BP 40: Systems Biology, Evolution and Neural Networks II

Time: Friday 9:30–12:00 Location: ZEU 250

BP 40.1 Fri 9:30 ZEU 250

Towards topological quantum computing in the brain — •Chrsitian Kerskens — Institute of Neuroscience, Trinity College Dublin

In recent years, the mathematical formalism of quantum theory has been adopted to formalise models of cognition. However, the success of quantum cognition over classical approaches opens up the following questions; Is the computing power of the brain sufficient to simulate quantum computation? And if yes, why would nature use so much computational power to simulate quantum computing resulting in a low reliability through non-commutative behaviour and human error? We believe that the brain doesn't simulate quantum computing. Instead, we propose that consciousness and cognition is based on topological quantum computing (TQC), which may be, if at all, the only way of quantum computing reliable enough in the wet and hot environment of the brain . The proposal relies on the experimental findings that the two main ingredients to realise TQC, topological defects and topological phase, may exist in the conscious brain. While the existence and important roles of topological defects are well established, we will focus on questions around the topological phase. Especially, we will discuss why our recent results which showed long-range quantum entanglement in the conscious brain can be interpreted as a topological phase. Further, we will discuss how the experimental results can shed light on the underlying mechanisms which provide the topological-like phase.

BP 40.2 Fri 9:45 ZEU 250

Multiscale activity in circadian clocks — •Pablo Rojas[1], Jenny A. Plath[2], Julia Gestrich[2], Bharath Ananthasubramaniam[3], Hanspeter Herzel[3], Monika Stengl[2], and Martin E. Garcia[1] — [1]Theoretical Physics, University of Kassel, Kassel, Germany — [2]Animal Physiology, University of Kassel, Kassel, Germany — [3]Institute for Theoretical Biology, Humboldt University of Berlin and Charité Universitätsmedizin, Berlin, Germany

The circadian clock orchestrate daily rhythms in physiology, metabolism and behavior. A group of neurons in the brain (the clock) is responsible for this ~24h rhythm. Single neurons in the clock show rhythms with periods ranging from milliseconds (action potential firing) to ~24 hours (circadian expression of clock genes). How cells interact and achieve synchronization in the clock is still a central question. To address this fundamental problem, we performed long term in-vivo electrophysiology (loose-patch clamp) recordings in the cockroach circadian clock. We developed and applied a method, based on wavelets, to detect and analyze electrical events of different timescales, an reduce the complexity of the datasets. We also provide tools for screening and detecting signatures of synchronization and network interaction episodes ranging from minutes to hours, promisingly closing the gap between the fastest and slowest timescales [1]. Our result over experimental datasets are combined with mathematical modeling, in order to describe internal configuration in the clock network.

[1] Rojas, P. et al, Network Neuroscience 2019

BP 40.3 Fri 10:00 ZEU 250

Dynamics and computation with anti-leaky integrate-and-fire neurons — •Paul Manz, Sven Goedeke, and Raoul-Martin Memmesheimer — Universität Bonn

Networks in the brain consist of different types of neurons. Here we investigate the influence of neuron diversity on the dynamics, phase space structure, and computational capabilities of spiking neural networks. We find that already a single neuron of a different type can qualitatively change the network dynamics and that mixed networks may combine the computational capabilities of ones with a single-neuron type. We study inhibitory networks of concave leaky (LIF) and convex "antileaky" (XIF) integrate-and-fire neurons that generalize irregularly spiking nonchaotic LIF neuron networks. Endowed with simple conductance-based synapses for XIF neurons, our networks can generate a balanced state of irregular asynchronous spiking as well. We determine the voltage probability distributions and self-consistent firing rates assuming Poisson input with finite-size spike impacts. Further, we compute the full spectrum of Lyapunov exponents (LEs) and the covariant Lyapunov vectors (CLVs) specifying the corresponding perturbation directions. We find that there is approximately one positive LE for each XIF neuron. This indicates in particular that a single XIF neuron renders the network dynamics chaotic. A simple mean-field approach, which can be justified by properties of the CLVs, explains the finding. As an application, we propose a spike-based computing scheme where our networks serve as computational reservoirs and their different stability properties yield different computational capabilities.

BP 40.4 Fri 10:15 ZEU 250

Dynamics, Statistics and Coding in Random Rate and Binary Networks — •Tobias Kühn[1,2,3], Christian Keup[2,3], David Dahmen[2], and Moritz Helias[2,3] — [1]Laboratoire de Physique Théorique de l'ENS, Paris, France — [2]INM-6, Forschungszentrum Jülich, Germany — [3]Department of Physics, RWTH Aachen, Germany

Cortical neurons communicate with spikes, discrete events in time. Functional network models often employ rate units that are continuously coupled by analog signals. Is there a benefit of discrete signaling? By a unified mean-field theory for large random networks of rate and binary units, we show that both models can be matched to have identical statistics up to second order. Their stimulus processing properties, however, are radically different: contrary to rate networks [Sompolinsky et al. 1988], the chaos transition in binary networks [van Vreeswijk & Sompolinsky 1998] strongly depends on network size, and we discover a chaotic submanifold in binary networks that does not exist in rate models. Its dimensionality increases with time after stimulus onset and reaches a fixed point that depends on the synaptic coupling strength. Low dimensional stimuli are transiently expanded into higher-dimensional representations that live within the manifold. We find that classification performance typically peaks within a single neuronal time constant, after which performance degrades due to variability in the manifold. During this transient, resilience to noise by far exceeds that of rate models with matched statistics, which are always regular. Our theory mechanistically explains all these observations.

BP 40.5 Fri 10:30 ZEU 250

Long-ranged signalling gradients generated by short-ranged molecular interactions — •Johanna Dickmann[1,2], Jochen Rink[2], and Frank Jülicher[1] — [1]Max Planck Institute for the Physics of Complex Systems, Dresden, Germany — [2]Max Planck Institute of Molecular Cell Biology and Genetics, Dresden, Germany

Embryonic development, regeneration, and tissue renewal require tissue organisation as can be provided by signalling molecules forming concentration profiles in space, i.e. signalling gradients. Planarian flatworms are a great model for tissue organisation as they constantly turn over their entire body and are able to regenerate from arbitrary amputation fragments. At a body length of up to 2 cm, they are orders of magnitudes larger than tissue organised during embryonic development in other species, yet, they employ signalling gradients for tissue organisation. In this project we investigate how such long-ranged signalling gradients can be formed. We chose the Wnt signalling gradient organising the main body axis of the worm as a model system. Building a discrete 1D model we account for signalling molecule levels in the extracellular space and signalling levels inside the cells. We consider diffusion and degradation of signalling molecules as suggested to explain signalling gradient formation during development. Motivated by observations in the worm, we add positive feedback. Thus, all cells become sources of signalling molecules. The directionality of the profile is organised by a signal-independent input from one side. The suggested mechanism can explain the formation of signalling gradients with a longer length scale compared to the diffusion/degradation mechanism.

BP 40.6 Fri 10:45 ZEU 250

Model for inference of cell dynamics from C14 data — •Julian Rode[1], Fabian Rost[2], Paula Heinke[1], Enikö Lazar[3], Lutz Brush[1], and Olaf Bergmann[1] — [1]Technische Universität Dresden, Dresden, Germany — [2]Max Planck Institute for the Physics of Complex Systems, Dresden, Germany — [3]Karolinska Institutet, Stockholm, Sweden

Carbon dating is an established method to determine the age of ancient artefacts. Traditionally, radioactive decay changes the C14 ratio of the sample which can be used to determine the age. Recently, a second route has become available as the drastic change of atmospheric C14

due to atomic bomb tests in the 60's allows to invert this classic C14 dating method. Now, the C14 decay is negligible, but the atmospheric C14 changes quickly, allowing an accurate age measurement even of human samples. This method allows to estimate the cell turnover in vivo using the C14 carbon ratio of the DNA from many cells. But a simple matching of C14 values is not sufficient because the measured C14 values are the average of cells with different ages. We introduce a C14-structured population model to predict the average C14 content and accounting for cell division, cell inflow from a fast cycling stem cell population and cell death. Additionally, a priori knowledge such as tissue growth has to be considered resulting in constrains for the model solution. We use variations of this model to analyse C14 data from human liver and muscle tissue.

30 min. coffee break

BP 40.7 Fri 11:30 ZEU 250

Testing developmental reaction-transport models by physical perturbations — •Moritz Kreysing — Max Planck Institute of Molecular Cell Biology and Genetics, Dresden, Germany

From Alan Turing we know that rates of biochemical reactions (in units of seconds) need to be coupled to physical processes to account for the generation of spatial structure (in units of meters) in biology. Suggested morphogenetic driving forces include: passive or active diffusion, directed motor-driven fluxes, and the enigma of cytoplasmic streaming. Since Turing, a great deal of causal insight into the biochemical basis of cellular organization and morphogenesis has been attained by genetic perturbations. In stark contrast to this, the functional role of physical transport in morphogenesis and homeostasis remains very poorly understood. Specifically, we lack the ability to test the functional role of these physical processes inside cells by appropriate perturbations, i.e.: how would one change direction, velocity or temporal persistence of flows within the cytoplasm of a developing embryo? This is clearly not possible by genetics. As a result of this methodological shortcoming, there is hardly one accepted proof of a reaction-transport system in biology. It is now time for experimental biophysics to catch up with molecular biologists and to test great quantitative models of patterning and morphogenesis. I will discuss challenges to test especially reaction-transport systems, while emphasizing chances to interactively guide early organism development via suitable physical perturbations.

Reference: M. Kreysing, Developmental Cell 51, 135-144 (2019)

BP 40.8 Fri 11:45 ZEU 250

Mitochondrial dynamics facilitates precise sensing of metabolic states — •Felix Jonathan Meigel[1], Philipp Mergenthaler[2], and Steffen Rulands[1,3] — [1]Max Planck Institute for the Physics of Complex Systems, Dresden, Germany — [2]Charite - Universitätsmedizin Berlin, Department of Experimental Neurology, Berlin, Germany — [3]Center for Systems Biology Dresden, Dresden, Germany

Cellular behaviour relies on robustly sensing and reacting to fluctuating environmental signals. In recent years, cellular organelles such as mitochondria were recognized as signaling hubs on which different environmental cues are integrated. While the formation of dynamic protein complexes on the outer mitochondrial membrane triggers cell death, the reversible aggregation of these complexes is embedded into a fusion and fission dynamics of the mitochondria themselves. Here, giving the example of the metabolic regulation cell death, we show how the interplay of mitochondrial and protein dynamics facilitates sensitive and specific sensing of fluctuating metabolite levels. By identifying collective degrees of freedom that resemble localised modes in Josephson junction arrays, we demonstrate that such multiscale dynamics form a kinetic low-pass filter that is able to distinguish between fluctuations and biologically informative signals. Our work shows paradigmatically how biological function relies on the integration of non-equilibrium processes on different spatial scales.