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COMPUTATIONAL NEUROSCIENCE / NEUROTECHNOLOGY
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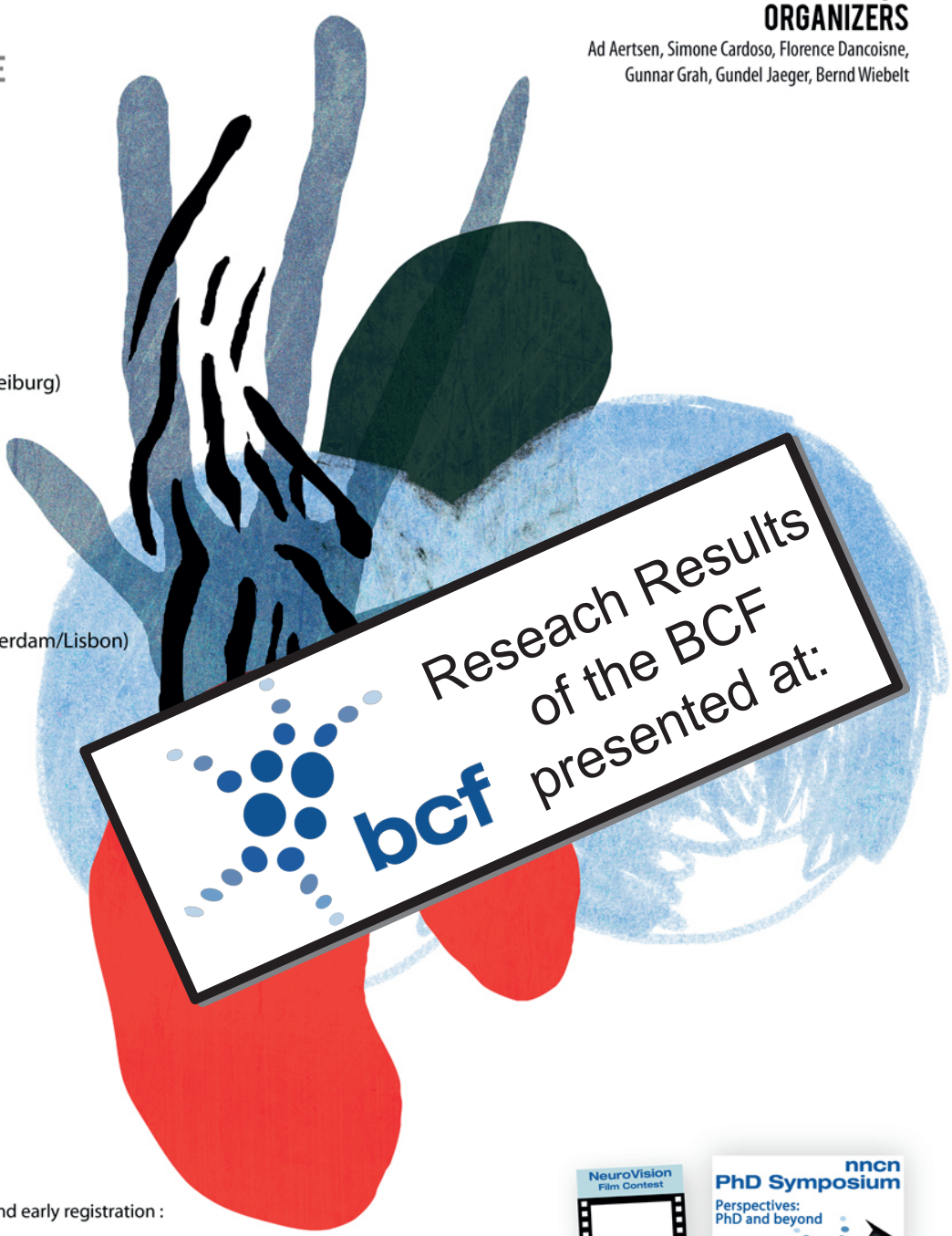

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EVENT ABSTRACT

Back to Event

Compartment-specific projection patterns onto pyramidal cells in rat neocortex

Mihael Zohar[1, 2*], Philipp Schnepel[1, 2], Ad Aertsen[1, 2] and Clemens Boucsein[1, 2]

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Pyramidal neurons in layer V of the neocortex constitute a major output population of the cortical network. Due to their good accessibility and their intricate morphology, showing a prominent apical dendrite spanning all cortical layers and terminating in an expanded tuft, these cells have been utilized to characterize general principles of cellular physiology of cortical pyramidal cells. In particular, it has been shown that the apical dendrite and the soma undergo functional decoupling during maturation, resulting in a strong attenuation of EPSP's arising from synapses located on the distal apical dendrite on their way to the soma. Furthermore, it was shown that apical dendrites of large layer V neurons are capable of active potential generation, such as Calcium- and NMDA-spikes. While connectivity of the somata was, up to now, extensively examined with anatomical, paired recording and photo stimulation methods, little is known about which populations of neurons project to the distal dendrite and are, thus, mainly involved in the generation of active potentials in the distal dendrite.

Here we used an experimental approach employing simultaneous distal dendritic and somatic patch-clamp recordings in vitro together with presynaptic glutamate uncaging to examine the properties and layer-dependent projection patterns onto the two compartments of layer V pyramidal neurons within the rat somatosensory cortex.

With this new combination of methods we were able to detect inputs from presynaptic neurons with a lateral distance of more than 1mm in acute brain slices and to compare the functional input maps from soma and apical dendrite. As reported previously, a substantial fraction of connections gave rise to EPSP's which underwent a strong attenuation along the apical dendrite and were hardly detectable at the soma. Surprisingly, however, the functional maps derived from dendritic vs. somatic recordings showed considerable differences in the layer in which the somata of the presynaptic neurons were located. While we could confirm the established projection patterns onto the soma, originating from cells in layers II/III, V and VI, the presynaptic neurons projecting onto the distal dendrite predominantly originate from supragranular layers. These compartment-specific projection patterns onto layer V pyramidal neurons point to a distributed integration of inputs coming from different cortical layers. These findings may help to further understand the role of dendritic integration in large layer V pyramidal cells, in particular the nonlinear mechanisms in the distal dendrite and the presynaptic populations involved in their generation.

Acknowledgements

This project received funding from the German Federal Ministry of Education and Research (Grants 01GQ0420 to BCCN Freiburg and 01GQ0830 to BFNT Freiburg-Tübingen) and from the German Research Council (DFG-SFB 780).

Keywords: compartment, connectivity, dendrite, neurons, networks and dynamical systems, photo stimulation

Conference: BC11 : Computational Neuroscience & Neurotechnology Bernstein Conference & Neurex Annual Meeting 2011, Freiburg, Germany, 4 Oct - 6 Oct, 2011.

Presentation Type: Poster

Topic: neurons, networks and dynamical systems (please use "neurons, networks and dynamical systems" as keywords)

Citation: Zohar M, Schnepel P, Aertsen A and Boucsein C (2011). Compartment-specific projection patterns onto pyramidal cells in rat neocortex. *Front. Comput. Neurosci. Conference Abstract: BC11 : Computational Neuroscience & Neurotechnology Bernstein Conference & Neurex Annual Meeting 2011.* doi: 10.3389/conf.fncom.2011.53.00127

Received: 23 Aug 2011; **Published Online:** 04 Oct 2011.

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Impact of intrinsic neuronal heterogeneity on firing rates and spike train correlations

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We studied the impact of neuronal heterogeneity on the spiking activities in a population of leaky integrate-and-fire neurons. In the high input regime, the sum of synaptic inputs to a neuron can be approximated by a fluctuating input noise, characterized by its mean and variance (Brunel & Hakim, 1999; Kuhn et al., 2004). Based on data from in vitro recordings (Padmanabhan and Urban, 2010) and new insights from mathematical analyses, we conclude that common input into heterogeneous neurons is better realized by an identical noise with different values of mean and variance than by the usual practice of adding independent noises to individual neurons. We identified the distinct roles of the mean and the variance for the spiking activity of a population of heterogeneous neurons. We found that the output firing rate of a neuron is largely shaped by the mean level of the noise, whereas the distributed values of the variance give rise to different degrees of imprecise spiking. To conclude, when receiving common input, heterogeneous neurons may differ considerably in their output firing rates, and their spikes may be jittered by several milliseconds, a phenomenon some researchers have termed “decorrelation” (Padmanabhan and Urban, 2010).

Acknowledgements

Supported by the German Federal Ministry of Education and Research (BMBF 01GQ0420 “BCCN Freiburg” and BMBF 01GW0730 “Impulse Control”).

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Padmanabhan, K., and Urban, N. N. (2010). Intrinsic biophysical diversity decorrelates neuronal firing while increasing information content. Nat. Neurosci. 13(10), 1276–1282.

Keywords: imprecise spiking, neuronal heterogeneity, white noise

Conference: BC11 : Computational Neuroscience & Neurotechnology Bernstein Conference & Neurex Annual Meeting 2011, Freiburg, Germany, 4 Oct - 6 Oct, 2011.

Presentation Type: Poster

Topic: neurons, networks and dynamical systems (please use "neurons, networks and dynamical systems" as keywords)

Citation: Yim M, Aertsen A and Rotter S (2011). Impact of intrinsic neuronal heterogeneity on firing rates and spike train correlations. *Front. Comput. Neurosci. Conference Abstract: BC11 : Computational Neuroscience & Neurotechnology Bernstein Conference & Neurex Annual Meeting 2011.* doi: 10.3389/conf.fncom.2011.53.00213

Received: 17 Aug 2011; **Published Online:** 04 Oct 2011.

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Quantitative models for stimulus-response relations in neuronal networks in vitro

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Electrical stimulation of nervous tissue is increasingly used in the treatment of CNS disorders, in neurotechnological devices or in examining the physiological properties of single cells and the function of networks of neurons. Interactions between ongoing and evoked neuronal activity render the stimulus' response sensitive to the network state and the history of previous activity. Here, we were interested in the interactions that arise between spontaneous and evoked network activity and how they shape and modulate stimulus-response relations. Our goal was to obtain analytical models for a user-defined interaction with neuronal activity.
We recorded and electrically stimulated rat cortical cell cultures on microelectrode arrays. Spontaneous network activity consisted of recurring periods of globally synchronized firing, so-called network bursts. The duration of intervals that preceded network bursts best predicted the length of the following network burst. Variable responses to electrical stimulation depended on the timing of stimulation relative to preceding network bursts. Response lengths increased exponentially and saturated with longer duration of pre-stimulus inactivity with $y(t) = A(1-e^{-\alpha t})$. Response delays, in turn, decreased exponentially and saturated at a low level with $y(t) = Be^{-\beta t} + C$. User-defined timing of stimulation relative to spontaneous activity significantly reduced trial-by-trial variability and thus facilitated further examinations on state-dependent stimulus-response relations.
Disinhibition by blockage of GABAA-receptors yielded ~183 % more spikes per network burst with ~88 % longer intervals and unchanged overall firing rates. The modulation of response delays by the duration of pre-stimulus inactivity persisted under disinhibition. The correlation between distance to stimulation site and response delay was enhanced and the speed of propagation clearly depended on stimulus-timing.
Facilitation of synaptic transmission by overexpressing DOC2B, a synaptic protein mainly involved in vesicle priming and docking, yielded ~39 % more spikes per network burst and ~72 % longer intervals. The rate parameter β that describes the relation between recovery from pre-stimulus, burst-induced depression and response delay decreased in young networks (< 20 DIV) whereas it increased in old networks (≥ 20 DIV) with DOC2B. This suggested slower and faster recovery, mediated by e.g. vesicle replenishment, depending on the network's maturation state.
In summary, we identified explicit rules for the modulation of evoked responses by spontaneous activity in generic neuronal networks in vitro. Our data support a process of network depression due to depletion of readily releasable vesicles during network bursts followed by subsequent recovery.

Acknowledgements

This work was supported by the German BMBF (FKZ 01GQ0420 & FKZ 01GQ0830) and the Boehringer Ingelheim Fonds.

Keywords: DOC2B, dynamical systems, electrical stimulation, microelectrode array, networks, neurons, pre-stimulus activity, response variability

Conference: BC11 : Computational Neuroscience & Neurotechnology Bernstein Conference & Neurex Annual Meeting 2011, Freiburg, Germany, 4 Oct - 6 Oct, 2011.

Presentation Type: Poster

Topic: neurons, networks and dynamical systems (please use "neurons, networks and dynamical systems" as keywords)

Citation: Weihberger O, Lavi A, Okujeni S, Ashery U and Egert U (2011). Quantitative models for stimulus-response relations in neuronal networks in vitro. *Front. Comput. Neurosci. Conference Abstract: BC11 : Computational Neuroscience & Neurotechnology Bernstein Conference & Neurex Annual Meeting 2011.* doi: 10.3389/conf.fncom.2011.53.00059

Received: 23 Aug 2011; **Published Online:** 04 Oct 2011.

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Neuron versus Time Clustering in the Identification of Cell Assemblies

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A major challenge in the analysis of neural activity data when considering spikes as the main information carrying unit is the detection of sets of neurons which act as functional groups. Such neuronal cell assemblies can be identified by clustering the spectrum of zero-lag cross-correlation between all pairs of neurons in a network or by dimensionality reduction of the similarity matrix of the spike trains.

Here we investigate how the identification of cell assemblies is dependent on the methodology chosen. We construct a self similar network of inhibitory adaptive exponential integrate-and-fire neurons that is stimulated with Poissonian excitatory input. For such a network one would expect that groups of neurons show a similar activity as the network as a whole. However, we observe that there is a difference between the evolution of network activity and sets of neurons clustered according to their correlation. When analyzing medium spiny neuron calcium imaging data, we again find that the results of the two methods are not in line.

Acknowledgements

Partially funded by the German Federal Ministry of Education and Research (BMBF 01GQ0420 to BCCN Freiburg, BMBF GW0542 Cognition and BMBF 01GW0730 Impulse Control), EU Grant 269921 (BrainScaleS), Helmholtz Alliance on Systems Biology (Germany), Neurex, the the Junior Professor Program of Baden-Württemberg and the Erasmus Mundus Joint Doctoral programme EuroSPIN.

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Keywords: Assemblies, Clustering, Dimensionality reduction, Medium Spiny Neurons, Self similar networks

Conference: BC11 : Computational Neuroscience & Neurotechnology Bernstein Conference & Neurex Annual Meeting 2011, Freiburg, Germany, 4 Oct - 6 Oct, 2011.

Presentation Type: Poster

Topic: data analysis and machine learning (please use "data analysis and machine learning" as keyword)

Citation: Toledo-Suárez C, Yim M, Kumar A and Morrison A (2011). Neuron versus Time Clustering in the Identification of Cell Assemblies. *Front. Comput. Neurosci. Conference Abstract: BC11 : Computational Neuroscience & Neurotechnology Bernstein Conference & Neurex Annual Meeting 2011.* doi: 10.3389/conf.fncom.2011.53.00073

Received: 23 Aug 2011; **Published Online:** 04 Oct 2011.

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Epileptiform Activity Reduces the Proteolytic Processing of Reelin in the Hippocampus

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The extracellular matrix protein Reelin, synthesized and secreted by Cajal-Retzius cells and GABAergic interneurons, is an important regulator for the formation of cortical layers during development and maintains this lamination in the adult hippocampus. In temporal lobe epilepsy (TLE) patients and in a TLE mouse model, Reelin levels are decreased which causes a migration defect of adult granule cells (Haas et al., 2002, Heinrich et al. 2006). However, not only absolute Reelin levels, but also proper proteolytic processing, giving rise to several Reelin isoforms, is important for its biological function. So far, it is unclear whether pathological processing of Reelin contributes to the malpositioning of dentate granule cells under epileptic conditions. To address this question, we used rat organotypic hippocampal slice cultures to investigate the effects of kainate (KA)-induced epileptiform activity on Reelin processing and the impact of Reelin cleavage on dentate granule cell layering.
As a prerequisite we showed that Reelin processing is decreased under epileptic conditions. Treatment of organotypic hippocampal slice cultures with KA resulted in an increase of high molecular weight Reelin isoforms in tissue and a significant decrease of the secreted 180 kDa Reelin fragment. This KA effect could be mimicked by incubation with protease inhibitors. Following epileptiform activity, we found a decrease of MMP-2 and MMP-9 (gelatinases) activity exclusively in the molecular layer and the granule cell layer of the dentate gyrus and elevated levels of the endogenous tissue inhibitor of MMPs-1 (TIMP-1). Both, epileptic conditions and impaired proteolytic cleavage of Reelin by inhibition of MMPs caused a significant widening of the granule cell layer and an extracellular accumulation of unprocessed Reelin. In summary, these experiments indicate that epileptic conditions impair the proteolytic processing of Reelin by an unbalance of MMPs and their inhibitor TIMP-1. As a consequence, Reelin accumulates extracellularly as a biological inactive form and contributes to granule cell dispersion.

Acknowledgements

Supported by the DFG, SFB TR3

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Heinrich C, et al. 2006. Reelin deficiency and displacement of mature neurons, but not neurogenesis, underlie the formation of granule cell dispersion in the epileptic hippocampus. J Neurosci 26: 4701-4713.

Keywords: dentate gyrus, extracellular matrix, granule cells, slice culture, TIMP-1

Conference: BC11 : Computational Neuroscience & Neurotechnology Bernstein Conference & Neurex Annual Meeting 2011, Freiburg, Germany, 4 Oct - 6 Oct, 2011.

Presentation Type: Poster

Topic: brain disease, network dysfunction and intervention (please use "brain disease, network dysfunction and intervention" as keyword)

Citation: Tinnes S, Ringwald J and Haas C (2011). Epileptiform Activity Reduces the Proteolytic Processing of Reelin in the Hippocampus. *Front. Comput. Neurosci. Conference Abstract: BC11 : Computational Neuroscience & Neurotechnology Bernstein Conference & Neurex Annual Meeting 2011.* doi: 10.3389/conf.fncom.2011.53.00214

Received: 17 Aug 2011; **Published Online:** 04 Oct 2011.

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The role of fast inhibition in AMPA and NMDA receptor mediated network burst dynamics in cortical cultures

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Synchronous patterns of activity are considered to play an essential role in the development of neuronal networks within a wide range of brain structures (Shatz, 1990). Similarly, in networks of dissociated cortical neurons spontaneous activity appears in form of network-wide bursts (NB) that are thought to be generated by recurrent excitatory pathways (Robinson et al. 1993; Jimbo et al. 2000). Inhibitory pathways, which suppress excitation, are however also considered important in shaping NB dynamics. As in native cortical tissue, glutamatergic fast AMPA receptors (AMPA-R) and slow NMDA receptors (NMDA-R) are the main mediators of excitatory synaptic transmission among neurons in vitro and fast inhibition is mediated via GABA_A receptors (GABA_A-R) (Legrand et al. 2004). Despite of a solid characterization of AMPA-R, NMDA-R, and GABA_A-R at the monosynaptic level, their respective contributions to particular phases during the NBs are still not well understood.
In this work we studied the role of both, fast and slow recurrent excitatory pathways, in initiating and maintaining NBs by blocking AMPA-Rs or NMDA-Rs in cortical cultures in vitro. Additionally, we investigated the influence of fast inhibition on shaping mainly AMPA-R or NMDA-R mediated NBs by additional GABA_A-R blockage. Network-wide activity was recorded with 59 planar extracellular electrodes under different pharmacological conditions. We analyzed the changes of overall network activity, NB rate and NB structures between the baselines and either AMPA-R or NMDA-R blocked activity and further between the latter states and GABA_A-R blocked activity.
Our results show that overall firing rates decrease after blockage of each of the excitatory receptors and in turn increase after block of fast inhibition as expected. Moreover, under all pharmacological conditions spontaneous activity was organized in NBs. Investigating the role of AMPA-Rs and NMDA-Rs on NB dynamics revealed that overall firing rates decrease three times more by NMDA-R blockage than by AMPA-R blockage. In contrast, burst rates decrease 10 times more by AMPA-R blockage than by NMDA-R blockage. This indicates that AMPA-Rs are in greater charge of initiating NBs than NMDA-Rs which mainly contribute in maintaining already initiated activity.
Our results further show that NMDA-R blocked bursts become shorter containing fewer spikes than control bursts and conversely, AMPA-R blocked bursts become longer containing more spikes. We suggested that this involves differences in time course and strength of feedback inhibition during NBs between both conditions. We therefore identified the influence of fast inhibition on recurrent glutamatergic network activity by blocking GABA_A-Rs, finding that overall firing rates are nearly ten times higher when NMDA-Rs were blocked compared to network state where AMPA-Rs were blocked. Moreover, disinhibition of NMDA-R blocked networks enhanced the early and late phases of bursts, while disinhibition of AMPA-R blocked networks shortened the late phase of bursts. This indicates that GABA_A-Rs effectively shut down AMPA-R mediated spiking and on the other hand further dampens the NMDA-R mediated network activity.

Acknowledgements

The authors would like to acknowledge the following fundings: Graduate School of Tampere University of Technology, Tampere Graduate School in Information Science and Engineering (TISE), Academy of Finland Grant (213462, 129657, 106030, and 124615), German BMBF (FKZ 01GQ0830, 01GQ0420), and EC (NEURO, 12788).

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Shatz, C. J. (1990). Impulse activity and the patterning of connections during CNS development. Neuron 5, 745-756.

Keywords: AMPA receptor, Cortical culture, Excitatory pathway, GABA_A receptor, Inhibitory pathway, Network dynamics, NMDA receptor, Recurrent network

Conference: BC11 : Computational Neuroscience & Neurotechnology Bernstein Conference & Neurex Annual Meeting 2011, Freiburg, Germany, 4 Oct - 6 Oct, 2011.

Presentation Type: Poster

Topic: neurons, networks and dynamical systems (please use "neurons, networks and dynamical systems" as keywords)

Citation: Teppola H, Okujeni S, Linne M and Egert U (2011). The role of fast inhibition in AMPA and NMDA receptor mediated network burst dynamics in cortical cultures. *Front. Comput. Neurosci. Conference Abstract: BC11 : Computational Neuroscience & Neurotechnology Bernstein Conference & Neurex Annual Meeting 2011.* doi: 10.3389/conf.fncom.2011.53.00043

Received: 23 Aug 2011; **Published Online:** 04 Oct 2011.

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EVENT ABSTRACT

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Transcranial DC stimulation (tDCS) improves voluntary modulation of mu-rhythm used for brain-machine interface (BMI) control

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The development of non-invasive and invasive BMI systems that translate electric or metabolic brain signals into control commands of external devices has experienced an impressive growth over the last years. They usually rely on the subjects' ability to control neural activity. The more rapid and accurate the control of such activity, the more effective BMI systems are. However, learning to control such activity often requires extensive training and, thus, it would be desirable to find strategies to facilitate learning.

In the motor domain, learning can be improved through designing practice protocols and/or by combining practice with stimulation of the central nervous system in the form of transcranial magnetic (TMS) or direct current stimulation (tDCS) that can facilitate learning effects. It was shown that these stimulation techniques can influence brain rhythms. Some of these rhythms, as for example the mu-rhythm (8-13Hz), were successfully utilized to control BMI systems. Here, we pose the hypothesis that application of non-invasive cortical stimulation over relevant brain regions facilitates control of neural activity in the mu-rhythm range used for online BMI control. If so, this could translate into better and more effective training protocols used in the context of assistive/biomimetic and restorative/biofeedback BMI systems.

Preliminary data support this hypothesis. We found that 20minutes of anodal tDCS delivered immediately before BMI training improved reliable production of event-related desychronization (ERD) of mu-rhythms. Analysis of neurophysiologic correlates of this improvement indicated that tDCS improves consistency of ERD production and optimal timing of mu-rhythm modulation.

We propose that brain stimulation might improve efficiency of BMI systems and help elucidating mechanisms underlying voluntary control of neural activity.

Acknowledgements

This work was supported by the Intramural Research Program (IRP) of the National Institute of Neurological Disorders and Stroke (NINDS), National Institues of Health (NIH), the German Federal Ministry of Education and Research (BMBF # 01GQ0831) and the Deutsche Forschungsgemeinschaft (DFG).

Keywords: brain stimulation, Brain-Machine Interface, ERD, mu-rhythm

Conference: BC11 : Computational Neuroscience & Neurotechnology Bernstein Conference & Neurex Annual Meeting 2011, Freiburg, Germany, 4 Oct - 6 Oct, 2011.

Presentation Type: Abstract

Topic: neurotechnology and brain-machine interface (please use "neurotechnology and brain-machine interface" as keyword)

Citation: Soekadar S, Witkowski M, Venkatakrishnan A, Ramos Murguialday A, Cohen LG and Birbaumer N (2011). Transcranial DC stimulation (tDCS) improves voluntary modulation of mu-rhythm used for brain-machine interface (BMI) control. *Front. Comput. Neurosci. Conference Abstract: BC11 : Computational Neuroscience & Neurotechnology Bernstein Conference & Neurex Annual Meeting 2011.* doi: 10.3389/conf.fncom.2011.53.00120

Received: 23 Aug 2011; **Published Online:** 04 Oct 2011.

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Distance and layer-dependent properties of horizontal projections onto layer 5 pyramidal neurons

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In order to elucidate the role of local, cortical networks in information processing in the brain, many studies both experimental and theoretical have been conducted over the past decades, spanning all levels and scales of investigation. Although the properties of local and inter-laminar synaptic connections have been investigated in great detail, the question remains if this is sufficient to describe more generic properties of cortical networks. Concomitantly, several neuroanatomical studies (Hellwig 2000; Binzegger et al., 2004; Stepanyants et al., 2009; Voges et al. 2010) have consistently suggested that an estimated 50-75% of the connections a neuron receives originate outside the local volume (radius: ~250µm). These connections have not been investigated in detail yet, which is mainly due to methodological constraints, but potentially have a strong impact on the local processing of information. Hence, they have received more and more interest over the past few years in order to complement the already well-established picture of local and laminar connectivity in terms of physiological properties and specificity. Their high number alone (up to 75%) together with potentially different connectivity patterns and synaptic properties might change the view of how cortical networks process information considerably.

Here, we used photostimulation to map long-range horizontal projections to layer 5B pyramidal neurons in acute cortical slices. For lateral distances of 200-1500µm, we found intact projections and characterized their physiological properties as well as their layer of origin. The average amplitude of EPSCs slightly dropped with distance, while strong connections were still present over long distances. Short and long range connections showed an equally high synaptic reliability of close to 100% in most tested synapses, the same level of amplitude variability, and an equally high temporal precision of <1ms. Indications for layer- and distance dependent differences in synaptic physiology are reported on especially for L5 and L6 projections. In summary, our data provide an initial parameterization of long-range connections, which could be used to refine structured models of cortical networks. We conclude that long-distance horizontal connections could represent a substantial fraction of inputs to the local, cortical network (Boucsein et al, 2011). Secondly, although they showed a slight drop in amplitude with increasing distance, they contribute with reliable and precise inputs to the single neurons in layer 5, thus impacting the local computation considerably.

Acknowledgements

Funded by the German Federal Ministry of Education and Research (Grants 01GQ0420 to BCCN Freiburg and 01GQ0830 to BFNT Freiburg-Tübingen), the German Research Council (DFG-SFB 780).

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Boucsein C, Nawrot MP, Schnepel P and Aertsen A (2011) Beyond the cortical column: abundance and physiology of horizontal connections imply a strong role for inputs from the surround. Front. Neurosci. 5:32. doi: 10.3389/fnins.2011.00032

Keywords: connectivity, neural variability, neurons, networks and dynamical systems, photo stimulation, synaptic transmission

Conference: BC11 : Computational Neuroscience & Neurotechnology Bernstein Conference & Neurex Annual Meeting 2011, Freiburg, Germany, 4 Oct - 6 Oct, 2011.

Presentation Type: Poster

Topic: neurons, networks and dynamical systems (please use "neurons, networks and dynamical systems" as keywords)

Citation: Schnepel P, Nawrot M, Aertsen A and Boucsein C (2011). Distance and layer-dependent properties of horizontal projections onto layer 5 pyramidal neurons. *Front. Comput. Neurosci. Conference Abstract: BC11 : Computational Neuroscience & Neurotechnology Bernstein Conference & Neurex Annual Meeting 2011.* doi: 10.3389/conf.fncom.2011.53.00149

Received: 23 Aug 2011; **Published Online:** 04 Oct 2011.

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EVENT ABSTRACT

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Contrast invariance in recurrent networks of multiplicatively interacting neurons

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Contrast invariance has been reported as an important property of orientation selective neurons in primary visual cortex. Here we show that in random recurrent networks of multiplicatively interacting neurons, this phenomenon occurs in a natural way. There is no need to impose rectification by a transfer function, as it is the case in networks using the classical Wilson-Cowan model, since rectification arises as a consequence of the mean-field description of the network in form of coupled Lotka-Volterra rate equations. We show that the shapes of the output tuning curves obtained by network stimulation are invariant upon input scaling (contrast invariance). We also show that sharp orientation tuning can be obtained in an inhibition dominant operating regime of the network. Finally, we compare this behavior with simulated neuronal responses in a more realistic network of spiking leaky integrate-and-fire neurons. Summarizing we argue that the multiplicative model provides a mathematically convenient and biologically realistic description for the networks in visual cortex.

Keywords: Contrast invariance, Lotka-Volterra equation, Multiplicative interaction, Recurrent network, Spiking neurons, Wilson-Cowan model

Conference: BC11 : Computational Neuroscience & Neurotechnology Bernstein Conference & Neurex Annual Meeting 2011, Freiburg, Germany, 4 Oct - 6 Oct, 2011.

Presentation Type: Poster

Topic: sensory processing (please use "sensory processing" as keyword)

Citation: Sadeh S, Cardanobile S and Rotter S (2011). Contrast invariance in recurrent networks of multiplicatively interacting neurons. *Front. Comput. Neurosci. Conference Abstract: BC11 : Computational Neuroscience & Neurotechnology Bernstein Conference & Neurex Annual Meeting 2011.* doi: 10.3389/conf.fncom.2011.53.00227

Received: 02 Aug 2011; **Published Online:** 04 Oct 2011.

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Inferring higher-order correlations from filtered spike activity

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Nonlinear response properties turn neurons into highly sensitive detectors for higher-order features of their input (see e.g. [1]). Whether or not higher-order correlations are important for cortical information processing, however, can only be decided by the analysis of experimental data.

Common data analysis methods (e.g. [2]) to investigate the potential role of higher-order correlations (HOCs) are devised for the application to spike recordings from multiple single neurons. A recent particularly promising approach is the cumulant based inference of higher-order correlations (CuBIC, [3]). CuBIC allows to infer a lower bound on the maximal order of correlations by employing the cumulants of the population spike count, i.e. the population spike activity “filtered” with a rectangular kernel. Compared to other methods, this approach can detect even weak HOCs in the activity of large neuronal populations based on realistic sample sizes.

However, describing HOCs in a neuronal population does not reveal its influence on the activity of a neuron at the next processing stage. In this respect, estimating cooperative dynamics in the presynaptic spike activity from an intracellular recording of a single neuron would be advantageous (cf. [4] for an approach based on pairwise correlations). To approach this issue, we represent the subthreshold activity as presynaptic activity filtered with a fixed kernel and adapt CuBIC accordingly. Studies on surrogate data revealed that the new method can reliably infer HOCs even from short stretches of membrane potentials.

Acknowledgements

Support by the German Federal Ministry of Education and Research (BMBF; grant 01GQ0420 to BCCN Freiburg) is gratefully acknowledged.

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Keywords: data analysis and machine learning, higher-order correlations, intracellular recordings, membrane potential fluctuations, shot noise, stochastic process, subthreshold activity

Conference: BC11 : Computational Neuroscience & Neurotechnology Bernstein Conference & Neurex Annual Meeting 2011, Freiburg, Germany, 4 Oct - 6 Oct, 2011.

Presentation Type: Poster

Topic: data analysis and machine learning (please use "data analysis and machine learning" as keyword)

Citation: Reimer IC, Staude B and Rotter S (2011). Inferring higher-order correlations from filtered spike activity. *Front. Comput. Neurosci. Conference Abstract: BC11 : Computational Neuroscience & Neurotechnology Bernstein Conference & Neurex Annual Meeting 2011.* doi: 10.3389/conf.fncom.2011.53.00108

Received: 23 Aug 2011; **Published Online:** 04 Oct 2011.

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Haptic Brain Computer Interface in Paralyzed Chronic Stroke Patients

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Incidence of a first stroke in Europe is about 1.1 million and prevalence about 6 million per year. Currently, about 75% of people affected by a stroke survive one year or more and this proportion will increase in the coming years due to enhanced quality in hyper-acute, follow-up acute and sub-acute care, and life-long treatment of these conditions. From all the stroke survivors showing no active upper limb motion at hospital admission, 14% showed complete recovery, while 30% showed partial recovery and 56% showed no recovery (Hendrick et al. 2002). Stroke survivors with chronic hand plegia and very low score in the Fugl-Meyer scale show limited residual muscle activity in the upper arm extensor muscles and normally no residual finger extension. Currently, there is no accepted and efficient rehabilitation strategy available that aims at reducing focal impairments in patients with chronic stroke and complete hand paralysis. In tight collaboration, the University of Tubingen (Germany) and the National Institutes of Health (NIH) demonstrated for the first time that stroke patients with complete hand paralysis can learn to control a magnetoencephalography (MEG) based Brain-Computer Interface (BCI) to drive a hand robotic orthosis (Buch et al. 2008). The BCI was used to move a cursor on a screen and depending on correct or incorrect response the hand orthotic device would or would not move the hand respectively. The results could not be translated out of the lab and patients needed the orthosis to move their hands. In a later study, we demonstrated that the combination of BCI and daily life-oriented physiotherapy can elicit functional recovery improving hand and arm movements as well as gait (Broetz et al. 2009). Furthermore, using a multimodal neuroimaging approach based on fMRI and diffusion tensor imaging (DTI) we investigated brain plasticity in the motor system along with longitudinal clinical assessments. We found a convergent association between functional and structural data in the ipsilesional premotor areas (Caria et al. 2010). Parallel to these findings we studied the effect of haptic feedback during the use of a sensorimotor rhythm (SMR) BCI. Here, an online EEG-based proprioceptive BCI was used for stroke rehabilitation (Ramos-Murguialday et al. 2009 & 20010) controlling a robotic exoskeleton online (250msec delay) using brain signals. In our study 36 chronic stroke patients with minimal residual hand extension underwent a 6-week daily online haptic-BCI rehabilitation therapy combined with goal-oriented physiotherapy. Several multimodal pre- and post-measurements were used to assess physiological and functional rehabilitation. The pre-measurements were conducted twice, two months before and immediately before the 6-week daily training. These two measurements allowed us to have a baseline of neurophysiological and psychophysiological data to check for stability and reliability of our measurements. The post-measurements were divided in two phases as well having one on the day after the last day of training and the second one six months later as a follow-up measurement. Magnetoencephalography (MEG) was used to measure sensory inputs using pneumatic vibrotactile actuators fixed to the index and pinky fingers and the lip. The ability to imagine and perform movement was assessed through a three-class protocol using MEG and functional magnetic resonance (fMRI). The MRI-scanner was also used to acquire important information related to anatomy and anatomical connectivity. To explore the corticospinal tract integrity, neuronavigated TMS was applied to the patients acquiring MEPs from lower and upper arm muscles and thus allowing us to have a more precise cortical map of flexors and extensors. TMS was applied following several protocols to elicit more stable and greater MEPs using pre contraction or imagination of movement. Several movements included in the Fugl-Meyer scale were used to generate a protocol to register muscle activity from the healthy and paretic side for further comparisons. EEG screenings were performed in order to identify most relevant oscillatory brain frequencies and electrode positions during hand opening and closing. This information was used to set up the online proprioceptive BCI classifier. Other psychological and physiotherapeutical tests (e.g. Wolf Motor Function Test, Fugl-Meyer Score, Ashworth Scale, SEIQL) were performed to correlate functional scales with neurophysiological data. Patients were assigned into one of three different feedback contingency groups (positive, negative and non-contingent feedback). Pilot results of this clinical study will be presented and discussed.

Acknowledgements

This study was supported by the German Ministry of Education and Research (Bernstein Fokus: Neurotechnologie, 01GQ0761, BMBF 16SV3783), ERC grant 227632-BCCI, the Deutsche Forschungsgemeinschaft (DFG) and the Intramural Research Program (IRP) of the National Institute of Neurological Disorders and Stroke (NINDS), National Institues of Health (NIH).

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Keywords: Brain Machine Interface, Haptic Feedback, Stroke Rehabilitation

Conference: BC11 : Computational Neuroscience & Neurotechnology Bernstein Conference & Neurex Annual Meeting 2011, Freiburg, Germany, 4 Oct - 6 Oct, 2011.

Presentation Type: Abstract

Topic: neurotechnology and brain-machine interface (please use "neurotechnology and brain-machine interface" as keyword)

Citation: Ramos-Murguialday A, Caria A, Broetz D, Läer L, Soekadar S and Birbaumer N (2011). Haptic Brain Computer Interface in Paralyzed Chronic Stroke Patients. *Front. Comput. Neurosci. Conference Abstract: BC11 : Computational Neuroscience & Neurotechnology Bernstein Conference & Neurex Annual Meeting 2011.* doi: 10.3389/conf.fncom.2011.53.00119

Received: 23 Aug 2011; **Published Online:** 04 Oct 2011.

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Identification of electrophysiological endpoints in stem cell-based systems for developmental neurotoxicity testing.

Grzegorz Podrygajlo[1, 2*], Samora Okujeni[1, 2, 3], Patrick Dini[1, 2, 3], Matthew Goddard[1, 2] and Ulrich Egert[1, 2]

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The toxicity of chemicals on brain development is of major concern. A predictive in vitro test for potential developmental neurotoxicity (DNT) needs to be an inexpensive, quick, standardized and predictive alternative to present in vivo methods. Here, we combine endpoints based on attachment, proliferation, differentiation and electrophysiological analyses, comparing four neuronal systems (murine embryonic stem cells, human neural progenitor cells and human teratocarcinoma cells) grown on microelectrode arrays (MEAs). Electrophysiological recordings show a spontaneous electrical activity of the networks derived from murine embryonic stem cells and human teratocarcinoma cells neuronal networks only. Comparison of their properties at different developmental stages shows activity increasing from 1st to 3rd week. Supplementary to the MEAs data we provided measurements of Ca+2 transient curves, which support existence of active neuronal networks in case of murine embryonic stem cells and human teratocarcinoma cells. Moreover, we optimized the differentiation of human teratocarcinoma cells, which now yields up to 2 times more neurons in a 3 to 5 days shorter time. In summary, we analyzed functional endpoints for electrical activity and network properties of 3 cell lines during the period of neuronal maturation.

Acknowledgements

NT2 cells were obtained from G. Bicker (Tiho, Hannover, Germany)
mESC cells were obtained from A. Seiler (BfR, Berlin, Germany)
hNPC cells were obtained from E. Fritsche (IUF, Duesseldorf, Germany)

Supported by the German BMBF FKZ 0315522B

Keywords: developmental toxicity DNT, microelectrode arrays MEAs, networks, neurons, stem cells

Conference: BC11 : Computational Neuroscience & Neurotechnology Bernstein Conference & Neurex Annual Meeting 2011, Freiburg, Germany, 4 Oct - 6 Oct, 2011.

Presentation Type: Poster

Topic: neurons, networks and dynamical systems (please use "neurons, networks and dynamical systems" as keywords)

Citation: Podrygajlo G, Okujeni S, Dini P, Goddard M and Egert U (2011). Identification of electrophysiological endpoints in stem cell-based systems for developmental neurotoxicity testing.. *Front. Comput. Neurosci. Conference Abstract: BC11 : Computational Neuroscience & Neurotechnology Bernstein Conference & Neurex Annual Meeting 2011.* doi: 10.3389/conf.fncom.2011.53.00057

Received: 23 Aug 2011; **Published Online:** 04 Oct 2011.

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Relation between spike correlations and network structure

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Correlations between neural spike trains are a widely studied phenomenon, due to their ubiquity and their influence on neural network dynamics and function. A state of relatively weak correlations and irregular spike trains is often assumed to be a good model for normal activity in cortical networks. 'Balanced' networks of leaky intergrate-and-fire neurons display low average correlations and irregular spike trains. There, the interplay between excitatory and inhibitory populations has been proposed as a key mechanism of correlation reduction. However, the variance of the correlations is typically still large.

We describe pairwise correlations in networks of integrate-and-fire neurons in the framework of point processes [1] considering only linear responses of all neurons. This approximation is applicable under a wide range of conditions, provided that spike activity is sufficiently irregular, and that reset effects are taken into account. The approach yields a simple analytical expression for correlations in the network and the connectivity matrix that encodes the synaptic topology of the network. Differences in correlations can be fully attributed to differences in the connectivity structure between neurons.

As correlations result from multiple direct and indirect synaptic connections [2], the inverse problem -- inference of network structure from correlations -- has no unique solution in general. We find that this fundamental ambiguity can be considerably alleviated, if a priori knowledge about specific features of the network structure, as for example sparse connectivity, is taken into account.

Acknowledgements

We gratefully acknowledge support by the German Research Foundation (CRC 780, subproject C4) and by the German Federal Ministry of Education and Research (BMBF grant 01GQ0420 to BCCN Freiburg).

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Keywords: correlations, integrate-and-fire neurons, network structure, neurons, networks and dynamical systems

Conference: BC11 : Computational Neuroscience & Neurotechnology Bernstein Conference & Neurex Annual Meeting 2011, Freiburg, Germany, 4 Oct - 6 Oct, 2011.

Presentation Type: Poster

Topic: neurons, networks and dynamical systems (please use "neurons, networks and dynamical systems" as keywords)

Citation: Pernice V, Cardanobile S, Staude B and Rotter S (2011). Relation between spike correlations and network structure. *Front. Comput. Neurosci. Conference Abstract: BC11 : Computational Neuroscience & Neurotechnology Bernstein Conference & Neurex Annual Meeting 2011.* doi: 10.3389/conf.fncom.2011.53.00065

Received: 23 Aug 2011; **Published Online:** 04 Oct 2011.

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Network inhomogeneity promotes spontaneous bursting in vitro

Samora Okujeni[1, 2, 3*], Nila Moenig[2], Steffen Kandler[1, 2, 3], Oliver Weihberger[1, 2, 3] and Ulrich Egert[1, 3]

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Spontaneously initiated synchronous bursting events (SBE) are widely observed in neuronal systems and are considered important in shaping fundamental neuronal circuitry in early cortical development (1,2). Likewise, SBE dynamics represent the predominant type of activity in developing networks of cultured neurons in vitro. Interestingly, theoretical models have shown that hierarchical network structures embedding clusters of strongly inter-connected neurons are optimal for initiating and sustaining spontaneous activity (3). We hypothesize that activity-dependent wiring supports the formation of clustered network structures supportive to SBE initiation. To test this we chronically manipulated activity-dependent structural plasticity in developing networks of cortical neurons in vitro by chronic inhibition of protein kinase C (PKC). Previous studies showed that PKC inhibition in developing cerebellum promotes dendritic outgrowth and arborization of Purkinje cells (4), and impairs pruning of climbing fibers (5), linking this protein closely to structural plasticity. We consistently found that developmental inhibition of PKC in cortical cell cultures increased dendritic outgrowth, impaired neurite fasciculation and clustering, and abolished network pruning. This resulted in more homogeneous and potentially better connected networks. In consequence, propagation of activity within SBE was faster and occurred in highly regular wave fronts. SBE were, however, triggered from fewer sites and at much lower rates suggesting that the homogeneous networks forming under blockade of activity-dependent wiring processes embed fewer SBE initiation zones. To further confirm that SBE frequencies in homogeneous networks were limited by reduced spontaneous network activation, we provided additional input by electrical stimulation. Interestingly, homogeneous networks formed under PKC inhibition achieved even higher activity levels when electrically stimulated than inhomogeneous control networks. Our data suggests that activity-dependent structural plasticity promotes network inhomogeneity which increases spontaneous activity levels during development. Based on recent evidence for a reciprocal scaling between synaptic strength and number of neuronal partners in vitro (6), we propose that locally more confined synaptic targeting within neuronal clusters promotes stronger and more recurrent coupling of neurons. The resulting connectivity patterns would more easily amplify spontaneous excitation beyond a critical threshold and thus serve as SBE initiation zones.

Acknowledgements

Funded by the German BMBF (FKZ 01GQ0420 & FKZ 01GQ0830) and by the EC (NEURO, No. 12788).

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Keywords: bursting, connectivity, culture, dendrites, in vitro, network, PKC, spontaneous activity

Conference: BC11 : Computational Neuroscience & Neurotechnology Bernstein Conference & Neurex Annual Meeting 2011, Freiburg, Germany, 4 Oct - 6 Oct, 2011.

Presentation Type: Poster

Topic: neurons, networks and dynamical systems (please use "neurons, networks and dynamical systems" as keywords)

Citation: Okujeni S, Moenig N, Kandler S, Weihberger O and Egert U (2011). Network inhomogeneity promotes spontaneous bursting in vitro. *Front. Comput. Neurosci. Conference Abstract: BC11 : Computational Neuroscience & Neurotechnology Bernstein Conference & Neurex Annual Meeting 2011.* doi: 10.3389/conf.fncom.2011.53.00051

Received: 23 Aug 2011; **Published Online:** 04 Oct 2011.

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Syntax, synfire, and synaptic plasticity: Modeling the generation of structure in birdsong and neuronal networks

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Adult Bengalese finches generate a variable song that obeys a distinct and individual syntax. The syntax is gradually lost over a period of days after deafening and is recovered when hearing is restored. In the first part of this talk I will present a spiking neuronal network model of the song syntax generation and its loss, based on the assumption that the syntax is stored in reafferent connections from the auditory to the motor control area. Propagating synfire activity in the HVC (high vocal center) codes for individual syllables of the song and priming signals from the auditory network reduce the competition between syllables to allow only those transitions that are permitted by the syntax. Both imprinting of song syntax within HVC and the interaction of the reafferent signal with an efference copy of the motor command are sufficient to explain the gradual loss of syntax in the absence of auditory feedback.

In the second part of this talk I will consider how the synfire chains assumed in the first part could develop. It has long been though that spike-timing dependent plasticity (STDP) provides an answer to the question of how the brain can develop functional structure in response to repeated stimuli. However, convincing demonstrations of this capacity in large, initially random networks have not been forthcoming; such demonstrations as there are typically rely on constraining the problem artificially. I will present a theoretical analysis based on a mean field approach of the development of feed-forward structure in random networks. An unstable fixed point in the recruitment dynamics prevents the stable propagation of structure in recurrent networks with weight-dependent STDP. The key theoretical predictions can be confirmed in large-scale simulations. The theory provides insight into the reasons why such development does not take place in unconstrained systems and enables the identification of candidate biologically motivated adaptations to the balanced random network model that might resolve the issue.

Keywords: Plasticity and Learning

Conference: BC11 : Computational Neuroscience & Neurotechnology Bernstein Conference & Neurex Annual Meeting 2011, Freiburg, Germany, 4 Oct - 6 Oct, 2011.

Presentation Type: Keynote

Topic: other

Citation: Morrison A (2011). Syntax, synfire, and synaptic plasticity: Modeling the generation of structure in birdsong and neuronal networks. *Front. Comput. Neurosci. Conference Abstract: BC11 : Computational Neuroscience & Neurotechnology Bernstein Conference & Neurex Annual Meeting 2011.* doi: 10.3389/conf.fncom.2011.53.00021

Received: 26 Sep 2011; **Published Online:** 04 Oct 2011.

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EVENT ABSTRACT

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An Online Brain-Machine Interface Using Decoding Of Movement Direction From The Human Electrocorticogram

Tomislav Milekovic[1, 2, 3, 4*], Jörg Fischer[4], Tobias Pistohl[3, 4], Johanna Ruescher[4, 5], Andreas Schulze-Bonhage[3, 5], Ad Aertsen[3, 4], Jörn Rickert[3, 4], Tonio Ball[3, 5] and Carsten Mehring[1, 2, 3, 4]

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4 University of Freiburg, Faculty of Biology, Germany
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A brain-machine interface (BMI) allows subjects to control an external actuator directly via their brain activity, without participation of the spinal cord or the peripheral motor system. BMIs can be characterized by the approach used to translate brain signals into effector movements. Here we use a “direct motor” BMI approach where movements of an artificial effector (e.g. movement of an arm prosthesis to the right) are controlled by motor cortical signals controlling the equivalent movements of the corresponding body limb (e.g. arm movement to the right). This approach has been successfully applied in monkeys and humans by accurately extracting parameters of movements from the spiking activity of multiple single-units. These spiking activities can only be recorded with electrodes implanted into the brain. Here we show that the same approach can be realized using brain activity measured directly at the surface of the human cortex (electrocorticogram, ECoG). Five subjects suffering from intractable pharmaco-resistant epilepsy voluntarily participated in the study after having given their informed consent (study approved by the Freiburg University Hospital's Ethics Committee). As part of pre-surgical diagnosis, all subjects had an 8x8 ECoG grid implanted subdurally over the hand/arm motor cortex. Subjects interacted with an experimental paradigm shown on a computer screen. Each trial consisted of a pause phase (1-2 sec) followed by a preparatory informative cue (1-2 sec) informing the subject to prepare for executing a hand/arm movement to the left or to the right (arm contralateral to the implantation site). After a delay of 2-3 sec, a go cue was presented and subjects executed the movement within the next three seconds. Subsequently, a cursor on the screen was moved according to the movement direction decoded from the subjects’ ECoG signals. Closed-loop BMI control of movement direction was realized using low-pass filtered ECoG signals during movement execution. Significant BMI control was achieved for 4 out of 5 subjects with correct directional decoding in 69%-86% of the trials (75% on average across all sessions). In one of the sessions BMI control was achieved using ECoG signals from only two neighbouring electrodes, indicating the feasibility of using smaller ECoG implants with smaller and/or denser electrode grids for direct motor BMI applications. Our results demonstrate the feasibility, in principle, of an online direct motor BMI using neuronal signals from the brain surface. Thus, for a direct motor BMI, ECoG might be used in conjunction with or an alternative to neuronal signals measured within the brain, with possible advantages due to reduced invasiveness.

Acknowledgements

Work supported by BMBF 01GQ0420 grant to BCCN Freiburg, BMBF 01GQ0830 grant to BFNT Freiburg and Tübingen and BMBF GoBio grant 0313891.

Keywords: ECoG, human, movement direction, neurotechnology and brain-machine interface, online

Conference: BC11 : Computational Neuroscience & Neurotechnology Bernstein Conference & Neurex Annual Meeting 2011, Freiburg, Germany, 4 Oct - 6 Oct, 2011.

Presentation Type: Abstract

Topic: neurotechnology and brain-machine interface (please use "neurotechnology and brain-machine interface" as keyword)

Citation: Milekovic T, Fischer J, Pistohl T, Ruescher J, Schulze-Bonhage A, Aertsen A, Rickert J, Ball T and Mehring C (2011). An Online Brain-Machine Interface Using Decoding Of Movement Direction From The Human Electrocorticogram. *Front. Comput. Neurosci. Conference Abstract: BC11 : Computational Neuroscience & Neurotechnology Bernstein Conference & Neurex Annual Meeting 2011.* doi: 10.3389/conf.fncom.2011.53.00198

Received: 19 Aug 2011; **Published Online:** 04 Oct 2011.

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EVENT ABSTRACT

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Brain-computer interfaces using field potentials

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A brain-computer interface (BCI) translates neuronal signals reflecting a subject's movement intentions into commands driving a machine (e.g. a prosthesis or a computer). In this talk I will present recent findings from our research on the development of BCIs that use non-spiking neuronal signals, e.g. signals measured directly from the surface of the human brain (Electrocorticogram, ECoG) or signals measured non-invasively (EEG / MEG). I will show that different parameters of natural hand/arm movements (e.g. movement direction, velocity and grasp) can be predicted from these signals and used for online control of external actuators. While this demonstrates the principle feasibility of the approach, current BCI control is of limited complexity and performance. To advance ECoG/EEG/MEG based BCIs a better understanding of movement encoding in these signals is essential and I will discuss our recent progress in this direction.

Acknowledgements

Contributions from A. Aertsen, T. Ball, J. Fischer, F. Galan, X. Liao, T. Milekovic, E.Neitzel, I. Novick, T. Pistohl, J. Rickert, G. Schalk, A. Schulze-Bonhage, E. Vaadia, S. Waldert and others are gratefully acknowledged.
Supported by the German Federal Ministry of Education and Research (BMBF Grant 01GQ0420, 0313891 and 01GQ0761).

Keywords: Motor Decoding and Brain-Machine Interfaces

Conference: BC11 : Computational Neuroscience & Neurotechnology Bernstein Conference & Neurex Annual Meeting 2011, Freiburg, Germany, 4 Oct - 6 Oct, 2011.

Presentation Type: Keynote

Topic: other

Citation: Mehring C (2011). Brain-computer interfaces using field potentials. *Front. Comput. Neurosci. Conference Abstract: BC11 : Computational Neuroscience & Neurotechnology Bernstein Conference & Neurex Annual Meeting 2011.* doi: 10.3389/conf.fncom.2011.53.00020

Received: 26 Sep 2011; **Published Online:** 04 Oct 2011.

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EVENT ABSTRACT

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High-gamma coupling between and within human pre- and primary motor cortex during movements

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In this study, we investigated neuronal interactions between pre- and primary cortical motor areas and their information about movements by analyzing partial directed coherence (PDC) and phase locking values (PLV) of high-gamma (80-200Hz) electrocorticographic (ECoG) signals recordings in humans during visually cued and self-paced motor tasks.

Five types of motor tasks were performed (each by 2-6 subjects): (1) cued individual finger flexion; (2) cued 8-directional center-out joystick movement; (3) cued brain-controlled 1D cursor movement based on motor imagery; (4) cued brain-controlled 1D cursor movement based on motor movements; (5) self-paced left/right joystick movements.

We computed the PDC to analyze the directional interactions of high-gamma activities between pre-motor cortex (PM) and primary motor cortex (M1). The PDC from PM to M1 increased briefly before and during the movements, consistently across all five motor tasks. Additionally, the involvement of different parts of dorsal and ventral PM depended on whether the task was cued or self-paced: for cued movements (1-4), we observed an increase in the PDC from dorsal pre-motor to primary motor cortex, while for self-paced movements (5), the most prominent observation was an increase in the PDC from ventral pre-motor cortex to primary motor cortex.

For movement tasks (1) and (5) we investigated the dependence of PM-M1 interaction on the movement type. To this end, we computed the PLV separately for index and little finger movements and separately for left/right joystick movements, and decoded the movement type from single-trial PLVs. On average, we found that the movement type could be inferred correctly from the PLVs in about 80% of the trials.

Our results indicate that the directed coherence patterns reflect information flow from pre- to primary motor cortex during different types of motor tasks, and the involvement of different parts of pre-motor cortex, depending on whether movements are externally cued or self-paced. Moreover, the high-gamma phase coupling between PM and M1 depends on the type of movement performed and could, therefore, potentially be used as a neuronal control signal for brain-machine interfaces.

Acknowledgements

German Federal Ministry of Education and Research (01GQ0420 to BCCN and 01 GQ 0830 to BFNT (AA,CM)), US Army Research Office (W911NF-07-1-0415 (GS), W911NF-08-1-0216(GS)) and the NIH/NIBIB (EB006356 (GS) and EB00856 (GS))

Keywords: Directed coherence, ECoG, Motor cortex, Neurotechnology and brain-machine interface, Synchronization

Conference: BC11 : Computational Neuroscience & Neurotechnology Bernstein Conference & Neurex Annual Meeting 2011, Freiburg, Germany, 4 Oct - 6 Oct, 2011.

Presentation Type: Poster

Topic: neurotechnology and brain-machine interface (please use "neurotechnology and brain-machine interface" as keyword)

Citation: Liao X, Schalk G, Miller KJ, Brunner P, Ojemann JG, Aertsen A and Mehring C (2011). High-gamma coupling between and within human pre- and primary motor cortex during movements. *Front. Comput. Neurosci. Conference Abstract: BC11 : Computational Neuroscience & Neurotechnology Bernstein Conference & Neurex Annual Meeting 2011.* doi: 10.3389/conf.fncom.2011.53.00115

Received: 23 Aug 2011; **Published Online:** 04 Oct 2011.

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Lotka-Volterra equations capture large-scale population activity in balanced random networks

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We study the rate dynamics of a sparsely connected recurrent network comprising excitatory and inhibitory neurons [1,3]. We describe its population dynamics by a system of Lotka-Volterra equations, which represent the mean-field equations for interacting populations of perfect integrators with exponential escape noise [2]. Here, we investigate how well this system of coupled nonlinear differential equations, and variants that can account for the membrane leak, reflects the large-scale dynamics of the network. Specifically, we attempt to identify the parameters of such a system from simulated activity in recurrent networks of leaky integrate-and-fire neurons, assess the goodness-of-fit, and compare the fitted parameters with the values obtained in an analytical approximation.

Previous work on such networks demonstrated that, depending on its parameters, several different activity states are displayed: synchronous regular (SR), asynchronous regular (AR), and asynchronous irregular (AI) activity [1]. The analysis was based on a diffusion approximation of input integration in single-neurons and a self-consistent mean-field description using a PDE-based Fokker-Planck formalism. We found that a bifurcation analysis based on coupled nonlinear ODEs leads to compatible results. In particular, we considered the relative strength of recurrent inhibition as a bifurcation parameter, which changes the excitation-inhibition balance. Another bifurcation parameter is the strength of external input, which is effective to induce AR states if synaptic delays are short.

Our analysis represents a first step toward analyzing the dynamics of more complex “networks of networks” that are implicated in various cognitive abilities of the brain.

Acknowledgements

Support by the German Federal Ministry of Education and Research (BMBF; grant 01GQ0420 to BCCN Freiburg) is gratefully acknowledged.

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Keywords: Integrate and fire model, large scale dynamics, Lotka-Volterra equations, neurons, networks, and dynamical systems

Conference: BC11 : Computational Neuroscience & Neurotechnology Bernstein Conference & Neurex Annual Meeting 2011, Freiburg, Germany, 4 Oct - 6 Oct, 2011.

Presentation Type: Poster

Topic: neurons, networks and dynamical systems (please use "neurons, networks and dynamical systems" as keywords)

Citation: Lagzi F, Cardanobile S and Rotter S (2011). Lotka-Volterra equations capture large-scale population activity in balanced random networks. *Front. Comput. Neurosci. Conference Abstract: BC11 : Computational Neuroscience & Neurotechnology Bernstein Conference & Neurex Annual Meeting 2011.* doi: 10.3389/conf.fncom.2011.53.00167

Received: 22 Aug 2011; **Published Online:** 04 Oct 2011.

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EVENT ABSTRACT

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Efficient spike tests for linear integrate-and-fire neuron models in time-driven simulations

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The characteristics of time-driven simulation are a fixed-size simulation step and a fixed-size communication interval [1]. The former defines update-and-check points, which are the discrete points in time when all neurons update their state variables and check for a super-threshold membrane potential. The latter defines the discrete points in time when all neurons communicate their spikes. The communication interval is a multiple of the simulation step size and limited only by the minimum synaptic transmission delay in the network.

Traditionally, spikes are incorporated, detected and emitted only at the pre-defined update-and-check points. However, the time-driven environment of the simulator NEST [2] provides an 'off-grid' framework that enables spikes to be be incorporated and emitted at any point in time [3,4]. For each neuron the arrival times of incoming spikes introduce additional update-and-check points.

As the detection of a threshold crossing can only take place at the next check point, time-driven simulation still bears the risk of missing a threshold crossing: a very brief excursion of the membrane potential above threshold may not be detected. This problem is more pronounced in networks with low connectivity and strong coupling as well as in the case of low firing rates.

Here, we investigate spike tests of increasing complexity and specificity that can supplement the standard test for a super-threshold membrane potential at each check point and that guarantee the detection of all threshold crossings. Firstly, we determine the specificities of simple sifting methods for a range of input scenarios. Secondly, we compare the performances of complex spike tests which faithfully indicate the existence of a threshold crossing between the last and the current check point. This stepwise analysis enables us to identify a cascade of tests which locates all threshold crossings at a low computational cost.

Acknowledgements

Partially funded by BMBF Grant 01GQ0420 to BCCN Freiburg, EU Grant 15879 (FACETS), EU Grant 269921 (BrainScaleS), the Helmholtz Alliance on Systems Biology (Germany), the Next-Generation Supercomputer Project of MEXT (Japan), Neurex, and the Junior Professor Program of Baden-Württemberg.

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Keywords: dynamical systems, networks, neurons, precise spike times, simulation technology, time-driven simulation

Conference: BC11 : Computational Neuroscience & Neurotechnology Bernstein Conference & Neurex Annual Meeting 2011, Freiburg, Germany, 4 Oct - 6 Oct, 2011.

Presentation Type: Poster

Topic: neurons, networks and dynamical systems (please use "neurons, networks and dynamical systems" as keywords)

Citation: Kunkel S, Helias M, Diesmann M and Morrison A (2011). Efficient spike tests for linear integrate-and-fire neuron models in time-driven simulations. *Front. Comput. Neurosci. Conference Abstract: BC11 : Computational Neuroscience & Neurotechnology Bernstein Conference & Neurex Annual Meeting 2011.* doi: 10.3389/conf.fncom.2011.53.00049

Received: 23 Aug 2011; **Published Online:** 04 Oct 2011.

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EVENT ABSTRACT

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Mechanisms of generation and suppression of oscillations in the basal ganglia

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Movement disorders in Parkinson's disease (PD) are commonly associated with slow oscillations and increased synchrony of neuronal activity in the basal ganglia. The neural mechanisms underlying this dynamic network dysfunction, however, are only poorly understood. Here, we show that the strength of inhibitory inputs from striatum to globus pallidus external (GPe) is a key parameter controlling oscillations in the basal ganglia. Specifically, the increase in striatal activity observed in PD is sufficient to unleash the oscillations in the basal ganglia.

This finding allows us to propose a unified explanation for different phenomena: absence of oscillation in the healthy state of the basal ganglia, oscillations in dopamine-depleted state and quenching of oscillations under deep brain stimulation (DBS). These novel insights help us to better understand and optimize the function of DBS protocols. Furthermore, studying the model behavior under transient increase of activity of the striatal neurons projecting to the indirect pathway, we are able to account for both motor impairment in PD patients and for reduced response inhibition in DBS implanted patients.

Acknowledgements

Funded in parts by the German Federal Ministry of Education and Research (BMBF grant 01GQ0420 to the BCCN Freiburg, BMBF GW0542 Cognition, and BMBF 01GW0730 Impulse Control).

Keywords: Basal Ganglia and Deep Brain Stimulation

Conference: BC11 : Computational Neuroscience & Neurotechnology Bernstein Conference & Neurex Annual Meeting 2011, Freiburg, Germany, 4 Oct - 6 Oct, 2011.

Presentation Type: Keynote

Topic: other

Citation: Kumar A, Aertsen A, Rotter S and Cardanobile S (2011). Mechanisms of generation and suppression of oscillations in the basal ganglia. *Front. Comput. Neurosci. Conference Abstract: BC11 : Computational Neuroscience & Neurotechnology Bernstein Conference & Neurex Annual Meeting 2011.* doi: 10.3389/conf.fncom.2011.53.00028

Received: 26 Sep 2011; **Published Online:** 04 Oct 2011.

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EVENT ABSTRACT

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Simultaneous multisite LFP and SUA recordings across the hippocampal formation in a mouse model of epilepsy

Antje Kilias[1, 2, 3*], Ulrich P. Froriep[1, 2, 3], Arvind Kumar[2, 3], Ute Häussler[4], Carola A. Haas[3, 4] and Ulrich Egert[1, 3]

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In temporal lobe epilepsy (TLE), physiological activity within the hippocampal-entorhinal loop is severely altered, visible as recurring epileptiform activity (EA). In many cases, this is accompanied by pathological restructuring of the anatomical substrate, including widespread cell death and aberrant connectivity. These changes, known as hippocampal sclerosis, are thought to underlie EA. However, hippocampal sclerosis is apparent at any time, not only during EA, so it must also have an impact on activity during EA-free periods between epileptic events.

An animal model which reproduces a severe case of focal hippocampal sclerosis, accompanied by recurrent EA, is the intrahippocampal kainate mouse model of TLE. Using this model, we previously showed that the relation of activity in the dentate gyrus (DG) and the entorhinal cortex (EC) is changed during such EA-free periods. In particular, the theta band activity (4-8 Hz) of the DG precedes that in the EC by ~ 25 ms in epileptic mice, whereas both are synchronized under healthy conditions (Froriep et al. 2010).

To investigate the mechanism underlying this delay, a higher spatial resolution of the recordings within the hippocampal formation and the analysis of phase relationships in the local field potential (LFP) between all recording sites are required. In addition, increased temporal resolution by recording the associated single unit activity (SUA) could reveal changes in spike timing with respect to the underlying LFP. A shift in spike timing relative to the local LFP might reflect pathological plasticity whereas spike timings shifted between hippocampal structures would point to altered network properties.

Here, we addressed these questions using newly developed, custom-made silicon multisite electrode probes (Herwik et al. 2009) that facilitate simultaneous acquisition of LFP and SUA on 16 channels throughout the hippocampal formation. Chronic implantation of these probes enabled us to record both from freely behaving epileptic and control mice.

We show that these probes allow for simultaneous LFP and SUA acquisition in the DG, the hippocampus proper and parahippocampal structures and analyzed the relationship of LFP rhythms between all these sites in EA-free periods. Furthermore, we investigate the spike time relation relative to the underlying theta cycle across these locations.

Acknowledgements

Supported by the German Federal Ministry of Education and Research (BMBF, FKZ 01GQ0420, 01GQ0830) and by the Deutsche Forschungsgemeinschaft (DFG, SFB TR3, SFB 780).

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Keywords: brain disease, network dysfunction and intervention

Conference: BC11 : Computational Neuroscience & Neurotechnology Bernstein Conference & Neurex Annual Meeting 2011, Freiburg, Germany, 4 Oct - 6 Oct, 2011.

Presentation Type: Poster

Topic: brain disease, network dysfunction and intervention (please use "brain disease, network dysfunction and intervention" as keyword)

Citation: Kilias A, Froriep UP, Kumar A, Häussler U, Haas CA and Egert U (2011). Simultaneous multisite LFP and SUA recordings across the hippocampal formation in a mouse model of epilepsy. *Front. Comput. Neurosci. Conference Abstract: BC11 : Computational Neuroscience & Neurotechnology Bernstein Conference & Neurex Annual Meeting 2011.* doi: 10.3389/conf.fncom.2011.53.00144

Received: 23 Aug 2011; **Published Online:** 04 Oct 2011.

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Developmental changes of activity in simple biological neuronal networks

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Correlated patterns of neuronal activity mark an early stage of cortical network development [1]. Accompanied by the formation of first functional circuits and neuron assemblies, this process establishes initial computational properties of a network. One open question is how this process is functionally and structurally reflected by individual neurons.

We approached this question by investigating the embedding of single neurons into the spontaneous bursting activity of dissociated networks. Obtained from cortical tissue of newborn rats and seeded on polyethylene iminine-coated microelectrode arrays (MEA), isolated neurons form haphazard networks within days and provide an established model for studying structure-function relations in simple networks [2]. Within the 1st week in vitro, these networks spontaneously generate network-wide bursts with characteristic spatiotemporal propagation patterns that can be recorded extracellularly at defined electrode positions [3]. In addition to sampling population activity with MEAs, we simultaneously recorded individual neuron activity with one or two intracellular patch-clamp electrodes.

Network differentiation caused pruning to a neuron density of max. 2,000 cells/mm2 in matured networks at 4 weeks in vitro. With dual patch-clamp recordings, we determined a local connection probability of 0.5 at pairwise distances of max. 450µm. 40% of these connections were bidirectional, revealing a high and recurrent connectivity of the networks. We found that pairwise correlation of spiking within network bursts (NB) increased with network age and across distance, suggesting an increase of overall connectivity. Within the 4th week in vitro, we found a homogenization of correlation degree reflected by a decrease at shorter and a further increase at longer distances. Pairwise correlation between intra- and extracellularly recorded NB activity was highly dependent on the synchrony of spiking at NB onset and the number of spikes/NB, and only to a minor degree on the spatial distance to NB onset location, suggesting that NB propagation involves inhomogeneities in the underlying network connectivity (e.g. functional clusters, long-range connections) and is not purely based on local propagation. In individual neurons, we detected average EPSP frequencies of 5Hz across all network ages. EPSP amplitude distributions, however, systematically decreased with age to average values of 1mV, indicating that synaptic weights scale with increased connectivity and degree of correlation in the matured networks.

In conclusion, our results suggest that fundamental developmental processes are widely preserved in generic neuronal networks in vitro. The homogenization of correlation together with the scaling of synaptic weights and the overall high connectivity imply that differentiation of a network in lack of functional input as observed in perinatal networks in vivo [4] provides a state in which prospective input can be processed reliably.

Acknowledgements

Clemens Boucsein is thanked for support with intracellular recording techniques.
Technical assistance by Patrick Pauli and Ute Riede is acknowledged.
Funded by the German BMBF (FKZ 01GQ0420) and by the EC (NEURO, No. 12788).

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Keywords: burst, connectivity, development, dissociated network, EPSP, microelectrode array, patch-clamp

Conference: BC11 : Computational Neuroscience & Neurotechnology Bernstein Conference & Neurex Annual Meeting 2011, Freiburg, Germany, 4 Oct - 6 Oct, 2011.

Presentation Type: Poster

Topic: neurons, networks and dynamical systems (please use "neurons, networks and dynamical systems" as keywords)

Citation: Kandler S, Okujeni S, Reinartz S and Egert U (2011). Developmental changes of activity in simple biological neuronal networks. *Front. Comput. Neurosci. Conference Abstract: BC11 : Computational Neuroscience & Neurotechnology Bernstein Conference & Neurex Annual Meeting 2011.* doi: 10.3389/conf.fncom.2011.53.00042

Received: 23 Aug 2011; **Published Online:** 04 Oct 2011.

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Biologically plausible connectivity features for the initiation and propagation of bursting in neuronal networks

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Neuronal cultures grown on microelectrode arrays (MEA) from dissociated tissue have been established as a useful biological model in the analysis of network dynamics. Present in their dynamics are periods of strongly synchronized spiking by the network, termed 'bursting', which have been demonstrated to contain different motifs and structure, refuting the possibility that they are merely chaotic activity. Of particular interest are the conditions required for bursting to be initiated and propagated throughout the entire network. Within cultures, burst initiation sites can be well characterized in their location and propagation waves display fairly regular patterns of neuron recruitment within the network burst.

Several proposed models of neuronal networks have displayed bursting and depend on the inclusion of a scale-free connectivity. However, the biological basis for this choice of connectivity is not clearly justified for cortical cultures, with evidence from physiological recordings and axonal tracings only providing limited support for such assumptions. Furthermore, scale-free connectivities cannot adequately be reconciled against the emergence of specific areas at the edge of cultures as burst initiation sites. Thus, while it is clear that bursting can be initiated and propagated within biological networks, exactly which features present in their connectivity are responsible for these traits is still undefined.

Thus, we investigate biologically plausible network features that can account for experimentally observed burst initiation and propagation patterns. Specifically, we examine the contribution of long-range connections by assuming a large-scale network of point neurons with a spatially anisotropic connectivity distribution, representing a mature dissociated cortical culture. Using this model and including a simple implementation of synaptic rescaling, we chart how the introduction long-range connections shifts the degree distribution and increases the amount of recurrent activity. Importantly, our model explains the emergence of burst initiation sites and the velocity of burst propagation observed experimentally when evoked by stimulation, when driven with low levels of background activity.

Acknowledgements

This work was supported by the German BMBF (01GQ0420 & FKZ 01GQ0830).

Keywords: bursting, cortical culture

Conference: BC11 : Computational Neuroscience & Neurotechnology Bernstein Conference & Neurex Annual Meeting 2011, Freiburg, Germany, 4 Oct - 6 Oct, 2011.

Presentation Type: Poster

Topic: neurons, networks and dynamical systems (please use "neurons, networks and dynamical systems" as keywords)

Citation: Jarvis S, Kandler S, Rotter S and Egert U (2011). Biologically plausible connectivity features for the initiation and propagation of bursting in neuronal networks. *Front. Comput. Neurosci. Conference Abstract: BC11 : Computational Neuroscience & Neurotechnology Bernstein Conference & Neurex Annual Meeting 2011.* doi: 10.3389/conf.fncom.2011.53.00048

Received: 23 Aug 2011; **Published Online:** 04 Oct 2011.

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Single-and Multi-unit recordings in humans at the Neurozentrum of the Albert Ludwigs University Freiburg

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Between April 2007 and May 2011 we implanted 25 patients (6 male and 19 female) with hybrid Macro-and Microelektrodes, so-called Behnke-Fried (BF) electrodes, at the Neurozentrum in Freiburg. While recordings of field potentials with intracerebral depth elctrode was indicated in these patients in order to localise their primary seizure focus, we could record activity from either Muti-units (MUA) or from Single-Units (SUA) in 22 out of 25 patients. The average recording time was 10,12 days (5 to 23 days). In 3 patients, each implanted only with a single BF electrode, failure of the microwire bundle lead to the complete absence of MUA or SUA. In contrast, the average failure rate was 19,5% (17 out of 87 BF electrodes) regarding all implanted patients. While none of the patients suffered from any complication of neurological significance, 3 of the patients revealed minor bleedings at a single site, presumably due to the implantation of the macroelectrode.. Failure to reach the brain area, targeted during presurgical planning occurred during implantation of 2 Behnke-Fried electrodes in 2 distinct patients.
In 18 out of 25 implanted patients we counted the number of putative single-as well as multi-units mainly during a single recording session. This count revealed a total number of 550 units (both SU and MUs) for 52 functional bundles, each composed of 8 recording wires. Hence the average unit yield was 0,756 units per recording channel in these 18 patients. Furthermore the average discharge frequency was 0,917 Hz for SUA, but only 0,835 for MUA.

Acknowledgements

This work was funded by the BFNT-B3 and by the SFB780

Keywords: Behnke- Fried electrodes, dynamic systems, human, multi-unit activity, networks, neurons, single unit activity, single-unit recordings

Conference: BC11 : Computational Neuroscience & Neurotechnology Bernstein Conference & Neurex Annual Meeting 2011, Freiburg, Germany, 4 Oct - 6 Oct, 2011.

Presentation Type: Abstract

Topic: neurons, networks and dynamical systems (please use "neurons, networks and dynamical systems" as keywords)

Citation: Hefft S, Hamid O, Brandt A, Schulze-Bonhage A and Trippel M (2011). Single-and Multi-unit recordings in humans at the Neurozentrum of the Albert Ludwigs University Freiburg. *Front. Comput. Neurosci. Conference Abstract: BC11 : Computational Neuroscience & Neurotechnology Bernstein Conference & Neurex Annual Meeting 2011.* doi: 10.3389/conf.fncom.2011.53.00153

Received: 22 Aug 2011; **Published Online:** 04 Oct 2011.

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EVENT ABSTRACT

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Septotemporal position in the hippocampal formation determines epileptic activity in Temporal Lobe Epilepsy

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Temporal Lobe Epilepsy (TLE) is associated with severe changes in cellular architecture of the hippocampus: cell loss in CA3, CA1 and the hilus, granule cell dispersion, mossy fiber sprouting, altered neurogenesis and gliosis. It is, however, still unclear how these changes contribute to the occurrence of epileptic seizures, in particular since some are supposed to have an anti-epileptic effect while others increase the excitability. In our approach, we characterize the strength and extent of epileptiform activity and then reconstruct the histological pattern at the respective positions along the septotemporal axis of the hippocampus to characterize potential interrelation.

To this end, we used the intrahippocampal kainate mouse epilepsy model, which recapitulates the main characteristics of TLE in humans: recurrent focal seizures, granule cell dispersion and selective cell death in the hippocampus. Following the focal injection of kainate into the hippocampus, we performed multi-site in vivo local field potential recordings along the septotemporal axis of the kainate-injected and in the contralateral hippocampus and quantified the strength of status epilepticus (SE) and recurrent epileptiform activity (EA). In addition, we used bromodeoxyuridine injections to monitor proliferative activity, immunohistochemistry and in situ hybridization to determine cell fate and interneuron loss and Nissl staining to measure granule cell dispersion and quantified all parameters.

We show that following kainate injection into the septal hippocampus, SE extended along the septotemporal axis of the hippocampus with stronger intensity at intermediate and temporal sites. Comparably, the intensity of recurrent EA was strongest in the intermediate hippocampus. The histological changes also showed septotemporal gradients: (1) Granule cell dispersion was strong in the septal hippocampus and ceased in the intermediate and temporal hippocampus. (2) Neurogenesis was completely lost in the septal hippocampus, but was strongly increased in the intermediate and temporal hippocampus. (3) Inhibitory interneurons were mostly lost in the septal hippocampus, still reduced in the intermediate hippocampus and back to normal numbers temporally. Notably, the site with strongest EA appeared to be the transition zone where neurogenesis reappeared but interneuron numbers were still reduced.

Therefore, we assume that the occurrence of strong EA requires increased excitation through the addition of hyperexcitable young granule cells and, in addition, decreased inhibition through the loss of inhibitory interneurons. In contrast, each change on its own has only minor effects.

Acknowledgements

Supported by the Deutsche Forschungsgemeinschaft (DFG, SFB TR3 and SFB780) and the Federal Ministry of Education and Research (BMBF FKZ 01GQ0420, 01GQ0830).

Keywords: Epilepsy, Epileptiform activity, Granule cells, Hippocampus, Inhibition, Interneurons, Neurogenesis

Conference: BC11 : Computational Neuroscience & Neurotechnology Bernstein Conference & Neurex Annual Meeting 2011, Freiburg, Germany, 4 Oct - 6 Oct, 2011.

Presentation Type: Poster

Topic: brain disease, network dysfunction and intervention (please use "brain disease, network dysfunction and intervention" as keyword)

Citation: Häussler U, Marx M, Bielefeld L, Froriep UP, Wolfart J and Haas CA (2011). Septotemporal position in the hippocampal formation determines epileptic activity in Temporal Lobe Epilepsy. *Front. Comput. Neurosci. Conference Abstract: BC11 : Computational Neuroscience & Neurotechnology Bernstein Conference & Neurex Annual Meeting 2011.* doi: 10.3389/conf.fncom.2011.53.00222

Received: 11 Aug 2011; **Published Online:** 04 Oct 2011.

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A Model of human dichromat color vision that may help to explain the evolution of trichromacy

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In humans, the basis of color vision is established by the cone photoreceptor system, which consists of three spectrally different cone photoreceptor types termed short (S), middle (M), and long (L) wavelength sensitive, respectively . In established models of color vison, signals from these cones are encoded in two opponent systems. One system compares messages from L and M photoreceptors, and signals in this system are assumed to result in color percepts of red and green. Another system compares signals from S cones with a combination of L and M cones and is thought to support percepts of blue and yellow.
In so-called red-green blind humans, either the M or the L cone type is missing and they are unable to discriminate certain colors that appear red and green to the color normal observer. Nevertheless these dichromats use the terms “red” and “green” in a consistent and meaningful way to describe their color percepts - a surprising finding considering that it is commonly assumed that the basis for their red-green color vision is an opponent combination of M and L cone signals.
We present a model for color vision that is consistent with the neurobiological processing of cone signals and accounts for the mapping of receptor excitation towards color percepts both in human dichromats and trichromats.
In the early primate visual system, cone signals are processed by neurons with ON- and OFF center-surround receptive fields. Following this pattern, our dichromat model combines the outputs of either M-cones (protanope case) or L-cones (deuteranope case) with their surround via horizontal cells, with subsequent rectifying into parallel ON- and OFF-midget pathways (Martin, 1998). Additionally, we consider luminance dependent input from a receptor sensitive in the shorter wavelength range, such as the rods (Lee et al.,1997).
Our model postulates, that in protanopes MON signals towards “green”, and MOFF towards “red”; likewise in deuteranopes, LON signals towards “green”, and LOFF towards “red”.
In a second stage of the model, two types of cortical cells receive and subtract signals from several adjacent M/LON– and M/LOFF-neuron. Again the summed OFF responses signal towards red the summed on responses towards green.
The blue-yellow mechanism in our model also assumes center-surround formation through horizontal cells, rectification into ON and OFF channels and a contribution of the rods with center like sign. Thereby a S dominated signal in the center is opponently connected with a mixed LM surround. Additionally, and in line with experimental findings, we propose a luminance dependent non-linearity that increases L+M-signals at higher luminance.
The model is able to predict color naming as measured with the hue scaling method in both human dichromats and trichromats. It also explains intensity dependent hue changes such as the well established Bezold-Brücke effect.
Assuming a processing similar to our dichromat scheme also for our dichromatic ancestors, provides an explanation for how the addition of a third cone opsin could lead to trichromacy and direct use of the additional spectral information.

Acknowledgements

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

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Keywords: Color vision model, Dichromacy, sensory processing, Trichromacy

Conference: BC11 : Computational Neuroscience & Neurotechnology Bernstein Conference & Neurex Annual Meeting 2011, Freiburg, Germany, 4 Oct - 6 Oct, 2011.

Presentation Type: Poster

Topic: sensory processing (please use "sensory processing" as keyword)

Citation: Garbers C, Wachtler T and Hertel R (2011). A Model of human dichromat color vision that may help to explain the evolution of trichromacy. *Front. Comput. Neurosci. Conference Abstract: BC11 : Computational Neuroscience & Neurotechnology Bernstein Conference & Neurex Annual Meeting 2011.* doi: 10.3389/conf.fncom.2011.53.00138

Received: 23 Aug 2011; **Published Online:** 04 Oct 2011.

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EVENT ABSTRACT

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Inverted theta-gamma coupling in a model of temporal lobe epilepsy

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We recently found that local field potential activity is phase shifted between the dentate gyrus (DG) and the entorhinal cortex (EC) in the kainate mouse model of temporal lobe epilepsy (TLE) (Froriep et al. 2010). In this model, features of human TLE, including recurrent epileptiform activity (EA) and major anatomical changes are reproduced by focal injection of kainic acid into the DG. Interestingly, the phase shift occured in episodes without any visible signs of EA in theta and alpha frequencies (4-12 Hz) but disappeared in higher frequency bands. This is likely to be the case as theta activity is considered a global rhythm, like a neural pacemaker, whereas higher frequencies reflect more local activity and synchronize under certain contiditions only. However, as the coupling of gamma band activity to a specific phase of the underlying theta cycle is a prominent feature of the hippocampal activity and the theta activity is shifted between DG and EC, local gamma activity could occur at a different phase of theta in epileptic animals as well.
We tested this hypothesis by analysing the average power of gamma activity with respect to the theta wave in the DG in activity between epileptic events. We chose the DG because its anatomical structure has changed whereas the EC is preserved in the kainate mouse model. We show that high gamma activity (70-140 Hz) is in fact altered under epileptic conditions. In particular, the highest gamma power in healthy control animals occurs at the trough of a theta cycle in the DG whereas in comparable activity from epileptic animals, it occurs at the peak. As gamma activity has been associated with fast inhibition, our finding suggests wrong timing of inhibition in the dentate gyrus of epileptic animals, which could lead to EA.

Acknowledgements

Supported by the German Federal Ministry of Education and Research (BMBF, FKZ 01GQ0420, 01GQ0830) and by the Deutsche Forschungsgemeinschaft (DFG, SFB TR3, SFB 780).

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Keywords: brain disease, network dysfunction and intervention

Conference: BC11 : Computational Neuroscience & Neurotechnology Bernstein Conference & Neurex Annual Meeting 2011, Freiburg, Germany, 4 Oct - 6 Oct, 2011.

Presentation Type: Poster

Topic: brain disease, network dysfunction and intervention (please use "brain disease, network dysfunction and intervention" as keyword)

Citation: Froriep UP, Kilias A, Kumar A, Haas CA and Egert U (2011). Inverted theta-gamma coupling in a model of temporal lobe epilepsy. *Front. Comput. Neurosci. Conference Abstract: BC11 : Computational Neuroscience & Neurotechnology Bernstein Conference & Neurex Annual Meeting 2011.* doi: 10.3389/conf.fncom.2011.53.00113

Received: 23 Aug 2011; **Published Online:** 04 Oct 2011.

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A PYTHON Package for Kernel Smoothing via Diffusion: Estimation of Spike Train Firing Rate

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Kernel smoothing is a powerful methodology to gain insight into data. It has wide applications in many different fields, ranging from Economics to Neurosciences. The most important basic application of kernel smoothing in Neuroscience is estimation of time-dependent firing rates from neuronal spike trains. Traditionally, this is achieved by the PSTH (Peri-Stimulus Time Histogram) or, alternatively, smoothing with a fixed kernel. The PSTH relies on the availability of multiple trials for averaging out trial-to-trial fluctuations. However, one can obtain a plausible estimate from a single trial as well, using kernel smoothing methods, where the bandwidth of the kernel is a parameter to be selected in analogy to the bin size of the histogram. The form of the kernel is rather unimportant, provided it is smooth, unimodal and normalized. Its bandwidth, in contrast, defines how smooth the resultant rate would be (Nawrot et al., 1999). A suboptimal kernel may result in over-smoothing or under-smoothing, where the optimal kernel is defined by a minimal deviation from the true rate profile. There may be no globally optimal kernel for strongly changing Poisson rates, though. As a cure to this problem one can optimize the estimate by locally adaptive bandwidth selection. To this end, Shimazaki and Shinomoto (2009) suggested a combinatorial way of optimizing MISE (mean square integrated error) as a method of local bandwidth estimation. This method, although effective, is computationally very costly and biased. Instead, we suggest an application of a new method by Botev et al. (2010), namely Kernel Density Estimation via Diffusion. The diffusion method offers a fast completely data driven algorithm for local bandwidth selection, avoiding the boundary bias and the assumption of Gaussianity. An implementation of the new method as a PYTHON package is made available.

Acknowledgements

Funding by the German Ministry of Education and Research (Bernstein Focus Neurotechnology Freiburg*Tübingen, FKZ 01 GQ 0830) is gratefully acknowledged.

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Keywords: Data Smoothing, Firing Rate, Kernel Density Estimation, PYTHON

Conference: BC11 : Computational Neuroscience & Neurotechnology Bernstein Conference & Neurex Annual Meeting 2011, Freiburg, Germany, 4 Oct - 6 Oct, 2011.

Presentation Type: Poster

Topic: data analysis and machine learning (please use "data analysis and machine learning" as keyword)

Citation: Deniz T, Cardanobile S and Rotter S (2011). A PYTHON Package for Kernel Smoothing via Diffusion: Estimation of Spike Train Firing Rate. *Front. Comput. Neurosci. Conference Abstract: BC11 : Computational Neuroscience & Neurotechnology Bernstein Conference & Neurex Annual Meeting 2011.* doi: 10.3389/conf.fncom.2011.53.00071

Received: 23 Aug 2011; **Published Online:** 04 Oct 2011.

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EVENT ABSTRACT

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Cooperative structural plasticity based on pre- and postsynaptic spike timing

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The structure of networks of mammalian neocortical neurons is subject to continuous remodeling. Synapse formation and pruning, however, do not occur randomly [1]. Rather, the relative abundance of multiple-synaptic connections between neurons indicates some cooperation [2]. It is yet unclear by which mechanism this cooperation is achieved. By extending current models of spine plasticity [1,3] we investigate whether spike timing dependent structural plasticity can explain the experimentally obtained distributions of synapse multiplicity [2]. Indeed, although assuming only generic mechanisms which might plausibly be realized locally in the dendritic spine, the model's spike timing dependence can explain cooperative formation and pruning of synapses. Furthermore, the study provides new insights into the possible functional role of silent synapses for structural plasticity, as well as an explanation for the existence of both transient and persistent dendritic spines.

Acknowledgements

Supported by BMBF grant 01GQ0420 to BCCN Freiburg.

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Keywords: learning and plasticity, multiple synapses, spike timing dependence, structural plasticity, synapse formation and pruning

Conference: BC11 : Computational Neuroscience & Neurotechnology Bernstein Conference & Neurex Annual Meeting 2011, Freiburg, Germany, 4 Oct - 6 Oct, 2011.

Presentation Type: Poster

Topic: learning and plasticity (please use "learning and plasticity" as keyword)

Citation: Deger M, Helias M, Rotter S and Diesmann M (2011). Cooperative structural plasticity based on pre- and postsynaptic spike timing. *Front. Comput. Neurosci. Conference Abstract: BC11 : Computational Neuroscience & Neurotechnology Bernstein Conference & Neurex Annual Meeting 2011.* doi: 10.3389/conf.fncom.2011.53.00095

Received: 23 Aug 2011; **Published Online:** 04 Oct 2011.

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Computational modeling and interpretation of epileptic activities

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Electrophysiological recordings play a crucial role in epilepsy treatment and research. To capture the neural mechanisms involved in the generation and propagation of epileptic activities, many recording techniques have been developed, allowing for the observation of neural activity at multiple spatial scales: microscopic (single neurons), mesoscopic (local networks), and macroscopic (global networks). The interpretation of these multiple observations is a key but complex issue. It requires the characterization of the relationships between recorded signals and the underlying neural activity, as well as the inter-relation of the different types of signals. Computational modeling may provide useful tools for addressing these issues. In particular, over recent years, new modeling approaches have been developed which allow for the simulation of electrophysiological signals from ensemble neuronal activity. They combine quantitative descriptions of neuronal activity in neural network models with biophysically inspired modeling of the field potentials recorded by electrodes from such network models (forward modeling). The approach allows for studying, on the one hand, the relationship between recorded signals and the underlying spatio-temporal organization of neuronal sources and, on the other hand, the relations between different types of recordings (LFP, ECoG, EEG, etc.). Two example studies using this modeling approach are shown.

In the first study, at a macroscopic level, the model is used to examine the impact of source-related parameters (source area, location, and synchrony) on the properties of epileptiform activities (interictal spikes) in depth-EEG and scalp-EEG signals, and to relate scalp activities to the underlying intracerebral field potentials. In the second study, at a microscopic level, the model is used to investigate the relationship between correlations in individual neurons' spiking activities and properties (amplitude spectrum, amplitude distribution) of resulting LFP signals, and to relate the different network activity states to the LFP profiles recorded in these network states.

Acknowledgements

Funding: Work supported by the French Ministry of Research (ACI "Neurosciences Intégratives et Computationnelles", 2002-2006) and by the German Federal Ministry of Education and Research (BMBF grants 01GQ0420 to BCCN Freiburg and 01GQ0830 to BFNT Freiburg/Tübingen).

Keywords: Dysfunctional Networks: Epilepsy

Conference: BC11 : Computational Neuroscience & Neurotechnology Bernstein Conference & Neurex Annual Meeting 2011, Freiburg, Germany, 4 Oct - 6 Oct, 2011.

Presentation Type: Keynote

Topic: other

Citation: Cosandier-Rimélé D and Cosandier-Rimélé D (2011). Computational modeling and interpretation of epileptic activities. *Front. Comput. Neurosci. Conference Abstract: BC11 : Computational Neuroscience & Neurotechnology Bernstein Conference & Neurex Annual Meeting 2011.* doi: 10.3389/conf.fncom.2011.53.00018

Received: 26 Sep 2011; **Published Online:** 04 Oct 2011.

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Stochastic models of recurrent neural networks

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Stochastic models of neural activity have a long standing tradition in the neuroscientific research, especially with regards to analysis of neural data. Analysis of recurrent neural networks models, in contrast, has been proven difficult. The main tools in the analysis are Fokker-Planck equations. Fokker-Planck theory have made possible to study stability properties and, at least partially, correlations in recurrent neural networks of leaky integrate-and-fire neurons.

Here we report on recent progresses regarding rate based, non Gaussian models of spiking neural networks. We address two different types of models: linear and nonlinear. The prototype for linear models of recurrent spiking networks are the Hawkes networks. In Hawkes networks, input spikes produce a linear transient in the rate of the post-synaptic neuron. Transients caused by different spikes superimpose linearly. A complete theory for pairwise correlations in Hawkes networks exist and can be exploited to study the links between structural properties and dynamical properties of networks.

Nonlinear models exist in several different forms and variants. We describe here multiplicatively interacting point processes and their connection to Lotka-Volterra equations, a type of equations that have been extensively used in the neuroscientific literature, based on phenomenological considerations. In recurrent networks of multiplicatively interacting processes, input spikes have a multiplicative effect on the rate of the post-synaptic neuron. They are linear in the logarithm of the rate and correspond to non leaky integrate-and-fire neurons with exponential escape noise. Recurrent networks of such neurons naturally display complex properties like multistability and chaos and can be used to construct networks with contrast invariant input-output tuning.

Acknowledgements

Supported by the German Federal Ministry of Education and Research, grant 01GQ0420 "BCCN Freiburg", and grant 01GW0730 "Impulse Control".

Keywords: Neural Dynamics in Cortical Networks

Conference: BC11 : Computational Neuroscience & Neurotechnology Bernstein Conference & Neurex Annual Meeting 2011, Freiburg, Germany, 4 Oct - 6 Oct, 2011.

Presentation Type: Keynote

Topic: other

Citation: Cardanobile S (2011). Stochastic models of recurrent neural networks. *Front. Comput. Neurosci. Conference Abstract: BC11 : Computational Neuroscience & Neurotechnology Bernstein Conference & Neurex Annual Meeting 2011.* doi: 10.3389/conf.fncom.2011.53.00017

Received: 26 Sep 2011; **Published Online:** 04 Oct 2011.

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EVENT ABSTRACT

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NeurOnline: A software to perform online analysis and control of electrophysiological recordings

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Project Summary
It is now standard to record the activity from large numbers of neurons simultaneously, both in behaving animals using acute or chronically implanted electrode arrays, and in brain slices or tissue cultures using substrate-integrated multi-electrode arrays. To enable control and intervention in an ongoing experiment, it is important to monitor certain critical parameters of the recorded activity in real-time. NeurOnline is a software that enables the researcher to perform online analysis of their electrophysiological recordings and to suitably interact with their experimental setups based on these analysis results. Specifically, our software supports optimizing the yield of experiments by providing new algorithms for online analysis that give comprehensive feedback about the status of the experiment in real-time. In addition, we hope to stimulate the development of novel experiments based e.g. on the possibility of fast adaptation of applied stimuli depending on the behavior of the system studied. The software is made publicly available under GPL.

Technical details
The software architecture chosen for this project consists in Python scripts that call C++ extensions provided by SIP. The C++ language allows high-performance computations that are crucial for time-critical “online” analyses and an easy use of multiple threads. The Python scripting language, on the other hand, enables experts and semi-skilled programmers at the same time to easily use and extend the envisaged software toolbox. The QT library is used to implement a signal/slot mechanism. A graphical user interface permits to control the analysis and displays the results of all computations performed on the recorded data.

Based on a set of open source drivers (“comedi”), NeurOnline can currently interact with 400 different data acquisition boards (e.g. National Instruments) that are commonly employed in electrophysiological setups. A TCP/IP client has been implemented to allow the communication with high-density multi-electrode arrays (HD-MEAs) currently developed at ETH Zurich/Basel. These devices currently allow sampling from 128 channels (selected from a set of 11,000) at a sampling frequency of 20 kHz per channel. Acquisition from Multichannel Systems multi-electrode arrays is also supported.

A butterworth IIR online filter has been developed to select the appropriate frequency bands of the recorded signals. Online spike sorting is performed on the detected spike waveforms by applying a dynamic template matching algorithm. The Python interface allows NeurOnline to send signals that depend on the result of online signal analysis to other processes, e.g. to update in real-time the visual stimulus displayed by some dedicated software (e.g. “VisionEgg”).

NeurOnline is currently used in two laboratories: At the Biomicrotechnology laboratory (IMTEK, University of Freiburg) the dynamics of dissociated cell cultures grown on HD-MEAs are studied. Those arrays have 11,000 recording sites, 128 of which can be recorded at the same time. NeurOnline is currently used to record the data from subsets of electrodes, to detect the spikes in the recorded signals and to organize the scan of the full set of electrodes depending on the recorded activity. At the Neurobiology and Biophysics laboratory (Faculty of Biology, University of Freiburg), extra- and intra-cellular recordings in the neocortex of anesthetized rats are performed. Visual stimuli that preferentially activate neurons in the thalamus (LGN) and in the primary visual cortex (V1) are selected by a method known as adaptive sampling. NeurOnline used in this laboratory for multi-channel signal recording, spike detection, spike sorting and visual stimulus updating.

Acknowledgements

Funding by the German Ministry of Education and Research (Bernstein Focus Neurotechnology Freiburg*Tübingen, FKZ 01 GQ0830) is gratefully acknowledged.

Keywords: data analysis, Electrophysiology, Online analysis

Conference: BC11 : Computational Neuroscience & Neurotechnology Bernstein Conference & Neurex Annual Meeting 2011, Freiburg, Germany, 4 Oct - 6 Oct, 2011.

Presentation Type: Poster

Topic: data analysis and machine learning (please use "data analysis and machine learning" as keyword)

Citation: Ambard M, Brandt A and Rotter S (2011). NeurOnline: A software to perform online analysis and control of electrophysiological recordings. *Front. Comput. Neurosci. Conference Abstract: BC11 : Computational Neuroscience & Neurotechnology Bernstein Conference & Neurex Annual Meeting 2011.* doi: 10.3389/conf.fncom.2011.53.00201

Received: 22 Aug 2011; **Published Online:** 04 Oct 2011.

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