Abstract book

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SUBCELLULAR

Poster ID: 1

Name: Lothar Baltruschat, DZNE

Title: Structural correlates of long-term memory formation in the Drosophila mushroom body calyx

Abstract: Memory consolidation is a complex and still elusive phenomenon involving structural and functional modifications in multiple circuits. However, which neuronal connections undergo specific changes associated with consolidation in a natural context has been proven difficult to pinpoint. We report that the establishment of olfactory appetitive long-term memory is accompanied by circuit reorganization in the adult Drosophila mushroom body. Axonal terminals delivering olfactory information form large synaptic complexes with the mushroom body neurons involved in the formation and retrieval of olfactory associative memories. Consolidation of appetitive olfactory memories resulted in an increased number of synaptic complexes formed specifically by the axonal boutons of the neurons conveying the conditioned odour. This protein synthesis-dependent change led to additional functional connections with the postsynaptic mushroom body neurons. Our data reveal a consolidation-associated structural and functional reorganization of identified neurons in the adult fly brain.

Poster ID: 2

Name: L. Niels Cornelisse, VU Medical Center, Amsterdam

Title: Frequency coding depends on suppression of asynchronous release by Synaptotagmin-1.

Abstract: Reliable transfer of firing-rate encoded information depends on synchronous synaptic transmission during sustained activity. We show that the fast Ca2+-sensor synaptotagmin-1 (Syt1) has the ability to suppress activity-induced asynchronous release essential to maintaining a continuous supply of synchronously releasable vesicles during high-frequency stimulation. While in WT neurons, release at near-physiological temperatures can remain synchronous at frequencies up to 40Hz, at room temperature it rapidly desynchronizes. Blocking asynchronous transmission by application of the slow calcium buffer EGTA can only partially rescue sustained release properties. Furthermore, we find that in the absence of Syt1 raising the temperature to near physiological levels does not suppress, but increases the remaining asynchronous component. This indicates that Syt1 independent processes such as faster calcium dynamics are not the dominant mechanism for synchronization of sustained release. However, when mutating Syt1's clamping ability through fixing the C2 domain linker, specifically the replenishment of the synchronous

component is reduced, and temperature dependent synchronization of release is impaired. Overall, we conclude that the release clamping function of Syt1 aids in maintaining a sufficient supply of synchronously releasable vesicles, and is crucial in preserving frequency-coded information transfer.

Poster ID: 3

Name: Vincent Huson, Vrije Universiteit Amsterdam, CNCR

Title: Post tetanic potentiation reduces the energy barrier for synaptic vesicle fusion in fast releasing vesicles

Abstract
Before fusion with the plasma membrane synaptic vesicles need to overcome an energy barrier. Regulation of this barrier is a powerful way to control synaptic efficacy, since linear modulation of the barrier leads to supralinear effects on the fusion rate. We hypothesize that modulation of the energy barrier occurs during post tetanic potentiation (PTP) and other forms of short-term synaptic plasticity. Furthermore, we predict that during PTP the energy barrier is reduced in a subset of the readily releasable pool that has a close interaction with the active zone, the fast releasing vesicle pool. To test this hypothesis, we developed a method to assess the activation energy for vesicle fusion and rate constants of vesicle priming, unpriming, fusion and transition to the fast vesicle pool by fitting a vesicle state model to synaptic responses to hypertonic solutions. We show that high frequency stimulation protocols indeed reduce the energy barrier for vesicle fusion. Moreover, by fitting evoked and hypertonic solution induced synaptic responses simultaneously, we found that reduction of the energy barrier in the fast releasing vesicle pool could explain the observed potentiation of synaptic responses, whereas an energy barrier reduction in the total synaptic vesicle pool could not. Interestingly, our model also provides an explanation for the observed non-linear relationship between the concentration of the hypertonic solution and the effect of PTP and genetic interventions on the energy barrier. In conclusion, this study indicates that PTP mainly increases the fusion willingness of fast releasing vesicles. Further research should focus on providing molecular evidence for this.

Poster ID: 4

Name: Joanna Komorowska, University of Bonn

Title: Molecular and behavioral consequences of the cannabinoid receptor 2 deletion

Abstract: Endocannabinoids are synthesized upon calcium influx into the postsynapse from phospholipid precursors and subsequently bind to cannabinoid receptors localized on the

presynapse. Two most abundant cannabinoid receptors in the brain are CB1 (cannabinoid receptor 1, localized primarily on neurons) and CB2 (cannabinoid receptor 2, believed to be mainly localized on glial and immune cells). Both of them are G-protein coupled receptors (GPCRs) that inhibit adenyl cyclase activity causing a decrease in neurotransmitter release. On the contrary to the canonical believe, recent publications showed the presence of the CB2 receptor also on the hippocampal neurons and its functional relevance in the CA2/3 area. Moreover the effects of Cnr2 (CB2 gene) deletion are understudied in comparison to Cnr1. In my project I am assessing the behavioral consequences of the Cnr2 deletion with the use of constitutive and cell-specific knock-out mice. Simultaneously, I am interested in the molecular changes caused by the deletion, especially regarding the inhibitory and excitatory balance in the hippocampal CA2/3 area. Preliminary results show no prominent shift in the balance, but an unexpected increase in the overall levels of Synapsin-1, a general synaptic marker, accompanied by a slight decrease in vesicular glutamate transporter 1 in both CA2 and CA3 areas.

Poster-ID: 5

Name: Dika Kuljis, Carnegie Mellon University

Title: Genetically encoded fluorescence-based tools for synapse quantitation and connectomics.

Abstract: Fluorescence-microscopy based approaches for synapse quantitation in brain tissue facilitate high-throughput mapping of synaptic connections across molecularly defined cells and synapses due to their capacity for rapid volumetric imaging of multiple fluorophores. We developed genetically-encoded, synaptically-targeted reagents to fluorescently label postsynaptic sites to examine the distribution and properties of synapses across four major classes of neocortical neurons: pyramidal (Pyr), parvalbumin (PV)-, somatostatin (SST)-, and vasoactive intestinal peptide (VIP)-expressing. Fluorescent synaptic puncta density distributions were similar across cell types, while fluorescence levels varied within and across cell types. Using Cre-recombinase reporter mouse lines for comprehensive labeling of PV, SST, and VIP inhibitory neurons, we aligned molecularly defined inputs to postsynaptic fluorescent puncta on L2/3 Pyr neurons. PV-assigned synapses dominated perisomatic and most dendritic compartments (~20% of puncta), SST synapses were soma-avoiding and showed a similar density across apical and basal dendritic branches (~10% of puncta), while VIP-synapses were sparsest across all compartments (~3%). Connections between SST neurons, which are electrophysiologically absent, were very rare (~2%). High-throughput synapse quantitation provides a powerful approach to visualize synapse organization and morphology in brain tissue and may be used to track synaptic plasticity across development, learning, and disease states.

Poster-ID: 6

Name: Bhuvaneswari Nagarajan, University of Bonn

Title: Zebrafish CNS myelin protein 36K regulates developmental oligodendrocyte differentiation

Abstract: In contrast to humans and other mammals, zebrafish can successfully regenerate and remyelinate central nervous system (CNS) axons following injury. In addition to the common conserved myelin proteins, 36K is a major component of fish CNS myelin protein, but not found in mammalian CNS. Although 36K is one of the most abundantly identified proteins in zebrafish brain, its function remains unknown. Understanding the function of 36K in fish myelin might help to develop therapeutic strategies for demyelinating diseases like Multiple Sclerosis in humans. We designed translation blocking Morpholinos (MO) to investigate the function of 36K. 36K knockdown larvae were found to have reduced body-length and fewer differentiated OPCs. The phenotypes could be rescued when co-injected with 36k mRNA to which the MO cannot bind anymore, which suggests the specificity of the MO. In 36K knockdown larvae, through a microarray analysis, we further found an upregulation in one of the signal-pathways involved in proliferation and differentiation of spinal cord progenitor cells. Further confirming these observations, reduction in OPC numbers in 36K knockdown larvae could be rescued when treated with an inhibitor that prevents the activation of this pathway. The focus of currently on-going work is, to find how 36K is affecting this signalling pathway, using different biochemical assays. Further studies will have to be carried out also to investigate the role of 36K on OPC differentiation during remyelination, which could open up new strategies for remyelination therapies.

Poster-ID: 7

Name: Till Moritz Schladt, caesar

Title: Gating Of The Proton Channel Hv1

Abstract: The Hv1 proton channel is found in a variety of species ranging from unicellular marine algae to mammals. In humans, Hv1 regulates the intracellular pH and is expressed in various cells, including leukocytes, airway epithelium cells, and macrophages. Although Hv1 belongs to the large family of voltage-gated ion channels, its architecture is unique. “Classical” voltage-gated ion channels (conducting K+, Na+, or Ca2+) contain a voltage-sensing domain (VSD) that is connected to a pore domain (PD). Hv1 contains a VSD, but lacks a PD. Therefore, ion permeation and gating in Hv1 must work differently than in classical voltage-gated ion channels. While key residues that form the selectivity filter of Hv1 and delineate the proton permeation pathway were identified, the opening and closing mechanisms that regulate the proton conduction in response to voltage are not well understood. Recent studies identified voltage-induced conformational changes within the VSD using environmentally sensitive fluorophores. In addition, open-channel blockers that bind Hv1 at the intracellular site were identified, suggesting that Hv1 contains an

intracellular gate. In the present study, I detect conformational changes at the intracellular gate of Hv1 with thiol-specific crosslinkers. I perform inside-out patch-clamp recordings on Hv1 channel mutants that contain pairs of engineered cysteines within the putative intracellular gate. Bifunctional crosslinkers can use the sulfhydryl groups of the cysteines as binding sites. A crosslink can change the proton current, e.g. lead to a block or potentiation of proton current, or altered opening or closing kinetics. At the same time, conformational changes are monitored at the extracellular site of the channel using the patch-clamp fluorometry technique.

Poster-ID: 8

Name: Reinhard Seifert, Caesar

Title: Potassium channel-based optogenetic silencing

Abstract: Optogenetics allows altering the activity of specific cell types with high spatio-temporal precision to control cellular networks or even animal behavior. In contrast to the panoply of excitatory opsins, the performance of inhibitory optogenetic tools is still limited. Hyperpolarizing light-driven chloride or proton pumps, or chloride-conducting channelrhodopsins either require high expression levels and persistent intense illumination, or suffer from paradoxical effects due to unintended changes in ion distribution. Shunting potassium conductances can generate inhibition without large changes in ionic distribution and thus do not show these limitations, but the single available light-gated potassium channel system BLINK1 does not well express in most mammalian cells and has therefore not been widely used. Here we report a two component optical silencer system (TCO silencer) comprising photoactivated adenylyl cyclases (PACs) and the small cyclic nucleotide-gated potassium channel SthK. Activation of the TCO by brief flashes of low intensity blue light causes robust and reversible hyperpolarization for several seconds in cultured cells, cardiomyocytes, and neurons, and allows repetitive, highly efficient and long-lasting silencing of cellular firing with minimal light exposure. Viral expression of the TCO silencer system is well tolerated in mouse and zebrafish, where blue light inhibits neuronal activity in vivo. In combination with red-shifted channelrhodopsins, the distinct photoactivation spectra of PACs allow bimodal, optically independent control of neuronal activity. Our TCO represents a reliable optogenetic silencer system with intrinsic amplification for persistent hyperpolarization by potassium efflux, conferring high operational light sensitivity to neurons and other cells of interest.

Poster-ID: 9

Name: Tomke Stürner, DZNE

Title: Actin Dynamics in Dendritic Arborisation Neurons: A Computational Approach

Abstract: The correct morphology of dendrites is essential for the function of the nervous system. The underlying cytoskeleton defines the shape and dynamics of dendritic branches under the control of complex protein networks. The aim of this study is to elucidate how regulators of the actin cytoskeleton define the diverse characteristic shapes and dynamics of dendritic arbors. Investigating the differentiation of Drosophila larva dendritic arborisation (da) neurons, we previously demonstrated that (WAVE) through recruitment of the Arp 2/3 complex promotes the formation of a branched actin patch at the base of a newly forming branchlet. To elucidate the mechanisms of actin organization following this initial step, we asked which additional actin cytoskeletal proteins are required for branching in these neurons. Here, we focus on the actin nucleators Spire and Capu/Formin2, the bundling factor Singed/Fascin the capping protein Ena/VASP and the depolymerisation factor Twinstar/Cofilin. We found that all these actin regulators play a role, in part exclusively, in the formation of the terminal branchlets of Class III da neurons. The dynamics of the highly actin enriched terminal branchlets of class III da neurons are essential for the neuron's function. By performing in vivo time-lapse recordings and developing quantification methods in the "Trees Toolbox" (http://www.treestoolbox.org/) in Matlab we were able to characterise the phenotypes further. Mutants of the different actin regulators all show reduced number of terminal branchlets. However, probing 44 different dendrite parameters we were able to quantitatively resolve the modified dendrite morphologies. Singed, for example, promotes the stabilization of tight uniparallel actin bundles and, correspondingly, mutants of singed3 show an increased tortuosity in class III terminal branches. With timelapse analysis we attempt to elucidate how the loss of the specific protein leads to the distinct dendritic phenotype. For example, while both spire1/2F or capu1/EE mutant larvae show reduced dynamics, spire1/2F mutant larvae have a significant defect in forming new branches, which goes in hand with the expected function of Spire in actin nucleation. Taken together, we show how a computational approach to dendrite morphology and a model of dendritic branch dynamics can help us not only analyse the function of a protein but also describe the interplay between actin nucleation, bundling, severing and elongation.

CELLULAR

Poster-ID: 10

Name: Therese Alich, University of Bonn

Title: Functional Imaging of Mouse and Human Neurons using a High-Fidelity Genetically Encoded Voltage Indicator

Abstract: Here we present a novel and highly accurate biocompatible sensor to monitor in vitro the functional activity of human iPSC-derived neurons. This hybrid genetically encoded voltage indicator (hGEVI) consists of a FRET pair with a fluorescent eGFP dye molecule anchored to the extracellular leaflet of the neuronal membrane via a GPI anchor and an diazo-benzene quenching molecule, D3. D3 rapidly moves in the membrane in a voltage-dependent fashion thereby quenching or unquenching eGFP fluorescence to generate a voltage response. We previously showed that this sensor follows with high-fidelity changes in membrane potential (MP) and outperforms previous hGEVI approaches by not altering intrinsic membrane properties of the cell. Here we demonstrate that our method enables fast and accurate monitoring of subtly graded subthreshold de- and hyperpolarizing changes in MP evoked by 4 s long chirp pulses. Wide ranging frequencies (10-100 Hz) and amplitudes (2-20 mV) of resting membrane potential (RMP) changes were accurately imaged in murine neuronal cultures. The excellent temporal resolution of the hGEVI is reflected in the < 300 µs time lag between phases of the electrophysiological and fluorescence signals. We also applied this sensor system for detecting neuronal activity in human induced pluripotent stem cell (iPSC) derived neurons. Upon expression of the eGFP-GPI construct, APs and more subtle changes in membrane potential induced by depolarizing and hyperpolarizing current injections could be accurately detected. We expect our method to be particularly useful for non-invasive, fast and deep analysees of neuronal activity in the context of iPSC-based disease modeling.

Poster-ID: 11

Name: Eleonora Ambrad Giovannetti, University of Bonn

Title: Long-range inhibition in the hippocampal system under healthy and Alzheimer's disease-like conditions

Abstract: In the mammalian brain, spatial navigation and episodic memory rely on the same anatomically and functionally dependent brain areas: the hippocampus (HPC) and the medial entorhinal cortex (mEC). These structures are interconnected by long-range inhibitory projections (LRIPs) that mediate disinhibition in both brain regions. It is not yet known whether and how these GABAergic interneurons contribute to spatial memory and/or navigation processes. This gains further relevance in light of crucial features

characterizing Alzheimer's disease (AD), such as the loss of spatial memory and dysfunction of inhibitory interneurons. We propose that LRIPs dysfunction contributes to neurophysiological and cognitive impairments observed in the APPswe/PS1dE9 mouse model of AD. Therefore, our aim is to characterize the activity of the LRIPs during spatial memory processes by means of two-photon calcium imaging and electrophysiological measurements, under healthy and diseased conditions.

Poster-ID: 12

Name: Chaitanya Chintaluri, University of Oxford

Title: Extracellular field potential map of the rat brain.

Abstract: Extracellular potentials serve as a window into the neural processes of the brain. While the origin of these potentials from the transmembrane currents is well understood, much remains to be explored on how the electrical properties of the tissue affect the recorded potentials in the brain volume. For example, white matter tracts which are myelinated along the axons are largely conductive along the axonal directions when compared to across them, further these tracts connect different brain regions in three dimensions complicating any generalization. It is not apparent how a subcortical neural activity spreads, if any, into the laminar probes placed in the cortex or vice versa. To address this, we developed a first of its kind - Finite Element Method (FEM) based forward model for a rat's brain. The model incorporates anisotropic conductivity of the brain obtained using high-resolution diffusion tensor image (DTI) scans. We processed these brain scans to add its anatomical labeling based on the Waxholm Space Atlas of the Sprague Dawley Rat Brain. The cerebrospinal fluid in the ventricles, the reference electrode, and the air surrounding the brain are also included with their respective conductivities. Then we study the placement of a point current source inside the brain region and measure the decay of the potentials generated due to this source in brain's volume. This was repeated for a three-dimensional spatial grid of locations, to reveal an extracellular potential map of the brain with precise atlas labels of brain regions. Next, we placed a morphologically detailed multicompartmental model of the CA1 pyramidal neuron, included as many point current sources, in the CA1 hippocampal region. We studied how a spike from this cell, observed as changes in the transmembrane currents, is revealed as the potentials in different brain regions. To test the next level of detail, we included a network of a detailed multicompartmental model of a thalamocortical column along a cortical normal. Once more, the spread of this network's activity into the subcortical areas is revealed. This framework provides ways to reveal the spread of neural activity due to spatial orientation and the relative position of the electrodes placed in the brain structures while including the distortions due to the white matter conductivity and nontrivial finite volume effects. It also serves as a prescription to design electrodes and where to place them, so as to capture the neural activity from brain regions of interest. It would also help needs of inverse current source density estimations, most of which rely on a forward model.

Poster-ID: 13

Name: Kara A. Fulton, Brown University, National Institutes of Health, Caesar Research Center

Title: Mapping the subtype-specific connectivity of periglomerular cells in the mouse olfactory bulb using correlative serial block-face electron microscopy

Abstract: A dense reconstruction of synaptic connectivity requires high-resolution 3D electron microscopy (EM) data. However, EM data alone typically lacks functional information about the neurons in the data. One way to add functional information to an EM dataset is to fluorescently immunolabel tissue for specific functionally relevant proteins and to optically image the distribution of proteins prior to preparing the tissue for EM. Unfortunately, current immunohistochemistry protocols rely on detergent permeabilization, which significantly compromises tissue ultrastructure and renders such protocols incompatible with high quality EM. By preserving extracellular space in acutely fixed sections, we have developed a permeabilization-free immunolabeling protocol compatible with serial block-face scanning electron microscopy (SBEM), which permits deep antibody penetration and preserves ultrastructure. SBEM, in combination with immunofluorescent labeling of neuron subtypes in the same piece of tissue, allows us to correlate the morphological and physiological properties of neurons through a dense reconstruction of the synaptic connectivity. We are utilizing this approach in the mouse olfactory bulb (OB) to investigate the differences in synaptic connectivity between neurochemically-distinct periglomerular cell (PGC) subtypes and OB principal neurons, mitral (MCs) and tufted cells (TCs), to understand if PGC subtypes have functionally distinct roles in odor processing. Due to the complexity of the circuit, it has been previously difficult to study the functional connectivity using standard recording methods. As a result, the functional roles of the PGC subtypes remain unknown due to a lack of knowledge about how they connect to MCs and TCs. Understanding the connectivity of PGC subtypes with MCs and TCs is important for future investigations into the subtype-specific involvement in regulating mitral versus tufted cell activity in the OB. For instance, the connectivity will serve as the foundation for generating a more detailed computational model of the glomerular layer. This experiment will challenge the canonical view of olfactory bulb circuitry by potentially revealing previously unidentified wiring specificity underlying OB computations.

Poster-ID: 14

Name: Mike Guest, Caesar

Title: In vivo structure and function of long-range inputs of pyramidal tract neurons

Abstract: Pyramidal tract neurons (PTs) represent the major output cell type of the neocortex. We have previously shown on the example of the rat barrel cortex that a functional property of PTs is to broadcast cortically processed sensory information to

downstream targets and that PTs relay this information in target related manner. In order to further explain the role of PTs in sensory information processing, we found it is necessary to investigate how this specific neuronal cell type receives sensory information. Therefore this study aims to focus on the structure and function of long range inputs of PTs using the example of the rat barrel cortex. We show that by injecting different subcortical regions with an anterograde virus expressing channel rhodopsin coupled with single cell attached in vivo recordings, allows for both mapping long range inputs to PTs and gives access to their electrophysiological responses during optogentic stimulation of these mapped inputs. By reconstructing the full dendritic morphology of individual recorded PTs and quantifying the number of synapses according to long range input region, we report for the first time the relationship between the 3D distribution of synapses along the dendrites of PTs according to input region and their electrophysiological responses to optogenetic stimulation of these different long range inputs.

Poster-ID: 15

Name: Maksims Ivanovs, Caesar

Title: Cell Type-Specific Organization Of The Rat Vibrissal Motor Cortex

Abstract: In rodents, vibrissal motor cortex (vM1) controls whisking and therefore is involved in the processing of tactile information. Study of interaction between the vM1 and the vibrissal primary somatosensory cortex (vS1) can contribute to the better understanding of sensorimotor integration and decision making. Here, we offer a preliminary classification of neurons in the rat vM1 on the basis of reconstructions of dendritic morphologies of 52 biocytin-labelled neurons. We identified similar types of neurons as previously found in vS1, namely, Layer 2 pyramids (L2py), Layer 3 pyramids (L3py), Layer 4 pyramids (L4py), putative Layer 4 spiny stellates (L4ss), Layer 5 slender-tufted pyramids (L5st), Layer 5 thick-tufted pyramids (L5tt), Layer 6 corticocortical cells (L6cc), and Layer 6 corticothalamic cells (L6ct). To investigate structure-function relationship in the vM1, we analyzed morphological features, spontaneous electrophysiological activity, and electrophysiological responses of vM1 neurons to whisker stimulation. We found a negative correlation between dendrite length and spontaneous activity in L4py, a positive correlation between dendrite length and spontaneous activity in L6ct, a positive correlation between dendrite complexity and pia-soma distance in L5tt, and a positive correlation in all neurons (L4ss excluded) between pia-soma distance and the ratio of apical dendrite length to overall dendrite length. As to responses to whisker stimulation, L2py showed a weak delayed response to the stimulus, L4ss responded to the stimulus by inhibiting their spiking activity, L5st showed no response, and L5tt and L6ct responded to the stimulus actively. We conclude that there are substantial similarities between morphologically defined cell types in vS1 and vM1, whereas observed correlations between morphological features and electrophysiological properties of vM1 neurons provide an insight into structure-function relationships in that brain region.

Poster-ID: 16

Name: Jonas Klußmann, Institute of Experimental Epileptology and Cognition Research

Title: Modulation of Intrinsic Properties of Locus Coeruleus Neurons by Dopamine

Abstract: For a long time the dopaminergic (DA) and the noradrenergic (NE) system were regarded as separate, with a clear separation of labor between the two neuromodulators. However, increasing results are indicating a coupling between the two catecholamine systems. The major nucleus of the NE system located in the locus coeruleus (LC) and the two main nuclei of the DA system located in the substantia nigra (SN) and the ventral tegmental area (VTA) were demonstrated to share projection targets like the prefrontal cortex. Additionally, reciprocal innervations between the VTA and the LC were identified. Nonetheless, a direct proof for the modulatory interaction of the two systems remains to be elucidated. Therefore, the present work focused on the DA modulation of LC neurons. In order to test the modulatory effect of DA on LC neurons perforated patch clamp recordings were performed on mice brain slices with post-hoc immunohistochemical stainings. Application of DA on LC neurons led to a bidirectional modulation. On the one hand a significant inhibition of the spontaneous activity was observed. On the other hand application of DA led to a significant increase of the excitability of LC neurons. However, in the presence of adrenoceptor antagonists application of DA, D1- or D2-like dopamine receptor agonists did not exhibit any significant effect. This indicates that the DA modulation of LC neurons is mediated by a cross reaction of DA and adrenoceptors. Additionally, immunohistological stainings for tyrosine hydroxylase and dopamine transporter (DAT) were performed on DAT-Cre tdTomato reporter mice demonstrating dopaminergic projections within the LC.

Poster-ID: 17

Name: Jinglin Li, Bielefeld University

Title: Adaptation in the visual motion pathway shapes representation of optic flow in aerial insects

Abstract: For volant insects, the retinal image motion (optic flow) elicited by their flight within an environment not only contains information about their self-motion, but also about the spatial layout of the environment with ecologically relevant objects. In insects, optic flow is processed in successive neuropile layers. The neurons in these layers share adaptive response features (i) to compensate for the limited operating range of neurons, (ii) to limit energy costs in generating neuronal responses, (iii) to provide a stable sensing platform even when physical parameters in nature vary widely, and (iv) for robustness against external and internal noise. While adaptation alters the response gain to the signal, some information needs to be retained consistent for behavioral control. By systematically modeling the adaptive motion pathway of the insect visual system based on electrophysiological data on adaptation at different processing layers of blowflies, and simulating the visual input during

flight in virtual and natural 3D environments, we could analyze the impact of adaptation on signal representation under various environmental conditions and flight dynamics. We find that brightness adaptation is essential for robust spatial vision under dynamic environmental conditions. Motion adaptation further enhances foreground-background segregation. Our studies on adaptation in population coding of global flow fields may give insights on what information is important to maintain for downstream neurons. Given the general similarity in the mechanisms of motion computation in a wide range of animal groups, our conclusions are not restricted to the model system of this study.

Poster-ID: 18

Name: Carlotta Martelli, University of Konstanz

Title: Slow presynaptic depression linearly rescales combinatorial odor representations to encode stimulus statistics

Abstract: Animals use the sense of smell to approach or avoid an odor source. While a brief exposure to the stimulus can be sufficient for an animal to identify the odor, information about source location can be inferred only through changes in stimulus intensity or in higher order statistics, as variance and temporal correlations. These properties of the stimulus influence the response of single olfactory neurons, whose combinatorial activity encodes a representation of the odor. Here we asked how the capability of olfactory neurons to temporally integrate and adapt to previous stimuli affects the combinatorial odor representation. To address this question we used in vivo optical imaging of calcium dynamics in the Drosophila Antennal Lobe in response to sustained fluctuating stimuli. We show that olfactory receptor neurons (ORNs) integrate past odor stimuli on glomerulus-specific timescales. Their dynamic properties are fairly concentration-invariant and non-adaptive, and odor representations at the population level are stationary. On the contrary, the responses of olfactory projection neurons (PNs), postsynaptic to ORNs, are adaptive and exhibit a slow (10-20 s) change in activity that linearly rescales odor representations. This adaptation is mediated by a slow presynaptic depression of vesicle release, is calcium independent and independent of inhibitory lateral inputs. In the adapted state the combinatorial population activity of PNs better encode mean and variance of dynamic odor stimuli. Similarly to other sensory modalities, this slow adaptation allows the olfactory system to sample the statistics of the concurrent stimulus and adjust the network response to match the stimulus properties.

Poster-ID: 19

Name: Nicola Masala, Institute of Experimental Epileptology and Cognition Research

Title: Degraded Ca1 Input Feature Detection During Chronic Epilepsy

Abstract: Neuronal dendrites receive thousands of synaptic inputs with the distribution and density of dendritic ion channels determining how they are integrated. A particularly intriguing feature of some dendrites is the capability to generate dendritic supralinear integration via dendritic spikes (Losonczy & Magee JC. Neuron, 2006). In CA1 neurons, D-spikes are elicited by spatiotemporally synchronous input patterns, and have been proposed to subserve input feature detection. The input integration mode depends on the expression of various ion channels and their distribution in the dendrite. However, very little is known regarding altered properties of small calibre dendrites during epilepsy (Royeck & Kelly, J. Neuroscience, 2015; Kelly, Epilepsia, 2017). Moreover, changes in supralinear integrative properties have so far not been addressed, despite the critical importance of D-spikes for neuronal integration. We have therefore set out to study changes in the integrative properties of CA1 apical oblique dendrites using patch-clamp recordings combined with multiphoton-uncaging of MNI-glutamate, in sham-control animals and in the kainate model of epilepsy. An examination of the intrinsic discharge behaviour revealed a significantly enhanced excitability and an increase in the maximal firing frequency in kainate-treated animals. In kainate-treated animals, d-spikes propensity was higher compared to sham-control animals. Interestingly, the d-spikes in CA1 neurons from kainate-treated animals were invariably elicited at reduced stimulated EPSP amplitudes, with much higher intensities being needed to elicit d-spikes in sham-control animals. This reduction in d-spike threshold may contribute to the increased propensity in CA1 neurons from epileptic animals. Furthermore, the strength of the d-spike measured as first temporal derivatives ($\delta V/\delta t$), was greater in cells from KA animals. An analysis of branch order suggests that primary apical oblique dendrites are particularly sensitive to these changes in supralinear integration. Moreover the requirements for input synchrony to generate d-spikes are degraded in KA animals and we also observed that supralinear integration fails to inhibit subsequent d-spikes in epileptic animals. What mechanisms underlie generation of increased dendritic spiking? We used selective pharmacological blockers to examine this question. We found that the Na v 1.3 specific blocker ICA-121431 affected dendritic spikes only in epileptic animals. Notably, ICA-121431 at concentrations of 100nM raised the dendritic spike threshold and decreased the rate of rise of dendritic spikes. These data suggest that a functional up-regulation of Na v 1.3 channels contributes to the increased dendritic excitability observed in kainate-treated mice. These results suggest that dendritic spikes can be generated at abnormally high frequencies in epileptic animals in-vivo. It is likely that this mechanism contributes to increased excitability of CA1 pyramidal cells. It is also likely that this mechanism contributes to degraded place coding of CA1 pyramidal cells, and thereby to impaired cognition and spatial memory.

Poster-ID: 20

Name: Arne Meyer, University College London

Title: Action-specific processing in mouse visual cortex

Abstract: As we move through our environment, circuits in our brain not only receive external sensory input but also non-sensory internally generated signals reflecting behavioural context. A major goal in sensory systems neuroscience is to understand how neural circuits integrate this wide range of inputs to produce flexible and adaptive behaviour. Traditional views posit that early sensory brain areas faithfully represent specific features of sensory stimuli, and that these representations are used by higher-level areas to guide decisions and actions. However, even in primary sensory cortical areas, neurons do not only respond to sensory input but are also modulated by behavioural context. For example, studies in head-fixed mice showed that locomotion has substantial effects on neural responses in primary visual cortex (V1), providing potentially important self-motion cues. However, it is not clear whether these modulatory signals carry information about specific actions, and how these signals are used during sensory-guided behaviour. Here we show that V1 responses convey action-specific information that is tightly locked to the animal's behavior. By combining chronic recordings with detailed behavioural tracking in freely moving mice, we found that neural activity in V1 is strongly modulated by head movements even in the absence of visual input (i.e. in the dark). This effect did not depend on variability in eye movements and could not be explained by whisking or locomotion. In total, more than half of the cells were significantly modulated by head movements. We observed both increases and decreases in firing rate in simultaneously recorded populations of cells. Importantly, the effect was substantially different from locomotion in most cells indicating action-specific processing in mouse V1. We also studied how responses depended on specific movement aspects by recording from populations of V1 neurons during active and passive movements. In both conditions V1 cells showed directionally tuned responses. Importantly, while response latencies were widely distributed, we found cells with very short responses latencies, often shorter than the visual response latency of the cell. Our results indicate that modulation of early sensory cortical areas by action is both more general (i.e. it occurs for multiple types of actions) and more specific (i.e. the effects depends on the type of action) than previously known. In addition, our results also suggest that movement-specific inputs may affect responses in sensory circuits before incoming primary sensory inputs associated with the action. We describe potential mechanisms of how action-specific signals can be used to selectively process sensory input by exploiting self-motion cues. In summary, our findings emphasize that activity linked to detailed aspects of behaviour can rapidly and flexibly shape processing of sensory input.

Poster-ID: 21

Name: Laura Bella Naumann, TU Berlin

Title: Presynaptic inhibition provides a rapid stabilization of recurrent excitation in the face of plasticity

Abstract: Hebbian plasticity, a mechanism believed to play a key role in learning and memory, detects and further enhances correlated neural activity. In recurrent networks this constitutes an inherently unstable positive feedback loop and therefore requires additional homeostatic control [1]. Recent computational work indicates that slow homeostasis, as observed in experiments, is insufficient to compensate the instabilities arising from Hebbian plasticity in recurrent neural networks [2]. We suggest presynaptic inhibition as a compensatory process, which does not suffer from this discrepancy of timescales. Experimental studies have revealed that excess network activity can trigger inhibition of transmitter release at excitatory synapses through activation of presynaptic GABAB receptors [3]. This effectively and reversibly attenuates recurrent interactions on timescales of 100s of milliseconds, thus serving as a candidate mechanism for the rapid compensation of elevated recurrent excitation induced by Hebbian changes. To study the network effects of presynaptic inhibition, we analyzed a rate-based recurrent network model, in which presynaptic inhibition is mimicked by a multiplicative reduction of recurrent synaptic weights in response to increasing firing rates. Using analytical and numerical methods, we show that presynaptic inhibition ensures a gradual increase of firing rates with growing recurrent excitation, even for very strong recurrence, whereas classical subtractive postsynaptic inhibition is unable to control recurrent excitation once it has surpassed a critical strength. We further demonstrate that this stabilizing power is conserved in networks subject to Hebbian synaptic plasticity using analytical mean-field approaches as well as network simulations. A computational study has shown that in recurrent networks subject to Hebbian plasticity, homeostatic mechanisms need to operate on timescales of seconds to minutes to prevent runaway activity [4]. By including presynaptic inhibition into such networks, we find that the timescale of homeostatic processes can be markedly increased without acquiring stability problems. Therefore the mechanism functions as an additional rapid compensatory process to stabilize recurrent plastic networks, even if homeostasis is as slow as observed in experiments. In summary, the multiplicative character of presynaptic inhibition provides a powerful compensatory mechanism to rapidly reduce effective recurrent interactions. As it conserves the underlying network connectivity, presynaptic inhibition might therefore set the stage for stable learning without interfering with plasticity at the level of single synapses. References [1] Abbott LF and Nelson SB. Synaptic plasticity: taming the beast. Nat Neurosci 2000, 3:1178-1490. [2] Zenke F and Gerstner W. Hebbian plasticity requires compensatory processes on multiple timescales. Phil Trans R Soc B 2017, 372(1715): 20160259. [3] Urban-Ciecko J, Fanselow EE, Barth AL. Neocortical somatostatin neurons reversibly silence excitatory transmission via GABAb receptors. Curr Biol 2016, 25(6):722-731. [4] Zenke F, Hennequin G and Gerstner W. Synaptic plasticity in neural networks needs homeostasis with a fast rate detector. PLoS computational biology 2013, 9(11), e1003330.

Poster-ID: 22

Name: Hyoungjun (Peter) Park, caesar

Title: Rabies-virus-based mapping of whisker-muscle-related cortical areas

Abstract
I present the precise localization of the cortical neurons that have the shortest synaptic distances to the intrinsic whisker muscles in rats, which are termed here as whisker-muscle-related (WMR) neurons. I also present a system of methods to reconstruct and standardize the distribution of the WMR neurons from a microscopy image stack. The WMR neurons in the cortex are detected as neurons retrogradely labeled in the third order of labeling by the rabies virus, which was injected into a single intrinsic whisker muscle as a trans-synaptic tracer. The locations of the cortical WMR neurons are then registered to a three-dimensional (3D) reference frame of the cortex, which is reconstructed from a fluorescent widefield microscopy image stack of either tangential slices or coronal slices. Based on the distribution of the WMR neuron population within a reference frame, the WMR neurons in the M1 region and S1 region of the cortex are identified as wmrM1 and wmrS1. As a method to standardize wmrM1 and wmrS1, labeled somata in wmrM1 and wmrS1 are registered into a designated 3D reference frame that is termed as the target reference frame. The standardization allows the precise localization of dense regions of average wmrM1 and average wmrS1 with respect to the olfactory bulb (or the bregma) and the midline. The centroid of the average wmrM1 is located 3.4 mm anteriorly to the bregma and 2.4 mm laterally to the midline, whereas the centroid of the average wmrS1 is located 0.85 mm posteriorly to the bregma and 5.2 mm laterally from the midline. Furthermore, since the WMR neuron distribution in the third order of rabies-labeling is confined within the cortical layer 5 (L5), the distribution of WMR neurons along the inner curvature of L5 is investigated using a surface-based coordinate system. The inner curvature of L5 is estimated by assuming the smoothed curvature of the pia and then re-parameterized as two-dimensional (2D) coordinates by flattening the approximated surface that forms the upper border of L5 using the multi-dimensional scaling. This technique is shown to be effective in accurately describing the neuron population within L5. By using the surface-based coordinate system, the locations of the average wmrM1 and wmrS1 thereby are also described in terms of the shortest traveling distances along L5 to the barrel field. Measured from the center of the barrel field, the centroid of the average wmrM1 is located 6.9 mm away anteriorly and the centroid of the average wmrS1 is located 2.2 mm away ventrally, both by traveling distance along L5. The precise localization by using the bregma, the midline, and the barrel cortex as the reference landmarks enables us to establish the reference frames of wmrM1 and wmrS1 that are consistent across different animals and experimental conditions.

Poster-ID: 23

Name: David Slabik, caesar

Title: Mapping of synaptic contact patterns between in vivo recorded neurons

Abstract: Most of what we know about structure-function relationships comes from electrophysiology measurements in acute brain slices from in vitro experiments. This methods are limited as they do not provide access to the brain of the living animal. Furthermore these experiments suffer severely from truncation of the pre- and postsynaptic neuronal structures. Similary, the limited extent of volumes accessible by dense circuit reconstructions at electron microscopy level questions whether present connectivity measurements can be extrapolated to larger volumes. We present a method to investigate synaptic contact patterns of multiple in vivo recorded and complete reconstructed neurons. Here we were even able to map contacts between up to six close by neurons. As a result, these locations, i.e. putative contacts, are visualized within a custom-designed 3D proof-editing environment for further investigation of spine-bouton overlap. This approach provides an upper and lower bound of the number, distribution and dendritic location of synaptic contacts between complete cell morphologies, thus providing quantitative relationships between cell-type identity, location, inter-somatic distance, connection probabilities and subcellular contacts.

Poster-ID: 24

Name: Vilim Štih, Max Planck Institute of Neurobiology

Title: Computational properties of visual stimulus-evoked activity in granule cells of the zebrafish cerebellum

Abstract
According to the prevailing view of cerebellar circuit function, cerebellar granule cells provide dimensional expansion, sparsification and decorrelation of input patterns, in order to enable learning of context dependent activity patterns in the downstream Purkinje cells. To examine these properties of granule cell representations, we recorded their activity using calcium imaging in larval zebrafish under a lightsheet microscope, while presenting a variety of visual stimuli. The lightsheet setup enables simultaneous recording of almost all granule cells (several thousands), which is not possible in any other animal. The stimuli consisted of localized flashes of light, uniform motion stimuli in different directions, and a video with natural scene statistics. Using this rich dataset, we investigate the dimensionality of granule cell responses and features of visual scenes which can be decoded from the activity patterns. We find that information-limiting correlations have a large impact on this decoding. We also compare stimulus-related information in the granule cells to neurons in visual areas such as the optic tectum. Finally, we repeated the stimulation protocol in the same animal over a period of 24 hours to quantify the stability of granule cell representations over time. These analysis together provide a comprehensive overview of the population coding properties of cerebellar granule cells for visual stimuli.

Poster-ID: 25

Name: Felipe Yanez, Caesar

Title: Laminar organization of morphologically, molecularly, and functionally defined inhibitory cell types in cortex

Abstract: Inhibition shapes the transfer of sensory-evoked thalamic inputs through sensory cortex in a still incomprehensible fashion. The building blocks of the underlying inhibitory mechanisms are GABAergic interneurons, which exhibit rich complexity that needs to be carefully handled. Here, on the example of the vibrissal part of rat primary somatosensory cortex (vS1), we quantitatively characterize morphological projection patterns and electrophysiological responses of molecularly identified inhibitory interneurons located throughout thalamorecipient layers (L)1-6.

Poster-ID: 25A

Name: Philipp Ranft, DZNE

Title: Investigation of a local microcircuit in the Mushroom Body calyx in the adult fly brain.

Abstract: The input region of the Drosophila mushroom body (MB), the calyx, is comprised of mainly three types of neurons: projection neurons (PN) emerging from sensory neuropiles, intrinsic neurons called Kenyon cells (KCs), and extrinsic neurons (EN), which connect the calyx with other brain regions. PNs display large boutons that form synaptic complexes, called Microglomeruli (MG), with KC claw-like dendrites. The size and number of MGs is under the control of activity within PNs and MGs have been suggested to be autonomous computational relays.

Previous studies using electron microscopy (EM) suggested that MGs represent complex synaptic networks that include ENs, but where unable to identify the types involved. Additionally, past light microscopy (LM) studies described various types of ENs, including modulatory and inhibitory, projecting into the calyx. However, which ones might contribute and to which extent to MGs remained unknown.

With the availability of a whole brain EM volume of an adult female fly (Zheng et al., in press), we reconstructed the complete circuitry of a MG and identified all the neurons that compose it and their local connections. We started of from a PN bouton and traced each pre- and postsynaptic cell within the MG synaptic complex to identification. Additionally, we annotated all synaptic connections, which allowed us to describe the local connectome of a single MG synaptic complex.

Our current data suggest a complex local microcircuit that could lead to MG-specific modulation, including the GABAergic APL and MB-C1 neurons.

BEHAVIOUR

Poster-ID: 26

Name: Georgia Christodoulou, CNCB, University of Oxford

Title: Dynamical memories: A new conceptual approach to memory networks.

Abstract: The ability to store and recall memories is the basis of many brain functions, but the notion of a memory is still not well understood. Models are traditionally based on the idea of 'Hebbian' cell assemblies with strong connections between a group of neurons representing a memory. More recently, the dynamical responses of neuronal networks have been explored in more detail, e.g., in 'Stability Optimized Circuits' (SOCs). In SOCs, neurons are strongly connected and stability is achieved by an optimization algorithm that changes the inhibitory weights to match the existing strong excitatory pathways. As as result, the background activity is stable, and interestingly, certain inputs that favour specific excitatory modes can transiently expose the excitatory pathways, generating strong amplified dynamics that decay back to baseline when inhibition matches excitation. Such networks, in which virtually all neurons are involved in the shape of the dynamics, have not been explored in the context of a memory system, and here we introduce a framework to explore the notion of dynamical memories. We define memories as the spatio-temporal transient outputs of a SOC - sets of all neuronal trajectories connecting two points in time. Thus, memories do not correspond to a specified pattern of neurons, but to a spatio-temporal response because of a structure already embedded in a given connectivity. To understand how a given connectivity affects memories we study stability optimized networks with small world structures of excitatory to excitatory connections. We follow their evolution through the transition from small world to random Erdos-Renyi and investigate how it affects the network dynamics. We quantify the effect of their structure on the dynamical properties of the memories they hold by the amplitude and length of the output transient. To discern further how architecture affects dynamics, we focus on graph-theoretical properties to also determine the network qualitatively, i.e., we assess memory degradation as a function of segregation (how modular the network is), integration (how well information is transmitted) and resilience (how robust the network is to node or edge loss). We find that segregation and integration are highly correlated with the dynamical properties of memories, indicating that different architectures change the amplitude and temporal length of memories. Finally, in order to analyse the network's storage capacity we consider both the spatial and the temporal dimensions of their memories. Specifically, we compare the dynamical responses using different "distance-metrics" (e.g., based on dimensionality reduction of the population activity or independent neuronal trajectories). Two different responses should be considered to be identical if they are similar by the given metric, i.e., a small perturbation of a dynamical response should not be seen as a different memory. To determine identical memories we define a threshold that depends on the context of a given task; the threshold is low if capacity is desired, and high when robustness is necessary. Our work is the starting point to establish a formal analysis of dynamical memories, and the development of tools to analyse neuronal activities as dynamic memories.

Poster-ID: 27

Name: Claire Eschbach, HHMI Janelia Research Campus

Title: Neuronal architecture for the adaptive control of reinforcement processing

Abstract: The ability to learn and update associations between stimuli and positive or negative reinforcement is essential for survival in an ever-changing environment. Across animal kingdom, modulatory neurons and especially dopaminergic neurons carry the signal about reinforcement that allows the formation and update of associative memories. We used electron microscopy reconstruction and experimental approach to identify and describe the neurons presynaptic to the modulatory neurons in an associative learning center, the mushroom body of Drosophila larva. We first characterized the way in which the modulatory neurons encode different punishment types, and mapped the afferent feedforward pathways all the way from the punishment-encoding nociceptive and mechanosensory neurons. We found 96 neuron types presynaptic to the modulatory neurons and discovered layers of feedback neurons that receive direct or indirect input from mushroom body output neurons and signal back to the modulatory neurons. Strikingly, most modulatory neurons received more than half of their total dendritic input from feedback pathways. We confirmed the feedback pathways were functional and could influence memory formation. Our study reveals the multilayered nature of the insect learning network and suggests reinforcement processing and learning in Drosophila are strongly influenced by previously formed memories, via the feedback pathways.

Poster-ID: 28

Name: Lisa Gill, caesar

Title: The social, the sexual and the genetic: proxies in behavioural research can be misleading

Abstract: Extra-pair parentage in socially monogamous species is an important subject of investigation because it sheds light on the evolution of reproductive strategies. Social monogamy has been shown to be profitable, for example when animals choose a compatible partner and raise their offspring together as a functional unit. Extra-pair parentage may, however, increase the genetic fitness of an individual through additional or high-quality offspring outside of the pair bond. The study of monogamy has come a long way. Until the 1970s it was believed that birds were exceptionally monogamous, because in many species, social pair bonds were found to persist for a lifetime. When genetic parentage analysis tools became available, this notion quickly faded. Based on the past 40 years of research, the view today is that strict genetic monogamy in socially monogamous birds is exceptional. However, since copulations in birds are often a matter of seconds, and occur visually concealed inside the nest cavity, few studies actually investigated sexual behaviour. Instead, the majority of studies today focus on the genetic component of monogamy because it is tightly linked with

evolution. But does this proxy alone draw a realistic picture of reproductive strategies? One of the supposed rare monogamous exceptions is the jackdaw, a group-living member of the corvid family (ravens, crows, jays etc.) renowned for its ecological flexibility and cognitive abilities. Jackdaw monogamy was previously assessed via social pairing data and its genetic output, without observations of sexual behaviour. Here we investigated monogamy of a colony of wild jackdaws at three different levels: social, sexual and genetic. To describe and quantify sexual behaviour, we used a combination of field observations, nest-box video recordings, and on-bird microphones. Nest checks and DNA analysis of adults and of all hatched chicks provided us with information on colony synchrony, a pair's nest success and extra-pair fertilisation rates. As previously described, we found that pairs stayed together for multiple breeding seasons, and we found no case of extra-pair parentage. However, we found substantial amounts of extra-pair sexual behaviour. Unlike in other species in which females seek extra-pair copulations, we found this behaviour to be male-driven and forced, i.e. the females vigorously defended against extra-pair males. Our results suggest the existence of different pre- and post-copulatory mechanisms (e.g. mate-guarding) involved in the mismatch between sexual and genetic levels of monogamy. Our results also show that using the genetic component alone does not allow a full view on the costs and benefits involved in the evolution of mating systems. Proxies may help us break down complex processes into more accessible pieces of information with which we can answer specific questions. But they cannot necessarily replace detailed observations, and should be used with caution if we are aiming to understand the bigger picture.

Poster-ID: 29

Name: Iris Grothe, Ernst Strüngmann Institute (ESI) for Neuroscience in Cooperation with Max Planck Society

Title: Selective gating of afferent input signals to area V4 by attention

Abstract: Depending on the behavioral context, the brain needs to channel the flow of information through its networks of massively interconnected neurons. Selective attention allows focusing on part of the incoming sensory information, which is reflected by neurons in visual cortex when two different stimuli are simultaneously present in their receptive fields. Their spiking response to the attended stimulus then resembles the response to that stimulus when presented in isolation. It is not clear where in the neuronal pathway attention intervenes to achieve such selective signal routing and processing. Three possible intervention points often discussed are: enhancing the gain in the presynaptic neurons (input gain), enhancing the gain in the postsynaptic neurons (output gain) or gating of the afferent signals (afferent signal gating). We designed an experiment which allows to causally asses routing of information originating from multiple visual stimuli. We tagged two equivalent visual stimuli by independent broadband luminance noise and used the spectral coherence of these behaviorally irrelevant signals with the field potential of a local neuronal population in macaque monkeys' area V4 as a measure for their respective causal influences. This novel experimental paradigm revealed that signal transmission was considerably

weaker for the not-attended stimulus. Furthermore, our results show that attention does not need to modulate responses in the input populations sending signals to V4 in order to selectively represent a stimulus, nor do they suggest a change of the V4 neurons' output gain depending on their feature similarity with the stimuli. Our results rather imply that selective attention 'gates' signals at the interplay between afferent fibers and the local neurons. A minimal model demonstrated that coherent gamma-rhythmic activity (~60-Hz) between local neurons and their afferent-providing input neurons can realize the gating: it replicated the attentional gating effect and the signals' spectral transfer characteristics. It supports the proposal that selective interareal gamma-band synchrony subserves signal routing and explains our experimental finding that attention selectively gates signals already at the level of afferent synaptic input. The method provides a useful tool to study mechanisms of dynamic network configuration underlying cognitive processes.

Poster-ID: 30

Name: Jan-Matthis Lückmann, caesar

Title: Can serial dependencies in choices and neural activity explain choice probabilities?
Autoren: Jan-Matthis Lueckmann, Jakob Macke, Hendrikje Nienborg

Abstract: During perceptual decisions the activity of sensory neurons co-varies with choice, a co-variation often quantified as "choice-probability". Moreover, choices are influenced by a subject's previous choice (serial dependence) and neuronal activity often shows temporal correlations on long (seconds) timescales. Here, we test whether these findings are linked. Using generalized linear models we analyze simultaneous measurements of behavior and V2 neural activity in macaques performing a visual discrimination task. Both decisions and spiking activity show substantial temporal correlations and cross- correlations but reflect two mostly separate processes: Removing history effects using semi-partial correlation analysis leaves choice probabilities largely unchanged. The serial dependencies in choices and neural activity therefore cannot explain the observed choice probability. Rather, serial dependencies in choices and spiking activity reflect two predominantly parallel processes, which are correlated by instantaneous co-variations between choices and activity. These findings provide important constraints for computational models of perceptual decision-making that include feedback signals

Poster-ID: 31

Name: Kevin Luxem, DZNE

Title: 3D behavioral analysis and optogenetics link medial septum and VTA during locomotion

Abstract: Locomotion is a fundamental behavior that comprises different specialized neuronal networks. However, how voluntary movement is initiated and which networks are involved is not fully understood. Glutamatergic neurons in the medial septum/diagonal band of Broca have been shown to be involved in locomotion-initiation and -speed. Thus, we wanted to investigate by which pathway the medial septal glutamatergic neurons initiate and control locomotion.

To find out, if there is a functional connection between the glutamatergic networks in the MSDB and the VTA we performed acute slice experiments in VGlut2-Cre mice, injected with a double floxed YFP channelrhodopsin (ChR) in the medial septum. Specific optogenetic activation of glutamatergic fibers from the medial septum and subsequent TTX application confirmed the existence of a monosynaptic glutamatergic connection between the two areas.

Optogenetic activation of glutamatergic neurons in the VTA in vivo could reliably initiate locomotion with the mouse head-fixed on a treadmill. We further analyzed the evoked locomotor behavior using the same stimulation paradigm in an open field arena, where the mouse could move freely. We used depth imaging and a machine learning approach to identify specific behavioral models related to the locomotion onset and modulation. This new approach will allow us to investigate not only the locomotion onset and speed but also further mouse pose dynamics on a sub-second timescale.

Poster-ID: 32

Name: Arne Monsees, caesar

Title: 3D Pose Recovery in Freely Moving Rats

Abstract: In order to study how the brain processes external stimuli and translates them into motor output, the analysis of behavior is essential, as it represents the ultimate output of the brain's neural computation. However, to date most studies have been limited to coarse and low-dimensional representations of animal behavior, both to simplify data analysis and because detailed behavioral measurements are technically challenging. For instance, when an animal is recorded with a single camera and it's center of mass is tracked, only two degrees of freedom are used to represent the full motor output. While sophisticated computer vision algorithms have been used to infer 3D poses from videography, these have usually been applied to human movements and relied on huge ground truth datasets that are lacking for other species. Furthermore, a particular challenge for 3D pose tracking in animals arises from the flexibility and diversity of body parts, such as soft tissue and fur in rats. This imposes the requirement to infer non-rigid motions and body deformations, potentially limiting the applicability of existing tracking algorithms. To bridge this gap, we show initial results demonstrating detailed 3D pose recovery in freely moving rats. The animals moved freely within an arena while their behavior was recorded by multiple cameras. After specific anatomical features were manually labeled, we reconstructed the

animal's 3D pose at each time point. The used algorithm aligns a 3D model of the rat's skeleton and surface to the labels by projecting the model to the image sensors of the cameras. We designed this method around gradient descent optimization of variables representing local anatomical scaling, translation, and rotation effects. The resulting reconstruction provides a detailed description of the animal's behavior, including head orientation and the positioning of individual limbs. Despite being widely successful in reconstructing the positioning of the rat's torso, head and limbs, our approach still has limitations. Consequently, we observe failure cases in different situations. Among the most frequent issues are the occasional misalignment of single body parts due to sparse labeling of the anatomical features and the abrupt repositioning of limbs in consecutive frames due to occlusions. To improve performance of our method, we plan to expand our analysis to combine high resolution MRI scans with video-based motion capture in a Bayesian framework, to allow for probabilistic pose reconstruction with increased accuracy, uncertainty estimation, and higher temporal and spatial resolutions.

Poster-ID: 33

Name: Martin Pofahl, University of Bonn

Title: State-dependent processing of dentate granule cell activity in-vivo

Abstract: The dentate gyrus (DG) is the first processing station within the canonical trisynaptic hippocampal circuit. It receives multisensory information via the medial and lateral perforant paths (MPP and LPP), and unidirectionally relays it to the CA3 region. DG main principal neurons, granule cells (GC), fire sparsely in-vivo, leading to the concept that the DG serves as a gate controlling information transfer from the entorhinal cortex to the hippocampus. Recent multiphoton Ca2+ imaging studies have examined activity in granule cells in-vivo and have found sparse, spatially tuned activity. However, information transfer has to be able to adapt to substantial differences in the richness and type of sensory information. In this study, we address the DG output properties in awake mice using 2-photon Ca2+ imaging. To enable Ca2+ imaging in the deeply located DG, we have firstly used a Thy1-GCaMP6 line (GP4.12Dkim/J) that expresses the Ca2+ indicator GCaMP6s in a subset (~50 %) of GCs. Secondly, we are using a specially designed hippocampal window, allowing us to record from several hundreds of GCs while the animal is advancing on a custom made horizontal linear treadmill. After habituation on the apparatus, we systematically vary the density of tactile cues in three successive recording sessions. During the subsequent three sessions we present different behaviorally salient stimuli, such as odors, or mild aversive stimuli. These data will allow conclusions regarding dentate input-output processing under conditions of differing amounts and quality of sensory stimulation.

Poster-ID: 34

Name: Federica Rosselli, caesar

Title: From the rat's perspective: Identification of the rat's visual fields by eye and head tracking

Abstract: Rats have a large binocular visual field that extends in front, over and behind the animal's head. We recently showed that the large binocular field of view is maintained overhead by eye movements that compensate changes in head position in freely moving conditions. We proposed that, in addition to stabilizing the overhead visual field during free behavior, the eye movements also minimize the part of the rear visual field that neither eye can cover, i.e. the area blind to either eye. To address the question of how the visual fields and posterior blind area maintain their relative coverage of the visual space around the animal's head during free behavior, we optimized our miniaturized ocular videography system and introduced a novel method for the direct quantification of the true anatomical relationship between head and eye positions, by triangulating head LEDs with a set of defined anatomical features for each individual animal. Such new method allows, with respect to our previous model, to avoid underestimation in the extent of the monocular visual fields, their overlap and the blind area. The visual fields were quantified in real time relative to a plane on which the animal was freely moving, and the visual fields projected onto an overhead screen used for the display of a 'threatening' visual input (i.e. an exponentially growing 'looming' dot) to ensure saliency, thus increase the likelihood of stimulus detection. Using this approach, we confirm that an overhead binocular coverage is constantly maintained by the eyes movements and head position, but much wider as estimated by our previous model, extending both overhead and behind the animal's head. On the contrary, the blind area is often minimal, and mainly falling outside of the overhead region of interest (i.e. the monitor).

Poster-ID: 35

Name: Sarah Schmidt, University of Bonn

Title: Network effects of Eslicarbazepine in the hippocampus

Abstract: Many anti-epileptic drugs (AEDs) act on voltage-dependent sodium channels, and the molecular basis of these effects is well understood. In contrast, how AEDs act on the level of neuronal networks, and how they affect different types of neurons is much less understood. The purpose of the present study was to determine the effects of eslicarbazepine (S-Lic), the active metabolite of the AED eslicarbazepine acetate on different types of neurons in the hippocampus. Experiments were performed in hippocampal slices from both sham-control and chronically epileptic pilocarpine-treated animals. Both 100 and 300 µM S-Lic significantly inhibited repetitive firing of excitatory CA1 pyramidal neurons. Interestingly 300 but not 100 µM S-Lic significantly reduced maximal firing rates in putative feed-forward interneurons located in the CA1 stratum radiatum of sham-control and

epileptic animals. Since S-Lic affects not only excitatory cells but also inhibitory interneurons in CA1 we were interested in the effects of S-Lic on on feed-back and feed-forward inhibition in the hippocampal CA1 region. We have previously shown that carbamazepine (CBZ), which is a use-dependent Na+ channel blocker, does not affect inhibitory circuits (Pothmann et al., J Neurosci. 2014; 34:9720). We examined S-Lic effects on feed-back and feed-forward inhibition in the hippocampal CA1 region by recording from CA1 pyramidal cells with the patch-clamp technique. Feed-back inhibition was selectively recruited by stimulating CA1 axons in the alveus, while feed-forward inhibition was recruited by stimulating CA3 Schaffer collaterals. In addition, we have recorded from identified interneurons to assess their recruitment into these circuits, and how it is affected by S-Lic. Feed-forward inhibition was significantly affected only at 300 µM, but not at 100 µM S-Lic in sham-control and epileptic animals. In addition, feed forward EPSCs in these interneurons were reduced by 300, but not 100 µM S-Lic. Interestingly feed-back inhibition was increased over time, even after wash-out of S-Lic at 300 µM concentration only in epileptic animals and not in sham-control animals. These results suggest that S-Lic affects inhibitory circuits in the CA1 hippocampal region. Depending on interneuron type and their special expression of channels and receptors, S-Lic affects feed-back and feed-forward inhibition differently. It remains to be seen how these effects on specific circuit elements translate to effects of S-Lic on cellular activity patterns during spontaneously developing epileptiform activity.

Poster-ID: 36

Name: Sylvia Schroeder, UCL

Title: Behavioural modulation of visual responses in mouse superior colliculus

Abstract: Sylvia Schröder, Nicholas A. Steinmetz, Michael Krumin, Matteo Rizzi, Kenneth D. Harris, Matteo Carandini Visual responses in multiple brain regions, including visual thalamus and primary visual cortex (V1), are modulated by non-visual factors such as locomotion and level of arousal. We asked whether this modulation also extends to the superficial layers of superior colliculus (SC), which receive direct visual input from the retina, and if so, where this modulation may originate. To record the activity of neuronal populations within the top 150 µm of SC, we used two-photon calcium imaging while mice were head-fixed and free to run on a treadmill. To drive visual activity we presented drifting gratings of varying orientations and directions, and to measure spontaneous activity we presented uniform gray screens. To track arousal level, we measured running speed and pupil diameter. We found that about 50% of neurons in superficial SC were significantly correlated with running and pupil diameter both during spontaneous and visually driven activity. Roughly equal numbers of neurons showed positive and negative correlations. Changes in arousal also had significant effects on direction tuning curves. These effects could be described with a linear model (additive shifts plus multiplicative effects) but were heterogeneous across neurons. To investigate whether these behavioral effects were inherited from V1, we inactivated V1 by optogenetically stimulating inhibitory neurons, while recording from ipsilateral SC neurons using Neuropixels probes. V1 inactivation

decreased average visual responses in SC, but it did not significantly decrease their modulation by locomotion and arousal. We then tested whether behavioral modulation originates in the retinal input to superficial SC. We expressed GCaMP in retinal ganglion cells (RGCs) and imaged calcium activity of RGC boutons in the superficial SC. To our surprise, bouton activity was correlated with locomotion and arousal and the boutons' direction tuning was modulated to a similar degree as seen in SC neurons. Correlation with locomotion even persisted during complete darkness, excluding the possibility that changing light levels reaching retina due to pupil dilation caused the change in responses. This observation might be explained by one or both of two mechanisms: modulation of RGC output by behavior, e.g. via backprojections from the rest of the brain, or modulation of retinal boutons in the superficial SC, e.g. via neuromodulators causing changes in calcium levels. We are currently testing the first hypothesis by recording from the optic tract to investigate whether retinal firing rates are modulated by running and arousal. Taken together, our results show that neuronal responses in the superficial layers of SC are not solely driven by visual inputs but are also modulated by locomotion and arousal. This modulation is not inherited from V1, but rather originates as early in the visual pathway as in the retinal input to superficial SC.

Poster-ID: 37

Name: Damian Wallace, caesar

Title: Eye movements during exploration and pursuit: oculo-videography in freely moving ferrets and tree shrews

Abstract: Coordination of eye movements is a critically important factor in the physiology of vision. Coordinated eye movements play a key role in stabilizing vision during movements, but also have central role in determining what visual information the central visual centers have to process. Coordination of eye movements is also important for stereopsis and binocular single vision. Here we quantify, using ocular-videography, the coordination of eye movements in two different mammalian species, the ferret and the tree shrew. Eye movements were simultaneously recorded from both eyes as the animals' explored a large (2 x 1 x 1m) arena, containing objects on which to climb and smaller objects to chase or interact with. Both animals have semi-laterally pointing eyes (both approx. 35 degrees lateral of frontal) but different life history strategies. Ferrets are carnivores and pursue and catch live prey, while tree shrews are omnivores, eating insects and small reptiles and fruits. Both species are preyed upon by larger predators birds in their respective ecosystems and therefore have pressure to vigilantly monitor the visual space above them. Ferrets maintain conjugate movements during pursuit of objects, but in addition can have strong horizontal divergent movements during nose-down head pitch that serve to shift the coverage of the visual field to be more above the animal. Tree shrews in contrast maintain continuous conjugate movements at all times. The eye movements observed in freely moving animals differ strongly between species and probably have a dramatic impact on the requirements placed on central visual processing.

COMPUTATION

Poster-ID: 38

Name: Alexandre René, caesar

Title: Inferring mesoscopic population models from population spike trains

Abstract: How does the interplay of single-neuron dynamics and neural connectivity give rise to the rich dynamical properties of neural populations? To tackle this question, it is desirable to have models which exhibit a wide range of population dynamics but remain interpretable in terms of connectivity and single-neuron dynamics. However, many commonly-used statistical models of neural population dynamics are based on generic models of dynamics (e.g. in Macke et al. 2011). Conversely, it has been challenging to link mechanistic spiking network models to empirical population data. To close this gap, we propose to model such data using mechanistic, but low-dimensional and hence statistically tractable models. We approximate neural populations as being composed of multiple homogeneous `pools' of neurons, and model the dynamics of the aggregate population activity within each pool. We derive the likelihood of parameters (both single-neuron parameters and inter-pool connectivity) given this activity, which can then be used to either optimize parameters by gradient ascent on the log-likelihood, or to perform Bayesian inference using Markov Chain Monte Carlo (MCMC) sampling. We illustrate this approach on a model based on generalized integrate-and-fire neurons (Schwalger et al., 2017). Using micro- and mesoscopic simulations of multiple neuron pools, we demonstrate that both single-neuron properties (membrane and adaptation constants) and connectivity-parameters (excitatory vs inhibitory connections and connection strengths) can be recovered on simulated data. Moving beyond point estimates, we compute the Bayesian posterior for combinations of parameters using MCMC sampling. Finally, we investigate how the approximations inherent to a mesoscopic population model impact the accuracy of the inferred single-neuron parameters. Ultimately, our method ensures compatibility between experimental multi-population data and mesoscopic dynamical models, by providing methods for statistical inference of low-dimensional mesoscopic models.

Poster-ID: 39

Name: Giacomo Bassetto, caesar

Title: Using Bayesian inference to estimate receptive fields from a small number of spikes.

Abstract: A crucial step towards understanding how the external world is represented by sensory neurons is the characterization of neural receptive fields. Advances in experimental methods give increasing opportunity to study sensory processing in behaving animals, but

also necessitate the ability to estimate receptive fields from very small spike-counts. For visual neurons, the stimulus space can be very high dimensional, raising challenges for data-analysis: How can one accurately estimate neural receptive fields using only a few spikes, and obtain quantitative uncertainty-estimates about tuning properties (such as location and preferred orientation)? For many sensory areas, there are canonical parametric models of receptive field shapes (e.g., Gabor functions for primary visual cortex) which can be used to constrain receptive fields – we will use such parametric models for receptive field estimation in low-data regimes using full Bayesian inference. We will focus on modelling simple cells in primary visual cortex, but our approach will be applicable more generally.
We model the spike generation process using a generalized linear model (GLM), with a receptive field parametrized as a time-modulated Gabor function. Use of the parametric model dramatically reduces the number of parameters, and allows us to directly estimate the posterior distribution over interpretable model parameters. We develop an efficient Markov Chain Monte Carlo procedure which is adapted to receptive field estimation from movie-data, by exploiting spatio-temporal separability of receptive fields. We show that the method successfully detects the presence or absence of a receptive field in simulated data even when the total number of spikes is low, and can correctly recover ground-truth parameters. When applied to electro-physiological recordings, it returns estimates of model parameters which are consistent across different subsets of the data. In comparison with non-parametric methods based on Gaussian Processes, we find that it leads to better spike-prediction performance.

Poster-ID: 40

Name: Wilhelm Braun, University of Bonn

Title: Understanding temporal correlations in networks of spiking neurons

Abstract: We consider temporal serial correlations in the spike trains of individual neurons in networks of purely inhibitory as well as inhibitory and excitatory integrate-and-fire neurons. This is different from spatial correlations between neurons, which are e.g. quantified by their membrane voltage or spike count correlation function. We show by performing extensive network simulations that in sparse networks, where spatial correlations are absent by definition, negative temporal correlations, as quantified by the neuron-averaged serial correlation coefficient (SCC), are pronounced and the network shows transitions from states of low to high temporal correlations as the amount of inhibition is increased. We proceed by considering the impact of a private adaptation current on temporal correlations and observe sharp dips of the SCC as a function of the timescale of the adaptation current. In the second part, we consider different methods to compute the network SCC. We perform an online full network simulation and record the spike times of all neurons, which we then replicate offline by adding recorded presynaptic spike trains to one particular neuron. We thus show that the SCC of one particular neuron is extremely sensitive to the number of its synapses, i.e. the true online SCC is only reproduced faithfully if nearly all synapses are taken into account. Therefore, we then consider two different ensembles for synaptic connectivity, the

'fixed p' scenario, where the average number of synapses per neuron, but not its precise number, is fixed, and the 'fixed C' scenario, where the exact number of synapses per neuron is fixed. We show that in the fixed-C scenario, the standard deviation of the SCC across neurons is much smaller than in the fixed-p scenario, and vanishes with increasing simulation time, in contrast to the fixed-p-scenario, where the standard deviation is strictly larger than zero. In light of these results, we explore the possibility of using a single effective neuron diffusion approximation, which has been applied with great success to understand first-order statistics of spiking networks, to compute temporal second-order statistics of the network. We develop a computational scheme to assess the reliability of the diffusion approximation for the computation of the SCC properties of the network. We show that the diffusion approximation for the SCC breaks down very quickly for medium to large-size networks, in contrast to first-order ISI quantities, and especially when a global oscillation of the network is present. In the presence of a strong adaptation current, the SCC is more faithfully produced by the diffusion approximation, even for non-sparse networks. We also compute temporal correlations in the network based on an appropriate inhomogeneous Poisson processes. Our results thus bear general relevance for the possibility of reducing spiking network dynamics to a rate-based formulation and show that for the second-order statistics of the individual neurons in the network, a mean-field approach such as the diffusion approximation has limited validity. This is joint work with André Longtin, University of Ottawa, Canada.

Poster-ID: 41

Name: Aditya Gilra, University of Bonn

Title: Local, stable learning in spiking neural networks for motor control

Abstract: How networks of spiking neurons in the brain can learn to plan and control body movements is an unsolved question. We propose a supervised local learning scheme, Feedback-based Online Local Learning Of Weights (FOLLOW), to train a network of heterogeneous spiking neurons with up to two hidden layers (distinct from input and output) to learn arbitrary non-linear dynamics. We construct an error feedback architecture and derive local, biologically-plausible, and stable learning rules from adaptive control theory. Strong error feedback entrains the activity of hidden neurons, enabling a modification rule on the internal weights proportional to pre-synaptic activity and feedback post-synaptic error, both of which are available locally in the post-synaptic neuron. We further propose how networks of neurons in the brain might learn forward and inverse models of the dynamics of the muscle-body system, here demonstrated for a two-link arm under gravity. We use a recurrent network to learn the forward model i.e. to predict the arm state given the time-varying control input, and a configuration termed differential feedforward to learn the inverse model, i.e. to infer the control input given the desired arm state trajectory. We use the inverse model in a further feedback loop to control the arm to reproduce a desired trajectory. We believe our FOLLOW learning scheme predicts not only a feedback architecture and learning rules in our brain for motor control, but also enables

computationally-inexpensive learning in energy-efficient neuro-morphic hardware for neuro-robotics.

Poster-ID: 42

Name: Pedro Goncalves, caesar

Title: Flexible Bayesian inference for mechanistic models of neural dynamics

Abstract: One of the central goals of computational neuroscience is to understand the dynamics of single neurons and neural ensembles. However, linking mechanistic models of neural dynamics to empirical observations of neural activity has been challenging. Statistical inference is only possible for a few models of neural dynamics (e.g. GLMs), and no generally applicable, effective statistical inference algorithms are available: as a consequence, comparisons between models and data are either qualitative or rely on manual parameter tweaking, parameter-fitting using heuristics, or brute-force search (Druckmann et al. 2007). Furthermore, parameter-fitting approaches typically return a single best-fitting estimate, but do not characterize the entire space of models that would be consistent with data. We overcome this limitation by presenting a general method for Bayesian inference on mechanistic models of neural dynamics. Our approach can be applied in a `black box' manner to a wide range of neural models without requiring model-specific modifications. In particular, it extends to models without explicit likelihoods (e.g. most spiking networks). We achieve this goal by building on recent advances in likelihood-free Bayesian inference (Papamakarios and Murray 2016, Moreno et al. 2016): the key idea is to simulate multiple data-sets from different parameters, and then to train a probabilistic neural network which approximates the mapping from data to posterior distribution. We illustrate this approach using single- and multi-compartment models of single neurons and models of spiking networks. On simulated data, estimated posterior distributions recover ground-truth parameters, and reveal the manifold of parameters for which the model exhibits the same behaviour. On in-vitro recordings of membrane voltages, we recover multivariate posteriors over biophysical parameters, and voltage traces accurately match empirical data. Our approach will enable neuroscientists to perform Bayesian inference on complex neural dynamics models without having to design model-specific algorithms, closing the gap between biophysical and statistical approaches to neural dynamics.

Poster-ID: 43

Name: David Greenberg, caesar

Title: Action potential inference from a calcium sensor protein through biophysical modelling

Abstract: With genetically encoded calcium indicators (GECIs), fluorescence changes arising from neural activity can be recorded from the same neurons for days to weeks. However, the relationship between action potential (AP) discharge and GECI fluorescence is complex, nonlinear and variable over neurons. To quantitatively characterize this relationship we developed a sequential binding model (SBM) describing the chain of physical effects through which APs cause fluorescence changes in the calcium indicator GCaMP6s. We then used the SBM as a basis for quantitative and reliable AP inference. The core idea of the SBM is to model calcium binding to each of the four GCaMP6s binding sites as a reversible chemical reaction with mass-action kinetics. These reactions, along with endogenous buffering and extrusion, define a system of coupled differential equations which we solved to determine concentrations over time for free calcium, calcium bound to endogenous buffers and GCaMP6s binding states with 0 to 4 calcium ions. This biophysical framework allowed the SBM to capture nonlinearity, describe the rising and falling phases of the AP response and incorporate variability in response amplitude and shape over neurons. In combined optical/electrical recordings of GCaMP6s-expressing cortical neurons, the SBM reliably and quantitatively predicted fluorescence signals from AP sequences. When the same biophysical framework was applied to GCaMP6s data from in vitro binding assays, it was able to explain the results of fluorescence spectroscopy, isothermal titration calorimetry and stopped flow fluorescence experiments using a single global fit, resulting in parameters which also accurately predicted fluorescence signals in vivo. To infer AP times and neuron-specific SBM parameters from in vivo fluorescence signals we used sequential Monte Carlo (SMC), an approach based on data-driven simulation. This algorithm outperformed previous AP-detection methods on GCaMP6s, with more accurate firing rates, AP counts and AP times while requiring less training data. These results demonstrate the utility of biophysically grounded models for quantitative analysis of complex biological data.

Poster-ID: 44

Name: Bettina Hein, Frankfurt Institute for Advanced Studies

Title: The role of network interactions in coordinating neuronal tuning properties across visual cortex

Abstract: Already early in development prior to the onset of normal visual input the visual cortex produces highly structured, spatially extended patterns of spontaneous activity. These distributed network activity states comprise pronounced correlations between distant cortical locations, whose role in development is currently unknown. One possible role of

such distributed network interactions could be to coordinate the stimulus response properties of individual neurons across visual cortex. For instance, different network elements, even if spatially separated by several millimetres in cortex may develop similar tuning properties over the course of cortical maturation if they tend to be coactive spontaneously at early stages in development. Here we test this hypothesis of co-activity-induced co-tuning by focusing on orientation preference in the visual cortex. Our approach combines chronic calcium imaging in the early developing visual cortex with computational analyses and modelling. We used widefield fluorescence imaging of GCaMP6s to record visually evoked responses with moving grating stimuli and spontaneous activity in ferret primary visual cortex, from four days prior to the natural time of eye-opening (~P30 in ferrets) until about a week after eye-opening. Prior to P30, the eye lids were transiently opened when probing the cortex with grating stimuli. This setup allows us to assess the emergence and refinement of orientation selectivity and its relationship to spontaneous activity during early development. We found that, already at the earliest time point measured (P26), visual stimuli robustly evoke network responses that are organised on a local and global scale. The responses are modular (i.e., patchy), extend over the whole field of view (about 10mm2), and are highly variable across trials. Typically, weak orientation tuning is evident at this stage, and its layout already coarsely resembles the mature organisation. However, we also observe a considerable refinement in the local organisation until a near mature layout is reached a few days after eye-opening. To investigate whether the network correlations evident in early cortical spontaneous activity could indeed drive the coordination of orientation preference between distant network elements, we formulated a computational model testing the hypothesis that network correlations induce a reorganization of orientation tuning over the course of development such that cortical locations tend to become more similar if positively correlated early on, while more dissimilar if negatively correlated. Using this model, the spontaneous correlation structure and layout of orientation preferences measured at an early stage in development allowed us to correctly predict aspects of the refinement in the layout of orientation preferences over subsequent days. The model predicts that spontaneous activity and the layout of orientation domains become increasingly similar in their layout during development, which we confirm experimentally. Interestingly, we find that with age specifically network interactions between more remote elements become more effective, suggesting a link to the system of long-range horizontal connections known to develop extensively around the time of eye opening. We conclude that large-scale network interactions in the developing visual cortex play a significant role in driving the refinement of distributed circuit elements by coordinating their visual response properties.

Poster-ID: 45

Name: Yaroslav Felipe Kalle Kossio, University of Bonn

Title: Growing critical: Self-organized criticality in a developing neural system

Abstract: A variety of neural systems generate neuronal avalanches: bursts of activity with characteristics typical for critical dynamics. A hallmark of such systems is the variability of the activity, which may be functionally relevant: An individual avalanche can involve any number of neurons, from one to the entire network; avalanches of all sizes occur with non-negligible frequency. A possible explanation for the occurrence of neuronal avalanches is that the underlying networks organize themselves into a critical state. Here we propose a simple spiking model for a developing neural system. It shows how networks may “grow into” the critical state during development. Consistent with earlier network growth models, the extents of neurites are represented by discs, with synaptic coupling strengths proportional to the discs' overlap; neurons are modeled as inhomogeneous, coupled Poisson processes. Inputs increase the Poisson spike rate of a neuron. Neurites grow if a neuron is silent and shrink when spikes are generated. Both processes balance at some spike rate, the network becomes stationary. Our analysis and numerical simulations show that the larger the stationary state's spike rate is compared to the spontaneous one of isolated neurons, the nearer the stationary network is to the critical point. The avalanche size distribution approaches a power law distribution with exponent -3/2. We also derive the avalanche duration distribution analytically and show that its tail approaches a power law with exponent -2. Both exponents have been reported in experiments. Refractoriness of neurons can be included, but tends to drive the system away from criticality. Our model can be viewed as a Markovian Hawkes process, which is used in life sciences, finance, social sciences and geophysics to model clustering phenomena. In particular our derivation of the avalanche duration distribution may thus prove useful in other scientific fields as well.

Poster-ID: 46

Name: Christian Klos, University of Bonn

Title: Modeling the altered function of canonical feedback inhibitory circuits in chronic epilepsy

Abstract: Canonical feedback inhibitory motifs play a key role in controlling and structuring the activity of principal cell ensembles. Despite the importance of these motifs and evidence for altered inhibition in chronic epilepsy, changes in the function of canonical feedback inhibition in chronic epilepsy have not been investigated. On the basis of experimental results from the hippocampal region CA1 in the pilocarpine model of chronic epilepsy, we use theoretical modeling to examine how the functional properties of the canonical feedback circuit motifs are changed and how this influences the filtering of signals from CA3. The experiments indicate that intrinsic excitability is reduced in both basket and pyramidal cells. Furthermore, the response of basket cells to stimuli mimicking feedback excitation

exhibits reduced initial excitation as well as reduced synaptic depression of the synapse between pyramidal and basket cells. Finally, the initial feedback inhibition to pyramidal cells is strongly reduced. Here we show that simple, biologically plausible neuron and synapse models for basket cells and for the pyramidal cell-to-basket cell-synapses match the data on basket cell responses for suitably chosen parameters. We use this to quantify and interpret the experimentally observed pathological changes. To visualize our results, we project the reproduction error nonlinearly onto the relevant model parameters. This shows a qualitative difference between the parameter sets characterizing the synapses on healthy and pathological basket cells. A similar approach can be employed for the pyramidal cells and the basket cell-to-pyramidal cell synapses. We combine the obtained models for the basket and pyramidal cells and their synapses to models of the complete feedback circuit motif in CA1 for healthy and epileptic animals. Probing them with inputs from CA3, we find that the entirety of the changes in the feedback motif leads to increased activity of the pyramidal cells in the epileptic case especially in case of steep rises of the signal, which are typical for the initial phase of epileptic bursts. This suggests that the changes in CA1 during development of epilepsy promote the transmission of epileptic bursts from CA3 to other parts of the brain. Acknowledgements: This work was supported by the German Federal Ministry for Education and Research BMBF through the Bernstein Network (Bernstein Award 2014), the Deutsche Forschungsgemeinschaft (SFB 1089), the BONFOR program of the University of Bonn Medical Center, and the ERANET Neuron grant ‘EpiNet’.

Poster-ID: 47

Name: Paul Manz, University of Bonn

Title: Dynamical stability and local phase space structure of networks containing neurons with negative dissipation

Abstract: We study the dynamical properties of inhibitorily coupled networks of integrate-and-fire neurons with mixed dissipation. In addition to regular leaky integrate-and-fire neurons (positive dissipation) we also consider neurons where the sign of the leak current is switched (negative dissipation). These networks can exhibit a balanced state of highly irregular spiking activity independent of the types of the neurons. Although the balanced state was shown to exist in networks of different spiking neuron models, its dynamical properties such as chaoticity vary considerably depending on the neuron model. In particular, inhibitory networks consisting only of neurons with positive dissipation are Lyapunov stable and thus non-chaotic despite their irregular dynamics. This property is robust for fast non-instantaneous synaptic couplings and a small number of excitatory connections. Here, we show that switching the sign of the dissipation of even one neuron in such a network renders the entire network dynamics unstable with respect to small perturbations in the initial conditions. Our metric of stability against generic perturbations is the largest Lyapunov exponent, whose sign determines whether the dynamics is stable or unstable. To further characterize the dynamics we consider the full spectrum of Lyapunov exponents, which are related to the covariant Lyapunov vectors. The norm of an

infinitesimal perturbation along the direction of a CLV grows or shrinks with a rate given by its corresponding Lyapunov exponent. We find that for each neuron with negative dissipation in a network there will be positive Lyapunov exponent. That observation can be explained by a simple mean-field approach where we consider the growth or shrinkage of a perturbations to neurons. In addition we find that the stable covariant Lyapunov vectors are concentrated in the subspace of neurons with positive dissipation whereas unstable CLV are concentrated in the subspace of neurons with negative dissipation.

Poster-ID: 48

Name: Mythreya Mysore Seetharama, caesar

Title: Reconstruction of the Synaptic and Cell Type Specific Brain-wide Organization of Neuronal Networks

Abstract: Delineating the organization of brain-wide neural networks across scales – from cell populations to individual synapses – has been challenging. Replication competent Rabies virus strains can potentially be applied to reveal hierarchical organization of multi-synaptic brain-wide networks. Here, we present a framework to reconstruct the cell type specific organization of whisker related networks at synaptic level. We inject the retrograde rabies virus into a single intrinsic whisker muscle and quantify the trans-synaptic spread across orders of labeling. Reconstruction of rabies labelled morphologies provides cell type specific identification of cells within the network. This approach can potentially be applied to reveal the differences in brain-wide networks due to pathology.

Poster-ID: 49

Name: Max Nolte, Ecole polytechnique fédérale de Lausanne

Title: Spike-time coding amidst high synaptic noise and chaotic network dynamics in neocortex

Abstract: Responses of neocortical neurons to sensory stimuli are often highly variable. Strategies of cortical coding depend on how much of this variability carries hidden signals from other brain areas, and how much arises internally from local circuitry. However, the amount of internally generated variability and its consequences for reliable coding remain unknown. We estimated this internally generated variability using the Blue Brain Project's detailed model of neocortical microcircuitry with biological noise sources such as synaptic noise and ion-channel noise. We find that the variable neurotransmitter release due to synaptic noise is amplified by recurrent connectivity to cause strong chaotic divergence. Notably, we found that, amid the chaos, relatively weak thalamocortical stimuli can induce

reliable packets of activity with millisecond spike-timing precision. These reliable events rely on the recurrent neocortical connectivity and are not a simple result of feed-forward thalamocortical input. The consequences of our findings are two-fold: instead of averaging out, synaptic noise is a critical component of cortical network dynamics that drives chaos; but at the same time, recurrent neocortical circuitry is equipped with intrinsic mechanisms to produce reliable spike timing amid this chaos.

Poster-ID: 50

Name: William Podlaski, University of Oxford

Title: Enhanced capacity and dynamic gating in a model of context-dependent associative memory

Abstract: An increasing amount of evidence suggests that memory formation and retrieval are modulated by contextual signals, such as behavioral or emotional state. However, typical models of associative memory do not incorporate this dependency. Here we propose an extension to the Hopfield network which takes into account contextual modulation. The network is divided into a set of overlapping subnetworks, each representing a different context with a separate set of memory patterns. Only one subnetwork is active at any given time, thereby reducing interference from memories found in other contexts, which remain dormant through inhibitory control. Using theoretical and numerical methods, we show that these context-modular Hopfield networks have substantially increased memory capacity, as well as robustness to noise and to memory overloading. Their performance depends on two parameters – the number of subnetworks, and their relative size – and when chosen optimally, the capacity is over ten times greater than the standard Hopfield model. Improved performance comes at the cost of limited retrieval, because only memories stored in the active subnetwork can be recalled. To address this, we propose a system in which a controller network dynamically switches the memory network to a desired contextual state before storage or retrieval. Through simulations, we successfully show that this system is able to bias memory retrieval based on context. Overall, our work illustrates the benefits of context-dependent memory, and may have implications for our understanding of cortical memories and their interaction with contextual signals in the prefrontal cortex and hippocampus.

Poster-ID: 51

Name: Hannes Rapp, University of Cologne

Title: Multispike Tempotron performance under different task-related neural spiking statistics

Abstract: The Multispike Tempotron [Gütig, 2016] is a synaptic-like model learning rule for spiking neurons. It is trained to elicit a precise number of spikes in response to a sequence of temporally precise presynaptic spike patterns that is embedded in background spiking activity. Each individual pattern elicits a specific number of spikes. As teaching signal, only the total number of output spikes for the complete input sequence is used. Thus, for successful learning, the neuron is required to arrange the total number of output spikes in time such that the occurrence times of the patterns are matched. The Multispike Tempotron model in [Gütig, 2016] used the homogeneous Poisson process, the commonly used stochastic model for single neuron spiking statistics in the neocortex, to simulate both, the embedded patterns and the background noise. However, the Poisson process is a mathematically convenient but deficient model for the spiking statistics of cortical neurons, which is likely more regular than Poisson (Stevens et al., 1998; Nawrot et al., 2007). Moreover, large and task-related trial-by-trial variability (Churchland et al., 2006; Rickert et al., 2009; Riehle et al. 2017) implies changing network conditions. In this work we examine how different spike train statistics of presynaptic input that deviate from Poisson, impact the learning and generalization capabilities of the Multispike Tempotron.

Poster-ID: 52

Name: Patrick Rose, caesar

Title: Measurement and correction of arbitrary scan pattern aberrations in mobile multi-photon microscopes

Abstract: Mobile multiphoton microscopy combined with genetically encoded fluorescent activity indicators enables measurement of both the spiking activity and spatial location of cortical neurons and their substructures in the freely moving animal. Given the reliance of this scanning method on precise movement of the excitation beam for accurate spatial reconstruction of the tissue, uniform scales throughout the imaging frame are essential in order to quantify distances, shapes, or spatial dependencies. Any distortion of this scanning path leads to a corresponding aberration in the recorded data. To address this problem we analyzed the scanning distortions introduced by the open-loop microscanners (both fiber-scanners and microelectromechanical systems (MEMS) mirrors) used in mobile miniature multi-photon microscopes. For quantification we developed a technique that measures the precise three-dimensional path of the scanning focus by imaging a constructed volume with well-defined fluorescence properties. This method can be applied to any existing scanning microscope and does not rely on assumptions regarding the spatial relationship between individual samples (pixels/voxels) nor the origin of the scanning errors. It allows the

calculation of scan pattern aberrations that can be used to reposition each pixel of recorded data. We applied this technique to activity measurements obtained from cortical neuronal populations expressing the genetically encoded calcium indicator, GCaMP6s. We show that this approach corrects for significant scan pattern aberrations and enables accurate quantification of the spatial-temporal organization of neuronal population activity in the freely moving animal.

Poster-ID: 53

Name: Vahid Rostami, University of Cologne

Title: Inhibitory assemblies play an important role in cortical attractor networks

Abstract: Vahid Rostami1*, Thomas Rost1, Sacha J. van Albada2, Martin P. Nawrot1 1Computational Systems Neuroscience, University of Cologne, Germany 2Institute of Neuroscience and Medicine (INM-6) and Institute for Advanced Simulation (IAS-6) and JARA Institute Brain Structure-Function Relationships (INM-10), Forschungszentrum Jülich, Germany Balanced networks of inhibitory and excitatory neurons with homogeneously random recurrent connectivity are often employed to model local cortical circuits. The balanced random network model exhibits irregular and asynchronous spiking activity similar to that observed in vivo. A recent series of studies [1-3] has extended the balanced random network model to incorporate clusters of strongly interconnected excitatory neurons, with no modularity in the inhibitory population. This clustered topology demonstrates a functionally desired multistability where different clusters become spontaneously activated and inactivated. The model captures a realistic high firing variability of single neurons and the reduction in trial-to-trial variability during stimulation of clusters as observed experimentally. We recently showed that, despite the multistability and trial-to-trial variability that emerge in the clustered excitatory network, this topology leads to widely separated firing rate states of single neurons and tends quickly towards firing rate saturation, which is inconsistent with experimental observations. To overcome this problem we introduced clusters of inhibitory neurons which are coupled to each excitatory cluster [4]. This connectivity scheme is not directly supported by experimental findings. However, recent anatomical and physiological studies point to increased local inhibitory connectivities and possible inhibitory clustering through connection strengths [5-7]. Here we model different architectures of inhibitory circuits, based on these recent experimental studies, and investigate the role of inhibitory clusters on the multistability and trial-to-trial variability of the spiking network when excitatory clusters have strengthened connections with different portions of the inhibitory population. Our model can be reduced to the case of exclusively excitatory clusters [2], or to a one-to-one correspondence of inhibitory and excitatory clusters [4], but we explore all different architectures in between these extreme cases. Such intermediate scenarios are more consistent with recent experimental observations. We find that inhibitory clustering is necessary to achieve realistic spiking activity under stimulation in terms of a biologically realistic firing rate, spiking regularity, and trial-to-trial spike count variability. Inhibitory clustering achieves the desired attractor dynamics over a wide range of

network parameters and thus makes networks robust against parameter fluctuations due to homeostasis or neuromodulation. Remarkably, when the stimulus is weak, without clustering of inhibitory neurons, the spiking network model fails to capture the reduction of trial-to-trial variability during stimulation. Acknowledgments This work is supported by the German Science Foundation under the Institutional Strategy of the University of Cologne within the German Excellence Initiative (DFG-ZUK 81/1). References 1.Deco, Hugues (2012) PLOS Comput. Biol., 8(3): e1002395. 2.Litwin-Kumar, Doiron (2012) Nat. Neurosci., 15(11), 1498–1505. 3.Mazzucato, Fontanini, La Camera (2015) J. Neurosci. 35(21):8214–8231. 4.Rost, Deger, Nawrot (2017) Biol. Cybern., doi: 10.1007/s00422-017-0737-7. 5.Xue, Atallah, Scanziani (2014) Nature, 511(7511), 596–600. 6.Lee, Marchionni, Bezaire, et al., (2014) Neuron 82, 1129–1144. 7.Morishima, Kobayashi, Kato, et al., (2017) Cereb. Cortex 27, 5846–5857.

Poster-ID: 54

Name: Florian Sandhäger, University of Tübingen

Title: Bridging the gap: Visual information in spikes, LFP, EEG and MEG

Abstract: Multivariate classification techniques allow for extracting the information content of neural signals rather than merely measuring activity levels. However, it remains unclear what these methods measure, when applied to MEG and EEG data, and what circuit level processes their results correspond to. To address this, we performed microelectrode recordings in several cortical areas of two macaque monkeys as well as macaque EEG and human MEG during identical visual stimulation with random dot patterns of varying colours and motion directions. We then applied identical multivariate classification analyses on all data, including source-reconstructed EEG and MEG. We found that both motion and colour information were accessible in all signal types. Tuning properties and latencies of the non-invasive signals reflected those of LFP and unit activity in V4 and IT, while diverging from those in dorsal and frontal areas, suggesting that MEG and EEG signals were dominated by early visual and ventral stream sources. Source level analysis of MEG and macaque EEG revealed corresponding gradients of information content and latencies, with earlier and higher information contents in early visual areas. In sum, we show how information-based methods can identify analogous properties of visual processing in signals spanning spatial scales from single units to MEG – a valuable framework for better relating human and animal studies.

Poster-ID: 54

Name: Artur Speiser, caesar

Title: Inference of discrete events from imaging data with variational inference

Abstract: Modern imaging methods in neuroscience and biology often rely on algorithms for extracting variables of interest from the recorded raw data. A common problem is the spatial or temporal localization of discrete events from noisy observations, e.g. extracting spike timings from calcium imaging recordings or localizing particles for super resolution microscopy or particle tracking. We present a framework based on deep neural networks (DNN) for solving these kind of tasks with high accuracy and speed.
In the scenarios we are interested in, ground truth data is often hard to obtain or completely unavailable, ruling out conventional approaches to training the DNN. However, we can instead utilize available biophysical models that describe how the discrete events lead to the observed measurements. The parameters of the network are adjusted together with the parameters of our biophysical model until the process of creating samples with our network and then applying the biophysical model to those samples recreates the observations accurately.
To highlight the potential of this algorithmic approach we adapted our framework for two imaging methods, Two-photon calcium imaging (2PCI) and single-molecule localization microscopy (SMLM). 2CPI is based on the fact that spiking activity in neurons leads to changes in intra-cellular calcium concentration which can be measured by fluorescence microscopy of calcium indicators. The data takes the form of fluorescence time series, or "traces". The variables we want to extract are the exact spike-timings. To this end we use a simple biophysical model that describes how spiking activity produces the observed fluorescence. In the case of SMLM the data consists of 2D images of photon counts where the underlying latents are the activated fluorophores. The generative model describes the purely physical process of a point spread function that models the distribution of the number of photons that can be collected from a single photon emitter. Most algorithms that were developed for this task process each image separately. We show that including information from other frames to account for the photo-activation dynamics of the fluorophores results in a large increase of accuracy. In both applications inference amounts to simply applying the trained DNN to the observations without any need for iterative optimization or sampling, resulting in very short runtimes that would be compatible with real-time applications.

Poster-ID: 56

Name: Daniel Udvary, caesar

Title: Axo-dendritic overlap impacts wiring specificity

Abstract: We present a structurally dense model of the primary somatosensory cortex of the rat. The model allows testing whether and how axo-dendritic overlap impacts synaptic wiring. We find that dense overlap between any two neurons in the model depends on the neurons' cell types, their absolute and relative location. The distribution of the dense overlap results in non-Poisson and non-Gaussian (i.e. specific) distributions. This specificity of the dense overlap at the meso- and microscale may partially result in and/or reflect wiring specificity at the nanoscale. To test the impact of dense overlap onto wiring, we generate a null hypothesis connectome model in which connectivity is solely based on dense overlap with no specificity at the nanoscale. We find that the null hypothesis-based predictions are consistent with a wide range of empirical observations from different experimental approaches like electron microscopy or pair-recordings in tissue slices. We show that the presently available pair-wise connectivity measurements cannot reject the null hypothesis. To provide means to falsify the null hypothesis, we predict first, second and higher-order wiring patterns, which are non-Poisson and non-Gaussian (i.e. specific) distributions. We conclude that wiring specificity at the nanoscale may originate from and/or reflect specificity at meso- and microscale. The present model and analysis provide a quantitative and statistically sound way to separate nanoscale specificity from other sources of wiring specificity.

Poster-ID: 57

Name: Martin Vinck, Ernst Struengmann Institute for Neuroscience in Cooperation with Max Planck Society

Title: Unsupervised clustering of temporal patterns in high-dimensional neuronal ensembles using a novel dissimilarity measure

Abstract: Temporally ordered multi-neuron patterns likely encode information in the brain. We introduce an unsupervised method, SPOTDisClust (Spike Pattern Optimal Transport Dissimilarity Clustering), for their detection from high-dimensional neural ensembles. SPOTDisClust measures similarity between two ensemble spike patterns by determining the minimum transport cost of transforming their corresponding normalized cross-correlation matrices into each other (SPOTDis). Then, it performs density-based clustering based on the resulting inter-pattern dissimilarity matrix. SPOTDisClust does not require binning and can detect complex patterns (beyond sequential activation) even when high levels of out-of-pattern ``noise'' spiking are present. Our method handles efficiently the additional information from increasingly large neuronal ensembles and can detect a number of patterns that far exceeds the number of recorded neurons. In an application to neural ensemble data from macaque monkey V1 cortex, SPOTDisClust can identify different moving

stimulus directions on the sole basis of temporal spiking patterns, but not firing rate patterns.

Poster-ID: 58

Name: Kay-Michael Voit, caesar

Title: Automatic Extraction of 2P Fluorescence Signals from Overlapped Neuronal Structures

Abstract: Multi-photon fluorescence imaging has been established as a standard technique for studying large neuronal populations. However, spatial overlap of neuronal features with each other and neuropil background due to limited z resolution may introduce significant contamination into ROI pixel averages used to derive fluorescence traces, and thus potentially lead to large errors in AP inference. This issue is intensified by recent protein-based calcium sensors like GCaMP6, which cover a very large dynamic range due to their nonlinear reaction to Aps. Therefore, even small overlaps can contribute transients similar to those evoked by multiple spikes in neighboring features. Based on ground truth through subsequent serial section electron microscopy and simultaneous patch-clamp recording and, we show that such influences are significant in real-world data and not reliably removed by existing techniques, leading to false positives in action potential inference. We present an algorithm to decompose a data movie into multiple, potentially overlapping features and their respective fluorescence traces, corresponding to physiological features like neurons, dendrites, and diffuse background fluorescence. The algorithm is based on non-negative matrix factorization (NMF), but features a novel regularization term, specifically tailored to address the challenges of physiological feature separation. We compare our approach to existing techniques and show it’s limits regarding motion, noise and nonlinear effects like saturation.