BP 38: Neurosciences

Time: Wednesday 9:30–11:15 Location: H45

Invited Talk BP 38.1 Wed 9:30 H45

Optogenetics: Basics, Applications and Chances — •Ernst Bamberg — Max Planck Institute of Biophysics Frankfurt

Optogenetics: Basics, Applications and Chances Ernst Bamberg Max-Planck Institute of Biophysics Frankfurt Abstract Optogenetics is the use of genetically encoded light activated proteins for manipulation of cells in an almost noninvasive way by light. The most prominent example is Channelrhodopsin2(ChR2), which allows the activation of electrical excitable cells via the light dependent depolarization. The combination of ChR2 with hyperpolarizing light driven ion pumps as the Cl- pump halorhodopsin (NphR) enables the multimodal remote control of neural cells in culture, tissue and living animals. Optogenetics has revolutionized neuroscience and is applied by more than 1000 Neurobiology oriented groups. Very soon it became obvious that this method will offer also the chance for a gene therapy for some neurodegenerative diseases. The basics of optogenetics and some applications are presented. Possible biomedical applications with the focus on blindness are discussed as well.

Invited Talk BP 38.2 Wed 10:00 H45

The mechanical control of CNS development and functioning — •Kristian Franze — University of Cambridge, Cambridge, UK

Throughout life, central nervous system (CNS) cells migrate and grow over large distances. During development and pathological processes, they are exposed to a multitude of signals determining where to move. Despite the fact that forces are involved in any kind of cell motion, our current understanding of the mechanical interactions of CNS cells and their environment is very limited. We used compliant cell culture substrates, traction force microscopy and calcium imaging to investigate how neurons and glial cells interact with their mechanical environment. Growth and migration velocities, directionality, cellular forces as well as neuronal fasciculation and maturation all significantly depended on substrate stiffness. Moreover, when grown on substrates incorporating linear stiffness gradients, axon bundles turned towards soft substrates while glial cells migrated in the opposite direction. In vivo atomic force microscopy measurements revealed stiffness gradients in developing brain tissue, which axons followed as well towards soft. Interfering with brain stiffness and mechanosensitive ion channels in vivo both led to similar aberrant neuronal growth patterns with reduced fasciculation and pathfinding errors, strongly suggesting that neuronal growth is not only controlled by chemical signals, as it is currently assumed, but also by the tissue's local mechanical properties.

BP 38.3 Wed 10:30 H45

Single-channel current of calcium channels in rat neocortical layer 5 pyramidal neurons at physiological calcium concentration: fluctuation analysis with voltage ramps — •Christian Scheppach[1,2] and Hugh P.C. Robinson[1] — [1]Physiological Laboratory, Department of Physiology, Development and Neuroscience, University of Cambridge, Cambridge, U.K. — [2]Institute of Physics, University of Freiburg, Freiburg, Germany

Voltage-gated Ca^{2+} channels are present in many neuronal membranes, playing important roles in presynaptic transmitter release, and in postsynaptic integration of inputs, e.g. by dendritic Ca^{2+} action potentials. Their single-channel current in physiological extracellular Ca^{2+} concentrations (1-2 mM) is not well known, and is too small to be resolved directly with standard patch-clamp. Yet it is a key parameter e.g. for stochastic effects arising from the random opening and closing of single Ca^{2+} channels. We recorded Ca^{2+} channel currents from neocortical L5 pyramidal neurons and used fluctuation analysis to obtain a single-channel current of 0.07 pA at −25 mV membrane voltage and 2 mM extracellular Ca^{2+}. A novel fluctuation analysis protocol was developed, whereby channel currents are recorded during voltage ramps. The presented technique is robust with respect to unstable experimental conditions like rapid run-down of the channel current. The data on the single Ca^{2+} channel current are relevant to a quantitative understanding of dendritic Ca^{2+} action potentials, especially their stochastic aspects.

Reference: C. Scheppach, H.P.C. Robinson (in preparation).

BP 38.4 Wed 10:45 H45

Emulation of the hippocampal circuit with memristive Hebbian Plasticity — •Nick Diederich[1,2], Annika Hanert[2], Thorsten Bartsch[2], Martin Ziegler[1], and Hermann Kohlstedt[1] — [1]Technische Fakultät, Christian-Albrechts-Universität zu Kiel, Germany — [2]Neurologie, Universitätsklinik Schleswig-Holstein, Germany

The hippocampus is one of the crucial brain areas for learning and consolidation of memory in human brains. In particular, it serves as a classical model for neuroplasticity. Therefore, important plasticity mechanisms such as long-term potentiation and depression have been identified and demonstrated in the hippocampal field. For the description of the working principles of the hippocampus a circuit model has been proposed which is based on auto- and heteroassociative networks. In this talk, a computational neural-network model of the hippocampal circuit is presented. Spiking neurons and memristive Hebbian synapses are employed into Hopfield- and feedforward network structures. The obtained network performance is discussed in the framework of pattern completion and recognition and recent studies of mnemonic processing.

Finanical support by the German Research Foundation through FOR 2093 is gratefully acknowledged.

BP 38.5 Wed 11:00 H45

Correlated activity of periodically driven binary networks — •Tobias Kühn[1], Michael Denker[1], Sonja Grün[1,2], and Moritz Helias[1,3] — [1]Inst. of Neuroscience and Medicine (INM-6), Inst. for Advanced Simulation (IAS-6) and JARA BRAIN Inst. 1, Jülich Research Centre, Germany — [2]Theoretical Systems Neurobiology — [3]Dept. of Physics, both Faculty I, RWTH Aachen University, Germany

Experiments showed that excess synchronous spike events are locked to the phase of LFP beta-oscillations more strongly than spikes not part of such events [Denker et al. 2011, Cereb. Cortex]. To identify the mechanisms by which correlations depend on the phase of the LFP, which primarily reflects input activity, we examine a balanced network of homogeneously connected binary model neurons [Ginzburg et al. 1994, PRE] receiving input from a sinusoidal perturbation. The Glauber dynamics of the network is simulated and approximated by mean-field theory. Treating the periodic input in linear response theory, the cyclostationary first two moments are analytically computed. They agree with their simulated counterparts over a wide parameter range. The zero-time lag correlations consist of two terms, one due to the modulated susceptibility (via the external input and network feedback) and one due to the time-varying autocorrelations. For some parameters, this leads to resonant correlations and non-resonant mean activities. Our results can help to answer the salient question how oscillations in mesoscopic signals and spike correlations interact.

Supported by the Helmholtz foundation (VH-NG-1028, SMHB); EU Grant 604102 (HBP). Simulations with NEST (nest-simulator.org).