Modelling Primary Visual Cortex Dynamics with a Dynamic Neural Field Based on Voltage Sensitive Dyes

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Contents

Introduction 1

1. The visual system of mammals 7
   1.1. The retina .............................................. 8
   1.1.1. The concept of receptive fields .......... 9
   1.2. The lateral geniculate nucleus ................. 10
   1.3. The primary visual cortex (V1) .................. 11
       1.3.1. Dorsal and ventral stream .............. 14

2. The functional architecture of cat’s primary visual cortex 15
   2.1. The composition of area 17 ....................... 16
   2.2. Non-pyramidal cells ............................... 18
   2.3. Functional columns in cat primary visual cortex 18
   2.4. Long-ranging lateral excitatory and inhibitory connections 19
   2.5. The cortical circuitry in cat area 17 .......... 20
   2.6. The excitatory output projects to higher brain areas 21

3. Voltage sensitive dye imaging data 23
   3.1. The visual stimuli ..................................... 26
       3.1.1. Voltage sensitive dye imaging of the line-motion effect in cat area 18 28

4. Previous work 33
   4.1. The Line-Motion illusion .......................... 33
       4.1.1. Where in the brain arises the line-motion effect? ... 34
       4.1.2. A recent model simulates the LM-effect .......... 35
   4.2. Latency compensation in the visual pathway .......... 36
       4.2.1. Differential latency times in neural processing .... 37
       4.2.2. Motion extrapolation at the retina ............ 38
       4.2.3. Electrophysiology in early visual cortex ........ 38
5. Simulating the primary visual cortex: a dynamic neural field 41
  5.1. Dynamic Neural Fields so far 43
    5.1.1. Wilson and Cowan’s excitatory-inhibitory network 43
    5.1.2. Amari’s dynamic neural field 45
    5.1.3. Erlhagen’s excitatory-inhibitory network 47
  5.2. A DNF to simulate VSDI-signals 48
    5.2.1. A dynamic neural field approach based on Wilson and
            Cowan’s excitatory-inhibitory network 49
  5.3. Divisive inhibition in the lateral geniculate nucleus 52
  5.4. Solving neural field equations, Euler’s method 54
  5.5. Runge-Kutta’s method 55

6. Optimising the parameters 57
  6.1. Application of the covariance matrix adaptation algorithm 57
  6.2. Redundant parameters in the DNF 58
  6.3. Calculating the parameter $\lambda$ 59
  6.4. Parameters used in the DNF 61
    6.4.1. Balancing the excitation and inhibition 62
    6.4.2. Ratio of excitatory and inhibitory activity in the dye-signals 64
  6.5. Analysis of stability 65

7. Results 73
  7.1. Square, bar, LM and drawn-out bar 74
    7.1.1. Non-linear effects in the dye-signals 78
    7.1.2. Propagation speed of high and low fluorescence signals 78
    7.1.3. Simulating the LGN: suppression effects by normalisation 80
    7.1.4. Effects of an imbalance of excitation and inhibition 82
  7.2. Simulating the excitatory and inhibitory activations separately 85
  7.3. Early excitation, late inhibition 87
  7.4. Latency compensation in the primary visual cortex predicted by
       the DNF 88
    7.4.1. Preactivation via lateral connections accelerates the processing
           of moving stimuli 93
  7.5. Model predictions: The reverse line-motion illusion 94

8. A comparison of model performances 97
## Contents

9. Discussion 101  
9.1. The dynamic neural field with preceding normalisation 102  
9.1.1. The simulations ........................................ 102  
9.2. The excitatory output transmits signatures of perceptual correlates 104  
9.3. Physiological interpretation of the parameters .......................... 105  
9.3.1. Lateral connectivity of excitatory and inhibitory neurons in the primary visual cortex .............................. 105  
9.3.2. Inhibition observed in the VSDI-signals .......................... 107  
9.4. The emergence of illusory motion in the primary visual cortex .. 108  

10. Outlook 111  

A. Mathematical background 113  
A.1. Fourier-transform ........................................... 113  
A.2. The correlation coefficient and the linear regression .............. 114  

B. The CMA evolution strategy 117
Abstract

Interaction dynamics of excitatory and inhibitory neural activities driven by visual input determine the information being transmitted from the primary visual cortex to higher brain areas. In this work, these interaction dynamics are simulated on the level of large populations of neurons. The basis of this modelling study form Voltage Sensitive Dye Imaging (VSDI) data derived from cat area 18 in which simple shaped stimuli, (1) a moving square, (2) a square drawn out to bar length and (3) a square briefly followed by a flashed bar, evoked similar dye signals. All three stimulus settings produced cortical responses, which were gradually drawn-out, forming ‘motion streak’-like activity patterns. Hence, measured cortical activity of the first stimulus mismatched perception of a localised moving square. The activity in response to the drawn-out bar resembled perception, also the activity pattern in response to the third paradigm: sensation of illusory line-motion, despite presentation of stationary discrete stimuli (square then bar).

In this thesis, a dynamic neural field (DNF) model of early visual processing simulates the observed VSDI dynamics and accounts for the respective perceptual phenomena. A preceding normalisation model of the lateral geniculate nucleus is used to compensate for too much spatial and temporal integration. Top-down mechanisms from higher brain areas were shown to be unnecessary to produce the desired cortical responses in the model.

The ambiguities in the dye-signal responses evoked by the three stimuli configurations could be solved by separately depicting the simulated excitatory and inhibitory signals. The simulated dye signals were dominated by effective inhibitory signals, however, the simulated excitatory signals correlated more strongly to perception than the patterns of inhibition. In the excitatory layer, the moving square stimulus (1) evoked responses of a local patch in motion, clearly distinguishable from the responses evoked by the drawn-out bar (2) and the square followed by a bar (3) stimulus. Thus, the excitatory activity, which is assumed to be read out by higher brain areas, is in accordance with the presumed perception.

Further, the model accounts for VSDI-signals evoked by square stimuli with different velocities. Latency compensation phenomena observed in these VSDI-
responses are simulated and discussed in the context of efficient computing at early stages of cortical visual processing.

In conclusion, the model accounts for the measured primary visual cortex dynamics and perceptual phenomena without involving higher brain areas by distinguishing between excitatory and inhibitory signals, which are aggregated in VSDI of cortical circuits.
Introduction

The perception of moving objects is an important task in our every-day life. However, the neural processing evoked by moving stimuli is not fully understood until now. Especially the role of the primary visual cortex in the processing of motion attributes reveals unknown.

In this work neural activities in early processing stages of the visual pathway are studied in response to artificial stimuli that move or apparently move. Apparent motion evokes the phenomenon of the perception of motion, although in fact stationary stimuli have been presented. Artificial visual stimuli can be used to evoke such illusory motion perception [Exn88, Wer12, Knk13]. In nature, objects do not usually appear, disappear, grow or shrink within a timespan of milliseconds. However, on a computer monitor we can create artificial visual stimuli with such properties. So how does the brain, which is trained on natural stimuli, perform in such an artificial environment? It can be observed that some artificial stimuli are able to outwit the brain: When presenting a bar after briefly flashing a square that is aligned to the top of the bar, the bar does not seem to appear at once, but as drawn-out from the place where the square stimulus has been presented. This illusory sensation of motion is termed line-motion illusion and was first reported by HIKOSAKA et al. [Hik93a].

Though artificial visual stimuli do not occur in nature, their experimental relevance is huge, as they can be used to reveal basic properties of brain functioning. An artificial stimulus that creates motion perception although a non-moving stimulus has been presented makes it possible to investigate origins and mechanisms underlying motion sensation. The question arised, where in the brain the line-motion effect is produced. While HIKOSAKA et al. argued for attention effects that are responsible for the line-motion sensation [Hik93a, Hik93b], JANCKE et al. proposed a bottom-up theory [Jan04a], where the line-motion effect is already observed in the primary visual cortex. JANCKE et al. used real time optical imaging with voltage sensitive dyes in area 18 of anaesthetised cats. The authors measured the responses to simple artificial stimuli configurations including the line-motion paradigm. They could show that the line-motion stimulus evokes in cat area 18 a ‘motion-streak’ like response indistinguishable from what real motion would evoke in this area.
In this work, simulations of \textit{voltage sensitive dye imaging (VSDI)} data measured by Jancke et al. at the \textit{The Weizmann Institute of Science} in Israel on anaesthetised cat’s area 18 are presented. ‘At present, imaging based on voltage-sensitive dyes (VSD) offers the highest spatial and temporal resolution for imaging neocortical functions in the living brain, and has paved the way for a new era in the functional imaging of cortical dynamics.’ [Gri04]. To reveal cortical functioning, both a high spatial and temporal resolution are needed. Grinvald and Hildesheim argued that a temporal resolution in the range of milliseconds is required as neural communication is mediated within this temporal regime and a spatial resolution should fall below 200 micrometres to comply with the columnar architecture of the cortex [Gri04]. Further, sub- as well as suprathreshold activities contribute to neural computations, so that an imaging of both is needed. VSDI is the imaging technique with the highest spatial and temporal resolution when imaging the living cortex, satisfying the mentioned resolution values. Further, VSDI depicts both sub- and suprathreshold activities. Herewith, VSDI allows to observe spatio-temporal patterns of sub- and suprathreshold activities in real-time of nearly the whole cat’s area 18. When studying and simulating motion processing in the primary visual cortex, an imaging with VSDs offers important information on the principles underlying the neural processing, as a real-time imaging with a high spatial resolution is necessary to investigate the responses evoked by moving stimuli.

The stimuli presented in the VSDI-experiments of Jancke et al. comprise simple shaped square and bar stimuli, including the line-motion stimulus and moving square stimuli with different velocities.

Some parts of the imaging data used in this study have been simulated by an integrate and fire approach of Rangan et al. [Ran05] and by a \textit{dynamic neural field (DNF)} study of Meyer et al. [Mey06]. These models account for the LM-effect without including simulated attention effects, which supports the bottom-up theory of Jancke et al. [Jan04a].

In this work, a DNF model based on Wilson and Cowan’s excitatory-inhibitory network [Wil72, Wil73] was used to account for primary visual cortex dynamics. The DNF has been designed as a model that accounts for the basic interplay between excitation and inhibition in the primary visual cortex with a well controllable set of biologically interpretable parameters. The output of the system was defined by a linear combination of the DNF’s excitatory and inhibitory responses. This allows a separate study of the excitatory and inhibitory activations without affecting interactions between both populations. The well controllable and biologically plausible parameter set was optimised by an evolution strategy algorithm [Han97, Schn04] and additional fine-tuning to fit the
VSD signals.

In VSDI, one image pixel represents the average response of hundreds of neurons including pre- and postsynaptic as well as glial cell activity. Therefore, a mean field approach in which one simulated neuron represents the mean activation of the population of neurons underlying one dye signal has been used. As the VSDI-technique does not allow a distinction between excitatory and inhibitory activities, simulating the interacting excitatory and inhibitory activities offers to depict both layers separately and herewith new insights into the interplay of excitation and inhibition and its mutual challenging and balancing.

Jancke et al. tested three stimuli that evoke similar VSDI-signals [Jan04a]: (1) a moving square, (2) a square gradually drawn-out to bar length and (3) a bar presented after briefly flashing a square aligned to the top of the bar (line-motion (LM) stimulus). As shown by Hikosaka et al., the LM and the drawn-out bar stimulus evoke similar perceptions in humans. Jancke et al. showed that this illusory motion effect can already be observed in the primary visual cortex of anaesthetised cats [Jan04a]. The dye-signals in cat area 18 evoked by the LM-stimulus are indistinguishable from the responses evoked by the real moving (growing) drawn-out bar stimulus. Surprisingly, the dye signals evoked by the presentation of the moving square stimulus are similar to the dye-signals evoked by the LM and the drawn-out bar stimulus: However, we can clearly distinguish a local patch in motion from an expanding line: So why do the dye signals resemble each other although the percept is presumably different?

Studies on cat’s area PMLS (posteromedial lateral suprasylvian region) and monkey’s area MT (medial temporal area) show that the excitatory neurons in the primary visual cortex carry information to these higher areas [Els97, Ein91, Ans98]. A study of Einstein and Fitzpatrick [Ein91] used intracellular dye injections to show that all labelled neurons projecting from cat area 17 to area PMLS are spine-bearing. Spine-bearing cells are supposed to be excitatory [Pay02]. Cat’s area PMLS is supposed to be the analogon to primate’s area MT (medial temporal area) [Pay93]. Both areas are concerned with the processing of moving stimuli. As the excitatory neurons project to an area that is concerned with the processing of moving stimuli, the excitatory neurons in the primary visual cortex are likely to carry the information on motion perception. With a separate study of simulated excitatory and inhibitory neural activities in response to the three stimuli moving square (1), drawn-out bar (2) and LM (3) it becomes possible to test this hypothesis. The percept of the three stimuli was compared to the disassembled excitatory and inhibitory activities in the simulated dye-signals in this study.

It shall be mentioned that throughout this work, it is assumed that the per-
Introduction

ception of the LM is equal in cats and humans. It is also assumed within this study that the primary visual cortical responses to simple shaped stimuli in the anaesthetised cat resemble that of cats and humans that are awake.

The functional architecture of the primary visual cortex has been discussed to be involved in latency compensation effects in response to moving stimuli [Jan04b]. In this study, dye signals evoked by moving square stimuli of different velocities were investigated in the context of latency compensation. Latency compensation describes mechanisms that compensate for processing times in the brain. Although the visual processing takes time, we are able to judge accurately the position of a moving object. Latency compensation phenomena in the visual system have been discussed in the context of the so called flash-lag effect [Nij94]. The flash-lag effect denotes the following illusory perception: when one square is presented in motion and another square is flashed aligned to this moving square, observers perceive the moving square displaced toward the moving direction [Nij94]. In this work, it was investigated if there was evidence for latency compensation effects in the VSDI-data of cat’s primary visual cortex. If there were latency effects in the primary visual cortex that could even be simulated by the DNF, the interesting question is, whether these are due to intracortical mechanisms that also accounted for the illusory line motion. If the responses to a moving square stimulus do not lag behind the stimulus presentation in the primary visual cortex, we can assume that there are mechanisms that preactivate regions in which the observed object is possibly assumed to be located in the future. This preactivation will also be discussed to be the potential origin of the illusory perception of line-motion in this work.

This work is structured as follows:

In chapter 1, an introduction to the mammalian visual system is given. As this work is based on the results of psychophysical studies on the LM-illusion ([Hik93a], [Hik93b], [vGr94], [Schm97]) and because we are mostly interested in an understanding of the human visual system, the stress is on the primate visual system in this first chapter.

As the real-time optical imaging of the data underlying this study have been derived from cat’s primary visual cortex, the state of the art of the findings on the functional architecture of this area is summarised in chapter 2, with special attention given to the lateral connectivity and circuitry of excitatory and inhibitory neurons.

In chapter 3, an introduction to the VSDI-technique is given. The specific aspect of this technique is that it offers in contrast to other imaging techniques (EEG, IMRI, PET, MEG, optical imaging based on intrinsic signals, radioactive imaging of changes in blood flow or electrode recordings) both a high spatial

4
and temporal resolution, which allows to study real-time activities evoked by moving stimuli. Further, the technique allows to observe membrane potentials (including sub- and suprathreshold activities) of nearly the whole cat’s area 18. In this chapter some first trial-averaged responses from this area evoked by the artificial stimuli are presented.

In chapter 4, previous work on the LM paradigm will be introduced by especially addressing the issue of where in the brain the LM is supposed to arise. Then, a recent modelling study by RANGAN et al. [Ran05] on the VSDI-data of JANCKE et al. [Jan04a] will be presented. Finally, findings on latency compensation phenomena within the visual pathway are given in this chapter.

A brief historical introduction into DNFs is given in chapter 5. The approaches of WILSON and COWAN [Wil72], AMARI [Ama77] and ERLHAGEN [Erl03] will be introduced. After that, the DNF used in the simulations in this study is compared to these approaches. WILSON and COWAN’s excitatory-inhibitory network is introduced, because it is the first neural field approach that considers two populations of excitatory and inhibitory neurons. AMARI’s neural field is introduced, as the stability analysis of this DNF constitutes a milestone in the theory of DNFs. ERLHAGEN’s DNF is a compact and powerful modification of WILSON and COWAN’s network with coevally being more consistent with physiology. Some of these modifications were used in the DNF used in this study.

It was further shown to be necessary to use a— to the DNF— preceding normalisation model of the lateral geniculate nucleus (LGN) according to HEEGER [Hee92], which has been inspired by the findings of suppression effects in the LGN by FREEMAN et al. [Fre02]. The normalisation model compensates for too strong spatial and temporal activity integration, which is introduced in this chapter. The DNF with preceding normalisation used in this study has been designed to be as simple and as biologically straightforward as possible to account for the basic neural interplay of excitation and inhibition.

At the end of this chapter, two common methods to solve the DNF equations computationally are proposed, the EULER- and the RUNGE-KUTTA-method. In the simulations of this study, the RUNGE-KUTTA-method is used, as it delivers better approximate solutions within the same time.

The techniques underlying the parameter optimisation in this study are presented in chapter 6. Some of the parameters of the DNF are shown to deliver redundant information. The parameter that describes the proportion of excitatory and inhibitory signals in the dye-responses can be calculated with a simple linear program. The remaining parameters have been adapted by the application of an evolution strategy algorithm proposed by HANSEN and ÖSTERMEIER.
Introduction

[Han97] and additional fine-tuning by visual judgement. The chapter is closed with an analysis of stability on the DNF equations. For this analysis, a method according to Malsburg and Cowan [vdM81] is used. The authors consider small deviations from a stable solution, then linearise the system, such that it becomes solvable. This enables the authors to investigate for a fixed parameter setting, whether the system comes back to the stationary point (stable point) or runs away (unstable point).

The results of the simulations of the DNF and preceding LGN model are shown in chapter 7. The simulations evoked by the basic stimuli square, bar, LM and drawn-out bar are presented. Further the need for normalisation effects in the LGN is shown. To show what happens when the system looses its balance, simulations with a variation of four parameters that encode the extent of the excitatory and inhibitory neural spread and its strengths of interactions are given.

Thereafter, the announced study on the simulations of excitatory and inhibitory signals evoked by the moving square (1), drawn-out bar (2) and LM-stimulus (3) is presented. The activities of both interacting neuron populations are depicted. The responses of the excitatory and of the inhibitory neuron population were compared for all three stimuli settings. Further, it was shown that, due to the time constants of the DNF, an early response of the excitatory neurons followed by a later inhibition was observed.

The dye-signals of three experiments in response to moving square stimuli of velocities of 4, 8, 16, 32 and 64 degrees per second are presented and discussed in the context of latency compensation.

As a last result, the dye signals in responses to the so called reverse Line-Motion (rLM) stimulus, a bar followed by a square that is aligned to the top of the bar is predicted by DNF-simulations. The rLM stimulus is perceived by most observers as a shrinking line toward the position in which the square has been presented [vGr94, Schm97]. As the activity within the primary visual cortex in response to this stimulus has not been measured with VSDI yet, the question of interest is then: Can in the case of the reverse LM a motion streak be observed in the primary visual cortex as in the case of the LM-paradigm?

In chapter 8, a comparison on the performances of the DNF and of the integrate and fire model [Ran05] on the data of JANCKE et al. is given.

A detailed discussion on the physiological interpretation of the parameters used in the model is given in chapter 9. The reason for the LM- and rLM-effect in the primary visual cortex is discussed and it is addressed how these effects are connected to latency compensation phenomena and efficient computing.
Chapter 1.

The visual system of mammals

A lot of knowledge about the mammalian visual system has been derived from psychophysical tests, EEG and fMRI-studies on humans and studies on the monkey visual system. Because an understanding of the primate visual system is of so great interest to us, this first chapter is devoted to the mammalian visual system and mainly concentrates on the primates. This work is build up on former psychophysical studies by Hikosaka et al. [Hik93a, Hik93b], which also makes an introduction to the primate visual system indispensable. The results of this work are also compared with an fMRI-study on humans by Larsen et al. [Lar06] making it also essential to consider the primate’s visual system.

In this chapter, an overview of the parts of the central nervous system that are concerned with vision is given. Visual processing starts when the eyeground, the so called retina catches images of the outer world. When these images reach the retina in form of light, axons of retinal ganglion cells transfer as optic nerve fibres action potentials over the optic chiasm to the lateral geniculate nucleus (LGN). In the optic chiasm, the optic nerve fibres are divided in two parts before synapsing into the intermediate station LGN. Nerve fibres that originate from the right side of each retina project to the right LGN. Neurons located in the right LGN send their axons to the right cerebral hemisphere. The area within the left and right cerebral hemisphere, to which neurons from both sides of the LGN project to, is termed primary visual cortex. The collection of axons originating in the LGN that project to the primary visual cortex is termed optic radiation. As the retina has a convex shape, the right cerebral hemisphere receives visual information from the left visual field, as depicted in Fig. 1.1.

Light that falls on the left site of each retina is processed over the optic chiasm to the left LGN and from there to the left cerebral hemisphere that processes herewith the right visual field.

Those three main visual processing stages, retina, LGN and the visual cortex will be discussed in more detail in the following.
Chapter 1. The visual system of mammals

1.1. The retina

Although spatially separated from the brain, the retina is part of the central nervous system connected via the optic nerve to the brain. The retina has a layered structure of roughly five different cell types. The photoreceptors are located farthest from incoming light on the retina. There are two types of photoreceptors, rod and cone cells, that are responsible for night and day vision respectively. The proportion of rod and cone cells vary from mammal to mammal. Compared to humans, cats have a higher portion of rod cells allowing better night vision but less acuity and colour vision. The cone cells are distributed over the whole retina and have the highest density in the middle of the retina, where the resolution is highest. This area in the middle of the retina is called fovea centralis in the primate and area centralis in the cat and it is the area where objects are focused. Rod and cone cells contain light sensitive pigments. Rod cells all have the same pigment. Humans have three different types of cone cells with different pigments which are sensitive for light with different
1.1. The retina

The connectivity of cell types in the retina is sketched in Fig. 1.2:

Figure 1.2.: Eye and retina inspired by [Hub89]. The blue box represents a cut of the retina. Photoreceptors (rod and cone cells) catch the incoming light, transform changes to bipolar cells and from there to the retinal ganglion cells which evoke action potentials that are carried over their axons (optic nerves) to the LGN. Horizontal cells connect photoreceptors and bipolar cells, whereas amacrine cells connect bipolar and ganglion cells. Ganglion and amacrine cells produce action potentials whereas photoreceptors, bipolar and horizontal cells respond with graded signals.

Although farthest away from incoming light, photoreceptors catch the incoming light. Photoreceptors then project to bipolar cells which in turn are connected to the ganglion cells whose axons result in the optic nerve fibres which leave the retina. There are two further types of cells in the retina, the horizontal and amacrine cells which have lateral connections with photoreceptors and bipolar cells (horizontal cells) and with bipolar and ganglion cells (amacrine cells). Only amacrine and ganglion cells evoke action potentials, all other cell types in the retina give local graded membrane potential signals.

1.1.1. The concept of receptive fields

The population of rod and cone cells that supplies a given cell in the visual pathway is pooled on a small area on the retina and is not distributed over the whole retina. This area of the retina, that alters the firing rate of that considered neuron is called receptive field [Har38]. Illuminating outside the receptive field of a cell has no effect on the spiking on the regarded cell by definition.

The size of the receptive fields of ganglion cells changes strongly from the fovea centralis to more peripheral areas. In and near the fovea centralis one cone cell
supplies one bipolar cell and one bipolar cell in turn supplies one ganglion cell. In the periphery, several cone cells can converge to one bipolar cell and several bipolar cells in turn converge to one ganglion cell, so that there are relations of 125 : 1 photoreceptors to one optical nerve fibre [Hub89].

1.2. The lateral geniculate nucleus

The LGN has a special laminar structure. In the primate, the LGN consists of six cell layers: Cells in layers 1 and 2 receive input from retinal ganglion cells of the magnocellular cell type and cells in layers 3, 4, 5 and 6 receive input from the smaller parvocellular ganglion cells. Cells in layers 1, 4 and 6 get input from ganglion neurons of the contra-lateral eye and cells in layer 2, 3 and 5 from the ipsi-lateral eye. Different stimulus properties like movement and form of objects are processed in different pathways: First these different properties originate in different types of ganglion cells, then they are processed in separated regions of the LGN and after that they are processed in different cortical areas. Those two different pathways are termed magnocellular and parvocellular pathways respectively. The magnocellular pathway consists of cells predominantly sensitive to moving stimuli, where the parvocellular cells are most sensitive to form, contour and colour vision properties.

The receptive field-type of geniculate cells resemble that of ganglion cells in the retina and are called on-centre and off-centre type receptive fields. Those receptive fields have a concentric form. In case of ‘on-centre’-cells, centre-illumination (in the middle of the circle) and no surround illumination on the concentric receptive field activates the geniculate and ganglion on-centre cells best, see Figs. 1.3 and 1.4. Off-centre cells fire most when illuminating the surround and not illuminating the centre of the receptive field, see Fig. 1.3.

A further characteristic of LGN neurons (also of cortical neurons, see next section) is their retinotopic order within its layers: Neighbouring regions of the retina form connections to neighbouring neurons in the LGN. Therefore the receptive fields of neighbouring neurons in the LGN can overlap on the retina [Hub89]. Neurons within a region of the area centralis project onto a larger area in LGN than more peripherally located neurons on the retina. This effect of a larger representation of the fovea centralis can also be observed in the cortex.

To summarise, the LGN sorts the axons of the ganglion cells of the retina, so that neighbouring neurons from the same visual field supply neighbouring neurons within one layer of the LGN and neurons that compute similar properties are clustered together.
1.3. The primary visual cortex (V1)

The primary visual cortex is organised in six layers (I-VI). Most axons of geniculate cells end in layer IV. In the macaque monkey, layer IV is further subdivided into IVA, IVB, IVCo and IVC/β. Magnocellular cells end in IVCo and parvocellular cells in IVC/β. Axons of cells in layer VI project back to the LGN. Cells in layer V project down to the superior culliculus (concerned with eye movements and eye-head coordination) that is part of the midbrain and down to the pons that is part of the brain stem. Cells in layers II and III project to higher cortical areas V2, V3, V4 and V5, that are responsible for the perception of movement and form properties, which will be introduced later. Further vertical connections within the primary visual cortex (V1) are summarised in Fig. 1.5. Lateral connections within the primary visual cortex will be discussed in section 2.4 on findings in the cat primary visual cortex.

Another phenomenon of the primary visual cortex is the ocular dominance structure: Neurons from one layer of the LGN, that perceive information from the one eye, project to layer IV cells in separate regions than those which receive signals from neurons of the LGN treating the other eye. This results in a striped arrangement of cells in the visual cortex that mostly receive signals from the one or the other eye. Below and above layer IV cells, neurons receive more...
Chapter 1. The visual system of mammals

Figure 1.4.: On-centre ganglion cell responses related to [Dud01]. On-centre cells are most sensitive to illumination in the middle of their receptive fields (orange colour) and no illumination in their surround (blue colour in a). In b) the ganglion cell responses to an illumination with a spot of light on the according locations 1 to 5 in a) are depicted, where at the bottom of b) the timing of illumination is shown. The graph at the bottom of a) shows the responsiveness (from resting membrane potential) of the cell to illumination on the respective location on the retina.

signals from both eyes, but also predominantly from that eye that supplies the corresponding neurons in layer IV.

The receptive field structure of neurons in the primary visual cortex is very characteristic and has been discovered by HUBEL and WIESEL [Hub62]. The cells in the visual cortex are discussed to be categorized into simple, complex cells and hypercomplex cells which describe the receptive field type of the neurons: simple cells result from converging geniculate cells with displaced receptive fields aligned on a straight line, so that simple cells are predominantly sensitive to bars with a certain orientation located at a certain position in the visual field, see Fig. 1.6 for an example. Complex cells arise from converging simple cells with receptive fields of the same orientation but different receptive field positions. Therefore complex cells are mostly sensitive to bar-stimuli with a certain orientation but with arbitrary positions. There are further types of complex cells
1.3. The primary visual cortex (V1)

Figure 1.5.: Connectivity in the primary visual cortex. An overview of vertical connections: within the cortex to other layers, to other cortical areas, from subcortical to cortical cells and vice versa.

![Diagram of connectivity in the primary visual cortex](image1.png)

Figure 1.6.: Examples of simple cells’ receptive fields. On-centre (left) and off-centre-type (right) receptive fields of simple cells are shown. In case of the on-centre cell, light that falls on the elongated area in the middle of the receptive field activates the neuron, whereas light that falls on the surround inhibits the simple cells’ responses.

![Examples of simple cells' receptive fields](image2.png)

that are sensitive to edges and lines or edges that stop. The last mentioned cells are called end-inhibited complex cells. Complex cells that are sensitive to
elongated stimuli of a certain length are often referred to as *hypercomplex cells*. Some simple and complex cells are termed *direction sensitive* because of their predominant sensitivity to stimuli that move in a certain direction. So called *functional columns* of such specificities will be introduced in section 2.3 exemplary for the cat’s primary visual cortex.

### 1.3.1. Dorsal and ventral stream

There are two main visual pathways from human area V1 to higher brain areas. The terms *dorsal* and *ventral stream* denote those two pathways to brain areas that are associated with the perception of movement and form respectively. As shown in Fig. 1.5 neurons originating from the magnocellular layers of the LGN project to layer IVcα. Those neurons in layer IVcα in turn project to neurons in layer IVb from there again to area V2 forward to V3 and next to the more parietal *mediotemporal cortex* (MT) and *medial superior temporal cortex* (MST). In all these areas neurons are mostly sensitive to the direction of motion of a stimulus. In area MT, neurons are especially sensitive to moving stimuli on stationary background. Neurons in area MST respond particularly when moving stimuli in spatial depth are presented. This is the case when a person tries to reach for an object for example.

The parvocellular cells in the LGN project, as depicted in Fig. 1.5, to layer IVcβ and from there to layer II/III where a first division of the parvocellular pathway takes place. Some cells project to areas in layer II/III in which the cells are sensitive to orientation but not to colour and not to contrast features. Other cells project to special areas in layer II/III where the cells are most sensitive to colour and contrast features of the stimuli. Both cell areas project to separate areas in V2. The colour and contrast sensitive cells in V2 synapse in the ventral part of V3 and from there to V4. The neurons located in area V4 are herewith sensitive to colour and form properties of stimuli.

Both pathways, the dorsal (occipital-parietal) pathway and the ventral (occipital-temporal) pathway process in parallel but however V4 neurons also project to MT neurons, so that interaction between both pathways is permitted.
Chapter 2.

The functional architecture of cat’s primary visual cortex

A lot of knowledge about the mammalian visual system has been derived from studies on cats. Payne states that in the last thirty years, more than 5000 articles have been published on the topic of the cat visual cortex, with a great majority about the primary visual cortex [Pay02]. As the data underlying this modelling study have been derived from cat primary visual cortex, the functional architecture of this area shall be introduced in more detail.

Until the early 1970’s, the cat primary visual cortex has been considered to be equivalent to area 17. This understanding was due to the assumption that areas 18 and 19 process later stages of visual processing. Hubel and Wiesel even extended this view in [Hub65] by correlating area 17 with V1, area 18 with V2 and area 19 with V3. However, in those studies, the proportion of direct projections from LGN to area 18 have not been considered. These projections have been revealed later by the introduction of axoplasmic pathway tracers in the 1970’s and early 1980’s [Cow71]. Intra-axonal injection of markers into LGN axons shows an innervation of the upper part of layer 4 in area 17 and the upper two thirds of layer 4 in area 18 [Tret75]. Therefore, today the term primary visual cortex comprises both areas 17 and 18.

In the cat, there are three main LGN cell layers, called A, A1 and C. The input to the layers A and C originate from ganglion cells of the contra-lateral eye, whereas A1 is supplied with ganglion cell input from the ipsi-lateral eye. One distinguishes between X, W and Y-fibres that supply different sublayers of A, A1 and C. The so called X-pathway is comparable with the primate parvocellular pathway, the Y-pathway can be associated with the magnocellular pathway. Therefore Y-type neurons mainly process movement features of the visual scene.

Tretter et al. showed that in contrast to Y-type signals, X-type signals only provide area 17 and do not innervate area 18 [Tret75], which is depicted in Fig. 2.1. Therefore area 18 is mostly provided with the larger magnocellular cells, but also with W-type cells. The magnocellular cells are sensitive to motion more than to form and colour attributes. Area 18 is hence organised in parallel
to area 17 and deals with other aspects of visual information [Tret75].

As in this work especially moving and illusory moving stimuli are investigated, the underlying data have been derived from cat area 18. The different innervation of X-type and Y-type neurons in cat areas 17 and 18 is summarised in Fig. 2.1.

Cat’s area 17 has been intensively studied in respect of its composition and circuitry whereas comprehensive data on area 18 are missing [Pay02]. Golgi studies and studies using intracellular horseradish peroxidase/dye injection methods show that the cell types in area 18 are comparable to those located in area 17 [Pay02]. For that reason the cellular structure of cat’s area 17 is described in the next section.

2.1. The composition of area 17

So called pyramidal and spiney stellate cells are supposed to act as the excitatory neurons in cat area 17. Those two non-GABAergic cell types account for 80 per cent of the total population of neurons in cat area 17 [Bin04]. The remaining 20 per cent inhibitory and therefore GABAergic neurons are composed by
2.1. The composition of area 17

smooth or sparsely spineous\(^1\) cells referred to as *stellate cells* or *interneurons*. Those stellate cells have mostly local axonal projections, whereas pyramidal cells project as well locally and long-ranging [Schm98].

Area 17 consists of mainly six layers as shown in Fig. 1.5 and 2.1. The upper part of layer II is populated by small pyramidal cells. The pyramidal cell size increases with increasing depth until layer III. There is no distinct boundary between layers II and III so that they are often unified to layer II/III. In area 17, layer II/III makes about one third of the thickness of the cortex, in area 18 even more. Those pyramidal cells have lateral connections (perpendicular to the cortical surface) whose axons most likely connect distant groups of pyramidal cells with similar physiological characteristics: ROERIG and KAO showed that excitatory connections in ferret primary visual cortex link mainly neurons with similar motion direction sensitivity [Roe99]. There have been several studies which show that excitatory pyramidal connections link neurons with the same orientation specificity [Gil89, Kis97]. Especially the extent of the lateral spread of cortical activity is investigated in this work. Hence, layer II/III becomes in this context of great interest. The imaging technique underlying the data used in this study mainly depicts layer II/III.

Layer IV comprises about the middle third of area 17’s cortical thickness. Neurons are here smaller and more dense than in layer II/III. Most cells in layer IV are small spiny stellate cells, but also a few pyramidal cells can be found [Pay02]. The neurons in layer IVA are larger than those in layer IVB. Layer IVB cells also have fewer long dendrites than layer IVA cells. The separation of layer IV into two sublayers is further justified because of the inputs they receive from the LGN. As depicted in Fig. 2.1, area 17’s layer IVB almost exclusively receives input from X-type neurons, whereas layer IVA receives input from both X and Y-type neurons, but it is dominated by the input of Y-type neurons.

Layer V contains pyramidal cells of different sizes and shall be divided into two layers VA and VB. Layer VA consists of small and medium sized neurons that project to layer II/III after shortly extending laterally within layer VA. Layer VB consists of large, solitary pyramidal cells, but mostly of small pyramidal cells that project to layer I. The large pyramidal cells first project to layer IV and then again to layer II/III.

\(^1\)Spines describe mushroom-like protuberances on the dendrites of neurons where afferent axons form their synapses. It is supposed that on the spines, synaptic plasticity takes place. There are usually several 10,000 spines on the dendritic arbor of those neurons [Schm98]. Mostly excitatory neurons are supposed to have spineous dendrites.
Chapter 2. The functional architecture of cat’s primary visual cortex

2.2. Non-pyramidal cells

The non-pyramidal neurons in area 17 are inhibitory and they are termed stellate cells or interneurons. They all use GABA as their neurotransmitter and do not have spiny dendrites. Their axons mostly project locally within the cerebral cortex. Two types of such stellate cells, that are common in layer II/III, will be described here, where one also projects to more distant targets.

The so called chandelier cells are most common in layer II/III, but they are also present in layer V. Their axons synapse at short distances on the initial segments of the axons of pyramidal cells [Som82]. For that reason chandelier cells are often called axoaxonic cells. The chandelier cells are a powerful inhibitor of a number of efferent pyramidal neurons, where ipsilateral projecting (not crossing the corpus collosum) pyramidal cells have 20 to 43 axoaxonal synapses with chandelier cells [Far91, Som82].

Large basket cells also project to more distant targets and occur in layers II-VI. They have an elongated cell body and vertically elongated dendritic trees. Their axon that originates from one pole of the cell body forms three to five branches that extend up to 1.5 millimetres parallel to the cortical surface [Mar83]. They synapse with up to five cell bodies or dendrites of predominantly pyramidal cells, but also with other inhibitory neurons. Basket cells are assumed to be the only neurons in area 17 that form synapses with cell bodies [Bin04]. Binzegger et al. calculated that 42 per cent of the GABAergic neurons are basket cells [Bin04].

2.3. Functional columns in cat primary visual cortex

Hubel and Wiesel [Hub62] found out in 1962 that neurons in cat visual cortex studied along vertically displaced electrodes showed almost the same orientation preference. By shifting the electrodes in horizontal directions, the neurons’ orientation preference changes with smooth continuous shifts. Those vertical regions that extend over all layers I-VI and respond to similar stimulus properties are called functional columns. In case of orientation selectivity those columns are termed orientation columns.

The so called ocular dominance columnar system describes the existence of functional columns containing neurons that are most sensitive to stimuli presented to one eye. This columnar structure is not so strict, as it is not so easy to attribute to a cortical neuron exclusively the one or the other eye. Layer IV neurons (as the main input layer of axons originating in the LGN) show mostly
a clear ocular dominance but neurons in more superficial or in more deep layers are more sensible to stimuli presented to both eyes with a slight preference to the one eye.

The columnar structure of the orientation selectivity of neurons is directly linked to the so called direction of movement dominance as many orientation selective neurons in areas 17 and 18 are most sensitive to the two moving directions perpendicular to the preferred orientation. Neurons that are displaced horizontally on the cortex have motion direction selectivity properties that change smoothly but with an abrupt shift of 180 degrees (to the other direction) periodically. The movement and orientation selectivity of neurons that are horizontally displaced have elegantly been shown by PAYNE in form of a graphic that shows both the orientation and direction selectivity of horizontally displaced neurons [Pay02].

The so called retinotopy can also be regarded as a functional columnar system. As mentioned in chapter 1, neurons in the visual cortex that are nearby are sensitive to nearby stimuli on the retina. Neurons within the same column even view the same visual field.

Most neurons in area 17 and 18 are also sensitive to spatial frequency properties. Neurons in area 17/18 do not clearly show a columnar structure in this context. Neurons in layer IV prefer the highest spatial frequency, neurons in layer II/III prefer intermediate spatial frequencies and neurons in layer V/VI prefer the lowest spatial frequencies [Maf77].

2.4. Long-ranging lateral excitatory and inhibitory connections

It has been shown that the large majority of long-ranging lateral connections are mediated by pyramidal cells [Gil79]. However, large basket cells filled with horseradish peroxidase have also been shown to have long collateral branches over distances of up to 1.6 millimetres [Mar83]. According to SENGPIEL and VOROBYOV, long-range connections are defined here by distances that are larger than 500 millimetres [Sen05]. In 1989, GILBERT and WIESEL could show that the excitatory long-range connections are selective for similar orientations [Gil89]. KISVARDAY et al. investigated both excitatory and inhibitory lateral connections with respect to their orientation specifity [Kis97]. The authors could show that the excitatory lateral connections are more biased toward iso-orientation and that the inhibitory connections are more biased toward non-isoorientation stimulus properties. They applied small iontophoretic injections of
biocytin to the superficial layers of cat area 17 and 18. The injections showed axonal projections of excitatory cells of up to 3.5 millimetres both in area 17 and 18. The authors further mark in some experiments chiefly large basket cells, which revealed lateral connections of up to 2 millimetres.

Other groups could even show that these inhibitory connections achieve up to 3 millimetres extent from the injection centre ([Alb75], [Mas92]).

**Gilbert** and **Wiesel** used in [Gil96] optical recordings and found long-range horizontal pyramidal axonal projections of up to 8 millimetres in cat visual cortex [Gil96]. The authors propose that the lateral spread of activity depends—due to spatial integration effects—on the visual stimulus presented. When activating the cortex by presenting a minimal visual stimulus, they could show that the spiking area is on average 0.75 millimetres and that of both sub- and suprathreshold activity 4 millimetres.

**Bringuier** et al. term the field of sub- and suprathreshold activity in response to a visual stimulus *synaptic integration field* [Bri99]. The authors assume that the subthreshold activity surrounding the classical discharge field result from the integration of activation waves caused by horizontal connections within primary visual cortex. With intracellular recordings they could show that a focal visual stimulus evokes a radial activity wave that spreads over a radius of 10 millimetres.

### 2.5. The cortical circuitry in cat area 17

The synaptic input of most neurons in layer 2/3 is dominated by layer 2/3 pyramidal neurons, which form between 40-60 per cent of all synapses on the dendritic trees. Each layer 2/3 neuron receives on average 2094 ± 857 synapses from pyramidal cells [Bin04]. **Binzegger** et al. termed projections with up to 300 synapses *weak projections*. On average the authors could show that these weak projections form 98 ± 78 synapses with a target neuron and that there are much more weak than strong projections.

The major input to the cat’s visual cortex is mediated by projections from the LGN. **Ahmed** et al. showed that only five per cent of the excitatory synapses arose from the LGN [Ahm94]. Therefore intra-cortical projections and especially lateral connections within layer 2/3 play a major role as many cells in area 17 project within one layer: for example pyramidal cells in layer 2/3 project to 78 ± 9 per cent to layer 2/3 neurons and basket cells in layer 2/3 project to 93 ± 6 per cent to layer 2/3 neurons.

The average dendritic length of smooth neurons has been shown to be 4.7
2.6. The excitatory output projects to higher brain areas.

millimetres and of spiney neurons 6.9 millimetres, where most of the dendritic
trees of neurons in layer 2/3 are contained in the layer of soma (75 per cent of
dendritic length corresponds to 5.2 millimetres in case of spiney neurons and
corresponds to 3.5 millimetres in case of smooth neurons) [Bin04]. Binzegger et al. could further show that at least 34 per cent of the synapses in cat’s area 17 are mediated by axons running horizontally within a cortical layer and are involved in the self-innervation of the individual cortical layers.

2.6. The excitatory output projects to higher brain areas.

Higher brain areas (like area MT in primates or area PMLS in cats) predominantly (if not even exclusively) read-out excitatory neurons in the primary visual cortex [Els97, Ans98, Ein91]. Elston and Rosa injected in monkeys’ area MT neurons Fluoroemerald tracers to reveal retrograde labelling in V1 [Els97]. Of 41 MT-projecting cells that were successfully reconstructed, 28 (68.3 percent) were pyramidal and 13 (31.7 per cent) were spiney multipolar neurons. Therefore all found axons were spiney and therefore excitatory with a great portion of pyramidal cells. Anderson et al. state that the source of V1 afferents in V5 is known to be the spiney stellate cells of layer IVB and pyramidal cells of layer VI [Ans98]. Einstein and Fitzpatrick used intracellular dye injections in cat’s cortical slices to examine the distribution and morphology of area 17 neurons that project to area PMLS [Ein91]. They could show that most of the projections to area PMLS arise from superficial layers (90 percent from layer 2/3). Retrogradely labeled neurons that were intracellularly filled with Lucifer Yellow showed that all these projecting neurons were spine-bearing. As spine-bearing cells are assumed to be excitatory, we can conclude that, in the cat, area 17 neurons that project to area PMLS are excitatory. Area PMLS in cat is like monkey’s area MT concerned with motion processing and assumed to be its analogon [Pay93].
Chapter 3.

Voltage sensitive dye imaging data

The data underlying this study have been derived by JANCKE et al. at the Department of Neurobiology at The Weizmann Institute of Science in Israel with a so called voltage sensitive dye imaging (VSDI) technique on the early visual cortex of anaesthetised adult cats.

First recordings of optical signals using voltage-sensitive dyes have been done in the late 1960’s, see [Tas68]. In-vivo VSDI began in the 80’s [Gri84]. The voltage-sensitive dye molecule design and the advances of very fast cameras in recent years allow today a high resolution imaging with a temporal resolution in the range of milliseconds [Gri01].

Fig. 3.1 sketches the setup of the VSDI-experiments underlying this study: JANCKE et al. stained area 18 of anaesthetised adult cats with a suitable voltage sensitive dye (here dye molecule RH1691), see Fig. 3.2. Those dye molecules bind to the external surface of excitable membranes and transform changes of membrane potentials into optical signals. These signals are linearly correlated with membrane potential signals of layer II and III cortical neurons [Pet03, Ste98, Jan04a]. The dye-signals are recorded with a fast camera, digitised by a computer and monitored on a screen, see Fig. 3.1.

The chemical structure of the dye molecule RH1691, that has been used by JANCKE et al., is depicted on the top of Fig. 3.2. The dye molecule is very sensitive to its microenvironment, which is emphasised by the picture of the four solvents in which the dye molecule has been dissolved. This dissolving of the dye molecule results in four dissimilar fluorescences depicted below the molecule. On the bottom of Fig. 3.2 the assumed binding of the molecule to the lipid bilayer is depicted.

VSDI offers both a very high temporal and spatial resolution. The data underlying this study provide a spatial resolution of about 50 micrometres and a temporal resolution of 10 milliseconds. This high temporal and high spatial resolution allows to observe spatio-temporal activation patterns, which are of great interest when investigating moving stimulus configurations. Table 3.1 summarises the temporal and spatial resolutions of different imaging techniques.
Chapter 3. Voltage sensitive dye imaging data

Figure 3.1.: The setup of VSDI. (Picture taken from [Gri01]) The primary visual cortex (area 18) is stained with a voltage sensitive dye. The dye molecules bind to the external surface of excitable membranes and transform changes in membrane potentials into optical signals. While the cat’s eyes are focused on a monitor that presents visual stimuli, the optical signals of the dye-molecules are recorded by a fast camera, displayed on a colour screen and saved on a computer.

It can be seen that VSDI offers the highest spatial resolution. Nevertheless, the temporal resolution of VSDI is also very high. Compared to imaging techniques that depend on the blood oxygen level, like functional magnetic resonance imaging (fMRI) and intrinsic optical imaging (Intrinsic OI), VSDI offers a much better temporal resolution, as this blood oxygen level dependency binds

![Diagram](image)

<table>
<thead>
<tr>
<th>Imaging Technique</th>
<th>Temporal Resolution</th>
<th>Spatial Resolution</th>
</tr>
</thead>
<tbody>
<tr>
<td>fMRI</td>
<td>&gt; 1sec</td>
<td>&gt; 1mm</td>
</tr>
<tr>
<td>EEG</td>
<td>1ms</td>
<td>&gt; 3mm</td>
</tr>
<tr>
<td>MEG</td>
<td>1ms</td>
<td>2mm</td>
</tr>
<tr>
<td>PET</td>
<td>50ms</td>
<td>4 – 5mm</td>
</tr>
<tr>
<td>Intrinsic OI</td>
<td>&gt; 1sec</td>
<td>50µm</td>
</tr>
<tr>
<td>VSDI</td>
<td>10ms</td>
<td>50µm</td>
</tr>
</tbody>
</table>

Table 3.1.: Temporal and spatial resolutions of different imaging techniques.
Figure 3.2: The molecular structure of the dye-molecule RH 1691. (A modified picture from [Gri01]) On the top, the molecular structure of the dye molecule RH 1691 is depicted. Below, four different solvents with different polarities in which the dye molecule has been dissolved are shown. The large changes in colour indicate the sensitivity of the dye molecule to its microenvironment. On the bottom, a schematic presentation of how the dye-molecules bind to a membrane is depicted. Once, the dye-molecule binds to the lipid bilayer (yellow colours), it becomes fluorescent, shown with red coloured dyes. A change in electric field across the bilayer causes changes in the intensity of the dye-molecule fluorescence in linear dependence [Ste98].

the temporal resolution to a scale of not better than one second. In contrast to these techniques, the so called electro encephalography (EEG) and magnetic encephalography (MEG) are bounded by the coarse spatial resolution.

VSDI-responses represent an overall activity of neurons, where one measured image pixel represents the average membrane potential of hundreds of neurons and axons irrespective of their type and function. This comprises pre- and post-synaptic neuronal activity as well as depolarisations of neighbouring glial cells. The glial cell binding of the dye-molecules can be neglected, as the dye signals can only show very slow changes on glial cells [Gri04] that is outside
the range of the timescale of the measured data. As the imaging technique measures average responses of membrane potentials, it can easily detect slow subthreshold synaptic potentials in the extensive dendritic arborisation [Gri01], which cannot be measured by single cell recordings. One image pixel measures an area of 50 to 50 micrometres and depicts herewith about 250-500 neurons and their processes [Gri01]. As VSDI measures the mean responses of hundreds of neurons, it is not possible to distinguish between excitatory and inhibitory neural contributions. As the interplay between the excitatory and inhibitory population, hidden in the VSDI signals, might deliver important information on brain functioning, we need a method to separate these responses from each other. A dynamic neural field (DNF) related to WILSON and COWAN’s excitatory-inhibitory network will simulate the VSDI-data and will coevally allow us to simulate excitation and inhibition separately without stopping the interaction between both populations, as shown later on in this work.

3.1. The visual stimuli

The visual stimuli presented to the anaesthetised cats can be categorised into stationary, moving and illusory moving stimuli. The stationary stimuli comprise a square and a bar stimulus, where both stimuli are of equal width and aligned with their upper edges. The bar stimulus is four times longer along the vertical axis. The two stationary stimuli, the square and the bar, are depicted in Fig. 3.3a and b in the two dark grey boxes. The two-dimensional stimulus presentation is given in the upper picture within each box. The so called space-time diagram, that results from the two dimensional stimulus presentation by averaging along the horizontal axis, is shown in each box on the bottom.

This projection from two dimensions to one dimension causes no information loss as all stimuli used in this study provide redundant information when moving along the horizontal axis.

The moving stimuli configurations are depicted in the brighter grey boxes in Fig. 3.3 in an analogous manner: Fig. 3.3d shows the so called drawn-out bar stimulus, a square that gradually draws out to bar length. In Fig 3.3e, square stimuli are sketched that move gradually from top to bottom on the screen with different velocities (4, 8, 16, 32 and 64 degree per second).

In the white box in Fig. 3.3d the illusory line-motion (LM) stimulus is shown, a square briefly presented before presenting an aligned bar stimulus. HIKOSAKA et al. have introduced the LM paradigm [Hik93a]: Observers perceive the line-motion stimulus, that is in fact a stationary stimulus, as if the square continu-
3.1. The visual stimuli

Figure 3.3.: Visual stimuli. Dark grey boxes show stationary, bright grey boxes moving stimuli, and the white box shows an illusory moving stimulus. a) The square, b) the bar, c) the LM, d) drawn-out bar and e) the moving square stimuli. Moving square stimuli are presented with velocities of 4, 8, 16, 32 and 64 degrees per second. On the top, the two-dimensional stimulus presentation is depicted, where in each lower picture the projection to a one-dimensional space (space-time diagram) is shown.

The temporal arrangement of the LM stimulus as well as the square and bar
Stimuli onsets and durations

<table>
<thead>
<tr>
<th>Time Interval</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>0-50ms</td>
<td>Square stimulus presented for 50 milliseconds.</td>
</tr>
<tr>
<td>50-60ms</td>
<td>Bar stimulus presented after 60 milliseconds and holds on for 130 milliseconds.</td>
</tr>
<tr>
<td>60-190ms</td>
<td>Inter stimulus interval (ISI) of 10 milliseconds in turn followed by the bar stimulus for 130 milliseconds.</td>
</tr>
</tbody>
</table>

**Figure 3.4:** Timing of the visual stimuli. The square stimulus is presented for 50 milliseconds. The bar stimulus is presented after 60 milliseconds and holds on for 130 milliseconds. In the line-motion setting, the square stimulus is presented for 50 milliseconds followed by an ISI of 10 milliseconds in turn followed by the bar stimulus for 130 milliseconds.

3.1.1. Voltage sensitive dye imaging of the line-motion effect in cat area 18

The space-time diagrams of average (averaged across 16 trials of the same experiment) dye signal responses are depicted in Fig. 3.5. The dye signals have been evoked by the presentation of the square (a), bar (b), LM (c) and drawn-out bar stimulus (d). On the left side, the stimuli projected to one cortical dimension are shown, whereas on the right side the corresponding stimulus evoked VSDI-responses are shown, that have been projected to one cortical dimension. In Fig. 3.6, this projection to one cortical dimension is schematically shown for the dye-signals evoked by the LM stimulus.

Presenting a square stimulus evokes an initial far reaching spread of low fluorescence areas (bluish colours), see 3.5a right. The high fluorescence signals (red colours) stay rather locally restricted about the corresponding stimulus size. The same is the case when presenting a bar stimulus with 60 milliseconds later
3.1. The visual stimuli

Figure 3.5.: Visual stimuli and VSDI data of the experiment of the 8th of May 2001. On the left side, the visual stimuli are shown in space-time diagrams. Colour represents light intensity. On the right side the evoked VSDI-responses (averaged across 16 trials) are depicted, where colour indicates fluorescence changes (blueish colours indicate low fluorescence values and reddish colour high fluorescence values, see colour-bars on the right). The following stimuli have been tested: a the square, b the bar, c the line-motion and d the drawn-out bar. The drawn-out bar stimulus was tested in a different experiment as the other three stimuli. Therefore the different colour-bars of the fluorescence signals (right) are needed.

onset. First, low fluorescence signals spread out fast and then high fluorescence signals stay rather locally covering about stimulus size, see Fig 3.5b.
Chapter 3. Voltage sensitive dye imaging data

Figure 3.6: Space-time diagram of the VSDI-signals evoked by the LM-stimulus. The two dimensional pictures are averaged over the horizontal dimension. Those averaged stripes placed in temporal course result in so called space-time diagrams.

When presenting first a square stimulus and then with 10 milliseconds ISI the bar stimulus (LM stimulus), see Fig. 3.5c, the low fluorescence signals caused by the presentation of the square stimulus spreads out very fast. When then the bar stimulus evokes its responses, these responses are ‘embedded’ into the square evoked low fluorescence signals, which causes the signals near by the square position to evoke high fluorescence (red area) earlier, so that the high fluorescence area gradually draws-out Fig. 3.5c. The gradual spread of high-fluorescence signals evoked by the LM stimulus resembles that of real motion when presenting a drawn-out bar stimulus, see Fig. 3.5d. This effect of observing motion correlates in the primary visual cortex although the stationary LM has been presented is referred to as LM-effect [Jan04a]. JANcke and others measured the responses to the drawn-out bar stimulus in a different experiment (also different cat), so that the stained and depicted area is slightly more shifted to the top. In Fig. 3.7, the dye-signal responses evoked by the moving square stimulus (32 degrees per second), drawn-out bar stimulus and LM stim-
3.1. The visual stimuli

Figure 3.7: VSDI-signals in response to the moving square, drawn-out bar and LM stimulus. Two-dimensional VSDI-signals (averaged across 16 trials) with according space-time diagrams, see Fig. 3.6, in response to the moving square (32 degrees per second), drawn-out bar and LM stimulus. The drawn-out bar stimulus evoked dye-signals have been measured in a different experiment than the moving square and LM evoked dye-signals.

For all three stimuli are depicted in two dimensional (upper) and one-dimensional space-time diagrams (below). It can be seen that all three stimuli evoke ‘motion-streak’
like responses, that are indistinguishable from each other. The response to the
drawn-out bar stimulus has been measured in a different experiment, in which
the responses tend to decay earlier and are a little bit weaker. These three qual-
itatively similar evoked dye-signals responses partly mismatch perception: The
moving square stimulus is perceived as a local patch in motion and not as an
expanding line. But the similarity between the LM and drawn-out bar stimulus
matches perception, as both stimuli are perceived as continuously growing out
to bar length.

Note that in all three cases, the fast spread of low fluorescence signals and
the continuous spread of the high fluorescence area can be observed. Note also
that JANCKE et al. used in some experiments extracellular recordings to prove
that high measured fluorescence areas correspond to spiking activity [Jan04a].
Chapter 4.

Previous work

This work is based on previous findings on the LM-paradigm, comprising several studies that investigate the origin of this illusion. One study additionally proposes a modelling of the effect that might account for the illusory motion perception. Furthermore, in the discussion of this work, see chapter 9, the reason for the emergence, that means the benefit from the mechanism underlying this illusion, is discussed and directly connected to latency compensation effects within the visual pathway. Several works have been proposed on latency compensation in the visual system, where this study is also based on.

The recent findings on these two main topics, ‘The Line-Motion illusion’ and ‘Latency compensation in the visual pathway’ are proposed in this chapter. An introduction to the ‘modelling-structure’ used in the simulations of this study, the so called dynamic neural fields, is given separately in the next chapter.

4.1. The Line-Motion illusion

The line-motion (LM) paradigm has been introduced by Hikosaka et al. to study effects of visual attention on perception [Hik93a]. The authors suggest that the preceding square stimulus focuses the observer’s attention and with that, information processing is accelerated at the side of attention. The authors claim that acceleration of attention causes the LM illusion, as the line is perceived as drawing out from the side of the preceding cue stimulus. Hikosaka et al. define attention as follows: ‘Attention may be defined as the state of mind in which a limited portion of sensory processing is temporarily facilitated while the other portion may be suppressed.’

Hikosaka and Miyauchi distinguish between active (voluntary) and passive (stimulus driven) attention [Hik93b]. In the active case, the test persons have been asked to direct their attention to one of two cue stimuli which are located on both sides of the aligned bar. In the passive case, one of the two cues flashes before the presentation of the bar without active attention of the observer. Hikosaka and Miyauchi tested the case where observers attend to the flashed
Chapter 4. Previous work

cue stimulus (pro-stimulus condition) and the case where observers attend to the non-flashed box (anti-stimulus condition). In case of the pro-stimulus condition, passive (flashing box) and active (attend to flashed box) attention conjoin but in case of the anti-stimulus condition, passive and active attention rival. The direction of the perceived line-motion is in the pro-stimulus condition in all subjects from the attended flashing box to the other side independently of how long the cue stimuli are presented. In the anti-stimulus condition, the perception of motion direction depends on the cue leading time. For short cue presentations (<200ms), the passive attention dominates, so that observers perceive the line drawing out from the flashing non-attended cue. For longer cue presentations (>400ms), the moving direction is perceived vice versa and the active attention dominates.

4.1.1. Where in the brain arises the line-motion effect?

Hikosaka et al. discuss where in the brain the line-motion effect could arise [Hik93a, Hik93b]. The result of an dichoptic experiment excludes the retina and the LGN as the areas where the line-motion effect could arise [Hik93a]. To prove this, the authors presented the cue stimulus to the test person’s one eye and the bar stimulus to the other eye. In this setting, the perception of a drawing out bar, the line-motion illusion, is maintained. As the retina and the LGN process the information of both eyes separately, the effect cannot be due to retinal or geniculate processes. They further argue that area MT is known to be correlated to motion perception in the monkey cortex, so that the line-motion effect has to arise on the way V1-V2-V3-MT, the dorsal stream, see section 1.3.1.

Baloch and Grossberg discussed which processes of their one-dimensional model of the V1-V2-V4 and the V1-V2-MT-MST pathways give rise to the LM-illusion [Balo97]. These processes comprise boundary completion and colour filling-in effects that are supposed to occur on the V1-V2-V4 pathway. Further, the processes involve form-motion interaction over the V2-MT and MT-V2 pathways and the building of a so called G-wave which is generated directly by long-range apparent motion processes on the motion pathway [Balo97]. The authors present model equations and argue that their model copes with the LM-effect.

Jancke et al. used in [Jan04a] real-time optical imaging in area 18 of anaesthetised adult cats when presenting simple stimuli configurations as shown in Fig. 3.3. They could show that signatures of the line-motion effect can already
4.1. The Line-Motion illusion

be observed in cat area 18 as the spiking activity zone gradually draws out from the square to the bar response due to long-ranging horizontal connections.

A recent fMRI study by Larsen et al. on illusory motion in humans primary visual cortex shows that the estimated centre of MT+ activation in response to real motion is close to the activation centre due to the presentation of an according illusory moving stimulus [Lar06]. But also area V1 shows a strong and similar innervation in response to the real moving (a square moving from left to right) and illusory moving stimuli (two squares flashing, first the left, then the right). Larsen et al. propose a so called three-stage theory of perception of long-range apparent motion: First, higher level areas (most likely MT+) identify parts of two images as being two successive views of the same objects, which is called correspondence problem. Second, the higher areas (MT+) compute the motion intervening between the two object presentations. And third, in V1, the computed motion trajectory is filled in by a sequence of visual representations of the objects in successive positions along the path from the first to the second object presentation.

A study by Eagleman and Sejnowski proposes that the LM-illusion can be reversed by a clever manipulation of the stimulus after the line has disappeared [Eag03]. The authors conclude from this that perception is an a posteriori reconstruction from an integration of information over a certain window of time. The authors propose a stimulus setting, in which they present at both of the two ends of the later bar presentation, grey lines. At one side of the bar, the grey lines change luminance and colour to catch the observer’s attention. At the other side, a dot is constantly shown. When the bar is presented, the dot changes its position from the one to the other (previously blinking) side. Observers report in this setting the LM toward the cued (flashing lines) position 73.9±14.9 per cent of the time. In trials when the dot stayed at place, the normal LM was seen (86.4±5.3 per cent of observers).

4.1.2. A recent model simulates the LM-effect

The LM-illusion has recently been investigated computationally by Rangan et al. [Ran05] who tested an integrate and fire model on the data of Jancke et al. [Jan04a]. The authors show that the so called model cortex (mC) simulates the continuous spread of high fluorescence signals in the LM-stimulus case. The mC consists of $10^6$ model neurons, with NMDA-, GABA- and AMPA-conductances and the orientation specificity of real neurons.

\[1\] In several experiments the authors used extracellular recordings to prove the border to sub- and suprathreshold activity zones in the dye-signals.
Chapter 4. Previous work

Their model has been constructed to account for the exciting effect of spontaneously appearing activity that is correlated to orientation specific responses [Ken04]. In the model, long-ranging orientation-specific NMDA-type neurons are responsible for the continuous spread of high-amplitude activity. The model is an integrate and fire model, with $V_i$ describing the membrane potential of the $i$th neuron:

$$\frac{\partial}{\partial t} V_i = -g^L(V_i - V^L) - (g^A_i + g^N_i)(V_i - V^E) - g^G_i(V_i - V^I) + g^L \Delta F \exp \left[ \frac{V_i - V^F}{\Delta F} \right].$$

(4.1)

The terms $g^A_i$, $g^N_i$, and $g^G_i$ indicate the AMPA, NMDA and GABA conductances respectively and $g^L = 0.05$ the leakage conductance. The terms $V^{E,I,L}$ denote the excitatory, inhibitory and leak reversal potentials, with $V^E = 0$ mV, $V^I = -80$ mV and $V^L = -70$ mV. The model is driven by background noise and the output of an LGN model, that couples into the equation that describes the conductance of the AMPA neurons ($g^A_i$). Every neuron is locally connected to 400 nearby neurons with a random connectivity pattern. The longer lateral interactions are mediated by Gaussian interaction kernels, that have a scale of $\sigma = 250 \mu m$. In the NMDA-conductances $g^N$, the long-range orientation specific lateral connections are mediated by a Gaussian interaction kernel with a scale of $\sigma = 1500 \mu m$.

With the use of the long-ranging NMDA-type coupling neurons Rangan et al. can show that the mC is able to simulate the LM effect observed in the early visual cortex [Jan04a] without any involvement of attention effects.

4.2. Latency compensation in the visual pathway

Within the processing of moving stimuli, latency compensation mechanisms are assumed to play a major role:

In the visual pathway information transmission takes time. Nevertheless, we are able to catch a ball for example and thus we are able to estimate the position of a moving object exactly. Therefore, there must be somewhere in the brain mechanisms to compensate for the latency in information processing from retina to higher brain areas. In the following an overview of the previous work on latency compensation in the visual system is given.

Maunsell and Gibson showed that striate responses in monkeys have response latencies of up to 100 milliseconds [Mau92]. Berry II et al. report on response latencies of salamander’s and rabbit’s retinal ganglion cells of about 50 milliseconds [Ber99]. During these tens of milliseconds, a moving object pos-
4.2. Latency compensation in the visual pathway

possibly moves several metres. Neural latency compensation is therefore crucial if we try to catch a moving object or if we try to pass a street with fast driving cars on it. If there was no latency compensation, tasks like this would be hard to fulfil.

*Motion extrapolation* denotes the effect proposed by [Nij94] that the visual system might extrapolate the position of a moving object to calculate its actual position. As this effect in fact is not really extrapolating, but more an effect of *compensating of latencies* in the visual pathway, in this work it is referred to the latter term.

The so called *flash-lag effect* refers to a visual illusion in which observers perceive the position of a moving object ahead of a briefly flashed object when both stimuli are actually co-localised [Nij94], which is depicted in Fig. 4.1. There is an ongoing discussion on where and how in the brain this flash-lag effect occurs and if it is due to latency compensation phenomena, which shall be summarised in the following.

![Figure 4.1.: Flash lag illusion.](image)

A moving and a flashed square presented aligned are perceived as if they are displaced. In this setting one square moves from left to right, whereas the other square is flashed aligned to the moving square in one position (left). The perception deviates from the setting, as the moving square is perceived more toward the moving direction (right).

4.2.1. Differential latency times in neural processing

Baldo and Klein introduced the idea that the flash-lag illusion might result from a longer time delay in the visual processing of flashing dots in contrast to moving dots [Bal95]. The authors tested two dots rotating about a fixation point while two further dots were flashed in alignment with the centred rotating
Chapter 4. Previous work

dots. The flash-lag effect also occurs in this setting but with increasing distance of the flashing dots from the fixating point, this effect even increases. BALDO and KLEIN conclude that an amount of time is required to bring the flashing dots to a sufficiently high level of awareness, which might involve attention.

WILKINSON et al. also argued for the visual latency effect to explain the flash-lag illusion [Wil98]. The authors report that visual latency varies inversely with its illumination: They tested a continuously rotating line with two flanking lines at 0 and 180 degrees by changing the illumination once of the inner rotating line and once of the outer flanking lines. By increasing the so called detectability of the inner rotating line, the temporal lead of the inner rotating line toward the outer flanking lines increases. By increasing the detectability of the outer lines, the flash-lag effect reduces and at some point even inverts to the opposite. From this, WILKINSON et al. conclude that flash-lag effects are due to latency-differences in processing of moving and stationary stimuli.

4.2.2. Motion extrapolation at the retina

BERRY II et al. showed that motion extrapolation already starts in the retina [Ber99]. The authors recorded simultaneously the spike trains of many ganglion cells in the separated retina of the salamander and the rabbit. They could show that the flash-lag effect can already be observed in the retina by measuring the responses to a flashed bar and a moving bar (from left to right) separately. The timestep at which the moving bar spatially covers the position where the bar is flashed is the interesting one: It can be seen that the response to the moving bar in this timestep is shifted toward the moving direction compared to the response to the flashed bar.

4.2.3. Electrophysiology in early visual cortex

JANCKE et al. provide electrophysiological data from cat’s area 17. They project cortical spiking activity to visual field coordinates by presenting flashed stationary squares and determining the neuron’s receptive field coordinates. With this technique, they could show that for the flash lag illusion, time-to-peak latencies in response to moving squares were shorter by about 16 milliseconds compared to the response to stationary flashes. However, the flash-lag effect is commonly described by a latency difference of 45 – 80 milliseconds [Kre01]. Therefore, to explain why we are able to catch a ball, additional neural mechanisms might be needed. JANCKE et al. have shown using VSDI that sub-threshold activity spreads out fast and wide in cat area 18 when presenting moving square stim-
4.2. Latency compensation in the visual pathway

Therefore, the idea of preactivating subthreshold activity causing receptive field responses to be shifted in moving direction is proposed [Jan04b]. JANCKE et al. further showed that latencies in processing of moving squares were even more reduced for peripheral-to-centre motion (38 milliseconds) compared to centre-to-peripheral motion (42 milliseconds) [Jan04b]. The latency for flashed stimuli was 54 milliseconds for comparison. The authors further report that moving square response latencies become smaller with increasing stimulus speed.
Chapter 5.
Simulating the primary visual cortex: a dynamic neural field

Dynamic neural fields (DNF)s are used to model large ensembles of cortical neurons in their temporal dynamics [Beu56, Wil72, Ama77, Erl03]. Since even in a small piece of cortex the number of neurons and synapses is huge, it became common to study neural networks as a space continuous neural tissue. This continuous description allows to handle the neural tissue as a dynamic field in a physical sense. However, implementing a DNF on a computer makes it necessary to discretise the field. As in this work VSDI-data are simulated, a reasonable level of modelling detail is implicitly given by the imaging method. Hence, one model neuron describes one measured dye signal activity (with a spatial resolution of 50 micrometres) that delivers the average membrane potential of a large number of neurons.

In this study, a two layered DNF—with one excitatory and one inhibitory neuron layer [Wil72, Wil73, Erl03]—is used to simulate the dynamics of cat’s primary visual cortex. The setting of the model, used in this work to simulate VSDI-data derived from cat area 18, is depicted in Fig. 5.1. Spatial and temporal integration effects within the DNF make normalisation effects necessary to compensate for too large response amplitudes evoked by larger visual input. According to Heeger and Freeman et al. LGN-suppression effects are used to account for these spatial and temporal integration and gain control effects [Hee92, Fre02]. The dye-signal responses are finally simulated by a linear combination of the DNF’s excitatory and inhibitory signals. One excitatory and one inhibitory neuron is each located at the position of one VSDI-pixel and models herewith the mean responses of a large number of neurons. As VSDI offers both a very high spatial and temporal resolution (see chapter 3), testing a DNF on such data offers new perspectives in investigating cortical network dynamics over several millimetres cortex between large populations of neurons in real time. This enables to observe a large part of cat’s area 18 in response to moving stimuli in real-time. Until now, no other imaging technique offers such a possibility with such a high spatial and temporal resolution, see chapter 3.
Figure 5.1: Setting of the model architecture. The visual input is first processed through an LGN-model that computes suppression (normalisation) effects [Hee92, Fre02]. The primary visual cortex is simulated with a variation of the excitatory-inhibitory network of [Wil72]. The output of the system is a linear combination of the excitatory (u) and inhibitory (v) responses, with \( \lambda \in [0, 1] \).

The first approach to describe a continuous approximation of neural activity is often attributed to BEURLE and later to GRIFFITH [Beu56, Grf63, Grf65]. WILSON and COWAN extended the model of BEURLE by considering both excitatory and inhibitory neural activity [Wil72, Wil73]. AMARI proposed a one layer neural network that is mathematically easier to handle, so that a stability analysis yields fruitful results [Ama77].

Before describing the DNF used in this study, related DNFs shall be introduced in the following section in historic order: the DNF’s by WILSON and COWAN [Wil72], AMARI [Ama77] and ERLHAGEN [Erl03] are presented.
5.1. Dynamic Neural Fields so far

In this section, first, Wilson and Cowan’s excitatory-inhibitory network [Wil72, Wil73] will be introduced, as it was the first approach that considered a two-layered DNF of excitatory and inhibitory neurons. The authors introduce a DNF that consists of two coupled nonlinear integrodifferential equations. One equation represents the dynamics of the excitatory neuron population and the other the dynamics of the inhibitory neuron population.

After that, Amari’s one-layered neural field is presented, as the analytical study [Ama77] on this network is a milestone in DNF-theory. A sketch of this theory will be given here.

The neural field model proposed by Erlhagen [Erl03] is presented because some modifications of the excitatory-inhibitory network realised in this model have also been used in the DNF of this study.

5.1.1. Wilson and Cowan’s excitatory-inhibitory network

The architecture of Wilson and Cowan’s excitatory-inhibitory-network is depicted in Fig. 5.2. The network consists of continuously distributed excitatory and inhibitory neurons. Characteristic for excitatory-inhibitory network is that there exists each type of combination of interaction between excitatory (E) and inhibitory (I) cells (E → E, E → I, I → E, I → I). For the reason of clarity, the picture

Figure 5.2.: Scheme of Wilson and Cowan’s network dynamics. There exist connections from each neuron to itself, from inhibitory to excitatory neurons and vice versa and within the excitatory neuron and inhibitory neuron population accordingly. Inhibitory efferent neurons reduce the activity of the afferent neurons, whereas excitatory efferent neurons enhance the activity of the postsynaptic cell, which is indicated with a circle sign (inhibition) and an arrow sign (excitation) on the axons’ endings. For the reason of clarity, just one inhibitory and five excitatory neurons are shown.
only shows one inhibitory and five excitatory neurons and their connections. Inhibitory neurons cause reduced activity on the according afferent neurons, which is depicted with a circular ending on the drawn axons. Whereas excitatory neurons enhance the afferent neurons’ activity, sketched with an arrow on the axonal endings.

As these excitatory and inhibitory neurons interact over time, a mathematical description in form of a dynamic system turns out to be advantageous. WILSON and COWAN propose two coupled integro-differential equations with one representing the excitatory and the other the inhibitory neuron population:

\[
\tau_u \frac{\partial u(x,t)}{\partial t} = -u(x,t) + \int f_u(u(x',t))w_{ee}(x,x')dx' - \int f_v(v(x',t))w_{ei}(x,x')dx' + S_u(x,t) \tag{5.1}
\]

\[
\tau_v \frac{\partial v(x,t)}{\partial t} = -v(x,t) + \int f_v(u(x',t))w_{ie}(x,x')dx' - \int f_v(v(x',t))w_{ii}(x,x')dx' + S_v(x,t) \tag{5.2}
\]

The location on the cortical space is expressed by the space variable \(x\). WILSON and COWAN describe the cortical tissue as a continuous space of neurons. For that reason the variable \(x\) can take values of any real number. The membrane potential of the excitatory neuron population, \(u(x,t)\), on cortical position \(x\) and time step \(t\) is expressed in its temporal dynamics indicated by the left side of equation 5.1. The inhibitory membrane potential is encoded in the term \(v(x,t)\) respectively. The axonal response to the membrane potential \(u(x,t)\) (or \(v(x,t)\)) is the so called \textit{gain function} or \textit{axonal response function}. The axonal response function \(f_{u(v)}\) returns for each membrane potential \(u(x,t)\) (\(v(x,t)\)) the resulting spiking activity of the neuron. It can be interpreted as a simulated axon hillock of the neuron. Weighting functions \(w_{\alpha\beta}\) (\(\alpha, \beta \in \{e, i\}\)) denote the connection strength of lateral couplings from a \(\beta\)-neuron to an \(\alpha\)-neuron. The lateral coupling function \(w_{ei}\) denotes herewith the coupling between neurons projecting from the inhibitory layer and synapsing to the excitatory layer. The external inputs, here the visual stimuli, are denoted with \(S_u\) in the excitatory layer and \(S_v\) in the inhibitory layer, respectively. The time constants \(\tau_u\) and \(\tau_v\) indicate the reaction time or latency of the corresponding neuron population.

WILSON and COWAN showed that the excitatory-inhibitory network works in three different modes, an \textit{active transient mode}, an \textit{oscillatory mode} and a \textit{steady-state mode}, which they associate in a somehow speculative way with dif-
ferent types of neural tissue: i.e. sensory neo-cortex, archi- or prefrontal cortex and thalamus [Wil73]. As the field equations are too complex to specify the three modes analytically, Wilson and Cowan show the three modes experimentally by a set of parameters evoking the different processing states [Wil73].

5.1.2. Amari’s dynamic neural field

Amari proposed a dynamic neural field [Ama77] that is mathematically simple compared to Wilson and Cowan’s excitatory-inhibitory network so that it permits an analytical treatment of its dynamical processes. The field equation consists of just one neural layer that incorporates excitatory and inhibitory activity by choosing the interaction kernel \( w(x, x') \) as a mexican hat function (see Fig. 5.3):

\[
\tau \frac{\partial u(x,t)}{\partial t} = \int w(x, x') f(u(x', t)) dx' + h + S(x, t) \quad (5.3)
\]

The use of the mexican hat function as the lateral coupling term implies that neurons that are far away in space inhibit each other and neurons that are nearby excite each other. To give a historical survey on DNFs, the famous analytical study of Amari on stable solutions of eq. 5.3 will be presented in the following [Ama77].

Amari studies the homogeneous solution of eq. 5.3 (\( \frac{\partial u(x,t)}{\partial t} = 0 \)) in the absence of input (\( S(x,t) = 0 \)):

\[
u(x,t) = \int w(x, x') f(u(x', t)) dx' + h \quad (5.4)\]
Chapter 5. Simulating the primary visual cortex: a dynamic neural field

The author considers three different types of solutions of the homogeneous equation. For this we need the definition of the excited region of the field:

**Definition 1.** Let \( R[u] = \{x|u(x) > 0\} \) be the excited region of the homogeneous solution \( u(x, t) \).

- Then we call an equilibrium \( u(x, t) \) satisfying \( R[u] = \emptyset \) a \( \emptyset \)-solution.
- An equilibrium for which \( R[u] = [−\infty, \infty] \) holds (the whole cortical region is excited) is called \( \infty \)-solution.
- A localised excitation denotes an excited region which is a finite interval, so that \( R[u] = (a_1, a_2) \) is the excited region of length \( a_2 - a_1 \) (\( a_1 < a_2 \)).

Hereby \( \emptyset \) denotes the empty set. The three solution types are characterised by the resting membrane potential \( h \) and the integral of the interaction kernel:

**Definition 2.** The integral of the interaction kernel is defined as
\[
W(x) = \int_{0}^{x} w(y)dy.
\]
With this we can define the terms
\[
W_m = \max_{x>0} W(x) \quad \text{and} \quad W_\infty = \lim_{x \to \infty} W(x).
\]

With these prerequisites AMARI proofs the following theorem on the three types of solutions:

**Theorem 1.** In the absence of input,

- there exist a \( \emptyset \)-solution if and only if \( h < 0 \).
- there exists an \( \infty \)-solution if and only if \( 2W_\infty > -h \).
- there exists an \( a \)-solution (a local excitation of length \( a \)) if and only if \( h < 0 \) and \( a > 0 \) satisfies \( W(a) + h = 0 \).

The proof and a detailed list of solutions in dependency of \( W(x) \) is given in [Ama77].

The stability of the system can be formulated in the following theorem that has been shown in [Ama77] and reported in compact form in [Goe00]:
5.1. Dynamic Neural Fields so far

**Theorem 2.** With the same notations as in Theorem 1, the following is true:

- The $\emptyset$-solution and the $\infty$-solution is always asymptotically stable.
- The $a$-solution is asymptotically unstable if $\frac{\partial W(a)}{\partial a} > 0$ holds and stable if $\frac{\partial W(a)}{\partial a} < 0$.

The asymptotically stable solution exists if and only if $W_m > h > W_\infty > 0$ or $W_m > h > 0 > W_\infty$. In both two conditions an $\emptyset$-solution exists also.

The proof can also be found in [Ama77].

5.1.3. Erlhagen’s excitatory-inhibitory network

A modified version of Wilson and Cowan’s excitatory-inhibitory network is used by Erlhagen and others to simulate dynamic cortical activity in the visual system ([Erl03], [Jan99], [Jan96]). The model resembles that of Wilson and Cowan, with some modifications:

\[
\tau_u \frac{\partial u(x, t)}{\partial t} = -u(x, t) + h + g(u(x, t)) \left( \int w_{ee}(x, x') f(u(x', t)) dx' \right) - v(x, t) + S(x, t) \quad (5.5)
\]

\[
\tau_v \frac{\partial v(x, t)}{\partial t} = -v(x, t) + \int w_{ie}(x, x') f(u(x', t)) dx' \quad (5.6)
\]

The axonal response function $f$ is a sigmoidal function which will be introduced in eq. 5.9 in the context of the DNF used in this work. ERLHAGEN et al. added the resting membrane constant $h$ to the right side of the equation describing the excitatory neuron dynamics, see eq. 5.5. Further a multiplicative term, the so called *shunting function*, $g(u(x, t))$, is introduced. The shunting function $g$ is assumed to be of sigmoid shape dependend on the membrane potential of the excitatory neurons at cortical position $x$. It is multiplied by the interaction terms in the excitatory neuron layer. This multiplicative term serves to gate the lateral interactions by feedforward activation [Erl03]. A further difference to Wilson and Cowan’s excitatory-inhibitory network is that the inhibition couples into the excitatory layer locally, as the inhibitory term $v(x, t)$ is not smoothed by a Gaussian interaction kernel. Local inhibitory couplings are physiologically supported as most inhibitory interneurons have local projections, see section 2.2. Furthermore, the term $\int w_{ii}(x, x') f_i(v(x', t)) dx'$, which describes the lateral interaction within the inhibitory neuron layer (eq. 5.2), is left out, so that the effect of local inhibition is further stressed.
Chapter 5. Simulating the primary visual cortex: a dynamic neural field

5.2. A DNF to simulate VSDI-signals

When choosing a computational model that best represents the dynamics of the fluorescence signals and that coevally realises basic principles of the functional architecture of the primary visual cortex, we have to consider the following points:

- **mean field approach**: As the data underlying this study are derived with a VSDI-technique, where one image-pixel represents the average membrane potential of a large number of neurons of different types, we should use a so called *mean field approach*. In this mean field approach, one model neuron represents the average membrane potential of a population of neurons, so that one model neuron simulates one VSDI pixel.

- **active transients**: The representation of a visual stimulus evokes a response that can sustain or even rise after offset of the stimulus. After reaching its peak response, the activity declines back to baseline-level. This constraint to the model response is called *active transient* (see Fig. 5.4 for an example). This active transient mode is common in sensory cortical areas and shall be possible with the model chosen.

- **excitation and inhibition**: We are especially interested in the interplay between excitatory and inhibitory neural activities. Hence, a model that offers the possibility to investigate both populations separately without affecting interactions is desired. As VSDI does not offer this separate study, a model with this ability can offer additional information and insights.

- **biologically plausible approach**: We intend to draw back conclusions from the used parameter setting of the model. Consequently, the parameters have to be directly biologically motivated, so that they encode the functional architecture of the primary visual cortex.

- **minimalist approach**: Using a huge number of parameters makes it easier to fit the VSDI data as the degree of freedom in the model increases. But at the same time, the expressiveness of each parameter gets lost and the calibration of the parameters becomes less manageable. As the VSDI gives an overall average response to large numbers of neurons, it is for instance not useful to simulate on the transmitter level as the VSDI technique does not offer this level of detail.

Wilson and Cowan’s excitatory-inhibitory network [Wil72] realises a neural mean field approach, that is able to work in an active transient mode [Wil73].
5.2. A DNF to simulate VSDI-signals

Figure 5.4: **Active transient response mode.** The response of one model neuron in its temporal course. The red rectangle indicates the time, the square stimulus has been presented (0-50 milliseconds). While the stimulus has been shown, the response begins to rise and still rises after stimulus offset, reaches then its peak at about 100 milliseconds and after that declines back to baseline level.

and moreover deals with two neuron populations of excitatory and inhibitory cells. For that reason an excitatory-inhibitory network with some modifications toward a more minimalist approach is introduced in this study.

5.2.1. A dynamic neural field approach based on Wilson and Cowan’s excitatory-inhibitory network

As Wilson and Cowan’s network serves our purposes best, the measured fluorescence signals are simulated with a dynamic mean-field approach based on Wilson and Cowan’s excitatory-inhibitory network [Wil72], see eqs. 5.1 and 5.2. Hereby one neuron represents one VSDI-pixel. To comply with the requirement of a ‘biological plausible’ model and of a ‘minimalist approach’, the equations are modified according to ERLHAGEN’s model, see eq. 5.5 and 5.6:

- The term $\int w_{ii}(x, x')f_v(v(x', t))dx'$ is left out.
Chapter 5. Simulating the primary visual cortex: a dynamic neural field

- The inhibition couples into the excitatory layer locally.

- Additional parameters describing the resting membrane potentials of the excitatory $h_u$ and inhibitory layer $h_v$ are introduced.

- The external input in the DNF couples only into the excitatory layer.

In contrast to Erlhagen’s equations the shunting term is left out. Further, the resting membrane potential constant is also added to the inhibitory layer. The coupling of the inhibitory term $v(x,t)$ into the excitatory layer is performed by first applying the axonal response function $f_v$ to the term to be more consistent with physiology.

With these modification, eqs. 5.1 and 5.2 turn to

$$
\tau_u \frac{\partial u(x,t)}{\partial t} = -u(x,t) + h_u + \int w_{ee}(x,x')f_u(u(x',t))dx'
$$

(5.7)

$$
\tau_v \frac{\partial v(x,t)}{\partial t} = -v(x,t) + h_v + \int w_{ie}(x,x')f_u(u(x',t))dx',
$$

(5.8)

where $u$ indicates the membrane potential of the excitatory neurons at cortical position $x$ and time $t$ and $v$ denotes the corresponding inhibitory membrane potential. The resting membrane potentials of the excitatory ($u$) and inhibitory ($v$) layers ($h_u$ and $h_v$) have been added to the equations. The external input $S$, that is the response of the LGN (see section 5.3) to the stimulus, couples into the excitatory neuronal layer. The axonal response $f_\gamma$ is chosen as a sigmoidal function

$$
f_\gamma(\gamma) = \frac{q_\gamma}{1 + e^{-c_\gamma(\gamma - \gamma_0)}},
$$

(5.9)

with $\gamma \in \{u, v\}$ and $c_\gamma > 0$ describing the slope of the response function, $q_\gamma \in \mathbb{R}$ implements its saturation and $\gamma_0 \in \mathbb{R}$ its threshold value. A typical sigmoidal axonal response function is depicted in Fig. 5.5. For membrane potentials $u$ that converge to $-\infty$ the axonal response gives no response ($f_\gamma(u) = 0$), whereas membrane potentials that converge toward $\infty$ evoke a saturated axonal response with saturation value $q_\gamma < \infty$. Naturally both converging cases will not occur in nature. The threshold value $\gamma_0$ has its position on the abscissa of the inflection point of the sigmoidal function. In general, the slope of the sigmoid function can be adjusted, so that there is a broad area of values around the threshold value where neurons which achieve these values are assumed to give spiking responses.
5.2. A DNF to simulate VSDI-signals

Figure 5.5.: The sigmoidal axonal response function. The axonal response function used in our simulations has a sigmoid shape. For large membrane potentials the sigmoid function returns full axonal activity (saturation value \( c_\gamma \)) and for small membrane potential values, the axonal response function gives no responses. The inflection point of the function is the threshold value \( \gamma_0 \).

Let \( \alpha \beta \in \{ee, ie\} \). The lateral coupling function \( w_{\alpha \beta}(x, x') \) is chosen as a Gaussian function

\[
 w_{\alpha \beta}(x, x') = a_{\alpha \beta} \frac{1}{\sigma_{\alpha \beta} \sqrt{2\pi}} \exp \left( -\frac{1}{2\sigma_{\alpha \beta}^2} \|x - x'\|^2 \right),
\]

with \( \| \cdot \| \) denoting the Euclidean norm, providing a higher synaptic influence between neurons that are closer to each other than between neurons that are spatially afar. The term \( w_{\alpha \beta} \) denotes the weight of the neurons coming from the \( \beta \)-layer and synapse into the \( \alpha \)-layer. The parameters \( \sigma_{\alpha \beta} \) and \( a_{\alpha \beta} \) describe the width and height of the Gaussian functions respectively and thereby the extent of lateral spread of the neurons on the cortex and the strength of interaction. Both parameters represent physiologically interesting values, as introduced in sections 2.4 and 2.5.

In eqs. 5.7 and 5.8 the cortical space is assumed to be continuous. As the dye-signals give pixel responses instead of continuous images and computers also have to simulate the cortical space discretely, the equations are rewritten...
discretely
\[
\tau_u \frac{\partial u(x, t)}{\partial t} = -u(x, t) + h_u + \sum_{x'=1}^{n} \omega_{ee}(x, x') f_u(u(x', t)) - g_v \cdot f_v(v(x, t)) + g_S \cdot S(x, t)
\]
\[
\tau_v \frac{\partial v(x, t)}{\partial t} = -v(x, t) + h_v + \sum_{x'=1}^{n} \omega_{ie}(x, x') f_u(u(x', t)),
\]
with \( n \) denoting the number of VSDI-pixels and thereby the number of model neurons used in this study.

The VSDI-data show an overall-response of both excitatory and inhibitory signals at the same time. Therefore, excitation and inhibition separated and the proportion of excitatory and inhibitory signals seen in VSDI becomes interesting. For that reason, the response of the system is defined by the linear combination of the DNF’s excitatory and inhibitory activations
\[
o(x, t) := \lambda u(x, t) + (1 - \lambda) v(x, t),
\]
with \( \lambda \in [0, 1] \) determining the proportion of excitatory and inhibitory signals in the dye-signal. Setting \( \lambda = 1 \), sets the output to the pure excitatory responses and setting \( \lambda = 0 \) simulates the pure inhibition by coevally conserving the interaction between both layers.

In the DNF proposed in this work, a so called local inhibition is used, as in eq. 5.11 the inhibitory neurons couple into the excitatory layer with the term \(-g_v \cdot f_v(v(x, t))\), without convolving with a Gaussian kernel (see chapter A.1, which is consistent with physiology as most interneurons have local projections, see section 2.2. However, the excitatory neurons project to the inhibitory neurons non-locally which is expressed by the use of an interaction kernel function \( \omega_{ie} \), making the inhibition acting also more ‘globally’.

5.3. Divisive inhibition in the lateral geniculate nucleus

Recently RUST and MOVSHON emphasised the importance of considering gain-control effects [Rus05]. The authors demanded with the use of the term ‘new standard models’ the use of gain control modelling. There exist several works on sensory gain control-mechanisms (see [Car97, Fre02, Gra00, Hee92, Schw01, Sim99]), an effect also observed in the data underlying this study.
5.3. Divisive inhibition in the lateral geniculate nucleus

Considering the excitatory-inhibitory network of Wilson and Cowan [Wil72], when presenting the square stimulus, activity integrates over space and time much less than when presenting the bar stimulus. However, there is no evidence that larger stimuli evoke much higher membrane potentials. This spatial integration-effect has been mentioned by Wilson and Cowan [Wil73]. When using the DNF proposed in this work (eq. 5.11 and eq. 5.12) without the use of any normalisation effect, the same spatial and temporal integration problem arises. Wilson and Cowan’s excitatory-inhibitory network does not capture phenomena like contrast-gain and activity-integration control. Indeed, the excitatory-inhibitory network is used as a cortex model in most cases. Therefore every contrast- and amplitude integration control phenomena taking place in lower processing stages like in the retina or in the LGN are not considered.

In this work a model of the LGN is used that first smoothes the stimulus by convolving the stimulus with a Gaussian kernel function over space dimension and second computes a suppression effect: Neurons in the LGN receive excitatory input from their neighbouring neurons which leads to a smoothing effect. But LGN-neurons also saturate in response and exactly this saturation is a kind of suppression [Fre02]. This form of suppression by response saturation can be approximated with a normalisation-technique that is inspired by Heeger who calls this effect ‘divisive normalisation’ [Hee92]: Let \( S(x, t) \) be the stimulus intensity at the cortical position \( x \) in millimetres and time \( t \) in milliseconds after stimulus onset. Let

\[
S'(x, t) = \int S(x, t)w_S(x', x)dx'
\]

be the smoothed stimulus response, with \( w_S(x', x) \) chosen as a Gaussian function (see eq. 5.10, with \( \{\alpha, \beta\} = S \)). We can now compute the normalised response

\[
S''(x, t) = \frac{g_{S'}S'(x, t)}{\int S'(x', t)dx' + R},
\]

(5.14)

where \( R \) denotes the synaptic saturation and \( g_{S'} \) is a stimulus-boosting parameter. By using this preceding divisive normalisation LGN method (nLGN), we can handle the spatial integration problem which will be discussed in section 7.1.3.
5.4. Solving neural field equations, Euler’s method

The DNF equations of the excitatory layer (see eq. 5.7) and of the inhibitory layer (see eq. 5.8) are termed integrodifferential equation as on the right side of the equations an integral operator occurs. Those integrodifferential equations have a complex structure, as they are in most cases highly nonlinear making it hard to solve these equations analytically. Therefore, DNF-equations are usually solved approximately.

The so called Euler-Method is a simple numerical method, that solves the equations approximately. With a fixed time step \( r > 0 \), the Euler-Method can be derived as follows: Let us start with a differential equation with initial function \( u(x, t_0) \) by

\[
\frac{\partial u(x, t)}{\partial t} = F(u(x, t)), \quad u(x, t_0) = u_0, \quad (5.15)
\]

with \( F(u(x, t)) \) denoting the right side of an autonomous differential equation. It is true that for small \( r > 0 \) (definition of the derivative of a function)

\[
\frac{u(x, t + r) - u(x, t)}{r} \approx \frac{\partial u(x, t)}{\partial t} = F(u(x, t)) \quad (5.16)
\]

holds, giving

\[
u(x, t + r) \approx u(x, t) + rF(u(x, t)). \quad (5.17)\]

With eq. 5.17 we can directly derive the Euler-Method:

For a given starting point, \( u(x, t_0) = u_0 \), we can calculate step by step an approximation of \( u(x, t) \):

Calculate for \( t_k = t_0 + kr \, (k = 0, 1, 2, \ldots) \) approximations \( y_k \) of \( u(x, t_k) \) iteratively as follows:

\[
y_{k+1} = y_k + rF(y_k, t_k), \quad y_0 = u(x_0) = u_0, \quad k = 0, 1, 2, \ldots \quad (5.18)
\]

Applying the Euler-Method to the integrodifferential eq. 5.11 yields

\[
u(x, t + r) = \frac{1}{\tau_u} \left( -u(x, t) + g_S \cdot S(x, t) \right. \\
\left. + \int f_u(u(x', t))w_{ee}(x, x')dx' \\
\left. -g_v \cdot f_v(v(x, t)) \right) + u(x, t). \quad (5.19)
\]
The inhibitory membrane potentials $v(x,t)$ (eq. 5.12) can be approximated accordingly.

### 5.5. Runge-Kutta’s method

For a given fixed step size, better approximate solutions than those derived with the **Euler** method can be obtained using the so called **Runge-Kutta method**. **Euler’s** method is based on a straight-line approximation which can be approved by expanding the first order **Taylor** series approximation:

$$u(x,t + r) \approx u(x,t) + r \frac{\partial u(x,t)}{\partial t}$$

The second order **Taylor** series approximation is

$$u(x,t + r) \approx u(x,t) + r F(u(x,t)) + \frac{r^2}{2} \frac{\partial^2 F(u(x,t))}{\partial t^2}.$$  \hspace{1cm} (5.20)

Substituting $\frac{\partial u(x,t)}{\partial t}$ using eq. 5.15 yields

$$u(x,t + r) \approx u(x,t) + r F + \frac{r^2}{2} \frac{\partial F(u(x,t))}{\partial t}.$$  \hspace{1cm} (5.21)

To simplify matters $F$ stands in the following for $F(u(x,t))$. The derivative of the right side of the differential equation, $F(u(x,t))$, might be difficult to compute. Therefore we avoid the calculation of $\frac{\partial F(u(x,t))}{\partial t}$. To avoid this, the following trick can be applied:

Consider the chain-rule

$$\frac{\partial F}{\partial t} = \frac{\partial F}{\partial u} \frac{\partial u}{\partial t} = F \frac{\partial F}{\partial u},$$

which leads with eq. 5.21 to

$$u(x,t + r) \approx u(x,t) + r F + \frac{r^2}{2} F \frac{\partial F}{\partial u}.$$  \hspace{1cm} (5.22)

To avoid now to calculate $\partial F/\partial u$ we are looking for an approximation of the form

$$u(x,t + r) \approx u(x,t) + ar F + br F(u + cr F).$$  \hspace{1cm} (5.23)

By choosing $a, b, c > 0$ adequately, it can be achieved that the equations 5.22
Chapter 5. Simulating the primary visual cortex: a dynamic neural field

and 5.23 become equal. To do so, the last term has to be expanded by using the first order TAYLOR series approximation

\[ F(u + crF) \approx F(u) + r\frac{\partial F}{\partial u} cF. \]

By substituting this in eq. 5.23 yields

\[ u(x, t + r) \approx u(x, t) + (a + b)rF + bc r^2 \frac{\partial F}{\partial u}. \]

(5.24)

Eq. 5.24 is identical to eq. 5.22 if \(a + b = 1\) and \(bc = 1/2\) holds. To specify \(a, b, c\) we set \(a = b\). Therefore we get \(a = 1/2, b = 1/2\) and \(c = 1\). Using these values in eq. 5.23 gives

\[ u(x, t + r) \approx u(x, t) + \frac{r}{2} F(u(x, t)) + \frac{r}{2} F(u(x, t) + rF(u(x, t))), \]

(5.25)

the so called second order RUNGE-KUTTA approximation which contains no derivative of \(F\), so that it can easily be applied computationally.

The second order RUNGE-KUTTA approximation has a three times smaller approximation error than the EULER-Method when using the same step size [Wil99]. Using a fourth order RUNGE-KUTTA method (TAYLOR series expansion of order four in eq. 5.20) leads for the same step size \(r\) even to a 15 times smaller error than EULER’s method [Wil99].

As mentioned, higher order RUNGE-KUTTA methods can be derived similarly to the second order RUNGE-KUTTA-method by applying higher order TAYLOR series expansions in eq. 5.20.

In this work, the MATLAB-toolbox function \texttt{ode45} has been used to solve the equations of the underlying DNF. It is a combined fourth and fifth order RUNGE-KUTTA-method.
Chapter 6.

Optimising the parameters

Beside designing the architecture of a model, the optimisation of its parameters is of no less importance. The DNF with the preceding normalisation model of the LGN incorporates 20 parameters ($\tau_u$, $\tau_v$, $h_u$, $a_{ee}$, $\sigma_{ee}$, $a_{ie}$, $\sigma_{ie}$, $c_u$, $c_v$, $g_S$, $g'_S$, $g_v$, $\sigma_S$, $\lambda$, $v_0$, $u_0$, $q_u$, $q_v$). Five of them ($q_u$, $g'_S$, $a_S$, $q_v$ and $v_0$) are kept fixed as they just deliver redundant information, see section 6.2, and one parameter ($\lambda$) can be computed with a linear program, see section 6.3. Hence, 14 parameters have to be optimised with an expedient strategy.

In this study an evolution strategy method called Covariance Matrix Adaptation Evolution Strategy (CMA-ES) algorithm [Han01] combined with manual fine tuning is used to optimise the parameters. The fitness function in the CMA-algorithm is the sample correlation—see appendix A.2—between the simulated DNF-output derived from eq. 5.13 and the VSDI-pictures. The CMA-ES has already been successfully applied to optimising dynamic neural fields by Igel et al. and Schneider et al. [Ige02, Ige03, Schn04].

6.1. Application of the covariance matrix adaptation algorithm

The individuals of a population are vectors of 14 real-valued elements ($\tau_u$, $\tau_v$, $h_u$, $h_v$, $a_{ee}$, $a_{ie}$, $c_u$, $c_v$, $g_S$, $g_v$, $\sigma_S$, $u_0$), which are modified by recombination and mutation of the CMA-ES, see appendix B for more details. The specific of this CMA-ES is that the mutation is realised by adding a normally distributed random vector with zero mean and adapting the whole covariance matrix for improving the searching strategy. The CMA algorithm implements important concepts for strategy parameter adaptation by realising two main concepts: First, the concept of derandomisation, where the mutation distribution is altered in a way where the distribution parameters are deterministically linked to the object parameter variations. Second, the idea of cumulation, which refers to the concept of taking a sequence of successive steps—a so called evolution path—into account.
The fitness function used to optimise the model of this study is defined as

$$fitness := \text{cor}(d, p), \text{ with } d = \begin{pmatrix} d_1 \\ \vdots \\ d_n \end{pmatrix} \text{ and } p = \begin{pmatrix} p_1 \\ \vdots \\ p_n \end{pmatrix}$$ (6.1)

with the indices $1 \leq i \leq n$ indicating the stimulus setting (square, bar and LM for example), $n$ the number of considered stimuli, $b_i$ the corresponding dye-signals and $d_i$ the simulated response in vector forms, that is all pixels of the matrix arranged into a vector. The vectors $d_i$ and $p_i$ for $i = 1, \ldots, n$ are concatenated to just two vectors $d$ and $p$. The function $\text{cor}$ denotes the sample correlation coefficient, see appendix A.2, between both vectors. As coevally the best (in respect of the mean squared error between simulation and dye-signals) linear transform of the simulated dye-signals $o_{\text{pictures}} = m_{\text{p}}o + b_{\text{p}}$ is calculated (see Appendix A.2), the vectors $d_i$ and $p_i$ for $i = 1, \ldots, n$ are concatenated to just two vectors $d$ and $p$. With this modification, the correlation coefficient can be jointly calculated, and the parameters $m_{\text{p}}$ and $b_{\text{p}}$ denote the conjoint linear transform of all simulations, see below.

The correlation coefficient is invariant to linear transforms of $d$, see appendix A.2. For that reason, the CMA-ES only has to cope with non-linear relationships between the simulations $d$ and the measured data $p$.

The correlation coefficient can be calculated from the so called linear regression of $d$ with respect to $p$ and the linear regression of $p$ with respect to $d$: The linear regression toward a data series denotes a linear transform of the other series that results in a minimal mean squared error between $d$ and $p$, see appendix A.2. To plot the simulations, the optimal linear transform of the simulations with respect to the measured data ($y = m_{\text{p}}p + b_{\text{p}}$) is calculated throughout this work, see appendix A.2:

$$o_{\text{pictures}} = m_{\text{p}}o + b_{\text{p}},$$

with $o$ taken from eq. 5.13. As the VSDI-signals are linearly correlated with membrane potentials [Pet03, Ste98], it is allowed to apply any kind of linear transform.

### 6.2. Redundant parameters in the DNF

It is not necessary to optimise all parameters used in the DNF, see eqs. 5.11 and 5.12. Some of the parameters deliver redundant information: Consider for
6.3. Calculating the parameter $\lambda$

From the DNF used in this work (eqs. 5.11 and 5.12, page 52) we can derive the following redundancies:

$q_v \propto g_v$:
- $q_v$ fixed: use $\tilde{g}_v := c_1 g_v$, with $c_1 = q_v$
- $\tilde{g}_v := c_2 a_{ee}$, with $c_2 = a_{ee}$

$v_0 \propto h_v$:
- $v_0$ fixed: use $\tilde{h}_v := h_v + c_3$, with $c_3 = -v_0$
- $\tilde{h}_v := h_v + c_4$

$u_0 \propto h_u$:
- $u_0$ fixed: use $\tilde{h}_u := h_u + c_4$, with $c_4 = -u_0$

$g_S \propto g_S \propto a_S : g_S$ and
- $g_S$ fixed: use $\tilde{g}_S := c_5 \cdot g_S$, with $c_5 = g_S a_S$

It can be seen that the new definitions of $h_v$ ($\tilde{h}_v$ and $\tilde{h}_v'$) deviate from each other when once describing the redundancy to $v_0$ and once to $u_0$. As $h_v$ cannot be redefined as both $\tilde{h}_v = h_v + c_3$ and $\tilde{h}_v' = h_v + c_4$, with $c_3 = -v_0$ and $c_4 = -u_0$, we have to decide which of the two redundancies to use. So, let us use the redundancy of $h_v$ to $v_0$.

Herewith, the four parameters ($q_v$, $q_u$, $g_S$, $a_S$ and $v_0$) encode redundant information and can be kept fixed in the optimisation.

6.3. Calculating the parameter $\lambda$

The parameter $\lambda$ from eq. 5.13 that describes the proportion of excitatory and inhibitory signals in the VSDI-pictures can be computed by minimising a simple linear program. Let $d = [d_1, ..., d_n]^T$ be the simulated dye signal and $t = [t_1, ..., t_n]^T$ the measured dye-signal both presented in vector form. By replacing $o$ with $d$ in eq. 5.13 we get:

$$d = (1 - \lambda)u + \lambda v,$$

with $u = [u_1, ..., u_n]^T$ and $v = [v_1, ..., v_n]^T$ the excitatory and inhibitory membrane potentials in vector forms respectively.

The minimisation problem is the following

$$\min_{a,b} [(ad + be) - t]^2, \quad (6.2)$$

which results from the definition of the correlation coefficient, with $e = [1, ..., 1]^T$.
the $n$-dimensional unit vector. Let us now define

$$x := [u, v] = \begin{pmatrix} u_1 & v_1 \\ \vdots & \vdots \\ u_n & v_n \end{pmatrix}, \quad w = \begin{pmatrix} \lambda_u \\ \lambda_v \end{pmatrix},$$

so that the minimising problem 6.2 can be reformulated as

$$\min_{x,b} [(xw + be) - t]^2. \quad (6.3)$$

Reformulate again the minimising problem 6.3 with

$$w' = \begin{pmatrix} \lambda_u \\ \lambda_v \\ b \end{pmatrix}, \quad x' = [u, v, e],$$

gives

$$\min_{w'} [x'w' - t]^2, \quad (6.4)$$

which is a simple linear problem.

Let us solve 6.4 by computing

$$\begin{pmatrix} \lambda_u \\ \lambda_v \\ b \end{pmatrix} = \text{pinv} \begin{pmatrix} u_1 & v_1 & 1 \\ \vdots & \vdots & \vdots \\ u_n & v_n & 1 \end{pmatrix}^{-1} \begin{pmatrix} u_1 & v_1 & 1 \\ \vdots & \vdots & \vdots \\ u_n & v_n & 1 \end{pmatrix}^T,$$

where \text{pinv} denotes the MOORE-PENROSE pseudo-inverse of a matrix, see [Moo20] and [Pen55]. In the output of the system (see eq. 5.13) it is aimed to address the proportion of excitatory neural signals $\lambda$ and derive the inhibitory proportion by $1 - \lambda$. For this we have to derive the following new values $a, b$ that comply $a + b = 1$ and $\frac{a}{b} = \frac{\lambda_u}{\lambda_v}$. Let us define $\lambda := a$. From solving these two equations with two unknowns we can derive $\lambda = \frac{1}{1 + \frac{\lambda_u}{\lambda_v}}$. 

60
6.4. Parameters used in the DNF

In addition to the evolution strategy [Han01] and the calculation of the parameter $\lambda$ (section 6.3), fine tuning of the parameters has been applied manually by comparing the simulations with the VSDI responses. The fitness function used in the CMA-ES is the sample correlation. As special attention has been payed to several detailed abilities of the DNF, additional fine tuning has been applied. The CMA-ES and the linear transform have been used to fit the data as best as possible from an objective point of view. Having this objective parameter fit, fine adaptation of some important parameters delivers the ‘icon on the cake’, that is a fit that satisfies our more special demands. After the fine-adaptation, additionally a complete grid search has been applied to the important parameters $\sigma_{ee}$ and $\sigma_{ie}$ as shown in Fig. 6.1 to bring out the best for this fixed parameter-set. The parameters used in the simulations (see eqs. 5.9, 5.7, 5.8, 5.10, 5.13, 5.14) are rounded off to two decimal places:

\[
\begin{align*}
\tau_u &= 1.70 \\
\tau_v &= 2.59 \\
a_{ee} &= 18.67 \\
a_{ie} &= 101.26 \\
\sigma_{ee} &= 13\pm1.66\text{mm} \\
\sigma_{ie} &= 18\pm2.29\text{mm} \\
g_S &= 299.71 \\
g_{S'} &= 8991.30 \\
h_u &= -63.63 \\
h_v &= -76.36 \\
\lambda &= 0.20 \\
c_u &= 0.5634 \\
c_v &= 0.5155 \\
g_e &= 4.6107 \\
\sigma_S &= 7.13\pm0.91\text{mm} \\
a_S &= 1 \\
u_0 &= v_0 = 33.47 \\
q_u &= q_v = 19
\end{align*}
\]

In the next two sections, some physiologically and computationally important parameters are discussed in detail.
Chapter 6. Optimising the parameters

6.4.1. Balancing the excitation and inhibition

The two parameters $\sigma_{ee}$ and $\sigma_{ie}$ describe the spatial extend of the lateral spread induced by long-ranging neural projections. The right tuning of the parameters comes out to be very important for the performance of the DNF used in this work (eqs. 5.7 and 5.8). The parameter $\sigma_{ee}$ describes the lateral spread of excitatory neurons that synapse to other excitatory neurons, whereas the parameter $\sigma_{ie}$ describes the projections of excitatory neurons that synapse to inhibitory neurons. An imbalance of these two parameters is able to destroy the expediency of the model.

To demonstrate this, a variation of these two parameters is performed keeping all other parameters fixed with values shown in section 6.4. The parameters are varied within the ranges of $\sigma_{ie} = 0$ to $\sigma_{ie} = 9$ millimetres and $\sigma_{ee} = 0$ to $\sigma_{ee} = 4$ millimetres. In Fig. 6.1 the respective correlation values (between VSDI-signals and simulations) are shown in case of the simulated square, bar and LM stimulus. The fourth picture (right, bottom) shows the mean correlation values between all three simulations and the according dye-signals. Red colours indicate high correlation values whereas blue colours indicate low correlation values (see colour-bars on the right of each picture). On the x-axis the $\sigma_{ie}$-values and on the y-axis the $\sigma_{ee}$-values are recorded. In the picture that shows the mean correlation values between simulations of all three stimuli and the according VSDI-data, a black line is printed: it shows the border where excitation and inhibition is of equal width. Left from the line excitation exceeds inhibition and right from the line inhibition exceeds excitation. Colour indicates the correlation values, see colour-bars on the right side of each correlation picture. When regarding the mean correlation values, most parts of the higher correlation values (red colours) are located right of the black line. However, there is one tail of red colour located left from the black line. This red tail is located at about $\sigma_{ie} = 2$ millimetres. Indeed, these two millimetres extent of the excitatory neurons projecting to the inhibitory neuron population are optimal within the system. In the model simulations, values of $\sigma_{ie} = 2.29$ millimetres and $\sigma_{ee} = 1.66$ millimetres are used which deliver a mean correlation value of 0.88 for the three stimuli (Square, Bar and LM). In this parameter setting, the height of the inhibitory interaction kernel $a_{ie}$ is more than five times bigger than the excitatory interaction kernel height $a_{ee}$ emphasising the strong effect of the inhibition. The interaction kernels used in our simulations are shown in Fig. 6.2, where the resulting spatial extent and the resulting amplitude can directly be compared.

The values of $\sigma_{ee}$ and $\sigma_{ie}$ correspond to the distance of the origin to the
6.4. Parameters used in the DNF

Figure 6.1.: Correlation values when balancing excitation and inhibition.

Correlation values (between simulations and dye-signals) are colour-coded for the bar (top left), LM (top right), square (bottom left) and averaged for all three stimuli (bottom right). The x-axis in each picture shows the $\sigma_{ie}$-values in millimetres, where the y-axis show the $\sigma_{ee}$-values in millimetres. The colour values correspond to the correlation values shown in the corresponding colour-bar right of each picture.

The inflection point of the Gaussian functions, as shown by the black dots in the two plots. The Gaussian function is also used as the normal distribution in probability theory, where the $\sigma$-value denotes the standard derivation. It is known that an area of about 95 percent under the normal distribution is located within the interval $[-2\sigma, 2\sigma]$.

When regarding the correlation values between simulated dye signals and real dye signals evoked by the square stimulus, it can be observed that in this setting,
Chapter 6. Optimising the parameters

Figure 6.2.: Gaussian weighting functions. In the simulations a very strong and long-ranging inhibitory kernel and a weaker and not so long-ranging excitatory kernel is used. The black dots on the x-axis denote the values $\sigma_{ee} = 1.66$ millimetres and $\sigma_{ie} = 2.29$ millimetres which are the abscissae values of the inflection points of the functions.

projections within the excitatory neuron population are advantageous when not exceeding the $\sigma_{ee}$-value bound of two millimetres. However, in this setting, highest correlation values arise when the $\sigma_{ee}$-values exceed $1.5$ millimetres. The square stimulus is a good measure for balancing excitation and inhibition, because it evokes very localised responses. Too much excitatory power leads very fast to an ‘overactivation’. For that reason a tuning of $\sigma_{ee} = 1.66$ millimetres is shown to be optimal within the DNF.

A more detailed physiological interpretation of the two parameters is given in section 9.3.

6.4.2. Ratio of excitatory and inhibitory activity in the dye-signals

As the output of the DNF is defined as a linear combination of the simulated excitatory and inhibitory activations, see eq. 5.13, the signals of the DNF show both inhibition and excitation. The ratio of excitation and inhibition is here expressed with the term $\lambda \in [0,1]$ that weights the excitatory signals. The inhibitory signals are weighted with $1 - \lambda$ accordingly. The parameter $\lambda$ has been calculated as described in section 6.3 for various parameter settings. Surprisingly, the value $\lambda$ tends always to be noticeable below 0.5, emphasising the
inhibitory part observed in the VSDI-pictures. Especially, the optimisation for
the used parameter set leads to a $\lambda$-value of $\lambda = 0.20$ that has been used in the
simulations.

6.5. Analysis of stability

In this chapter an analysis of stability will be performed on the equations of
the DNF, see eqs. 5.7 and 5.8 on page 50. The goal of the stability analysis is
to see how the DNF behaves when it is close to a stationary point. For this
purpose, one considers small deviations from a stable solution and linearises
the equations [vdM81]. With this technique one yields two linear equations that
are easy to solve. The system can then show two behaviours: it comes back
to the stationary point (stable point) or it runs away from the stationary point
(unstable point). The DNF should be stable as the absence of input or very
little input should not evoke responses far away from the resting membrane
potentials.

Hence, the DNF equations 5.7 and 5.8 are analysed analytically by a linear
stability analysis, where the approach of von der Malsburg [vdM81] (see
also [Ige03, Wen02]) is used. Let us start from the DNF-equations

\begin{align}
\tau_u \frac{\partial u(x,t)}{\partial t} &= -u(x,t) + h_u + \int w_{ee}(x,x') f_u(u(x',t)) dx' \\
\tau_v \frac{\partial v(x,t)}{\partial t} &= -v(x,t) + h_v + \int w_{ie}(x,x') f_u(u(x',t)) dx'
\end{align}

and reformulate the eqs. 6.5 and 6.6 by using the symbol of ‘$*$’ (see appendix
A.1). In the following the weights $w_{ie}$ and $w_{ee}$ will only depend on one variable,
indicating $w(y) := w(x - x') := w(x, x')$. Defining $\hat{u}(x,t) := \frac{\partial u(x,t)}{\partial t}$, $\hat{v}(x,t) := \frac{\partial v(x,t)}{\partial t}$, $f_u(u) := f_u(u(x,t))$ and $f_v(v) := f_v(v(x,t))$ yields:

\begin{align}
\tau_u \hat{u}(x,t) &= -u(x,t) + h_u + f_u(u) * w_{ee}(x) \\
&\quad - g_v \cdot f_v(v(x,t)) + g_s \cdot S(x,t) \\
\tau_v \hat{v}(x,t) &= -v(x,t) + h_v + f_u(u) * w_{ie}(x)
\end{align}

Considering now the homogeneous solution of 6.7 and 6.8 with $u(x,t) = \tilde{u}_0$
Chapter 6. Optimising the parameters

and $v(x,t) = \tilde{v}_0$ in the absence of external input ($S(x,t) = 0$):

$$
0 = -\tilde{u}_0 + h_u + f_u(\tilde{u}_0)W_{ee} - g_v f_v(\tilde{v}_0) \tag{6.9}
$$

$$
0 = -\tilde{v}_0 + h_v + f_u(\tilde{u}_0)W_{ie} \tag{6.10}
$$

with $W_{ee} = \int w_{ee}(x')dx'$ and $W_{ie} = \int w_{ie}(x')dx'$. To investigate the stability of the system, let us consider small disturbances $\epsilon(x,t) = u(x,t) - \tilde{u}_0$ and $\eta(x,t) = v(x,t) - \tilde{v}_0$ at timestep $t$:

$$
\tau_u \dot{\epsilon}(x,t) = -(\tilde{u}_0 + \epsilon(x,t)) + h_u + (f_u(\tilde{u}_0 + \epsilon) \ast w_{ee})(x,t) - g_v f_v(\tilde{v}_0 + \eta(x,t))
$$

$$
\tau_v \dot{\eta}(x,t) = -(\tilde{v}_0 + \eta(x,t)) + h_v + (f_u(\tilde{u}_0 + \epsilon) \ast w_{ie})(x,t)
$$

By applying the first order Taylor expansion on $f(\tilde{u}_0 + \epsilon)$ the system is linearised

$$
\tau_u \dot{\epsilon} \approx -\tilde{u}_0 - \epsilon + h_u + (f_u(\tilde{u}_0) + \epsilon f_u'(\tilde{u}_0)) \ast w_{ee} - g_v f_v(\tilde{v}_0 + \eta(x,t)) \tag{6.11}
$$

$$
\tau_v \dot{\eta} \approx -\tilde{v}_0 - \eta + h_v + (f_u(\tilde{u}_0) + \epsilon f_u'(\tilde{u}_0)) \ast w_{ie} \tag{6.12}
$$

where the dependencies of $x$ and $t$ are removed for simplicity. It is true that $(g + h) \ast f = g \ast f + h \ast f$, so that 6.11 and 6.12 become

$$
\tau_u \dot{\epsilon} \approx -\tilde{u}_0 - \epsilon + h_u + f_u(\tilde{u}_0)W_{ee} + f_u'(\tilde{u}_0)\epsilon \ast w_{ee} - g_v f_v(\tilde{v}_0 + \eta(x,t)) \tag{6.13}
$$

$$
\tau_v \dot{\eta} \approx -\tilde{v}_0 - \eta + h_v + f_u(\tilde{u}_0)W_{ie} + f_u'(\tilde{u}_0)\epsilon \ast w_{ie}. \tag{6.14}
$$

By subtracting the homogeneous solution 6.9 and 6.10 from 6.13 and 6.14 we get

$$
\tau_u \dot{\epsilon} \approx -\epsilon + f_u'(\tilde{u}_0)(\epsilon \ast w_{ee})(x,t) - g_v \eta f_v'(\tilde{v}_0) \tag{6.15}
$$

$$
\tau_v \dot{\eta} \approx -\eta + f_u'(\tilde{u}_0)(\epsilon \ast w_{ie})(x,t). \tag{6.16}
$$

As $F(f \ast g) = \hat{f} \hat{g}$ holds (see appendix A.1), where $\hat{f}$ and $\hat{g}$ are the Fourier transforms of $f$ and $g$ respectively, applying the Fourier transform in the spatial dimension in eqs. 6.15 and 6.16 makes the calculation of the convolution
6.5. Analysis of stability

terms easier:

\[
\tau_\nu \dot{\epsilon}(k, t) \approx -\dot{\epsilon}(k, t) + f'_u(\tilde{u}_0)\dot{\epsilon}(k, t)\hat{w}_{ee}(k) \\
- g_v \dot{\eta}(k, t) f'_v(\tilde{v}_0)
\]

\[
\tau_\nu \dot{\eta}(k, t) \approx -\dot{\eta}(k, t) + f'_u(\tilde{u}_0)\dot{\epsilon}(k, t)\hat{w}_{ie}(k),
\]

with

\[
\hat{w}_{ee}(k) = a_{ee} \frac{\sqrt{2\pi}}{\sqrt{\sigma_{ee}^2 k^2}} e^{-\frac{k^2}{2\sigma_{ee}^2}},
\]

when \(w_{ee}(x) = a_{ee} \frac{\sqrt{2\pi}}{\sqrt{\sigma_{ee}^2}} e^{-\frac{x^2}{2\sigma_{ee}^2}}\) and

\[
\hat{w}_{ie}(k) = a_{ie} \frac{\sqrt{2\pi}}{\sqrt{\sigma_{ie}^2 k^2}},
\]

when \(w_{ie}(x) = a_{ie} \frac{\sqrt{2\pi}}{\sqrt{\sigma_{ie}^2}} e^{-\frac{x^2}{2\sigma_{ie}^2}}\).

Reformulating 6.17 and 6.18 in vector form gives

\[
\begin{pmatrix}
\dot{\epsilon}(k, t) \\
\dot{\eta}(k, t)
\end{pmatrix}
\approx
A(k)
\begin{pmatrix}
\dot{\epsilon}(k, t) \\
\dot{\eta}(k, t)
\end{pmatrix},
\]

with

\[
A(k) =
\begin{pmatrix}
-\tau_u^{-1} + \tau_u^{-1}f'_u(\tilde{u}_0)\hat{w}_{ee}(k) & -\tau_u^{-1}g_v f'_v(\tilde{v}_0) \\
\tau_v^{-1}f'_u(\tilde{u}_0)\hat{w}_{ie}(k) & -\tau_v^{-1}
\end{pmatrix}.
\]

(6.19)

If the matrix \(A(k)\) has two distinct eigenvalues, the homogeneous and linear differential equation 6.19 can be solved as

\[
\begin{pmatrix}
\dot{\epsilon}(k, t) \\
\dot{\eta}(k, t)
\end{pmatrix}
\approx
T(k)
\begin{pmatrix}
e^{\lambda(k)_+} & 0 \\
0 & e^{\lambda(k)_-}
\end{pmatrix}
T^{-1}(k)
\begin{pmatrix}
\dot{\epsilon}_0(k) \\
\dot{\eta}_0(k)
\end{pmatrix},
\]

with the starting value \((\dot{\epsilon}_0(k), \dot{\eta}_0(k))^T\).

The columns of the matrix \(T(k)\) are the eigenvectors, where \(\lambda(k)_+\) and \(\lambda(k)_-\) are the corresponding eigenvalues. It holds that \(\lambda(k)_+^2 - \text{tr} A(k)\lambda(k)_+ + \text{det} A(k) = 0\) if \(\lambda(k)\) is an eigenvalue, where \(\text{tr} A(k)\) stands for the trace of the matrix. Therefore we get

\[
0 = \lambda(k)_+^2 - \lambda(k)_+ \left( \tau_u^{-1}f'_u(\tilde{u}_0)\hat{w}_{ee}(k) - \tau_v^{-1} - \tau_u^{-1} \right) + \tau_u^{-1}\tau_v^{-1}(1 - f'_u(\tilde{u}_0)\hat{w}_{ee}(k)) + \tau_u^{-1}\tau_v^{-1}g_v f'_v(\tilde{v}_0)f'_u(\tilde{u}_0)\hat{w}_{ie}(k).
\]

(6.20)
Solving this quadratic equation gives

\[
\lambda_{\pm}(k) = \frac{1}{2} \left( \tau_u^{-1} f'_u(\tilde{u}_0) \hat{w}_{ee}(k) - \tau_v^{-1} - \tau_u^{-1} \right) \\
\pm \left[ \frac{1}{4} \left( \tau_v^{-1} f'_v(\tilde{u}_0) \hat{w}_{ee}(k) - \tau_v^{-1} - \tau_u^{-1} \right)^2 \\
- \tau_u^{-1} \tau_v^{-1} \left( 1 - f'_u(\tilde{u}_0) \hat{w}_{ee}(k) \right) - \\
\tau_u^{-1} \tau_v^{-1} g_v f'_v(\tilde{v}_0) f'_u(\tilde{u}_0) \hat{w}_{ee}(k) \right]^{1/2}.
\]

(6.21)

For convenience, we write

\[
\lambda_{\pm} = B \pm \sqrt{B^2 + C}.
\]

If the real part of the largest eigenvalue is negative, then the system is asymptotically stable. To analyse the stability of the system, those points have to be considered where (local) bifurcations occur. Local bifurcations denote points where a small change of a parameter causes the stability of an equilibrium to change. This happens when the real part of an eigenvalue of the linearisation around the fixed point passes through zero. In our system, we have to consider a Hopf bifurcation, in which a pair of complex conjugate eigenvalues \((\lambda_{\pm})\) of the linearisation cross the imaginary axis of the complex plane, and a saddle point bifurcation, where two fixed points, one stable and one instable, collide and annihilate each other.

For the first case, we assume that the imaginary part of the eigenvalues is non-zero. This means \(B^2 + C < 0\), which implies \(C < 0\). The bifurcation occurs when the real part of \(\lambda\) is zero (i.e., \(\lambda_{\pm} = 0 \pm i\omega\), with \(\omega \in \mathbb{R}, \omega \neq 0\)). In this case, we have

\[
\tau_u^{-1} f'_u(\tilde{u}_0) \hat{w}_{ee}(k) - \tau_v^{-1} - \tau_u^{-1} = 0
\]

and therefore

\[
f'_u(\tilde{u}_0) \hat{w}_{ee}(k) = \frac{\tau_u}{\tau_v} + 1
\]

and applying this to the Gaussian kernel gives

\[
f'_u(\tilde{u}_0) \frac{a_{ee}}{\sqrt{2\pi}} e^{-\sigma^2 \frac{x^2}{4}} = \frac{\tau_u}{\tau_v} + 1.
\]

(6.22)

Thus, under the assumption that the discriminant \(B^2 + C\) is negative, the system
6.5. Analysis of stability

is asymptotically stable if $B < 0$ (the real part of the largest eigenvalue has to be negative, see above). That is, the following inequality has to be true

$$f'_u(\tilde{u}_0) \frac{a_{ee}}{\sqrt{2\pi}} e^{-\sigma^2 \frac{\tilde{r}^2}{2}} < \frac{\tau_u}{\tau_v} + 1.$$  \hspace{1cm} (6.23)

Now we look at the second case where $B^2 + C \geq 0$. Then the system is obviously not asymptotically stable for $B \geq 0$, therefore we consider $B < 0$. The bifurcation point is $\lambda \pm \lambda = 0 + 0i$. From eq. 6.20 we see that this equality holds if $C = 0$:

$$-\tau_u^{-1} \tau_v^{-1} (1 - f'_u(\tilde{u}_0) \hat{w}_{ee}(k)) - \frac{\tau_u^{-1} \sigma^2}{\tau_v} g_u(f'_u(\tilde{u}_0)f'_u(\tilde{u}_0)\hat{w}_{ie}(k)) = 0$$

For non negative discriminant and $B < 0$ this leads to the stability condition

$$f'_u(\tilde{u}_0) \hat{w}_{ee}(k) - 1 < g_u f'_u(\tilde{u}_0)f'_u(\tilde{u}_0)\hat{w}_{ie}(k).$$  \hspace{1cm} (6.24)

To summarise, the analysis reveals that the system is asymptotically stable if both inequality 6.23 and inequality 6.24 are fulfilled.

The inequality 6.23 is satisfied with the parameter setting in this study: The axonal response $f_u$ is a sigmoid function with $c_u = 0.56$, $q_u = 19$ and $u_0 = 33.47$, see section 6.4, as plotted in Fig. 6.3. With this parameter setting, the slope of the function $f_u$ at $\tilde{u}_0 < 20$ is nearly 0, so that 6.23 holds if $\tau_u > 0$ and $\tau_v < \infty$, as $\tilde{u}_0 \approx h_u = -63.63 < 20$ when no stimulus has been presented. The second inequality 6.24 is also satisfied with the parameter setting as the inhibition is chosen much stronger than the excitation. For the system to be stable, it has been shown that the inhibition needs to be stronger than the excitation in the sense of eq. 6.24. This seems to be intuitively clear as an overexcitation that cannot be stopped by the inhibitory power causes instability. The parameter optimisation result also supports this. However, that the stability of the DNF (5.11, 5.12) depends on the slope of the excitatory axonal response function at the point of the homogeneous solution (see eq. 6.22) seems to be surprising at first sight. But this can be understood when considering the not activated system or neural tissue when no stimulus has been presented. If little disturbances of membrane potentials occur, one expects a population of excitatory neurons not to fire (remember that we model the mean responses of thousands of neurons). This can be formalised with an axonal response function that does not fire when the system is about in rest (resting membrane potential $u_0 = -63.63$). The axonal function shows a firing behaviour when its derivative clearly deviates from zero, as it has a sigmoidal shape. In Fig. 6.4 simulations are shown when
no stimulus has been presented. One can expect that the system is stable the greater the distance from threshold to the resting membrane potential is in the rightward direction. For that reason, the threshold has been pushed leftwards toward the resting membrane potential. It can easily be seen that when the threshold reaches the membrane potential \( (u_0 = -70 < h_0 = -63.63) \) oscillations directly occur (Fig. 6.4 top left). When the threshold value is \( u_0 = -30 \), oscillations sum up over time and become clearly visible at about 200 milliseconds (Fig. 6.4 bottom left). When using a threshold value of \( u_0 = 30 \) that is far away from the membrane potential, no oscillations occur (Fig. 6.4 right), as the slope of the axonal response function about resting level is nearly zero.

When investigating what happens if the threshold value is continuously changed within a parameter regime of \(-400\) to \(300\) millivolts, we can observe that for the very large and very small membrane potentials the smallest oscillations occur. For this purpose, the maximal difference of outputs when no stimulus is presented is plotted against the threshold value (see Fig. 6.5). This is in accordance with the observation that the system becomes stable when the derivative at the threshold value becomes zero: when shifting the axonal response function to the very right and very left results in the two cases, where either the resting membrane potential is at the place where the sigmoid converges to zero or the saturation. In both cases, the derivative of the sigmoid has values that converge to zero.

It can be concluded that with the parameter setting used in this work, see section 6.4, the DNF, see eqs. 5.7 and 5.8 is proved to be stable.
6.5. Analysis of stability

Figure 6.4: Emerging oscillations when decreasing the threshold value. The simulations are shown when presenting no stimulus to the DNF. From top left to bottom right, the threshold values have been increased, see the according axonal response functions in the lower rows (white backgrounds). First, a threshold value $u_0 = -70$ is tested that is smaller than the resting membrane potential of the excitatory layer $h_u = -63.63$ (compare red and black dot). Oscillations emerge right from the beginning of the simulations. After that, a threshold value of $u_0 = -70$ is tested (top right), which causes oscillations after about 250 milliseconds. When testing a threshold value of $u_0 = -30$ (bottom left) oscillations emerge after about 200 milliseconds. When using a threshold value of $u_0 = 30$ (bottom right) no oscillations are observed.
Chapter 6. Optimising the parameters

Figure 6.5: Quotients between maximal and minimal values. The differences (max./min) between maximal and minimal values of the simulations when no stimulus has been presented with varying threshold values. The red dot denotes the resting membrane potential, the black dot the used threshold value and the grey dots indicate the threshold values used in Fig 6.4.
Chapter 7.

Results

The performance of the DNF is tested on data derived from VSDI on cat’s area 18. The output of the model—a linear combination of the DNF’s excitatory and inhibitory activations—is used to simulate the responses of the dye-signals. The dye-signals show overall activities comprising excitatory and inhibitory contributions at the same time. Therefore, a separate study of excitation and inhibition with the DNF is used to make predictions on the neural responses of the two populations.

Our data basis consists of VSDI experiments in which stationary, moving and illusory moving stimuli have been presented, see Fig. 3.3, page 27. In this work, three visual stimuli are especially important: the moving square (1), the drawn-out bar (2) and the LM stimulus (3). The stimulus of a square gradually drawn-out to bar length equals in perception the LM-stimulus, a bar that follows a square, briefly presented at one of the bar ends: The bar is not perceived as appearing at once, but as gradually expanding from the former square position [Hik93a, Hik93b], see chapter 4. JANCKE et al. showed that this illusory motion effect can already be observed in the primary visual cortex of cats: the authors could show that the dye-signal in response to the LM are ‘indistinguishable from real motion in this area’ [Jan04a]. The moving square stimulus is differently perceived: The perception equals a local square moving from top to bottom on the screen and not a square drawn-out to bar length. Surprisingly, despite this perceptual difference, the dye signals evoked by the three stimuli resemble each other, see Fig. 3.7, page 31.

In this chapter, first, simulations of the dye-signals evoked by the stimuli square, bar, LM and drawn-out bar are presented. Herein, non-linear effects within the dye signals and the DNF and the different propagation speeds of high and low-fluorescence signals are studied. The necessity for normalisation effects in the LGN is shown for the simulations evoked by these four stimuli. After that, two physiologically important parameters that encode the lateral spread of excitatory and inhibitory neuron populations (see section 2.4) are varied. This offers an insight into what happens when the interplay between excitation and
inhibition becomes out of balance within the DNF.

The DNF responses when stimulating with the three special stimuli, moving square, drawn-out bar and LM are particularly studied by simulating the excitation, the inhibition and the linear combination of both populations. After that it will be shown that the parameter setting of the DNF predicts an early excitation and a late inhibition.

After that it will be tested how the DNF performs on moving square stimuli evoked responses. The term ‘moving squares’ denotes square stimuli moving from top to bottom on a screen with velocities covering values between 4 and 64 degrees per second, see Fig. 3.3e on page 27.

The so called reverse LM stimulus, denotes a bar followed by the presentation of a square that is aligned to the top of the bar. The responses evoked by the presentation of the reverse LM has not been measured yet with VSDI. Predictions are made by simulating the responses to this stimulus with the DNF in this work. It has been reported that the reverse LM stimulus is mostly perceived by humans as a shrinking bar toward the square position ([vGr94], [Schm97]). It therefore becomes interesting, if the DNF-responses evoked by the rLM show any kind of motion-effect in the simulations of excitation and inhibition.

7.1. Square, bar, LM and drawn-out bar

In this section, the performance of the DNF will be shown on the VSDI-data already presented in Fig. 3.5 in chapter 3. In the simulations with the DNF, the parameter setting proposed in section 6.4 is used to account for the dye-signals. The optimisation of the parameters has been performed by the CMA-algorithm that optimises the correlation between the simulations and the dye signals (see section 6.1 for more details) for the square, bar and LM-evoked dye-signals of the experiment of the 8th of May 2001. All other simulations presented in this study are predictions made by the DNF without using further optimisations. In the case it is clearly stated, slight changes of particular parameter settings have been applied to account for the different qualitative behaviour of dye-signals from other experiments than that of the 8th of May 2001. These changes were necessary as with changing the experiment also the cat tested changes (including different eye-sights of the cats for example) and possibly also the behaviour of the dye-signals applied to the cortex as the application of the dyes is a manual task.

In Fig. 7.1 the simulated and real dye signals evoked by the square (a), bar
7.1. Square, bar, LM and drawn-out bar

(b), LM (c) and drawn-out bar (d) stimulus taken from the first experiment (8th of May) are depicted. Red and yellow colours indicate high fluorescence changes whereas blueish colours indicate low fluorescence changes in simulated (second column) and real dye-signal responses (third column), see legend on the right. Throughout this work, the simulated responses are presented in grey background boxes, whereas the VSDI-data are shown in yellow background boxes.

Figure 7.1.: Simulated and real dye-signal responses (8th of May 2001). Simulations and the according data from the experiment of the 8th of May 2001 (averaged across 16 trials). The underlying visual stimuli are a) the square, b) the bar, c) the LM and d) the drawn-out bar stimulus. In the second column, the DNF responses to the according stimuli are shown, where in the third column the corresponding real dye-signals averaged across 16 trials are taken up for comparison. The white arrows in c indicate the continuous spread of high fluorescence changes (see colour bars right) in simulations and real data when presenting the LM stimulus. This continuous spread is comparable to that evoked by the drawn-out bar stimulus, see simulation in d.
Chapter 7. Results

Figure 7.2: Simulated and real dye-signal responses 22nd of April 2003. Simulations and the according data from the experiment of the 22nd of April 2003 (averaged across 16 trials).

All three stimuli settings, square, bar and LM evoke DNF-responses that look similar to the fluorescence changes observed in the VSDI-data (compare second and third column). The white arrows in the simulation and real data presentation in response to the LM (c) stress the gradual spread of fluorescence signals, i. e. the line-motion effect. The correlation coefficient between the simulations of the square, bar and LM stimulus and the corresponding VSDI-data (about 200 milliseconds from stimulus onset considered) is 0.88. The drawn-out bar stimulus evokes simulated activity as depicted in Fig. 7.1d. The VSDI signals in response to the drawn-out bar stimulus have not been measured in this experiment. Therefore, the according measured data are missing. In the second experiment, the drawn-out bar evoked responses have additionally been measured. Fig. 7.2 depicts the VSDI-data (right column), the simulations (middle...
7.1. Square, bar, LM and drawn-out bar

column) and the according stimuli (left column). To account for the data of this second experiment, it became necessary to reduce the excitatory interaction kernel height $a_{ee}$ to 57 per cent of its original height. As with experiments the cat tested changes, this adaptation may be necessary because of different eyesights of the cat or different features or the bahaviour of the dye applied to the cortex for example. With this adaptation, the dye-signals evoked by the four

![Visual Input](image)

**Figure 7.3.: ‘Best of’ simulated and real dye-signal responses.** Simulations and the according data from the experiment of the 8th of May 2001 and the 22nd of April 2003 (drawn-out bar) (averaged across 16 trials). The black and white arrows in the DNF simulations of the LM-stimulus indicate the spread of lower and higher fluorescence changes. Note that the angle decreases with increasing fluorescence intensity (from left to right arrow).

stimuli are simulated, compare second and third column in Fig. 7.2a-d. It can
be observed that in this experiment, the measured VSDI-signals in response to the LM stimulus show a not so clear gradual drawing out of higher fluorescence signals, compare yellow and red colours in Figs. 7.1c and 7.2c right columns. A tendency towards a separate response evoked by the preceding square stimulus can be observed in the simulations of the LM-responses in the second experiment, see 7.2c second column first yellow colours. It can further be observed that all responses tend to decay earlier in the second than in the first experiment although the same stimuli have been presented, compare VSDI-responses in both experiments. This qualitative change in the responses seems to be arbitrary and is rudimentarily simulated by changing the excitatory kernel height. The responses to the square and drawn-out bar stimulus are simulated with decaying high fluorescence signals similar to the real dye signals, compare second and third column in 7.2a and d, but the LM is simulated with a later decay than observed in the real dye signals, see 7.2c.

In Fig. 7.3 the ‘best of’ experiments are plotted in one figure.

7.1.1. Non-linear effects in the dye-signals

The question whether nonlinear effects play a role when simulating VSDI-signals with a DNF is tested on the square and bar stimulus by plotting the sum of the responses to the two stimuli versus the response to the LM stimulus, see Fig. 7.4. It can be observed that in both simulations and dye-signals, the superposition of the two responses differs from the response to the LM stimulus. It is obvious that the superposition is more biased toward the response evoked by the later bar presentation (see Fig. 7.4 upper row), whereas the response evoked by the LM-stimulus starts earlier with a more gradual drawing out of high fluorescence signals (see Fig. 7.4 lower row). It can be concluded that non-linear effects play a role in creating the LM effect in both the real primary visual and its simulations.

7.1.2. Propagation speed of high and low fluorescence signals

It can be seen that the effect of continuously moving out high fluorescence changes evoked by the LM-stimulus in the VSDI-data is exactly simulated with the DNF, which is stressed with the white arrows in Fig. 7.3c. The white arrows in both pictures have a gradient of about 3mm/70ms which corresponds to about 0.04m/s moving speed.

JANCKE et al. showed that with increasing amplitudes of dye-signals the propagation speeds of the activities decrease [Jan04a]. The model simulations support this observation: In the simulated responses to the LM two further arrows
7.1. Square, bar, LM and drawn-out bar

Figure 7.4: Superposition of the responses evoked by the square and bar stimulus. In the upper row the superpositions of the DNF-simulations (left) and the VSDI-signals (right) in response to the square and bar stimulus is shown. In the lower row the responses to the LM stimulus is presented for comparison.

are added by visual judgement, indicating very high (grey arrow) and low (black arrow) fluorescence changes, see Fig. 7.3c. It can clearly be seen that the gradient of the three arrows decreases from left to right. As the dye-signals are linearly correlated with membrane potentials, thesis of decreasing propagation speed with increasing membrane potentials can be supported. A mathematical calculation of the propagation speeds in the simulation in response to the square stimulus is shown in Fig. 7.5. In the left picture of the figure, contour lines of the simulated dye signals are plotted. The most leftward point of each contour line has been linearly connected to the most downward point of each according contour line (see the two white lines). The derivatives of these linear lines have been used as the propagation speeds (in metre per second) of the according levels of fluorescence changes. These speeds have been plotted against the according fluorescence levels in the right picture of Fig. 7.5. It can clearly be seen that with increasing fluorescence signals, the propagation speeds decrease, which supports the observation by JANCKE et al. [Jan04a].
7.1.3. Simulating the LGN: suppression effects by normalisation

When simulating neural population activities, we have to cope with spatial and temporal integration effects. Stimuli of smaller sizes evoke signals that integrate over time much less than responses to bigger stimuli in LGN as well as cortex. Therefore, the square and the bar stimulus evoke different simulated amplitudes of dye signals.

In the VSDI-data of cat area 18, an amplitude difference between maximal square and maximal bar responses of a factor of 1.24 can be observed. Without normalisation in the LGN and with the optimised parameter setting (see section 6.4) a simulated response increase from square to bar case of a factor of 3.40 arises, whereas with normalisation, an increase of just a factor of 1.47 is achieved, see Fig. 7.6. In Fig. 7.6 the temporal course of one fluorescence pixel in response to the square stimulus (a) and in response to the bar stimulus (b) is shown for the real dye-signal (second column), the DNF without normalisation (third column) and with normalisation (fourth column) with the used parameter setting.
7.1. Square, bar, LM and drawn-out bar

**Figure 7.6.: Fluorescence amplitudes.** The responses of one VSDI-pixel (averaged across 16 trials) to the presentation of a) a square and b) a bar stimulus. In the VSDI-data a response increase of 1.24 of maximal activity from square to bar case is observed, see value in the last row under the VSD-measurements. When simulating the dye-signals with a DNF without normalisation a response increase of 3.40 occurs, whereas the same simulation with normalisation leads to a response increase of 1.47.

The quotient of maximal response to the bar stimulus and maximal response to the square stimulus is depicted in the lowest line of Fig. 7.6. Hence, using the DNF without normalisation results due to temporal and spatial integration with this parameter setting in a too large response increase from square to bar-case.

Fig. 7.7 depicts the simulated LGN- and dye-signal responses once without normalisation in the LGN (dark grey box) and once with normalisation in the LGN (bright grey box) in response to the square (a), bar (b), LM (c) and drawn-out bar stimulus (d). It can be seen that due to spatial and temporal integration, the response to the square stimulus in the LGN without normalisation is much weaker than the response to the bar stimulus. Using the normalisation, this amplitude difference is even reversed, as in the simulated LGN the maximal response amplitude evoked by the square is stronger than the maximal response amplitude evoked by the bar stimulus. In the cortex, this reversed effect gets lost, because of further spatial and temporal integration effects. But the square stimulus evokes nevertheless enough response amplitude, so that the bar evokes just of a factor of 1.47 a higher maximal response fluorescence changes, which is
Chapter 7. Results

Figure 7.7.: The effect of LGN-normalisation on amplitudes. LGN- and DNF simulations once without normalisation effects (darker grey box) and once with normalisation effects (brighter grey box). Note that the square stimulus evokes no high fluorescence changes (no red colours) in the DNF-response without normalisation (a, third column). Simulated DNF-response with normalisation evokes higher fluorescence signals (a, fifth column), which is comparable to the real dye-response (a, sixth column).

approximately comparable to the change observed in the real dye-signals of 1.24. In the last column of Fig. 7.7 the VSDI-signals are depicted for comparison.

7.1.4. Effects of an imbalance of excitation and inhibition

The widths of the interaction kernels in both layers have been fine balanced between excitation and inhibition, see section 6.4.1. But what happens if the kernels are imbalanced toward the one or other direction? To account for this question, a variation of both kernel widths ($\sigma_{ee}$ and $\sigma_{ie}$) and kernel heights ($a_{ee}$ and $a_{ie}$), see eq. 5.10, page 51, is performed by keeping all other parameters fixed.

First, let us vary the width of the Gaussian kernels, so that an imbalance between excitation and inhibition emerges. To do so, let us decrease the inhibitory kernel width from $\sigma_{ie} = 2.29$ to $\sigma_{ie} = 0.12$ millimetres. The resulting effect is shown in Fig. 7.8c. It can be seen that in each stimuli configuration case, the simulated high fluorescence signal (red areas) spreads out very fast and
too wide, as the inhibition looses control over the excitatory activity (compare c and e). In case of the LM-stimulus (third row), the angle with which the high fluorescence area (red colours) spreads out is similar in the upper and lower direction, so that the typical LM-effect is lost.

\[ \sigma_{ie} = 0.12 \text{ millimetres}, \]
\[ \sigma_{ee} = 0.38, \sigma_{ie} = 12.47. \]

Figure 7.8.: Excitatory and inhibitory kernel width imbalances. a) The stimuli, b) the balanced DNF-responses, c) the imbalanced DNF-responses, in which the inhibitory kernel width has been reduced \( (\sigma_{ie} = 0.12 \text{ millimetres}) \), d) imbalanced DNF-responses in which the excitatory kernel width has been reduced and the inhibitory kernel width has been tremendously enlarged \( (\sigma_{ee} = 0.38, \sigma_{ie} = 12.47.) \) e) The according VSDI-signals.

In the next setting, a decrease of the excitation width to \( \sigma_{ee} = 0.38 \text{ millimetres} \) and an increase of the inhibition width to \( \sigma_{ie} = 36.91 \text{ millimetres} \) is applied. In Fig. 7.8d it can be seen that in case of the square and LM-stimulus, only a very short excitatory response occurs followed by a long-lasting inhibition. In case of the square stimulus, nearly no high fluorescence signals (red colours) can be evoked, but low fluorescence signals spread out too wide representing the extremely wide-ranging inhibitory kernel. In case of the LM stimulus, the high fluorescence response evoked by the square stimulus is separated from the high fluorescence area evoked by the bar stimulus, see red areas in Fig. 7.8d (third row).
Chapter 7. Results

Figure 7.9: Excitatory and inhibitory kernel height imbalances. a) The stimuli, b) the balanced DNF-responses, c) imbalanced DNF-responses. The excitatory kernel height has been enlarged ($a_{ee} = 28.67$). d) Imbalanced DNF-responses. The excitatory kernel height has been reduced ($a_{ee} = 5.0$). e) The according VSDI-signals.

Second, let us vary the strength of interactions by variation of the Gaussian kernel heights. For this purpose, the excitatory kernel height is varied in two directions: Increasing the excitatory kernel height to 154 per cent of the original kernel height evokes the responses shown in Fig. 7.9c. The effect of increasing the excitatory kernel height is similar to increasing the excitatory kernel width, compare Fig. 7.8c and Fig. 7.9c. The high fluorescence areas spread out too fast and too wide, so that the responses to the square, bar and LM look nearly similar, compare red areas in the pictures. Reducing the excitatory kernel height to about 30 per cent of the original kernel height evokes the responses depicted in Fig. 7.9d, which are comparable to the responses evoked by reducing the excitatory kernel width and enlarging the inhibitory kernel width, compare Fig. 7.9d and Fig. 7.8d. The square stimulus also evokes nearly no high fluorescence, see Fig. 7.9d, first row. When presenting the LM stimulus, the spiking response to the square is separated from the spiking response to the bar stimulus, therefore the LM-effect is not so clearly observed (third row).
To summarise, with the used parameter setting, enhancing (reducing) the height of the interaction kernels has similar effects as enhancing (reducing) the kernel widths.

### 7.2. Simulating the excitatory and inhibitory activations separately

The output of the DNF used in this study is a linear combination of its simulated excitatory and inhibitory activities, see eq. 5.13, page 52. This makes it possible to observe the excitatory and inhibitory activations separately without affecting the interaction between both layers (see eqs. 5.11 and 5.12, page 52).

The three stimuli, particularly focused on in this study, the moving square, the drawn-out bar and the LM stimulus, have been shown to evoke similar VSDI-signals, see Fig. 3.7 on page 31. In Fig. 7.10, the separate responses of the DNF’s excitatory and inhibitory activations are depicted. In the figure, each row shows the responses of one layer, stressed with different contrasts of the model neurons on the left. The last row shows the simulated dye-signals, which are a linear combination of both layers. As the VSDI-signals might be dominated by inhibition ($\lambda = 0.20$ in eq. 5.13, page 52), the simulated VSDI-signals (fourth row) resemble that of the simulated inhibitory neuron population (third row) strongly. The visual stimuli are presented in the first row of Fig. 7.10: a) the LM, b) the drawn-out bar and c) the moving square (32 degrees per second) stimulus. The second line depicts the corresponding simulations of the excitatory neuron layer ($\lambda = 1$), the third row the corresponding simulations of the inhibitory layer ($\lambda = 0$) and the fourth row the simulations of the dye-signals ($\lambda = 0.20$).

It can be seen that the simulated excitatory activation evoked by the moving square (c) differs from the responses evoked by the LM (a) and drawn-out bar (b) stimulus. This difference exactly equals the perceptual scenario: The LM and drawn-out bar stimulus are perceived equally as a ‘motion streak’, whereas the moving square stimulus is perceived differently as a local patch in motion. On the most right, the excitatory response to the bar stimulus is presented, so that the stationary bar response can directly be compared to the ‘motion-streak-like’ response evoked by the LM-stimulus. The inhibitory responses (third line of Fig. 7.10), evoked by the three stimuli resemble each other, which is comparable to the inhibitory dominated simulated dye-signal responses (fourth line of Fig. 7.10).

It can therefore be concluded that in case of stimulating with the moving square, the inhibition dominated simulated dye-signals (and also the real dye-
Chapter 7. Results

Figure 7.10. Excitation read out for perception. The stimuli tested are the a) LM, the b) drawn-out bar and c) the moving square stimulus (32 degrees per second speed). The last row shows the respective DNF-simulations of the dye signal ($\lambda = 0.20$), the second row depicts the excitatory activations (by setting $\lambda = 1$ in eq. 5.13) and the third row depicts the separate responses of the inhibitory layer ($\lambda = 0$). Like in perception, in case of the excitatory activity, the drawn-out bar and the LM-responses look similar (compare a and b in the second row), whereas the excitatory response to the moving square stimulus (c) looks quite different. But we can see no essential differences in the simulations of the dye signal and of the disassembled inhibitory responses in all three cases (see third and last row a-c). The bar stimulus response of the excitatory layer is depicted in d), so that the continuous drawing-out of activity in case of the LM-stimulus in a can be compared to the stationary bar response.

signals) mismatch the perceptual scenario. The simulated excitatory neuron contributions deliver responses that match much better the perceptual scenario. Physiological reasons that might be responsible for this observed phenomenon are presented in section 2.6: several works support the hythesis that excitatory neurons project from the primary visual cortex to the higher brain area PMLS, an area that is supposed to be concerned with the processing of moving stimuli in the cat.

86
7.3. Early excitation, late inhibition

The optimisation of the time constants in the DNF lead to values for which $\tau_u < \tau_v$ holds. This has the effect that the excitatory neurons respond faster than the inhibitory neurons, which is also supported by the fact that the stimulus couples into the excitatory neuron layer. In the model, the inhibitory neurons are activated by the excitatory neurons and can herewith respond proportional to their time constant $\tau_v$. In Fig. 7.11, the excitatory and inhibitory activations evoked by the a) square, b) bar and c) LM stimulus are shown separately. It can be observed that—as expected—the excitatory response appears earlier than the inhibitory response, compare second and third row in Fig. 7.11. This effect of an *early excitation and late inhibition* is especially strong when presenting the square stimulus, which covers a smaller portion of the visual field. When presenting larger stimuli like a bar stimulus, spatial and temporal integration effects tend to compensate the late onset of activities in the inhibitory responses by boosting the signals to reaching earlier higher fluorescence signals. That the excitation responds comparably fast is especially important when considering that it is read-out from higher areas for perception, see section 2.6.

**Figure 7.11.: Early excitation and late inhibition.** a) The square, b) the bar and c) the LM stimulus depicted with the evoked disassembled excitatory and inhibitory responses and the simulated dye-signals. The excitatory responses tend to respond faster than the inhibitory signals, compare second and third row.
7.4. Latency compensation in the primary visual cortex predicted by the DNF

The performance of the DNF is tested on stimuli called ‘moving squares’. This denotes square stimuli moving with velocities of 4, 8, 16, 32 and 64 degrees per second downward on the screen. Data derived from three different experiments are available: in the first experiment (8th of May 2001) moving square stimuli with the velocities of 4, 8, 16 and 32 degrees per second have been tested (see Fig. 7.12) whereas in the other two experiments (22nd of April 2003 and 19th of March 2001), moving square stimuli with the velocities of 8, 16, 32 and 64 degrees per second have been tested (see Figs. 7.13 and 7.14).

As the processing of moving stimuli within the visual pathway and especially in the primary visual cortex is not fully understood, two questions concerning the moving square stimuli evoked dye-signals are in the centre of interest:

- Is the DNF with preceding LGN model able to account for the VSDI-pictures evoked by moving square stimuli?
- Does the VSDI-pictures and/or the simulations give evidence for latency compensation phenomena, especially concerning the excitatory neural responses in the simulations?

The phenomenon of latency compensation has been described in section 4.2. To investigate latency compensation phenomena in this study, eccentricity effects are considered when presenting the projected stimuli at different positions on the cortex: With increasing distance from the area centralis, visual stimuli are processed in smaller regions. This has an effect on the positions of the stimuli on the cortex, see Figs. 7.12, 7.13 and 7.14: The stimulus position is projected in form of dashed white lines in the pictures. Eyed, it can be seen that the stimuli do not move linearly but slightly curved due to the mentioned eccentricity effects.

As mentioned above, when observing fluorescence signals in different experiments, the dye-signals behaviour changes from experiment to experiment making it necessary to adopt some of the parameters accordingly: As with experiment the cat tested changes, all individual differences like different eye-sights for example influence the responses. Further, it is not possible to apply the dye-molecules absolutely identical on the cortex of all cats. This might also have effects on the dye signal behaviour. All these factors influence the dye-signals so that when simulating the dye-signals from experiment to experiment some of the DNF’s parameters have to be changed. This parameter change concerns
7.4. Latency compensation in the primary visual cortex predicted by the DNF

mainly the setting of the time constants and the power of excitation \( a_{ee} \) and inhibition \( g_v \).

**Figure 7.12.:** Moving squares with different velocities, experiment 8th of May 2001. **Upper line:** VSDI-responses averaged across 16 trials to the according moving stimuli. **Second line:** DNF-Simulations to moving square stimuli with velocities 4, 8, 16 and 32 degrees per second (from left to right). **Third line:** Simulated excitatory activations. Dashed lines indicate the stimulus positions.

The DNF-simulations of the dye-signals evoked by moving square stimuli in the experiment of the 8th of May are shown in Fig. 7.12. In the upper row, the VSDI-signals are shown with increasing moving speed of the underlying square stimuli from left to right. In the second row, the according simulations of the dye-signals are presented, and in the last row, the simulated excitatory neural activities are depicted. It can be observed that until the velocity of 16 degrees per second, both simulated and VSDI high fluorescence areas (red and yellow colours) clearly cover the projected stimuli positions. At the velocity of 32 degrees per second, simulations and VSDI high-amplitude areas begin to lag behind the stimuli positions. However, when considering the activation of the excitatory neurons in the simulations, the stimulus position is covered by the highest fluorescence values in all the cases of underlying stimuli speeds.

To account for the second experiment (22nd of April 2003), the kernel height \( a_{ee} \) has been reduced to 14.3 per cent of its original height, the time constants have been enlarged (\( \tau_{ee} = 4.40, \tau_{ie} = 4.79 \)) and the strength of inhibition has been reduced \( g_v = 3.01 \). With this adaptation, the dye signals of this exper-
Chapter 7. Results

iment can be simulated. The same effect as in the first experiment can be observed, see Fig. 7.13: until the stimulus velocity of 16 degrees, the high fluorescence areas (red and yellow colours) of both simulations and VSDI-responses cover the stimuli positions (see Fig. 7.13). At 32 degrees per second stimulus speed, the high fluorescence areas again begin to lag behind the area of stimulus presentation. At 64 degrees per second moving speed, the area covered by the stimulus presentation nearly completely outpaces the high fluorescence areas. When observing the excitatory neural activity, we can see the same effect as in the experiment of the 8th of May 2001. The highest fluorescence areas even seem to outrun the stimuli positions. It must be noted that this ‘outrun’ is also supported by the fact that the eccentricity effects are not considered in the simulations. The eccentricity has been used to be consistent with the VSDI-data in the plots.

Figure 7.13.: 22nd of April 2003. Same data presentation as in Fig. 7.12 with data from another experiment also averaged across 16 trials. Here, the velocities of 8, 16, 32 and 64 degrees per second have been tested.

To account for the third experiment (19th of March 2001), a new parameter setting has been used. This is due to the fact that this experiment shows another different characteristic in the responses than the other two experiments: For this, four values have been changed: The time constants have been enlarged from $\tau_u = 1.46$ to 15.27 and from $\tau_v = 8.22$ to 11.99 and the excitatory kernel height $a_{ee}$ has been reduced to 81.70 per cent of its original kernel height and
the inhibitory power is slightly enlarged $g_i = 4.61$. The effects described in the previous two experiments are also supported with this setting (Fig. 7.14): The high fluorescence areas (red colours) of both simulations and VSDI-responses cover the stimuli positions until the velocity of 16 degrees per second. At 32 degrees per second the border of loosing this covering ability can be observed, whereas at 64 degrees per second high fluorescence areas completely lag behind stimuli positions. When simulating the excitatory activations, highest fluorescence areas cover the corresponding stimuli positions in the case of all presented moving stimuli until the highest presented moving speed.

![Figure 7.14: 19th of March 2001. Same data presentation as in Fig. 7.12 averaged across 16 trials with data taken from a further experiment. Here, the velocities of 8, 16, 32 and 64 degrees per second have been tested.](attachment:figure7.14.png)

A quantification of the temporal lag between fluorescence signals of a special level and stimulus position is given in Fig. 7.15. For 30, 50 and 65 per cent of the maximal fluorescence signal, the contour-lines of these levels have been calculated. The mean temporal position of the stimulus is subtracted from the mean temporal value of these contour-pixels. This value is used to calculate the temporal lag between the responses of the different levels and stimulus positions. In Fig. 7.15 the ‘lag-curve’ for the simulations is shown in grey colours, whereas the ‘lag-curve’ for the VSDI-data is given in yellow colours. It can clearly be seen that over 32 degrees per second stimulus speed (black vertical lines in the pictures) both simulations and dye-signals show a positive lag. Until 16 degrees per second stimulus speed, the lag can be found around zero and is also partly
Figure 7.15.: Temporal lags for different stimulus velocities. For two experiments (upper and lower row), the temporal lags between the simulated responses and the stimulus position (grey lines) and between the real dye signals and stimulus position (yellow lines) have been calculated. From left to right different levels of response activity have been considered. Most left, 30 per cent of maximal fluorescence level, in the middle 50 per cent and most right 65 per cent of maximal fluorescence level have been considered in the calculation of the lags.

Even in the negative timescale in both experiments and simulations for all three observed fluorescence levels. Furthermore, a tendency of increasing lag with increasing velocity is present when taking all pictures into account.

Hence, we have observed that fluorescence areas in response to moving square stimuli cover the region of the stimuli representation until the velocity of about 32 degrees per second. Considering the neural processing time needed by information to reach the early visual cortex, one should expect the VSDI-signals to lag behind stimulus position. However, in our experiments, this is not the case.
for moving square stimuli until the velocity of about 32 degrees per second. In case of the excitatory neuron population, we even observed no latency effects. It can therefore be supposed that there occur latency compensation phenomena—here observed in cat area 18—on the way from retina to primary visual cortex, which shall be investigated in the next section.

7.4.1. Preactivation via lateral connections accelerates the processing of moving stimuli

Not only the VSDI pictures, also the simulations show latency compensation effects. According to JANCKE et al., it might be suggested that those compensation phenomena are due to the activity spread of sub- and suprathreshold activities [Jan04b] within the early visual cortex.

The fast spread of low-amplitude activities (especially subthreshold activities) has the effect that future incoming stimuli, which are near the retinal position of already activated regions (as in the case of moving objects), evoke high-amplitude activities extremely fast. This effect has been called preactivation [Jan04b]. The fact that our simulations also show this latency compensation effect supports this hypothesis as the DNF has been optimised especially to simulate the interplay of both high- and low fluorescence dynamics.

In addition to the preactivation theory, further effects may contribute to this latency compensation: BALDO and KLEIN and WILKINSON et al. propose that moving stimuli are processed faster than flashed stimuli from retina to cortex [Bal95, Wil98]. However, those proposed 16 milliseconds cannot account alone for the up to 100 milliseconds latency difference [Jan04b] reported from the flash-lag effect [Mau92]. Therefore, it has to be assumed that a second major part to explain why we are, despite neural processing times, able to estimate the position of a moving object lies in the functional architecture of the primary visual cortex. Long-range lateral connections pave the way to fast and wide spreading sub-threshold activity making it easier for moving stimuli to evoke high-amplitude activities faster. This can be explained by a preactivation with subthreshold activity near already spiking regions which then raises membrane potentials faster toward threshold values. Therefore, this effect not only explains illusory motion perception, like in the LM-paradigm [Jan04a] but also accounts for motion latency compensation phenomena in the visual system.
Chapter 7. Results

7.5. Model predictions: The reverse line-motion illusion

The so called reverse LM (rLM) denotes a stimulus of a bar that is briefly flashed before presenting a square, that is aligned to the top of the bar. VON GRÜNAU and FAUBERT report that observers perceive the bar as shrinking toward the square position [vGr94]. The authors tested a setting, in which first the square is flashed, then the bar is presented and after that the square is presented again (square-bar-square). They report that observers first perceive the square as growing out to the bar and then, when the bar is removed, as shrinking back to the square position. SCHMIDT and KLEIN reported [Schm97] that in their setting (just first bar then square stimulus) 75 per cent of the test persons perceive motion toward the square position. In the square-bar-square experiment, 85 per cent of the observers perceive the square first growing out to the bar and then shrinking back to the square stimulus.

HIKOSAKA and MIYAUCHI argued for attention effects that cause the LM illusion [Hik93a]. However, in the rLM paradigm this explanation cannot hold as there is no cue position (a square catching observer’s attention). Nevertheless, the bar is not perceived as disappearing at once. SCHMIDT and KLEIN extended the theory of HIKOSAKA et al.: They propose that the presentation of the square after the presentation of the bar extends the duration of signal transmission, so that the bar is perceived as shrinking toward the square position [Schm97].

In this section it shall be investigated whether the rLM might be due to processes within the primary visual cortex. Provided that this were the case, the DNF should show the rLM-effect in its response: simulated high fluorescence (red and yellow areas) should gradually shrink upwards toward the position, the square has been presented. In the left column of Fig. 7.16 different variants of the rLM stimulus are presented in space-time diagrams. The simulated fluorescence signals which are dominated by inhibition are shown in Fig. 7.16a. A gradual shrinking of high-fluorescence areas can clearly be observed, which is stressed by a white arrow in the simulated response. In addition to the simulated dye signals, the according excitatory activations have been simulated separately. As in these activations, the LM-effect is not as clearly observed, several settings of the reverse LM have been tested: In Fig. 7.16b-e some variations on the duration of the presentation of the square and bar stimulus are depicted. In Fig. 7.16c and d the inter stimulus interval (ISI) has been left out. In all the tested cases, the gradual shrinking of high fluorescence values is not as clearly observable as in the case of simulated dye-signals (see Fig. 7.16a). But in one
7.5. Model predictions: The reverse line-motion illusion

Figure 7.16: Reverse LM (rLM). A bar is presented followed by a square that is aligned to the top of the bar. **a)** The simulated VSDI-signals evoked by the rLM. **b)** The according separately simulated excitatory activities. **c-e)** Different variations on the ISIs and the duration of the presentation of the square and bar when simulating the excitatory activities.
case (d, when the ISI is left out and the bar stimulus is presented longer), we can observe a tendency in the yellow fluorescence colours to gradually move out, which is stressed by a white arrow in the picture.

It can be hypothesised that the rLM-illusion [Schm97] might be due to processes within the primary visual cortex though the motion effect is not as clearly observable as in the LM case. As Schmidt and Klein have reported on 75 per cent of observers who perceived the illusion, it can be supposed that in the rLM case additional effects might be responsible for the illusion. In contrast to the LM illusion, it could not be shown that intracortical mechanisms can alone account for the effect in the excitatory signals. However, a result of 75 per cent of observers that perceive the illusion is likewise a not so clear result. There is a majority of observers, that perceive the illusion, but there is also a not neglectable part who does not perceive the illusion at all.

Concluding, it can be summarised that simulated intracortical processes give a tendency but no clear indication to a rLM effect in the excitatory activations. It might be that this tendency is enhanced in higher areas (like area MT in primates or area PMLS in cats), such that, in the setting of Schmidt and Klein, 75 per cent of observers report on perceiving the bar as gradually shrinking toward the square position.
Chapter 8.

A comparison of model performances

In this chapter, the performance of the DNF will be compared with the performance of the two-dimensional integrate and fire neuron model of RANGAN et al. Both models, the DNF of this study and the so called model Cortex (mC) [Ran05], simulate the LM-effect [Jan04a] reported by JANCKE et al. The mC further models the effect of spontaneously appearing activity correlated with stimulus orientations [Ken04].

The simulations of the spiking neuron approach to the square, bar, LM and moving square stimulus are depicted in Fig. 8.1. The VSDI responses are shown in Fig. 8.1 a-d2, whereas the according stimuli are presented in Fig. 8.1a-d1.

When comparing the simulations of the mC with the simulations of this study, it can be observed that the integrate and fire approach has the spatial and temporal integration-problem discussed in section 5.3: The square-stimulus evokes only low fluorescence changes, see Fig. 8.1 a3. Considering the colour-scale on the right, yellow colours in the simulation correspond to blueish colours in the VSDI-data, so that no high fluorescence signals are observed. In the case of the bar-stimulus, simulated spreading-out high fluorescence changes can be observed (see Fig. 8.1 b3). Compared to the VSDI-data (see Fig. 8.1 b2), this area is too large and arises about 20 milliseconds earlier than it is the case in the real VSDI-responses. JANCKE et al. stressed that the data show stationary high fluorescence areas in contrast to low fluorescence signals, which spread out very fast [Jan04a]. This has been stressed in this work by the fact that also the simulated dye signals show a faster propagation than the higher dye-signals, see Fig. 7.5.

Compared to the VSDI-signals, the simulated high fluorescence signals evoked by the LM show no spatial border when the bar stimulus evokes its responses (compare Fig. 8.1 d2 and d3). Further, at the beginning when the square stimulus has been presented, the high fluorescence changes arise 20 milliseconds later than in the VSDI-data.

Similar problems arise when the moving square stimulus is presented. First, the moving square hardly achieves high fluorescence signals (Fig. 8.1 d3), so
Chapter 8. A comparison of model performances

Figure 8.1.: Activity patterns of the mC of [Ran05]. Rangan et al. tested the mC a-d3 on the VSDI-data a-d2 of [Jan04a]. a-d1 depict the tested stimuli: a1) the square, b1) the bar, c1) the LM and d1) the moving square stimulus.

that 10-20 milliseconds later than in the VSDI-data (Fig. 8.1 d2) higher signals arise and then spread out with almost no spatial border.

The DNF simulates the different propagation speeds of higher and lower levels of fluorescence signals, see Fig. 7.5. Without the additional LGN-normalisation (section 5.3, eq. 5.14, page 53), the DNF has also to struggle with smaller responses evoked by smaller stimuli like the square stimulus, see section 7.1.3. These problems arise because of spatial and temporal integration effects that boost the responses of larger stimuli. In the mC, the stimulus couples into the equations of their LGN-model. In this LGN-model, the stimulus is convolved with a Gaussian kernel. Therefore, similar LGN responses as shown in the second column of Fig. 7.7 are likely to arise with the LGN model of Rangan et al. Using the additional normalisation method proposed in this study, see section 5.3, could help to enhance the responses evoked by the square stimulus and could reduce the responses of the larger stimuli.

However, even without the LGN normalisation, it was possible to evoke localised high fluorescence signals with the DNF in response to the bar, LM and drawn-out bar stimulus, see third column in Fig. 7.7. The only problem in these simulations has been the weak response evoked by the square stimulation. In the simulations of the integrate and fire model, a general problem of simulating
localised responses, as they are found in the dye signals, can be observed. It can be suggested that this cannot be compensated alone by the use of the additional normalisation in the LGN and seems to be caused by the integrate and fire architecture.

A further noticeable difference in the simulations is that the orientation-specific long-range connections lead in the mC to a lattice-like response in the simulated high fluorescence signals (see Fig. 8.1 b-d3), that is not present in the VSDI-data (see Fig. 8.1 a-d2).

Both the DNF and the mC can simulate the gradual spread of high-amplitude activity in the LM-stimulus case. In the case of the mC this effect is due to the use of long-range orientation-specific NMDA-type neurons [Ran05]. In contrast to the integrate and fire model, the DNF stresses the most important components by using only few parameters, that describe basically the interplay of excitatory and inhibitory neuron populations, which have long-range excitatory lateral connections.

Comparing the parameters that describe the physiological architecture of the primary visual cortex, we get the following results:

The kernel function in the excitatory layer (see eq. 5.11, page 52) has a scale of about $\sigma^{exc} = 1650$ micrometres, where RANGAN et al. assume about a similar scale of $\sigma_{ee} = 1500$ micrometres. The kernel function in the inhibitory layer has in our DNF a scale of even about $\sigma_{ie} = 2290$ micrometres. In the integrate and fire model, the GABAergic neurons are assumed to have a scale of $\sigma_{GABA} = 250$ micrometres in their Gaussian spreading of activities within the GABAergic system. As the $\sigma_{ie}$-value in the DNF models the activity spread from the excitatory neuron population to the inhibitory population, these two values $\sigma_{GABA}$ and $\sigma_{ie}$ cannot be compared easily. In the DNF the inhibitory neurons only project locally to the excitatory neurons. The spread within the inhibitory population is kept out in the modelling to reduce the complexity of the system. As the excitatory neurons have far reaching connections to the inhibitory neurons, this spread is implicitly encoded.

In the DNF, the chosen inhibition is very strong, as the kernel in the inhibitory layer ($a_{ie}$ in eq. 5.7) is almost six times higher than the kernel height in the excitatory layer ($a_{ee}$ in eq. 5.7), see Fig. 6.2, page 64.

As a linear combination of the excitatory and inhibitory responses is visualised, see eq. 5.13, the signals of the DNF show both inhibition and excitation. The $\lambda$-value is 0.20, so that the simulation is dominated by inhibition. RANGAN et al. assumed [Ran05] 80 per cent excitatory and 20 per cent inhibitory neurons in their integrate and fire model. At the first sight, these statements seem to be nearly the contradictory. However, the number of excitatory and inhibitory
neurons cannot be counted in the excitatory-inhibitory network of Wilson and Cowan. It is a mean-field architecture, where both populations are potentially assumed to be on all cortical positions. Nevertheless, it is of great interest whether in the VSDI-responses more excitatory or more inhibitory signals are observed (see section 6.4.2 for a more detailed discussion). The DNF supposes that, although there are just 20 per cent inhibitory neurons in the cortex, these 20 per cent neurons are more effective, so that they dominate the VSDI signals.

Furthermore, the DNF enables a separate study of excitatory and inhibitory activations which reveals some exciting hints toward perception. A separate study of excitation and inhibition in the integrate and fire model is also of great interest. The question arises: Does approach of Rangan et al. reveal the same results on the similarity of separated excitatory activation and the perceptional scenario?

To summarise, the mC of Rangan et al. has the advantage to simulate further effects of spontaneously appearing activities. But the performance of the DNF proposed in this work shows some other important advantages: The qualitative behaviour of high and low fluorescence changes (propagation speed, extent and amplitude) observed in the VSDI-signals is simulated with the DNF proposed here. Further, a separate study of excitation and inhibition, which could also be performed with the integrate and fire approach proposed by Rangan et al., has been proposed in this study.
Chapter 9.

Discussion

‘Processing of visual information in the mammalian primary visual cortex (V1) is thought to arise from an interplay between the pattern of external projections and intrinsic cortical dynamics.’ [Blu06].

Blumenfeld et al. well describe the processing within the primary visual cortex especially when deriving information with VSDs. VSDI enables a study of interactions over several millimetres on the cortex of as well sub- and suprathreshold activities, revealing important information on brain functioning in its dynamics. In this study, a dynamic neural field (DNF) with one excitatory and one inhibitory layer [Wil72] was used to account for the cortical population dynamics in the primary visual cortex. The ‘intrinsic cortical dynamics’ are modelled by the strength and extent of the lateral excitatory and inhibitory interactions within the DNF. The ‘pattern of external projections’ arise in this work from a visual stimulation with artificial, simple shaped stationary, moving and illusory moving stimuli.

It was shown that the balancing of the mutual interaction of the excitatory and inhibitory neuron population came out to be the prominent feature of the DNF. A linear combination of the simulated well-balanced excitation and inhibition could be shown to account for the real-time optical imaging data [Jan04a] derived with VSDI from anaesthetised cat’s area 18 in response to stationary, moving and apparently moving stimuli.

Further, the DNF allowed a separate simulation of the excitatory and inhibitory activations. The separate study of the two simulated populations led to the hypothesis that in the primary visual cortex the excitatory activation is more related to perception than the pattern of inhibition. Nevertheless, the dye signals are, from the simulations, predicted to be dominated by effective inhibition.

In this chapter, the DNF, its simulations, the excitatory projections from the primary visual cortex to higher brain areas, the physiological interpretation of the model parameters and the emergence of illusory motion in the primary visual cortex are discussed.
Chapter 9. Discussion

9.1. The dynamic neural field with preceding normalisation

A DNF with one excitatory and one inhibitory neuron layer according to Wilson and Cowan [Wil72, Wil73] was used to simulate the cortical dynamics observed in the VSDI of cat’s area 18. The DNF of Wilson and Cowan was modified to yield a compact set of biologically straightforward parameters. Some of these modifications were chosen according to Erlhagen’s DNF [Erl03]. The simulation of the dye-signal was defined as a linear combination of the modelled excitatory and inhibitory membrane potential states. Additionally, a normalisation model [Hee92] of the LGN according to Freeman et al. [Fre02] was introduced to compensate for too much spatial and temporal integration of activities in response to large stimuli.

The parameters of the system were optimised by an evolution strategy algorithm of Hansen and Ostermeier [Han97] and additional manual fine-tuning. This evolution strategy algorithm has already been applied successfully in the optimising of parameters of dynamic neural fields [Ige03, Ige02, Schn04]. The parameter that encodes the proportion of the excitation observed in the dye-signals could be calculated by a simple linear program.

A stability analysis on the DNF equations was performed, that showed that a necessary condition for stability is that the resting membrane potential of the excitatory population is safely smaller than the threshold value considering the used sigmoidal axonal response function of the excitatory neurons. The optimised parameter set satisfies this assumption. It was shown that when pushing the excitatory resting membrane potential toward the threshold value, oscillations arise when the system receives no external stimulation.

As the output of the system was defined as a linear combination of the excitatory and inhibitory membrane potentials, it was possible to vary the simulated proportion of excitation in the dye-signals.

The imaging technique did not allow a distinction between excitatory and inhibitory activities. Therefore, a separate study of both populations without affecting its interactions became possible and provided additional information on intra-cortical dynamics, that in case of VSDI until now no other modelling study provided.

9.1.1. The simulations

The DNF has been shown to account for the real-time optical imaging data evoked by the artificial simple shaped stimuli. High- as well as low fluorescence
signals have been simulated with their different propagation speeds with the DNF.

The LM-stimulus which causes illusory perception of motion in humans [Hik93a] has been shown to evoke simulated responses similar to the VSDI signals. VSDI-data as well as the simulations show in response to the LM a 'motion-streak'-activity pattern, indistinguishable from real motion in this area. The DNF did not involve any attention effects of higher brain areas to account for this LM-effect. The real moving stimulus, a square drawn-out to bar length, has also been shown to evoke 'motion-streak'-responses in dye-signals [Jan04a] and simulations. Surprisingly, a moving square (with a moving speed of 32 degrees per second) evoked the same 'motion-streak'-responses in simulations and dye-signals. The question arose, whether the same similarities can be found in the activations of the excitatory neurons.

The VSDI technique did not allow a distinction between excitation and inhibition. The integrate and fire model proposed by Rangan et al. (which has been used to simulate VSDI-data derived by Jancke et al.) makes it not possible to simulate both populations separately. Hence, another model which offers a separate study of excitation and inhibition became necessary.

The output of the DNF proposed in this work has been chosen as a linear combination of excitation and inhibition. This made a separate study of excitatory and inhibitory neural activations possible without affecting the interaction between both populations. It was shown that the simulated inhibitory responses evoked by the three special stimuli (moving square, LM and drawn-out bar) resembled each other. However, the excitatory simulations showed different responses to the moving square stimulus compared to the LM and drawn-out bar evoked activations. In the excitatory responses, the moving square evokes activity of a local patch in motion in contrast to a 'motion-streak'-like activity pattern evoked by the other two stimuli. Hence, the excitatory responses matched the perceptual scenario, as observers perceive a moving square stimulus completely different from a drawn-out bar and a LM stimulus.

The DNF has further been used to account for moving-square stimuli evoked responses. The VSDI of three different experiments have been presented and compared with the simulated activities. Both simulations and dye-signals showed compensation of latency effects: When observing activity patterns of moving stimuli in the primary visual cortex, it shall be expected, because of processing times that the responses lag behind stimulus positions. However, in this study it was shown that this is neither the case in simulations nor in VSDI until a square stimulus velocity of 32 degrees per second. When observing the excitatory responses, nearly no lagging behind stimulus presentations is
observed for all velocities tested (4, 8, 16, 32 and 64 degrees per second). Spatial and temporal activity integration of excitation and inhibition mediated by simulated lateral interactions of both populations was shown to be responsible for this compensation phenomenon. It can therefore be assumed that—according to Jancke et al. [Jan04b]—the primary visual cortex is involved in latency compensation in the processing of moving stimuli.

When presenting the rLM (reverse LM) stimulus, a bar followed by a square that is aligned to the upper edge of the bar, a ‘motion-streak’ like activity pattern can be observed in the simulations. When simulating the excitatory responses separately, this ‘motion-streak’ response was weakened. The according VSDI-data have not been measured until now, so that the simulations can be used as a prediction on the dye-signal behaviour.

9.2. The excitatory output transmits signatures of perceptual correlates

It was worked out that previous studies suggest that the projections from cat’s primary visual cortex to area PMLS are performed by excitatory cells (see section 2.6). Cat’s area PMLS is assumed to be the analogon to monkey’s area MT [Pay93], an area which is involved in the processing of moving stimuli in primates. Because in this study motion perception was in the centre of interest, the excitatory activity in the primary visual cortex became therefore very important when trying to simulate and explain motion perception phenomena like the LM-illusion.

It can hence be assumed that the perceptual scenario (LM equals drawn-out bar, LM differs from moving square) is reflected in the simulated excitatory neural activities: We can clearly distinguish between a drawn-out bar and a moving square stimulus, as the drawn-out bar is perceived as an expanding line and the moving square stimulus is perceived as a local patch in motion. In contrast, the LM deviates from physical input, as the bar does not appear at once but seems to be drawn out, leading to an illusory sensation of motion [Hik93a] similar when presenting the drawn-out bar stimulus.

Indeed, in the simulations it can be seen that when simulating the excitatory responses separately, the response to the LM resembles that of the drawn-out bar, but these responses deviate from the response to the moving square. The response to the moving square stimulus can be clearly accepted as a local patch in motion, whereas the responses to the LM and drawn-out bar reflect the responses to the perception of an expanding line. The simulated inhibitory
neuron responses resemble each other in all three stimuli cases. As the VSDI-simulations are assumed to be dominated by inhibition, the same similarity in all three responses appears also in the inhibition dominated simulations of the dye-signals. Therefore, in the simulations, both inhibitory activations and simulated dye-signals mismatched the perceptual scenario in the case of the moving square stimulus.

To summarise, according to physiology, higher brain areas receive excitatory activities from the primary visual cortex. This modelling study proposes that VSDI-signals show a lot of inhibition. However, the excitatory neuron responses allow conclusions about perception which is in accordance with physiological findings. Therefore, in this study it is concluded that inhibition has a very strong and important part in the balancing of the excitation, but the excitation carries the information correlated to perception.

9.3. Physiological interpretation of the parameters

The DNF used in this work implements a basic model of the interplay between excitation and inhibition. The parameters have been chosen to be biologically plausible, few in number and reasonable in the level of detail considering the imaging technique. Two parameters of the DNF that encode the extent of the lateral spread of excitatory and inhibitory signals are discussed in detail and compared to physiological findings. After that, the parameter $\lambda$ that encoded the proportion of excitatory compared to inhibitory signals is interpreted.

9.3.1. Lateral connectivity of excitatory and inhibitory neurons in the primary visual cortex

In the parameter setting of the DNF, the lateral connectivity was implicitly encoded in the Gaussian interaction kernels $w_{ie}$ and $w_{ee}$, see eq. 5.10, page 51. As shown in section 6.4, the values $\sigma_{ee} = 1.66$ millimetres and $\sigma_{ie} = 2.29$ millimetres are used in the simulations. These $\sigma$-values denote the distance from the origin of the Gaussian interaction function to the inflection point (see Fig. 6.2) and encode herewith the lateral extent of the axonal projections in the primary visual cortex. To compare these $\sigma$-values with physiological findings, we should consider that the $\sigma$-values just denote this distance from the origin to the inflection point of the Gaussian functions. Considering the normal distribution, 95 per cent of the area under the curve is within the interval $\mu \pm 2\sigma$, with $\mu$ denoting the expectation value and $\sigma$ the standard derivation. Therefore, we
have to multiply the values by at least a factor of two to account for the approximate whole activity spread. In the case of the excitatory neurons we get a scale of about 3.3 millimetres which is in accordance with Kisvarday et al. who report on excitatory neurons with up to 3.5 millimetres axonal projections revealed with biocytin tracing [Kis97].

Gilbert et al. investigate the extension of the pyramidal cells which they show to link neurons over distances of 6 – 8 millimetres in cat primary visual cortex [Gil89]. The assumed approximate radius of 3.3 millimetres lateral spread predicted by the parameter setting of the DNF is in accordance with the results of Gilbert et al.: As the 3.3 millimetres (2 × 1.66 millimetres) in the model just denote the radius of spread we have to multiply this value again by a factor of two (yielding 6.6 millimetres), as the interaction kernel is assumed to be symmetric. Therefore, the resulting spreading property between excitatory neuron populations within the DNF is in accordance with physiology.

Inhibitory lateral spread by large basket cells has been reported in the literature as reaching up to 2 millimetres (see section 2.4), where Kisvarday report on just up to 1.5 millimetres lateral spread [Kis97]. In the simulations, a scale of $\sigma_{ie} = 2.29$ millimetres is assumed that denotes the spread of excitatory projections to the inhibitory neuron population. This factor is multiplied by the factor two even 4.58 millimetres (interaction radius) and therefore much larger than the values given in literature for the spread formed by both the excitatory and the inhibitory neurons. However, there is an amount of inhibitory interneurons in the primary visual cortex as introduced in section 2.1. Those interneurons act mostly locally (see section 2.2), which has been implemented in the DNF by the local incoupling of the inhibitory term into the excitatory field equation, see eqs. 5.11 and 5.12, page 52. These interneurons are activated—in physiology as well as in the DNF equations—by widespreading lateral excitatory connections ($w_{ie}$).

Inhibitory activity is therefore remotely controlled and can be activated over long distances by projections of excitatory neurons. Interneurons mostly form synapses to axons of pyramidal cells, but also to dendrites (see section 2.2). When a pyramidal cell has been activated over long-range connections, also inhibitory neurons around the cell are activated. Therefore a large region of activated inhibitory signals around the activated excitatory signals is observed. This large inhibitory region that is activated by excitatory neurons is encoded by the interaction kernel $w_{ie}$ that describes the remote control of inhibition by wide spreading excitatory projections. After being activated, the inhibitory interneurons suppress the activity of the excitatory neurons in its direct neighbourhood.
9.3. Physiological interpretation of the parameters

9.3.2. Inhibition observed in the VSDI-signals

Measuring with VSDs offers real-time optical imaging of membrane potentials with one dye-signal representing the average potential of a large number of both excitatory and inhibitory cells. An interesting finding of this modelling study when simulating the considered dye-signals is that the choice of a small value of the parameter $\lambda = 0.20$ in eq. 5.13, page 52 is optimal to fit the data. This choice of the parameter hypothesises a large portion of inhibitory signals (about 80 percent) in the measured dye signals. In the cat primary visual cortex the proportion of inhibitory (GABA-immunoreactive) neurons is about 20 per cent [Gab86]. At first sight these statements seem to be the opposite. But it has to be considered that in the DNF the number of excitatory and inhibitory neurons cannot be assessed. It is a mean-field architecture, where both populations are assumed to be in all cortical positions. The setting $\lambda = 0.20$ exclusively hypothesises that the inhibition is more effective in the dye-signal responses than the excitation and therefore better seen. In the VSDI-setting, the number of observed excitatory and inhibitory neurons can also be hardly assessed: the measured fluorescence signals in one image pixel represents the mean membrane potentials of a large number of neurons of different types, comprising excitatory and inhibitory neurons.

To investigate the word more effective in this context, let us consider the conductance behaviour of excitatory and inhibitory neural activity: Considering the inhibitory and excitatory conductance behaviour, we find that inhibitory conductance takes more than a nine-fold time to decay before returning back to baseline level [And00]. Further, the inhibitory conductance is much stronger than the excitatory conductance, which can be observed in conductance measurements in cat area V1 [And00] and has been predicted by the V1 neuron model of Delmore especially for responses to stimuli with non-preferred orientation [Del03].

Because the inhibitory conductance is stronger and has a longer timespan than the excitatory conductance, the entire conductance is more biased toward the inhibitory than to the excitatory one, which could account for a great portion of inhibitory signals observed in the VSDI-pictures and predicted by the DNF. Anderson et al. show that the inhibitory conductance is in most measurements nearly exactly the negative response to the membrane potential [And00].

From these observations it can be concluded that synaptic inhibition plays a major role when observing membrane potentials as in the case of VSDI.
9.4. The emergence of illusory motion in the primary visual cortex

The LM illusion has been assumed to emerge on the way V1-V2-V3-MT [Hik93a]. With a dichoptic experiment on humans, Hikosaka et al. could exclude subcortical areas as being responsible for this illusory effect, see section 4.1.1. The question arises, whether higher brain areas like area MT (or PMLS in cat) are responsible for this effect. One main result of this work is that the LM-effect observed in the primary visual cortex of cats has been well simulated with a DNF-model of the primary visual cortex (with preceding normalisation in the LGN), without involving any higher brain areas like area MT or area PMLS in the simulations. This result supports that cat’s primary visual cortex might alone have the ability to produce the LM effect, without the involvement of higher brain areas.

Larsen et al. recently argued that illusory motion perception is a kind of observed filling-in activity that has been computed by higher brain areas and represents not more than a perceptual filling-in [Lar06]. With the simulation of the DNF, it was shown in this work that the LM-effect can be simulated by bottom-up and lateral interaction processes already and alone. The model results of Rangan et al. shows that an integrate and fire-model was also able to simulate the LM-effect without the involvement of higher are modelling [Ran05].

Larsen et al. presented a so called three-stage theory (see section 4.1.1): in the first step of the theory, the visual system identifies two images as being successive views of the same objects. In their setting (two squares flashed in successive order), the identification of the two objects (square and square) is intuitively clear. However, in the LM-paradigm, this correspondence problem is not so intuitively applicable, as in nature objects rarely expand and increase their sizes by the factor of four (square-bar). Fluids can for example change their forms but they never expand in size by a factor of four within milliseconds in our every-day life. Therefore, the emergence of illusory motion perception cannot be explained alone by the three stage theory of Larsen et al. Especially when explaining the LM-illusion, this theory is not applicable.

It cannot be excluded that there are additional top-down-effects in the measured VSDI-responses, like effects from back projections of cat’s area PMLS to the primary visual cortex for example. But from the simulations, we have strong evidence that the interplay mediated by lateral connections of the excitatory and inhibitory neurons lead—in the case of the LM paradigm—to responses of the primary visual cortex that are indistinguishable from real motion in this area.
Larsen et al. further report on a strong innervation of the MT-complex (MT+) when presenting illusory or real moving stimuli to the test persons. From the modelling result of this study, it can be supposed that this strong innervation of MT+ involves also a great portion of cells that just recognise motion and do not initiate motion perception: It was shown that the excitatory activity shows motion-like responses and it is true that the excitatory population is read out from area PMLS in cat and area MT in primates, see section 7.2.

Eagleman and Sejnowski presented a stimulus setting, see section 4.1.1, in which the LM-perception could be reversed by a stimulus presented after the line (bar) has disappeared [Eag03]. The authors argue that this reversing of the direction of movement is due to an integration of information after the presentation of the LM. They conclude that motion awareness is an a posteriori reconstruction. However, in their setting, several influences play together: First, they use lines that change colour and illuminance to cue the position of attention with coevally presenting at the other side, also lines (that do not change colour and illuminance) and additionally a dot. In our setting, we just use the presence of a square that might catch the observers attention. Further, the dot that moves from the one to the other side is recognized as the same object, so that in this setting, higher brain areas are involved to recognize that the same object appears. In the setting used in this work, those influences that might involve higher brain areas are reduced as we just use the flashing of one square stimulus before presenting the line. Therefore, a general conclusion from the setting of Eagleman and Sejnowski that motion induction always involves higher brain areas might not commonly be true, also in the case of the LM-illusion.

The question arises: if illusory motion emerges in the primary visual cortex, what might this effect be good for? To answer this question, it is useful to consider the compensation of latency effects introduced in section 4.2 (previous work) and in section 7.4 (observed in the VSDI-data of this study). An object’s visual information is processed within a certain time-span over the visual pathway until the object is perceived. If this object is in motion, it might have a completely different position in visual space when the information receives perception. If the primary visual cortex with its functional architecture encodes activity spread that boosts moving stimuli faster to spiking activity (see section 7.4.1 about preactivation of activity), a side effect of this fast processing of moving stimuli by preactivating neighbouring areas might be the following: a stimulus that is directly followed by another larger stimulus of equal or directly adjacent retinal position, can evoke preactivating activity spread, so that the second larger stimulus can ‘surf’ on this preactivated activity induced by the first stimulus. This surfing activity then causes illusory perception of motion
although non-moving stimuli have been presented. In this work, this effect is referred to as *surfing effect* in this context. This preactivation was shown in this study to emerge because of lateral activity spread within the primary visual cortex.

If the second stimulus is smaller in size than the first stimulus (bar-square in the rLM paradigm, see section 7.5) this surfing effect explanation cannot hold: There is no special position in the rLM setting (bar-square), in which activity is preactivated to a greater extend. SCHMIDT and KLEIN propose the following bottom-up theory [Schm97] which is consistent with our results: The presentation of the square after the bar (rLM stimulus) leads to an extension of signal transmission in the visual cortex. Activity, which without the presentation of the square stimulus decreases, is therefore prolonged by the later presentation of the bar stimulus. It can therefore be assumed that, when the square is presented after the bar, an extension of activity near the square position is facilitated, whereas activity far away from the square position returns faster back to baseline level. This effect of extension of activity is referred to as *refreshing-effect*, as the second stimulus ‘refreshes’ the activity of the first stimulus, so that it is expanded.

Hence, the question arises: *what is this refreshing-effect good for?* If observers perceive a stationary object, this object is under normal circumstances not expected to disappear. Therefore it is advantageous that the primary visual cortex holds activity for a certain period of time, so that the object is perceived as being continuously present. If the object disappears nevertheless (as it becomes possible with artificial stimulation on a computer screen, like in the rLM setting), activity is sustained for a while and possibly appearing smaller stimuli can refresh this activity, so that the area around the second stimulus is sustained for a longer period.

It can be concluded that the illusory motion perception phenomena in both the LM and the rLM-paradigm can be understood as a side-effect of efficient computing in the brain. In the case of the refreshing-effect in the rLM-paradigm it might be a side effect of efficient computing of stationary stimuli. In the case of the surfing-effect in the LM-paradigm, it might be a side-effect of efficient computing of moving stimuli.
Chapter 10.

Outlook

The biologically straightforward DNF model based on basic interactions between excitation and inhibition proposed in this study, accounts for the primary visual cortex dynamics revealed with VSDI. The model is obviously valid for the one-dimensional cortical setting. A two dimensional modelling, when considering the used stimuli setting, does not deliver additional information, as all stimuli used in the VSDI underlying this study deliver redundant information in the second cortical dimension. The extension of the model to two dimensions is a technical task that is now under construction within the scope of a diploma thesis at the ‘Institut für Neuroinformatik’ at the Ruhr-University Bochum. Starting from a two dimensional modelling, the consideration of more complex such as natural stimuli becomes possible. When simulating two dimensions, orientation tuning effects could also be considered.

By showing that the DNF accounted without any additional involvement of top-down modelling for the LM-effect in the primary visual cortex, we have evidence that the LM might be initiated in this area. Further evidence for this hypothesis could be given, when presenting the LM and drawn-out bar stimuli to the blind field of patients with lesions in the primary visual cortex or its thalamic afferents. These patients report that they are not able to see anything in this lesioned visual field, but in decision making tasks they nevertheless decide for the right alternative with higher than chance level. This effect has been termed blindsight [Wei74]. One theory to account for the abilities in blindsight patients asserts that information from the retina reaches the extrastriate cortex via pathways that bypass V1 [Rod89, Rod89, Scho02]. Especially the neurons in the extrastriate areas of the dorsal stream (concerned with motion processing) ‘retained much of their visual responsiveness’ [Rod89]. If it is the primary visual cortex that induces the illusory LM, those patients with lesions in this area or it’s thalamic afferents (the optic radiation) might not produce the LM-effect and therefore might not see the illusion. When presenting either the LM-stimulus or the drawn-out bar stimulus to the patient’s blind fields, it could be possible that those patient are in contrast to normal controls able to distinguish these two
stimuli. If this were the case, another strong evidence would be given for the bottom-up theory of the induction of motion within the primary visual cortex in the LM-scenario.
Appendix A.

Mathematical background

A.1. Fourier-transform

The Fourier transform is essential in medical imaging studies and other image processing procedures: For example, two-dimensional pictures are represented in matrix form with the values indicating the grey value and the position within the matrix showing the image location. But global phenomena concerning the whole picture, like noise effects can be defined better in the so called frequency domain [Ehr97]. The transform of a function in its frequency domain with the so called Fourier transform has further advantageous properties that have been used in section 6.5. As in this work the Fourier transform is applied within the $n$-dimensional Euklidian space, in this appendix the basic facts are summarised accordingly.

First of all we need the following

**Definition 3.** A function $f : \mathbb{R} \rightarrow \mathbb{C}$ is called absolutely integrable, if the integral

$$\int_{-\infty}^{\infty} |f(t)| dt$$

exists.

With this it becomes possible to introduce the definition of the Fourier transform:

**Definition 4 (Fourier transform).** Let $f$ be an absolutely integrable function with $n$ real variables. Then its Fourier transform, denoted by $\hat{f}$, is defined as

$$\hat{f}(x) = \frac{1}{\sqrt{2\pi}} \int_{\mathbb{R}^n} e^{-ix \cdot t} f(t) dt. \tag{A.1}$$

In the context of the Fourier transform it is worth to mention the so called convolution which is as well of great importance in imaging processing like in medical imaging studies [Ehr97]:

113
Appendix A. Mathematical background

Definition 5. For two absolutely integrable functions $f$ and $g$ of $n$ real variables, the convolution is defined through

$$f \ast g := \int_{\mathbb{R}^n} f(x - y)g(y)dy, \quad x \in \mathbb{R}^n.$$ 

Applying the Fourier transform on the convolution of two functions gives the following useful

Theorem 3. For two absolutely integrable functions $f$ and $g$ of $n$ real variables, the Fourier transform of the convolution satisfies

$$\hat{f} \ast \hat{g} = \hat{f} \cdot \hat{g}.$$ 

The proof can be found in [Che01].

A.2. The correlation coefficient and the linear regression

In this work the correlation between two images, a measured image and a simulated image has been calculated. All these images have two dimensions, one space and one time dimension. In this appendix, it is clarified what is meant by using the term correlation between two pictures in this work. For that reason, the so called sample correlation is introduced.

In probability theory, the so called correlation coefficient indicates the strength and direction of a linear relationship between two random variables. In this study we have two data series: a measurement and a simulation and assume that those series are derived from two random variables. Therefore the data matrices are transformed into two vectors of the form $p_1, \ldots, p_s \cdot l$ and $d_1, \ldots, d_s \cdot l$, with $s$ denoting the number of pixels in the cortical space dimension and $l$ denoting the number of timesteps considered. To simplify matters, let us set $n = s \cdot l$ to give the following

Definition 6 (sample correlation). The so called sample correlation between two series $d = d_1, \ldots, d_n$ (simulated data) and $p = p_1, \ldots, p_n$ (measured data) is defined as

$$\text{cor}(d, t) := \frac{\sum_{i=1}^{n}(d_i - \bar{d})(p_i - \bar{p})}{\sqrt{\sum_{i=1}^{n}(d_i - \bar{d})^2} \cdot \sqrt{\sum_{i=1}^{n}(p_i - \bar{p})^2}},$$

with $\bar{d}$ and $\bar{p}$ denoting the sample expectation values of $d$ and $p$ respectively,
A.2. The correlation coefficient and the linear regression

that are defined as the mean values of the vectors: \( \bar{d} = \frac{1}{n}(d_1 + \ldots + d_n) \), \( \bar{p} = \frac{1}{n}(p_1 + \ldots + p_n) \).

The sample correlation is a measurement for the strength and direction of a linear relationship. The correlation coefficient covers values between \(-1\) and \(1\). The correlation \(0\) indicates no linear relationship, \(1\) a complete positive and \(-1\) a complete negative relationship.

The correlation coefficient is invariant to lineal transforms of \(d\) and \(p\). To show this, the so called linear regression is introduced in this appendix: For the simulated responses \(d_1, \ldots, d_n\) exist a linear transform of the form

\[
y_d = m_p d + b_p,
\]

that minimises the mean squared error:

\[
\text{error}_{d,p} = \sum_{i=1}^{n} (d_i - p_i)^2
\]

It can easily be shown by setting the derivatives of the error function with respect to \(m\) and \(b\) to zero that this error is minimal if the slope \(m_p\) and the shift \(b_p\) satisfy

\[
m_p = \frac{\sum_{i=1}^{n} (d_i - \bar{d})(p_i - \bar{p})}{\sum_{i=1}^{n}(d_i - \bar{d})^2} \tag{A.2}
\]

and

\[
b_p = \bar{p} - m_p \bar{d}. \tag{A.3}
\]

In eqs. A.2 and A.3, the values \(m_p\) and \(b_p\) are calculated with respect to the measured data \(p\). Let us reformulate the equations to derive the regression line \(y_p = m_d p + b_d\), with

\[
m_d = \frac{\sum_{i=1}^{n} (d_i - \bar{d})(p_i - \bar{p})}{\sum_{i=1}^{n}(p_i - \bar{p})^2} \tag{A.4}
\]

and

\[
b_d = \bar{d} - m_d \bar{p}. \tag{A.5}
\]

The correlation coefficient can now be interpreted as

\[
\text{cor}(d, p) = \sqrt{m_d \cdot m_p}.
\]

Hence, the correlation coefficient is the geometrical median between the slope of the linear regression line with respect to the measured data and the slope of
Appendix A. Mathematical background

the regression line with respect to the simulations. As $m_d$ and $m_p$ result from a linear regression, it can easily be concluded that the correlation is invariant to linear transforms of $d$ or $p$.

As the VSDI-signals are linearly correlated with membrane potentials ([Ste98], [Pet03]), the correlation coefficient is a good measure to calculate the similarity between simulations and the data. Note that the simulations shown throughout this work are in each case the optimal linear fit to the data: $o_{pictures} = m_p o + b_p$, with $o$ taken from eq. 5.13.
Appendix B.

The CMA evolution strategy

In the following we present the CMA-ES with weighted recombination [Han97, Han01, Han04]. Each individual represents an $n$-dimensional, real-valued, object-variable vector. These variables are altered by two variation operators, intermediate recombination and additive Gaussian mutation. The former corresponds to computing the weighted centre of mass of the $\mu$ individuals in the parent population. Mutation is realised by adding a normally distributed random vector with zero mean. The complete covariance matrix of the Gaussian mutation distribution is adapted during evolution to improve the search strategy.

The CMA implements important concepts for strategy parameter adaptation. These allow for highly accurate adjustment of the search distribution, and therefore fast and accurate optimisation, while requiring only small population sizes. The first concept is derandomisation. That is, the mutation distribution is altered in a deterministic way such that the probability of reproducing steps in the search space that have led to the actual population is increased.

Thereby the algorithm detects correlations between object variables and becomes invariant under orthogonal transformations of the search space (apart from the initialisation). The second important concept is cumulation, which means using the information from previous generations efficiently by taking into account the search path of the population over a number of past generations.

The object parameters $x_k^{(g+1)}$ of offspring $k = 1, \ldots, \lambda$ created in generation $g + 1$ are given by

$$x_k^{(g+1)} = \langle x \rangle_w^{(g)} + \sigma^{(g)} B^{(g)} D^{(g)} z_k^{(g)},$$

where the $z_k^{(g)} \sim \mathcal{N}(0, I)$ are independent realizations of an $n$-dimensional normally distributed random vector with zero mean and covariance matrix equal
Appendix B. The CMA evolution strategy

... to the identity matrix $I$ and

$$\langle x \rangle_{w}^{(g)} = \sum_{i=1}^{\mu} w_{i} x_{i: \lambda}^{(g)}$$

is the weighted mean of the selected individuals with $\sum_{i=1}^{\mu} w_{i} = 1$ and $w_{i} > 0$ for $i = 1, \ldots, \mu$. The index $i: \lambda$ denotes the $i$-th best individual. We use superlinear weighted recombination and set $w_{i} := \ln(\mu + 1) - \ln i$. The covariance matrix $C^{(g)}$ of the random vectors

$$B^{(g)} D^{(g)} z^{(g)}_{k} \sim \mathcal{N}(0, C^{(g)})$$

is a symmetric positive $n \times n$ matrix with

$$C^{(g)} = B^{(g)} D^{(g)} \left( B^{(g)} D^{(g)} \right)^{T}.$$ 

The columns of the orthogonal $n \times n$ matrix $B^{(g)}$ are the normalised eigenvectors of $C^{(g)}$ and $D^{(g)}$ is a $n \times n$ diagonal matrix with the square roots of the corresponding eigenvalues.

The strategy parameters, both the matrix $C^{(g)}$ and the so-called global step size $\sigma^{(g)}$, are updated online using the CMA method. The key idea of the CMA is to alter the mutation distribution in a deterministic way such that the probability of reproducing steps in the search space that have led to the current population is increased. This enables the algorithm to detect correlations between object variables and to become invariant under orthogonal transformations of the search space (apart from the initialisation). In order to use the information from previous generations efficiently, the search path of the population over a number of past generations is taken into account.

In the CMA-ES, rank-based $(\mu, \lambda)$-selection is used. That is, the $\mu$ best of the $\lambda$ offspring form the next parent population. After selection, the strategy parameters are updated:

$$p^{(g+1)} = (1 - c_{c}) \cdot p_{c}^{(g)} + \sqrt{c_{c}(2 - c_{c})} \frac{\mu_{\text{eff}}}{\sigma^{(g)}} \left( \langle x \rangle^{(g+1)} - \langle x \rangle^{(g)} \right),$$

$$C^{(g+1)} = (1 - c_{\text{cov}}) \cdot C^{(g)} + c_{\text{cov}} \cdot p^{(g+1)} \left( p^{(g+1)} \right)^{T}.$$

Herein, $p^{(g+1)} \in \mathbb{R}^{n}$ is the evolution path—a weighted sum of the centres of
the population over the generations starting from \( p^{(0)} = 0 \) (the factor \( \sqrt{\mu_{\text{eff}}} \) compensates for the loss of variance due to computing the centre of mass). The parameter \( c_c \in [0, 1] \) controls the time horizon of the adaptation of \( p \). The constant \( \sqrt{c_c(2 - c_c)} \) normalises the variance of \( p \) (viewed as a random variable) as \( 1^2 = (1 - c_c)^2 + \sqrt{c_c(2 - c_c)}^2 \). The index \( i : \lambda \) is the index of the offspring having the \( i \)-th best fitness value of all offspring in the current generation. The parameter \( c_{\text{cov}} \in [0, 1] \) controls the update of \( C^{(g)} \). The vector \( p \) does not only represent the last (adaptive) step of the parent population, but a time average over all previous adaptive steps. The influence of previous steps decays exponentially, where the decay rate is controlled by \( c_{\text{cov}} \). The update rule for the covariance matrix shifts \( C^{(g)} \) toward the \( n \times n \) matrix \( p^{(g+1)} (p^{(g+1)})^T \), which has rank 1, making mutation steps in the direction of \( p^{(g+1)} \) more likely.

The adaptation of the global step-size parameter \( \sigma \) is done separately on a shorter timescale (a single parameter can be estimated based on less samples than the complete covariance matrix). We keep track of a second evolution path \( p_{\sigma} \) without the scaling by \( D \):

\[
p^{(g+1)}_{\sigma} = (1 - c_{\sigma}) \cdot p^{(g)}_{\sigma} + \sqrt{c_{\sigma}(2 - c_{\sigma})} \cdot B^{(g)} (D^{(g)})^{-1} (B^{(g)})^T \sqrt{\mu_{\text{eff}}} \sigma^{(g)} \cdot \left( \langle x \rangle^{(g+1)} - \langle x \rangle^{(g)} \right),
\]

\[
\sigma^{(g+1)} = \sigma^{(g)} \cdot \exp \left( \frac{c_{\sigma}}{d_{\sigma}} \left( \frac{\|p^{(g+1)}_{\sigma}\|}{\hat{\chi}_n} - \hat{\chi}_n \right) \right),
\]

where \( \hat{\chi}_n \) is the expected length of a \( n \)-dimensional, normally distributed random vector with covariance matrix \( I \). It is approximated by \( \sqrt{n}(1 - \frac{1}{4n} + \frac{1}{21n^2}) \). The damping parameter \( d_{\sigma} \) decouples the adaptation rate from the strength of the variation. The parameter \( c_{\sigma} \in [0, 1] \) controls the update of \( p_{\sigma} \).

If there were no selection (i.e., if the new parents were selected from the offspring uniformly at random), the evolution path \( p_{\sigma} \) would be a weighted sum of independently normally distributed random variables starting from \( p_{\sigma}^{(0)} = 0 \). Because of the normalisation, its expected length would tend to \( \hat{\chi}_n \) for growing \( g \). Hence, the update rule basically increases the global step size if the evolution path \( p_{\sigma} \) is larger than expected under uniform random selection and decreases the step size in the opposite case. If the path \( p_{\sigma} \) is shorter than \( \hat{\chi}_n \) then the steps that led to selected individuals cancelled out each other more strongly than expected (i.e., they tended to be anticorrelated) or the selected
Appendix B. The CMA evolution strategy

steps were smaller than expected. Thus, the step size $\sigma$ should be decreased. Many successive steps in the same direction, which do not cancel out in $p_\sigma$ and could have been realised by a single long step, lead to an evolution path $p_\sigma$ that is larger than expected and the step size should be increased.

The parameters are set to the default values given in [Han04] (with $\mu_{\text{cov}} = 1$, i.e., do not use the extension proposed in [Han03], because we use only small populations): $\lambda = 4 + \lceil 3 \ln n \rceil$, $\mu = \lfloor \lambda/2 \rfloor$, $c_\sigma = \frac{10}{n+20}$, $d_\sigma = \max \left( 1, \frac{3\mu_{\text{eff}} n}{n+10} \right) + c_\sigma$, $c_c = \frac{4}{4+n}$, and $c_{\text{cov}} = \frac{2}{(n+\sqrt{2})^2}$. 
## List of abbreviations

<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Description</th>
</tr>
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<tbody>
<tr>
<td>CMA-ES</td>
<td>Covariance Matrix Adaptation Evolution Strategy</td>
</tr>
<tr>
<td>DNF</td>
<td>Dynamic Neural Field</td>
</tr>
<tr>
<td>ISI</td>
<td>Inter Stimulus Interval</td>
</tr>
<tr>
<td>LGN</td>
<td>Lateral Geniculate Nucleus</td>
</tr>
<tr>
<td>nLGN</td>
<td>normalization LGN method</td>
</tr>
<tr>
<td>LM</td>
<td>Line-Motion</td>
</tr>
<tr>
<td>MT</td>
<td>Medial Temporal</td>
</tr>
<tr>
<td>PMLS</td>
<td>Posteromedial lateral suprasylvian</td>
</tr>
<tr>
<td>rLM</td>
<td>reverse Line-Motion</td>
</tr>
<tr>
<td>VSDI</td>
<td>Voltage Sensitive Dye Imaging</td>
</tr>
</tbody>
</table>
List of symbols

\( a_{\alpha\beta} \) height of the interaction kernel \( w_{\alpha\beta} \)
\( c_{u,v} \) \(c\)-value (slope of the axonal response function) \( f_{u,v} \)
\( cor \) sample correlation
\( f_{u,v} \) axonal response function of the excitatory \((u)\) and inhibitory \((v)\) neurons
\( g_s \) stimulus boosting parameter
\( g_v \) inhibition boosting parameter
\( h_{u,v} \) resting membrane potential of the excitatory \((u)\) and inhibitory \((v)\) neurons
\( \lambda \) portion of excitatory signals, \( \lambda \in [0,1] \)
\( o(x,t) \) simulated dye-signal at position \( x \) and time \( t \)
\( q_{\alpha\beta} \) height of the axonal response function \( f_{u,v} \) (synaptic saturation)
\( S(x,t) \) stimulus at cortical position \( x \) and time \( t \)
\( S'(x,t) \) smoothed stimulus
\( S''(x,t) \) smoothed and normalized stimulus
\( \sigma_{\alpha\beta} \) \(\sigma\)-value in the interaction kernel \( w_{\alpha\beta} \)
\( T \) transpose of a vector or matrix
\( \tau_{u,v} \) empirical membrane time constants used in the neural field equations
in the excitatory \((u)\) and inhibitory \((v)\) neuron layer
\( u(x,t) \) membrane potential of the excitatory neurons at position \( x \) and time \( t \)
\( u_0 \) threshold value of the excitatory neurons
\( \bar{u}_0 \) homogeneous solution of \( u(x,t) \)
\( v(x,t) \) membrane potential of the inhibitory neurons at position \( x \) and time \( t \)
\( v_0 \) threshold value of the inhibitory neurons
\( \bar{v}_0 \) homogeneous solution of \( v(x,t) \)
\( w_{\alpha\beta} \) weighting function that denotes the strength of lateral couplings
from the \( \beta \) to the \( \alpha \)-layer
Bibliography


Bibliography


Bibliography


Bibliography


Bibliography


Bibliography


Index

absolutely integrable, 113
active transient, 48
apparent motion, 1
area centralis, 8
artificial stimuli, 1
attention, 33
  active, 33
  passive, 33
axon response, 51
balancing excitation and inhibition, 63
bar stimulus, 27
biologically plausible model, 48
blindsight, 111
blood oxygen level, 24
bottom-up theory, 1
cell
  large basket cells, 18
cells
  amacrine, 9
  bipolar, 9
  chandelier, 18
  complex, 12
  ganglion, 9
  horizontal, 9
  hypercomplex, 12
  magnocellular, 10
  on-centre, 12
  parvocellular, 10
  pyramidal, 16, 17
  simple, 12
  stellate, 16, 17
CMA evolution strategy (CMA-ES), 57, 117
columns
  functional, 18
  movement direction, 19
  ocular dominance, 18
  orientation, 18
compensation of latency, 36
cone cell, 8
convolution, 113
correlation, 114
correlation coefficient, 114
correspondence problem, 35
cortex
  medial superior temporal, 14
  mediotemporal, 14
  primary visual, 7, 11
cortical circuitry, 20
cumulation, 57, 117
derandomisation, 57
divisive inhibition, 52
dorsal stream, 14
drawn-out bar stimulus, 27
dynamic neural field (DNF), 41, 43, 45, 49, 51
Index

electro encephalography (EEG), 25
Euler’s method, 54
evolution path, 57
evolution strategy, 5, 117
evolution strategy algorithm, 2
excitatory-inhibitory network, 43
excitatory-inhibitory network by
Erlhagen, 47
excitatory-inhibitory network by
Wilson and Cowan, 43
external input, 44
extracellular recordings, 32

fitness function, 58
flash-lag effect, 37
flash-lag illusion, 37
Fourier-Transform, 113
fovea centralis, 8
functional magnetic resonance
imaging (fMRI), 24

Gaussian function, 51

homogeneous solution, 45
hypercomplex cells, 14

illusory motion, 26
integrate and fire model, 36, 97
inter stimulus interval (ISI), 28
interneurons, 17, 18
intrinsic optical imaging, 24

latency compensation, 4, 36, 88
lateral coupling, 45, 51, 105
lateral coupling function, 51
lateral geniculate nucleus (LGN), 7
layer
excitatory, 44
inhibitory, 44

layers of the primary visual cortex,
11
line-motion effect, 30, 76, 111
line-motion illusion, 1, 26
line-motion stimulus, 1, 27
linear regression, 58, 114
linear transform, 58, 114

magnetic encephalography (MEG),
25
magnocellular pathway, 10
mean-field approach, 48
mexican hat function, 45
motion extrapolation, 37
moving square stimuli, 27, 88

natural stimuli, 1
neural field, 41

based on Wilson and Cowan, 51
by Amari, 45
by Erlhagen, 47
by Wilson and Cowan, 44
non-pyramidal cells, 18
normalisation, 42, 53, 80

off-centre cells, 11
on-centre cell, 12
on-centre-cells, 11
optic chiasm, 7
optic radiation, 7
output of the DNF, 52

parvocellular pathway, 10
photoreceptors, 8
preactivation of activity, 93

projections
lateral, 17
local, 17
long range, 17
strong, 20
Index

weak, 20
receptive field, 9
resolution of VSDI, 23
retina, 8
reverse line-motion (rLM), 74, 94
rod cell, 8

sample correlation coefficient, 57, 114
sigmoidal function, 51
smoothing the stimulus, 53
space-time diagram, 30
spatial integration effect, 53
spatial resolution, 24
spatio-temporal resolution, 23
spiking activity, 32
square stimulus, 27
stability analysis, 65
superior colliculus, 11
suppression effect, 53
synaptic integration field, 20
synaptic saturation, 53

temporal resolution, 2, 24
time constants, 44, 87

ventral stream, 14
voltage sensitive dye imaging (VSDI), 23, 28
voltage sensitive dye imaging data, 28

weighting function, 44
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### Curriculum vitae

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</tr>
</tbody>
</table>

141
Declaration

To the PhD Commission of the International Graduate School of Neuroscience, Ruhr-University Bochum

Hereby I submit my PhD thesis entitled ‘Modelling Primary Visual Cortex Dynamics with a Dynamic Neural Field Based on Voltage Sensitive Dyes’. I guarantee that I have written this dissertation autonomously and without any illegitimate aids, the references and aids used are cited in their entity. This dissertation has not been submitted to another faculty, it has not been published yet. I guarantee that I will not publish the dissertation before completion of the promotion procedure. I have complied with the regulations laid down in the latest version of the ‘Guidelines for Good Scientific Practice’.

(Jennifer Meyer)
Bochum, 29th of December 2006