

## AKB 10 Neuroscience

Time: Tuesday 11:30–13:30

Room: ZEU 255

**Invited Talk**

AKB 10.1 Tue 11:30 ZEU 255

**Synaptic Plasticity and Memory from an Optimality Viewpoint** — ●WULFRAM GERSTNER<sup>1</sup>, TARO TOYOIZUMI<sup>1,2</sup>, JEAN-PASCAL PFISTER<sup>1</sup>, and KAZUYUKI AIHARA<sup>2</sup> — <sup>1</sup>Ecole Polytechnique Federale de Lausanne, EPFL — <sup>2</sup>University of Tokyo

Connections between neurons change and these changes (potentiation and depression of synapses) are thought to be the basis of learning and memory. Despite a diversity of experimental facts, we believe that the rule controlling changes of synapses should be simple. More precisely, we studied the hypothesis that synaptic dynamics is controlled by three basic principles:

(A) Synapses adapt their efficacies so that neurons can effectively transmit information;

(B) homeostatic processes stabilize the mean firing rate of the postsynaptic neuron; and

(C) weak synapses adapt more slowly than strong ones while maintenance of strong synapses is costly.

Our results show that a synaptic update rule derived from these principles depends on spike timing, is sensitive to correlations in the input, and is useful for synaptic memory.

AKB 10.2 Tue 12:00 ZEU 255

**The role of inhibitory feedback for information processing in thalamocortical circuits** — ●JÖRG MAYER, HEINZ GEORG SCHUSTER, and JENS CHRISTIAN CLAUSSEN — Institut für Theoretische Physik und Astrophysik, Christian-Albrechts Universität, Olshausenstraße 40, 24098 Kiel, Germany

The information transfer in the thalamus is blocked dynamically during sleep or deep anaesthesia, in conjunction with the occurrence of spindle waves. We analyze two modeling approaches for a recent experiment by Le Masson *et al.* on the thalamocortical loop. In a first step, we use a conductance-based neuron model to reproduce the experiment computationally. In a second step, we model the same system by using an extended Hindmarsh-Rose model, and compare the results with the conductance-based model. In the framework of both models, we investigate the influence of inhibitory feedback on the information transfer in a typical thalamocortical oscillator. We find that the extended Hindmarsh-Rose neuron reproduces the experiment better than the conductance-based model. Further, inhibitory feedback leads to stable self-sustained oscillations which mask the incoming input, and thereby reduce the information transfer significantly.[1]

[1] Jörg Mayer, Heinz Georg Schuster, and Jens Christian Claussen, The role of inhibitory feedback for information processing in thalamocortical circuits, arxiv.org e-print q-bio/0510040

AKB 10.3 Tue 12:15 ZEU 255

**Electrical field parameters for the electrical stimulation of isolated neurons on wide planar interfaces** — ●A. REIHER<sup>1</sup>, H. WITTE<sup>1</sup>, A. KRITSCHIL<sup>1</sup>, S. GÜNTHER<sup>1</sup>, A. KROST<sup>1</sup>, A. DE LIMA<sup>2</sup>, and T. VOIGT<sup>2</sup> — <sup>1</sup>Inst. of Experimental Physics, University of Magdeburg, PO Box 4120, 39016 Magdeburg — <sup>2</sup>Inst. of Physiology, University of Magdeburg, Leipziger Str. 44, 39120 Magdeburg

Embryonal neocortical neurons form physiologically active, synaptically interconnected networks after two weeks in vitro. In order to stimulate larger groups of neurons we developed an interdigitated electrode structure for electrical stimulation of the whole network. This interface consists of a planar finger structure of gold on a 5 nm thin Ti-undercoating with a gap of 300  $\mu\text{m}$  between the electrodes exhibiting an overall electrode thickness of 55 nm. Stimulation conditions leading to synchronous network activity were analyzed in [1]. Here we show that the network activity generated by previous stimulation parameters is not due to the direct activation of single neurons, but depends on widespread synaptic interactions. The parameters for the stimulation of single neurons independent from synaptic interactions were analyzed either by inhibiting glutamate and *GABA<sub>A</sub>* receptors in synaptically interconnected networks, or in immature networks with still non-interconnected neurons. The electrical field strength distribution for the interface is calculated with the finite-elements-method. These results are compared with a position dependent analysis of separated firing neurons in order to determine the critical electrical field strength for single neuron stimulation. [1] A. Reiher, et al., Appl. Phys. Lett. 86, 103901 (2005)

AKB 10.4 Tue 12:30 ZEU 255

**Analytical approach to correlation- and feedback-induced oscillations in biological neural networks** — ●BENJAMIN LINDNER<sup>1,2</sup>, BRENT DOIRON<sup>2</sup>, and ANDRE LONGTIN<sup>2</sup> — <sup>1</sup>MPI fuer Physik komplexer Systeme, Noethnitzer Str. 38, 01187 Dresden — <sup>2</sup>Department of\*Physics, University of Ottawa, 150 Louis Pasteur, Ottawa, Canada\*K1N-6N5

A network of leaky integrate-and-fire neurons with global inhibitory feedback and under the influence of spatially correlated noise is studied. We calculate the spectral statistics of the network (power spectrum of the population activity, cross spectrum between spike trains of different neurons) as well as of a single neuron (power spectrum of spike train, cross spectrum between external noise and spike train) within the network. As shown by comparison with numerical simulations, our theory works well for arbitrary network size if the feedback is weak and the amount of external noise does not exceed that of the internal noise. By means of our analytical results we discuss the quality of the correlation-induced oscillation in a large network as a function of the transmission delay and the internal noise intensity. It is shown that the strongest oscillation is obtained in a system with zero internal noise and adiabatically long delay (i.e. the delay period is longer than any other time scale in the system).

REFS.: Doiron, Lindner, Longtin, Maler, Bastian Phys. Rev. Lett. 93, 048101 (2004), Lindner, Doiron, Longtin Phys. Rev. E (2005, in press)

AKB 10.5 Tue 12:45 ZEU 255

**Postsynaptic signaling at the Drosophila neuromuscular junction during development** — ●THOMAS BITTIG<sup>1</sup>, VERONICA DUDU<sup>2</sup>, EUGENI ENTCHEV<sup>2</sup>, ANNA KICHEVA<sup>2</sup>, MARCOS GONZÁLEZ-GAITÁN<sup>2</sup>, and FRANK JÜLICHER<sup>1</sup> — <sup>1</sup>Max Planck Institute for the Physics of Complex Systems, Noethnitzer Strasse 38, 01187 Dresden, Germany — <sup>2</sup>Max Planck Institute for Molecular Cell Biology and Genetics, Pfotenhauer Strasse 108, 01307 Dresden, Germany

Cellular signaling systems play an important role during development to determine cell fates and regulate cellular properties. Muscle development is affected by cell-to-cell communication at the synapse which involves the TGF-beta signaling pathway. Synaptic plasticity and the role of growth factors can be studied in the Drosophila neuromuscular junction (NMJ), which is formed during embryogenesis and grows during larval stages to adjust itself to the growth of the muscle. There TGF-beta signaling is transduced by the phosphorylation of transcription factors in the cytosol, which causes their accumulation in the nucleus where they activate gene expression.

We use a simple kinetic model to describe cytoplasmic and nuclear pools of phosphorylated and non-phosphorylated transcription factors that mediate TGF-beta signaling in the muscle cells. This model is used to interpret the time dependence of the concentrations of these molecules as observed in FRAP (Fluorescence Recovery after Photobleaching) experiments. We determine several rate constants of intracellular signaling and show that synaptic signaling via neural action potentials modulates the developmental signaling via growth factors.

AKB 10.6 Tue 13:00 ZEU 255

**Analysis of the Neuron-Silicon Interface with a 2D Transistor Array** — ●RALF ZEITLER<sup>1</sup>, ARMIN LAMBACHER<sup>1</sup>, ROLAND THEWES<sup>2</sup>, and PETER FROMHERZ<sup>1</sup> — <sup>1</sup>Max Planck Institute for Biochemistry, Department of Membrane and Neurophysics, Martinsried/München — <sup>2</sup>Infineon Technologies, Corporate Research, München

A two-dimensional transistor array with a pitch of 7.8  $\mu\text{m}$  is used to study the electrical interfacing of neurons and silicon chips. The 128x128 array is implemented in extended CMOS technology. The open gates of the sensor transistors are insulated by titaniumdioxide. Identified neurons from *Lymnaea stagnalis* are cultured on the chip. The extracellular voltage in the cell-chip junction is recorded as it is induced by action potentials. The response is not homogeneous in the cell-chip contact, but exhibits dramatic variation in shape and amplitude.

For an interpretation we modelled the ionic membrane current on the basis of literature data and own patch-clamp measurements. Computed action potential shapes agree well with experimental intracellular records. The electrolyte in the narrow cleft between cell and chip was described by a two-dimensional Nernst-Planck equation. By comparing computed

extracellular voltage patterns with experimental data it is possible to draw conclusions about the distribution of ion channels in the adhesion membrane.

AKB 10.7 Tue 13:15 ZEU 255

**Capacitive Stimulation of Neurons on Silicon Chips: Discrimination of Channel Opening and Electroporation** — •FRANK WALLRAPP and PETER FROMHERZ — Department of Membrane and Neurophysics, Max Planck Institute for Biochemistry, Martinsried, Germany

The implementation of neuroelectronic systems requires a stimulation of neurons from silicon chips by capacitive interaction. The displacement current through a dielectric layer may give rise to extracellular and intracellular voltages that enhance the membrane conductance. Two mechanisms are feasible: (i) Opening of ion channels. (ii) Transient electroporation. Here, we show that both processes can be induced capacitively and discriminated with proper protocols of the applied voltage. In the model experiments, we use HEK293 cells, stably transfected with the  $K^+$  channel Kv1.3. We cultured them on  $TiO_2$ -insulated silicon chips. By applying voltage ramps to the chip, we induce an extracellular voltage in the narrow cleft between cell and chip. For a given amplitude of negative voltage ramps, membrane current is solely due to  $K^+$  channels if the slope is beneath a certain threshold. The selectivity is checked with a toxin for Kv1.3. Above the threshold, a current component appears that is not blocked by the toxin. It is also observed for positive voltage ramps above a threshold of the slope. We assign it to electroporation. With respect to neuronal stimulation we conclude that rectangular voltage pulses applied to a capacitor inevitably elicit reversible electroporation. Selective activation of ion channels is achieved with slower voltage ramps of long duration that require a large amplitude and large capacitance.