Human and Animal cognition

Société des Neurosciences

From behavioral observation to underlying neuronal mechanisms

26 · 27 May 2026
CBI, Toulouse

Program

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26 May 2026

9:00 - 9:30 — **Coffee and opening remarks**

9:30 - 10:30 — **Plenary lecture**

Julien Bastin (GIN, Grenoble, France)
How the human brain learns and chooses: evidence from intracerebral recordings

10:30 - 12:30 — **Symposium - Learning signals and processes**

Chaired by **Lionel Dahan** (CRCA, Toulouse) and **Mehdi Senoussi** (CLLE, Toulouse)

Antoine Bergel (ICM, Paris)
Hippocampus-cortex communication during sharp-wave ripples observed at the whole-brain scale with functional ultrasound imaging

Elsa Karam (IGF, Montpellier)
Differential temporal dynamics of distinct dopaminergic neuronal populations during contextual fear extinction learning

Jacqueline Scholl (CRNL, Lyon)
Gamified tasks, emotions and decision-making

Jonathan Curot (CerCo / CHU, Toulouse)
Are We Overlooking a Building Block of Memory? The Posterior Cingulate Cortex

Adrien A. Causse (University of Oxford, Oxford, United Kingdom)
A learning-evoked slow-oscillatory architecture paces population activity for offline reactivation across the human medial temporal lobe

12:30 - 14:00 — **Lunch and poster session**

14:00 - 16:00 — **Symposium - Exploring the Neural and Cognitive Foundations of Social Interactions**

Chaired by **Vincent Fourcassié** (CRCA, Toulouse)

Rui F. Oliveira (ISPA, Lisbon, Portugal)
Of (zebra) fish and flies: studying the evolution of social cognition in two model organisms

Philippe Faure (ESPCI, Paris)
Dopaminergic modulation of social specialization in mouse micro-societies

Guillaume Dumas (Ste Justine, Montreal, QC, Canada)
From Dyadic Coupling to Social Networks: Multiscale Neural Mechanisms of Social Interaction

Julie Carcaud (EGCE, Paris)
Neural bases of pheromonal communication in the honey bee

Guillaume Bouisset (Miguel Hernández University, Alicante, Spain)
Cortical geometry of neural representations of social preference

16:00 - 16:30 — **Coffee break**

16:30 - 17:30 — **Plenary lecture**

Ewelina Knapska (Nencki Institute of Experimental Biology, Warsaw, Poland)
Emotional echoes: neural mechanisms by which others' emotions shape adaptive behavior

27 May 2026

8:30 - 9:30 — **Plenary lecture**

Aya Goldshtein (Max Planck Institute of Animal Behavior, Konstanz, Germany)
Decision-making in the wild

9:30 - 10:30 — **Coffee break and poster session**

10:30 - 12:30 — **Symposium - Positive or deleterious effects of the exposome on cognition**

Chaired by **Elsa Suberbielle** (Infinity lab, Toulouse)

Yvonne Nolan (Cork University, Ireland)
Lifestyle factors and the gut-brain axis: relevance to Alzheimer's

Elsa Suberbielle (Infinity lab, Toulouse)
Pathogen's driven chronic low grade inflammation and neuroimmune mechanisms behind cognitive impairment

Enikő Csata (CRCA, Toulouse)
Collective self-medication in ants

Lucile Capuron (NutriNeuro, Inrae, Bordeaux)
The role of diet in regulating immune function and mental health

Maxime Linard (Institut des Biomolécules Max Mousseron - CNRS, Montpellier, France)
Bees, insecticides and neurodeveloppement: affected even before the first flower is visited?

12:30 - 14:00 — **Lunch**

13:30 - 14:00 — **General Assembly of the French Neuroscience Society**

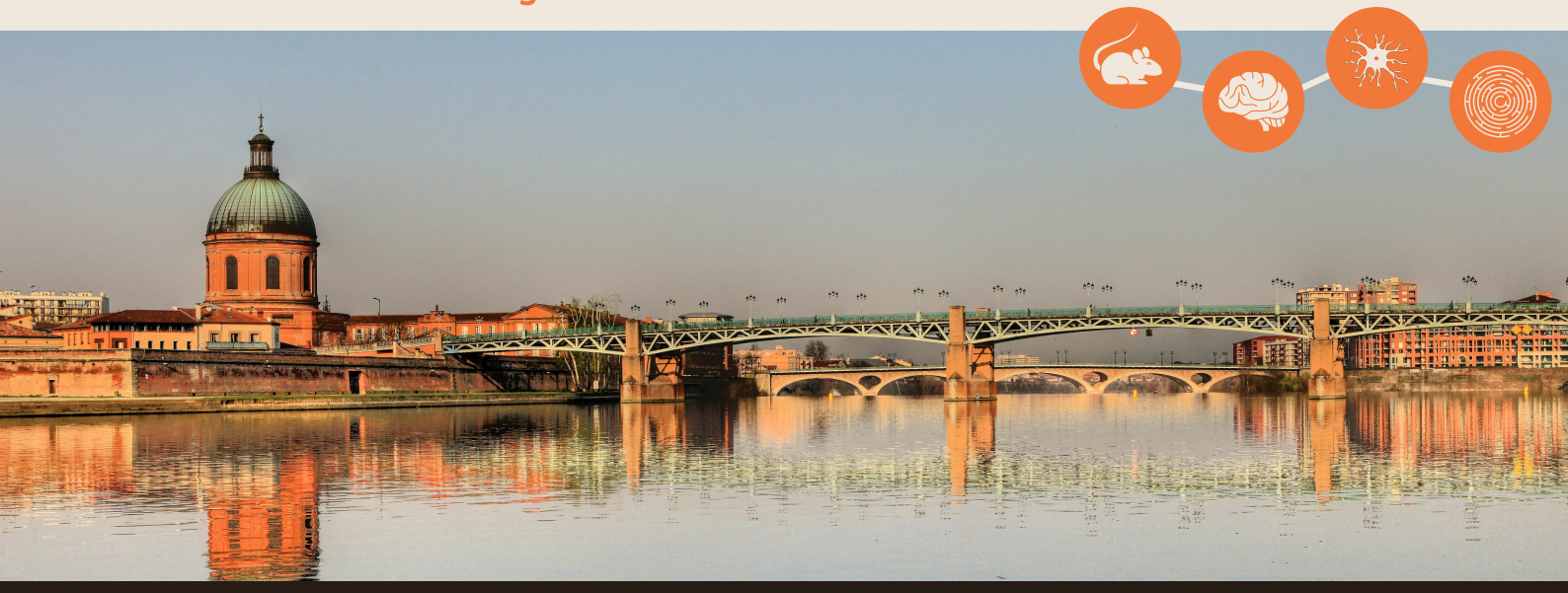
14:00 - 15:00 — **Alfred Fessard Lecture**

Claire Rampon (CRCA, CBI, Toulouse)
How adult neurogenesis changed our understanding of memory

15:00 - 16:30 — **Roundtable - How to address the ecological validity of our research?**

Chaired by **Mathieu Lihoreau** (CRCA, Toulouse)

16:30 — **Concluding remarks**



Poster session

Poster #1
From neural circuits to behavior: Sweeteners reshape feeding in Drosophila

Enisa Aruci, Pierre-Yves Musso

Centre des Sciences du Goût et de l'Alimentation, Univ. Bourgogne - CNRS - INRAe - Institut Agro, Dijon, France

Sweeteners are commonly used as sugar substitutes because they provide sweetness without, or with very little, caloric content. Although widely consumed, their effects on feeding regulaBon remain insufficiently understood.
In particular, the uncoupling between sweet taste perception and caloric intake has emerged as an important factor influencing feeding behavior across species, from insects to humans. Using Drosophila melanogaster as a model organism, we investigated how sweeteners affect feeding behavior, metabolic responses and the neuronal circuits involved in food evaluation. To do so, we combined two complementary behavioral approaches: the Proboscis Extension Response (PER), to assess feeding initiation and the fly Activity Detector (flyPAD), to quantify feeding dynamics with high temporal resolution.
Our data show that sweetener consumption induces major alterations in feeding behavior. Notably, it promotes the formaBon of Sweet Taste Devaluation(STD) caused by the uncoupling between sweet taste perception and the expected caloric reward. This experience subsequently leads to abnormal feeding responses, suggesting that sweeteners can durably alter food valuation. These findings support the idea that sweet taste-calorie uncoupling not only affects immediate behavior, but also effects the integration of sensory and post-ingestive signals within the brain.
This work identifies Drosophila as a relevant model to study the biological impact of sweeteners and provides new insight into how non-caloric sweet compounds influence feeding through behavioral and neural mechanisms.

Poster #2
Does the social environment influence behaviour in a Drosophila model of Alzheimer's disease?

Mariana Da Silva Malheiro

Université de Toulouse, Centre de Recherches sur la Cognition Animale, EXPLAIN, Toulouse, France

Alzheimer's disease (AD) is a neurodegenerative disorder that leads to behavioural deficits in some patients, including reduced motor skills. This disease is characterised by the accumulation of the Aβ protein, the most toxic form of which is the Aβ42 peptide. Studies have shown that loneliness increases the risk of developing AD, while staying mentally active and participating in social activities may help prevent cognitive decline and delay the onset of the disease. Currently, no medication has been shown to be effective in preventing the progression of AD. It is therefore important to identify factors that may delay certain behavioural symptoms.
Using an animal model of AD, we aim to understand the effect of Aβ42 at the cellular level in relation to locomotor activity, and to show how this effect may be mediated by the social environment. Drosophila provides an effective model for this study, with a wide range of

behaviours that are easy to study, and numerous genetic tools that allow the manipulation of gene expression and neuronal activity. In addition, Drosophila has already been used to study the relationship between Aβ42, amyloid plaques and memory, making it a particularly relevant model.
Our results show that the expression of Aβ42 in the mushroom bodies, the main center of learning and memory in insects, reduces locomotor activity when Drosophila males are tested alone, but increases locomotor activity when they are tested in groups with conspecifics. Our results indicate that social conditions may interact with the Aβ42-mediated locomotion deficit.

Poster #3
Decoding Human Intracranial Stimulation: Cognitive Responses Are Driven by Long-Range Single-Unit Dynamics

Aube Darves-Bornoz[1], Laure Balguerie[1,2], Loïs Oundjian[1], Mathis Grosjean[1], Idaya Fauser[1], Tia Girard[1], Marie Denuelle[1,2], Amaury De Barros[2], Jean-Albert Lotterie[2], Luc Valton[1,2], Emmanuel Barbeau[1], Jonathan Curot[1,2]

[1]Brain and Cognition Research Center (CerCo), Centre National de la Recherche Scientifique, UMR5549, Toulouse, France
[2]Departments of Neurology and Neurosurgery, Toulouse University Hospital, Toulouse, France

Electrical brain stimulation (EBS) is widely used during stereo-EEG to study brain functions and identify epileptic networks in humans. However, cognitive responses (memories, language disturbances...) remain unpredictable, and underlying neuronal mechanisms across spatial scales are poorly understood. While local-field potential studies show that EBS engages distributed networks, with connectivity increasing with cognitive complexity, corresponding dynamics at the single-unit level remain unknown. Growing evidence shows that local neuronal events can exert functionally specific effects on remote brain regions. We therefore investigated how EBS modulates neuronal activity from local microcircuits to distant regions. We hypothesized that cognitive effects arise from specific neuronal dynamics, locally and remotely, shaped by the type of cognitive response, EBS sites, and neuron–EBS distance. Single-unit activity was recorded using depth electrodes with tetrodes in 63 human subjects undergoing clinical EBS. EBS were classified as “Cog+” (cognitive effects, with subtypes), “control” (non-cognitive responses), or “negative”. For each EBS–single-unit pair, we computed responsiveness (Wilcoxon tests, 10 s pre-/post-EBS), and normalized firing rates (FRz, FRz(t)). Temporal dynamics were analyzed across response types and distance. We identified 363 EBS in 31 patients. Preliminary analyses in 11 patients (134 EBS, 125 single-units, 849 pairs) revealed heterogeneous neuronal responses—both excitation and inhibition—across all conditions and distances. Some responses were detected only at micro-scales. FRz were more negative locally than remotely. Additionally, specific cognitive phenomena were associated with regionally-selective activation patterns. Findings suggest that EBS-induced cognitive effects arise from distributed, distance-dependent neuronal modulation, linking local microcircuits to large-scale networks.

Poster #4

What are the relationships between the temporal pole and the hippocampus in humans? Effective connectivity using cerebro-cerebral evoked potentials

Vincent Dornier[1], Maël Lavigne-Stieremans[1], Caroline Stamm[1], Luc Valton[1,2], Marie Denuelle[1,2], Jean-Albert Lotterie[3,4], Amaury de Barros[3,4], Annabelle Goujon[5], Jonathan Curot[1,2], Emmanuel J. Barbeau[1]

[1]CerCo, Univ Toulouse, CNRS, Toulouse, France
[2]Brain Electrophysiology, Epilepsy and Sleep Unit, Neurology Department, Toulouse University Hospital, Toulouse, France
[3]Toulouse Neuro Imaging Center, INSERM, UMR 1214, Toulouse, France
[4]Department of Neurology and Neurosurgery, Toulouse University Hospital; Toulouse, France
[5]Université Marie & Louis Pasteur, UMR 1322 INSERM, LINC, Besançon, France

Models of declarative memory have long focused on the hippocampus. However, it is the temporal pole that is considered the key region for the storage of semantic representations. Humans may therefore have two hubs underlying declarative memory rather than only one as is usually thought. However, information regarding the interactions of these two hubs is scarce or contradictory. Some models of semantic memory do not even refer to the hippocampus. We therefore sought to determine precisely the interactions between these two structures. We studied cerebro-cerebral evoked potentials (CCEP) obtained via intracranial recording in 26 patients with drug-resistant epilepsy. Using the latest data-driven method (Canonical Response Parametrization, Miller et al., 2023), we demonstrate that the medial, but not the lateral, temporal pole is functionally connected to the hippocampus. We also show that the relationship between the medial temporal pole and the hippocampus is bidirectional, but with a preferential connection from the temporal pole to the hippocampus, as revealed by CCEP latencies. These results were replicated in an independent analysis using a large database of 613 patients (F-tract database, Jedynak et al., 2023). Intriguingly, we did not find any relationships between interhemispheric temporal poles or hippocampi in neither analysis at the latencies of CCEP (<200 ms). Overall, these results demonstrate robust intrahemispheric effective connectivity between the medial temporal pole and the hippocampus in humans, with a stronger and more rapid influence of the medial temporal on the hippocampus. This is intriguing given that no model clearly incorporates these two regions.

Poster #5

Selective Modulation of Nicotinic Acetylcholine Receptors in the Prefrontal Cortex in the Control of Behavioral Symptoms of Autism Spectrum Disorder

Manuel Felline[1,2], Ana Paula Rodrigues de Paulo[1,2], Helena Janíčková[1]

[1]Institute of Physiology of the Czech Academy of Sciences, Prague, Czech Republic
[2]Charles University, Prague, Czech Republic

Nicotinic acetylcholine receptors (nAChRs) play key roles in cognitive and behavioral regulation and are implicated in neurodevelopmental disorders such as autism spectrum disorder (ASD). However, their widespread distribution across the brain complicates

therapeutic targeting. The prefrontal cortex (PFC), essential for social behavior and cognitive control—both disrupted in ASD—contains diverse neuronal populations with distinct connectivity, physiology, and functions. This project tests the hypothesis that selective modulation of nAChRs in specific PFC neuronal populations can reverse ASD-related behavioral symptoms, including social impairment and cognitive inflexibility. We focus on three neuronal types: glutamatergic (VGLUT1+) neurons, VIP interneurons, and Chandelier cells, examining how α7 and β2 nAChR subunits influence behavior in BTBR mice (ASD mouse model). With RNAscope we will map nAChR expression across these populations. We will then selectively manipulate nAChRs subunits using VGLUT1-Cre–driven AAVs for glutamatergic neurons and enhancer-specific-driven AAVs for VIP and Chandelier cells. Following validation of targeting specificity, we will perform knockdown (via shRNA) or overexpression (via CRISPR-dCas9 activation) of α7 and β2 nicotinic subunits. Behavioral assessments conducted before and after genetic manipulation will evaluate cognition, anxiety-like behavior, repetitive behavior and social interaction. These experiments aim to define how cell-type-specific modulation of nAChRs affects ASD-relevant behaviors in BTBR mice. Preliminary data show enrichment of α7 and β2 subunits in VIP interneurons relative to other PFC cell populations. We also demonstrate efficient AAV targeting of Chandelier cells in the PFC and confirm distinct behavioral phenotypes between BTBR and wild-type mice, particularly in terms of locomotor activity, cognition and exploratory behavior.

Poster #6

Role of neuronal DNA double-strand break signaling in engram reactivation during memory recall

Yoann Fraysse, Elisa Roitg, Maïlys-Cassy Baudet, Mathilde Periou, Elsa Suberbielle

Institut INFINITY, Inserm UMR1291, CNRS 5051, Université Toulouse 3, Toulouse, France

Background: Neuronal DNA double-strand breaks (DSBs) exhibit a dual function in cognitive processes. Physiologically, transient DSBs are essential for the expression of immediate-early genes governing learning (Suberbielle et al., 2013; Madabhushi et al., 2015). Conversely, in Alzheimer's disease (AD), chronic neuroinflammation exacerbates DSB accumulation, and the associated H2A.X signaling drives cognitive deficits (Belloy et al., 2025). While ablating H2ax prevents inflammation-induced spatial consolidation deficits, our preliminary data suggest it may also compromise long-lasting memory. We hypothesize that H2A.X signaling is critical for the accurate reactivation of the memory trace during recall.

Methods: We employed a viral "tag-and-manipulate" strategy in mice. We used a cFos-dependent Tet-Off system to tag active hippocampal neurons with an excitatory DREADD-mCherry reporter.

Experimental Design: H2ax neuron KO vs WT mice were taken off Doxycycline shortly prior to the encoding phase of a Novel Object Recognition task. This window allows the historic tagging of the "encoding engram" (mCherry+). Doxycycline was immediately restored post-encoding. Memory retention was assessed at several points. Brains were collected to immunolabel endogenous cFos (retrieval activity). We calculated the engram reactivation index by quantifying the overlap between the encoding (mCherry+) and retrieval (cFos+) ensembles.

Conclusion: These ongoing experiments aim to determine if DSB signaling is required for the stability and natural accessibility of memory engrams.

Bibliography:
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R. Madabhushi … L.-H. Tsai https://doi.org/10.1016/j.cell.2015.05.032
E. Suberbielle … L. Mucke https://doi.org/10.1038/nn.3356

Poster #7

Investigating the role of the gut microbiota in honey bee brain maturation

Anais Garreau[1], Marlène Botella[2], Laurie Thouvenel[2], Grégoire Pasquier[2], Amélie Cabirol[2]

[1]Université de Toulouse - Centre de Biologie Intégrative, Centre de Recherches sur la Cognition Animale, CNRS UMR 5169, Toulouse, France
[2]Centre de Biologie Intégrative, Centre de Recherches sur la Cognition Animale, Toulouse, France

The gut microbiota plays an important role in brain functions via the gut microbiota–brain axis and is increasingly studied in different models. The honeybee Apis mellifera, is a relevant model due to the simplicity of its microbiota and its advanced cognitive abilities. It has already been shown that microbiota-deprived bees have deficits in social behavior, learning and memory, but the neural mechanisms are poorly understood. Furthermore, these studies were mostly conducted under controlled laboratory conditions.
We sought to determine whether the gut microbiota impacts brain maturation in bees and developed a novel semi-ecological rearing environment to favor natural brain maturation in a complex social and sensory context.
Honeybees raised in mini-hives with access to the outside foraging environment were divided into three treatment groups: newly emerged bees (NB), microbiota deprived (MD) and colonized with the natural gut microbial community (CL). Five brains were sampled at emergence (NB group) and 7 days post-emergence for synapsin immunostaining in whole mount-brains.
Confocal microscopy images were acquired and allowed volumetric measurements of the olfactory and visual neuropils of the mushroom bodies as well as the quantification of synaptic boutons in these regions.
We did not find a significant effect of the microbiota on brain maturation, but a significant variability between the two experimental replicates. These results suggest that an ecological environment exposes bees to uncontrolled environmental factors and call for additional replication. This study highlights the importance of considering the ecological context in studies on gut microbiota–brain interactions.

Poster #8

Maternal stress drives long-term offspring behavioral deficits through corticosterone-dependent developmental perturbations of nociceptors

Nadine Gheziel[1,2], Tiphaine Voisin[1], Célia Changenot[1], Nasser Salem Abdullah[1], Celine El Samrout[1,2], Alexia Loste[1], Nadine Serhan[1], Chloé Granat[3], Anne-Alicia Gonzales[1], Ruçhan Ekren[1], Elisa Roitg[1], Elsa Suberbielle[1], Marthe Moldes[4], Elodie Labit[1], Amaury François[5], Nicolas Cenac[2], Nicolas Gaudenzio[1], Lilian Basso[1]

[1]University of Toulouse, Inserm, CNRS, Toulouse Institute for Infectious and Inflammatory Diseases (Infinity), Toulouse, France
[2]Institut de Recherche en Santé Digestive (IRSD), Université de Toulouse-Paul Sabatier, INSERM, INRAe, ENVT, UPS, Toulouse, France
[3]Institut de Génomique Fonctionnelle (IGF), Université de Montpellier, CNRS, INSERM, Montpellier, France
[4]Centre de recherche Saint-Antoine, UMR_938, Inserm, Sorbonne université, Paris, France; Institute of CardioMetabolism and Nutrition, ICAN, Inserm, Sorbonne université, Paris, France
[5]Institut de Génomique Fonctionnelle (IGF), Université de Montpellier, CNRS, INSERM, Montpellier, France

Neurodevelopmental disorders (NDDs), including autism spectrum disorder (ASD), represent a major public health concern, affecting approximately 1 in 100 children worldwide. These disorders are characterized by impairments in social behavior and are frequently associated with sensory dysfunctions. Prenatal stress has emerged as a significant environmental risk factor for NDDs, yet the underlying biological mechanisms remain poorly understood.

Recent evidence suggests that peripheral sensory neurons, particularly nociceptors, may play a role in shaping behavioral outcomes associated with NDDs. We hypothesized that prenatal stress induces early and persistent alterations in nociceptor development and function, contributing to sensory and behavioral abnormalities.

Using a murine model of chronic prenatal stress, we show that offspring exhibit significant transcriptomic alterations across multiple nociceptor populations. These changes are detectable at birth and persist into adulthood, indicating that prenatal stress disrupts developmental programs in utero. Notably, several dysregulated genes are associated with NDDs. Behavioral assessments reveal that prenatally stressed mice display mechanical hypersensitivity, reduced social interactions, repetitive behaviors, neophobia, and anhedonia.

Mechanistically, we demonstrate that glucocorticoid signaling is a key driver of these effects. Conditional deletion of the glucocorticoid receptor Nr3c1 specifically in nociceptors prevents several behavioral and sensory deficits induced by prenatal stress. Furthermore, pharmacological modulation of the GABAergic system restores normal behavioral responses in stressed offspring.

Altogether, our findings identify a novel peripheral mechanism linking prenatal stress to NDD-related phenotypes and highlight nociceptors as key contributors and potential therapeutic targets.

Poster #9
Neuronal representation of birdsong variability signals in a Basal Ganglia–Cortical circuit for vocal learning

Carmen Guerrero Marquez, Julien Braine, Arthur Leblois

Institut des Maladies Neurodégénératives - CNRS UMR 5293, Université de Bordeaux, Bordeaux, France

The basal ganglia–thalamo–cortical (BGT) circuit is critical for motor skill acquisition across vertebrates, including speech learning in humans, a process thought to rely on reinforcement learning (RL). Songbirds provide a powerful model for studying skill learning, as they acquire their songs by imitating adult tutors through trial and error. Their specialized song system, comprising both a BGT loop and a cortical motor pathway, underlies song learning and production.

Within this circuit, the lateral magnocellular nucleus of the anterior neostriatum (LMAN) acts as the cortical output of the BGT loop, introducing variability into song production through projections to the motor pathway. RL models propose that LMAN conveys an efference copy of these vocal variations to Area X, the songbird basal ganglia nucleus homologous to mammalian motor BG circuits. Yet, the functional organization of LMAN and Area X, as well as the neuronal coding of vocal variability, remain poorly understood.

To address this, we examined functional connectivity and neural dynamics in LMAN and Area X during singing, using chronic and acute in vivo electrophysiology in zebra finches. Our analyses revealed no straightforward linear correlations between single-neuron activity in these regions and acoustic song features. However, population-level analyses and nonlinear modeling may capture more complex relationships. In parallel, we are mapping functional connectivity from LMAN to Area X to estimate the neural population size required for encoding vocal variability. Together, these findings shed light on how BGT circuits represent vocal features and provide broader insights into sensorimotor learning and speech acquisition mechanisms.

Poster #10
Effect of social living environment on sleep and memory in rodents

Iris Guggenbuhl, Sébastien Arthaud, Nadine Ravel, Philippe Litaudon, Christelle Peyron

Centre de Recherche en Neurosciences de Lyon (CRNL), CNRS UMR5292, Inserm U1028, UCBL, UJM, Bron, France

In rodents, sleep is traditionally studied in isolated impoverished laboratory conditions, far removed from their natural social living environment. However, memory consolidation is supported by sleep and the living environment is known to influence cognition, behavior, and physiology of rodents, especially their memory. Previous ecological evaluation of sleep/activity have used actimetry which does not allow analysis of the precise architecture of the different states of the sleep-wake cycle. To tackle this challenge, we used newly developed biologgers (Manitty), that allow us to simultaneously record a colony of 20 rats

moving freely in a unique 2m2 naturalistic living environment (NLE). This NLE provides 20 rats with a rich social environment, allowing them to express most of their global behavioral repertoire and exercise control over their environment. Rats were implanted with a network of electrodes (EEG, EOG, EMG) connected to a biologger, meaning that sleep was recorded continuously and simultaneously on the 20 rats without disturbing natural behaviors of the colony. To study the impact of living environment, we performed a longitudinal study where animals were first recorded in a conventional laboratory environment (2 rats/cage for 96 hrs) and then transferred to NLE for 3 months. Sleep was evaluated immediately after transfer in the NLE for 48hrs and again 6 weeks later. To monitor the rats' social life, the space is equipped with an automated behavioral tracking system (Live Rat Tracker, https://livemousetracker.org/) and radio frequency identification antennae for individual tracking. Our preliminary results show that animals in the naturalistic environment sleep less than in the conventional environment during the light phase. Data reporting the impact of the environmental conditions on sleep and spatial memory, will be presented. This innovative approach will enable us to better understand the impact of housing conditions, and in particular the social dimension, on the quantity and quality of sleep and its consequences on memory performance.

Poster #11

Distinct contributions of dorsomedial and ventromedial prefrontal cortices to attentional behavior

Laëtitia Lageyre, Rémi Proville, Karine Guillem

University of Bordeaux, CNRS, INCIA, UMR 5287, Bordeaux, France

In order to survive in an ever-changing world, organisms must adapt their behavior optimally. In this context, attention is a fundamental adaptation mechanism, enabling the allocation of mental resources to salient environmental stimuli, while filtering out distractions. The medial prefrontal cortex (mPFC) plays a critical role in attentional control, yet its functional organization and underlying mechanisms remain partially understood. In particular, the respective contributions of the dorsomedial (dmPFC) and ventromedial (vmPFC) prefrontal cortices in attentional processes, as well as their neuromodulatory regulation, require clarification. To address this issue, we used fiber photometry recordings in rats performing a 5-choice serial reaction time task (5-CSRTT), a well-established paradigm for assessing attentional performance. Our results reveal a functional dissociation between these two subregions: the vmPFC being preferentially activated during light stimulus detection and successful execution (correct responses), while the dmPFC is activated during error signaling following incorrect, omission and premature responses, suggesting a role in performance monitoring. Given the pivotal role of acetylcholine (ACh) in attentional modulation, we next investigated whether ACh signaling contributes to these region-specific patterns using a genetically encoded cholinergic sensor. We found that ACh release increased during stimulus detection and correct responses in both subregions, while no changes in ACh levels were observed after incorrect, omission or premature responses, suggesting that ACh signaling is specifically linked to successful attentional processing. Together, these findings provide a multilevel characterization of attentional processing in the mPFC, highlighting how distinct subregions and their neuromodulatory control contribute to different aspects of attention.

Poster #12

Dorso-ventral hippocampal reactivation increase during sleep following an aversive experience

Juan Facundo Morici, Azul Silva, Izabela Lima-Paiva, Eleonore Pronier, Gabrielle Girardeau

Institut de biologie Paris-Seine, NeuroSU, Sleep and Emotional Team, Paris, France

Our brains continuously generate internal representations of the environment, integrating spatial and emotional information. The hippocampus plays a central role in this process, particularly during sleep, a reversible state critical for memory stabilization across species. Within the dorsal hippocampus (dHPC), sharp-wave ripples (SWRs) during non-REM sleep facilitate spatial memory consolidation through place cell reactivation. The hippocampal formation is functionally heterogeneous: the dHPC primarily processes contextual information, whereas the ventral hippocampus (vHPC) encodes emotional valence and anxiety. Although the role of sleep in processing emotional experiences in the dHPC has been extensively studied, the contribution of the vHPC and its coordination with the dHPC in the sleep-dependent processing of aversive experiences remains unexplored. We propose that communication between these regions during sleep supports the consolidation of emotional memories by integrating or segregating contextual and emotional content. To test this, we recorded electrophysiological activity from the dHPC and vHPC in rats during reward- and aversion-motivated exploration of a linear track followed by sleep. Analysis of hippocampal coordination at the neuronal level revealed dorsal, ventral, and joint (dorso-ventral) neuronal assemblies. Notably, joint aversive assemblies showed stronger reactivation during non-REM sleep than reward-related assemblies, with their reactivation preferentially associated with coordinated dorso-ventral SWRs. These results highlight the critical role of dorsal-ventral hippocampal communication in reactivating emotionally salient experiences during sleep, potentially sustaining the increased consolidation of aversive memories.

Poster #13

Low-grade chronic hippocampal inflammation promotes microglial reactivity and blunts exercise-induced neurogenesis in rats

Sarah Nicolas

Université de Toulouse - Centre de Biologie Intégrative, Centre de Recherches sur la Cognition Animale, CNRS UMR 5169, Toulouse, France

Adult hippocampal neurogenesis (AHN), the ability of the hippocampus to generate new neurons across the lifespan, maintains memory and the regulation of emotion. The pro-inflammatory cytokine interleukin-1β (IL-1β) is elevated in the hippocampus with age and Alzheimer's disease and reduces AHN and impairs memory. Conversely, exercise enhances AHN and associated behaviours in healthy rodents, however its effects in an inflamed hippocampus remain unclear. Here, we investigated the impact of chronic hippocampal IL-1β

overexpression and exercise on memory, AHN and microglia reactivity in adult male rats. After lentiviral delivery of IL-1β or scrambled control in the hippocampus, animals were housed with or without running wheels for 8 weeks. IL-1β impaired pattern separation in both sedentary and exercising animals. Exercise increased the number of doublecortin+ cells (AHN) in the hippocampus, an effect blunted by IL-1β. Microglial analyses revealed that exercise reduces IL-1β-associated increases in Iba+ cells and soma size, and modulated lipid droplet accumulation in the hippocampus. Transcriptomic analysis of isolated hippocampal microglia showed that exercise downregulated chemotactic and pro-inflammatory gene expression programs in control rats but upregulated immune pathways, including leukocyte migration and cytokine signalling in IL-1β-treated rats. While exercise promotes AHN and suppresses microglial inflammation under physiological conditions, prior IL-1β-induced neuroinflammation negates its proneurogenic and anti-inflammatory effects. Our findings highlight inflammatory context as a critical determinant of exercise efficacy relevant to ageing and neurodegeneration.

Poster #14

Chronic latent Toxoplasma Gondii infection precipitates cognitive decline in a TAU mouse model of dementia

Elisa Roitg[1], Mathilde Periou[1], Charlotte Paut[1], Yoann Fraysse[1], Amel Aïda[1], Emilie Bassot[1], Renzo Gutierrez-Loli[1], David Blum[2], Sabrina Marion[3], Nicolas Blanchard[1], Elsa Suberbielle[1]

[1]Institut INFINITY, Inserm UMR1291, CNRS 5051, Université Toulouse 3, ViNeDys, Toulouse, France
[2]Lille Neuroscience & Cognition (LilNCog), University of Lille, Inserm, CHU Lille, UMR-S1172, Lille, France
[3]Center for Infection & Immunity of Lille - Institut Pasteur de Lille, Lille University, Centre National de la Recherche Scientifique (CNRS) UMR9017, Inserm U1019, Centre Hospitalier Universitaire (CHU)

Neuroimmune dysregulations contribute to the pathophysiology of Alzheimer's Disease (AD). Because pathogens shape the immune system throughout life, their association with AD has been suggested. Increasing evidence supports a role for the prevalent, brain-persisting parasite Toxoplasma gondii (Tg) in chronic neurological diseases. However, our knowledge of the impact of chronicTg infection on AD pathology and clinical progression remains sparse. While Tg infection has been examined in constitutive mouse amyloidosis models, its contribution to tau pathogenesis and associated cognitive deficits has not been characterized. Given the temporal relationship between natural Tg infection and AD onset, it seems legitimate to evaluate the effects of a strain of Tg leading to latent infection, on the later development of cognitive deficits associated with tauopathy.

To tackle this question, we combined infection by transgenicTg (Tg.GRA6-OVA), which results in a persisting and subclinical infection, with a slowly progressive mouse model of tauopathy(tau).Tg-infected THY-Tau22 mouse displayed accelerated tau hyperphosphorylation and insoluble forms. Behavioral, immune and biochemical analyses were performed at a time when non-infected (NI) THY-Tau22 micehave not yet developed cognitive deficits. We found thatTg-infected THY-Tau22 mice displayed behavioral and cognitive impairments earlier than non-infected tau mice, characterized by disinhibition and impaired task learning, while spatial learning and memory remained preserved. Flow

cytometry analyses of brain-isolated immune cells revealed significant alterations in immune cell populations inTg-infected tau mice compared to non-infected controls.

Together, chronic latent Toxoplasma gondii infection accelerates tau-related cognitive decline through neuroimmune alterations.

Poster #15

Mapping microbiota-derived metabolites from the gut to the brain in honey bees

Laurie Thouvenel[1], Julie Pacull[1], Hanna Barbier[2], Floriant Bellvert[2], Simon Duthen[1], Amélie Cabirol[1]

[1]Centre de Recherches sur la Cognition Animale, CNRS, Université de Toulouse, Toulouse, France
[2]MetaToul, Toulouse Biotechnology Institute, INSA, Toulouse, France

Honey bee behaviour is under the influence of the microorganisms located in their gut and the metabolite they produce. While gut bacteria have evolved unique metabolic functions to degrade the nutrients and non-nutritive compounds ingested by the bee, few microbiota-produced metabolites have been identified for their effect on the neural functions supporting behaviour. Here, we applied an integrative metabolomics framework to investigate gut–brain metabolic communication in microbiota-deprived bees and in bees colonized with either a native microbiota or a defined synthetic community of native gut bacteria.
To characterize microbiota-dependent shifts in both gut and brain metabolism, we performed 13C-glucose labelling, enabling the tracing of carbon incorporation into metabolites. We used desorption electrospray ionization (DESI) spatial metabolomics to map the localisation of differentially regulated metabolites within the brain. By integrating these complementary approaches, we reconstruct the trajectory of microbiota-associated metabolites from their production in the gut to their detection and spatial distribution in the brain. This strategy allows us to distinguish candidate metabolites potentially transferred across compartments from those arising through local metabolic reprogramming.
Together, our results provide a systems-level view of microbiota-driven metabolic changes along the gut–brain axis and establish a framework for linking microbial metabolism to brain biochemistry in honey bees. This work advances our understanding of host–microbiota interactions and their potential impact on neural function in this key pollinator species.

Poster #16

Pregnancy induces physiological angiogenesis in the adult brain

*Gabrièle Lienhard[1], Daxiang Na[4], Daniela Fernandois[2], Julie Poisson[1], Giorgia Barra[1], Maxime Boyer[1], Thibaud Charrier[1], François-Xavier Lejeune[1], Charly Rousseau[1], Vincent Prévot[2], Sophie Pezet[3], Marc Schneeberger Pane[4], **Ahlem Assali[1*]** & Nicolas Renier[1*]*

[1] Paris Brain Institute, Paris, France
[2] Lille Neuroscience and cognition center, Lille, France
[3] Physics for Medicine, ESPCI, Paris, France
[4] Yale university, New Haven, Connecticut, USA

Pregnancy induces a wide range of plastic adaptations to the mother's brain. These include structural changes at all scales, ranging from synaptic plasticity to changes in the volumes of brain regions visible from MRI scans. The contribution of the neurovascular system to this brain plasticity has however seldomly been considered. Here, we used 3D whole-brain clearing (iDISCO), light-sheet imaging, and computational analysis tools (ClearMap), to reconstruct the evolution of the whole pregnant female mouse brain vasculature throughout gestation, at the capillary level. Gestation induced an increase in blood flow in the mPOA, an increase in the number of capillaries largely reversed after weaning, and an increase in vascular endothelial growth factor (Vegfa) expression in several brain regions, notably the mPOA and the primary somatosensory cortex. Gestation also triggered an increase in the number of endothelial tip cells in different brain regions, particularly in the mPOA, suggesting an active vascular remodeling. Estradiol implants in virgin females were sufficient to recapitulate part of gestation-induced vascular plasticity. Vegfa downregulation by shRNA in the mPOA of pregnant mice blocked the gestation-induced mPOA increase in capillaries' number and led to impairments in pup survival, pup retrieval, and nesting after delivery. Taken together, our data demonstrate that gestation induces steroid-dependent vascular plasticity in physiological conditions in brain regions critical for reproductive fitness.

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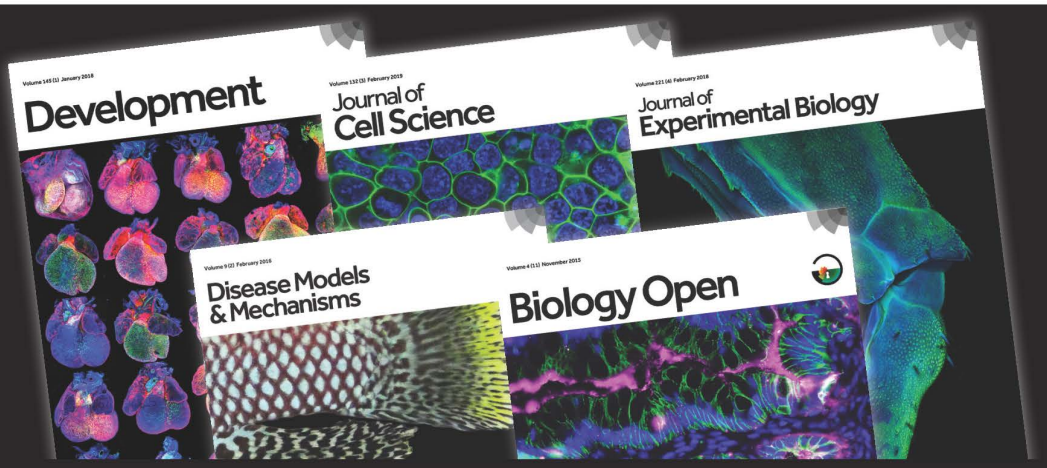

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Société des Neurosciences

Université de Bordeaux
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colloque@societe-neurosciences.fr
www.neurosciences.asso.fr