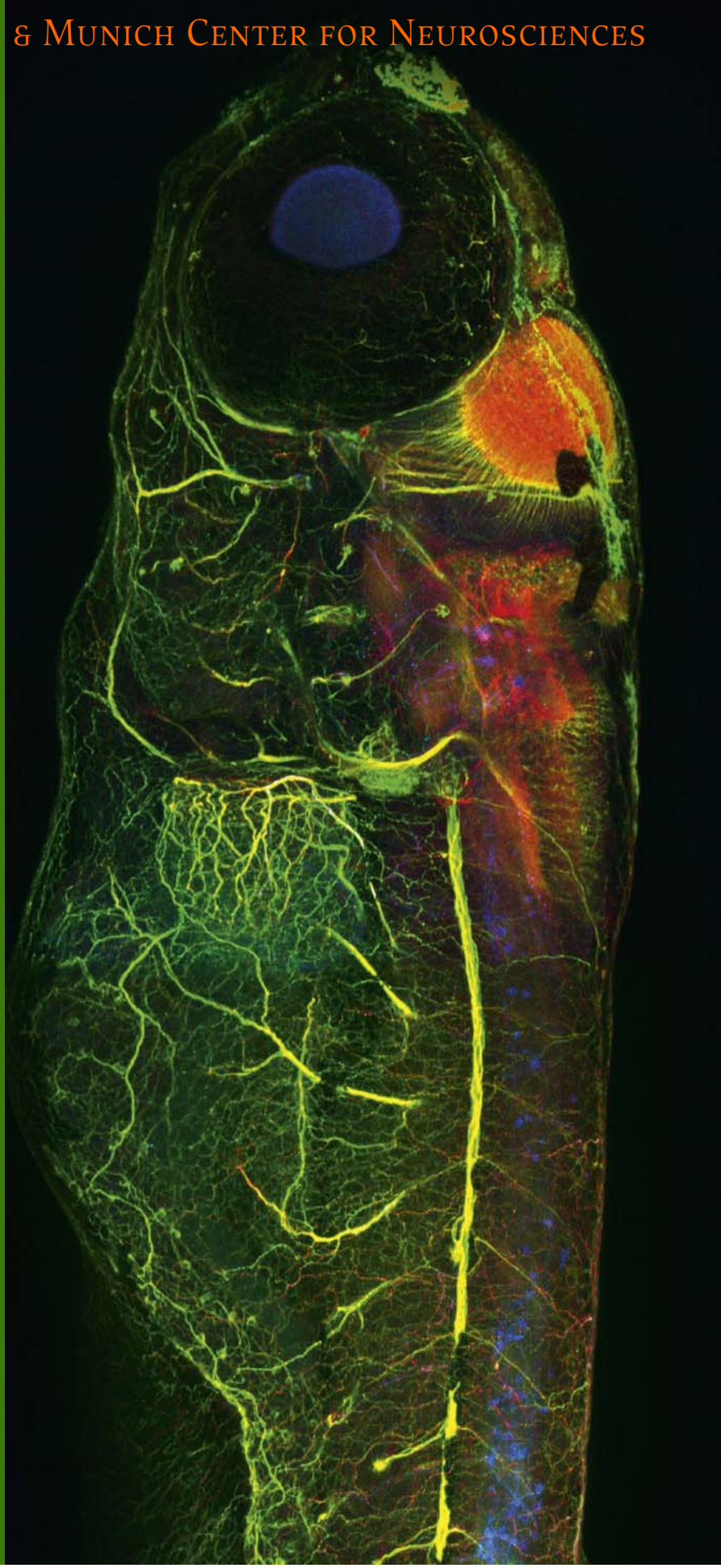
Queensland Brain Institute & Munich Center for Neurosciences

Systems Neuroscience

2nd QBI-MCN Symposium



9-12 October, 2012

Ludwig-maximilians-Universität
Munich, Germany

Supported by

The University Of Queensland AUSTRALIA | qbi queensland brain institute

MCN LMU Munich Center for Neurosciences

Graduate School of Systemic Neurosciences LMU Munich

The 2nd Systems Neuroscience Symposium, hosted this year by the Munich Center for Neurosciences (MCN), brings together leading researchers in sensory, cognitive, cellular and molecular neuroscience to share their recent findings with the neuroscience community.

Further information available at www.mcn.lmu.de

- **2nd QBI-MCN Symposium at LMU Munich**: Systems Neuroscience, October 9-12, 2012
- **Academic Board:** Prof. Perry Bartlett, Prof. Benedikt Grothe
- **Program Management:** Liz Atwood, Prof. Oliver Behrend, Sylvia Zehner
- **Participating Academic Institutes:** Graduate School of Systemic Neurosciences (GSN-LMU), Harvard University, Helmholz Zentrum München (HGMU), Munich Center for Neurosciences (MCN-LMU), Max Planck Institute of Neurobiology (MPIN), Max Planck Institute of Psychiatry (MPIPsy), Queensland Brain Institute (QBI), Technische Universität München (TUM), Signal Processing in Neurons PhD Program (SPIN – Univ. Innsbruck)
- **Academic Program:** Prof. Perry Bartlett, Prof. Oliver Behrend
- **Institutional Responsibility:** Munich Center for Neurosciences, LMU Munich
- **Cover Photo:** Courtesy of Prof. Christian Haass

Systems Neuroscience

2nd QBI-MCN Symposium

9-12 Oct. 2012; LMU Biocenter, Grosshadernerstr. 2, 82152 Martinsried

Objective

Together with the Queensland Brain Institute (QBI), the Munich Center for Neurosciences (MCN) and the embedded Graduate School for Systemic Neurosciences (GSN) co-founded the QBI-MCN Symposia Series. The symposia were instigated as a platform for students and faculty to explore two world-class neuroscience institutions of Australia and Germany, thereby providing opportunities for exchange in research and teaching.

The initiative aids the transition from the doctoral to the postdoctoral phase and potentially scouts for well educated students from abroad on advanced doctoral and postdoctoral levels. The inaugural symposium on Systems Neuroscience between QBI and MCN-LMU took place at the University of Queensland, Brisbane, Australia in September 2011. The event is followed by this year's meeting in Munich that showcases the Munich neuroscience community as a promising environment for well educated young scientists.

The symposia aim to enhance national and international visibility for both institutions. The future concept of collaboration embraces an exchange of MSc and PhD students for summer schools (planned for 2013), research projects including long term student exchange (1-2 years) and ongoing short-term exchange (2-3 weeks), as well as regular bilateral faculty visits.

Program

Tuesday, 9th October 2012 - Arrivals	
16:30	Munich City Tour (delegates and faculty) pick-up at Carlton Astoria Hotel
19:00	Welcome Dinner (delegates and faculty) Hosted by Prof. Benedikt Grothe & Dr. Stefan Lauterbach Spatenhaus (Residenzstr. 12, 80333 Munich)

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Wednesday, 10th October 2012 – Scientific Sessions I (Room B01.027)	
10:00	**Welcome address** Prof. Perry Bartlett (Director, QBI) Prof. Benedikt Grothe (Speaker, MCN) Dr. Stefan Lauterbach (Director, LMU International Office)
Session 1 - Neural Circuits (Chair: Stefan Glasauer, LMU)	
10:15	PD Dr. Felix Felmy (MCN; LMU): *Duration of "persistent inhibition" is set by synaptic activity and passive integration*
10:45	Prof. Martin Biel (MCN; LMU): *Ion channel diseases of the retina: from pathomechanisms to treatment*
11:15	Coffee & Tea Break
Session 2 – Neurogenesis (Chair: Christian Haass, LMU)	
11:45	Dr. Jovica Ninkovic (MCN; HMGU): *Essential role of BAF complex interacting with Pax6 in establishment of a core cross-regulatory neurogenic network*
12:15	Prof. Perry Bartlett (QBI): *Activation of different neurogenic precursor populations in the hippocampus: Potential for Dementia and Depression Therapy*
12:45	Lunch – Freshmaker (delegates and faculty)
Session 3 – Neural Regulation (Chair: Stefan Lichtenthaler, TUM)	
14:00	Assoc. Prof. Elizabeth Coulson (QBI): *Regulation of neuronal survival and death signalling by the cleaved fragments of p75 neurotrophin receptor*
14:30	Dr. Charles Claudianos (QBI): *MicroRNA regulation of olfactory learning and memory in honeybees*
15:00	Coffee & Tea Break
Session 4 – Sensory-Motor Control (Chair: Benedikt Grothe, LMU)	
15:30	Prof. Alexander Borst (MCN; MPI): *Genetic dissection of the fly visual course control system*
16:00	Dr. Ingo Schiffner (QBI): *A birds view: Control and precision in flight and landing*
16:30	Prof. Mandyam Srinivasan (QBI): *Of bees, birds and robots*
17:00	Afternoon Tea
17:30	**Strategy meeting (delegates and faculty; room D00.013)**
19:00	Dinner (delegates and faculty) Trattoria 4Mori (Heiglhofstr. 3, 81377 Munich)

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Thursday, 11th October 2012 – Scientific Sessions II (Room B01.027)	
Session 5 – Cognition I (Chair: Hermann Müller, LMU)	
10:00	Prof. Stefan Glasauer (MCN; LMU): *Spatial behaviour as probabilistic inference*
10:30	Prof. Jason Mattingley (QBI): *Enhanced steady-state neural responses to task-relevant features at ignored locations during visual search*
11:00	Coffee & Tea Break
Session 6 – Cognition II (Chair: Lutz Wiegrebe, LMU)	
11:30	Dr. Donatas Jonikaitis (MCN; LMU): *"What" and "where" of spatial attention*
12:00	Will Harrison (QBI): *Oculomotor preparation mitigates visual crowding*
12:30	Lunch – Freshmaker (delegates and faculty)
Session 7 – Modulation (Chair: Christian Leibold)	
14:00	PD Dr. Mathias Schmidt (MCN; MPI): *Unravelling the molecular mechanisms of short- and long-term stress-induced cognitive deficits*
14:30	Dr. Judith Reinhard (QBI): *Investigating neurexin and neuroligin behavioural functions in vivo using Drosophila melanogaster*
15:00	Coffee & Tea Break
Session 8 – Olfactory processing (Chair: Georg Dechant, SPIN)	
15:30	Dr. Hiromu Tanimoto (MCN; MPI): *Memory formation and mushroom bodies in the fly brain*
16:00	Prof. Pankaj Sah (QBI): *Olfactory processing in the medial amygdale*
16:30	Prof. Venkatesh Murthy (MCN; Harvard University): *Feedback control of circuits and codes in the mammalian olfactory bulb*
17:00	Closing Remarks
17:05	Afternoon Tea
17:30	**Joint poster session (details below; D00.003 foyer)**
19:00	Informal Dinner Buffet (LMU Biocenter, D00.003 foyer)

Friday, 12th October 2012 – Excursion: Bavarian Alps Herrenchiemsee and Kampenwand (event subject to current weather conditions)	
9:00	Pick-up at hotel Carlton Astoria
21:00	Return to Munich

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Posters

QBI – Queensland Brain Institute (University of Queensland)

Amelia Douglass:
DCC Functions in multiple aspects of corpus callosum development

Shao-Chang Huang:
Prey capture ability in damselflies: function of ommatidia structure, viewing angle, and visual resolution

Gavin Taylor:
Combining the senses: Looking at the interaction of wind and vision on the honeybees' streamlining response

MCN – Munich Center for Neurosciences (LMU/ MPI/ HMGU/ TUM)

Filippo Calzolari & Julia Schwausch (Götz lab):
Clonal analysis of neural stem cell output in the adult mouse subependymal zone

Boris Chagnaud (Straka lab):
Intrinsic and network properties of a highly synchronous hindbrain motor nucleus

Graziana Gatto (Klein lab):
Receptor tyrosine phosphatase PTPRO inhibits trigeminal axon growth and branching by repressing TrkB and Ret signaling

Rene Gilster (Deubel lab):
Motor selection and visual attention in manual grasping

Lars Kunz (Grothe lab):
1) Metabolic maturation of auditory brainstem neurones
2) The impact of the endocannabinoid system on sound localisation

Christian Leibold:
Spatial behavior of mongolian gerbils in complex virtual environments

Sónia Paixão (Klein lab):
EphA4 marks a population of spinal cord dorsal interneurons required for left-right coordination of locomotion

Gregor-Alexander Pilz (Götz lab):
Amplification of progenitors in the mammalian telencephalon includes a novel radial glia cell type

Dragan Rangelov (Müller lab):
Pop-out visual search mechanisms: Not all display densities are made equal

Juhi Sardana (Kadow lab):
Eph, Ephrin and Ephexin instruct dendritic patterns of specific olfactory projection neurons in fly

Ludwig Wallmeier (Wiegrebe lab):
Echolocation vs. echo suppression: Influence of the precedence effect on the human-sonar localization of reflective surfaces

SPIN – Signal Processing in Neurons (University of Innsbruck)

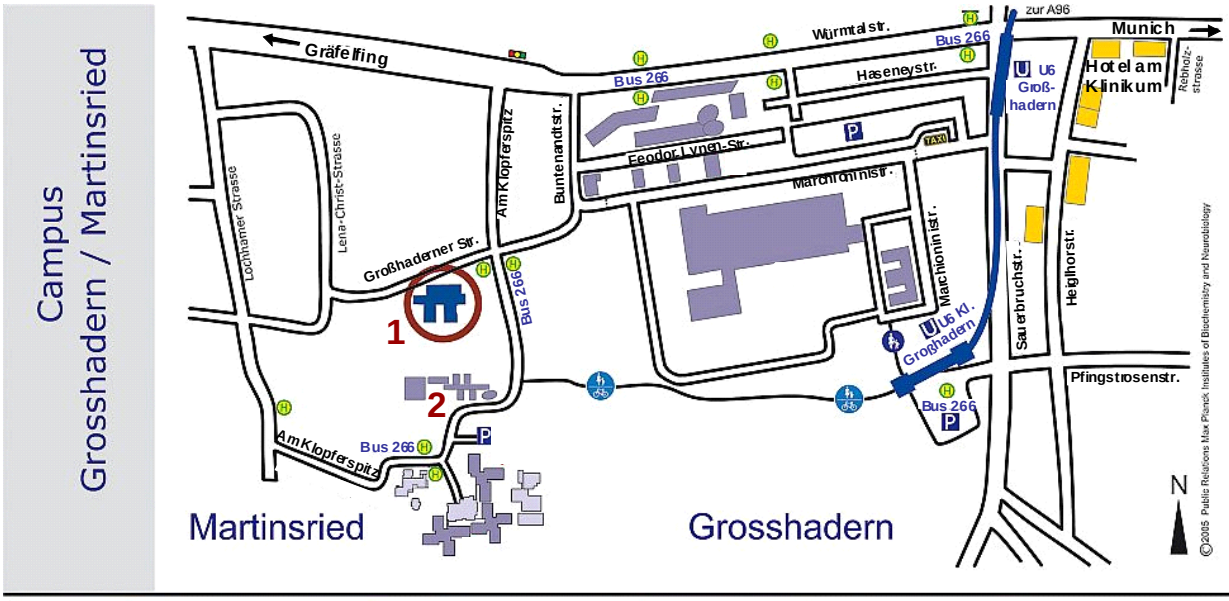
Kai Kummer (Dechant lab):
Social interaction reverses both cocaine conditioned place preference and associated brain activation patterns

Claudia Schmuckermair (Dechant lab):
Behavioral and neurobiological effects of deep brain stimulation in a mouse model of high anxiety- and depression-like behavior

GSN – Graduate School for Systemic Neurosciences (LMU/ MPI/ HMGU/ TUM)

50+ additional posters by current GSN students (GSN IntroWeek; details in separate booklet)

Martinsried Campus Map



1
LMU BioCenter
Großhadernerstr. 2
82152 Plannegg- Martinsried

2
Café Restaurant *Freshmaker*
Am Klopferspitz 19a
82152 Planegg - Martinsried

Freshmaker:
Lunch location for delegates and faculty on Wednesday (Oct. 10th) and Thursday (Oct. 11th)

Participants

Queensland Brain Institute (QBI)

- **Perry Bartlett**, Director
  QBI, Univ. of Queensland
- **Charles Claudianos**, Principle Investigator
  Molecular Mechanisms of Senses and Synapses, QBI
- **Elizabeth Coulson**, Associate Professor
  Nerve Cell Survival, QBI
- **Amelia Douglass**, Senior Research Technician
  Cortical Development and Axon Guidance (Prof. Richards), QBI
- **Will Harrison**, PhD Student
  Cognitive Neuroscience (Prof. Mattingley), QBI
- **Shao-Chang Huang**, PhD Student
  Olfactory Function and Behaviour (Dr. Reinhard), QBI
- **Jason Mattingley**, Professor
  Cognitive Neuroscience, QBI
- **Judith Reinhard**, Principle Investigator
  Olfactory Function and Behaviour, QBI
- **Pankaj Sah**, Professor
  Synaptic Plasticity, QBI
- **Ingo Schiffner**, Post-doctoral fellow
  Visual and Sensory Neuroscience (Prof. Srinivasan), QBI
- **Mandyam Srinivasan**, Professor
  Visual and Sensory Neuroscience, QBI
- **Gavin Taylor**, PhD Student
  Visual and Sensory Neuroscience (Prof. Srinivasan), QBI
- **Linda J. Richards**, Professor
  Cortical Development and Axon Guidance (Prof. Richards), QBI

Munich Center for Neurosciences (MCN-LMU)

Helmholtz Zentrum München (HMGU), Munich
Ludwig-Maximilians-Universität München (LMU)
Max Planck Institute of Neurobiology (MPIN)
Max Planck Institute of Psychiatry (MPIPsy)
Technische Universität München (TUM)

- **Oliver Behrend**, Managing Director
  Munich Center for Neurosciences - Brain & Mind, LMU
- **Martin Biel**, Professor
  Dept. Pharmacy, LMU
- **Alexander Borst**, Professor
  Dept. Systems and Computational Neurobiology, MPI of Neurobiology
- **Filippo Calzolari**, PhD Student
  Institute Stem Cell Research (Prof. Götz), HMGU
- **Boris Chagnaud**, Post-doctoral fellow
  Div. Neurobiology (Prof. Straka), LMU
- **Heiner Deubel**, Professor
  Dept. Psychology and Pedagogy, LMU
- **Felix Felmy**, Principle Investigator
  Div. Neurobiology, LMU
- **Graziana Gatto**, PhD Student
  Dept. Molecules - Signaling - Development (Prof. Klein), MPI of Neurobiology
- **René Gilster**, Research assistant
  Dept. Psychology and Pedagogy (Prof. Deubel), LMU
- **Stefan Glasauer**, Professor
  Institute for Clinical Neuroscience, LMU
- **Magdalena Götz**, Professor
  Institute Stem Cell Research, HMGU
- **Benedikt Grothe**, Professor
  Div. Neurobiology, LMU
- **Christian Haass**, Professor
  German Center for Neurodegenerative Diseases & Adolf Butenandt Institute, LMU
- **Donatas Jonikaitis**, Post-doctoral fellow
  Dept. Psychology and Pedagogy (Prof. Deubel), LMU

- **Ilona Grunwald-Kadow**, Principle Investigator
  Sensory Neurogenetics, MPI of Neurobiology
- **Rüdiger Klein**, Professor
  Dept. Molecules - Signaling – Development, MPI of Neurobiology
- **Lars Kunz**, Principle Investigator
  Div. Neurobiology, LMU
- **Stefan Lauterbach**, Director
  LMU International Office
- **Christian Leibold**, Professor
  Div. Neurobiology, LMU
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  Dept. Medicine, TUM
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  Institute Stem Cell Research (Prof. Götz), HMGU
- **Dragan Rangelov**, Post-doctoral fellow
  Dept. Psychology and Pedagogy (Prof. Müller), LMU
- **Juhi Sardana**, PhD Student
  Sensory Neurogenetics (Dr. Grunwald-Kadow), MPI of Neurobiology
- **Mathias Schmidt**, Principle Investigator
  Neurobiology of Stress, MPI of Psychiatry
- **Hiromu Tanimoto**, Principle Investigator
  Behavioral Genetics, MPI of Neurobiology
- **Hermann Müller**, Professor
  Dept. Psychology and Pedagogy, LMU
- **Hans Straka**, Professor
  Div. Neurobiology, LMU
- **Ludwig Wallmeier**, PhD Student
  Div. Neurobiology (Prof. Wiegrebe), LMU
- **Lutz Wiegrebe**, Professor
  Div. Neurobiology, LMU

Signal Processing in Neurons PhD Program (SPIN), University of Innsbruck

- **Georg Dechant**, Director SPIN
  Institute for Neuroscience, Innsbruck Medical Univ.
- **Kai Kummer**, PhD Student
  Div. Experimental Psychiatry, Innsbruck Medical Univ.
- **Claudia Schmuckermair**, PhD Student
  Institute of Pharmacy: LFUI, Innsbruck Medical Univ.
- **Veronika Schuchter**, Coordinator
  Institute for Neuroscience: SPIN Office, Innsbruck Medical Univ.

Harvard University (special guest)

- **Venkatesh Murthy**, Professor
  Dept. of Molecular and Cellular Biology, Havard Univ.

Abstracts
Scientific Talks

Session 2 - Neurogenesis, Wednesday Oct. 10th

Activation of different neurogenic precursor populations in the hippocampus: Potential for Dementia and Depression Therapy

Perry Bartlett[1]

1 Queensland Brain Institute (QBI), University of Queensland, Brisbane, Australia

The production of new neurons in the hippocampus is thought to underpin aspects of learning and memory. Defining how neurogenesis is regulated is central to our understanding of the learning process and to the future development of neurogenic-based therapeutics aimed at ameliorating cognitive loss.

Recently, we identified a large precursor pool in the dentate gyrus of the mouse hippocampus, including a small number of true stem cells, which is normally dormant but can be activated by depolarizing levels of K+ to produce large numbers of neurogenic neurospheres. *In situ* stimulation of the perforant pathway also activates this precursor population and leads to an increase in newly born neurons. Importantly, this population can be activated in the aged mouse, uncovering the potential for significant neurogenesis in the ageing brain.

Further, synaptic activity stimulates precursor activity through the release of a number of soluble factors and the neurotransmitter, norepinephrine (NE). These factors act directly on the precursors with NE activating through a novel adreno-receptor pathway. Interestingly, different stimuli led to the activation of different pools of precursors and stem cells, suggesting production of hippocampal neurons in the dentate gyrus with distinct properties reflective of a specific stimulation process. This provides a mechanism by which the functional capacity and the number of newly generated neurons can be directly influenced by the type and complexity of environmental stimuli.

Session 1 - Neural Circuits, Wednesday Oct. 10th

Ion channel diseases of the retina: from pathomechanisms to treatment

Martin Biel[1]

1 Department of Pharmacy – Center for Drug Research, LMU Munich, Germany

The retina is a neuronal network dedicated to converting visual stimuli (photons) into spatially and temporally controlled patterns of action potentials that are transmitted to the visual cortex. Ion channels play a fundamental role in both principal visual transduction and intraretinal information processing. Dysfunction of these proteins gives rise to various retinal diseases (retinal "channelopathies") that can eventually lead to blindness. In my lecture I will summarize our recent work on retinal cyclic nucleotide-gated (CNG) channels. These channels are specifically expressed in photoreceptor outer segments where they play a fundamental role in transducing light-evoked changes of the cGMP concentration into electrical signals. I will discuss the mechanisms that link dysfunction of these channels with specific types of blindness and retinal degeneration. Recently, our laboratory has developed viral vectors to rescue loss of retinal ion channels and restore vision in genetic mouse models. The impact of gene replacement on the therapy of human blinding eye diseases will be discussed.

Session 4 - Sensory-Motor Control, Wednesday Oct. 10th

Genetic dissection of the fly visual course control system

Alexander Borst[1]

1 Dept. of Systems and Computational Neurobiology, MPI of Neurobiology, Martinsried, Germany

Visual navigation has been studied extensively in flies, both in tethered as well as in freely flying animals. As neural control elements, the tangential cells of the lobula plate seem to play a key role: they are sensitive to visual motion, have large receptive fields, and, with their spatial distribution of preferred directions, match the optic flow as elicited during certain types of flight manoeuvres. However, several key questions have remained unanswered for long:
1. What is the neural circuit presynaptic to the tangential cells responsible for extracting the local direction of motion? 2. Do the lobula plate tangential cells indeed control turning responses of the fly? 3. Is there a separate visual course control system allowing the fly to detect and track individual objects? I will present recent progress towards answering these questions made by combining whole-cell patch recording and behavioral studies with silencing and optogenetic stimulation of genetically targeted candidate neurons in Drosophila.

Session 3 - Neural Regulation, Wednesday Oct. 10th

MicroRNA regulation of olfactory learning and memory in honeybees

Alexandre S. Cristino[1], Stephanie D. Biergans[1], Judith Reinhard[1], Charles Claudianos*[1]

1 Queensland Brain Institute (QBI), University of Queensland, Brisbane, Australia

We investigated how learning and long-term memory formation affects gene expression in the honeybee brain using an olfactory conditioning paradigm (proboscis extension reflex, PER). A microarray gene expression analysis comparing groups of conditioned and control bees found 53 genes differentially expressed between these two groups. Most of these genes were down regulated in trained bees, with only a few non-coding RNAs upregulated. A total of 16 genes were validated using qRT-PCR analysis. Among the down-regulated group were genes encoding chromatin remodelling (*Histone-1, Histone-2B*), RNA interference (*Headcase*), cytoskeleton (*Actin, Heatshock 83*), protein transport (Sec61β) and metabolism (*Asparagine Synthetase*). These down-regulated genes were enriched with binding sites for microRNAs (miRNAs), indicating miRNAs might be responsible for the inhibition of these genes during memory formation. Indeed, qRT-PCR analysis validated that seven miRNAs were upregulated in trained bees. Of these seven miRNAs, we further investigated miR-210, which is associated with foraging, miR-928 and miR-932, which are embedded in key neurological genes, *ether a go-go (eag)* and *neuroligin 2 (Nlg2)*, respectively. We suggest that 'modules of miRNAs may regulate synapse development during learning and memory processes'. To test this hypothesis we used small interference RNAs (cholesterol conjugated antagomers) to inhibit miR210, miR928, and miR932. Inhibition of miR928 and miR932 impressively impaired the formation of olfactory long-term memory while inhibition of miR210 had no effect. Our results show that miRNAs associated with key synaptic genes are involved in control of long-term memory formation in honeybees.

* *speaker*

Session 3 - Neural Regulation, Wednesday Oct. 10th

Regulation of neuronal survival and death signalling by the cleaved fragments of p75 neurotrophin receptor

Elizabeth Coulson[1]

1 Queensland Brain Institute (QBI), University of Queensland, Brisbane, Australia

$p75^{NTR}$ is well known to mediate both neuronal survival and cell death depending on the ligand and the expression of its coreceptors Trk and sortilin (which bind to pro-neurotrophins). $p75^{NTR}$ undergoes regulated cleavage (RIP) by a metalloprotease and γ-secreatase, producing first a membrane bound C-terminal fragment and subsequently a free intracellular domain fragment ($p75^{ICD}$). We have been studying the effects of the co-receptors on $p75^{NTR}$ RIP as well as the functional outcomes following modulation of $p75^{NTR}$ cleavage events.

We have evidence that the conformation of the $p75^{NTR}$ transmembrane domain, through which it homodimerises, regulates the ability of $p75^{NTR}$ and to be cleaved by γ-secreatase. We have found that both sortilin co-expression and Trk activation promotes $p75^{NTR}$ RIP and have mapped their interactions. $p75^{NTR}$ and sortilin interact via their extracellular domains, suggesting modulation of cleavage occurs through a change in protein trafficking of the full-length receptors. The interaction between $p75^{NTR}$ and Trk receptors occurs via their intracellular domains, with $p75^{ICD}$ facilitating neurotrophic signalling by triggering a conformational change that alters the neurotrophin binding affinity of Trk receptors.
We are interested in understanding more about the endogenous regulation of $p75^{NTR}$ cleavage and its relationship to $p75^{NTR}$ function, particularly in the context of neurodegenerative conditions.

Session 1 - Neural Circuits, Wednesday Oct. 10th

Duration of “persistent inhibition” is set by synaptic activity and passive integration

Felix Felmy[1]

1 Department Biology II, Division of Neurobiology, LMU Munich, Germany

The dorsal nucleus of the lateral lemniscus (DNLL) is part of the binaural system and is thought to play a crucial role in echo suppression. To serve this role the physiological relevant hallmark of the DNLL, the generation of an exceptionally long lasting GABAergic inhibition appears crucial. This “persistent inhibition” suppresses activity for tens of milliseconds in the contra lateral DNLL. *In vitro* it has been shown that the kinetics of single GABAergic IPSC decay within ~ 4 ms, being too fast to explain the persistent inhibition. The generation of the appropriate inhibitory time course might be adjusted in an activity dependent manner as was proposed in juvenile gerbils. In this study we describe the cellular basis of the persistent inhibition and its dependence on activity using *in vitro* patch clamp recordings in acute brain slices from P30-35 Mongolian Gerbils.

To probe for an activity dependence of the GABAergic decay time constants commissural inputs were stimulated at different frequencies, pulse numbers and intensities. Higher frequencies, more successive pulses and more incoming fibres lead to an increase in the decay time constant of GABAergic IPSCs. Pharmacologically dissection of the synaptic mechanisms showed that transmitter spill over and asynchronous release is enhanced in an activity dependent manner prolonging GABAergic decay time constants. To understand how the IPSC time course is transformed into the suppression of activity it is crucial to understand the factors that influence the conversion of this inhibitory conductance into effective IPSPs. Here, as the chloride reversal potential is ~ -90 mV the GABAergic IPSPs are strongly hyperpolarizing. Furthermore, the membrane properties between the resting potential and the reversal potential indicate that synaptic GABA conductances are integrated in a passive manner. From this it follows that larger synaptic conductances cause slower IPSPs. The injection of simulated synaptic conductances shows that these membrane properties transform the activity dependent slowing of IPSCs into a long lasting hyperpolarization. This hyperpolarization is indeed sufficient to suppress action potentials for tens of milliseconds. Together, the activity dependent increase in decay time based on spill over and asynchronous release is passively integrated to generate a GABAergic hyperpolarization that mimics the persistent inhibition *in vivo* underlying the suppression of space information during echo perception.

Session 5 - Cognition I, Thursday Oct. 11th

Spatial behavior as probabilistic inference

Stefan Glasauer[1]

1 Center for Sensorimotor Research, Clinical Neurosciences, LMU Munich, Germany

Perception and action are the result of the integration of various sources of information, such as current sensory input, prior experience, or the context in which a stimulus occurs. Yet, how do we combine such diverse information to guide our actions? Here we propose an iterative Bayesian framework to fuse sensory input, symbolic cues, and prior experience and to learn unknown stimulus statistics from experience. We show for a simple spatial task that humans (1) integrate sensory information and prior experience into their perceptual estimate and (2) learn the mapping of a verbal, symbolic cue onto the stimulus dimension. Our model replicates the behavioral results on a trial-by-trial basis and explains common psychophysical phenomena such as range effects, order effects, and regression to the mean. We further show that (1) under realistic noise conditions, the performance of our adaptive model is close to a normative 'optimal' model that takes the actual stimulus statistics into account and (2) if the stimuli are generated by a random walk, the systematic errors are predicted to vanish.

Session 6 - Cognition II, Thursday Oct. 11th

Oculomotor preparation mitigates visual crowding

Will Harrison*[1], Roger W. Remington[1], Jason B. Mattingley[1]

1 Queensland Brain Institute (QBI), University of Queensland, Brisbane, Australia

Our ability to recognize a visual target in the periphery is impaired when the target is flanked by non-target objects, a phenomenon referred to as crowding. Crowding has been attributed to the obligatory pooling of visual signals, such as orientation information, from the target and flanker stimuli within primary visual cortex. Here, we made a crowded object the target of an eye movement. This enabled us to test if the magnitude and spatial extent of crowding is reduced by the precise spatial selection of the target required by the oculomotor system to execute accurate saccades. Compared with trials in which no saccade was planned, we found that when a crowded target was the goal of a planned saccade, observers' target discrimination accuracy increased to the extent that target recognition was equal to that of an uncrowded target at the same location during constant fixation. We next measured the critical distance of crowding - the zone around a target within which flankers reduce target identification - and found that this distance shrinks just prior to a saccade. Finally, we found that the spatial characteristics of saccade landing points were similar to the spatial characteristics of crowding. Contrary to the widely held view that crowding depends on retinal signals alone, our findings reveal an important role for eye movement signals. These pre-saccadic changes may enable enhanced recognition of visual objects in the periphery during active search of visually cluttered environments.

** speaker*

Session 6 - Cognition II, Thursday Oct. 11th

“What” and “where” of spatial attention

Donatas Jonikaitis*[1], Heiner Deubel[1]

1 Dept. of Psychology, LMU Munich, Germany

Spatial attention is defined as a mechanism which is related to both, improved perceptual performance within a specific part of the visual field and higher neural firing rates within receptive fields of neurons representing that part of space. Typically, it is assumed that spatial attention originates in the saccadic system (e.g., frontal eye fields, lateral intraparietal area, superior colliculus).

In a series of experiments we investigated the allocation of spatial attention and its relationship to saccade plans and found evidence that spatial attention does not originate exclusively within the saccadic system. First, top-down controlled spatial attention is tightly bound to saccade planning, and competes for shared processing resources. Second, bottom-up driven spatial attention is independent of saccade preparation, and does not affect saccadic planning. Third, spatial attention benefits are independent of feature-based attention, which acts within the whole visual field. Combined, our findings refine what and where spatial attention is - only some subsystems of spatial attention are manifested within the saccadic system, whereas others are not.

** speaker*

Session 5 - Cognition I, Thursday Oct. 11th

Enhanced steady-state neural responses to task-relevant features at ignored locations during visual search

Jason Mattingley[1,2]

1 Queensland Brain Institute (QBI), University of Queensland, Brisbane, Australia
2 School of Psychology, University of Queensland, Brisbane, Australia

During visual search, observers can select targets on the basis of their spatial locations or their elementary features, such as color. It is currently unknown to what extent feature-based selection within the search array affects neural representations of objects that fall outside the spatial focus of attention. We investigated the effects of feature-based attention on ignored peripheral stimuli as observers searched rapidly changing foveal arrays. In separate blocks, observers monitored these arrays for targets in the cued color, and ignored concurrently presented peripheral checkerboards that were always task-irrelevant. Within the foveal arrays, targets were distinguishable from distractors either by a unique color (unique-feature search) or by a combination of color and shape (conjunction search). We used frequency tagging in which flickering visual stimuli produce steady-state neural oscillations at the flicker frequency, detectable at the scalp via electroencephalography (EEG). This allowed us to separate neural responses to target and distractor items in the task-relevant search arrays from those elicited by the checkerboards. The checkerboards contained interleaved elements of three different colors: those that matched the target color, those that matched the distractor color, and those of a third, neutral color. During unique-feature search, the magnitude of checkerboard-evoked cortical oscillations was independent of the search color. During conjunction search, however, checkerboard elements matching the target color evoked enhanced cortical oscillations relative to those associated with distractor and neutral colors. This suggests that competitive interactions at the fovea drive featural enhancement in the periphery. Our findings provide strong evidence that feature-based selection spreads across the visual field during search to enhance cortical representations of spatially unattended stimuli that share target defining features.

Session 8 - Olfactory processing, Thursday Oct. 11th

Feedback control of circuits and codes in the mammalian olfactory bulb

Venkatesh Murthy[1]

1 Harvard University, Cambridge, MA, USA

The formation of sensory percepts in our brain is often described as a feed-forward process that involves serial transformation of sensory stimuli into neural activity. Yet our everyday experience clearly shows that higher brain areas can actively modulate how sensory information is processed even in the earliest stages. In the mammalian olfactory system, the first stages of synaptic processing are in the olfactory bulb, which receives massive feedback projections from the cortex as well as midbrain neuromodulatory regions. In this talk, I will present our studies on the circuitry of these feedback projections as well as their influence on odor representation in rodents. These studies involve two feedback systems, the serotonergic innervation from the raphe nucleus and glutamatergic projections from the olfactory cortex, and exploit new developments in optogenetics and optophysiology.

Session 2 - Neurogenesis, Wednesday Oct. 10th

Essential role of BAF complex interacting with Pax6 in establishment of a core cross-regulatory neurogenic network

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The molecular mechanisms of neurogenic fate determination are of particular importance in light of the need to regenerate neurons. Here we describe the regulation of neurogenic fate by the transcription factor Pax6 acting together with the Brg1-containing BAF chromatin remodeling complex in the adult brain. Pax6 physically interacts with the Brg1-containing BAF complex and genetic deletion of either Pax6 or Brg1, in the neural stem cells of the adult mouse subependymal zone results in a strikingly similar fate conversion from late neuronal progenitors, neuroblasts, to distinct glial subtypes. The newly identified complex mediates neurogenesis by directly activating the transcription factors Sox11, Nfib and Brn4, which form a cross-regulatory network that maintains neurogenic fate downstream of the Pax6-BAF complex. Our work identifies a novel concept of stratification in neural fate commitment with the highly specific role of chromatin remodeling complex in initiating effector network and the maintenance of the lineage decision.

Session 7 - Modulation, Thursday Oct. 11th

Investigating neurexin and neuroligin behavioural functions in vivo using Drosophila melanogaster

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As the brain develops, huge numbers of neurons synapse precisely with each other to form complex networks. Even after these initial connections are made, the circuitry of the brain is plastic and is altered to reflect an animal's environmental experiences. Neurexins and neuroligins are cell adhesion molecules thought to be vital for the formation, modulation, activity-dependent maturation and specification of synapses. Their functional importance is underlined by the fact that mutations in these genes have been associated with a number of cognitive disorders, including autism, in humans. *Drosophila melanogaster* is a particularly useful model system in which to study the function of these genes *in vivo*, due in large part to the availability of a wide array of genetic tools with temporal and spatial control. We are employing a range of behavioural assays to assess function of neurexin and neuroligins *in vivo*, in an effort to relate abnormalities in synaptic communication and circuitry imbalances to not only basic activities, but also more complex cognitive tasks. Results show that overexpression and/or gene disruption of *neurexin 1* and *neuroligin 2* affects grooming behaviour, as well as perturbs sleep patterns. We are currently investigating potential involvement of these genes in learning and memory processes, social interaction, and general motor function using fully automated Ctrax assays. Importantly, our results suggest that behavioural changes and defects caused by disruption of these genes are relatively subtle, possibly due to compensatory genetic mechanisms.

** speaker*

Session 8 - Olfactory processing, Thursday Oct. 11th

Olfactory processing in the medial amygdale

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The medial nucleus of the amygdala (MeA), referred to as the vomeronasal amygdala, plays a key role in defensive and reproductive behaviour. It receives processed information from the associational cortical amygdala (CoA) as well as direct olfactory inputs from the accessory olfactory bulb (AO). Little is known about the processing of olfactory sensory information in the MeA. In this study we examined the physiological properties CoA and AO inputs to neurons in the posteroventral nucleus of the MeA (MePV). Whole cell recordings were made from neurons in acute coronal brain slices made from adult male GAD67-eGFP knock-in mice**.** Synaptic responses were evoked by electrical stimulation of the afferents in the CoA and/or in the MeA molecular layer where the axon terminals of projections from the AO are located. Cells were filled with biocytin during recording and later visualized using immunohistochemistry. Stimulation of either CoA or AO inputs evoked excitatory synaptic inputs to MeA neurones. Both CoA and AO stimulation activated dual component (AMPA/NMDA) glutamatergic synapses. In voltage clamp, AMPA-receptor mediated excitatory synaptic currents (EPSCs) evoked by stimulation of AO axons showed significantly slower kinetics as compared to those evoked by CoA stimulation. The rise times were 2.04±0.24ms vs. 0.96±0.08 ms; (n=24, $p<0.001$) and decay time constants were 10.1±1.35ms vs. 4.06±0.27ms (n=24, $p< 0.001$). Upon reconstruction of neurones, we found their dendrites extend to the molecular layer forming distal dendritic tufts. Focal pressure application of TTX (1μM) at the distal dendritic tuft that blocked the accessory olfactory synaptic input, leaving the CoA evoked responses intact (80±0.6 % block vs. 6.2±1.6% block; n=6, $p<0.0001$). Calcium imaging of the dendritic tree while stimulating the AO afferents revealed synaptically evoked calcium transients that were restricted to dendrites that extend to the molecular layer. These results show that MePV neurons receive convergent AO and CoA inputs at separate locations on their dendritic tree, with the AO inputs synapsing at the distal dendrites and the associational afferents from CoA synapsing mainly at the proximal dendrites. We suggest that MeA neurones process olfactory and cortical inputs using distinct dendritic compartments.

* *speaker*

Session 4 - Sensory-Motor Control, Wednesday Oct. 10th

A birds view: Control and precision in flight and landing

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The budgerigar, *Melopsitactus undulatus*, is a highly social parrot inhabiting the bushlands of central Australia. Birds aggregate in large flocks of up to several 100 individuals, in which they traverse an environment that can be extremely cluttered. Due to their social behaviour and their skilful flight they are the ideal model organism to investigate questions regarding organisation of flight and landing in large groups and obstacle avoidance.

In two sets of experiments we looked into (a) the potential idea that landing patterns may be controlled by biases on the population level and (b) look into the precision necessary to perform extreme manoeuvres, such as flying through narrow gaps.

Our findings suggest that biases observed during the task of landing are highly context specific, depending on the task, the individual and also on interactions with conspecifics. Our results also show that birds are highly aware of their own body size and can estimate the size of an aperture with a precision of +/- 1cm, thus allowing them to tailor their flight appropriately and traverse even very narrow gaps safely.

* *speaker*

Session 7 - Modulation, Thursday Oct. 11th

Unraveling the molecular mechanisms of short- and long-term stress-induced cognitive deficits

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Stressful live events are commonly accepted as risk factors for the development of psychiatric disorders. Social and work stress in particular is prevalent in western societies and psychopathologies emanating from such stressors result in high economic losses. Chronic stress paradigms in animal models produce a variety of behavioral, physiological and neuroendocrine changes that are related to clinical symptoms of psychiatric disorders. One major indication of these disorders is cognitive impairment, and numerous studies have investigated the interactions of stress and cognitive dysfunction at different stages in life. Early life stress, chronic stress and chronically elevated circulating glucocorticoids (GCs) have been shown to induce cognitive impairments. In contrast, acute effects of stress and GCs critically depend on the timing and magnitude of the stressor or GC exposure and the stage of memory formation, consolidation or retrieval. One major aim of our work is to identify the molecular mechanisms that lead to stress-induced cognitive deficits. In this talk, I will first present data illustrating how metabotropic glutamate receptors interacting with their intracellular partner Homer1 mediate short-term stress-induced cognitive deficits. As a second example of our work, I will show how stress early in life can affect cognition in adulthood via long-term modulation of specific synaptic cell adhesion molecules. Finally, I will present possible intervention strategies, which can rescue or prevent the stress-induced phenotype.

Session 4 - Sensory-Motor Control, Wednesday Oct. 10th

Of bees, birds and robots

Mandyam V. Srinivasan[1,2]

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2 School of Information Technology and Electrical Engineering, University of Queensland, Brisbane, Australia

Flying insects and birds are remarkably adept at seeing and perceiving the world and navigating effectively in it, despite possessing a brain that weighs less than a milligram and carries fewer than 0.01% as many neurons as ours does. This presentation will describe our recent progress in understanding how honeybees use their vision to control regulate their flight speed, avoid mid-air collisions with other flying insects, and perform smooth landings, using computational principles that are often elegant, simple, and unprecedented. It will also outline our recent progress in understanding visually guided flight in birds, and conclude with an update of our advances in the design, construction and testing of biologically inspired vision systems for autonomous aerial vehicles.

Session 8 - Olfactory processing, Thursday Oct. 11th

Memory formation and mushroom bodies in the fly brain

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Animals can adapt their behaviour to given environment according to memories of their former experiences. To address neural circuits underlying adaptive behaviour, we chose associative memory of *Drosophila melanogaster* as a model system. Flies form positive or negative memories of an odour by paired presentation of sugar reward or electric shock punishment. Over the past years, we have worked on the neuronal mechanisms that endow positive or negative values with the odour and found the important roles of neuromodulator dopamine. The value signals by dopamine converge with the odour signal in the mushroom body that consists of second-order olfactory interneurons, thereby the fly forms associative memories. Since dopamine is synthesized in ~280 neurons in the fly brain and involved also in other brain functions, it is important to identify individual responsible neurons for value signalling. I will summarize our most recent findings particularly how we identified different types of neurons for signalling positive and negative values and discuss about the subcellular modulation of synapses at the formation of odour memories.

Abstracts
Posters

Joint Poster Session - Thursday Oct. 11th

Clonal analysis of neural stem cell output in the adult mouse subependymal zone

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3 Hubrecht Institute, University Medical Center Utrecht, Netherlands

In mammals, adult neural stem cells (NSCs) reside in restricted regions in the forebrain, the subependymal zone (SEZ) along the lateral ventricles and the subgranular zone of the hippocampus. The NSC population of the SEZ generates a heterogeneous population of neurons, which are destined to the olfactory bulb circuitry. It is however unclear how spatiotemporal patterns of proliferation and differentiation at the clonal level contribute to determine the diversity and magnitude of the output from this neurogenic population. To address this crucial issue we aimed to trace the progeny of single adult NSCs of the SEZ under physiological conditions, by using genetic fate mapping based on a multicolor reporter ('confetti') and the inducible CreERT2 expressed in adult NSCs and a few other glial cell types (GLAST::CreERT2).

We first examined the frequency of expression of each of the 4 reporter genes by constitutively expressing Cre in the forebrain of ´confetti´ mice (Emx1::Cre/confetti). This revealed that cells expressing nuclear the GFP reporter are underrepresented, as already reported. We then examined the reliability of the GLAST::CreERT2 mediated recombination in stem cells and glia, but not transit-amplifying progenitors and neuroblasts, by using high doses of tamoxifen in GLAST::CreERT2/confetti mice. Lastly we titrated the tamoxifen dose to label only few clones. Proper dosing was further confirmed by each olfactory bulb containing cell groups of a single color, consistent with an origin of these cells from a single NSC. Our preliminary observations reveal surprising dynamics at the transit-amplifying stage and profound differences in the self-renewal capacity of NSCs. These data suggest that the continuous generation of neurons by the SEZ represents a population property rather than reflecting the activity of individual NSCs. Ongoing experiments aim to clarify, among other aspects, the life span and output composition of individual NSCs.

** presenter*

Joint Poster Session - Thursday Oct. 11th

Intrinsic and network properties of a highly synchronous hindbrain motor nucleus

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Synchronous, high frequency firing is basic to neuronal function, so far mainly shown at cortical and subcortical levels. Here, intracellular single cell analyses identified intrinsic and network properties determining synchronous, ultrafast-gamma firing in a hindbrain motor nucleus dedicated to vocalization in fish. Individual, neurobiotin-filled vocal motoneurons exhibited modest dendritic arbors of 3-4 main branches extending bilaterally throughout, and contained mainly within, paired midline motor nuclei. Motoneurons lacked spontaneous activity, only firing action potentials at frequencies matched to excitatory vocal pacemaker input resulting in highly synchronous motoneuron activity and a pulsatile nerve volley directly determining natural call frequency. Intracellular current injection revealed low somato-dendritic, voltage-dependent excitability with rapid accommodation of action potential firing rate. Electrotonic coupling was demonstrated in collision tests of antidromic motoneuron activation via the vocal nerve and intracellularly evoked action potentials. Action potentials showed a strong after-hyperpolarization during vocal activity, with intracellular chloride injections revealing a background membrane hyperpolarization blocked by extracellular bicuculline, a GABAA receptor antagonist. Together with immunocytochemical evidence for dense GABAergic input throughout both vocal motor nuclei, the results support prominent roles for synchronizing inhibition, synchronous excitatory input and electrotonic coupling in determining vocal oscillatory output in the ultrafast-gamma frequency range. Axonal recordings further showed differential recruitment of variably sized vocal motoneurons without disrupting motoneuron synchronicity, but accounting for natural variation in call amplitude.

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** presenter*

Joint Poster Session - Thursday Oct. 11th

DCC Functions in multiple aspects of corpus callosum development

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The corpus callosum is a large fibre tract that connects the cerebral hemispheres. Corpus callosum development commences embryonically and continues after birth with multiple mechanisms facilitating axon growth across the telencephalic midline. One molecule implicated in this process is DCC (Deleted in Colorectal cancer), a transmembrane receptor, that binds the guidance ligands Netrin1 and Draxin. DCC can also interact intracellularly with another transmembrane guidance receptor called Robo1 via their P3 and CC1 intracellular domains, respectively. Purpose: To investigate the role of the DCC P3 domain throughout callosal development using the mouse model, DCCKanga, which lacks this domain. Methods: Immunohistochemical phenotypic analysis of the DCCKanga mouse was performed at embryonic, postnatal and adult ages. The phenotype of the DCCKanga mouse was compared with the DCC knockout mouse, which is known to display defects in corpus callosum formation. The development of axonal tracts, cortical lamination and glial development were assessed in both mutants. Results: Both homozygous DCCKanga mice and DCC knockout mice display similar embryonic phenotypes, where the corpus callousm is completely absent (n . 3). DCCKanga mice also display this phenotype into adulthood (n . 3). Both mouse lines also display defects in midline glial development, telencephalic fusion and cingulate pioneering axon pathfinding, but cortical lamination occurred normally (n . 3). Conclusion: These results suggest that the DCC P3 domain is required for formation of the entire corpus callosum across embryonic and postnatal development. Furthermore, DCC appears to function in multiple aspects of callosal development, with the P3 domain essential for DCC function. Importantly, these findings provide insight into the mechanisms that underlie brain wiring and may shed light on human congenital disorders of corpus callosum formation.

** presenter*

Joint Poster Session - Thursday Oct. 11th

Receptor tyrosine phosphatase PTPRO inhibits trigeminal axon growth and branching by repressing TrkB and Ret signaling

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During development neurons need to coordinate the activities of growth factors and guidance cues in order to reach their optimal growth rates and their correct synaptic targets. Among the prominent signaling pathways, tyrosine phosphorylation appears to be particularly important. To date, receptor tyrosine kinases (RTKs) have been extensively investigated, but much less is known about receptor tyrosine phosphatases (RPTPs) and their requirements during neural development. In chick, PTP receptor type O (PTPRO) is required for motor axon outgrowth and Eph receptor-dependent retinotectal axon guidance. We have found that, in mice, PTPRO does not have the same *in vivo* functions, suggesting that chick and mouse PTPRO have different substrate specificities. Previous work demonstrated that $PTPRO^{-/-}$ mice show abnormal spinal pathfinding and decreased survival of specific trunk nociceptive neurons, but the molecular mechanism underlying the phenotypes has not been elucidated. We found that during embryonic development of the mouse trigeminal ganglion and lumbar DRGs, PTPRO is expressed in BDNF- and GDNF-sensitive mechanoceptive neurons, but not in NGF-sensitive nociceptive neurons. Consistently, primary cultures of trigeminal neurons from $PTPRO^{-/-}$ embryos were more responsive than $PTPRO^{+/+}$ neurons to BDNF and GDNF, but not NGF. *In vivo,* $PTPRO^{-/-}$ embryos showed increased arborization of the ophthalmic branch of the trigeminal nerve, and defasciculation of the maxillary branch. Additionally, we observed a loss of NGF-sensitive nociceptive neurons in $PTPRO^{-/-}$ newborn mice, possibly due to the competitive growth advantage that BDNF- and GDNF-sensitive neurons have. To address the underlying mechanism, we overexpressed PTPRO, TrkB and Ret to look at their interaction. PTPRO directly dephosphorylated TrkB and Ret and inhibits their signaling upon BDNF and GDNF stimulation. In contrast and consistent with the *in vivo* results, EphA4 phosphorylation was only reduced by the co-expressed chick isoform of PTPRO, but not with the mouse isoform of PTPRO. These results indicate that PTPRO acts as a specific negative regulator of BDNF/TrkB and GDNF/Ret signaling during mouse neural development.

** presenter*

Joint Poster Session - Thursday Oct. 11th

Motor selection and visual attention in manual grasping

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Many studies have suggested that goal-directed movements are preceded by covert shifts of visual attention to the movement target. We recently extended these approaches to investigate visual attention before grasping movements, focusing on how grasping points are chosen and on how this selection is related to the spatial deployment of attention during the preparation of grasps. Our results confirm the import role of attention also in grasp planning and provide evidence for divided attention. They also provide evidence for a dissociation of overt attention (eye movements) and covert attention: While the eyes during grasp preparation tend to fixate the centre of the object, attention is split and is focused on those locations where the fingers will touch the object. These findings are consistent with the conjecture that the planning of complex movements enacts the formation of a flexible "attentional landscape", which tags all those locations in the visual lay-out that are relevant for the impending action.

* *presenter*

Joint Poster Session - Thursday Oct. 11th

Prey capture ability in damselflies: function of ommatidia structure, viewing angle, and visual resolution

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Odonata (dragonflies and damselflies) are insects well known for their amazing visual capacities and flight control. These insects rely predominantly on vision to search among conspecifics when choosing mates for reproduction, for visual signaling during male-male competitions, and to identify prey during search for food. The mechanisms underlying prey detection and interception has been well studied in Odonata regarding behavioural, neuroethological, and physiological aspects. However, most research has focused on dragonflies, and damselflies have received comparatively less attention regarding the role of their visual abilities during prey catching. Here, we test the prey catching ability of the Australian common blue tail damselfly, *Ischnura heterosticta* using a multi-disciplinary approach. First, we measured the movements of pseudopupils of the ommatidium to determine the inter-ommatidial angles in different regions of the eyes. Based on these data we are calculating the resolution of their visual system. Secondly, *in-situ* observations of live hunting damselflies are providing data regarding the preferred approach path towards prey and preferred predation angle. Preliminary observations suggest that the frontal eye region may have better viewing angles and a higher resolution, thus being the most relevant eye region for prey detection. Finally, histological studies are used to generate a clear view of the eye structure and differentiations in each eye region. Taken together this will give first insights into the visual resolution of damselfly compound eyes via their predation angle choices.

** presenter*

Joint Poster Session - Thursday Oct. 11th

Social interaction reverses both cocaine conditioned place preference and associated brain activation patterns

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A main challenge in the therapy of drug dependent individuals is to help them reactivate interest in non-drug-associated activities. Among these activities, social interaction is doubly important because treatment adherence itself depends on it. We recently developed a rat experimental model based on the conditioned place preference (CPP) paradigm in which only four 15-min episodes of social interaction with a sex- and weight-matched male conspecific (i) reverse CPP from cocaine to social interaction and (ii) inhibit the reinstatement of cocaine CPP.

Behavioral analyses revealed that the rats already demonstrated the full amount of 'friendly'social interaction and engaged in adult forms of play from the first training session onward, spending more than 75% of the 15-min session time in direct contact with each other. The behavioral reversal was paralleled by a reversed expression of the immediate early gene Zif268: Social interaction counterconditioning reversed the cocaine CPP-associated increase of Zif268 expression in the lateral nucleus accumbens core (AcbC), the basolateral (BLA) and central (CeA) amygdala, and the ventral tegmental area (VTA), but not in the medial AcbC or the medial accumbens shell (AcbSh).

In concurrently trained animals for CPP pairing cocaine with one compartment and social interaction with the other (i.e., mutually exclusive stimulus presentation during CPP training), excitotoxic lesioning of AcbC or BLA shifted CPP toward social interaction, whereas AcbSh inactivation shifted CPP toward cocaine.

Altogether, these results support our hypothesis that different neuron ensembles in the nucleus accumbens mediate different types of reward, e.g., drug- vs "natural" reward.

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** presenter*

Joint Poster Session - Thursday Oct. 11th

Metabolic maturation of auditory brainstem neurones

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The energy a neurone has at hand determines to a great deal the firing behaviour the neurone can engage in. Therefore, neurones generally adjust their mitochondrial numbers according to their specific energy demand in a tightly regulated fashion, so that energy is neither lacking nor abundant. We studied the role of metabolic maturation of auditory brainstem neurones in the medial nucleus of the trapezoid body (MNTB), the lateral superior olive (LSO) and the medial superior olive (MSO) during the period of hearing onset and refinement of neuronal circuits in the Mongolian gerbil (Meriones unguiculatus). Using immunohistochemical and histological stainings of brain slices we show that mitochondrial density as well as cytochrome c oxidase activity is subject to an increase around the onset of hearing and the subsequent refinement period in all nuclei investigated. In particular the MNTB shows a very early upregulation of mitochondrial markers when compared to MSO and LSO. Also Na+/K+-ATPase, which is known to consume most of the adenosinetriphosphate produced by the neurone, is upregulated along with the mitochondrial density in all investigated nuclei. In addition, the observed changes in mitochondria and Na+/K+-ATPase correlate well with the final arrangement and distribution of synaptic inputs as visualised by synapsin labelling. In contrast, the expression of GLUT3, which is the most important neuronal glucose transporter, is drastically upregulated only after the onset of hearing in all nuclei investigated. The expression levels of all metabolic markers investigated saturate after the refinement period of auditory circuits. Our findings are in line with the assumption that only after the onset of hearing, when neuronal activity is functionally important, does computation of these neurones become relevant and, thus, lead to a rise in cellular metabolism.

* *presenter*

Joint Poster Session - Thursday Oct. 11th

The impact of the endocannabinoid system on sound localisation growth and branching by repressing TrkB and Ret signaling

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For sound localisation animals exploit differences in arrival time and amplitude of sound waves at both ears. These cues are computed in the auditory brainstem in the medial and lateral superior olive (MSO and LSO), respectively. Despite the required temporal precision, dynamic changes induced by neuromodulators are of great importance in this system.

We studied the role of the endocannabinoid system in the MSO of the Mongolian gerbil using immunohistochemical stainings and patch-clamp recordings from neurones in acute brain slices.

Immunohistochemically, we found a predominantly presynaptic localisation of CB1 during the period of hearing onset, i.e. P10-P15. This distribution completely reverted during late postnatal development to almost exclusively postsynaptic localisation of CB1. In addition, a glial subpopulation expresses high amounts of CB1. The endocannabinoid-synthesising enzymes Diacylglycerol lipase α/β were localised to the soma of postsynaptic cells at all developmental stages tested.

In accordance with immunohistochemical results, depolarisation-induced suppression of inhibition and excitation were successfully elicited between P10-P15. In animals older than P20 physiological evidence for presynaptically located CB1 receptors could not be found, however a CB1-dependent hyperpolarising effect on the resting membrane potential by endocannabinoids was found. Voltage-clamp recordings suggest that an increased K^+ conductance underlies this hyperpolarisation. In addition, we could show that endocannabinoids modulate glycinergic currents by directly binding to postsynaptic glycine receptors.

Our results suggest that the endocannabinoid system plays an important role in the physiology of auditory neurones. In animals aged P10-P15 over-excitation of these neurones might suppress CB1 expressing inputs retrogradely, whereas in older animals endocannabinoids seem to adjust the temporal tuning of these neurones by postsynaptic mechanisms.

** presenter*

Joint Poster Session - Thursday Oct. 11th

Spatial behavior of mongolian gerbils in complex virtual environments

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Virtual reality (VR) environments are increasingly used to study spatial behavior in rodents. In many approaches so far the design of the environments did not go beyond simple open fields or linear tracks. How to make rodents learn to navigate in more complex virtual environments is hardly investigated. We use a VR setup with a spherical treadmill that is surrounded by a 360° toroidal screen onto which the virtual environment is projected. This design permits a fixation of the animal such that it can freely rotate around its vertical body axis. We trained gerbils (Meriones unguiculatus) to perform spatial tasks in virtual mazes of different complexity. First the animals learned to run back and forth between the two ends of a virtual linear track. At the ends they received a food reward. They had to learn to orient with only visual information, not to collide with the virtual walls and optimize the path from one reward to the next. Performance increased within about ten training sessions to almost optimal values: The animals avoided virtual walls and ran straight paths from one track end to the other. Running speed was consistent with what is typically observed in real environments. When afterwards presenting more complex mazes such as U- or 8-shaped arenas the animals were able to transfer their previously acquired skills to the new tasks after only a few trials. We furthermore implemented two-alternative choice tasks using a virtual y-maze were animals were required to run to the end of one arm depending on their decision.

* *presenter*

Joint Poster Session - Thursday Oct. 11th

EphA4 marks a population of spinal cord dorsal interneurons required for left-right coordination of locomotion

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Mice lacking the EphA4 receptor tyrosine kinase show a remarkable motor dysfunction, they walk with synchronous movements of the hindlimbs producing a hopping gait. Previous work in isolated spinal cords points to an intrinsic spinal cord defect, suggesting that EphA4 is important for the development of the circuitry of central pattern generators (CPGs) involved in left-right alternation. EphrinB3 ligand serves as a midline repulsive cue, preventing EphA4-positive ipsilateral neurons to aberrantly cross the midline. It was suggested that in the absence of EphA4 excitatory neurons form ectopic connections on the contralateral spinal cord, leading to synchronous activity of the CPGs, and the hopping gait.

The identity of EphA4 positive interneurons responsible for the synchronous gait is not known to date. In order to characterize the EphA4 circuit controlling locomotion, we have undertaken a conditional KO approach to ablate EphA4 in specific spinal cord neuronal populations. Removing EphA4 from the whole spinal cord (with HoxB1-Cre) phenocopies the hopping gait phenotype seen in EphA4 null mice, confirming the involvement of local spinal CPGs in the abnormal locomotive behavior. Interestingly, removal of EphA4 from the dorsal spinal cord (with Pax7-Cre), or from the more restricted dI4-6 population (with Lbx1-Cre) results in a partial loss of left-right limb alternation. These results suggest that a previously uncharacterized class of dorsal interneurons either integrate the synchrony/alternation circuit of CPGs or modulate CPG function. In addition we confirm that EphA4 has a cell-autonomous role in corticospinal tract (CST) axon guidance (with Emx1-Cre). Further, we propose that EphA4 contributes to the development of CST projections through a non-cell autonomous mechanism. By preserving the midline, EphA4 affects supraspinal and sensory axonal tract projections. We find that EphA4 dorsal spinal cord KO mice present a shallow dorsal funiculus, and aberrant projections of CST and nociceptive axons across the midline. In summary, our work suggests the involvement of dorsal interneurons in tuning CPG activity and instructing developing CST axons.

** presenter*

Joint Poster Session - Thursday Oct. 11th

Amplification of progenitors in the mammalian telencephalon includes a novel radial glia cell type

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To investigate the mechanisms regulating expansion of neuron numbers during development we examined the mouse ventral telencephalon, which is characterized by a large subventricular zone (SVZ). Utilizing long-term imaging of progenitor cells labelled in embryonic brain slices (embryonic day 14) and immunohistochemistry, we discovered novel subapically dividing radial glia that contribute pivotally to the extensive progenitor amplification generating this expanded proliferative zone. This amplification of progenitor cells initiates already within the ventricular zone (VZ) and coincides with a shortened cell cycle length in the intermediate progenitor population. Importantly, subapical radial glia are found in increasing numbers in mammalian cortices with increased gyrification, like the ferret and the sheep, implying key roles in ontogeny and phylogeny.

* *presenter*

Joint Poster Session - Thursday Oct. 11th

Pop-out visual search mechanisms: Not all display densities are made equal

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In visual search tasks participants select the task-relevant item among several distractors and report some of the target's features, e.g., its shape: recorded reaction times (RTs) reflect durations of target selection processes and of post-selective processes. When the target is a singleton, e.g., red target and green distractors, RTs are fast and do not increase as the number of distractors, or display density, increases: the singleton "pops out" of display and is always the first selected item. Present study investigated whether or not singletons indeed pop-out in displays of different densities, varied by increasing the number of distractors in a fixed presentation area. Participants performed a search task with a color singleton embedded in either *sparse* or *dense displays*. Prior to display onset, a spatial cue, announcing the location of the upcoming singleton target, was briefly presented on half of the trials: as on cued trials no target selection processes were necessary, the RT difference between *un-cued* and *cued* trials indexed the duration of target selection (T_{sel}). Distributional analyses showed T_{sel} to be constant in dense displays while T_{sel} for sparse displays varied considerably. Dissociation between sparse and dense displays suggests different selection mechanisms to operate in different densities, a finding which cannot be accommodated by the dominant theories of spatial attentional selection.

** presenter*

Joint Poster Session - Thursday Oct. 11th

Eph, Ephrin and Ephexin instruct dendritic patterns of specific olfactory projection neurons in fly

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The olfactory system of Drosophila offers an excellent opportunity to study the molecular mechanisms of neuronal wiring specificity. To date less than a handful of guidance receptors have been identified in Drosophila to play a role in ORN (olfactory receptor neuron) connectivity. Examples include trans-membrane molecules Dscam (Hummel, Vasconcelos et al. 2003), Plexin (Lattemann, Zierau et al. 2007; Sweeney, Couto et al. 2007), and Ncadherin (Hummel and Zipursky 2004). Thus, the field of molecular mining to search for the components of the so called combinatorial cell-surface code that instructs discrete olfactory map formation is wide open. A class of molecules known to be involved in axon guidance in different nervous systems is the Eph receptor tyrosine kinase family members and their ligands, the ephrins (Eph receptor interacting proteins). The activities of Rho-GTPases are important for re-arranging the actin cytoskeleton during axon path finding downstream of Eph and ephrin signaling. Ephexin acts as a Rho specific guanine nucleotide exchange factor (GEF) during growth cone retraction. It was recently shown that Drosophila Ephexin is important in maintaining synapse homeostasis and morphology. Eph/ephrin signaling is known to be involved in the optic system for guiding retinal projections via a continuous gradient. We addressed whether Eph, ephrin and/or Ephexin play a role in the rather dis-continuous and non-graded olfactory system. Here, olfactory receptor neurons (ORNs) axons are sorted by bundling together to target properly. A repulsive cue (like Eph/ephrin signaling) could play a role in the process of sorting. We analyzed previously published mutants of the Eph receptor, ephrin ligands (Boyle, Nighorn et al. 2006) and Ephexin. Although the study is still under progress, we found that these molecules are involved in the correct targeting of the ORNs. Also we could show through rescue and MARCM experiments, that it's the projection neurons (PNs) that require these molecules and then ORNs follow their PN partners and mis-target. The requirement of these molecules for wiring specificity is restricted to certain ORN classes. All these molecules seem to be expressed during crucial stages of development suggesting their potential role. From the evidences we have until now, our study indicates an involvement of these molecules in olfactory targeting of Drosophila in a very specific manner.

Joint Poster Session - Thursday Oct. 11th

Behavioral and neurobiological effects of deep brain stimulation in a mouse model of high anxiety- and depression-like behavior

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Recent evidence suggests that high-frequency deep brain stimulation of the nucleus accumbens (NAcb-DBS) may represent a novel therapeutic strategy for individuals suffering from treatment-resistant depression although underlying mechanism of action remains largely unknown. Using a unique psychopathological mouse model of enhanced depression- and anxiety-like behavior (HAB) we investigated behavioral and neurobiological effects of NAcb-DBS. HAB mice underwent either chronic treatment with selective-serotonin-reuptake-inhibitors (SSRIs) or stereotactic surgery to implant DBS electrodes into the NAcb. NAcb-DBS was applied for 1h daily for seven consecutive days (130Hz, 100µA, 60µs pulse width) and sham-stimulated animals were used as controls. Anxiety- and depression-related behaviors were assessed using established tests with predictive anxiolytic or antidepressant validity. Furthermore, the effects of NAcb-DBS on challenge-induced immediate-early-gene expression and hippocampal neurogenesis were investigated.

The enhanced depression-like behavior of HAB mice was not influenced by chronic SSRI-treatment. In contrast, repeated, but not single, NAcb-DBS induced robust antidepressant and anxiolytic responses in HAB animals while these behaviors remained unaffected in normal depression/anxiety animals (NAB), suggesting a preferential effect of NAcb-DBS on pathophysiologically deranged systems. NAcb-DBS caused a modulation of challenge-induced activity in brain regions implicated in stress and depression, including an increase in c-Fos expression in the dentate gyrus and enhanced the blunted hippocampal neurogenesis in HABs.

Taken together we show that the normalization of pathophysiologically enhanced, SSRI-insensitive depression-like behavior by repeated NAcb-DBS was associated with reversal of reported aberrant brain activity and rescue of impaired adult neurogenesis in HAB mice, indicating that NAcb-DBS affects neuronal activity and plasticity in a defined, mood-associated network. Finally, it is suggested that SSRI-insensitive HAB mice represent a clinically relevant model for elucidating the neurobiological correlates underlying the observed behavioral effects of NAcb-DBS.

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** presenter*

Joint Poster Session - Thursday Oct. 11th

Combining the senses: Looking at the interaction of wind and vision on the honeybees' streamlining response

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In their flight through the world, honeybees are exposed to complex combinations of sensory cues. For example, the bee perceives at least two sources of velocity information -- derived from optic flow, and from air flow -- that it can use to control its flight. As these cues can even conflict if a gust of wind blows over the insect, reliable integration of information from the two senses poses a challenge.

To investigate how honeybees combine information from vision and air flow to control flight, we used a virtual reality flight simulator in which tethered honey bees were exposed to controlled combinations of moving visual stimulus, and air flow. The insect's abdomen angle was used as an indicator of its perception of the strength of the combined stimuli. In the absence of wind, honeybees actively raise their abdomens as the rate of optic flow simulating forward flight increases, apparently attempting to streamline the body's posture to reduce energy consumption. Optic flow is required to elicit sustained flight in tethered bees. With only wind stimulation, bees are not guaranteed to fly, and are unlikely to hold a stable abdomen position.

When exposed to optic flow and wind concurrently, wind speed was generally found to combine additively with optic flow, resulting in higher abdomen positions as wind speed increased, until a plateau was reached at a wind speed of around 3 m/s. The wind-evoked component of the response would aid the honeybee to further reduce energy consumption for flight through streamlining. Experiments with freshly deceased bees show that the wind-evoked raising of the abdomen is mediated by sensory mechanisms, and unlikely to be caused by passive, drag-induced lift. However, the curve of abdomen pitch versus wind speed (for any given optic flow) is complex, showing a minimum, at around 1.5 m/s, flanked by two maxima (at 0.5 and 3 m/s), suggesting that this response may serve additional purposes to streamlining.

Honeybees with amputated antennae or immobilized Johnston's organs do not modulate their abdomen angle in response to air flow. These bees displayed an elevated response at lower wind speeds, and no minimum at 1.5 m/s. Despite the apparent additive nature of wind and optic flow on the response at high wind speeds in normal bees, this finding indicates that feedback from the antenna acts to inhibit the streamlining response at low wind speeds.
Surprisingly, although wind results in the abdomen being raised at high wind speeds, it actually inhibits the response at low wind speeds. These findings reveal the importance of exposing tethered insects to the broad range of sensory modalities that they normally experience during flight, as multiple sensory inputs may act to regulate even seemingly straight-forward responses, such as streamlining.

** presenter*

Joint Poster Session - Thursday Oct. 11th

Echolocation vs. echo suppression: Influence of the precedence effect on the human-sonar localization of reflective surfaces

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Echolocation is an effective perception-action ability of humans, especially used by blind people. However, the precedence effect predicts a conflict of echo analysis and echo suppression: when localizing sound sources, the human auditory system suppresses spatial information of echoes, but just this information underlies effective echolocation.

A common approach to investigate the precedence effect is the arrangement of two sound sources presenting a direct sound (lead) and a delayed copy (lag). Several experiments on lag-discrimination suppression have quantified the deterioration of spatial information of the lag produced by the lead.

This study investigates the interaction of echolocation and precedence effect in terms of discrimination suppression. Sighted subjects performed two experiments of an azimuth-discrimination experiment in virtual acoustic space: In the 'Listening' experiment, subjects had to discriminate between positions of a single sound source, the leading, or the lagging of two sources, respectively. In the 'Echolocation' experiment, the sound sources were replaced by sound reflectors. Here, subjects evaluated echoes generated in real-time from self-produced vocalizations and thereby discriminated between positions of a single reflector, the leading, or the lagging of two reflectors, respectively.

Our results show that sighted subjects can learn to discriminate reflective surfaces echo-acoustically with accuracy comparable to sound-source-discrimination. In the Listening experiment, the presence of a lagging source impaired lead-discrimination only slightly by a factor of 1.60, while a leading source impaired lag-discrimination considerably by a factor of 8.58. The asymmetry between lead- and lag-discrimination shows strong influence of the precedence effect, which facilitates localization of the lead at the expense of the lag. In the Echolocation experiment, however, this asymmetry was significantly weaker: lead- and lag-discrimination deteriorated by a factor of 4.75 and 6.06, respectively. These data indicate that the precedence effect is weakened in an echolocation context.

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