Dynamical Properties of Self–Regulating Neurons

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December 14, 2003

Abstract

The phenomenon of learning is still an open problem. The present work performs one step towards a model of learning that is inspired by early ideas of cybernetics and constructivism and makes use of the mathematical formalisms of dynamical system theory and artificial neural network theory. Homeostasis is proposed as principle of synaptic learning in recurrent neural networks, and an analysis of stabilisation properties of the suggested neuron model are investigated, by means of phenomenological analysis of system dynamics.

The model of synaptic regulation is placed in a larger framework of an artificial life approach to adaptation and cognition. It is conjectured that the proposed mechanism of synaptic regulation, if employed in a structurally appropriate nervous system, can realise adaptive behaviour.

Acknowledgements

I want to thank the INDY group at the Fraunhofer Institute for Autonomous Intelligent Systems — without your help, your expertise, your patience and your commitment, I could never have become acquainted with the matter as rapidly and as profoundly.

Contents

1 Preamble

The human adaptive capacity is stunning. There is much more to the phrase "you live and learn" than one might think on the first glance. Not only that you go to school or to university, or that you learn from mistakes you have committed. There is much more subtle forms adaptive processes can take. People adapt their manner and language to interlocutors. If it gets darker, their eyes adapt to the light. If they move to another house, soon, they do not notice anymore they have not always lived there. The longer one thinks about the amazing amount to which our environment changes every day, the more astonishing seems the fact, that we hardly notice these differences and how *we* change with them, in order to live on. Because of its tacitness, many prominent learning models neglect the persistent and automatic manner in which adaptation frequently takes place. They focus instead on special situations that are explicitly conceived as learning situations, e.g. such that involve teaching.

The approach adopted in this work is inspired by the vision to provide a general explanation of adaptive capacity in humans and animals. Homeostasis, i.e. maintenance of an internal variable at a desired value, is proposed as principle that underlies synaptic learning. Far from suggesting a whole learning theory, this thesis investigates a homeostatic mechanism to regulate neural activation in a simple neuron model. The investigation is performed by means of phenomenological analysis of the convergence behaviour that small networks of such neurons exhibit.

The introductory section 2 of this thesis will cover the theoretical and motivational background of the investigations performed. It will characterise the problem and will introduce the methodological and ideological commitments made. It will specify the exact purpose of this work within the outlined approach to learning.

The theoretical model itself will be presented in section 3. The homeostatic neuron will be defined and some of its formal properties will be shortly

discussed. Also, it will be specified how changes in the exterior can affect the neurons.

In section 4, the results of the experiments are presented. Homeostatic domains in recurrent networks of a single neuron and of a two neuron network are investigated by means of phenomenological analysis of asymptotic behaviour. Stable domains will be characterised.

The conclusion, section 5, will discuss the findings obtained. Firstly, the results as such will be evaluated, with respect to formal advantages and disadvantages of the model. Then, the findings will be interpreted in the context of learning and behaviour sketched in section 2.

2 Introduction

This section is devoted to the specification of the problem addressed in this thesis and to the considerations that lead to the adopted approach.

Section 2.1 will start with a definition of the terms "learning" and "adaptation". In section 2.2, the language of description, dynamical system theory, and its key concepts will be introduced. The motivational and methodological background will be provided in section 2.3, and it is explained how the present work is intended to contribute in defining a learning theory committed to the outlined ideas. In section 2.4, the regulatory mechanism of homeostasis is presented and integrated in the described framework. Finally, in section 2.5, the present approach is characterised and located within the scientific landscape.

2.1 Adaptation and Learning

Learning and adaptation are among the terms that are used most ambiguously, in cognitive science and in everyday life. Intuitively, adaptation might be understood as the noun corresponding to the verb "to adapt", which is used in phrases as different as e.g. "to adapt to light", "to adapt to living in a foreign culture" or "to adapt to the needs of another person".

Scientifically, adaptation is normally understood in an evolutionary sense, e.g. the MIT Encyclopaedia of the Cognitive Sciences defines (biological) adaptation as "a trait whose form can be explained by natural selection" ([20], lemma "Adaptation and Adaptationism" by Paul Griffith), although it is pointed out that non–evolutionary usages of the term "adaptation" exist. This definition obviously does not capture the various colloquial meanings listed above.

The same dictionary defines "learning" as a "change in an organism's capacities or behaviour brought about by experience" ([20], lemma "Learning" by Daniel Reisberg). It is admitted that this definition does include phenomena which are normally not considered as learning, such as increases in

muscular strength due to exercise. Another problem with this definition is that some behavioural changes, such as development of psychoses, can very well be brought about by experience, but would hardly be called "learning". Apparently, learning somehow includes a notion of a rise in "quality". Also it is questionable, what "experience" is supposed to be. If a person experiences the injection of steroids and is hence able to run faster, this would probably neither be considered an instance of learning. In order to be called learning, behavioural changes have to be attributable to the learner itself, in a way.

There is not much sense in further stressing the fuzziness of these and other related terms by providing more problematical definitions. Instead I will give my own working definitions, which are perhaps not universally applicable, but they roughly comply with the intuitive grasp of the notions and suffice the current needs of distinctiveness and accuracy.

Paying tribute to the colloquial usage of the term, *adaptation* will be understood as a general term to subsume changes that lead to rise in evolutionary fitness in agent behaviour. The reliance on fitness gives the required measure of quality. Since this work will not be concerned principally with issues of fitness, this term will be maintained as vague as it is. The evolutionary adaptation of populations across generations, as outlined above, will be explicitly referred to as *biological adaptation*. Increase of fitness within a single individual will be called *behavioural adaptation*, if it is triggered by external changes, but is realised by an intrinsic change of the individual. Such underlying changes themselves will be called *self–adaptation*. Self–adaptation and behavioural adaptation together will be called *learning*, since they are just two sides of the same coin. Obviously, this definition does not agree with definitions that consider learning as a "high level" process. For instance, changes in muscular strengths, as described above, are embraced by the given definition.

How these natural language definitions are interpreted formally will be

addressed later on in this work.

2.2 Dynamical System Theory

The model proposed in this thesis will be specified in the terms of dynamical system theory. The formal language of dynamical system theory qualifies for the description of adaptive processes, because it focuses on changes across time that are essential to adaptation. This section will give a brief and semi–formal introduction to the key ideas and terms of dynamical system theory that the reader will encounter in this thesis (compare [14],[19],[3]).

The theory of dynamical systems is centred around the notion of a *state*. A *state* x of a system is a set of system quantities that allows the complete description of the system's development across time. Formally, a state is a variable assignment to a set of variables, the so called *state variables* of a dynamical system. If a dynamical system is supposed to explain an actual dynamical process, the state variables have to correspond to measurable quantities. The space of possible assignments of values to state variables is called the *state space* (or *phase space*) M of the dynamical system, the number of state variables is called its *dimension* d.

Dynamical systems can either be given as a set of *ordinary differential equations* or as a set of *difference equations*. The former model the development of a dynamical system time–continuously, while the latter describe the development of a dynamical system in discrete time steps. In this thesis, only time–discrete modelling will be employed, and the following terms will be explained as they account for time–discrete dynamical systems.

A difference equation is an equation of the following kind:

$$x(t+1) = f(x(t))$$

where x is a system state, t denotes the time and $f(x)$ is a map $M \mapsto M$. The state x_0 a system is in at time $t_0 = 0$ is called the *initial condition*, its development across time is computed by iterated application of f to x_0.

Let $f^1(x(t)) = f(x(t)) = x(t+1)$ and $f^n(x(t)) = f^{n-1}(x(t+1))$, $\forall n \, \epsilon \, \mathbb{N}$. The sequence of states $x_0, x_1, \ldots x_n$ a system traverses when being iterated is called an *orbit* or a *trajectory*.

If for a state $x^* \, \epsilon \, M$ it holds that $f(x^*) = x^*$, x^* is called a fixed point of the system, or a *trivial orbit*. If a system returns to a state x_1^* after a finite number n of system iterations, i.e. $f^n(x_1^*) = x_1^*$, the traversed states $x_1^*, x_2^*, \ldots, x_n^*$ are called periodic points of the system. Such a cyclical orbit is called a *periodic orbit*, and the minimum number n for which this condition holds is called the *period* of the orbit. Trivial and periodic orbits are kinds of *invariant sets*. Invariant sets are sets of states $I \subset M$ in which orbits remain for all future times. Other kinds of invariant sets are circles or fractal sets on which orbits are lying dense. Orbits that remain either on circles or fractal sets are called quasi–periodic orbits or chaotic orbits respectively.

Invariant sets can be *unstable, stable* or *asymptotically stable*. An invariant set I is called stable, if there exists a neighbourhood I' of I such that orbits of states $x' \, \epsilon \, I'$, remains in this neighbourhood I' for all future times. Otherwise, it is called unstable. If the orbits of neighbouring states $x' \, \epsilon \, I'$ additionally converge to the invariant set I for all future times, the invariant set I is not only stable, but even asymptotically stable. The stability of invariant sets can be determined by graphical or mathematical analysis. Asymptotically stable invariant sets are called *attractors* of a dynamical system. They are called *fixed point attractors, periodic attractors, quasi–periodic attractors* or *chaotic attractors* respectively. The set of states $B \subset M$, whose orbits converge towards an attractor are called the *basin* of an attractor, the converging orbits themselves are called the *transients* of an attractor. The attractors are an important qualitative property of a dynamical system, and they will be in the centre of interest here.

A system is *globally stable* if all system states $x \, \epsilon M$ converge to a single attractor, it is *multistable* if it has more than one attractor. A *convergent* (*dynamically trivial*) dynamical system is one that has only fixed point at-

tractors. A dynamical system is called an *open system* if it interacts with the environment, otherwise, it is called a *closed system*.

Apart from state variables, a system can have *control parameters* r, which change on a larger time scale than the state variables, i.e. in between control parameter changes, a system is left time to converge to an attractor. Control parameters define a parametrised set of different dynamical systems.

2.3 Methodological and Ideological Background

The approach taken in this thesis involves a number of methodological and ideological commitments, out of which the choice to model a learning agent as dynamical system is one. The principle to study learning in situated and embodied models is adopted, as well as the paradigm of artificial neural network theory. The considerations and observations that lead to these commitments will be explained in this section.

The cyberneticist Ross Ashby gave a definition of the learning problem, which will serve as guideline in doing so:

> *"What cerebral changes occur during the learning process, and why does the behaviour usually change for the better?"* ([1], p. 4)

The first thing to notice is the noun phrase "cerebral changes" in Ashby's first question. There is very little doubt that behavioural changes in humans and animals are usually realised as cerebral changes. Straight forwardly, this implies that modelling learning corresponds to modelling neural plasticity within the nervous system. It is not denied that behavioural modelling on a more abstract level of description is valid, but in this thesis, it will be stuck closer to the physical correlate of cognitive processes in biological agents. Still, it has to be stressed that is not intended to give a biologically plausible model of the brain or of a neuron. The assumption presupposed in the theory of artificial neural networks is adopted, that relevant to the constitution of a human's or an animal's behaviour is primarily the architectonic feature of the brain to be composed out of a large number of interconnected simple

units (neurons) that simultaneously transduct signals (compare [10] p. 55f). Also, the present approach subscribes to the idea first uttered by Donald Hebb that learning is realised as changes in the efficacy of synapses (compare [21], p. 18).

Another characteristic of Ashby's definition that should be noticed is that it is composed out of two questions. The first question, "What cerebral changes occur during the learning process" translates to the problem of self–adaptation, while the second question, "why does the behaviour usually change for the better?" corresponds to the problem of behavioural adaptation. As stated in section 2.1, these problems are just two sides of the same coin: You cannot tear them apart, but still, you have to flip the coin in order to see the other side.

In order not to confuse these two questions, it helps to take different stances when approaching them. While being concerned with the second question, why behaviour improves, it is reasonable to take the classical "cognition as information processing" stance, in which an agent is seen as having sensors, effectors and in between a computational (oftentimes called "cognitive") device that causally links them. From that perspective, notions of behaviour, input, output and fitness can be established, *with respect to the environment the agent is located in.* It is important to see that notions like fitness and behaviour depend on the environment in which an agent interacts, since this brings in the commitment to situated and embodied study of cognition. An agent is embodied, if it has defined sensory and effectory systems, it is situated, if it is integrated in a closed sensorimotor–loop[1]. If and why self–modification of an agent yield improved fitness of behaviour depends on the environment an agent is in and how it affects and is affected

[1]Closure of the sensorimotor–loop means that causal relations between inputs and outputs are determined both internal and external to the agent. E.g. in a simplified example, if a ringing phone makes my arm move to pick it up, this is an internal causal connection. But that the ringing then ceases, which I perceive via my sensors, is an external causal connection

by the agent.

On the other hand, in order to answer the first question, it might be helpful to leave the "cognition as information processing"–stance in favour of a more relativist one. The Chilean constructivist Humberto Maturana proposed to view the nervous system as "a closed system"[2] that "generates only states [...] of relative activity between its component neurons" ([9], p.35) instead of relations between inputs and outputs. This view does not mean to deny that there is a causal connection between the nervous system and its environment, it only points out that labelling certain surface components of the system "input sensors" or "output effectors" is not the denomination of intrinsic system properties, but an interpretation by the observer. Such interpretations are always based on certain assumptions about the environment in which an agent interacts (or will interact). Restricting one's view to the system internal dependencies, i.e. neglecting external closure of the sensorimotor loop, helps in answering the first question, because it impedes violation of the constraint that adaptive changes shall be system intrinsic. But even if Maturana's internal view of the system is adopted, it is important to allow perturbation of the system, because otherwise there would be no way to detect environmental changes, to which an agent is supposed to adapt. The learning system can hence not be a closed system in the strict sense of dynamical system theory (compare section 2.2).

The modelling of a situated adaptive agent as dynamical system suggests itself, because dynamical system theory, as explained before, qualifies for describing the development of a system across time. Paying tribute to the hypothesis of neural network theory, the state variables will be those neural properties that are assumed to be involved in signal transduction (compare section 3.1). The constraint that adaptive changes should be intrinsic translates to the formal demand that their dynamics shall be specified within the system.

[2] "closed system" in this context is not equivalent to the term introduced in section 2.2 on dynamical system theory.

Changes in the exterior will be realised as parameter changes, i.e. they are expected to take place on the time scale of control dynamics, such that the system can settle in an attractor in between environmental changes. Pasemann ([12], p. 197, [14], p.12) outlines how in such a scenario the attractors of a system could correspond to different functional modes of the brain. In that framework, the basins of attractors define classes of different system states leading to one behavioural mode. Taking that point of view, learning can be seen as trajectory in the dynamics of synaptic weights, that takes place on the time scale of control dynamics.

Within the just outlined approach to learning, this thesis covers only a very limited part. It is concerned with the proposal of homeostasis (see section 2.4) as regulatory mechanism that realises synaptic learning, on the basis of internal values, an idea that is to be attributed to Pasemann (personal communciation). But it will be restricted to specifying and analysing a homeostatic neuron model, in order to see whether and how it is promising to be applicable in a theory of synaptic learning. Behavioural issues, i.e. an answer to Ashby's second question, will not be addressed at all. The proposed model is far from being a learning model itself, it is just a first step on the way to one.

To give a prospect on how the second question could be answered: The long term vision is to employ the homeostatic neuron model (or an improved variant) in artificial evolution, to evolve adaptive robot controllers in simulated environments. Artificial evolution is a programming technique in which so called "evolutionary algorithms" mimic the process of biological evolution (compare e.g. [15]).

2.4 Homeostasis

Homeostasis is a mechanism of self–regulation. The purpose of a homeostatic mechanism is to maintain a variable as close as possible to a certain desired value. In this thesis, it is conjectured that the principle, on which synaptic

learning is based, is homeostatic maintenance of a target neural activation level. A closer explanation of the term "homeostasis" and how it is included in the approach to learning outlined so far will be performed in this section.

Homeostasis is originally a physiological term, the name was introduced by Walter Cannon in 1932, but the mechanism of maintenance of an internal milieu in spite of environmental changes has been known even before (compare [8], p. 961f). A popular example of homeostatic regulation is the regulation of body temperature, which is successfully maintained at approximately 37° in human beings. This example serves also to introduce the *negative feedback loop*, i.e. "increased effectiveness of the factor or the factors that resist the change" (Shannon, quoted in [8], p. 962), which is the key mechanism of homeostatic regulation: If it is hot, the body sweats in order to cool down, if it is cold, shivering warms the body up.

What makes homeostasis a good candidate for the present purpose is the fact that it promotes a stable state, which meets well with the idea of synaptic weights settling in fixed point attractors. Also, it is an internal regulatory mechanism that does not rely on any presuppositions about external factors, such as reinforcement (see discussion on learning principles below). Empirical support comes from neuroscientific findings on homeostatic regulation in biological cells (compare e.g. summaries in [17] and [6]). Another appeal of homeostasis is the simplicity of the principle, since it centres around a single variable.

Proposing homeostasis as regulatory mechanism that underlies adaptive behaviour is not at all an innovative proposal. It has been popular in the days of cybernetics, e.g. in his book "Design for a Brain" from 1954 [1], Ross Ashby suggests that animals should be seen as homeostats that "maintain the essential variables within physiological limits" ([1], p.57), where essential variables are considered to be variables such as pulse and body temperature, that are directly linked to survival.

An important observation by Ashby is that a homeostatic mechanism

"promotes, but does not guarantee, survival" ([1], p.65), because some environmental changes might be just too drastic to be regulated. This does not contradict the principal adaptive capacity that a homeostatic regulatory mechanism can provide. Actually, a homeostat will even be required to have a limited homeostatic domain, if it is supposed to model a biological organism, because otherwise, the system modelled would be immortal.

This thesis proposes neural activation as a variable that a neuron aims at maintaing stable at a certain target value, by means of adjustment of synaptic weights. Single neurons are considered as autonomous entities that self–regulate locally. Neural activation is not considered as an essential variable in the Ashbyan sense, i.e. loss of homeostasis is not associated with cell death.

Homeostatic adjustment as such cannot be guaranteed to realise learning. It depends on the sensorimotor–loop in which an agent is situated, whether an internal homeostatic mechanism benefits fitness of behaviour. In order to make a system adaptive, homeostatic mechanisms have to be set up in a purposeful way that suits the environmental conditions.

2.5 Related Work

This section will be devoted to a comparison of the outlined approach to learning with others, to point out in how far they are similar or different. It will begin with a short discussion of general types of learning theories, and will then go on with a comparison to research that heads in a similar direction.

A prominent classification of learning systems is the distinction between models of *supervised learning, unsupervised learning* and *reinforcement learning* made in the theory of machine learning and in the theory of artificial neural networks. *Supervised learning* is inspired by the idea that a system learns from a teacher (hence supervised). Technically, a supervised learner is one that has a feedback channel providing it with the percept of the output

it should have produced. A similar approach paying tribute to the idea that frequently there is no unique "correct" response is *reinforcement learning*, in which a learning system is provided with an evaluation measure (reinforcement) of its output, rather than with a certain correct output pattern. Technically, "drawing the line between supervised and reinforcement learning is somewhat arbitrary"([18], footnote p. 528), because a reinforcement stimulus can be seen as a "less informative feedback signal" ([18], footnote p. 528). Both paradigms rely on the idea that external evaluation triggers self–adaptation. A learning system that does not receive an external feedback signal about the correctness of its outputs is called an unsupervised learning system. In unsupervised learning, a system adapts "solely on the basis of its intrinsic connections and dynamics" ([4], p.283), a process that is oftentimes called *self–organisation*. (compare [18], [4], [21])

Supervised and reinforcement learning systems normally learn in an "off–line" training phase from a set of supposedly representative examples. After this training phase, a system is tested on accuracy with another set of examples, and if it performs well, it is maintained the way it is and applied to problems of the learned type. The scientific question addressed in these approaches is normally whether an algorithm can approximate a certain mapping from input patterns to output patterns. These techniques are useful to generate systems to perform in specific domains in which a correct mapping from inputs to outputs exists, such as game playing or pattern recognition. But they do not qualify for modelling learning in an agent that is exposed to an ever changing environment, to which it needs to adapt autonomously and on–line, as it is the case in biological organisms.

Another important property of learning theories is whether they presuppose *context–sensitivity* (or *state–sensitivity*) of behaviour. An assumption closely linked to the one that learning is functional approximation is that a specific sensory input pattern should be mapped uniquely to a specific output pattern. This claim is not adequate, since agents sometimes are bet-

ter off if their actions are chosen sensitive to internal states, in which past experiences can be reflected[3].

This implies that the neural network model implemented has to allow *recurrent* connections, since feed–forward networks, i.e. networks that only transduct signals in one direction, are known to be context–insensitive ([18], p.570). The term "recurrent" is frequently used to refer to self–coupling in neurons, but here it will account for all kinds of feed–back (or *recursive*) connections, i.e. connections that lead to a cycle in network connectivity. Recurrent networks can, other than feed–forward networks, exhibit non–trivial dynamics (compare [14], p. 14f).

Among the theories and approaches to explain learning, there are many that are related to the one adopted in this work.

One example is the *homeokinetic* principle investigated by Der and his group (compare e.g. [16], [5]): They employ robots with a *homeokinetic* control mechanism, in which output patterns are opposed to feedback patterns of actually expected outcomes. The feed–back patterns are derived directly from sensory input, such that their robots controllers can be placed in the realm of unsupervised learning systems, in spite of superficial similarities to supervised learning approaches. Their work is not a competitive approach to the one sketched here, since the homeokinetic principle describes self–regulation on a different level of description, i.e. not with reference to a single variable. It is imaginable that homeokinetic regulation is locally based on homeostatic principles, or, the other way around, that homeostatic networks implement homeokinetic adaptation.

A very related approach that of DiPaolo and his group (compare e.g. [6], [2]). They investigate adaptation in agents controlled by networks of

[3]As an example, consider the protagonist in the movie "Memento" who suffers from hippocampal amnesia: He finds himself in a situation where he and another man are running over a parking site. From the mere percept he concludes that he is chasing the other man, while actually, it is the other way around. His malfunction in memory could be seen as a loss of context–sensitivity of behaviour.

homeostatic neurons. In spite of the similar theoretical background to their research, they have another emphasis: They model adaptation to sensorimotor disruptions (such as the adaptation to wearing goggles that invert the visual field) on the behavioural level. Concerns of underlying neural dynamics are not in the focus of their interest. Their findings provide further support for the hypothesis that the homeostatic neuron model can be employed to realise adaptive behaviour.

Reimann (compare e.g. [17]) investigates dynamical properties of homeostatic neurons from the viewpoint of theoretical neuroscience: His main aim is to explain the homeostatic mechanisms observed in biological brains and the system stability it promotes, whereas its functional role in the adaptation of behaviour is only addressed in the margin.

Although this thesis itself will not be concerned with behavioural issues neither, the approach in which it is embedded could be located in between the ones represented by DiPaolo and Reimann respectively: Other than in DiPaolo's work, it is aimed at explaining processes on a sub–behavioural level, while still, functional role is considered more crucial than biological plausibility, which delimits the approach from Reimann's.

3 The Theoretical Model

Based on the ideas developed so far, this section will introduce the homeostatic neuron model. Section 3.1 will be concerned with the formal definition of the neuron and the network, while section 3.2 will treat the representation of external disruptions of the system, that is adapted to.

3.1 The Neuron and Network Model

The neural network is modelled as a time–discrete dynamical system. Time–discrete modelling has been chosen because it can be assumed that the observed dynamical properties exist as well in corresponding time–continuous dynamical systems, but it is easier to simulate (compare [12], p.196). A neuron is defined as a three–dimensional dynamical system, with state variables a, ξ, η and a state x defined as triple $x := < a, \xi, \eta >$. The state variables will be indexed with a subscript i for neuron n_i, $i = 1, \ldots, N$ in a network of N neurons. a_i is considered as the activation level of a neuron. η_i and ξ_i can be roughly interpreted as the transmitter level and the receptor level of a neuron.

The activation level a_i is assumed to be the essential variable to be regulated homeostatically, as outlined in section 2.4. This implies that the system is supposed to have a fixed point attractor $x_i^{\circledast} = < a_i^{\circledast}, \xi_i^{\circledast}, \eta_i^{\circledast} >$, such that $a_i^{\circledast}$ is the target activation value.

The connectivity structure in a network of N neurons is given by a $N \times N$ connectivity matrix C. The entries are $c_{ij} = 1$ if there is a connection from n_j to n_i and $c_{ij} = 0$ if there is none. All possible connections are allowed. It is deliberated that recursive projections, i.e. those leading to cycles within the connectivity, occur, in order to make non–trivial dynamics possible (compare [14], p. 14).

The strength of a synapse w_{ij} from neuron n_j to neuron n_i is defined as

$$w_{ij}(t) = c_{ij} \cdot \eta_j(t) \cdot \xi_i(t) \tag{1}$$

Hence, it is the dynamics of ξ_i and η_j that realise learning.

The activation level of a neuron is simply defined as the sum of the synaptic inputs and a constant bias term:

$$a_i(t+1) = \theta_i + \xi_i(t) \cdot \sum_{j=0}^{N} (c_{ij} \cdot \eta_j(t) \cdot \sigma(a_j(t))) \tag{2}$$

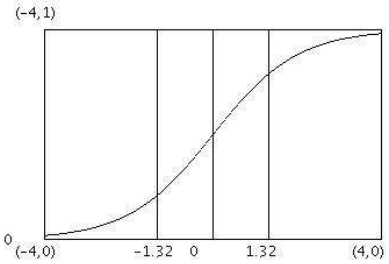

Figure 1: The standard sigmoidal output plotted against the neural activation. The target activation levels $\pm a^{\circledast}$ are marked.

where σ is the standard sigmoidal transfer function $\sigma(a) = \frac{1}{1+e^{-a}}$ (see figure 1). It is interpreted as an idealised approximation of the average firing rate in the postsynapse of a neuron. θ_i is a constant bias term.

There are many possible and plausible ways how to choose $a_i^{\circledast}$, the target activation value of a neuron. Here it is assumed that either $a_i^{\circledast} = +1.31696$ or $a_i^{\circledast} = -1.31696$ for all neurons n_i. It will be referred to these values as $+a_i^{\circledast}$ and $-a_i^{\circledast}$ respectively. These two values were picked because they have the maximum "non–linearity" in the sigmoidal transfer function, i.e. they satisfy $\sigma'''(\pm a_i^{\circledast}) = 0$. This choice was made because, if the system achieves homeostasis, it operates at a point where slight variation of the activation level leads to a substantial variation in slope of $\sigma(a_i(t))$. If a neuron remains at such a point, rich and versatile dynamics are probable[4]. The corresponding output rates are $\sigma(+a^{\circledast}) \approx 0.79$ and $\sigma(-a^{\circledast}) \approx 0.21$.

The equations for the transmitter and receptor levels, η_i and ξ_i, are the equations realising changes in synaptic weight. Although, as has been stressed earlier, the homeostatic neuron is not guaranteed to implement learning, for convenience, the adjustment of synaptic weights will be referred to as learning of synaptic weights.

[4]The same strategy is e.g. exploited in audio amplifiers to cause distortion effects.

The equation for ξ_i is designed in order to achieve homeostasis, i.e. to establish a fixed point attractor $x_i^{\circledast} =< a_i^{\circledast}, \xi_i^{\circledast}, \eta_i^{\circledast} >$ in the dynamical system, such that $a_i^{\circledast}$ is the target activation level.

The equation for η_i is more inspired by Hebbian ideas of an increase in synaptic efficacy upon persistent stimulation. Both equations are designed obeying "Dale's law", that says that a neuron has the same combination of transmitter substances in all its synapses (compare [7], p.214), which implies that the outgoing synapses of a neuron are either all inhibitory or all excitatory in effect. This property is realised in the present approach by fixing the sign of η_i in a neuron and by maintaining ξ_i strictly positive.

The difference equation for the receptor level is given by

$$\xi_i(t+1) = \varepsilon + \xi_i(t) \cdot (1 + \beta \cdot (a_i^{\circledast} - a_i(t)) \cdot \text{sign}\,(a_i(t) - \theta_i)) \tag{3}$$

where β is a learning parameter $0 < \beta < 1$. The receptor potential was intended to be strictly positive, in rare cases where it could drop below 0 ($\beta \cdot (a_i^{\circledast} - a_i(t)) < -1$), it is maintained artificially at 0. This drawback led to the consideration to delimit the amount of change in ξ_i in future models (compare section 5.1). The change in receptor level is computed taking into account the difference $(a_i^{\circledast} - a_i(t))$, which indicates, if and to what amount the activation has to be increased or decreased, and with respect to the sign of the net–internal input $(a_i(t) - \theta_i)$ that indicates, whether an increase or decrease of receptor level leads to the desired outcome. ε is a small real number ($\varepsilon = 10^{-5}$, in the present experiments) that prevents the receptor level from being trapped if it once decreases to 0. It is not assumed to relevantly contribute to the determination of synaptic weights, and will be neglected in the below computations. The receptor level does not saturate.

The difference in the receptor potential $\Delta\xi_i(t) := \xi_i(t+1) - \xi_i(t)$ in a neuron n_i is given by

$$\Delta\xi_i(t) = \beta \cdot \xi_i(t) \cdot (a_i^{\circledast} - a_i(t)) \cdot \text{sign}\,(a_i(t) - \theta_i) \tag{4}$$

The transmitter level η_i is varied depending on the stimulation of the cell.

A neuron has a fixed sign of output. The transmitter level is defined as

$$\eta_i(t+1) = (1-\gamma_1)\cdot\eta_i(t) + \text{sign}\,(\eta_i)\cdot\gamma_2\cdot\sigma(a_i(t)) \tag{5}$$

$\gamma_{1,2}$ are learning parameters $0 < \gamma_i < 1$. $(1-\gamma_1)$ can be interpreted as a decay term, $\gamma_2\cdot\sigma(a_i(t))$ is an increase in transmitter level triggered by the activation of the neuron. The transmitter level does neither saturate.

The difference in transmitter level $\Delta\eta_i(t) := \eta_i(t+1) - \eta_i(t)$ in a neuron n_i is given by

$$\Delta\eta_i(t) = -\gamma_1\cdot\eta_i(t) + \text{sign}\,(\eta_i(t))\cdot\gamma_2\cdot\sigma(a_i(t)) \tag{6}$$

The difference in synaptic weight $\Delta w_{ij}(t) := w_{ij}(t+1) - w_{ij}(t)$ in a synapse with $c_{ij} = 1$ is then given by

$$\begin{aligned}
\Delta w_{ij}(t) &= \eta_j(t+1)\cdot\xi_i(t+1) - \eta_j(t)\cdot\xi_i(t) && (7)\\
&= -\ \gamma_1\cdot\eta_j(t)\cdot\xi_i(t) && (8)\\
&\quad +\ \gamma_2\cdot\text{sign}\,(\eta_j)\cdot\sigma(a_j(t))\cdot\xi_i(t)\\
&\quad +\ \text{sign}(a_i(t)-\theta_i)\cdot\text{sign}(\eta_j)\cdot(a_i^{\circledast} - a_i(t))\\
&\qquad \cdot\beta\cdot\xi_i(t)\cdot((1-\gamma_1)\cdot|\eta_j(t)| + \gamma_2\cdot\sigma(a_j(t)))
\end{aligned}$$

The three addends making up $\Delta w_{ij}(t)$ identified in equation (8) display the different factors taking part in the determination of synaptic weight. The first one is very easy to identify, it denotes a general decay in $|w_{ij}|$. The second one is a roughly hebbian term, it reflects growth of $|w_{ij}|$, depending on presynaptic activation and receptor density in the postsynapse. The third term is the one that promotes homeostasis. The product of the two sign terms is 1 in the synapses that were dominant in the determination of the sign of the summed synaptic input ($\text{sign}(a_i(t)-\theta_i)$) at time t. The adjustment of synaptic weights is then directed by the term $(a_i^{\circledast} - a_i(t))$: If $a_i(t)$ has been lower than desired, the synaptic weight is lifted, if it was higher, it is lowered. Synapses with a reverse sign of input ($\text{sign}(\eta_j) = -\text{sign}(a_i(t)-\theta_i)$) are adapted in a counterproductive way, which can be

explained by the fact that receptor levels in a neuron n_i have to be adjusted globally for all its incoming synapses. The amount of change in this addend is biased with the absolute synaptic strength and presynaptic activation.

The dynamical analysis will be employed to show that a single self–coupled neuron is homeostatic for some parameter settings, i.e. that there is a fixed point attractor $x_i^{\circledast} \epsilon M$ such that the target activation level $a_i^{\circledast}$ is a component of that fixed point. In the following it will be argued that if the target activation level is maintained homeostatically, the other system variables are stationary too, i.e. stability on $a_i^{\circledast}$ guarantees a system fixed point $x_i^{\circledast} =< a_i^{\circledast}, \xi_i^{\circledast}, \eta_i^{\circledast} >$. Furthermore, dependencies of $a_i^{\circledast}$,$\xi_i^{\circledast}$ and $\eta_i^{\circledast}$ on each other and on the network parameters will be analysed.

For the receptor potential it is true by definition that $a_i(t) = a_i^{\circledast} \Rightarrow \xi_i(t+1) = \xi_i(t)$, if the auxiliary term ε is neglected. Unlike $a_i^{\circledast}$, $\xi^{\circledast}$ is not one fixed value for all possible situations, but depends on synaptic inputs and system parameters. Let $I_i^{syn}(t) := \sum_{j=0}^{N}(c_{ij} \cdot \eta_j(t) \cdot \sigma(a_j(t)))$, the sum over the synaptic inputs to a neuron n_i. If a network is stationary and is not distorted by external changes (i.e. I_i^{syn} is constant), the fixed point component $\xi_i^{\circledast}$ of the receptor level is given by

$$\xi_i^{\circledast} = \frac{a_i^{\circledast} - \theta_i}{I_i^{syn}} \tag{9}$$

$\xi_i(t)$ converges towards $\xi_i^{\circledast}$ as $a_i(t)$ converges to $a_i^{\circledast}$. It should be noted that $\xi_i^{\circledast}$, according to this equation, would have to be negative for some parameter settings. The parameter domains in which this is the case are those domains in which self–regulation is in principle impossible, of course the receptor level does not drop below $\xi_i = 0$. The point at which $\xi_i^{\circledast} = 0$ will be referred to as the *natural limit of homeostasis*.

Given $a_i(t) = a_i^{\circledast}$ and $\xi_i(t) = \xi_i^{\circledast}$ are stationary, η_i also has to be stationary on a fixed value $\eta_i^{\circledast}$, if the synaptic inputs and system parameter stay the same. Indeed, there is one fixed point η_i^* for all stationary states a_i^* of the activation level, be they target activation or not. η_i^* depends sigmoidally

on a_i^*, scaled by the relation between the learning parameters γ_1 and γ_2 and its own sign (inhibition or excitation).

$$\eta_i^*(a_i^*) = \text{sign}\,(\eta_i) \cdot \frac{\gamma_2}{\gamma_1} \cdot \sigma(a_i^*) \tag{10}$$

If a_i is stationary at some value a_i^*, η_i converges to $\eta_i^*(a_i^*)$. The η-component of the system's target fixed point $x_i^{\circledast}$ will be referred to as $\eta_i^{\circledast} = \eta_i^*(a_i^{\circledast})$.

It has been decided to fix the relation $\frac{\gamma_1}{\gamma_2}$, such that the relation between $\eta_i^{\circledast}$ on $a_i^{\circledast}$ does not change in between experiments. Let $\frac{\gamma_2}{\gamma_1} := 2$. With this choice, if the neuron is not activated at all, i.e. $a_i(t) = 0$, it fires with an average output rate of $\sigma(0) = 0.5$. The corresponding fixed point of the transmitter level is normalised at $|\eta_i^*(\sigma(0))| = 1$. At a maximum stable synaptic output of $\sigma(\pm\infty)$, the transmitter level has fixed points $|\eta_i^*(-\infty)| = 0$ or $|\eta_i^*(+\infty)| = 2$, respectively. Although these dependencies only hold for stationary activation levels of the system, it is assumed that the absolute values of the transmitter level $|\eta_i(t)|$ in most settings will not substantially exceed the interval $[0, 2]$. According to equation (10), the fixed point component $\eta_i^{\circledast}$ result in $|\eta_i^{\circledast}(+a_i^{\circledast})| \approx 1.58$, and $|\eta_i^{\circledast}(-a_i^{\circledast})| \approx 0.42$ respectively. It is not necassary to specify both, γ_1 and γ_2 in the experiments, if their relation is fixed. For convenience, only γ_1 will be defined, the subscript 1 will be omitted, i.e. $\gamma_1 := \gamma$, $\gamma_2 := 2 \cdot \gamma$.

The convergence to $\xi_i^{\circledast}$ and $\eta_j^{\circledast}$ is accompanied by a convergence of w_{ij} to

$$w_{ij}^{\circledast} = \eta_j^{\circledast} \cdot \xi_i^{\circledast} = \text{sign}(\eta_j) \cdot 2 \cdot \sigma(a_j^{\circledast}) \cdot \frac{a_i^{\circledast} - \theta_i}{I_i^{syn}} \tag{11}$$

3.2 External Input

As discussed in section 2.3, there has to be a way how the environment takes influence on the system state, and this environmental effect is supposed to change on the time scale of control dynamics. Since the neuron model is supposed to homeostatically maintain a target activation value $a_i^{\circledast}$, it is reasonable to model environmental disruptions such that they affect the

activation level. As has been anticipated in section 2.3, environmental inputs are supposed to change on the time–scale of control parameters, i.e. they parametrise a family of dynamical systems.

In the introduced neuron model, there is at least two ways how to induce external distortive input: Directly or via other neurons. Formally, these variants yield the following difference: Direct input can be set against the bias term θ_i, i.e. $\theta_i' := I_{ext} + \theta_i$ is varied. In the experiments where direct induction of inputs is investigated, θ_i' will be simply referred to as θ_i. Neuronal inputs operate via additional addends in the summed synaptic input $I_i^{syn}(t)$. In this set–up, the sum of outputs of a group of k neurons o_0 will be varied. Let $o_0 := \sum_{j=0}^{k}(c_{ij} \cdot \eta_j(t) \cdot \sigma(a_j(t)))$, the external synaptic input to a neuron n_i.

There is a crucial difference between these two options: In the case of distortion via synapses, the neuron has means to directly regulate this input by adjustment of ξ_i. If activation is induced directly in the cell, the neuron can only compensate for distortion by means of regulation of the net–internal input.

Both of the variants have advantages and disadvantages: Direct input decreases system complexity, because of the reinterpretation of θ_i, but on the other hand, it is questionable, why input from the exterior, other than net–internal input, should not be directly regulatable. This work will take into consideration both models.

Recapitulating section 2.4, a homeostat ought to have a limited space of environmental circumstances in which it achieves to regulate. For the single autonomous neuron, this demand translates to the demand to find and characterise an interval of external inputs, in which $x_i^{\circledast}$ is a fixed point attractor of the system, and enclosing intervals in which it is not. The analysis of networks of several coupled homeostatic neurons is performed in order to characterise network attractors and their stability domains in the parameter space.

4 Results

The investigation of neural dynamics will be restricted to very small networks, i.e. to a single neuron and networks of two neurons with mutual coupling and all possible variants of self–coupling.

Section 4.1 will introduce the investigatory tools employed and how the diagrams have to be read. Section 4.2 will present findings on dynamical properties of corresponding hard–wired neural networks, since dynamics in stable intervals are likely to obey the regularities found there. In section 4.3, equation (3) and equation (5), will be investigated independently, in order to understand their effects in synaptic regulation. The single neuron network will be treated in section 4.4, while section 4.5 will deal with two neuron networks in different possible set–ups. Section 4.6 will sum up the findings obtained.

4.1 Employed Methods

As already explained, the focus of investigation will be on attractors and asymptotic behaviour of the system upon variation of an external input. The analysis will be primarily a phenomenological one, evaluating bifurcation and attractor diagrams (see below), although sometimes it will be referred to mathematical findings.

In bifurcation diagrams (e.g. figure 2), a system variable x is plotted against a control parameter r, that is slowly increased or decreased. After the system has been left time to relax, a number of successive values of x is plotted, visualising the attractor the system has converged to. Typically, there are intervals of r, in which the system has attractors that are qualitatively equal. The limits of such intervals are called *bifurcation points*. Bifurcation diagrams exhibit characteristic patterns that display attractors and bifurcation points of a dynamical system. Figure 2 shows an interesting bifurcation diagram of neural output that depicts a variety of different attractors, with bifurcations points marked as t_i (bifurcation points will al-

ways be marked this way). Intervals of r, in which lines appear, represent intervals in which the system converges to a fixed point attractor (if it is one line) or a period n attractor (if there are n different lines). Quasi–periodic and chaotic attractors occur in bifurcation diagrams as regions in which plotted states do not form lines, but remain dense (e.g. 2, $[t_2, t_3]$, the area that looks shaded on the first glance). To determine whether an attractor is chaotic or quasi–periodic, it is oftentimes necessary to employ mathematical analysis, for instance calculation of the Liapunov–exponents, or to closer investigate specific orbits.

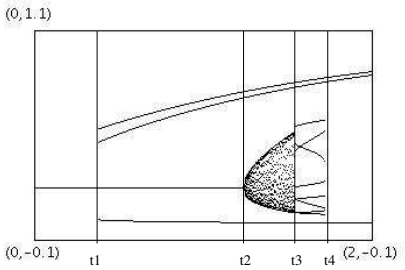

Figure 2: Example bifurcation diagram for the output of a self–coupled neuron upon variation of θ_i, with hysteresis: For increasing θ_i, the system has a fixed point attractor ($[0, t_2]$), a chaotic or quasi–periodic attractor ($[t_2, t_3]$), a period 8 attractor ($[t_3, t_4]$) and a period 3 attractor ($[t_4, 2]$). For decreasing θ_i, the system is in the period 3 attractor throughout the interval $[t_1, 2]$, then the system jumps to the fixed point attractor ($[0, t_1]$).

Coexistence of attractors cannot be displayed, if bifurcation diagrams are employed as outlined so far, since computation follows a single orbit. If the control parameter r is first increased and then decreased, and the outcome is plotted in the same diagram, coexistent attractors can be detected. A special situation in which attractors coexist is the so called hysteresis effect, a type of irreversible change: Sometimes, after a system has crossed a bifurcation point t_i, if r is then varied in the reverse direction, the system crosses t_i without showing a bifurcation. Characteristically, there occurs a bifurcation at another point t_j that has not been a bifurcation point on the way there. At that point t_j the system returns to the attractor it was in on the way there, the system is thus bistable in an interval $[t_j, t_i]$. Figure 2 also contains an example of such a hysteresis, the system is bistable in the interval $[t_1, t_4]$, with a period three attractor for decreasing r and a

sequence of different attractors (a fixed point, a period eight and a chaotic or quasi–periodic attractor) for increasing r.

In the present work, bifurcation diagrams will be employed to find and characterise homeostatic domains in the parameter space given for neural networks of the type outlined in section 3. The control parameter r varied will normally be the external input, i.e. the bias term θ_1, if direct input is chosen, or else the external synaptic input o_0 (compare section 3.2). Stability analysis will ordinarily not exceed an interval $[-10, 10]$, since very high external inputs are not of interest, homeostatic domains are assumed and desired to be located at low absolute values of input. The state variables monitored are the neural outputs $\sigma(a_i)$, the transmitter levels η_i or the receptor levels ξ_i. The convergence time at each value r normally is 2000 time steps, then, 20 successive variable values are plotted.

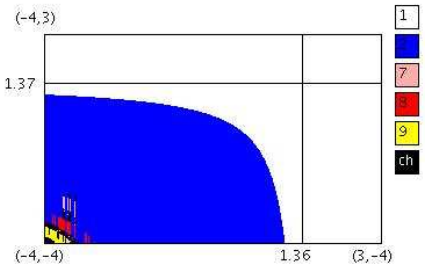

Figure 3: Example of an isoperiodic plot of a two–neurone network with varied θ_1, θ_2, with different isoperiodic domains (white: stationarity, coloured: periodic oscillation, see legend. Black: higher periods, quasi–periodic oscillation or chaotic oscillation). The stripes in the bottom left corner allude to a coexistence of attractors.

Another kind of diagram used are isoperiodic plots (see figure 3). Isoperiodic plots are computed similarly as bifurcation diagrams, but they have control parameters r_x, r_y plotted on both, the horizontal and on the vertical axis. Convergence is computed at every point (r_x, r_y), starting from the same random initial condition. The system is iterated for slowly increasing values of r_y, while r_x is fixed. Then, r_y is reset and the computation is repeated for a slightly higher value r_x, etc. The period of the resultant orbit is encoded as a colour in lack of a third dimension. Bifurcation points are then normally located at the boundary line of areas of the same colour. Sets of bifurcation points delimiting a

domain of qualitatively equal attractors are called *bifurcation sets*. In isoperiodic plots, there is no information displayed on distances between fixed points, and no means to directly display multistability. Stripes, such as in the bottom left corner of figure 3, indicate coexistence of attractors. In this thesis, isoperiodic plots will be computed for two neuron networks, with $r_x = \theta_1$ and $r_y = \theta_2$.

Isoperiodic plots in this thesis display stability of the network, not of a single neuron. If neurons converge to qualitatively different attractors, the respectively more complex attractor will be depicted. All periodic attractors of period greater than 9, as well as chaotic or quasi–periodic attractors will be drawn black, the "ch" (see figure 3) in the legend does not necessarily mean chaos.

4.2 Dynamical Properties of Hard–Wired Neural Networks

During homeostasis, the networks are stationary, which means they have stationary synaptic weights. It is probable that in homeostatic domains, the network dynamics correspond to the dynamics observed for hard–wired neural networks.

The dynamical properties of small time–discrete neural networks with additive neurons, sigmoidal transfer functions and fixed synaptic weights, have e.g. been investigated by Pasemann. Three of his publications, the papers "Dynamics of a Single Model Neuron."[13], "Characterisation of Periodic Attractors in Neural Ring Networks."[11] and "Complex Dynamics and the Structure of Small Neural Networks."[12] will be briefly summarised insofar as they are relevant for the present enterprise.

Figure 4 shows the stability domains in the (θ_i, w_{ii})–space, Pasemann has discovered for a single self–coupled neuron n_i in [13]. Self–coupled autonomous neurons converge to global fixed point attractors for a large part of the (θ_i, w_{ii})–space (region I in figure 4). For some θ_i and an excitatory weight $w_{ii} > 4$, coexistence of two fixed point attractors is found (region

II in figure 4). If θ_i is slowly increased and decreased, hysteresis can be observed when crossing that domain. For some θ_i and inhibitory weights $w_{ii} < -4$, a self–coupled neuron oscillates at period 2 (region III in figure 4).

Attractors in ring networks, i.e. networks in which neurons are connected in a one–directional single cycle, have been investigated in [11]. It was found out that hard–wired two neuron rings have parameter domains in which non–trivial dynamics occur. Two neuron rings in which either both neurons are inhibitory or both neurons are excitatory (also called "even" two neuron rings) have a domain in which a period two attractor coexists with two fixed point attractors, while odd two neuron rings, i.e. two neuron rings in which one of the neurons is inhibitory and the other one excitatory, show global period four oscillation in non–stationary domains. The two neuron ring set–up will be investigated in this work as well, it will be compared if the findings agree.

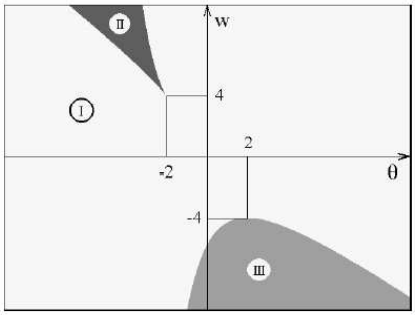

Figure 4: Attractor domains for a single self–coupled hard–wired neuron in the (θ_i, w_{ii})–space. I: global stability on a fixed point attractor II: hysteresis of two fixed point attractors III: a period two–attractor. (From [13])

In [12], recurrent networks of two and three neurons have been investigated. For two neurons, Pasemann has analysed the networks with respect to stability conditions on the determinant D and the trace T of the Jacobian matrix of their neural dynamics. The domain in the (D,T)–space of a dynamical system in which there are only stable fixed points corresponds to the triangular region in figure 5. The exact mathematical background is not subject to the present work. It suffices to know how T and D are computed in a network of two homeostatic neurons while it is stationary, to

determine the location of a system in the (D,T)–space. It is presupposed that $c_{12} = c_{21} = 1$.

$$T(t) = c_{11} \cdot \xi_1(t) \cdot \eta_1(t) \cdot \sigma'(a_1(t)) + c_{22} \cdot \xi_2(t) \cdot \eta_2(t) \cdot \sigma'(a_2(t)) \quad (12)$$

$$D(t) = (c_{11} \cdot c_{22} - 1) \cdot \xi_1(t) \cdot \xi_2(t) \cdot \eta_1(t) \cdot \eta_2(t) \cdot \sigma'(a_1(t)) \cdot \sigma'(a_2(t)) \quad (13)$$

From these equations, it can already be seen that $c_{11} = c_{22} = 1 \Rightarrow D(t) = 0$ and $c_{11} = c_{22} = 0 \Rightarrow T(t) = 0$.

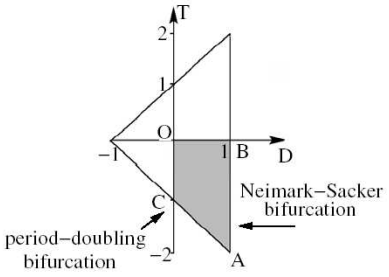

Figure 5: Stability domain for a fixed point (triangular shaped area) in the (D, T)–space, where D is the determinant, T the trace of the Jacobian matrix of the linearised dynamical system. (From [12])

Loss of homeostasis is expected to occur in the homeostatic networks upon leaving the triangular area in the (T, D)–space. The shaded area is not of special interest for this thesis.

It has to be remarked that in spite of stationarity during homeostasis, networks of self–regulating neurons are still higher dimensional dynamical systems than their hard–wired pendants. The findings introduced in this section can only be used to generate estimates about dynamical phenomena that are likely to occur. It will be investigated if and how the dynamics of homeostatic neurons deviates from the dynamics of hard–wired neurons.

4.3 An Analysis of the Learning Equations in Isolation

This section will investigate the equations for η_i and ξ_i in isolation, to get an impression about how they take part in the adjustment of a synapse. For this purpose, the respectively other state variable will be clamped to either $\xi_i = 1$ or $\eta_i = 1$. The changes in synaptic weight will be examined and the effect of the equations in a self–coupled neuron will be discussed.

As derived in section 3.2, there is a scaled sigmoidal dependency between fixed activation levels a_i^* and corresponding fixed transmitter levels η_i^*. Also,

$\xi_i^{\circledast}$ depends on the synaptic inputs I_i^{syn} and the bias term θ_i. These dependencies are expected to be reflected in the following experiments.

Figure 6 shows the bifurcation diagram of η_i for varying a_i. It displays the dependency of η_i^* on a_i^*. The curve shows a scaled sigmoidal function, as it was expected from equation (10). For an inhibitory neuron, the curve is mirrored about the horizontal at $\eta_i = 0$.

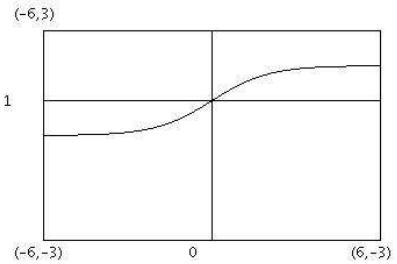

Figure 6: Bifurcation diagram for η_i, with variation of a_i, $\gamma = 0.01$ and $\text{sign}(\eta_i) = 1$

Figure 7 shows the interplay in convergence of transmitter level and activation level in a self–coupled neuron with $\xi_i = 1$. Compared to the standard sigmoidal output (figure 7 (b)), if a neuron is excitatorily self–coupled (figure 7 (a)), the slope gets steeper, if it is inhibitorily self–coupled (figure 7 (c)), it gets shallower. If the relation $\frac{\gamma_2}{\gamma_1}$ is chosen differently, the neuron can enter the hysteresis or oscillatory domain as characterised in [13] and depicted in figure 4.

The receptor equation is investigated accordingly in isolation, with respect to how it relates to parameter changes. Figure 8 (b) shows the bi-

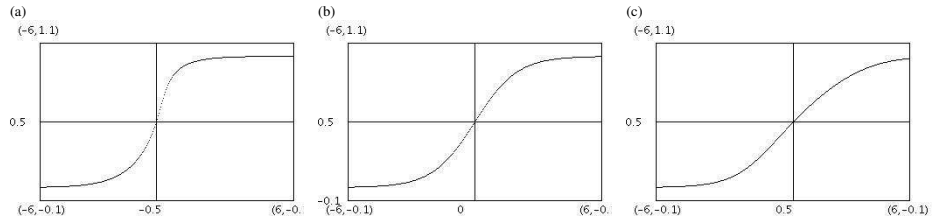

Figure 7: Output bifurcation diagrams of n_i with $\xi_i = 1$ and variation of θ_i and $\gamma = 0.01$. (a): self–excitation ($w_{ii} = 1$, $\text{sign}(\eta_i) = 1$), (b): no synapses (sigmoidal output), (c): self–inhibition ($w_{ii} = 1$, $\text{sign}(\eta_i) = -1$).

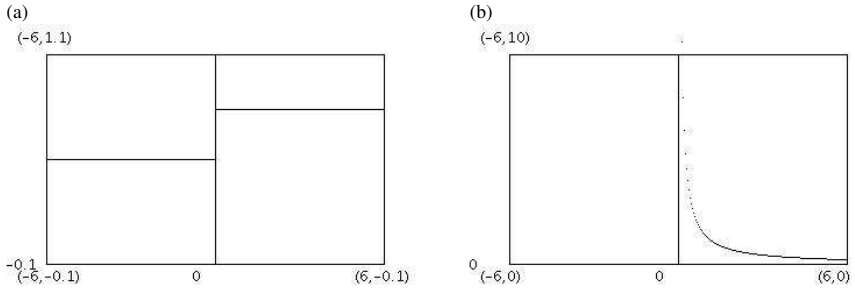

Figure 8: Output (a) and receptor (b) bifurcation diagrams of a neuron upon varied synaptic input I_i^{syn}. $\theta_i = 0.0$, $\beta = 0.01$

furcation diagram of ξ_i, as I_i^{syn} is varied, $\theta_i = 0$. In figure 8 (a) the corresponding output of the postsynaptic neuron, $\sigma(a_i)$, is plotted. As predicted by equation (9), the converged ξ_i is hyperbolically dependent on I_i^{syn} for $I_i^{syn} > 0$, to the right of the natural limit of homeostasis at $I_i^{syn} = 0$, where ξ_i has a vertical asymptote. According to equation (9), $\xi_i^{\circledast}$ would have to be negative for $I_i^{syn} < 0$, in order to achieve homeostasis. In this interval, ξ_i is stationary at $\xi_i^* = 0$. The output diagram (figure 8 (a)) shows homeostasis (i.e. stationary output of $\sigma(+a^{\circledast}) = 0.79$) for $I_i^{syn} > 0$ and resting activation ($\sigma(a_i) = \sigma(\theta_i) = 0.5$) for $I_i^{syn} < 0$. These findings confirm the principal regulatory capacity of the system, realised by the receptor equation (3). In domains where homeostasis is not possible, a positive ξ_i would only allow synaptic inputs to draw activation even further away from $a_i^{\circledast}$, hence the stabilisation on $\xi_i = 0$.

The explosive growth of receptors, as I_i^{syn} approaches 0 from the right reflects the desperate effort to maintain homeostasis upon approaching the natural limit of the homeostatic interval. From the biological viewpoint, it is not plausible that the receptor level grows to infinity, in future trials a saturation level should be implemented (compare section 5.1).

In the present model, obeying Dale's law (compare section 3.1) has the side–effect that a neuron can only remain homeostatic if it receives inputs

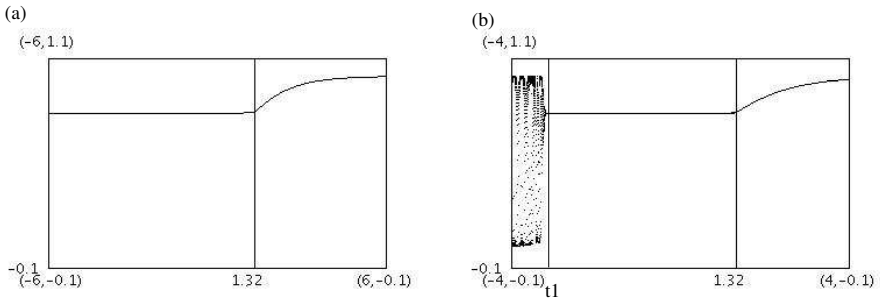

Figure 9: Output bifurcation diagrams for a single neuron with $a_i^{\circledast} = +a^{\circledast}$, variation of θ_i. (a) No synapse, $I_i^{syn} = 4$. (b) A self–synapse ($c_{ii} = 1$), $\eta_i = 1$, no other synaptic input.

of a fixed sign, namely $\mathrm{sign}(a_i^{\circledast} - \theta_i)$. Neurons that have inhibitory and excitatory input synapses have to be investigated with special attention, since the synaptic input can potentially change its sign.

Figure 9 shows the bifurcation diagrams of neural outputs upon variation of the bias term θ_i, in a neuron with constant synaptic innervation $I_i^{syn} = 4$ (a) and excitatory self–coupling (b), η_i is clamped to $+1$ in both trials.

In both set–ups, homeostasis could be observed. The homeostatic intervals are limited to the right by the point $\theta_i = a_i^{\circledast} \approx 1.32$. For $\theta_i > a_i^{\circledast}$, self–regulation is not possible (compare section 3.1). The receptor level ξ_i (not depicted) decreases to 0 and consequently, the output is stationary on $\sigma(\theta_i)$. Within the homeostatic intervals, the receptor level ξ_i grows linearly as θ_i decreases, as predicted by equation (9).

If the neuron only receives external input (figure 9 (a)), homeostasis is achieved practically to infinity for $\theta_i < a_i^{\circledast}$. In the self–coupled neuron (figure 9 (b)), homeostasis ceases at $\theta_i = t_1$ and non–trivial dynamics can be observed. The bifurcation point t_1 seems to be identifiable with a bifurcation point found for hard–wired self–coupled neurons, i.e. it can be located in the bifurcation set between regions I and II in figure 4. Whereas hard–wired neurons enter a hysteresis domain of two fixed points crossing that

bifurcation set (compare section 4.2), the homeostatic neuron settles in a chaotic or quasi–periodic attractor. The self–coupled neuron with constant η_i already has a limited homeostatic domain, as demanded from a homeostat in section 2.4.

With respect to the two different possibilities of inducing synaptic input (via input synapses or via variation of the bias term θ_i), the investigation of ξ_i in isolation provides some insights:

The variation I_i^{syn} can be seen as a special case of variation of o_0, in which there is no other synaptic input. In a single self–coupled neuron, there will be just one additional internal synapse, whose input will be added to o_0. The findings can hence be expected to be similar to those obtained for variation of I_i^{syn}. The low receptor levels for large $|I_i^{syn}|$ (compare figure 8(b)) allude that in self–coupled neurons, non–trivial dynamics will not occur in intervals with large $|o_0|$, since absolute values of synaptic weights $|w_{ii}|$ have to be > 4 to cause non–trivial dynamics in hard–wired neurons (compare section 4.2). On the other hand, the hyperbolic growth of receptors, if I_i^{syn} approaches the natural limit of homeostasis at $I_i^{syn} = 0$, predicts an interval of non–trivial dynamics there, since $|w_{ii}|$ are assumed to be large in that interval.

When θ_i is varied, which is exactly the technique applied if input is induced directly, the adjustment of ξ_i is linear, as could be seen in the experiments depicted in figure 9, ξ_i decreases to 0 if it approaches the natural limit of homeostasis, it grows far from that point. Thus, non–trivial dynamics are predicted to occur far from that limit, because synaptic strengths are expected to be larger there.

4.4 A Single Self–Coupled Neuron

The foremost purpose in the investigation of a single self–coupled neuron is to give evidence that the introduced neuron model actually defines a homeostat as defined in section 2.4, i.e. it is anticipated to find parameter settings for which there is an interval of I_{ext} in which the desired state $x^{\circledast}$ is a

fixed point attractor of the self–coupled neuron. Further, it is expected that non–trivial attractors delimit the homeostatic interval, in the domains in the (w_{ii}, θ_i)–space that were characterised for hard–wired self–coupled neurons (compare section 4.2). Section 4.4.1 will investigate the single self–coupled neuron upon variation of synaptic input, while section 4.4.2 will discuss the set–up in which θ_i is varied.

4.4.1 External Input as Synaptic Input

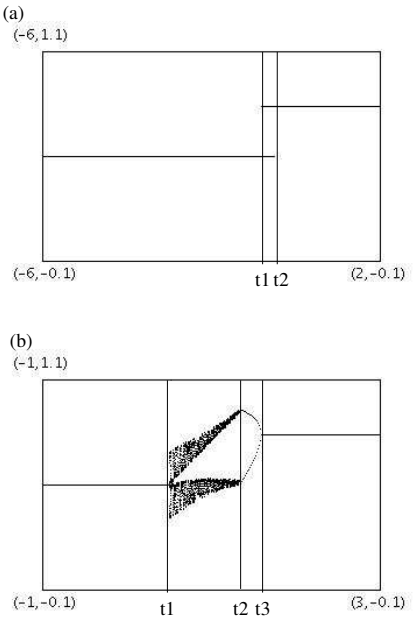

Figure 10: Output bifurcation diagrams for self–coupled neurons with $a_i^{\circledast} = +a^{\circledast}$, $\theta_i = 0$, $\beta = \gamma = 0.01$. Variation of o_0. (a) excitatory ($\text{sign}(\eta_i) = 1$), (b) inhibitory ($\text{sign}(\eta_i) = -1$)

In the following experiments, the control parameter varied will be the postsynaptic output o_0 of a group of k input neurons (see section 3.2), projecting onto n_i, the self–coupled neuron under investigation.

Figure 10 shows two typical patterns of output diagrams for an excitatory (a) and an inhibitory (b) self–coupled neuron. As predicted in the last section, the findings obtained are similar to those found for variation of I_i^{syn} in a neuron with $c_{ii} = 0$. Still, non–trivial dynamics occur in the vicinity of the natural limit of homeostasis at $I_i^{syn} = o_0 + \eta_i^{\circledast} \cdot \sigma(a_i^{\circledast}) = 0$, as anticipated. The homeostatic interval is delimited to one side by the natural limit of homeostasis at $I_i^{syn} = 0$ ($o_0 = t_2$ in the excitatory neuron (a), $o_0 = t_1$ in the inhibitory neuron (b)).

The other limit point (t_1 in (a) and t_3 in (b)) of the non–trivial interval seems to correspond to a point in one of the bifurcation sets found for

hard–wired neurons (compare section 4.2, figure 4), if the contribution of the external synaptic input to neural potential is considered, i.e. if θ_i in the diagram is substituted for $\theta'_i = \theta_i + \xi_i^{\circledast} \cdot o_0$. The length of the interval o_0 with non–trivial dynamics depends on the difference $(a_i^{\circledast} - \theta_i)$. This is because this difference linearly influences $w_{ii}^{\circledast}$ (compare equation (11)), and has a strong impact on θ'_i, the two variables that have to exceed certain values to enter the domain of non–trivial oscillation characterised in figure 4.

The non–trivial interval is enclosed by two intervals of stationary activation. In agreement with the findings on neurons with $c_{ii} = 0$ in section 4.3, activation is homeostatic to the side of that dynamically non–trivial interval, where $\text{sign}(a_i^{\circledast} - \theta_i) = \text{sign}(\eta_i)$ and stationary on $a_i^* = \theta_i = 0$ to the other side. Non–trivial dynamics in inhibitory neurons generally involve period two oscillation, while excitatory neurons tend to exhibit hystereses, which agrees with the finding on hard–wired neurons introduced in section 4.2, but higher order, quasi–periodic or chaotic oscillation occurs as well (e.g. in figure 10 (b), interval $[t_2, t_3]$).

Thus, as predicted, if the control parameter varied is o_0, non–trivial dynamics only occur close to the natural limit of the homeostatic interval, where I_i^{syn} switches sign in a self–coupled neuron. The homeostatic interval is practically not delimited to the other side.

4.4.2 External Input Directly Added to Activation

If the input varied is directly induced into the self–coupled neuron (i.e. variation of θ_i), the neuron generally shows a homeostatic interval enclosed by a dynamically non–trivial interval and another interval in which the output is stationary on $\sigma(\theta_i)$, as it was already observed in a self–coupled neuron with $\eta_i = 1$ (compare figure 9, (b)).

Figure 11 shows the output (a), receptor (b) and transmitter (c) bifurcation diagrams of an inhibitory neuron with $a_i^{\circledast} = -a^{\circledast}$. In the output diagram (a), there is a homeostatic interval $[a_i^{\circledast}, t_1]$ in which the output is

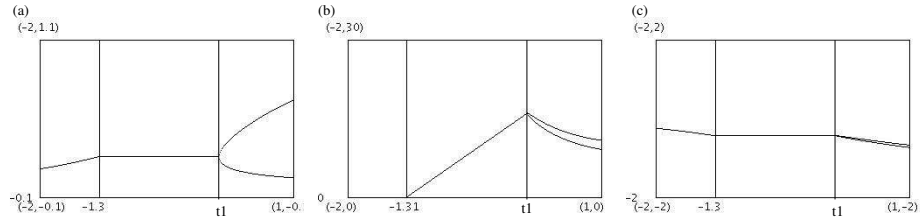

Figure 11: Output, receptor and transmitter bifurcation diagrams for an inhibitory ($\mathrm{sign}(\eta_i) = -1$) self–coupled neuron. Variation of θ_i. $a_i^{\circledast} = -a^{\circledast}$, $\beta = 0.1$, $\gamma = 0.1$

maintained on $\sigma(a_i^{\circledast})$. The receptor diagram (b) shows that in this interval, $\xi_i^{\circledast}$ grows proportional to θ_i, as expected from equation (9). For $\theta_i < a_i^{\circledast}$ (i.e. beyond the natural limit of homeostasis), the output stabilises on $\sigma(\theta_i)$, since receptors remain stable at $\xi_i^* = 0$. For $\theta_i > t_1$, the neuron oscillates at period two. The bifurcation point t_1 can apparantly be identified with a point at the border of region I and region III in figure 4. Also, the oscillation at period two corresponds to the findings obtained for hard–wired neurons. The transmitter equation displays the dependency of η_i^* on a_i^* expressed in equation (10).

Analogous attractor patterns (see figure 12) could be observed for set–ups with $a_i^{\circledast} = +a^{\circledast}$ ((b) and (c)) and excitatory neurons ((a) and (b)). The bifurcation points t_1 apparantly can always be identified with points on

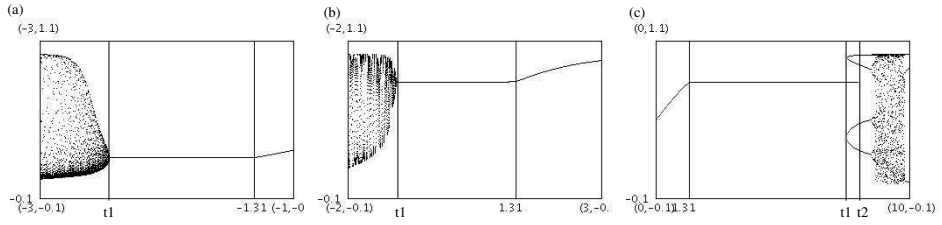

Figure 12: Output bifurcation diagrams for single self–coupled neurons. Variation of θ_i. (a): $\mathrm{sign}(\eta_i) = 1$, $a_i^{\circledast} = -a^{\circledast}$, $\beta = 0.1$, $\gamma = 0.01$. (b): $\mathrm{sign}(\eta_i) = 1$, $a_i^{\circledast} = +a^{\circledast}$, $\beta = 0.01$, $\gamma = 0.5$. (c): $\mathrm{sign}(\eta_i) = -1$, $a_i^{\circledast} = +a^{\circledast}$, $\beta = 0.1$, $\gamma = 0.1$.

one of the borderlines between region I and region II or III respectively in figure 4. The homeostatic interval is delimited to the other side by the natural limit point $\theta_i = a_i^{\circledast}$ (compare equation (9)).

In general, the learning parameters β, γ can be changed in a broad range without substantially changing the qualitative behaviour of the system. Only for big β, an unfavourable phenomenon occurs: Some quasi–periodic or chaotic attractors exceed the artificial threshold $\xi_1 = 0$, if β is not sufficiently small (see figure 13). As already mentioned, a discussion of how to fix the drawback that ξ_i can drop below 0 according to equation (3) in future models will be performed in section 5.1. The dynamics in these cases develop in an odd way. It is hard to say what "big β" are, it seems that for very long intervals, this odd behaviour occurs for all β. The bigger β is chosen, the shorter gets the interval of "non–odd–but–non–trivial" dynamics (interval $[t_1, t_2]$ in figure 13). Cases in which such artefacts occur were excluded from analysis, since they do not display "natural" dynamics, as described by the state equations.

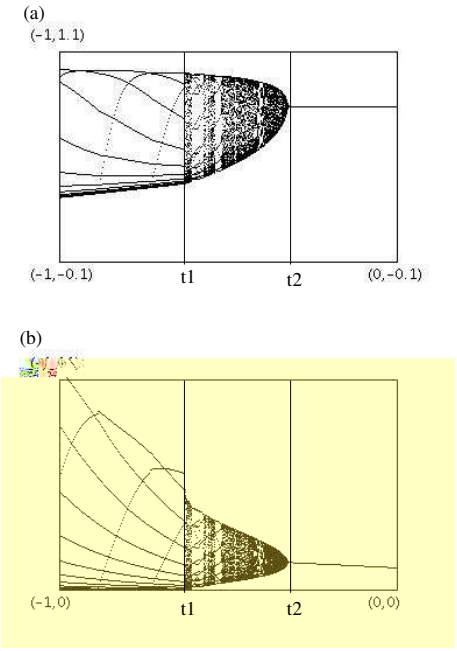

Figure 13: Output and receptor diagrams of an excitatory ($\mathrm{sign}(\eta_i) = 1$) self–coupled neuron with $a_i^{\circledast} = +a^{\circledast}$, $\beta = 0.4$, $\gamma = 0.01$. Variation of θ_i. The quasi–periodic or chaotic attractor changes quality at t_1 because ξ_i is forcibly maintained positive

The presented findings showed that a neuron that receives external inputs as variation of the bias term θ_i define a robust homeostat, with a homeostatic interval that is little influenced by slight parameter changes. Also, within the homeostatic interval, receptors do not grow without bounds, but linearly to

the environmental changes. Although in the variant where input is induced via synapses, homeostasis was also achieved, the practically infinite length of the homeostasic interval is not desirable. As described in section 2.4, the conditions under which a homeostat can regulate ought to be limited.

The next section will investigate how the homeostatic neurons behave if they are coupled. There will be no further analysis of neurons with external input induced via synapses. The explosive growth upon approaching $I_i^{syn} = 0$ and the unlimited capacity to regulate inputs made that variant less interesting for dynamical analysis. An investigation of the interaction of these two variants of inducing inputs would be interesting, but would exceed the scope of this thesis.

4.5 A Network of Two Homeostatic Neurons

When coupling two neurons n_1 and n_2, a six–dimensional dynamical system $< a_1, a_2, \xi_1, \xi_2, \eta_1, \eta_2 >$ yields. The networks investigated here will always be mutually coupled, i.e. they will have $c_{12} = c_{21} = 1$, but the recurrent connections will be varied, i.e. $c_{11}, c_{22} \epsilon \{0, 1\}$. The networks are assumed to have an identical homeostatic activation level $a_1^{\circledast} = a_2^{\circledast}$. β and γ will also be fixed within a network.

It is hoped to find parameter domains in which the whole network achieves homeostasis. First, in section 4.5.1 the case of two neuron networks where both of the neurons are excitatory or inhibitory will be discussed. Section 4.5.2 investigates the set–up in which an excitatory neuron is coupled to an inhibitory neuron.

4.5.1 A Network of Two Neurons of Equal Sign

In two neuron networks with $\text{sign}(\eta_1) = \text{sign}(\eta_2)$, the synaptic input ($I_i^{syn}$) has a fixed sign in n_1 and n_2, which makes analysis more relaxed, because synaptic inputs will never cross $I_i^{syn} = 0$. Probably, they will not even get close to that point. $I_i^{syn} \approx 0$ has turned out to be a critical value in section

4.3, because the homeostatic receptor levels $\xi_i^{\circledast}$ explode if they approach the natural limit of homeostasis at $I_i^{syn} = 0$.

A "smooth" loss of regulatory capacity is expected to occur if θ_i enters domains where $\text{sign}(I_i^{syn}) = -\text{sign}(a_i^{\circledast} - \theta_i)$, i.e. domains where $\xi_i^{\circledast}$ had to be negative in order to promote homeostasis. As already seen, neurons tend to maintain $\xi_i = 0$ in these intervals. The respectively other neuron n_j, which will, in these cases, receive a synaptic input of $\eta_i^*(\theta_i) \cdot \sigma(\theta_i)$, where $\eta_i^*(\theta_i)$ can be computed with equation (10), is expected to behave accordingly (compare sections 4.3 and 4.4).

The natural loss of regulatory capacity occurs if $\theta_i > a_i^{\circledast}$ in an excitatory network and if $\theta_i < a_i^{\circledast}$ in an inhibitory network (compare section 3.1 and equation (9)). Thus, if the (θ_1, θ_2)–space is divided with axes $\theta_1 = a_1^{\circledast}$ and $\theta_2 = a_2^{\circledast}$, four quadrants will be obtained: one in which both neurons are expected to have $\xi_i^* = 0$, two in which either one of the neurons is expected to have $\xi_i^* = 0$ and one in which both neurons are in a domain in which they can regulate in principle. This quadrant is expected to be governed by interactive dynamics and is hence most interesting.

As an advance, there will be no exceptions to that expectation. Neurons will never show any behaviour other than stationarity on $a_i^* = \theta_i$ in domains in which they cannot regulate, because $\text{sign}(I_i^{syn}) = -\text{sign}(a_i^{\circledast} - \theta_i)$ (compare equation (9)). Hence, there is no homeostatic stability found in quadrants other than the quadrant labelled "interesting" above. This regularity accounts for both, two neuron ring networks and networks in which self–coupling is involved. Therefore, findings will only be explained with respect to the "relevant" quadrant, in which homeostasis of the network is possible. Expectations about homeostatic intervals in that quadrant can be derived from the findings on hard–wired neurons presented in section 4.2.

First, the case of an even ring of two neurons, i.e. a network in which the two neurons are mutually coupled ($c_{12} = c_{21} = 1$), but not self–coupled ($c_{11} = c_{22} = 0$) will be treated. Figure 14 shows the isoperiodic plot of an

inhibitory two neuron ring with $a^{\circledast}_{1,2} = -a^{\circledast}$, with reference axes painted at $\theta_1 = a^{\circledast}_1$ and $\theta_2 = a^{\circledast}_2$.

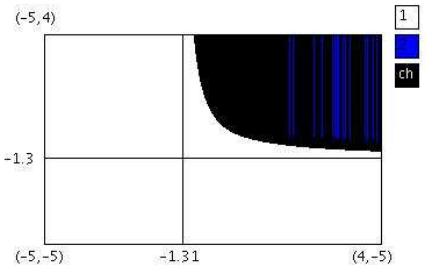

Figure 14: Isoperiodic plot of an inhibitory ($\mathrm{sign}(\eta_{1,2}) = -1$) two ring ($c_{11} = c_{22} = 0$) network. Variation of θ_1 (horizontal) and θ_2 (vertical). $a^{\circledast}_{1,2} = -a^{\circledast}$, $\beta = \gamma = 0.01$.

Homeostasis of the whole network is achieved in the white area in this relevant quadrant (top right) in figure 14. This homeostatic area is delimited by a hyperbolically shaped region of non–trivial dynamics, i.e. higher period, quasi–periodic or chaotic oscillation. The blue stripes allude coexistence of a period two orbit. Hard–wired even two neuron rings have no non–trivial dynamics other than period two–oscillation, the self–regulating neuron model disagrees with its hard–wired counterpart in this concern. But it agrees with respect to the bifurcation set: The borderline of the dynamically non–trivial domain can apparently be described by means of the trace T and the determinant D of the Jacobian matrix of the linearised network, just as in hard–wired networks (compare section 4.2): In an even ring network, the trace $T = 0$ (according to equation (12)) and the determinant $D = -\xi_1 \cdot \xi_2 \cdot \eta_1 \cdot \eta_2 \cdot \sigma'(a_1(t))\sigma'(a_2(t)) < 0$ (according to equation (13)). Therefore, stability is expected to cease at $D < -1$ (compare figure 5). During homeostasis, this condition solely depends on the inputs θ_1 and θ_2, since synaptic inputs are fixed as $I_i^{syn} = \eta^{\circledast} \cdot \sigma(a^{\circledast}_{1,2})$ for both n_1 and n_2, because of the stationarity on equal fixed points. From equations (9) and (13) and the dependency $\frac{\sigma'(x)}{\sigma(x)} = \sigma(-x)$, the determinant of a two neuron ring can be derived as

$$D^{\circledast} = -(a^{\circledast}_{1,2} - \theta_1)(a^{\circledast}_{1,2} - \theta_2) \cdot \sigma(-a^{\circledast}_{1,2})^2 \tag{14}$$

where $\sigma(-a^{\circledast})^2 = 0.0441$ and $\sigma(+a^{\circledast})^2 = 0.6241$. The form of $D^{\circledast}$ explains the hyperbolic shape of the curve. It also predicts the confirmed observation

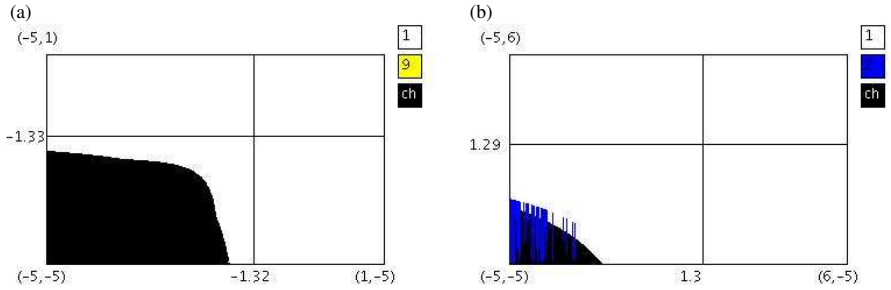

Figure 15: Isoperiodic plots for excitatory ($\mathrm{sign}(\eta_{1,2}) = 1$) two neuron ring ($c_{11} = c_{22} = 0$) networks. Variation of θ_1 (horizontal) and θ_2 (vertical). $\beta = \gamma = 0.01$. (a): $a^{\circledast}_{1,2} = -a^{\circledast}$. (b): $a^{\circledast}_{1,2} = +a^{\circledast}$.

that for $a^{\circledast}_{1,2} = +a^{\circledast}$, the homeostatic intervals are respectively much longer.

Figure 15 shows the isoperiodic plots of excitatory two neuron rings with $a^{\circledast}_{1,2} = -a^{\circledast}$ (a) and $a^{\circledast}_{1,2} = +a^{\circledast}$ (b). For excitatory neurons, the relevant quadrant is located bottom left accordingly. If $a^{\circledast}_{1,2} = -a^{\circledast}$, the bifurcation set can only partially be described by means of the conditions for stationarity in the (D, T)–space. The boundary curve follows in parts the hyperbola derived from this condition, but it has a "lump" (compare figure 15 (a)) that makes the homeostatic domain smaller than predicted.

If $a^{\circledast}_{1,2} = +a^{\circledast}$, there is again coexistence (indicated by the stripes) of a period two and a quasi–periodic or chaotic attractor. The corresponding bifurcation diagrams mostly showed hystereses of a period two attractor and a sequence of attractors (homeostatic stationarity followed by a quasi–periodic or chaotic attractor). Bifurcations delimiting the homeostatic domain occurred with $D^{\circledast} > -1$ for variation of θ_1 in both directions. Stationarity in excitatory two neuron rings was hence lost earlier than expected from the condition of stationarity in the (D,T)–space for hard–wired neurons. The quasi–periodic or chaotic oscillation of the networks is another such disagreement.

Figure 16 displays the isoperiodic plots of an inhibitory network, in which

one of the neurons is self–coupled, i.e. $c_{11} = 1$. Again, homeostasis is achieved in the white area in the relevant quadrant (top right).

The conditions for non–trivial dynamics in this set–up get a bit more complicated, since for a network with one self–coupling, neither the trace $T = 0$ nor the determinant $D = 0$ (compare equations (12) and (13)). Neural outputs are fixed during homeostasis and $a_1^{\circledast} = a_2^{\circledast}$, therefore the synaptic input to n_1 during homeostasis is given by $I_1^{syn} = 2 \cdot \eta^{\circledast} \cdot \sigma(a_{1,2}^{\circledast})$, while the input to n_2 yields $I_2^{syn} = \eta^{\circledast} \cdot \sigma(a_{1,2}^{\circledast})$. From equations (12), (13) and (9) and the fact that $\frac{\sigma'(x)}{\sigma(x)} = \sigma(-x)$, the trace T and the determinant D of such a network during homeostasis can then be derived as

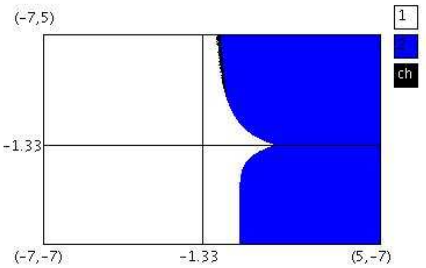

Figure 16: Isoperiodic plot of an inhibitory ($\text{sign}(\eta_{1,2}) = -1$) two neuron network with $c_{11} = 1$, $c_{22} = 0$. Variation of θ_1 (horizontal) and θ_2 (vertical). $a_{1,2}^{\circledast} = -a^{\circledast}$, $\beta = \gamma = 0.01$.

$$T^{\circledast} = \frac{1}{2}(a_{1,2}^{\circledast} - \theta_1) \cdot \sigma(-a_{1,2}^{\circledast}) \tag{15}$$

$$D^{\circledast} = -\frac{1}{2}(a_{1,2}^{\circledast} - \theta_1) \cdot (a_{1,2}^{\circledast} - \theta_2) \cdot \sigma(-a_{1,2}^{\circledast})^2 \tag{16}$$

The conditions for non–trivial dynamics are $T + D = -1$ for inhibitory neurons and $T - D = 1$ for excitatory neurons (compare section 4.2, figure 5). Hence, an overall condition to be met would be $|T| - D = 1$. The bifurcation set in the relevant quadrant for the inhibitory network can apparently be described by means of this condition, bifurcation to period two oscillation occurs. In an excitatory two neuron network with a single self–coupling, stationarity again was lost earlier than expected.

In an inhibitory network with full connectivity (i.e. $c_{ij} = 1 \quad \forall i, j \epsilon \{1, 2\}$), the dynamics in the interesting quadrant are bounded by a bifurcation set that yields a straight line in the (θ_1, θ_2)–space (compare figure 17), which is

due to the fact that $D = 0$ (compare section 4.2). Hence, the condition for leaving the stationary area depicted in figure 5 solely depend on the trace T. In a two neuron network with $\text{sign}(\eta_1) = \text{sign}(\eta_2)$ and full connectivity, the synaptic input is equal for both neurons upon homeostasis, it yields $I^{syn}_{1,2} = 2 \cdot \eta^{\circledast} \cdot \sigma(a^{\circledast}_{1,2})$. The trace T is given by

$$T^{\circledast} = (a^{\circledast}_{1,2} - \frac{\theta_1}{2} - \frac{\theta_2}{2}) \cdot \sigma(-a^{\circledast}_{1,2}) \tag{17}$$

Non–trivial dynamics can be expected if $|T| > 1$. Again, in the inhibitory network, this condition appears to be met, while in corresponding excitatory networks, it is not, oscillation starts earlier. Investigation of the hysteresis effect, which could again be observed in bifurcation diagrams of the excitatory network, showed that the later bifurcation point was located where it was expected from the condition.

A special remark on excitatory networks has to be made. These networks oftentimes have quasi–periodic or chaotic attractors that exceed the threshold at $\xi_i = 0$, even for very small β, e.g. $\beta = 0.01$. This drawback further reinforces the aim to modify the receptor equation in future trials (see section 5.1).

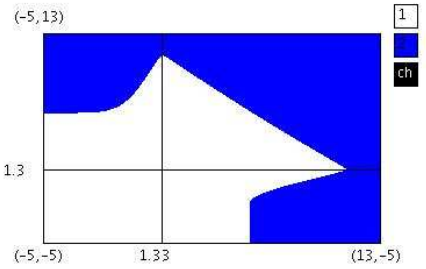

Figure 17: Isoperiodic plot of an inhibitory ($\text{sign}(\eta_{1,2}) = -1$) two neuron network with $c_{11} = c_{22} = 1$. Variation of θ_1 (horizontal) and θ_2 (vertical). $a^{\circledast}_{1,2} = +a^{\circledast}$, $\beta = \gamma = 0.01$.

The investigation of networks with $\text{sign}(\eta_1) = \text{sign}(\eta_2)$ in general agrees with the findings on single neurons (section 4.4) and complies roughly with the findings on networks with fixed synaptic weights (section 4.2). Taking the fixed points as reference axes in the (θ_1, θ_2)–space, there is just one area in which homeostasis of a two neuron network can be achieved and actually is achieved within a finite domain of parameter settings.

4.5.2 A Network of an Excitatory and an Inhibitory Neuron

Networks with neurons of different sign(η_i) are expected to behave more "awkward" than those in which both of the neurons produce output of the same sign. If there are neurons with $c_{ii} = 1$ in such a network, homeostasis is in principle impossible in the strict set–up underlying the present experiments, which will be discussed below. The section will begin with the easy case, i.e. with the odd two neuron ring network.

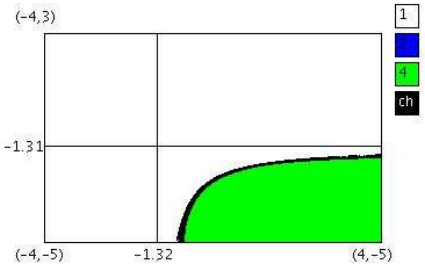

Figure 18: Isoperiodic plot of a two neuron ring ($c_{11} = c_{22} = 0$) network with sign(η_1) = 1 and sign(η_2) = -1. Variation of θ_1 (horizontal) and θ_2 (vertical). $a^{\circledast}_{1,2} = -a^{\circledast}$, $\beta = \gamma = 0.01$.

Figure 18 shows the isoperiodic plot of an odd two neuron ring. In this case, I_i^{syn} has fixed (though different) signs in both n_1 and n_2 as before, since each neuron receives synaptic input from a single synapse. Homeostasis is achieved in the white area in the relevant quadrant (bottom right). Since in ring networks, $T^{\circledast} = 0$ (compare equation (12)), the conditions for non–stationarity of dynamics depend on $D^{\circledast}$ solely, which can be computed with equation (14) that gives the general determinant for ring networks. It is expected that stationarity is lost at $D^{\circledast} = 1$, and actually, stationarity appears to be lost exactly when meeting that condition.

On the first glance, the black rim of the period four attractor looks as if it was composed of long transients that would settle upon further iteration of the system. Graphical analysis of the bifurcation diagram (see figure 19 (a)) could not resolve this question. Examination of how an exemplary orbit (see figure 19, (b)) evolves, suggests that the system actually has a chaotic attractor there. The orbit remains close to periodic orbits, but it is always repelled after a certain time span, because the respective periodic orbits are

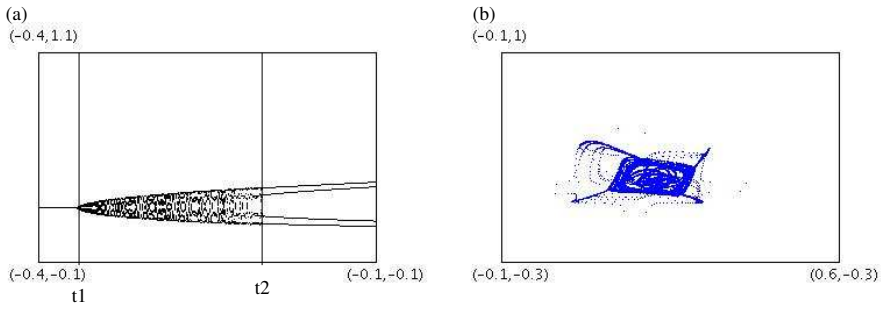

Figure 19: Chaotic attractor of a ring ($c_{11} = c_{22} = 0$) network with $\text{sign}(\eta_1) = 1$ and $\text{sign}(\eta_2) = -1$. $a^{\circledast}_{1,2} = -a^{\circledast}$, $\gamma = \beta = 0.01$, $\theta_2 = -3$ (a) Bifurcation diagram of $\sigma(a_1)$. Variation of θ_1 (b) A corresponding orbit in the $(\sigma(a_1), \sigma(a_2))$–space (plot started after 5000 convergence iterations), with $\theta_1 = -0.3$

not stable. Such behaviour is typical for chaotic orbits. The period four oscillation that follows the chaotic attractor is again an agreement with the findings on hard–wired two neuron rings.

More complicated get things if self–couplings are added to a network with an inhibitory and an excitatory neuron. Taking into account the fixed absolute value of $\eta_i^{\circledast}$ and the fixed output rate during homeostasis, it becomes obvious that homeostasis in a two neuron network is in principle impossible, if $\text{sign}(\eta_1) = -\text{sign}(\eta_2)$ and one of the neurons is self–coupled: The synaptic input in a self–coupled neuron during homeostasis would yield $I_i^{syn} = (-|\eta_i^{\circledast}| + |\eta_i^{\circledast}|) \cdot \sigma(a_{1,2}^{\circledast})$ (remember: $a_1^{\circledast} = a_2^{\circledast}$), which corresponds to $I_i^{syn} = 0$. Without synaptic input, there is no regulation possible. Furthermore, since both neurons aim at maintaining homeostasis it can be anticipated that inputs to the self–coupled neuron(s) will be close to $I_i^{syn} = 0$. Therefore, dynamics cannot be predicted to develop in a controlled way, because receptors explode to one side of that point and drop to $\xi_i = 0$ on the other side (compare section 4.3).

Figure 20 shows two isoperiodic plots for the respective cases ((a): n_1 is self–coupled, (b): both neurons are self–coupled). The white areas depict domains in which one or both of the neurons have $\xi_i^* = 0$. It has to be added

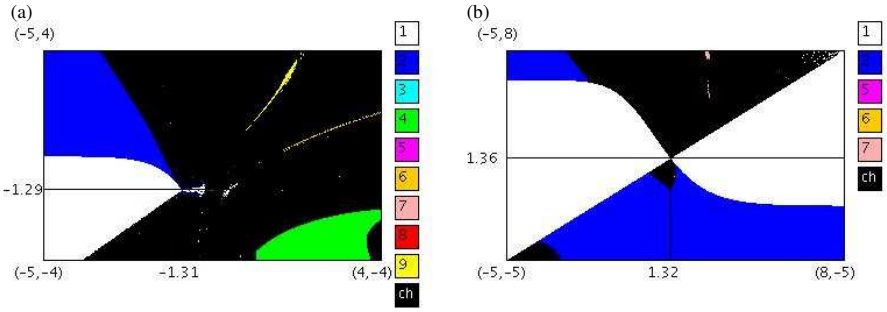

Figure 20: Isoperiodic plots for a two neuron network with $\text{sign}(\eta_1) = 1$ and $\text{sign}(\eta_2) = -1$. Variation of θ_1 (horizontal) and θ_2 (vertical). $\beta = \gamma = 0.01$. (a): $a^{\circledast}_{1,2} = -a^{\circledast}$, $c_{11} = 0$, $c_{22} = 1$ (b): $a^{\circledast}_{1,2} = +a^{\circledast}$, $c_{11} = c_{22} = 1$

that for some conditions, the neurons operate at an average activation level close to $a^{\circledast}_i$, but ever in a non–stationary fashion.

But before judging these finding, it should be considered that homeostasis could again be achieved in a three neuron network with one inhibitory and two excitatory neurons, which is a network containing neurons with different $\text{sign}(\eta_i)$ as well. Also, if $a^{\circledast}_1 = -a^{\circledast}_2$ homeostasis could be achieved for some parameter settings in a two neuron network with different $\text{sign}(\eta_i)$ and self–couplings, independent of which of the neurons takes which of the fixed points. Finally, if a self–coupled neuron receives a small extra synaptic input, the network could stabilise on the desired state $x^{\circledast}$ for some parameter settings, without explosion of ξ_i. It would be interesting to further investigate these cases, but there will be no such analysis performed here, since this would exceed the scope of the work.

4.6 Summary of the Results

The analysis lead to some important generalisations and characterisations about how and to what extend the investigated neuron model is homeostatic.

In the investigation of the single neuron, it could be shown that the aim to settle in a stationary state $x^{\circledast}_i$, in which activation maintains stable at a

target level $a_i^{\circledast}$, could be achieved for a reasonable domain in the parameter space.

Also, in two neuron ring–networks, the homeostatic capacity of the single neurons was preserved, a homeostatic domain could be detected and characterised. In two neuron networks, where at least one of the neurons was coupled to itself, homeostasis could only be achieved if $\text{sign}(\eta_1) = \text{sign}(\eta_2)$.

Concerning the limits of homeostatic domains, the networks were observed to roughly comply with the stationarity criteria that account for hard–wired neural networks. The homeostatic domains lie within the fixed point domain in the (D, T)–space of the Jacobian matrix of the linearisation of the system, depicted in figure 5. The homeostatic domains could be approximately characterised with respect to the system parameters θ_i and $a_i^{\circledast}$. For different structural settings, different conditions for stationarity could be derived, which are reflected in the different shape of homeostatic domains as they are displayed in the isoperiodic plots in the (θ_1, θ_2)–space.

Changes in the learning parameters β and γ were generally not relevant for the qualitative outcome, if not chosen extremely high or low. In excitatory networks, loss of homeostasis frequently resulted in settlement in a quasi–periodic or chaotic attractor that would have dropped below $\xi_i = 0$, if not impeded. In these cases β had to be chosen sensitively and possibly very small.

Inhibitory networks followed the expectations derived from findings on hard–wired neural networks closely. Bifurcations seemingly occurred at the exact limits predicted. They oscillated at period two, if there was self–coupling in the network. Excitatory networks dominantly exhibited chaotic or quasi–periodic oscillation and generally left the homeostatic interval earlier as expected from the computed conditions for stationarity. Frequently, hysteresis of the homeostatic fixed point attractor and a non–trivial attractor could be observed in excitatory networks.

Odd ring networks complied the conditions for stationarity computed

and oscillated at period 4, in agreement with their hard–wired pendants. Still, the transition from stationarity to periodic oscillation involved a small chaotic domain.

In two neuron networks with $\text{sign}(\eta_1) = -\text{sign}(\eta_2)$ and self–coupling, homeostasis could not be achieved. This incapacity is due to the fact that equal target activation levels of the two neurons imply an equal magnitude of inhibitory and excitatory outputs during homeostasis that cancel out each other in neurons that are innervated by both, the other neuron and themselves. Asymptotic behaviour of such networks was diverse. If the strict experimental restrictions are softened, (e.g. by allowance of more neurons, external synaptic stimulation or different target activation levels), even in such networks, homeostasis is possible.

The investigation of single neuron networks included a juxtaposition of synaptic and direct induction of external input. The receptor levels ξ_i that realise homeostatic adaptation were observed to grow linearly with θ_i and hyperbolically with o_0. This observation lead to a neglection of the latter variant in the investigation of two neuron networks, because the hyperbolic dependency lead to an explosion of receptor levels.

5 Conclusion

This section is dedicated to the evaluation of the findings presented in the previous section, with respect to how they could form a baseplate, on which to build up a theory of learning. Section 5.1 is devoted to a discussion of how and why the proposed neuron model came short and how it could be extended and improved. What the findings obtained in the dynamical analysis add up to in the context of the adopted approach to learning (see section 2.3), will be discussed in section 5.2.

5.1 Discussion of the Theoretical Model

The dynamical analysis of the homeostatic neuron model lead to a variety of ideas and insights about its qualities, its drawbacks and about possible extensions. In general, it could be shown that the defined dynamical system achieves homeostasis under diverse conditions. Still, some shortcomings were encountered during the investigation, some of them more drastic than others. Critical issues to pay attention to in future experiments can be educed from these experiences. Some of the most important points and possible extensions of the proposed model will be discussed in this section.

Perhaps the most severe drawback of the dynamical system proposed is the fact that the receptor level in some situations would drop below $\xi_i = 0$, if it was not forcibly detained. Having detected this phenomenon in the investigation of a single neuron, it was first not considered relevant, which turned out to be a mistake later, in the analysis of networks of excitatory neurons. A simple manoeuvre to fix this shortcoming is to use the difference in the output rates $(\sigma(a_i^{\circledast}) - \sigma(a_i(t)))$ instead of the difference in activation levels $(a_i^{\circledast} - a_i(t))$. With this move, the maximal negative change of receptor levels would be $\Delta\xi_i(t) = \beta \cdot \xi_i(t) \cdot (\sigma(a_i^{\circledast}) - 1)$, which is smaller than $\xi_i(t)$, if $\beta < 1$.

Another weak point is the possibly unbounded growth of ξ_i, which became evident if the neural inputs were very close to $I_i^{syn} = 0$. Bounding of the

receptor level could e.g. be performed by using the sigmoidal (or multiples m) of the receptor level in the computation of synaptic weight, i.e. $w_{ij}(t) = \eta_j \cdot m \cdot \sigma(\xi_i)$. Implementing a saturation level for the receptor equation is a sensitive issue, because $\eta_i^{\circledast}$ is already fixed, depending on $a_i^{\circledast}$ (compare equation (10)). If $\xi_i^{\circledast}$ had a fixed limit, too, the absolute values of synaptic strengths during homeostasis would be strictly bounded. Such bounds would principally cut off large parts of the parameter space.

The potential unboundedness of the transmitter level η_i did not cause any problems, the estimate that converged transmitter levels would rarely leave the interval given by equation (10) was right.

Another unfavourable effect, which was rather a shortcoming of the network model than of the neuron model, is that odd rings with recurrent connections do not achieve to regulate at all. The prospects on how slight changes in the set–up (more neurons, different fixed points, low external synaptic inputs), make incapacity to regulate disappear, allude that this shortcoming can be attributed to the laboratory conditions, in which the model was tested. Still, it cannot be assumed that situations in which there is a synaptic input of $I_i^{syn} \approx 0$ do not occur if a neuron receives excitatory and inhibitory inputs, and the consequent explosion of receptor levels is, as already mentioned, a severe drawback.

Some general considerations concern the principal idea to employ a fixed receptor potential for all incoming synapses of a neuron. Individual learning of receptor levels at each synapse is unfavourable, because computational complexity of the anyway complex network model would explode. Since neurons achieve to regulate in large parameter domains, it does on the first glance not even appear necessary. But analysis in this work was restricted to neurons that receive either excitatory or inhibitory input, apart from the set–up in which an a network contained reflexive couplings and had $\text{sign}(\eta_1) = -\text{sign}(\eta_2)$, which turned out to be problematic. Although the prospect on achievement of homeostasis in a three neuron network with different $\text{sign}(\eta_i)$

anticipates that competitive inputs do not generally impede homeostasis, it could be thought of implementing a compromise, inspired by observations of biological brains. There are two principal chemical neurotransmitters employed in synaptic signal transduction, which are glutamate in excitatory and GABA in inhibitory synapses. These transmitters bind to different types of receptors (glutamate receptors and GABA receptors respectively) (compare [8], chapter 12, pp. 207-228, on Synaptic Integration)[5]. Hence, the obvious proceeding would be to determine the change in receptor density of GABA-ergic and glutminergic receptors independently. Such a model promises relief for several of the encountered problems: Changes in sign of the overall input would not lead to explosion of receptor levels anymore. Also, the observed phenomenon that inputs cancel each other in an networks with different sign(η_i) would no more be a matter.

It remains questionable, whether such a network would be too powerful, because neurons that receive inputs of both types would not have domains in which regulation is in principle impossible anymore. It can be anticipated that in such a set–up, one of the receptor levels would always be at $\xi_i = 0$, the neuron would pick only the input of the desired type to enter the cell. Still, neurons could exhibit non–trivial dynamics, probably, if the stationarity constraints outlined in this thesis are violated. Such neurons would at least not be universally homeostatic.

Another consideration refers back to section 2.4, in which homeostasis was discussed: It is assumed that the activation level not as such as an essential variable, i.e. loss of homeostasis does not imply cell death. This is a reasonable decision insofar as if a network only operated homeostatically, it would output at a fixed rate, which would not allow variation of behaviour (compare section 5.2). But still, it could be thought about defining circumstances that are considered as mortal, i.e. if a neuron enters them, it is

[5]This scenario is, of course, just the prototypical one: there are more neurotransmitters, even some that can bind to different kinds of receptors, etc., but, as mentioned before, the network model is a strong idealisation.

excluded from network computation.

In spite of the mentioned problems and restrictions, the neuron model met well with the requirements specified for a homeostatic neuron. For a contiguous domain within the parameter space, robust maintenance of homeostasis could be observed. Even if the proposed changes would be realised, it can be assumed that the principle findings would be preserved, i.e. the analysed criteria for homeostasis would probably still account, if the changes are respected in the computation. The concrete homeostatic domains would, of course, be modified.

A last theoretical remark has to be made with respect to the variants of synaptic or direct inputs. This difference as such is only a computational one, how it is interpreted is up to the reader. In the input synapse case, interpretation is straight forward, but direct modification of the activation level could be imagined in different ways. The association alluded by the term "direct input" is probably an electrode applied to the cell body. But other possible interpretations are e.g. innervation by synapses that carry a neurotransmitter which binds to receptors of a fixed density, or direct stimulation of specific sensory neurons.

5.2 Prospect: Situated Homeostatic Neural Networks

The proposed model — or a corresponding improved model of homeostatic adaptation in neurons — is meant to realise adaptivity in situated agents. Thus, we will flip the coin and have a short prospect on how the findings obtained could relate to behavioural issues, if a network of homeostatic neurons is integrated in a sensorimotor loop.

The first question to address is how such an integration would look like. As it is common for neural network applications, sensory and effectory sub–systems would have to be specified, i.e. it had to be determined, how the system affects and is affected by the environment. It is hoped that in a clever structural set–up, the implemented desire of the neurons to be homeostatic

would lead to an adaptive behaviour. Further, it is hoped that evolutionary algorithms (compare section 2.3) would be able to generate such a clever set–up. The long term vision of how adaptive agents are developed is evolution of network structures (number, connectivity and parametrisation of neurons), in which synaptic strengths are learned by means of homeostatic self–regulation.

In principle, it has to be considered thoroughly how this is imagined to work: If neurons were universally able to maintain homeostasis, they would always output at a fixed rate, independent of the input. Thus, there *has* to be the possibility to distort homeostasis, in order to yield alteration of the system's behaviour. The proposed model leaves space for such distortion in at least three different ways:

1. Since loss of homeostatic activation is not considered to be mortal, neurons could be stimulated in a way that directs them in non–homeostatic domains, to achieve an alteration of their behaviour.

2. The assumption that the environment changes on a much slower time scale cannot be expected to hold true in general. Neural dynamics in a driven system would probably be *transient dynamics*.

3. A mechanism that modifies the target activation levels could be established.

Naïvely imagined, all three possibilities could lead to adaptive behaviour. Concerning point 1., the fact that non–trivial dynamics occurs in biological brains alludes that not only stationary dynamics can play a functional role in the determination of behaviour. Periodic or chaotic oscillation of neurons might be desirable in some situations.

With respect to point 2., the naïve vision is that hostile environmental factors move the activation level in a neuron such that behaviour is altered before homeostasis can be re–achieved. Still, long term changes, such as slow changes in illumination, due to wheather or daytime, could be adapted to

homeostatically. Situated homeostatic networks are not desired to actually achieve homeostasis in all possible situations, fast processes are expected to move the activation level. But the homeostatic mechanism is supposed to maintain the activation value *close* to the target activation, on the long run.

A mechanism to reset target activation levels, as described in point 3., could be implemented as hormonal control, following own dynamics: a switch between the two different fixed points could e.g. be interpreted as wake and sleep mode that are alternated in a situated agent.

For the first two options, it would be important to choose parameters such that homeostasis is not too robust, i.e. to impede that homeostatic domains are very large, or to locate the neuron close to a bifurcation set at rest.

There are many more models imaginable, e.g. mixing homeostatic and non–homeostatic neurons, but the evidence on homeostatic regulation in biological neurons leaves space for the hope that such complication of the model is not necessary. There are difficulties connected with the neuron model, because it is strictly constrained with respect to the domains, the synaptic weight matrix can enter: Dale's rule already imposes some contraints, the interdependencies of synaptic weiths due to fixed receptor and transmitter levels within a single neuron restrict possible synaptic weight matrices even more. It remains to be investigated, how networks of homeostatic neurons perform in driven agents. It is hard to predict the dynamic behaviour in driven networks that will probably be much larger than the investigated ones and in which external and net–internal inputs will interact and change in a much less controlled way.

But still, with homeostasis, there is a mechanism found that adapts synapses locally, according to a principle that do not rely on any presupposition about external factors. The homeostatic neuron has proven to stabilise and maintain neural dynamics in a fixed point attractor, robustly and in a large part of the parameter domain. Nevertheless it allows versatile dy-

namics within the network, including oscillation and hystereses, that could be employed to cause sequences of output signals or context sensitivity of behaviour. The different attractors have limited and defined domains within the parameter space. These properties define a rich tool kit on which an evolutionary algorithms could draw. I personally would be really surprised if homeostatic networks models — even in this preliminary form — could not be set–up with the help of artificial evolution to fulfil simple tasks.

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Hereby I confirm that I wrote this thesis independently and that I have not made use of any other resources or means than those indicated.

Hiermit bestätige ich, dass ich die vorligende Arbeit selbständig verfasst und keine anderen als die angegebenen Quellen und Hilfsmittel verwendet habe.

Bonn, December 18, 2003