invariance of response variables like orientation, spatial frequency and relative spatial phase. Orientation was highly clustered, closely followed by spatial frequency. One response variable did, however, not show any evidence of clustering, namely, the relative spatial phase. Furthermore, an 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.

The Bayesian Confidence Propagation Neural Network model (BCPNN) has been developed in analogy with the known generic cortical structure [25]. 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. We hypothesize that normalization can be carried out by large basket cells [28]. We assume that these cells inhibit an area that corresponds to a hypercolumns.

A patch of layer 4 of cat area 17 has been modeled. This developmental network model is based on the modular structure of the neocortex. BCPNN incremental learning algorithm develops the connections between the units. The correlation-based network captures some of the known properties of area 17 of cats, such as dense local and sparse distal connectivity. The network has two different types of interneurons. Large basket cells are responsible for keeping the total activity within a hypercolumn constant. The second group of interneurons, the inhibitory simple cells, mediate local and distal inhibition through targeting excitatory cells that are located in their close surroundings and have opposite absolute and relative spatial phase (relative to the interneuron). Excitatory local connections seem to be biased towards the iso-orientation domain. However, excitatory long-range horizontal connections target all orientation domains in a balanced manner, thus there is a strong crosstalk between all orientation domains. Note however that some of the targets of the long-range horizontal connections are excitatory cells, whereas some are inhibitory simple cells, since the network is correlation-based. Furthermore, the excitatory long-range horizontal connections are mildly elongated along the orientation axis, most likely as a result of the stimulus configuration. During the learning phase the stimuli were contrast edges. Note that on the contrary to layer 2/3, there has not been any report on axial specificity of the layer 4 long-range horizontal connections. Nevertheless, we believe that the network behavior supports the existence of elongated summation pools in visual cortex, and gives a simple explanation for how it might be carried out within area 17. There are a variety of feedforward and recurrent models of area 17 (see review [3]), however, very few of them address the questions related to response facilitation as a consequence of elongated summation pools. Recently, Grossberg and Raizada [31] modeled perceptual grouping based on long-range horizontal interactions within area 17. In that model, layer 2/3 complex cells are responsible for contrast-sensitive perceptual grouping.

9.2 Network Model

As stated before the columnar organization of the primary visual cortex [15] is the main influence of our network model. We hypothesize that area 17 is composed of repetitive structures, i.e. orientation minicolumns. We assume further that the orientation minicolumns can be grouped around hypothetical centers, the so-called pinwheels [18], to form modules we refer to as hypercolumns. We hypothesize that a hypercolumn can be built from a finite number of orientation minicolumns each representing a unique orientation (Fig. 9.1*B*). The network model used during the simulations consists of 220 (11x20) hypercolumns arranged to form a hexagonal array (Fig. 9.1*A*). The diameter of the circular hypercolumns, and thus the distance between two adjacent hypercolumns is 0.7 mm [19]. The size of the network model in cortical dimensions is 5.6x14.4 mm (Fig. 9.1*A*). The distance between the RF centers of two adjacent hypercolumns corresponds to 0.2º of visual angle (at 2º of eccentricity [19]). The visual world covered by the model is 2.4x5.4º. Note that the modeled cortical patch and hence the visual field covered by the network model is elongated. This shape was chosen after observing the mildly elongated shape of the excitatory long-range horizontal connections, and having the computational limitations in mind. However no artifacts related to corner effects due to the elongated shape of the network model was noticed during the simulations.

Each hypercolumn consists of 12 units, representing 6 orientation minicolumns (Fig. 9.1*B*). The difference in orientation preference between two successive units inside a model hypercolumn is 30°. Observe that having this configuration, we represent every orientation twice with two so-called anticorrelation units. These anticorrelation units represent same orientation. However, they have opposite absolute and relative spatial phases, so that their subfields with opposite sign overlap. The magnified RF figures inside the middle hypercolumn are detecting vertical lines and have opposite absolute and relative spatial phases (Fig. 9.1*B*), thus they qualify as anticorrelation pairs.

The RF centers of the units belonging to a model hypercolumn are positioned in the center of their host hypercolumn. As a consequence of this arrangement the units belonging to a hypercolumn are analyzing the same spot of the visual field. The RF of the units are designed as contrast edge detectors, and hence composed of two elongated subregions with opposite sign (Fig. 9.1*B*). Orientation tuning of the LGN input is 40º at half-width at half-height [20], suggesting a subfield aspect ratio of 3:1. The RF width is 1º [19], and hence the RF height is 1.5º. Figure 9.1*A*. shows how a RF, in this case belonging to the units located in the middle hypercolumn detecting vertical lines (Fig. 9.1*B*), are related to the rest of the network. We can see that there is an overlap between the RF:s of these two units and the rest of the network. Furthermore, all units are tuned for the same spatial frequency of 1 cycle/degree [19]. The thalamic input of the units is computed using a model developed by Troyer et al. [23].

9.3 Simulation Results

The simulations are divided into two parts. Firstly, BCPNN incremental learning algorithm [15] is used to train the fully connected recurrent network of units. The objective is to see if the BCPNN algorithm can develop a network that resembles the primary visual cortex [9–14]. Later we address the question of whether or not this network can explain some of the reported psychophysical observations, especially those related to response facilitation [5].

Briefly the BCPNN incremental learning algorithm behaves in the following way. If two units are correlated during a time step, the connection between them strengthens. This corresponds to the creation of an excitatory connection. However, anticorrelation between two units will result in an inhibitory connection (via a local inhibitory interneuron [26,27]). Hirsh and colleagues [26] have reported that there are inhibitory simple and complex cells in cat area 17. Furthermore, according to the study by Ferster [27] interneurons mediate inhibition between excitatory simple cells, which have opposite absolute spatial phase. Based on the results by Ferster [27] and Hirsh et al. [26] we assume that interneurons inhibit excitatory cells located in their close surroundings. We hypothesize further that the interneurons and their postsynaptic excitatory cell targets have overlapping receptive fields and opposite absolute spatial phases, relative each other. The implication of this scheme is that any given excitatory cell can, through an interneuron, inhibit another excitatory cell. Note that both local and distal inhibition can be mediated by these interneurons, since excitatory cells located in an arbitrary place can target an interneuron. Kayser and Miller [22] have proposed a similar model called the opponent inhibition model based on [27]. Their model assumes, however, that exclusively excitatory cells that have similar receptive field profile, i.e. same orientation preference and same absolute spatial phase, target the interneurons. Kayser and Miller [27] also assumed that the excitatory cell's receptive field and its inhibitory target's receptive field coincide in visual space. Hypothetically the whole network is local and can be located within an orientation minicolumn.

According to the BCPNN incremental learning algorithm, if units are uncorrelated, the connection value will fluctuate around zero value. This means that the weight between two units is a measure of the correlation between them. From this follows also that the weight matrix is symmetric, and hence can be interpreted both as projections from one unit to all units in the network, and projections from all units into one. Note that normalization limits the total activity within a hypercolumn to one. This is done mathematically by dividing activity level of each unit within a hypercolumn by the total activity of its host hypercolumn. It is, however, assumed that large basket cells are responsible for normalization within area 17 [28].

Training of the fully connected network lasts for 1000 simulation steps. The time step is defined as 1 second, and hence the simulation duration corresponds to

1000 seconds. The learning rate, which defines the degree of weight modification, is 0.005 [15]. At every time step, the activity levels of the units are initiated with a new arbitrary contrast edge stimulus. The position and the orientation of the stimulus are sampled from a uniform distribution. The stimulus width is 1º, and its spatial frequency is 1 cycle/degree. Observe that for convenience the width and the spatial frequency of the stimulus is the same as that of the RF.

Noise is added to the activity levels through several steps. First a normally distributed noise with a standard deviation of 10% of the so-called bias value is added. The bias value is defined as the value of all units inside a hypercolumn in absence of any stimulus. The activity levels are rectified so that all negative activity levels are set to zero, and a 5-10% uniform distribution noise is added to all units to simulate the background activity. Later the activity levels are normalized so that the sum of activities in each hypercolumn is equal to one, as required by the BCPNN incremental learning algorithm. The contrast of the stimulus is 100%, though the effect of high noise in combination with the normalization procedure lowers this level considerably.

We see that in general the local connections do have higher amplitude than the long-range horizontal connections. This indicates strong local and sparse distal connectivity (Fig. 9.2*A*). The developed network is correlation-based and incorporates a second group of interneurons (after the large basket cells), which are hypothesized to be the inhibitory simple cells described by Hirch et al. [26] (see also [27]).

Not surprisingly, the ‘reference unit’ is most correlated with itself (Fig. 9.2*A*). Units that are correlated excite each other (Fig. 9.2*A*). Units that are anticorrelated (not only anticorrelation pairs) inhibit each other through inhibitory simple cells, which have opposite absolute and relative spatial phase compared to their postsynaptic targets (Fig. 9.2*A*). We hypothesize that these interneurons could be driven by both local and distal excitatory cells that are anticorrelated with the targets of the interneurons. We assume further that the connection strengths between the inhibitory simple cells and their excitatory targets are constant.

The mildly elongated shape (along the orientation axis) of the connections made by the reference unit reveals the axial specificity of the excitatory long-range horizontal connections (Fig. 9.2*A*). This result is in agreement with the results of Schmidt et al. [12] and others on layer 2/3 of several species. Note however that the layer 4 network seems to be less elongated than layer 2/3.

As shown by Yousef et al. [11], all distal orientation domains are targets of the long-range horizontal connections. The connection matrix shows that, some of the excitatory long-range horizontal connections are targeting excitatory cells located in iso-orientation domains, whereas others are targeting local interneurons positioned in all three orientation domains (2*A*). Obviously there is an intense crosstalk between all orientation domains, made by the excitatory long-range horizontal connections. Note further that the inhibitory long-range horizontal

connections are not the subject of study, since all long-range horizontal connections are considered to be excitatory.

A more detailed analysis of the excitatory long-range horizontal connections uncovers the type of these connections. We see that the strength of the excitatory connections between the reference unit, and units having same orientation preference and absolute (and relative) spatial phase tend to decrease along the axis, which corresponds to the preferred orientation of the reference unit. Along the orthogonal axis, connection type is switched from excitatory to inhibitory, as an effect of anticorrelation (Fig. 9.2*A*). Note also that again along the orthogonal axis, the reference unit is correlated with the units having opposite relative spatial phases, but similar absolute spatial phases (Fig. 9.2*A*). These observations indicate that units with similar absolute spatial phases excite each other. DeAngelis et al. [17] showed recently that relative spatial phase did not show any evidence of clustering, and local pooling across simple cells with different spatial phases was proposed to improve signal quality. These ideas support the connectivity pattern between the iso-orientation domains.

Above, we showed that the network captures important properties of the connection pattern found within area 17. In the second part of the simulations, we will see if this network can predict some of the psychophysical effects especially those related to elongated summation pools in visual cortex [5]. But first, a brief explanation of the balance between the excitatory cortical input and the thalamic input received by the units. Chung and Ferster [20] reported that cortical suppression left only 46% of the visually evoked response of the striate cells receiving monosynaptic input from the thalamus. This balance is preserved in our network model such that at 100% contrast the input to a unit is 46% of maximum theoretical input, and the cortical input is scaled accordingly. The maximum theoretical input is defined as the strongest excitatory input that is possible to receive by a unit from the rest of the network. Normally distributed noise with a standard deviation of 10% of the maximum theoretical input is added to the total input of every unit. The simulation time step is 10 ms, and the ‘membrane time constant’ of the units is 50 ms [15].

The simulation consists of two phases. During these both phases the stimulus is a vertical contrast edge, positioned in the center of the network. This stimulus is identical to the stimuli used during the training of the network. In the first phase, the units receive both thalamic input and cortical excitatory and inhibitory input (Fig. 9.*2B*). The thalamic input is amplified by the cortical connections and all hypercolumns are converging after 50 ms, even those receiving weak input. In the second phase, the units receive only thalamic input (Fig. 9.*2C*). However, the thalamic input is not sufficient for the majority of the hypercolumns; only those receiving maximum thalamic input can converge. Note the difference in convergence speed and quality between these two cases after 50 ms. The role of excitatory long-range horizontal connections in sharpening of the responses of the units receiving weak thalamic input is evident.

The experiment by Polat and Norcia [5] shows that elongation of a Gabor patch along the orientation axis results in facilitation of the responses of the striate cells in visual cortex. As a result, elongated physiological summation pools are proposed. We hypothesize that the mildly elongated shape of the long-range horizontal connections along the orientation axis (Fig. 9.2*A*) can explain the facilitation described by Polat and Norcia [5]. Furthermore, pooling across simple cells with different, in our network model opposite, relative spatial phases (same absolute spatial phase) might also play an important role improving signal quality [17]. Finally, we presume that at least qualitatively, the simulations show the facilitatory effect of the excitatory long-range horizontal connections (Fig. 9.2B and 9.2C).

9.4 Discussion

A patch of layer 4 of cat area 17 has been modeled. This developmental network model is based on the modular structure of the neocortex. BCPNN incremental learning algorithm develops the connections between the units. The correlation-based network captures some of the known properties of area 17 of cats, such as dense local and sparse distal connectivity. The network has two different types of interneurons. Large basket cells are responsible for keeping the total activity within a hypercolumn constant. The second group of interneurons, the inhibitory simple cells, mediate local and distal inhibition through targeting excitatory cells that are located in their close surroundings and have opposite absolute and relative spatial phase (relative to the interneuron). Excitatory local connections seem to be biased towards the iso-orientation domain. However, excitatory long-range horizontal connections target all orientation domains in a balanced manner, thus there is a strong crosstalk between all orientation domains. Note however that some of the targets of the long-range horizontal connections are excitatory cells, whereas some are inhibitory simple cells, since the network is correlation-based. Furthermore, the excitatory long-range horizontal connections are mildly elongated along the orientation axis, most likely as a result of the stimulus configuration. During the learning phase the stimuli were contrast edges. Note that on the contrary to layer 2/3, there has not been any report on axial specificity of the layer 4 long-range horizontal connections. Nevertheless, we believe that the network behavior supports the existence of elongated summation pools in visual cortex, and gives a simple explanation for how it might be carried out within area 17.

During the learning phase exclusively contrast edges stimuli were used. We believe that this affected the layout of the local and long-range horizontal connections significantly. One obvious question is how other types of stimuli like

sinusoidal gratings or other shapes might affect the layout of these connections. Our intention is to address this question in the near future.

9.5 References

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

[2] Reid, R.C. & Alonso, J.M. (1995) Specificity of monosynaptic connections from thalamus to visual cortex. *Nature*, 378:281–284.

[3] Ferster, D. & Miller, K.D. (2000) Neural Mechanisms of Orientation Selectivity in the Visual Cortex. *Annual Reviews of Neurosci.*, 23:441–471.

[4] Polat, U. & Norcia, A.M. (1996) Neuropsychological Evidence for Contrast Dependent Long-range Facilitation and Suppression in Human Visual Cortex. *Vision Res.*, 36, 2099–2109.

[5] Polat, U. & Norcia, A.M. (1998) Elongated physiological summation pools in the human visual cortex. *Vision Res.*, 38, 3735–3741.

[6] Adini, Y., Sagi, D. & Tsodyks, M. (1997) Excitatory-inhibitory network in the visual cortex: psychophysical evidence. *Proc. of the National Academy of Sciences USA*, 94, 10426–10431.

[7] Solomon J.A. & Morgan M.J. (2000) Facilitation from collinear flanks is cancelled by non-collinear flanks. *Vision Research*, 40:279–286.

[8] Solomon, J. A., Watson, A. B. & Morgan, M. J. (1999). Transducer model produces facilitation from opposite-sign flanks. *Vision Research*, 39, 987–992.

[9] Gilbert, C.D. & Wiesel, T.N. (1989) Columnar specificity of intrinsic horizontal connections and corticocortical connections in cat visual cortex. *J. Neurosci.*, 9:2432–2442.

[10] Kisvárday, Z.F., Tóth, E., Rausch, M. & Eysel, U.T. (1997) Orientation-specific Relationship Between Populations of Excitatory and Inhibitory Lateral Connections in the Visual Cortex of the Cat. *Cerebral Cortex*, 7, 605–618.

[11] Yousef, T., Bonhoeffer, T., Kim, D.-S., Eysel, U.T., Tóth, E. & Kisvárday, Z.F. (1999) Orientation topography of layer 4 lateral networks revealed by optical imagining in cat visual cortex (area 18). *E. J. of Neurosci.*, 11:4291–4308.

[12] Bosking, W.H., Zhang, Y., Schofield, B. & Fitzpatrick, D. (1997) Orientation Selectivity and the Arrangement of Horizontal Connections in Tree Shrew Striate Cortex. *J. Neurosci.*, 17(6):2112–2127.

[13] Rockland, K.S. & Lund, J.S. (1982) Widespread periodic intrinsic connections in the tree shrew visual cortex. *Science*, 215, 1532–1534.

[14] Schmidt, K.E. & Löwel, S. (2002) Long-range Intristic Connections in Cat Primary Visual Cortex. In B.R. Payne, A. Peters (eds.). *The Cat Primary Visual Cortex*, Chapter 10. Ac. Press.

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

[16] Mountcastle, V.B. (1997) The columnar organization of the neocortex, *Brain*, 120, 701–722.

[17] DeAngelis G.C., Geoffrey, M.G., Ohwaza, I. & Freeman, R.D. (1999) Functional Micro-Organization of Primary Visual Cortex: Receptive Field Analysis of Nearby Neurons. *J. Neurosci.*, 19(9):4046–4064.

[18] Braitenberg, V. & Braitenberg, C. (1979) Geometry of the orientation columns in the visual cortex. *Biological Cybernetics*, 33:179–186.

[19] De Valois, R.L. & De Valois, K.K. (1990) Striate Cortex. *Spatial Vision*. Oxford Sci. Pub.

[20] Chung, S. & Ferster, D. (1998) Strength and Orientation Tuning of the Thalamic Input to Simple Cells Revealed by Electrical Evoked Cortical Suppression. *Neuron*, 20, 1177–1189.

[21] Albrecht, D.G., Geisler, W.S., Frazor, R.A. & Crane, A.M. (2001) Visual Cortex Neurons of Monkeys and Cats: Temporal Dynamics of the Contrast Response Function. *J. Neurophysiol.*, 88:888–913.

[22] Kayser, A.S. & Miller, K.D. (2002) Opponent Inhibition: A Developmental Model of Layer 4 of the Neocortical Circuit. *Neuron*, 33, 131–142.

[23] Troyer, T.W., Krukowski, A.E. & Miller, K.D. (2002) LGN Input to Simple Cells and Contrast-Invariant Orientation Tuning: An Analysis. *J. Neurophysiol.*, 87:2741–2752.

[24] Grossberg, S. & Raizada, R.D.S. (2000) Contrast-sensitive perceptual grouping and object-based attention in the laminar circuits of primary visual cortex. *Vision Science*, 40, 1413–1432.

[25] Sandberg, A., Lansner, A., Petersson, F.M. & Ekeberg, Ö. (2002) A Bayesian attractor network with incremental learning. *Network: Computing in Neural Systems*, 13, 179–194.

[26] Hirch, J.A., Alonso, J-M, Pillai, C. & Pierre, C. (2000) Simple and complex inhibitory cells in layer 4 of cat visual cortex. *Soc. Neurosci. Abstr.*, 26, 1083.

10 Paper IV – Quantitative Assessment of the Local and Long-Range Horizontal Connections within the Striate Cortex

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Abstract

We present a quantitative assessment of the local and long-range horizontal connections within two separate models of layer 4 and layer 2/3 of the striate cortex. Connections between the orientation minicolumns, building the models, are developed during exposure to visual input. Layer 4 long-range horizontal connections target all orientation domains in a balanced manner, whereas local connections are biased towards the iso-orientation domain. However, both local and long-range horizontal connections of the layer 2/3 network are biased towards the iso-orientation domains. We hypothesize that the patchy layout of the layer 2/3 long-range connections is a consequence of excitatory cells targeting mainly other excitatory cells located in distal iso-orientation domains. Furthermore, both networks demonstrate dense local and sparse distal connectivity.

10.1 Introduction

The columnar organization of the neocortex [6,15] is one of the most influential findings in neuroscience [16]. Hubel and Wiesel [15] found that the striate cortex (primary visual cortex) is composed of orientation minicolumns each one comprising some hundreds of excitatory cells and a smaller number of inhibitory interneurons of different kinds. Vertical penetration of the cortex showed that contrast edge orientation is coded such that the cells in each orientation minicolumn respond selectively to a broad range of orientations. Furthermore, orientation selectivity was shifted smoothly during horizontal penetration. One decade earlier, Hubel and Wiesel [1] addressed the fundamental question of the orientation selectivity of the cortical cells. The proposed model of orientation selectivity, which is known as the 'feedforward' model, relies heavily on the arrangement of the thalamic afferents. According to this arrangement, the ON-center LGN cells converge on the ON-subregions of a simple cell's receptive field (RF). The OFF-subregions of a simple cell were constructed in the same way by the OFF-center LGN cells (see also [2]). Still today the Hubel and Wiesel feedforward model serves as a model of the thalamocortical circuitry. However, some of the orientation selectivity properties are not possible to predict by the feedforward model [3]. Contrast invariance of orientation tuning is one such example. As contrast increases the height of the response curve increases while the width remains almost constant [21]. Furthermore, the psychophysical studies examining the long-range spatial interactions within the visual cortex clearly

demonstrate that the cortical circuitry plays a major role in cortical cell responses. Polat and Norcia [5] demonstrated that elongation of a Gabor patch along the orientation axis results in facilitation of the responses of the cells in visual cortex, probably as an effect of summation fields.

Since the discovery of the patchy layout of the layer 2/3 long-range horizontal connections [13], the layout and the function of intracortical connections have started to drawn more attention. It is now widely accepted that the long-range horizontal connections of the superficial layers are a prominent feature of the visual cortex [7–14]. More recent studies on a variety of species have confirmed the patchy, iso-orientation biased, layout of these connections [7,8,10,12]. A study by Kisvárday et al. [10] shows that 56.2% of the excitatory local connections target the iso-orientation (±30º) domain. Oblique- (±30–60º) and cross-orientation (±60–90º) domains receive 28.4 and 15.3 per cent of the connections respectively. Long-range connections show a similar pattern, i.e. 40.0% of them descending from the injection site are targeting the iso-orientation domains, 36.9% the oblique-, and 23.1% the cross-orientation domains. Same study reveals also that only 30% of the excitatory connections from an injection site, in area 17 of cat, are defined as distal (>0.5 mm), and thus the majority of the connections target nearby cells. A recent study by Chisum et al. [8] reveals the fall-off in bouton distribution as a function of distance from an injection site. Number of boutons along the preferred axis is down to 15% of the maximum only 0.7 mm from the injection site. At 1.4 mm the number of boutons is roughly 5% of the maximum. The decrease along the orthogonal axis is even more dramatically. Thus, axial specificity of the layer 2/3 long-range connections is prominent. Chisum et al. [8] show that the dramatic fall-off with distance implies that most long-range connections are between neurons for which the RFs overlap by at least 50%. Furthermore, the long-range horizontal connections are to be responsible for the summation field of the neurons. Chisum et al. [8] observe also the similarity between the elongated RF and axial specificity of the long-range connections.

Surprisingly, the long-range horizontal connections found in the layer 4 have drawn much less attention. Layer 4 is the main recipient of the thalamic input, and the simple cells found in the cat layer 4 are well tuned for orientation [1,2]. Thus, the interplay between the orientation map and the layout of long-range connections is essential for understand the origins of orientation selectivity. The findings suggest that the layout of the layer 4 long-range horizontal connections is different from those found in the superficial layers [8,10,11,14]. Their extent is only 50% of the long-range connections of the superficial layers [10]. Furthermore, Yousef et al. [11] found that layer 4 long-range connections (of area 18) are hardly biased towards iso-orientation domains. 35.4% of the connections (>740 μm) targeted the iso-orientation domain. Oblique- and cross-orientation domains received 33.7% and 30.9% of the connections respectively. The pattern shown by the local connections (<740 μm) is different. The local iso-orientation domain receive

60.0% of the connections, while 22.0% of the connections are targeting the oblique- and 18.0% the cross-orientation domains. Note that as much as 40% of the local connections are targeting oblique- and cross-orientation domains. Inhibitory long-range horizontal connections extent only one third to one half of the excitatory network, and the excitatory ones out number them. Thus, majority of the long-range connections are considered to be excitatory in both layer 4 and layer 2/3 [10]. It seems that independent of the layer, the excitatory long-range horizontal connections are neither random nor restricted to iso-orientation domains. Consequently cross talk between different orientation domains is a prominent feature of both the layer 4 and the superficial layers.

The Bayesian Confidence Propagation Neural Network (BCPNN) has been developed in analogy with the known generic cortical structure [24]. This is an abstract neural network architecture in which each unit corresponds to a cortical minicolumn. The learning rule is based on Hebb's ideas on synaptic plasticity and emergence of cell assemblies [26]. Thus, correlated activity reinforces the connections between the units. Anti-correlated activity results in weakening of a connection, and emergence of an inhibitory one. The network is partitioned into hypercolumn-like modules and the summed activity within each hypercolumn is normalized to one. A biologically plausible model of a hypercolumn module based on the BCPNN was presented recently [28].

A quantitative assessment of the local and long-range horizontal connections within two separate models, of layer 4 and superficial layers, of the striate cortex is presented. Long-range horizontal connections of the layer 4 network target all orientation domains in a balanced manner, whereas local connections are biased towards the iso-orientation domain. The pattern shown by the layer 2/3 network is different. Both local and long-range horizontal connections are biased towards the iso-orientation domains. We hypothesize that the patchy layout of the layer 2/3 long-range connections is a consequence of excitatory cells targeting mainly other excitatory cells located in distal iso-orientation domains. Both networks demonstrate dense local and sparse distal connectivity as a result of fall-off with distance. Furthermore, tests with contrast edges indicate that both layer 4 and layer 2/3 networks can detect contrast edges, which have low contrast. However, these tests need to be verified quantitatively.

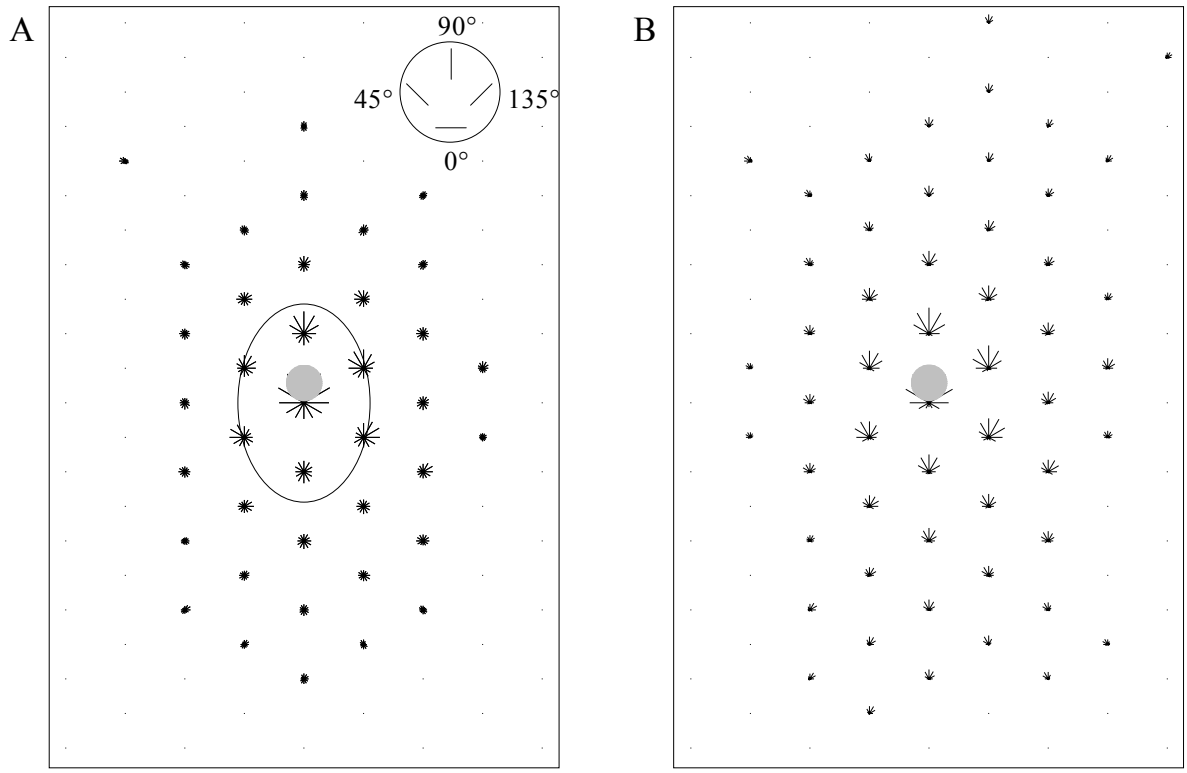

Figure 10.1. *A*, Layer 4 network connections, which originate from the abstract injection positioned at 90º (illustrated with a gray circle), visualized by 9x11 polar-plots organized as the network model. Each polar-plot represents a hypercolumn module. The legend (top right) shows the orientation of the units in each polar-plot, consequently the relative spatial phases are ignored. Distance from the origin is proportional to the strength of the connection. The oval marks the RFs of units detecting vertical lines inside the host hypercolumn. We see that these units' RF overlap with many other hypercolumns, and hence indicate strong overlap between RF of units situated in neighboring hypercolumns. Note the elongated shape of the long-range connections. *B*, Same as *A* but for the layer 2/3 network connections.

10.2 Network Model

As stated before the columnar organization of the striate cortex [15] is the main influence of our network model. We hypothesize that V1 is composed of repetitive structures, i.e. orientation minicolumns. We assume further that the orientation minicolumns can be grouped around hypothetical centers, the so-called pinwheels [18], to form modules we refer to as hypercolumns. We hypothesize that a

hypercolumn can be built from a finite number of orientation minicolumns each one representing a unique orientation. The network model used during the simulations consists of 99 (9x11) hypercolumns arranged to form a hexagonal array. The diameter of the circular hypercolumns, and thus the distance between two adjacent hypercolumns is 0.7 mm [19]. The size of the network model in cortical dimensions is 5.6x8.1 mm. The distance between the RF centers of two adjacent hypercolumns corresponds to 0.5º of visual angle (at 5º of eccentricity [19]). The visual field covered by the model is 4.5x7.3º. Note that the modeled cortical patch, and hence the visual field covered by the network model is elongated. This shape was chosen after observing the axial specificity of the long-range horizontal connections, and having the computational limitations in mind. However no artifacts related to corner effects due to the elongated shape of the network model was observed during the simulations.

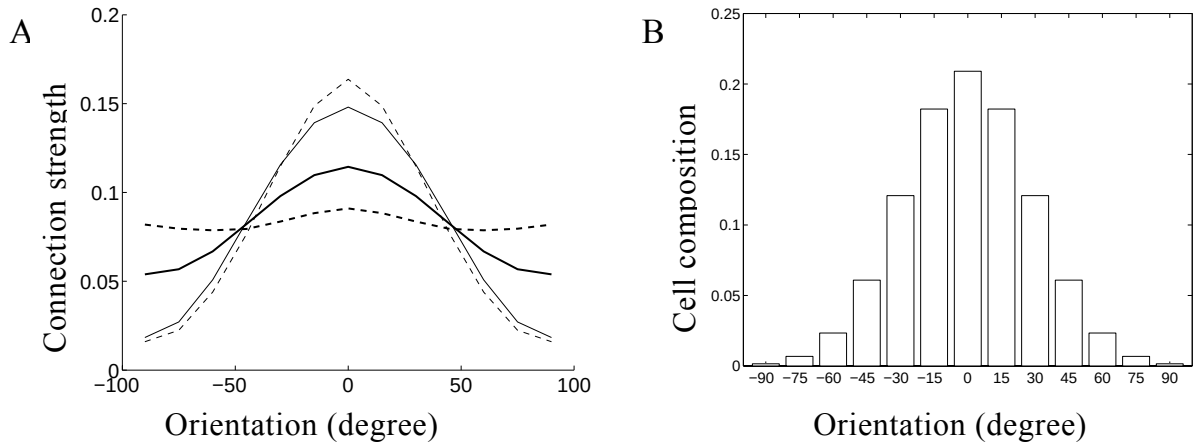

Figure 10.2. *A*, Fraction of the layer 4 local (solid thick line) and long-range (dashed thick line) connections as a function of the deviation from the mean orientation selectivity at the abstract injection site. Long-range connections are equally distributed, whereas local connections are biased towards the iso-orientation domain. Furthermore, both layer 2/3 local (solid thin line) and long-range (dashed thin line) connections are biased towards the iso-orientation domains. *B*, Composition of the abstract injection sites for layer 4 and layer 2/3 networks. Spread of orientations inside these regions correspond roughly to that of an iso-orientation domain and is less than those in *A*.

Each layer 4 hypercolumn consists of 24 units, representing 12 orientations. The difference in orientation selectivity between two successive units inside a model hypercolumn is 15°. Observe that having this configuration, we represent every orientation twice with two so-called anti-correlation units. These anti-correlation units represent the same orientation. But they have opposite relative and absolute

spatial phases, so that the units' subfields with opposite sign overlap. The RF centers of the units belonging to a model hypercolumn are positioned in the center of their host hypercolumn. As a consequence of this arrangement, the units belonging to a hypercolumn analyze the same spot of the visual field. Furthermore, as shown by the oval, which represents RFs of units detecting vertical lines, there is a strong overlap between the RF of units situated in neighboring hypercolumns (Fig. 10.1*A*). The RFs of the units are designed as contrast edge detectors, and hence are composed of two subregions with opposite sign. Orientation tuning of the LGN input is 40º at half-width at half-height [20], suggesting a subfield aspect ratio of 3:1. The RF width is 1º [19], and hence the RF height is 1.5º. All units are tuned for the same spatial frequency of 1 cycle/degree [19]. The thalamic input of the units is computed using a model developed by Troyer et al. [23].

The inhibition is mediated by local interneurons. Recently, Hirch et al. [4] reported inhibitory simple cells, which have good orientation tuning, and complex inhibitory cells that are untuned for orientation. We assume that the inhibitory simple cells can mediate local inhibition to excitatory simple cells. We hypothesize further that the opponent inhibition theory proposed by Ferster [22] can explain the local circuitry of inhibitory and excitatory simple cells. Opponent inhibition implies that the presynaptic inhibitory simple cell has opposite absolute spatial phase as the postsynaptic excitatory cell. Furthermore, the cells' subfields with opposite sign overlap, i.e. they have opposite relative spatial phase. We hypothesize that the inhibitory complex cells reported by Hirch et al. [4] can regulate the total activity of the excitatory cells in a region, which corresponds to a hypercolumn.

10.3 Simulation Results

The simulations are divided into two phases. During the first phase, we intent to show that the incremental BCPNN learning rule can develop a network that quantitatively resembles layer 4 local and long-range connectivity. During the second phase, the objective is to show that layer 2/3 local and long-range connectivity can be derived based on the results from the first phase and additional simulations.

The incremental BCPNN learning rule [24] develops the fully connected recurrent network of units. The result is a weight matrix, which contains information on the degree of correlation between pairs of units located in the different regions of the network. Briefly, the incremental BCPNN learning rule behaves in the following way. If two units are correlated during a time step, the connection between them strengthens. This corresponds to the creation of an excitatory connection. However, anti-correlation between two units will result in an inhibitory connection (via a local inhibitory interneuron [4,22]). We assume

that the excitatory long-range connections can mediate long-range inhibition through local inhibitory interneurons. We hypothesize that interneurons can inhibit excitatory simple cells in their close surroundings as proposed by the opponent inhibition theory [22]. If units are uncorrelated, the weight will fluctuate around zero value. Thus, the weight between two units is a measure of the correlation between them during the training sequence. The weight matrix is symmetric, and hence can be interpreted both as connections from one unit to all units in the network, and connections from all units into one. Training of the fully connected network lasts for 1000 simulation steps. The time step is defined as 1 second, and hence the simulation duration corresponds to 1000 seconds. The learning rate, which defines the degree of weight modification, is 0.005 [15]. At every time step, the activity levels of the units are initiated with a new arbitrary contrast edge stimulus. The position and the orientation of the stimulus are sampled from a uniform distribution. The stimulus width is 1º, and its spatial frequency is 1 cycle/degree. Observe that for convenience, the width and the spatial frequency of the stimulus is the same as that of the RFs. Noise is added to the activity levels through several steps. First a normally distributed noise with a standard deviation of 10% of the so-called bias value is added. The bias value is defined as the mean activity of an arbitrary unit inside a hypercolumn in absence of stimulus. The activity levels are rectified so that all negative activity levels are set to zero, and a 5-10% uniform distribution noise is added to all units to simulate the background activity. Later the activity levels are normalized so that the sum of activities in each hypercolumn is equal to one, as required by the incremental BCPNN learning rule. The contrast of the stimulus is 100%, though the effect of high noise in combination with the normalization procedure lowers this level considerably.

Recently we reported the behavior of a layer 4 network similar to the current network [25]. We saw that the long-range connections have facilitory effect on the units. This result was in line with the elongated summation pools proposed by Polat et al. [27]. The network could detect a noisy contrast edge much faster in presence of the intracortical connections. As revealed by the weight matrix, the modular specificity of the long-range connections targeting excitatory cells was prominent. Cross-orientation domains were inhibited through excitatory long-range connections targeting local inhibitory interneurons. However, quantitative values on corticocortical connections reported by Yousef et al. [11] and others are based on connections from cells located inside an injection site, which can be 200–400 μm across, i.e. roughly an iso-orientation domain. Inside such a patch we find cells with a broad range of orientation preferences. Note that the information available as the weight matrix is fundamentally different from the data on the connections from an injection site. As a consequence, an abstract injections site, which corresponds to a typical iso-orientation domain, is constructed (Fig. 10.2*B*). Spread of orientation preferences inside an orientation minicolumns is also an important factor while constructing an abstract injection site. Note that Murphy and Sillito [17] reported that orientation selectivity could vary 9–18º inside an

orientation minicolumn. To enable a quantitative assessment, the connections originating from the abstract injection site are divided into local connections, i.e. connections to the units inside the so-called host hypercolumn, and long-range connections, i.e. connections to units located in all other hypercolumns. The abstract injection site is located inside the host hypercolumn. Connection strengths from this abstract injection site is defined as the weighted sum of connections from each one of the units participating in the connection. The size of this weight corresponds to a fraction that defines the occurrence of neurons with a given orientation selectivity inside the injection site, which has the size of an iso-orientation domain (Fig. 10.2*B*). The strength of the connections is visualized as polar-plots (Fig. 10.1*A*). Connections to distal hypercolumns, which are >15% of the connections within the host hypercolumn are plotted and used during the quantitative assessment. Calculations show that 43.3% of the local connections target the iso-orientation domain (Fig. 10.2*A*). Oblique-orientation domain's share of the local connections is 33.1%, and the cross-orientation domain receives 23.6% of the local connections. However, the distribution of the excitatory long-range connections differs prominently. Only 35.5% are targeting the iso-orientation domains. The oblique- and cross-orientation domains receive, respectively, 32.4 and 32.1 per cent of the connections. These values are close to reported values of Yousef et al. [11]. To test the parameter dependencies, the abstract injection site was set to infinity, i.e. nearly the whole model. The distributions were almost equal for all three orientation domains, both for local and distal connection. Iso-orientation domains were targeted by 34.6% of the connections, while 33.7 and 31.7 per cent of the connections targeted oblique- and cross-orientation domains respectively.

The patchy layout of the layer 2/3 long-range connections is investigated by a series of simulations. The layer 2/3 network used during the simulations has similar set up as the previous networks, which were used to investigate the layer 4 connectivity. However, now the units have complex cell RFs, and hence only 12 units are needed in each hypercolumn instead of 24 as earlier. Input to a complex cell unit is defined as the sum of rectified inputs to the anti-correlation unit pairs, found in the layer 4 network, having the same orientation selectivity as the complex cell unit. Note that the anti-correlation pairs have same orientation selectivity but opposite relative spatial phase, i.e. they detect the same orientation but opposite phase. We hypothesize that the patchy layout of the layer 2/3 network is, at least partially, a consequence of excitatory cells targeting mainly other excitatory cells located in distal iso-orientation domains. To test this hypothesis the excitatory connections targeting the inhibitory interneurons are removed (Fig. 10.1*B*). The distribution of the long-range connections from the abstract injection site is 58.4% to iso-, 31.4% to oblique- and 10.2% to cross-orientation domains (Fig. 10.2*A*). Local connections show a similar pattern. The iso-orientation domain receives 54.3% of the connections. Oblique- and cross- orientation domains receive 33.4 and 12.3 per cent respectively. Kisvárday et al. [10] report a similar

distribution for the local connections, whereas the long-range connections reported by Kisvárday et al. [10] are less orientation specific.

The fall-off with distance for both layer 4 and layer 2/3 networks is less than the reported values by Chisum et al. [8]. We found that the degree of overlap between RFs of units situated in adjacent hypercolumns is one decisive factor that controls the degree of fall-off. When the distance between the RF centers of two adjacent hypercolumns increased, the extent of the long-range connections is reduced (not shown here). A consequence of the fall-off with distance is dense local and sparse distal connectivity demonstrated by both networks. Furthermore, the elongated shape of the long-range connections of both networks matches well the RF aspect ratio of the units. These findings are in line with the reported similarities between RF shape and the extent of the long-range connections by Chisum et al. [8]. Furthermore, preliminary results indicate that both networks can detect low contrast (15%) stimulus, defined as contrast edge. However, to confirm this observation and investigate the optimal extent of the long-range horizontal connections, in relation to the RF sizes and shapes, additional simulations must be carried out.

10.4 Discussion

We presented a quantitative assessment of the layer 4 and layer 2/3 local and long-range horizontal connections based on two separate models. Our results show that layer 4 long-range horizontal connections target all orientation domains in a balanced manner, whereas layer 4 local connections are biased towards the iso-orientation domain. However, the layer 2/3 network is significantly different. Both local and long-range horizontal connections of the layer 2/3 network are biased towards the iso-orientation domains. We hypothesize that the patchy layout of the long-range connections is a consequence of excitatory long-range connections targeting mainly other excitatory cells located in distal iso-orientation domains. Furthermore, the fall-off with distance results in dense local and sparse distal connectivity for both networks. Preliminary results indicate that the layer 2/3 network, like the layer 4 network, can detect low contrast stimulus. We intend to confirm this observation by further simulations in the near future. The optimal extent of the long-range horizontal connections, in relation to the RF sizes and shapes, needs also be investigated more carefully.

10.5 References

[1] 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.

[2] R.C. Reid & J.M. Alonso, Specificity of monosynaptic connections from thalamus to visual cortex. *Nature*, 378:281–284, 1995.

[3] D. Ferster & K.D. Miller, Neural Mechanisms of Orientation Selectivity in the Visual Cortex. *Annual Reviews of Neurosci.*, 23:441–471, 2000.

[4] J.A. Hirch, J.-M., Alonso C. Pillai & C. Pierre, Simple and complex inhibitory cells in layer 4 of cat visual cortex. *Soc. Neurosci. Abstr.*, 26, 1083, 2000.

[5] U. Polat & A.M. Norcia, Elongated physiological summation pools in the human visual cortex. *Vision Res.*, 38, 3735–3741, 1998.

[6] V.B. Mounthcastle, Modality and topographic properties of single neurons of cat's somatic sensory cortex, *J. Neurophysiol.*, 20:408–434, 1957.

[7] K.E. Schmidt, D.-S. Kim, W. Singer, T. Bonhoeffer & S. Löwel, Functional Specificity of Long-Range Intrinsic and Interhemispheric Connections in the Visual Cortex of Strabismic Cats, *J. Neurosci.*, 17(14):5480–5492, 1997.

[8] H.J. Chisum, F. Mooser & D. Fitzpatrick, Emergent Properties of Layer 2/3 Neurons Reflect the Collinear Arrangement of Horizontal Connections in Tree Shrew Visual Cortex, *J. Neurosci.*, 23(7):2947–2960, 2003.

[9] C.D. Gilbert & T.N. Wiesel, Columnar specificity of intrinsic horizontal connections and corticocortical connections in cat visual cortex. *J. Neurosci.*, 9:2432–2442, 1989.

[10] Z.F. Kisvárday, E. Tóth, M. Rausch & U.T. Eysel, Orientation-specific Relationship Between Populations of Excitatory and Inhibitory Lateral Connections in the Visual Cortex of the Cat. *Cerebral Cortex*, 7, 605–618, 1997.

[11] T. Yousef, T. Bonhoeffer, D.-S. Kim, U.T. Eysel, E. Tóth & Z.F. Kisvárday, Orientation topography of layer 4 lateral networks revealed by optical imagining in cat visual cortex (area 18). *E. J. of Neurosci.*, 11:4291–4308, 1999.

[12] W.H. Bosking, Y. Zhang, B. Schofield & D. Fitzpatrick, Orientation Selectivity and the Arrangement of Horizontal Connections in Tree Shrew Striate Cortex. *J. Neurosci.*, 17(6):2112–2127, 1997.

[13] K.S. Rockland & J.S. Lund, Widespread periodic intrinsic connections in the tree shrew visual cortex. *Science*, 215, 1532–1534, 1982.

[14] K.E. Schmidt & S. Löwel, Long-range Intrinsic Connections in Cat Primary Visual Cortex. In B.R. Payne, A. Peters (eds.). *The Cat Primary Visual Cortex*, Chapter 10. Ac. Press, 2002.

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

[16] V.B. Mountcastle, The columnar organization of the neocortex, *Brain*, 120, 701–722, 1997.

[17] P.C. Murphy & A.M. Sillito, Continuity of orientation columns between superficial and deep laminea of cat primary visual cortex, *J. Physiol.*, 381, 95–110, 1986.

[18] V. Braitenberg & C. Braitenberg, Geometry of the orientation columns in the visual cortex. *Biological Cybernetics*, 33:179–186, 1979.

[19] R.L. De Valois & K.K. De Valois, *Spatial Vision*, Chapter 4, Oxford Sci. Pub, 1990.

[20] S. Chung & D. Ferster Strength and Orientation Tuning of the Thalamic Input to Simple Cells Revealed by Electrical Evoked Cortical Suppression. *Neuron*, 20, 1177–1189, 1998.

[21] G. Sclar & R.D. Freeman, Orientation selectivity in the cat's striate cortex is invariant with stimulus contrast, *Exp. Brain Res*., 46:457–461, 1982.

[22] D. Ferster, Spatially opponent excitation and inhibition in simple cells of the cat visual cortex, *J. Neurosci.*, 8, 1172–1180, 1988.

[23] T.W. Troyer, A.E. Krukowski & K.D. Miller, LGN Input to Simple Cells and Contrast-Invariant Orientation Tuning: An Analysis. *J. Neurophysiol.*, 87:2741–2752, 2002.

[24] A. Sandberg, A. Lansner, F.M. Petersson & Ö. Ekeberg, A Bayesian attractor network with incremental learning. *Network: Computing in Neural Systems*, 13(2):179–194, 2002.

[25] B. Çürüklü & A. Lansner, Layout and Function of the Distal Projections within the Striate Cortex, *submitted to NIPS*, 2003.

[26] D.O. Hebb, *The Organization of Behavior*, New York: Wiley, 1949.

[27] U. Polat & A.M. Norcia, Elongated physiological summation pools in the human visual cortex. *Vision Res.*, 38, 3735–3741, 1998.

[28] B. Çürüklü & A. Lansner, An Abstract Model of a Cortical Hypercolumn, *Proc. of Int. Conf. on Neural Information Processing*, 80–85, 2002.