
Special Theme:
Neuroinformatics!
CNS
2009
Reseach Results of the
BCCN Freiburg
presented at:
bccn
Freiburg
Eighteenth Annual Computational Neuroscience Meeting
July 18th - 23rd 2009, Berlin, Germany
Hilton Hotel and Berlin-Brandenburg
Academy of Sciences, Gendarmenmarkt
C-NS 09
Organization for Computational Neurosciences, Inc. http://www.cnsorg.org

WORKSHOP TALKS AND ORGANIZATIONS

Symposium on Neuroinformatics - Data Sharing and Data Analysis in Neurophysiology

organized by: Andreas Herz, **Martin Nawrot**, & Thomas Wachtler

Workshop: Python in Neuroscience

organized by: Eilif Muller, **Jens Kremkow**, Andrew Davison, Romain Brette

Experimental data from dissociated cortical cell cultures on self-organized criticality

Samora Okujeni

Talk in Workshop: Activity-Dependent Structural Plasticity – from cell cultures to cortical networks (Butz & Van Ooyen)

Structural plasticity controlled by calcium based correlation detection

Moritz Helias, Stefan Rotter

Talk in Workshop: Activity-Dependent Structural Plasticity – from cell cultures to cortical networks (Butz & Van Ooyen)

Variability and co-variability of spiking activity in cortical networks

Stefan Rotter

Talk in Workshop: Modern Mathematical Neurodynamics: Bridging Single Cells to Networks (Timme)

Higher-Order Correlations in Large Neuronal Populations

Benjamin Staude, Sonja Grün and Stefan Rotter

Talk in Workshop: Methods of Information Theory in Computational Neuroscience (Lazar & Dimitrov)

Intermediate-range projections in non-columnar circuits of rat neocortex

Clemens Boucsein

Talk in Workshop: Cortical Microcircuit Models of Information Processing and Plasticity (Cutsuridis & Wennekers)

Impaired structural plasticity increases connectivity in developing cortical networks

Samora Okujeni, Steffen Kandler, Oliver Weihberger, Ulrich Egert

The principle of self-organization is fundamental for the adaptive formation and modification of functional circuits in many parts of the nervous system. At a cellular level, cortical micro-circuitry evolves on the basis of activity-dependent biochemical processes that guide neuronal wiring and that are differentially regulated in the course of development.

Protein kinase C (PKC) plays a key-role in this morphological differentiation of neurons, since it cross-links many biochemical pathways involved in structural regulation and targets many cytoskeletal proteins directly. In a simplified model, activation of PKC via metabotropic glutamate receptor downstream signaling phosphorylates and mobilizes cytoskeletal proteins and thereby promotes structural plasticity [1]. Antagonistic pathways that involve NMDA receptor mediated activation of protein phosphatases in turn promote cytoskeletal assembly and stabilization.

We explore this concept of structural homeostasis in dissociated cortical cell cultures developing on micro-electrode arrays. These generic random networks display a self-regulated maturation process with similar phases as the developing cortex. Within this period of network formation we interfered with the structural homeostasis by inhibiting PKC activity. Previous studies showed that inhibition of PKC activity in cerebellar slice cultures promotes dendritic outgrowth and arborization in Purkinje cells [2] and that climbing fiber pruning is impaired in PKC deficient mice [3]. Further in vitro data demonstrate the importance of PKC activity for the experience-dependent modulation of synaptic weights on the basis of AMPA receptor trafficking [4], suggesting reduced synaptic plasticity under PKC inhibition.

To assess possible functional consequences of these dependencies, we chronically inhibited PKC activity in cortical cell cultures and compared network activity and connectivity characteristics. Applying new morphometrics, we found significantly increased arborization and extent of dendrites as well as increased synapse density, indicating increased connectivity in these networks. Further, we observed reduced neuronal clustering suggesting impaired cell migration. These findings indicate changes in the connectivity statistics in networks developing under inhibited PKC activity that fit to the assumed homeostatic model of structural regulation.

Irrespective of PKC inhibition, spike activity remained organized in network-wide bursting that characteristically emerges in cortical cell cultures. Bursts were, however, more synchronized across the recording area and contained more spikes, suggesting a faster propagation of activity through the networks and longer reverberations due to increased connectivity. Differences in the spatio-temporal spread of activity at different stages of development further indicate a more homogeneous topology under PKC inhibition. This could be the result of a stronger conservation of the highly connected and unclustered immature network structure.

In summary, consistence between our morphological and electrophysiological data support the idea that coordinated regulation of PKC activity is required for a proper formation of functional pathways in early network development.

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Supported by the German Federal Ministry of Education and Research (BMBF grant 01GQ0420), and the EU (NEURO, 12788)

A Model of Free Monkey Scribbling Based on the Propagation of Cell Assembly Activity

Alexander Hanuschkin, J. Michael Herrmann, Abigail Morrison, Markus Diesmann

Recent findings on a free scribbling task in monkeys [1] reveal that planar trajectories can be decomposed into elementary movements. Typical patterns in the experimental data are parabolic segments that correspond to piecewise constant acceleration of the end effector.

We present a biologically plausible spiking neuronal network model of free monkey scribbling that maps accelerations to positions via a representation of velocities, where the unperturbed propagation of synchronous activity represents a parabolic segment. The control architecture can be visualized as a graph in velocity space: vertices correspond to constant velocities and are connected by edges that correspond to constant accelerations. The edges are formed by synfire chains (SFCs) [2] that transform the velocity of the trajectory linearly between the start and end vertex, i.e. local neuronal groups code a velocity that depends on its position along the edge. A trajectory is generated by integrating the population vector of all neurons. The neurons in the network are driven by independent Poisson input such that in the absence of synfire activity, an asynchronous irregular dynamic state is maintained. Activity in the final group of a SFC can activate the first group of any SFC whose start vertex coincides with the end vertex of the previously active chain, thus changing the direction of acceleration. Reliable switching to select one of several candidate SFCs with the same start vertex is achieved by mutual inhibition among the SFCs. The scribbling activity is sustained by a background network of highly recurrent backward and forward connected chains (BFCs). Self-ignition from the background activity initiates synfire activity in the model. The activity of the BFCs is suppressed during active scribbling.

The assumptions of the model are derived from a perceptual, rather than muscular, control approach. The model provides an explanation for the segmentation of the trajectory [1] and guarantees on-going parabolic movement trajectories with smooth transitions and an endeffector dynamics that obeys the experimentally observed two-thirds power law [3].

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Supported by the German Federal Ministry of Education and Research (BMBF grants 01GQ0420 and 01GQ0432), the EU (FACETS, 15879), DIP F1.2. All simulations are performed using NEST [4].

Reservoir computing methods for functional identification of biological networks

Tayfun Gürel, Stefan Rotter, Ulrich Egert

Introduction

The complexity of biological neural networks (BNN) necessitates automated methods for investigating their stimulus-response and structure-dynamics relations. In the present work, we aim at building a functionally equivalent network to a reference BNN. The response signal of the BNN to various input streams is regarded as a characterization of its function. Therefore, we train an artificial system that imitates the input-output relation of the reference BNN under the applied stimulus range. In other words, we take a system identification approach for biological neural networks. Generic network models with fixed random connectivity, recurrent dynamics and fading memory, i.e. reservoirs, were shown to have a strong separation property on various input streams. Equipped with additional simple readout units, such systems have been successfully applied to several nonlinear modeling and engineering tasks [1].

Methods

Here we take a reservoir computing approach for functional identification of simulated random BNNs and neuronal cell cultures [2]. More specifically, we utilize an Echo State Network (ESN) of leaky integrator (non-spiking) neurons with sigmoid activation functions to identify a BNN. We propose algorithms to adapt the ESN parameters for modeling the relations between continuous input streams and multi-unit recordings in BNNs.

Selected Results

Our findings indicate that the trained ESNs can imitate the response signal of a reference biological network for several tasks. For instance, we trained an ESN to estimate the instantaneous firing rate (conditional intensity) of a randomly selected neuron in a simulated BNN. Receiver Operating Characteristic (ROC) curve analysis showed that the ESN can estimate the conditional intensity of this selected neuron (see Figure 1).

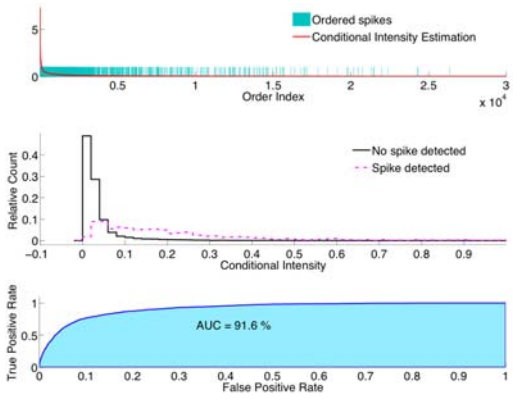

Fig. 1: Estimated conditional intensity for a selected biological neural network. Conditional intensity estimations, ƒÉ, _for all time steps in the testing period are shown in decreasing order (top). A bar is shown if there was indeed a spike observed in the corresponding time step (top). Distributions of conditional intensity for time steps with observed spikes and without spikes (middle). By a varying threshold on ƒÉ_, true positive rates vs. false positive rates can be calculated (bottom).

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Supported by the German Federal Ministry of Education and Research (BMBF grant 01GQ0420), and the EU (NEURO, 12788).

Dynamical emergence of fear and extinction cells in the amygdala – A computational model

Ioannis Vlachos, Arvind Kumar, Ad Aertsen

During a typical fear conditioning experiment a neutral stimulus is paired with a fearful one and after multiple trials the former acquires aversive properties. This learning can be suppressed by repeated presentations of the initially neutral stimulus alone (fear extinction). The major brain structure involved in these fear related processes is the amygdaloid complex, but the exact functional mechanisms are not well understood. Recently, Herry et al. (2008) described two distinct fear and extinction cell populations within the basal nucleus of the amygdala (BA) that are exclusively activated during fear conditioning and extinction respectively [1].

The aim of this project is to explain how these two neural subpopulations arise and what their causal role is in the acquisition and suppression of fear memories.

For this purpose we built a spiking neuron network model using the NEST simulator [2]. We modeled the BA based on known anatomical data as a recurrent network consisting of excitatory and inhibitory neurons. The excitatory neurons received sensory input from the adjacent lateral nucleus (LA) and contextual input from the hippocampus and in turn excited the surrounding inhibitory neurons. We propose short-term plasticity at the sensory afferents of the excitatory neurons as one of the main mechanism underlying the formation of the experimentally observed neuron subgroups within the BA. This type of plasticity is governed by contextual inputs and regulated by neuromodulators.

Endowed with this plasticity mechanism the recurrent network model is able to replicate three key experimental findings: (i) Emergence of fear cells during fear conditioning in one context; (ii) Emergence of extinction cells during extinction training in a different context; (iii) Post-extinction activation of same fear cells in the original conditioning context. These results are evidently based on the assumption that fear extinction is a new learning rather than unlearning, that is fear and extinction memories coexist and compete with each other. Herry et al. reported a distinct functional connectivity of fear and extinction cells to hippocampus and pre-frontal cortex, which suggests a rather rigid organization of those cells. In contrast, our model shows that fear and extinction cells can emerge dynamically as a result of the identical learning mechanism being applied to the population as a whole.

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Supported by the German Federal Ministry of Education and Research (BMBF grant 01GQ0420).

Control of the temporal interplay between excitation and inhibition by the statistics of visual input

Jens Kremkow, Laurent Perrinet, Cyril Monier, Yves Frégnac, Guillaume S. Masson, Ad Aertsen

In the primary visual cortex (V1), single cell responses to simple visual stimuli (drifting gratings) are usually strong but with a high trial-by-trial variability. In contrast, when exposed to full field natural scenes with simulated eye movements, the firing patterns of these neurons are sparse but highly reproducible over trials [1]. So far the mechanisms behind these two distinct different response behaviours are not yet fully understood. Different mechanisms are candidates for controlling spike timing precision and models are needed to clarify their respective contribution, which may be of thalamic or intracortical origin.

As a first step, we investigated which aspects of the neuronal dynamics can be explained by balanced feedforward excitation and inhibition and its dependency upon the spatio-temporal statistics of the different stimuli. We built a simple model of the early visual system (LGN,V1). The thalamocortical connectivity was similar to the gpush-pull h architecture used in [2], with additional depressing thalamocortical synapses [3]. The model was written in PyNN [4] using NEST[5] as simulator.

Indeed, the model can reproduce the main response characteristics of first-order thalamo-cortical neurons in layer 4 of cat V1. During drifting gratings, excitatory and inhibitory conductances of cortical neurons were anti-correlated [6,7], such that excitation can be freely integrated and induce multiple spikes. In contrast, during natural scenes the conductances were correlated, with inhibition lagging by few milliseconds [1,8]. This small lag between excitation and inhibition induces a strong selectivity to synchronous inputs, with a consequence that the responses became sparse and precise.

However, some key aspects of the in vivo recordings in cat area V1 cannot be explained, such as selective reduction of stimulus-locked subthreshold noise during natural scene viewing, precise firing during fixational eye-movements and center-surround non-linearities, opening the door for future investigation about the role of intra-cortical recurrent connectivity in further shaping the neuronal responses to natural images.

In conclusion, our study points that correlated inhibition can explain, at least in part, sparse and reliable spiking activity as observed in response to natural scenes. This is consistent with its role reported from other sensory modalities and cortical areas [8]. Thus correlated excitation and inhibition could be a general mechanism to induce sparse and precise spiking in the nervous system.

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Supported by the German Federal Ministry of Education and Research (BMBF grant 01GQ0420), the CNRS, the Agence Nationale de la Recherche (ANR - Natstats), and the EU (FACETS, 15879).

The computational role of the feedforward inhibition in the striatum network

Man Yi Yim, Arvind Kumar, Ad Aertsen

Basal ganglia are a major neural system implicated in important behavioral tasks such as action selection, reward-based learning and motor control. Striatum is the main input stage of the basal ganglia and receives massive convergent cortical afferents. Although a wealth of anatomical and electrophysiological data have been accumulated about the striatum, computational models to understand information processing in the striatum network are lacking.

To understand information processing in the striatum, we developed a spiking neuron network model of the striatum based on existing physiological and anatomical data. The network was simulated using a Python interface to NEST [1]. We considered two populations of neurons, namely, medium spiny neurons (MSNs) and fast spiking interneurons (FSIs). MSNs are the main cell type in the striatum and represent over 95% of the neuron population. The recurrent connections among MSNs provide weak and sparse feedback (FB) inhibition. The major type of GABAergic interneurons in the striatum is FSI, which projects to the MSNs extensively and forms strong feedforward (FF) inhibition.

First we studied the role of FF and FB inhibitions in shaping the dynamics of the striatum network. The FF inhibition set a brief time window for the MSNs to spike and thus reduced the overall excitability of the network. Further, strong FF inhibition synchronized the firing pattern of the MSNs. On the other hand, the weak and sparse FB inhibition among the MSNs had a desynchronizing effect.

In addition, we characterized the input-output transfer function of the striatum network for correlated and uncorrelated inputs which mimic the cortico-striatal afferents, by selectively activating a subset of MSNs and FSIs. Such activation resulted in a corresponding decrease in the spiking of unstimulated neurons mainly mediated by the FF inhibition. This is similar to the neural activity recorded in striatum in behaving rats [2]. Thus the FF inhibition plays a critical role in improving the signal-to-noise ratio of the cortical inputs in the striatum.

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Supported by the German Federal Ministry of Education and Research (BMBF grants 01GQ0420 and01GW0542 Cognition).

The Poisson process with dead time captures important statistical features of neural activity

Moritz Deger, Stefano Cardanobile, Moritz Helias, Stefan Rotter

Stochastic point processes are widely used in computational neuroscience to model the spiking of single neurons and neuronal populations. The choice of a particular point process is critical for statistical measures of neural activity and has impact on the subthreshold dynamics of neuron models.

Here we show that the Poisson process with dead time, a particular simple point process, already captures important features of the spiking statistics of neurons [1,2] (Fig. A).

On the level of single neurons, we apply a step change to the rate of a Poisson process with dead time, keeping the refractory time constant. The expected PSTH is computed by numerically solving the partial differential equation of the corresponding non-homogeneous renewal process [3], and we also give an analytical approximation. We observe a very sharp transient in the firing -rate (Fig. B) that resembles experimental results of [4].

On the level of neuronal populations, we employ the superposition of many Poisson processes with dead time as a model of the population activity in a network. We compute the explicit form of the inter-spike-interval (ISI) distribution and the coefficient of variation for superimposed processes and compare them to direct simulations. The ISIs of the superimposed spike trains show negative serial correlations that correspond to those we observe in population recordings of simulated integrate -and -fire (i&f) neurons (Fig C).

For the single Poisson process with dead time and superpositions alike, we can determine the variance of shot noises driven by them, like the associated spike count in a certain time window or the free membrane potential of an i&f neuron. This enables us to show how empirical approximations of the Fano factor depend on the width of the counting window, and how the statistical properties of the driving point-process influence the variance of the subthreshold dynamics of neurons.

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Supported by the German Federal Ministry of Education and Research (BMBF grant 01GQ0420), and DIP F1.2.

Higher-order correlations in non-stationary parallel spike trains: statistical modeling and inference

Benjamin Staude, Stefan Rotter

Understanding the cooperative dynamics of large neuronal groups is a major topic in current brain research. A particularly controversial issue has been the extent to which groups of neurons exhibit higher-order correlations in their firing patters [1]. Higher-order correlations are a signature of the temporal coordination of action potentials across neurons, which is considered a powerful mechanism to cooperatively compute and transmit information in neuronal pools [2]. Currently available analysis tools, however, require vast sample sizes [3], rendering the analysis of massively parallel spike trains (N>10) for higher-order correlations essentially impossible.

We have recently presented a novel method for a cumulant-based inference of higher-order correlations (CuBIC) that avoids the need for extensive sample sizes [4,5]. This is achieved by a) exploiting constraining relations among correlations of different orders and b) estimating correlations among spike trains by the cumulants of the superimposed and discretely sampled spiking activity of all recorded neurons (population spike counts). Combining these concepts, CuBIC infers the presence of higher-order correlations from only few lower-order cumulants, which drastically reduces the requirements with respect to sample size as compared to previous approaches.

CuBIC employs the Compound Poisson Process as a statistical model for the population spike counts, where correlations are induced by the insertion of additional coincident events in continuous time, i.e. before the binning is applied [6]. In its current form, CuBIC furthermore assumes all spike trains to be stationary, an assumption which is often violated in standard experimental protocols.

Here, we present a non-stationary version of the compound Poisson process by decoupling the correlation structure from the intensity of the population. Using the "law of total cumulance", we incorporate common, population-wide non-stationarities into the computation of the cumulants of the population spike counts. These rate-adjusted cumulants are then utilized to adapt CuBIC to infer higher-order correlations even from non-stationary data stretches. The performance of the proposed adaptation is illustrated by numerical simulations.

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Supported by the German Federal Ministry of Education and Research (BMBF grant 01GQ0420).

A spiking temporal-difference learning model based on dopamine-modulated plasticity

Wiebke Potjans, Abigail Morrison, Markus Diesmann

Making predictions about future rewards and adapting the behavior accordingly is crucial for any higher organism. One theory specialized for prediction problems is temporal-difference (TD) learning. Experimental findings suggest that TD learning is implemented by the mammalian brain. In particular, the resemblance of dopaminergic activity to the TD error signal [1] and the modulation of corticostriatal plasticity by dopamine [2] lend support to this hypothesis. We recently proposed the first spiking neural network model to implement actor-critic TD learning [3], enabling it to solve a complex task with sparse rewards. However, this model calculates an approximation of the TD error signal in each synapse, rather than utilizing a neuromodulatory system.

Here, we propose a spiking neural network model which dynamically generates a dopamine signal based on the actor-critic architecture proposed by Houk [4]. This signal modulates as a third factor the plasticity of the synapses encoding value function and policy. The proposed model simultaneously accounts for multiple experimental results, such as the generation of a TD-like dopaminergic signal with realistic firing rates in conditioning protocols [1], and the role of presynaptic activity, postsynaptic activity and dopamine in the plasticity of corticostriatal synapses [5]. The excellent agreement between the predictions of our synaptic plasticity rules and the experimental findings is particularly noteworthy, as the update rules were postulated employing a purely top-down approach.

We performed simulations in NEST [6] to test the learning behavior of the model in a two dimensional grid-world task with a single rewarded state. The network learns to evaluate the states with respect to its reward proximity and adapt its policy accordingly. The learning speed and equilibrium performance are comparable to those of a discrete time algorithmic TD learning implementation.

The proposed model paves the way for investigations of the role of the dynamics of the dopaminergic system in reward based learning. For example, we can use lesion studies to analyze the effects of dopamine treatment in Parkinson's patients. Finally, the experimentally constrained model can be used as the centerpiece of closed-loop functional models.

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Supported by the German Federal Ministry of Education and Research (BMBF grant 01GQ0420), the EU Grant (FACETS, 15879), Next-Generation Supercomputer Project of MEXT, Japan, and the Helmholtz Alliance on Systems Biology.

Implications of the specific cortical circuitry for the network dynamics of a layered cortical network model

Tobias Potjans, Tomoki Fukai, Markus Diesmann

The local cortical network consists of specifically interconnected neuronal populations (see [1] for review). This microcircuitry determines the possible interactions between neurons and thus may play a crucial role in shaping neuronal activity. We investigate the dynamical implications of the specificity of connections in the local network by means of large-scale simulations [2] of a spiking layered network model. To this end, we quantify the specificity of connections measured by diverse experimental techniques.

We identify a hierarchy of specificity: Layer-specific connections depend on the layers of the preand post-synaptic populations. Among the most prominent layer-specific connection profiles is a feed-forward pattern of connections (layer 4 (L4) to L2/3 to L5 to L6) which is closely linked to the tuning properties of cells in the primary visual cortex [1]. Target-specific connections, in addition, depend on the neuronal type of the target neuron [3]. Projection-specificity, finally, classifies neuronal subpopulations of a layer according to the main target layer of their axonal projection (see [4] for review).

Our layered cortical network model consists of 80,000 I&F neurons and explains about 90% of the synapses constituting the local cortical microcircuit. As we focus on the relationship of connectivity and network activity, we use identical dynamics and parameters for all neuron types in the network. Despite this homogeneity, we observe that the layer specific connections alone cause layer- and type-specific firing rates: excitatory firing rates are lowest in L2/3 (often below 1 Hz, comparable to [5]) and highest in L5 and inhibitory firing exceeds excitatory rates. Furthermore we find that a small subset of target-specific connections [3] is relevant for the stability of network activity. Finally, the incorporation of projection-specificity enables us to investigate the influence of fine-scale connectivity on global activity patterns. We conclude that specific connections represent a structural correlate of the experimentally observed network dynamics.

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Supported by the German Federal Ministry of Education and Research (BMBF grant 01GQ0420), the EU Grant (FACETS, 15879), Next-Generation Supercomputer Project of MEXT, Japan, and the Helmholtz Alliance on Systems Biology.

Dynamics of non-linear cortico-cortical interactions during motion integration in early visual cortex: A spiking neural network model of an optical imaging study in the awake monkey.

Jens Kremkow, Laurent Perrinet, Alexandre Reynaud, Ad Aertsen, Guillaume S. Masson, Frederic Chavane

Lateral interactions are crucial mechanisms in contextual modulation of visual processing, including visual motion. We have recently shown [1], using voltage-sensitive dye imaging (VSDI), that a local static stimulus (Gaussian blob) first activates a restricted cortical area, followed by slow horizontal propagation of activity along the cortex. In a sequence of two local static stimuli, 2 stroke apparent motion, the two waves of horizontal activation interact non-linearly in V1, giving rise to a gradual and smooth wave of normalized activity.

The signature of this non-linear integration was a wave of suppression travelling from the representation of the second stimulus towards the first stimulus.

To investigate the cellular and network mechanisms underlying these non-linear lateral interactions, we constructed a two dimensional cortical network model using spiking neurons with conductance based synapses. The model represents cortical layer 2/3, the main source of the VSDI signals. The connectivity of the inhibitory neurons was restricted to the local neighbourhood, whereas the excitatory neurons could, in addition, also make long-range horizontal connections. The physiology of these horizontal connections was adjusted to induce balanced excitation/inhibition at high activity levels [2]. To compare the model dynamics to the in vivo VSDI signals we extracted a model VSDI signal by recording the membrane potentials of many neurons, arranged on a fine rectangular grid. The model was written in PyNN [3] using NEST[4] as a simulator.

Our simulations reproduced the experimental observations of slow horizontal propagation when stimulating the network by a single static stimulus. In the 2 stroke apparent motion paradigm the model reproduced the non-linear integration by a wave of suppression. The origin and dynamics of this suppression were caused by the activity-dependent amount of local inhibition balancing the effect of excitatory lateral connections.

Physiological data shows that similar wave of suppression could be observed with a wide range of spatio-temporal two stroke input, but also with “real motion” stimuli. We therefore generalized our study to conditions where the stimulus speed varies according to the suppressive spread velocity.

Thus, our model suggests that the wave of non-linear integration observed in vivo could be caused by local inhibition balancing the integration of horizontal inputs. Moreover, it highlights the importance of contextual modulation for visual processing.

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Supported by the German Federal Ministry of Education and Research (BMBF grant 01GQ0420), the EU Grant (FACETS, 15879), and the CNRS.

Finite synaptic potentials cause a non-linear instantaneous response of the integrate-and-fire mode

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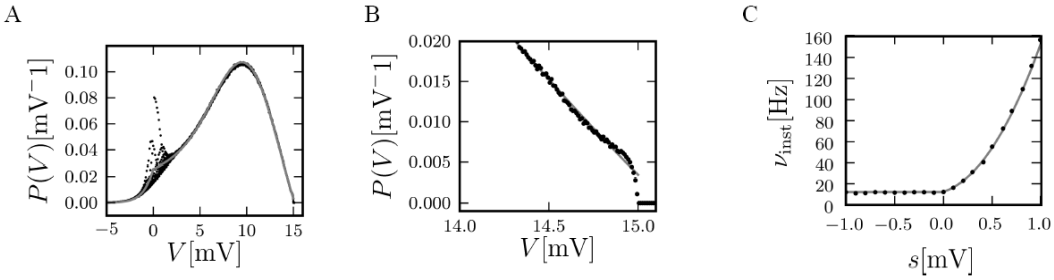
The integrate-and-fire neuron model with exponential postsynaptic potentials is widely used in analytical work and in simulation studies of neural networks alike. For Gaussian white noise input currents, the membrane potential distribution is described by a population density approach [1]. The linear response properties of the model have successfully been calculated and applied to the dynamics of recurrent networks in this diffusion limit [2]. However, the diffusion approximation assumes the effect of each synapse on the membrane potential to be infinitesimally small. Here we go beyond this limit and allow for finite synaptic weights. We show, that this considerably alters the absorbing boundary condition at the threshold: in contrast to the diffusion limit, the probability density goes to zero on the scale of the amplitude of a postsynaptic potential (Fig B). We give an analytic approximation for the density (Fig A) and calculate how its behavior near threshold shapes the response properties of the neuron. The neuron with finite synaptic weights responds arbitrarily fast to transient positive inputs. This differs qualitatively from the behavior in the diffusion limit, where the neuron acts as a low-pass filter [3]. We extend the linear response theory [3] and quantify the instantaneous response of the neuron to an impulse like input current. Even for realistically small perturbations (s) of the order of a synaptic weight, we find a highly non-linear behavior of the spike density (Fig C). Direct simulations in continuous time [4] confirm the analytical results. For numerical simulations in discrete time, we provide an analytical treatment which quantitatively explains the distortions of the membrane potential density. We find that temporal discretization of spikes times amplifies the effects of finite synaptic weights. Our demonstration of a non-linear instantaneous response amends the theoretical analysis of synchronization phenomena and plasticity based on the diffusion limit and linear response theory.



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Supported by the German Federal Ministry of Education and Research (BMBF grant 01GQ0420), the EU (FACETS, 15879), DIP F1.2, and Next-Generation Supercomputer Project of MEXT, Japan. All Simulatons are perfomed using NEST [5].

Self-organized criticality of developing artificial neuronal networks and dissociated cell cultures

Christian Tetzlaff, Samora Okujeni, Ulrich Egert, Florentin Wörgötter, Markus Butz

Self-organized criticality (SOC) Bak et al. (1987) was first described in neuronal cell cultures by Beggs & Plenz (2003). Neuronal networks being in a critical state produce avalanche-like discharges that are power-law distributed. The assessment of avalanches in neuronal networks is a new way of looking at neuronal activities apart from bursts, synchronization etc. The main novelty of our approach is to assess the avalanche distribution at different developmental stages of neuronal networks. For this, we used dissociated post-natal cell culture taken from the rat cortex (Experimental data was provided by the Ulrich Egert group, BCCN Fribourg, Germany). We found that different network states as subcritical, critical or supracritical specify a time and spatial activity profile that is linked but not equivalent to low, moderate or high levels in neuronal activity, respectively. We are the first who show that the activity profile in cell cultures develop from supracritical states over subcritical into critical states. To shed light to the dependency of SOC on network development, we used a self-organizing artificial neuronal network model based on a previous model by Van Ooyen and Abbott (Van Ooyen & Van Pelt, 1994; Van Ooyen et al., 1995; Abbott & Rohrkemper, 2007). An important novelty of our model is that it is more detailed with respect to representing seperate axonal and dendritic fields (Butz et al., 2008; Butz & Wörgötter, 2009). The model network aims to develop towards a homeostatic equilibrium in neuronal activity which is achieved by growth and retraction of axonal and dendritic fields. This abstract model already reproduces the transient behaviour as seen in cell cultures from supracritical over subcritical to critical states. However, we found that some cell cultures remain in a subcritical regime. The model offers a simple explanation as depending on the strength of inhibition, equivalent to the friction in self-organizing systems (Lauritsen et al., 1996), neuronal networks may or may not reach criticality even though they are homeostatically equilibrated.

Supported by the German Federal Ministry of Education and Research (BMBF grant 01GQ0420).

German Money