The Sleeping Brain

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The expression of serotonin 5-HT2B receptors in microglia is required for spatial memory and learning-induced spine remodeling in the hippocampus.

Giulia Albertini * 1, Vincenzo Mastrolia 1, Fanny Etienne 1, Jean-Christophe Poncer 1, Luc Maroteaux 1, Anne Roumier 1

1 Institut du Fer à Moulin – Institut National de la Santé et de la Recherche Médicale : U1270, Sorbonne Université – France

The hippocampus receives dense innervation by serotonergic projections from the raphe nuclei and several studies demonstrated that serotonin (5-HT) modulates hippocampal plasticity and memory formation. We previously showed that, besides neurons, microglia are able to sense and respond to 5-HT via 5-HT2B receptors, the main 5-HT receptors expressed by microglia. Microglia are dynamic cells that constantly survey the environment by physically interacting with neurons and shaping synaptic connections: as normal synaptic and structural plasticity supports cognition, altered microglial functions might cause synaptic dysfunction and memory deficits. In this study, we investigated whether disturbed communication between microglia and neurons due to conditional invalidation of microglial 5-HT2B receptors in adolescent mice affects hippocampal-dependent memory processes. We demonstrate that microglial 5-HT2B receptor ablation impairs spatial memory tasks, such as the discrimination of object’s location or the fear response to a context, while having a weaker effect on non-spatial paradigms. In contrast, hippocampal LTP induced in CA1 by high frequency stimulations of Schaffer collaterals is slightly increased in mice lacking microglial 5-HT2B receptors. This unexpected finding might be related to the increased spine head volume in CA1 principal neurons of conditional knockout mice. As larger spines are known to be more stable, we investigated the dynamics of spine remodeling induced by fear conditioning. Intriguingly, we show that the fear response observed 24 hours after contextual fear conditioning and the subsequent increase in spine density and length are strongly diminished in the hippocampus of mice lacking 5-HT2B receptors in microglial cells. Finally, we show that the correlation between spine growth and fear intensity observed in control mice is abolished upon invalidation of 5-HT2B receptors on microglia. Altogether, these findings point to an important role of microglial 5-HT2B receptors in spatial memory and structural plasticity. Since the serotonergic system modulates sleep also via 5-HT2B receptors and sleep is tightly associated with learning-related spine remodeling and memory, we are currently investigating whether disrupted serotonergic modulation of microglia might affect the sleep/wake cycle.

*Speaker
Neurofunctional ultrasound reveals new insights in functional coupling between fast gamma oscillations and blood flow in sleeping rodents

Antoine Bergel *, Thomas Deffieux 1, Charlie Demene 1, Mickaël Tanter 1, Ivan Cohen 2

1 ESPCI Paris, PSL Research University, CNRS UMR 7587, INSERM U979 – ESPCI Paris, PSL Research University, CNRS UMR 7587, INSERM U979 – France
2 Institut de Biologie Paris Seine (IBPS) – Institut de Biologie Paris Seine (IBPS) – France

Functional Ultrasound (fUS) imaging is a novel neuroimaging modality, which probes the activity of deep vascular networks in a wide range of experimental contexts including mobile, head-fixed and sleeping animals. Its key features include large field of view, good spatial (150 microns) and temporal (200 ms) resolutions, lightweight apparatus and compatibility with electrophysiological recordings. In rodent studies, fUS imaging is becoming a tool of choice as it provides an insight into brain-wide functional networks in an unbiased fashion. During REM sleep, we demonstrate the close association between massive hyperemic events and fast gamma. We show that vascular activity divides into tonic and phasic regimes. The phasic component of this vascular activity contained vascular surges (VS), i.e. large-amplitude spatially-extended hyperemic waves. The amplitude of these VS outmatched waking levels and were robustly preceded by sustained theta (6-10 Hz) and fast gamma oscillations (80-110 Hz), the power of fast gamma being strongly correlated to the amplitude and duration of each subsequent VS. Our findings question the evolutionary benefit of such high energy-demanding vascular patterns and opens the way for the combined local controlled manipulation of brain rhythms and global imaging of sleep hemodynamics.

*Speaker
rsleep, an open-source R package to analyse polysomnography data.

Paul Bouchequet * 1, Geoffroy Solelhac 1,2, Damien Léger 2,3

1 Université Paris Descartes - EA7330 VIFASOM – Université Paris Descartes - Paris 5 – France
2 Assistance publique - Hôpitaux de Paris (AP-HP) – Assistance publique - Hôpitaux de Paris (AP-HP)
   – France
3 Université Paris Descartes - EA7330 VIFASOM – Université Paris Descartes - Paris 5, Université
   Paris Descartes - Paris 5 – France

rsleep is an open-source R package providing implementations of common tools and function for sleep analysis, available on the Comprehensive R Archive Network and Github (https://github.com/boupetch/rsleep). rsleep reads sleep data in European Data Format (EDF), Morpheo Data Fromat (MDF) format, and writes records as MDF. MDF is a simple, fast and interoperable data format relying on the filesystem architecture.

rsleep provides tools for spectral analysis such as Welch and multitaper density estimators. Hearth Rate Variability analysis can be performed on raw electrocardiogram data using a R peak detector based on the Pan & Tompkins method.

rsleep automatically scores sleep stages from polysomnographies using a pretrained neural network. This model has been trained on more than 100 records from the patients of the Center for Sleep and Vigilance of the Hôtel-Dieu, Paris, France, using the architecture described by Chambon & Al. in ”A deep learning architecture for temporal sleep stage classification using multivariate and multimodal timeseries” (2018). Functions to create a training dataset and train a new model from a new database are also available.
In addition to the visualization opportunities offered by R, rsleep provide simples functions to draw hypnogram and hypnodensities from raw data using ggplot2.

*Speaker
Inside the mind of a dreamer: two way communication during lucid dreams

Emma Chabani 1, Basak Turker 1, Jean-Baptiste Maranci 2, Isabelle Arnulf 1,2, Delphine Oudiette * 1

1 Institut du Cerveau et de la Moelle Epinière = Brain and Spine Institute – Institut National de la Santé et de la Recherche Médicale : U1127, CHU Pitié-Salpêtrière [APHP], Sorbonne Université : UM75, Centre National de la Recherche Scientifique : UMR7225 – France
2 Service des Pathologies du Sommeil – Hôpital Pitié-Salpêtrière – France

Dreams are one of the widest unexplained mystery of human cognition, partly because of their private nature and the inability for scientists to directly access dream experience. Contrary to typical dreamers, lucid dreamers are aware of being in a dream. They can signal lucidity with lateral eye movements, but research has been limited to one-way communication so far, with no online intervention of the experimenter. Would it be possible to establish a dialogue between the experimenter and the lucid dreamer while she is within the dream?

In this study, we performed several two-way communication attempts with one experienced lucid dreamer using sensory stimulation. The participant was monitored during a daytime nap, using polysomnography and two additional EMG sensors recording facial muscles (zygomatic and corrugator). The subject was informed that tactile (e.g. taps on the hand), light stimuli (e.g. green or red), and auditory (yes/no questions) would be presented during the nap, but did not know in which order. He was instructed to contract either the zygomatic or the corrugator muscles accordingly to the number of taps, the color of the light or whether the response was yes or no.

The participant was not able to discriminate light (no responses). However, he correctly counted and signalled the number of taps applied in 9 cases out of 12 and successfully answered 3 yes/no questions out of 5 using appropriate facial contractions. Our results are a proof of concept that two-way communication may be possible in multiple modalities and different levels of cognitive complexity. This finding paves the way for a new era of investigations into dream cognition.

*Speaker
Neuropeptide S inhibits sleep-promoting neurons into the VLPO to favor wakefulness

Frédéric Chauveau *, Damien Claverie, Emma Lardant, Christophe Varin, Augustin Walter, Éléonore Hardy, Frédéric Canini, Nathalie Rouach, Armelle Rancillac *

1 IRBA (Armed Biomedical Research Institute), Brétigny-sur-Orge. – Institut de Recherche Biomédicale des Armées, Institut de recherche biomédicale des armées – France
2 ULB Neuroscience Institute – Belgium
3 Brain Plasticity Unit, CNRS, UMR 8249, ESPCI-ParisTech, PSL Research University, Paris, 75005 – ESPCI Paris, PSL Research University – France
4 Neuroglial Interactions in Cerebral Physiopathology, CIRB, UMR 7241/ U1050, Paris, F-75005. – Collège de France – France
5 Ecole du Val de Grâce, 1 place Laveran, 75005 Paris – Ecole du Val de Grâce – France

Regulation of sleep-wake cycles is crucial for brain’s health and cognitive skills. Among the various neurochemicals known to control different states of alertness, neuropeptide S (NPS) would play an important role in promoting awakening. However, NPS signaling pathways remain elusive.

In this study, we characterized the effects of NPS in the ventrolateral preoptic nucleus of the hypothalamus (VLPO), one of the major brain structures regulating non-rapid eye movement (NREM) sleep. Using polysomnographic recordings, we demonstrated that in mice, local and bilateral infusion of NPS into the VLPO significantly increases awakening while reducing both quantity and quality of NREM sleep. At the cellular level, we demonstrated by patch-clamp recordings that NPS hyperpolarizes VLPO sleep-promoting neurons, indirectly via the depolarization of local GABAergic neurons. Moreover, we have established that the application of NPS on acute brain slices induces a strong and reversible TTX-sensitive constriction of blood vessels in the VLPO. This vasoconstrictor effect suggests that NPS down-regulates the activity of VLPO neural networks.

Altogether, our results highlight for the first time in the VLPO that NPS exerts a direct role by controlling local neuronal activity. Following the depolarization of local GABAergic neurons, NPS indirectly causes a feed-forward inhibition onto sleep-promoting neurons, which translates into a decrease in NREM sleep to favor arousal.

*Speaker
Monitoring calcium dynamics in astrocytes of freely-moving mice over sleep-wake cycles

Valentyna Dubovyk\textsuperscript{1}, Tatiana Peleh\textsuperscript{2}, Thomas Kuner\textsuperscript{3}, Michal Slezak\textsuperscript{*}

\textsuperscript{1} BioMed X Innovation Center – Germany
\textsuperscript{2} Boehringer Ingelheim – Germany
\textsuperscript{3} University of Heidelberg – Germany

Astrocytes are key cells in the brain that function as homeostatic regulators. Having the contact with blood vessels and neuronal synapses they influence local homeostasis through regulation of ionic homeostasis, neurovascular communication, metabolic uptake and neurotransmitter clearance. Importantly, these processes consistently change across the sleep-wake cycle, suggesting that astrocytes function at different rates during different vigilance states. Moreover, it is believed that astrocyte activity in large part is correlated to intracellular calcium dynamics. The discovery of norepinephrine-mediated astrocytic calcium responses has raised the possibility that astrocytic activity is driven by changes in behavioral state, which are known to be regulated by norepinephrine among other neuromodulators. In the current work, we aim to establish a system for monitoring calcium dynamics in astrocytes of freely-moving mice that will allow us to test the hypothesis whether astrocytic activity is regulated by the sleep-wake cycle.

\textsuperscript{*}Speaker
Synchronized population activity in cortex is a hallmark of slow-wave sleep, quiet wakefulness, or anesthesia. Rhythmic fluctuations of neuronal membrane excitability are reflected in extracellular field potentials as delta (1-4 Hz) or large-amplitude slow (~1Hz) oscillations (SO); the latter are indicative for Up-to-Down-states transitions. The Up-states comprise periods of neuronal membrane depolarization accompanied by sustained spiking activity; the Down-states are associated with membrane hyperpolarization and neuronal silence. The level of cortical synchronization depends on the input from a number of subcortical neuromodulatory centers, including the brainstem nucleus Locus Coeruleus (LC). The LC regulates cortical excitability via its direct or indirect ascending projections and norepinephrine (NE) release in the target regions. We have previously demonstrated that phasic LC activation causes transient cortical desynchronization. Here, we sought to characterize the effect of transient NE release on the cortical population dynamics at a fine temporal scale. To this end, we quantified the effects of the direct electrical stimulation of LC (LC-DES) on the Up/Down-state transition in the medial prefrontal cortex (mPFC) in urethane-anesthetized rats. Biphasic electric pulses (0.4 ms, 0.02-0.05 mA) were applied to the LC unilaterally at 50 Hz for 200 ms while mPFC activity was monitored ipsilateral to the simulation site. The effect of LC-DES on cortical population activity depended on the phase SO. The LC-DES presented within an Up-state prolonged the ongoing Up-state for 94.3 ± 21.3 ms and caused ~20% increase of the firing rate in the mPFC. In addition, Down-states coincided with LC-DES were followed by a rapid transition to Up-state in ~20% of trials. Our results suggest that the effect of NE release on cortical population

*Speaker
dynamics strongly depends on the cortical state preceding LC activation.
Hypothalamic MCH neurons contribute to forgetting of hippocampus dependent memory during REM sleep

Shuntaro Izawa *, Srikanta Chowdhury 1, Toh Miyazaki 1, Yasutaka Mukai 1, Daisuke Ono 1, Ryo Inoue 1, Thomas Kilduff 2, Akihiro Yamanaka 1

1 Dept. of Neurosci. II, Res. Inst. of Env. Med., Nagoya, Japan – Japan
2 Cntr for Neurosci., SRI Intl., Menlo Park, CA – United States

Memory regulation during sleep is a diverse and complicated process, and the neural mechanisms underlying this regulation are not yet fully understood. Here, we demonstrate that rapid eye movement (REM) sleep-active melanin-concentrating hormone (MCH)-producing neurons in the hypothalamus actively contribute to forgetting hippocampus-dependent memory. Hypothalamic MCH neurons densely innervate the dorsal hippocampus, and activation or inhibition of MCH neurons using chemogenetics or optogenetics significantly impaired or improved hippocampus-dependent memory, respectively. Activation of MCH nerve terminals in vitro inhibited firing of hippocampal pyramidal neurons by increasing inhibitory inputs. In vivo calcium imaging indicated that wake- and REM sleep-active MCH neurons are distinct populations that are randomly distributed in the hypothalamus. Sleep/wakefulness state-dependent optogenetic inhibition of MCH neurons revealed that REM sleep-active MCH neurons impaired hippocampus-dependent memory without affecting sleep architecture or quality. Together, these results demonstrate that REM sleep-active MCH neurons in the hypothalamus are involved in active forgetting in the hippocampus.

*Speaker
Selection of informative stimuli during human rapid eye-movement sleep

Matthieu Koroma *, Thomas Andrillon 2, Célia Lacaux 3, Guillaume Legendre 4, Damien Léger 5,6, Sid Kouider 1

1 Laboratoire de Sciences Cognitives et Psycholinguistique, Dept d’Etudes Cognitives, ENS, PSL University, EHESS, CNRS – École normale supérieure [ENS] - Paris – France
2 IBRO Research Fellow, School of Psychological Sciences, Monash University – Australia
3 Sorbonne Université, IHU@ICM, INSERM, CNRS UMR7225, équipe MOVIT, AP-HP, Hôpital Pitié-Salpêtrière, Service des Pathologies du Sommeil, F-75013 Paris, France – AP-HP [Groupe hospitalier Pitié - Salpêtrière] – France
4 Laboratory for Neurology and Imaging of Cognition, Departments of Neurology and Neurosciences, Centre Médical Universitaire, University of Geneva, CH-1211, Geneva, Switzerland – Switzerland
5 Assistance publique - Hôpitaux de Paris (AP-HP) – Assistance publique - Hôpitaux de Paris (AP-HP) – France
6 Université Paris Descartes - EA7330 VIFASOM – Université Paris Descartes - Paris 5 – France

Sleep is a period of sensory disconnection. When we dream, we are conscious of something else than our direct environment. Is this because of a competition between our dream content and external stimuli or because we cannot access external stimuli? We first observed that external stimuli are still being selectively processed during REM sleep. We additionally show that eye movements, a marker of dreaming activity, are concomitant with a selective suppression of informative stimuli. These results support the "informational gating hypothesis", i.e. a competition between internal activity and stimulus processing during REM sleep (Andrillon et al, 2016).

*Speaker
Breathing coordinates network dynamics underlying memory consolidation

Nikolaos Karalis * ¹, Anton Sirota ²

¹ Friedrich Miescher Institute for Biomedical Research – Switzerland
² Ludwig-Maximilians-University Munich – Germany

During offline states, cortical and hippocampal networks are dominated by nonlinear dynamics, such as hippocampal ripples and neocortical DOWN/UP states. The coordination of these network dynamics underlies memory reactivation and information transfer between these regions and is believed to be critical for the consolidation of memories, giving rise to prominent models of systems memory consolidation. However, the mechanisms underlying such persistent coordination of the dynamics across global brain networks are poorly understood and the existing frameworks based on pair-wise interactions cannot explain the large-scale brain synchrony during offline states. From a theoretical perspective, a global pacemaker has been postulated as an effective solution to the coupling of distinct nonlinear network dynamics, but the neural implementation of such a mechanism remains elusive.

Here we address the hypothesis that respiration acts as an oscillatory master clock, persistently coupling distributed brain circuit dynamics. Using large-scale recordings from the prefrontal cortex, hippocampus, amygdala, nucleus accumbens, and thalamus in behaving mice, we demonstrate that respiration entrains the neuronal activity in all these regions, giving rise to global synchronization. In parallel, it mediates the interaction of local nonlinear network dynamics, including hippocampal ripples and cortical DOWN/UP states, as well as gamma oscillations, during quiescence and sleep, effectively providing the substrate for coherent systems memory consolidation across distributed brain structures.

Further, using pharmacological manipulations and analytical methods, we identify a novel joint circuit mechanism underlying the respiratory entrainment of the limbic circuits, in the form of an intracerebral respiratory corollary discharge (RCD) and a respiratory olfactory reafference (ROR), suggesting a distributed predictive signaling mechanism across cortical and subcortical networks.

These results highlight breathing, a perennial brain rhythm, as an oscillatory scaffold for the functional coordination of the limbic circuit, enabling the segregation and integration of information flow across neuronal networks and coordinating memory consolidation processes.

*Speaker
Sleep dynamics in the dorsal hippocampus and amygdala

Billel Khouader * 1,2, Gabrielle Girardeau 3

1 Institut du Fer à Moulin – Institut National de la Santé et de la Recherche Médicale : U1270, Sorbonne Université – France
2 Ecole de l’inserm liliane bettencourt – Institut National de la Santé et de la Recherche Médicale - INSERM – France
3 Institut du Fer à Moulin – Institut National de la Santé et de la Recherche Médicale, Sorbonne Université – France

Sleep is a physiological state of reduced vigilance and alertness known to be important for brain homeostasis, memory consolidation and emotional regulation. During sleep, the brain cycle trough two main stages: Non-REM sleep (also called slow-wave sleep) and REM sleep. The neuronal dynamics of REM and Non-REM sleep have been extensively studied, notably in the hippocampus and neocortex, in link with homeostasis and plastic processes related to memory consolidation. The amygdala is a critical brain region for the processing of emotions. However, the sleep dynamics of the amygdala are comparatively understudied, despite a hypothesized role for both REM and Non-REM sleep in emotional memory consolidation. Here, using large-scale recording of LFPs and neuronal assemblies in the hippocampus and basolateral amygdala (BLA) of freely moving rats, we extensively describe the dynamics of neuronal firing in the BLA at transitions between states (Wakefulness/REM/Non-REM), and within REM-sleep. In addition, we analyze how local (BLA) and hippocampal theta oscillations during REM-sleep influence activity in the amygdala. Preliminary results indicate increased firing rates of both pyramidal neurons and interneurons in the BLA at Non-REM to REM transitions. Moreover, subsets of BLA neurons are modulated by hippocampal theta oscillation during REM-sleep. Unique firing patterns in the BLA during REM and Non-REM sleep might underlying the specific roles of these two sleep stages in emotional regulation and memory.

*Speaker
Population dynamics in the cortico-basal pathway of behaving and sleeping songbirds

Corinna Lorenz *, 1,2, Ezequiel M. Arneodo 1, Richard Hahnloser 1, Nicolas Giret *

1 Institute of Neuroinformatics, UZH/ETH Zürich – Switzerland
2 Institut de Neurosciences Paris-Saclay – CNRS : UMR9197, CNRS UMR 9197 – France
3 Institut de Neurosciences Paris-Saclay (Neuro-PSI) – CNRS UMR 9197 – Batiment 446 Université
Paris Sud 91400 Orsay, France

Sleep occupies roughly one-third of our lives, yet we do not fully understand its purpose or function. Despite our reduced external behavior during sleep, paradoxically our brains remain highly active. The widespread activity that occurs in the brain during sleep has a purpose; however, there is still no consensus on what that might be. One view, which is well supported by data, is that sleep benefits learning and memory. Sleep plays for example a critical role in birdsong learning. Birdsong learning is a sensorimotor imitation process during which birds first memorize the song of a tutor as a template and then modify their own song according to the auditory feedback. Songbirds have evolved a set of interconnected brain nuclei dedicated to song learning, production and perception. Active processes occur during sleep in these nuclei and argue for a critical role of offline neuronal processes on song motor learning in songbirds. However, we are still missing a clear understanding of the offline neuronal code expressed while birds are learning their song. To gain insights on bird sleep neurophysiology, we are performing large-scale (384 channels) electrophysiological recordings with Neuropixels probes of cortical-like and basal ganglia areas in freely moving birds while they are singing, sleeping or listening passively to playbacks of their own song. Beyond singing and hearing related neuronal activity, our preliminary data also shows transitions between states of low activity with highly precise synchronized bursting activity of several neuronal sites and states of high activity with little temporal correlation in the sleeping birds.
A recent study (A. Bergel et al., 2018) showed during Rapid-Eye-Movement sleep (REMS) a spectacular increase in the cerebral blood volume, far above wake and non-REMS levels, composed of a tonic component and a phasic one tightly correlated to an increase in hippocampal theta and gamma activity. Characterizing the vascular activity across the whole brain will help us understand better this peculiar state whose functions are still not well known.

Using the same setup, functional ultrasound imaging combined with electroencephalography, we were able to measure the variations of cerebral blood volume across several coronal and sagittal planes and the hippocampal local field potential (LFP), in three rats, creating a large REMS dataset (37 planes, 145 recordings, 218 REM episodes).

First analyses of the dataset showed that some brain regions present only a sustained tonic activity during the whole REM episode, such as the septum, a brain area, known to be at the origin of the REMS’s characteristic theta oscillation. It also appears that REM episodes do not always present a tonic and phasic component, and when it happens, the REM episode is divided in two: one part with both tonic and phasic components, and one part with only the tonic component. In that later case, the amygdala are more active than the rest of the brain.

Imaging for the first time the vascular activity of the whole brain shows us peculiar vascular patterns in different brain regions, thus increasing our knowledge on brain activation patterns during REMS.
Is histamine increased in animal models of narcolepsy type 1?

Silvia Melzi *, Anne-Laure Morel, Roland Liblau, Yves Dauvilliers, Christelle Peyron

1 Centre de Recherche en Neurosciences de LYON, CNRS UMR5292, INSERM U1028, Université Lyon-1, LYON France. – Centre de Recherche en Neurosciences de Lyon – France
2 Centre de Physiopathologie de Toulouse-Purpan, INSERM U1043, CNRS UMR5282, Université Toulouse-3, TOULOUSE, France. – Centre de Physiopathologie de Toulouse-Purpan – France
3 Centre National de Référence de la Narcolepsie et des Hypersomnies, CHU Gui-de-Chauliac, INSERM U1061, MONTPELLIER, France. – Centre National de Référence de la Narcolepsie et des Hypersomnies – France

Narcolepsy type 1 (NT1) is a rare central hypersonnia characterized by the selective loss of orexin(ORX)-producing neurons that results in excessive daytime sleepiness and cataplexy. Although surprising, an increase in the number of histaminergic (HDC) neurons in human NT1 was found by two independent studies (Vaiko et al, 2013; John et al, 2013). However, the milder increase in genetic animal models of narcolepsy has been described by only one of the study and is controversial. Interestingly, studied animal models of NT1 are all genetic while human NT1 is suspected to be of autoimmune origin. The increased number of HDC cells in human NT1 might thus be the consequence of a non-specific neuro-inflammatory process.

To evaluate whether the HDC cells increase seen in humans is linked to a compensation phenomenon or to an inflammatory process, we quantify HDC cells and HDC mRNA in genetic and neuroinflammatory models of NT1 in mice, namely Orex-KO (genetic) and Orex-HA (neuroinflammatory). The number of HDC cells in WT and NT1 mice models is assessed using immunohistochemistry while the level of mRNA HDC expression is assessed by qPCR. Number of cells and mRNA expression for ORX and MCH are also evaluated as controls because ORX is impaired while MCH remains intact in NT1.

Our preliminary data, deriving from the counting of HDC immunostained cells, shows that the number of HDC neurons is not increased in any animal models of genetic and neuroinflammatory NT1 studied. As immunohistochemistry is not the best technique for quantification, qPCR quantification of HDC, orexin and MCH mRNA expression levels are currently evaluated and will be presented. These original results will potentially provide key data to further our understanding of NT1 etiology.

*Speaker
Sleep and prospective memory in breast cancer patients: a neglected link?


Breast cancer (BC) patients have frequent complaints of sleep difficulties, however few studies have used PSG in these patients. Sleep difficulties can have deleterious impact over prospective memory (PM), a form of memory needed to remember what we are supposed to do at the right moment. PM is essential for medical adherence, autonomy, and return to social and professional life. Previous PM laboratory tasks used in BC patients lacked ecological validity. In contrast, virtual reality recreates naturalistic situations of daily life while maintaining experimental rigor that is difficult to uphold in naturalistic PM tasks. We aimed to evaluate the role of sleep disturbances in PM functioning difficulties in BC patients using virtual reality. Eighteen BC patients treated by radiotherapy and hormonal therapy and twenty-one post-menopausal healthy controls matched in age and education completed the study. Sleep was evaluated by questionnaires and by PSG. The PM task consisted of learning intentions in the evening and recalled them the next morning. Intentions were ever Event-Based (EB), i.e. when an event occurs and triggers the remembering of the action; or Time-Based (TB), referring to self-initiated retrieval of the action after a period of time has elapsed or at a certain time. A neuropsychological test battery included measures of retrospective episodic memory, working memory, binding process and executive functioning.

*Speaker
Questionnaires revealed greater sleep complaints and subjective sleep difficulties in BC patients than in healthy controls. Sleep architecture results revealed significant lower sleep maintenance index, greater number of wake after sleep onset, greater number of wake greater than 15 seconds and greater wake index per hour in BC patients than in controls. Patients had lower scores than controls for EB linked intentions and performed worse than controls for processing speed, executive functioning, retrospective episodic memory, and binding. Awakening parameters correlated positively with TB intentions scores and negatively with executive functioning scores in patients only.

Our results provide support for sleep disruptions, PM and executive functioning difficulties in BC patients treated with hormonal therapy. Larger awakenings seem to be related to both PM and executive functioning performances in patients as suggested by correlations.
Microglia-dependent GABAAR plasticity during sleep

Maria Joana Pinto ∗ 1, Lucy Bizien 1, Valentin Lepetz 1, Antoine Triller 1, Alain Bessis 1

1 Institut de biologie de l’ÉNS Paris (UMR 8197/1024) – École normale supérieure - Paris, Institut National de la Santé et de la Recherche Médicale, Centre National de la Recherche Scientifique : U1024 – France

The sleep-wake cycle is associated with global changes in synaptic strength, with potentiation during wake and depression during sleep [1]. These daily adjustments in synaptic strength have been attributed to plasticity of excitatory synapses, particularly glutamate receptors [2, 3], but little is known about inhibitory connections. The common use of sleeping pills acting on GABAARs [4] hints to the likely modulation of GABAergic inhibition through GABAARs plasticity in the sleep-wake cycle, which has however remained elusive.

In order to examine GABAARs plasticity we analyzed the accumulation of GABAARs at synapses by confocal microscopy in the prefrontal cortex of mice at different stages of their sleep-wake cycle. We found that accumulation of GABAAR at synapses increases during sleep in upper (L1) but not deeper cortical layers (L5). Notably, this form of GABAARs plasticity during sleep resembles an in vitro paradigm of long-term potentiation of inhibitory synapses (iLTP), which is specific to somatostatin+ interneuron inputs [5], known to induce sleep and slow wave activity [6].

Microglia, the immune cells of the brain, are gifted sensors of their surroundings. Recent work is starting to reveal their exquisite ability to sense neuronal activity and tune synapses [7]. Therefore, we explored the involvement of microglia in GABAARs plasticity by inducing their depletion with PLX3397. Synaptic enrichment of GABAARs, both during sleep and iLTP, was abolished upon such treatment. Together, our findings attribute a novel role for microglia in modulating cortical GABAergic postsynaptic plasticity, likely to control network activity during sleep.


∗Speaker

Reciprocal interaction of hippocampal ripples and cortical slow waves leads to coordinated spiking activity during NREM Sleep

Pavel Sanda *, Paola Malerba, Xi Jiang, Giri Krishnan, Sydney Cash, Eric Halgren, Maxim Bazhenov

1 Institute of Computer Science of the Czech Academy of Sciences – Czech Republic
2 Department of Medicine, University of California, San Diego – United States
3 Department of Cognitive Sciences, University of California Irvine – United States
4 Neurosciences Graduate Program, University of California at San Diego – United States
5 Canadian Centre for Behavioural Neuroscience, University of Lethbridge – Canada
6 Departments of Neurology, Massachusetts General Hospital and Harvard Medical School – United States
7 Department of Radiology, University of California, San Diego – United States

The dialogue between cortex and hippocampus is known to be crucial for sleep dependent memory consolidation. During slow wave sleep, memory replay depends on slow oscillations (SO) and spindles in the (neo)cortex and sharp wave-ripple complexes (SWR) in the hippocampus. The mechanisms underlying interaction of these rhythms are poorly understood. We examined the interaction between cortical SOs and hippocampal SWRs in a model of the hippocampocortico-thalamic network and compared the results with human intracranial recordings during sleep. We observed that ripple occurrence peaked following the onset of SO and that cortical input to hippocampus was crucial to maintain this relationship. A small fraction of ripples occurred during the Down-state and shaped initiation of the next Up-state. We observed that the effect of ripple depends on its precise timing which is in line with the idea that ripples occurring at different phases of SO might serve different functions, particularly in the context of encoding the new and reactivation of the old memories during memory consolidation. The study revealed complex close-loop interaction of SWR and SO in which early hippocampal ripples influence transitions to Up-state, while cortical Up-states control occurrence of the later ripples, which in turn influence transition to Down-state.

*Speaker
Is cataplexy a dissociated state of paradoxical (REM) sleep? Role of the GABA/Glycinergic neurons of the ventromedial medulla in brainstem in a mouse model of narcolepsy type 1

Manon Villalba *, Alexis Roman 1, Anne-Laure Morel 1, Christelle Peyron 1

1 Center for Research in Neuroscience of Lyon (CRNL) – CNRS : UMR5292, Institut National de la Santé et de la Recherche Médicale - INSERM : U1028 – France

Narcolepsy type 1 is a chronic neurology disorder due to degeneration of the hypocretin/orexin neurons of the lateral hypothalamus. It is characterized by excessive daytime sleepiness and episodes of cataplexy. Cataplexies are depicted by a bilateral loss of muscle tone during wakefulness that is triggered by a strong positive emotion, without loss of consciousness. Because of the many common points between paradoxical sleep (PS) muscle atonia phenotype and cataplexy, it has been classically proposed that the neuronal network recruited for the expression of muscle atonia during PS would also be involved during cataplexy. However, it has never been demonstrated. The ventromedial medulla (VMM) in the brainstem, where are located GABAergic and glycinergic inhibitory pre-motoneurons responsible for muscle atonia during PS, contains neurons active during PS and also during cataplexy in narcoleptic dogs (Siegel et al, 1991). We thus hypothesize that these GABAergic/glycinergic pre-motoneurons would play a key role during cataplexy. To test this hypothesis, we abolished specifically and permanently the GABA/Glycinergic transmission in the VMM of narcoleptic Orex-KO mice (deficient in hypocretin/orexin), using short-hairpin RNA method (shRNA) directed against vGAT the vesicular transporter of GABA and Glycine. Mice were injected with the experimental (shVGAT) or a control (shCTRL) AAV, locally in the VMM, and implanted with electrodes for polysomnographic recordings. Vigilance states, loss of muscle atonia during PS and cataplexies are evaluated with polysomnographic and video recordings in baseline condition and in a protocol of cataplexy induction with chocolate at 1, 4, 6 and 8 weeks after viral injection. Our preliminary results indicate that blockage of the VMM GABA/Glycinergic transmission induced episodes of REM sleep without atonia in shVGAT mice as expected. Furthermore, cataplexy seemed to be reduced in its total amount or bouts number, in shVGAT mice compare to shCTRL, and this during baseline condition or after cataplexy-induced protocols. These preliminary data need to be confirmed but suggest that GABA and Glycinergic pre-motoneurons of the VMM might be involved in cataplexy.

*Speaker
EREMAD project : Epileptiform activity during REM sleep in Alzheimer Disease

Lionel Dahan * 1

1 Centre de Recherches sur la Cognition Animale [Toulouse] (CRCA) – CNRS : UMR5169, Université Paul Sabatier [UPS] - Toulouse III, Université Paul Sabatier (UPS) - Toulouse III – UFR - S.V.T 118 route de Narbonne 31062 TOULOUSE CEDEX 4, France

We showed that Tg2576 mouse model of Alzheimer Disease (AD) present epileptiform activity specifically during sleep, with a prominent increase during REM-sleep. This phenotype is specific to AD mice since REM-sleep usually prevents seizures and epileptiform activity. Thus, epileptiform events during REM-sleep, if present in patients, could be used as a specific biomarker of AD. We thus launched a clinical monocentric study aiming at evaluating seizures and epileptiform activity during sleep in 40 patients at early to moderate stages of AD and 40 matched healthy participants. Participants undergo a high resolution MRI scan and a neuropsychological evaluation including episodic memory tests before an overnight video-EEG session which is followed by a test for memory consolidation. This should allow precise the incidence of sleep related epileptic events in AD patients and to correlate these events cognitive decline and/or anomalies in brain structure and functional resting state connectivity.

*Speaker
Histaminergic control of cortical activity during wakefulness and sleep

Francois David *, Amandine Ferret 1, Manon Villalba 1, Régis Parmentier 1, Paul A. Salin 1, Christelle Peyron 1, Gaël Malleret 1, Jian-Sheng Lin 1, Luc J. Gentet 1

1 Lyon Neuroscience Research Center – Université Claude Bernard Lyon 1, Institut National de la Santé et de la Recherche Médicale : U1028, Centre National de la Recherche Scientifique : UMR5292 – France

Histaminergic neurons of the TMN (Tuberomammillary Nucleus) constitute a major component of the ascending activating system and promote arousal and maintenance of wakefulness. However, it remains unknown how their firing activity affects neuronal cortical dynamics during wake-specific tasks and/or different vigilance states. Here, using DREADD pharmacogenetic transgenic mouse models, we investigated the impact of TMN histaminergic neuronal activation/inactivation, at the level of both, behavioral performance and cortical dynamics of neuronal ensembles. Activating or inhibiting histaminergic TMN neurons reliably promoted wakefulness or sleep durations respectively. On the other hand, in awake mice, both activation and inactivation of TMN neuronal activity did not impair their cognitive performance on a T-maze choice task, but increased their latencies under the latter condition. Putative suppression of TMN neuronal activity led to reduced EEG power spectral amplitudes in the 4-100Hz frequency range during both wakefulness and sleep, while TMN activation had little impact. These effects were accompanied by a significant silencing of cortical neuronal ensemble activity in both the somatosensory and prefrontal cortices. Furthermore, intracellular recordings revealed a significant reduction in membrane potential amplitudes of UP- and DOWN-state fluctuations. DOWN- and UP-state durations respectively increased and decreased, while action potential firing was considerably reduced during TMN neuronal inactivation.

Taken together, our results indicate that disturbing the arousal system via modulation of histaminergic neuronal activity creates an oscillatory imbalance at the level of cortical dynamics which may impair some aspects of cognitive performance and which extends over subsequent sleep periods. Whether TMN histaminergic neurons exert their control over cortical dynamics through direct projections, or via indirect pathways (eg: thalamic neuronal regulation) remains to be determined.
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