Computational Neuroscience

Prof. Dr. Stefan Rotter

Bernstein Center Freiburg & Faculty of Biology

1 Research interests

The Computational Neuroscience Laboratory at the Bernstein Center Freiburg comprises a team of theoreticians from mathematics, physics, biology and various engineering sciences. We are interested in the relations between structure, dynamics and function of the neuronal networks of the brain, with a specific focus on the mammalian neocortex. Our work involves modeling and data analysis of (i) multi-scale neuronal network topology, (ii) spiking activity dynamics of recurrent networks, and (iii) biological function and dysfunction of neuronal networks. Mathematical (deterministic and stochastic) as well as computational methods (large-scale numerical simulations) are employed. The goal is to develop a theory of the brain which also supports a better understanding of the neuronal mechanisms underlying brain diseases.

2 Interdisciplinary teaching & training

Summer term:

- "Computational Neuroscience" (with Carsten Mehring) lecture (4 hours/week) + exercises (2 hours/week)
- "Simulation of Biological Neuronal Networks" practical course (1 week block)
- "Biological Learning & Machine Learning" (or similar) seminar (2 day block)

Winter term:

- Research Internship (individual arrangements)

3 Current lab members

Lena Baum, Neuroscience (MSc candidate)
Nebojša Gašparović, Theoretical Physics (PhD candidate)
Michael Gerlach, Physics & Biology (PhD candidate)
Mohammad Joudy, Physics (PhD candidate)
Ali Mahdavi, Neuroscience (PhD candidate)
Benoit Scholtes, Theoretical Physics (PhD candidate)
Wenqing Wei, Neurobiology (PhD candidate)

4 Dynamic self-organization in recurrent networks

Nerve cells employ pulsed electrical signals (spike trains) to communicate with other neurons. Signals are transmitted over cables (dendrites and axons) to other neurons nearby or at distant sites. If axons contact dendrites (synapses), spikes are transferred from one neuron to another. Due to the electro-chemical nature of transmission, neuronal communication is directed. The recurrent networks formed by neurons and synapses are very complex, and it is hard to predict the dynamics of networks. We use simplified models to compute how the coordination of spikes takes place. Assuming stationarity, we can compute pairwise correlations and correlations of higher order explicitly.

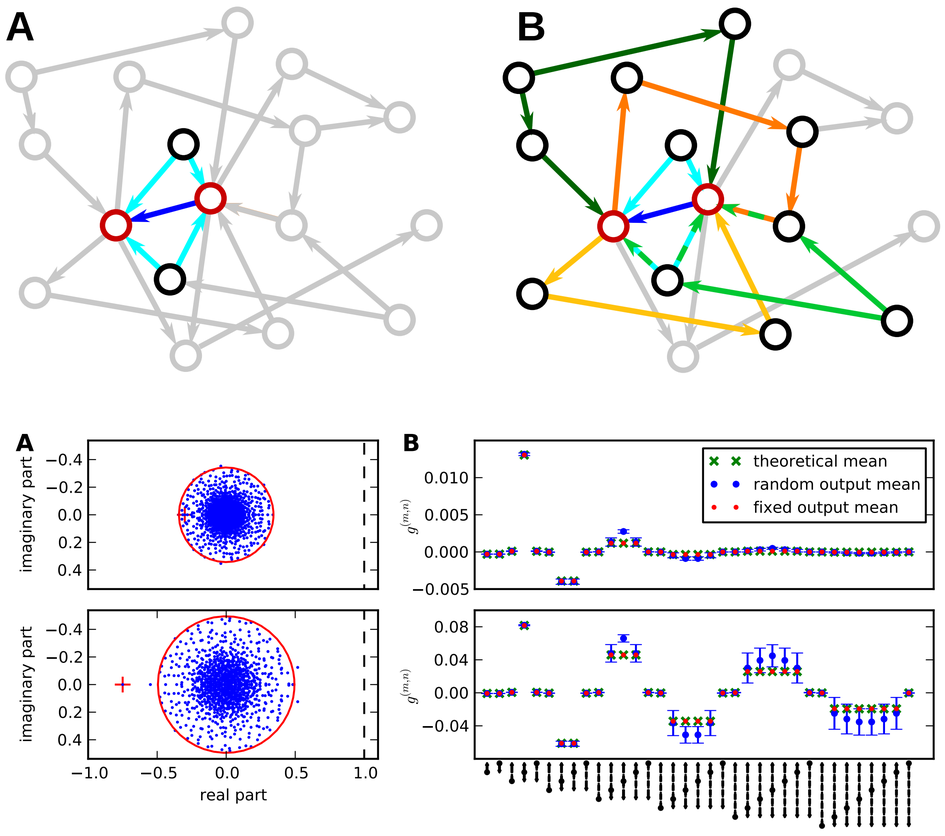

Figure 1: Synaptic interactions (arrows) induce pairwise correlations of neuronal spike trains. The theory of Hawkes processes, an approximation to nonlinear spiking neuronal networks, allows us to quantify the impact of direct contacts, shared input, but also complex muli-synaptic pathways to pairwise correlations. The theory makes explicit statements about the contribution of specific network motifs. The theory can also be generalized to correlations of higher order.

[Publications: Pernice et al. PLOS CB 2011, PRE 2012; Jovanović et al. PRE 2015, PLOS CB 2016]

5 Network models of primary sensory processing

How does the brain process sensory information? In 1981, David H. Hubel and Torsten N. Wiesel received the Nobel Prize in Physiology or Medizine for their discoveries concerning information processing in the visual system. However, the neuronal mechanisms, which underly these observations made in experiments, and which cause the tuning properties of neurons, are still unclear. This is due, among other things, to experimental liminations, as only a very small fraction of neurons can be observed in an experiment. Neuronal network models can make an important contribution here, in particular, when it comes to disentangling system properties of distributed networks. In ongoing projects we study the impact of different spectral sensitivities of light-sensitive cells in the retina for color vision, the contribution of statistics and geometry of thalamo-cortical projections to the emergence of orientation selectivity, and the significance of the layered structure for visual information processing in cortical networks.

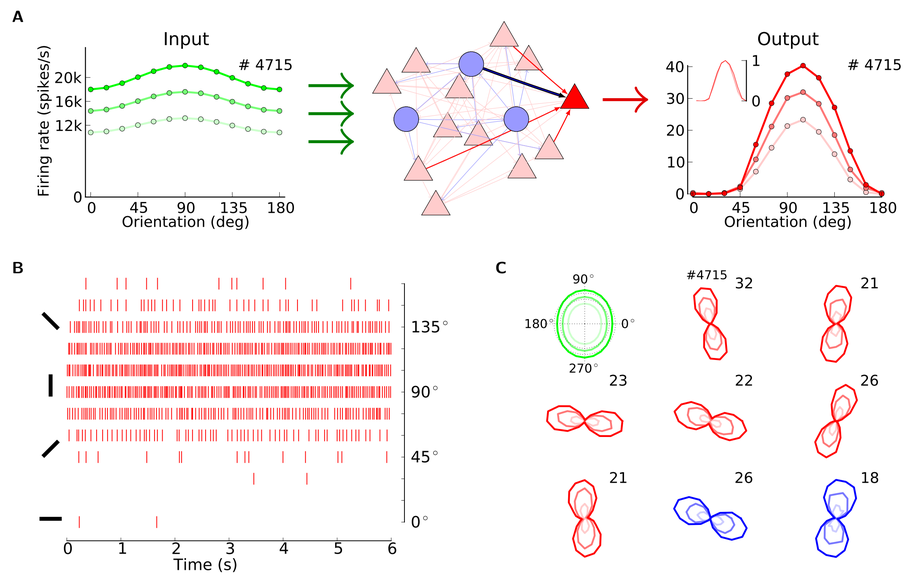

Figure 2: Numerical simulation of a network model of primary visual cortex. The presentation of oriented light bars leads to a small orientation bias of the input (left), and pronounced orientation-specific responses at the output (right). An inhibition-dominated randomly coupled network (middle) performs these computations in a natural way, based on selective attenuation of the non-oriented baseline input.

[Publications: Sadeh et al. Biol. Cyb. 2014, SpringerPlus 2014, PLOS ONE 2014 & 2015, PLOS CB 2015]

6 Structural plasticity and network remodeling

The wiring of brain networks is not fixed, but undergoes continuous remodeling throughout life. New synapses are formed, and existing synapses are removed. As a model, we consider structural plasticity which is homeostatically controlled, keeping neuronal activity at a prescribed level. Experience-dependent structural plasticity shapes the networks during development, but also during learning in the adult. In certain brain diseases (e.g. Epilepsy, Parkinson's Disease, Alzheimer's Disease), pathological connectivity emerges and causes some of the symptoms. It is likely that long-term brain modulation exerted through pharmaceuticals (e.g. in Major Depressive Disorder) or electrical stimulation (e.g. in Parkinson's Disease) also causes structural changes.

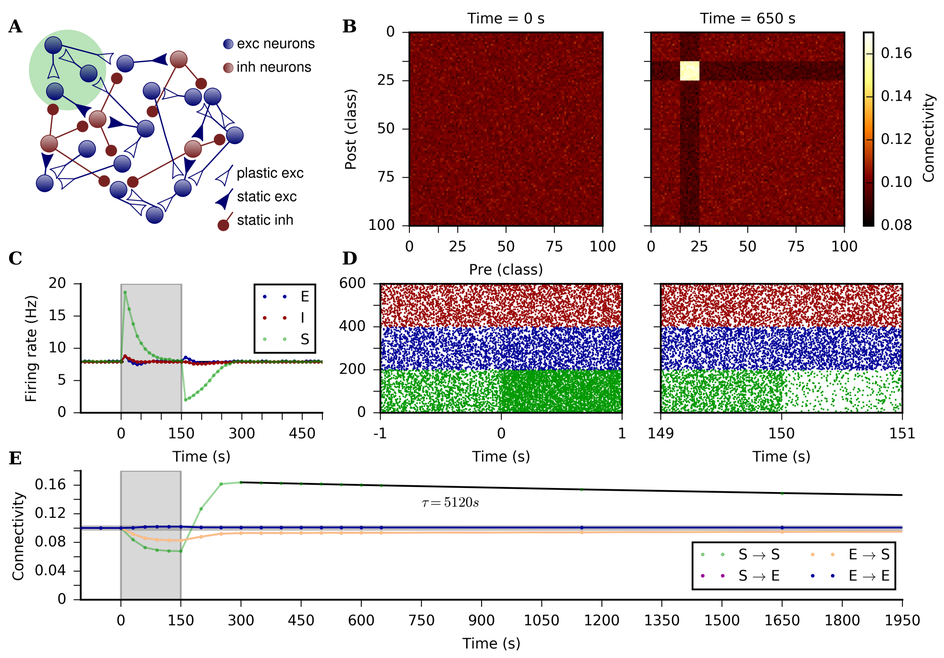

Figure 3: Selective stimulation of a subgroup of excitatory neurons leads to the formation of a neuronal assembly, if homeostatic structural plasticity controls network remodeling. Interestingly, the formation of the assembly is much faster than its decay.

[Publications: Sadeh et al. 2015; Gallinaro et al. 2018, 2020; Lu et al. 2019]

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stefan.rotter@bio.uni-freiburg.de | https://www.bcf.uni-freiburg.de/about/people/Rotter