Abstract Booklet

Young Swiss Society for Neuroscience

4th Annual Early-Career
Researchers Symposium 2022

"Ontogeny of Cognition and Gender Bias in Neuroscience Research"

University of Fribourg
Bd de Pérolles 90, 1700 Fribourg
PER21, C120
June 10, 2022

How to get to the University of Fribourg, Bâtiment PER 21 and 22?

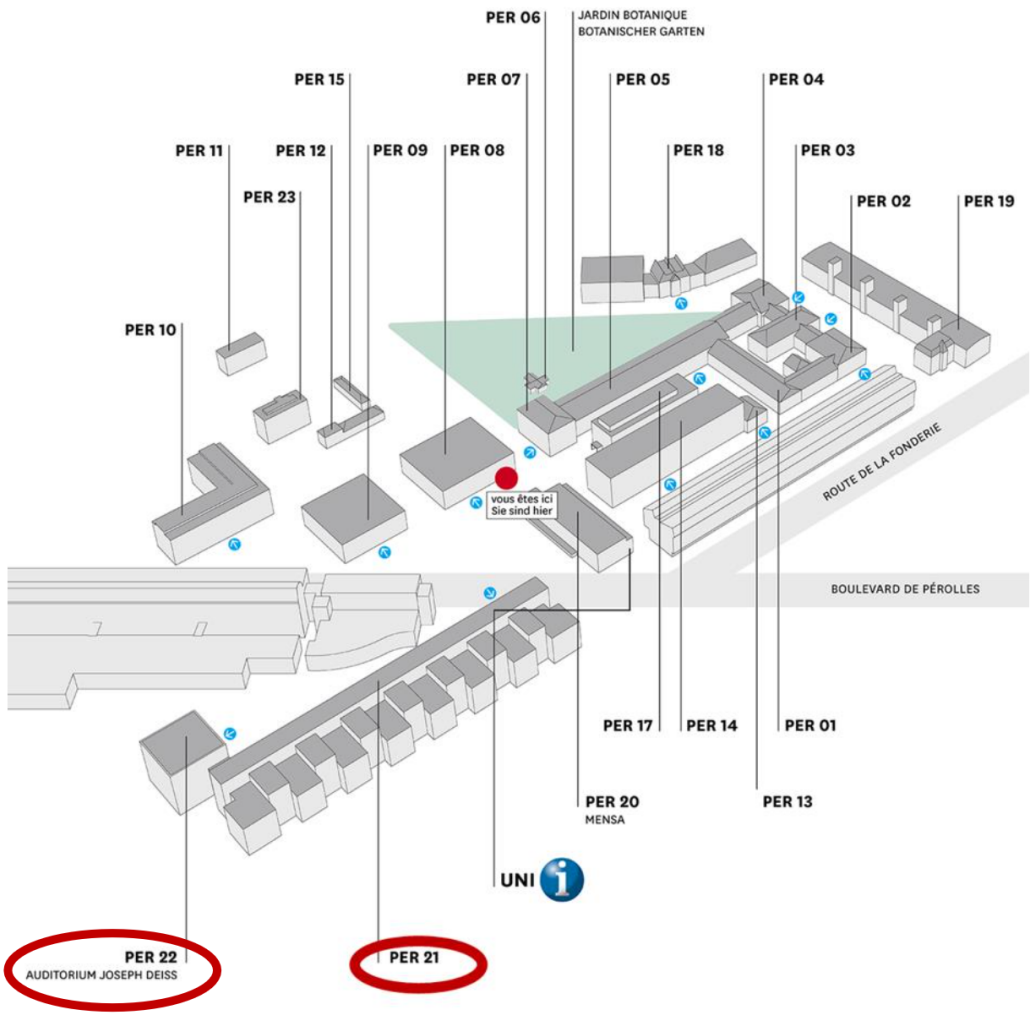
The meeting will take place at the *University of Fribourg, Building PER21& PER22 Boulevard de Pérolles 90, Fribourg*

You can reach it by a 16 min **walk** from the train station

With the **bus**: BUS1(direction: Marly) which stops directly in front of the University (Stop: Plateau de Pérolles)

The **entrance** to the ySSN Registration desk is in the building PER22.

Map of the Campus



Map of the PER21 building

Programme

08:00	**Check-In and Welcome Coffee.** *Entrance from PER22.*
08:25	**Opening Remarks** - *Dr. Maria Reva, ySSN President*
08:30	**Keynote Lecture - *Dr. Flavio Donato*** *chair: Kristina Slabeva* “How do you build a cognitive map? Assembling circuits to navigate through space and create memories”
09:30	**Talks - Session 1 -** *chair: Dr. Maria Reva* “Visual and tactile integration of object location in the mouse cortex” - *Adrian Hoffmann* “Sleep-dependent modulation of somatosensation in the Anterior Cingulate and Somatosensory Cortices in mice” - *Raquel Adaia Sandoval Ortega* “Modulation of brain circuits for sensory processing during sleep states” - *Ida Boccalaro* “Introducing principles of synaptic integration in the optimization of deep neural networks” - *Giorgia Dellaferrera*
10:30	**Coffee Break**
10:50	**Talks - Session 2 -** *chair: Dr. Samy Rima* “Motor control in the human brain. The neural signature of the sense of agency revealed by EEG in healthy subjects” - *Dr. Giuseppe Angelo Zito* “Differences in sleep neurophysiology between adolescent girls and boys: a longitudinal high-density sleep EEG study” - *Dr. Andjela Markovic* “Electrophysiological exploration of functional vision restoration in blind retinas treated with ON-bipolar-cell-specific optogenetic gene therapy” - *Jakub Kralik* “Associations between brain age, body composition, and reproductive span, in 8,878 post-menopausal females” - *Louise Schindler*
11:50	**Poster Session 1 and Lunch**

13:10	**Sponsored Talk by Roche - *Dr. Markus Britschgi*** *chair: Dr. Maria Reva* *“* Insights into scientific career paths in the pharma industry: examples, opportunities and how to go about it”
13:30	**Keynote Lecture - *Dr. Maria Teresa Ferretti*** *chair: Dr. Alberto Antonietti* “Sex and gender differences in brain and mental diseases - the gateway to precision medicine”
14:30	**Talks - Session 3 -** *chair: Pedro Espinosa* “The role of autophagy in parvalbumin-expressing neurons” - *Theodora Chalatsi* “Activation of basal forebrain parvalbumin neurons exerts frequency dependent effects on auditory processing and behavior in the rat” - *Hamid Azimi* “Paradoxical somatodendritic decoupling supports cortical plasticity during REM sleep” - *Dr. Mattia Aime*
15:30	**Poster Session 2 and Coffee**
16:40	**Best Presentation and Best Poster Awards -** *Dr. Samy Sima* **Closing Remarks -** *Dr. Maria Reva*
17:00	**Film**: “Cinq Nouvelles Du Cerveau”, *Directed by Jean-Stephane Bron*
19:00	**Apero**, Hosted by ySSN @ Café du Belvédère

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Poster Sessions

Poster Session 1 - *11:50-13:30*

- “Local slow-wave activity in regular sleep reveals individual risk preferences” - *Mirjam Studler*
- “Real-time fMRI neurofeedback improves pattern separation in healthy aging and mild cognitive impairment” - *Dr. Katharina Klink*
- “Converting thoughts into actions: restoring communication between the motor cortex and the lumbosacral region of the spinal cord using a Brain-spine-interface in an individual with chronic tetraplegia” - *Andrea Galvez Solano*
- “Retrosplenial Cortex activity: a hub in a paradoxical sleep network?” - *Micaela Borsa*
- “Impact of long-term and short-term exposure of environmental enrichment on pain-related depression in adolescent mice” - *Marta Falkowska*
- “A contextual neuronal activity of ventral hippocampus integrating reward and aversive memories” - *Joana Duarte*
- *“*Inhibiting the Anterior Cingulate Cortex to modulate behavioral responses to pain” - *Margot Renard*

Poster Session 2 - *15:30-16:40*

- “Probing cortical excitability and seizure resilience under GABAergic modulation” - *Gregory Lepeu*
- “The role of the Melanin-Concentrating Hormone (MCH) neurons in increased REM sleep propensity and cataplexy in narcolepsy” - *Bianca Viberti*
- “A hardware-software co-design approach to minimize the use of memory resources in multi-core neuromorphic processors” - *Vanessa Leite*
- “Potassium channels in retinal interneurons: impact on healthy retina, degenerated retina and vision restorative approaches” - *Giulia Schilardi*
- “Resolving decision-making during emotional conflicts by ventral hippocampal circuits” - *Carlo Cerquetella*
- “Functional dissociation of ventral hippocampal inhibitory circuits during anxiety and fear behaviors” - *Kaizhen Li*

Visual and tactile integration of object location in the mouse cortex

Adrian Hoffmann (1,2), Fritjof Helmchen (1,2)

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For a stable and coherent perception of the world, the brain combines sensory information from different modalities using distinct reference frames. In the mouse brain, tactile information from the snout's vibrissae passes through the thalamus to the somatotopically organized primary whisker somatosensory cortex (wS1) while the primary visual cortex (V1) contains a retinotopic map of the visual field. For nearby objects, these two sensory streams likely converge in the rostro-lateral (RL) area of the posterior parietal cortex. How such converging multisensory inputs are integrated on a single-cell level, especially for naturalistic stimuli, remains unclear.

Here, we investigated how neurons in mouse wS1, V1, and RL integrate visuotactile information about a pole in reach of the whiskers that can be seen and/or felt. Using two-photon calcium imaging, we recorded several thousand neurons in L2/3 of head-fixed mice while tracking whisker-pole interactions. Simultaneously, we recorded neural spikes and local field potentials from the primary and secondary thalamus (VPM, PO) by chronically implanting a 64-channel linear array of flexible electrodes.

By comparing average activities at different pole locations, we find that subsets of neurons in RL and wS1 show selectivity for specific locations in the near space based on tactile and/or visual input. Using Generalized Linear Models, we find neural activity in RL better predicted by object locations than by whisker kinematics, whereas the opposite holds for wS1. Ongoing work explores how artificial neural networks can be used to explain this transformation of whisker to head coordinates, as well as to predict the influence of thalamic activity on cortical neurons. Together, these findings corroborate the notion of RL representing object locations based on visual and tactile information, potentially in a more modality-invariant manner.

Sleep-dependent Modulation of Somatosensation in the Anterior Cingulate and Somatosensory Cortices in Mice

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Sensory disconnection from the environment is a hallmark of sleep. Yet, disengagement is not absolute as auditory and visual information can reach the cortex and be processed during sleep. Somatosensation is a crucial sensory modality for survival. Interestingly, dysregulation in the somatosensory system, such as in chronic pain, impairs sleep and sleep impairments modulate somatosensation. However, despite this bidirectional interaction, the somatosensation processing during sleep has not been comprehensively characterized yet.

To evaluate the effect of sleep on somatosensation, we simultaneously recorded local field potential (LFP) in the anterior cingulate cortex (ACC; affective component of pain) and the somatosensory cortex (S1; sensory component of pain) in mice while animals received painful and non-painful stimuli to the hind paws throughout their sleep cycle. We hypothesized that sleep modulates neural processing of painful stimuli.

The event-related potentials (ERPs) revealed a differential information flow to the cortex in NREM sleep and Wake. In addition, ERPs showed the presence of early and late responses even in the absence of a behavioral response. Interestingly, the presence or absence of a behavioral response can be determined by the spectral properties of the ERP in NREM sleep, but not in Wake. Furthermore, the phase and the power of slow frequencies (<10 Hz) at the time of the stimulation onset predicts the presence and type of evoked behavior.

In summary, here we show for the first time in naturally sleeping animals that painful and non-painful somatosensation reach both the ACC and S1 during sleep. Additionally, the late tonic responses during sleep suggest the presence of sustained integration of the affective and sensory processing of pain.

Modulation of brain circuits for sensory processing during sleep states

Luisa Boccalaro1, Florence Aellen2, Thomas Rusterholz1, Ivan Bozic1, Athina Tzovara2, Antoine R. Adamantidis1

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Sleep is associated with a sensory disconnection from the environment, thought to be mediated by a thalamic gating of sensory-motor processing. However, recent studies suggested that the gate is located downstream of thalamic relays (Issa and Wang 2008), yet the underlying mechanisms remain unclear. Here, we investigated the thalamo-cortical circuit dynamics upon auditory stimulation across sleep states (i.e. wakefulness, NREM, and REM sleep) using single-unit activity and local field potential (LFP) activities recorded from chronically implanted electrodes in the primary auditory cortex (Au1), the central medial thalamus (CMT), the medial geniculate (MG), and hippocampus (HP) in freely-moving wild-type mice. We found that auditory stimuli-evoked LFP responses during wakefulness and REM sleep were similar whereas their amplitude was significantly higher during NREM sleep. Furthermore, we investigated the modulation of auditory-evoked awakenings by SW activity (slow waves and spindles) and neural activity. Our results showed that animals significantly wake up from NREM when the stimuli were delivered on the negative-to-positive slope of CMT slow waves when the CMT neurons are likely hyper-excitable, suggesting a key role of the CMT in mediating sensory-evoked arousals confirmed by an optogenetic silencing and a computational approach. Finally, to test whether the information associated with auditory cues – i.e., danger (conditional stimuli, CS+) versus safety (CS-) – is differentially processed during sleep, we performed an auditory cued fear conditioning followed by re-exposure to the CS+ and CS- cues during subsequent sleep. Our results showed an increase in the percentage of awakening from the CS+ after the conditioning, suggesting that the discriminative ability persisted during NREM, but not during REM. Taken together, our results showed that the effect of environmental auditory cues to induce awakening, in particular those associated with danger, strongly depends on the ongoing brain state, in particular the presence of slow waves (SWs) and spindle, and the activity of CMT neurons.

Introducing principles of synaptic integration in the optimization of deep neural networks

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3 Axelera AI

Plasticity circuits in the brain are known to be influenced by the distribution of the synaptic weights through the mechanisms of synaptic integration and local regulation of synaptic strength. However, the complex interplay of stimulation-dependent plasticity with local learning signals is disregarded by most of the artificial neural network training algorithms devised so far. Here, we propose a novel biologically inspired optimizer for artificial and spiking neural networks that incorporates key principles of synaptic plasticity observed in cortical dendrites: GRAPES (Group Responsibility for Adjusting the Propagation of Error Signals). GRAPES implements a weight-distribution-dependent modulation of the error signal at each node of the network. We show that this biologically inspired mechanism leads to a substantial improvement of the performance of artificial and spiking networks with feedforward, convolutional, and recurrent architectures, it mitigates catastrophic forgetting, and it is optimally suited for dedicated hardware implementations. Overall, our work indicates that reconciling neurophysiology insights with machine intelligence is key to boosting the performance of neural networks.

Motor control in the human brain. The neural signature of the sense of agency revealed by EEG in healthy subjects

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* these authors contributed equally to the work

"Background
The sense of agency (SoA) is a fundamental aspect of motor control that allows us to consciously own our motor actions. Neuroimaging studies on the SoA have shown an involvement of frontal and parietal areas, as well as premotor and subcortical structures, in response to various aspects of agency processing. However, the specific neural correlates of the SoA are still object of debate.
We performed a behavioural experiment with simultaneous electroencephalography (EEG) recording to test the hypothesis that such neural correlates are proportional to the conscious experience of motor control, i.e., the higher the agency, the stronger the neural response from specific brain regions.
Methods
Twenty healthy volunteers performed a motor task while we recorded their brain activity with EEG. In the task, participants controlled a cursor on a screen to catch some moving targets. The visual feedback was artificially manipulated, so that the cursor responded with different degrees of disrupted movements. Participants judged how well they could control the cursor on a continuous bar (judgement of agency – JoA).
We implemented a mixed-model regression analysis to estimate whether the variability in the EEG power was explained by JoA and performance on the task, respectively.
Results
We identified distinct frequency bands in the central areas in response to JoA and performance, respectively. JoA negatively correlated with EEG power in the theta band (4 – 8 Hz), and positively in the upper beta band (19 – 27 Hz). Performance positively correlated with EEG power in the frequency range 10 – 21 Hz.
Conclusions
The theta band is associated to various aspects of cognition and behaviour, including spatial navigation, and may represent a neural signature of the SoA, decreasing in power with increasing agency. Increased beta power with the increasing agency may encode higher concentration on motor planning as a consequence of a disrupted control.
These results are relevant, as we are currently investigating the modulatory properties of the SoA by means of real-time neurofeedback. In particular, we will test whether patients affected by abnormal SoA can learn to self-regulate their motor control by modulating their theta activity, and thus improve their perception of agency. "

Differences in Sleep Neurophysiology between Adolescent Girls and Boys: A Longitudinal High-Density Sleep EEG Study

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Gender differences in sleep neurophysiology have long been described in adults. The age at which these differences emerge remains unknown. In this study, we compared 58-channel sleep EEG power, coherence and spindle activity between 31 girls (mean age = 12.13; SD = 1.67) and 30 boys (mean age = 12.83; SD = 0.75) between 9 and 14 years of age in a longitudinal design with two time points 6 months apart. We found greater spindle activity in girls as compared to boys reflected in higher non-rapid eye movement (NREM) sleep sigma (11–16 Hz) power as well as spindle amplitude, frequency and density across widespread brain regions. Furthermore, sleep EEG power between 16.2 and 44 Hz in both NREM and REM sleep was larger in girls in comparison to boys. Finally, girls also demonstrated greater sleep EEG coherence, a measure of functional brain connectivity, across frequencies (1–44 Hz) and sleep states (i.e., NREM and REM sleep) with the exception of the alpha (8–10.8 Hz) band where coherence did not differ (REM sleep) or was higher (NREM sleep) in boys as compared to girls. Taken together, our study shows robust gender differences in several measures of the adolescent sleep EEG as demonstrated through large effect sizes that persist when controlling for age and pubertal effects. Increased sleep spindle activity in girls can most likely be attributed to gender differences in the thalamocortical circuit, the brain network that generates sleep spindles. This notion is further supported by the fact that the differences in coherence in our study were most pronounced in the delta and sigma bands, two frequency ranges associated with thalamocortical activity. Given the importance of the thalamocortical network for brain function and information processing as well as its involvement in several brain disorders, our findings have broad implications for cognition and mental health. Finally, our observations are in line with similar studies in adults suggesting that such gender differences prevail over the life course. Our study thus adds to recent efforts to advocate for taking gender into account when designing and interpreting studies in the field of neurosciences.

Electrophysiological exploration of functional vision restoration in blind retinas treated with ON-bipolar-cell-specific optogenetic gene therapy

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1 University of Bern, Department of Physiology, Translational optogenetics group

Approximately 2.4 million people suffer from heredetery retinitis pigmentosa (RP) and 3.8 million from multi-factorial age-related macular degeneration (AMD), both being currently untreatable causes of progressive photoreceptor degeneration. Nevertheless, even in advanced stages of retinal degeneration, when the retina is essentially photoreceptor-less, treatment strategies such as optogenetic gene therapy can be used to transform remaining retinal cells into artificial light sensors.

Here we report the success of an ON-bipolar cell targeted AAV optogenetic gene therapy restorative approach. Bipolar cells undoubtedly represent an attractive target given the potential to restore inner retinal processing – which is impossible to achieve via retinal ganglion cell targeted therapy. Moreover, we use a designer chimeric opsin Opto-mGluR6 that couples directly into the signalling cascade of ON-bipolar cells.

We here evaluate the functional outcome of an Opto-mGluR6 gene therapy at the retinal and cortical levels using a variety of electrophysiological techniques. We evaluate general response properties, also in the light of progressive retinal degeneration, confirming longevity and independence of intervention timepoint of the therapy.

Associations between brain age, body composition, and reproductive span, in 8,878 post-menopausal females

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7 Western Norway University of Applied Sciences, Department of Health and Functioning
8 AMRA Medical AB
9 Linköping University, Department of Health, Medicine and Caring Sciences
10 Linköping University, Center for Medical Image Science and Visualization (CMIV)
11 University of Oxford, Department of Psychiatry
12 University of Oxford, Wellcome Centre for Integrative Neuroscience
13 Max Planck Institute for Human Cognitive and Brain Sciences, Department of Neurology
14 University of Oslo, NG Jebsen Centre for Neurodevelopmental Disorders

Background: The menopause transition involves increased risk of obesity and metabolic diseases, which may contribute to the observed post-menopausal risk for neurodegeneration and dementia. Although higher levels of adipose tissue may involve health risks, body fat acts as a source of oestrogen post-menopause, potentially protecting against neural decline. Therefore, it is unclear whether certain types of adipose tissue may be more or less detrimental to brain health in postmenopausal females. This may also depend on lifetime oestrogen exposure, which is shown to have lasting effects on body composition and brain structure even after menopause. These relationships between oestrogen exposure, body composition, and brain health in females are complex and largely unexplored.
Methods: Using the UK Biobank sample, we investigated associations between different types of adipose tissue and brain characteristics in 8,878 post-menopausal females, and assessed whether the relationships varied depending on length of reproductive span (age at menarche until age at menopause). Adipose tissue included visceral adipose tissue (VAT) and abdominal subcutaneous adipose tissue (ASAT) measured using body MRI. Age prediction models were run using T1 and diffusion-weighted imaging data to estimate grey matter (GM) and white matter (WM) brain age gap (BAG), and white matter hyperintensity (WMH) volumes were extracted based on T2-weighted FLAIR images. Bayesian multiple linear models, adjusting for age, were run to assess relationships between the brain measures and VAT, ASAT, and reproductive span.
Results: The results showed that higher VAT and ASAT were both associated with higher GM and WM BAG, and higher WMH volume. A shorter reproductive span was weakly associated with higher GM and WM BAG, and higher WMH volume. The relationships between VAT/ASAT and the brain measures varied positively with reproductive span, indicating that higher levels of adipose tissue may be less detrimental in females with a shorter reproductive span. These results could not be explained by common genetic variance or relevant confounders.
Conclusions: The findings indicate that associations between body fat and brain health in females may partly depend on individual differences in cumulative oestrogen exposure, emphasising the complexity of neural and endocrine ageing processes in females.

The role of autophagy in parvalbumin-expressing neurons

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Macroautophagy is a highly conserved cellular recycling pathway that sequesters cellular constituents in autophagic vesicles (AVs) and delivers them to the lysosome for degradation. Previous studies demonstrated that ablation of core autophagy genes, such as atg5 or atg7, in pallial progenitors compromises the survival of their glutamatergic neuron progeny, suggesting that autophagy may be required for neuronal survival. Here, we examine the effects of atg5 conditional ablation in neurons expressing parvalbumin (PV-atg5KO), which in the forebrain mainly represent fast-spiking inhibitory neurons that provide peri-somatic inhibition onto the principal pyramidal cells. Contrary to the prevailing view, we show that autophagy-deficient PV neurons survive throughout the brain, with the exception of Purkinje neurons in the cerebellum, which rapidly degenerate. However, proteomic analysis of forebrain PV neurons indicated that autophagy deficiency leads to aberrant proteostasis of synaptic and other proteins. Consistently, in the hippocampus, PV-interneurons show altered synaptic properties and the PV-atg5KO animals exhibit memory deficits. Our findings reveal a neuronal type-specific vulnerability to autophagy deficiency, while indicating a role of autophagy for the proper function of PV interneurons.

Activation of basal forebrain parvalbumin neurons exerts frequency dependent effects on auditory processing and behavior in the rat

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The basal forebrain (BF) has been implicated in attention and the modulation of information processing. While the BF provides the main cholinergic input to the cerebral cortex, it also harbors substantial GABAergic populations. Here we study how activation of the GABAergic population in the posterior nucleus basalis (pNB) of the BF impacts auditory pathway activity and behavior. We utilized a PV-Cre rat line to express channel rhodopsin (ChR2) uniquely in PV GABAergic neurons and recorded LFP and single unit activity simultaneously in the pNB, the medial geniculate nucleus (MGN), and the primary auditory cortex (A1). We utilized auditory band-pass noise stimuli covering 1kHz to 22kHz frequency range, which was presented in conjunction with broad-band masking stimulus during the behavioral study. We present evidence for a tonotopic organization of auditory responsive neurons within pNB. Consistent with this, we show that optogenetic activation of pNB PV neurons leads to an increase in the spiking activity of single neurons in both MGN and A1 at their preferred frequency band as well as at adjacent frequency bands, but not distant bands. Detection of a narrow-band target sound in the presence of broad-band noise was compromised during pNB PV activation, consistent with a boosting of neural responses to both target and adjacent masking stimulus. Our findings support the idea that pNB modulation of auditory processing encompasses frequency-specific aspects, permitting precise regulation of sensory inputs rather than the mere enhancement of overall excitability of neurons in the auditory pathway.

Paradoxical somatodendritic decoupling supports cortical plasticity during REM sleep

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REM sleep is associated with the consolidation of emotional memories. Yet, the underlying neocortical circuits and synaptic mechanisms remain unclear. We found that REM sleep is associated with a somato-dendritic decoupling in pyramidal neurons of the prefrontal cortex. This decoupling reflects a shift of inhibitory balance between PV neurons-mediated somatic inhibition and VIP-mediated dendritic disinhibition, mostly driven by neurons from the central medial thalamus. REM-specific optogenetic suppression of dendritic activity led to a loss of danger versus safety discrimination during associative learning and a lack of synaptic plasticity, whereas optogenetic release of somatic inhibition resulted in enhanced discrimination and synaptic potentiation. Somato-dendritic decoupling during REM sleep promotes opposite synaptic plasticity mechanisms that optimize emotional response to future behavioral stressors.

Local slow-wave activity in regular sleep reveals individual risk preferences

Mirjam Studler*+ (1), Lorena R.R. Gianotti+ (1), Katharina Koch (1), Jan Hausfeld (1,2), Leila Tarokh (3,4), Angelina Maric (5) & Daria Knoch (1)

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In many everyday life situations, we have to make decisions under varying degrees of risk. Even though previous research has shown that the manipulation of sleep affects risky decision-making, it yet remains to be understood how regular, healthy sleep relates to risk preferences. Therefore, we investigated the relationship between individual, temporally stable neural sleep characteristics and individual differences in risk preferences in healthy adults. Sleep data were collected using a portable high-density EEG system at participants' home. Results revealed a significant negative correlation between local sleep depth, as reflected in slow-wave activity (SWA) in a cluster of 5 electrodes located over the right prefrontal cortex and risk-taking behavior. This finding remained significant when controlling for total sleep time. Moreover, the association between SWA over the right prefrontal cortex and risk preferences was very similar in all sleep cycles. Our findings suggest that sleep depth in the right prefrontal cortex, an area involved in self-regulation, might serve as a dispositional indicator of lower self-regulatory abilities, which is expressed in greater risk-taking behavior.

Real-time fMRI neurofeedback improves pattern separation in healthy aging and mild cognitive impairment

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Introduction: Task fMRI studies repeatedly found hippocampal hyperactivity in patients with mild cognitive impairment (MCI; prodromal stage of dementia due to Alzheimer's disease). This is associated with memory deficits and a faster clinical progression. Reducing the hippocampal activity using an antiepileptic drug improved memory performance (Bakker et al., 2015). An alternative approach might be real-time fMRI neurofeedback, during which patients with MCI learn to down-regulate hippocampal hyperactivity.
Method: We use a pattern separation task (e.g. Bakker et al., 2015) to measure changes in functional activity levels of the hippocampus, comparing baseline and post-intervention fMRI performance. So far, n=30 healthy elderly volunteers (18 females, age mean + SD, 69.5 + 5.8) and 7 patients with MCI (4 females, age mean + SD, 74.8 + 2.3) down-regulated activity in the hippocampus, while n=14 healthy elderly volunteers (8 females, age mean + SD, 66.9 + 4.6) down-regulated activity in the control region (intraparietal sulcus) during the two sessions of neurofeedback training.
Preliminary results: Comparable to the study by Bakker et al., 2015, patients with MCI show reduced hippocampal activation and an improved discrimination performance on the pattern separation task. The healthy control groups also show improved discrimination performance after neurofeedback, regardless of whether they regulated the hippocampus or the control region (intraparietal sulcus).
Interim conclusion: For the currently small sample size, patients with mild cognitive impairment show a reduced functional hippocampal activity and improved pattern separation task performance after two sessions of neurofeedback training (learning down-regulation of hippocampal activity). There was no such effect on functional hippocampal activity in healthy elderly controls, but task performance also improved after the intervention

Converting thoughts into actions: Restoring communication between the motor cortex and the lumbosacral region of the spinal cord using a Brain-spine-interface in an individual with chronic tetraplegia

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Spinal cord injury interrupts the communication between the brain and the spinal cord, having a devastating impact on the motor contol and sensory function leading to paralysis. A digital bridge between the motor cortex and the dormant neurons located in the spinal cord could in principle restore mobility and promote neurological recovery in people who are chronically paralyzed.

Methods: We designed a Brain-Spine Interface (BSI) to restore communication between the motor cortex and the lumbosacral region of the spinal cord in an individual with chronic tetraplegia (Incomplete lesion at C5-C6). We implemented this digital bridge with fully-implantable recording and stimulation systems which translate electrocorticographic signals into analog modulations of epidural electrical stimulation targeting the region of the spinal cord involved in walking.

Results: The brain signals were streamed in real time to a computing unit that uses iterative classification algorithms to generate online predictions of motor intentions. We were able to classify motor attempts for the different joint movements (hip, knee and ankles bilaterally) with high accuracy. The decoded predictions were converted into electrical stimulation commands delivered by the implantable pulse generator via an epidural array of 16 – electrodes targeting the different dorsal roots of the lumbosacral region of the spinal cord responsible for lower limb movements. The participant used the BSI system over a 40 session rehabilitation period.

Conclusion: A BSI system can be used as a neurorehabilitation therapy to promote neurological recovery allowing participants to regain natural control of walking after spinal cord injury.

Retrosplenial Cortex Activity: a Hub in a Paradoxical Sleep Network?

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Rapid eye movement (REM, also called paradoxical) sleep correlates with enhanced cellular activity of region-specific thalamo-cortical circuit and subcortical structures including the hippocampus, midbrain or hypothalamus. This REM sleep specific neuronal activity is hypothesized to promote structural plasticity and provide a window for the consolidation of contextual and emotional memories previously acquired during wakefulness, yet the underlying mechanism remains unclear.
Amongst the cortical structures, the activity of neurons located in the retrosplenial cortex (RSC) is increased during REM sleep. Yet, cortical single cell-to-whole brain circuit connections and its role in REM sleep function remains unknown. Here we characterized the activity of RSC microcircuit across the sleep-wake cycle using simultaneous 2-photon calcium imaging and electrophysiological recordings in spontaneously head-restrained sleeping mice. We observed a REM sleep-specific reduction of pyramidal cell somatic activity concomitant to the activation of the interneurons expressing either parvalbumin, somatostatin or vasoactive intestinal peptide. Collectively, these observations suggested a region-specific regulation of excitatory/inhibitory balance in RSC during REM sleep that may contribute to information integration, memory consolidation and ultimately behavioural optimization.

Impact of long-term and short-term exposure of environmental enrichment on pain-related depression in adolescent mice.

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Chronic pain significantly impacts well-being of more than 30% of the world's population. Its comorbidities, such as depression, anxiety, cognitive impairments or sleep disturbances may intensify and exacerbate the overall experience of pain, which consists of physiological as well as psychological aspects. Pharmacological treatments are often effective in reversing these changes, yet carry the risk of side-effects and addiction. Moreover, prescription drugs may be costly, therefore inaccessible to number of individuals. This project aims to characterize the impact of environmental manipulation on animal model of chronic pain, and evaluate if this can prevent the development of anxiodepressive symptoms. In a pilot study, we confirmed the presence of depressive-like behaviors in adult mice after Spared Nerve Injury (SNI) causing long-lasting pain in one hind paw. Subsequently, we randomly assigned 64 mice at 21 days of age into one of eight experimental groups, following two different protocols. Each protocol is applied to four groups: SNI (pain) + standard housing, SNI + enriched environment, sham (control) + standard housing, and sham + enriched environment. Protocols differ in the duration of exposure to enrichment: a) enrichment is interrupted after 6 weeks, simultaneously to conducting SNI/sham surgery; or b) all four groups of animals are housed in the enriched environment until the end of the experiment. Six weeks after surgery, we evaluate mechanical hypersensitivity and depression phenotype, using multiple behavioral tests: von Frey, marble burying, novelty suppressed feeding, splash test, and forced swimming task.

A contextual neuronal activity of ventral hippocampus integrating reward and aversive memories

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Assessing and developing on memories of rewarding and aversive events is critical for animal survival. This behavior depends on the brain capability to simultaneously construct salient emotional experiences and internal representations of the spatial environment in which they occur. The ventral hippocampus (vHip) is a high-order cortical area involved in emotional behaviors associated with positive and negative stimuli, and so a potential candidate to create these emotional spaces.
In this study, recording the neuronal activity of freely moving mice during a social reward preference and a contextual fear conditioning task, we found that activity among ventral hippocampal CA1 neurons discriminates between emotional learning contexts. This activity pattern was selective to the emotional context after learning and able to predict context identity.
We next aimed to identify an input to vHip necessary for the formation of emotional contextual memories. For that purpose, we optogenetically manipulated, the locus coeruleus (LC), a brain region known to respond to novelty and to regulate hippocampal plasticity. We observed that suppression of LC input to vHip during learning impaired social reward memory formation. Contrastingly, the inhibition of LC projections to vHip did not impair contextual fear memory.
These findings identify contextual discrimination activity patterns as a principal feature of vCA1 that encodes behaviorally relevant emotional contextual memories. Moreover, this study provides evidence that LC input to vHip plays a crucial role in social reward memory. Additional experiments will address the impact of LC for the emergence of the emotional context discrimination activity patterns observed in vCA1.

Inhibiting the Anterior Cingulate Cortex to modulate behavioral responses to pain

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The Anterior Cingulate Cortex (ACC) is part of the medial pain pathway, drives the emotional component of pain and our conscious body response to unpleasant stimuli. However, the thalamocortical circuit of pain processing is intricate and the precise functional contribution of the ACC in building the pain experience remains unclear.
Recent research in the lab, using intracranial tetrode recordings in the mouse, hints that the ACC activity differently modulates the behavioral responses to painful stimuli in the hind paw. These two behavioral responses are withdrawal of the paw and awakening from NREM sleep. Interestingly, these two responses, do not seem dependent on one another. Therefore, the aim of this study is to determine the contributions of the ACC in the behavioral outcome of the stimuli. To do so, we use optogenetic tools to inhibit the activity of this region, at the time of the stimulation, combined with electroencephalogram recordings to evaluate changes in the behavioral outcomes. These experiments are implemented in a larger study investigating - by electrophysiological recordings and behavioral testing - the interplay of pain processing and sleep in the mouse thalamocortical networks.

Probing cortical excitability and seizure resilience under GABAergic modulation

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Background: Cortical excitability, the variable response to a given cortical input, is widely studied in neuroscience, from slice experiments and in silico modeling work to human clinical settings. However, a unifying definition and a translational approach to the phenomenon are currently lacking In particular, defining and quantifying the boundaries between physiological and pathological cortical dynamics is an active field of research. Recent modeling and experimental work suggested that transitions to seizure occur by the alignment of small stochastic perturbations and of a slower decrease in cortical resilience, also called the slow permittivity variable. However, this work mostly relied on measurements of excitability and resilience in in-vitro models of epilepsy, and in-vivo validation of these concepts and their broader applicability to the non-epileptic brain are currently lacking. Aim: To test In-Vivo the theorical predictions that changes in cortical resilience are accompanied by precursor sings measurable in the cortical response to small perturbations. Methods: In this study, we used in-vivo optogenetic stimulation in awake, freely-moving mice to assess whether changes in cortical excitability, quantified as evoked response to small perturbation, correlate to changes in resilience to seizures, measured as the duration of stimulation necessary to induce a seizure (Time to Seizure). We explored how these markers varied in the presence of GABAergic agonist (Diazepam (DZ)) and antagonist (Pentylenetetrazol (PTZ) and Picrotoxin (PTX)). Results: We found that evoked cortical response to single opto-pulse was reduced by 29.3% 95%CI [27.6-31.2] in presence of DZ and increased with GABAergic antagonist (5.1% [3.0-7.1] for PTZ and 9.3% [7.1-11.3] for PTX). GABAergic drugs also modulated cortical resilience with a 78.1% [54.5 113.0] increase in time-to-seizure in presence of Diazepam and a respectively 19.6% [3.0-10.8] and 10.6% [-4.0-18.5] decrease with PTZ and PTX, corroborating the changes observed in cortical excitability. Conclusion: In this study, we provide strong in vivo experimental evidence for a direct relevance of using minute perturbations of ongoing activity as markers of cortical excitability and show their correlation with resilience to epileptic seizures.

The Role of the Melanin-concentrating Hormone (MCH) Neurons in increased REM Sleep Propensity and Cataplexy in Narcolepsy

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The lateral hypothalamic melanin-concentrating hormone (MCH) neurons play a regulatory role in REM sleep, including their ability to dynamically increase REM sleep expression during thermoneutral ambient temperature (Ta) warming (Komagata et al., 2019). Moreover, our prior data have shown that thermoneutral Ta warming increases REM sleep but decreases cataplexy in narcoleptic hypocretin knock-out (Hcrt-KO) mice. Given the reciprocal (indirect vs direct) inhibition between the Hcrt and MCH systems, we hypothesize that loss of Hcrt may disinhibit MCH activity resulting in the increased REM sleep propensity characteristic of narcolepsy, whereas periods of low MCH activity may exacerbate boundary state instability and favor cataplexy. Using fiber photometry calcium imaging, we first investigated the normal dynamics of MCH activity across the sleep-wake cycle in MCH:cre mice at both constant Ta and thermoneutral Ta warming. We then evaluated the role of the MCH system in the expression of REM sleep and cataplexy in MCH:cre/HcrtKO narcoleptic mice. During the light phase, fiber photometry approach revealed that MCH-dependent signal markedly increased in anticipation and during REM sleep but dropped during NREM sleep and wake states in both animal models. Exposure to thermoneutral warm Ta pulsing increased the time spent in REM sleep compared to constant Ta condition for MCH:cre mice as expected, an effect that is not observed during the dark phase.
However, MCH:cre/HcrtKO increased REM sleep expression during the dark phase during the Ta warming condition, consistent with the increased REM sleep propensity of narcolepsy. However, cataplexy expression was significantly decreased during Ta warming as seen in our prior experiments. MCH activity in narcoleptic mice increased in anticipation of REM sleep with maximum dF/F values near the beginning of REM sleep onset. Surprisingly, MCH activity also increased during cataplexy but with maximum values observed near the end of the cataplexy bouts. MCH activity then decreased at the transition from REM sleep or cataplexy to the wake state. These data are consistent with our working hypothesis that increased MCH activity in narcolepsy may drive increased REM sleep propensity, whereas low MCH activity may favor cataplexy onset. The role of increasing MCH activity during cataplexy bouts as a potential reflection of state transition from cataplexy to REM sleep requires further investigation.

A hardware-software co-design approach to minimize the use of memory resources in multi-core neuromorphic processors

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There are a diversity of applications where Artificial Intelligence (AI) exceeds human capabilities; however, they also require the processing of large amounts of data, which in turn place significant demands on computer speed and power efficiency that are limited by the hardware. We know that computers are faster to perform calculations, are more accurate to make deterministic decisions, and are reliable. Yet, human brains outperform computers in cognitive information processing tasks, such as object recognition or complex scene analysis and understanding. Even more, relying only on a few data or multiple modalities combined. The rapid development of AI pushes the development of domain-specific hardware, which leads to the development of multi-core Spiking Neural Network (SNN) chips. Neuromorphic computing chips integrate a range of features inspired by neurobiological systems and can provide an energy-efficient approach to AI computing workloads.

Biological neural networks are often highly recurrent and have dense connections for nearby neurons and sparse connections to specific/far away neurons, showing an exponential decay in the number of connections with increasing distance, following a small-world network structure. We understand that computation, and other functions, emerge from the interaction of neural areas. And that the structural wiring of brain areas is highly correlated with brain functions such as memory, vision, and motor control. Those differences support a wide range of cognition and behaviors. Focusing on specific motifs of networks, we can reduce the connectivity space without limiting (too much) the SNN developer. We fix a network architecture to find the balance between configurability and memory costs.

In this work, we investigate how to optimize resource (memory) allocation both within cores and across cores to derive specifications to guide chip designs and map SNN onto neuromorphic hardware. For this, we propose a hardware-software co-design approach that takes inspiration from biological neural networks. We use this approach to design a new routing scheme optimized for small-world networks and, at the same time, to present a hardware-aware placement algorithm that optimizes the allocation of resources for small-world network models.

Potassium channels in retinal interneurons: impact on healthy retina, degenerated retina and vision restorative approaches

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Background: Bipolar cells have become successful targets for optogenetic gene therapy to restore vision in photoreceptor degenerative diseases. However, degeneration was shown to cause changes in neuronal connectivity and protein expression, which may impact the quality of synthetically restored signaling.
Materials and Methods: We here investigated channel activity in rod bipolar cells employing a transgenic mouse model of retinitis pigmentosa expressing the optogenetic actuator Opto-mGluR6 in the ON-bipolar cell population with patch-clamp technique, genetic and immunoistochemical analysis.
Results: We identified two functional types of rod bipolar cells within the same morphological class that were interconvertible with the membrane potential acting as a toggle switch for functional classification. With progressing degeneration, the dominant outward rectifying currents of type 2 RBCs significantly decreased. We confer this loss of conductivity to downregulation of BK channel expression in RBCs of the degenerated rd1 retina, which normally tune the membrane potential to the optimal functional range.
Conclusions: A better understanding of the changes of rod bipolar cell physiology during retinal degeneration may pave the way to optimize future treatment strategies of blindness.

Resolving decision-making during emotional conflicts by ventral hippocampal circuits

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How the brain computes decisions to support adaptive behaviours under different demands is a long-standing question. Decision-making does not solely rely on cognitive judgements but is likewise influenced by internal states. The ventral hippocampus (vHip) is a higher-order cortical brain region critical for processing emotions such as anxiety. Here, we examined the influence of anxiety levels on decision-making while recording neuronal activity in the vHip and medial prefrontal cortex (mPFC) as mice performed decision-making tasks under emotional conflicts. We observed that the activity of vHip neurons was scaling according to anxiety levels with concomitant remapping of firing fields. This effect was modulated by trajectories with different anxiety levels but was not a mere reflection of novelty. We additionally identified vHip neurons with preferential ‘deliberating’ and ‘anxiety’ features as mice made decisions under emotional conflicts. Using selective optogenetic inhibition of vHip terminals in the mPFC, we showed that mice exhibited biased decision-making selectively during trials with higher emotional conflicts. Collectively, these results suggest that vHip circuits targeting mPFC mediate decision-making under emotional conflicts.

Functional dissociation of ventral hippocampal inhibitory circuits during anxiety and fear behaviors

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The ability to predict potentially harmful environments and escape from dangerous circumstances is vital for wildlife. Mounting evidence suggests that while the dorsal subdivision of the hippocampus is associated with spatial and episodic memory formation, the ventral hippocampus is mostly involved in emotional behaviors. However, the neural circuits and mechanisms within the ventral hippocampus underlying innate or learned emotional behaviors are poorly understood. In the present work, we investigated whether anxiety and conditioned fear are represented by distinct ventral CA1 (vCA1) neural circuits. By utilizing cell-type-specific expression of calcium indicator GCaMPs and in vivo calcium imaging in freely behaving mice via miniature fluorescence microscope, we monitored the neuronal activity of vCA1 pyramidal cells and three sub-classes of GABAergic interneurons (PV+, VIP+, and SST+) during innate anxiety and conditioned fear behaviors. Our data indicated that vCA1 pyramidal cells and interneurons have distinct activity patterns during anxiety and fear behaviors. Different subpopulations of vCA1 pyramidal cells showed preferential responses to either anxiogenic experiences or fear-conditioned cues. The majority of PV+ interneurons were recruited during anxiety behavior but barely during a cued fear test. By contrast, about half of VIP+ interneurons were involved in conditioned fear learning but not in anxiety behaviors, while SST+ interneurons displayed inhibition during fear learning, suggesting that an inhibitory microcircuit may gate pyramidal cell activity during fear conditioning. Altogether, our data suggested a division of labor among various vCA1 GABAergic interneurons during different forms of emotional behaviors.

Who we are

The Young Swiss Society for Neuroscience is an organization funded by the Swiss Society for Neuroscience focused on the promotion of early-career neuroscientists in Switzerland.

We believe science communication is a fundamental part of the professional development of a researcher. Following this belief, we organize annual meetings and events to provide trainees the opportunity to share their research with the neuroscience community. The ySSN brings together neuroscientists from institutes across Switzerland with the goal of fostering scientific exchange and networking in a vibrant and friendly environment.

Our Mission

The primary mission of the ySSN is to create a dynamic scientific network for the young neuroscience community in Switzerland.

To attain this goal, we organize events throughout the year which serve as a platform for young neuroscientists to increase their visibility and audience. We provide environments where early-career researchers can develop professional skills to aid their success in research. Specifically, we aim to:

- Increase knowledge transfer between trainees, laboratories, and institutions.
- Build effective communication skills to engage the audience in thinking and discussing the research of early-career neuroscientists.
- Create a diverse network that works together to train and promote outstanding future neuroscientists.

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