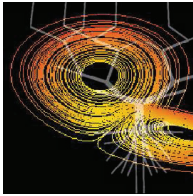


Applied Computational Neuroscience: Sleep, Neuroengineering and Dynamics



Scientific programme



Monday, 20. October 2010

Introduction and Overview

Jens Christian Claussen (Lübeck)

Mathematical Neuroscience: from neurons to networks

Stephen Coombes (Nottingham, UK)

The tools of dynamical systems theory are having an increasing impact on our understanding of patterns of neural activity. In the first part of my talk I will introduce some of the more popular single neuron models and explain their behaviour in terms of bifurcation diagrams, phase-planes and phase-response curves. For limit cycle oscillators I will review the coupled oscillator approach that has provided a framework for understanding behaviour in neural networks with weak synaptic and gap junction coupling. I will then show how results for strong coupling can be obtained by focusing on a specific class of spiking neural models, namely (non-smooth) planar integrate-and-fire models. In the second part of my talk I will describe how to build tractable tissue level models that maintain a strong link with biophysical reality. These models typically take the form of nonlinear integro-differential equations. Their non-local nature has led to the development of a set of analytical and numerical tools for the study of waves, bumps and patterns, based around natural extensions of those used for local differential equation models. Here I will present an overview of these techniques, and discuss the relevance of neural field models for describing the brain at the large scales necessary for interpreting EEG data.

Onset of excitability in single neurons and in neural populations

Alistair Steyn-Ross (Waikato, NZ)

An “excitable” membrane is so-called because it is capable of generating an action potential spike. This transition from subthreshold linear fluctuations to a nonlinear spiking dynamics is an abrupt change of state whose onset is characterized by extreme sensitivity to stimulus. This increase in neural sensitivity is also observed in the EEG signals generated by populations of cooperating neurons as they approach a large-scale state change such as that between slow-wave and REM stages of sleep, or on approach to anaesthetic-induced changes in states of consciousness. In this talk we explore the subthreshold properties of both complex and simple single-neuron models, and identify the minimum requirement for a neural model to be “excitable”.

What is an appropriate model for calcium dynamics in a complete neuron simulation?

Erik De Schutter (Okinawa)

I will cover different models of calcium dynamics from the phenomenological pool model to detailed biophysical models. I will briefly describe how calcium dynamics change strongly in the presence of buffers. These changes are not captured very well by the pool model but can be well approximated by models including buffers but no diffusion.

Network mechanisms generating slow and fast cortical rhythms

Maria Sanchez-Vives (Barcelona)

Basal network excitability and recurrent connectivity in the cerebral cortex network allows neuronal firing that reverberates in the circuit, resulting in an emergent network activity. During slow-wave sleep and anesthesia, cortical spontaneous activity is organized into a slow (μ Hz) rhythmic pattern consisting of interspersed Up (or active) and Down (or silent) states. A very similar activity emerges from cortical slices, as far as some level of excitability allows spontaneous firing (Sanchez-Vives and McCormick, *Nat Neurosci* 3:1027, 2000). Further, local activity is spontaneously synchronized in fast, beta and gamma rhythms during Up states both in vivo (Steriade et al., *J Neurosci* 16: 392, 1996; Hasenstaub et al., *Neuron* 47:423) and in cortical slices (Compte et al., *J Neurosci* 28: 13828, 2008). In this talk I will first discuss some of the cellular and network mechanisms that determine the generation and propagation of cortical rhythmic activity. Next, how rhythmicity and synchronicity are transformed when there is a transition to a different brain state or alteration of physiological variables such as temperature. An emphasis will be made on the cortical excitatory / inhibitory balance and how progressive alterations of this balance induces parametric alterations of rhythmicity (Sanchez-Vives et al., *J Neurophys* epub 2010). Progressive reduction of inhibition in the in vitro cortical network induces linear changes in different parameters of Up and Down states, linearity that is lost when the activity becomes epileptiform. The mechanistic and connectivity rules that support our experimental findings are explored using a biologically inspired computer model of the cortical network.

A model of synaptic tagging:

Claudia Clopath (Paris)

Changes in synaptic efficacies need to be long-lasting in order to serve as a substrate for memory. Experimentally, synaptic plasticity exhibits phases covering the induction of long-term potentiation and depression (LTP/LTD) during the early phase of synaptic plasticity, the setting of synaptic tags, a trigger process for protein synthesis, and a slow transition leading to synaptic consolidation during the late phase of synaptic plasticity. We present a mathematical model that describes these different phases of synaptic plasticity. The model explains a large body of experimental data on synaptic tagging and capture, cross-tagging, and the late phases of LTP and LTD. Moreover, the model accounts for the dependence of LTP and LTD induction on voltage and presynaptic stimulation frequency. The stabilization of potentiated synapses during the transition from early to late LTP occurs by protein synthesis dynamics that is shared by groups of synapses. The functional consequence of this shared process is that previously stabilized patterns of strong or weak synapses onto the same postsynaptic neuron are well protected against later changes induced by LTP/LTD protocols at individual synapses.

Stochastic modeling of signaling pathways in a dendritic spine

Erik De Schutter (Okinawa)

Stochastic modeling of signaling pathways in a dendritic spine I will first describe different stochastic simulation methods for reaction-diffusion modeling, with an emphasis on the Gillespie method. I will then briefly describe STEPS, a stochastic reaction-diffusion simulator, followed by an example implementation: the induction of long-term depression at the parallel fiber to Purkinje synapse.

Welcome Reception at Rathaus

Bernd Saxe (Major of the City of Lübeck): Welcome from the Hanse City Lübeck

Cornelius Borck (Institute for History in Medicine, Lübeck): Surfing on the Sea of Brain Waves

Tuesday, 21. October 2010

Mean-field modelling of the cortex: applications to phase transitions

Moira Steyn-Ross (Waikato, NZ)

This talk describes a mean-field model of the cortex envisioned as sets of interacting populations of excitatory and inhibitory neurons which communicate via chemical (neurotransmitter controlled) and electrical (gap junction) synapses. The model consists of a closed set of stochastic differential equations describing the spatially averaged behaviour of the firing rates of neurons. For low levels of gap junction diffusivity, and in the limit in which soma response is slow relative to dendritic events, the model predicts first-order phase transitions at (a) the point of loss of consciousness and at return to consciousness during general anaesthesia, and (b) at the transition between slow-wave and REM sleep states.

Modelling anaesthetic-induced change in brain state and emergence of hysteresis in animal experiments

Alistair Steyn-Ross (Waikato, NZ)

We review some historical animal and human anaesthesia studies highlighting the surprisingly nonlinear brain response to increases in anaesthetic drug concentration. By altering the strength of the inhibitory postsynaptic potential in a drug-dependent manner, we are able to demonstrate similar nonlinear EEG responses in a mean-field model of the cortex, including both the biphasic surge in activity at low concentrations, and the prediction of hysteresis: recovery of consciousness should occur at a lower drug concentration than that required to induce it. Traditionally, such drug-effect lag has been interpreted as a purely pharmacokinetic (PK) delay with no deeper significance. We analyze a recent sheep propofol experiment carefully instrumented to minimize PK-lag errors, and find a robust hysteresis at lower EEG frequencies. We comment briefly on a recent finding of neural inertia in anaesthetized mice and flies, and suggest that a first-order phase-transition picture provides a natural explanation for these observations.

Bridging the gap between anaesthetic action in synaptic receptors and the EEG

Axel Hutt (Nancy)

To understand the neural origin of anaesthesia and sleep, the corresponding electroencephalographic data (EEG) has been studied in detail in recent years. In general anaesthesia (GA), the microscopic action of the anaesthetic drug on synaptic receptors is known, but it is difficult to understand the origin of the macroscopic EEG based on the microscopic drug action. Hence a successful model of GA has to bridge over the different spatial and temporal scales. At first, the talk discusses the similarities and differences of sleep and anaesthesia and then introduces briefly to the generation of EEG by neural sources. These sources might be modelled by neural populations and the talk will discuss such a population model that takes into account the synaptic action of anaesthetic drugs.

Slow oscillations orchestrating system-consolidation of memory during sleep

Jan Born (Lübeck)

Slow-wave sleep (SWS) facilitates the consolidation of declarative memory (for facts, episodes), i.e., a system-level consolidation process assumed to involve the redistribution of the memory representations from temporary hippocampal to neocortical long-term storage sites. Evidence will be provided indicating that this consolidation relies on a dialogue between neocortex and hippocampus which is essentially orchestrated by the ≈ 1 Hz EEG slow oscillation (SO). The SOs characterising SWS originate from neocortical networks. Their amplitude depends partly on the use of these networks for encoding of information, i.e., the more information is encoded during waking, the higher the SO amplitude during subsequent SWS. The SOs temporally group neuronal activity into up-states (of strongly enhanced activity) and down-states (of neuronal silence). This grouping is induced not only in the neocortex but also, via efferent pathways, in other structures relevant to consolidation, i.e., in the thalamus, generating 10-15 Hz spindles, and in the hippocampus, generating sharp-wave ripples which are known to accompany a replay of newly encoded memories taking place in hippocampal circuitries during SWS. The feedforward synchronizing effect of the SO enables memory-related inputs to be synchronously fed back from these (hippocampus, thalamus) and other structures to the neocortex. The co-occurrence in the neocortex of these feedback-inputs possibly plays a critical role for the long-term storage of memories in neocortical networks. Indeed, induction of slow oscillations during NonREM sleep (but not during REM sleep or waking) by slowly alternating transcranial current stimulation not only enhances and synchronizes spindle activity but also improves the consolidation of declarative memory.

Neural fields: derivation and application to anaesthesia and feedback systems

Axel Hutt (Nancy)

The talk first motivates and derives the neural field equations. Afterwards, it discusses the application to the synaptic action of general anaesthetics. Finally, a nonlocal feedback model is discussed which is motivated by the sensory feedback loop in weakly electric fish.

Frequency and Spike-Timing dependent plasticity interact in a recurrent network

Claudia Clopath (Paris)

Electrophysiological connectivity patterns in cortex often show a few strong connections in a sea of weak connections. In some brain areas a large fraction of strong connections are bidirectional, in others they are mainly unidirectional. In order to explain these connectivity patterns, we use a model of Spike-Timing-Dependent Plasticity where synaptic changes depend on presynaptic spike arrival and the postsynaptic membrane potential, filtered with two different time constants. The model describes several nonlinear effects in STDP experiments, as well as the voltage dependence of plasticity under voltage clamp and classical paradigms of LTP/LTD induction. We show that in a simulated recurrent network of spiking neurons our plasticity rule leads not only to development of localized receptive fields, but also to connectivity patterns that reflect the neural code: for temporal coding paradigms with spatio-temporal input correlations, strong connections are predominantly unidirectional, whereas they are bidirectional under rate coded input with spatial correlations only. Thus variable connectivity patterns in the brain, mainly unidirectional in barrel cortex versus bidirectional in visual cortex, could reflect different coding principles across brain areas.

Role of Potassium Currents in the Auditory Adaption in the Awake Rat

Maria Sanchez-Vives (Barcelona)

Responses to sound in the auditory cortex are influenced by the preceding history of firing. We studied the time course of auditory adaptation in primary auditory cortex (A1) from awake, freely moving rats. Two identical stimuli were delivered with different intervals ranging from 50 ms to 8 s. Single neuron recordings in the awake animal revealed that the response to a sound is influenced by sounds delivered even several seconds earlier, the second one usually yielding a weaker response. To understand the role of neuronal intrinsic properties in this phenomenon we obtained intracellular recordings from rat A1 neurons in vitro and mimicked the same protocols of adaptation carried out in awake animals by means of depolarizing pulses of identical duration and intervals. The intensity of the pulses was adjusted such that the first pulse would evoke a similar number of spikes as its equivalent in vivo. A1 neurons in vitro adapted with a similar time course, but less than in awake animals. At least two potassium currents participated in the in vitro adaptation: a Na⁺-dependent K⁺ current and an apamin-sensitive K⁺ current. Our results suggest that potassium currents underlie at least part of cortical auditory adaptation during the awake state.

Biological pattern generators for design of oscillatory motor control systems

Ranu Jung (Arizona)

Computational Neuroscience seeks to utilize quantitative methods to investigate and analyze the behavior and function of neural systems at multiple scales. Non-linear mathematical models are often used to describe the systems. The models can also be used in the design of biomedical control systems. In this lecture, I will discuss small networks that form central pattern generators (biological neural networks that can produce periodic output without periodic forcing) for control of rhythmic motor movement. We will look at examples from physiological systems and discuss ways to model them. Topics may include: Respiratory and locomotor pattern generators; van der Pol, Fitzugh-Nagumao, Morris-Lecar models, Coupled nonlinear oscillators for quadruped gait; Network oscillators with embedded pacemakers.

Poster session (with fingerfood)

Lower Foyer (1 floor below reception entrance)

Wednesday, 22. October 2010

Lab rotation

Visit of Neuroengineering labs on campus (in small groups)

What can we learn about neural activity from extracellular potentials recorded in the brain?

Gaute Einevoll (Ås)

Mathematical modeling rely on experimental data to make progress, both to constrain and to test the models. In neuroscience the dominant experimental method in vivo has so far been single-unit extracellular recordings: when a sharp electrode is placed sufficiently close to the soma of a particular neuron, the recorded potential reliably measures the firing of individual action potentials in this neuron. This information is contained in the high-frequency part of the recorded potentials. The low-frequency part, that is, the local field potentials (LFP), has proved much more difficult to interpret and has typically been discarded.

Other experimental methods, particularly methods that measure population-level activity in vivo, are needed to facilitate development of biologically relevant cortical network models. Large-scale electrical recordings using various types of multielectrodes, that is, electrodes with many contacts, are one such option. As techniques for such recordings are rapidly improving, there is a need for new methods for extraction of relevant information from such data. Extracellular potentials in the brain are in general due to complicated weighted sums of contributions from transmembrane currents, and the potentials can be calculated by a combination of compartmental modeling providing the transmembrane currents following neural activity and electrostatic forward modeling using the quasistatic version of Maxwell's equations.

In the seminar I will present results from several projects in our group using this computational scheme to (i) elucidate the link between recorded extracellular potentials and the underlying neural activity and (ii) develop new analysis methods for multielectrode data.

Thursday, 23.October 2010

Modeling the thalamocortical system of mammals

Gaute Einevoll (Ås)

Sensory input to the brain typically reaches the primary sensory cortices via thalamic nuclei. For example, visual signals reach primary visual cortex (V1) via relay cells in the lateral geniculate nucleus (LGN), while tactile signals from whiskers reach the barrel cortex (S1) via neuron in a thalamic nucleus called VPM. A striking feature of the organization of these sensory pathways is the significant feedback from the primary sensory cortices to the corresponding thalamic nuclei providing the feedforward input.

The functional role of these thalamocortical circuits are still poorly understood, and in the seminar two different modeling projects aimed at elucidating their properties are described: one for the thalamocortical circuit in the rat whisker system, the other for the corresponding circuit in the cat visual system.

Anaesthesia and brain - neurophysiological monitoring

Hartmut Gehring (Lübeck)

Main columns of anaesthesia are the deep sleep (narcosis) and analgesia. The pharmacological effects of anesthetic drugs in the brain are not explained in detail. Control of the depth of anaesthesia with respect to rapid induction and recovery are determined also by economical considerations. Therefore indirect parameters including neurophysiological monitoring are chosen for assessment of anaesthesia function. This demand of neurophysiological monitoring is underlined when cerebral deficits pre-exist or interventions with a high risk of a cerebral damage are scheduled. The role of neuromonitoring in the prevention of cerebral damage associated with cardiosurgical interventions has not yet been clearly elucidated. Reliable randomised studies from evidence-based medicine showing a clear reduction of risk do not exist. Numerous studies and reviews however, have confirmed that non-invasive procedures for monitoring neuronal or neurophysiological changes before, during and after interventions within the heart or the major thoracic vessels are available and provide early indications of damage. In the presentation technological modalities and clinical indications for the non invasive cerebral monitoring were evaluated:

- 1.Electroencephalography (EEG) with processed EEG, bispectral index (BIS) and the evoked potential for use with spinal cord function
- 2.Near infrared spectroscopy (NIRS) for assessment of cerebral perfusion and oxygenation
- 3.Transcranial Doppler sonography (TCDS) for assessment of cerebral circulation and perfusion
- 4.Multimodality monitoring as a combination of EEG, NIRS and TCDS.

Interacting Hopf and Turing instabilities in the cortex: resting states and seizure

Moira Steyn-Ross (Waikato, NZ)

This presentation explores the dynamical behaviour of a mean-field model of the cortex as the relative balance between chemical and electrical synapses is altered. When the soma response is slow relative to dendritic events, the model predicts the emergence of a Turing instability, generated by inhibitory gap-junction mediated diffusion, manifesting as spatial patterning of neural activation. In addition, a low-frequency temporal oscillation, in the form of a Hopf instability, emerges when the inhibitory dendritic rate-constant is reduced. Nonlinear interactions between the Turing and Hopf instabilities give rise to an ultra-slow spatiotemporal oscillation in cortical activity reminiscent of the resting state of the cortex. Altering the balance between the Turing and Hopf modes provides a mechanism for seizure initiation and control.

Sleep oscillations and the complex dynamics of the sleeping brain - insights from the time series analysis of the human sleep EEG

Eckehard Olbrich (Leipzig)

The dynamics of the sleeping brain is governed by multiple time scales: The typical EEG oscillations in the frequency range from 1 to 40 Hz, the so called slow oscillation (0.1-1 Hz), the transitions between the sleep stages at a scale of 10s to a few minutes, the NREM-REM sleep cycle (60-90 min) and the dynamics of sleep regulation with the homeostatic component and the 24h circadian rhythm. The ultimate aim is to integrate these different time scales in one dynamical model. This can be done either by starting from the physiological mechanisms and constructing appropriate models or by analyzing the dynamics from time series such as the EEG - which is my approach. The starting point is the fastest time scale - the sleep oscillations. The most important oscillations in the sleep EEG are slow waves and the sleep spindles. They are essential in the definition of sleep stages and received recently additional attention because of their hypothesized involvement in processes related to neuronal plasticity, learning and memory. I will present a method based on modelling the EEG time series by adaptive linear models that allows to identify and characterize such oscillations. The parameters of the model can be mapped to the frequencies and dampings of these oscillations which then can be related to the oscillatory eigenmodes of the underlying thalamocortical networks. The changes of event frequencies and incidences as a function of sleep stage and cycle as well as effects of sleep deprivation can be considered as indicators for specific changes in the underlying neural networks governed by a slower modulatory dynamics.

References:

E. Olbrich and P. Achermann Analysis of oscillatory patterns in the human sleep EEG using a novel detection algorithm, *Journal of Sleep Research* 14, (2005), 337-346.

E. Olbrich and T. Wennekers Dynamics of parameters of neurophysiological models from phenomenological EEG modelling, *Neurocomputing* 70, (2007), 1848-1852.

Evidence of plasticity and learning during recovery in a chronic animal model of ischemic stroke

Winnie Jensen (Ålborg)

Stroke is third leading cause of mortality following heart disease and cancer in the Western industrialized countries. Cerebral ischemia is caused by occlusion of the vascular supply, and 80-85% of all strokes are ischemic in nature. A blockage of the blood supply initiates a series of events, but will typically result in an ischemic area that are damaged within minutes or hours (i.e. the core), and a surrounding area that are less ischemic (also referred to as the peri-infarct zone or penumbra). The neural basis for motor recovery has not been fully elucidated, however, it likely involves cerebral reorganization or neuroplasticity, which can include functional compensation within residual or contra-lateral cortical areas or structural changes. Cerebral reorganization following stroke has typically been investigated using neurophysiologic, neuroanatomic and neuroimaging studies. The use of intra-cortical recordings is today a well-established and widely used method to study single or multi-unit cell functionality, coding and plasticity in animals. However, placement of micro-electrode arrays in the cortical tissue has not traditionally been used to evaluate the direct, intra-cortical response following brain injury such as stroke. The presentation will first discuss basic concepts of plasticity, neurological mechanisms and rehabilitation in stroke, and secondly how the plasticity and neurological mechanisms may be monitored through intra-cortical recordings from the motor cortex in acute and chronic animal studies.

Boosting slow oscillations during sleep potentiates memory

Lisa Marshall (Lübeck)

There is compelling evidence that sleep contributes to the long-term consolidation of new memories¹. This function of sleep has been linked to slow (<1 Hz) potential oscillations, which predominantly arise from the prefrontal neocortex and characterize slow wave sleep. However, oscillations in brain potentials are commonly considered to be mere epiphenomena that reflect synchronized activity arising from neuronal networks, which links the membrane and synaptic processes of these neurons in time⁵. Whether brain potentials and their extracellular equivalent have any physiological meaning per se is unclear, but can easily be investigated by inducing the extracellular oscillating potential fields of interest. Here we show that inducing slow oscillation-like potential fields by transcranial application of oscillating potentials (0.75 Hz) during early nocturnal non-rapid-eye-movement sleep, that is, a period of emerging slow wave sleep, enhances the retention of hippocampus-dependent declarative memories in healthy humans. The slowly oscillating potential stimulation induced an immediate increase in slow wave sleep, endogenous cortical slow oscillations and slow spindle activity in the frontal cortex. Brain stimulation with oscillations at 5 Hz another frequency band that normally predominates during rapid-eye-movement sleep decreased slow oscillations and left declarative memory unchanged. Our findings indicate that endogenous slow potential oscillations have a causal role in the sleep-associated consolidation of memory, and that this role is enhanced by field effects in cortical extracellular space.

Friday, 24. October 2010

Nonlinear time series analysis of neural data - problems, limitations and challenges

Eckehard Olbrich (Leipzig)

First I will give a short introduction to nonlinear time series analysis with the emphasis on phase space reconstruction and the estimation of dynamical invariants, such as attractor dimension, KS entropy and Lyapunov exponents. In the following I will critical review some early attempts to apply these methods to neural data and in particular to sleep EEG. Finally I will discuss some more recent developments using concepts from nonlinear time series analysis for the analysis of functional connectivity and synchronization in neural systems.

References:

Y. Shen, E. Olbrich, P. Achermann and P. F. Meier Dimensional complexity and spectral properties of the human sleep EEG, *Clin. Neurophysiol.* 114, (2003), 199-209. C.J. Stam Nonlinear dynamical analysis of EEG and MEG: Review of an emerging field, *Clin. Neurophysiol.* 116 (2005), 2266-2301.

Neural implants in the peripheral nervous system

Ranu Jung (Arizona)

Neural recording and stimulation systems are increasingly being interfaced with the nervous system in order to influence it or access information from the nervous system. In this lecture we will look at interfaces to the peripheral nervous system. We will look at different electrodes that can directly or indirectly activate the peripheral nerves and discuss some of the applications. We will see how modeling may be used for designing appropriate electrode-nerve interfaces and we will examine an adaptive control system for activating the peripheral nervous system for rhythmic motor control via functional electrical stimulation.

Current challenges in invasive BCI - decoding of intracortical signals

Winnie Jensen (Ålborg)

Intra-cortical brain computer interfacing (BCI) aims at restoring movement control in severely disabled people (e.g. spinal cord injury patients). The method consists in creating direct communication pathways between the brain and an external device, independently of the normal output pathways of the brain based on peripheral nerves and muscles systems. A typical closed-loop invasive BCI system usually consists of a neural interface to collect ensemble recordings from motor parts of the brain, signal-to-noise enhancement and spike detection modules, decomposition of multi-unit recordings into their constituent sources to identify the discharge patterns of each neurons, extraction of motor commands from neuronal discharge rhythms and control of an actuator that can be sensed by the subject to link between the intention of the movement and the action. This presentation will first provide an overview of the current invasive BCI systems and discuss the advantages and limitations in the clinic. Secondly, a specific example signal-to-noise enhancement and classification of units based in intracortical recordings will be presented.

A mathematical Model of Orexin/Hypocretin Effects in Sleep-Wake Regulation

Hans A. Braun (Marburg)

Transitions from wake to sleep are accompanied by significant alterations of impulse patterns and synchronization in thalamocortical circuits and distinct activity changes in diverse hypothalamic nuclei. The question is whether and how these alterations can be related to the generally accepted concept of sleep regulation which postulates that sleep-wake transitions result from the interaction between a circadian and a homeostatic process. While the circadian process can be ascribed to the neuronal activity in the suprachiasmatic nucleus, the mechanism of the homeostatic process are still unclear. Here we present a new concept of orexin/hypocretin-based control of sleep homeostasis which has been realized in a mathematical model with Hodgkin-Huxley-type neurons and physiology-based synapses (Postnova et al., J Biol Rhythms 2009, 24: 523-535, Postnova et al., Pharmacopsych 2010, 43: S82-S91). We propose that 1) high frequent impulse activity of hypocretin/orexin neurons during wakefulness is sustained by reciprocal excitatory connections; 2) transition to the silent state (sleep) results from activity dependent weakening of the synaptic efficacy of orexin/hypocretin; 3) sustained activity can be reinstated by the circadian pacemaker when the synaptic efficacy has sufficiently recovered; 4) depending on the input from the orexin/hypocretin neurons the thalamic neurons undergo transitions between asynchronous tonic firing activity during wakefulness and synchronized bursting discharges during sleep. In combination with a circadian input, the model mimics the transitions between silent and firing states of hypothalamic neurons as well as thalamic transitions between asynchronous tonic firing and synchronized bursting in full agreement with sleep-wake cycles.

(Supported by the European Union through the Network of Excellence BioSim contract No LSHB-CT-2004-005137.)

References:

Svetlana Postnova, Karlheinz Voigt, and Hans A. Braun, J. Biol Rhythms 2009, 24: 523-535

Svetlana Postnova et al., Pharmacopsych 2010, 43: S82-S91

Understanding hierarchies in human odor quality by analysis of rat olfactory bulb activity patterns

Amir Madany Mamlouk (Lübeck)

On the way to quantify the sense of smell, some key questions remained unanswered. Especially, it is still not clear what are the neuronal correlates of human odor perception. In previous work, we proposed a set of maps to quantify human odor quality, based on verbal descriptions only. To interpret these results in a sensory context, we will discuss the derivation of functional module maps from rat olfactory bulb uptake images, which correspond to input specific activity patterns. Grouping the activities of different odorants in the context of their chemical properties and psychophysical descriptions of odor quality, we used linear support vector machines to derive meaningful decision images and motivate how these results can be used to organize the verbal odor descriptors in an hierarchical context. Such modules might have an twofold impact: They might help to systematically localize target regions for specific binding receptor genes, just the same as they will give insights into the potential coding strategies of the olfactory system up into the pyriform cortex.

Round table, wrap-up and Closing

Ulrich Hofmann (Lübeck)

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