



## Introductory Remarks to Symposium 1

### Astrocytes as new targets for antiepileptic drugs

*Peter Bedner and Kjell Heuser, Bonn and Oslo (Norway)*

Epilepsy is a disorder of the brain characterised by unprovoked, recurrent seizures and affects about 1% of the population worldwide. A deeper understanding of the cellular basis of the epilepsies would be essential for the identification of novel targets for therapeutic intervention. Since neuronal hyperexcitation and hypersynchronization are hallmarks of epilepsy, the search for new antiepileptic drugs (AEDs) has concentrated so far mainly on compounds that affect neuronal functions. However, the efficacy and tolerability of these drugs have not substantially improved over the past decades, and all known AEDs merely suppress the symptoms without treating the underlying disorder. Hence, new strategies for the development of more efficacious AEDs are required. In this context glial cells, astrocytes in particular, have attracted increasing attention. These cells play essential roles in brain physiology: they modulate synaptic transmission by release, uptake, degradation and recycling of transmitters, and control ion homeostasis and blood–brain-barrier integrity. Impairment of these functions has been associated with the pathophysiology of epilepsy. This symposium will highlight an alternative view on the etiopathogenesis of epilepsy and challenge the commonly accepted neurocentric view of epileptogenesis. We will bring together scientists with basic research background and practising clinicians working on different aspects of neuron-glia interactions in epilepsy. The symposium will be opened by Peter Bedner with a brief introduction to clinical and neuropathological characteristics of epilepsy and the possible role of dysfunctional astrocytes in the development and progression of the disorder. The first speaker, Tore Eid, will talk about the astrocytic regulation of glutamate homeostasis in epilepsy. Eleonora Aronica will reveal the role of astrocyte immune responses in epilepsy. Kjell Heuser will present new evidence of altered astrocytic  $\text{Ca}^{2+}$  signaling in the early stage of epileptogenesis. Finally, Peter Bedner will discuss the involvement of impaired astrocytic gap junction coupling in the development and progression of temporal lobe epilepsy.

## Symposium 1

*Wednesday, March 18, 2015*  
*14:30 – 16:30, Lecture Hall 10*

Chair: Peter Bedner and Kjell Heuser,  
Bonn and Oslo (Norway)

14:30 **Opening Remarks**

14.40 Tore Eid, New Haven, USA  
ASTROCYTES, GLUTAMINE SYNTHETASE AND  
EPILEPSY (S1-1)

15:05 Eleonora Aronica, Amsterdam, The Netherlands  
ASTROCYTE IMMUNE RESPONSES IN EPILEPSY  
(S1-2)

15:30 Kjell Heuser, Oslo, Norway  
STIMULATION-INDUCED CHANGES OF  
ASTROCYTIC  $CA^{2+}$  SIGNALING DURING THE  
LATENT PERIOD OF MESIAL TEMPORAL LOBE  
EPILEPSY (S1-3)

15:55 Peter Bedner, Bonn  
ASTROCYTE UNCOUPLING AS A CAUSE OF  
HUMAN TEMPORAL LOBE EPILEPSY (S1-4)

16:20 **Concluding Remarks**



## Introductory Remarks to Symposium 2

# Neuronal basis of vocal communication in vertebrates - from genes to physiology to behavior

*Boris Chagnaud and Steffen R. Hage, Planegg-Martinsried and Tübingen*

Vocal behavior is a fundamental aspect of vertebrate communication and of uttermost importance in our everyday's life. In general, vocal communication can be subdivided into learned vocal patterns such as bird song or human speech and genetically pre-programmed vocalizations, which include most other vertebrate vocal utterances. At any time, vocal production mechanisms are inherently linked with the auditory system at each level of the brain. Such audio-vocal integration processes are crucial for proper vocal output since animals do rely on auditory feed-forward and feedback mechanisms to adapt their vocal behavior. Birds, for example, do not learn their songs without auditory perception, bats highly rely on a tight link between vocal production and auditory perception to perform proper echolocation and there is no meaningful vocal communication without auditory input in any vertebrate.

Within this conceptual framework vocalization is an ideal model with high behavioral relevance to study adaptive behavior at all organizational levels of the brain. Recent findings across vertebrate species gave novel insights into vocal production and perception mechanisms as well as into how vocal motor and auditory networks interact. The major purpose of the proposed symposium is to frame these developments into the context of auditory circuit modulation by vocal production networks across vertebrate classes (from fish to birds to bats to primates) rather than only to provide an overview of recent developments in the field of vocal pattern generation.

Research within the field of vocal production and perception mechanisms as well as audio-vocal processing strongly benefits from a wide diversity of animal models each of which is ideally suited to answer specific questions which will be reflected within this symposium. The symposium will guide the audience from brainstem-based to higher order motor pattern generating processes to cognitive control of vocal output. In addition, we will particularly focus on audio-vocal interactions and auditory-motor modulation of vocal output on each brain level.

## Symposium 2

Wednesday, March 18, 2015  
14:30 – 16:30, Lecture Hall 101

Chair: Boris Chagnaud and Steffen R. Hage,  
Planegg-Martinsried and Tübingen

- 14:30 **Opening Remarks**
- 14:35 Andrew H. Bass, Ithaca, USA  
CENTRAL PATTERN GENERATOR FOR VOCA-  
LIZATION: IN SEARCH OF A VERTEBRATE  
MORPHOTYPE (S2-1)
- 15:00 Susanne Seltmann, Seewiesen  
THE INFLUENCE OF SLEEP ON SONG-  
RELATED NEURONAL ACTIVITY IN RA –  
WHAT ROLE DOES MELATONIN PLAY? (S2-2)
- 15:10 Richard Mooney, Durham, USA  
MOTOR-AUDITORY INTERACTIONS FOR  
LISTENING AND LEARNING (S2-3)
- 15:35 M. Jerome Beetz, Frankfurt  
ABOUT HOW CORTICAL NEURONS OF  
BATS COPE WITH FAST ECHOLOCATION  
SEQUENCES: MULTI-ELECTRODE AND  
SINGLE-ELECTRODE RECORDINGS WITH  
NATURAL ECHOLOCATION STIMULI (S2-4)
- 15:45 Steffen R. Hage, Tübingen  
AUDIO-VOCAL INTEGRATION AND COGNI-  
TIVE CONTROL OF VOCAL BEHAVIOUR IN  
MAMMALS (S2-5)
- 16:05 Constance Scharff, Berlin  
NEUROGENETIC CONTRIBUTIONS TO VOCAL  
PRODUCTION LEARNING (S2-6)



## Introductory Remarks to Symposium 3

### DBS-underlying mechanisms

*Anaïs Djodari-Irani and Christine Winter, Berlin and Dresden*

Deep brain stimulation (DBS) has shown to be an efficient treatment-option in therapy-resistant neurological disorders like Parkinson's disease or dystonia. DBS is a neurosurgical treatment involving the implantation of a brain pacemaker, sending electrical impulses to specific parts of the brain. As such, DBS allows focal intervention in disturbed neuronal networks in a modifiable and reversible way, therefore successfully replacing former ablative irreversible techniques. In the last years, increasing attention has also been drawn to DBS as an alternative treatment option for therapy-refractory psychiatric diseases like obsessive-compulsive disorder and depression. However, despite the fact that this technique is widely accepted by now in the neurological field and is even approved by the Food and Drug Administration (FDA) as a treatment for essential tremor, Parkinson's disease and also dystonia, its underlying principles are not fully understood. The application of DBS in the psychiatric field led to somewhat inconsistent results due to lack of consent as to which brain areas are the most promising as DBS targets, different stimulation intensities used and various observed beneficial effects. Altogether further investigation into the underlying mechanisms of DBS is of great necessity. Consequently, in this symposium we wish to address the question of how DBS works, using different strategies. We will present different approaches from (i) the examination of neurotransmissional network plasticity over (ii) in-vivo strategies exhausting diverse animal models for evaluation of DBS-effects employing different targets and stimulation criteria and finally we will discuss (iii) cellular approach examining the effects of DBS on the immune competent cells of the brain - the microglia.

**Symposium 3 - cancelled**

Wednesday, March 18, 2015  
14:30 – 16:30, Lecture Hall 102

Chair: Anaïs Djodari-Irani and Christine Winter,  
Berlin and Dresden

- 14:30    **Opening Remarks**
- 14:35    Sabrina Boulet, Grenoble, France  
MOTIVATIONAL DISORDERS IN PARKINSON'S  
DISEASE AND HIGH FREQUENCY STIMULA-  
TION OF THE SUBTHALAMIC NUCLEUS: PRE-  
CLINICAL STUDY IN THE RAT (S3-1)
- 15:00    Nicolas Singewald, Innsbruck, Austria  
DEEP BRAIN STIMULATION IN PSYCHOPATHO-  
LOGICAL MOUSE MODELS OF FEAR AND  
AFFECTIVE DISORDERS (S3-2)
- 15:25    Ravit Hadar, Dresden  
FROM RATS TO MEN: DEEP BRAIN STIMULA-  
TION IN RODENT MODELS OF PSYCHIATRIC  
AFFLICTIONS (S3-3)
- 15:50    Anaïs Djodari-Irani, Berlin  
MICROGLIA: THE MISSING LINK IN DBS'  
MECHANISM? (S3-4)
- 16:15    Águida Förster, Göttingen  
EFFECTS OF DIFFERENT TRANSCRANIAL  
DIRECT CURRENT STIMULATION (TDCS) PO-  
LARITY ON MOTOR LEARNING INDUCED OF  
MENTAL PRACTICE  
(S3-5)
- 16:25    **Concluding Remarks**



## Introductory Remarks to Symposium 4

# Timing and valence in associative learning

*Markus Fendt, Ayse Yarali and Bertram Gerber, Magdeburg*

Memories relating to a negative event are adaptive when supporting pre-emptive avoidance, escape or attack. However, such memories can also become overwhelmingly powerful. They may trigger excessively negative psychological states and uncontrollable, maladaptive behaviours. Clearly, any process to counteract such effects will be of value. Recent research emphasizes the notion that negative events are 'Janus-faced' in the sense that there are actually two aspects worth remembering about them: what made them happen, and what made them cease.

This symposium will present the latest research from fruit flies, rats and humans to show that both aspects, respectively related to the beginning and the end of a negative event, induce distinct and oppositely valenced memories: stimuli experienced before e.g. an electric shock acquire negative value as they warn of potential threat (this process is also called fear or punishment learning), whereas stimuli experienced after an electric shock acquire positive value because they promise relief.

During this symposium we will discuss what is known, and what should be asked, about the mechanisms of such fear and relief learning. One focus will be how both these forms of learning relate to reward as well as to safety learning. We selected speakers such that perspectives also will be offered as to how this mnemonic organization relates to applied psychology. This is timely, because despite the rich literature on fear learning, little is known about the neurobiological mechanisms or the psychological corollaries of relief learning. Such knowledge would be important, however, in particular from an applied perspective: the more distinct the underlying opponent processes of fear- and relief learning are, the more likely they contribute independently to pathology, and the easier it will be to selectively interfere with either of them.

## Symposium 4

Wednesday, March 18, 2015  
14:30 – 16:30, Lecture Hall 9

Chair: Markus Fendt, Ayse Yarali and Bertram Gerber,  
Magdeburg

14:30 **Opening Remarks**

14:35 Ayse Yarali, Magdeburg  
RELIEF LEARNING IN FRUIT FLIES (S4-1)

14:55 Markus Fendt, Magdeburg  
RELIEF AND SAFETY LEARNING IN RATS:  
BEHAVIORAL CHARACTERIZATION AND  
NEURAL BASIS (S4-2)

15:15 Anushka Fernando, Oxford, UK  
SAFETY SIGNALS INHIBIT FEAR BUT ARE THEY  
REINFORCING? (S4-3)

15:35 **Coffee Break**

15:45 Siri Leknes, Oslo, Norway  
RELIEF AND REWARD IN THE HUMAN BRAIN  
(S4-4)

16:05 Marta Andreatta, Würzburg  
PAIN RELIEF LEARNING IN HUMANS (S4-5)

16:25 **Concluding Remarks**





## Introductory Remarks to Symposium 5

# When the effect determines the cause - sensory consequences of self-action and their relevance for planning, control, and perceptual interpretation of one's behaviour

*Alexander Gail and Axel Lindner, Göttingen and Tübingen*

In this symposium we highlight the idea that the expected sensory effects of action play a profound role in processes related both (i) to the perceptual distinction of self-produced from external sensory events, and (ii) to the cause of these effects – namely action planning and control.

How is an organism able to distinguish sensory events that are self-produced from events that arise from the environment? It has been suggested that the brain uses corollary discharge of motor commands to predict the sensory consequences of motor acts. By subtracting such sensory predictions – also referred to as forward models - from the actual sensory afference, sensory information that is self-produced could be attenuated. This is needed, for instance, to guarantee the visual percept of a stable environment despite eye movements.

Psychophysical evidence clearly supports the concept of corollary discharge in explaining perceptual stability. Yet, only quite recently we started to understand the neural underpinnings of corollary discharge pathways in primates. In this symposium Robert Wurtz will address how the perception of space is modified by interrupting corollary discharge in monkeys.

But sensory predictions of action consequences are not just relevant for perceptual stability. They also mark an essential concept in adaptive motor control. Reza Shadmehr will show how the cerebellum represents predictive forward models, underlying the high accuracy of eye movement execution.

Finally we will address how the motor system solves the inverse problem, namely finding the “correct” motor cause for a desired sensory effect. Computational theories suggest that so-called inverse models serve this ability. Richard Hahnloser will provide us important computational and empirical insights into inverse models for motor control in songbirds.

The speakers in our symposium represent different disciplines and take complimentary perspectives on sensorimotor behaviour. Thereby, their converging evidence underlines the importance of treating perception and action as an integrated, mutually dependent closed-loop system.



## Symposium 5

*Wednesday, March 18, 2015  
14:30 – 16:30, Lecture Hall 104*

Chair: Alexander Gail and Axel Lindner,  
Göttingen and Tübingen

- 14:30    **Opening Remarks**
- 14:35    Robert Wurtz, Bethesda, USA  
VISUAL PERCEPTION DEPENDS ON AN  
EFFERENCE COPY OF SACCADDES (S5-1)
- 15:00    Reza Shadmehr, Baltimore, USA  
CEREBELLAR CONTRIBUTIONS TO LEARNING  
SENSORY CONSEQUENCES OF ACTION (S5-2)
- 15:25    Richard Hahnloser, Zurich, Switzerland  
INVERSE MODELS FOR MOTOR CONTROL -  
A SONGBIRD PERSPECTIVE (S5-3)
- 15:50    Manuel J. Roth, Tübingen  
A DOMAIN-GENERAL ROLE OF THE CEREBEL-  
LUM IN FINE-TUNING SENSORY PREDICTIONS  
(S5-4)
- 16:05    Matthias Nau, Tübingen  
AREA V3A ENCODES OBJECTIVE MOTION  
VELOCITY REGARDLESS OF EYE MOVEMENT  
VELOCITY (S5-5)
- 16:20    **Concluding Remarks**



## Introductory Remarks to Symposium 6

# Neural mechanisms underlying spatial orientation in insects

*Uwe Homberg and Keram Pfeiffer, Marburg*

Orientation in space includes sophisticated behaviors such as long-range migration and vector navigation, as well as simpler behaviors like maintaining a straight heading. Research on different insect species increasingly points to the central complex (CX) in the insect brain as the neural substrate mediating various aspects of spatial orientation. The symposium presents recent advances in understanding this neuropil's role in spatial orientation. Roy Ritzmann studies the neural control of turning and climbing behavior in cockroaches. Multi-unit recordings show that activity in units of the CX is closely correlated with walking speed and suggest a key role of the CX in locomotor decisions. Roland Strauss investigates short- and long-term memories in visual orientation of fruit flies. Wild type flies decide on a target and maintain walking direction even when a second target is presented in alternation. Specific mutants of the CX lack this memory and perform zigzag movements toward the two alternating targets. In addition, a specific part of the fly CX is essential to learn and use information about the fly's own body size for decisions on gap crossing. Keram Pfeiffer and Basil el Jundi analyze neural mechanisms in sky compass orientation. The sky polarization pattern and colour gradient are used by insects as orientation cues. Keram Pfeiffer reports on the integration of chromatic and polarization cues in the CX of bumblebees. Basil el Jundi shows that nocturnal dung-beetles roll dung balls away from a dung pat in a straight compass course determined by the low light levels of polarized moon light. Intracellular recordings suggest that polarization-sensitive neurons of the CX play a major role in this behaviour. Two student presentations address issues in fly optomotor behavior (Franziska Toepfer) and processing of sky compass signals in locusts (Tobias Bockhorst). Taken together, the comparative approaches illustrate that the CX serves a variety of spatial orientation tasks, including sky compass orientation and visual landmark memory.

## Symposium 6

Wednesday, March 18, 2015  
14:30 – 16:30, Lecture Hall 8

Chair: Uwe Homberg and Keram Pfeiffer,  
Marburg

- 14:30 Roy E. Ritzmann, Cleveland, USA  
CENTRAL COMPLEX ACTIVITY ASSOCIATED  
WITH CONTEXT AND STATE DEPENDENT  
SPATIAL ORIENTATION (S6-1)
- 14:55 Roland Strauss, Mainz  
SHORT-LIVED AND LONG-TERM MEMORIES  
IMPROVE SPATIAL ORIENTATION IN *DROSOPHILA*  
(S6-2)
- 15:20 Franziska Toepfer, Würzburg  
PERCEPTUAL HYPOTHESES IN *DROSOPHILA*  
VISION (S6-3)
- 15:30 Keram Pfeiffer, Marburg  
PROCESSING OF CHROMATIC AND POLA-  
RIZED LIGHT STIMULI IN THE CENTRAL BRAIN  
OF THE BUMBLEBEE (S6-4)
- 15:55 Basil el Jundi, Lund, Sweden  
NEURAL CODING OF THE HIERARCHY OF  
CELESTIAL COMPASS CUES IN AN INSECT  
BRAIN (S6-5)
- 16:20 Tobias Bockhorst, Marburg  
CONTEXT-DEPENDENT SIGNALING OF SKY-  
COMPASS CUES IN AN INSECT BRAIN  
(S6-6)



### *Introductory Remarks to Symposium 7*

## **Contribution of astrocyte connexins to neuroglial interaction in the healthy and diseased brain**

*Christian Steinhäuser, Bonn*

The last decade has seen the emergence of a new concept of brain signaling that has challenged the prevailing “neurocentric” view. Indeed, increasing evidence has established that synaptic plasticity, neuronal activity and survival should also be considered as resulting from an active interplay of neurons with glial cells. Such dynamic and metabolic neuroglial interactions have been identified in healthy as well as in diseased situations suggesting a role of glia in normal brain functions and pathologies. Interestingly, compared to neurons, a characteristic feature of glial cells, in particular astrocytes, is their high expression level of connexins. Once at the membrane, these proteins support two channel functions: formation of gap junction intercellular channels and hemichannels allowing, respectively, direct communication between the cytoplasm of adjacent cells and exchanges between the intra- and extracellular medium. The participants to this symposium will cover several aspects of neuro-glial interactions in which astroglial connexins are involved either in a physiological function or in a pathological context. This will include: i) the analysis of connexin 30-mediated gap junctional communication in thalamic panglial networks between astrocytes and oligodendrocytes which will be presented by a young PhD student, Stephanie Griemsmann (Bonn, Germany); ii) the role of connexin 43 in the regulation of sleep homeostasis presented by Phil Haydon (Boston, USA), an internationally recognized expert in the field who has initially proposed the concept of the “tripartite synapse” associating an astrocyte to the pre- and postsynaptic elements; iii) the interaction between connexin 43 and c-Src activation, proliferation and glucose uptake in astroglioma presented by a young investigator, Arantxa Taberner (Salamanca, Spain) and iv) the contribution of connexin 43 hemichannel activity in reactive astrocytes to neuronal dysfunctions studied in a murine model of Alzheimer’s disease, the APP/PS1 mouse, which will be presented by a postdoctoral investigator Chenju Yi (Paris, France). This program should be of interest to a large audience of neuroscientists: i) by offering a broad view of several aspects of neuro-glial interactions in normal brain function and diseases, but also ii) by putting the light on an important characteristic of astrocytes, which concerns their high content of connexins, providing the basis of intercellular networks as well as of a pathway for the release of active molecules.

## Symposium 7

Thursday, March 19, 2015  
11:30 – 13:30, Lecture Hall 10

Chair: Christian Steinhäuser, Bonn

11:30 **Opening Remarks**

11:40 Stephanie Griemsmann, Bonn  
CHARACTERIZATION OF PANGLIAL GAP  
JUNCTION NETWORKS IN THE MURINE  
BRAIN (S7-1)

12:00 Phil G. Haydon, Boston, USA  
DELETION OF ASTROCYTIC CONNEXIN 43  
CAUSES A NARCOLEPSY-LIKE PHENOTYPE  
(S7-2)

12:20 Arantxa Tabernero, Salamanca, Spain  
RELEVANCE OF THE INTERACTION BET-  
WEEN CONNEXIN43 AND C-SRC IN ASTRO-  
CYTOMA CELLS (S7-3)

12:40 Chenju Yi, Paris, France  
CHRONIC HEMICHANNEL ACTIVATION IN  
ASTROCYTES CONTRIBUTES TO NEURONAL  
SUFFERING IN A MURINE MODEL OF ALZ-  
HEIMER'S DISEASE (S7-4)

13:00 Anne C. Wolfes, Göttingen  
CALCIUM SIGNALLING AND VESICLE-RELATED  
PROTEINS IN DIFFERENT ASTROCYTE CULTURE  
TYPES (S7-5)

13:10 Alenka Gucek, Ljubljana, Slovenia  
FUSION PROPERTIES OF GLIOTRANSMITTER  
VESICLES IN CULTURED ASTROCYTES (S7-6)

13:20 **Concluding Remarks**



## Introductory Remarks to Symposium 8

# The ontogeny of entorhinal circuitry and function

*Ileana Hanganu-Opatz and Dietmar Schmitz, Hamburg and Berlin*

The ability to internally represent the external space and to accordingly guide the navigation-based behavior mainly relies on the specific activation of several areas in the medial temporal lobe. The identification of place cells in the hippocampus as well as of grid, head-direction and border cells in the entorhinal cortex led the research focus on these two brain areas and their functional interplay. While the neuronal mechanisms underlying the spatial memory in adult have been largely investigated, only recently their ontogeny in relationship to the maturation of entorhinal-hippocampal networks has been experimentally addressed. The proposed symposium aims at providing a comprehensive overview of recent key findings on the emergence of information processing and encoding in the entorhinal networks. The lecture by Menno Witter will set the anatomical framework of early hippocampal-entorhinal communication and introduce the patterning mechanisms of parahippocampal and hippocampal projections during neonatal and juvenile development of rodents. This emerging connectivity represents the substrate of functional coupling within neuronal networks, including the hippocampus, entorhinal cortex and prefrontal cortex. The ability of oscillatory patterns of activity to mediate the directed interactions between these areas and set the initial wiring of neuronal networks involved in mnemonic abilities will be highlighted in the talk by Ileana Hanganu-Opatz. The last two talks will focus on the establishment of circuitry responsible for the neural representation of space. The lecture by Rhiannon Meredith aims at identifying early topographic modules in the entorhinal cortex, the coupling by synchrony of which will lead to grid cell entrainment at juvenile age. The talk by Francesca Cacucci will highlight that the maturation of spatial representation involves not only the entorhinal networks, but results from a tight interplay within entorhinal-hippocampal networks in response to environmental boundaries. These recent insights from neuroanatomy, electrophysiology, imaging, and behavior represent a synopsis of current understanding of how mnemonic ontogeny is encoded into developing neural circuits.

The symposium is supported by the DFG Priority Program SPP 1665 "Resolving and manipulating neuronal networks in the mammalian brain".

## Symposium 8

Thursday, March 19, 2015  
11:30 – 13:30, Lecture Hall 103

Chair: Ileana Hanganu-Opatz and Dietmar Schmitz,  
Hamburg and Berlin

- 11:30    **Opening Remarks**
- 11:40    Menno Peter Witter, Trondheim, Norway  
POSTNATAL DEVELOPMENT OF PARAHIPPO-  
CAMPAL-HIPPOCAMPAL CONNECTIVITY  
(S8-1)
- 12:00    Ileana Hanganu-Opatz, Hamburg  
SEARCHING FOR FEEDBACK: CONTRIBUTION  
OF THE ENTORHINAL PROCESSING TO THE  
DEVELOPMENT OF PREFRONTAL-HIPPOCAM-  
PAL COMMUNICATION (S8-2)
- 12:20    Rhiannon Meredith, Amsterdam, The Netherlands  
PACEMAKERS AND WAVES: DEVELOPING  
PRE-GRID CELL NETWORKS OF RODENT  
MEDIAL ENTORHINAL CORTEX (S8-3)
- 12:40    Francesca Cacucci, London, UK  
THE DEVELOPMENT OF THE NEURAL MAP OF  
SPACE IN THE HIPPOCAMPAL FORMATION  
(S8-4)
- 13:00    Franziska Bender, Berlin  
OPTOGENETIC CONTROL OF HIPPOCAMPAL  
THETA OSCILLATIONS REVEALS THEIR FUNC-  
TION IN LOCOMOTION VIA HIPPOCAMPUS  
TO LATERAL SEPTUM PATHWAY (S8-5)
- 13:10    Anne-Kathrin Theis, Berlin  
BACKPROPAGATING ACTION POTENTIALS  
MEDIATE PLASTICITY OF SPINE CALCIUM  
DYNAMICS IN THE MEDIAL ENTORHINAL  
CORTEX (S8-6)
- 13:20    **Concluding Remarks**





## Introductory Remarks to Symposium 9

### Processing of acoustic pulse patterns: Common themes in different brains?

*Berthold Hedwig and Stefan Schöneich, Cambridge (UK)*

The processing of sound patterns is essential for acoustically communicating species throughout the animal kingdom and therefore a specific function of many nervous systems. One fundamental aspect is how the temporal pattern of amplitude modulated signals is transformed into a neural representation of signal periodicities. Recent progress in analysing the underlying neuronal processing may point to some common principles in terms of cellular properties and neural algorithms. The symposium aims at a comparative approach that draws its examples from vertebrate, invertebrate and computational models. It will elucidate the neuronal underpinning of transformation of amplitude modulated sound patterns into a signal representation by neural activity. The central question we like to pose is: To what degree have different types of brains come up with similar solutions for the same problem?

Our intention is to bring together scientists who work on the processing of temporal sound patterns in different acoustically communicating animals (e.g. mouse, frog and cricket) and with a theoretical approach. The recent progress in analysing the neuronal mechanisms underlying temporal auditory processing may point to some common principles in terms of cellular properties and neural algorithms. These principles for the detection and recognition of amplitude modulated sound patterns have a strong behavioural relevance for acoustic communication and also for processing the temporal aspects of speech and music.

## Symposium 9

*Thursday, March 19, 2015  
11:30 – 13:30, Lecture Hall 8*

Chair: Berthold Hedwig and Stefan Schöneich,  
Cambridge (UK)

- 11:30    **Opening Remarks**
- 11:40    Stefan Schöneich, Cambridge, UK  
NEURAL NETWORK AND MECHANISM FOR  
SOUND PATTERN RECOGNITION IN THE  
CRICKET BRAIN (S9-1)
- 12:00    Jakob Christensen-Daalsgard, Odense, Denmark  
TEMPORALLY SELECTIVE PROCESSING OF  
COMMUNICATION SIGNALS BY FROG  
AUDITORY MIDBRAIN NEURONS (S9-2)
- 12:20    Anna K. Magnusson, Stockholm, Sweden  
KEEPING TIME: PROCESSING OF AUDITORY  
COMMUNICATION CUES IN THE BRAINSTEM  
(S9-3)
- 12:40    Dominik F. Aschauer, Mainz  
CHRONIC CALCIUM IMAGING OF NEURONAL  
ENSEMBLES IN THE MOUSE AUDITORY CORTEX  
(S9-4)
- 13:00    Leo van Hemmen, Munich  
HOW TO MEASURE SIGNAL PERIODICITY,  
IF YOU MUST? (S9-5)



## Introductory Remarks to Symposium 10

# Microcephaly and developmental defects of the brain

*Angela M. Kaindl, Berlin*

Microcephaly, the clinical sign of reduced brain size, can be caused by congenital neurodevelopmental disorders. It has a high prevalence of about 2% in the general population and is frequently associated with intellectual disability. In corresponding pedigrees, a multitude of mutant genes has been identified in the past decades, but further genetic heterogeneity exists, and mechanisms by which these regulate cognitive function and brain size remain to be elucidated. For congenital microcephaly, various disorders of stem cell survival, proliferation and specification as well as defects in the maintenance of postmitotic cells have been reported. In this symposium, we discuss pathomechanisms underlying developmental defects of the brain that lead to microcephaly and intellectual disability. Specifically, we discuss 1) novel hereditary microcephalies, 2) defects in neuronal specification of stem cells in cortical development that lead to microcephaly, 3) evolutionary concepts and stem cells in microcephaly, and 4) models of brain development and microcephaly. The subject has a broad appeal for all aspects of basic research and also a high clinical relevance.

## Symposium 10

*Thursday, March 19, 2015*  
*11:30 – 13:30, Lecture Hall 104*

Chair: Angela M. Kaindl, Berlin

11:30    **Opening Remarks**

11:40    Wieland B. Huttner, Dresden  
NEURAL STEM AND PROGENITOR CELLS AND  
NEOCORTEX EXPANSION IN DEVELOPMENT  
AND EVOLUTION (S10-1)

12:05    Pierre Vanderhaeghen, Brussels, Belgium  
MECHANISMS OF MICROCEPHALY, AND THEIR  
LINKS TO DEVELOPMENT AND EVOLUTION  
OF THE HUMAN BRAIN  
(S10-2)

12:30    Magdalena Renner, Jürgen A. Knoblich, Vienna,  
Austria  
MODELING HUMAN BRAIN DEVELOPMENT  
AND DISEASE IN 3D CULTURE (S10-3)

12:55    Angela M. Kaindl, Berlin  
MICROCEPHALY – FROM BEDSIDE TO BENCH  
(S10-4)

13:20    **Concluding Remarks**



## Introductory Remarks to Symposium 11

# Ultramicroscopy for imaging the central nervous system and its pathological alterations

*Edgar R. Kramer, Hamburg*

Being able to visualize neuronal connections is the basis to understand the physiological function of complex cellular networks such as the central nervous system. And imaging pathological alterations in the brain and spinal cord enables investigating the etiology of their neurological malformations during development and aging.

Ultramicroscopy is an excellent tool to accomplish these two tasks by using light sheet illumination to image fluorescently labeled single cells, cell populations, neuronal networks but also protein aggregates and cell lesions in complete brains or spinal cords in subcellular resolution without the need for the cumbersome classical histology. The critical steps are first the fluorescently labeling of the structure of interest in the nervous system, second the clearing procedure to transform the brain or spinal cord into a glass body without bleaching the fluorescent signal and third the high resolution imaging and data processing to obtain a representative 3D animation. Although this novel technique has revealed already exciting insides into the nervous system development and maintenance and ultramicroscopic setups are now commercially available, it is still pioneering work to develop this technique further and elucidate its full potential.

In this symposium we will present on one hand new technical developments that improve and widen the use of ultramicroscopy to study the central nervous system of rodents and on the other hand new applications of these method to address central questions in the neuroscience field such as the monosynaptic connection map to the entorhinal cortex, the absolute quantification of dopaminergic neurons effected in Parkinson's disease patients, the distribution of Alzheimer plaques in the brain, and the stimulation of axonal regeneration in the spinal cord.

This symposium is supported by LaVision BioTec GmbH in Bielefeld ([www.lavisionbiotec.com](http://www.lavisionbiotec.com)).

## Symposium 11

Thursday, March 19, 2015  
11:30 - 13:30, Lecture Hall 9

Chair: Edgar R. Kramer, Hamburg

- 11:30 **Opening Remarks**
- 11:35 Nina Jährling, Vienna, Austria  
TECHNICAL ADVANCES IN ULTRAMICROSCOPY AND THEIR APPLICATION FOR INVESTIGATING NEURONAL DEVELOPMENT AND DISEASE (S11-1)
- 11:55 Günter Giese, Heidelberg  
VISUALIZING NEURONAL STRUCTURES IN TRANSLUCENT ADULT MOUSE BRAIN WITH LIGHT SHEET FLUORESCENCE MICROSCOPY (S11-2)
- 12:20 Andrea Tedeschi, Bonn  
RNA-SEQ SCREEN IDENTIFIES CRITICAL REGULATORS OF AXON GROWTH AND REGENERATION (S11-3)
- 12:45 Ulrich Leischner, Jena  
IMAGING OF WHOLE-MOUNT SAMPLES WITH  $\mu\text{M}$  RESOLUTION USING LIGHT-WEDGE-MICROSCOPY (S11-4)
- 13:05 Edgar R. Kramer, Hamburg  
IMAGING AND QUANTIFICATION OF DOPAMINERGIC NEURONS OF THE MOUSE USING ULTRAMICROSCOPY (S11-5)
- 13:25 **Concluding Remarks**



## Introductory Remarks to Symposium 12

### Breaking News I

*Carmen Smarandache-Wellmann, Cologne*

Registered students had the choice to either register with a poster presentation or apply for an oral communication. The program committee has selected the young investigator presentations from these submissions and assigned them either to a symposium or to one of the two Breaking News symposia (symposia 12 and 23).

The following students were selected to give a short communication in Symposium 12 – Breaking News I:

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- 14:30    **Opening Remarks**
- 14:35    Anil Annamneedi, Magdeburg  
CONDITIONAL MUTANTS OF BASSOON IN EXCITATORY FOREBRAIN SYNAPSES AND DOPAMINERGIC SYNAPSES, TO STUDY THEIR CONTRIBUTION IN LEARNING AND MEMORY PROCESSES (S12-1)
- 14:45    Sophie Batsching, Würzburg  
LEARNED HELPLESSNESS IN *DROSOPHILA MELANOGASTER*- DOES IT TRANSFER TO OTHER BEHAVIOR? (S12-2)
- 14:55    Lisa K. J. Clausen, Oxford, UK  
EXPLORING THE EFFECTS OF  $\beta_2$ -ADRENERGIC RECEPTOR AGONISTS IN DOK7 CONGENITAL MYASTHENIC SYNDROME (S12-3)
- 15:05    Bettina Hein, Frankfurt/Main  
CHRONIC STUDY OF SPONTANEOUS ACTIVITY AND ORIENTATION SELECTIVITY IN VISUAL CORTEX AROUND EYE OPENING (S12-4)

## Symposium 12

Thursday, March 19, 2015  
14:30 – 16:30, Lecture Hall 105

Chair: Carmen Smarandache-Wellmann, Cologne

- 15:15 Jan-Hendrik Heyne, Magdeburg  
OPPOSING EFFECTS OF CAMP-EFFECTORS  
PKA AND EPAC ON ACTIVITY-DEPENDENT  
BDNF SECRETION IN DISSOCIATED HIPPO-  
CAMPAL NEURONS (S12-5)
- 15:25 **Break**
- 15:35 Johannes Mayer, Rostock  
INFLUENCE OF MTDNA SINGLE NUCLEOTIDE  
POLYMORPHISMS ON AGE DEPENDENT  
CHANGES OF MEMORY FUNCTION (S12-6)
- 15:45 Julia Michely, Saarbrücken  
THE IMPACT OF MICRORNAs IN MEMORY  
FORMATION PROCESSES IN THE HONEYBEE  
(*APIS MELLIFERA*) (S12-7)
- 15:55 Esther Nibbeling, Groningen, The Netherlands  
IDENTIFICATION OF NOVEL SPINOCEREBELLAR  
ATAXIA DISEASE GENES USING NEXT GENE-  
RATION SEQUENCING APPROACHES (S12-8)
- 16:05 Steffen Platschek, Frankfurt/Main  
COMPUTATIONAL MODELING OF LESION  
INDUCED DENDRITIC REORGANIZATION  
(S12-9)
- 16:15 Kerstin Wernecke, Magdeburg  
THE OLFACTORY HOLE-BOARD TEST: A NEW  
PARADIGM TO STUDY BEHAVIOR TO BIOLO-  
GICALLY-RELEVANT ODORS (S12-10)
- 16:25 **Concluding Remarks**





## Introductory Remarks to Symposium 13

# Functional consequences of sensory loss and restoration

*Stephen Lomber, London, Canada*

Plasticity is the neural mechanism by which complex nervous systems adjust themselves to their environment. Adaptive, or compensatory plasticity is a part of this overall process resulting from the loss of a class (or modality) of sensory inputs that is accompanied by a corresponding expansion of the remaining systems. Not only does this process provide some substitute for the lost modality, but the additional circuitry also conveys enhanced abilities to the remaining systems. Developmental studies of the deaf and blind, as well as recent studies in mature subjects, demonstrate remarkable multisensory plasticity throughout the cerebrum. As hearing can be restored through cochlear implants, and more recently sight can be restored with retinal prosthetics, sensory deprivation represents an opportunity to study the capacity of cortical plasticity within and between modalities. The symposium brings together information from both human and animal studies examining functional compensations following deafness and blindness, and the changes that occur following the initiation of hearing and sight. Speakers will describe psychophysical, imaging, electrophysiological, and anatomical studies performed to reveal the functional consequences and underlying mechanisms of crossmodal plasticity.

## Symposium 13

Thursday, March 19, 2015  
14:30 – 16:30, Lecture Hall 104

Chair: Stephen Lomber, London (Canada)

- 14:30    **Opening Remarks**
- 14:35    Stephen Lomber, London, Canada  
DEAF AUDITORY CORTEX MEDIATES EN-  
HANCED FACE PERCEPTION IN THE CON-  
GENITALLY DEAF (S13-1)
- 15:00    Andrej Kral, Hannover  
PLASTICITY WITH SINGLE-SIDED DEAFNESS:  
REPRESENTATIONAL MAPS AND BINAURAL  
INTERACTIONS (S13-2)
- 15:25    Brigitte Roeder, Hamburg  
SENSITIVE PHASES FOR THE DEVELOPMENT  
OF MULTISENSORY PROCESSES (S13-3)
- 15:50    Amir Amedi, Jerusalem, Israel  
THE NEURAL CORRELATES OF HEARING CO-  
LORS AND SHAPES: INSIGHTS FROM DAR-  
KNES ON BRAIN PLASTICITY AND STABILITY  
(S13-4)
- 16:15    Blake Edward Butler, London, Canada  
CORTICAL PLASTICITY FOLLOWING SENSORY  
DEPRIVATION: CHARACTERIZING THE PAT-  
TERNS OF THALAMOCORTICAL AND CORTI-  
COCORTICAL PROJECTIONS IN EARLY- AND  
LATE-DEAF CATS (S13-5)
- 16:25    **Concluding Remarks**



## Introductory Remarks to Symposium 14

# Recent advances in basal ganglia research: action selection, movement and pathologies

*Robert Schmidt and Arvind Kumar, Freiburg and Freiburg/Stockholm (Sweden)*

Recent discoveries have changed our view on how movements are selected and executed through basal ganglia pathways. In particular, classic concepts of processing in the direct and indirect pathways have been challenged and modified. For instance, the massive backprojection of so-called arky pallidal neurons from the globus pallidus to the striatum calls for an update in the simplified feed-forward description of the basal ganglia. Specifically, the arky pallidal neurons might inhibit a striatal "Go" signal during the successful suppression of movements (Schmidt). Therefore, it is crucial to understand the integration of multi-sensory excitatory and inhibitory inputs in the striatum to guide action selection (Silberberg). On a systems level the striatum can function as a decision threshold to facilitate action selection (Bahuguna). Besides these functional roles of basal ganglia circuits in normal behavior, the classic description of direct and indirect pathways was also instrumental to understand behavioral deficits in neurological disorders like Parkinson's disease. However, the limitations of this simplified concept have also become apparent. An extended approach includes the oscillatory dynamics of the basal ganglia. Neural oscillations in basal ganglia circuits have been strongly associated with Parkinson's disease and advances in understanding the pathological activity are complemented by pioneering advances in treatment (Brown). Finally, de la Crompe presents how the selective optogenetic manipulation of different basal ganglia subregions affects neural oscillations related to Parkinson's disease. The goal of this symposium is to bring together clinical, experimental and theoretical researchers interested in basal ganglia function to discuss these recent developments in the field.

## Symposium 14

*Thursday, March 19, 2015*  
*14:30 – 16:30, Lecture Hall 8*

Chair: Robert Schmidt and Arvind Kumar, Freiburg and  
Freiburg/Stockholm (Sweden)

14:30    **Opening Remarks**

14:35    Robert Schmidt, Freiburg  
DYNAMICS OF BASAL GANGLIA CIRCUITS  
DURING MOVEMENT INITIATION AND  
SUPPRESSION (S14-1)

15:00    Gilad Silberberg, Stockholm, Sweden  
MICROCIRCUITS UNDERLYING MULTISEN-  
SORY INTEGRATION IN THE MOUSE STRIATUM  
(S14-2)

15:25    Jyotika Bahuguna, Freiburg  
EXISTENCE AND CONTROL OF GO/NO-GO  
DECISION TRANSITION THRESHOLD IN THE  
STRIATUM (S14-3)

15:40    Peter Brown, Oxford, UK  
PATHOLOGICAL NEURONAL SYNCHRONI-  
SATION IN PARKINSON'S DISEASE AND ITS  
CONSEQUENCES (S14-4)

16:05    Brice de la Crompe, Bordeaux, France  
OPTOGENETIC MAPPING OF NETWORK  
DYNAMIC IN BASAL GANGLIA (S14-5)

16:20    **Concluding Remarks**



## Introductory Remarks to Symposium 15

# Is insect odor transduction primarily based upon an ORCO-dependent ionotropic mechanism or on metabotropic cascades?

*Monika Stengl, Kassel*

Insect odor transduction is still under lively debate. Insect olfactory receptors (ORs) are 7TM receptors which adopt an inverse membrane topology. A conserved coreceptor (ORCO) is a chaperon which locates ORs to dendritic membranes of sensory neurons. In addition ORCO is suggested to form a heteromeric ligand-gated ion channel together with ORs. Contradicting results suggest either solely ionotropic, or solely metabotropic, or both ionotropic and metabotropic mechanisms for insect odor transduction. In this symposium the four main talks report the involvement of different metabotropic transduction cascades in insect odor transduction even in the same insect species. We will focus mainly on odor transduction in *Drosophila* and on the crosstalk between different signal transduction cascades. We will discuss the apparently contradicting results and different hypotheses to come to a conclusion how odor transduction in insects is solved by evolution.

## Symposium 15

Thursday, March 19, 2015  
14:30 – 16:30, Lecture Hall 10

Chair: Monika Stengl, Kassel

- 14:30 **Opening Remarks**
- 14:35 Robin Schumann, Kassel  
THE CONTRIBUTION OF METABOTROPIC SIGNAL TRANSDUCTION CASCADES IN INSECT OLFACTION (S15-1)
- 14:55 Eva Neuhaus, Jena  
THE STIMULATORY  $G\alpha S$  PROTEIN IS INVOLVED IN OLFACTORY SIGNAL TRANSDUCTION IN *DROSOPHILA* (S15-2)
- 15:15 Giovanni Galizia, Konstanz  
OLFACTORY TRANSDUCTION IN *DROSOPHILA MELANOGASTER* - THE CONTRIBUTION OF SOME G PROTEINS (S15-3)
- 15:35 Dieter Wicher, Jena  
FUNCTION AND REGULATION OF INSECT ODORANT RECEPTORS (S15-4)
- 15:55 Alpha Renner, Hilzingen  
RAPIDLY RESPONDING OLFACTORY RECEPTOR NEURONS IN *DROSOPHILA MELANOGASTER* (S15-5)
- 15:15 **Outlook**



## Introductory Remarks to Symposium 16

# Molecular, neuronal and behavioural effects of oxytocin: a translational approach

*Inga D. Neumann and Valery Grinevich, Regensburg and Heidelberg*

The neuropeptide oxytocin (OXT) - often popularly dubbed the "love hormone" - currently attracts scientific attention due to its profound pro-social, anxiolytic and anti-stress effects. Although an abundant variety of human studies describes its use after intranasal application, underlying neurobiological mechanisms of these behavioural and physiological effects are less well understood. The symposium will bring together experts in the fields of neuroanatomy (V. Grinevich, Heidelberg), electrophysiology (R. Stoop, Lausanne), and behavioural/molecular neuroendocrinology (I. Neumann, Regensburg) with a human researcher (J. Bartz, Montreal) to discuss important aspects of OXT actions, their variability and their translational value.

V. Grinevich will focus on the morphological transformations of the OXT system during vertebrate evolution, in particular the co-development of OXT immunoreactive projections in the brain and the establishment of social and emotional behaviours. Using virus-based vectors he explores anatomical features of OXT neurons in rodents, including their projections to forebrain and limbic regions. Using optogenetics in combination with *in vitro* electrophysiology, he shows neuronal responsiveness and fear-reducing effects after optically evoked axonal OXT release. R. Stoop combines fluorescent retrograde tracing of projections from the central amygdala (CeA) to areas modulating behavioural and autonomic fear responses with *in vitro* electrophysiology. He identified CeA projections from separate neuronal populations with different electrophysiological characteristics and OXT responsiveness *in vitro* and *in vivo* in the context of fear conditioning. I. Neumann will focus on the brain OXT system and its involvement in non-social and social anxiety and fear in rodent models. Her group monitored the activation of subsequent intracellular signaling cascades in response to acute or chronic OXT and binding to its G-protein coupled receptors; activation of the MAP kinase and CREB pathways underlie the anxiolytic and anti-stress effects of OXT, respectively, both in a social and non-social context. J. Bartz will present evidence from human data showing that OXT effects are highly variable due to the individual, partly genetically determined, variability of the endogenous OXT system with the emergence of both pro- and anti-social effects. These human data are essential for the development of OXT as a potential treatment option in diseases such as social anxiety disorders, schizophrenia or autism.

## Symposium 16

Thursday, March 19, 2015  
14:30 – 16:30, Lecture Hall 9

Chair: Inga D. Neumann and Valery Grinevich,  
Regensburg and Heidelberg

- 14:30 Valery Grinevich, Heidelberg  
CENTRAL OXYTOCINERGIC PATHWAYS AND  
THEIR INVOLVEMENT IN SOCIALITY (S16-1)
- 14:55 Ron Stoop, Lausanne, Switzerland  
NEUROMODULATION BY OXYTOCIN IN  
THE CENTRAL AMYGDALA: AN IN VITRO AND  
IN VIVO OPTOGENETIC AND ELECTRO-  
PHYSIOLOGICAL DISSECTION OF THE UN-  
DERLYING CIRCUITRY (S16-2)
- 15:20 Inga Neumann, Regensburg  
INTRANEURONAL SIGNALING CASCADES  
MEDIATING OXYTOCIN ON ANXIETY AND  
STRESS REGULATION: EFFECTS OF CHRONIC  
TREATMENT (S16-3)
- 15:45 Jennifer A. Bartz, Montreal, Canada  
OXYTOCIN, ATTACHMENT AND THE SELF IN  
RELATION TO OTHER (S16-4)
- 16:10 Rohit Menon, Regensburg  
EPIGENETIC ADAPTATIONS OF OXYTOCIN  
SYSTEM DURING SOCIAL FEAR CONDITIO-  
NING (S16-5)
- 16:20 Ferdinand Althammer, Heidelberg  
FEAR ACTIVATED-GENETIC TARGETING OF  
OXYTOCIN NEURONS AND THEIR BEHA-  
VIORAL EFFECTS (S16-6)





## Introductory Remarks to Symposium 17

# Regeneration in the injured spinal cord - hopes and perspectives

*Antal Nógrádi, Szeged, Hungary*

Spinal cord injury leads to severe loss of grey and white matter and subsequent deficit of motor and sensory functions below the lesion. In the mammalian CNS very little regeneration occurs following spinal cord injury, therefore these deficits remain permanent and often fatal. On the other hand, anamniotes possess a remarkable regenerative and neurogenic capacity, which may carry very important knowledge for the strategies aiming at the repair of the injured mammalian cord. Apart from the immediate morphological damage and functional loss there are late-onset consequences, such as incontinence, spasticity and chronic pain which often are more difficult for the patients to tolerate than the sudden loss of motor function. As to our present knowledge there are very limited treatment strategies for spinal cord injured patients. However, at the preclinical level there are very promising approaches which suggest that the regeneration inhibiting effects within the mammalian spinal cord can be overcome and lost function can be at least partially restored.

The symposium speakers will focus on the complexity of the spinal cord injuries from various aspects. Thomas Becker (University of Edinburgh) will present their results on zebrafish that underwent spinal cord injury but showed extensive axonal regeneration and neurogenic capacity to generate motoneurons. Laurent Vinay (Institut de Neurosciences de la Timone, Marseille) is interested in the basic mechanisms of spasticity and chronic pain which develop several weeks or months following spinal cord injury. Recent results from his laboratory may lead to successful treatment of these terrible conditions. Urszula Slawinska (Nencki Institute, Warsaw) has been working for many years on the restoration of locomotor function below the level of a transection injury. Her laboratory, in close collaboration with Larry Jordan in Winnipeg has successfully transplanted embryonic 5-HT tissue into the injured cord or has applied 5-HT agonists to activate the hind limb central pattern generator. Our laboratory at University of Szeged has provided evidence that transplantation of a neuroectodermal stem cell line dramatically reduces micro- and astroglial upregulation in the injured spinal cord and thus prevents secondary damage and deposition/expression of axonal growth-inhibiting molecules around the injury. These favorable processes in the spinal cord microenvironment promote axonal regeneration and reorganization of the local circuitry.

## Symposium 17

Thursday, March 19, 2015  
14:30 – 16:30, Lecture Hall 105

Chair: Antal Nógrádi, Szeged (Hungary)

14:30 **Opening Remarks**

14:35 Thomas Becker, Edinburgh, UK  
NEURONAL REGENERATION IN THE SPINAL  
CORD OF ADULT ZEBRAFISH (S17-1)

15:00 Laurent Vinay, Marseille, France  
NEW PERSPECTIVES FOR THE TREATMENT OF  
SPASTICITY AND NEUROPATHIC PAIN AFTER  
SPINAL CORD INJURY (S17-2)

15:25 Urszula Slawinska, Warsaw, Poland  
SEROTONINERGIC CONTROL OF LOCOMO-  
TOR HINDLIMB MOVEMENTS – PROSPECTIVE  
STRATEGY FOR RESTORING LOCOMOTION  
AFTER SPINAL CORD INJURY (S17-3)

15:50 Antal Nógrádi, Szeged, Hungary  
REGENERATION IN THE INJURED RODENT  
CORD INDUCED BY GRAFTED STEM CELLS:  
MULTIPLE MECHANISM (S17-4)

16:15 Krisztián Pájér, Szeged, Hungary  
HUMAN iPS CELLS MEDIATE TISSUE SPARING  
WITH MODERATE FUNCTIONAL IMPROVE-  
MENT AFTER SPINAL CORD CONTUSION  
INJURY IN RATS (S17-5)

16:25 **Concluding Remarks**





## Introductory Remarks to Symposium 18

# Cellular adaptations for temporal precision in the auditory system

*Felix Felmy, Thomas Künzel and Ivan Milenkovic,  
Planegg-Martinsried, Aachen and Leipzig*

Sensory systems use temporal variations of stimuli to construct the perception of the world. The auditory system is ideally suited to study the impact of temporal variations, as multiple temporal cues are simultaneously processed on different time scales. In this symposium the role of different cellular adaptations in specific temporal processing tasks of the auditory system are discussed.

Auditory nerve fibers convey sensory information from the cochlea and form the endbulb of Held (EoH) synapse on bushy cells in the cochlear nucleus. These giant terminals relay information with high fidelity and temporal precision in the sub-millisecond time range. Matthew Xu-Friedman will focus on the activity-dependent regulation of synaptic depression and the synaptic mechanisms regulating the reliability of this synapse. The second talk by Ivan Milenkovic will address the role of inhibition for the information transfer at the EoH synapse. Again at the EoH, the student talk by David Goyer will present how cholinergic modulation affects the sub- and supra-threshold excitability of these neurons. Upstream of the EoH auditory information is processed in the superior olivary complex with temporal precision ranging from the sub- to the millisecond time range. Ian Forsythe will elaborate on the role of voltage gated ion channels in regulating the firing properties of neurons in the superior olivary complex. The discussion of this processing stage is complemented by the student talk by Alexander Fischer, illuminating how inhibition affects the temporal precision in the millisecond range during binaural processing in the lateral superior olive. Along the ascending auditory pathway the complexity of processing increases by adding information about the time-variable context ranging from milliseconds to tens of milliseconds. At this level, the mechanisms underlying the adjustment of processing time scales become particularly important. Ida Siveke will elucidate how different synaptic components account for context dependent processing in the dorsal nucleus of the lateral lemniscus, a structure implicated in the suppression of sound sources during reverberations.

## Symposium 18

Friday, March 20, 2015  
11:30 – 13:30, Lecture Hall 102

Chair: Felix Felmy, Thomas Künzel and Ivan Milenkovic,  
Planegg-Martinsried, Aachen and Leipzig

11:30    **Opening Remarks**

11:40    Matthew Xu-Friedman, Buffalo, USA  
ACTIVITY-DEPENDENT, HOMEOSTATIC REGU-  
LATION OF SYNAPTIC DEPRESSION AT THE  
ENDBULB OF HELD (S18-1)

12:00    Ivan Milenkovic, Leipzig  
DYNAMIC FIDELITY CONTROL TO THE  
CENTRAL AUDITORY SYSTEM: SYNERGISTIC  
GLYCINE/GABAERGIC INHIBITION IN THE  
COCHLEAR NUCLEUS (S18-2)

12:20    David Goyer, Aachen  
CHOLINERGIC SIGNALING INFLUENCES  
GERBILS SPHERICAL BUSHY CELLS EXCITABI-  
LITY IN VITRO (S18-3)

12:30    Ian Forsythe, Leicester, UK  
THE ROLE OF VOLTAGE GATED ION CHANNELS  
IN BRAINSTEM AUDITORY PROCESSING (S18-4)

12:50    Alexander U. Fischer, Kaiserslautern  
THE ROLE OF INHIBITION FOR TEMPORAL  
PRECISION IN THE LATERAL SUPERIOR OLIVE  
(S18-5)

13:00    Ida Siveke, Planegg-Martinsried  
CELLULAR MECHANISMS OF CONTEXT DE-  
PENDENT SIGNAL PROCESSING IN THE LATE-  
RAL LEMNISCUS (S18-6)

13:20    **Concluding Remarks**



## Introductory Remarks to Symposium 19

# Novel mechanisms influencing synaptic plasticity at GABAergic synapses

*Shiva Tyagarajan and Anne McKinney, Zurich (Switzerland) and Montreal (Canada)*

Homeostatic plasticity can be described as a mechanism through which neurons adjust the strength of their synapses in response to global or local changes in excitability. It is becoming increasingly clear that functional alteration at a given synapse is often accompanied by compensatory adaptation at other synapses at both local and network level. A major tenet of our proposal is that synaptic homeostasis depends on signaling cascades regulating the efficacy of GABAergic transmission. These signals converge onto postsynaptic protein scaffolds to regulate synaptic function by means of posttranslational modifications on specific target proteins, recruitment of specific GABAAR subunits, control of local protein degradation and actin remodeling via changes in local pH. This symposium comprising of two female and two male scientists will address four emerging concepts in the field of inhibitory neurotransmission.

1. Shiva Tyagarajan (University of Zurich, Switzerland) will present data showing how neuronal activity shapes mRNA splicing, protein degradation, in turn affecting GABAergic synapse structure.

2. Co-organizer Anne McKinney (McGill University, Canada) will present data showing molecular adaptations at GABAergic post-synapse in response to changes in BDNF signaling.

3. Patricia Seja (University of Helsinki, Finland) will provide evidence showing that carbonic anhydrase CA VII is effective in promoting HCO<sub>3</sub><sup>-</sup>-dependent excitatory GABAAR responses<sup>3</sup>. Mechanism(s) that allow CA VII to control actin remodeling, neuronal excitability, synaptic structure via pH regulation has shed new light into synaptic transmission and plasticity.

4. Derek Bowie (McGill University, Canada) has recently demonstrated that mitochondrial- reactive oxygen species (mROS) regulates the strength of postsynaptic GABA<sub>A</sub> receptors at inhibitory synapses. He will provide evidence to show that cellular metabolism can be coupled to synaptic plasticity changes.

## Symposium 19

Friday, March 20, 2015  
11:30 – 13:30, Lecture Hall 8

Chair: Shiva Tyagarajan and Anne McKinney,  
Zurich (Switzerland) and Montreal (Canada)

- 11:30    **Opening Remarks**
- 11:40    Derek Bowie, Montreal, Canada  
MITOCHONDRIAL REACTIVE OXYGEN SPECIES COUPLES CELLULAR METABOLISM TO NEURONAL COMMUNICATION (S19-1)
- 12:00    Anne McKinney, Montreal, Canada  
BDNF REGULATES SYNAPSE MAINTENANCE AFTER OXYGEN-GLUCOSE DEPRIVATION IN THE HIPPOCAMPUS (S19-2)
- 12:20    Shiva Tyagarajan, Zurich, Switzerland  
ADAPTATIONS AT GABAERGIC POSTSYNAPSES IS FACILITATED BY GEPHYRIN POSTTRANSLATIONAL MODIFICATIONS (S19-3)
- 12:40    Dr. Patricia Seja, Helsinki, Finland  
KCC2 AND CA7: NEURONAL ION-REGULATORY PROTEINS WITH A MORPHOGENIC FUNCTION (S19-4)
- 13:00    Marta Carus-Cadavieco, Berlin  
COORDINATION OF INNATE BEHAVIOURS BY GABAERGIC CELLS IN LATERAL HYPOTHALAMUS (S19-5)
- 13:10    Florian Walker, Göttingen  
INTEGRATION OF MARTINOTTI CELLS INTO DIS-/INHIBITORY CORTICAL CIRCUITS (S19-6)
- 13:20    **Concluding Remarks**



## Introductory Remarks to Symposium 20

# Actin cytoskeleton in neuronal morphogenesis and plasticity

*Britta Qualmann and Michael Kessels, Jena*

The astonishing morphological intricacy neurons acquire during their differentiation is a structural basis for the complex architecture of the neuronal networks, cellular arrays, and layers. This complex architecture of neuronal networks and the organized connectivity of neurons within neuronal arrays and layers is a key requisite for higher brain function. Cytoskeletal elements are crucially involved in bringing about the morphological complexity that neurons develop during their differentiation. Such cell shape changes predominantly occur in brains of embryos and newborns but to a lesser extent also are occurring life-long. Compelling evidence has emerged that actin filament organization and dynamics are not only pivotal for control of early neuronal morphology but are also crucially involved in establishing, maintaining and remodeling the complex and dynamic environment of postsynaptic specializations during processes indispensable for neuronal plasticity. Although these remodeling processes are an important basis for regeneration and plasticity of the brain, astonishingly little is known about how actin filament formation giving rise to the forces initiating and promoting the complex morphologies of neuronal cells are brought about. The speakers of this symposium will highlight that the neuronal actin cytoskeleton has indispensable functions in neuromorphogenesis and synaptic plasticity. They will furthermore unravel how the pivotal spatial and temporal control of cytoskeletal organization and dynamics underlying brain structure and function is brought about. The symposium aims to emphasize the role of the neuronal actin cytoskeleton in synaptic plasticity and its possible contribution to pathological changes in neurologic and psychiatric diseases.

## Symposium 20

Friday, March 20, 2015  
11:30 – 13:30, Lecture Hall 105

Chair: Britta Qualmann and Michael Kessels, Jena

- 11:30    **Opening Remarks**
- 11:40    Gaia Tavosanis, Bonn  
THE ARP2/3 COMPLEX IS REQUIRED FOR DE NOVO FORMATION OF DENDRITIC BRANCHES (S20-1)
- 12:00    Britta Qualmann, Jena  
ACTIN NUCLEATION AND MEMBRANE REMODELLING IN NEUROMORPHOGENESIS AND SYNAPTIC PLASTICITY (S20-2)
- 12:20    Martin Korte, Braunschweig  
REGULATION OF ACTIN-DYNAMICS IN PROCESSES OF NEURONAL PLASTICITY, MEMORY FORMATION AND SYNAPSE STABILIZATION (S20-3)
- 12:40    Britta Eickholt, Berlin  
PTEN REGULATES DENDRITIC SPINE FUNCTION BY TARGETING THE ACTIN BINDING PROTEIN DREBRIN (S20-4)
- 13:00    Niklas Lonnemann, Braunschweig  
FAST NOGO-A SIGNALING ACUTELY MODULATES NEURONAL STRUCTURE AND FUNCTION IN THE MATURE MOUSE HIPPOCAMPUS (S20-5)
- 13:10    Torsten Götz, Berlin  
UNCONVENTIONAL MYOSIN AFFECTS PRE-SYNAPTIC ASSEMBLY (S20-6)
- 13:20    **Concluding Remarks**





## Introductory Remarks to Symposium 21

# Neuronal mechanisms of behavioral timing

*Christian Wegener and Wolfgang Rössler, Würzburg*

The brain is the master organ in timing neurophysiological processes controlling behavior. Timing is also a key component for the internal functioning of the brain itself, like rhythmic oscillations, temporal aspects in learning and memory, synchronization of decision-making processes, and a variety of cognitive processes. Not surprisingly, the activity of neurons, neuronal networks and brains must be timed in the ranges of milliseconds to hours and days to years. Brains also generate timing signals or produce rhythmic output across this wide range of time scales including ultradian neuroendocrine output. At the same time, brains are able to adjust or synchronize these activities with external input to generate appropriate timing of behavior. The past years have seen an impressive progress in understanding molecular and neuronal underpinnings of the central circadian clock across animal taxa. However, most studies have separately addressed circadian or interval timing at the neurobiological or behavioral levels and respective computational models have been developed. Until now it remains largely unexplored how circadian and interval timers interact with each other and the environment and feed their information into neuronal networks to generate appropriate timing of adaptive behaviour. As doing the right thing at the right time is crucial for the survival of all animal species, it seems reasonable to assume that timing mechanisms are positively selected by evolution and may share common molecular, cellular and functional principles across brains from different taxa.

This symposium intends to shed light on current concepts and research progress on neuronal mechanisms of behavioural timing from different angles: from synchronising input to neuronal activity and coding all the way to the generation of behaviorally-relevant output. The symposium presents a perspective from different phylogenetic standpoints to address common neuronal principles of neuronal timing. A major focus of the symposium is on insect models with smaller brains that offer good experimental access to integrative approaches aiming at understanding neuronal mechanisms and behavioral consequences of timing.



This symposium proposal is an initiative out of the SFB1047 Insect timing.

## Symposium 21

Friday, March 20, 2015  
11:30 – 13:30, Lecture Hall 10

Chair: Christian Wegener and Wolfgang Rössler,  
Würzburg

- 11:30 Valter Tucci, Genova, Italy  
NEURONAL MECHANISMS OF BEHAVIORAL  
TIMING (S21-1)
- 11:50 Monika Stengl, Kassel  
CIRCADIAN RHYTHMS IN SECOND MESSENGERS  
AND BIOGENIC AMINES SET OLFACTORY  
THRESHOLDS IN INSECT ANTENNAE  
(S21-2)
- 12:10 Ralf Stanewsky, London, UK  
SENSORY SYSTEMS AND MOLECULAR MECHANISMS  
INVOLVED IN SYNCHRONIZING THE  
*DROSOPHILA* CIRCADIAN CLOCK (S21-3)
- 12:30 Mareike Selcho, Würzburg  
TIMING OF THE PEPTIDE-ORCHESTRATED  
ECLOSION BEHAVIOR IN *DROSOPHILA* (S21-4)
- 12:50 Martin Strube-Bloss & Martin Nawrot, Würzburg  
and Berlin  
CHRONOLOGICAL INTERACTIONS BETWEEN  
ANTENNAL LOBE AND MUSHROOM BODY:  
EXTRACTING THE BEHAVIORALLY RELEVANT  
STIMULUS (S21-5)
- 13:10 Katrin Vogt, Martinsried  
VARIABLE EVENT TIMING IN VISUAL  
CONDITIONING LEADS TO MEMORIES WITH  
OPPOSITE VALENCE IN *DROSOPHILA* (S21-6)
- 13:20 Matthias Schlichting, Würzburg  
THE HOFBAUER-BÜCHNER-EYELET SIGNALS  
TO THE VENTRO-LATERAL NEURONS AND  
THEREBY MEDIATES SIESTA AND PHASE-SHIFTS  
IN *DROSOPHILA* (S21-7)



## Introductory Remarks to Symposium 22

# Recognition molecule-associated glycans in synaptic plasticity and regeneration after trauma

*Melitta Schachner, Hamburg*

Melitta Schachner will describe the functional roles of alpha 2,8 polysialic acid (PSA) and the human natural killer antigen-1 (HNK-1) in mice and zebrafish. These glycans can influence the functions of different types of synapses. For instance, HNK-1 regulates the activity of the GABA<sub>B</sub> receptor. In regeneration after trauma, PSA and HNK-1 enhance regeneration in mouse and zebrafish models of spinal cord injury, respectively. HNK-1 improves preferential motor reinnervation after mouse femoral nerve injury, thus regulating axonal regrowth into appropriate nerve branches. For these studies, glycan mimetic peptides and small organic compounds were crucial.

Alexander Dityatev will discuss the importance of PSA in activity and plasticity of central nervous system synapses in vitro and in vivo.

Andreas Faissner will show that the stem cell niche contains factors in cerebrospinal fluid and in blood or factors originating from endothelial cells or cells in or at the niche which contribute to differentiation of glial progenitors and affect glial tumors and their stem cells. Specialized chondroitin sulfates and particular variants of the LewisX glycan, as well as the corresponding carbohydrate presenting proteins regulate proliferation and differentiation of neural stem cells.

Rita Gerardy-Schahn and Herbert Hildebrandt will present data on how NCAM's functions are modified by PSA added to the NCAM protein backbone by two polysialyltransferases, ST8SIA2 and ST8SIA4. Abnormal levels of NCAM or PSA and polymorphisms in NCAM and ST8SIA2 have been reported to be relevant to schizophrenia. Complete loss of PSA by deletion of both polysialyltransferases causes severe malformations of major fiber tracts in the brain. Even minor reductions of PSA during brain development in ST8SIA2-deficient mice cause enlarged ventricles, reduced size of some brain nuclei, and disorganized patterns of fiber tracts connecting thalamus and cortex. Loss of PSA affects interneuronal populations in the prefrontal cortex in different polysialyltransferase-deficient mouse lines, leading to abnormalities in different GABAergic interneuronal subtypes in the prefrontal cortex in the transgenic mice.

## Symposium 22

Friday, March 20, 2015  
11:30 – 13:30, Lecture Hall 103

Chair: Melitta Schachner, Hamburg

- 11:30 Melitta Schachner, Hamburg  
RECOGNITION MOLECULE-ASSOCIATED  
GLYCANS IN SYNAPTIC PLASTICITY AND  
REGENERATION AFTER TRAUMA (S22-1)
- 11:55 Alexander Dityatev, Magdeburg  
NCAM-ASSOCIATED POLYSIALIC ACID RE-  
GULATES HIPPOCAMPAL AND CORTICAL  
SYNAPTIC PLASTICITY  
(S22-2)
- 12:20 Andreas Faissner, Bochum  
COMPLEX GLYCANS AND THEIR CARRIER  
PROTEINS IN THE NEURAL STEM CELL NICHE  
(S22-3)
- 12:45 Rita Gerardy-Schahn, Hannover  
POLYSIALIC ACID ON NCAM: REGULATOR  
OF CORTICAL DEVELOPMENT WITH RELEVANCE  
TO SCHIZOPHRENIA (S22-4)
- 13:10 Dina Safina, Bochum  
LOW DENSITY LIPOPROTEIN RECEPTOR-RELA-  
TED PROTEIN 1 (LRP1) – A NOVEL MODULATOR  
OF THE NEURAL STEM CELLS' PROLIFERATION,  
DIFFERENTIATION AND SURVIVAL (S22-5)
- 13:20 Nina Westphal, Hamburg  
NUCLEAR IMPORT OF POLYSIALIC ACID CAR-  
RYING FRAGMENTS OF THE NEURAL CELL  
ADHESION MOLECULE NCAM (S22-6)



## Introductory Remarks to Symposium 23

### Breaking News II

*Marc Spehr, Aachen*

Registered students had the choice to either register with a poster presentation or apply for an oral communication. The program committee has selected the young investigator presentations from these submissions and assigned them either to a symposium or to one of the two Breaking News symposia (symposia 12 and 23).

The following students were selected to give a short communication in Symposium 23 – Breaking News II:

- 
- 11:30    **Opening Remarks**
- 11:35    Benedikt Bausewein, Bayreuth  
REPRESENTATION OF VISUAL INFORMATION  
IN THE ARCHERFISH MAUTHNER-CELL (S23-1)
- 11:45    Stephanie D. Biergans, Konstanz  
TEMPORAL DYNAMICS AND GENOME-WIDE  
TARGET REGIONS OF CYTOSINE METHYLATION  
AND HYDROXYMETHYLATION DURING LONG-  
TERM MEMORY FORMATION IN HONEYBEES  
(S23-2)
- 11:55    Christoph Bode, Leipzig  
DEVELOPMENTAL CHANGES OF STRIATAL IN-  
TERNEURONS IN AN ANIMAL MODEL OF  
PAROXYSMAL DYSTONIA (S23-3)

## Symposium 23

Friday, March 20, 2015  
11:30 – 13:30, Lecture Hall 104

Chair: Marc Spehr, Aachen

- 12:05 Karolina Can, Göttingen  
RETT SYNDROME PROVOKES REDOX IMBALANCE ALREADY IN NEONATAL NEURONS, AFFECTING THE CYTOSOL AND THE MITOCHONDRIA (S23-4)
- 12:15 Rainer Engelken, Göttingen  
INPUT SPIKE TRAINS SUPPRESS CHAOS IN BALANCED NEURAL CIRCUITS (S23-5)
- 12:25 **Break**
- 12:35 Carola Wormuth, Bonn  
A YOUNG PILOCARPINE MODEL FOR EPILEPSY (S23-6)
- 12:45 Lars Emil Larsen, Ghent, Belgium  
EFFECTS OF VAGUS NERVE STIMULATION ON HIPPOCAMPAL NEUROPHYSIOLOGY IN FREELY MOVING RATS (S23-7)
- 12:55 Uta Pegel, Marburg  
INTEGRATION OF SKY COMPASS CUES IN THE BRAIN OF THE DESERT LOCUST (S23-8)
- 13:05 Sarah Starosta, Bochum  
DYNAMIC CODING PATTERNS IN SINGLE UNITS OF THE FOREBRAIN ACROSS THREE STAGES OF LEARNING (S23-9)
- 13:15 Lena Veit, Tübingen  
LEARNING OF ARBITRARY VISUAL ASSOCIATIONS IN THE CORVID ENDBRAIN (S23-10)
- 13:25 **Concluding Remarks**



## Introductory Remarks to Symposium 24

# The emerging etiopathogenic role of infections and inflammation in chronic CNS diseases

*Wolfgang Löscher and Wolfgang Baumgärtner, Hannover*

Infectious diseases as well as neurodegenerative and inflammatory disorders of the central nervous system (CNS) represent major medical challenges of the health care systems in the coming decades. Numerous CNS diseases are triggered directly or indirectly by infections or a misdirected immune response against their causative agents. Additionally, some emerging diseases, many of them arising from zoonotic pathogens, like spongiform encephalopathy (new variant of Creutzfeldt-Jacob disease in humans), influenza, tick-borne encephalitis, and West Nile disease, are neurotropic to a varying degree. Moreover, several neuro-degenerative diseases including Alzheimer's disease and multiple sclerosis (MS) are suspected to be caused or aggravated by infections. To develop new strategies for diagnosis, prevention, and treatment of these and other CNS disorders, the complex interactions between CNS and pathogens urgently require a more profound understanding. This is not limited to a better understanding of the pathogenesis of neurological and psychiatric disorders and associated infections, but should also encompass their epidemiology by studying routes of transmission within and between species. The chairs and main speakers of this symposium are principal investigators of the novel research network N-RENNT (Niedersachsen-Research Network on Neuroinfectiology), which brings together a unique consortium of experts and institutions in the integrated fields of neuroscience and infectious diseases in Lower Saxony.

The talks give examples of the N-RENNT research, which is funded by the Ministry of Science and Culture of Lower Saxony and the VolkswagenStiftung in Germany.



## Symposium 24

Friday, March 20, 2015  
14:30 – 16:30, Lecture Hall 105

Chair: Wolfgang Löscher and Wolfgang Baumgärtner,  
Hannover

- 14:30    **Opening Remarks**
- 14:35    Albert Osterhaus, Hannover  
EMERGING VIRUS-INDUCED CNS DISEASES  
(S24-1)
- 14:55    Christian Hammer and Hannelore Ehrenreich,  
Göttingen  
ROLE OF AUTOANTIBODIES IN NEUROPSY-  
CHIATRIC DISEASES (S24-2)
- 15:15    Alexander Flügel, Göttingen  
ROLE OF LYMPHOCYTE INVASION IN CNS  
DISEASES (S24-3)
- 15:35    Kristin Michaelsen and Martin Korte, Braun-  
schweig  
ROLE OF CNS INFECTIONS FOR NEURO-  
DEGENERATIVE DISEASES (S24-4)
- 15:55    Marianna Weller, Braunschweig  
LONG-TERM INFLUENCES OF AN IMMUNE  
STIMULATION ON NEURONAL STRUCTURE  
AND PLASTICITY (S24-5)
- 16:05    Christin Schifani, Ludwigshafen  
VALIDITY OF A "TWO-HIT" DEVELOPMENTAL  
MODEL OF SCHIZOPHRENIA (PRENATAL POLY  
I:C AND NEONATAL PCP) (S24-6)
- 16:15    **General Discussion and Concluding  
Remarks**





## Introductory Remarks to Symposium 25

# Regulation of normal and impaired sleep

*Axel Steiger and Mayumi Kimura, Munich*

Recent research accumulated much knowledge about regulation and function of sleep and the pathophysiology of impaired sleep. This symposium brings together leading experts who contributed distinctly to the recent development in this area.

Tarja Stenberg (Helsinki) reports molecular mechanisms of sleep homeostasis. Adenosine increases in basal forebrain (BF) during wakefulness and decreases during sleep. Nitric oxide increases in BF through activation of inducible nitric oxide synthase coupling adenosine increase to immunological activation. These effects on sleep are connected to cortically projecting cholinergic neurons in BF, as inactivation of these cells abolishes sleep homeostasis.

Jian-Sheng Lin (Lyon) deals with the control of wakefulness under different behavioral situations focusing on brain histamine and orexin neurons. The regulation of wakefulness depends on behavioral context. Each arousal system contributes complementarily and synergistically to the maintenance of cortical activation during wakefulness. In different behavioral and cognitive context their individual participation and specific role are distinct.

Mayumi Kimura (Munich) presents a mechanism of sleep impairment by stress. In healthy condition, after long wakefulness deeper sleep increases representing sleep homeostasis. During stress, however REM sleep appears more frequently than nonREM sleep during recovery. Such REM sleep disinhibition occurs in animal models of depression and depressed patients. Corticotropin-releasing hormone is a major modulator of stress. Its brain-site specific effects on sleep under stress are discussed with a possible cholinergic enhancement in prefrontal-limbic structures.

Martin Dresler (Nijmegen) addresses sleep related memory consolidation in depressed patients. In depression, it is decreased for procedural, but not declarative tasks. Neither sleep disturbances nor REM suppression by drugs underlie these impairments, whereas high-dose corticosteroids led to impaired procedural memory consolidation. Sleep-related memory impairments in depression seem to be related to stress hormone dysfunction rather than to sleep changes.

## Symposium 25

Friday, March 20, 2015  
14:30 – 16:30, Lecture Hall 104

Chair: Axel Steiger and Mayumi Kimura, Munich

- 14:30    **Opening Remarks**
- 14:40    Tarja Stenberg, Helsinki, Finland  
THE MOLECULAR MECHANISMS OF SLEEP  
HOMEOSTASIS (S25-1)
- 15:00    Jian-Sheng Lin, Lyon, France  
THE MULTIPLE FACETS OF WAKEFULNESS,  
CONTROL BY HISTAMINE AND OREXINS (S25-2)
- 15:20    Mayumi Kimura, Munich  
THE ROLE OF CRH IN STRESS-INDUCED SLEEP  
IMPAIRMENT (S25-3)
- 15:40    Martin Dresler, Nijmegen, The Netherlands  
SLEEP-RELATED NEUROPLASTICITY IN HEALTHY  
SUBJECTS AND PSYCHIATRIC PATIENTS (S25-4)
- 16:00    Christian Schmidt, Magdeburg  
TARGETING THE SEROTONERGIC AND NOR-  
ADRENERGIC BRAIN SYSTEM TO TREAT NAR-  
COLEPSY IN A MOUSE MODEL (S25-5)
- 16:10    Nikolaos Karalis, Munich  
HIGH-DENSITY ELECTROPHYSIOLOGICAL  
CHARACTERIZATION OF THE HIPPOCAMPAL  
AND CORTICAL NETWORK ACTIVITY IN THE  
AWAKE AND SLEEPING MOUSE (S25-6)
- 16:20    **Concluding Remarks**



## Introductory Remarks to Symposium 26

# Nanostructure and function of presynaptic active zones

*Tobias Moser and Carolin Wichmann, Göttingen*

Presynaptic active zones are highly specialized membrane nanodomains that mediate transmitter release onto the postsynaptic neurons.  $\text{Ca}^{2+}$  channels and vesicular release sites are spatially well-organized at the active zones for efficient  $\text{Ca}^{2+}$ -triggered exocytosis. Recent technical advances have enabled gaining insights into the molecular nanoanatomy and -physiology of the active zone. The speakers of the symposium will present progress towards understanding the sophisticated supramolecular organization of active zones that accomplishes spatiotemporally well-defined  $\text{Ca}^{2+}$  signalling,  $\text{Ca}^{2+}$  triggered membrane fusion, vesicle replenishment and maturation. In their contributions they will relate nanoanatomy to -physiology and address concepts such as synaptic vesicle priming and vesicle pool dynamics using electron tomography combined with high-pressure freezing, cell physiology, high-resolution fluorescence and super-resolution fluorescence microscopy.

## Symposium 26

Friday, March 20, 2015  
14:30 – 16:30, Lecture Hall 8

Chair: Tobias Moser and Carolin Wichmann,  
Göttingen

- 14:30    **Opening Remarks**
- 14:40    Carolin Wichmann, Göttingen  
ULTRASTRUCTURAL DETERMINATION OF  
DYNAMIC VESICLE POOLS AT INNER HAIR  
CELL RIBBON SYNAPSES (S26-1)
- 15:00    Thomas Kuner, Heidelberg  
NANOARCHITECTURE OF ACTIVE ZONES AT  
THE CALYX OF HELD  
(S26-2)
- 15:20    Benjamin Cooper, Göttingen  
THE MORPHOLOGICAL AND MOLECULAR NA-  
TURE OF SYNAPTIC VESICLE PRIMING AT  
PRESYNAPTIC ACTIVE ZONES (S26-3)
- 15:40    Jens-Karl Eilers, Leipzig  
MUNC13-3 SUPERPRIMES SYNAPTIC VESICLES  
AT GRANULE CELL-TO-BASKET CELL SYNAPSES  
IN THE MOUSE CEREBELLUM (S26-4)
- 16:00    Rituparna Chakrabarti, Göttingen  
ACTIVITY DEPENDENT NANOSTRUCTURE OF  
INNER HAIR CELL RIBBON SYNAPSES (S26-5)
- 16:10    Tanvi Butola, Göttingen  
ROLE OF PICCOLO IN HIGH FREQUENCY  
TRANSMISSION AT THE ENDBULB OF HELD  
SYNAPSE (S26-6)
- 16:20    **Concluding Remarks**



## Introductory Remarks to Symposium 27

# Brain tumors strongly interact with different cell-types in the CNS: biological mechanisms and therapeutic impact

*Michael Synowitz, Berlin*

High-grade gliomas are malignant, incurable brain tumors. Our understanding of the cellular and molecular mechanisms promoting the formation of high-grade gliomas and their interaction with their microenvironment are rapidly advancing and can lead to new therapies. Furthermore, investigating the interplay of gliomas with cells of the adaptive or the innate immune system, neural precursors (NPCs) or with endothelial cells provides important insights into cell biological reactions to CNS pathology. Gliomas are immunosuppressive and uncovering the signalling mechanism modulating T-cell responses in the neoplastic brain identifies the cytokines that are important for coordinating immunity in the CNS. Johannes vom Berg (Zurich, Switzerland) will show how interleukins control the function of regulatory T (Treg) and effector (Teff) cells in the brain. His experiments showed that local delivery of specific interleukins together with systemic blockade of a co-inhibitory T-cell receptor determines the ratio of Treg and Teff and primes the adaptive immune system towards an efficient antitumor immune response. Stefan Momma (Frankfurt, Germany) found that small vesicles secreted from tumor cells and from physiological brain cells carry mRNA and miRNA that is taken up by target cells. Microvesicular shedding has large implications for signal transduction in the CNS and also mediates under-acknowledged effects in transgenic mouse models. Roland Kälin (Munich, Germany) will show how signalling pathways that are important for embryonic development, like the G-protein coupled receptor APJ and the cognate ligand apelin, function in endothelial cells of the brain. During glioma angiogenesis the apelin/APJ system accelerates tumour angiogenesis and constitutes a new therapeutic target. Rainer Glass (Munich, Germany) will present new data on the anti-tumorigenic effect of NPCs against gliomas. Recent evidence indicates that NPC-mediated tumor suppression is relevant to the human brain and that glioma cell-death induction is a function specifically related to NPCs, but not other stem and precursor cells. We are confident that this topic is of interest for a broad audience of neuroscientists, stem cell researchers and clinicians. With this symposium we hope to stimulate discussions and also collaborations between researchers of these different fields, since only the combined effort of researchers with different background may lead to progress in glioma research.

## Symposium 27

Friday, March 20, 2015  
14:30 – 16:30, Lecture Hall 102

Chair: Michael Synowitz, Berlin

14:30 **Opening Remarks**

14.40 Stefan Momma, Frankfurt/Main  
GENERATION OF NEURONAL PROGENITOR  
CELLS IN RESPONSE TO TUMORS IN THE  
HUMAN BRAIN (S27-1)

15:05 Johannes vom Berg, Zurich, Switzerland  
SITE MATTERS - IMMUNOTHERAPY OF  
MALIGNANT BRAIN TUMORS USING  
PRO-INFLAMMATORY CYTOKINES AND  
SYSTEMIC IMMUNOSTIMULATION (S27-2)

15:30 Roland Kälin, Munich  
DISSECTING THE ROLE OF APELIN SIGNALING  
IN GLIOMAGENESIS (S27-3)

15:55 Rainer Glass, Munich  
THE DUAL ROLE OF NEURAL PRECURSOR  
CELLS (NPCS) IN TUMORIGENESIS: NPCS  
ARE THE POINT OF ORIGIN FOR GLIOMAS  
AND ALSO CONSTITUTE A FIRST LINE OF  
DEFENCE AGAINST BRAIN TUMORS (S27-4)

16:20 **Concluding Remarks**



## Introductory Remarks to Symposium 28

# Processing of temporal stimulus cues in the insect olfactory system

*Paul Szyszka, Konstanz*

In order to generate a dynamic representation of the outside world, sensory systems have to encode both the static quality of a stimulus (e.g. color or shape) as well as its kinetics (e.g. speed and direction). The processing of stimulus kinetics is well understood in vision and audition, but less in olfaction. Airborne odors occur in turbulent plumes that break them into thin filaments, so that flying insects encounter odors as short and intermittent stimuli. In this symposium we will challenge the common notion that olfaction has rather long integration times relative to other senses and we will address the following questions: How is temporal stimulus information represented in olfactory receptor neurons and in the brain? What are the time scales of temporal stimulus information that insects can use for odor source identification? What are the neuronal mechanisms underlying the extraction of temporal stimulus information? This topic is only just emerging in the neuroscience research community, but we foresee that it will gain increasing attention in the near future. While we still have to understand how temporally complex stimuli are coded at the sensory level, the next step will be to investigate how temporal information is preserved and processed in the brain.

We have put together an international team of researchers to elucidate these problems from different angles, and in different species: Martin Andersson investigates the effect of olfactory receptor neuron co-localisation on beetles' capability to discriminate between closely separated odor sources. Carlotta Martelli will report on the use of single sensillum recordings in fruit flies to study the kinetics of olfactory receptor neuron responses and how they depend on the identity and intensity of odorants. Georg Raiser and Paul Szyszka probe the limits of insects' temporal resolution in the transduction, central processing and perception of odors. Thomas Nowotny uses computational approaches to investigate neural mechanisms underlying odor segregation based on stimulus-onset asynchrony, and how the brain can disentangle intrinsic time stamps from external temporal information.

## Symposium 28

Friday, March 20, 2015  
14:30 – 16:30, Lecture Hall 10

Chair: Paul Szyszka, Konstanz

- 14:30 **Opening Remarks**
- 14:35 Martin Andersson, Lund, Sweden  
CO-LOCALISATION OF INSECT OLFACTORY SENSORY CELLS IMPROVES THE DISCRIMINATION OF CLOSELY SEPARATED ODOUR SOURCES (S28-1)
- 15:00 Carlotta Martelli, Göttingen  
INTENSITY INVARIANT DYNAMICS AND ODOR-SPECIFIC LATENCIES IN OLFACTORY RECEPTOR NEURON RESPONSE (S28-2)
- 15:25 Paul Szyszka, Konstanz  
HIGH SPEED SMELLING AND ODOR OBJECT SEGREGATION IN INSECTS (S28-3)
- 15:50 Georg Raiser, Konstanz  
*DROSOPHILA* KENYON CELL RESPONSES TO TEMPORALLY COMPLEX ODOR MIXTURES GENERATED WITH A NOVEL HIGH-BAND WIDTH OLFACTORY STIMULATOR (S28-4)
- 16:05 Thomas Nowotny, Brighton, UK  
EXPLORING NEURAL MECHANISMS OF ODOR OBJECT-SEGREGATION IN COMPUTATIONAL MODELS (S28-5)





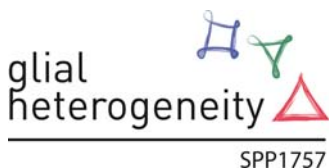
## Introductory Remarks to Symposium 28/2

# Role of glial heterogeneity in brain function

*Frank Kirchhoff and Christine Rose, Homburg and Düsseldorf*

Neuroscience research has long established that the major classes of neurones such as projection neurones or interneurones each consist of a multitude of specialized subtypes adapted to performing defined tasks in the network. Also the major classes of glial cells, namely astrocytes, oligodendrocytes and microglial cells, have important, but diverse functions. Therefore, each class of glia should not be considered a homogeneous population of cells. Recent studies provided compelling evidence that the picture of "the" astrocyte or "the" oligodendrocyte is way too simplistic. Each class of glial cells embodies a diverse cell population. Many new discoveries were possible due to increasing use of electrophysiology, imaging, and gene modification approaches *in vitro* and *in vivo*, as well as due to development of new transgenic mouse lines specific for glia. This work revealed distinct physiological properties of glia in different brain regions, at different developmental stages and at different activity levels of the organism. Functional specializations of glia apparently emerge to meet the specific requirements of distinct networks which might as such be critical determinants of brain activity. This new concept will change the way we think about brain function and puts glial cells into an even more prominent focus of attention. It is, however, still based on rather anecdotal evidence and as such, research on glial heterogeneity is in its infancy. In this symposium we will address this fundamental question of neuroscience. We will try to understand glial cell specialization and to elucidate its contribution to brain function and behavior *in vitro*, *in vivo* and *in silico*.

This symposium will be a joint meeting of the SPP 1757 and the Study Group Molecular Neurobiology of the Gesellschaft für Biochemie und Molekularbiologie (GBM).



## Symposium 28/2

Friday, March 20, 2015  
14:30 – 16:30, Lecture Hall 101

Chair: Frank Kirchhoff and Christine Rose,  
Homburg and Düsseldorf

- 14:30 Leda Dimou, Munich  
NG2<sup>+</sup>GLIA: A JOURNEY THROUGH THEIR  
DIVERSITY IN THE ADULT BRAIN (S28/2-1)
- 14:50 Maria Kukley, Tübingen  
DEVELOPMENTAL CHANGES IN SYNAPTIC  
COMMUNICATION BETWEEN AXONS AND  
OLIGODENDROCYTE PRECURSOR CELLS IN  
CORPUS CALLOSUM (S28/2-2)
- 15:10 Christian Henneberger, Bonn  
HETEROGENEITY OF ASTROCYTE COVERAGE  
OF HIPPOCAMPAL SYNAPSES (S28/2-3)
- 15:30 Kerstin Lenk, Tampere, Finland  
INEX – A COMPUTATIONAL MODEL TO SI-  
MULATE SPATIAL NEURONAL-ASTROCYTIC  
ACTIVITY (S28/2-4)
- 15:50 Ralf Dringen, Bremen  
UPTAKE AND TOXICITY OF METAL OXIDE NA-  
NOPARTICLES IN GLIAL CELLS (S28/2-5)
- 16:10 Swetlana Sirko, Munich  
HETEROGENEITY IN THE RESPONSE OF  
ASTROCYTES FOLLOWING CNS INJURY  
(S28/2-6)
- 16:20 Daniela Dieterich, Magdeburg  
ROLE OF PROTEIN TRANSLATION AND PRO-  
TEIN TURNOVER FOR ASTROCYTE HETERO-  
GENEITY IN THE HIPPOCAMPUS, STRIATUM  
AND PREFRONTAL CORTEX (S28/2-7)



## Introductory Remarks to Symposium 29

# Mechanisms of synchronization and coordination of neural oscillators

*Carmen Smarandache-Wellmann, Cologne*

Studying the mechanisms of synchronization of central pattern generators (CPGs) is of eminent importance if we want to understand the generation and functional outputs in the central nervous system. All rhythmic activity is driven by coupled neuronal oscillators that have to be synchronized for proper functioning. In the nervous system, we find different levels of such activity, for example, starting from the visceral system where CPGs of respiration or peristaltic movement which have to be active at the correct timing. Another conglomeration of synchronized neuronal oscillators are those responsible for coordination of locomotor patterns. Their activity is coordinated on segment-by-segment and network-by-network bases, but also governed through descending control from higher centers. We also find coordinated networks in cortical areas, where we are just starting to understand why these neuronal oscillators fire in the synchrony of theta, gamma and beta patterns. In this symposium, we aim to start discussions between scientists who are all interested in synchronization of neural oscillators but work with different animal model systems. The different perspectives range from: (I) coordination of locomotion CPGs, (II) coordination between locomotion and respiration, or (III) synchronization of neural oscillators in cortical networks, which can enhance cooperation. Finally, we want to show an example of a small network where the mechanisms of coordination were studied on cellular level. Here we understand how the neuronal oscillator is able to encode coordinating information and how it is able to decode and integrate this information in the neighboring neuronal oscillators.

## Symposium 29

Saturday, March 21, 2015  
8:30 – 10:30, Lecture Hall 10

Chair: Carmen Smarandache-Wellmann, Cologne

- 08:30    **Opening Remarks**
- 08:40    Karen Mesce, St. Paul, USA  
KEEPING IT TOGETHER BEFORE AND AFTER  
NERVE CORD INJURY: HOW SINGLE NEU-  
RONS HELP TO COORDINATE LOCOMO-  
TOR OSCILLATORS (S29-1)
- 09:00    Réjean Dubuc, Montreal, Canada  
LINKING RESPIRATION TO LOCOMOTION  
(S29-2)
- 09:20    Carmen Smarandache-Wellmann, Cologne  
MECHANISMS OF COORDINATION IN DIS-  
TRIBUTED NEURAL CIRCUITS: FROM ENCO-  
DING THROUGH DECODING TO INTEGRA-  
TION OF COORDINATING INFORMATION  
(S29-3)
- 09:40    Anna Caren Schneider, Cologne  
COORDINATING CENTRAL PATTERN GENE  
RATORS: TWO NEURONS, TWO STRATEGIES  
(S29-4)
- 09:50    Andreas Kreiter, Bremen  
ATTENTION DEPENDENT ROUTING BY SYN-  
CHRONY AND DYNAMIC COORDINATION  
OF NEURONAL PROCESSING IN MONKEY'S  
VISUAL CORTEX (S29-5)
- 10:10    Zahra Bahmani Dehkordi, Teheran, Iran  
THE ROLE OF NEURAL SYNCHRONY AND  
OSCILLATIONS IN FEATURE-BASED ATTENTION  
IN THE PRIMARY VISUAL CORTEX OF THE  
MACAQUE MONKEY (S29-6)
- 10:20    **Concluding Remarks**



## Introductory Remarks to Symposium 30

# Adaptation and plasticity in a distorted sense of hearing during tinnitus and hyperacusis

*Manuela Nowotny and Marlies Knipper, Frankfurt/Main  
and Tübingen*

Plasticity is an essential characteristic of the brain. It allows adaptation to new circumstances and relearning after an injury such as a stroke. In case of acoustic overstimulation (trauma), however, damage-induced adaptation processes and plasticity of the brain can have fatal consequences like the emergence of hyperacusis and tinnitus. Trauma-induced changes emerge along the entire auditory pathway beginning in the ear and across the entire auditory brain areas and accessory areas. Therefore our symposium starts with topics related to noise-induced changes in the ear including the development of tinnitus and successively change focus to more central processes.

Factors including aging and stress-related changes in the inner ear and the brain interact with tinnitus at the time of generation. Therefore compensating homeostatic processes that occur during restoration of the hearing function in animal models are investigated. To understand processes leading to tinnitus and hyperacusis, computational modeling approaches can help to test different candidate mechanisms potentially related to both, plastic changes in the auditory system after hearing loss and the development of tinnitus.

Finally, studies in humans, based on magnetic resonance methods, allow to investigate gray-matter and activity differences across diverse brain areas. These studies clearly demonstrate that not only a single brain area, but a variety of brain areas is involved in tinnitus perception. Comparing hearing-impaired patients with and without tinnitus reveals activity pattern in the human brain specifically related to tinnitus.

This symposium is supported by the Hertie-Foundation (<http://www.ghst.de>).

Gemeinnützige

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## Symposium 30

*Saturday, March 21, 2015*  
8:30 – 10:30, Lecture Hall 102

Chair: Manuela Nowotny and Marlies Knipper,  
Frankfurt/Main and Tübingen

- 08:30    **Opening Remarks**
- 08:40    Manuela Nowotny, Frankfurt/Main  
NOISE-INDUCED HEARING LOSS AND THE  
DEVELOPMENT OF TINNITUS IN MONGO-  
LIAN GERBILS (S30-1)
- 09:05    Lukas Rüttiger, Tübingen  
INTERACTIONS OF AROUSAL AND TRAUMATIC  
STRESS WITH TINNITUS RELATED HEARING  
DISORDERS IN ANIMAL MODELS  
(S30-2)
- 09:30    Roland Schaette, London, UK  
TINNITUS AND HIDDEN HEARING LOSS  
(S30-3)
- 09:55    Pim van Dijk, Groningen, The Netherlands  
ABNORMAL SOUND PROCESSING IN TINNI-  
TUS PATIENTS SUGGESTS THALAMIC DYS-  
FUNCTION: RESULT FROM FMRI (S30-4)
- 10:20    **Final Remarks**



## Introductory Remarks to Symposium 31

# Integrative study of the social insect brain - combining neuro-ethological and computational approaches

*Hiroyuki Ai, Hidetoshi Ikeno and Thomas Wachtler, Fukuoka and Hyogo (Japan) and Planegg-Martinsried*

The honeybee is known as an excellent model for inquiries into social learning and communication. Honeybees can learn associations between odors and rewards, even though their brain is much smaller than those of mammals. And it was recently discovered that some pheromones used by honeybees modulate the learning behaviors. Furthermore, they can communicate using their own unique behavior, the waggle dance (von Frisch, 1967). During the waggle dance, they produce airborne vibrations induced by wing-beat, encoding direction and distance to the nectar-bearing flower. Honeybee foragers also learn odors associated with reward, and transfer such information to their hive mates by species-specific stereotyped in-hive behaviors. With its impressive performance despite its small size, the honeybee brain is experimentally accessible and computationally attractive. Here, we bring together a number of scientists working at the interface of experimental and theoretical approaches to study the biology of olfactory learning and vibration communication underlying social communication in the honeybee. Randolph Menzel will discuss research on the function of the mushroom body as a recording device taking into account the evaluated history of experience. Martin Nawrot will present a neural network model for olfactory associative learning in the honeybee. Hiroyuki Ai will talk about the morphological characteristics of the Johnston's organ which is the vibration detector of honeybee and primary center receiving spatial information encoded in the waggle dance. Alison Mercer will talk about the adaptive value of social modulation of learning in honeybees. Student talks by Ajayrama Kumaraswamy and Anna Beer will highlight developmental aspects of behavior-relevant adaptations in communication signal processing and circadian rhythms, respectively. This symposium will make a unique contribution to the field of social insect brain, exposing synergies between experimental and theoretical approaches to systems neuroscience. The speakers will present a combination of established methods and new approaches that can be inspiring for experienced researchers and young scientists alike.

## Symposium 31

Saturday, March 21, 2015  
8:30 – 10:30, Lecture Hall 9

Chair: Hiroyuki Ai, Hidetoshi Ikeno and Thomas Wachtler,  
Fukuoka and Hyogo (Japan) and Planegg-Martinsried

08:30 **Opening Remarks**

08:40 Randolph Menzel, Berlin  
EXPLORATORY LEARNING IN BEES, AND THE  
SEARCH FOR NEURAL CORRELATES (S31-1)

09:00 Martin Nawrot, Berlin  
FROM INSECT NEUROETHOLOGY TO NEU-  
ROTECHNOLOGY: COMPUTATIONS IN SMALL  
BRAINS (S31-2)

09:20 Hiroyuki Ai, Fukuoka, Japan  
THE PARALLEL SYSTEMS IN THE PRIMARY  
AUDITORY CENTER OF THE HONEYBEE (S31-3)

09:40 Ajayrama Kumaraswamy, Munich  
EVIDENCE FOR MORPHOLOGICAL REFINE-  
MENT OF NEURONS ENCODING WAGGLE  
DANCE COMMUNICATION SIGNALS IN THE  
HONEYBEE (S31-4)

09:50 Alison Mercer, Dunedin, New Zealand  
DOPAMINE SIGNALLING AND THE SURVIVAL  
OF HONEY BEE QUEENS (S31-5)

10:10 Anna Beer, Würzburg  
HOW THE CLOCK DEVELOPS: THE PDF-  
NETWORK IN HONEYBEE BRAINS OF DIFFE-  
RENT DEVELOPMENTAL STAGES (S31-6)

10:20 **Concluding Remarks**





## Introductory Remarks to Symposium 32

# Microglia and brain Tumors: Friends or foes?

*Nicolai E. Savaskan, Erlangen*

Microglia, the brain resident macrophages, are abundant in the malignant brain tumor (glioma) microenvironment and execute tumor-promoting as well as tumor-destructive capacities. Beyond their phagocytic capacity microglia influence important biological hallmarks of gliomas including angiogenesis, adaptive immunity, resistance to therapy and cell migration.

This symposium aims at integrating various aspects of microglial function in the glioma microenvironment to critically discuss technical approaches and current concepts of microglial plasticity and function.

## Symposium 32

Saturday, March 21, 2015  
8:30 – 10:30, Lecture Hall 104

Chair: Nicolai E. Savaskan, Erlangen

- 08:30 **Opening Remarks**
- 08:35 Michael Platten, Heidelberg  
TARGETING THE IMMUNOSUPPRESSIVE  
GLIOMA MICROENVIRONMENT (S32-1)
- 08:55 Peter Vajkoczy, Berlin  
MICROGLIA AND MACROPHAGES AS MO-  
DULATORS OF GLIOMA VASCULARIZATION  
AND PROGRESSION (S32-2)
- 09:15 Janka Held-Feindt, Kiel  
BUZZING THE BUDDY: CHEMOKINES IN THE  
INTERPLAY OF TAMs AND GLIOMA CELLS  
(S32-3)
- 09:35 Nicolai E. Savaskan, Erlangen  
MIF SIGNALING AND THE BRAIN TUMOR  
MICROENVIRONMENT (S32-4)
- 09:55 Anne Régnier-Vigouroux, Mainz  
MICROGLIA IN GLIOMA BIOLOGY (S32-5)
- 10:15 Ali Ghoochani, Erlangen  
MIF SIGNALING AND THE BRAIN TUMOR  
MICROENVIRONMENT (S32-6)
- 10:25 **Concluding Remarks**



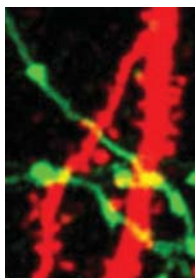
## Introductory Remarks to Symposium 33

# Balancing change and stability: homeostatic plasticity in the central nervous system

Corette Wierenga and Andreas Vlachos, Utrecht (The Netherlands) and Frankfurt/Main

Synaptic connections within our brain are highly dynamic structures that continuously change in response to experience adjusting strength and number. During the past 40 years enormous effort has been spent to dissect the cellular and molecular mechanisms of Hebbian forms of plasticity granting experience-dependent synaptic changes. Conversely, it has been recognised that despite ongoing synaptic changes functional stability of neuronal networks needs to be assured. Emerging evidence suggests that various forms of homeostatic plasticity keep the activity of neuronal networks within a dynamic range and are therefore essential to balance change and stability in the brain. Homeostatic mechanisms have been reported to occur at different synapses, at different developmental stages, or under disease conditions and we are undoubtedly only starting to appreciate the full scope of these fundamental compensatory mechanisms.

In our symposium we will discuss different forms of homeostatic plasticity that occur in the central nervous system under physiological and pathological conditions. The first speaker, Juan Burrone is an expert on synapse formation and homeostatic plasticity during functional network formation in culture. Corette Wierenga will discuss how plasticity of inhibitory and excitatory synapses may interact within dendrites. Tara Keck studies homeostatic adaptations in inhibitory neurons in the visual cortex after retinal lesion. Andreas Vlachos will elaborate on homeostatic synaptic changes occurring upon partial deafferentation. Finally, we have two contributions from young scientists working on homeostatic adaptations.



Our symposium brings together German and international neuroscientists and forms an excellent platform to examine and discuss an essential topic in neuroscience: how functional stability of networks is maintained in the brain despite ongoing synaptic changes.

## Symposium 33

Saturday, March 21, 2015  
8:30 – 10:30, Lecture Hall 8

Chair: Corette Wierenga and Andreas Vlachos,  
Utrecht (The Netherlands) and Frankfurt/Main

- 08:30    **Opening Remarks**
- 08:40    Juan Burrone, London, UK  
ACTIVITY-DEPENDENT PLASTICITY OF THE  
AXON INITIAL SEGMENT AND ITS SYNAPSES  
(S33-1)
- 09:00    Corette Wierenga, Utrecht, The Netherlands  
INHIBITORY AXONS AS DYNAMIC STRUC-  
TURES ADAPTING TO ACTIVITY (S33-2)
- 09:20    Tara Keck, London, UK  
HOMEOSTATIC PLASTICITY OF SUBNET-  
WORKS OF EXCITATORY AND INHIBITORY  
NEURONS IN MOUSE VISUAL CORTEX IN VIVO  
(S33-3)
- 09:40    Andreas Vlachos, Frankfurt/Main  
STABILITY MATTERS - HOMEOSTATIC PLASTI-  
CITY IN DENERVATED NEURONAL NETWORKS  
(S33-4)
- 10:00    Santosh Pothula, Magdeburg  
HOMEOSTATIC REGULATION OF SYNAPTIC  
FUNCTION AND RECONFIGURATION OF  
GENE EXPRESSION UPON KETAMINE TREAT-  
MENT: RELEVANCE TO ANTIDEPRESSANT  
EFFECTS (S33-5)
- 10:10    Sara Leijon, Stockholm, Sweden  
STAGGERED DEVELOPMENT OF SPON NEU-  
RONS IN MICE LACKING L-TYPE  $Ca^{2+}$ -  
CHANNELS (S33-6)
- 10:20    **Concluding Remarks**



## Introductory Remarks to Symposium 34

# Modeling evolution, neuronal development and neurodegenerative disorders using mammalian induced pluripotent stem cells

*Marisa Karow, Beate Winner and Jürgen Winkler, Munich and Erlangen*

Induced pluripotent stem cells (iPSC) exhibit an embryonic stem cell-like state, which allows for their differentiation into neural cells that in turn can be used to study various neurodegenerative diseases including Parkinson's disease (PD). Our symposium will discuss how to apply pluripotent reprogramming and direct conversion strategies from somatic cells for modelling evolution, neuronal development, and neurodegeneration. Identifying cellular and molecular differences between human and non-human primates is essential to the basic understanding of the evolution and diversity of our own species. Thus, M. C. Marchetto will show how to use iPSC as a unique biological resource to study relevant phenotypical differences between human and non-human primates. Those differences could have potential adaptation and speciation value also in the context of neurodegenerative diseases. Next, S. Cappello will focus on neuronal migration disorders and the emerging role of neural stem cells (NSC) as key players in developmental malformations. iPSC-derived NSC from patients carrying genetic aberrations are used to study the putative changes in their molecular, cellular, and functional properties. Furthermore, strategies for the development of therapeutic approaches such as the re-expression of mutant genes will be discussed. B. Winner will show how to use iPSC to investigate the pathogenesis underlying neurodegenerative diseases. Specifically, she will present how to model a motor neuron disease characterized by a progressive paraparesis and defined as hereditary spastic paraplegia (HSP) using iPSC-derived cells with a mutation in the SPG4 gene, encoding Spastin. Finally, M. Karow will present strategies for direct reprogramming of pericyte-derived cells isolated from the adult human cerebral cortex via forced expression of developmentally inspired transcription factors. Here, a special focus will be laid on the generation of human striatal interneurons as an approach to restore the basal ganglia activity that is detrimentally affected in patients with PD. In summary, we will cover a wide range of potential applications for iPSC-based techniques to model evolution, neuronal development, and neurodegeneration.