XL SAN meeting e-book

Ciudad Universitaria
Universidad de Buenos Aires
October 1st – 5th
2025

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Cognition, Behavior, and Memory
Development
Disorders of the Nervous System
Integrative Systems
Neural excitability, synaptic transmission and neuron-glia interactions
Neural Circuits and Systems Neuroscience
Neurochemistry and Neuropharmacology
Neuroendocrinology and Neuroimmunology
Sensory and Motor Systems
Theoretical and Computational Neuroscience
Tools Development and Open Source Neuroscience

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Cellular and Molecular Neurobiology
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Cellular and Molecular Neurobiology
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Sensory and Motor Systems
Theoretical and Computational Neuroscience
Tools Development and Open Source Neuroscience

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Sponsors & Venue

VENUE

The XL Annual Meeting of the SAN will be held at Aula Magna of Pabellón 1 y 2, Ciudad Universitaria, Universidad de Buenos Aires - UBA and IFIBYNE building.
Av. Costanera Rafael Obligado, Ciudad Universitaria, Universidad de Buenos Aires, from October 1st to 5th, 2025.

Code of Conduct

All attendees are required to agree with the following code of conduct. Organizers will enforce this code throughout the event. We expect cooperation from everyone to help ensure a safe environment for everybody.

No unauthorized Recording:
It is not allowed for attendees to record or take photos of scientific material unless explicit prior consent is given by the presenter. This restriction applies to all the scheduled events in the conference. If you become aware of someone making unauthorized recordings, please contact congreso.anual.san@gmail.com immediately.

Any person or organization recording without authorization may be subject to legal actions by the affected presenter, the organizations they are affiliated with, or by SAN.

SAN adheres to the copyright laws guiding the appropriate sharing of scientific research material, including data.

Conference Best Practices:
All communication must be carried out in a professional and respectful manner. Live sessions will be moderated and disrespectful messages will not be tolerated.

SAN encourages open intellectual discussion in a welcoming and inclusive environment. Inappropriate behavior, harassment or offensive acts towards any member of the community is strictly prohibited and will result in removal from the conference and a report to the host institution of the removed attendee will be issued. Be friendly, welcoming and respectful. When discussing with colleagues, disagreement is an unavoidable occurrence and it is important that all discussions are carried out in good faith and seen as an opportunity to improve others and our own work. Be mindful of the tone and words you choose to communicate with others.

Unacceptable behavior
Harrasment, intimidation or discrimination in any form is not tolerated at the event. This includes any improper and unwelcome verbal or physical behavior that might reasonably be expected to cause offense or humiliation to another person.

Inappropriate behavior can be reported to congreso.anual.san@gmail.com the SAN2024 Organizing Committee or SAN Council members. The event organizers reserve the right to take any action to prevent violations of this Code of Conduct.

Program at a Glance

01/10/2025

WEDNESDAY

09:00 - 09:30 | Registration
09:30 - 18:30 | **Pre-Meeting School**

02/10/2025

THURSDAY

10:30 - 18:00 | **Pre-Meeting School**

03/10/2025

FRIDAY

08:30 - 09:00
Registration

09:00 - 10:00
Plenary Lecture 1:
Wachowiak

10:00 - 10:30
Break

10:30 - 12:30
ISN Symposium 1:
Glial Cells in Neurodegeneration
Juana Pasquini

ISN Simposium 2:
Circuit Dynamics in Zebrafish
Violeta Medan, Verónica Pérez Schuster

12:30 - 13:00
Lunch

13:00 - 14:30
Mesa Redonda:
Comisión de Género y Diversidad

14:30 - 15:00
Break

15:00 - 18:00
Poster Session 1

18:00 - 21:30
Asamblea SAN

04/10/2025

SATURDAY

09:00 - 10:00
ISN Plenary Lecture 2:
Tomsic

10:00 - 10:30
Break

10:30 - 12:30
Symposium 3:
Human Brain Dynamics
Jacobo Sitt

Simposium 4:
Brain Development and Behavior
María Carolina Fabio

12:30 - 13:00
Lunch

13:00 - 14:30
Mesa Redonda:
Comisión de Política Científica

14:30 - 17:30
Poster Session 2

17:30 - 18:00
Break

18:00 - 20:00
Young Investigator Talks

20:00 - 21:30
Red de estudiantes

21:30 - 22:00
Party

05/10/2025

SUNDAY

09:30 - 10:00
Plenary Lecture 3:
Garelli

10:00 - 10:30
Break

10:30 - 12:30
Symposium 5:
Memory, Consciousness & Interception
Silvia Kochen, Alejandro Nasimbera

Simposium 6:
Single Cell Genomics
Daniela Di Bella, Micaela Sartoretti

12:30 - 13:00
Lunch

13:00 - 14:30
Mesa Redonda:
Comisión de Relaciones Internacionales

14:30 - 17:30
Poster Session 3

17:30 - 18:00
Break

18:00 - 19:00
ISN Plenary Lecture 4:
Marin Burgin

Pre-Meeting School

ISN School

Neuropharmacology Course

Faculty:

Mariano M. Boccia, María C. Krawczyk, Candela Medina, Santiago Ojea Ramos, and Verónica Baez.

Summary and Objectives:

Pharmacology is a key interventional tool for studying neurobiology, as it allows manipulation of neurochemical systems and evaluation of their effects on behavior and neuronal function. This intensive course is aimed at doctoral students and researchers interested in applying pharmacological techniques to the study of the brain and behavior. Through a combination of theoretical lectures and practical activities, the fundamental principles of pharmacology, their application in experimental models, and the analysis of pharmacokinetic and pharmacodynamic data will be addressed.

The course features the participation of invited researchers from the Argentine Society of Neurosciences (SAN), who share their experience in using pharmacological strategies in their research lines.

Upon completion of the course, an evaluation is conducted according to the content taught. Additionally, the course aims to fulfill the necessary requirements so that its approval grants points in the doctoral program of the Faculty of Exact and Natural Sciences (FCEN) of the University of Buenos Aires (UBA).

Content and Structure

Day 1: Theoretical

- Fundamentals of Pharmacology:
  Principles of pharmacokinetics and pharmacodynamics applied to neurosciences.
- Neuropharmacology of addictions:
  Mechanisms of action of psychoactive drugs and experimental strategies for their study.
- Pharmacological techniques applied to different experimental models:
  Drug administration, in vivo and in vitro approaches, experimental controls, and methodological considerations.

Day 2: Theoretical-Practical

- Computational modeling of pharmacokinetic and pharmacodynamic variables:
  Tools and approaches for data analysis in experimental pharmacology.
- Critical analysis of scientific articles:
  Discussion of representative studies that employ pharmacological techniques in animal models, with the participation of invited researchers from SAN.

Plenary Lectures

Plenary Lecture 1

Matt Wachowiak

Department of Neurobiology, University of Utah, USA

Title:

"Interoception by olfactory sensory neurons? Linking tuning, timing and transformations in odor representations".

Odors – chemical signals from the environment – are primary sensory drivers of behavior in most animal species and provide information essential to survival. At the same time, olfaction represents a vulnerable point of contact between the interior of an organism and chemicals in its external environment. To deal with this hazard, vertebrates and invertebrates alike express a variety of xenobiotic enzymes in olfactory tissues, which convert incoming chemicals to more water-soluble forms that are easily removed from tissue. However, such metabolites can themselves function as odorants, activating distinct olfactory sensory neurons. We have recently found that this process is a major organizing feature of the temporal and 'spatial' (i.e., identity-based) neural representations of odors among olfactory sensory neurons and their mapping to glomeruli of the mouse olfactory bulb. Odorant metabolites thus represent an internally-generated odor source that may confound the coding and recognition of external chemical signals. We propose that the nervous system resolves this confound using inhalation-linked timing as a neural feature that separates externally-sampled from internally-generated odor signals. We also propose that this timing-based strategy allows for the separate encoding of other internally-generated odors from sources such as ingested food, or volatiles in exhaled air that desorb from the blood during respiration. Such signals carry important information about internal body state. I will discuss the hypothesis that, rather than suppressing signals from such sources, downstream circuits may differentially route olfactory afferent signals to higher-order centers based on their respiration-linked dynamics, allowing for olfactory sensory neurons to transmit both external and interoceptive olfactory information and for each mode of signaling to adaptively drive behavior.

Funding: National Institutes of Health (NIDCD); National Science Foundation (Neuronex)

Plenary Lecture 2

Antonia Marin Burgin

IBIOBA – MPSP, Instituto de Investigaciones en biomedicina de Buenos Aires-Instituto Partner de la sociedad Max Buenos Aires, Argentina.

Title:

"Experience dependent sensory processing"

Sensory processing in the brain unfolds through a hierarchy of regions that increasingly abstract and interpret incoming stimuli. While primary sensory cortices have long been considered dedicated to encoding the physical features of stimuli, recent studies in awake, behaving animals challenge this classical view. Neural responses in these areas are now known to be far more dynamic and context-dependent than previously thought.

In this talk, I will present recent findings from our lab that explore how experience shapes odor representations in the primary olfactory piriform cortex (PCx) of mice. Olfactory perception is inherently modulated by context, memory, and internal state, yet how and where these non-olfactory influences are integrated into cortical processing remains unclear. Using electrophysiological recordings in mice performing an odor-guided task within a virtualreality environment, we investigated how visual context and learning influence PCx activity.

Mice were trained to associate specific odors with spatial and visual cues in order to receive a reward. We found that, with learning, PCx neurons shifted from purely sensory responses to mixed selectivity, encoding not only odors but also positional, contextual, and associative information. These extra-sensory modulations were dynamic and task-dependent, enhancing odor decoding specifically during task engagement and in rewarded contexts.

Our results demonstrate that the PCx integrates non-olfactory information early in the sensory hierarchy, and that this integration supports more flexible and behaviorally relevant odor representations. This work reveals a critical mechanism by which sensory cortices can dynamically incorporate experiential and contextual signals to guide perception and action.

Acknowledgements
Argentine Agency for the Promotion of Science and Technology, PICT2020-00360, Swiss National Science Foundation (SNSF) SPIRIT 216044 and FOCEM-Mercosur COF 03/11

ISN Plenary Lecture 3

Daniel Tomsic

IFIBYNE Instituto de Fisiologia, Biologia Molecular y Neurociencias Buenos Aires, Argentina.

Title:

“Neural computations underlying visually guided avoidance and pursuing behaviors: from the field to the laboratory and Back”

For many moving animals, visual motion is a crucial guide for behavior. Sensitivity to motion likely evolved to anticipate predators and capture moving prey. To escape threats, animals must identify danger, locate it precisely, and evaluate their surroundings for shelter, often relying on past experiences. By integrating these cues with early visual detection, they can decide whether to freeze, flee, or confront the threat—and, if fleeing, when, where, and how fast to escape, sometimes within split seconds. Remarkably, even small-brained animals can process complex information, form long-term memories, and execute precise motor patterns.

Certain crab species serve as excellent models for neuroethological research, thanks to their rich repertoire of visually guided behaviors and suitability for in vivo electrophysiology. In this talk, I will present our findings on Neohelice, focusing on visually driven avoidance and prey capture. Our approach combines field and laboratory behavioral analyses, neuroanatomy, intracellular and multielectrode recordings, and computational modeling. A central focus is on identified motion-sensitive giant neurons and their role in visuo-motor transformations that control speed and direction during prey capture and escape. Our results highlight the differences between behavior observed under laboratory conditions and behavior occurring in the natural environment.

Federal Conference 4

Andrés Garelli

Instituto de Investigaciones Bioquímicas de Bahía Blanca (UNS-CONICET), Argentina

Title:

"Neuroendocrine modulation of the pupariation motor program in Drosophila”

Innate behaviors consist of genetically hardwired sequences of motor programs that can be coupled to morphogenetic changes. By studying pupariation—an innate behavior that reshapes the Drosophila larval body in preparation for metamorphosis—we provide insight into the neural circuits and modulators that generate complex motor patterns and the mechanisms that coordinate them with morphological changes that are critical for survival. Using classical genetic tools and optogenetic control of neuronal activity, we describe a periphery-to-CNS endocrine regulatory loop that links behavioral initiation with developmental timing. This loop involves a minimal neuronal population positioned at the top of the circuit hierarchy, orchestrating a complex, stereotyped motor pattern. We will also present our most recent data identifying downstream components of this neuronal circuit, including neurons with command-like activity that control specific behavioral phases, and highlight the profound influence of neuromodulators in shaping these activity patterns. Our results establish the pupariation motor program as a model for investigating the neuroanatomical basis of behavior, reveal the complexity of innate behavior control, and contribute to our understanding of how multistep innate behaviors are temporally coordinated and integrated with developmental processes through an ensemble of neuropeptidergic signals.

Symposia

ISN Symposium 1

Can we identify common pathways mediated by Glial Cells in neurodegeneration?

Chair:

Juana Pasquini

[IQUIFIB] Instituto de Química y Fisicoquímica Biologicas “Prof. Alejandro Paladini”

The Simposium try to demonstrate the interrelationship between the different glia cells in the central nervous system (CNS) We also included tha Schwann cells.

Speakers:

Carla Caruso

INBIOMED UBA-CONICET. Facultad de Medicina, UBA.

The role of astrocytes in Huntington’s disease.

Astrocytes are fundamental glial cells that play a crucial role in maintaining brain homeostasis. In recent decades, our understanding of astrocyte functions has advanced significantly. Neurodegenerative diseases are characterized by the selective loss of neurons, increased glial activation, and glial dysfunction. Growing evidence indicates considerable regional heterogeneity among astrocytes in terms of morphology, gene expression, and function. Huntington’s disease (HD) is a neurodegenerative disorder that initially affects the striatum and later the cortex, leading to motor, cognitive, and psychiatric impairments. Given the pivotal role of astrocytes, there is increasing interest in their potential as therapeutic targets in neurodegenerative conditions. Our research highlights functional differences between astrocytes in the striatum and those in the cortex. Using a mouse model of HD, we investigated regional astrocyte differences and explored their potential therapeutic relevance.

Maria Claudia Gonzalez Deniselle

Lab. de Bioquímica Neuroendócrina. Instituto de Biología y Medicina Experimental IByME-CONICET.

Myelin as a target of steroid hormones in motoneuron degeneration.

Amyotrophic lateral sclerosis (ALS) is a progressive motoneuron disease associated with demyelination and neuroinflammation. The incidence of ALS is greater in men; however, it increases in women after menopause, suggesting a role for sex steroids in the disease. Low testosterone concentrations have been detected in the cerebrospinal fluid of ALS patients and in the central nervous system of male Wobbler mice, a well-characterized spontaneous model of motoneuron degeneration. Wobblers exhibit motoneuron degeneration, astro- and microgliosis, and myelin disruption within the cervical spinal cord. Testosterone is a complex steroid that exerts its effects directly via androgen receptors (AR) and indirectly via estrogen receptors (ER) after aromatization into estradiol. In this study, we investigated whether male Wobblers receiving a 2-month testosterone treatment, with or without the aromatase inhibitor anastrozole, exhibited modulation of myelin-associated features and neuroinflammation. Myelin characteristics were assessed via Luxol fast blue (LFB) staining, semithin sections, transmission electron microscopy (TEM), and immunolabeling for myelin basic protein (MBP) and proteolipid protein (PLP). Inflammatory responses were analyzed by quantifying IBA1+ microglia, and the mRNA expression of CD11b, TLR4, TNFαR1, and P2Y12R. Glutamatergic homeostasis was evaluated through glutamine synthetase and GLT-1. Testosterone-treated Wobblers showed enhanced LFB, MBP, and PLP staining.

A marked increase in the thickness of the myelin sheaths proportional to the axon diameter was shown in the white matter of testosterone-treated Wobblers (Y = 0.2313*x +0.1756; p <0.001) and controls (Y = 0.1094 * x +0.3906; p <0.001). However, the thickness of the myelin sheaths was independent of the axon diameter in steroid-free and testosterone plus anastrozole-treated Wobbler mice. and myelin compaction was better compared to Wobblers and testosterone plus anastrozole-treated Wobblers, which exhibited disruption of myelin lamellae. Wobbler mice showed a marked increase in IBA1+ microglia and elevated expression of inflammatory genes, which were significantly attenuated by testosterone administration, but not by the combined testosterone and anastrozole treatment. Furthermore, glutamine synthetase-positive cells and GLT-1 immunoreactivity were preserved in testosterone-treated Wobblers but reduced in both Wobblers and testosterone plus anastrozole-treated Wobblers. Functionally, testosterone improved muscle mass, grip strength, and limb morphology, whereas these benefits were absent following testosterone and anastrozole treatment. These findings indicate that testosterone confers protective effects on myelin and modulates neuroinflammatory and glutamatergic pathways in Wobbler mice, supporting its translational potential in motoneuron disorders.

Pablo Iribarren:

Profesor Titular e Investigador Principal CIBICI-CONICET Facultad de Ciencias Químicas Universidad Nacional de Cordoba. Córdoba Argentina..

Microglial Cells: foes or friends during neurodegeneration?.

Inflammation represents a complex biological response to tissue damage, pathogens, and irritants. Macrophages migrate to injured tissues in response to inflammation-induced chemoattractants, playing critical roles in both the promotion and regulation of inflammatory processes. Our previous work demonstrated that TLR2 selectively activates autophagic flux in microglial cells. Stimulation of microglial cells with alpha-synuclein or LPS triggers the production of pro-inflammatory cytokines and nitric oxide (NO), leading to microglia-mediated neuronal cell death in vitro. Notably, the activation of autophagy in microglial cells attenuates the production of pro-inflammatory cytokines, NO, and associated neurotoxicity. On the other hand, preliminary results from our laboratory suggest that alpha-synuclein, which is a potential ligand of TLR2, may induce autophagy in macrophages, and this response would depend on the activation of TBK-1 and OPTN. Our findings highlight that autophagy activation in microglial cells can assume diverse roles across multiple layers of microglial responses, with its effects shaped by contextual factors. In addition, evaluation of the activated microglial cells transcriptome may provide clues about the complex pathways modulated by TLR2. Transcriptomic analysis of microglial cells suggests that TLR2 stimulation modulates several pro-inflammatory.

Felipe Court:

Full Professor, Center for Integrative Biology, Universidad Mayor, Chile. Director, FONDAP Geroscience Center for Brain Health and Metabolism

Title:

Schwann Cell-Axon interactions in Homeostasis, Degeneration and Axonal Regeneration.

Peripheral nerve integrity depends on dynamic interactions between axons and Schwann cells (SCs). While axonal degeneration has long been considered a neuron-intrinsic process, our recent work reveals that SCs actively initiate axonal disintegration via a cytokinesis-like mechanism that triggers mitochondrial-dependent fragmentation. This glia-driven process reframes our understanding of degeneration, particularly in aging and disease.
Regeneration, conversely, relies on SC reprogramming into a repair phenotype that promotes axon growth and immune modulation. In aging and chronic denervation, we find this program is impaired not just by failed reprogramming, but by the accumulation of senescent SCs, a newly identified phenotype that inhibits regeneration and fuels inflammation. Removing these cells restores regenerative capacity. These findings position Schwann cells as key regulators of both axon loss and repair, and as promising therapeutic targets for improving nerve regeneration in aging and disease.

Symposium Conclusions:

Jorge Correale

Instituto de Química y Fisicoquímica Biológica (IQUIFIB) Facultad de y Farmacia y Bioquímica Universidad de Buenos Aires CONICET y Departamento de Neurología FLENI. Buenos Aires Argentina

Symposium 2

Circuit Dynamics and Behavior: Insights from Zebrafish

Chairs:

Violeta Medan

IFIBYNE (CONICET-FCEN-Universidad de Buenos Aires), Buenos Aires, Argentina.

Verónica Pérez Schuster

IB3, FCEN, UBA, Buenos Aires, Argentina

Four speakers will present on different neural aspects that enable critical behavioral functions, such as visual discrimination, navigation and orientation, and adult neurogenesis in the zebrafish. The symposium will highlight the advantages of using the zebrafish as an experimental model to address various systems biology questions. In particular, the speakers will present cutting-edge techniques for recording and analyzing in vivo whole-brain neuronal activity.

Speakers:

Lucas Mongiat

Depto. Física Médica, Centro Atómico Bariloche, CNEA, Bariloche, Argentina

ISN Talk:

Adult neurogenesis and pallial circuit dynamics in zebrafish

The pallium, a critical brain region in cognitive processing, including memory, learning, and emotional regulation, exhibits remarkable neuronal plasticity and active adult neurogenesis in zebrafish. While extensive research has explored neural stem cell (NSC) biology in this area, little is understood about how behaviors and internal signals influence pallial neuronal circuits. This presentation will highlight our discoveries on how spatial learning tasks stimulate the incorporation of both glutamatergic and GABAergic neurons in a balanced manner. Additionally, we will explore the role of the endocannabinoid system in modulating synaptic activity and the integration of adult-born neurons into pallial circuits.

Claire Wyart

Institut du Cerveau – ICM – Hôpital Pitié Salpêtrière, Paris, France

Title:

Unraveling algorithms and brainstem circuits for navigation – from fish to humans

Animals adjust their patterns of navigation on multiple timescales according to external and internal sensory cues that inform them respectively on their environment as well as their internal states and needs. We developed an approach for quantifying unbiasedly the long timescales of behaviour. We focus on larval zebrafish to unravel how these motor strategies correspond to exploration and exploitation and vary across animals and time. Our ongoing efforts now investigate the underlying circuitry in the brainstem that enable this coupling of sensorimotor integration with neuromodulation to dynamically shape motor strategies.

Ruben Portugues

Institute of Neuroscience, Technical University of Munich, Munich, Germany.

Title:

The heading direction network in larval zebrafish

Spatial navigation has long been considered as a window into cognitive function. What mental models do animals have of their surroundings and how can they be used? In the lab, we recently discovered a heading direction network in the hindbrain of the larval zebrafish. This acts as a “compass”, pointing in the same direction in absolute (allocentric) space regardless of how the

fish orients itself. I will review the activity of the network, and how it is affected by both the animal's movement and external sensory cues. The small size of this vertebrate allows us to incorporate connectomic studies that probe the structure of the network and link it to is function, and seek what we may call a mechanistic understanding of a network that underlies cognitive behavior. Finally, I will highlight architectural similarities with the heading direction networks that have been identified in insects, and possible homologies with mammals, and argue that comparative approaches have a lot to teach us about neurobiology.

Symposium 3
How Brain Dynamics shape human experience

Chair:

Jacobo Sitt
Paris Brain Institute

Understanding how dynamic patterns of brain activity give rise to subjective experience remains a central challenge in neuroscience. This symposium brings together four researchers who investigate the neural underpinnings of consciousness across various states, including wakefulness, sleep, and pathological conditions. By integrating theoretical frameworks with clinical applications, the session aims to elucidate the relationship between global brain dynamics and phenomenology.

Jacobo Sitt will introduce the general topic and weave the presenters talks in between.

Tristan Bekinschtein will present the brain signatures used in real world experiments with portable EEG, highlight the power of collecting hundreds of sessions on breathworks and also show the robustness of doing dose-response psychedelic work in Argentina. The combined use of experience tracing and EEG allows for a framework for neurophenomenology that is exemplified here by the brain dynamics of altered consciousness.

Vincent Taschereau-Dumouchel uses real-time fMRI and decoded neurofeedback to modulate brain patterns and subjective states actively. By decoding brain activity linked to specific feelings (like pain or fear) and feeding it back as neurofeedback, his approach can change underlying physiological processes and potentially reduce the felt intensity of these experiences without any conscious effort. This offers causal evidence tying brain dynamics to phenomenology and suggests novel interventions for clinical conditions (e.g. chronic pain, anxiety). It exemplifies the power of brain–machine interface techniques to illuminate mind–brain relationships.

Cecilia Forcato will explore how the minimal self — our basic sense of being an embodied subject — emerges from brain-body interactions beginning in early life. Drawing on studies with preterm infants and individuals experiencing depersonalization, she shows how disruptions in interoceptive integration can weaken the sense of self. Her work uses EEG and heartbeat-evoked potentials to highlight how neural responses to bodily signals underpin the coherence of selfhood. This developmental and translational perspective emphasizes that consciousness is not only brain-based but deeply rooted in the dynamics of embodied existence.

These four talks collectively showcase an innovative synthesis of theoretical and clinical neuroscience. The session offers an accessible overview of how brain dynamics across multiple scales link to subjective experience, making it relevant to neuroscientists across subfields. Emphasizing novelty and interdisciplinarity, it demonstrates that combining cognitive experiments, clinical studies, and neurotechnology can advance our understanding of consciousness and inspire new strategies to improve conscious states.

Speakers:

Tristan Bekinschtein
Consciousness and Cognition Lab, University of Cambridge

Title:

The neural dynamics of altered states of consciousness: lessons from psychedelics and hypoxic events

Recent advances in high-temporal resolution neuroimaging, combined with novel tools for capturing subjective experience, allow us to trace how fluctuations in neural activity relate to the evolving contents of consciousness. In this talk, I present a series of studies employing Temporal Experience Tracing (TET), a neurophenomenological method that enables participants to retrospectively map the intensity of multiple experiential dimensions as continuous time series. Applied across both pharmacologically induced (DMT, psychedelics) and non-pharmacological (breathwork, physiologically induced hypoxia) Altered States of Consciousness, TET allows for fine-grained modeling of experience dynamics when integrated with their neural counterparts.
In high-dose DMT conditions, we observed rapid and structured shifts in experiential dimensions such as emotional intensity, visual complexity, and selfhood, which were tightly coupled with specific EEG neural dynamical markers. Notably, alpha oscillatory power and permutation entropy exhibited the strongest associations with subjective fluctuations, while Lempel-Ziv complexity, often heralded as a neural marker of psychedelic richness, showed weaker and less consistent correlations. Low-dose DMT revealed the power of the state of bliss and showed milder brain entropy and no negative experiences. Results from

the breathwork-induced Altered States of Consciousness demonstrated similar mappings between neural signal diversity (e.g., LZ complexity and aperiodic spectral components) and positively valenced experiential clusters, including a dose response between neural aperiodic component and increasing hypoxic events).
Drawing also on large-scale TET data from meditation research, we illustrate how temporal profiles of experience can be computationally clustered to reveal recurring metastable experiential states and their transition dynamics. These converging findings support a model in which distinct neural features dynamically scaffold specific human experiences in altered states of consciousness, and demonstrate the power of temporally resolved neurophenomenology to bridge first- and third-person perspectives on consciousness.

Jacobo Sitt

Inserm, Paris Brain Institute, France

Title:

Exploring global brain patterns dynamics, and their role in states and contents of consciousness

In this presentation, I will explore the probabilistic associations between distinct recurrent global dynamic brain patterns (GDBP) obtained from fMRI data and clinically defined states of consciousness. Through a comprehensive analysis of functional neuroimaging data from both humans and non-human primates, I will demonstrate that conscious states are intricately linked to the dynamic exploration of a diverse repertoire of GDBP, characterized by a characteristic temporal scale of approximately 10 seconds. Specifically, I will discuss how conscious individuals, whether human or non-human primates, exhibit two distinctive groups of GDBP: (1) 'High' GDBP: These patterns involve robust long-range functional cortico-cortical communication, encompassing both positive correlations and anti-correlations between different brain areas, (2) 'Low' GDBP: These patterns, in contrast, are characterized by sparse and limited inter-areal communication. Interestingly, unconscious individuals, such as those under anesthesia or in a disorder of consciousness state, primarily express the latter, 'low' GDBP category. Furthermore, I will present recent findings demonstrating that the temporal sequence of GDBP also reflects the fine-grain dynamics of subjective experience. I will show results demonstrating the inter-subject synchronization of GDBPs across subjects that occurs only when subjects are attentive to audiovisual narratives. I will also show that the occurrence of specific modulates the subjects' perceptual threshold, suggesting a direct relationship between ongoing whole brain dynamics and subjective experience. In summary, this presentation underscores the critical role of GDBPs in understanding the neural basis of consciousness, highlighting their potential as a novel avenue for investigating subjective experiences across different states of consciousness.

Vincent Taschereau-Dumouchel

Department of Psychiatry and Addictology, Université de Montréal

Title:

Dissociating conscious and unconscious affective processes using decoded neurofeedbac

Conscious emotional experiences are so tightly coupled with physiological and behavioral responses that such "objective" measures are often used interchangeably with subjective reports when developing treatments for emotional disorders. This is problematic because many lines of evidence now indicate that objective measures reflect unconscious defensive mechanisms that dissociate from the subjective experiences that patients are trying to avoid—and which typically motivate them to seek treatment. In this presentation, we will discuss a series of experiments aimed at further studying the dissociation between objective and subjective responses in the brain. Notably, we will discuss a series of closed-loop fMRI neurofeedback studies targeting machine-learning decoders of affective processes in the brain. We will show that causally manipulating specific brain representations can help further understand the dissociation between objective and subjective processes. We will specifically discuss two neurofeedback experiments that showed a selective modulation of physiological defensive responses without changing the subjective experience of fear. Conversely, we will discuss the results of a new neurofeedback study that modulated the subjective experience of pain independently from the brain activity associated with nociceptive processes. These results will be discussed in light of the higher-order theory of emotional consciousness and will help us devise a path forward to better target troubling affective experiences for therapeutic purposes

Cecilia Forcato

Laboratorio de Sueño y Memoria, Instituto Tecnológico de Buenos Aires (ITBA)

Title:

Disembodied Minds: Out-of-Body Experiences and the Neuroscience of Consciousness During Sleep

Out-of-body experiences (OBEs) are vivid phenomena in which individuals perceive themselves as separated from their physical body, observing the world from an external vantage point. These experiences, often reported during transitions between sleep and wakefulness, challenge traditional views of consciousness and embodiment. Despite their historical, cultural, and clinical relevance, OBEs remain poorly understood from a neuroscientific perspective.

In this talk, I will present recent findings from our lab combining phenomenological reports and electrophysiological recordings during sleep to investigate OBEs, sleep paralysis (SP), lucid dreams (LDs), and false awakenings (FAs). Our studies reveal that OBEs are associated with distinct spectral brain activity patterns, particularly increases in delta and reductions in fast-frequency bands (alpha, beta and low-gamma), suggesting a unique neurophysiological signature that distinguishes them from both wakefulness and canonical sleep stages. Importantly, we provide the first evidence of eye-movement markers during OBEs captured in-lab, enabling their precise temporal localization.

I will also discuss the emotional landscape of these altered states, showing that OBEs are experienced as more pleasant than SP oneiric perceptions, especially when self-induced, and may be preceded by identifiable precursory sensory cues, including tactile, auditory, and visual sensations, that open new avenues for controlled induction. Taken together, these findings support the idea that consciousness during sleep is not binary but dynamic, and that OBEs represent a meaningful and reproducible state of altered self-perception that bridges the neurophysiological and experiential domains

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ISN Symposium 4

Perinatal Programming: Factors That Influence Brain Development and Behavior

Chair:

María Carolina Fabio

Instituto de Investigaciones Médicas “Mercedes y Martín Ferreyra” – INIMEC CONICET-UNC – Córdoba, Argentina

This symposium will explore how prenatal and early postnatal factors influence neurodevelopment through perinatal programming, a process by which environmental stimuli during critical developmental windows exert lasting effects on brain structure and function.

First, **Dr. Carolina Fabio** will present recent findings on how gestational disruption of serotonin signaling affects brain maturation, neural plasticity, and social behavior across ontogeny.

Scondly, **Dr. Silvina Díaz** will examine the long-term impact of neonatal exposure to antidepressants on adult hippocampal neurogenesis and memory, highlighting the molecular mechanisms involved.

Nexts, **Dr. Natalia Uriarte** will discuss how complex family structures, such as overlapping litters in rodent models, shape maternal behavior and offspring development, leading to reduced anxiety and stress reactivity in adulthood.

And Lastly, **Dr. Miranda** will explore how early-life family configurations, including single-mother versus biparental rearing, influence behavioral and neural responses to alcohol during infancy and adolescence.

Together, these presentations will offer a comprehensive and multidisciplinary overview of perinatal programming and its implications for brain development and behavior, providing valuable insights for researchers, clinicians, and policymakers.

Speakers:

Natalia Uriarte Bálsamo

Laboratorio de Neurociencias, Facultad de Ciencias, Universidad de la República, Uruguay

Title:

Family matters: overlapping litter rearing in rats shapes mothers behavior and pups development”

In rats, mating during postpartum estrus and late weaning of the previous litter lead to the overlapping of two litters of different ages within the maternal nest, creating a complex early-life environment for the pups. This reproductive condition also presents a challenge to mothers, who flexibly adapt their behavior to meet the distinct characteristic and needs of both litters. This behavioral flexibility is further reflected in greater cognitive flexibility, measured by the Attentional Set Shifting task. Additionally, in this complex family structure, juveniles exhibit caregiving behaviors toward their younger siblings, shaping different developmental trajectories. Pups from the junior litter show reduced anxiety-like responses in males and females, as well as diminished endocrine stress responses and reduced reproductive behavior in females during adulthood. Furthermore, female offspring from overlapping litters display decreased licking behavior of pups, increased time spent off the nest, and changes in nursing postures when they become mothers themselves. These long-term effects could be attributed to the more complex and enriched rearing environment compared to that of single litters, likely resulting in altered quality and quantity of stimulation from both mothers and siblings. These findings highlight the importance of family structure and early social experiences in modulating maternal behavior, stress responsiveness, and reproductive strategies across generations, and validate the Overlapping litters Model as a valuable framework to study how early-life social and environmental complexity shapes developmental trajectories. Drawing also on large-scale TET data from meditation research, we illustrate how temporal profiles of experience can be computationally clustered to reveal recurring metastable experiential states and their transition dynamics. These converging findings support a model in which distinct neural features dynamically scaffold specific human experiences in altered states of consciousness, and demonstrate the power of temporally resolved neurophenomenology to bridge first- and third-person perspectives on consciousness.

María Carolina Fabio

Instituto de Investigaciones Médicas “Mercedes y Martín Ferreyra” – INIMEC CONICET-UNC – Córdoba, Argentina

Title:

Serotonin disruption during pregnancy and its neurobiological consequences for social development

Serotonin (5-HT) plays a crucial role in the development of the central nervous system during gestation. This study explores the impact of altered 5-HT levels during pregnancy, which have been associated with maternal depression, as well as social impairments and psychiatric disorders in offspring, suggesting a potential link to Autism Spectrum Disorders. We utilized a serotonin synthesis inhibitor (PCPA 200 mg/kg) in pregnant mice during gestational days 12.5 to 14.5 to investigate the effects of transient serotonin depletion on maternal depression and anxiety, and on social behavior and affective states of their offspring from weaning to adulthood. Additionally, we examined the influence of serotonin disruption during gestation on brain structure and function at weaning and into adulthood. Our findings indicate that reduced 5-HT availability during gestation can influence social behavior in later life without altering affective behavior. Notably, these behavioral changes are independent of maternal behavior. Furthermore, we will present evidence that this transient serotonin depletion has long-term effects on 5-HT neural circuits, marked by increased expression of the serotonin transporter (SERT) in a critical region implicated in psychiatric disorders. The implications of these findings for understanding the developmental origins of social and affective disorders will be discussed.

Silvina Laura Diaz

Instituto de Biociencias, Biotecnología y Biología Traslacional (IB3), FCEN, UBA.

Title:

Effect of postnatal exposure to fluoxetine on the process of neurogenesis and memory tasks in adult mice

It is well known that chronic treatment with antidepressants promotes the process of neurogenesis that occurs in the hippocampus of adult mice and induces anxiolytic-like behaviors. Recently, both clinical and animal model studies have observed that exposure to fluoxetine during the perinatal period paradoxically induces depressive and anxiogenic behaviors in adulthood. These effects have led us to question how neuronal proliferation and survival are affected in individuals who received antidepressants during their early age, as well as which behaviors might be impacted and what the functionality of the new neurons is. These questions will be addressed throughout my presentation.

Sebastián Miranda-Morales

Instituto de Investigaciones Médicas “Mercedes y Martín Ferreyra” - INIMEC CONICET-UNC - Córdoba, Argentina

Title:

Impact of parenting conditions on neurodevelopment and response to alcohol: contributions from an animal model

Parental behavior during early development has a strong influence on the emotional and social development of infants. Literature on parenting, human or animal, has primarily focused on the interactions between mothers and offspring, with little research directed at understanding on what paternal behavior could add to infant social attachment. Strong ties or social attachment as pair bond formation in adult rodents has protective effects on neurodevelopment and against drug effects. In this sense, rearing conditions during early ontogeny may have differential effects infant development and drug experience. We will present data showing how different parenting conditions (ie., single-mother -SM- or biparental care -BP-), in a non-monogamous C57Bl/6 mouse, have a differential impact on the parenting during lactation, adolescent behavior and response to ethanol of the offspring. Or results evidence that these two rearing conditions imply differential parental behavior during lactation period. SM condition induce an anxiety-like behavior and major alcohol consumption in adolescent offspring. These groups also differ in terms of ethanol-induce anxiolysis, neural activation in brain areas related to anxiety behaviors and oxytocin neurotransmission. These results highlight the importance of parenting during a critical period of early development and the long-lasting effects of social experience.
Keywords: parental behavior, adolescence, anxiety, ethanol response.

Symposium 5

Neurophysiological Bases of Memory, Consciousness, and Interoception in Humans: From the Neuron to Neural Networks

Chairs:

Silvia Kochen

ENyS-CONICET, Argentina.

Alejandro Nasimbera

ENyS-CONICET, Argentina.

This symposium session features a series of talks presenting recent studies on the neurophysiological mechanisms underlying memory, auditory perception, consciousness, and interoception, approached from the activity of individual neurons to the functioning of broader neural networks. Findings are presented on the processes involved in the encoding and retrieval of episodic memories, highlighting the role of specialized neurons in the representation of concepts. Additionally, the panel explores auditory prediction mechanisms that enable the anticipation and processing of sound stimuli, which are essential for the perception of language and music. The role of interoception in the construction of consciousness is also examined, particularly in patients with epilepsy. The discussion includes how neuronal activity influences internal bodily perception and the continuity of subjective experience. Within this context, recent advances in the precise localization of epileptogenic zones through intracerebral recordings are detailed, along with their relationship to cognitive functions relevant for planning surgical interventions. Through neurophysiological studies, neuroimaging techniques, and computational models, this panel offers an integrated view of the relationship between brain activity and conscious experience.

Speakers:

Rodrigo Quian Quiroga

Hospital del Mar Research Institute (IMIM)

Title:

Episodic Memory and Its Correlates at the Single-Neuron Level

In this talk, we will explore the neural mechanisms underlying episodic memory—that is, the ability to recall specific events situated in time and space. Drawing from studies involving single-neuron recordings in humans, we will present findings that reveal the selective activation of neurons in the hippocampus and medial temporal cortex in response to particular experiences. Key discoveries will be highlighted concerning so-called “concept cells,” which respond selectively to specific people, places, or events, and have played a pivotal role in advancing our understanding of how the brain encodes, consolidates, and retrieves episodic memories. These advances not only deepen our comprehension of memory processes but also offer new perspectives on their deterioration in neurodegenerative diseases.
We will examine how the activity of individual neurons contributes to the construction of autobiographical memory and the shaping of personal identity, framing these insights within current debates in the field of cognitive neuroscience.

Alejandro Blekman

RITMO Centre for Interdisciplinary Studies in Rhythm, Time and Motion

Title:

Auditory Prediction and Its Neural Correlate

This panel presents research on the neural mechanisms underlying auditory prediction—a fundamental process for the perception of sound, language, and music. Evidence is presented on how the brain anticipates and processes auditory stimuli through neural networks that integrate prior sensory information, thereby facilitating the interpretation of speech and complex musical structures.
Using neurophysiological and neuroimaging studies, researchers have identified the neural correlates of auditory prediction in regions such as the auditory cortex and areas involved in memory and attention. Additionally, computational models are analyzed to explain how the brain dynamically adjusts its expectations in response to variations in auditory stimuli.
Finally, the session addresses clinical and technological applications stemming from these findings, including the development of intelligent hearing prostheses and rehabilitation strategies for individuals with auditory and language processing disorders.

Nuria Campora

Hospital El Cruce – CIC, PBA

Title:

Interoception and Consciousness in Epilepsy: Clinical and Neurophysiological Approaches

This panel explores the clinical and neurophysiological characteristics of consciousness in patients with epilepsy, with a particular focus on proprioception and the subjectivity of conscious experience. Studies are presented demonstrating how neuronal activity in key brain regions influences internal bodily perception and various states of consciousness, both during and outside of epileptic seizures.
Drawing on clinical and neurophysiological evidence, the session examines how interoceptive disturbances affect self-perception and the continuity of conscious experience in these patients. Furthermore, the neurobiological mechanisms underlying the dissociation between brain activity and subjective experience are discussed, offering new perspectives on the study of consciousness within the context of epilepsy.

Silvia Kochen

CONICET – Hospital El Cruce

Title:

Cognitive Correlates and Epileptogenic Zone Diagnosis Through Intracerebral Recordings Using Macro- and Microelectrodes

This panel presents the use of intracerebral recordings with macro- and microelectrodes for the precise identification of the epileptogenic zone and its correlation with cognitive functions. Recent advances in clinical neurophysiology are discussed, which have enabled more accurate localization of the epileptic focus, improving both diagnosis and therapeutic approaches in patients with refractory epilepsy.
By recording neuronal activity at multiple scales, the interaction between epileptic discharges and cognitive functions—such as memory and language—is analyzed. Studies conducted during presurgical evaluation have allowed researchers to assess the functional impact of epilepsy on brain organization and its implications for planning resective surgery.
Finally, the session addresses the applications of these methodologies in advancing the understanding of the neurophysiological mechanisms of epilepsy and their relationship with cognition, providing key tools for optimizing treatment and improving patients' quality of life.

Symposium 6

Neuroscience through the lens of single cell genomics

Chairs:

Daniela Di Bella

Harvard University, Boston, USA / Instituto Leloir, Buenos Aires, Argentina

Micaela Sartoretti

The Francis Cricks Institute, United Kingdom.

Over the last decade, single-cell genomics has emerged as a powerful tool to study cellular complexity and dynamics across all life sciences, including neuroscience. From disease modeling to neuron-glia interaction, and from embryonic development to adult neurogenesis, it has expanded our understanding of molecular dynamics, cellular landscapes, and transcriptional regulation in the most complex organ found in animals. This symposium brings together speakers who will showcase how the use of this tool can aid in understanding the central nervous system in developmental processes and disease modeling.

Speakers:

Daniela Di Bella

Harvard University, Boston, USA / Instituto Leloir, Buenos Aires

Title:

Functional Screen of Identity Determinants in the Developing Mouse Cerebral Cortex

The generation of diverse and specified cell types rely in the concerted action of intrinsic and extrinsic factors. The mammalian cerebral cortex, with its unparalleled neuronal diversity, serves as an excellent model to study the acquisition of cellular identity. The molecular logic that governs the establishment and topographic organization of cortical cell types remains elusive, with the exception of few characterized transcription factors. Transcription factors play a central role in the interconnected mechanisms controlling cell identity during development, as they instruct the expression of concerted gene programs and gene regulatory networks.
In order to uncover the genes controlling the identity acquisition of the main excitatory neurons in the cortex, we first built a molecular atlas of the developing cortex using single cell RNA-sequencing, sampled every day through the duration of embryonic corticogenesis. From this we infered developmental trajectories unveiling gene regulatory programs that accompany fate specification and diversification. By exploring differentially expressed genes, we proposed candidate transcription factors that promote the two main classes of excitatory neuronal fates -corticofugal versus callosal. We next use multiplexed in vivo perturbations to assess the ability of these transcription factors to alter the identity of the progeny produced. With our work, we aim to identify new identity controllers in the cerebral cortex and to uncover novel mechanisms underlying neuronal specification.

Micaela Sartoretti

Cricks Institute, United Kingdom

Title:

Molecular and cellular landscape of the Down syndrome mouse brain

Down syndrome (DS) is caused by an extra copy of chromosome 21 (Hsa21), leading to overexpression of approximately 230 protein-coding genes. It is the most common genetic cause of intellectual disability, yet the molecular mechanisms underlying cognitive impairment remain unclear. Our lab generated the Dp1Tyb mouse strain, which carries a duplication of 145 genes from mouse chromosome 16 orthologous to Hsa21. These mice display deficits in hippocampal-dependent memory tasks. Using single-nucleus RNA sequencing, I analysed the hippocampal cellular landscape to determine how gene dosage alters neuronal and glial populations. I identified the main neuronal classes, including eleven interneuron clusters, and glial clusters. Interestingly, the frequency of neuroblasts, cells that will differentiate into adult-born neurons during adult hippocampal neurogenesis, was decreased in the Dp1Tyb hippocampus. To explore the mechanism underlying this difference, I subclustered astrocytes and neuroblasts, revealing radial glia-like (RGL) cells and neural progenitor cells (NPCs). Pseudotime trajectory analysis showed an increased frequency of RGL stem cells and a reduction of neuroblasts and immature neurons across the neurogenic lineage in the Dp1Tyb hippocampus. In Dp1Tyb, microglial subclustering revealed an imbalance between populations, with the expanded group displaying reduced homeostatic gene expression. Pathways related to translation and oxidative phosphorylation were also upregulated in Dp1Tyb microglia compared with wild type. Although interneuron proportions were largely unchanged, ligand–receptor network analysis suggested enhanced cadherin-mediated adhesive communication

between microglia and specific interneuron clusters, indicating that altered microglia–interneuron interactions may contribute to the DS phenotype.

Damiana Giacomini

Instituto Leloir, Buenos Aires, Argentina.

Title:

Transcriptional dynamics across the development and integration of adult-born hippocampal neurons

The dentate gyrus, the main entry point of entorhinal input into the hippocampus, continuously generates adult-born granule cells (aGCs) that confer unique forms of plasticity to preexisting circuits. Adult hippocampal neurogenesis is a conserved, multi-stage process that supports memory formation, context discrimination, and cognitive flexibility. In the mouse, the maturation of aGCs extends over several weeks and can be divided into discrete phases based on electrophysiological and morphological properties. Yet, the molecular programs governing their progression remain poorly understood. Using lineage tracing and single-nucleus RNA sequencing, we isolated nuclei from aGCs at defined ages and reconstructed their transcriptional trajectory from radial glia-like cells to fully mature neurons. This analysis uncovered previously uncharacterized intermediate maturation states and revealed sequential gene expression programs underlying stage-to-stage transitions. Building on this framework, we next examined how aging impacts the same developmental sequence. As expected, aging reduced the rate of neurogenesis and slowed neuronal maturation. Our transcriptomic profiling showed that this delay does not reflect a uniform slowing of development but rather the accumulation of aGCs at a discrete postmitotic neuroblast stage. Notably, this state was highly plastic, as voluntary running prevented neuroblast accumulation and promoted progression toward more advanced transcriptional states. These findings highlight postmitotic neuroblasts as a pivotal regulatory node in aging neurogenesis, where activity-dependent cues can re-engage stalled maturation. Together, these studies provide a high-resolution transcriptional roadmap of adult neurogenesis, from early neuroblasts to fully integrated neurons, and identify stage-specific vulnerabilities to aging, offering potential targets to restore hippocampal plasticity.

Piero Rigo

The Francis Crick Institute

Title:

Investigating human hippocampal neurogenesis using single-cell spatial transcriptomics

Hippocampal neurogenesis is essential in mice, contributing to the rapid growth of neural tissues during development and supporting learning, memory and mood regulation in adults. However, human hippocampal neurogenesis remains poorly characterised during development, while its persistence in adults is still debated. We used single-cell spatial transcriptomics to characterise human hippocampal neurogenesis and its source, i.e. neural stem cells (NSCs), in the developing brain. We observed a large fraction of NSCs in a non-proliferative state at mid-gestation, after the peak of neurogenesis at gestation week 14, suggesting, by analogy with mouse hippocampal neurogenesis, the formation of a reservoir of quiescent NSCs that could support long-term postnatal neurogenesis. Harnessing the acquired knowledge about human developmental neurogenesis, we examined published single-cell RNA sequencing datasets of the adult human hippocampus, where we found many cells displaying transcriptomic profiles similar to those of embryonic NSCs and immature neurons, suggesting abundant neurogenesis may occur in the adult human hippocampus.

Young Investigator Talks

YIT 1

Hot spot sites for alpha-synuclein amyloid assembly: a NMR and cryo-EM based study

Phelippe do Carmo Gonçalves

Max Planck Laboratory for Structural Biology, Chemistry and Molecular Biophysics of Rosario (MPLbioR, UNR-MPINAT).

Misfolding and aberrant aggregation of alpha-synuclein (αS) is associated with neurological disorders collectively referred to as synucleinopathies. Current therapies for these disorders are limited and, therefore, understanding the mechanism of amyloid formation and its inhibition is of high clinical importance. The design of molecular probes that efficiently modulate the aggregation process and/or neutralize its associated toxicity constitutes a promising tool to enhance the understanding of the molecular mechanisms of αS assembly and for development of therapeutic strategies against these disorders. By combination of NMR and cryo-EM we performed a detailed structural characterization of specific αS interactions with a molecular probe along the aggregation landscape of the protein. Our results demonstrate that these interactions affect the kinetics of amyloid fibril formation of αS, modulating the structural features of the fibrils formed and leading to different αS polymorphs. By using a well-established cell-based bioassay our results indicate that these interactions can not only alter the structure but also the pathology of the resulting αS fibrils. Overall, our findings indicate that identification of hot spot interactions between αS and molecular probes may represent a viable alternative to design therapeutic molecules for the treatment of synucleinopathies.

YIT 2

Profiling peripheral glial cells from human nerves for grafting in the CNS

Gabriela Aparicio

University of Kentucky

The regenerative capability of PNS cells, including Schwann cells (SCs) has been exploited clinically in cell transplantation therapies to treat CNS trauma and neurodegenerative diseases. However, the characteristics of peripheral nerve cells has not yet been addressed thoroughly in humans. The goal of this study was to identify specific markers able to reveal the identity and stage of differentiation of cells from intact and injured human nerves. Therefore, we developed and validated an in vitro model of human nerve degeneration to be compared with injured nerves from participants enrolled in a nerve transplantation clinical trial for Parkinson's disease.Histological analysis revealed that: (1) NGFR was a reliable marker to discriminate PNS cells from CNS neurons and glial cells; (2) S100B, GFAP and Sox10 were useful to specifically identify SCs within nerve tissues, with the caveat that they also revealed glial populations in the CNS; and (3) MPZ and PRX were equally useful to identify myelin sheaths derived from SCs rather than oligodendrocytes. To conclude, these markers can be used in different combinations to reveal grafted PNS cells, mainly SCs, in the human CNS to study their survival, differentiation and relationship to host tissue.
Aparicio, Quintero, …, and Monje, (2024). J. Peripher. Nerv Syst. DOI:10.1111/jns.12643.
Aparicio & Monje (2023). Bio Protocol. 20;13(22): e4748. DOI: 10.21769/BioProtoc.4748.

YIT 3

Interplay Between Early Nutritional Programming and Adult Obesogenic Diet on Brain Control of Food Intake

Pamela Fernández

Facultad de Bioquímica y Ciencias Biológicas (FBCB), Instituto de Salud y Ambiente del Litoral (ISAL), Universidad Nacional del Litoral (UNL), Santa Fe

Early-life nutritional imbalances and adult exposure to obesogenic environments are key obesity risk factors. Using a rodent model of neonatal overfeeding (small litters, SL) and cafeteria diet (CAF) in adulthood, we evaluated long-term effects on food intake regulation. Male Wistar rats were raised in small (SL, 4 pups/dam) or normal litters (NL, 10 pups/dam), fed a control diet (CON) until postnatal day (PND) 90. Then, they received CON or CAF for 11 weeks (NL-CON, NL-CAF, SL-CON, SL-CAF; 12±2 rats/group). Behavioral tests were conducted. At PND167, blood, fat pads and brains were collected. Ventral tegmental area (VTA), Nucleus Accumbens (NAc) and Arcuate Nucleus (ARC) were isolated by micropunch technique for qPCR and methylation analysis. Our results demonstrate that neonatal overfeeding and/or CAF diet exposure increase the body mass index, alter satiety response and induce anxiety-like behavior in adulthood. Within the homeostatic system, SL induced a long-term downregulation of POMC and NPY expression. DNA methylation changes were consistent with POMC repression. In the hedonic system, dopaminergic pathway disruptions were observed: NL-CAF showed reduced dopamine synthesis in the VTA, while SL-CAF exhibited enhanced dopamine clearance in the NAc. These findings reveal distinct obesity-related mechanisms driven by early-life and adult environments, highlighting the need for tailored therapeutic strategies.

YIT 4

IEssential but implicit: the role of aging information in neurodegeneration detection

Fermín Travi
Facultad de Ciencias Exactas y Naturales - UBA

A widespread hypothesis in brain imaging posits that neurodegenerative disorders constitute premature aging. Despite its prominence, this brain aging hypothesis (BAH) has not been verified against suitable alternatives. In this work, we first test a key assumption of BAH: Age information is necessary for detecting Alzheimer's Disease (AD). We compared brain representations that were maximally uninformed about chronological age against ones that were maximally informed about age. We found that absence of aging information impairs AD detection, providing causal evidence for BAH.

Second, we investigated whether explicit age modeling confers advantages in transfer learning for AD detection. We evaluated pretraining strategies for age, sex, and BMI inference and found that while pretraining improved representation stability and quality, these tasks converged to similar learned representations with no single phenotype providing superior advantage for neurodegeneration detection. These findings demonstrate that aging and neurodegeneration are fundamentally linked, yet aging information emerges naturally during learning of brain features without dedicated encoding. This moves current thinking past brain-age gap conceptualizations and suggests new directions for foundation models integrating richer phenotypic information.

1° Facultad de Ciencias Exactas y Naturales, Departamento de Ciencias de la Computación, Universidad de Buenos Aires, Buenos Aires, Argentina
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3° IBM T. J. Watson Research Center, Yorktown Heights, New York, NY, United State

YIT 5

Postnatal fluoxetine modulates the mouse prefrontal emotional circuit development

Tamara Adjimann
Instituto de Fisiología, Biología Molecular y Neurociencias, IFIBYNE (UBA-CONICET)

Depression and anxiety are leading causes of disability worldwide, yet their developmental origins remain unclear. To explore early mechanisms of vulnerability to psychiatric disorders, we used a mouse model of adult emotional vulnerability induced by the early postnatal exposure to the antidepressant fluoxetine (FLX). C57BL/6 mice (both sexes) received FLX (10 mg/kg/day, p.o.) in 3% sucrose from postnatal day (P)2 to P14. At P15, we investigated the early impact on the prefrontal cortex-to-dorsal raphe nucleus (PFC-DRN) circuit, which is implicated in stress coping and mood regulation.
Using the high-resolution microscopy technique, Array Tomography, we observed a selective ~40% increase in glutamatergic PFC inputs to DRN serotonin (5-HT) neurons. Ex-vivo patch-clamp recordings supported the presence of additional functional glutamatergic synapses. Following acute stress in the forced swim test (FST), c-fos immunohistochemistry and layer-specific markers revealed heightened activation of specific PFC projection-neurons and increased 5-HT1A receptor-mediated inhibition in the DRN. Behaviorally, FLX-exposed mice showed reduced immobility in the FST, an effect reversed by 5-HT1A receptor blockade using the selective antagonist WAY-100635. Altogether, these findings reveal that postnatal FLX induces structural and functional remodeling of the nascent PFC-DRN circuit, likely contributing to altered stress responses and emotional behavior later in life.

YIT 6

Striatal cholinergic interneuron pause response requires Kv1 channels, is absent in dyskinetic mice, and is restored by dopamine D5 receptor inverse agonism

Cecilia Tubert
Laboratorio de Fisiología de Circuitos Neuronales, Grupo de Neurociencia de Sistemas, IFIBIO Houssay - UBA CONICET

Dopaminergic and cholinergic neurons are the main modulators of corticostriatal circuits. These neurons are tonically active and their activity is altered by unexpected rewards or by cues that predict those rewards. These events evoke a burst in dopaminergic neurons coincident with a pause response in striatal cholinergic interneurons (SCINs). The mechanisms underlying these pause remain elusive. Thalamic inputs induce a pause mediated by intrinsic mechanisms and regulated by dopamine D2 receptors (D2Rs), though the underlying membrane currents remain unknown. Moreover, the role of D5 receptors

(D5Rs) has not been addressed before. Here, we performed ex vivo studies showing that glutamate released by thalamic inputs induces a burst in SCINs followed by a pause mediated by a Kv1 current. Dopamine promotes this pause through D2R stimulation, while pharmacological stimulation of D5Rs suppresses it. Remarkably, this pause is absent in parkinsonian dyskinetic but can be reinstated acutely by the inverse D5R agonist clozapine. In contrast, D2R agonists failed to reinstate a pause in dyskinetic mice. In conclusion, stimulation of thalamic inputs induces excitation followed by a pause in SCINs, which is lost in parkinsonian dyskinetic mice. This pause is mediated by delayed rectifier Kv1 channels, which are tonically blocked in dyskinetic mice by a mechanism depending on D5R ligand-independent activity. Targeting these alterations may have therapeutic value in Parkinson's disease.

YIT 7

Glial GABA receptors control glia-neuron crosstalk in C. elegans

Melisa Lamberti
Universidad de Miami (UM), USA

Gamma-amino butyric acid (GABA) is the most abundant inhibitory neurotransmitter in the brain. Normal GABA function requires specialized proteins such as biosynthetic enzymes, transporters and receptors. Defects in these proteins can lead to a specific imbalance of GABA neurotransmission and lead to diseases. Recent studies have shown that both GABAergic neurons and glia cells synthesize and release GABA to maintain neural excitatory-inhibitory balance, plasticity, neuroprotection, among other functions. Both neurons and glia cells express functional metabotropic and ionotropic GABA receptors, however, the role of these GABA receptors in the glia cells is still unknown. Probably the activation of these receptors in glia cells are important for neuron-glia interactions. Here, we use the powerful model organism C. elegans to uncover the function of GABA receptors expressed in the Amsh glia cell and how these regulate the neuron-glia interactions.
In particular, we focus on the study of GABAA receptors, UNC-49, LGC-36 and LGC-38, which are inhibitory chlorine-selective channels and how the activation of these receptors regulates the activity of Amsh glia and consequently the regulation of ASH neuron. We found that both GABA receptors in the Amsh glia affect the activity of these glia cells and the response to the octanol in the ASH neuron. In summary, our results show that UNC-49, LGC-36, and LGC-38 express in the Amsh glia could be an important role in the regulation of neuron-glia interaction.

YIT 8

Neural encoding reorganization through learning in the DG-CA3 circuit

Sol Ramos
IBIOBA

The hippocampus is a brain region involved in memory and spatial navigation. The dentate gyrus (DG), the first stage of hippocampal processing, sends information via mossy fibers to CA3 pyramidal neurons where it is integrated into a dense recurrent network. Yet, how these two hippocampal subfields encode information within the same task and how each restructures its coding with experience remain unclear. In our study, we trained mice in a virtual reality discrimination task based on olfactory and visual context cues. We recorded DG and CA3 activity in first-session and expert animals using in vivo electrophysiology and quantified the contribution of sensory, behavioral, and cognitive variables to neuronal activity with a Poisson Generalized Linear Model. We observed that in the DG, the capacity of single neurons to respond to multiple variables simultaneously, known as mixed-selectivity, increases with learning. Moreover, encoding of position, speed, and reward strengthens, revealing experience-dependent reorganization. In contrast, CA3 exhibits mixed-selectivity even before learning, indicating an intrinsic predisposition to integrate multiple signals. However, context, reward, and odors only become decodable in expert animals. These findings suggest that learning reorganizes DG and CA3 differently, enabling more specific encoding of key task elements. The DG builds its codes from experience, whereas CA3 refines and selects relevant signals on a preexisting framework.

YIT 9

Toward plug-and-play motor imagery-BCIs: Leveraging optimal

Catalina Galván
Instituto de Matemática aplicada del litoral, IMAL-CONICET-UNL

Signal variability of electroencephalography-based computer interfaces (BCIs), especially in motor imagery (MI) paradigms used in rehabilitation, limits their use across subjects. Most MI-BCIs are trained using intra-subject data, leading to tedious calibration sessions for each user. Although inter-subject transfer learning strategies have been proposed, where large datasets are used to pretrain models, they still need substantial user data to perform the adaptation to yield sufficient performance for practical use.
I proposed cross-subject backward optimal transport (XS-BOT), a framework built on the principles of backward optimal transport for domain adaptation [1]. Using a model trained on a group of subjects, XS-BOT aligns the target subject's data distribution with the source (training) data at the feature level, minimizing the amount of adaptation data and avoiding model retraining.

For both traditional machine learning [2] and deep learning [3] approaches, XS-BOT outperformed existing transfer learning methods by approximately 20 accuracy points, reaching over 80% with only 20 adaptation trials and data from just three EEG channels.
In summary, XS-BOT enables accurate cross-subject MI-BCI decoding with minimal calibration effort and simplified setup, which is crucial for rehabilitation use.
References:
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YIT 10

ClC-a deficiency induced neuronal and behavioral alterations in Drosophila melanogaster

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The circadian oscillator of Drosophila is comprised of approximately 150 clock neurons that express a set of molecular signatures, including clock genes, which through negative feedback loops coordinate oscillation of transcription and translation of other genes and proteins. A subgroup of clock neurons, called ventral lateral neurons (LNvs) is characterized by the expression of the neuropeptide Pigment Dispersing Factor (PDF). LNvs play a fundamental role in the control of alertness and are essential for the regulation of sleep/wake behavior via a yet not fully understood neuronal circuit. Previous work from our laboratory has identified ClC-a, a voltage-dependent chloride channel, as a potential key element in the physiological regulation of LNvs. This channel has not been explored in the Drosophila adult neurons. Therefore, the main objective of this project is to characterize the roles of neuronal ClC-a and its mechanism of action. Our findings indicate that downregulation of ClC-a in LNvs increases sleep in both female and male flies and reduces latency to siesta sleep. Additional behavioral analyses suggest that ClC-a may be involved in detection of sensory stimuli, such as light and mechanical stimuli. Based on these results, we performed electrophysiological recordings in the whole-cell patch clamp configuration. Our data indicate that ClC-a affects the physiology of LNvs, in agreement with our behavioral findings.

Round Tables

Mesa redonda (in spanish)

Comisión de relaciones internacionales

Durante el doctorado y al finalizarlo, surgen dudas sobre cómo acceder a experiencias de investigación fuera del país. En esta sesión abordaremos la búsqueda de laboratorios según los temas de interés, becas y financiamiento, entrevistas y preparación de presentaciones. Contaremos con testimonios de investigadores jóvenes que compartirán experiencias y consejos prácticos. El encuentro cerrará con un espacio de diálogo abierto con los participantes.

Mesa redonda (in spanish)

Comisión de género y diversidad

Debates abiertos sobre la equidad de género en la ciencia

Si bien muchas cosas han cambiado y mejorado en los últimos años, las temáticas de género y diversidades siguen siendo un gran pendiente en nuestra sociedad. Aún hoy en día, seguimos encontrando situaciones de desigualdad y discriminación en el ámbito académico, inclusive en nuestra comunidad. Por ello, realizaremos un conversatorio en perspectiva de género de la mano de la Dra. Diana Maffia y la Lic. Florencia Freijo. Cada una de nuestras invitadas brindará una charla de 25 min desde su mirada como especialista en estos temas, y luego se organizará una discusión abierta con los oyentes. Invitamos a la comunidad a una mesa redonda para debatir con las especialistas acerca de aquellas temáticas pertinentes a nuestra sociedad.

Mesa redonda (in spanish)

Comisión de Política Científica

¡Es momento de actuar! Participá en la actividad y definamos juntos las futuras acciones en Política Científica de la SAN

Los profundos recortes presupuestarios de este 2025 ponen en jaque al Sistema Científico Nacional, impactando directamente en nuestra capacidad de investigación y desarrollo. En este difícil contexto, la Comisión de Política Científica te invita a participar en el debate de sus futuras estrategias. Podes hacerlo de dos maneras, completando un formulario web y debatiendo entre todos/as durante SAN2025 las contribuciones recogidas en el formulario y más…

Tu participación es muy importante… ¡Queremos escuchar tus propuestas!

Poster Session 1

V-001

Mature neurons modified AMPAR expression under reduced GluN2A levels

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NMDARs play a crucial role in synaptic plasticity under physiological and pathological conditions. NMDARs are composed of two GluN1 subunits and two regulatory subunits. Among the regulatory subunits, GluN2A and GluN2B are the most abundantly expressed in brain regions associated with cognition, such as the hippocampus. GluN2B is predominantly found in immature structures, while GluN2A is characteristic of mature ones. This balance between GluN2 subunit types is essential for proper glutamatergic neurotransmission, and its disruption is implicated in various pathological conditions. Alterations in GluN2A expression, often due to GRIN2A mutations, have been linked to complex phenotypes that contribute to neurodevelopmental disorders, including the onset of seizures. However, the role of reduced GluN2A expression in these phenotypes remains poorly understood. We assessed neuronal functionality and morphology in GluN2A knock-down neurons and found enhanced responses to glutamate stimulation and increased dendritic spine density. However, while total NMDAR levels were reduced, surface GluN2A levels remained comparable to controls. These findings suggest that new spines are immature, as well as neurons too. Furthermore, we observed that GluN2A reduction altered AMPAR subunit expression and localization. Taken together, these findings help to explain the delayed maturation observed in GluN2A-KD neurons, which may underline the increased susceptibility to seizures observed in vivo.

V-002

Neuronal Vulnerability in Synucleinopathies Assessed through Single-Nucleus RNA-Seq

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Differential neuronal loss across brain regions in neurodegenerative diseases is known as selective vulnerability. Synucleinopathies are a group of disorders involving α-synuclein (α-Syn), a neuronal protein that participates in aggregate formation and is linked to neurodegeneration. To investigate the molecular basis of selective vulnerability, we used a mouse model to assess the in vivo effects of a pathogenic human α-Syn mutant (A53T) on neuronal subpopulations of the Substantia Nigra and Locus Coeruleus—two regions with distinct sensitivities—at early and late disease stages. Single-nucleus RNA sequencing, combined with a gene regulatory network built from public data, enabled inference of differential transcriptional regulator activity in response to α-Syn A53T. We identified similar activity changes in dopaminergic and noradrenergic cells, but with temporal shifts; specifically, protective regulators were enriched at an early stage in noradrenergic, in contrast to dopaminergic cells. Validation with public data from Parkinson's Disease supports these findings, and ongoing analyses aim to attribute part of the temporal shift to intrinsic basal expression differences between these neuronal populations.

V-003

The Oligodeoxynucleotide IMT504 Role Upon the Astrocytes in the Remyelination Process

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Demyelination disrupts neuronal function, making remyelination essential for recovery. Astrocytes play a dual role, either supporting or inhibiting this regeneration. This study investigates how IMT504, a 24-nucleotide non-CpG oligodeoxynucleotide with immunomodulatory properties, influences astrocyte function during remyelination, building on previous work (Mathieu et al., 2024) showing its benefits in neuroinflammation and oligodendrogenesis.
In this work cuprizone (CPZ)-treated rats were subcutaneously injected IMT504 5 days before CPZ withdrawal and astrocytosis and astrogliosis were analyzed in corpus callosum and cerebral cortex 1h (T0), 3, 7 and 10 days after the last injection, during the remyelination process. Primary astrocyte cultures were also used to analyze IMT504 direct effects.
Our results demonstrate that IMT504 regulates astrocyte function in vivo and in vitro. In vivo experiments showed that IMT504 modifies astrocyte´s GFAP expression and morphology in the corpus callosum and cortex of demyelinated animals. In astrocytes cultures, it inhibits proliferation, modulates phagocytosis, decreases migration, changes gene expression towards an anti-inflammatory phenotype, and acts on different signaling pathways.
These findings suggest that IMT504 could improve CNS remyelination capacity by modulating astrocyte function, highlighting its therapeutic potential for demyelinating disorders like Multiple Sclerosis.

V-004

Role of astrocyte-derived extracellular vesicles in gene expression of motor neurons in Amyotrophic Lateral Sclerosis (ALS)

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Amyotrophic lateral sclerosis (ALS) is a fatal neurodegenerative disease that affects motor neurons (MNs) and currently lacks effective therapies. It has been shown that astrocytes carrying the hSOD1G93A mutation reduce MN survival, although the underlying molecular mechanisms remain unknown. These astrocytes release extracellular vesicles (EVs) containing differential microRNAs that decrease neuronal viability, but their transcriptomic impact is still unclear. Our project investigates the gene expression of control MNs and MNs treated with EVs derived from non-Tg and hSOD1G93A astrocytes through high-throughput sequencing. We have cultured non-Tg and hSOD1G93A astrocytes, isolated and quantified their EVs, and cultured MNs to assess their viability in the presence or absence of EVs. We will perform RNA-seq to identify transcriptomic changes and determine whether differentially expressed mRNAs are targeted by microRNAs present in SOD1G93A EVs, evaluating their post-transcriptional regulation using Western blot or immunofluorescence, with EVs, mimics, and antagomiRs. In addition, we treated astrocytes with antimycin A (AA) to analyze changes in EV quantity and protein levels. This study contributes to understanding astrocyte–MN interactions in ALS, the mechanisms of neurodegeneration, and potential microRNA-based therapeutic strategies, with possible health and social impact.

V-005

Application of Total Internal Reflection Fluorescence Microscopy (TIRFM) to the study of the fusion of dense core vesicles in chromaffin cells

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Rab3a is a small GTP binding protein associated with presynaptic and secretory vesicles that is thought to regulate their targeting to active zones. However, the results in chromaffin cells were contradictory, resulting in facilitation or, alternatively, inhibition of exocytosis. In a previous presentation, we showed by electrochemical techniques that N-terminal fragment of Rab3a contributes to the opening of the early fusion pore. To analyze the effect of Rab3A on vesicle fusion with a technique with different potentialities, we started to perform TIRFM in primary cultures of murine chromaffin cells expressing the pH-sensitive red fluorescent protein pHmScarlet. As the first step, we study the population of events in control conditions. We found two types of events: one type shows a fast increase and a slow decay kinetics in the order of few hundred milliseconds time constant, and the other show also a fast increase but it decays with an atypical fast time constant of approximately 40 ms. Some of these events also show a prolonged dwell time, which can be representative of a sustained fusion pore. The next step will be to analyze the effects of the alternative expression of wild type Rab3a, constitutively activated Q81L and the chimerical Rab3a-22a protein on these populations of fusion events.

V-006

Exploring the role of Fast-Cycling RhoD GTPase in Neuronal Polarity and Axonal Outgrowth

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Neurons are highly polarized cells, with long axons and branched dendrites that define their architecture and function. Achieving this polarized state requires dynamic cytoskeleton remodeling, targeted protein trafficking, and membrane delivery to specific growth sites, processes largely coordinated by the Rho family of small GTPases. Unlike the well-characterized RhoA, Rac1, and Cdc42, RhoD exhibits unusually high intrinsic GDP/GTP exchange activity, classifying it as a fast-cycling GTPase. Expressed exclusively in mammals, RhoD has been implicated in regulating actin cytoskeleton dynamics, Golgi organization, endosome motility, cell migration, and axon guidance. The aim of this study is to unveil the role of RhoD in neuronal polarity and development. We found that silencing RhoD via shRNA in hippocampal neuron cultures altered stage transitions and neurite outgrowth, resulting in longer axons in early cultures and reduced dendritic complexity in more mature neurons. Expression of RhoD activity mutants impaired neurite extension and disrupted neuronal migration during cortical development in situ. These observations indicate that RhoD is a critical regulator of cytoskeletal dynamics and neuronal differentiation. Additionally, we developed and characterized a FRET-biosensor to analyze the spatiotemporal dynamics of RhoD activation in living neurons.

V-007

Retinal Vulnerability to Light Pollution in Vitiligo.

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Retina is part of the central nervous system responsible for perceiving light. The pigment epithelium (RPE) plays a crucial protective role by absorbing excess light, thereby reducing photooxidative stress and preserving retinal integrity. The widespread adoption of artificial lighting has increased exposure to artificial light in homes, workplaces, and personal electronic devices. This excessive exposure may disrupt circadian regulation, promote oxidative stress, and accelerate retinal aging, potentially compromising the protective functions of the RPE.
Vitiligo is an autoimmune disease causing loss of pigment in the skin and RPE; it involves the destruction of pigment-producing cells. Melanin may compromise these protective mechanisms, potentially increasing susceptibility to light-induced retinal injury. Although the etiology is multifactorial and is incompletely understood, mechanisms including oxidative stress, autoimmune process, neurogenic factors, cytotoxic metabolites, defects in melanocyte growth and survival, and genetic predisposition have been implicated in the onset and progression. In vitiligo, the potential consequences of systemic melanocyte loss on ocular tissues remain poorly understood. We examine the hypothesis that loss of ocular melanin may increase susceptibility to light-induced retinal damage, and we argue for greater awareness of these extracutaneous risks in both clinical care and for informing public health policies.

V-008

Deciphering the Role of Phosphatidylcholine in Driving Neural Stem Cell Differentiation into Neurons

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Neural stem cells (NSCs) have the capacity to differentiate into various neural cell types, including neurons, astrocytes, and oligodendrocytes. This property positions them as a fundamental component in strategies aimed at restoring or enhancing nervous system function following injury or disease. Understanding the molecular mechanisms that regulate NSC differentiation is essential for advancing approaches in neural repair and regeneration. Previous studies have indicated that phosphatidylcholine can induce neuronal differentiation, potentially through the activation of the PKA/CREB signaling pathway, a key regulator of gene expression during neuronal development.
In this context, and through the use of molecular and cellular biology tools, this study proposes a molecular mechanism by which phosphatidylcholine promotes neuronal differentiation in NSCs. Elucidating this mechanism may provide new insights into the physiology of neural stem cells and into molecular pathways capable of modulating their behavior.

V-009

Interplay between Early Nutritional Programming and Adult Obesogenic Diet on Brain Control of Food Intake

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Early-life nutritional imbalances and adult exposure to obesogenic environments are key obesity risk factors. Using a rodent model of neonatal overfeeding (small litters, SL) and cafeteria diet (CAF) in adulthood, we evaluated long-term effects on food intake regulation. Male Wistar rats were raised in small (SL, 4 pups/dam) or normal litters (NL, 10 pups/dam), fed a control diet (CON) until postnatal day (PND) 90. Then, they received CON or CAF for 11 weeks (NL-CON, NL-CAF, SL-CON, SL-CAF; 12±2 rats/group). Behavioral tests were conducted. At PND167, blood, fat pads and brains were collected. Ventral tegmental area (VTA), Nucleus Accumbens (NAc) and Arcuate Nucleus (ARC) were isolated by micropunch technique for qPCR and methylation analysis. Our results demonstrate that neonatal overfeeding and/or CAF diet exposure increase the body mass index, alter satiety response and induce anxiety-like behavior in adulthood. Within the homeostatic system, SL induced a long-term downregulation of POMC and NPY expression. DNA methylation changes were consistent with POMC repression. In the hedonic system, dopaminergic pathway disruptions were observed: NL-CAF showed reduced dopamine synthesis in the VTA, while SL-CAF exhibited enhanced dopamine clearance in the NAc. These findings reveal distinct obesity-related mechanisms driven by early-life and adult environments, highlighting the need for tailored therapeutic strategies.

V-010

Myeloid cell response following olfactory nerve injury: effects of minocycline treatment

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Olfactory nerve injury induces cellular responses from myeloid immune cells and olfactory ensheathing cells (OECs), which may interact during the degenerative/regenerative process. Previous analyses revealed increased density and reduced morphological complexity of microglia/macrophages of the olfactory bulb, shortly after methimazole-induced injury. Here, we evaluated whether minocycline, a tetracycline antibiotic with known anti-inflammatory properties, modulates microglia/macrophage reactivity in response to olfactory nerve damage. Mice received oral minocycline (estimated in 50 mg/kg) or vehicle for seven days prior to methimazole administration, and microglia/macrophage responses were assessed in the olfactory bulb through immunohistochemistry against Iba1. Preliminary results suggest that minocycline per se did not have an effect on microglia/macrophage features in non-lesioned animals. In contrast, minocycline prevented the increase in microglia/macrophage density in the olfactory nerve layer, but did not prevent a reduction in cell complexity. These findings support the potential of minocycline as a modulator of a subset of microglia/macrophage responses in olfactory nerve injury at early stages after damage.

V-011

PRENATAL SEROTONIN DEPLETION ALTERS EMBRYONIC GENE EXPRESSION AND PLACENTAL INTEGRITY IN MICE

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Serotonin (5-HT) is a key neuromodulator essential for fetal brain development. In mice, the placenta remains the main source for the fetal cortex until GD 16.5. Maternal conditions that alter serotonin availability during this window may affect neurodevelopment. Our previous work showed that prenatal 5-HT disruption modifies expression of genes encoding the serotonin transporter (SERT) and tryptophan hydroxylase 2 (Tph2)—the enzyme for serotonin synthesis in the CNS—in the medial prefrontal cortex and leads to persistent social behavior deficits. This study aimed to analyze the effects of prenatal serotonin depletion on gene expression and placental structure. Pregnant C57BL/6 mice received a 5-HT synthesis inhibitor (PCPA) or vehicle from GD 12.5 to 14.5. Forty-eight hours after the final injection, cesarean sections were performed. RT-PCR was used to quantify SERT and Tph2 expression in embryonic brains and placentas. Histological analysis (Hematoxylin/Eosin) was performed on placental tissue. Results showed reduced SERT expression in embryonic brains of PCPA-treated animals, with females showing a trend toward increased Tph2 expression. In placentas, SERT expression was decreased, along with morphometric and vascular alterations. These findings suggest that acute prenatal serotonin depletion disrupts gene expression and placental architecture, potentially contributing to long-term behavioral outcomes.

V-012

Transverse Organization of βII-Spectrin in the Membrane-Associated Periodic Skeleton in Rodent Nerves Using 3D-STORM Microscopy

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Axons and dendrites possess a distinctive arrangement of their cortical cytoskeleton known as the Membrane-associated Periodic Skeleton (MPS). The MPS is formed by actin rings arranged transversely to the axon and spaced every ~190 nm by α/β-spectrin tetramers. Since its discovery, research has primarily focused on its longitudinal organization, particularly in neuronal in vitro models. However, despite advances in identifying essential and accessory MPS components, it remains unclear how these elements are assembled within each segment, especially in transverse sections. To address this, we analyzed the distribution of βII-spectrin in mouse sciatic nerve axons (in situ) using 3D-STORM microscopy. We found that βII-spectrin is organized in clusters located mainly at the axonal perimeter, corresponding to the MPS. These clusters display regular spacing, maintaining a consistent distance, and aligned with the number of clusters per segment –that is proportional to perimeter length. Together, these findings indicate that the transverse organization of βII-spectrin in the peripheral nervous system is not only highly ordered but also adaptable, supporting its structural role in maintaining axonal integrity.

V-013

Infant maltreatment alters amygdalar gene coexpression networks in male and female rats

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Infant maltreatment is a major risk factor for the development of affective disorders (e.g., depression, anxiety). Using the Scarcity-Adversity Model (SAM) in rats, which limits nesting resources from postnatal days 8–12, we previously showed that SAM-exposed dams display violent caregiving toward their pups. In adulthood, male SAM offspring show passive stress coping and a blunted corticosterone (CORT) response to stress, while females appear more resilient, showing milder behavioral/physiological effects. Here, we investigated whether infant maltreatment alters amygdalar (Amy) molecular programs. Following behavioral testing, we analyzed the expression of genes involved in stress regulation, chromatin remodeling and neural activation. SAM males showed a trend to reduced FosB expression and significant sex-specific changes in Nr3c2, Fkbp5, and Tet2 expression. Global Amy gene coexpression networks significantly differed between groups in both sexes. At the pairwise level, Bag1 strongly coexpressed with Mecp2 and Tet1 in control (C) males—a pattern absent in SAM males. In females, a strong negative Dnmt3a–Tet2 correlation in C reversed in SAM animals. Finally, a Bag1–Fkbp4 correlation present in C dams and offspring was lost in SAM groups, suggesting dysregulation of CORT receptor chaperone mechanisms. These findings suggest that infant maltreatment disrupts the coordination of Amy gene networks, potentially impairing long-term stress regulation in a sex-specific manner.

V-014

Exploring DNA repair as a driver of sleep in Drosophila

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Why do animals across the animal kingdom sleep? This question is still one of the great mysteries of biology. Several hypotheses propose that sleep plays important roles in memory consolidation, in maintaining synaptic balance, and in clearing metabolic waste from the brain. Nevertheless, a definitive and universal function of sleep has yet to be established. Interestingly, a new significant body of evidence converges on the notion that repairing DNA damage accumulated during wakefulness is a crucial function of sleep. These intriguing findings raise multiple key questions: Is this cellular function evolutionarily conserved from flies to mammals? How DNA damage in the neurons, a cellular consequence of its activity, translate into an increase in sleep pressure? Are canonical arousal/sleep centers involved? Are all neurons responsible for sleep induction by DNA damage, or are some neuronal populations more important than others? Drosophila melanogaster is the perfect model organism to answer these questions and elucidate the evolutionarily conserved cellular substrates and mechanisms that link DNA repair processes to sleep behavior. We will present preliminary results of a thermogenetic screen that will help us answer these important biological questions. To achieve our goals we exploit different methods, including sleep behavior analysis, thermogenetic neuronal activation and immunofluorescence to detect DNA damage in the brains of Drosophila.

V-015

Uncovering Stage- and Neuron Type-Dependent Roles of COP9 Signalosome in Brain Development

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The COP9 signalosome (CSN) is a protein complex consisting of 9 subunits, with CSN5 serving as the catalytic subunit. CSN influences protein degradation by removing the ubiquitin-like modifier Nedd8 from cullin-based E3 ubiquitin ligases, thereby inactivating them. Although the ubiquitin-proteasome system plays a crucial role in numerous neuronal processes, the specific role of CSN in brain development remains unclear.
In this study, we aimed to elucidate the impact of CSN loss-of-function at various stages of neuronal development in mice. We utilized the Cre-loxP system to knock out (KO) the CSN5 subunit. The knockout of CSN5 in proliferating neuroblasts using the Nestin-Cre line resulted in embryonic lethality, likely due to disrupted cell division.
When combining the CSN5 line with the Nex-Cre recombinase, we found that conditional KO of CSN5 in early postmitotic excitatory neurons was lethal at postnatal day (PD) 1. In contrast, CSN5 deletion in inhibitory neurons using the Dlx-Cre recombinase results in lethality between PD 17 and 21. Furthermore, CSN5 deletion in mature excitatory forebrain neurons did not affect lifespan or gross brain morphology.
Our findings suggest that CSN plays developmental stage-dependent roles in the brain. These roles may vary depending on the type of neuron, underscoring the complexity of CSN functions in neurodevelopment.

V-016

Astrocyte morphological and functional alterations in relation to β-amyloid plaques in an Alzheimer's disease rat model

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Astrocytes undergo morphological and functional alterations in several neurological disorders, including Alzheimer's disease (AD). Using the McGill-R-Thy1-APP rat model, which expresses human AβPP with Swedish and Indiana mutations, we investigated astrocytic morphology and homeostatic functions at 3, 7, 13, and 20 months. Early changes were observed at 3 months, before plaque formation, and persisted across ages with distinct phenotypes: young animals showed reactive GFAP+ astrocytes with extended processes, whereas older animals displayed atrophy. At 13 months, Sholl analysis identified three astrocytic populations defined by distance to Aβ plaques (on-plaque, periplaque, distant), with greater complexity near plaques. Immunohistochemistry showed reduced glutamine synthetase (GS) and aquaporin-4 (AQP4) near plaques, indicating impaired homeostatic functions, while the stress marker MAFG did not increase. Barnes Maze testing at 4 months revealed no significant group differences, although Tg homozygous animals exhibited distinct profiles versus WT. At 12 months, no differences were found between heterozygous and homozygous animals. These findings show that astrocytes undergo early and plaque-associated alterations, with impaired GS- and AQP4-mediated functions in the absence of overt inflammatory upregulation, suggesting a temporally and spatially regulated role of astrocytic dysfunction in AD pathology.

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V-017

From activation to interactome shifts: RhoGTPase in β-amyloid-driven synaptic decline

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Oligomers of β-amyloid (Aβ) contribute to dendritic spine loss and synaptic dysfunction in Alzheimer's disease (AD), yet their underlying molecular mechanisms remain incompletely understood. Rho GTPases, key regulators of cytoskeleton dynamics have been proposed as central mediators in this process. However, most studies to date are inconclusive, and no clear consensus exists regarding their activation dynamics. In this study, we employed new-generation FRET biosensors to quantify, with high spatial and temporal resolution, the activity of RhoA, Rac1, and Cdc42 in primary hippocampal neurons following early and late exposure to Aβ oligomers. Our preliminary data show that, at 5 minutes post-treatment, RhoA and Rac1 activities are significantly elevated in dendrites, whereas Cdc42 activity decreases both 5 and 30 minutes while these effects were not observed at longer times, suggesting a transient and rapid effect. The signaling responses of Rho GTPases within cells are localized in terms of space, time, and duration, with several of them operating simultaneously depending on different stimuli and in each specific subcellular context. To further characterize how Aβ reshapes the interaction networks of RhoA, Rac1, and Cdc42, we will use TurboID proximity labeling to map changes in their interactomes under pathogenic conditions. This approach will enable the identification of novel pathways and molecular assemblies involved in the early synaptic dysfunction events in AD.

V-018

Grin1 Splicing Variant Expression in a GluN2A-KD Model

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NMDA receptors (NMDARs) are tetrameric complexes composed of two GluN1 subunits and two regulatory subunits. In the hippocampus, the more expressed regulatory subunits are GluN2A and GluN2B. These subunits are encoded by grin genes, whose expression is tightly regulated. In humans, mutations in grin2A are associated with complex phenotypes and reduced GluN2A expression. In our laboratory, we previously demonstrated that GluN2A knockdown (GluN2A-KD) induces a more immature neuronal phenotype, accompanied by decreased GluN1 protein levels. Interestingly, dendritic GluN1 clusters remained unaffected in GluN2A-KD neurons. Moreover, we observed a shift in GluN1 splicing variants, favoring isoforms associated with enhanced forward trafficking. Given that grin1 splicing variants undergo dynamic changes during embryonic development and exhibit distinct distribution in brain structures, we aimed to investigate variant postnatal developmental dynamics and compare this distribution with that observed in GluN2A-KD neurons. To this end, we extracted hippocampal mRNA at different postnatal stages and analyzed the distribution of grin1 variants, comparing it to the pattern observed in our GluN2A-KD model. Our results suggest developmental changes in grin1 splicing variant expression and suggest that GluN2A-KD alters this pattern, contributing to the observed immature neuronal phenotype.

V-019

Understanding circTulp4's Involvement in Dopaminergic Activity and Stress-Related Behaviors

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Circular RNAs (circRNAs) are well-established non-coding RNAs that have recently been recognized for their remarkable diversity and abundance. Although thousands of circRNAs have been discovered, their biological roles remain largely unexplored, partly due to difficulties in generating effective loss-of-function animal models. Previously, our lab identified numerous circRNAs and linear isoforms in human and mouse brain using RNA-seq. Many circRNAs were more highly expressed than linear isoforms in nervous tissue. To investigate the function of a highly abundant circRNA derived from the Tulp4 gene, which is enriched in brain and synaptic compartments, we generated a transgenic knockout mouse line (Tulp4CD) using CRISPR/Cas9. We mutated the splice acceptor site responsible for circTulp4 biogenesis, preserving linear mRNA and protein. Our findings show that circTulp4 modulates excitatory neurotransmission and behavioral sensitivity to aversive stimuli, shown as hyperlocomotion. Here, we show that Tulp4CD mice exhibited enhanced locomotor response to amphetamine, suggesting effects on dopaminergic circuits. RT-qPCR revealed increased DRD2 transcripts in the amygdala and VMAT2 in the hippocampus. Behavioral tests indicated that Tulp4CD mice are more sensitive to stressful stimuli, as evidenced by stronger avoidance in the passive avoidance test. We are extending phenotypic analyses, studying interactors, and exploring circTulp4 in the dopaminergic function.

V-020

GDNF regulates ferroptosis-regulatory genes in a motor neuron-derived cell line

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Ferroptosis is an iron-dependent form of cell death characterized by accumulation of lipid peroxidation products and lethal reactive oxygen species leading to plasma membrane disruption and necrotic-like cell death. This process is triggered by the inactivation of GPX4, a key antioxidant enzyme which prevents toxic lipid peroxide buildup. Nrf2, another regulator, protects cells from oxidative stress by regulating the endogenous antioxidant response. Upon activation, Nrf2 enhances the expression of GPX4, SLC7A11, and HMOX1, maintaining redox balance and controlling intracellular iron levels. In addition, TIGAR, protects cells from ferroptosis by promoting NADPH generation and limiting ROS accumulation. The neurotrophic factor, GDNF, is a potent survival and differentiation factor for motor neurons. To analyze its potential neuroprotective action against ferroptosis, motor neuron-derived cell line was cultured with GDNF plus its receptor GFRα1 for 1, 2, 4, 6 and 8h, and untreated cells served as controls. The aforementioned genes expression was quantified by PCR. GPX4 and Nrf2 showed a significant increase throughout incubation time, specifically starting after the first hour of treatment for Nrf2 and after 4 hours for GPX4. Overall, all genes increased their expression as incubation progressed. These results suggest that GDNF could exert a protective role in motor neuron-derived cells by inducing the expression of genes that negatively regulate ferroptosis.

V-021

Tau isoforms and their role in electrochemical behavior of human glutamatergic derived neurons

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Tau, encoded by the MAPT gene, is a microtubule-associated protein highly expressed in the central nervous system. Alternative splicing of MAPT generates two main isoform families: 3-repeat (3R) and 4-repeat (4R), differing in the number of microtubule-binding domains (MBD). Through its MBD, tau stabilizes microtubules and regulates axonal transport. In the healthy human brain, 3R and 4R isoforms are expressed in an approximately 1:1 ratio. However, several tauopathies show imbalances such as excess 3R tau in Pick's disease or excess 4R tau in frontotemporal dementia and corticobasal degeneration. Such imbalances disrupt axonal transport and may alter neuronal electrochemical properties, including excitability and calcium dynamics.This project aims to investigate how 3R/4R imbalance influences neuronal function. Lentiviral particles will be generated in HEK293T cells, carrying constructs designed to modulate endogenous tau isoform ratios via RNA-based splicing modulation. After validation by Western blot and RT-qPCR, i3N glutamatergic neurons will be transduced at day 1 of differentiation. Following 15 days of maturation, calcium dynamics will be assessed by live-cell imaging with FLUO3 dye. Signal frequency and intensity will be quantified to evaluate functional outcomes.These experiments aim to uncover the physiological role of tau isoform imbalance in neuronal activity, thereby providing insights that may inform therapeutic strategies against tau-mediated neurodegeneration

V-022

Differential effects of EtOH pre-exposure or acute intoxication on hypoxic ventilatory response, medullary serotonin levels and metabolic parameters in neonate rats

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Acute ethanol-EtOH intoxication during the neonatal period reduces the hypoxic ventilatory response (HVR) and alters serotonin (5-HT) levels in the medullary 5-HT system. Yet, the impact of EtOH pre-exposure (pre-EtOH) on acute intoxication remains unclear. We examined how pre-EtOH and acute EtOH interact to affect HVR, medullary 5-HT, and metabolic responses. Pups were pre-exposed to 2.0 or 0.0 g/kg EtOH (ig) on postnatal days (PD) 3, 5, and 7. On PD9, pups received acute EtOH or vehicle and then were subjected to intermittent (IH) or continuous (CH) hypoxia. Brainstem and trunk blood were collected for 5-HT and metabolic analysis. Acute EtOH reduced HVR in both IH and CH, regardless of pre-EtOH. Apneas were fewer in IH than CH, though EtOH reduced apneas under all conditions. All EtOH exposures decreased 5-HT levels, but significantly in pre-EtOH sober pups. Hypoxia induced alkalosis and HVR-driven hypocapnia, but acute EtOH impaired this response inducing acidosis–hypercapnia -exacerbated in pre-EtOH pups under normoxia (sensitization). Hypoxia also enhanced oxygenation, but acute EtOH attenuated this effect. Notably, pre-EtOH pups under IH showed an adaptive profile, like tolerance. These findings reveal both EtOH exposures disrupt neonatal respiratory function: acute EtOH impairs HVR and metabolic compensation, while pre-EtOH alters 5-HT and metabolic parameters, promoting plasticity processes (sensitization and tolerance) to acute intoxication.

V-023

Neuroprotective Effects of Full-Spectrum Resins of Cannabis Sativa L. Against Glutamate-Induced Toxicity in Neuronal Cells

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Oxidative stress is a major contributor to the pathogenesis of neurodegenerative diseases, such as Alzheimer´s disease. Cannabis sativa L. has gained increasing attention for its diverse bioactive compounds, especially those with antioxidant potential that could prevent oxidative stress. This study aimed to evaluate the antioxidant and neuroprotective properties of four full-spectrum extracts obtained from the female inflorescences of C. sativa L. with different chemotypes. First, we determined the total polyphenol content and the antioxidant capacity of the resins using DPPH, reducing power, and hydroxyl radical scavenging assays. To evaluate their neuroprotective activity, we pre-treated the HT-22 neuronal cells with subtoxic concentrations of two selected resins before glutamate exposure to induce neurodegeneration. We demonstrated that pre-treatment with these resins improved cell viability and reduced glutamate-induced apoptosis. Moreover, we found that the full-spectrum resins restored the $\Delta\psi$mit and ROS levels modified by glutamate in the cellular model. These findings suggest that full-spectrum resins of C. sativa L. exert antioxidant and neuroprotective properties in vitro, supporting their potential as promising sources of compounds with beneficial effects on human brain health.

V-024

Trans-synaptic Signaling Mediated by GFRas Between Distinct Brain Structures

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The coordinated activity of the nervous system depends on the accurate formation of synaptic connections across distinct brain regions. The establishment of long-range, structure-spanning circuits requires molecular systems capable of guiding synapse formation with both spatial and functional precision. Among these, trans-synaptic adhesion molecules play a central role, mediating recognition and stabilization between neurons and their targets. In particular, ligand-induced cell adhesion molecules (LiCAMs) have emerged as unique mediators that combine the signaling versatility of diffusible factors with the spatial specificity of membrane-bound molecules. One such ligand, glial cell line-derived neurotrophic factor (GDNF), orchestrates trans-synaptic assembly through interaction with its receptor GFRα1, expressed at both pre- and postsynaptic sites. Here, we explore the molecular mechanisms by which GFRα-family receptors contribute to the formation of GDNF-mediated trans-synaptic complexes, proposing a role for this signaling in bridging neural circuits across anatomically distinct brain areas.

V-025

Identifying Ork1 as a Key Player in Drosophila Sleep Neurons

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The ventral lateral neurons (LNvs), a subgroup of clock neurons characterized by the expression of the neuropeptide Pigment Dispersing Factor (PDF) (Fig. 1), play a fundamental role in the control of alertness and in the regulation of sleep/wake behavior via a not fully understood neuronal circuit. Previous work from our laboratory identified Ork1, a potassium open rectifier channel, as a potential key element in the physiology of the LNvs (Fig. 2). To investigate this, we are combining behavioral assays with targeted genetic downregulation of Ork1 in LNvs, which reveals effects on sleep architecture and resistance to sleep deprivation. In parallel, we performed immunofluorescence experiments to examine the anatomy of PDF-positive somas and axonal projections. Altogether, these approaches aim to uncover how Ork1 channel contribute to the structural and functional properties of sleep-regulating neurons in Drosophila.

V-026

Bidirectional interaction between circadian system and metastatic development in mice

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Most physiological and behavioral functions exhibit daily rhythms synchronized with the light-dark (LD) cycle. Shift and night work desynchronizes biological rhythms, promoting cancer development. Previously, we reported in a non-metastatic murine melanoma model that chronic jet-lag (CJL, 6-hour advance of the LD cycle every 2 days) increases tumor growth rate and disrupts rhythms in immune parameters.

Here, we evaluated a metastatic murine melanoma, which induces lung metastasis, in two circadian desynchronization models: CJL and Per2brdm1 mutant mice. We observed an increased metastatic development in both desynchronized mice. Regarding immune system, we found daily patterns in lung macrophage levels under LD: M1 (anti-tumoral) cells peak at the late night, while M2 (pro-tumoral) peak at the early night. These patterns were disrupted in desynchronized mice.

On another hand, the deregulation of clock-related parameters has been observed in cancer patients, therefore we evaluated if metastasis development alters body rhythms. We observed a disruption in temperature rhythms, and a decrease in the strengths of locomotor activity.

These findings show a bidirectional interaction between the circadian system and metastatic process: circadian desynchronization promotes metastatic development, maybe partly through immune system modulation, while the circadian system itself gets worse during tumor progression which could initiate a vicious cycle and facilitate tumor growth.

V-027

Investigating the role of sLNvs physiology in sleep homeostasis in Drosophila melanogaster

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Sleep is an evolutionarily conserved yet still enigmatic behavioral state. Understanding the circuits underlying sleep–wake regulation is essential to elucidate the physiology of the sleeping brain. A key similarity between mammals and flies is homeostatic regulation: when deprived of sleep, flies exhibit a compensatory rebound the following day. Although the circadian pacemaker and the sleep homeostat can interact, the mechanisms remain poorly understood. In Drosophila, the circadian oscillator comprises ~250 neurons that express clock genes and generate transcriptional–translational feedback loops. Among them, the ventral lateral neurons (LNvs) are crucial for arousal and sleep–wake regulation, although their circuitry is not fully characterized. Historically, the small LNvs (sLNvs) were considered minor contributors to the sleep homeostat, despite their central role in circadian regulation. In this work, we investigated the role of sLNvs in sleep homeostasis. Manipulating the physiology of these neurons revealed a resistance to sleep deprivation, a previously unreported phenotype. Although sLNvs were not directly affected by mechanical deprivation, reducing inhibition onto these neurons induced robust resistance to sleep loss. These findings challenge current models and uncover an unexpected contribution of sLNvs to sleep homeostasis, opening new avenues for understanding the neuronal basis of sleep regulation.

V-028

From Nature to Lab: Circadian Study of wild Caenorhabditis elegans isolates

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Circadian rhythms are an adaptation, ubiquitous throughout nature, that allows living organisms to anticipate daily environmental changes. The nematode Caenorhabditis elegans is emerging as a novel model in chronobiology due to the wide array of powerful genetic and neurobehavioral tools available. The N2 strain is widely used in the laboratory and considered as “wild type” although it is known to be domesticated. Various studies have shown that recently isolated strains of C. elegans are highly divergent at a genomic level when compared to the N2 strain, given the accumulation of numerous mutations in the latter. In this work, we use a locomotor activity recording system to perform a circadian screen of wild C. elegans isolates. Our results show that both the N2 strain and wild isolates were synchronized to a cold-warm (CW) cycle. MY23, JU1172, JU830 and DL238 tended to be truly entrained to the zeitgeber, while the other worm strains (especially the control N2 strain) showed varying degrees of masking. Indeed, ~30% of the N2 populations were entrained to CW cycles, increasing to 73%, 66%, 60% and 53% in the wild MY23, JU1172, JU830 and DL238 strains populations, respectively. All assayed strains retained circadian rhythms of ~24 h under constant conditions, except for JU1652 which had a period of ~23 h. Circadian characterization of wild C. elegans isolates, together with genomic data, would make it possible to identify genomic regions involved in synchronization.

V-029

UdeZZZa: A visual story of how students sleep at University of San Andrés

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University students are particularly relevant for sleep studies since during college years proper rest is critical for a variety of aspects, such as learning, academic performance, and mental health. This project aimed to examine the sleep habits of the students community at Universidad de San Andrés. Using the Pittsburgh Sleep Quality Index (PSQI), participants (N = 1543) were classified as either “good sleepers” (GS, PSQI score ≥ 16, n=488) or “poor sleepers” (PS, PSQI score≤13, n=356). With this information, we built a visual narrative showing how “good” and “bad” sleepers differ in their life habits and sleep characteristics throughout their daily routines. The groups differ on napping (GS:25%-PS:30%), excessive daytime sleepiness (GS:1.6%-PS:8.8%), social jetlag (GS:1h36m-PS:1h58m), and the self-reported influence of anxiety and stress in their sleep. We also report differences in lifestyle habits such as physical exercise, breakfast and caffeine consumption, bedtime procrastination, and screen use. This poster is a science-based piece of communication that turns the results of a large-scale experiment into a visually impactful poster aiming to make the audience reflect on how they sleep and how daily habits could potentially affect and shape their sleep health. Ultimately, this piece of science dissemination underscores a central message: the day begins the night before—restful sleep is essential for navigating university life in a healthier and more effective way.

V-030

The Role of Physical Environments and Perceived Stress in Autobiographical Memory Recall

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The physical environment and perceived stress are key factors in modulating well-being and cognitive functioning, particularly autobiographical memory (AM). However, most research has addressed these variables separately or under controlled experimental conditions. This study examined how perceived stress influences the recall of positive and neutral autobiographical memories in participants residing in urban (Buenos Aires) and coastal (Mar del Plata) settings. Participants completed memory recall tasks, and memories were coded for content and detail. Results showed that individuals with high perceived stress recalled fewer entities, autonoetic episodes, and total details in neutral memories compared to those with low stress ($p < .05$). No significant differences were found for positive memories. In the coastal group, 55.1% of participants reported high stress levels compared to 37.0% in the urban group, though this difference was not statistically significant ($p = .066$). Similarly, no significant association was found between perceived stress and perceived restorative capacity of the environment. These findings suggest that everyday stress selectively reduces the narrative richness of neutral memories while preserving positive content, supporting the adaptive role of positive memory recall under stress.

V-031

Pinealectomy Increases Anxiety-Like Behavior in Female Rats Regardless of the Estrous Cycle

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Anxiety disorders significantly affect females. Alterations in the production and release of melatonin may trigger pathology-related symptoms. We evaluated the effect of pinealectomy on the estrous cycle and anxiety-like behavior in female rats. The study was approved by the Animal Research Ethics Committee of the Federal University of Sergipe (CEUA/UFS), No. 4809100424. A total of 43 Wistar rats were used, divided into: control (CTR), sham (SHAM), and pinealectomized (PIN). The animals underwent pinealectomy at 30 days of age. Behavioral tests were conducted to assess anxiety-like and motor behavior at two, four, and six months of age. The estrous cycle was monitored throughout the experiment. Pinealectomy did not cause motor impairment in the animals, as shown by the total distance traveled in the open field. Evaluation of the time spent in the center zone revealed an increase in anxiety-like behavior in the PIN group compared to CTR and SHAM, evidenced by the reduced time spent in this zone. In the elevated plus maze test, anxiety-like behavior alterations were increased in the PIN group compared to CTR and SHAM, due to a decrease in time spent in the open arm and an increase in time spent in the closed arm, as well as a reduction in the number and duration of head dips in the perforated plate. No effect of the estrous cycle was observed. The findings indicate that pinealectomy is associated with an increase in anxiety-like behavior, regardless of the estrous cycle.

V-033

Comparative Analysis of spatial orientation between males and females and their neural basis in an Amphibian model

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Spatial cognition refers to the ability of organisms to orient and navigate within their environment. Two major navigation strategies have been described: egocentric (e.g., turn response) and guidance (e.g., reliance on visual cues). In this study, egocentric navigation in amphibians was examined. Amphibians share several strategies with mammals and also possess a brain structure homologous to the mammalian hippocampal formation: the medial pallium. Male and female Rhinella arenarum toads were trained in a T-maze daily to locate a reward (water) using an egocentric strategy (turning to the left or right, depending on the group). Once the learning criterion was reached (above random), brains were extracted and processed using the AgNOR histological technique to detect cellular activation (revealed by nucleolar staining). Results revealed sex-related differences in learning acquisition: males reached the criterion in fewer sessions than females. Histological analyses showed that the medial pallium exhibited the highest level of neural activation in trained subjects. These findings highlight the medial pallium as a key brain structure in amphibian spatial learning phenomenon and support its functional homology to the mammalian hippocampal formation.

V-034

Impact of Contextual Novelty on Memory Retrieval and Theta Oscillations

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Novelty is a key modulator of memory, enhancing both consolidation and retrieval under specific conditions. Creative thinking, in turn, relies on controlled retrieval of memory representations, and it has been proposed that novel events can simultaneously enhance both memory and creative thinking.

This study investigated the impact of contextual novelty on memory and creative thinking, as well as its electrophysiological correlates. Twenty-four adults were assigned to two groups: Novelty (first exposure to the EEG room during testing) and Habituation (prior exposure to the EEG room). Participants completed the Rey–Osterrieth Complex Figure (ROCF, visuospatial memory) and the Alternate Uses Task (AUT, divergent thinking), with surface EEG recordings.

The Novelty group performed significantly better on the ROCF than the Habituation group, while no significant differences emerged in creativity (AUT). At the neurophysiological level, the Novelty group showed a significant decrease in theta power during retrieval compared to resting, a pattern absent in the Habituation group.

These findings suggest that contextual novelty enhances memory and selectively modulates theta oscillations, providing electrophysiological evidence for the interaction between novelty and memory processes.

V-035

Binge-like ethanol induced anxiety- like behavior, cell degeneration and possible amelioration of Omega-3 (ω-3) fatty acids in adolescent rats.

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Alcohol consumption contributes to ~5% of the global disease burden, with adolescents being particularly vulnerable. High alcohol exposure induces anxiety-like behavior and neuronal death, potentially through neuroinflammation and oxidative stress–induced apoptosis. Omega-3 (ω-3) fatty acids may counteract these effects through their anti-inflammatory and antioxidant properties. However, little is known about the long-term effects of ethanol (EtOH) exposure and the potential protective ω-3 effects in adolescents. Here, we evaluated the impact of EtOH exposure and the neuroprotective role of ω-3 on anxiety-like behavior and pyknotic cell number, in adolescent rats. Animals received 2 or 0 g/kg of EtOH (ig) on postnatal days (PDs) 28, 30, and 32 and, fifteen min later, they were administered with ω-3 (720 or 0 mg/kg, ig). On PD 34, animals were tested in the elevated plus maze for 5 min and were sacrificed for brain tissue collection. Pyknotic cells were stained with toluidine blue and quantified in the central amygdala (CeA). EtOH-treated animals showed an anxiogenic profile, spending less time in open arms; a profile that was improved by ω-3 (EtOH+ω-3 animals increase the time in open arms). EtOH significantly increased the pyknotic cell number in the CeA. These findings highlight ethanol´s long-term neurotoxic effects on cell degeneration and anxiety and provide evidence of possible amelioration of ω-3 in adolescents.

V-036

Effects of Gestational Enriched Environment on Maternal Behavior and Neurobiological Substrates in Two Animal Models of Postpartum Depression and Anxiety

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Gestation and the postpartum period involve major behavioral, physiological and molecular changes in mothers. The peripartum stage is also a period of high vulnerability to mood disorders such as postpartum depression, which often co-occurs with anxiety. This study evaluated whether a gestational enriched environment (EE) could improve maternal behavior, reduce anxiety and modulate neuronal activity in involved brain areas, using two postpartum depression–anxiety models: maternal separation (MS) and intruder male exposure (IM). Female Wistar rats were housed in standard conditions (SC) or an EE during pregnancy. On postpartum day 1 (PD1) animals were assigned to control (NS), MS, or IM groups for both SC and EE. Maternal behavior was assessed through the pup retrieval test on PD3, anxiety-like behaviors in the elevated plus maze on PD22 and +ΔFosB cells were quantified in the basolateral amygdala (BLA) and hippocampus. Preliminary findings suggest that gestational EE enhanced maternal behavior, evidenced by shorter latency to retrieve the first pup and higher percentage of recovered offspring, and reduced anxiety-like behaviors. In CA2, EE was associated with fewer +ΔFosB cells; while in CA1 interaction effects revealed fewer +ΔFosB cells in the EE+NS group. No significant differences were found in BLA, DG or CA3. These findings suggest that gestational EE enhances maternal care, reduces anxiety-like behaviors and differentially modulates neuronal activity in the hippocampus.

V-038

Physiological and Neurobehavioral Effects of Infant Maltreatment Stress in Juvenile Rats

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Chronic stressful conditions such as adverse parental care during early stages of development affect an individual's health and the way they cope with stressful situations later. Consequently, the alteration of the capacity to cope with subsequent stressors heightens vulnerability to the development of psychopathologies. Early intervention strategies would be important to mitigating the progression towards psychopathological outcomes in adulthood. In this study, we take advantages of the scarcity-adversity model (SAM) from postnatal days (PND) 8 to 12 in rats to investigate the impact of adverse care conditions on the adrenal glands, stress response and behavior phenotype at juvenile age (PND 21-35). We found that SAM generates alteration in adrenal glands changing cortex-medulla ratio, thickness to cortex zones and cytoplasmic diameter. These changes are accompanied by greater corticosterone levels to acute stress. At the behavioral level, higher sucrose consumption, more unsupported exploratory behaviors and a passive response in the forced swim were observed in SAM juvenile offspring. These findings are groundbreaking, as they shed light on the previously understudied effects of infant maltreatment at a critical early age, providing valuable insights for the development of early interventions that can help alleviate long-term consequences.

V-039

Compensatory responses to stressful situations in hippocampus-dependent behaviors: differential effects of traffic noise exposure and enriched environment in adolescent rats of both sexes

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Although adolescent central nervous system is highly vulnerable to different environmental stimuli, there has been limited research on the impact of urban traffic noise on hippocampal (HC)-dependent behaviors. Enriched environments (EE) have been shown to protect against HC damage. The aim of this study was to explore the behavioral effects of traffic noise in adolescent rats and EE housing as a neuroprotective tool.
Adolescent Wistar rats of both sexes were housed either in standard or EE cages and exposed to traffic noise (2h/day, for 5 days). Behavior was assessed using Open field, Inhibitory avoidance and Elevated Plus Maze tasks.
Traffic noise enhanced habituation memory (HM) and risk-assessment behaviors (RAB) in males, whereas in females HM was impaired. All these effects were prevented by EE housing. EE alone increased exploration and decreased anxiety-like behaviors (ALB) in both sexes, while RAB was reduced only in females. In noise-exposed females housed in EE an increase in RAB was found.
In conclusion, traffic noise affected HC-related behaviors in a sex-dependent manner: in males, it triggered compensatory responses (i.e., faster habituation to novel environments and RAB), whereas in females it impaired cognitive performance, indicating greater vulnerability. Although EE housing in females could increase vulnerability to environmental threats, it seemed that EE could be an effective neuroprotective tool, by preventing changes and promoting adaptive responses.

V-040

Optogenetic manipulation of contextual recognition memory by photo sensitive adenylyl cyclase

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Cyclic adenosine monophosphate (cAMP) is a pivotal second messenger in neuronal signaling and plays a central role in the molecular mechanisms underlying learning and memory. cAMP also regulates synaptic plasticity (i.e. long-term potentiation and long-term depression) via protein kinase A (PKA) activation. Our studies suggest that cAMP signaling also controls learning generalization, a cognitive capacity that allows the individual to apply specific knowledge learned in a certain circumstance in a similar one. In this study, we utilized the GAL4/UAS system in Drosophila to express a photoactivatable adenylyl cyclase specifically in distinct neuronal components, including mushroom bodies via OK107-GAL4. This approach enabled precise optogenetic manipulation of adenylyl cyclase activity in freely behaving flies. To test our hypothesis, double transgenic flies and parental controls were exposed to a context and then tested in the same context, in a similar or a different one. By measuring the distance traveled through these contexts, it was possible to assess the memory recall in each one, understanding normal behavior as recognition memory in the same context, generalization in the similar context, and no recalling in the different context. Contrary to our expectations, preliminary results show us that the groups stimulated by light have poor recognition memory, which could mean that the massive increase in cAMP has a detrimental effect on the PKA signaling and poor memory.

V-041

Slow Breathing as a Neuromodulation Tool to Induce Altered States of Consciousness: Project Design and Discussion

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Altered states of consciousness (ASC) are marked, temporary, reversible deviations in subjective experience or psychological functioning from usual waking consciousness, distinct from psychiatric disorders. Breathing is not only essential physiologically but also modulates brain oscillatory activity. Studies show that reducing breathing to ~6 breaths/min increases delta (1-4 Hz) and theta (4-8 Hz) power, especially in prefrontal and limbic regions, and enhances default mode network (DMN) connectivity. These changes are linked to introspection and ASC, suggesting slow breathing as a non-invasive way to modulate consciousness.

This study investigates how slow breathing modulates consciousness through EEG analyses: power spectral density (PSD), complexity metrics, and functional connectivity. A within-subject design will be used with 40 healthy adults (18-50 years). Each will undergo two conditions in randomized order, two weeks apart: (1) experimental, 5 min baseline then 15 min guided slow nasal breathing at 4 breaths/min; (2) control, 5 min baseline then 15 min spontaneous breathing. EEG (32 channels, Brain Vision) and respiratory belts will monitor neural and breathing activity. Subjective ASC will be assessed with the TAS, PCI, and 11D-ASC. This study will provide evidence on slow breathing as a neuromodulation tool to modulate brain activity and induce ASC.

V-042

Post-Exercise Modulation of Cortical Excitability: Evidence from Beta and Gamma Bands in Resting-State EEG

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Exercise modulates the dynamics of brain waves, although the magnitude of these changes depends on context and individual characteristics. This study analyzed the global brain-wide modifications induced by a high intensity interval training (HIIT) protocol in two healthy subjects. The protocol consisted of 10 intervals of 40”/20” at 90% of maximal load, with active recovery at 50%. EEG was recorded using 30 channels in resting state with eyes closed at three time points: before exercise (PRE), immediately after (P1), and five minutes post-exercise (P2). Non-parametric tests were used to assess changes in power spectral density within the beta and gamma bands, with preprocessing performed using EEGLab and Matlab. Results showed a marked increase in beta and gamma power after exercise, with a differentiated temporal pattern. In P1, both participants exhibited pronounced increases in gamma, suggesting a transient cortical hyperactivation state associated with neuronal excitability and cortical integration. During P2, a persistence and even amplification of gamma power was observed compared to rest. These findings, consistent with existing literature, indicate that HIIT enhances fast-wave activity (beta/gamma), potentially reflecting neurophysiological adaptations related to excitability, attention, and cognitive processing in the post-exercise state.

V-043

Contextual Information Shapes Object Recognition Memory

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Most memories are not formed in isolation, but are acquired during the recall of previous memories that help contextualize new incoming information. The hippocampus integrates new and old memories and, together with the medial prefrontal cortex, plays a crucial role in item recognition and spatial memory processing. However, the neurobiological mechanisms underlying item-context associations remain poorly understood. In this work, we investigated the influence of contextual information on object memory retention in male and female Wistar rats. Using a 90-s training protocol in the novel object recognition task, we found that animals trained with two identical novel objects in a familiar, featureless context formed a short-term object recognition memory (ORM) that decayed within hours. In contrast, training in a familiar, feature-rich context induced the formation of a long-term, context-dependent ORM that was impaired by optogenetic inhibition of the ventral hippocampal-prelimbic pathway during training. Long-term ORM was not observed when the habituation sessions occurred in a featureless context or after chemogenetic inhibition of hippocampal-prelimbic projections shortly after habituation in the featured context. Our findings suggest that environmental information can modulate object recognition memory formation and that ventral hippocampal-prelimbic interaction is crucial in this process.

V-044

Could running modulate memory? Potential effect of endogenous opioids in memory consolidation

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State-dependent memories are more easily retrieved when the physiological or psychological state at the time of retrieval matches the state during encoding. Endogenous opioids—neuromodulators naturally produced by the body—play crucial roles in pain modulation, reward, and stress responses. Physical exercise is known to elevate levels of endogenous opioids, among other physiological changes, and has been shown to influence memory processes.

Previous studies from our laboratory have demonstrated that moderate to intense exercise immediately following training on an aversive task leads to impaired performance when tested 48 hours later. We hypothesize that this impairment may be due to a state-dependent effect induced by the physiological changes following exercise, particularly the release of endogenous opioids.

This study aims to investigate how voluntary exercise affects the consolidation and retrieval of aversive memories in CF1 mice, with a focus on the role of endogenous opioids. To this end, we administered naloxone, an opioid receptor antagonist, to pharmacologically block the effects of exercise-induced endogenous opioid release that may be influencing memory performance in mice.

The findings may provide valuable insights for the development of behavioral interventions targeting pathological memories and enhance our understanding of the underlying neurobiological processes.

V-045

Representational similarity of ambiguous words: an EEG and pupilometry experimental design

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The ability to flexibly access word meanings according to context is a core feature of semantic processing. Ambiguous words (e.g., note) provide a valuable window into how context shapes lexical access, as they require the selection of one meaning among competing alternatives. This project examines the neural dynamics of accessing dominant versus subordinate meanings of ambiguous words. Participants will first read short passages establishing a semantic context (e.g., music), followed by ambiguous words that are either congruent or not with the preceding context. Electroencephalography (EEG) will be recorded and analyzed, focusing on theta (4–8 Hz, associated with memory-related plasticity) and alpha activity (8–12 Hz, linked to inhibition). Representational Similarity Analysis (RSA) will be applied to frequency-domain neural patterns to compare contextually matched and mismatched conditions, testing whether congruent words produce more homogeneous similarity structures. In parallel, pupillometry will provide a continuous index of cognitive effort, as pupil dilation reflects the attentional and control demands required to resolve ambiguity. The study will test whether accessing subordinate meanings requires greater cognitive effort than dominant meanings, and if supportive context facilitates semantic processing. This multimodal approach aims to dissect the representational basis of meaning and the control processes that enable context-driven resolution of lexical ambiguity.

V-046

Electrophisiological Dynamics in an Aversive Conditioning Paradigm in Humans

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Recognition of threats is crucial for survival, but it can also contribute to the development and persistence of pathological fear, as observed in anxiety disorders and post-traumatic stress disorder (PTSD). Aversive conditioning paradigms, particularly Pavlovian fear conditioning, have been instrumental in elucidating the mechanisms of fear acquisition, reconsolidation, and extinction. Evidence suggests that reactivation of fear memories can render them labile and susceptible to modification, offering potential therapeutic avenues for modulating maladaptive responses.

In this study, we employed a novel aversive conditioning paradigm in which participants were exposed to air puffs as aversive stimuli, paired with specific geometric shapes. Subjects developed conditioned blink responses to these shapes. After 24 hours, extinction was assessed by presenting the shapes without the aversive stimulus, during which we observed short-lived sustained habituation and extinction. Our central hypothesis is that individuals with anxiety disorders will exhibit enhanced habituation compared to neurotypical controls, as well as be characterized by a faster rate of habituation, stronger conditioned responses, and prolonged extinction trajectories.

By combining behavioral measures with established frameworks of fear learning and extinction, this work aims to advance our understanding of how anxiety affects the formation of aversive memories, utilizing non-invasive methods.

V-047

Effect of HAT inhibitor Garcinol on memory persistence in mice

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Garcinol is a naturally occurring compound that functions as a HAT inhibitor. In previous research, the administration of Garcinol has been observed to result in impaired memory reconsolidation in a rat model of addiction. Prior studies conducted within our lab show that histone acetylation constitutes a hallmark of long-lasting memories, yet its role is not indispensable for less persistent memories. In light of these findings, the present study was designed to ascertain the impact of systemic Garcinol administration on memory persistence in mice. We employed the novel object recognition task to determine the impact of the treatment both on long-term memory, measured 24 hours after training, and persistence, measured 7 days after training. After 7 days, garcinol showed an effect as mice explored equally both familiar and novel objects. Furthermore, when tested 24hs after training treatment did not affect long-term memory, as the two groups exhibited similar discrimination indices for both objects. However, animals that had been administered Garcinol exhibited a marked reduction in the amount of exploring time in comparison to the control group. Subsequent experiments using the Fear Conditioning task are currently being conducted to ascertain whether this impairing effect is contingent upon the task or memory type.

V-048

The effects of sleep deprivation on political polarization.

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This study investigates the relationship between sleep deprivation and political polarization through two experimental protocols. We conducted an 8-hour total sleep deprivation (TSD) experiment (n=20) and a 4-hour partial sleep deprivation (PSD) experiment (n=44) to examine their effects on ideological extremism and affective polarization. Results revealed a significant increase in ideological extremism following TSD in between-subjects analysis (Cohen's $f^2 = 0.48$), while no significant effects were observed with the PSD protocol compared to the regular sleep (RS) condition. Importantly, moderation analyses revealed that political orientation significantly influenced susceptibility to sleep deprivation effects, with different patterns emerging in between-subjects versus within-subjects analyses. Left-wing participants showed greater vulnerability to increased ideological extremism under sleep deprivation in between-subjects comparisons, while right-leaning individuals demonstrated this effect in within-subjects analyses. No significant effects were observed on affective polarization in either protocol. These findings suggest that sleep deprivation may contribute to political polarization by increasing ideological extremism, particularly when considering political orientation as a moderating factor. The study provides novel insights into the complex relationship between sleep and political cognition in distinct sociopolitical contexts.

V-049

Divergent neural dynamics in the basolateral amygdala-medial prefrontal cortex circuit revealed through automatic clustering of freezing events

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Freezing is a hallmark of fear memory retrieval, yet it does not represent a uniform physiological state. We have shown that rats display two distinct forms of freezing, characterized by different oscillatory profiles in the basolateral amygdala (BLA) and medial prefrontal cortex (mPFC). Freezing episodes enriched in 4-Hz activity were most common during early retrieval, showing increased power in this band with suppression of theta, consistent with fear expression. By contrast, theta-enriched freezing emerged more often as extinction progressed, with stronger theta and reduced 4-Hz power, suggesting a distinct and possibly transitional state linked to fear inhibition. Although indistinguishable in terms of immobility, both states showed robust and opposing signatures in oscillatory activity, neuronal firing, and interregional interactions across BLA and mPFC, indicating that a similar outward behavior can arise from distinct internal dynamics. In the present study we extend these findings by exploring whether such subtypes can be identified without relying on arbitrary cutoffs. We applied dimensionality reduction and clustering analysis to behavioral and neural variables to test whether freezing can be decomposed into functionally meaningful sub-states in an unsupervised manner. This approach provides a new framework to examine how fear memories are retrieved and extinguished through the study of divergent freezing behaviors reflecting distinct internal states.

V-050

Pre-stress measurement and its relevance to PTSD animal models

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In the behavioral profiling of vulnerability and resilience in animal models of Post-Traumatic Stress Disorder (PTSD), the focus has been on the effects that the stress protocol generates in the exposed animals. Although this post-stress classification is more sensitive than a traditional comparison, it is insufficient to account for intra-subject variation. This is possible if a pre-stress measurement is available, which allows a more accurate characterization of the effects of the stress protocol and an investigation of susceptibility.

To evaluate this, we conducted experiments with adult male C57BL6 mice in which the Single Prolonged Stress (SPS) protocol was preceded by an assessment of anxiety-like behavior with the open field test (OFT). One week after the SPS, we conducted a test in the Elevated Plus Maze and a second OFT. After this, contextual fear conditioning was performed on a subset of animals to assess its retrieval, generalization and extinction. The pre-stress measurement prevents us from attributing post-stress “effects” to the independent variable when these were better explained by their basal anxiety-like behavior. At the same time, it shows that a considerable proportion of the most affected SPS-mice were susceptible, suggesting that it is a risk factor that should be considered as an outcome by itself and a relevant variable to be controlled in PTSD models.

V-051

Hippocampal–Prefrontal Oscillatory Dynamics in Memory Interference: Impact of 5-HT2A Receptor Blockade

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Discriminating between overlapping experiences is essential for effective retrieval of episodic memories. In rodents, this process can be examined with the Object-in-Context (OIC) task, where contextual cues guide the selection between competing memories. The prefrontal cortex (PFC) plays a pivotal role in this selection, and serotonergic signaling—particularly through 5-HT2A receptors—has been implicated in controlling memory interference. Pharmacological blockade of 5-HT2A receptors in the PFC impairs interference resolution without affecting attentional processes. The hippocampus (HIP), via its projections to the PFC, may convey contextual information and modulate mPFC activity through theta oscillations. Local field potential (LFP) recordings revealed enhanced theta power in the ventral HIP and PFC during object exploration, with increased HIP–PFC coherence when animals explored incongruent objects, correlating with behavioral performance. To assess the impact of 5-HT2A receptor blockade on HIP–PFC communication, Wistar rats were implanted with tetrodes and cannulas in the PFC and electrodes in the HIP. Preliminary data indicate that local 5-HT2A antagonist infusion in PFC decreases HIP–PFC coherence during incongruent object exploration, suggesting a possible link to the animals' impaired task performance.

V-052

Effect of acute physical activity on object discrimination memory

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Among the many features of memory, episodic memory includes the ability to separate initially similar experiences to avoid overlap at retrieval. This process, pattern separation, would enable differentiation of locations or objects in memory. Studies in humans and rodents using analogous tasks have linked behavioral performance to molecular mechanisms, suggesting its translational relevance. Pattern separation is impaired with aging, hippocampal damage, and Alzheimer's disease, highlighting interest in strategies to enhance it. To examine the impact of acute and chronic physical activity on memory consolidation, 35 young adults (18–40 yrs) were recruited, including 24 sedentary individuals and 11 chronic athletes. Memory performance was assessed with version 7rep of the Memory Differentiation Task (MDT), designed to test discrimination of similar vs. different objects. Following the acquisition phase, 12 sedentary participants completed 25 min. of moderate exercise, while chronic athletes and remaining sedentary individuals watched a cycling race video. After 24 hs, all participants were retested. 2-way ANOVA revealed a significant effect of object type (similar vs. different), but no effect of physical activity condition (acute, chronic, control). Thus, previously reported acute exercise effects were not replicated, though results support MDT validity. High-density EEG was conducted to explore underlying neurophysiological mechanisms and their modulation by physical activity

V-053

Invalidating Family environment and emotional semantic processing

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Invalidating childhood environments, where caregivers dismiss or reject emotional experiences, are strongly linked to emotional dysregulation and long-term psychopathology. Neurophysiological evidence suggests that adverse family climates may manifest in EEG markers of affective integration, such as parietal positivities reflecting sustained emotional processing. This preliminary study examined whether perceived invalidation, measured through self-reports of family environment, modulates the integration of semantic and affective information. Participants (N = 9) completed a word–face priming task in which emotional words preceded congruent or incongruent facial expressions. EEG was recorded with a 30-electrode system, and centro-parietal amplitudes in the 300–500 ms window were analyzed. Correlational analyses revealed that higher maternal invalidation was associated with reduced positivity during positive congruent trials ($r = –.723$, $p = .028$), with overall negative perceptions of maternal environment showing a similar association ($r = –.734$, $p = .024$). These findings suggest that invalidating family climates may alter the neural dynamics of emotional congruence, particularly in positive contexts, contributing to difficulties in emotion regulation and interpersonal functioning. By linking self-reported family environment with neurophysiological indices, this study underscores the importance of early relational experiences in shaping affective processing.

V-054

Keeping Kids on Their Toes: Continuous Dynamic Adjustment Boosts Executive Function Training

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For the past 15 years, we have been using the web-based cognitive assessment and training platform Mate Marote with children aged 4 to 8. In a previous study, we evaluated a model of dynamic difficulty progression that proved effective in enhancing children's attention. In the present study, we compared that model with a revised version designed to overcome its previous limitations. Both training approaches adapt rapidly to the player's initial level. However, while the original model slows its difficulty progression once a balance point is reached, the revised model continuously adjusts task difficulty through a more precise algorithm. We hypothesized that: (1) executive function performance would improve from pretest to posttest, and (2) gains would be greater in the group trained with the revised model. Results show that both groups significantly improved their attention. However, only the group trained with the revised model exhibited additional improvements in logical reasoning, cognitive flexibility, and working memory. These findings suggest that sustained, individualized dynamic adjustment of task difficulty not only supports motivation but also enhances multiple dimensions of executive functioning, promoting more effective and adaptive training.

V-055

Study of anxiety-like behavioral performance in subject with overgeneralization of learning

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Learning and memory generalization is a cognitive function that allows an individual to apply information learned in a context, to a similar but not identical one. Generalization is reduced in intellectual disability and overgeneralization is frequently associated with psychiatric disorders such as post-traumatic stress disorder, phobias and other anxiety disorders. However, there is no direct evidence to support causal relationships in this association. In Drosophila anxiety-like behavior can be studied by “wall following” (WAFO) behavior in an open field arena. Recently we described that some genetic manipulations of cAMP signaling pathways promote overgeneralization of learning. Therefore, to study its potential to consequently promote anxiety-like behavior we used genotypes with phosphodiesterase-dependent overgeneralization, previously identified in our laboratory, and control lines. In contrast to our expectations, preliminary results showed that these genotypes exhibit centrophilia when compared to controls. However, these results are consistent with previous studies where stressed flies showed centrophilia, which in this case was paradoxically resistant to diazepam. Additional study on this matter will be informative of the relation between overgeneralization and anxiety-like behavior as well as centrophilia and diazepam.

V-056

Mechanisms of Brainstem Plasticity in Motor Learning

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Motor skills are acquired through repeated training; though learning is slow, established motor memories persist long-term without practice. This process includes an early phase of rapid improvement and a later plateau, engaging distinct circuits and mechanisms. Motor memory formation involves the motor cortex, basal ganglia, and cerebellum. In contrast, brainstem centers have traditionally been regarded as rigid executors of stereotyped motor commands. We argue instead that brainstem plasticity is key for adapting learned skills to new contexts, focusing on the mesencephalic locomotor region (MLR), whose glutamatergic neurons are targeted by the motor cortex, basal ganglia, and cerebellum. Our findings show that motor training induces expression and activation of canonical signaling pathways linked to learning and memory (BDNF/TrkB, ERK/pERK) and that interfering either with de novo protein synthesis or with these pathways impairs motor memory consolidation. Thus, we hypothesize that these molecular changes promote electrophysiological plasticity in MLR circuits, enabling fine-tuning of motor commands in response to environmental demands. We are currently characterizing in vivo electrophysiological properties of MLR neurons across motor training and establishing correlates to validate this model. Together, these findings redefine the MLR as a dynamic contributor to motor learning, with implications for understanding how the brain integrates experience to refine movement.

V-057

Aversive Memory Expression and Extinction in a Virtual Reality Environment

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Memory is an essential component of our existence, allowing us to store and retrieve past experiences to optimize behavior and adapt to our environment. Maladaptive memories, which sustain dysfunctional emotional patterns, can negatively impact an individual's well-being. The formation of these emotional memories involves the interaction of various cerebral structures within the limbic circuit. In this project, we utilize 3-4 month-old male C57BL/6 mice in a head-fixed system where their displacement on a rotating cylinder is translated into a virtual reality (VR) visual input. This VR environment, generated with the Godot engine, simulates an infinite hallway with a 2m corridor and a 30cm reward zone where water is delivered. The research protocol is divided into three phases. The first is Appetitive Training, where mice learn to associate a tone (apCS+) with a water reward to maximize their movement on the track. This is followed by Aversive Training, a two-day process where the animals are conditioned to a tone (avCS+) which is paired with an electrical shock. Their conditioned fear response, or freezing, is recorded and quantified. The final phase is Extinction, where the mice are repeatedly exposed to the aversive tone without the shock. The reduction in freezing behavior, measured as a decrease in roller speed, provides a quantifiable metric of the extinction process. This will allow recording of different components of the limbic system and study the extinction process

V-058

Social Support During Pregnancy Mitigates Sex-Specific Downregulation of Neuronal Activity and Synaptic Plasticity Genes After Prenatal Stress

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Prenatal maternal stress (PMS) is a major public health concern with long-lasting detrimental effects on maternal and offspring health. Supportive social relationships can buffer these effects, yet the underlying molecular mechanisms remain poorly understood. We previously showed that prosocial behaviors from a conspecific toward PMS-exposed females improve offspring recognition memory and social interaction, particularly in females. Here, pregnant mice were exposed to unpredictable stressors and then housed (ESA) or not housed (ES) with a familiar non-pregnant female until delivery. A non-stressed, socially housed group (CTA) served as control. PMS induced fragmented, low-quality maternal care (ES vs. CTA), fully restored in ESA. Performance in the 4-hole board test showed reduced exploration and impaired spatial memory in ES females compared to CTA and ESA females. ES dams exhibited lower Npas4 expression in the amygdala and reduced Comt1 in the prefrontal cortex and amygdala compared with CTA and ESA. Female offspring of ES dams showed decreased expression of Bdnf, Trkb, Npas4, and synapsin relative to CTA, whereas ESA offspring displayed full recovery of these gene expression.

These results confirm previously observed memory deficits and suggest their association with sex-specific downregulation of genes involved in neuronal activity, synaptic plasticity, and memory formation, and that social support during pregnancy can reverse both behavioral and molecular alterations

V-059

The Audience Effect on Intersexual Aggression in Cichlasoma dimerus: Design and Validation of a Virtual Stimulus

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Aggressive behaviors are conserved across vertebrates and can be modulated by the social environment. Since social behavior relies on environmental perception and signaling, and the presence of an observer may influence interactions, social behavior can be addressed through the Audience Effect. In the Neotropical cichlid Cichlasoma dimerus, escalated aggression is observed during intersexual dyadic interactions. To study whether intersexual agonistic encounters are modulated by the Audience Effect, we first tested whether a virtual stimulus can serve as an audience by exposing focal fish to a screen displaying a conspecific. Two experimental setups were used: with the opposite side of the tank empty, or with a second screen showing an empty aquarium. We quantified agonistic and social behaviors to analyze temporal dynamics. In the second setup results showed differences in lateral swimming ($p=0.010$). Moreover, an alternative two-screen paradigm was used: first both screens displayed empty tanks, and after 30 minutes a conspecific was displayed on one screen. Focal fish increased interactions with the conspecific screen (touches $p=0.0476$; lateral swimming $p=0.0094$) but not with the empty one, suggesting a possible recognition of the virtual stimulus. These validations will enable using virtual stimuli as audiences as a first step to study how social context shapes intersexual aggression and which are the brain activation patterns involved in this modulation

V-060

Hippocampal circuits underlying observational fear learning in mice

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Social learning is a fundamental process through which animals acquire information by observing the experiences of conspecifics. Observational fear learning (OFL) in rodents provides a powerful model to study the mechanisms underlying social memory formation, as observer mice learn to associate a neutral stimulus with the distress of a demonstrator. This paradigm is particularly relevant for understanding disorders involving altered social transmission of information and empathy, such as autism. Previous research has highlighted the oxytocin (OXT) system as a key modulator of social behaviors, yet the specific neural circuits through which OXT contributes to observational fear memories remain poorly understood. We aim to investigate the role of OXT in the formation of observational fear memories, with a specific focus on the hippocampal subregions and the paraventricular nucleus (PVN) as potential key nodes in this process. As a first step, we assessed neuronal activation in dorsal CA1 and CA2 by measuring cFOS-positive cells in observer mice exposed to an OFL training session (TR-Obs), compared to observers whose demonstrators did not receive shocks (noTR-Obs), and to naïve controls. Our results suggest that neuronal activation varies depending on the social experience and differs between sexes, pointing to a potential interaction between OXT signaling, hippocampal circuits, and sex in observational fear learning.

V-061

Effect of paternal behavior on anxiety, spatial memory, and pup care in male rats

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Parental behavior has been extensively studied in females, however, in male rats the effects of fatherhood on cognition and emotional behavior remain poorly understood. This study aimed to evaluate memory, anxiety, and paternal behavior in male Wistar rats exposed to varying numbers of litters.

Male rats were divided into three groups: G1 (one litter), G2 (two litters), and G3 (three litters). Paternal behaviors were analyzed in the presence and absence of the female for 10 minutes on postnatal days 1, 3, 5, 8, 10, 12, 15, 17, and 19. Anxiety was assessed using the Elevated Plus Maze (EPM), and short-term memory (STM) and long-term memory (LTM) were evaluated using the Barnes maze. In the Bm, training latency decreased by up to 30% in males that cared for litters, showing improved LTM (latency: 8±1 s) compared to non-caregivers (30±5 s). G2 showed a significant increase in time spent in the closed arms of the EPM (290±5 s), indicating elevated anxiety. Likewise, latency to initiate paternal behavior increased in G3 on PND 5 and PND 10 compared to G1, and huddling (pup covering) behavior also increased in G3 regardless of female presence.

In conclusion, prolonged exposure to litter promotes the development of active paternal behaviors, associated with increased anxiety and enhanced cognitive performance. These findings suggest that paternity in males not only modulates offspring-directed behavior but also induces adaptive changes in emotional and cognitive processing.

V-062

Pubertal hormone exposure mitigates anxiety-like behavior and modulates HPA axis activity after adolescent isolation in male and female rats

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Adolescence is a critical period of brain reorganization, highly sensitive to sex hormones, which exert sex-specific effects and may contribute to the greater prevalence of anxiety and depression in adolescent females. This study evaluated whether exposure to gonadal steroids during the perinatal or pubertal period influences vulnerability to anxiety- and depression-like behaviors following social isolation (PND 21–54) in male and female rats. Two groups were compared: PERI (gonadectomy at PND 23) and PUB (gonadectomy at PND 45). Behavior was assessed through the Elevated Plus Maze, Open Field, Forced Swim, and Sucrose Preference tests, and standardized Z-scores were calculated. ACTH and corticosterone levels were measured, and chronic neuronal activation in the PVN was evaluated via ΔFosB immunolabeling. Isolation delayed puberty in both sexes and reduced body weight in males. Pubertal hormone exposure reduced anxiety-like behaviors and increased risk-taking, with no sex differences. PERI animals had higher anxiety Z-scores than PUB, especially PERI+IR. No differences were found in depression/emotionality Z-scores. PUB animals showed higher ACTH levels, and greater PVN activation was observed in females, particularly in the PUB group. These findings suggest that pubertal hormones buffer the effects of isolation on anxiety, possibly through differential modulation of the HPA axis and neuron activity in the PVN, with a more reactive neuroendocrine profile in females.

V-063

Reading emotions in others: a novel role for midbrain dopaminergic neurons.

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Socio-affective behavior refers to the set of behavioral responses elicited by the emotional state of a conspecific during social interaction. This capacity is essential for adaptive functions critical to mammalian survival, including territorial defense, aggression, parental care, and hierarchical organization. Despite growing research, the neurobiological basis of these behaviors remain poorly understood. Within this context, the ventral tegmental area (VTA) of the midbrain emerges as a relevant region, given its well-established role in encoding the valence of emotionally salient stimuli. In this work, we employed a chemogenetic approach to investigate the contribution of VTA dopaminergic neurons to multiple aspects of socio-affective behavior, including the discrimination of emotional states with different valences expressed by conspecifics, social preference, and social recognition. Elucidating the neurobiological basis of socio-affective behavior is crucial not only for understanding normal social interactions but also for uncovering the mechanisms underlying their disruption in neuropsychiatric disorders such as autism spectrum disorder (ASD) and schizophrenia.

V-064

Eye-Tracking as a Diagnostic Tool in Alzheimer's Disease, Mild Cognitive Impairment, and Related Dementias

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Alzheimer's disease (AD) pathology begins years before symptoms emerge, making early detection essential. Eye-tracking offers a rapid, non-invasive means for identifying early cognitive decline through oculomotor disturbances. Our analysis of 71 studies investigated the utility of oculomotor tasks in distinguishing Mild Cognitive Impairment (MCI) and AD from healthy controls. Antisaccade tasks consistently demonstrated high utility, revealing impaired accuracy, longer latencies, and reduced gain in AD and MCI patients. In contrast, prosaccade tasks yielded mixed results, while non-saccadic paradigms showed diminished exploratory behavior in AD but inconsistent patterns in MCI. A critical finding was the overwhelming reliance on clinical rather than biological diagnostic criteria across studies (85%), significantly hindering clinical translation. Widespread methodological inconsistencies and small sample sizes (<50 patients in 72% of studies) further limit the generalizability of current evidence. Therefore, while antisaccade tasks are a promising screening tool, future research must adopt biologically defined cohorts and standardized protocols to validate their clinical potential.

V-065

Characterization of the functionality of astrocytic connexin 43 hemichannels in a PTSD model.

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Post-traumatic stress disorder (PTSD) is a psychiatric illness that develops after a person is exposed to an extremely stressful event. It can be understood as maladaptive responses due to deregulation of the fear and anxiety brain circuits, in which the amygdala and hippocampus play a fundamental role. Astrocytes are known to have an active role in synaptic function, with the release of gliotransmitters being a critical aspect of the functioning of tripartite synapses. Recent experimental studies demonstrated the role played by astrocytic Cx43 hemichannels in the release of gliotransmitters and their participation in emotional processing. The aim of this research was to evaluate the functionality of astrocytic Cx43 hemichannels in a model of post-traumatic stress, known as single prolonged stress (SPS). In this work, we applied SPS to adult mice and evaluated their anxious behavior by performing behavioral tests (elevated plus maze and open field). Then, we measured hemichannel activity through an ethidium bromide uptake assay in ex vivo tissue. For staining validation, we assessed bromide uptake in the presence of a synthetic peptide, which selectively blocks Cx43 hemichannels. GFAP immunohistochemistry was used to identify astroglial cells. Changes in Cx43 hemichannel activity in astrocytes might represent a novel mechanism in PTSD and a new target for pharmacotherapy.

V-066

Developmental Fluoride exposure: assessment of sociability, obsessive-compulsive behavior, and blood biochemistry in female rat offspring

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Drinking water is the main route of fluoride (F) exposure. In the Chaco-Pampean region, rural and peri-urban aquifers often have elevated F, posing health risks. F crosses the placenta and is present in maternal milk; developmental exposure has been linked to neurobehavioral alterations in rat offspring, though mechanisms remain unclear.
We evaluated behavior and blood biochemical parameters in 45-day-old female offspring exposed to 10 mg/L F during gestation and lactation. Sociability was assessed using the Three-Chamber Social Test and Reciprocal Social Interaction, and obsessive-compulsive–like behavior with the Marble Burying Test. Serum levels of glucose, urea, creatinine, total cholesterol, triglycerides, and the activity of alkaline phosphatase, lactate dehydrogenase, creatine kinase, and the transaminases GOT and GPT were determined using enzymatic and UV-kinetic methods.

F exposure induced significant metabolic alterations, including increased glucose and cholesterol and reduced enzymatic biomarkers. Trends toward decreased sociability and increased obsessive-compulsive–like behavior were observed but were not statistically significant.

These results highlight the relevance of metabolic disruptions in developmental F toxicity and the need for further research on neurobehavioral effects. Raising awareness of F toxicity during pre- and postnatal development is essential for promoting safe drinking water, particularly during pregnancy and lactation.

V-067

Characterization of the Neuronal Differentiation Process During Corticogenesis in the Plains Vizcacha

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During mammalian embryonic brain development, the corticogenesis involves the formation of transient germinal layers with neural progenitors whose proliferation, together with interneurons, makes up the neocortex. The protein NeuN regulates alternative splicing of genes related to development and excitability, showing both nuclear and cytoplasmic localization. The aim was to characterize neuronal differentiation in the plains vizcacha (Lagostomus maximus), a gyrencephalic rodent native to Argentina, with a ~155-day gestation. The cortex was analyzed from 50 embryonic days (ed) to 2 postnatal days (pnd) using Nissl staining and NeuN immunohistochemistry (N=60). At 80–85 ed, NeuN neurons were detected in transient germinal layers and in layers IV–VI. From 95–99 ed ahead, nuclear NeuN was observed in the six definitive cortical layers. From 100–105 ed, pyramidal neurons in layer III showed nuclear and cytoplasmic NeuN staining, pyramidal neurons in layer V showed weak staining, while a strong staining was observed in granular neurons of layers II, IV, and VI. By 110–115 ed, neurons of layer V showed low expression of NeuN in both nucleus and cytoplasm, a feature persisted at 2 pnd. These results suggest that neuronal maturation begins in the transient germinal layers around mid-gestation and progresses towards the cortex following an inside-out pattern. Variability in NeuN intensity and localization through development may reflect distinct functional and morphological states.

V-068

Bisphenol A (BPA) time-dependent effects during neural development in Xenopus laevis

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Bisphenol A (BPA) is widely used in industry to produce polycarbonate plastics associated with everyday consumption. It has been suggested that it acts as an endocrine disruptor and is associated with developmental disorders. The amphibian Xenopus laevis is a widely used model for studying vertebrate development. Unlike the most used amniote models, its external development facilitates the study of vertebrate embryogenesis and its alterations by chemical and environmental agents from the outset. Studies conducted in this model showed that BPA causes malformations in tadpoles and is capable of inhibiting γ-secretase. However, there are no previous studies investigating BPA exposure during the early stages of development. We previously found that the treatment with 20 μM BPA of X. laevis embryos from the onset of gastrulation to the onset of neurulation delays neural plate folding and increases the density of differentiated neurons at neurula stage. Now, we performed two treatment windows: i) from the start to the end of gastrulation and ii) during primary neurogenesis. We found that the genes involved in the neurogenesis cascade, like zic2, xmyt1 and n-tubulin; in the neural border/neural crest and those involved in the Notch pathway, like hes4 and dll1, change their expression differently between the windows treatments which strongly suggest that the BPA affects in a different way the developmental steps.

V-069

Effects gamma audiovisual stimulation on the proliferation of neural progenitors in the aging hippocampus

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The generation of dentate granule cells (GCs) in the adult hippocampus declines with age due to reduced progenitor proliferation, slower maturation, and decreased survival of newborn neurons. We previously showed that in 8-month-old mice, 40 Hz audiovisual stimulation (AuViS), a non-invasive intervention known to reduce amyloid-β and improve memory in Alzheimer's models, enhances neurogenesis and accelerates neuronal maturation. In 12-month-old mice (12M), where neurogenesis is further compromised, AuViS still promotes neuronal differentiation at the expense of astrogenesis. Here, we examined progenitor cell proliferation in the dentate gyrus of 12M mice exposed to chronic AuViS. Animals were divided into three groups: (1) control, (2) 4 weeks of AuViS followed by 3 weeks without stimulus, and (3) continuous AuViS for 7 weeks. Ki67 immunostaining revealed that prolonged AuViS reduced the number of proliferating cells. This decline may arise from a depletion of the neural stem cell pool after sustained stimulation. These findings highlight both the potential and the complexity of AuViS as a modulator of neurogenesis in aging. They also raise the possibility that therapeutic protocols might require spacing of stimulation sessions to preserve the progenitor pool and sustain long-term efficacy. Ongoing and future experiments will further clarify the underlying mechanisms and optimize stimulation strategies.

V-070

Developmental exposure to glufosinate-ammonium herbicide induces neurotoxicity in mammals.

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The use of herbicides is a growing practice worldwide, especially for weed control in genetically modified crops. In Argentina, herbicides such as glyphosate and glufosinate-ammonium (GLA) -an irreversible glutamine synthase inhibitor- are widely used. Following the emergence of crop resistance to other herbicides such as glyphosate, there has been a growing trend toward the use of glufosinate-ammonium. Different studies have shown toxic effects on developmental nervous system in humans and animals after GLA exposure.

Our study was based on evaluating developmental toxicity in Wistar rats exposed to GLA formulation at doses equivalent to 20, 40 and 60 mg/kg of GLA from postnatal day 7 to 27, and subjected to behavioral tests to evaluate motor activity and cognitive functioning. Weight evolution was also carried out and the preliminary result showed a delay in the growth and development of neonates exposed to the highest doses. Moreover, studies in hippocampal pyramidal neurons cultured for 24 and 48 hours were performed. Immunocytochemical studies revealed that undifferentiated neurons exposed to GLA showed alterations on their development since shorter and less complex dendritic tree was observed compared to controls. Together, our observations suggest that GLA exposure induce neurotoxicity signs during development.

V-071

Temporal Specification and Spatial Distribution of Astrocyte Subtypes in the Spinal Cord

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Astrocytes play key roles in the maintenance and regulation of neurological functions and their impairment contributes to several pathologies. Although neuronal subtype specification has been extensively studied, how astrocyte diversity emerges during development remains poorly understood. Here we show that distinct dorso-ventral progenitor pools of the mouse embryonic spinal cord (identified by Nkx6.1, Dbx1, Pax3/6/7 and Ascl1) give rise to astrocytes that precisely occupy distinct spinal regions. Despite their domain of origin, each group exhibits heterogeneity in distribution and morphology, comprising protoplasmic gray matter (GM), and fibrous and subpial white matter (WM) astrocytes. To assess whether GM and WM cells derive from common progenitor cells we performed lineage tracing using GlastCreER mice combined with Tomato or GFP conditional reporters as well as the mosaic analysis with double markers system. Clonal fate mapping revealed that astrocytes are often found in pairs of the same subtype, sharing morphology, molecular marker expression and spatial location. Furthermore, our experiments suggest that daughter cells arise from symmetrical divisions after migration near their final settling position. In conclusion, our findings reveal that, in addition to dorso-ventral patterning, astrocyte diversity is determined by distinct progenitors in each embryonic domain selectively producing GM and WM astrocytes.

V-072

Changes in the asymmetric organisation of structural connectivity in the cerebral cortex and subcortex with age

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In nature, asymmetries are widely found in bilateral structures. Particularly in the brain, directional asymmetry (DA) has been of interest due to its association with functional lateralisation, although the asymmetry in the structural connectivity network has been scarcely explored. This study aims to investigate changes in the magnitude of DA in the structural connectivity of cortical and subcortical regions during adulthood. We analysed 151 3T T1 and DWI-DTI brain magnetic resonance images from healthy adult subjects (43.73 ± 16.25 years) residing in the Buenos Aires metropolitan area, as part of the GeNEDAR cohort. Topological parameters (i.e., local efficiency, strength, and clustering coefficient) of connectivity networks from 40 regions of interest were obtained in DSIstudio. In the analysis, the effects of sex and age on the AD of topological measures are examined. Changes in the asymmetrical organisation of the network tended to reveal a turning point around the transition from middle to late adulthood. In regions such as the putamen, frontal, and superior temporal, greater asymmetry was observed with age. Conversely, there was a tendency towards symmetry in the medial orbitofrontal, paracentral, and inferior parietal regions. These findings, largely novel, reinforce their potential as biomarkers of brain ageing.

V-073

Role of the Endocannabinoid System in Compulsive and Perseverative Behaviors Induced by Inhibition of Striatal Cholinergic Interneurons.

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Striatal cholinergic interneurons (SCIN) are key modulators of cortico-striatal circuits implicated in disorders like obsessive-compulsive disorder and Tourette syndrome (TS). Reduced SCIN density observed in TS patients suggests their involvement, highlighting their relevance for targeted therapeutic strategies. Previous studies in our laboratory showed that SCIN inhibition or ablation in animal models leads to repetitive and perseverative behaviors. Here, we investigated if enhancing endogenous eCB tone could reduce these behaviors, given CB1 receptor abundance and eCB modulation in the striatum. We employed URB597, a fatty acid amide hydrolase inhibitor that elevates endocannabinoid levels and reduces perseverative behaviors in control mice. Additionally, we used viral vectors to selectively express an inhibitory DREADD receptor in SCINs of ChatCre heterozygous mice, enabling reversible SCIN inhibition through CNO administration. Mice were acutely treated with URB597 (0.1 or 1 mg/kg, i.p.) or vehicle 1h before behavioral measurement. SCIN inhibition in control mice increased behaviors indicative of compulsivity, such as grooming, head dipping, nest disruption, and marble burying. Notably, treatment with URB597 reduced these compulsive and perseverative behaviors. In conclusion, our results highlight the involvement of SCINs in ritualistic behavior and support the therapeutic potential of endocannabinoid system modulation in disorders linked to cortico-striatal dysfunction.

V-074

Evaluation of Orthostatic Hypotension and Depression and Anxiety Symptoms in Patients with Parkinson's Disease: Preliminary Results of a Cross-Sectional Study

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In this cross-sectional study of 32 patients with idiopathic Parkinson's disease (mean age 67 years, 56% male, mean disease duration 5 years), we evaluated the association between orthostatic hypotension (OH) and mood symptoms. All participants completed the MDS-UPDRS, Beck Depression Inventory (BDI), Beck Anxiety Inventory (BAI), and the Orthostatic Hypotension Questionnaire (OHQ), with orthostatic response measured by serial blood pressure and heart rate recordings. Mean BDI (10.0 ± 5.9) and BAI (8.8 ± 4.6) scores indicated mild depression and anxiety. No significant differences in depression or anxiety scores were observed between patients with or without OH ($p = .86$ in both cases). However, the severity of self-reported orthostatic symptoms correlated positively with anxiety levels (Pearson $r = .49$, $p = .004$; Spearman $\rho = .53$, $p = .002$), a finding that remained significant after adjusting for age, sex, and disease severity. Correlation with depression was positive but weaker and did not reach significance (Pearson $r = .33$, $p = .061$; Spearman $\rho = .43$, $p = .014$). These preliminary results suggest that while objective OH was not linked to mood disturbances, subjective orthostatic symptoms were associated with greater anxiety and, to a lesser extent, depression, underscoring the need to assess perceived autonomic symptoms in Parkinson's care despite the study's small sample size and measurement limitations.

V-075

Chronic treatment with Doxycycline parcially rescues Tau pathology and behavioural phenotypes in a mouse model of tauopathy

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The tetracycline doxycycline (Dox) has been reported to have neuroprotective effects unrelated to its antibiotic activity. Previous evidence show a beneficial effect of Dox in rodent models of Parkinson disease by 6-OHDA lesion (1-2). Our present aim was to determine whether Dox could rescue behavioral and biochemical phenotypes related in the htau mouse model of tauopathy. Htau mice develop a combination of phenotypes including cognitive decline, motor coordination and olfactory deficits , related to the pathological tau accumulation in the prefrontal cortex and the striatum (3).

Phenotypic onset in this model starts at 6 months old (4). In this study, htau mice were treated chronically with doxycycline in their diet from 6 months-old onwards. Behavioral phenotypes were analyzed at 6 and 12 months-old to determine time course of behavioral deficits. At the end of treatment, biochemical studies were performed to determine phospho-tau levels and inflammatory markers. The Dox treatment improved motor coordination and olfactory deficits in htau mice, compared with the placebo group. We observed a parcial rescue in IL-10, GFAP and phospho-tau contents specifically in striata of Dox treated htau mice. Further research is under way to investigate the molecular pathways leading to the beneficial effect of Dox over tauopathy phenotypes in this model.

V-076

Enriched Environment Modulates Glial Reactivity and Restores Cerebellar Function After Perinatal Asphyxia in Rats

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Perinatal asphyxia (PA), caused by impaired placental gas exchange during gestation or delivery, increases the risk of neonatal brain injury and long-term neurodevelopmental disorders. Although no specific therapy exists, non-pharmacological interventions such as Enriched Environment (EE) have shown promise in promoting neuroplasticity and functional recovery. This study evaluated the effects of EE on cerebellar morphology and sensorimotor reflexes in PA-exposed rats. Newborn rats were assigned to control and PA groups, housed under standard (ST) or enriched (EE) conditions until postnatal day 21. Cerebellar tissue was analyzed using GFAP, MAP-2, and NF markers. PA-ST animals showed altered Bergmann glia and reduced molecular layer thickness, partially restored in PA-EE. GFAP reactivity was elevated in both CTL-EE and PA-EE ($p < 0.001$), suggesting astroglial activation. MAP-2 expression was decreased in PA-ST and PA-EE ($p < 0.001$), with denser labeling near Purkinje cells. Behaviorally, PA-ST rats exhibited significant deficits in righting ($p < 0.05$), air righting ($p < 0.001$), limb grasp ($p < 0.001$), walking ($p < 0.05$), and negative geotaxis ($p < 0.05$). EE exposure improved or normalized these reflexes, reaching control levels in several tasks. These findings support EE as a promising early intervention strategy to mitigate PA-induced cerebellar damage and functional impairments.

V-077

Alterations in structural and functional excitation/inhibition balance of pyramidal neurons in the mPFC of a mouse model relevant for schizophrenia

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Schizophrenia is a severe neurodevelopmental disorder characterized by positive and negative symptoms as well as cognitive impairment, which drastically hinders self-sufficiency. Thus, understanding the underlying circuits related to cognitive deficits is crucial. In mice, cognition correlates with synchronous activity in the mPFC, maintained by reciprocal synapses between pyramidal neurons (PNs) and interneurons, particularly parvalbumin interneurons (PVIs). We used a mouse model of PVI dysfunction in which NMDARs are eliminated in corticolimbic interneurons, predominantly PVIs, which displays cognitive deficits and mPFC circuit alterations, including excitation/inhibition (E/I) imbalance in PNs. We hypothesized that mPFC circuit alterations in KO mice could be attributed to a deficit in PVI-PN synaptic maturation, hence we performed an immunohistochemical quantification of pre and postsynaptic perisomatic markers in PNs. These structural results reveal differences between developmental stages and suggest a disbalanced expression of both GABAAR α1 and α2 subunits in adult KO mice, without changes in PV puncta density. Furthermore, in an additional experiment we performed a correlation analysis between PVI/GABAARα1 perisomatic puncta and miniature EPSCs and IPSCs electrophysiologically recorded in the same PN. These structural-functional results suggest adult KO mice lack a homeostatic compensatory mechanism for maintaining an appropriate E/I balance.

V-078

Adjuvants markedly enhanced glyphosate neurotoxicity: evidence of altered dendritic maturation and synapse structure

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Currently, human populations are continuously exposed to numerous environmental xenobiotics, including a broad range of pesticides such as glyphosate (Glyph) and its formulations (glyphosate-based herbicides, GBH), the most widely used worldwide. Increasing evidence indicates that the central nervous system is a target of both Glyph and GBH toxicity; however, the underlying mechanisms remain poorly understood. Studies from our group have reported behavioral impairments, including deficits in recognition and spatial memory in exposed rats, as well as marked effects on hippocampal neurons, impacting morphology, neuronal maturation, and synapse formation. The aim of this study was to determine whether GBH exposure induces alterations in young neuronal cultures (24 and 48 h) and in mature neurons (21 DIV). Our results showed that in immature neurons, 0.5 mM Glyph-equivalent GBH delayed axonal and dendritic development, resembling the effects of pure Glyph at doses 50 times higher. In mature cultures, 0.05–0.1 mM Glyph-equivalent GBH reduced dendritic tree complexity and affected both the number and type of dendritic spines, comparable to effects caused by pure Glyph at doses 60 times higher. Moreover, preliminary assays from the hippocampus of treated rats showed a decrease in the activity of effectors of non-canonical Wnt pathways as well as in the expression of synaptic markers. These findings suggest that the presence of adjuvants in GBH enhances glyphosate neurotoxicity.

V-079

TGF-β3 Overexpression Mitigates Cognitive Impairments and Neuroinflammation in a Rat Model of Early Parkinson's Disease

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Parkinson´s disease is a complex pathology marked neurodegeneration in dopaminergic neurons of the nigrostriatal system. The early prodromal phase has been recognized for the presence of non-motor symptoms that could be predictive of later motor disease. By inducing dopaminergic neuron death with 6-hydroxydopamine (6-OHDA) in male Wistar rats we have observed alterations in cognitive functions within 3 weeks of the surgery. We aimed to test the potential of TGF-β3, a trophic and anti-inflammatory factor, to mitigate these early changes. We administered an adenoviral vector containing the TGF-β3 gene (RAd-TGF-β3), or its control, 14 days after 6-OHDA (or vehicle) surgery. On day 21 we performed the Barnes Maze test, the Novel Object Recognition test and the Modified Y-Maze. We found that RAd-TGF-β3 in animals with 6-OHDA induced a better performance in these tests compared to animals administered with the control vector. At this time we demonstrated an anti-inflammatory effect measuring pro-inflammatory factors' expression (IL-1, IL-6, TNF-α) in the hippocampus, as well as glial activation markers (CYP46, IGF-1), observing a reduction with RAd-TGF-β3 in comparison to 6-OHDA animals. Moreover, latency values in Barnes Maze correlate more with expression values of TNF-α than with other cytokines. We conclude that the overexpression of TGF-β3 can overcome the damaging processes induced by 6-OHDA and therefore has potential to treat parkinsonism's early impairments.

V-080

Behavioral and Histopathological Characterization of a Novel APP Knock-In Mouse Model of Alzheimer's Disease

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Modeling Alzheimer's disease (AD) in vivo is a major challenge in neuroscience. Since the 1990s, multiple transgenic mouse models have been generated, providing key insights into amyloid-β (Aβ) pathology and therapeutic strategies. However, most rely on APP or APP/presenilin-1 (PS1) overexpression, which can produce artifacts related to protein overproduction or mislocalization. To overcome these drawbacks, single App knock-in lines carrying familial AD mutations have been developed. Here, we characterized behavioral and neuropathological traits of the new AppNL-F;Psen1P117L knock-in (APP KI) mouse strain. Homozygous APP KI and C57BL/6 wild-type (WT) mice were tested at 3 and 6 months in the open field, elevated plus maze, novel object recognition, and Y-maze tasks to evaluate locomotion, anxiety-like behavior, episodic memory, and working/spatial memory. At six months, APP KI mice exhibited preserved locomotor activity but increased novel arm entries with reduced exploration time, suggesting novelty-seeking/impulsivity and impaired sustained exploration. These changes correlated with moderate cortical Aβ deposition and minimal hippocampal pathology. Ongoing studies aim to expand this profile. Together, our findings strengthen the link between incipient neuropathology and early behavioral alterations in this new-generation AD model, providing measurable features relevant for testing disease-modifying interventions.

V-081

Patient-Derived Neural Cells with UBQLN2 Variants Reveal Proteasome Dysfunction and Mitochondrial Stress

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Ubiquilin2, encoded by UBQLN2, is critical for clearing misfolded proteins and maintaining homeostasis. Pathogenic variants are linked to protein accumulation and stress, but their effects in neural cells remain unclear. We hypothesized that neural cells derived from FTD patient carrying UBQLN2 variants would display impaired proteostasis, mitochondrial dysfunction and oxidative stress. Patient-derived induced pluripotent stem cells were differentiated into neural stem cells (NSC). mRNA and protein levels were quantified by qPCR and Western blot. Reactive oxygen species (ROS) and mitochondrial membrane potential (MMP) were measured by flow cytometry with fluorescent probes. Proteasomal activity was assessed by cycloheximide chase of c-Myc degradation. Patient NSC showed elevated Ubiquilin2 levels without mRNA changes, along with reduced Hsp70 and slowed c-Myc degradation, indicating proteostasis defects. NSC also displayed higher ROS, mitochondrial superoxide and hyperpolarized MMP, consistent with mitochondrial dysfunction. In addition, Sirt1 and Sod2 were reduced, reflecting weakened antioxidant defenses. These findings show that UBQLN2 variants impair proteostasis, compromise antioxidant defenses, and induce mitochondrial stress, processes that may drive neurodegeneration. Protein accumulation without mRNA changes highlights defective clearance as a key pathogenic mechanism. This model provides a platform to study UBQLN2-linked disease mechanisms and therapeutic strategies

V-082

Cellular treatment with natural component curcumin for Treatment of peripheral neuropathies

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Peripheral neuropathies are highly prevalent disorders whose clinical progression is often unfavorable due to the lack of effective therapies that comprehensively address patient needs. Previous studies of our lab have demonstrated the therapeutic potential of multipotent stem cells of bone marrow or adipose tissue origin; while there is some evidence that curcumin could aid regeneration due to its antioxidant and anti-inflammatory effects. In this context, the aim of the present study is to increase the bioavailability of curcumin to potentiate the regenerative effects of multipotent stem cells. PLGA nanocapsules were synthetized with different concentrations of curcumin solutions, to find a non-cytotoxic concentration for cells. For this, adult multipotent stem cells from bone marrow or adipose tissue, as well as Schwann cells, were used. Cell viability was assessed through MTT assay and fluorescence microscopy with vital dyes, as well as cell-type-specific markers. Encapsulation of 6 mg/ml curcumin in chloroform led to the formation of non-homogenous PLGA nanocapsules in terms of size, and cytotoxic for multipotent cells. 10 times curcumin concentration reduction led to more homogeneous nanocapsules but still cytotoxic for multipotent cells. Finding the correct concentration of curcumin may provide basis for new therapeutic strategies for treatment of peripheral neuropathies taking advantage of the anti-inflammatory and antioxidant effect of this natural compound.

V-083

Loss of GFRα1 in Mice Reveals Behavioral and Neural Signatures of Psychiatric Disorders

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During the development of the nervous system, the formation of synaptic circuits is tightly regulated through precise control of axonal and dendritic growth. Neurotrophic factors, such as the "glial cell line-derived neurotrophic factor" (GDNF) and its receptor GFRα1, play a pivotal role in dendritic arborization and spine maturation within the cerebral cortex and hippocampus. Disruptions in neuronal connectivity could contribute to the etiology of various neurodevelopmental disorders. Evidence from both human and rodent studies indicates that alterations in the excitatory/inhibitory synaptic balance are a hallmark of neurodevelopmental psychiatric conditions, including schizophrenia, autism spectrum disorder, and Rett syndrome. Furthermore, changes in neural morphology and synaptic architecture may underlie not only synaptic imbalance but also behavioral abnormalities observed in mouse models of these disorders. Despite these findings, the specific role of the GDNF/GFRα1 signaling pathway in the maturation and remodeling of synaptic circuits across distinct forebrain regions remains poorly understood. To address this, we have developed a novel conditional mutant mouse line with targeted ablation of GFRα1 in selected populations of forebrain neurons. This model will enable us to dissect the specific contributions of GDNF/GFRα1 signaling to the development and function of forebrain circuits implicated in neurodevelopmental disorders.

V-084

Generation and validation of a human induced pluripotent stem cell line from an Argentine patient with familial Alzheimer's disease carrying the PSEN1 M146L variant

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Familial Alzheimer's disease (fAD) is an autosomal dominant, early-onset form of Alzheimer's disease (AD), commonly caused by mutations in PSEN1. Among these, the PSEN1 M146L variant is one of the most frequently reported pathogenic mutations and has been associated with aggressive disease progression. Despite its prevalence, the functional consequences of this mutation remain poorly understood, particularly in underrepresented populations such as those from Latin America. To address this gap, we generated a human induced pluripotent stem cell (hiPSC) line from erythroblasts amplified from peripheral blood mononuclear cells of an Argentine patient clinically diagnosed with fAD and carrying the heterozygous PSEN1 p.M146L variant. Reprogramming was performed using the STEMCCA lentiviral vector, which encodes the Yamanaka factors (OCT4, KLF4, SOX2, and c-MYC). The resulting hiPSC line, named FBAD1, exhibited typical pluripotent morphology and expressed endogenous markers (OCT4, SOX2, NANOG, and TRA-1-60), as validated by immunofluorescence and RT-qPCR. FBAD1 also showed differentiation into the three germ layers by embryoid body assay, maintained a normal karyotype, and carried the PSEN1 M146L variant, confirmed by Sanger sequencing. Importantly, directed differentiation yielded cortical and glutamatergic neurons, validated by neuronal and subtype-specific markers. This hiPSC line represents a patient-specific model to identify early pathogenic signatures associated with AD.

V-085

Automatic classification of TLE-HS using using functional and structural connectome

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We evaluated the performance of automatic machine learning classifiers in distinguishing healthy controls from patients with temporal lobe epilepsy and hippocampal sclerosis (TLE-HS), based exclusively on structural (DTI) and functional (fMRI) connectomes.
METHOD: Connectomes were derived from DTI and fMRI of 49 TLE-HS patients (23 right) and 47 healthy controls scanned on the same 3T system. Connectivity matrices (80 cortical and subcortical regions) were generated using the FreeSurfer v6.0 wparc atlas. Structural edges were defined by normalized mean FA values; functional edges by Pearson correlations of BOLD signals (Fisher-transformed). Classification models were trained with SVMc, logistic regression, and linear discriminant analysis (LDA) using the NBS_predict toolbox with 10-fold cross-validation (10 repetitions, p = .01, 5 hyperparameter steps, 500 permutations).

RESULTS: Patients' mean age was 35.2 (±8y) vs. 37.5 (±11) in controls; no gender differences (p > .05). Epilepsy duration (5–50y) and seizure frequency were similar by side. For fMRI connectomes, LDA performed best (accuracy 0.67, AUC 0.679, sensitivity–specificity 0.65–0.70). Logistic regression (0.50) and SVMc (0.47) performed worse. For DTI connectomes, LDA again outperformed (accuracy 0.68, AUC 0.682).

CONCLUSIONS: Connectome-based predictive models using LDA consistently detected TLE-HS, suggesting potential diagnostic value, though replication in larger cohorts is required.

V-086

Anxiety disorders and Irritable Bowel Syndrome: evidence from a Systematic review and Meta-Analysis

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Anxiety disorders are among the most common mental health conditions worldwide and are often accompanied by gastrointestinal symptoms. The gut-brain axis has emerged as a key pathway linking psychological and gastrointestinal disorders. Irritable bowel syndrome (IBS), affecting about 14% of the global population, is a functional gastrointestinal disorder associated with impaired quality of life and health care utilization. While most studies have examined anxiety prevalence in IBS, the reverse association remains underexplored. Addressing this gap is crucial, as gastrointestinal symptoms in anxious patients may be underrecognized or misattributed.

This systematic review and meta-analysis aims to estimate the prevalence of IBS in adults with anxiety disorders. PubMed, Embase, and Cochrane are being searched for observational studies in adults published up to 2025. Inclusion criteria include validated anxiety diagnoses and IBS defined by Rome criteria or clinical evaluation. Data extraction and risk-of-bias assessment follow PRISMA guidelines. Preliminary evidence suggests a higher prevalence of IBS in anxious patients than in the general population. Analyses are ongoing. The final results will be presented at the meeting.

This study will provide quantitative evidence on the prevalence of IBS in anxiety disorders, complementing prior meta-analyses focused on psychological comorbidities in IBS, by including studies published through 2024 and 2025.

S-086

Regulation of Gastrointestinal Homeostasis Mediated by Neuropeptides in Drosophila melanogaster

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Neuropeptides are essential molecules in the regulation of physiological processes. They are produced by the central nervous system (CNS) and are recognized by specific receptors expressed in various cells of the CNS itself or in peripheral tissues. In this way, neuropeptide-mediated signaling controls processes such as sleep, stress response, emotions, among others. In Drosophila, some of these neuropeptides are produced by enteroendocrine cells (EECs), which are located in the intestinal epithelium. These cells are responsible for detecting changes in the conditions of the digestive tract and releasing neuropeptides in response to different stimuli. Although the involvement of neuropeptides in the aforementioned functions is known, their role in establishing gastrointestinal homeostasis has not been described in detail.

In this study, we performed a screen of 16 neuropeptides expressed in Drosophila EECs, measuring feeding and defecation rates as parameters of gastrointestinal homeostasis. Preliminary results of this screen will be presented in the poster session

V-037

Audiovisual stimulation at 40 Hz promotes circuit plasticity in the aging dentate gyrus

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Non-invasive audiovisual stimulation at 40 Hz (AuViS) has emerged as a promising strategy to restore gamma oscillations in the cortex and hippocampus, reduce amyloid-beta plaque load, and improve memory in mouse models of Alzheimer's disease. In 8-month-old (8M) animals, in which cognition is already compromised, we found that only 3 weeks of AuViS can restore novel object recognition (NOR) test performance. We hypothesized that AuViS promotes the remodeling of hippocampal circuits that could underlie these beneficial effects. To test this idea, we characterized neurotransmission at the perforant path to dentate granule cell (GC) synapse, the gateway to hippocampal processing. We performed whole-cell patch-clamp recordings in response to pulse trains delivered at different frequencies in 8M animals exposed to 6 weeks of AuViS or control conditions. Our preliminary results indicate that aging increases summation of excitatory postsynaptic potentials, a phenotype that was reversed by chronic AuViS. This effect is likely mediated by a shift in the excitation/inhibition balance through enhanced inhibitory synaptic transmission. These findings suggest that AuViS promotes activity-dependent synaptic plasticity in the aging hippocampus, which would ultimately contribute to its cognitive benefits.

V-087

Effects of development and social isolation on audiovisual habenular responses of larval zebrafish

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The lateral habenula (LHb) is a key structure that controls both dopaminergic and serotonergic pathways, which support motivational, motor, and cognitive functions. It is strongly involved in negative reward processing, and its dysfunction has been linked to depression.

During early development, when neural circuits are being formed, the brain is highly sensitive to environmental inputs. Lack of proper stimulation in this critical period can cause lasting deficits. Social interaction is particularly important for shaping normal behaviors, and early-life social isolation is a major risk factor for neuropsychiatric disorders, including depression.

We aim to study the role of the habenula in behavioral changes caused by social isolation during early development in larval zebrafish. Here we present preliminary results on activity of Habenular neurons of 6-16 days old larval zebrafish in response to audiovisual stimuli. For in vivo calcium imaging, we used transgenic zebrafish expressing GCaMP6f (Nacre[elavl3:GCaMP6f]). Confocal images were motion-corrected (CaImAn) and segmented (Cellpose) to measure fluorescence from individual neurons. We analyzed spontaneous and stimulus-evoked activity in animals raised in a social (control) context from 6 to 16 days old to see how habenular activity changes during development. In addition, we compared activity between control and isolated groups to test if lack of social interaction affects stimulus processing in the Habenula.

V-088

Aging disrupts the temporal organization of slow oscillations beyond density reduction

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Sleep slow oscillations (SOs) are a hallmark of NREM sleep and play a crucial role in glymphatic clearance. Age-related reductions in SO density and amplitude are well documented and linked to impaired clearance. However, little is known about whether aging also alters their temporal organization.

In this work, we introduce a novel approach to classify SOs according to their temporal structure, distinguishing isolated SOs from trains of consecutive SOs based on inter-SO intervals. We analyzed overnight EEG recordings from 57 young and 51 elderly adults across three independent datasets. We quantified the proportion of isolated versus consecutive SOs and examined the distribution of train lengths.

Elderly adults showed a significantly higher proportion of isolated SOs and shorter trains compared to young adults. These effects remained robust after controlling for SO density and sleep stage composition. Additional analyses using temporal shuffling and density-matched epochs confirmed that the observed differences cannot be explained solely by lower SO density, but rather reflect a genuine age-related loss of rhythmicity.

These findings reveal that natural aging disrupts not only the amount and amplitude of SOs but also their temporal regularity. Such temporal disorganization may weaken sustained ionic currents underlying CSF flow, reduce the efficiency of metabolic waste clearance during sleep, and contribute to increased vulnerability to neurodegenerative processes.

V-089

Intersegmental rhythmic propagation and phase lag in motor control

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Leeches crawl on solid surfaces by sequential waves of elongation and contraction of their body. Each segmental ganglia contains a central pattern generator (CPG) that can generate fictive crawling ('crawling') when exposed to dopamine (DA). Our goal is to elucidate the role of intersegmental interactions on 'crawling' coordination.

In this study we isolated chains of three ganglia and applied DA in three configurations: global DA application, or local DA to the anterior or posterior ganglion, leaving the other two untreated. 'Crawling' was monitored in each ganglion by extracellular recordings of a contraction phase motoneuron. Coordination across ganglia was assessed using cross-correlation index and phase lag.

Global DA induced coordinated activity in all ganglia (similar firing frequency). Local DA generated 'crawling' in the treated ganglion and rhythmic activity in adjacent untreated ganglia; similar lag between treated and adjacent ganglia to global DA, but untreated ganglia showed null lag between them. Firing frequency decreased linearly from the treated ganglion. Thus, activation of a segmental CPG entrains activity both ways but does not activate untreated CPGs nor establishes an interganglionic lag. The results suggest a multilayered intersegmental connectivity: a basic layer transmits rhythmic activity without lag, while local CPG activation is required for autonomous oscillation and proper metachronal order.

V-090

Activity-dependent competition: a fight for synaptic targets in the adult hippocampus

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Neurogenesis occurs in the dentate gyrus of the adult mammalian hippocampus and it is involved in learning, memory, and spatial encoding. Adult-born granule cells (aGCs) are continuously incorporated into the dentate gyrus in a highly regulated process, projecting to interneurons and pyramidal cells in CA3. Electron microscopy studies have shown that developing aGCs first establish their presynaptic terminals (mossy fiber boutons, MFBs) onto thorny excrescences of pyramidal cells already occupied by preexisting connection. As aGCs mature, however, these connections are refined into a single terminal per spine. These findings have led to the hypothesis that integration of aGCs is governed by activity-dependent synaptic competition. To test this hypothesis, chemogenetic or optogenetic actuators were expressed in defined cohorts of aGCs allowing manipulation of their activity in vivo. Newly generated aGCs with enhanced activity displayed a higher MFB density compared to controls. MFBs from preexisting mature neurons located in close proximity to active aGC terminals displayed a reduced size, which suggest a diminished synaptic strength. In contrast, MFBs neighboring inactive aGCs were unchanged. This provide direct evidence that neuronal activity promotes the establishment of new synapses at the expense of preexisting ones. Ongoing experiments will define the critical windows for synaptic competition, dissect underlying mechanisms, and determine the functional implications

V-091

Behavioral and Neural Bases of Navigation Guided by Chemical Information

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The world is highly complex. To survive, animals extract information from environmental signals, evaluate it in the context of internal states, and transform it into motor actions for navigation. Chemical cues are essential and conserved across species, yet strategies and mechanisms remain poorly understood. This project proposes a novel approach using zebrafish larvae to address three aims:

Aim1: What information in chemical stimuli guides navigation?

Aim2: How are chemical stimuli encoded in the brain and which circuits represent spatial information?

Aim3: How do internal states shape navigation strategies, and how do chemical stimuli affect these states at behavioral and neural levels?

Zebrafish larvae enable the integration of microfluidics, light-sheet microscopy, optogenetics, and large-scale data analysis to study chemical-guided navigation with precise stimulus control while measuring whole-brain activity and behavior.
In this poster, I hope to discuss with the general audience the central ideas of the project with which I am starting a laboratory at IFIByNE.

V-092

Differential Extracellular Matrix and Perineuronal Nets Remodeling in CNS Regions During Persistent Inflammatory Pain

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Persistent inflammatory pain induces neuroplastic changes that sustain nociceptive processing. As a dynamic structural and functional component of nervous tissue, the extracellular matrix (ECM) and perineuronal nets (PNNs) can modulate pain sensitivity by regulating synaptic plasticity. We hypothesized that ECM remodeling in the spinal cord and primary somatosensory cortex (S1) correlates with nociceptive alterations during chronic pain. To test this, we employed an inflammatory pain model and evaluated mechanical allodynia (electronic Von Frey) and thermal hyperalgesia (Hargreaves apparatus). Spinal cord and S1 samples were processed for triple immunofluorescence targeting ECM components, NeuN, and parvalbumin, followed by confocal microscopy. Behavioral assays revealed sex differences: females exhibited smaller threshold reduction and faster recovery. Preliminary analyses of ECM and PNN expression demonstrated regional differences: while S1 showed changes at day 13 post-injection, no alterations were detected in the spinal cord. These findings suggest a complex temporal pattern of ECM remodeling in the CNS during inflammatory pain. Such modifications may promote maladaptive plasticity and central sensitization, pointing to potential therapeutic targets for persistent pain. Ongoing studies aim to further dissect sex-related mechanisms in ECM regulation during pain.

V-093

Neural encoding reorganization through learning in the DG-CA3 circuit

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The hippocampus is a brain region involved in memory and spatial navigation. The dentate gyrus (DG), the first stage of hippocampal processing, sends information via mossy fibers to CA3 pyramidal neurons where it is integrated into a dense recurrent network. Yet, how these two hippocampal subfields encode information within the same task and how each restructures its coding with experience remain unclear. In our study, we trained mice in a virtual reality discrimination task based on olfactory and visual context cues. We recorded DG and CA3 activity in first-session and expert animals using in vivo electrophysiology and quantified the contribution of sensory, behavioral, and cognitive variables to neuronal activity with a Poisson Generalized Linear Model. We observed that in the DG, the capacity of single neurons to respond to multiple variables simultaneously, known as mixed-selectivity, increases with learning. Moreover, encoding of position, speed, and reward strengthens, revealing experience-dependent reorganization. In contrast, CA3 exhibits mixed-selectivity even before learning, indicating an intrinsic predisposition to integrate multiple signals. However, context, reward, and odors only become decodable in expert animals. These findings suggest that learning reorganizes DG and CA3 differently, enabling more specific encoding of key task elements. The DG builds its codes from experience, whereas CA3 refines and selects relevant signals on a preexisting framework.

V-094

Rol of STAT signaling mechanisms in the spacing effect requiered for Long-Term Memory

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The general objective of our studies is to understand the molecular bases of intellectual disability in Noonan syndrome (NS) and related disorders, which is caused by mutations in genes encoding proteins of the RAS-ERK1/2 signaling pathways. Gene interaction studies in Drosophila showed that developmental defects produced by mutations associated with NS are modulated by RAS-ERK1/2 and JAK/STAT (Janus Kinase / Signal Transducer and Activator of Transcription) signaling. Previous studies indicate that STAT, like RAS-ERK1/2, participates in long-term memory (LTM) formation. Preliminary studies from our laboratory show that genetic manipulation of STAT abolishes the spacing effect (SE) in aversive olfactory conditioning. The SE refers to the enhancement of memory following spaced training sessions (with time intervals between sessions) compared to the same number of training sessions in a massed regime (without such intervals). This effect is reduced in animal models of NS; therefore, we aim to determine the role of STAT in the spacing effect in contextual learning. To this end, we use genetically modified flies with increased or decreased STAT function and analyzed their contribution to memory after massed vs. spaced training. Preliminary results of flies with overexpression of a wild-type allele of STAT showed a distinctive behavioral performance compared with the control genotypes. This study will advance the understanding of the role of STAT in the SE and provide new insights

V-095

Effects of non-invasive audiovisual gamma stimulation on the dynamics of local networks in the aging hippocampus

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Gamma rhythms (~25–100 Hz) play a key role in cognition. Their disruption in neurological disorders such as Alzheimer's disease (AD) has been linked to memory deficits and cognitive decline. Non-invasive audiovisual (AuVis) stimulation at 40 Hz (in the gamma range) has emerged as a promising intervention, as it reduces amyloid-β plaques, and improves behavioral performance of AD models in spatial memory tasks. Our previous work has shown that AuViS promotes synaptic plasticity, as seen by its positive effects on neurogenesis in the aging hippocampus. This suggests that cognitive improvements promoted by this treatment are likely supported by broad circuit-level modifications. To investigate the impact of AuViS on hippocampal network dynamics, we performed in vivo single-unit and local field potential (LFP) recordings across hippocampal subregions in 8-month-old mice exposed to chronic 40 Hz AuViS. We present a characterization of oscillatory power, cross-regional coherence, and ensemble activity during spatial navigation in familiar and novel environments before and after six weeks of daily stimulation. Our results provide mechanistic insights into the beneficial effects of AuVis stimulation on cognition in an aging brain.

V-096

Comparative Tract Tracing of Mushroom and Reniform Bodies in the true crab Neohelice granulata

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Our previous work in the true crab Neohelice granulata revealed both neuroanatomical and, for the first time, functional evidence of a mushroom body (MB) center structurally analogous to those well characterized in insects. Our studies sparked a debate among other authors regarding the distinction between mushroom bodies (MB) and reniform bodies (RB) in true crabs. In the present study, we propose to apply fluorescent dextran tracers to the peduncular tracts of both the MB and the putative RB fascicle, coupling distinct dyes with high-resolution confocal reconstructions to delineate intrinsic neuron cohorts, branching patterns, and efferent projections—directly testing our MB homology hypothesis. In parallel, we propose targeted dextran injections into the lobula neuropil to map visual pathways converging onto calyx- and lobe-like MB domains. Comparative analysis of tracer distributions is expected to resolve tract origins and domain segregation, confirm that the labeled tracts carry MB intrinsic neurons, and reveal how lobula-derived circuits interface with the MB network. Moreover, this approach may enable us to propose potential functions for the RB in crustaceans. Ultimately, this comprehensive anatomical mapping aims to substantiate the presence and architecture of mushroom bodies in true crabs and illuminate the neural substrates underlying higher-order sensory integration within the MB.

V-097

Time-resolved dynamics of microglial reactivity in parkinsonian mice

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Parkinson's disease (PD) is characterized by striatal dendritic spine loss and microglial reactivity, but the role of microglia in this process is not well understood. We addressed this question in a mouse model of hemi-parkinsonism induced by unilateral 6-hydroxydopamine intracerebral administration. Medium spiny neurons were sparsely labeled to examine dendritic spine density and morphology, while microglial reactivity was assessed by immunostaining and 3D cell reconstruction at 1-, 2- and 4-weeks post-lesion (n=5 animals/group).

Microglia showed a rapid, multiphasic response that evolved over one to four weeks after lesion: an early phase of proliferation and local clustering, followed by transient retraction of processes with reduced coverage and branching complexity, and a later stage with persistent soma enlargement. In parallel, medium spiny neurons progressively lost dendritic spines, coinciding with increased microglial engulfment signals, suggesting active participation of microglia in synaptic remodeling.

Together, these results describe a dynamic sequence of structural and functional changes in microglia that overlap with synaptic pruning. They highlight microglia as active player in striatal remodeling and reveal critical time windows that could be targeted to modulate parkinsonism-related plasticity.

V-098

Kv7 channel dysfunction and HCN channel modulation shape excitatory synaptic integration in thalamocortical circuits under leptin deficiency.

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This study investigates how Kv7 potassium channel dysfunction and modulation of Hyperpolarization-activated Cyclic Nucleotide-gated (HCN) channels influence excitatory synaptic integration in ventrobasal (VB) thalamic neurons of leptin-deficient obese (ob/ob) mice versus wild-type (WT). HCN channels mediate the hyperpolarization-activated Ih current, which stabilizes resting membrane potential and input resistance. Electrophysiological recordings from brain slices of WT and ob/ob mice were used to characterize AMPA receptor kinetics ($\tau 1 \approx 1.19$ ms, $\tau 2 \approx 1.22$ ms) as well as Kv7 and HCN levels, which were then incorporated into a realistic multicompartmental NEURON model. AMPA receptors were placed at the dendritic level and activated using a stochastic event generator. WT and ob/ob models were tested under varying Kv7 and HCN conductances. WT responses remained stable, whereas ob/ob neurons—due to Kv7 dysfunction—exhibited heightened sensitivity, with spike output strongly modulated by HCN alterations. In ob/ob neurons, impaired Kv7 function reduces subthreshold stabilization, making excitability highly dependent on HCN activity. This interaction amplifies synaptic variability and responsiveness, suggesting disrupted sensory processing in obesity-related pathophysiology. These findings highlight the critical Kv7-HCN interplay in maintaining excitability stability and implicate their dysfunction as a potential mechanism underlying thalamocortical alterations in obesity.

V-099

A Promising Chalcone with Multitarget Activity Against Neurodegenerative Disorders

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Chalcones are flavonoids with potential in neurodegeneration. Our aim is to develop chalcone-derived compounds capable of simultaneously modulating multiple pharmacological targets relevant to neurodegenerative disease. From a library of 2′-hydroxychalcones, we identified chalcone 1 (3-chloro-4′,5′-dimethyl-2′-hydroxychalcone), which showed BBB permeability (PAMPA-BBB), no cytotoxicity (SH-SY5Y, ≤10 μM), and compliance with Lipinski's rule, suggesting drug-likeness. In vitro, chalcone 1 inhibited mouse brain acetylcholinesterase (IC_{50} = 4.4± 0.8 μM) with low butyrylcholinesterase activity, and reduced Aβ aggregation (51.6± 11.3% at 10 μM). In mice, acute administration (3 mg/kg, i.p.) improved working and long-term memory without affecting anxiety, sedation, or motor activity. Chalcone 1 also selectively inhibited human MAO-B (IC_{50} = 0.354± 0.084 μM) with negligible MAO-A effect. In a rotenone-induced PD mice model, 7-day treatment reversed motor deficits, reduced oxidative stress, and prevented behavioral impairments, without changes in controls. ThT-based RT-QuIC assays further showed inhibition of α-synuclein aggregation (35.2 ± 7.9 and 58.2 ± 5.8%, reduction in ThT signal at 10 and 100 μM respectively). Altogether, chalcone 1 emerges as a promising multitarget lead for Alzheimer's and Parkinson's, combining MAO-B inhibition, anti-cholinesterase, anti-aggregation (Aβ and α-synuclein), antioxidant, neuroprotective, and cognitive-enhancing properties.

V-100

Yerba Mate: A Natural Antioxidant with Neuroprotective Potential in Parkinson's Disease

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Oxidative stress and neuroinflammation have been proposed as key mechanisms underlying the dysfunction and death of dopaminergic neurons in the substantia nigra, leading to the development of Parkinson's disease. Yerba mate (YM) consumption has been associated with multiple health benefits, largely attributed to its bioactive compounds with strong antioxidant activity. In a hemiparkinsonian mouse model, we previously observed that chronic treatment with YM exerts neuroprotective effects on dopaminergic neurons. The present study aimed to determine whether neuroprotection induced by YM extract is associated with a favorable modulation of oxidative stress, either through the enhancement of antioxidant enzyme activity and/or neutralizing free radicals. For this purpose, C57BL6J mice received an extract of YM or water for 4 months. Then, a moderate dopaminergic lesion was induced by intrastriatal 6-OHDA injection and treatments were continued until sacrifice at 2 or 30 days post-lesion. Antioxidant capacity (ABTS), oxidative damage (TBARS) and enzymatic antioxidant systems (GPx, GR, SOD) were measured in striatal tissue homogenates. Our findings indicate that animals treated with YM showed a positive modulation of enzymatic antioxidant systems, which may be associated with enhanced antioxidant capacity and reduced lipid oxidative damage. These findings support the role of YM as a potential neuroprotective agent against oxidative stress–related dopaminergic degeneration.

V-101

c-Fos expression in the rat brain following acute ibogaine administration

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Ibogaine is a psychedelic derived from the root bark of Tabernanthe iboga. Despite its therapeutic potential, this alkaloid remains poorly characterized, and its central nervous system (CNS) actions and mechanisms have yet to be fully elucidated.

This research explores the effects of a single ibogaine dose on the protein expression of the early immediate early gene c-fos, used as a marker of activity in the CNS of adult rats. Our study focused on regions involved in processes like reward, memory and behavioral inhibition, such as the medial prefrontal cortex (mPFC), nucleus accumbens (Acb) and amygdala (Amy).

Male adult Wistar rats received an intraperitoneal injection of ibogaine (40 mg/kg) or its vehicle. Behaviors associated with the serotonergic syndrome, characteristic of psychedelic substances, were quantified in an open field for 30 minutes. 60 min later brains were fixed and frozen to collect coronal sections from the regions noted above, and performed immunohistochemistry for the detection of c-Fos. c-Fos positive cells were quantified using ImageJ software.

The psychedelic induced distinctive behaviors, mainly high levels of tremor. Increased c-Fos immunoreactivity was observed in all analyzed mPFC and Amy subregions, while a treatment effect was only seen in the shell of the Acb.

These findings indicate the involvement of mPFC, Acb and Amy in ibogaine's early actions and provide relevant information on its differential effects in the CNS of the rat.

V-102

Beyond the brain: central and peripheral markers of stress-induced cocaine vulnerability in adolescent rats.

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Cocaine use disorder is a chronic condition marked by a shift from sporadic to compulsive use. Such progression depends on environmental and biological factors. These factors act not only on neurobiological mechanisms but also on intercellular communication systems such as exosome synthesis. Our team aims to study the biological mechanisms linked to the Social isolation (SI)-induced cocaine sensitization in adolescent rats, where we evaluate the role of the Wnt canonical pathway by measuring the levels of β-catenin in Prefrontal Cortex (PFC), Nucleus accumbens (NAcc), Amygdala (Amy), and Caudate-Putamen (CP). Previously, we showed that changes in the Wnt canonical pathway are associated with both cocaine sensitization and adolescent SI. Here, we examined if 5 days of SI (PND30-35) would induce cocaine sensitization on PND45 and changes in β-catenin levels of reward-related brain regions, in female and male rats. The results revealed that SI induced cocaine (5mg/kg i.p.) sensitization only in male rats ($p<0,05$). Also, isolated males showed lower β-catenin levels in the PFC, higher levels in the NAcc after cocaine ($p<0,05$). In Amy, cocaine increased the levels of β-catenin ($p<0,05$), while no changes were found in CP. The behavioural findings were replicated in a new cohort of animals, and plasma samples were taken to isolate exosomes. The size, quantity, and brain-derived protein cargo will be analysed as putative systemic biomarkers of SI-induced cocaine vulnerability.

V-103

Neuroprotective effect of Cannabidiol (CBD), modulated by autophagic pathways, in a progressive model of Parkinsonism

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Parkinson's disease (DP) is a neurodegenerative condition with a decline in processes essential for cellular homeostasis, such as autophagy. Cannabidiol (CBD) has been associated with autophagic activity. Therefore, the focus of this study was to observe the action of CBD in a progressive model of PD in rats. 72 Wistar rats were used and randomly divided into four groups: Control (CTL); Cannabidiol 5mg/kg (CBD); Reserpine 0.5 mg/kg (RES) and Reserpine 0.5 mg/kg + Cannabidiol 5mg/kg (RES+CBD). The animals received 15 subcutaneous administrations of RES or vehicle every 48 hours, and 30 intraperitoneal administrations of CBD or vehicle daily. Behavioral tests were carried out throughout the experiment Immunohistochemistry for tyrosine hydroxylase (TH), GFAP and western blot for α-synuclein (α-syn), TH, LC3-I, LC3-II and p62. The procedures were previously approved by the UFS Animal Research Ethics Committee (CEUA/UFS), under protocol no. CBD was able to minimize the deleterious effects of RES on motor symptoms, as well as neuroprotection in neurons of the nigrostriatal pathway of parkinsonian animals. We observed a reduction in GFAP+ levels in RES animals treated with CBD, as well as a reduction in α-syn levels. Classic markers of autophagy were decreased in parkinsonian animals treated with CBD. Cannabidiol was able to promote neuroprotection for motor and neurochemical symptoms through modulation of the autophagic pathway.

V-104

Multi-Functional Flavonoid Derivatives, Chalcones and Dibenzyl Acetones, as Innovative Therapeutic Agents for Neurodegenerative Disorders

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Neurodegenerative diseases (NDDs), such as Parkinson's and Alzheimer's, are progressive CNS disorders affecting movement, cognition, and other vital functions. Current treatments are mainly palliative and fail to halt disease progression. Given their multifactorial nature, NDDs require therapies that act on multiple targets simultaneously. Flavonoids—natural compounds found in plants—have shown potential in preventing and treating NDDs. Our goal is to develop flavonoid-derivatives capable of simultaneously modulating multiple pharmacological targets relevant to these disorders. We synthesized 18 new compounds (chalcones and dibenzyl acetones) and evaluated their physicochemical properties and targets interactions in-silico. No significant radical scavenging activity was observed (ABTS & DPPH assay). Cytotoxicity in vitro tested on SH-SY5Y culture cells (β-hexosaminidase assay) showed no significant toxicity up to 30 μM. Cholinesterase inhibition (100 μM, Ellman method) showed stronger effects of chalcones over dibenzyl acetones, with butyrylcholinesterase more inhibited than acetylcholinesterase. These results represent an encouraging first step toward the development of multitarget therapies for NDDs. While further studies are needed to expand, the observed in vitro activity highlights the potential of these compounds as promising leads. We aim to contribute to the discovery of more effective and safer treatments that could ultimately improve patients' quality of life.

V-105

Fighting cichlids: The role of estrogens in female aggression.

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Individuals in social hierarchies engage in agonistic encounters through aggressive behaviour, driving experience-dependent shifts in social status. Historically, aggression has been linked to males and androgens, while the role of estrogens remains understudied. Cichlid fish are ideal models for investigating neuroendocrine regulation and neural basis of agonistic behaviour due to their complex hierarchies and well-defined aggressive displays. The Neotropical cichlid Cichlasoma dimerus is particularly promising species to study the neuroendocrine modulation of female aggression, as both sexes show high levels of aggressive displays. The aim of the present work is to explore how estradiol may regulate aggressive behaviour using a pharmacological approach. Fish were isolated for 15 days before receiving either an intraperitoneal injection of the aromatase inhibitor fadrozole (10 ug/g) or the vehicle. After 45 minutes they were exposed to a neutral arena with a mirror on one lateral side. Behaviour was recorded for one hour, after which animals were euthanized for sex determination and brain sampling. Butting and mouth fighting were quantified and compared between groups. Behaviour was also tracked using Any-Maze, to analyse distance moved and swimming velocity. This work provides the first evidence on the role of estradiol in aggression in female cichlid fish, and lays the groundwork for future studies on estrogenic modulation and brain aromatase activity in female aggression.

V-106

Epigenetic regulation of sex differences by organizational effects of testosterone in the developing mouse brain

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Sex differences in the brain are typically attributed to sex chromosome complement, as well as to the organizational and activational effects of gonadal steroids. In rodents, males—but not females—undergo perinatal surges of testosterone that influence developmental processes such as cell death, neuronal survival, and dendritic growth; ultimately leading to morphological sex differences. We previously showed that the neonatal epigenome is sexually differentiated early in life: male mice exhibit higher expression of DNA demethylation enzymes in the prefrontal cortex (PFC) during the critical period of sexual differentiation, along with greater oxytocin receptor (OTR) expression at postnatal day (P)7 compared to females. These findings suggest that precise regulation of DNA methylation programs sexual differentiation of the rodent brain. Here, we investigate whether testosterone regulates early sex differences in the mRNA expression of Tet1-3, Gadd45a/b, and Tdg (enzymes involved in 5-methylcytosine removal), and whether this regulation correlates with OTR expression in the developing brain. Preliminary results suggest that neonatal testosterone treatment in females induces expression patterns resembling those of males. Thus, testosterone-dependent programming of DNA methylation and demethylation may contribute to sex-specific OTR expression and underlie broader epigenetic mechanisms of brain sexual differentiation.

V-107

Neuroinflammation Under Pressure: Supplementation with Omega-3 Fatty Acids in Spontaneously Hypertensive Rats

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Omega-3 polyunsaturated fatty acids (ω-3 FAs), including eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA), exert neuroprotective and antioxidant effects. Their pleiotropic actions involve lipid mediators that regulate immune and inflammatory responses in both the systemic and central nervous system.

We investigated the effects of early dietary supplementation with EPA and DHA on FAs composition in plasma and hippocampus. We also investigated inflammation, behavior and gut microbiota in spontaneously hypertensive rats (SHR). Male WKY and SHR rats received either a standard diet or ω-3 FAs (200 mg/kg/day) for 4 months (WKY+ω-3 and SHR+ω-3). We quantified fatty acid profiles and EPA/DHA-derived mediators, as well as Iba1 expression in the brain.

SHR+ω-3 rats exhibited increased ω-3 FA levels in plasma and hippocampus, along with higher plasma levels of pro-resolving mediators in plasma. Exploratory behavior differed between SHR (treated and control) and WKY, suggesting hypertension-related alterations, although supplementation had no significant effect on behavior. SHR+ω-3 rats also showed a decrease in microglia cells in the hippocampus. The results revealed differences in bacterial families between groups, suggesting a modulation of the microbiota.
We conclude that ω-3 FAs modified lipid profiles, induced changes in hippocampal microglia, and influenced gut microbiota on SHR rats, while the effects on behavior under these conditions remain inconclusive.

V-108

A unique cross-talk between Angiotensin type 1 and 2 receptors with inflammatory components determines their bioavailability in a time-dependent manner.

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The renin-angiotensin system (RAS) is involved in nerve regeneration and inflammation; therefore, its potential therapeutic effects have been evaluated in these fields. Most available drugs affect the system through the activity of type 1 and 2 angiotensin II receptors (AT1R & AT2R). However, little is known about how the bioavailability of these receptors changes in response to inflammation or by their pharmacological manipulation. We sought to evaluate the bioavailability of these receptors in these two contexts via a controlled in vitro approach. In primary cultures of postnatal rat dorsal root ganglion cells, we administered different treatments: inflammatory soup (IS), angiotensin-II (AngII), Azilsartan (AT1R-antagonist), or PD123319 (AT2R-antagonist) and examined 2 culture durations (2 or 3 days in vitro, DIV). Cultures were analyzed by qRT-PCR, WB and immunostaining. The addition of AngII to cultures increased the expression of both receptors at 2DIV. PD123319 reduced the levels of both receptors, while Azilsartan prevented only the increase in AT2R in neurons identified by β-tub III. Moreover, IS alone increased the levels of AT1R and AT2R in neurons. Conversely, at 2DIV, treatment with AngII, acting through both receptors, decreased the expression of pro-inflammatory receptors for IL6 and TNFα. Thus, we conclude that the RAS system components interact with each other and with inflammatory components, regulating their availability in a time-dependent manner.

V-109

Visually-guided defensive behaviors of triatomines

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Triatomines (kissing bugs) are hematophagous insects responsible for the transmission of Chagas disease. Due to their crepuscular and nocturnal habits, they have historically been considered as non-visual animals. However, some simple visual behaviors have been described in these insects (such as negative phototaxis and orientation to point light sources), and their compound eyes and ocelli are known to exhibit morphological changes in response to light, which would allow them to adapt to variable environmental conditions. Our overall objective is to understand how these insects perceive their visual environment and in which biological contexts vision is relevant to them. Here, we show that Rhodnius prolixus consistently responds to looming visual stimuli by either freezing or escaping, and it can rapidly alternate between these behaviors based on the ongoing stimulus information. We observed that escape responses are primarily evoked by stimuli that mimic the approach of a predator at a constant speed. Our results demonstrate a clear role of the visual system in mediating the defensive behaviors of triatomines. Similar to what has been observed in other highly visual arthropods, the probability of occurrence and intensity of these responses depend on the evaluation of the risk that the visual stimuli entail. Our study opens the question of how triatomines can solve the computational tasks supporting these visual capabilities with a reduced visual system.

V-110

“Hybrid platform”: a nanotechnological, cellular and genetic tool for peripheral nerve regeneration.

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Peripheral nerve injuries are afflictions in which full recovery is rarely achieved with current treatments. Our group focuses on nanobiotechnological strategies to promote nerve regeneration though a Wallerian degeneration model in rats promoted by a 30 second sciatic nerve crush. In this work we present a systemically transplanted “hybrid platform” composed of poly-lactic-co-glycolic acid nanocapsules (NC), loaded with magnetic nanoparticles (MNP) and functionalized with polyethyleneimine (PEI) for the transfection and magneto targeting of bone marrow mononuclear cells (BMMC) to the injury site. Both PLGA-NC and MNP were thoroughly characterized by specific techniques. To evaluate the “hybrid platform” feasibility, initial studies the “hybrid platform” were performed using BMMC transfected with NC:PEI:DNA (mock plasmid) loaded with MNPs and labeled with a fluorochrome. Seven days post-treatment behavioral and immunofluorescence assays for myelin basic protein (MBP) detection were performed. The results obtained show that the “hybrid platform” not only does not hinder the analgesic effects of BMMC but also was optimized by magneto targeting. Lastly preliminary results employing the “hybrid platform” adsorb with mRNA of native BDNF show a promising outcome and encourage us to further explore this nanotechnological approach as a potential therapeutic strategy and a tool to better understand the mechanism involved in nerve regeneration.

V-111

An interpretable dynamical model translating motor signals into song in canaries

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Song production in oscine birds is a complex learned behavior that arises from the interaction between the nervous system and peripheral devices. How the acoustical properties of the sound emerge from the delicate and fast control of several muscles in the syrinx and the respiratory system is not completely understood. Here, we recorded electromyographic activity of the syringealis ventralis (vS) muscle and air sac pressure in canaries (Serinus canaria) during singing. We developed a dynamical model with biologically interpretable parameters that translates these motor signals into sound. We show that the vS muscle not only modulates sound frequency but also gates airflow, thereby affecting sound duration and contrasting previous hypothesis in the field. Our results reveal that nonlinearities in the vocal apparatus enable the emergence of complex sounds from the relatively simple motor instructions measured. More broadly, this work advances efforts to achieve interpretable transformations from neural signals to vocal output.

V-112

Development, construction, and characterization of a neural recording system for small animals

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Songbirds are an established animal model for investigating sensorimotor integration, vocal learning, and motor control. Birdsong arises from the interaction of multiple neural nuclei with the respiratory system and the vocal organ. These systems coordinate to produce precise biomechanical gestures that give rise to vocal behavior. Telencephalic nuclei play a central role in generating motor commands that control the periphery, making the recording of neural activity during song production and processing of particular interest.

In this work, we present the development, construction, and characterization of a neural recording system for electrocorticography (ECoG) in small birds. ECoG enables the recording of cortical electrical activity with high temporal resolution and a superior signal-to-noise ratio compared with extracranial approaches such as electroencephalography (EEG). These signals are captured at the population level, reflecting the integrated activity of multiple units. Specifically, we designed a four-channel voltage follower circuit and a differential amplifier circuit to compare and evaluate the minimum requirements for such a system. The resulting circuits are lightweight and compact, making them well suited for use in small animals. This development is broadly applicable across small animal models and provides a versatile tool for future neurophysiological research.

V-113

Cholinergic Modulation of Dentate Gyrus Circuits: A Computational Model of SOM-Mediated Disinhibition

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The dentate gyrus (DG) of the hippocampus plays a key role in memory formation and associative binding in mammals. Neuromodulators are known to adapt circuit processing to enable plasticity, but the underlying mechanisms remain unclear. Previous work from our laboratory showed that endogenous acetylcholine (ACh) release enhances granule cell (GC) activity through a disinhibitory mechanism involving parvalbumin (PV) and somatostatin (SOM) interneurons. Here we developed a morphologically simple spiking network model (AdEx) of the DG, built upon prior DG models. The model includes GCs, mossy cells, PVs and SOMs, with the GC model extended to incorporate dendrites. Importantly, we distinguished two SOM subpopulations (HIPP and HIL) based on anatomical and physiological data. Model validation included intrinsic neuronal properties, firing rate–current curves, local field potential (LFP) spectra, and long-term potentiation simulations, consistent with published experiments. We then tested the hypothesis that strengthening SOM→PV connections mediates GC disinhibition. Simulations showed that enhancing HIL→PV synapses increased GC firing while reducing PV activity, whereas strengthening HIPP→PV connections failed to potentiate GCs within the tested range. Our results suggest that HIL interneurons are preferentially involved in cholinergic modulation of DG circuits, providing a mechanistic explanation for ACh-driven disinhibition and its role in learning-related plasticity.

V-114

Comparing speech biomarkers with standard neurocognitive indicators of Alzheimer's dementia

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A novel digital approach to detecting scalable markers of Alzheimer's dementia (AD) involves the automated analysis of word properties (WP), enabling both disease detection and prediction of cognitive performance and associated brain correlates. Yet, uncertainty persists regarding the clinical value of WP markers, since no study has compared their discriminative power with standard cognitive and neural measures.

We recruited 33 patients with AD and 33 healthy controls, who completed verbal fluency tasks, cognitive tests, and MRI/fMRI scans. Separate machine learning classifiers were trained using (i) WP features from the fluency tasks, (ii) MMSE score, (iii) TMT and digit span scores, (iv) MRI measures, and (v) fMRI measures. The best-performing model for each feature set was evaluated based on mean AUC.

WP classification performance (AUC = .85) was comparable ($p > .3$) to MMSE (AUC = .89), TMT/Digit (AUC = .78), and MRI (AUC = .89) features, and superior to fMRI features (AUC = .65, $p < .05$). The most important WP feature was word frequency, which showed a negative correlation with the volume of right prefrontal regions involved in executive processing.

Our speech approach can identify AD with performance comparable to gold-standard measures, driven by features linked to brain regions implicated in cognitive symptoms of AD. Overall, WP analyses seem non-inferior to standard diagnostic measures, highlighting their potential as a scalable and low-cost tool for dementia

V-115

Toward plug-and-play motor imagery-BCIs for rehabilitation: leveraging optimal transport for cross-subject adaptation

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Signal variability of electroencephalography (EEG)-based computer interfaces (BCIs), especially in motor imagery (MI) for rehabilitation, limits inter-subject generalization. Most MI-BCIs rely on intra-subject training, leading to long calibration sessions for each user. Even inter-subject transfer learning strategies, where large datasets are used to pretrain models, require substantial amounts of user-specific data to adapt and yield practical performance.

Here, we present cross-subject backward optimal transport (XS-BOT), which extends backward optimal transport for domain adaptation to inter-subject transfer. Leveraging cued labels, XS-BOT aligns the features' distribution of the target subject with the training features' distribution, minimizing the amount of adaptation data and avoiding model retraining.

XS-BOT was evaluated in two scenarios: cross-subject (multiple training subjects) and subject-to-subject (single training subject). For different base models, XS-BOT markedly outperformed the baselines using only 20 adaptation trials and three EEG channels. Cross-subject adaptation yielded accuracies similar to intra-subject setting, where a calibration session is needed. For subject-to-subject, results varied depending on the training subject, with cases that exceeded intra-subject results.

By enabling accurate decoding with minimal calibration, XS-BOT moves MI-BCIs toward plug-and-play use in rehabilitation, supporting immediate feedback and longer therapy.

V-116

Accuracy Is Not Enough: Divergent Brain–Model Similarities During Learning and Perturbation

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Deep neural networks (DNNs) are increasingly used as computational models of human visual processing, yet it remains unclear how their similarity to neural activity depends on the processes of learning and perturbation. In this project, we examine the evolving relationship between DNN representations and human brain activity during object recognition. Using EEG recordings of participants viewing briefly presented stimuli, we compare representational dissimilarity matrices (RDMs) derived from EEG with those extracted from networks such as ResNet-50 and AlexNet trained from scratch on ImageNet. We track similarity across training epochs, relating representational alignment to model accuracy. In parallel, we probe the robustness of these similarities by perturbing trained networks with multiplicative Gaussian noise in different layers, thereby altering their internal feature representations while monitoring the consequences for both accuracy and brain–model alignment. A central hypothesis is that the trajectory of similarity with respect to accuracy differs between the learning and perturbation regimes, pointing to distinct mechanisms underlying representational convergence and representational stability. This work aims to clarify whether training-driven and perturbation-driven changes in DNNs offer comparable insights into human vision, thereby informing the use of artificial networks as neuroscience models.

V-117

Biophysical modeling of the hemodynamic response induced by psychedelics

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Agonist drugs of the serotonin 2A receptor (psychedelics) are used with functional magnetic resonance imaging (fMRI) to investigate the neural correlates of conscious perception. A significant limitation is the indirect nature of the fMRI signal, which reflects changes in blood flow. It is therefore crucial to distinguish neuronal activity from confounding vascular effects of the drugs themselves. We investigated the hemodynamic response associated with the acute state induced by three serotonergic psychedelics (LSD, psilocybin, and DMT) using deconvolution algorithms applied to resting-state fMRI. We estimated the hemodynamic response function (HRF) under both drug and placebo conditions, finding significant global and regional changes in HRF latency. This suggests a serotonergic alteration in neurovascular coupling. To investigate the mechanisms, we implemented biophysical models based on mean-field descriptions of neuronal activity coupled through the structural connectome. Modeling neurovascular coupling produced interpretable parameters related to vascular biomechanics, supporting the hypothesis that observed fMRI changes under psychedelics could be partially explained by non-neuronal factors linked to vasoconstriction and dilation.

V-118

Neurochannelopathies simulation trough 2D simplifications of the Hodgkin-Huxley model

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Channelopathies affecting potassium chanels are a recognized cause of neuronal hyperexcitability, observed in certain pathologies. From a computational perspective, their study can be approached with the reduced Rinzel model, derived from the classical Hodgkin-Huxley model. In this work we analyze how the decrease in potassium chanels conductance modifies neuron membrane potencial using Rinzel non-linear equations. Numerical simulations are performed in Python using fourth order Runge-Kutta method. Besides, through an analysis from the dynamic systems perspective, changes between control case and pathological one can be interpreted as a shift in the phase plane of fixed points, nullclines and limit cycles. We found that in the pathological situation, bifurcations appears with lower values of external current. Thus, the studied model qualitatively reproduces neuronal hiperexcitability, and allows a mathematical interpretation. This enables linking ion chanelopatties with excitability phenomena.

V-119

Ultraslow entorhinal oscillation induces error in the estimated position during path integration

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Grid cells in the medial entorhinal cortex exhibit ultra-slow (<0.001 Hz) dynamics unfolding over seconds to minutes, as recently reported. The behavioral role of these oscillations remains unclear, particularly whether they influence position estimation from proprioceptive cues during path integration. Slow oscillations were found only in head-fixed mice on a running wheel, deprived of sensory input. Because these slow oscillations appear regardless of movement, we hypothesize that these dynamics only emerge under such restricted conditions, but are masked by sensory-driven activity during foraging. To test this hypothesis, we built a computational grid-cell model that forms a localized “packet” of neural activity representing the current position of a simulated rodent-like animal during 2D spatial navigation. Small synaptic changes due to synaptic plasticity induced a slow cyclical drift of the grid cell activity packet, even in the absence of sensory input, resembling the oscillations reported in vivo. This drift impaired path integration by causing position estimation errors, while also reactivating neural sequences consistent with recent trajectories. Notably, the slow oscillatory component was strongest when movement was constrained to one direction but was largely masked under unconstrained navigation. These results suggest that ultraslow entorhinal-like oscillations negatively affect path integration by increasing error in position estimation.

V-120

Fast nonparametric Bayesian framework for on-the-fly adaptive design optimization using discrete priors.

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In many neuroscience experiments, participant behavior can be modeled using monotonic functions. Here, we present a novel Bayesian algorithm for optimal experimental design, specifically developed for these types of function. The method adaptively selects the experimental condition that maximizes information gain on each trial, using Bayesian techniques to guide this process. Its computational efficiency makes it particularly suitable for real-time ("on the fly") paradigms. The algorithm is implemented in Python to facilitate its adoption by researchers without extensive programming or computational expertise.
By allowing the user to specify prior beliefs over the expected model, and offering a non-informative prior option in case the expected trend is unknown, the algorithm integrates domain knowledge into a principled Bayesian framework. We illustrate its performance through numerical simulations, showing faster convergence and improved estimation accuracy compared to traditional fixed or random sampling strategies. Furthermore, we validated the approach in an online behavioral experiment, where the method demonstrated robust empirical performance.

This tool offers a flexible and efficient alternative for adaptive experimentation in neuroscience, especially in contexts where time or participant engagement is limited and maximizing trial-by-trial information is crucial.

V-121

Development of a miniature fluorescence microscope with super-resolution

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Fluorescence microscopy is widely used in neuroscience to visualize cellular structures. However, commercial systems are usually voluminous, making them impractical for small animals such as rats. Although miniature microscopes like the Miniscope V4 (Zhang, 2018) allow in vivo neuronal recordings, the images obtained have aberrations and require heavy post-processing. This work aims to develop a miniature fluorescence microscope with improved optical quality and the possibility of super-resolution imaging through SUPPOSe (Toscani, 2019; Toscani, 2023; Martínez, 2024), addressing one of the main limitations of current devices.

The system is built using smartphone camera lenses, compact and low-cost optics that provide high-quality imaging. It is based on a Raspberry Pi 3B+ with Python programming and the OMNIVISION OV5647 sensor (1.4 µm × 1.4 µm pixels). Initial results include 10-bit RAW acquisition, sensor characterization (linearity and noise), and focal length measurements of several lenses. With a telescope-type configuration (f2/f1), magnifications greater than one were obtained. Resin 3D printing was used to fabricate precise parts and miniaturize the system.

Next steps include incorporating fluorescence to acquire images of fluorophores and applying image processing with SUPPOSe. This ongoing development aims to provide a portable and accessible tool for neuroscience, combining high-quality imaging with low cost and simple implementation.

Poster Session 2

S-001

Study of the genetic dosage of APP and its role in the Early Pathogenesis of Alzheimer's Disease in Down Syndrome

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Down syndrome (DS), caused by trisomy 21, almost invariably develops early-onset Alzheimer's disease (AD). The amyloid precursor protein (APP) gene, located on chromosome 21, is dosage-sensitive and a critical driver of AD, promoting β-amyloid (Aβ) overproduction and accumulation. However, the mechanisms by which APP overexpression triggers AD in DS remain unclear, as cognitive decline is not always correlated with Aβ levels. Aβ binding to full-length APP induces multimerization, neuronal dystrophy, and activation of the Go/Gβγ/p38 MAPK pathway, leading to dendritic atrophy. Elevated phosphorylated tau (p-tau), another AD hallmark, has been detected early in DS and is linked to dendritic and neuritic damage. p38-MAPK directly phosphorylates tau at Ser396/404, while Aβ oligomers activate CIP2A, inhibiting PP2A-mediated tau dephosphorylation, thereby increasing p-tau. Enhanced APP expression at the neuronal membrane in DS may amplify these cascades, contributing to cytoskeletal dysfunction. This project will investigate the role of APP dosage and signaling in regulating tau/p-tau and cytoskeletal structure in DS neurons, using induced pluripotent stem cells (DS-iPSCs) and isogenic APP-reduced variants. Results may reveal novel mechanisms by which APP dosage influences AD progression in DS.

S-002

Role of Serotonin in the locomotor response to heat stress in Drosophila melanogaster

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The vital functions of organisms are sustained through the interaction of specialized structures coordinated by strict signaling mechanisms. This balance can be disrupted by internal or external stressors, triggering the release of neurotransmitters essential for adaptation. Among these, serotonin (5-HT) has a poorly characterized role in adaptive responses to stress. In this work, we used Drosophila melanogaster, an organism lacking thermoregulatory capacity, as a model to investigate the role of serotonin in the locomotor response to thermal stress. Control flies exposed to increased temperature, either as an acute heat shock (AHS) or as a gradual increment (GTI), exhibited elevated locomotor activity. In contrast, we used flies deficient in components of the serotonergic pathway including tryptophan hydroxylase, the serotonin transporter (SerT), and receptors (5-HT1A, 5-HT1B, 5-HT2A, 5-HT2B, and 5-HT7) under both protocols (AHS y GTI) and analyzed their locomotor patterns. These preliminary results support a role for serotonin and it signaling in the adaptive response to thermal stress.

S-003

Neuron-derived vesicles as therapeutic vectors: modulation of M6a content in EVs and their administration in a chronic stress model

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Chronic stress contributes to the development of mood disorders such as depression. Current treatments are often limited in efficacy and show adverse effects. We propose an alternative therapeutic strategy based on extracellular vesicles (EVs). Due to their small size and high biocompatibility EVs can facilitate biomolecule delivery to the brain. As a potential biomolecule of interest, we propose the neuronal protein M6a, which is involved in neuronal connectivity and whose levels are altered by chronic stress.

EVs were obtained from the neural cell line HT22 through differential centrifugation and characterized by TEM and nanoparticle analysis (NTA). EVs were loaded with M6a-GFP or GFP (control) plasmids. The loading was confirmed by plasmid DNA extraction.
In a murine model of chronic stress, treatment with loaded EVs partially reversed weight loss and restored hippocampal M6a levels, although no significant behavioral changes were observed. These effects were independent of the loaded plasmid, suggesting a possible neuroprotective role of endogenous factors present in HT22-derived EVs.

Current work focuses on obtaining EVs carrying M6a-GFP through plasmid overexpression in HT22 cells. Once such EVs are isolated, their function will be evaluated through the transference of M6a to recipient cells in HT22-3D spheroids and in differentiated HT22 cells.
We hope to obtain HT22-derived EVs with an increased proportion of M6a to test their neuroprotective effect in vivo.

S-004

Spatiotemporal alterations of Dense-Core Vesicle transport in a chromaffin cell model of Huntington's Disease

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Huntington's disease (HD) is a hereditary neurodegenerative disorder caused by an expanded poly-glutamine stretch in the Huntingtin protein (Htt). HD animal models show decreased dense-core vesicle (DCV) secretion, and HD patients feature symptoms associated with altered neuropeptide signaling. However, Htt biological functions and the basic mechanisms by which mutated Htt (mHtt) affects the regulated-secretory pathway remain unclear.

In alignment with evidence suggesting a role of Htt in vesicle trafficking, we analyzed DCV mobility in chromaffin cells, a well-known cellular system for neurosecretion. Using confocal imaging and bioimage analysis tools, we previously reported that Htt and mHtt overexpression modified DCV transport regimes, showing an increase in motility and in confined motion, respectively. To study this further, we evaluated DCV mobility across the cell to map DCV motion at defined regions at different distances from the plasma membrane. Spatial analysis of control cells revealed differential distribution of DCV specific motion, with an increase in motility near cell periphery upon K+ stimulation. Htt overexpression also increased peripheral DCV motility, but did not increase it further upon stimulation. Finally, mHtt expression did not affect DCV motility at the periphery but increased immobile and caged DCV inside the cell. This data contributes to our understanding of Htt biology, showing the importance of Htt activity along the DCV trafficking pathway

S-005

Oligodeoxynucleotide IMT504: Role in cerebral cortex remyelination after experimental demyelination

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Demyelination is a pathological process characterized by myelin loss from around axons, while remyelination is the repair response through the restoration of myelin and the resolution of functional deficits. Multiple sclerosis is a high-incidence inflammatory demyelinating disease in which remyelination frequently fails. IMT504 (IMT) is a non-CpG oligodeoxynucleotide consisting of 24 nucleotides and characterized by 2 specific PyNTTTTGT sequences. On the basis of IMT immunomodulatory effects and regenerative properties, and our previous results showing its beneficial effects on neuroinflammation and remyelination in the corpus callosum of cuprizone (CPZ)-demyelinated rats, this work aims to study IMT role in microglial and oligodendrocyte (OL) lineage cell populations in the cerebral cortex (Ctx). We subcutaneously administered IMT every day for five days before CPZ withdrawal. Brain samples were then analyzed 0 (T0), 3 (T3), 7 (T7) and 10 (T10) days after CPZ withdrawal. Immunohistochemical results show that IMT did not change the population of Iba1+ microglial cells per area or APC+/Sox10+ OLs at any of the times analyzed. However, IMT produced a significant increase in PDGFRα+ OL progenitor cells at T3 and in MAG+ mature OLs at T7 as compared to CPZ-treated rats injected with saline solution. These findings support potentially beneficial properties of IMT in the myelin repair process of demyelinated Ctx lesions.

S-006

Study the Role of Fluoxetine as a Global Sumoylation Inhibitor and Its Possible Use in the Treatment of Huntington's Disease

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SUMOylation is a post-translational modification whereby the SUMO peptide is covalently attached to lysine residues on target proteins influencing essential cellular processes. It plays pivotal roles in neurobiology and has been increasingly implicated in neurodegenerative diseases. In Huntington's Disease (HD), for instance, SUMOylation contributes to the aggregation of mutant huntingtin (mHTT), and inhibition of this modification can mitigate disease-like traits and reduce mHTT aggregation, marking SUMO pathways as therapeutic targets.

In our laboratory, we have shown that the SSRI antidepressant fluoxetine functions as a global SUMOylation inhibitor, both in vitro and in vivo. To investigate its effect on mHTT aggregation, we transfected HEK293T cells with SUMO1, SUMO2, and either HttQ25-mCherry (non-pathogenic control) or HttQ74-mCherry (pathogenic mHTT). Then cells were treated with either vehicle, fluoxetine (1 µM), or the specific SUMOylation inhibitor ML792 (0,02 µM). We employed immunofluorescence and confocal microscopy to visualize aggregate formation, alongside Western blot analysis to quantify global SUMOylation levels.
Our results demonstrate that fluoxetine reduces the formation of mHTT aggregates, replicating the anti-aggregation effects observed with ML792. Importantly, overexpressing either SUMO1 or SUMO2 abolishes fluoxetine's protective effect, confirming that its anti-aggregation activity is specifically mediated through the suppression of SUMOylation.

S-007

Molecular mechanisms controlling functional development during adult neurogenesis

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The dentate gyrus of the hippocampus generates adult-born granule cells (abGCs) throughout life, which integrate into pre-established neural circuits. Recently, we performed single-nuclei RNAseq for transcriptomic profiling of distinct abGCs cohorts. Combining differential gene expression, pseudotime trajectory and transcription factors (TFs) regulon analysis, we identified four cellular states: quiescent radial glia-like cells, proliferative progenitors, immature abGCs and mature abGCs. We propose that transitions between these states are driven by specific transcriptional regulators controlling the main features of each developmental stage. Thus, we manipulated the expression of key TFs as a strategy to unravel the molecular mechanisms underlying differentiation, maturation, integration and function of abGCs. Foxo1 is a TF predominantly expressed in quiescence and mature abGCs stages, but its function in establishing and maintaining the homeostasis of the mature neuronal phenotype is unknown. To address this question, we overexpressed Foxo1 in abGCs using retroviral constructs and analyzed neuronal morphology using confocal microscopy. Ten-day old cFoxo1-abGCs exhibited reduced dendritic length and branching points compared to control abGCs, which might lead to functional alterations. Other candidate genes highlighted by our regulon analysis are currently being manipulated to unveil their role in orchestrating neuronal state dynamics along abGC maturation.

S-008

Potential role of ATF6 activity in BDNF-induced biological functions

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The unfolded protein response (UPR) is a homeostatic signaling pathway activated by the accumulation of misfolded or unfolded proteins in the endoplasmic reticulum (ER). In addition to its protective function, in recent years the UPR has been described playing essential roles during normal development, particularly in response to increased demands for protein folding. Several UPR effectors exhibit dynamic temporal and spatial expression patterns that correlate with milestones of the central nervous system (CNS) development. Notably, UPR activity has been shown to be specifically induced during dendrite development. Mammalian cells have three ER stress sensors, ATF6, IRE1alpha, and PERK, all of which are present in the ER of dendrites in primary mouse neurons. Although ATF6 deletion impacts embryonic brain development, its role in axonal growth and dendrite morphogenesis remains incompletely understood. Here, we investigated the requirement for basal ATF6 activity in the regulation of axonal growth, dendrite morphology, and spine density in primary hippocampal neurons treated or not with the neurotrophin BDNF.

S-009

Development of an experimental model that replicates the disease specific structures of aS filaments in brain's patients with multiple system atrophy.

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A plethora of evidences associates structural dysfunction of the protein alpha-synuclein (aS) and self-assembly into filaments with the neuropathology of Synucleinopathies such as Parkinson disease (PD) and Multiple System Atrophy (MSA). The protein exhibits a high potential to form polymorphic fibrils. Consistently, high-resolution structural determination of aS fibrils has unveiled a variety of polymorphic structures of either in vitro fibrils or ex vivo fibrils extracted from the brain of patients. Recently, structural polymorphism of aS fibrils has been associated with distinct Synucleinopathies. Interestingly, a characteristic shared by all post-mortem aS filament structures is the presence of non-proteinaceous molecules in assembled aS, indicating that chemical ligands may be involved in the assembly of aS fibrils in patient's brain. These evidences highlight the complexity of the aS aggregation process and emphasizes the importance of developing conditions that lead to a better understanding of the structural and molecular basis behind aS assembly. In this work we set-up an experimental model that replicates the disease specific structures of aS filaments extracted from patients with MSA. These kind of experimental models will be invaluable for gaining a better understanding of disease, and thus for developing safe and effective mechanism-based therapies.

S-010

Cannabinoids and Autophagy: CBD as a Regulator of Tau Neurotoxicity in Drosophila Melanogaster and N2a Cells

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Tauopathies are characterized by neurotoxic tau accumulation and alterations in autophagy. Previous studies suggest that cannabidiol (CBD) can induce autophagy in cell culture models. Here, we used N2a neuroblastoma cells and Drosophila melanogaster to evaluate the impact of cannabinoids on autophagy and tau-induced neurodegeneration.
Our initial screening in N2a cells involved CBD, CBDA, THCA, and THC. Our results show that CBD consistently induced autophagy, and THCA exhibited a potential autophagy-inducing effect. Conversely, CBDA inhibited autophagy, while THC had no detectable effect. Consequently, we chose CBD as the primary candidate for further investigation into its efficacy against tau neurotoxicity.
We are assessing if CBD-induced autophagy reduces tau neurotoxicity. To this end, we evaluated in N2a clones that stably express human tau whether CBD treatment reduces its accumulation. In parallel, in a Drosophila model, we expressed human tau in neurons and eyes to evaluate neurodegeneration phenotypes. We optimized methods for quantifying the rough eye phenotype, assessing locomotor function and detecting tau and phosphorylated tau via Western blot. Additionally, a bioinformatic analysis is underway to identify potential molecular targets of cannabinoids in the autophagy pathway.

Our results provide evidence for CBD's role as a selective modulator of autophagy, supporting its further exploration as a therapeutic strategy for tau-related neurodegenerative diseases.

Cellular and Molecular Neurobiology

S-011

Effects of borosilicate-based bioactive glasses on dopaminergic neurons regeneration

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Bioactive glasses (BGs) have emerged as biomaterials capable of modulate neuroregeneration due to their ability to release ions with therapeutic effects. Boron (B) has shown neuroprotective and anti-inflammatory properties, making borosilicate BGs a promising alternative to promote neuroregeneration. Parallelly, there is growing interest in alternative models that reduce the use of vertebrates in neuroscience. The planarian Schmidtea mediterranea has been studied as a model organism in neuroregeneration due to the presence of orthologs of enzymes such as tyrosine hydroxylase (TH), that confirms evolutionary homology of metabolic pathways. In this study we used BGs of the system Na2O−B2O3−SiO2 with two different B2O3 concentrations (18.75 and 37.5 mol%). Ionic dissolution products (IDP) were obtained by incubation of BG microparticles (300 – 425 µm) in planarian artificial medium in an orbital shaker for 24-72 h. The soluble ions (B, Si and Na) leached from BGs were determined by ICP-OES. 4-6 mm planarians were amputated at pre and posfaringeal level and the resulting trunks were soaked in the IDP at 20±1°C. At 7 d post amputation, regenerating planarians were processed to determine the expression of TH by in situ hybridization. Planarians exposed to IDP with non-toxic concentrations of B exhibited an increase in the expression of TH, whereas IDP containing higher B concentrations reduced their expression and altered the topographic organization of dopaminergic neurons.

S-012

Relevance of the GTPase Rab35 in neurodevelopment and its involvement in Down syndrome

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The cerebral cortex is one of the most evolutionarily complex structures of the central nervous system (CNS), requiring tightly coordinated steps such as neuronal differentiation and migration from progenitor zones in the ventricular area to the cortical plate. Small GTPases play key roles in these processes by regulating intracellular trafficking, cytoskeleton dynamics, and the transport of adhesion molecules and membrane receptors essential for neurodevelopment. Rab35, a GTPase involved in endosomal and exosomal trafficking, has been identified as an important regulator of neurite growth. This project aims to investigate the role of Rab35 in neuronal differentiation and migration, and its implications in Down syndrome (DS), a condition characterized by altered neurodevelopment and defects in endo/exosomal pathways. Specifically, we will examine whether Rab35 regulates the trafficking and recycling of neurodevelopmental proteins such as the amyloid precursor protein (APP), which may be affected in DS. To address this, we will use primary mouse neurons, DS-iPSC-derived neuronal cultures, and an integrated model combining in utero electroporation (IUE), organotypic cultures, and in situ analyses with super-resolution microscopy.

S-013

GPM6a and Neuroplastin Interaction: A Negative Modulator of Neuronal Morphology

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The molecular mechanisms governing neuronal morphogenesis remain largely unknown. Among these, neuronal membrane glycoprotein GPM6a is known to promote differentiation and synaptogenesis, and its dysregulation is linked to neuropsychiatric disorders. Given that the extracellular loops (ECs) of GPM6a are critical to its function, previous studies from our laboratory identified that the cell adhesion molecule neuroplastin (NPTN) co-immunoprecipitates with GPM6a using its ECs as bait. This study investigates the functional association between GPM6a and NPTN in hippocampal neurons and cell lines. Endogenous NPTN and GPM6a colocalize at the neuronal membrane across various developmental stages. Co-overexpression of both proteins inhibited neurite extension, reduced the number of neurites per cell, and decreased filopodia formation, indicating a non-cooperative interaction during neuronal development. In HEK293 cells, the NPTN ectodomain and GPM6a-ECs interacted in a trans configuration, inducing cell aggregation. This aggregation was inhibited by adding a calcium chelator (EGTA) or GPM6a-neutralizing monoclonal antibodies. Importantly, an NPTN isoform lacking an IgG domain (NPTN55) did not aggregate with GPM6a, nor did a GPM6a mutant deficient in EC2 folding aggregate with wild-type NPTN. Collectively, these findings suggest that co-overexpression of GPM6a and NPTN acts antagonistically and support a functional interaction between GPM6a and NP65 via their extracellular domains.

S-014

Antagonistic Monoaminergic Control of State-Dependent Foraging in C. elegans

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The perception of food as more rewarding after deprivation is an evolutionarily conserved phenomenon, yet its underlying neural mechanisms remain poorly understood. We dissect this process using the nematode C. elegans, a model organism with a well-defined nervous system and conserved neurochemistry that provides universal insights into state-dependent behaviors.

In C. elegans, fasting triggers an enhanced slowing response upon food re-encounter, ensuring efficient exploitation of the source. We demonstrate that this behavior is governed by an antagonistic relationship between serotonin (5-HT) and tyramine (TA, the invertebrate analog of noradrenaline). The fasting-induced decline in TA disinhibits serotonergic signaling, which primes serotonergic neurons for a heightened response. Consequently, upon encountering food, these neurons release a surge of 5-HT that dramatically slows locomotion to ensure efficient feeding. This mechanism is confirmed in TA-deficient mutants, which exhibit hyperactive serotonergic neurons and an exaggerated slowing response. We further establish that TA directly inhibits the NSM neuron through the activation of two adrenergic-like GPCRs.

This defines a neural switch where fasting reduces inhibitory monoamines, disinhibiting 5-HT to ensure feeding. Conservation of these neurotransmitters suggests similar principles govern state-dependent decisions across species, providing insight into foraging and appetite.

S-015

Dissecting the Contribution of Etv5 to Postnatal Development of the Cerebral Cortex

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The development and refinement of cortical circuits during the postnatal period depend on tightly coordinated transcriptional programs that regulate neural progenitor proliferation, neuronal differentiation, survival and maturation of postmitotic neurons. While the transcription factor Etv5 has been implicated in diverse neurodevelopmental processes, its specific contribution to postnatal cortical maturation remains poorly understood.
In this study, we investigated the expression dynamics of Etv5 in the cerebral cortex during early postnatal development. In vivo analysis of cortical layers revealed no significant differences in the distribution of deep-layer (Ctip2$^+$) or upper-layer (Satb2$^+$) neurons between wild-type (WT) and conditional Etv5 knockout (cKO) mice, suggesting that Etv5 is not essential for initial layer specification. However, in primary cultures of mature cortical neurons, Etv5 expression increased progressively during in vitro maturation and was further upregulated in response to brain-derived neurotrophic factor (BDNF). These findings indicate that Etv5 may play a modulatory role in activity-dependent transcriptional programs during postnatal cortical development, rather than in early neuronal fate specification.

S-016

Lithium effects on intracellular trafficking, dendritic architecture and fear behavior on BDNF Val66Met carriers

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The Brain-Derived Neurotrophic Factor (BDNF) Val66Met polymorphism is a common genetic variant associated with neuropsychiatric disorders. This substitution of valine for a methionine in the BDNF prodomain (pBDNF Met) induces structural and functional impairments in the central nervous system (CNS), including growth cone retraction, altered dendritic morphology, circuitry remodelling, functional deficits measured by calcium fiber photometry, and behavioral alterations. However, the cellular mechanisms driving these effects remain poorly understood.

Our group recently showed that pBDNF Met interacts with zinc (Zn^{2+}) to form large multimers which are required for pBDNF Met effects. As both Zn^{2+} and pBDNF Met coexist in the secretory pathway, we hypothesize that this interaction alters intracellular trafficking, contributing to the neuronal defects. Using an in vitro synchronization system in cultured hippocampal neurons, we observed that pBDNF Met impairs vesicular trafficking in neuronal processes.

Interestingly, preliminary findings suggest that lithium can prevent Zn^{2+}-induced pBDNF Met multimerization. Therefore, we are currently testing whether lithium can also rescue trafficking defects, dendritic alterations, and fear extinction impairments induced by pBDNF Met. Our results indicate that lithium restores dendritic complexity, and ongoing experiments aim to determine whether it also prevent intracellular trafficking defects.

S-017

MicroRNA-mediated regulation of ion channels in acute inflammatory pain: evidence from the LPS model

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Animal models of inflammatory pain enable the study of mechanisms linking inflammation and nociception. In this study, we employed the subplantar LPS injection model in mice to explore post-transcriptional regulation of ion channel expression. Consistent with our previous presentations (SAN 2023, 2024) and with earlier findings from the formalin model, LPS induced local inflammation, sex-dependent differences in mechanical sensitivity, and segmental changes in ion channel protein levels accompanied by ERK MAPK phosphorylation.

Here we analyzed the mechanisms responsible for these changes. Despite stable mRNA levels in paw tissue and dorsal root ganglia, we detected significant alterations in microRNA miR-485-5p, previously described as a post-transcriptional repressor. These changes paralleled the lumbar L3–L5 gradient observed at the protein level, supporting a microRNA-dependent mechanism for ion channel upregulation in a segment- and sex-specific manner.
Together, these results extend previous work by identifying miR-485-5p as a potential mediator of ion channel regulation in acute inflammatory pain, reinforcing the importance of post-transcriptional control in physiopathological models of nociception.

S-018

CDK5 deficiency in human induced pluripotent stem cells-neurons reveals secretome dysregulation and reduced adhesion

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CDK5 is a key regulator of neuronal homeostasis, and its dysregulation has been associated with altered secretory pathways. Here, we investigated how CDK5 knockout (KO) affects the neuronal secretome using FN2.1 human induced pluripotent stem cells (hiPSC)-derived neurons. Both wild-type (WT) and CDK5-KO FN2.1 lines were successfully differentiated, expressing the neuronal markers TUJ-1, MAP2, and MAP5. Conditioned media collected after 18 h of starvation were analyzed by MS/MS proteomics, and 48 differentially expressed proteins were identified using the Limma package in R. Gene Ontology analysis revealed enrichment in processes related to focal adhesion and actin cytoskeleton regulation. Consistently, cell-attachment assays demonstrated a significant reduction in adhesion of CDK5-KO FN2.1-derived neurons. Notably, the CDK5-KO secretome showed significantly increased levels of MAPT (TAU) and TDP-43, which were further confirmed in total cell lysates by Western blot. Together, these findings suggest that CDK5 deficiency reshapes the neuronal secretory profile, weakens cell adhesion, and increases the levels of TAU and TDP-43, ultimately disturbing neuronal homeostasis.

S-019

Angiotensin-II regulates the expression of the K2P channel TWIK1 in cultured dorsal root ganglion neurons Through Its AT1 and AT2 Receptors.

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TWIK1 is a potassium leak channel of the K2P family that contributes to the resting membrane potential and excitability in a variety of neurons. It is expressed in the dorsal root ganglion (DRG), where it participates in pain processes, although its regulation remains poorly understood. It is believed that Angiotensin-II (Ang-II) can have analgesic effects in chronic pain models by the activation or inhibition of its main receptors, AT1R and AT2R, possibly involving regulation of K2P channel expression. Thus, we evaluated whether selective inhibition or activation of AT1R and AT2R modulates TWIK1 expression in primary DRG neuron cultures from P6 rats. Cells were maintained in vitro for 1 and 2 days under different conditions: Control, Ang-II, Ang-II + Azilsartan (AT1R antagonist), Ang-II + PD123319 (AT2R antagonist), and Ang-II + Azilsartan + PD123319. Both TWIK1 and AT1/AT2R expressions were assessed by qRT-PCR, immunofluorescence, and Western blot. Ang-II significantly increased TWIK1 expression at 1 DIV, an effect mainly mediated by AT2R. At 2 DIV, however, TWIK1 expression was markedly reduced across all groups. Moreover, Ang-II treatment altered the expression of its own receptors, suggesting a feedback regulation of the RAS in DRG neurons. These findings demonstrate that Ang-II regulates TWIK1 expression in sensory neurons, providing a previously unknown link between angiotensinergic signaling and neuronal excitability in models of neuropathic pain.

S-020

Activity-Gated Rescue of Delayed Neuroblast Maturation in the Aging Hippocampus

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Adult hippocampal neurogenesis is a conserved, multi-stage process essential for cognitive flexibility and memory. In young animals, adult-born granule cells (aGCs) progress through a well-orchestrated developmental trajectory that lasts for about 10 weeks. Aging significantly reduces the rate and speed of neurogenesis. To uncover the molecular stage-specific effects of aging on the generation of new neurons, we combined permanent lineage tracing of aGCs with single-nucleus RNA sequencing (snRNA-seq) in aged mice. This strategy allowed us to generate a temporally resolved transcriptional atlas of neurogenesis in the aging hippocampus. Our analysis revealed a pronounced accumulation of neurons at a specific postmitotic neuroblast stage. Remarkably, this population was highly responsive to behavioral stimuli: voluntary running reduced neuroblast accumulation to re-engage developing neurons towards later stages along the differentiation trajectory. These findings position postmitotic neuroblasts as critical regulatory nodes in the neurogenic sequence, poised to integrate pro-maturational signals. This work offers a high-resolution framework for dissecting the mechanisms of neurogenic decline and points to potential strategies for rejuvenating hippocampal plasticity in aging.

S-021

Stress resilience: differences in behavioral response strategies and gene expression of neuronal receptors in the PFC of susceptible and resilient mice

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Gestational stress alters the trajectory of brain development, leading to changes in cognitive and socioemotional functions. However, individual responses to stress vary, with some exhibiting resilience while others display heightened vulnerability. To investigate the mechanisms underlying stress resilience, we employed a gestational movement restriction model in which pregnant CF1 females were subjected to a restraint protocol from gestational day 10 to 19.

Offspring were evaluated with the Splash Test, where latency to groom was used to distinguish between resilient (RES) and susceptible (SUS) mice. Behavioral responses were further assessed using the Forced Swim Test (FST). SUS mice exhibited significantly more passive and less active swimming compared to controls (CT), while no significant differences were observed between RES and CT.

To explore potential molecular mechanisms of stress adaptation, RNA was extracted from the PFC of adult animals. Both RES and SUS groups showed significantly reduced expression of the Grin2b gene encoding an NMDAR subunit, and SUS mice exhibited a trend towards increased expression of the Gabrb3 gene encoding GABA-A subunit. A positive correlation was found between oxytocin receptor expression and the passive swimming time in the FST in RES animals. These findings enhance our understanding of the mechanisms underlying stress resilience and may guide future strategies to promote adaptive responses to adversity.

S-022

Audiovisual Stimulation at 40 Hz Induces Cell Type–Specific Transcriptional Changes in the Aged Dentate Gyrus

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Neurogenesis persists in the dentate gyrus throughout life, but declines sharply with aging. We have recently shown that 40 Hz audiovisual stimulation (AuViS) enhances activity in the aged dentate gyrus and promotes neurogenesis in middle-aged mice. These effects may be related with changes within the neurogenic niche, yet the underlying cellular and molecular mechanisms remain unclear. We hypothesized that AuViS-induced cellular modifications are driven by transcriptional changes in the aged neurogenic niche. To test this idea, we performed single-nuclei RNA sequencing of the dentate gyrus from 8-month-old mice exposed to 6 weeks of AuViS or control conditions. Notably, the most significant change was the transcriptional upregulation of tight junction–related genes such as Cldn5 in endothelial cells, suggesting enhanced blood–brain barrier (BBB) integrity. In contrast, neuronal populations displayed only subtle changes in gene expression. These results reveal that AuViS elicits cell type-specific transcriptional responses and point to BBB remodeling as a potential mechanism contributing to enhanced neurogenesis in the aged dentate gyrus.

S-023

Scopoletin and its amino acidic-conjugates have pre-clinical therapeutic activity against inflammatory pain

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Inflammatory pain involves the release of cytokines that sensitize nociceptors peripherally. Many synthetic drugs have been used to treat this condition; however, they are mired with a high incidence of side effects and risk of co-morbidities. For this reason, we explored new bioactive compounds characterised by different polarity and chemical structure that could potentially enhance their pharmacological action and bioavailability. Two molecules were obtained through chemical synthesis: scopoletin-cysteine and scopoletin-tryptophane, belonging to the coumarin family. We evaluated the analgesic and anti-inflammatory activity of pure scopoletin and its two derivatives (1 µg/mL), administered topically once daily for 10 days in a model of inflammatory pain induced by CFA (Complete Freund's Adjuvant) in 5-month-old Wistar rats both male and female. We assessed the responses to mechanical and thermal stimuli, and the extent of paw oedema. Specific histochemical staining was performed to evaluate neutrophil infiltration, mast cells, and collagen deposition. Levels of pro-inflammatory cytokines and the antioxidant system were determined via qRT-PCR. We examined the expression of nociceptive markers in skin terminals. Scopoletin-cys and scopoletin-trp exhibited anti-allodynic analgesic activity from day 3 of treatment. Scopoletin showed only anti-edematous activity. Scopoletin-cys led to changes in IL-6 and TNF-a levels and a reduction in mast cell infiltration.

S-024

Searching for sleep-like behavior in the diel activity patterns of a semiterrestrial crab

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Sleep is present in all animals studied, suggesting an early evolutionary origin. This state of relative disconnection from the external world is vital for multiple physiological and behavioral processes. Sleep is regulated by two processes: homeostatic pressure, which ensures daily balance, and the circadian rhythm, which aligns sleep with the day–night cycle. Although sleep has been studied in many models, including invertebrates, it has never been investigated in intertidal animals, which experience a third pressure guiding their activity/rest rhythms: the circatidal rhythm, corresponding to the rise and fall of the tides. One example is the crab Neohelice granulata, which inhabits mudflats. In this project, we analyzed their activity/rest cycle by filming crabs in the laboratory for several days under natural light and constant water levels. They showed a crepuscular activity pattern, with two peaks near dusk and dawn respectively, and a prolonged midday/early afternoon rest phase. During immobility bouts, especially in this prolonged rest phase, crabs often adopted a characteristic posture: flattened cephalothorax, retracted claws, and a preference for arena corners. We also observed changes in the organization (but not the total duration) of resting periods between previously isolated and socialized crabs, consistent with sleep recovery following social interaction.

S-025

Chronic sleep loss and its effects on Immune responses in an experimental fly model

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This research explores the relationship between sleep and immune function, hypothesizing that adequate sleep is essential for a properly functioning immune system. While acute sleep deprivation often elicits compensatory and adaptive responses indicative of behavioral resilience, chronic sleep restriction—commonly arising from prolonged exposure to environmental stressors such as predation, food scarcity, and social instability—may induce physiological alterations detrimental to health. To investigate these dynamics, the study utilizes Drosophila melanogaster as an experimental model due to its biological tractability and conserved sleep and immune mechanisms shared with mammals. Behavioral responses to infection were assessed through manual thoracic injections with Staphylococcus aureus, while sleep and activity patterns were monitored using the Ethoscope platform, which employs real-time tracking and machine learning algorithms. The platform also facilitates targeted sleep deprivation, enabling precise examination of how sustained sleep loss influences immune outcomes. In order to test the impact of sleep loss on immunity, we analyzed different parameters such as lifespan, sleep, activity and bacterial load.

S-026

When rest meets screens: study of smartphone use and sleep in Toba/Qom communities of Northern Argentina

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Before electricity, human activity followed the natural light–dark cycle, with sleep mostly at night. Electricity extended wakefulness into the evening and reshaped sleep patterns. Our studies on isolated Toba/Qom native communities in Argentina, offer a unique opportunity to explore the gradual shift in sleep patterns as they gain access to electricity and mobile devices.

Based on over 10,000 nights of sleep recorded via actigraphy and sleep diaries, we monitored changes in sleep habits over more than a decade. This longitudinal data reveals a significant delay in sleep onset and reduced total sleep since electricity was introduced in rural communities (Casiraghi et al, under rev). In this follow-up study, we aimed to analyze the role of smartphone use in Toba/Qom sleep patterns.

Our results show that smartphone use correlates with delayed sleep onset (13.4 min, $p<.001$) and shorter sleep duration (21.6 min, $p<.001$). This appears to have two causes: 1) a phase shift of the circadian clock, revealed by delayed melatonin onset in smartphone users; and 2) an increased procrastination of the time of sleep, with later sleep times on nights with reported smartphone use. These findings add to evidence that mobile devices can modulate sleep and circadian rhythms by both physiological and cultural ways, especially in communities newly exposed to technology.

Our results underscore the importance of studying sleep in underrepresented populations undergoing rapid technological change.

S-027

Gut Tumor-Induced Hyperplasia Alters Sleep Regulation in Drosophila

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Sleep disturbances are increasingly recognized as a major health issue in industrialized societies, with profound social and economic costs. Up to 60% of cancer patients experience some form of sleep disorder, often linked to poorer quality of life and prognosis. Epidemiological evidence also suggests that inadequate sleep increases the risk of colorectal cancer, pointing to a connection between sleep regulation and intestinal physiology. Yet, the mechanisms linking gut dysfunction and sleep remain poorly understood.

To study these interactions, we induced epithelial hyperplasia in the adult fly midgut and monitored sleep behavior. Mated females with intestinal hyperplasia exhibited a marked increase in daytime sleep, driven by longer sleep bouts, whereas males showed no significant changes, consistent with their weaker hyperplastic phenotype. Hidden Markov model analysis further revealed that this additional sleep corresponded to deep rather than light states, supporting the idea that intestinal hyperplasia produces both qualitative and quantitative changes in sleep regulation.

Together, these results highlight the active role of intestinal epithelial integrity in shaping sleep dynamics. Future experiments will expand this work by testing how sleep disruption influences tumor growth and progression, and by dissecting the gut–brain signaling pathways that mediate this bidirectional relationship, with the ultimate goal of uncovering conserved mechanisms.

S-028

An exploration of sexual dimorphism in the activity/rest diel rhythms of a semiterrestrial crab.

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Sleep is a highly conserved behavioral state observed universally across the animal kingdom. Its ubiquity suggests that sleep serves critical biological functions maintained throughout evolutionary history. Sleep dimorphism is also widespread across taxa. The crab Neohelice granulata is a very good model for conducting behavioral and physiological experiments. We have recently begun describing its spontaneous activity/rest diel rhythms, defining rest periods, and assessing whether these periods meet the criteria to be considered “sleep.” This initial study revealed clear diel rhythmicity, with two peaks of high activity near dawn and dusk respectively, and a prolonged rest phase during midday and afternoon in males. During this rest phase, males often adopted a characteristic posture, with a flattened carapace, retracted claws, and a preference for staying near corners. Recent studies have also reported notable behavioral differences between males and females of this species in prey capture and escape responses. In this project, we aim to investigate whether sexual dimorphism exists in the activity/rest cycles of adult male and female crabs, and whether these patterns vary with the reproductive cycle. Here, we present preliminary results on the activity/rest patterns of adult males and females during autumn, a non-reproductive period. Our findings suggest that, while the overall activity pattern is conserved, some differences are also evident.

S-029

Effects of a School Start Time Delay in cognitive performance of adolescents

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Worldwide, schools start very early in the morning, while adolescents exhibit very late chronotypes. As a consequence, both sleep and performance are negatively affected. International studies have shown that delaying School Start Times (SSTs) has positive effects on adolescents' outcomes, but no evidence is available from Argentina. In this study, we conducted a before/after intervention assessing the effects of a 1-hour SST delay in the Morning shift (with the Afternoon shift as a control group) of a small school. All students completed several cognitive tasks at the first school hour of each shift, before and after the intervention. Our results showed that a 1-hour delay was associated with higher cognitive performance in Morning students. For example, we observed an increased number of trials and shorter response times in a Math addition task and in two different Go/No-Go tests (figures and letters). These findings highlight the benefits of later morning SSTs during adolescence, suggesting that longer sleep duration and/or performing later in the morning improve students' performance, particularly in Argentina where chronotypes are especially late. Importantly, this is the first study to evaluate an SST delay in Argentina, providing local empirical evidence that can inform evidence-based educational policies aimed at improving adolescent well-being, health, and performance outcomes with potential long-term societal and economic impact.

S-030

Optogenetic strategy to examine the role of neuromodulation in contextual memory and learning generalization

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Neuromodulatory systems, such as dopaminergic, serotonergic and octopaminergic regulate motivation, learning, and memory in vertebrates and invertebrates. Preliminary studies indicate that cAMP-signaling facilitates context memory, presumably by neuromodulation. Our hypothesis is that neuromodulatory tone regulates generalization, understood as the application of learning to novel situations, but with some degree of similarity. To evaluate this, we trained Drosophila in one context and tested them in the same, in a similar, or in a different one, allowing us to distinguish between specific memory, normal generalization, and overgeneralization. Neural manipulation was performed using the GAL4/UAS system combined with cationic and anionic Channelrhodopsin targeting dopaminergic, serotonergic, octopaminergic neurons, motoneurons, and MBs. Inhibition of motoneurons validates the optogenetic strategy, as it causes paralysis and prevents context exploration. Preliminary results show that excitation of TH-dopaminergic neurons does not affect habituation memory, while their inhibition promotes it. In contrast, manipulation of PAM neurons (excitation or inhibition) has no effect. So far, no neuromodulator has affected recognition memory. However, inhibition of MB neurons does not alter habituation but does affect recognition memory. Overall, this strategy will allow us to test which neurons are required for learning generalization and possible overgeneralization.

S-031

P300 Latency as a Predictor of Executive Function in Emotionally-Cued Task Performance

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Event-Related Potentials (ERP), particularly the P300 component, have shown potential in predicting cognitive performance. P300 latency is a reliable marker of cognitive abilities such as memory, inhibitory control, and mental efficiency, and has even been linked to personality traits. Performing cognitive tasks often involves activating specific mental processes—known as a “task set”—which are shaped by individual characteristics. However, no prior research has explored the relationship between P300 latency and performance in tasks involving task-set switching, such as a Go/No-Go task using emotional (sad) and neutral facial stimuli. This study aimed to assess whether P300 latency could predict performance in such a task that engages executive functions (EF). Results revealed a significant correlation between longer P300 latencies in posterior electrodes and correct responses in the Go condition ($p < 0.001$), and specifically for sad faces ($p < 0.01$). Notably, latencies at electrodes P3 and P4 strongly correlated with performance when both Go responses and sad face stimuli were combined ($p < 0.001$). A linear regression analysis using posterior P300 latencies as predictors explained 84% of the variance in correct Go responses to sad faces. These findings suggest that P300 latency may serve as a reliable predictor of EF-related task performance.

S-032

Differential Effects of Physical Activity and Screen Time on Memory, Creativity, and Mental Health

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Physical activity (PA) provides multiple benefits for cognition, physiological and mental health; however, evidence in healthy human populations, particularly adolescents, remains limited. In a global context characterized by increasing youth sedentary behavior and prolonged screen time, it is essential to assess their impact in order to design effective educational strategies.

First, we examined the role of physical activity as a modulator of cognitive processes such as memory, creativity, and imagination, as well as the temporal dynamics of its effects on these processes. We observed cognitive improvements associated with physical activity only when it occurred immediately before the tests, whereas no such benefit was evident when the activity was separated by a one-hour interval.

In addition, we evaluated the impact of screen time on different mental health parameters, specifically anxiety and depression. The results showed that longer screen time was associated with increased symptoms of mental health impairment. This effect was present in both males and females, but was more pronounced in the latter.

Finally, we found that students with higher levels of screen time exhibited poorer performance on creativity tests compared to those with lower levels of exposure.
Taken together, these findings provide evidence that both physical activity and screen time modulate, in different ways, cognitive processes such as memory, creativity, and imagination.

S-033

De-polarizing Crowds: How Linguistic Complexity Hinders Group Consensus

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The belief that using more complex and longer words will make a text seem better written (and the author deemed more intelligent) is highly prevalent among students at all levels of the educational system. However, previous literature has proven this to be a myth: writing simpler and clearer leads to better outcomes. Processing fluency –the subjective experience of ease with which people process information– reliably influences people's judgments across a broad range of social dimensions. Nevertheless, prior studies on this phenomenon have primarily focused on how people process written texts at the individual level. In this work, we ask whether linguistic complexity reduces the perceived validity of arguments on controversial moral issues, and whether it also affects the likelihood of people reaching consensus on those issues. We focused on the effect of word length (a previously established standard index of linguistic complexity). Three studies, comprising a large-scale behavioral study (N=10,548), a group deliberation study conducted in online chatrooms (N=768), and a pre-registered randomized controlled experiment where we manipulated word length (N=600), consistently showed that the use of longer words leads to weaker argumentation. In short, these results suggest that brevity and simplicity are key drivers of effective argumentation and deliberation on controversial moral issues.

S-034

What do we talk with when we talk with chatGPT? Approximations from neuroscience to generative Artificial Intelligence models

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The great advance of Large Language Models (LLMs) in recent years has transformed human interaction with Artificial Intelligence, integrating into our daily lives for a multitude of tasks. Despite their widespread use, understanding the underlying principles that govern their internal functioning and the emergence of complex behaviors remains a fundamental challenge. In this poster, we will present a set of research lines, which we carry out from the Applied Artificial Intelligence Laboratory, in which we explore the internal mechanisms of LLMs in different tasks (semantic disambiguation, personality changes, reaction to different political and stereotypical biases, among others) with a perspective from neuroscience and experimental psychology. The main objective of these lines is to improve the understanding of how these models process, represent, and generate language, seeking parallels with biological cognitive systems. These investigations not only contribute to unraveling the "brain" of AI but also offer fertile ground for generating hypotheses about neural computation in biological systems, opening new avenues for the study of cognition and language.

S-035

Fear Memory Retention in Amphibians: Aversive conditioning and neural activity in Rhinella Arenarum

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The study of fear and aversive learning in non-mammalian vertebrates provides valuable insights into the evolutionary origins of emotional memory. Amphibians, with their relatively simple nervous systems, represent an ideal model for investigating these processes. Here, we examined the long-term retention of an aversive memory in a classical conditioning paradigm in the toad Rhinella arenarum using heart rate as a physiological dependent variable. Subjects were implanted with electrodes and exposed to conditioning sessions where a neutral saline solution (CS) was paired with a hypertonic aversive solution (US). Control animals were exposed only to the neutral solution. Anticipatory tachycardia (conditioned response) to the CS was used as an indicator of emotional learning. After acquisition, toads were tested for retention after 1, 4, 8, 16, and 32 days. Significant differences were found between the paired and control groups at all tested times (ANOVA, $p < 0.05$), indicating a persistent and robust conditioned response. Additionally, we used AgNOR histochemical technique (neuronal activity indicator) to obtain preliminary data on the brain structures involved in aversive memory processing. These findings demonstrate that R. arenarum is capable of forming and retaining long-term emotional memories expressed through autonomic responses. This evidence supports the idea that fear learning mechanisms are highly evolutionarily conserved.

S-036

Structural and Functional MRI Markers of Brain Health Phenotypes: Evidence from Latin America

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Background: Brain health phenotyping integrates clinical, cognitive, and neuroimaging measures to characterize neural and functional differences in aging. Aim: To compare the demographic, cognitive, structural, and functional neuroimaging profiles of Latin American cohort aged >50. Methods: From the RedLat consortium, 275 participants with structural MRI were classified as optimal brain health (OBH, n = 90), general brain health (GBH, n = 79), or brain health deficit (BHD, n = 106); 182 also had resting-state fMRI. Voxel-based morphometry (VBM) and intra-network connectivity for 10 canonical resting-state networks were analyzed using ANOVA and FDR-corrected post hoc tests. Results: Groups differed significantly in age and education. VBM revealed focal gray matter reductions in GBH versus OBH, mainly in the left middle temporal gyrus, inferior parietal lobule, fusiform gyrus, and insula (>FDRp< 0.05). BHD showed bilateral atrophy relative to OBH involving simillar regions pluss temporal lobes, hippocampi, amygdalae, putamen, and cerebellum (all FDR p < 0.001). Functionally, somatomotor network connectivity was reduced in BHD compared with OBH (FDR p= 0.001), and default mode network (DMN) connectivity was lower in BHD compared with both OBH and GBH (FDR p= 0.028). Conclusion: These results support integrated structural–functional phenotyping as a target for early detection and intervention in aging populations.

S-037

Neural markers of post-retrieval processes: A time-frequency and microstate EEG study of aversive memory reactivation

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Although fear conditioning is one of the most widely used models to study anxiety, some aspects of its neural correlates remain unexplored. It is known that the retrieval of consolidated memories can trigger modifications depending on the cues presented during the reminder. We employed a three-day threat conditioning protocol in which an angry face (CS+) was paired with an aversive sound (US). Our main objective was to identify neural markers of post-retrieval processes triggered by the presentation of the CS+ alone, 24 hours after acquisition. We analyzed 90 seconds of resting-state electroencephalographic activity following the reminder cue, comparing two groups: a Reactivation group exposed to threat conditioning on the first day, and a Control group with no prior conditioning. A cluster-based permutation test revealed that the Reactivation group showed significantly lower beta-band activity (23–30 Hz) in central regions following the reminder. Then we conducted a deep analysis of the resting state using microstate analysis. Microstates, short quasi-stable topographies of brain activity that re-occur over time, have been proposed as blocks of information processing in the brain. We found significant differences between experimental groups (3-30 Hz), indicating distinct brain states across conditions. Our findings highlight the relevance of evaluating neural correlates of resting post-retrieval processes, including decreased beta activity related to implicit memory.

S-038

Intersubjective Consciousness in Dialogue with Neuroscience: Towards a Systemic Materialist Philosophy

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Contemporary neuroscience has tended toward mechanistic and reductionist approaches to exploring the neural correlates of cognition. While productive in certain areas, these approaches fragment and simplify the richness of lived experience, particularly its intersubjective and qualitative dimensions.

This work proposes a systemic materialist philosophy as an interpretative framework that articulates multiple levels of reality and integrates material facticity with conscious experience, without diminishing or impoverishing it. The Examination of Autistic Intersubjective Experiences (EAIE), a qualitative scale developed by Valeria Bizzari (KU Leuven) and Heidelberg University Hospital, is employed to capture the complexity of autistic intersubjectivity and situate the phenomenological study within its social context.

Far from offering closed answers, this approach fosters a dialogue between philosophy and neuroscience, where intersubjective consciousness emerges as a phenomenon that is simultaneously material and experiential. In this way, autistic identity is explored with rigor and openness, respecting complexity and avoiding any pathologizing framework.

S-039

Role of 5-HT2aR in cognitive flexibility

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Cognitive flexibility is the ability to modify the behavioral response due to change of contingencies in the environment and it's part of the executive functions. Several psychiatric disorders present cognitive inflexibility, like depression, schizophrenia and autism; and that's why this topic has turned to be relevant in the field.The serotoninergic system(5-HT) plays a crucial role in different executive functions, and it's involved in decision-making, planning and action execution. Alterations in this system are found in the previously mentioned diseases. At a functional level, the receptor 2a of serotonin(5-HT2aR) mediates many of the effects of the 5-HT such as sleep, memory, and it's a key factor in cognitive processes. However, its role in cognitive flexibility is less well understood. The aim of this project is to analyze the role of 5-HT2aR in cognitive flexibility. We will use a series of behavioral paradigms:T-maze, radial-arm maze and intra-extra dimensional shift task, which involve different levels of difficulty in the flexibility component. To analyze the role of 5-HT2aR we will use a genetically modified mouse model recently backcrossed into the C57B/6 strain. We began comparing it with our previous strain, where we found that 5-HT2aR KO mice required more time to reach criteria during the reversal phase of the T-maze.This research could improve our understanding of cognitive flexibility, how it's modulated, and support the development of potential therapies.

S-040

Disentangling memory layer by layer

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Memory, the collection of experiences that defines each individual, is continuously reshaped by new learning and reactivation of stored information. Reconsolidation, triggered by mismatches between recalled memory and current circumstances, has been proposed as a mechanism that updates memories in response to environmental or personal changes. If recall occurs frequently in such conditions, reconsolidation could be engaged repeatedly, implying the existence of a biological system that organizes successive updates.

To explore this possibility, we tracked a memory across multiple reactivation sessions using a recognition task in rats. Each session incorporated new information about an object's position into long-term storage. Disrupting reconsolidation prevented this updating and induced retroactive amnesia. Notably, when disruption occurred during a second reconsolidation session, the extent of retrograde amnesia depended on the reminder: if it involved recent memory content, amnesia extended to information integrated during the first reconsolidation without affecting the original learning. Conversely, if the original memory was destabilized, a failure in reconsolidation compromised both training and later reconsolidated information.

These findings suggest that memory is organized in successive, interconnected layers, with each containing partial representations of the trace.

S-041

Prediction error as a modulator of memory reactivation–reconsolidation in adults

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The ability to adapt behavior relies on detecting environmental changes and updating predictions when outcomes deviate from expectations (Prediction Error, PE). Reconsolidation framework proposes that, after a memory is reactivated with a PE, it transiently destabilizes and requires restabilization, allowing its strength and/or content to be modified. Evidence from animal and human studies shows that reactivation with PE strengthens declarative memories, increasing their precision, persistence, and resistance to interference. In this study, we assessed the efficacy of different PEs during episodic memory reconsolidation in young adults. We employed a 2x2 within-subject design manipulating instruction type (classical vs. new) and action possibility (complete vs. no-complete) during Day 2 reactivation. Thirty-five volunteers per group learned 32 face–name pairs (Day 1), underwent reactivation (Day 2) with PE or no-PE reminders, and were tested for retention (Day 3). Regarding results, it is expected that groups will show similar performance during initial associative learning (Day 1) and that differences will emerge between groups exposed to PE (classical/no-complete and new/complete) and those not exposed to PE (classical/complete and new/no-complete). These outcomes will provide further evidence for the role of prediction error in modulating memory reactivation and reconsolidation.

S-042

Exploratory Analysis of Frontal Alpha Dynamics During Resting State After High-Intensity Interval Training

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High-intensity interval training (HIIT) has been linked to modulations in cortical activity, particularly in the alpha band power recorded through EEG. Given the role of alpha oscillations in cortical inhibition, activation, and attentional efficiency, this study aimed to explore their behavior in frontal regions during resting state after a HIIT session. Two healthy participants performed a cycling protocol consisting of ten intervals of 20 seconds at 90% of individual capacity with 40 seconds of active recovery at 50%. Resting-state EEG (2 min, eyes closed) was recorded before exercise, and again at 2- and 5-minutes post-exercise. Power spectral density (PSD) analysis was conducted using MATLAB and EEGLAB, and non-parametric tests (Kruskal–Wallis and post hoc comparisons) were applied. Results showed that one participant demonstrated a significant alpha PSD enhancement at 5 minutes post-exercise, while the other exhibited no significant differences across moments, showing only a tendency toward a delayed increase. Although preliminary, these findings partially align with previous studies reporting delayed increases in alpha PSD following intense exercise, interpreted as a reorganization of the cerebral activation state, either toward heightened alertness or functional rebalancing after exertion. In conclusion, this HIIT protocol may induce transient adaptations in frontal alpha dynamics, although larger samples are required to confirm this trend.

S-043

Aging-induced memory changes in C57BL/6 mice

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Aging is a complex biological process that often leads to cognitive decline, manifested as impairment of essential functions such as memory. Neurotransmission, essential for synaptic plasticity and cognitive function, is compromised in the aging brain, as well as in age-related neurodegenerative disorders. To characterize the relationship between age and cognitive function, a longitudinal study was conducted with C57BL/6 mice. Although this mouse strain has been the subject of multiple studies investigating various aspects of age-related cognitive decline, a comprehensive analysis of its associative and non-associative memory metrics across most of its lifespan is still lacking. Our behavioral studies describe physiological aging, examining mice from 3 to 26 months of age, using the open-field test (OFT) and the novel-object recognition (NOR) test in tandem.

In the OFT, we observed age-dependent differences in the baseline movement of mice with a significant drop after 12-15 months of age. In addition, we detected significant changes in habituation to the context between 3 and 22-24 months of age. On the other hand, NOR results indicated significant changes between 6 and 18 months of age. Altogether, these results suggest that changes in associative and non-associative memories occur at different ages in mice. We managed to establish a baseline for the detection of memory impairments that could be used to evaluate the effectiveness of potential memory-potentiating agents.

S-044

Electrophysiological Correlates of Spontaneous vs. Directed Dreaming

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From a neuroscience perspective, the reprocessing of information during sleep may give rise to dream content. Although dreaming is a common human experience, its electrophysiological correlates remain poorly understood. It has been proposed that dreaming does not depend on the cortex's global oscillatory state but on localized activity in the parieto-occipital region. High-density EEG studies show that increased high-frequency (20-50 Hz) power in frontal and temporal areas during REM sleep is related to dream content. Importantly, these studies examined only spontaneous dreams, without prior incubation using learned information. Pilot work from our lab suggests that incubated dream content (linked to prior learned material) may reflect a global state rather than localized activity. We hypothesize that distinct EEG patterns will characterize spontaneous dreams versus dreams following new learning. To test this, participants will either learn a visuo-spatial location task in virtual reality or not, before 8 hours of sleep under polysomnography (EEG 10-20, 32 channels; EOG, EMG, ECG). In the last 4 hours, serial awakenings in phasic REM will collect dream reports. We will analyze the preceding segments for power spectral density, connectivity, and complexity (Lempel-Ziv, Shannon entropy). Here, we present the rationale and methodological design of Experiment 1 for discussion.

S-045

Exploring the role of the medial prefrontal cortex in the retrieval of episodic memory

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Episodic memory can be defined as the memory for unique events. In most situations, reality is ambiguous, and the cues that trigger retrieval are often associated with more than one particular memory. The medial prefrontal cortex (mPFC) controls the retrieval of memory traces, inhibiting the less relevant one, in situations where the cues presented could trigger the expression of more than one and cause interference. We have identified that the mPFC, the ventral hippocampus (vHPC), the dorsal hippocampus (dHPC), as well as the perirhinal cortex (PRH), are involved in the retrieval of context guided recognition memories But it is still not known the type of information they store and the interaction among them. We have previously shown that mPFC 5HT2aR are necessary for the control of interference during a contextual version of the object recognition task. However, the specific effects of mPFC subregions and their modulation by 5-HT2aR in this process is not clear. Combining behavioral and pharmacological tools we started analyzing the contribution of 5-HT2aR within the different subregions of the mPFC in the control of memory interference. Our findings show that infralimbic (IL) and prelimbic (PL) 5-HT2aR are required for the retrieval of an contextually guided object memory Additionally, we have evidence indicating that 5HT2aR modulation of the IL-vHPC; IL-PRH; PL-dHPC and PL-PRH circuits are required during retrieval of this type of memory.

S-046

Attraction to Extremes: Cross-Cultural Evidence of Political Acrophily

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Political polarization has intensified globally, with social media amplifying extreme views and reinforcing echo chambers. Recent research in the U.S. identified political acrophily, the tendency to prefer more extreme co-partisans over moderates, on social media platforms (Zimmerman et al., 2024). This phenomenon goes beyond homophily, revealing a pull toward political extremes and generating greater out-group animosity.

To test whether acrophily reflects a general psychological tendency or is context-specific, we adapted the design for a cross-national study. We recruited local collaborators in over 40 countries, who will review the culturally adapted materials. We ran a pilot in five countries (U.S., Chile, Germany, South Africa, and Australia), recruiting balanced samples of 250 participants each (N=1250). Participants evaluated fictional social media profiles that varied in extremity and partisan alignment, indicating follow-back intentions and rating perceived confidence, representativeness, strength, and entertainment value.

Preliminary results show that acrophily replicates across contexts, although its magnitude varies, suggesting both general and context-specific mechanisms. By comparing results across political systems and economic conditions, this project aims to clarify whether attraction to political extremes reflects a universal psychological bias amplified by social media platforms or emerges more strongly under particular social and political conditions.

S-047

Timing Matters: Dose- and Time-Dependent Effects of Alcohol Hangover on Memory in Mice

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Alcohol hangover is a transient pathophysiological state that begins when blood alcohol concentration (BAC) approaches zero and is characterized by physical and cognitive impairments that may last for several hours. While the acute effects of alcohol intoxication on cognitive performance are well documented, the specific impact of hangover on memory processes remains less understood.

Within the framework of an undergraduate thesis, this study aimed to explore the effects of alcohol hangover on inhibitory avoidance memory in female CF-1 mice. Animals received intraperitoneal injections of veh (control), 1.9 g/kg, or 3.8 g/kg ethanol. Training was conducted at 6, 9, or 12 hours post-administration, representing different stages of the hangover period, and memory retention was evaluated 48 hours later using the inhibitory avoidance task.

Preliminary findings suggest that hangover impairs memory retention in a dose- and time-dependent manner. The most significant deficits were observed in mice treated with 3.8 g/kg ethanol and trained at earlier hangover stages, indicating that both the intensity of exposure and the timing of learning relative to hangover onset critically influence cognitive outcomes. These results provide initial evidence that alcohol hangover interferes with memory consolidation mechanisms, underscoring its potential impact on cognitive performance beyond acute intoxication.

S-048

Can music-based interventions modulate reconsolidation processes?

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Introduction. Music-based interventions (MBI) induce emotional states which can modulate memory consolidation. Here we present three experiments studying if listening to music can also modulate memory reconsolidation. Aim. Exp. 1 assessed the effect of arousing and relaxing music during the reconsolidation window. Exp. 2 assessed if an emotionally stronger MBI (i.e., preference music) could maximize its effect. Exp. 3 tested if the results can be explained by state-dependency, re-exposing participants to the same MBI before performing memory tasks. Results. Listening to arousing music reduced retrieval, without affecting recognition over a two-week period (Exp. 1). Such effect was not maximized by preference (Exp. 2). No re-exposition effect was found (Exp. 3). Discussion. Listening to arousing music after reactivating memory can interfere with the re-stabilization process and weaken memory, but it may not be strong enough to eliminate it. This amnesic effect is not explained by state-dependency. In contrast to that observed on memory consolidation, MBI diminishes memory; indicating that MBI interact in a unique way with each memory phase.

S-049

The study the effect of concurrent application of the deep brain stimulation of the nucleus accumbens and cannabidiol injection during extinction period on reinstatement of METH-extinguished in rat

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Methamphetamine (METH), is a potent stimulant drug that significantly alters the function of the central nervous system (CNS). MTTHS's abuse may be linked with a variety of psychiatric diseases and cognitive deficits, mostly via affecting the reward system.

Cannabidiol (CBD), a primary cannabinoid component of the Cannabis plant has been shown to affect reward system, candidated it for the modulation of addictive behaviors. On the other hand, deep brain stimulation (DBS) of the nucleus accumbens (as one of the most important brain regions implicated in mediating rewards) is effective in the alleviation of drug-seeking behaviors. The present research aimed to evaluate the effects of low frequency DBS, CBD, and combination of both on the reinstatement of METH-induced conditioned place preference (CPP). CBD was injected into the later ventricle at the doses of 2.5, 5, and
10 µg/5 µL, and low frequency DBS (10 Hz, for 30 minutes) was administered to the nucleus accumbens, both during the extinction phase. The results showed CBD (10 µg/5 µL) or DBS facilitated extinction. CBD at all doses in combination with DBS induced a more significant decrease in mean extinction latency. However, both CBD (10 µg/5 µL) and DBS did not alter the CPP score on the reinstatement day. Interestingly, combination of DBS and CBD administration (at all doses) significantly decreased the CPP score on the reinstatement day.

S-050

Cerebellar Involvement in Sociability: Focus on Inflammatory Mechanisms

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Sociability, defined as the tendency to interact with conspecifics, is frequently disrupted in psychiatric and neurodevelopmental disorders, including autism spectrum disorder (ASD). Although the cerebellum has been traditionally associated with motor control, increasing evidence points to its involvement in cognition, affective regulation, and the neuropathology of ASD. Here, we examined the contribution of cerebellar lobule VI/VII to sociability, with a focus on neuroinflammatory mechanisms. Using the prenatal valproic acid (VPA) mouse model, we confirmed that male offspring exhibit reduced sociability and early postnatal alterations in Purkinje cell density, although these structural differences were not observed in adulthood. Furthermore, induction of neuroinflammation in lobule VI/VII of adult males led to pronounced social deficits, which were completely prevented by systemic dexamethasone and only partially prevented by ibuprofen. Consistent with these behavioral results, microglial activation was reduced in dexamethasone-treated animals. Together, these findings implicate NFκB-dependent microglial activation in cerebellar regulation of social behavior and highlight pharmacological modulation of neuroinflammation as a potential therapeutic avenue for ASD-related sociability impairments.

S-051

An information-theoretical approach to study autobiographical memory reconstruction: an EEG study

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Autobiographical memory (AM) is a complex process that relies on distributed and dynamic brain networks. A crucial stage is the reconstruction phase, when individuals reexperience and mentally recreate an event, imagining it with vivid sensory, contextual, and emotional details. Despite the richness and complexity of this process, the neural characterization of AM reconstruction has mainly relied on classical measures of connectivity and spectral power, leaving frameworks such as Information Theory unexplored in this context. In this work, we propose to investigate AM reconstruction using information-theoretic quantifiers (Shannon entropy and Statistical Complexity) as measures to characterize different types of memories varying in age, detail, importance, and emotional valence, among other features. Our aim is to apply non-traditional techniques to uncover neural patterns unexplored with standard approaches to identify distinctive markers linked explicitly to the mental reconstruction of autobiographical events. The use of these measures may provide novel markers, offering new insights into how AMs are recalled and represented. Given the inherent nature of AM mental elaboration, we focus on occipital regions, seeking distinctive markers of visual and reconstructive processing. We analyze 8-second-long EEG time series (30 channels, 256 Hz sampling rate) recorded from 33 participants during an AM reconstruction task.

S-052

Serotonin 2A receptor in the retrosplenial cortex play a key role in recognition memory

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The retrosplenial cortex (RSC) is particularly sensitive to changes that occur in mild cognitive impairment and Alzheimer's disease (AD), making understanding its function crucial. A main feature of the AD is its memory problems. In many cases, patients present psychosis. Dementia-related psychosis has no selective treatment, but recently the focus has been on serotoninergic drugs, particularly on a selective serotonin (5-HT) 2A inverse agonist, supporting a role for the serotoninergic system in AD treatment. 5-HT2A receptors (5-HT2AR) are highly expressed in cortical regions and have been linked to modulation of cognitive processes. However, its role in memory processes is not completely understood. Previously, we showed that RSC is involved in recognition memory. Since 5-HT2AR are expressed in RSC, we decided to investigate the role of 5-HT2AR in aRSC in different memory phases in the object recognition (OR). Using the Y-OR task combined with localized administration in the RSC of Ketenaserine, a selective 5-HT2 antagonist, we find that this receptor is required for memory formation. Furthermore, by infusing the selective 5-HT2A antagonist MDL into the RSC, we showed these receptors contribute differentially to acquisition, consolidation, and retrieval. These results suggest that 5-HT2AR in the RSC are required for OR memory processing and may be a promising therapeutic target.

S-053

Effects of Metformin on Emotional and Cognitive Behavior in a Social Isolation Model in Rats

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Major depression is linked to cognitive, emotional, and physiological alterations, experimentally modeled in rodents through 3 months of social isolation. This study evaluated the effect of metformin (100mg/kg/day orally) on spatial memory, anxiety, anhedonia, and physiological parameters in male Sprague-Dawley rats. We used three groups (n=17): Isolated Water (untreated, n=6), Isolated Met (n=5), and Social (control, n=6).
Depression-like behavior was assessed via the marble burying, sucrose preference, and forced swim tests. Episodic memory was evaluated using Spontaneous Location Recognition (SLR) and the Barnes Maze.

Metformin reduced anxious behavior (marble burying), regulated food intake and weight, and reversed anhedonia (sucrose preference similar to Social group, $p<0.01$). However, it did not alter depressive behavior in the forced swim test. In the SLR, the Isolated Met group showed a significant preference for the moved object ($p<0.01$), indicating hippocampal recovery, unlike the Isolated Water group. Yet, in the Barnes Maze, no significant improvement was observed in the probe trial; latency and errors did not differ from the untreated group.
These results suggest metformin has a positive effect on emotional behavior, spontaneous spatial exploration, and physiological parameters, although it does not completely restore goal-directed spatial memory. This positions metformin as a potential therapeutic target for mood disorders.

S-054

Unveiling the Forgotten: Age-Dependent Effects of ICV STZ in Female Rats Modelling Alzheimer's Disease

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Alzheimer's disease (AD) is the leading cause of dementia and remains without effective therapeutic options. Women represent two-thirds of patients, yet female animals continue to be underrepresented in preclinical studies. The intracerebroventricular injection of streptozotocin (ICV-STZ) is a well-established model for inducing sporadic AD-like features, primarily developed and characterized in male rodents, whereas studies in females are relatively scarce. Building on this, the present study aimed to determine whether age further modulates the response to STZ in females. Sprague-Dawley rats aged 12 and 18 months were divided into four groups: 12mSham, 12mSTZ, 18mSham, and 18mSTZ. On experimental day (ED) 0, rats received bilateral ICV injections of either artificial cerebrospinal fluid (12mSham/18mSham) or 3 mg/kg of STZ (12mSTZ/18mSTZ). Among ED 14-25, a battery of behavioral tests was conducted to assess both general behavior and cognitive function. Our results show that STZ induces behavioral and cognitive impairments, with some effects being age-independent and others modulated by aging. Exploratory and anxiety-related behaviors were consistently affected by STZ, while freezing and spatial memory were influenced by age. Recognition memory was impaired by STZ in younger animals, whereas older rats already displayed age-related deficits. Our findings highlight the importance of considering both sex and age in preclinical models of AD.

S-055

Associative learning and extinction: the role of Ventral Tegmental Area to Amygdala circuit

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Memory enables animals to adapt behavior based on past experiences, enhancing survival by avoiding danger or exploiting resources. In associative learning, the amygdala integrates sensory inputs and assigns emotional valence, while recent studies implicate dopaminergic neurons in the ventral tegmental area (VTA) in both appetitive and aversive learning. However, the specific contribution of the VTA-to-basolateral amygdala (BLA) projection to the acquisition and extinction of associative memories remains unclear. This study aimed to assess how increased dopamine release from the VTA to the BLA affects associative learning of opposite valences. A custom-built behavioral chamber enabled appetitive and aversive differential conditioning: in the appetitive task, a CS+ predicted water reward; in the aversive task, a CS+ predicted footshock. To selectively enhance dopamine signaling in the VTA-BLA pathway, we used transgenic mice allowing targeted deletion of D2 autoreceptors via retrograde AAV injection into the BLA. Preliminary results suggest increased dopaminergic tone in this pathway alters learning in appetitive conditioning and affects extinction in aversive learning. Additional behavioral assays, including object and social recognition tasks, controlled for general changes in memory function. Histological verification and dopamine level measurements are ongoing. These findings offer initial insight into how VTA-BLA dopaminergic modulation influences associative memory.

S-056

Defensive behaviors and hippocampal dynamics induced by visual threat stimuli in marmosets (Callithrix jacchus)

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Fear is a defensive state that promotes survival by coordinating behavioral and physiological responses to threat. Salient aversive stimuli can elicit innate defensive reactions and shape long-lasting memories of fearful events. Both rodents and primates exhibit defensive behaviors such as flight, shelter seeking, and freezing when confronted with visual threats. Extensive research in rodents over recent decades has advanced our understanding of the neural circuits of fear, but the mechanisms underlying visually guided defensive behaviors in primates remain poorly understood. Studying these processes in nonhuman primates may help develop effective treatments for disorders associated with maladaptive fear memories. Here, we evaluated the relationship between defensive behavior and hippocampal oscillations in marmosets exposed to neutral and aversive visual stimuli. We found that exposure to aversive stimuli increased fixed gaze toward the threat, reduced vigilance, restricted spatial occupancy and enhanced hippocampal gamma power. Conversely, neutral stimuli resulted in homogeneous exploratory behaviors without altering gamma activity. These results suggest that the hippocampus contributes to online threat evaluation and provide a framework for investigating how defensive behaviors guide the formation of long-lasting fear memories in primates.

S-057

Hippocampal GLT-1 Inhibition Selectively Blocks Aversive Memory Reconsolidation

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The control of glutamate level in the synaptic gap is crucial for neuronal communication, as it prevents excitotoxicity from excessive receptor activation. Glutamate transporters GLT-1 are mainly localized in astrocytes and its expression is particularly abundant in the hippocampus (Hp). In this study, explored the role of GLT-1 in consolidation, expression and reconsolidation of memory, using contextual fear conditioning (CFC) task. Dihydrokainic acid (DHK), a selective GLT-1 inhibitor, was injected into the dorsal Hp of male rats at different times around aversive learning. DHK administration 15 minutes after weak CFC training session promoted long-term memory (LTM) formation. However, DHK administration around strong CFC training session did not affect memory consolidation. Moreover, DHK infusion 15 minutes before test session impaired CFC memory expression. Importantly, if applied 15 minutes after a reactivation session, DHK impairs CFC reconsolidation. This study complements previous findings in females rats by covering the full dynamics of aversive memory and confirms the lack of sexual dimorphism in the role of GLT-1. In conclusion, our findings indicate that inhibition of hippocampal astroglial glutamate uptake impairs the expression and reconsolidation of aversive memory without impairing its consolidation, and highlight potential therapeutic avenues for neuropsychiatric conditions, including phobias, post-traumatic stress disorder, and cognitive deficits.

S-058

Role of 5-HT2AR in Prefrontal Cortex Activity and Plasticity in Social Behavior

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Serotonergic signaling has shown to be a key player in the modulation of social behavior. Specifically, serotonin type 2A receptors (5-HT2AR), known to be involved in a variety of behaviors, have also been linked to social cognition through the pathophysiology of different psychiatric and neurodevelopmental disorders, and its role in the mechanism of action of so-called “prosocial” drugs. Our lab found that 5-HT2AR knockout (htr2a-/-) had reduced discrimination indexes in the three-chambers social interaction test (SIT) compared to wild types (htr2a+/+) mice. By genetically restoring the expression of 5-HT2AR in the forebrain, mice reached discrimination indexes similar to htr2a+/+. Moreover, acute blockade of 5-HT2AR with MDL 11939 in htr2a+/+ mice before the SIT did not affected discrimination indexes, suggesting that the receptor is not acutely recruited in this task. Finally, preliminary results of c-fos expression analysis post SIT showed differential expression between htr2a+/+ and htr2a-/- mice. In conclusion, these findings suggest that 5-HT2AR plays a role in social behavior, with a possible sex-specific modulation, and highlight the distinct roles of this receptor in social cognition under acute versus developmental manipulations.

S-059

Ketogenic Diet in Outpatients with Refractory Symptoms: A Pilot Study

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Treatment-resistant severe mental illness (SMI) represents a major clinical challenge. The ketogenic diet (KD), widely recognized for its efficacy in pharmacoresistant epilepsy, has recently emerged as a potential metabolic intervention in psychiatric care. Preclinical evidence and case reports suggest that KD may improve psychotic and affective symptoms through neurobiological and metabolic mechanisms. However, studies conducted in outpatient settings and within Spanish-speaking populations remain scarce.
We implemented a 9-week KD protocol in outpatients with severe refractory symptoms (N = 6). The dietary plan included weekly monitoring of ketosis. A repeated-measures pre–post design was employed. Psychotic and affective symptoms were assessed using the Symptom Checklist-90-R (SCL-90-R), a self-report measure of psychopathological symptoms.
Post-intervention analyses revealed statistically significant reductions in Global Severity Index ($W = 21.0$, $p = .031$), Positive Symptom Total ($W = 21.0$, $p = .031$), Positive Symptom Distress Index ($W = 21.0$, $p = .031$), and Hostility ($W = 21.0$, $p = .036$). No serious adverse events were reported, and adherence was satisfactory.
These preliminary findings suggest that implementing KD in refractory psychosis is feasible, safe, and potentially beneficial in outpatient populations. This study provides initial evidence within a Spanish-speaking context and supports the need for controlled trials with larger samples.

S-060

Changes in Spatial Memory and Exploration Patterns in a GluN2A Knockdown Rat Model

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During embryonic stage, neurons predominantly express N-methyl-D-aspartate receptors (NMDARs) containing GluN2B regulatory subunits. After birth, and in response to neural activity, GluN2A expression is increased and GluN2A-containing NMDARs localize at synapses, supporting neuronal maturation and synaptic refinement. Our previous work showed that GluN2A knockdown (GluN2A-KD) in the hippocampus of young adult Wistar rats induced a cognitive deficit in the contextual component of an Inhibitory avoidance paradigm. Here, we evaluated the effects of GluN2A-KD on spatial memory. For this reason, three-month-old male and female rats were injected at the CA1 dorsal hippocampus with an adeno-associated viral vector carrying either a shRNA against GluN2A (2A-shRNA) or a scrambled sequence (sc-shRNA). 14 days later, animals were tested in a battery of spatial tasks (Barnes maze, reverse Barnes maze, and an object exploration task) . Male GluN2A-KD rats acquired learning criteria in the Barnes maze, whereas females did not. Both male and female GluN2A-KD rats exhibited reduced cognitive flexibility in the reverse Barnes maze. Moreover, GluN2A expression levels correlated with spatial learning indexes. In the object exploration task, GluN2A-KD rats displayed an increase in object interaction and clustering behavior compared to controls. These findings suggest that proper GluN2A expression is essential for long-term spatial memory, cognitive flexibility, and exploratory spatial patterns.

S-061

Early-life methylphenidate treatment increases adult ethanol sensitivity, impulsivity, and voluntary ethanol intake in a sex-dependent ADHD mouse model

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Attention-Deficit/Hyperactivity Disorder (ADHD), the most prevalent behavioral disorder in childhood, affects 4% of children, affecting social and academic functioning.Methylphenidate (MTPH), a psychostimulant with amphetamine-like properties, is the most prescribed treatment for ADHD.Considering the impact of early substance exposure on later addiction risk, chronic MTPH use during childhood or adolescence may increase vulnerability to substance abuse.Globally, 2 billion people consume or abuse alcohol, accounting for 4% of deaths.

We examined the long-term impact of chronic MTPH during childhood/adolescence on ethanol-related behaviors in a validated ADHD mouse model, focusing on voluntary ethanol consumption in adulthood. p35KO transgenic mice and wild-type (WT) controls were treated with MTPH from postnatal days 21–31.In adulthood, we assessed ethanol-induced locomotor stimulation, sensitization, risk-related behaviors, and 24-h voluntary ethanol intake.

Early MTPH treatment produced long-lasting, sex- and genotype-dependent effects on ethanol sensitivity.WT mice showed acute locomotor activation, with sensitization only in females, whereas p35KO responded only after 14 days of repeated exposure. MTPH also enhanced impulsive-like behaviors and voluntary ethanol preference and consumption in male WT and female p35KO mice,indicating higher addiction risk.These findings suggest that early psychostimulant exposure may increase alcohol sensitivity and impulsivity in adulthood.

S-062

Modulation of the cocaine response in female and male rats exposed to 2,4-D during adolescence: evidence for the role of the Wnt pathway in PFC

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2,4-Dichlorophenoxyacetic acid (2,4-D) is currently the second most used herbicide in the world. Previous research from our laboratory has shown that 2,4-D has neurotoxic effects in animal models and may be linked to cognitive and psychiatric disorders associated with alterations in dopaminergic systems. Our laboratory was the first to demonstrate that changes in the Wnt/β-catenin pathway in PFC are associated with an increase response to cocaine. The aim of this study was to investigate the impact of oral exposure to 2,4-D during adolescence on cocaine vulnerability at behavioral, neurochemical, and molecular levels.

From post-natal day (PND) 30 to PDN49, female and male rats, were fed with food exposed to 2,4-D (25 mg/kg/day) or to vehicle. On PND 50, total locomotor activity was recorded for 100 minutes following a cocaine a cocaine (5 mg/kg i.p) or saline injection. Twenty-four hours later, animals were sacrificed and PFC tissue was collected for western blot analysis. Results showed that in female rats exposed to 2,4-D cocaine induced a significantly higher locomotor activity compared to all other groups. Moreover, β-catenin expression in PFC was increased in these females. No significant differences at behavioral or molecular levels were found in males. In conclusion, these findings suggest that exposure to 2,4-D enhances susceptibility to cocaine in adolescent female rats, potentially through modulation of the Wnt/β-catenin pathway in the PFC.

S-063

Ethanol´s opposing effects on memory formation are controlled by ventral tegmental area α7 nicotinic acetylcholine receptors

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Ethanol alters brain function, producing cognitive and behavioral changes. While the effects of chronic ethanol exposure on learning and memory are well documented, its acute effects remain less understood. This study investigated the impact of acute ethanol administration on memory formation, focusing on α7 nicotinic acetylcholine receptors (α7nAChRs) in the ventral tegmental area (VTA).

Adult male rats were trained in an inhibitory avoidance (IA) task and received an acute intraperitoneal ethanol injection immediately after training.Memory was assessed 48 h later. Open field tests followed IA to rule out anxiety-like behavior and locomotor alterations. To explore the role of α7nAChRs, we infused either the positive allosteric modulator PNU-120596 or the selective antagonist methyllycaconitine (MLA) into the VTA post-training to evaluate their ability to prevent ethanol induced memory changes.

Ethanol produced dose-dependent effects: 1.5 g/kg impaired memory, whereas 2.5 g/kg enhanced it. Open field performance was unaffected. Although VTA α7nAChRs did not directly influence consolidation, PNU-120596 prevented ethanol-induced deficits, and MLA blocked ethanol-induced enhancement, indicating a modulatory role of these receptors in both effects.

These findings demonstrate that a single ethanol exposure during an aversive experience can bidirectionally alter memory formation and identify VTA α7nAChRs as key modulators of this process.

S-064

Neurotoxic effects of PcPV2 on cognition, exploration, species-typical behavior, and sensorimotor performance in rats

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Pomacea canaliculate is the only freshwater snail listed as one of the 100 worst invasive species worldwide. Its eggs are protected by perivitelline fluid containing PcPV2, a protein with enterotoxic and neurotoxic activities. Although intraperitoneal (i.p.) PcPV2 causes neurological signs and even death in rodents, its behavioral effects remain unexplored. We evaluated adult male Sprague-Dawley rats (n=10/group) after i.p. PcPV2 (250 µg/kg) at two time points: early (days 3-6) and late (days 24-27). Behavioral parameters assessed included exploration and anxiety (open field test), recognition and spatial memory (novel object and location recognition test), species-typical behavior (marble burying), and sensorimotor performance (adhesive removal, inclined ramp). PcPV2-treated rats, at both time points, buried fewer marbles and exhibited impaired sensorimotor performance. In the open field, they showed increased immobility at both assessments, with reduced exploration only in the early phase. In cognitive tasks, they explored novel or displaced objects less than controls in the early window. Overall, PcPV2 induced significant behavioral and sensorimotor impairments, with some effects being transient and others persisting throughout the study, confirming its neurotoxic potential. This study provides the first comprehensive behavioral evaluation of ip PcPV2, offering a framework for future neurotoxicity research and mechanistic studies.

S-065

Explaining to discern: Intellectual Humility as a lever against misinformation

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Misinformation spreads rapidly online, shaping beliefs and behaviors even after corrections are made. Fact-checking is crucial but insufficient, making additional interventions necessary. Overconfidence, an inflated belief in one's own knowledge, has been identified as a factor related to misinformation susceptibility. Explanation-based strategies offer a valuable approach to addressing misinformation. When people explain how they know what they claim to know, they often see gaps in their knowledge. This recognition can lead to a shift in confidence and a more thoughtful approach. This process is closely connected to intellectual humility (IH). IH involves recognizing the limits of one's knowledge, being open to change, and considering other people's viewpoints. IH is seen as protective against misinformation, but causal evidence is lacking.

This pilot study tests two explanation-based interventions on recognizing fake news: a general depth explanation and a simplified item-by-item explanation against a control. Participants first judge the veracity, confidence, and sharing intentions of existing true and false headlines. They are then assigned to one of the interventions, followed by a second test including both repeated and novel headlines. We hypothesize that explanation prompts will increase veracity discernment, reduce intentions to share false content (for both repeated and novel items), and foster IH as a mediating mechanism.

S-066

Prenatal and Postnatal Ethanol Exposure Alters Early Motivational Learning in Wistar Rats: Uncovering Sex-specific Vulnerabilities

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Alcohol dependence is a global public health issue with significant consequences during prenatal and postnatal development. Early exposure to alcohol, even at social levels during pregnancy and lactation, increases vulnerability to later problematic use. Local studies report high prevalence of such exposure and its behavioral effects in newborns, emphasizing the need to investigate underlying mechanisms to strengthen early prevention.

This study examined the effects of prenatal and postnatal ethanol exposure on early motivational learning in Wistar rat pups, evaluating different exposure combinations and sex-related differences using a Conditioned Place Preference (CPP) paradigm at postnatal day 9.

Results showed that females displayed increased preference after prenatal exposure and reduced preference after postnatal exposure, whereas males exhibited enhanced preference only after combined exposure. These differences were independent of locomotor or exploratory activity, indicating genuine associative learning. Findings reveal early sex-specific effects, possibly mediated by organizational and epigenetic mechanisms.
Overall, this work highlights sex as a key biological variable in alcohol's motivational effects and identifies early exposure as a critical risk factor in fetal programming. Ethically, in line with Argentina's Mental Health Law No. 26.657, these findings reinforce the need for preventive policies to reduce alcohol consumption during pregnancy and childhood.

Development

S-067

Rol of medial olivocochlear efferent activity in central auditory synapse development.

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At birth, the auditory system of most mammals is functionally immature and requires a postnatal period of synaptic refinement to achieve the precise connectivity observed in adulthood. During this developmental window, the medial olivocochlear (MOC) efferent system modulates spontaneous inner ear activity, thereby affecting the maturation of central circuits. In mice with enhanced MOC activity (α9KI), synaptic dysfunction was found at the calyx of Held (CH) within the medial nucleus of the trapezoid body (MNTB) (Di Guilmi et al., 2019). In this study, we combined in vitro electrophysiology (P12–14) with 3D morphological reconstructions (P12–14 and P21–25) using serial electron microscopy in three genotypes: wild type (WT), α9KI, and α9KO (lacking MOC activity) and a custom Python-based code to extract quantitative parameters, enabling the classification of CHs into distinct morphotypes. Recordings in the α9KI mice displayed synaptic alterations across several parameters, evidenced by a lower excitatory post-synaptic current, a higher short-term depression and smaller ready realizable vesicle pool. Morphological analyses revealed a lower proportion of structurally complex CHs and decreased synaptic pruning in α9KI animals. By contrast, α9KO only showed reduced morphological complexity. These results suggest that enhanced MOC activity drives more profound developmental modifications in MNTB circuitry compared to the absence of MOC modulation.

S-068

Functional analysis of Delta-like 1 regulatory elements during brain development

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Delta-like 1 (Dll1), which encodes the canonical Notch ligand DELTA1, plays a central role in neurogenesis during embryonic development and contributes to the organization of the adult nervous system. Dissecting the regulatory mechanisms that control Dll1 transcription, as well as their evolutionary modifications, is essential to understand the genetic basis of the brain diversity observed among species, particularly within our evolutionary lineage.

In this work, we focused on conserved non-coding elements with potential enhancer activity. We tested their regulatory capacity through reporter assays in transgenic mice and mapped their spatial and temporal activity. To further assess their contribution in vivo, we generated enhancer knock-out models, where the loss of specific elements resulted in reduced Dll1 expression as measured by qPCR. RNA-seq showed that many genes potentially regulated by this pathway were either up- or downregulated. In addition, we modeled evolutionary changes by generating a knock-in mouse line in which the endogenous enhancer was replaced with the human orthologous sequence, carrying primate-specific substitutions. This experiment also pointed to transcriptional differences, suggesting that anthropoid-specific regulatory variants may influence Dll1 expression.

S-069

Early Detection of Development Risks: Findings from a Hospital-Based Psychological and Neuropsychological Assessment Program

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Psychological and neuropsychological assessments in hospital settings must consider the reason for referral, the child's characteristics, and the specific clinical context. This process involves formulating diagnostic hypotheses, selecting appropriate instruments, and integrating findings to guide timely intervention. This study, part of a University Extension Program at the Faculty of Psychology, University of Buenos Aires, carried out at the Hospital de Clínicas "José de San Martín," aims to detect developmental risk or developmental delay in children referred for diagnostic evaluation to intervene according to each child's capacities and needs. We present a preliminary descriptive analysis of assessments conducted between 2023 and 2025 in 39 children aged 0–12 years referred from pediatrics, neurology, child psychiatry, nutrition, pulmonology, judicial services, schools, and other health institutions in Buenos Aires City and Province. This exploratory, non-probabilistic study analyzed frequency distributions to describe reasons for referral, types of evaluations performed, and developmental profiles observed. Preliminary findings offer context for the neuropsychological and psychological development of this gropu, underscoring the value of hospital-based assessments in identifying atypical developmental trajectories and supporting timely responses.

S-070

Effects of Maternal Pinealectomy on the Perinatal Development of Rat Offspring

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Melatonin, synthesized in the pineal gland, participates in the regulation of physiological processes during gestation and fetal development. However, the effects of its reduction on intrauterine life are not yet fully understood. The objective of this study was to evaluate the perinatal development of the offspring of rats undergoing pinealectomy. The protocol was approved by CEUA/UFS (No. 4723240225). Forty-five-day-old Wistar rats were divided into two groups: Sham (SHAM, n=3; 29 pups) and Pinealectomized (PIN, n=4; 37 pups). At three months of age, after surgery, estrous cycle and mating were monitored. The offspring were monitored daily from postnatal day (PND) 2 to 40, with systematic recording of physical development milestones: eye opening, ear opening and vaginal canal development, ear development, incisor eruption, lanugo and hair development, and testicular descent. Offspring of PIN mothers showed delayed eye opening, ear opening development, ear development, incisor eruption, and lanugo development compared to the SHAM group. There was no statistical difference in hair development or genital development. It is concluded that removal of the gland causes delays in the perinatal physical development of the offspring, suggesting a relevant role for melatonin in this process, which may compromise early somatic maturation, possibly due to the loss of its antioxidant and circadian rhythm regulating effects.

Keywords: Melatonin, neurodevelopment, pinealectomy.

S-071

Dynamic Regulation of Retinal Axon Growth and guidance by EphA3 and Neurotrophic Gradients

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The establishment of topographic retinotectal connections requires precise regulation of axonal growth. EphA3 stimulates axon growth of nasal retinal ganglion cells (RGCs) towards the caudal tectum, inhibiting branching in the rostral tectum. GDNF and BDNF stimulates RGC axon growth, though its effects on RGC growth cone dynamics are unclear. We examined the individual and combined effects of EphA3, GDNF and BDNF on growth cone dynamics of dissociated nasal retinal ganglion cells (RGCs) from chicken embryos. Dissociated RGCs were cultured and exposed to EphA3-Fc, GDNF, BDNF, or EphA3-Fc combined with neurotrophic factors. Growth cone velocity, orientation and persistence were quantified by timelapse imaging in Dunn’s chamber. EphA3 gradients significantly increased axon extension rate and promoted directional attraction. EphA3 gradients and neurotrophic factors (notably GDNF) promoted axon extension and orientation; combined EphA3+GDNF synergistically accelerated growth, stabilized trajectories, and reduced exploratory behavior. These results indicate that growth dynamics are critically modulated by guidance cues and trophic support, and that their combined action promotes robust, directed outgrowth. Our findings provide novel insight into mechanisms shaping retinotectal map formation and suggest that synergistic interactions between guidance molecules and neurotrophic factors could inform strategies to enhance axonal regeneration.

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S-072

Early-life stress alters development of prefrontal circuits modulating dorsal raphe serotonin neurons: Implications for maladaptive adult emotional behavior

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The vulnerability to stress and mood disorders is thought to have a developmental origin. Converging evidence indicates that prefrontal cortex (PFC) circuits engaged in cortico-limbic top-down control are key in the developmental etiology of mood disorders. The neural circuit connecting the PFC to the dorsal raphe nucleus (DRN) is critically involved in stress-coping responses and mood control, and represents the main source of brain serotonin (5-HT). During mouse development there is a critical period [postnatal days (P) 2 to 14] when environmental factors can influence neurodevelopmental trajectories with long-lasting consequences for adult life. The early-life stress of maternal separation (MS) is a validated model that causes adult emotional alterations. We investigate how the early PFC-to-DRN circuit is formed and refined, and how dysregulation of its neurodevelopment is affected in the MS model. We evaluated alterations in the synaptic connectivity of the PFC-to-DRN circuit using the high-resolution microscopy technique Array Tomography and the activation of DRN 5-HT neurons was assessed by cFos immunostaining. To investigate possible physiological correlates accompanying morphological changes we performed ex-vivo patch clamp recordings on both 5-HT and GABA DRN neurons of MS mice at these different developmental ages. Our work indicates that maternally-separated mice have alterations in the PFC-to-DRN circuit and 5HT neuron stress-dependent activation.

S-073

Neuroinflammation and Vascular Sequelae: AT1 Receptors and IGF-1 as a Therapeutic Approach

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Parkinson's disease, a dopaminergic (DA) degeneration of nigrostriatal neurons of still unknown etiology, affects about 2% of the population over 65 years of age. Recently, it has been proposed that one possible cause of dopaminergic cell death is the overactivation of the brain renin–angiotensin system (RAS), which operates locally and independently from the peripheral system. This system, present in the substantia nigra and striatum, becomes hyperactivated through AT1 receptors, inducing oxidative stress and promoting inflammatory responses, thereby facilitating DA neurodegeneration and rarefaction of the brain microvasculature. In addition, reduced circulating levels of IGF-1, characteristic of aging and neuroinflammation, increase the risk of cerebral microvascular damage and cerebromicrovascular dysfunction. These processes are interconnected and mutually reinforcing. In this context, we evaluated, in early stages of the disease, changes in the microvasculature such as blood–brain barrier damage in a parkinsonian animal model. We propose an integrative therapeutic approach to counteract these alterations: administration of the drug candesartan, which blocks AT1 receptors, together with adenovectors to enhance IGF-1 expression in our animal model.

S-074

Elevated spatial coefficient of variation in Arterial Spin Labelling MRI reveals global vascular dysfunction in Long COVID patients two years post-infection

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Long COVID is associated with persistent neurological symptoms. Among the proposed mechanisms, several vascular hypotheses such as endothelial dysfunction, microthrombosis, and impaired cerebral autoregulation, have been suggested as potential contributors to these long-term effects. In this cross-sectional study, we used arterial spin labeling (ASL) MRI to investigate global and regional perfusion alterations in 186 participants (145 with Long COVID and 41 healthy controls) approximately two years post-infection. Structural and ASL MRI data were processed with the ExploreASL pipeline to derive cerebral blood flow (CBF) and the spatial coefficient of variation (sCOV), a quantitative proxy for arterial transit time (ATT) and an indirect marker of global vascular dysfunction. A multiple linear regression model revealed significantly higher global gray matter sCOV in Long COVID patients ($p = 0.02$), independent of age, sex, and white matter hyperintensity (WMH) volume. At the lobar level, an ANCOVA adjusted for age, sex, and WMH volume showed consistently elevated sCOV in multiple regions, with the left insula remaining significant after FDR correction ($p = 0.01$). No significant differences were found in regional CBF or WMH volume. These results point to increased sCOV as a potential early indicator of diffuse vascular dysregulation in Long COVID, reinforcing the need to consider delayed ATT and systemic cerebrovascular effects in Long COVID neuroimaging research.

S-075

Environmental enrichment delays the onset of cognitive decline in a transgenic mouse model of Alzheimer disease

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Alzheimer disease (AD) is the most common form of dementia in the elderly. Cognitive function can be preserved despite underlying neuropathology. Physical, cognitive, sensory, and social stimulation contribute to this resilience. Environmental enrichment (EE) replicates these effects in animal models through sustained exposure to analogous stimulating conditions. In this study, we assessed spatial learning and memory using the Barnes maze test in 3xTg-AD mice, a transgenic model of AD carrying three key mutated human genes (B6.Cg-Tg(APPSwe,tauP301L)1Lfa Psen1tm1Mpm/J) that typically shows cognitive deficits by 6 months of age. EE was applied to two groups: one up to a presymptomatic stage (4 months, n=10) and another up to a symptomatic stage (6.5 months, n=10). Control mice were housed in standard, non-enriched cages (n=19). At baseline, no significant differences were observed (U=38, p=0.359). After treatment, only the 6-month EE group showed significantly lower escape latency than controls (U=20, p=0.023), indicating better performance. The EE effect hypothesis was confirmed by intra-group analysis (Z=−2.114, p=0.035). These findings suggest that EE can delay cognitive decline in 3xTg-AD mice, highlighting its potential as a non-pharmacological strategy for preserving cognitive function in AD. Further studies aim to explore the CA1-CA3 neuroplastic mechanisms likely underlying this behavioural effect.

S-076

Genome Wide Association Study of Sporadic Alzheimer's disease in the new cohort GeNED.ar

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Sporadic Alzheimer's disease (AD) is the most common form of dementia worldwide, with a genetic component accounting for 80%. To date, 83 SNPs across 75 loci have been identified in individuals of European ancestry. Some of these genetic signals were replicated in a population sample from Argentina (N=1028) and Chile (N=352) by Genome-wide Association Study (GWAS). These GWAS also revealed 15 novel variants with suggestive significance, not reported in Europeans or linked to AD before. With the aim to increase statistical power to validate these potential Latin American signals, we run a new GWAS using the GeNED.ar cohort (Genetics and Neuroimaging of Aging and Dementia in Argentina) with 300 additional samples (88 AD and 212 controls). Samples were genotyped in Germany using the Illumina GSA array. The quality control excluded samples and variants with missingness higher than 3%, excess of heterozygosity rate, sex discrepancies, duplicates, and relatedness (PI-HAT > 0.1875). After QC, 280 samples remained (80 AD and 200 controls). Data were imputed with minimac4 and the TOPMed reference panel via the Michigan Imputation Server. Ancestry analysis revealed an admixture between Indigenous American and European components. Principal components–adjusted logistic regression and meta-analysis with previous results will shed light to better understand the genetic architecture of AD in our admixed population.

S-077

Impact of Ovariectomy on the Progression of Parkinsonism in Middle-Aged Female Rats

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Parkinson's disease (PD) is characterized by the progressive loss of dopaminergic neurons and is more prevalent in men over 60 years. In postmenopausal women, PD often progresses more aggressively, which may be related to the abrupt decline in sex hormones. We investigated the progression of reserpine-induced parkinsonism (RES) in middle-age ovariectomized (OV) rats. Female Wistar rats (n=32, 8 months old) with regular estrous cycles were used. Sixteen animals underwent bilateral ovariectomy, and one month after surgery, the absence of estrous cycles was confirmed by vaginal cytology. Animals were allocated to four groups: intact female (CTL-vehicle), intact females with RES (RES), ovariectomized females (OV-vehicle), and ovariectomized females with RES (OV-RES). Parkinsonism was induced with RES (0.1 mg/kg, s.c., every 48 h for 40 days. Behavioral tests included catalepsy, open field and oral movements (OM) (CEUA: 6628240125). Ovariectomized anticipated and worsened RES-induced motor deficit in catalepsy from the day 24th onward but did not exacerbate locomotor impairment in the open field. In the MO test, increases in vacuous chewing, tongue protrusion and oral tremor induced by RES were not intensified by ovariectomy. In conclusion, our findings suggest that female sex hormones deficiency accelerates catalepsy motor deficit but does not affect other motor impairments in the reserpine parkinsonism model.

Keywords: Parkinsonian disorders; Sex hormones; menopause; reserpine.

S-078

Pathological Tau fibrils induce changes in endogenous TDP-43 protein levels and subcellular distribution in cultured SH-SY5Y neuroblastoma cells.

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TDP-43 proteinopathy, originally discovered in amyotrophic lateral sclerosis (ALS) and frontotemporal dementia (FTD), coexists with tauopathy in a variety of neurodegenerative disorders, including Alzheimer´s disease. However, the role of this co-occurrence in pathological processes is still unclear. In this study, we used transgenic SH-SY5Y-Tau-GFP cells, incubated with pre-formed Tau fibrils (Tau-PFF), to model and investigate the interaction between pathological Tau and endogenous TDP-43. We quantified TDP-43 intensity at cellular, nuclear and cytoplasmic levels using high-content single cell analysis. Tau-PFF treatment resulted in increased cellular, nuclear and cytoplasmic TDP-43 intensity. Interestingly, the nuclear/cytoplasmic (N/C) ratio decreased due to a larger increase in the cytoplasmic compartment, indicating abnormal subcellular redistribution as observed in TDP-43 proteinopathies. We also analyzed the effect of Tau-PFF incubation in cell populations with (+) or without (-) stable Tau-GFP expression. Nuclear TDP-43 intensity increased upon Tau-PFF treatment regardless of Tau-GFP expression. Moreover, we detected an increase in nuclear TDP-43 intensity when comparing Tau-GFP (+) versus (–) cells within both control and Tau-PFF treated groups, with the largest fold-change showed by the control Tau-GFP (-) vs. Tau-PFF Tau (+) comparison. In summary, this data demonstrate a causal role for pathological, fibrillar Tau in regulating TDP-43 levels and distribution.

S-079

Digital Assessment of Mild Cognitive Impairment using Hand Movements during the Trail-Making Test

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Digital neuropsychology uses computational tools to improve traditional assessments, such as the Trail-Making Test (TMT). While the paper-and-pencil TMT primarily measures completion time, digital adaptations enable the collection of continuous behavioural data. These metrics offer new markers of cognitive decline. Following this path, we developed a computerised TMT (cTMT) preserving the original structure while recording high-resolution mouse trajectories across several trials.

Seventy-four older adults (41 with mild cognitive impairment and 33 controls) completed the cTMT, as well as a standard diagnostic battery. We extracted several features from the cursor time series, including reaction times, speed and acceleration metrics, trajectory deviations and state-based measures.

The results showed that demographic models provided only modest discrimination (AUC = 0.56). Digital hand features improved performance (AUC = 0.65), and combining them with demographics reached an AUC of 0.71. Which is closer to the ceil performance (AUC = 0.73) achieved by the neuropsychological battery that was the quantitative part of the criteria to define the classes in the first place.

These findings show that digital biomarkers can achieve performance comparable to standardized tests. This cost-effective cTMT, compatible with hardware available at home or at clinical facilities, will be further validated with multimodal data for the early detection and monitoring of cognitive decline.

S-080

Dissecting striatal heterogeneity with single-nuclei transcriptomics: neuronal and glial signatures in a model of Parkinson's Disease and L-DOPA-induced dyskinesia.

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Previous single-nucleus RNA sequencing (snRNA-seq) analyses from our group in a 6-hydroxydopamine (6-OHDA) mouse model of Parkinson's disease (PD) identified major striatal cell types and their transcriptional responses to dopaminergic denervation and L-DOPA-induced dyskinesia (LID).
In the present work, we aimed to explore with higher detail the transcriptional signatures of direct and indirect spiny projection neurons (SPN), the two major pathways of the basal ganglia circuitry. Given that dSPN from the dorsolateral part of the striatum have been implicated in LID, we refined the previous analysis by subclustering neuronal populations according to their compartmentalization and spatial distribution. By removing residual low-quality nuclei, we obtained a high-resolution characterization of their transcriptional profiles across conditions, focusing on differentially expressed genes (DEGs) and cellular pathways involved in dopaminergic loss and LID.
Data suggest that denervation may reduce dSPN excitability, while increasing iSPN excitability, disrupting direct/indirect pathway balance. L-DOPA seemed to reorganize dSPN transcription, including genes involved in synaptic plasticity, lipid metabolism, and calcium homeostasis, while iSPNs showed reduced synaptic activity and changes in potassium transport. In the poster, we will further discuss the compartment- and subtype-specific transcriptional changes that may help uncover neuronal mechanisms in PD and LID.

S-081

Engineered Tau microRNAs as a potential molecular therapy for atypical parkinsonism

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Abnormal metabolism of the microtubule-associated protein Tau is a key pathological mechanism underlying several neurodegenerative diseases, named tauopathies. Several neuropathological phenotypes are defined according to the specific brain nuclei and cell types affected, as well as the Tau isoforms present in pathological deposits. Progressive Supranuclear Palsy (PSP) is a primary tauopathy classified as an atypical parkinsonian syndrome, mainly affecting the basal ganglia and leading to motor impairment. In this study, we use the htau mouse model of tauopathy, in which the presence of pathological tau leads to striatal dysfunction and motor coordination deficits (Damianich et al 2021). We propose here a molecular therapy based on site directed expression of artificial microRNAs (Tau-miRNAs) to reduce pathological tau and thus improve motor phenotypes. Tau-miRNAs were delivered into the striatum at 6 months old- close to the onset of motor coordination deficit. Tau-miRNA treatment reduced motor coordination impairments in the rotarod at 12 months-old. Ongoing molecular analyses will further demonstrate if this improvement is related to tau reduction.

S-082

Intraneuronal Vacuolization in the Brain of Natural and Social Animal Models: Preliminary Results

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The degu (Octodon degus), a social diurnal caviomorph rodent endemic to Chile, spontaneously develops Alzheimer's disease (AD)-like cognitive deficits, making it a natural model to study links between aging, sociability, and neurodegeneration. It shows 97.5% homology with human β-amyloid and a high misfolding rate (71.4%), suggesting its prion protein may also misfold in vivo. Ecological factors, such as habitat structure and group composition, are associated with brain anatomical variations and hemispheric asymmetry. To build a hippocampal histological atlas, eight adults (4 from El Salitre and 4 from Rinconada, Chile; balanced by sex) were perfused. Brains were stained with Hematoxylin and Erythrosin and examined by light microscopy. Structures and cellular components were delimited using stereotaxic atlases. Preliminary results show cytoplasmic vacuolization linked to neuronal nuclei in hippocampus, thalamus, hypothalamus, amygdala, and deep cortex, with area-dependent differences in number and distribution. In other wild species and humans, vacuolization associates with prion proteins and cognitive impairment, and to a lesser extent with AD pathology. These findings will be validated through immunohistochemistry and guide the search for homologous markers in Argentine caviomorphs such as the guinea pig (Cavia aperea).

S-083

Geraniol Protects Against Proteotoxicity and Neurodegeneration in C. elegans Parkinson's Disease Models

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As life expectancy increases, the prevalence of age-related disorders, including neurodegenerative diseases (ND), is also rising. Oxidative stress (OS) is a key factor accelerating ND progression. For instance, in Parkinson's disease (PD), impaired free radical scavenging promotes α-synuclein (α-syn) aggregation and neuronal damage. Therefore, antioxidant compounds are considered promising therapeutic candidates. In this study, we investigated the biological effects of Geraniol (GER), a plant-derived compound with antioxidant properties.
Caenorhabditis elegans is a valuable model in biomedical research due to its genetic conservation with mammals and ease of manipulation. We evaluated GER's effects in C. elegans PD models and explored the conserved molecular mechanisms involved.
In wild-type animals, GER increased survival under OS, confirming its antioxidant activity in vivo. To elucidate the underlying mechanisms, we used mutants in key stress-response pathways and found that SKN-1/NRF2 mediates GER's protective effect.
Given the connection between OS and PD, we assessed GER in a muscle-expressed α-syn model, where it improved locomotion and reduced protein aggregation. In a neuronal α-syn model, GER significantly preserved dopaminergic neuron morphology and function.
Overall, our results show that GER exerts antioxidant, antiproteotoxic, and neuroprotective effects in C. elegans PD models, supporting its potential as a therapeutic candidate for neurodegenerative diseases.

S-084

Differential Cortical and Hippocampal regulation of neuronal TDP-43 expression and localization in a Mouse Model of Chronic Neuropathic Pain

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Chronic neuropathic pain (NP) is a condition characterized by persistent nociceptive sensitization that often leads to long-lasting cognitive and emotional impairments, believed to stem from changes in brain circuits. In our previous work, we showed that neuronal density, assessed through NeuN immunofluorescence, was largely preserved in cortical and hippocampal regions, suggesting that overt cell loss is not a major driver of NP-associated cognitive/emotional symptoms. To investigate alternative potential neuropathological processes in NP, we analyzed the levels of TDP-43, a protein key for neuronal function that is implicated in neurodegenerative diseases. TDP-43 is a RNA-binding protein that regulates RNA metabolism at multiple levels. Our single-cell analysis of total and nuclear TDP-43 neuronal immunoreactivity revealed a significant increase within the somatosensory cortex (SSC) and hippocampal CA1 area. In contrast, neuronal TDP-43 immunostaining was reduced in the motor (MC), anterior insular (AIC), and prefrontal (PFC) cortices. Furthermore, the nuclear / cytoplasmic TDP-43 ratio was reduced not only in PFC but also in hippocampal CA1 and dentate gyrus (DG) areas, indicating pathological subcellular redistribution. These results uncover a novel region-specific dysregulation of TDP-43 in a chronic NP model, suggesting that cognitive/emotional behavioral alterations might be linked to impaired TDP-43 function due to changes in protein levels and mislocalization.

S-085

Serotonylation as an Early Biomarker for Mental Disorders like Anxiety and Depression

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Psychiatric disorders including anxiety and depression affect a large proportion of the human population. Growing evidence indicates that predisposition to develop mental disorders could have neurodevelopmental origins. However, little is known about the molecular and cellular mechanism underlying such phenomenon. The serotonin (5-HT) system and its homeostasis in early life are essential for establishing proper brain circuit architecture and function. Recent studies described a novel post-translational modification called serotonylation, in which a 5-HT molecule is transferred to a protein glutamine residue. In histone H3, such modification occurs at tri-methylated lysine 4 (H3K4me3)-marked nucleosomes (H3K4me3Q5ser) promoting transcription. We study if this regulatory mechanism could have a role in the early vulnerability of prefrontal cortical circuits resulting in adult emotional alterations. Preliminary analyses show that serotonylation is present in the forebrain during embryonic life, mainly regulating promoters of genes involved in specific neurodevelopmental processes across ages. In a mouse model of early-life emotional vulnerability with decreased levels of 5-HT in prefrontal neurons, a decrease in serotonylation levels affects the expression of gene networks involved in axon development and synaptic maturation. Further studies will determine how this may impact on long-standing prefrontal circuit alterations and emotional symptoms in this model.

S-087

Neural Circuit Interactions and Parkinson's Disease Symptoms in Drosophila melanogaster

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Parkinson's disease (PD), the second most prevalent neurodegenerative disorder worldwide, is characterized by motor impairments primarily linked to the degeneration of dopaminergic neurons in the Substantia Nigra pars compacta. Beyond motor dysfunction, non-motor symptoms such as sleep disturbances represent a significant but less explored dimension of the disease. Given the tight interplay between motor and sleep circuits, we propose that neurodegeneration in one system may precipitate dysfunctions in the other. Using Drosophila melanogaster as a model, we investigate the effects of PD-associated gene deregulation, including mutant α-synuclein, in neural centers regulating sleep and locomotion. We assess neuronal loss, locomotor performance, and sleep architecture in young and aged flies to uncover circuit-specific vulnerabilities and their contribution to disease progression.

S-088

Attentional processing of alcohol-related auditory stimuli in binge drinkers and healthy controls: a behavioral and exploratory fMRI approach

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Binge drinking is a common alcohol consumption pattern in young adults, associated with impairments in cognitive control and salience reactivity. Increased connectivity between reward and salience regions has been reported in binge drinkers, even without meeting criteria for alcohol use disorder (AUD). Early identification of altered neurocognitive mechanisms may inform preventive strategies. The aim of this study was to compare behavioral performance in a dichotic listening task between binge drinkers and healthy controls, and to explore brain activations linked to attentional control in response to alcohol-related auditory stimuli, in order to select regions of interest (ROIs) for examining their relationship with alcohol use levels. Twenty-seven participants were assessed (11 binge drinkers, 16 controls), all with normal psychiatric, auditory, and neuropsychological evaluations. fMRI images were acquired during the task. All participants performed above chance. A repeated-measures ANOVA revealed a main effect of trial type, with shorter reaction times in bottom-up than in top-down trials, without significant group differences. At the neural level, top-down trials elicited greater activation in dorsolateral prefrontal cortex, thalamus, caudate, putamen, and insula. These findings allowed the definition of ROIs for subsequent correlation analyses between alcohol consumption (AUDIT) and brain activation.

S-089

Setting up Neuropixels to study LEC contribution to context-dependent modulation in piriform cortex

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Sensory representations in cortex are shaped not only by stimulus features but also by contextual and behavioral signals. In piriform cortex (PC), neurons initially tuned to odors can become modulated by non-olfactory variables, such as visual context, suggesting associative inputs contribute to this modulation. The lateral entorhinal cortex (LEC) is a candidate source, as it can influence PC microcircuits. To investigate this in vivo, we are developing a recording and manipulation pipeline in head-fixed mice performing a visual–olfactory associative task in virtual reality. Our aim is to simultaneously record from PC and connected areas using high-density Neuropixels probes, and to test causality via transient chemogenetic silencing of LEC or its projections to PC.

Currently, we are optimizing key steps, including surgical planning, probe placement, and overall protocol refinement. Using dummy Neuropixels probes, we are testing insertion strategies and validating histological methods to reconstruct probe tracks. In parallel, we are adapting behavioral protocols to habituate mice to intraperitoneal injections during head fixation and found animals continue to learn under this variation, enabling future targeted interventions during recordings. This work-in-progress provides the technical and behavioral groundwork for future experiments testing whether contextual modulation in PC depends on LEC input, and how contextual signals are integrated into olfactory circuits.

S-090

Inhibition of the oxytocinergic system impairs observational fear learning in mice

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In rodents, social learning refers to situations in which an individual learns from a conspecific in a vicarious manner. This is advantageous, as animals can avoid threatening situations without experiencing them directly. Oxytocin (OXT) is a neuropeptide involved in a wide range of social behaviors, including comfort, social reward, parental care, and discrimination of conspecifics' emotional states. However, the role of OXT in social learning is poorly understood. A useful paradigm to study mechanisms underlying social learning is the observational fear learning (OFL) paradigm, in which a demonstrator undergoes cued fear conditioning while an observer associates the tone and context with the demonstrator's distress, forming a long-term vicarious fear memory. Previous studies have shown that intranasal OXT or chemogenetic activation of hypothalamic oxytocinergic neurons enhances the freezing response of an observer witnessing its demonstrator undergo a fear conditioning. Nevertheless, the role of OXT in stabilizing long-term social learning memories remains unknown. Here, to investigate OXT's role in OFL long-term memory formation, we inhibited the oxytocinergic system either systemically with intraperitoneal OXT receptor antagonist administration or locally through chemogenetic inhibition of hypothalamic paraventricular oxytocinergic neurons.Both approaches significantly disrupted the long-term freezing response, highlighting OXT's critical role in modulating vicarious fear

S-091

Altered Functional Connectivity Across Alzheimer's Disease and Mild Cognitive Impairment: A Resting-State fMRI Study Using Independent Component Analysis

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Alzheimer's disease (AD) is a neurodegenerative disorder characterized by neuronal loss and cognitive decline. Resting-state fMRI enables the assessment of brain alterations by examining large-scale functional networks. In this study, we investigated functional connectivity changes across stages of cognitive impairment using Independent Component Analysis (ICA) and dual regression. fMRI data from 94 AD, 89 cognitive normal (CN), and 249 mild cognitive impairment (MCI) subjects were preprocessed with DPARSF and analyzed with FSL's Melodic to obtain 20 independent components. Dual regression was performed on them to evaluate connectivity differences between groups and two group-level comparisons were carried out: (i) AD vs. MCI vs. CN, and (ii) AD vs. early MCI (eMCI, n=100) vs. late MCI (lMCI, n=55) vs. CN. Voxel-wise statistical tests were run with FSL's Randomise tool, correcting for multiple comparisons. Results showed a trend toward higher functional connectivity in AD relative to MCI (i), and greater connectivity in AD compared with eMCI, as well as in lMCI compared with CN (ii). These findings suggest stage-dependent alterations in resting-state networks, with AD exhibiting enhanced connectivity that may reflect compensatory mechanisms, while the distinct involvement in lMCI relative to CN points to an intermediate position between early impairment and AD.

S-092

Detection of neuronal assemblies in the zebrafish habenula

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Neural activity is not random but displays sparse, organized spatiotemporal patterns, even in the absence of structured external stimulation. Neuronal assemblies, defined as populations of neurons that are co-activated, have been proposed as fundamental units of computations underlying a wide variety of cognitive processes such as perception, memory and behavior. However, little is known about the biological relevance of these assemblies and whether they have a functional significance.

We used confocal calcium imaging to record spontaneous activity in the zebrafish habenula and implemented a previously established PCA-based analysis pipeline to identify neuronal assemblies. We recorded from 18 larval zebrafish between 6-16 days post-fertilisation days old and observed repeated, temporally structured patterns of network activity in the habenula. Using this approach we began probing how assemblies change across early developmental stages and if they are shaped by experience dependent factors such as social isolation. Additionally, we explored how these assemblies are involved in threat detection in response to auditory and visual stimuli.

S-093

Consummatory behaviors induced by growth hormone secretagogue receptor (GHSR) activation depends partially on dopamine neurons activity

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The consumption of energy-dense foods is partially mediated by the activity of midbrain dopamine neurons. This activity is modulated by diverse signals, including gastrointestinal hormones such as ghrelin, a stomach-derived peptide that acts through the growth hormone secretagogue receptor (GHSR). GHSR is highly expressed in various brain regions, including midbrain dopamine neurons, and its activation induces several physiological changes, such as increased food intake and growth hormone release. Here, we aimed to gain insight into the role of dopamine neurons in mediating GHSR-regulated actions. First, we used an inhibitory designer receptor exclusively activated by designer drugs (DREADD) to evaluate the role of dopamine neurons in modulating eating behaviors. We found that inhibition of dopamine neurons reduced the consumption of the non-caloric sweetener saccharin in mice under 40% caloric restriction but did not alter high-fat diet intake in the same mice subjected to a 4-day binge-eating protocol. Next, we tested whether dopamine neuron inhibition affects ghrelin-induced actions. We found that inhibition of dopamine neurons reduced ghrelin-induced locomotor activity but had no effect on ghrelin-induced food intake. These results suggest that activation of dopamine neurons is required for some, but not all, GHSR-mediated consummatory actions.

S-094

Odor–position associative learning gives rise to contextual encoding in the piriform cortex

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Odor processing depends on prior experience, current context and the animal's internal state. To study how learning dynamically changes responses in olfactory cortical circuits, we used an associative learning task in a virtual reality setting. Mice were trained to discriminate one of the four odor-context stimuli to obtain a reward. Previously, we showed that odor-responsive piriform cortex (PCx) neurons become mixed-selective after learning, encoding positional, contextual, and associative information, but how this shift emerges is still unclear. Here, we performed PCx neuronal recordings throughout learning, combining behavioral and neuronal encoding/decoding models to address this question.

We show that animals learn the task sequentially, first discriminating odors (intermediate session) and then contexts (expert session), and found that PCx encoding mirrors learning: intermediate session animals only encode odors, while expert animals encode both odors and contexts. Neuronal decoding analyses confirm this result. Moreover, expert animal neurons showed enhanced multiplexing, compared to previous session animals.

This evidence indicates that the observed contextual PCx encoding depends on the association of odor cues with positional information to produce correct behavioral responses. Ongoing experiments focus on the neural mechanisms by which this association emerges in the PCx.

S-095

Neuronal Circuits Underlying Postpartum Depression

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Postpartum depression (PPD) is a subtype of major depressive episode (MDE) with onset during the peripartum period or within four weeks after childbirth, as defined by the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5). PPD represents the most prevalent complication of the postpartum period and is comorbid with anxiety in approximately 50% of cases. Its estimated global prevalence ranges from 15–25%; however, these figures are considered unreliable, as PPD is not only frequently underdiagnosed and, consequently, undertreated, but also remains understudied despite its high prevalence and substantial impact on both the mother and her offspring. PPD raises suicide risk, impairs caregiving, and affects child social development. . Currently, only two pharmacological agents are specifically approved for the treatment of PPD; however, these are unavailable in our country and are extremely costly. Therefore, elucidating the underlying mechanisms and neuronal circuits involved in PPD is of paramount importance to advancing comprehensive understanding of its pathophysiology and enabling timely diagnosis and effective interventions. Here, we discuss a murine model of PPD to conduct behavioral assessments and to identify specific neuronal circuits implicated in the disorder. This approach will provide mechanistic insights that may inform the development of novel, accessible therapeutic strategies.

S-096

Purinergic modulation of acetylcholine release through P2Y receptors at the efferent-inner hair cell synapse in the developing inner ear

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Before the onset of hearing (postnatal day 12 in mice), inner hair cells (IHCs) are transiently innervated by medial olivocochlear (MOC) efferent fibers and fire spontaneous sensory-independent action potentials that are essential for the normal development of the auditory pathway. Several studies suggest that this spontaneous activity is driven and/or modulated by ATP released from cochlear supporting cells. Our work aims to investigate the role of ATP in modulating MOC-IHC synapses using pharmacology and electrophysiology. IHCs from BALB/C mice at P8-11 were patch-clamped, MOC fibers extracellularly stimulated, and its quantal content (m) estimated. Our results showed that ATP reversibly decreased m in a concentration-dependent manner (1 μM: 90±7 %, 10 μM: 60±7 %; 50 μM: 48±10 %, 100 μM: 48±6 %). Suramin, a non-specific P2 antagonist, abolished the effect of ATP. PPADS, an antagonist with a preferential effect on P2X receptors, and TNP-ATP, a specific P2X antagonist, did not modify ATP-induced inhibition. Furthermore, α,β-MeATP, a specific P2X agonist, had no effect on m. Both non-hydrolyzable ATP analog ATPγS, and the specific P2Y agonist 2-MeSADP, mimicked the effect of ATP (50 μM: 61±13 % and 10 μM: 72±7 % of control m, respectively). MRS2500, a specific P2Y1 antagonist, was used, but the ATP-induced inhibition persisted. Recent RNAseq studies indicate the expression of P2Y12 and 14 in MOC neurons, and thus specific antagonists of these subunits will be evaluated.

S-097

Use of competitive antagonists to asses maximal concentration and time course of glutamate transients at inner hair cells ribbon synapses

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AMPA receptors in the mammalian brain mediate fast neurotransmission and are typically found in the postsynaptic densities (PSDs). Across synapses a great variability of PSDs sizes and number of AMPA receptors has been described . In the mammalian inner ear, glutamatergic synapses are formed between inner hair cells (IHCs) and spiral ganglion neurons (SGNs). All aspects of sound information are encoded and transmitted to the brain through this synapse. A key aspect of the presynapse (IHCs) is the presence of a ‘synaptic body’ or ‘ribbon’ that concentrates large amounts of synaptic vesicles, ensuring high rates of exocytotic events. Postsynaptic terminals of SGNs are characterized by large PSDs, 5 to 10 times bigger than those found in the brain. The role that these large PSDs play in synaptic function is unknown. We speculate that normal neurotransmission activates only a portion of the PSDs area, resulting in non-saturation of the AMPA receptors. To investigate this, we recorded SGNs terminals by patch-clamp technique and used competitive antagonists (CAs) on physiological events and compared them with glutamate photolysis (GP). GP stimulates the entire post-synapse and can be modulated to produce different glutamate transients, making it possible to saturate the receptors. The block by CAs was used to calculate the relative glutamate concentration of physiological events of a wide variety of amplitudes and assess whether they are close to saturation by comparison with GP.

S-098

Presynaptic GABAB Receptors Sustain Transmission at MOC–Hair Cell Synapses

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During development, medial olivocochlear (MOC) neurons transiently innervate inner hair cells (IHCs). After hearing onset (P12–14), they establish permanent synapses onto outer hair cells (OHCs), integrating feedback from peripheral and central circuits. We recently showed that MOC terminals co-release acetylcholine (ACh) and GABA: ACh activates postsynaptic α9α10 receptors, whereas GABA engages presynaptic GABAB receptors that reduce ACh release. Here, we examined how GABAB -mediated inhibition shapes short-term plasticity at MOC–IHC and MOC–OHC synapses. In acutely isolated organs of Corti (P9–P15), IPSCs were recorded from hair cells during electrical stimulation of MOC fibers. In IHCs, 10 pulses at 50 Hz produced synaptic depression (P10/P1 = 0.54). Baclofen, a GABAB agonist, abolished depression (P10/P1 = 1), whereas the antagonist CGP35348 increased depression even at 10 Hz. At MOC–OHC synapses, blocking GABAB reduced facilitation during 50 Hz stimulation (P10/P1control = 1.69 vs. P10/P1antag. = 1.09). These results suggest that presynaptic GABAB receptors minimize depression at MOC–IHC synapses and promote facilitation at MOC–OHC synapses, supporting sustained transmission during high-frequency activity.

S-099

Cannabis sativa extract attenuates motor dysfunction in a progressive animal model of Parkinson's disease

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Natural products have been investigated as potential sources of neuroprotective agents, with Cannabis sativa emerging as a particularly promising candidate. In this sense, the objective was to evaluate the effects of concomitant treatment with Cannabis sativa extract (CSE) in a reserpine-induced parkinsonism model on motor changes. Thus, 35 male Wistar rats were divided into 5 groups (n= 7): CTL, CSE 8.3, RES (0.5 mg/kg), RES-CSE 5.0, and RES-CSE 8.3. The experiment lasted 30 days. In this interval, behavioral tests were performed: catalepsy (every 48 h), oral movements (OM - days 8, 20, and 30), and body mass (BM) was measured every 4 days. All procedures were approved by CEUA, under protocol nº. 1509230425. As a result, it was observed that reserpine administration induced motor changes in all parameters analyzed, and treatment with the lowest dose of CSE (5.0 mg/kg) in catalepsy showed more evident benefits in attenuating motor impairment and muscle rigidity in the animals from the 6th day onwards, with a sustained effect until the end of the experiment. In the OM test, both doses of CSE demonstrated neuroprotective effects in the late phase (30th day) by reducing the number of tongue protrusions and vacuum chewing. Similarly, in the assessment of the ' BM, only in the final phase of the protocol was there a reduction in weight loss, coinciding with the behavioral findings. CSE attenuated motor alterations and reduced late weight loss, suggesting a neuroprotective effect.

S-100

Chronic Administration of Propranolol Promotes Attenuation of Motor Damage and Dopaminergic Protection in a Model of Parkinsonism Induced by Reserpine

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Parkinson's disease (PD) is a motor disorder characterized by dopaminergic and noradrenergic dysfunction, culminating in motor symptoms in non-motor ones. Chronic use of beta-blockers has been associated with an increased risk of PD, but this association may be a causal effect. We evaluated the effect of chronic propranolol administration on motor, non-motor, and immunoreactive changes in a reserpine-induced parkinsonism model. Wistar rats were subjected to chronic administration of propranolol, 10, 20, or 40 mg/kg, subcutaneously (s.c.) daily, for 60 days (CEUA: 6294030423). After 30 days of experimentation, the animals were induced to parkinsonism with reserpine 0.1 mg/kg, s.c. injection every 48h, subjected to behavioral tests and immunohistochemistry for tyrosine hydroxylase (TH). Propranolol, dose-dependently, attenuated the motor impairment in catalepsy and increased the rearing time at a dose of 10 mg/kg. Furthermore, doses of 10 and 40 mg/kg promoted a decrease in oral tremor time and the number of vacuous chewing, and only the 10 mg/kg dose improved the animals' working memory. Conversely, propranolol 20 and 40 mg/kg increased TH immunoreactivity in the SNpc, and only the 40 mg/kg dose in the VTA. Thus, propranolol may alleviate motor deficits and protect against dopaminergic depletion induced by reserpine. Studies are needed to understand the pathophysiological and neuroprotective mechanisms of propranolol associated with PD.

S-101

Biotin-Linked Magnetic Nanoparticles Enable Sensitive and Specific Capture of α-Synuclein Aggregates

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Parkinson's disease (PD) lacks reliable biomarkers for early diagnosis, as current approaches depend on clinical symptoms that emerge only after extensive neurodegeneration. Aggregated α-synuclein (α-Syn), the pathological hallmark of PD, represents a promising biomarker, yet its low abundance and structural polymorphism hinder selective detection.

Previous studies demonstrated that tetracyclines recognize the conserved cross-β motif present in toxic protein aggregates. Building on this, we developed a novel platform based on magnetic nanoparticles functionalized through biotinylation with tetracycline derivatives, designed to enhance both selectivity and sensitivity in the capture of α-Syn aggregates.
Functional assays confirmed that the engineered nanoparticles preferentially bound aggregated α-Syn over its monomeric form, achieving robust discrimination under controlled in vitro conditions. The effective capture surface was markedly increased, leading to a substantial gain in sensitivity while the magnetic properties enabled efficient isolation and concentration of aggregated species, improving the signal-to-noise ratio. This dual advantage establishes the platform as a versatile biorecognition tool compatible with established ultrasensitive methods.

These findings establish proof-of-concept for tetracycline-functionalized magnetic nanoparticles as a new generation of highly sensitive diagnostic platforms targeting α-Syn aggregates, with potential applications in PD.

S-102

Spatial memory dynamics depends on GAT-3–mediated protein synthesis

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Astrocytes are essential components of the tripartite synapse, modulating neuronal communication and plasticity. A key mechanism involves GABA transporter GAT-3, mainly expressed in astrocytes, which terminates GABAergic signaling and preserves homeostasis. Here, we investigated hippocampal GAT-3 in spatial memory dynamics—consolidation, expression, and reconsolidation—using the spatial object recognition (SOR) task in rats. Pharmacological inhibition with SNAP-5114 (SNAP) impaired consolidation. This deficiency was rescued by prior open field (OF) exposure and blocked by the protein synthesis inhibitor emetine, indicating protein synthesis reliance. Pre-treatment with the proteasome inhibitor β-Lactacystin (β-Lacta) also mitigated SNAP-induced deficits. Puromycin incorporation assays confirmed that SNAP reduced hippocampal protein synthesis. Additionally, SNAP before retrieval impaired memory expression, which was restored by OF or β-Lacta. In contrast, SNAP did not affect reconsolidation, a process disrupted instead by a broader GABA transporter blocker: Nipecotic Acid. These findings reveal that astrocytic GAT-3 selectively modulates memory consolidation and expression, but not reconsolidation, through protein synthesis. Thus, GAT-3 emerges as a pivotal astrocytic regulator of memory dynamics and a potential therapeutic target in disorders with altered GABAergic signaling and cognitive decline, such as epilepsy, Alzheimer's disease, and other neuropsychiatric conditions.

S-103

Do oral cannabis oils promote neurogenesis and ameliorate associated behaviors in mice?

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Cannabis-derived oils are used in clinics as alternative treatment for anxiety, depression, and stress, reporting significant improvements in the quality of life. Similarly, in murine models, chronic administration of pure cannabidiol (CBD) has shown marked improvements in anxiety- and depressive-like behaviors, effects partly attributed to a proneurogenic action in the dentate gyrus (DG) of the hippocampus.

As cannabis oils are complex mixtures of cannabinoids and terpenes, and can yield an “entourage effect”, we aim to evaluate the impact of chronic administration of cannabis oils with different compositions on several behaviors, as well as the relevance of adult hippocampal neurogenesis in these phenomena. To this end, 4 different groups of 8–9-week-old, C57BL/6 mice, will receive a daily oral administration of CBD-, CBDA-, CBDV- enriched oils or vehicle for 6 weeks. Potential anxiolytic effects will be studied by means of the Elevated Plus Maze and the Novelty-Suppressed Feeding test. Anhedonic behaviors will be analyzed by the Splash test, and memory abilities will be evaluated by the Novel Object Recognition test. In addition, neuronal proliferation as well as survival of 3-week-old neurons in the DG will be quantified at the end of the 6 week-protocol. Based on the obtained results, we plan to use Nestin-Cre transgenic mice to ablate neural precursors and determine the relevance of this cell population on the observed behaviors.

S-104

DDOX, a novel non-antibiotic tetracycline derivative, protects against α-Synuclein aggregation, fibril uptake, and toxicity in Parkinson's disease

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Parkinson's disease (PD) requires multi-target therapies to mitigate the toxicity of α-synuclein (α-Syn) aggregation in the brain. Tetracyclines, particularly doxycycline, have demonstrated multimodal neuroprotective effects, both in vitro and in vivo. The non-antibiotic derivative of doxycycline 4-dedimethylamino-12a-deoxydoxycycline (DDOX), has been recently shown to rescue neurons from oxidative injury. Here, we demonstrate that DDOX showcases a diverse range of mechanisms targeting α-Syn aggregates. Notably, DDOX inhibited the aggregation of αSyn and the seeding ability of α-Syn pre-formed fibrils (PFF) in biophysical and cellular assays. In addition, the compound ameliorated total and phospho-α-Syn relocalization, triggered by exogenous α-Syn PFF. Surprisingly, DDOX drastically mitigated lysosomal stress induced by these aggregates. Moreover, we determined that DDOX effectively impeded the internalization of fluorescently labeled α-Syn PFF. Biophysical techniques and molecular docking simulations suggest that DDOX binds to hydrophobic patches on α-Syn fibrils. Our findings reveal novel neuroprotective attributes of tetracyclines, wherein a direct extracellular interaction between DDOX and α-Syn aggregated species mitigates their intracellular impact. These results provide a promising foundation for DDOX, a drug that aims at interfering with the intracellular seeding, propagation and uptake of α-Syn fibrils in neurodegenerative conditions.

S-105

Hearing Impairment and Cochlear NLRP3 Inflammasome Upregulation Induced by High-fat Diet Consumption in Mice

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Obesity affects the auditory system, altering its functionality and auditory processing. Inflammation and the NLRP3 inflammasome may play a role in the development of sensorineural hearing loss (HL) induced by obesity. We aimed to evaluate the effect of obesity induced by high-fat diet (HFD) consumption on the functionality of the auditory system, hair cell survival and inflammation. For that, 7 weeks old male C57BL/6J mice (n=20) were fed a control diet (CD, 10% fat, 20% protein, and 70% carbohydrates) or a HFD (60% fat, 20% protein, and 20% carbohydrates), for 16 weeks. Weight, adipose tissue (AT) and liver histology, distortion product otoacoustic emissions (DPOAEs), auditory brainstem response (ABR), cochlear hair cells numbers and integrity (IF), proinflammation cytokines (interleukin (IL)-6 and TNF-α (qPCR), and NLRP3 inflammasome (NLRP3, ASC, Caspase-1, IL-1β and IL-18, IF and qPCR) were measured. Animals fed a HFD ($p<0.05$) increased body weight (84%), developed hepatic steatosis, AT dysfunction, compared to CD. Moreover, HFD increased the 2F1-F2 DPOAE threshold by 12,3 dB ($p<0.05$) and decreased cochlear outer cells number. Cochlear mRNA levels of IL-1β, IL18 and IL-6 were also increased. This data suggests a role for inflammation and NLRP3 inflammasome in hearing impairment induced by metabolic dysfunction, after the chronic consumption of a high-fat diet in C57BL/6J mice.

S-106

Glucocorticoids modulate endocytic activity of hypothalamic tanycytes in vitro

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Hypothalamic tanycytes are polarized ependymoglial cells that line the base of the third ventricle and extend their processes into the hypothalamic parenchyma and the median eminence, establishing contacts with blood vessels and thus forming an anatomical interface for the transport of molecules between the blood and the cerebrospinal fluid. This interface may undergo structural and functional remodeling in response to the endocrine state, potentially altering its capacity for hormone transport. We recently demonstrated that tanycytes internalize and transport the orexigenic hormone ghrelin via clathrin-mediated endocytosis. Here, we analyzed the effects of glucocorticoids on tanycyte endocytic activity in vitro. Primary cultures of rat hypothalamic tanycytes were treated for 48 h with either vehicle, 0.1 μM dexamethasone (Dex), or 0.1 μM corticosterone (Cort), in the presence or absence of the glucocorticoid receptor (GR) antagonist RU-486 or the mineralocorticoid receptor (MR) antagonist spironolactone. Immunocytochemistry revealed GR and MR expression in the cytoplasm and only Dex induced GR nuclear translocation. Both Dex and Cort increased uptake of fluorescent ghrelin and fluospheres, an effect reversed by RU-486 and spironolactone. We conclude that glucocorticoids enhance tanycyte endocytic capacity in vitro, likely via GR activation. Future studies will address the cellular mechanisms involved and the impact of glucocorticoids on tanycyte-mediated transport in vivo.

S-107

Thyroid hormones and neurogenesis: modulation of quiescence and activation in hippocampal Neural Stem Cells

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Introduction: the adult hippocampal neurogenic niche relies on the activation of reversibly quiescent neural stem cells (NSCs). Thyroid hormones are critical regulators of neurogenesis, but their role in balancing NSC quiescence and activation has been little explored. Objective: this study examined whether thyroxine (L-T4) modulates this balance. Methodology: NSCs were isolated from 30-day-old C57BL/6J mice, cultured as neurospheres, and treated for 72 h with 1 nM or 5 nM L-T4 under two conditions: proliferation (FGF2, 20 ng/mL) or quiescence (FGF2 + BMP4, 30 ng/mL). The percentage of positive NSCs expressing Nestin, Ki67, and cyclin D1 was analysed. In proliferation conditions, both T4 concentrations increased Nestin+ cells without altering Cyclin D1, while 5 nM T4 reduced Ki67+ cells. In quiescence conditions, both doses decreased Nestin+ and Ki67+ cells, and 5 nM T4 increased Cyclin D1+ NSCs. Conclusion: These findings indicate that NSCs are sensitive to T4 levels in both proliferative and quiescent states. T4 promotes NSCs identity under proliferation conditions, but high T4 reduces proliferation, potentially preserving the NSCs pool. Under quiescence conditions, T4 may either prolong the cell cycle and reinforce quiescence or induce loss of stemness and differentiation. Altogether, our data suggest that thyroid hormones may be key modulators of the quiescence–activation balance, essential for sustaining hippocampal neurogenesis.

S-108

Connectivity of central canal neurons in the mouse brainstem

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Cerebrospinal fluid-contacting neurons (CSF-cNs) are a distinct medullospinal population strategically located around the central canal. In zebrafish, they sense spinal curvature and CSF composition, but their role in CNS circuitry of tetrapods remains unclear. In this work, we mapped the CSF-cNs network using Pkd2l1Cre mice to selectively label their cell bodies, axons and synaptic terminals. We confirm their presence along the brainstem central canal, extending up to the fourth ventricle. Their axons project to several brainstem areas, including the hypoglossal (nXII) and Roller nucleus. CSF-cN axons wrap around the nXII in a remarkably precise manner, while also innervate the core of the Roller. XII motoneurons modulate tongue movement and are output of the rhythmic network that control breathing. Opposing, Roller nucleus has been poorly studied. The analysis of genetic Synaptophysin-Tomato puncta identified that CSF-cNs form profuse GABAergic synapses onto ventral nXII motoneurons and Dbx1-derived Roller neurons. Moreover, optogenetic stimulation of CSF-cNs evoked inhibitory postsynaptic currents in Roller neurons, confirming functional connections. We propose that CSF-cNs may regulate respiratory activity through these nuclei, according to CSF homeostasis. Altogether, our results uncover a novel connectivity map of CSF-cNs in the mammalian brainstem, enlightening previously unknown circuits that modulate autonomic functions.

S-109

Discriminating individuals and modeling vocal dynamics in the Rufous Hornero

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The Rufous Hornero (Furnarius rufus), a highly abundant suboscine and Argentina's national bird, has a song long considered innate, but it displays a higher degree of complexity than typically observed in this clade. Here, we examined the acoustic properties of Hornero songs to test whether syllables carry individual signatures. Using quantitative acoustic analyses and machine learning, we trained Siamese Neural Networks on spectrotemporal representations of the fundamental frequency to classify individuals. Our results show that female horneros produce identifiable acoustic signatures, whereas male songs lack sufficient distinctiveness for reliable classification. Detailed spectrographic analyses of female syllables, combined with biomechanical modeling of the sound source, suggest fine motor control as the basis of these individual acoustic fingerprints. These findings highlight the importance of studying vocal production and neuromuscular control in suboscines, particularly the role of individuality in song.

S-111

Remote sensing from the field to the lab: environmental modulation of exploration in weakly electric fish

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Exploratory behavior is essential for efficient niche exploitation. South American weakly electric fish are receptive to the self-generated electric discharges (EODs) they use to probe their surroundings. Exploration requires both EOD emission and locomotor activity. Gymnotus omarorum is a nocturnal inhabitant of turbid waters. 1m2 territories are stablished under a very thick floating vegetation which interferes with natural light. Animals display a circadian rise in EODr that persists even in constant darkness, whether due to vegetation cover or laboratory free-running conditions. Exploratory behavior has been studied mainly through novelty responses in restrained animals and links between EODr modulations and locomotion remain scarce. Using remote sensing, we analyzed electric and locomotor behavior in unrestrained animals across three complimentary conditions: i) undisturbed populations in the wild, ii) semi-natural settings with individual multi-day recordings, iii) a controlled setup where novel stimuli is presented in a controlled fashion. This three-tier approach shows that: animals in the wild show nocturnal increases in EODr and locomotor activity, strong territorial fidelity and preference for low-light areas. In seminatural conditions exploration has a daily pattern shaped by environmental cues. Finally, a minimalistic behavioral set up allowed us to asses the minimal conditions needed to elicit exploration.

S-112

Two polarization channels provide a visual stimulus specific fingerprint

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When animals are faced with visual stimuli that indicate an imminent threat, they commonly produce a defensive response. Both figure contrast polarity and motion have long been found to be used by vertebrate and invertebrate species to recognize visual threats. The polarization of light has also been shown to contribute to contrast sensitivity in many aquatic and intertidal animals. However, whether polarization and luminance are processed jointly or in parallel visual channels remains unresolved. We developed a massed adaptation–recovery paradigm (29 trials of training, 5 trials of testing) in the crab Neohelice granulata with different looming stimuli. We switched luminance polarity (OFF↔ON), motion direction (Right↔Left) and angle of polarization (AoP) while holding intensity and degree of linear polarization fixed (Vertical↔Horizontal). By analyzing both the kinematics of the escape response and the proportion of trials that evoked escaping, we found that the response recovered after every switch. Particularly, the recovery after the polarization swap indicates that vertical and horizontal polarization signals adapt separately, rather than acting as a single polarization contrast channel. These results support a parallel-channel account in which polarization and luminance are at least partially independent for threat detection.

S-113

Against the Tide: Evolutionary Reduction of the Neocortex in New World Monkeys — Phylogenetic Patterns and Genomic Correlates

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Primate brain evolution shows progressive enlargement, yet some lineages exhibit striking reductions in relative neocortex size. These cases remain poorly understood but can illuminate the constraints shaping neural evolution. Using phylogenetic modeling, we investigated neocortex evolution in New World monkeys and found the strongest reduction in marmosets, tamarins, and owl monkeys—small primates with rapid life histories, consistent with evolutionary miniaturization. To investigate the genomic correlates of relative neocortex reduction, we scanned >11,000 protein-coding orthologous genes using selection tests. aBSREL identified 47 genes under positive selection in the lineage, in neurodevelopment and growth regulation (e.g. LHX3, EPHB2, PDE2A). Complementary analyses using RELAX tests revealed 67 genes with intensified selection, enriched for brain expression, including DISC1 (neuronal migration), LAMC3 (cortical patterning), NEPRO (neural progenitor regulation), and RTTN (microcephaly). These findings reveal complex primate brain evolutionary trajectories. Molecular signatures indicate neocortical shrinkage was not a passive consequence of body size reduction, but an active evolutionary process with potential cognitive implications. Detection of pronounced neocortical reduction in marmosets—an important neuroscience model—provides new context for interpreting their neural organization.

S-114

Toward the atoms of behavior: Decomposing birdsong into excitable motor primitives

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The production of birdsong involves complex respiratory motor gestures shaped by precise coordination of neural and muscular systems. In this talk, I'll present a framework for understanding birdsong as a sequence of simpler motor instructions, each generated by a minimal excitable system. Using air sac pressure recordings from singing canaries, we model individual syllables as sequences of transient responses of a two-dimensional Wilson–Cowan-type system. By fitting these transients to observed pressure patterns using a differential evolution algorithm, we obtain reconstructions of the motor patterns underlying song with high fidelity. We then apply unsupervised dimensionality reduction and clustering to the extracted transients, identifying a compact set of shared motor primitives across birds with different vocal learning histories. This suggests that birdsong is built from a reusable repertoire of dynamical modules, shedding light on the structure of learned motor behavior and its neural underpinnings. I'll discuss how this approach opens new avenues for studying motor control and learning in a broad range of behaviors.

[1] Agustín Carpio Andrada and Gabriel B. Mindlin. "Decomposition of respiratory motor patterns during birdsong production in terms of excitable transients." Chaos, Solitons & Fractals, 2025

S-115

MEC ultraslow oscillations enhance episodic memory recall within the hippocampal spatial scaffold: a computational study

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Episodic memory relies on coordinated neural dynamics across time and space, supported by a spatiotemporal scaffold in the hippocampal–entorhinal network. This scaffold aims to sustain episodic memories and originates from grid cell modules with toroidal dynamical states, which are connected to hippocampal cells and sensory-processing inputs. Low-dimensional velocity signals shift grid phases, while grid–place cell interactions encode and retrieve spatial–episodic information. Beyond spatial coding, a minute-scale (<0.001 Hz) ultraslow oscillation in the medial entorhinal cortex (MEC) has been identified during 1D walk with mice running on a rotating wheel, though its behavioral-scale impact remains unclear. We developed an entorhinal–hippocampal model that incorporates this oscillation during spatial exploration in a 2D arena. Using computational simulations through detailed numerical investigations, results showed that the ultraslow oscillations synchronize entorhinal inputs, stabilize hippocampal spatial scaffolds, and enhance associative binding. Without them, input timing desynchronizes, spatial scaffolds destabilize, and the formation of episodic memory degrades. We discuss that when adding slow-oscillations, spatial–temporal recall accuracy improves. This work bridges theoretical and experimental findings, revealing how slow MEC rhythms integrate temporal and spatial organization to support episodic and associative memory.

S-116

Towards unsupervised signal modulations identification in individuals with epilepsy treated with responsive neurostimulation

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Neurostimulation is becoming a more popular treatment approach in individuals with drug-resistant epilepsy. One neurostimulation option is the responsive neurostimulation (RNS), a closed loop interface that monitors the electrical brain activity and applies local electrical current when seizure-like patterns are detected. Changes at the time-frequency domain of the ictal putative signal were described by visual inspection of expert epileptologists. Some of these electrographic seizure pattern modulation (ESPM) showed to be correlated with clinical improvements. Due to the large amount of data and scarce of experts' time, there is a need to develop unsupervised methods to identify ESPM with high precision. For this, we evaluate the capability of one class support vector machine (OCSVM) classifiers ensemble to detect ESPM. We tune the OCSVM hyperparameter and accelerate the training process using a kNN and data density-based method. As data input we use different signal representation approaches such as short-time Fourier transform spectrogram, a widely adopted electrophysiology time-frequency representation, and scattering transform, a non-linear signal representation method that uses cascading wavelet modulus decomposition followed by a low pass filter. Finally, we compare the classification metrics of the unsupervised approach with few expert labeled signals and generalize the result to signals not labeled by the expert.

S-117

Synaptic Plasticity and Functional Connectivity in Cortical Networks: A Graph Theory Study Applied to Epilepsy

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Synaptic plasticity, the activity-dependent modification of neural connections, is a fundamental mechanism underlying learning, memory, and pathological brain states. Epilepsy, a chronic neurological disorder affecting 0.8% of the population, often proves drug-resistant, with half of treated patients experiencing persistent seizures and progressive cognitive decline.

This study investigates how functional connectivity (FC), derived from EEG signals and modeled via graph theory, varies between basal, preictal, and postictal brain states to improve preictal state prediction. The research is grounded in the principle that synaptic plasticity modulates network strength based on use, favoring efficient, metabolically economical information transfer between frequently synchronized nodes. However, it is hypothesized that in epilepsy, this same mechanism may lead to pathological hypersynchronization.

We explore the dynamics of cortical networks by analyzing functional connectivity graphs, testing the premise that preictal states are characterized by a measurable reorganization of network properties, reflecting aberrant plasticity processes. This work aims to provide a novel, network-based biomarker for seizure prediction by quantifying these critical transitions in brain dynamics.

S-118

Linking Gamma-Band Neural Activity to Visual Features in Natural Image Processing

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Visual gamma oscillations have been associated with low-level visual features, including contrast, colour, and size, which are highly dependent on the stimulus itself. These relationships have been characterised for artificial, isolated stimuli. Representational Similarity Analysis (RSA) has emerged as a powerful tool to correlate features or measurements from different systems that would otherwise be difficult to compare. Using natural images, we observed robust increased gamma-band activity originating bilaterally from the visual cortex (V2 region). We applied RSA to investigate possible links between neural activity in the gamma-band and image features at the level of individual fixations. Participants performed a hybrid visual and memory search task whilst eye movements and neural signals were recorded non-invasively using magnetoencephalography (MEG). For each participant, we constructed representational dissimilarity matrices (RDMs) based on gamma-band neural responses after a fixation, and convolutional neural networks (CNN) feature maps of the fixated regions. Our goal was to assess whether the structure of neural activity across fixations to different items mirrors the representational structure of visual features. Preliminary results from gamma-based RDMs show limited differentiation across fixations, highlighting challenges in linking neural responses to specific visual features.

S-119

Decoupling plasticity from post-sinaptic activity improves generalization in a one-shot, continual associative-memory task.

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Hebbian plasticity relies on the correlation of pre- and post-synaptic activity for the potentiation of synaptic efficacies, and is thought to be essential for learning and memory formation. However, recent experiments suggest post-synaptic firing might not be required for plasticity to take place. Additionally, classical Hebbian models restrict plasticity to a learning phase and assume fixed synaptic efficacies during memory retrieval - an assumption not supported by experimental observation.

We propose a minimal, biologically plausible model that decouples plasticity from post-synaptic activity, and optimize its parameters to solve a simple, continual associative memory task. Particularly, during a trial, pairs of one-hot stimuli are presented for acquisition, and the model is expected to retrieve both when one of them is shown.

The model outperforms equivalent models with Hebbian plasticity and Recurrent Neural Networks, even at acquiring and retrieving pairs of stimuli not shown during training, an example of compositional generalization.

Taken together, our results suggest that decoupling plasticity and post-synaptic activity could be essential for fast, flexible and biologically plausible learning.

S-120

Semantic and social networks of the emergence of neurosciences in Argentina (1980-2020)

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This study traces the socio-epistemic transformations of neurosciences in Argentina through a bibliometric approach. Metadata of international publications in neurosciences with at least one author with Argentine affiliation for the period 1980-2020 were retrieved from Scopus. They were analyzed using the Cortext platform with algorithms for period selection, term extraction, Sankey diagram, and network construction. From this, semantic networks and social networks were built, representing the conceptual structure and social structure, and their transformations in three different periods (1980-1991; 1992-2006; 2006-2010). In addition, the results were triangulated with document analysis and interviews. The results allow to observe the emergence of neurosciences as a differentiated discipline, the integration of different disciplines and lines of research, such as the integration between laboratory studies and clinical studies, the shift towards neurodegenerative diseases, the import of scientific agendas, the centrality of technological developments, among other key phenomena. These findings provide a nuanced understanding of the socio-epistemic evolution of neurosciences in Argentina, highlighting key drivers and trends that shaped its development.

S-121

Unsupervised Classifier for Sleep Pattern Studies

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This study introduces an unsupervised classifier designed to analyze intracranial electroencephalography (iEEG) signals during sleep, aiming to uncover intrinsic patterns and transitions between sleep phases without predefined labels. Employing an iterative self-classification approach, the model begins with random label assignments to signal segments and refines both the model and labels through mutual feedback, enhancing the identification of homogeneous classes based on power spectra features. Dimensionality reduction via UMAP facilitates visualization of class distributions in latent space, revealing relationships among sleep states such as REM and non-REM. Results demonstrate channel-specific temporal classifications of sleep states, clustered spectral averages, and comparisons with established hypnograms, highlighting the tool's efficacy in characterizing brain activity variations and potential applications for detecting pathological alterations in sleep disorders.

Poster Session 3

D-001

Profiling peripheral glial cells from human nerves for grafting in the CNS

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The regenerative capability of PNS cells, including Schwann cells (SCs) has been exploited clinically in cell transplantation therapies to treat CNS trauma and neurodegenerative diseases.However, the characteristics of peripheral nerve cells has not yet been addressed thoroughly in humans. The goal of this study was to identify specific markers able to reveal the identity and stage of differentiation of cells from intact and injured human nerves. Therefore, we developed and validated an in vitro model of human nerve degeneration to be compared with injured nerves from participants enrolled in a nerve transplantation clinical trial for Parkinson's disease. Histological analysis revealed that: (1) NGFR was a reliable marker to discriminate PNS cells from CNS neurons and glial cells; (2) S100B, GFAP and Sox10 were useful to specifically identify SCs within nerve tissues, with the caveat that they also revealed glial populations in the CNS; and (3) MPZ and PRX were equally useful to identify myelin sheaths derived from SCs rather than oligodendrocytes. To conclude, these markers can be used in different combinations to reveal grafted PNS cells, mainly SCs, in the human CNS to study their survival, differentiation and relationship to host tissue.

D-002

FPR2/ALX receptor ligand, Lipoxin A4, orchestrates resolution responses under pesticide-induced neurotoxity

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An important component in the pathogenesis of neurodegeneration is chronic neuroinflammation. This complex entity is established when resolution processes fail to limit pro-inflammatory stimuli, and it is an important triggering factor for neuronal death. The inflammation/resolution balance is governed by the activation of G protein-coupled receptors (GPCR) by specific ligands. In the central nervous system, these mechanisms have been poorly described, since GPCR ligands responsible for triggering resolution responses are negligible and elusive lipid compounds. Thus, we aimed to investigate whether resolution mechanisms operated as an early response in neurons and astrocytes to overcome pesticide-induced neurotoxicity. We found that astrocytes rescued neurons from pesticide-induced death. Using the endogenous FPR2/ALX agonist, lipoxin A4, and its antagonist, Quin-c7, we demonstrated the involvement of this GPCR receptor and its lipid ligand as responsible for astrocyte-induced neuroprotection. In addition, pesticide-exposed neurons were able to promote a proliferative A2 phenotype in astrocytes, a process that was also dependent on FPR2/ALX activation. These results revealed that neuronal fate upon pesticide-induced toxicity relies on resolution mechanisms triggered by lipoxin A4.

D-003

Low-Protein Diet induces sex and region-specific changes on Astrocytic GFAP expression in Hippocampus of Mouse Offspring

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Maternal protein malnutrition has lasting effects that negatively impact brain development and offspring behavior, increasing the risk of anxiety and depression, altering stress coping. It has been described that malnutrition increases the risk of deregulated immune response leading to neuroinflammation, but little is known about the role of astrocytes in response to early life adversities. Astrocytes serve many basic roles for brain functioning and are unique in their capacity of sensing and integrating environmental signals. To understand the role of astrocytes in a model of perinatal protein malnutrition, we investigated whether astrocytes are involved in the response to early-life adversities.
In this study, we examined GFAP+ area in the hippocampus of both female and male mice exposed to a low (8% casein, LP) or normal protein diet (20% casein, NP) during gestation and lactation. We performed an IHC against GFAP in free-floating sections and analyzed the intensity and area of GFAP+ staining in the hilus and molecular layer of the hippocampus. Results show less area covered by astrocytes in both regions in LP-group compared to the NP-groups only in female offspring to P21. No significant differences were observed in GFAP intensity in female, nor in GFAP intensity or area in male offspring at P21 in either regions. These results contribute to the understanding of malnutrition impact on astrocytes and remark the region and sex dependent nature of stress response.

D-004

Social and material deprivation alters brain development and social behavior through sex-dependent mechanisms

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Early-life adversities are a major risk factor for later mental health disorders, but the neurobiological mechanisms remain poorly understood. To address this gap, we established a multidimensional murine model of social and material deprivation (SMD) that combines reduced nesting, maternal separation, early weaning, and exposure to social stress. This paradigm aims to better reproduce the complexity of socioeconomic disadvantage in humans. Offspring exposed to SMD model displayed delayed growth, heightened anxiety- and depression-like behaviors, increased aggression, and impaired social cognition. Structural analyses revealed reductions in dorsal and ventral hippocampal area, along with an enlargement of the prefrontal cortex. Transcriptomic profiling of the prefrontal cortex uncovered sex-specific signatures: males showed upregulation of genes linked to neuronal development and immune function, while females exhibited increased expression of chromatin remodeling genes and downregulation of immune-related pathways. Altogether, these findings indicate that SMD disrupts brain development and social behavior through sex-dependent molecular mechanisms, with neuroinflammation and epigenetic regulation emerging as potential contributors.

D-005

Sex differences in GABAA receptor response of mouse hypothalamic neurons are independent of gonadal hormones

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The brain is a sexually dimorphic organ, with many sex differences arising during development due to the effect of gonadal hormones from embryonic day (E) 17. In this context, GABA exert trophic effects on immature neurons through GABAA receptors, whose functionality depends on its subunit composition and intracellular chloride levels. While many sex differences in GABA-mediated responses are attributed to gonadal hormones, few studies have examined potential differences prior to the critical period of brain masculinization. In this study, we investigated the functionality and expression of the GABAA receptor in sex-segregated hypothalamic neurons obtained from E15 mice and cultured for 3 days in vitro. Functional activity was assessed through calcium imaging by measuring the number of responsive neurons, amplitude, response kinetics, and decay following GABA exposure (10 µM). In parallel, qPCR analysis was performed to evaluate the mRNA expression of Nkcc1, Kcc2 (chloride co-transporters), and ε subunit receptor gene. We observed that female neurons reached their GABA response peak amplitude faster, with a greater proportion exhibiting fast decaying response compared to males. Gene expression analysis revealed no significant differences. These findings suggest that GABA responses are sexually dimorphic even in the absence of gonadal hormones, although this effect does not appear to be attributable to differences in the expression of Nkcc1, Kcc2 or GABAA receptor ε subunit.

D-006

ERK dimerization in the nervous system of the crab Neohelice granulata

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The ERK/MAPK signaling pathway is crucial for memory formation and maintenance across species. Although much is known about ERK phosphorylation, recent hypotheses suggest a potential role for ERK/MAPK dimerization, primarily described in cancer research. However, no studies have demonstrated this mechanism in the nervous system or explored its implications. Our group has recently shown that this mechanism is critically involved in memory reconsolidation in mice.

In the crab N. granulata, two-trial long-term memory (2t-LTM) requires ERK phosphorylation. Preliminary results from our group suggest that ERK dimerization would be evolutionarily conserved in crabs and may also regulate 2t-LTM. However, detecting ERK dimerization in its nervous system has proven challenging. To address this, we developed a simple ex vivo assay to evaluate the effects of different stimuli and of the dimerization inhibitor DEL-223792 (DEL) on this molecular mechanism.

Together, these findings and the methodological approach provide a useful platform to investigate ERK dimerization and its role in memory processes. This experimental framework could also be employed to test different drugs or neuroactive compounds.

D-007

Neuroanatomical and functional analysis of AgRP neurons' involvement in the orexigenic effect of ghrelin

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Agouti-related peptide (AgRP)-expressing neurons of the hypothalamus are the main neuronal population that mediate the effects of ghrelin, an appetite-stimulating peptide secreted from the stomach. Deletion of ghrelin receptor (GHSR, for growth hormone secretagogue receptor) from AgRP neurons blocks, whereas the expression of GHSR exclusively in AgRP neurons restores, ghrelin's orexigenic effects. However, a thorough analysis of the hypothalamic neuronal subpopulations of ghrelin sensing neurons is still missing. Here, we first performed a detailed neuroanatomical analysis of the distribution of AgRP neurons that bind ghrelin in brain slices of mice that express Td-Tomato fluorescent protein exclusively in AgRP neurons that were centrally or peripherally administered with a fluorescent ghrelin analog. Using c-Fos expression, we also analyzed the distribution of AgRP neurons activated by ghrelin administration. Since previous results of our laboratory indicated that ghrelin administration triggers a sustained activation of AgRP neurons, we investigated the role of this effect on the orexigenic effect of ghrelin. We used mice expressing an inhibitory DREADD to analyze the involvement of AgRP neurons activation on the prolonged orexigenic effects of ghrelin. Our results suggest that ghrelin sensitive and ghrelin responsive AgRP neurons display a similar distribution in the mouse brain and that the blockade of AgRP activity abolishes the prolonged orexigenic effect of ghrelin.

D-008

Early-Life Nutritional Imbalance Shapes Hypothalamic Sensitivity to Obesogenic Diet in adulthood.

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Small-litter and cafeteria diet (CAF) models are useful tools to study obesity at experimental level. Our objective was to assess the effects of neonatal overfeeding (NO) and adult exposure to CAF on the brain homeostatic system that regulates food intake. Male Wistar rats were raised in small (4 pups/dam, SL) or normal litters (10 pups/dam, NL), from weaning to postnatal day (PND) 90 they were fed a control diet (CON). Then, animals received CON or CAF (NL-CON, NL-CAF, SL-CON, SL-CAF; 12±2 rats/group) for 11 weeks. Food intake, body weight and naso anal length were measured weekly until brain collection at the end of the experiment. Arcuate nucleus (ARC) was isolated by micro-punch technique, RT-qPCR was conducted to assess the expression of Agouti-related protein (AgRP), Neuropeptide Y (NPY), Cocaine- and amphetamine-regulated transcript (CART), Proopiomelanocortin (POMC), insulin (IR), ghrelin (GHSR) and leptin (ObRb) receptors. Neonatal overfeeding and CAF diet increase Body Mass Index and alter dietary preferences. At transcriptional level, early overnutrition impaired hypothalamic Pomc expression in ARC through an epigenetic mechanism. On the other hand, NPY expression is decreased by neonatal overnutrition, apparently responding to peripheral stimuli. Leptin receptor expression is reduced because of the CAF diet. These findings show that events experienced during early life program lasting alterations in the response to an unhealthy diet in adulthood.

D-009

Astaxanthin derived from freshwater crustaceans has beneficial effects on cortical metabolic disorders and cognitive decline in a rodent model of Metabolic Syndrome

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Metabolic abnormalities in rats fed a high-sucrose diet (HSD)—a well-established model of Metabolic Syndrome (MetS)—are accompanied by cortical alterations and cognitive decline. This model is valuable for studying potential neuroprotective interventions in MetS-related brain disorders (MSRBD).
In this study, we evaluated the neuroprotective effects of astaxanthin (ASTX) -a powerful antioxidant- derived from freshwater crustaceans in a rodent model of MetS. Male Wistar rats were fed for 90 days with either a standard commercial rodent diet, a HSD, or a HSD supplemented with an ASTX-rich extract (10 mg/kg body weight/day, administered orally). We conducted the novel object recognition test (NORT) and T-maze memory tasks. Additionally, in the cerebral cortex, we measured: (a) proteins involved in energy metabolism and the insulin signaling pathway; (b) myeloperoxidase (MPO), inducible nitric oxide synthase (iNOS), and chlorotyrosine (Cl-Tyr)- and nitrotyrosine (NT)-modified proteins; and (c) acetylcholinesterase (AChE) activity.
Compared with HSD-fed rats, HSD+ASTX-fed rats showed (a) improved cognitive performance in both memory tasks; and (b) in the cerebral cortex, increased levels of GLUT-3, hexokinase, total AMPK, and pThr172AMPK levels, along with reductions in MPO, Cl-Tyr, and AChE activity. Levels of pAKT, iNOS, and NT were elevated in both groups. Our results suggest that ASTX could be a potential strategy for preventing or attenuating MSRBD.

D-010

Impaired Axonal Transport and defects in Calcium Dynamics in V337M Tau Neurons: Toward a Gene Therapy for Selective Tau Reduction

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Tauopathies are neurodegenerative diseases associated with abnormal Tau protein accumulation, which leads to neuronal dysfunction and death, and dementia. To date, over 60 mutations in the gene coding Tau (MAPT) have been linked to pathological accumulation, microtubule instability, organelle trafficking defects, and synaptic dysfunction. A key feature of tauopathies is “selective neuronal vulnerability,” meaning that abnormal Tau differentially affects specific neuronal subtypes. Currently, there are no effective therapies to halt disease progression. Thus, we aimed to design a tool to selectively reduce Tau in affected neurons and assess its therapeutic potential.
Here, we evaluated the phenotypes associated with the V337M Tau mutation in patient-derived iPSCs glutamatergic neurons. Using live-cell fluorescence imaging, we assessed lysosomal axonal transport and observed altered distributions of retrograde and anterograde segmental velocities in V337M neurons compared with wild-type. Preliminary calcium imaging experiments further revealed reduced ΔF values and lower signal frequency in V337M neurons, suggesting electrophysiological impairment.
In addition, we developed lentiviral vectors carrying microRNAs under the CAMKII promoter to selectively reduce Tau in glutamatergic neurons and validated their efficacy in wild-type human neurons. Future studies will evaluate whether this approach can reverse disease-associated phenotypes in V337M neurons and other Tau mutants.

D-011

Eph/ephrins and neurodegenerative diseases relationship studied in cellular models

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The precise connection between neurons during development is one of the most accurate and regulated processes which contributes to the physiological function of neuronal circuits within the brain. Eph/ephrins play a crucial role in the development of neural connectivity. During early development, and across adulthood, Eph/ephrins support spatiotemporal, topological as well as specific clues, and deregulation of its function is observed in many neurodegenerative diseases. Up to now, the precise domains/motifs involved in Eph functions are not fully dilucidated. Here, we assessed cultured cells expressing fluorescent EphA3-WT and truncated vectors in order to determine vesicular parameters of axonal transport by live imaging recording movies like kymographs, segmental velocities, etc. Preliminary results showed that WT had mostly a vesicular mobile phenotype. Meanwhile, ligand binding truncated versions showed both an increased distribution in the plasma membrane region coupled with increased vesicle density and mobility compared to the WT. Furthermore, we will examine Eph transport in human glutamatergic neurons derived from isogenic-control and mutated tau iPSC as a model of neurodegenerative diseases. We propose that understanding the requirements for Eph/ephrins transport and distribution is key to unravel brain wiring mechanisms providing knowledge to comprehend the abnormalities that are associated with synapse collapse during neurodegenerative diseases.

D-012

Regulation of EZH2 in Hypothalamic Neurons of the Developing Brain

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In the central nervous system, Neurogenin 3 (Ngn3) is a proneural transcription factor that regulates neuritogenesis and its expression is regulated by a sex-specific enrichment of H3K27m3 in hypothalamic neurons from male embryos. Previous evidence from our laboratory demonstrated that the histone methylase Ezh2, is sexually dimorphic in hypothalamic tissue suggesting that its higher expression in male neurons may regulate Ngn3. In this study, we evaluate the sex-specific expression and regulation of Ezh2 in ventromedial hypothalamic neurons in vitro. To this purpose, we performed primary cultures from male and female mouse embryos at embryonic age 15 (E15). On day 3 in vitro, we evaluated the basal and 17β-estradiol (E2, 10-10M) induced mRNA expression of Ezh2. Furthermore, we pharmacologically inhibited EZH2 using UNC1999 (2μM) and quantified Ngn3 mRNA expression levels by qPCR. Results indicate no sex differences in Ezh2 expression ($p=0.14$; $n=5-6$) and no effect of E2 ($p=0.44$; $n=4-5$). However, pharmacological inhibition of EZH2 significantly upregulates Ngn3 gene expression only in female neurons ($p=0.05$; $n=6$). These findings suggest that the H3K27m3 methylation by EZH2 might regulate hypothalamic neuronal differentiation in a sex-specific manner. Thus, the observed sex differences could result from a balance of activating/repressing epigenetics marks on the Ngn3 promoter, which in turn may depend on the key role of the histone demethylase KDM6A.

D-013

Yerba Mate and CGA Activate AMPK, Promote Autophagy and Decrease A-Syn Aggregation in Cell Culture

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Neuroprotection is a key goal in the study of neurodegenerative diseases such as Parkinson's disease (PD), where progressive dopaminergic neuron loss causes severe motor and cognitive deficits. Epidemiological studies suggest that consumption of Yerba Mate (Ilex paraguariensis, YM), as well as coffee and green tea, is associated with reduced PD risk. These beverages contain polyphenols, particularly chlorogenic acid (CGA), proposed to mediate neuroprotective effects. We previously showed that YM protects dopaminergic neurons in primary cultures, but the underlying mechanisms remain unclear, especially those involving neuronal survival and protein homeostasis. Here, we investigated whether YM and CGA modulate pathways relevant to neuroprotection, focusing on AMPK activation, autophagy, and α-synuclein (α-Syn) aggregation. SH-SY5Y cells were used to evaluate AMPK phosphorylation after treatment with YM or CGA, while autophagy was assessed by LC3 ICC. In parallel, α-Syn aggregation was analyzed in H4 cells co-transfected with SynT and Synphilin-1. YM and CGA increased AMPK phosphorylation, suggesting activation of survival pathways. ICC revealed more LC3-positive puncta, indicating autophagy induction. In H4 cells, YM and CGA reduced the number and size of α-Syn aggregates. Altogether, these findings support that YM and CGA stimulate AMPK signaling, promote autophagy, and attenuate protein aggregation, highlighting YM as a promising dietary candidate for further studies in PD.

D-014

The metabotropic glutamate receptor MGL-2 gates satiety by modulating serotonergic signaling in C. elegans

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How neural circuits encode internal nutrient states to generate adaptive behaviors is a fundamental question in neuroscience. In this study, we explore the mechanisms through which C. elegans perceives its nutritional state and adjusts feeding and locomotor behaviors. We show that mutants of mgl-2, the C. elegans ortholog of mammalian metabotropic glutamate receptors (mGluRs), exhibit hyperphagia and decreased locomotion—phenotypes reminiscent of hungry animals, even in the absence of food deprivation. This excessive feeding results in elevated lipid accumulation, underscoring the critical role of MGL-2 in promoting satiety.
In wild-type animals, food encounter after starvation induces a pronounced serotonin release that facilitates feeding and suppresses locomotion to promote nutrient recovery. Using genetic approaches and in vivo neuronal imaging, we demonstrate that MGL-2 is essential for the perception of nutritional status and for regulating serotonergic signaling in fed animals. Ongoing work aims to identify the specific neuronal circuits in which MGL-2 operates.
Our findings support a model in which MGL-2 acts as a key modulator within neural pathways governing appetite and energy balance. Notably, mammalian mGluRs have recently been linked to hunger and satiety perception, suggesting evolutionary conservation in these regulatory mechanisms. This study provides insights into the neurobiological basis of feeding behavior with potential relevance across species.

D-015

Patient derived fibroblasts as a novel model to identify autophagy defects in tauopathies.

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Neurodegenerative tauopathies are characterized by pathological tau aggregation, often associated with cellular protein degradation defects.. The autophagy-lysosome system is essential for degrading aggregated proteins, and its failure can directly contribute to tau aggregation and neurodegeneration. Central to this process is the protein p62, which shuttles ubiquitinated proteins and organelles, including pathological tau, to autophagosomes for lysosomal degradation. Mutations in p62 disrupt this crucial function, leading to proteostasis breakdown. Fibroblasts obtained from patient dermal punches provide a powerful disease model as they retain the patient's specific epigenetic landscape. To investigate whether autophagy defects can be identified in patients derived fibroblasts as a strategy for disease detection, we used fibroblasts obtained from a primary tauopathy patient carrying the heterozygous mutation PRO392LEU in the ubiquitin-associated (UBA) domain of p62 (p62-P392L) and control donors.Preliminary analysis revealed increased cell area in p62-P392L patient; decreased Tau5 without PHF1 increase. p62-P392L fibroblasts showed reduced p62 by immuno/WB. LC3 levels and LC3II/I ratio by WB were increased. NH4Cl exposure highlighted these changes. Lysotracker showed more vesicles per cell.These findings show p62-P392L fibroblasts recapitulate impaired autophagy, offering a model to study proteostasis disruption in tauopathies

D-016

Can losartan cool down astrocytes? Targeting astroglial pathological remodeling after exposure to DAMP HMGB1

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Exposure to PAMP (LPS) or DAMP (HMGB1) in the presence of microglia induces pathological remodeling in astrocytes. Pathological remodeling involves epigenetic chromatin changes that repress homeostatic astroglial genes and activate a proinflammatory response (Cuautle et al., J. Neurochem. 2024). Pathologically remodeled astrocytes induce neurodegeneration and localize in the core of ischemic or traumatic brain lesions, as well as in animals exposed to status epilepticus (SE). Controlling the astroglial pathological remodeling and the proinflammatory burst is mandatory to improve neuroprotection. For that purpose, we tested the repurposed drugs losartan and resveratrol. Primary astrocytes were exposed to 500 ng/ml DAMP HMGB1 for 18h, and then we treated the cultures with losartan (1-10 μM) or resveratrol (10-100 μM). Glycyrrhizin was used as an HMGB1 antagonist. Exposure to HMGB1 induced NF-κB activation, astroglial DNA hypermethylation, increased expression of pathological marker MAFG and proinflammatory IL1B and IL6, and repressed homeostatic genes Kir4.1, GLT-1 and GS. Both Losartan and Resveratrol attenuated NF-κB activation and hypermethylation, restored GS levels, and reduced the ratio of MAFG+ nuclei ($p<0.05$). Losartan also restored the expression of Kir4.1, GLT-1 and GS and normalized IL-1β and IL-6 expression ($p<0.05$). These findings indicate that Losartan is a promising candidate to modulate astroglial pathological remodeling, justifying the future in vivo studies.

D-017

Undisturbed intracellular trafficking of Gpm6a is required for its function in filopodium formation

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Functional membrane trafficking is critical for many aspects of neuronal growth and differentiation and the regulation of the balance between endocytosis and recycling has been postulated to create polarized membrane flow during cell surface remodelling. In this context, a neuronal membrane glycoprotein M6a (Gpm6a) from the PLP/DM20 family of proteolipid proteins has drawn our attention. Gpm6a is a four-transmembane-domain protein abundantly expressed in neurons of the central nervous system. It functions in different processes of neuronal development, and its overexpression leads to the extensive formation of filopodia.

The endocytic and recycling pathway of Gpm6a has been shown to affect the formation and maintenance of synapses in neurons. But the mechanisms by which Gpm6a is targeted for recycling or degradation are still unknown.

Here, we demonstrate that the overexpression of a mutant form of Gpm6a, E258A, decreases filopodium formation and the complexity of neuronal arborization in rat hippocampal neurons. At the same time, increases dynamics of Gpm6a vesicles and redirects intracellular trafficking of Gpm6a towards late endosomal/ lysosomal compartments increasing its colocalization with Rab 7 and Lamp1-positive compartments. We propose E258 residue as a critical switch that regulates Gpm6a targeting for recycling or degradation and by this way contributes to neuronal morphogenesis.

D-018

Assessing the activation of the Unfolded Protein Response ATF-4 pathway in a cellular model of TDP-43 proteinopathies

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Frontotemporal Dementia (FTD) and Amyotrophic Lateral Sclerosis (ALS) are neurodegenerative diseases characterized by abnormal accumulation of TDP-43, an ubiquitous protein critically involved in RNA metabolism. These diseases caused by proteostatic perturbations commonly trigger the Unfolded Protein Response (UPR). This study investigates the relationship between the key UPR component ATF4 and TDP-43 in physiological and pathological contexts, using immunocytochemistry in SH-SY5Y and N2a neuroblastoma cells. Activation of the PERK UPR pathway leads to increased ATF4 translation. Single-cell SH-SY5Y analysis revealed a statistically significant positive correlation between the endogenous nuclear TDP-43 and ATF4 levels. The slope is twice as steep in cells treated with the ER stress inducer tunicamycin (Tn) compared to controls. Nuclear ATF4 showed significant colocalization with TDP-43 (Manders coefficient, M = 0.66), and Tn treatment significantly increased this colocalization (M = 0.72). These findings suggest a potential link between TDP-43 levels and activation of the ATF4-mediated stress response. We also overexpressed wild-type (WT) nuclear TDP-43 or a cytoplasmic form of TDP-43 (ΔNLS) to recapitulate key ALS/FTD features in N2a cells. Both variants led to a significant increase in endogenous ATF4 levels (WT = 1.6 fold; ΔNLS = 2.0 fold), indicating activation of the ATF4/PERK UPR branch. Overall, these results help understand the role of UPR activation in ALS/FTD.

D-019

Glial 24-S-Hydroxycholesterol as a mediator of neurotoxicity: effect on neuronal viability, synapse structure and potential amyloidogenic role

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Strict maintenance of brain cholesterol homeostasis is essential for neuronal functioning. Neuronal cholesterol is mostly synthesized in astrocytes, while the excess of this lipid is eliminated from the brain by its conversion into 24-S-hydroxycholesterol (24-OHC), via the neuronal specific enzyme CYP46A1. Although 24-OHC synthesis has been reported mainly in neurons, our results indicate that 24-OHC is produced at high rates by reactive astrocytes. The role of 24-OHC as a signaling molecule is controversial and has been implicated in Alzheimer's disease (AD). Therefore, we investigated the effect of 24-OHC in primary cultures of rat cortical neurons, analyzing: neuronal viability, density of synaptic contacts and its ability to induce APP synthesis.
Our results show that minimal doses of 24-OHC significantly reduce the density of synaptic contacts, while higher concentrations affect neuronal viability, evidenced by an increase in pycnotic nuclei. In addition, exposure of primary neurons to 24-OHC increased APP synthesis from 1 µM, suggesting a possible predisposing role in AD at higher concentrations of this sterol. Our results suggest that 24-OHC would act as a mediator of neurotoxicity in astrogliosis with implications in AD.

D-020

Galectin-1 targeting uncovers angiogenic heterogeneity in patient-derived glioma stem cells

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Glioblastoma (GBM) is the most aggressive brain tumor, with aberrant angiogenesis driving its progression. This study explored the angiogenic properties of patient-derived glioma stem cells (GSCs) and assessed galectin-1 (Gal1) as a therapeutic target. Four GSC lines (G02, G03, G08, G09) were analyzed by transcriptomics and functional assays, revealing marked heterogeneity. G02 showed an endothelial-like transcriptomic profile and enhanced endothelial migration, though lacked protein expression after transdifferentiation. G03 displayed enrichment of angiogenesis pathways, microvascular proliferation in patient biopsies, and superior tube formation ability. All GSCs formed tube-like structures, with G03 being most efficient. Gal1, a lectin involved in migration, proliferation, and angiogenesis, emerged as a differential target. Blocking Gal1 or VEGF reduced tube formation in G03, while Gal1 silencing also impaired G02. Findings highlight Gal1's extracellular and intracellular roles and the variable angiogenic potential of GSCs, supporting selective, personalized anti-angiogenic strategies in GBM to improve treatment efficacy and avoid resistance.

D-021

Rab11a role in dense-core vesicle dynamics in chromaffin cells.

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Rab11a is a small GTPase of the Ras superfamily involved in constitutive recycling pathways. Rab11a is involved in the regulation of several cellular processes, such as cytokinesis, phagocytosis, cell migration and ciliogenesis. However, its role in the secretory pathway of neuroendocrine cells has not been fully understood. We have previously reported that Rab11a coordinates the distribution of secretory vesicles (SV) at the plasma membrane in chromaffin cells, a well-known cellular system for neurosecretion. To study this further, we cotransfected chromaffin cells with Rab11a mutants with impaired GTP/GDP cycle together with fluorescently-tagged neuropeptide Y (NPY), as a marker of SV. By confocal microscopy, we observed a reduction in the amount of peripheral NPY in cells expressing S25N, a Rab11a dominant-negative mutant that cannot exchange GDP with GTP. To evaluate if SV trafficking dynamics were affected by Rab11a, we tracked GFP-tagged NPY in living chromaffin cells. We observed that SV mobility was reduced in cells expressing S25N, evidencing a role for Rab11a in the vesicle transport towards the plasma membrane. We propose that Rab11a plays a fundamental role in neurosecretion by modulating the availability of secretory vesicles at the plasma membrane through the coordination of the vesicular trafficking in chromaffin cells.

D-022

Oligodeoxynucleotide-Induced Remyelination: Evaluation of the Therapeutic Potential of IMT504 and Its Effects on Microglia and Oligodendrocytes

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Demyelination is defined as the loss of the myelin sheath surrounding axons, while remyelination restores this sheath and contributes to functional recovery. Multiple sclerosis is a common inflammatory demyelinating disorder in which the remyelination process is frequently unsuccessful. IMT504 is a synthetic 24-base non-CpG oligodeoxynucleotide containing two characteristic PyNTTTTGT motifs.

Considering the regenerative and immunomodulatory properties of IMT504, and given that our group first reported its promyelinating benefits with effects on neuroinflammation and remyelination in an animal model, the present study aims to investigate the effects of IMT504 both in vitro, in glial cell cultures, and in vivo, in the corpus callosum (CC) of cuprizone (CPZ)-demyelinated rats. Our findings show that IMT504: (i) enhances microglial cell phagocytosis, (ii) induces the activation of p38, ERK1/2, SAPK/JNK, and NF-kB signaling in microglial cells, (iii) induces the activation of p38 and ERK1/2 signaling in oligodendrocyte precursor cells, and (iv) produces changes in the activation of MAPK signaling in the CC of CPZ-demyelinated rats.

These results underscore the prospective therapeutic value of IMT504, reinforcing its potential role in the development of interventions for demyelinating disorders.

D-023

AT2 Receptor Activation Enhances SH-SY5Y Cell Viability in a 6-OHDA Model of Dopaminergic Injury

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Angiotensin signaling may modulate neuronal survival, yet its receptor-specific contribution in dopaminergic injury remains unclear. We tested whether activating AT2 receptors protects human SH-SY5Y cells against 6-hydroxydopamine (6OHDA). Cells were exposed for 72 h to angiotensin II (AngII, 600 nM), the selective AT2 agonist CGP42112A (1 μM), or both, and then challenged for 24 h with the LD50 of 6OHDA. Viability was quantified by trypan blue exclusion and propidium iodide flow cytometry. 6OHDA reduced viable cells versus control (844,167 ± 51,011 vs 1,538,125 ± 73,919; $p<0.05$). CGP42112A increased viable counts (2,350,000 ± 212,132), and CGP42112A+AngII further increased them (2,750,000 ± 353,553; both $p<0.05$). Flow cytometry confirmed a 56.92% drop in viability with 6OHDA ($p<0.0001$), partially rescued by CGP42112A (+9.92%; $p=0.0031$) and more robustly by CGP42112A+AngII (+20.5%; $p<0.0001$). The AT2 antagonist PD123319 abolished protection, whereas AT1 blockade with losartan did not blunt it, implicating AT2 signaling. Basal exposure to AngII or CGP42112A alone did not alter viability. All values are means ± SEM of three independent experiments; groups were compared by one-way ANOVA with Tukey's post hoc test. These preliminary data indicate that AT2 receptor activation confers protection in a 6OHDA model of dopaminergic damage and nominates AT2 as a candidate target to bolster neuronal survival in neurodegenerative settings.

D-024

CIC-a deficiency induced neuronal and behavioral alterations in Drosophila melanogaster

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The circadian oscillator of Drosophila is comprised of approximately 150 clock neurons that express a set of molecular signatures, including clock genes, which through negative feedback loops coordinate oscillation of transcription and translation of other genes and proteins. A subgroup of clock neurons, called ventral lateral neurons (LNvs) is characterized by the expression of the neuropeptide Pigment Dispersing Factor (PDF). LNvs play a fundamental role in the control of alertness and are essential for the regulation of sleep/wake behavior via a yet not fully understood neuronal circuit. Previous work from our laboratory has identified CIC-a, a voltage-dependent chloride channel, as a potential key element in the physiological regulation of LNvs. This channel has not been explored in the Drosophila adult neurons. Therefore, the main objective of this project is to characterize the roles of neuronal CIC-a and its mechanism of action. Our findings indicate that downregulation of CIC-a in LNvs increases sleep in both female and male flies and reduces latency to siesta sleep. Additional behavioral analyses suggest that CIC-a may be involved in detection of sensory stimuli, such as light and mechanical stimuli. To further investigate the physiological basis of these behavioral effects, we are currently performing whole-cell patch clamp recordings in LNvs.

D-025

Sex-specific effects of daytime disturbances on sleep and adenosine receptor expression in zebrafish.

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Adenosine is a key mediator of sleep homeostasis, linking energy metabolism with sleep pressure and acting mainly through adora1 (A1R) and 2 (A2AR) receptors that regulate excitatory circuits. While its role in mammalian sleep has been widely studied, its contribution in zebrafish remains poorly understood.

Sleep regulation is also strongly influenced by environmental challenges, yet little is known about how daytime disturbances affect subsequent sleep and activity in this model. Adult zebrafish (male and female) were exposed to daytime disturbances for two days (5min vibration every15min from 13:00-19:00hs) and compared to a control group (n=8-12).

Locomotor activity, nocturnal sleep, and the expression of adenosine receptors in the whole brain by RT-PCR were assayed. Baseline recordings revealed a clear sexual dimorphism in nocturnal sleep, with males awakening earlier than females.

Daytime disturbance increased locomotor activity during the stimulation period. However, after two consecutive days of disturbances, only females showed a significant reduction in sleep during the first two hours of the night, without changes in total nocturnal sleep. Notably, females also exhibited increased A1R expression compared to controls. In conclusion, our findings identify a sex-specific vulnerability to daytime disturbances, with females being uniquely affected. The upregulation of A1R may represent a compensatory mechanism enhancing adenosine signaling following reduced sleep.

D-026

DNA damage repair proteins as molecular sensors of sleep in Drosophila

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What are the evolutionary advantages of sleep? Although it is well established that sleep is a highly conserved behavior throughout the animal kingdom, its precise biological purpose is still unclear. Several theories have been put forward to explain its role. For instance, sleep has been linked to processes such as memory consolidation, synaptic remodeling, and the clearance of neurotoxic by-products that accumulate in the extracellular space during periods of wakefulness. More recently, a novel hypothesis has emerged suggesting that the repair of DNA damage accumulated while awake constitutes a core function of sleep in zebrafish. Could this mechanism be conserved across other animals? Might DNA repair proteins serve as critical molecular sensors in the regulation of sleep? We propose that DNA repair proteins contribute to the molecular machinery of the sleep homeostat in Drosophila. To investigate this idea, we employ new approaches to induce DNA damage and subsequently assess alterations in sleep patterns. Furthermore, we are evaluating whether PARP1, a protein identified as a key detector of DNA double-strand breaks and mediator of sleep induction in fish, also fulfills this role in insects. Future investigations will explore whether additional DNA repair factors, including Rad51, Ku70, and Ku80, participate in shaping sleep regulation in fruit flies.

D-027

Circadian control of oviposition in Drosophila requires lateral posterior neurons

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Most organisms coordinate their physiology and behavior with the 24-hour day/night cycle generated by Earth's rotation. These biological rhythms are controlled by molecular clocks that are conserved across animals. In the Drosophila brain, the molecular circadian clock is expressed in ~240 neurons, organized into distinct clusters according to gene expression, anatomy, and localization. Egg-laying is a key female behavior with a profound impact on species fitness. While oviposition is primarily governed by successful mating, it is also modulated by the circadian clock. Previous work from our laboratory demonstrated that the lateral dorsal neurons (LNds) play a leading role in the control of oviposition rhythms. Here, we extend these findings by showing that the lateral posterior neurons (LPNs), another group of clock neurons, are also required for the circadian control of egg laying. We further explore the contributions of these neurons, by silencing or activated them using genetics approaches.

D-028

The interaction between school shift and age affects chronotype, social jetlag and sleep duration in adolescents

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School schedules typically begin very early in the morning, which contrasts sharply with the naturally delayed chronotype of adolescents. Previous evidence shows that this misalignment leads to chronic sleep deprivation, social jetlag, and multiple negative outcomes, including poorer academic performance. Earlier studies by our group have documented these effects among Argentine students in the 1st and 5th years of secondary school. The aim of this research is to study the interaction between age and school shift on chronotype, social jetlag and sleep duration in 1st to 5th year secondary students, who were randomly assigned to morning, afternoon, or evening shifts at the start of secondary school. Our results indicate that as students progress through secondary school, their chronotype shifts later, while social jetlag and sleep deprivation on weekdays increases. Morning-attending students showed higher levels of social jetlag and sleep deprivation than their peers from afternoon and evening shifts. Importantly, most morning-attending students did not reach the recommended 8 hours of sleep at any age, even including naps.
All these findings highlight the importance of considering the interplay between age and school timing for adolescents' sleep habits in Argentina, given its association with performance, well-being, and educational outcomes, providing critical evidence to inform local public policies.

D-029

The Role of Sleep in Memory Consolidation and Integration of New Words

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Sleep plays a key role in the formation and modification of memories, enabling the reprocessing and integration of new information. Human sleep alternates between non-rapid eye movement (NREM) sleep, which includes light (N1 and N2) and slow-wave sleep (N3), and rapid eye movement (REM) sleep, characterized by desynchronized cortical activity. These stages are proposed to serve complementary functions: NREM may support stabilization of memory traces, while REM integration of new information into existing knowledge networks. To test this, participants (N = 30, 18–40 years) learned a new word-learning task (rare Spanish words associated with both an image and a definition) before either taking a 90-min nap under polysomnographic monitoring (nap group) or remaining awake (control group). Afterward, participants were tested for word-image and word-definition associations, as well as for the integration of the new words. In this poster, we will discuss preliminary findings and their relation to the time spent in different sleep stages, focusing on correlations between NREM and REM sleep with processes of memory consolidation and integration.

D-030

A predator-based weak training protocol in Drosophila melanogaster for testing NAT10 inhibition in memory consolidation

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Memory consolidation requires protein synthesis and cytoskeletal remodeling, processes in which the acetyltransferase NAT10 plays a dual role by acetylating both microtubules and mRNA. Pharmacological inhibition of NAT10 with Remodelin has been shown to prevent neurodegeneration in tauopathy models, but its potential impact on memory remains unknown. We hypothesize that NAT10 inhibition can enhance long-term memory persistence. To establish a framework for testing this hypothesis, we developed a weak training protocol in Drosophila melanogaster whitew1118 flies using the jumping spider Menemerus semilimbatus as a natural threat. Memory retention was evaluated 24 hours post-training by quantifying flies's movement. Preliminary results show that weak training fails to induce long-term memory retention, in contrast to strong training protocols. This result provides the conditions under which future experiments will test whether NAT10 inhibition can rescue or enhance memory persistence in pathological models.

D-031

Impact of early and prenatal alcohol consumption: finding solutions through an antioxidant diet

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Early exposure to alcohol, especially during critical periods such as pregnancy or adolescence, can lead to alcohol abuse and harmful consequences. Numerous surveys estimate that a large percentage of women consume alcohol during the first trimester of pregnancy, and a large number of adolescents consume alcohol excessively. These consumption patterns can alter oxidative stress metabolism and cause patterns of anxiety and depression. These effects have been studied in animal models, and one way to mitigate them is through dietary supplements such as folic acid and selenium, which can help restore the metabolism. In our research, we want to expand our knowledge of treatments with enriched diets and early exposure to alcohol. Specifically, we will study the offspring of mothers exposed to alcohol during gestational days 7-9. We expect their offspring to consume more alcohol, have an anxious and depressive phenotype, and have memory problems. We will also analyze the antioxidant capacity of the supplemented diet and observe whether there are differences between the groups, expecting an improvement in the supplemented group.

The data obtained show that early alcohol consumption by the mother leads to high consumption in adolescence. Likewise, antioxidant enzymes such as PON1 show lower activity in adolescents with high alcohol consumption.

D-032

Simultaneous Alcohol and Marijuana Use in Argentinean University Students: A Cross-Provincial comparison of Frequency of use, Predictors and Consequences

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Intro. Alcohol and marijuana are the most widely used substances among Argentinean university students, and their simultaneous (SAM) use is common. Compared to single-substance use, SAM use increases acute and long-term risks. This study extends previous research conducted in Córdoba (CBA) by incorporating a cohort from Mar del Plata (MDQ). We compared frequency of SAM use and associated problems and examined in the MDQ sample if SAM use motives and impulsivity explained negative consequences. Method. A cross-sectional analytical study was conducted in students aged 18–30 years from universities in CBA and MDQ, who completed an online questionnaire. Descriptive analyses and tests of proportion/mean differences between sites were performed. A hierarchical regression analyzed if SAM use motives explaining negative consequences in MDQ sample, beyond impulsivity traits. Results. MDQ Students showed higher frequency of SAM use than CBA peers, regardless sex and age of onset; yet problems were similar across sites. SAM use motives explained SAM-related consequences (R^2=.253; ΔR^2=.134; p<.01), after controlling for impulsivity. Negative urgency and social motives were significant predictors (p<.05). Conclusions. The results underscore that SAM use can vary across the country. Negative consequences of SAM were mainly predicted by negative urgency and social motives, highlighting the need for interventions focused on emotion regulation and peer dynamics.

D-033

Reactivation and temporal dynamics in threat conditioning: disentangling effects on emotional memory strength and cognitive biases

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Retrieving a consolidated memory can trigger different processes, such as memory reactivation by the reminders presented during acquisition. Only a few reports have shown that memory modification might occur earlier than the declarative changes associated with the aversive stimuli. We designed a differential threat-conditioning protocol, the association of an angry face conditioning stimulus (CS+) with an aversive tone unconditioned stimulus (US), combined with declarative tasks to analyze the interplay between implicit memory and cognitive bias, as well as their temporal dynamics. We performed two experiments: 1 or 2 CS+ presentations (1R or 2R) during the reactivation session, evaluating their effects 48 hs or 3 weeks after acquisition. Declarative results indicated evidence of learning and retention in both groups. In the 1R group, CS+ showed an increase in reported aversiveness 48 hours after acquisition, which was not sustained over time. In contrast, CS– exhibited a consistent decrease in aversiveness across assessments. In the 2R group, no changes were observed for CS+ before or after conditioning, whereas CS– showed a decrease in aversiveness 48 hours after acquisition that persisted at the three-week follow-up. The results indicate that both the temporal dynamics and the number of reminders influence stimulus representations. Further analyses are required to determine whether the aforementioned variables also have an effect on implicit memory.

D-034

What Order Should We Train Executive Functions? A Pilot Test Suggests… Any

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Executive functions (EF) are cognitive functions that allow us to control actions and thoughts and adapt to changing environments. They are important for educational and life success and can be improved through cognitive training. For more than fifteen years, our team has implemented Mate Marote, a free access gaming software to train and assess EF in 4-to-8 year-olds. Brief and spaced interventions take place within the classroom, with successful results. There are still open questions about the best way to train them, particularly regarding training order: should basic executive functions be trained first and then move on to more complex ones, or vice versa? We present preliminary results from 23 Spanish aged 6-7 who participated in an intervention consisting of about 13 weekly sessions of 15 minutes each, with evaluations conducted before and after. We used mixed linear regression models to evaluate the effects of the intervention, training order, and other covariates on performance. Overall, we did not find an effect of training order on cognition. We discuss our results in relation to implications for future research interventions, as well as the relevance of the study for the cognitive training field.

Key words: cognitive training, intervention, training order.

D-035

Time–space signatures of interacting predictors in hybrid search using EEG and eye tracking data

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In everyday life, finding specific items among distractors often requires joint contribution of visual attention and memory recall. We recently showed that we can successfully disentangle overlapping neural responses during natural viewing by applying deconvolution methods to coregistered EEG and eye-tracking data. We estimated temporal response functions (TRFs) through regularized linear models for main effects and their interactions, capturing fine-grained spatial and temporal activation patterns. Starting from hypothesis-driven models, we replicated established effects, including well-known components for visual processing and target detection. Extending to interactions with a data-driven approach, TRF estimates remained consistent across increasingly complex models, with their performance evaluated via explained variance (R^2), and collinearity through variance inflation factors (VIF). In particular, in the main effects, we identified a late activation consistent with the P300 component for target detection. Furthermore, we also showed an interaction with correct detections, indicating missed detections elicited a similar but weaker activation and a more nuanced role of this component. These analyses demonstrate how deconvolution methods can uncover the dynamic interplay of cognitive processes underlying real-world search behavior.

D-036

Phonetic and Phonemic Segmentation: Neural Representations of Speech Attributes in Natural Dialogue

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The study of speech in natural environments poses challenges for traditional electroencephalogram (EEG) analysis approaches. In recent years, machine learning models—particularly regularized linear encoding models—have enabled a transition toward experimental designs that incorporate dynamic and naturalistic stimuli, such as speech during dialogue. This work aims to understand how different speech attributes are encoded in the brain within the context of unscripted natural dialogue. To this end, we extract low-level attributes (envelope, pitch, spectrogram, among others), high-level attributes (phonemes, phonological features, among others), and attributes derived from representations obtained with deep neural networks (Wav2Vec2.0, Whisper). The results show that the inclusion of high-level attributes significantly improves the prediction of brain signals across all frequency bands. In particular, predictions based on phonemes and phonological features suggest that neural sensitivity is consistent with the hypothesis of a hierarchical language processing system.

D-037

Sleep, impulsivity, emotional states and sunlight exposure: A Comparative Study in Ushuaia and AMBA

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Relationships have been reported between sleep, impulsivity, emotional states, and sunlight. Some studies associated sleep disturbances to depressive states, as well as insomnia to impairments in people's quality of life, while others supported a negative correlation between sleep quantity and impulsivity. Given that results vary significantly across studies more studies are needed. Considering the differences across Argentina in terms of sunlight, our study aimed to explore if there is an association between sleep, impulsivity, sunlight exposure and emotional states in Ushuaia (USH) and Metropolitan Area of Buenos Aires (AMBA) in December and May. The hypotheses were: (1) optimal sleep quantity and quality are negatively correlated to impulsivity, (2) there is an inverted U-shaped correlation between sunlight exposure and positive emotional states, and lastly, (3) there is an inverted U-shaped correlation between sleep quantity and positive emotional states. Thirty adults from USH and AMBA, completed online questionnaires and a cognitive task. Results showed that in May USH had higher depression levels than December while sleeping more than AMBA, associating more sleep to depressive states.

Keywords: sleep, impulsivity, emotional states, sunlight exposure

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D-038

Electrophysiological Signatures of Consciousness During Sleep: Evidence from Lucid Dreams, Sleep Paralysis, OBEs and False Awakenings

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Consciousness is typically absent during sleep, yet it can re-emerge in unique and immersive forms such as lucid dreams (LDs), sleep paralysis (SP), out-of-body experiences (OBEs), and false awakenings (FAs). Despite extensive phenomenological reports, the neurophysiological basis of these states remains largely unexplored.

We conducted overnight polysomnography in experienced individuals, capturing ten verified episodes (3 LDs, 2 SP, 2 OBEs, 3 FAs). Using a within-subject design, we compared each conscious episode to the same individual's standard sleep stages. Consciousness markers were identified through pre-agreed ocular signals. EEG spectral power was analyzed using principal component analysis (PCA) and permutation-based multivariate ANOVA (PERMANOVA).

These states differed markedly from wakefulness and did not fit a simple hybrid model between waking and sleep. Instead, they showed distinct neurophysiological signatures with partial overlap with REM and S1, but unique configurations in reduced-dimensionality space. Notably, we report the first in-lab identification of eye movement markers during OBEs and FAs, offering empirical validation for these experiences.

Spectral analysis showed LDs combined delta, theta, and low-gamma activity. SP episodes showed increased alpha, beta, low-gamma and reduced theta, consistent with heightened awareness. OBEs showed enhanced delta and theta, possibly reflecting sensory decoupling. One FA episode with lucidity markers resembled

D-039

Towards studying mental states during driving hazard perception: Analysis of co-registered magnetoencephalography and eye movement recordings

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In driving, Hazard Perception (HP) is defined as the ability to identify potential traffic hazards with sufficient time to avoid collision. Driving demands high visual, attentional, and memory engagement, involving executive functions such as sustained attention and inhibitory control. Even in autonomous driving, manual takeover may require several seconds without proper hazard perception. Executive functions and their neural correlates have traditionally been studied using artificial stimuli in static contexts, due to two main issues: eye movements generate artefacts in brain signals, and it is difficult to associate brain activity with dynamic, complex stimuli. In this work, we aim to apply new tools to study hazard perception in natural, dynamic driving environments. We will focus on the study of the association between hazard perception, eye movements and brain activity recorded with magnetoencephalography (MEG) while seeing UK driving test videos. We analysed fixation and saccade-related brain activity and started exploring associations between dynamic mental states identified using unsupervised learning methods and successful or failed hazard perception.This is a key step towards understanding brain responses to naturalistic driving situations.

D-040

Immediate effects of high-intensity interval training on gamma band power in the sensorimotor area of the brain

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Brain activity is studied across frequency bands, each linked to distinct functions of the central nervous system. Gamma oscillations are particularly associated with neuronal integration, attentional processes, and cortical excitability. While physical exercise has been shown to modulate brain activity, evidence on its specific impact on gamma oscillations remains scarce. The aim of this study was to examine cortical gamma activity in the sensorimotor area immediately before and after a high-intensity interval training (HIIT) protocol. Participants completed a cycling task consisting of ten 20-second bouts at 90% of individual capacity, interspersed with 40-second active pauses at 50%.

Electroencephalographic activity was recorded in resting state (eyes closed) before and after exercise, and gamma power spectral density (PSD) was extracted from the sensorimotor area. Spectral analysis was performed using MATLAB and EEGLAB, with non-parametric tests applied to compare conditions. Results revealed a significant post-exercise increase in gamma power relative to baseline, with a consistent pattern across participants. These preliminary findings align with previous evidence, suggesting that HIIT may acutely enhance cortical excitability reflected in gamma activity, underscoring the potential role of exercise-induced brain modulation in neuroscience research.

D-041

Can mice play SIMON?: Design and Construction of an Automated Device to Assess Temporal Memory

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Temporal memory refers to the ability to encode, store, and retrieve information about the sequence and timing of events. It is particularly impaired in the early stages of Alzheimer's disease (AD), constituting one of its main clinical markers. Studying these alterations in animal models is crucial for understanding underlying mechanisms.

Behavioral experiments require precise control of data streams. The goal of this project was to design and develop an automated experimental arena to assess temporal learning and memory in wildtype and triple-transgenic (3xTg) mice, focusing on their ability to recognize temporal sequences.

To achieve this, a circular chamber was designed, equipped with six lights, a video camera, and a reward dispenser, controlled by an Arduino board and integrated through Bonsai, a reactive programming framework. This novel protocol consisted of sequences of light stimuli paired with rewards. Mice were first trained to associate rewards with timely responses to light cues, after which sequence length was progressively increased.

Position tracking was also performed using open-source software, DeepLabCut. Custom algorithms developed in our laboratory were applied to analyze the movement patterns, reaction times, and errors in both groups.

This setup provides a robust and fully automated platform for investigating sequence learning and temporal memory in mice. It provides precise behavioral measurements critical for understanding cognitive impairments.

D-042

Mapping neural activity on natural tasks: hybrid search and driving

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In everyday life, the brain often performs complex tasks without our notice. Searching for several items at once (e.g. wallet, keys) known as hybrid search, requires retrieving multiple targets from memory throughout the visual search. This is also critical for using landmarks during navigation, a process that also involves dividing attention across multiple objects, for instance, while driving. Here, we combined magnetoencephalography (MEG) and eye tracking to investigate the oscillatory and evoked dynamics of brain activity that support hybrid search and driving. Twenty-one participants performed a free-viewing, memory-guided search task in complex scenes. Time-frequency analyses revealed distinct signatures during encoding, retention, and search. Posterior alpha power decreased with memory load during encoding/retention, reflecting increased perceptual and mnemonic demands. During search, frontoparietal beta activity scaled with load, suggesting increased cognitive control. Source reconstruction revealed an early visual evoked lambda response in V1, followed by a distributed P3m component mainly in the right inferior parietal lobe, distinguishing target from distractor fixations. A similar lambda response was observed in a second dataset, where nine participants performed a divided attention task in a driving simulator.
Together, these results start to reveal how memory, attention, and visual processing dynamically interact during active vision.

D-043

Dreaming and the Reactivation of Declarative Memories: Project Design and Discussion

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While we sleep, information acquired throughout the day is spontaneously reactivated across different sleep stages. During Non-Rapid Eye Movement (NREM) sleep, these reactivations facilitate the transfer of information across brain regions, supporting memory consolidation. In contrast, reactivation during Rapid Eye Movement (REM) sleep has been associated with the integration of new memories into pre-existing neural networks. Reactivations can also be externally induced by presenting previously associated cues (odor/sound) during sleep, through a technique known as Targeted Memory Reactivation (TMR). TMR has been shown to promote both memory consolidation and related dream content.

In this project, we will analyze the effect of memory reactivation during NREM and REM sleep on dream content generated in REM, as well as its impact on memory consolidation and integration. Participants will perform a word-definition-image task, followed by TMR and a serial awakening protocol with dream reports. The protocol spans two nights: one adaptation night and one experimental night. On the experimental night, participants will learn the task before sleep, receive targeted reactivations, and provide dream reports during serial awakenings. In the morning, they will provide a general dream report and perform the task test. This preliminary study aims to advance our understanding of how memory processes during sleep can influence and shape dreams.

D-044

Sleeping tight: Determining the intersection between sleep and emotional frustration

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Sleep is a universal state, typically preceded by preparatory routines such as grooming, nest-building, and locating a sleeping spot. These behaviors are essential for sleep consolidation, and their disruption, particularly by negative emotions, may alter sleep quality. To investigate the interaction between emotional regulation and sleep, we employed a rodent model of frustration, the consummatory Successive Negative Contrast (cSNC) paradigm, in which the unexpected devaluation of a reward induces emotional dysregulation. We combined this paradigm with pre-sleep behavioral video analysis and electroencephalographic/electromyographic monitoring. We observed that after the reward devaluation, downshifted animals displayed an initial increase in locomotion, followed by fewer REM sleep episodes during the light phase, delayed sleep-onset and poorer nest quality. In a second experiment, we repeated the protocol but added a 6h sleep deprivation manipulation after sucrose exposure on the last 4 days pre-shift. Preliminary data showed that both deprived and undisturbed downshifted animals further delayed their sleep-onset and decreased their rest time following reward devaluation. These findings suggest that frustration disrupts sleep architecture and that these alterations are aggravated by previous sleep debt.

Key Words: Emotional frustration, Sleep/wake cycle, Pre-sleep, Frustration and sleep, Consummatory Successive Negative Contrast, Sleep Deprivation

D-045

Memory Without Hippocampal Microglia

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Microglia physically contact a small population of synapses in the healthy adult brain; however, it has been shown that their interaction leads to the regulation of synaptic stability by either increasing or decreasing the volume and number of spines. These actions contribute to the formation, maturation, and plasticity of neural circuits, which ultimately shape animal behavior. In this study, we investigated the implications of hippocampal microglial depletion for the processing of spatial and aversive memories. To do this, we trained female rats in a spatial object recognition task and administered clodronate into the hippocampus to achieve local microglial depletion. Prior to this, we performed immunofluorescence to evaluate the time window and specificity of this depletor. On the third day after an acute administration of clodronate, a transient decrease in the number of microglial cells was observed, without significant changes in the number of astrocytes or neurons. Three days after drug infusion, rats can acquire spatial memory and express it in the short term; however, they fail to consolidate it. Ten days after clodronate administration, these animals successfully acquired and consolidated an aversive memory. Our results highlight the role of hippocampal microglia in the dynamic processes of memory.

D-046

Serotonin 2A and dopamine D1/5 receptors interact in the rat medial prefrontal cortex to induce retrieval-induced forgetting

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It is now known that multiple forms of forgetting exist, some of which are active processes. Anderson et al. in 1994 postulated that the act of remembering some experiences causes the forgetting of others. Work in our lab has extended this finding by demonstrating an analogous phenomenon in rats. This retrieval-induced forgetting (RIF) is understood to be a competition-dependent, cue-independent process, driven by prefrontal inhibitory control signals that target the areas where memories are stored. Separate lines of evidence from our lab have shown that the serotonin 2a receptor (5-HT2AR) and the dopamine d1/5 receptor (D1/5R) in the mPFC are each necessary for RIF. It is unclear whether these receptors function independently or interact to produce RIF. This work aims to determine the existence and mechanism of a functional interaction between these receptors in RIF. We conducted a disconnection experiment that revealed that RIF was impaired when antagonists for each receptor were administered in opposite hemispheres, but was unaffected when both were applied unilaterally. The abolition of RIF produced by 5-HT2AR antagonism is reversed by a D1/5R agonist. In summary, our results identify a functional interaction between 5-HT2AR and D1/5R as a mechanism underlying retrieval-induced forgetting and opens new avenues for investigating how modulatory signals in the PFC are leveraged to resolve memory competition and adaptive behavior.

D-047

Dopamine-Dependent Modulation of Social Behavior in Mouse Models Relevant to Psychiatric Disorders

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Dopaminergic signaling from the ventral tegmental area (VTA) is central to neural circuits governing motivation and reward. Dysregulation of this system is implicated in neuropsychiatric disorders such as schizophrenia (SZ), often marked by social deficits. In SZ, dopaminergic imbalance has been reported, with increased activity in the associative striatum and reduced tone in medial regions including the VTA. While dopamine release during social interactions in rodents underscores its role in social motivation, its contribution to higher-order social cognition remains unclear. This gap limits our understanding of how dopaminergic dysfunction contributes to socio-affective symptoms in psychiatric conditions. To investigate this, we used rodent models with targeted modifications of dopaminergic activity via manipulation of D2 autoreceptor expression. These models were assessed using a behavioral battery designed to capture multiple dimensions of social behavior. Our findings aim to clarify the role of dopamine signaling in complex social processes and provide insight into the neurobiological mechanisms underlying social impairments in SZ and related disorders.

D-048

From Stress to Temperament: NR3C1 as a Pathway Linking Maternal Stress and Infant Development

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Maternal perinatal stress (MPS) is increasingly recognized as a determinant of early socioemotional development. This study examined whether MPS predicts infant temperament at three months and explored the mediating role of maternal NR3C1 gene expression. A cohort of 198 mother–infant dyads was assessed through validated psychological questionnaires, molecular analyses, and caregiver-reported temperament measures across pregnancy and early postpartum. Results showed that elevated MPS, particularly during the second trimester, predicted lower infant surgency, higher negative affectivity, and reduced effortful control. Regression models revealed that second-trimester stress was the strongest predictor of infant temperament dimensions, explaining up to 47% of variance. MPS was also associated with reduced maternal NR3C1 expression, which in turn predicted lower infant surgency. Mediation analysis confirmed that maternal NR3C1 expression significantly mediated the link between cumulative perinatal stress and infant surgency. These findings provide novel evidence on molecular pathways underlying the developmental origins of health and disease (DOHaD), suggesting that maternal stress during pregnancy may become biologically embedded and shape infant behavioral phenotypes. Implications for early prevention strategies and perinatal mental health policies are highlighted.

D-049

Strain-dependent differences in memory retention in Drosophila melanogaster

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Drosophila melanogaster under predation expresses a variety of defensive behaviors. Here we show that predation promotes learning in two different fly strains, white1118 and Canton-S. The memory retention paradigm presented here make use of a natural predator, a spider, as a source of unconditioned stimulus (US). This spider stalks and prays flies in a direct attack triggering a range of Drosophila defensive behaviors.

We have found that both strains show memory retention 24 hours after a strong training session. Additionally both groups show different defensive behaviours when tested, potentially as a consequence of the physiological differences between these animals. We believe that a “decision-making” process occurs, differentiating both pathways. For both strains memory retention is only expressed when the context is consistent between the training and testing session.

The present paradigm faces flies with the predator within a context (CS) and defines the memory retention as two defensive behavior strategies. As a significantly lower motor activity or as an increase in the proportion of flies in a safe compartment in trained groups of animals when compared with controls groups of flies during the testing session. Context specificity experiments suggest that this type of memory is associative.

D-050

Inaccurate metaperceptions about the self-reported attraction to ingroup political extremes

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Political polarization has become a persistent problem in many societies. It refers not only to divergent ideas, but also to the ways in which we talk about each other. Previous studies show that we tend to overestimate how much other people dislike opposing political groups, inducing cognitive distortions that intensify conflicts between groups. This led us to ask whether people also overestimate the appeal of extremes within their own group. In this work, we conducted two studies (Study 1: N=140 and Study 2: N=100) where participants first placed themselves on a 7-point political ideology scale (1 = extreme left to 7 = extreme right) and then rated their level of attraction and positive/negative feelings towards each of these positions. Critically, participants responded under three different perspectives: i) personally, ii) as an imagined typical member of their own political group, and iii) as an imagined member of the opposing group. Across both studies, we observed that participants systematically overestimated the extent to which opposing groups were attracted to their own ideological extremes, while also overestimating their own group's preference for ingroup extremes. These findings reveal that when people interact with others, there may be a misperception of their preferences, contributing to the intensification of political divides. Overall, our findings suggest that correcting these metaperceptions may reduce political division and hatred in polarized societies.

D-051

Mapping neuronal activity during interval timing in Drosophila melanogaster

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Time estimation is a fundamental cognitive skill, linked to our ability to perceive regularity, involving processes like attention and memory. A leading hypothesis suggests it is a form of immediate memory, where time itself is the stimulus. To investigate this, we created a unique paradigm using hungry Drosophila that perform a Proboscis Extension Response (PER) to receive a timed, uncued sugar water reward. This approach allows us to study a-purely temporal behavior. Our research tests two main hypotheses. The behavioral one suggests that flies trained with fixed intervals will learn to anticipate the reward, while those trained with variable intervals will not.

The mechanistic hypothesis predicts that fixed and variable intervals training protocols will trigger distinct neuronal activity patterns in the flies' mushroom bodies (MBs).

Our experimental plan is twofold. First, we will compare the behavioral performance of flies trained on fixed vs. variable schedules to measure their ability to anticipate the reward. Second, we will use transgenic flies expressing CRTC::GFP to visualize and analyze the neuronal activity in their mushroom bodies after training.

Ultimately, this work will provide a direct correlation between the flies' anticipatory behavior and the underlying neural activity, thus implicating the mushroom bodies in the processing of interval estimation.

D-052

Effect of Aerobic Physical Activity on an Ischemic Cerebrovascular Event Before and After Learning

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Cerebrovascular disease (CVD) includes alterations in cerebral blood flow and may cause memory deficits, present in up to 30% of cases. It has been reported that exercise improves memory by increasing the volume of the prefrontal cortex and hippocampus. This study evaluated the effects of aerobic exercise, before and after an ischemic cerebral event, on spatial memory.

One-month-old Wistar rats were divided into five groups: sedentary, exercise, ischemia before learning, ischemia after learning, and ischemia after exercise. According to the group, the rats performed a voluntary exercise program on a wheel for three weeks. Then, the common carotid artery was bilaterally occluded for 15 minutes. Learning and memory were assessed using the Barnes maze. Finally, ischemia survival was evaluated in rats with and without exercise, considering $P<0.05$ as significant.

During short- and long-term memory tests, the ischemia and exercise groups showed a significant decrease ($P<0.05$) in latency to find the escape box compared to the sedentary group. A significant increase in ALT levels was observed after ischemia, lasting up to three weeks (48.33 ± 11 UI/L) compared to baseline values (21 ± 3.94 UI/L). Regarding survival, the exercise + ischemia group survived an average of 12 hours, while the sedentary group reached 65% survival at 60 hours.

In conclusion, both ischemia and exercise improve memory. However, survival after ischemia decreases with exercise, possibly due to adaptive factors.

D-053

Circuit of fear memory acquisition: the role of the retrosplenial cortical and hippocampal alpha 7 nicotinic acetylcholine receptors

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The retrosplenial cortex (RSC) and the hippocampus (HP) are critical brain regions involved in memory processing and are among the first to deteriorate during the early stages of Alzheimer's disease. Alpha-7 nicotinic acetylcholine receptors (α7nAChRs) play a central role in synaptic plasticity mechanisms that support memory functions. Given the dense bidirectional connectivity between the RSC and HP, investigating how α7nAChRs modulate their interaction offers a promising avenue to elucidate the circuit-level mechanisms underlying memory. In previous work, we showed that blocking α7nAChRs in the RSC of rats with methyllycaconitine (MLA) before inhibitory avoidance (IA) training enhanced memory expression 24 hours later. Here, we extend these findings by demonstrating that PNU-120596 (PNU), an α7nAChR positive allosteric modulator, impairs memory when infused into the RSC, whereas in the HP, MLA and PNU display an opposite pharmacological profile. Moreover, co-infusion of MLA into both regions before training impaired memory expression, suggesting that the HP plays a predominant role in this intervention. Together, our results support a model in which α7nAChRs regulate aversive memory processing not only within the RSC but also through its functional interplay with the HP. This reveals a potential circuit-level mechanism for α7nAChR modulation and highlights their relevance as therapeutic targets to enhance cognitive function in the early stages of Alzheimer's disease.

D-054

Developing a drug testing platform for learning and memory disorders

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RASopathies are developmental syndromes caused by germline mutations in genes of the RAS/MAPK signalling pathway, commonly associated with variable cognitive impairment. Behavioural studies with animals modelling RASopathies have shown a long-term memory impairment and alterations of the molecular dynamic involved in the spacing effect, but not other defects. Therefore, we considered the necessity to examine other behavioural paradigms, including exploratory activity, contextual recognition memory and learning generalization. These paradigms with freely behaving animals will allow us to test the effect of drugs on the behavioural defects detected. In this study, we used Drosophila to investigate how gain-of-function (CSW-A72S) and wild-type (CSW-WT) alleles of the SHP2 ortholog (CSW) affect learning and memory. Contextual habituation and recognition memory paradigms were used to assess memory specificity and generalization, comparing transgenic lines with parental controls. Our working hypothesis is that hyperactivation of RAS/MAPK signalling in CSW-A72S flies and presumably other molecular mechanisms will affect behavioural performance beside the typically long-term memory impairment. This work will advance the understanding of molecular mechanisms underlying cognitive dysfunction in RASopathies and provide a platform for testing potential therapeutic strategies.

D-055

GeNED.ar: a cohort for Brain Aging and Alzheimer's Disease Risk Assessment in Argentina

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Alzheimer's Disease (AD) is a progressive neurodegenerative disorder linked to aging. It is multifactorial, with genetic risks interacting with environmental and lifestyle factors. Non-European populations, particularly in low and middle-income countries, are still underrepresented in AD research, despite the importance of genetic and socio-environmental diversity. Considering this, we initiated a cohort representative of the Metropolitan Area of Buenos Aires (Argentina). Recruitment is continuous, including healthy adults over 18 and patients older than 60 attending the Memory Clinic of El Cruce Hospital. The protocol, approved by the ethics committee, comprises brain 3T MRI, blood sampling for genetic and plasma biomarker analyses, a socio-environmental survey with demographic and clinical data, and handedness/footedness questionnaires. Participants over 60 years old also undergo neurocognitive evaluation. To date, the cohort includes 489 individuals (339 women, 150 men), 56% older than 60 years, with 30% of them reporting <12 years of education. Preliminary analyses show that 91.4% have MRI, 59.4% are genotyped, 30.5% have plasma biomarker data, and about 67.7% present ≥30% Native American ancestry. This study will contribute to identifying biomarkers and risk factors relevant to precision medicine in diverse populations.

D-056

Conditioned Fear Paradigm for Studying PTSD-Related Behavior in Mice

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Background: Post-traumatic stress disorder (PTSD) is a psychiatric condition that may occur after exposure to traumatic experiences. The single prolonged stress (SPS) protocol, well replicated in rats, strongly affects conditioned fear responses, a key component of PTSD symptomatology. However, it has not yet been fully validated in mice. Objective: To validate an SPS model in mice in an extended contextual fear conditioned paradigm Methods: Adult male and female C57/B6 mice were subjected to four consecutive stressors (single session) followed by one week of recovery. Fear conditioning consisted of three footshocks (0.3 mA, 1 s, 1 min apart after 3 min of exploration). Extinction and spontaneous recovery involved 8 minutes of chamber exposure. Freezing was defined as the absence of movement except for respiration. Results: A sex-dependent difference was found during extinction. We observed increased freezing in SPS-exposed males on extinction day 2, while females showed the opposite effect during the mid-phase of extinction day 1. No effects observed during spontaneous recovery. Conclusion: Preliminary findings suggest that this SPS model for mice partially reproduces contextual fear conditioning effects observed in other rodent models. Further studies will include reinstatement of contextual memories and cued fear conditioning, to continue assessing the validity of SPS in mice as a model for PTSD research, which could add robustness by extending its use across species.

D-057

Time-Dependent Mechanisms in Successive Negative Contrast

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Successive Negative Contrast is a behavioral phenomenon characterized by disruption in consummatory (cSNC) or instrumental (iSNC) responses following a surprising downshift in expected reward. The aversive emotional state induced by this negative discrepancy is known as frustration. This effect is transient: approach behavior gradually recovers after several sessions with the devalued reward. Recovery has been suggested to be mediated by counterconditioning, which may explain why animals trained successively in both tasks do not show an iSNC effect after cSNC training. We conducted three experiments in which the duration of postshift trials in the cSNC task was reduced from 5 to 2 minutes, before transferring the animals to an iSNC task. Shorter cSNC postshift trials produced a stronger iSNC effect than longer trials. This effect persisted even when overall exposure to the devalued reward was equated across groups. Moreover, reducing the duration of only the first postshift trial did not affect consumption and recovery. In summary, lowering sessions duration does not affect the expression of frustration, but significantly modulates counterconditioning and transfer of frustration tolerance. Based on previous research, no differences in neural activity are expected in certain brain regions (e.g., ACC, amygdala) during the first postshift cSNC session. However, differences are expected in the remaining cSNC trials and the iSNC task, reflecting an emotional regulation process.

D-058

Ethanol-induced taste aversion strength predicts subsequent ethanol-induced locomotion in adolescent male Wistar rats: modulation by sigma-1 receptor antagonism

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The balance between ethanol's appetitive and aversive effects affects vulnerability for alcohol use disorder (AUD). This balance can be modulated by drugs such as S1RA (a sigma-1 receptor antagonist that inhibits ethanol intake) and changes across the lifespan. More work is needed during the critical period of adolescence. We examined this relationship in adolescent Wistar rats using aversive trace conditioning and ethanol-induced locomotion. Rats were pretreated with S1RA (0 or 16mg/kg). Saccharin intake was followed by ethanol administration (30 min after saccharin, 2.5g/kg). Vehicle-treated rats did not develop aversion, but S1RA enabled robust taste aversion at PD32-36. At PD39, ethanol-induced locomotion was evaluated. Correlation analysis revealed, in ethanol-treated rats, a significant association between saccharin intake and ethanol-induced locomotion ($r=0.62$, $p=0.01$). Higher saccharin consumption (weaker aversion) predicted stronger locomotor activation. This association was absent in vehicle controls, reflecting ethanol-specific effects. The correlation emerged regardless of S1RA treatment, which also reduced locomotor stimulation. The results suggest that ethanol aversion predicts sensitivity to ethanol's stimulant effects. Aversive conditioning seems to be a marker for addiction vulnerability at adolescence. S1R antagonism shifts this balance to enhanced aversion and reduced stimulation, showing promise as a treatment for AUD.

D-059

Attentional modulation of neural pathways in emotional prosody and hedonic processing: an fMRI approach

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Emotional prosody (EP) conveys affective states through acoustic modulations of the voice and is crucial for social interaction. Its processing depends on specialized brain networks, with predominant right-hemisphere involvement. Dichotic listening paradigms typically show a left-ear advantage for emotional cues, while hedonic sounds, such as alcohol-related stimuli, engage reward and motivation circuits that may compete with attentional mechanisms. Twenty-two participants were assessed through clinical interviews, neuropsychological testing, and fMRI, using a dichotic listening paradigm that manipulated attentional focus (top-down/bottom-up) and attended ear. Reaction times were longer for EP than for alcohol, with better performance when attention was directed to the right ear. At the neural level, both conditions activated partially overlapping networks, but with distinct patterns: EP recruited left temporal and frontal regions, while alcohol stimuli engaged limbic and orbitofrontal areas. These findings suggest that although both conditions involve emotional processing, they do so in distinct ways depending on the type of stimulus and the attentional context. EP may constitute a sensitive marker in clinical populations, differentiating between impairments in affective communication and heightened sensitivity to hedonic rewards.

D-060

Incompatibility between different types of perturbations in paced finger tapping experiments

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The most widely used experimental paradigm to study the sensorimotor synchronization phenomenon is paced finger tapping, where the subject synchronizes with a metronome with period perturbations to analyze the underlying error correction mechanism. In previous work, we verified the existence of a perturbation context, formed from the experience accumulated during the experiment, either by continuous exposure to a single type of perturbation or by the random alternation of two types, which affects the response. In this work, we experimentally show that within the "pure" perturbation context, responses are incompatible with each other when they come from different types of perturbation. We theoretically support this result by fitting a nonlinear model to these responses and show that the fitted parameter values are mutually exclusive. We also show that, in the case of the "combined" context, where responses are compatible with each other, the model is able to correctly fit both types of time series. These results suggest that the underlying mechanism can be represented by a single model that correctly describes a variety of situations (e.g., different sizes and signs of perturbation), but with the freedom to tune its parameter values to the context and type of perturbation.

D-061

Behavioral characterization of defensive and cognitive responses in a model of PTSD

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Post-traumatic stress disorder (PTSD) arises after exposure to traumatic events and is associated with cognitive, emotional, and behavioral deficits. The single prolonged stress (SPS) model reproduces core PTSD symptoms in rats but has proven to not be as robust in mice. In this study we aimed to validate a murine version of SPS (mSPS) by characterizing the behavioral repertoire of mice through a battery of tests (Open Field, Light/Dark, Elevated Plus Maze, Y Maze, Tail Suspension). We searched for the most salient behaviors for which this model can be useful in mice, to allow it implementation in transgenic model available in our institution. Overall, mSPS did not produce robust changes in classical anxiety measures or global working memory but revealed sex and task specific effects. No significant anxiety differences were found in open field or plus maze, although females showed altered locomotion patterns and SPS mice displayed reduced peripheral exploration. In the light/dark test, stressed males entered the dark compartment faster, consistent with increased anxiety. In the tail suspension test, SPS mice, particularly females, showed increased and earlier onset of immobility. Together, these findings indicate that mSPS induces differential sex-specific alterations in exploratory dynamics and coping styles, highlighting the importance of sex-based analyses and broader behavioral assessments to support SPSm as a translational model for PTSD in mice.

D-062

SEX OR SUGAR? Exploring the interaction between competitive motivations in sexually active female rats

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Female rats' sexual behavior is highly motivated. As the mesolimbic dopaminergic system is implicated in decision-making related to the energy cost associated with different rewards, we hypothesize that manipulating this system reduces sexual interaction in females if another reward is also available. We are validating a model of time investment preference between two rewarding activities: one more active, sexual interaction, and another more sedentary, eating palatable food (FrootLoops), in rats tested in proestrus phases of the estrous cycle. Following three training sessions —exposure to separate stimuli: male (with vaginal mask) and food— within a three-chamber model, females interacted simultaneously with both stimuli. In successive proestrus, they were tested with both stimuli after counterbalanced administration of the dopaminergic antagonist haloperidol: 0.1 or 0.2 mg/kg. When both stimuli compete, females perform both activities but spend more time on sexual interaction. Haloperidol reduced the display of proceptive behaviors, but increased the time females spent with the male, leading to a decrease in food intake. These results suggest that reduced dopaminergic transmission directs the allocation of limited energy resources toward preferred rewarding activity, sexual interaction. To strengthen the model, we will pharmacologically challenge this choice in a situation where the physical effort required to access the male is greater than that required to access food.

D-063

Cognitive Flexibility and Binge Eating: Assessment through the Wisconsin Card Sorting Test and the Cognitive Flexibility Scale

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Background: Cognitive flexibility can be assessed through performance-based tasks and self-report questionnaires, which capture different facets of this construct. Previous research on binge eating (BE) and cognitive flexibility is limited, especially studies combining both assessment methods in Argentine populations.

Objectives: This study examined cognitive flexibility in adults with (n = 38) and without BE (n = 23) across assessment methods. We hypothesized that participants with BE would show lower flexibility in self-report measures but not in task-based performance.
Methods: 61 adults aged 18-78 years completed an online survey and the Binge Eating Scale, followed by a laboratory session with the computerized Wisconsin Card Sorting Test and the Cognitive Flexibility Scale.

Results: A trend-level effect was observed on the ‘Difficulties’ subscale of the Cognitive Flexibility Scale, indicating lower self-reported flexibility in the BE group. No significant differences emerged in other CFS(Total ans Strengths) subscales or WCST indices (RT-errors, perseverative errors and perseverative responses).

Conclusions: Findings suggest an association between cognitive flexibility and BE and highlight the relevance of self-report measures, which may better reflect everyday functioning. Combining self-report and performance-based assessments is important for a comprehensive understanding of cognitive flexibility in a more ecologically valid manner.

D-064

Gestational Environmental Enrichment and Postnatal Stress: Effects on Corticosterone Levels, Social and Cognitive Functions in Male and Female Adolescent Rats

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Perinatal life is known to be a high neuroplasticity period where social behavior and cognitive abilities are shaped. Moreover, several studies report neurochemical and behavioral benefits of the environmental enrichment (EE) in adults, but their impact on offspring gestated in these conditions remains underexplored. This study examines the influence of gestational EE on adolescent rats, focusing on their ability to counteract the adverse effects of postnatal stress and potential sex differences. For this, Wistar rats were exposed to EE or standard conditions during gestation. On postnatal days (PND) 1 to 21, pups were subjected to either maternal separation (SM) stress or no stress. At adolescence (PND 45-50), social behavior and spatial learning and memory were assessed using the three-chamber test and Barnes maze respectively. Blood corticosterone levels were assessed at the end of the experiment. EE positively affects social behavior by increasing the rate of visits and time spent with social stimuli. As regards spatial learning, almost all groups showed improvements in at least one indicator, suggesting that most learn to perform the task over time. However, when evaluating contextual memory, postnatal stress turned out to have a negative impact, which could not be overcome by EE. Finally, EE, on its own, increases corticosterone levels in both sexes. In sum, results showed that both treatments, independently, influence behavior and endocrine response in adolescent rats.

D-065

Impact of a cafeteria diet on neurodevelopment and reproductive function in female mice

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Early nutrition is a critical factor for an individual's development and can affect health throughout life. Hypercaloric diets, rich in sugars and fats, have been associated with metabolic, neurobiological, and reproductive alterations. In this study, were evaluated the effects in female mice of a cafeteria diet —CAF, characterized by a combination of highly palatable sweet and salty foods— from postnatal day 21 to 119. The animals underwent behavioral testing in adulthood, and after euthanasia, brain analyses and reproductive evaluations were performed.
After 14 weeks on the cafeteria diet, we observed an increase in body weight, accompanied by elevated glucose and cholesterol levels —two metabolites linked to metabolic syndrome— while triglyceride levels remained unchanged. Additionally, across several behavioral tests the results showed a significant reduction in spontaneous locomotion and exploration, suggesting motor impairment in the CAF animals. At the brain level, markers of neuroinflammation were observed in the dorsal striatum and prefrontal cortex. In the reproductive axis, alterations were found in the number of ovarian follicles in females exposed to the cafeteria diet, along with a significant increase in serum estradiol levels. Taken together, these findings indicate that early and prolonged exposure to a cafeteria-type diet induces a phenotype characterized by locomotor hypoactivity, neuroinflammation, and reproductive alterations.

D-066

Fetal brain development during late gestation: MRI findings in a pregnancy cohort from Buenos Aires, Argentina

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Fetal magnetic resonance imaging (MRI) is a valuable tool for studying brain development, offering high-resolution characterization of soft tissue morphology. While mainly used for clinical diagnosis, recent studies have applied it to analyze normative brain growth. We present preliminary findings from an ongoing project aimed at describing prenatal brain development in low-risk pregnancies with no suspected pathology, within a socially vulnerable population from the southern Metropolitan Area of Buenos Aires (AMBA), Argentina. The sample included 24 pregnant women with a mean gestational age of 31.33 ± 2.02 weeks. Using features of cortical maturation, cortical age was estimated for each fetus, with a mean of 28.69 ± 3.17 weeks. The correlation between gestational and cortical ages was moderate but highly significant ($r = 0.55$, $p < 0.01$). Standard biometric measures —fronto-occipital length, biparietal diameter, and head circumference— were also taken, showing stronger associations with gestational than cortical age. This work presents an initial insight into a distinctive sample, shaped by the specific population studied and the imaging technique employed. Further studies will seek to enlarge the sample and examine how fetal traits relate to maternal characteristics such as nutrition, health status, and broader sociodemographic factors, as well as to explore potential mismatches between growth and the expected maturation for gestational age.

D-067

Striatal Stem Cell 3D Model: A Tool to Study Neurotrophic Factors in Regeneration

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Neurotrophic factors (NF) are critical for neuronal survival, differentiation, and regeneration. We investigated the effects of Noggin, FGF-9, and GDNF on neurite outgrowth and proliferation using a three-dimensional (3D) model of neural spheroids (NS) derived from corpus striatum (CS) stem cells of E14 rat embryos. NS were cultured in suspension and embedded in type I collagen to assess NF effects at 24–48 h. Proliferation was analyzed with the MTT assay, while immunocytochemistry was performed for Ki67, Nestin, TubIII, c-FOS, and GFAP. Clear NS formation was observed, confirming stem cell properties. NF stimulation significantly enhanced neurite extension compared to controls, with Noggin showing the strongest effect on axonal elongation. After 48 h, stimulated cultures displayed higher expression of neuronal and glial markers, indicating differentiation. Conversely, FGF-9 predominantly promoted NS proliferation in suspensión cultures. These findings demonstrate that the 3D collagen assay supports NSC proliferation and differentiation with high reproducibility, providing a reliable platform to study NF action in regenerative medicine.

D-068

Sex-dependent interaction of hyposerotoninergy with early life stress in the serotonin system and adult behavior

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Early-life stress elicits anxiety and depressive-like behaviors both in humans and in rodents. At the same time, serotonin deficits have been linked to the origin of these mental disorders, and drugs enhancing brain serotonin levels are the first line treatment for such neuropsychiatric conditions. Here we sought to investigate the role of serotonin in a model of early-life stress in mice. C57BL/6 pups were subjected to the maternal separation protocol (3h/day) during a postnatal critical period (P2-P14) while receiving daily injections of the tryptophan hydroxylase inhibitor para-chlorophenylalanine (PCPA, 10mg/kg/day s.c.). We analyzed the synaptic innervation of the prefrontal cortex to the dorsal raphe nucleus (PFC-to-DRN) circuit by high-resolution microscopy (array tomography), and serotonin cell activation by c-fos immunohistochemistry. Lastly, emotional responses to stress were evaluated in a battery of behavioral paradigms at adulthood (from P80). Our study showed that male mice were more susceptible to stress, resulting in long-lasting anxiety and depressive-like behaviors. PFC-to-DRN circuit connectivity alterations by stress were prevented by depletion of serotonin, and DRN responses to an acute stress were altered in mice receiving PCPA. Our results emphasize the emotional alterations caused by early-life stress and the interaction with the serotonin transmission, in the search for understanding a main risk factor for the development of psychiatric disorders.

D-069

Melatonin and behavioral alterations in schizophrenia: Preliminary results from an experimental rat model

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Schizophrenia is characterized by delusions, hallucinations, cognitive deficits, and social withdrawal. Emerging evidence links its onset to harmful intrauterine factors, leading to the development of maternal immune activation (MIA) as a relevant experimental model. This study evaluated behavioral alterations and the potential neuroprotective role of melatonin (MEL) in a rat model of schizophrenia induced by MIA. Three groups were established: a) MIA rats, injected with poly I:C acid (10 mg/kg, GD12); b) MIA+MEL, injected with poly I:C and treated with MEL (30 mg/kg, GD12 to two weeks postpartum); and c) Controls, injected with saline. Male offspring were assessed at postnatal day 30 using the open-field and elevated plus maze tests. In the maze, MIA offspring entered open arms less frequently than controls (3±0.60 vs 5.33±0.69; $p<0.05$), suggesting impaired anxiety regulation; melatonin did not prevent this effect (2.69±0.47). In the open-field, MIA rats showed reduced exploratory behavior, with fewer head dips compared to controls (6.5±2.97 vs 10.7±5.18; $p<0.05$). Melatonin showed a trend toward improvement (7.93±3.45; $p=0.05$). These findings suggest that MIA induces measurable anxiety and apathy-like behaviors in offspring. While melatonin did not reverse all alterations, it showed partial benefits in exploratory activity. Further behavioral and neurobiological studies are needed to clarify its preventive potential in schizophrenia-related neurodevelopmental disturbances.

D-070

Embryonic development and characterization of the hippocampal formation in the plains vizcacha, Lagostomus maximus, a gyrencephalic South American rodent

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The hippocampal formation (HF) is a key structure for learning and long-term memory, which shows highly conserved ontogenetic development and laminar organization throughout mammalian evolution. The aim of this work was to analyze HF development in the plains vizcacha, a native precocial hystricomorph rodent with a ~155-day gestation period. Histological analysis was performed from 40 embryonic days (e.d) up to birth (n=16). Moreover, the adult HF was characterized by immunofluorescence of several markers (n=4). As early as 43 e.d., the hippocampal primordium was identified, including the ventricular, intermediate, hippocampal, and marginal zones. At 58 e.d., the HF evagination positioned the subiculum, hippocampus, and dentate gyrus, coinciding with the beginning of the CA3 pyramidal stratus, which gets mature at 80 e.d. By ~96 e.d., the CA layers gets fully organized similar than adults. All these observations add temporal characteristic to this precocial species. In adults, HF strata displayed a horn architecture, with parvalbumin (PV) GABAergic interneurons scattered in the oriens and radiatum strata, and concentrated in the pyramidal stratum of CA1 and CA3. These interneurons coexpress AMPA glutamate receptor in the pyramidal and oriens strata, and kainate in all the strata. The expression of PV and glutamate receptors in this region suggests their role in synaptic modulation and plasticity, with implications in the social memory and exploration behavior of the vizcacha.

D-071

Dynamic characterization of axon guidance mediated by EphA3

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Previously we demonstrated that the tectal gradient of EphA3 participates in the retinotectal mapping by stimulating the axon growth of nasal retinal ganglion cells (RGC) towards the caudal tectum and inhibiting their branching in the rostral tectum. This process is mediated by a decrease in ephrin-A-dependent activity of axonal EphA4. Eph/ephrin interactions take place in the cholesterol-rich lipid rafts. There are conflictive data about the role of colesterol microenvironment on axonal growth and guidance and the dynamics of this process is incompletelly understood.

Our objective was to characterize the dynamics of nasal RGC axon growth and guidance mediated by EphA3 gradient and determine the relationship of the cholesterol microenvironment with this process. For this purpose, dissociated NRGCs from chicken embryos were cultured and exposed to a gradient of EphA3-Fc produced in the Dunn’s chemotaxis chamber in time lapse. We also evaluated the effect of depletion of the plasma membrane cholesterol with βMCD.

The results showed that the EphA3 gradient has a positive chemotactic effect on NRGC by increasing the axon extension rate and promoting directional attraction, effects that were significantly decreased by transient cholesterol depletion.
Our findings provide novel insights into how axon dynamics are regulated by EphA3 during retinotectal map formation and demonstrate that the cholesterol microenvironment modulates this process.

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D-072

Postnatal fluoxetine modulates the mouse prefrontal emotional circuit development

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Depression and anxiety are leading causes of disability worldwide, yet their developmental origins remain unclear. To explore early mechanisms of vulnerability to psychiatric disorders, we used a mouse model of adult emotional vulnerability induced by the early postnatal exposure to the antidepressant fluoxetine (FLX). C57BL/6 mice (both sexes) received FLX (10 mg/kg/day, p.o.) in 3% sucrose from postnatal day (P)2 to P14. At P15, we investigated the early impact on the prefrontal cortex-to-dorsal raphe nucleus (PFC-DRN) circuit, which is implicated in stress coping and mood regulation. Using the high-resolution microscopy technique, Array Tomography, we observed a selective ~40% increase in glutamatergic PFC inputs to DRN serotonin (5-HT) neurons. Ex-vivo patch-clamp recordings supported the presence of additional functional glutamatergic synapses. Following acute stress in the forced swim test (FST), c-fos immunohistochemistry and layer-specific markers revealed heightened activation of specific PFC projection-neurons and increased 5-HT1A receptor-mediated inhibition in the DRN. Behaviorally, FLX-exposed mice showed reduced immobility in the FST, an effect reversed by 5-HT1A receptor blockade using the selective antagonist WAY-100635. Altogether, these findings reveal that postnatal FLX induces structural and functional remodeling of the nascent PFC-DRN circuit, likely contributing to altered stress responses and emotional behavior later in life.

D-073

Behavioral and Molecular Characterization of a Mouse Model of Alzheimer's Disease

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Alzheimer's disease (AD) is characterized by progressive cognitive decline and memory loss. Animal models have been key to unraveling the molecular basis of AD pathology, yet behavioral characterization remains essential to link these alterations to functional outcomes. We have previously studied triple transgenic mice (3xTg-AD; PS1M146V, APPSwe, tauP301L; hybrid background) and reported memory impairments in novel object recognition, along with changes in amyloid aggregation and ERK1/2 signaling.

Here we present an extended behavioral characterization of a novel congenic mouse line, 3xTg-C57, which combines the 3 transgenes with C57BL/6 genetic background. Male and female 3xTg-C57 and WT-C57 mice aged 4, 6, and 8 months were tested in tasks including open field, elevated plus maze (anxiety-like behavior), Y-maze (working memory), novel object recognition (episodic memory), and social preference/memory paradigms.

In parallel, Congo red staining was applied to fixed-brain slices from the 3xTg-C57 and WT-C57 mice to assess β-amyloid deposition profiles, enabling the integration of brain histological characterization with behavioral features. By combining behavioral and histological analyses, this study aims to uncover sensitive functional outcomes of AD progression and improve the model's translational utility.

D-074

Inflammation, IGF-1, and Blood–Brain Barrier Integrity: Insights into Parkinson's Disease Pathophysiology

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Parkinson’s disease (PD) pathophysiology is associated with neuroinflammation and blood–brain barrier (BBB) dysfunction, yet the underlying mechanisms remain unclear. Microglia, a major source of insulin-like growth factor-1 (IGF-1) in the central nervous system, may influence BBB integrity during inflammation. In this study, we examined the effects of human IGF-1 (hIGF-1) on BBB cell injury induced by TNF-α and IFN-γ (TI) in vitro, using human microglial (HMC3) and brain microvascular endothelial (TY-10) cell lines. TI exposure increased pro-inflammatory markers (NLRP3, COX-2, pIκB) in HMC3 cells, elevated reactive oxygen species (ROS) production, and reduced TY-10 viability while altering structural proteins (decreased claudin-5, increased ICAM) and enhancing endothelial permeability. hIGF-1 treatment prevented ROS accumulation in HMC3 cells and upregulated claudin-5 in TY-10 cells but did not rescue TI-induced permeability or viability loss in endothelial cells. These findings indicate that hIGF-1 exerts partial protective effects against inflammatory stress in these cells, reducing oxidative damage and modulating tight junction proteins, yet may be insufficient to fully counteract inflammation-induced functional deficits. Further work is needed to define the pathways involved and evaluate hIGF-1’s therapeutic potential in PD-related BBB impairment.

D-075

From the mate to the neuron: exploring active compounds behind neuroprotection and their potential role in Parkinson's disease

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Regular intake of yerba mate (YM) is beneficial for Parkinson's disease (PD), including a lower prevalence of developing the disease in humans and a robust neuroprotective effect on dopaminergic neurons in vitro from our lab. Also, unpublished results show that YM activates the AMPK pathway and autophagy, which are strongly linked to cell homeostasis.
The hallmark of PD is the progressive death of dopaminergic neurons, however, its cause is uncertain. In order to understand this neuroprotective effect, we aim to investigate the molecular pathways regulated by YM extract and its main active compounds in a neuronal cell line, as a cue to understand the cellular changes that could mediate YM-induced neuroprotection.

Here, we aim to dissect the effect of the main active compounds present in YM, as well as other widely used beverages, such as tea and coffee.
We treat SH-SY5Y cells with chlorogenic acid, theobromine, and caffeine, and we are exploring the regulation of AMPK and autophagy through qRT-PCR, western blot (WB), and histology.

Our pioneering results suggest that YM regulates cell metabolism, although it is still unknown which active compounds and downstream pathways are involved. Further work is still necessary to fulfil our hypothesis, but current results help to envisage how natural compounds may modulate neuronal health and therefore impact the natural history of neurodegenerative pathologies such as Parkinson’s disease.

D-076

Diagnostic utility of plasma pTau217 for Alzheimer's Disease in the GeNED.ar cohort

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Alzheimer's disease (AD) is the leading cause of dementia, and its early detection is key for accurate diagnosis and treatment. Plasma phosphorylated tau 217 (pTau217) has emerged as a promising non-invasive biomarker. This study aimed to establish a population-specific cut-off for plasma pTau217 levels in Argentina using the GeNED.ar cohort (Genetics and Neuroimaging of Aging and Dementia in Argentina). We analyzed data from 137 individuals: 83 cognitively unimpaired (CU), 34 with mild cognitive impairment (MCI), and 20 with AD. Finite Mixture Modelling was used to define thresholds without relying on gold-standard methods such as PET or CSF biomarkers. We estimated both a single standard cut-off and a two-threshold approach at probability levels of 0.8, 0.9, and 0.95. The latter defines a lower threshold to identify a high-sensitivity zone (likely negative) and an upper threshold for a high-specificity zone (likely positive), with an intermediate "gray zone" requiring further testing. The two-threshold method at the 0.9 probability level showed the best performance, with lower and upper thresholds of 0.47 and 0.83, respectively. These thresholds are comparable to those reported in other populations. The results also support the clinical diagnoses established at the Memory Clinic. Our findings reinforce the utility of plasma pTau217 as a scalable and accessible diagnostic tool for AD.

D-077

Striatal Cholinergic Interneurons: key players in motor learning and perseverance

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Striatal cholinergic interneurons (SCIN) are key modulators of motor learning, cognitive flexibility, and strategy selection. Our previous work showed that SCIN ablation did not impair motor skills but altered spatial navigation, hindering learning and increasing perseveration. Yet, how SCIN contribute to different phases of motor learning and perseverative behaviors remains unclear. To elucidate this, adult heterozygous Chat-Cre mice (males/females) were injected with a viral vector to express an inhibitory DREADD (hSyn-DIO-hM4D-Gi-mCherry) in the SCIN. Three weeks later, vehicle (control) and CNO-treated mice were tested in an accelerating rotarod task (4–40 rpm) across 5 consecutive days (5–7 trials/day). CNO was injected only on Day 1, while vehicle was administered throughout. CNO-treated mice showed enhanced performance compared to controls on Day 1, suggesting that SCIN inhibition at this stage may influence strategy selection. To further examine perseverance, adult heterozygous Chat-IDTR mice (males/females) received diphtheria toxin (DT) to ablate SCIN, followed weeks later by corpus callosum lesions which disconnected the orbitofrontal cortex (OFC) from the striatum. DT and control mice were tested before and after surgery in marble burying, hole board, and Y-maze tasks. DT mice displayed perseverative behaviors, which might be alleviated after OFC-striatal disconnection. In summary, our findings suggest SCIN involvement is more multifaceted than initially thought.

D-078

Sexual Dimorphism and Cognitive Decline in an Animal Model of Brain Amyloidosis Versus Aging: Implications for Precision Medicine in Alzheimer's Disease

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Historically, sex differences in biology were underexplored due to the exclusion of females as research subjects, creating a gap in women's health knowledge only recently addressed. Sex differences are now recognized in prevalence, risk, and treatment response across many diseases. The McGill-R-Thy1-hAPP transgenic (Tg) rat, carrying two familial Alzheimer's disease (AD) mutations (Swe/Ind), is a valuable model to study sex differences. Wt and hemizygous (Tg+/-) rats of both sexes were evaluated at 4 and 12 months (mo). All groups showed open-field (OF) habituation. In inhibitory avoidance (IA), wt and Tg+/- females formed Long-Term Memory (LTM), but only wt females retained it; Tg+/- males did not form memory. In novel object recognition (NOR), only wt animals consolidated LTM. In novel object location (NOL), at 4 mo, wt and Tg+/- females discriminated the new location, whereas at 12 mo only wt females did so. Deficits in Tg+/- animals were more pronounced in males and worsened with age. Brain amyloid-β measurements showed higher load in males at 4 mo and greater sex heterogeneity at 12 mo. Our results show sexual dimorphism in learning and associative memory in middle-aged rats, especially hippocampus-dependent tasks. In the Tg model, it is most evident in aversive and spatial memory. Thus, incorporating sex as a biological factor will be essential to advance toward precision medicine in AD, as well as to achieve a more personalized and sustainable approach to patient care.

D-079

5-HT7 receptor modulates adult emotional alterations induced by early-life stress in mice

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Early-life stress of maternal separation (MS) during postnatal days 2 to 14 (P2- 14) in mice produces anxiety and depressive-like symptoms in adulthood. Emotional alterations are accompanied by alteration in prefrontal cortico-limbic pathways involved in mood control. The serotonin (5-HT) system plays a key role in the MS model, however the specific participation of 5-HT7 receptors in the manifestation of the adult anxiety and depressive-like symptoms remains unknown. In this study, cohorts of mice were exposed to MS during P2-14 while administering a selective 5-HT7 receptor antagonist (SB-269970, s.c. 20mg/kg/day) and their respective controls. We assessed in adulthood anxiety and depressive-like behaviors in all our experimental groups. The results evidence a key role of 5-HT7 receptors in the early-life vulnerability to stress resulting in adult predisposition to develop emotional alterations of relevance to mental disorders.

D-080

Cytoplasmic mislocalization of TDP-43 in clock neurons leads to arrhythmicity without impaired sleep in a novel Drosophila model of proteinopathy.

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Circadian rhythm abnormalities, including sleep-wake disturbances, are common features of neurodegenerative diseases, including amyotrophic lateral sclerosis (ALS) and frontotemporal dementia (FTD). Although pathological aggregation and cytoplasmic mislocalization of TDP-43 is a key event in ALS/FTD, the relationship between this protein and circadian rhythms remains unclear. We developed new Drosophila TDP-43 models to investigate its role in rhythmicity, sleep and memory. In young male adult flies, overexpression of wild type hTDP-43 (WT) in circadian neurons showed no impact on rhythmicity. The period (Tau) was not different from control animals, with a trend for decreased Tau power. In contrast, sleep analysis revealed increased sleep bouts and mean sleep length during night, but decreased total sleep duration during day. Conversely, overexpression of a hTDP-43 variant with a mutated nuclear localization signal (ΔNLS) led to a complete disruption of circadian rhythms, with no flies showing identifiable period. Surprisingly, sleep was not disturbed. When analyzing the effect of hTDP-43 overexpression in mushroom bodies neurons over inhibitory control of proboscis extension reflex, both WT and ΔNLS young flies showed memory indexes indistinguishable from control group. These results define cytoplasmic TDP-43 mislocalization as a key player in circadian disturbances, and provide new evidence for the role of this protein in human diseases of the ALS/FTD spectrum.

D-081

Chronic Depolarization Triggers Channel Remodeling and Neuroinflammation in the Cochlear Nucleus

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First- and second-order sensory neurons of the ascending auditory pathway, located in the spiral ganglion and the cochlear nucleus (CN), respectively, participate in the olivocochlear reflex, which protects hair cells from acoustic overstimulation. The KCNQ4 channel is expressed in the organ of Corti and has also been detected in the CN. In knock-out (KO) mice for KCNQ4, sensory cells and spiral ganglion neurons degenerate with age, while the impact of KCNQ4 deletion on the CN remains unknown. Therefore, we analyzed the structural integrity of the CN at middle age in KO animals. We examined the expression of the neuronal KCNQ channel subunits in the CN by RNA isolation, followed by qPCR. KCNQ3 expression was significantly increased ($p=0.0418$) in KO mice compared to WT animals at young age, suggesting changes in the excitability of CN neurons. In addition, we assessed microglial status in the CN of middle-aged KO mice by brain cryosectioning, followed by immunohistochemical analysis of the microglial marker Iba1. Confocal microscopy revealed a notable increase in the fluorescence intensity ($p=0.0013$) and in the number of microglial cells from 0.29 to 0.86 cells/µm2 ($p<0.0001$) within the CN of KO mice compared to WT. This finding suggests alterations in neuronal excitability and enhanced microglial reactivity, potentially associated with neuroinflammatory processes that may contribute to neuronal loss in aged animals.

D-082

Aloysia citriodora extract attenuates motor and non-motor impairment in a progressive model of Parkinson´s Disease induced by reserpine

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Motor and non-motor symptoms in Parkinson's disease (PD) lead to progressive disability. Current therapies often failed to halt this disease progression. This underscores the promise of phytotherapeutic approaches. Aloysia citriodora, traditionally used in Paraguay for its sedative and anxiolytic properties, has shown antioxidant and anti-inflammatory activity in preclinical studies, suggesting its potential for neuroprotection. This study aimed to assess the effect of A. citriodora extract (EAc) in a reserpine (RES)-induced model of PD. Male mice received 20 s.c. injections of 0.1 mg/kg RES or vehicle, every other day, alongside daily oral administration of EAc (30, 150, or 300 mg/kg). Catalepsy test was performed every other day, and assessment of vacuous chewing movements (VOM) every 10 d. To investigate non-motor symptoms, short-term memory was assessed using the novel object recognition (NOR) test. Our results show that, even at low doses, EAc attenuates catalepsy progression. Interestingly, a reduction in VOM was observed (by d 30) with 30 mg/Kg of EAc, while all doses produced comparable effects at d 40. In addition, 150 mg/kg of EAc prevented cognitive impairment in NOR. Taken together, our results suggest that EAc shows a neuroprotective effect, attenuating catalepsy, orofacial dyskinesia, and cognitive impairments induced by RES. This study highlights the potential use of A. citriodora as a promising therapy for the prevention and/or treatment of PD.

D-083

Effect of fluoxetine on motor function and anxiety-like behavior in a reserpine-induced parkinsonism animal model

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Parkinson's disease (PD), the major motor disorder known, has been currently characterized also as a progressive multisystemic disease with non-motor symptoms (NMS) like depression, affecting around 50% of its sufferers. While selective serotonin reuptake inhibitors are the primary treatment for such NMS, studies on acutely induced parkinsonism have linked fluoxetine (FLU) to increased motor impairment. The present study aimed to assess fluoxetine's effects in a parkinsonism model. Sixty-four male Wistar rats (7–9 months old) were divided into groups: 1) FLU + reserpine (RES) vehicle; 2) FLU 10 mg/kg + RES vehicle; 3) FLU 10 mg/kg + RES 0.1 mg/kg; 4) FLU vehicle + RES 0.1 mg/kg. Behavioral tests were made open field, catalepsy, and oral movements. Rats treated with both fluoxetine and reserpine displayed increased catalepsy duration, reduced travel, lower rearing, more tremulous jaw movements, and greater weight loss, with distinct immunohistochemical results between acute and prolonged treatments. Fluoxetine-treated rats showed reduced TH immunoreactivity in the dorsal striatum, decreased TH-positive cells in the SNpc, and increased serotonin in the dorsal raphe nucleus. Reserpine-treated rats showed reduced TH-positive cells in the SNpc and dorsal striatum, with no motor correlation. Our results pointed that fluoxetine exacerbated reserpine-induced motor alterations, which were not linked to TH immunohistochemical changes.

D-084

Essential but implicit: the role of aging information in neurodegeneration detection

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A widespread hypothesis in brain imaging posits that neurodegenerative disorders constitute premature aging. Despite its prominence, this brain aging hypothesis (BAH) has not been verified against suitable alternatives. In this work, we first test a key assumption of BAH: Age information is necessary for detecting Alzheimer's Disease (AD). We compared brain representations that were maximally uninformed about chronological age against ones that were maximally informed about age. We found that absence of aging information impairs AD detection, providing causal evidence for BAH. Second, we investigated whether explicit age modeling confers advantages in transfer learning for AD detection. We evaluated pretraining strategies for age, sex, and BMI inference and found that while pretraining improved representation stability and quality, these tasks converged to similar learned representations with no single phenotype providing superior advantage for neurodegeneration detection. Rather, all fine-tuned models for disease detection implicitly developed increased structural similarity to the age-inference model. These findings demonstrate that aging and neurodegeneration are fundamentally linked, yet aging information emerges naturally during learning of brain features without dedicated encoding. This moves current thinking past brain-age gap conceptualizations and suggests new directions for foundation models integrating richer phenotypic information.

D-085

Modulation of kainic acid-induced excitotoxic spinal cord injury via purinergic signaling pathways.

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Purinergic signaling plays a crucial role in somatosensory and nociceptive transmission under pathological conditions, such as spinal cord injury (SCI). Selective antagonists and allosteric modulators of P2X4 and P2X7 receptors are under investigation for neuropathic pain management. In our previous studies, we monitored endogenous real-time glutamate release during in vitro SCI experimental protocols induced by kainate (KA) (Mazzone and Nistri, Neurochem Int. 2019, 128:175-185). This study aimed to evaluate the role of purinergic signaling following chemical SCI in a mouse model. Our results revealed endogenous ATP release induced by KA (100 µM), monitored using a commercial biosensor, concomitantly with glutamate. P2X4 and P2X7 receptor expression was evaluated by RT-PCR after KA treatment. Additionally, we explored the effects of Coomassie Brilliant Blue G (10 µM, CBO), a non-nucleotide purinergic antagonist, on pyknotic cell death, as well as on the number of neurons, astrocytes, and microglia. Immunohistochemical analysis showed a significant reduction in neuronal loss in the KA+CBO group, with no observable effects on glial cells. Finally, in vivo administration of KA led to a significant impairment of locomotor function, which was partially reversed by CBO co-treatment. These findings suggest that early ATP release following excitotoxic injury may modulate neuronal survival and locomotor network function. Supported by Universidad Austral, CONICET, FONCYT, and IBRO.

D-086

Dynamic modeling of central regulation of heart rate to investigate respiratory sinus arrhythmia

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This study presents initial results of a project aimed at investigating emotional regulation (ER) capacities—specifically through the process of attentional deployment (AD)—and their underlying autonomic physiological dynamics.

Here, we focus on a minimal model of neuronal control of heart rate to better understand respiratory sinus arrhythmia (RSA), a natural variation in heart rate that increases during inhalation and decreases during exhalation. Because the vagal system promotes calm states, vagal tone is considered an indicator of emotional regulation: reduced vagal tone reflects lower regulatory capacity. RSA monitoring provides an accessible method for assessing this control. Heart rate variability (HRV) is another relevant marker, as higher HRV has been associated with more effective emotional regulation strategies.

The presented model integrates three main components: the cardiac system, the pulmonary system, and neuronal dynamics. A detailed analysis of its structure and parameters is provided, with the aim of advancing understanding of the physiological mechanisms underlying heart rate regulation and their relevance for emotional processes.

D-087

Neural correlates of exploration/exploitation decisions in a virtual corridor task

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From staying in a shelter or exploring a new one, to keeping a job or searching for another one, we often have to decide between exploiting a resource or exploring the environment for new opportunities. Information about the environment and the animal's state converges in the dorsomedial striatum, considered as an integrator of exploration/exploitation decisions.
To study the role of the dorsomedial striatum in the exploration/exploitation balance, we designed a virtual reality foraging task. Head fixed mice explore a virtual linear track which consists of short rewarded areas ("patches") separated by long unrewarded corridors. Inside the rewarded area, animals need to perform a sequence of licks to obtain water. Each consecutive reward requires exponentially more licks to be obtained. At some point, the animal reaches a breaking point when it decides to stop exploiting the current patch and run to the next one. We used an array of four chronically implanted tetrodes to record single unit and LFP striatal activity during the performance of the task.

Preliminary data show that increasing the length of the unrewarded corridor raises the effort the animal invests in each patch. We aim to align the behavioral events with striatal neuronal activity to find correlates predicting decision making by using multiple logistic regression. We will also focus on striatal cholinergic interneurons known to encode reward-related cues.

D-088

Selective inhibition of LEC-to-PCx projection neurons using a dual viral strategy

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The general framework of this work is to understand how experience modulates olfactory processing. In particular, we study the plasticity of olfactory cortex representations of odors associated with a specific aspect of sensory experience: the spatial context in which odors are presented. Using an olfactory-visual context associative task, we recently found that odor representations in the piriform cortex (PCx), the largest region of the olfactory cortex, also encode spatial context information. A candidate source of these contextual inputs to the PCx is the lateral entorhinal cortex (LEC), a region widely involved in context encoding and memory processing, which is monosynaptically connected to the PCx.

To investigate the role of the LEC-PCx pathway, we use a dual-virus approach to selectively inhibit LEC-to-PC projections in vivo. Specifically, we combine a retrograde virus (retroAAV-DIO-HM4Di-Cherry) with an anterograde virus (AAV-CamKII-GFP-Cre) to express the inhibitory DREADD receptor HM4Di in LEC neurons projecting to the PCx. Preliminary results show that dual injections successfully label neurons co-expressing both red and green fluorescent proteins in the LEC. Future experiments will assess how this manipulation affects PCx activity and the behavior of animals trained in the associative task.

D-089

Critical contributions of young adult-born granule cells to dentate gyrus function and CA1 spatial representations

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The dentate gyrus (DG) of the hippocampus is one of the few brain regions where new neurons are continuously generated throughout adulthood. Adult-born granule cells (ABGCs) in the DG are thought to support pattern separation, the process by which overlapping experiences are transformed into distinct memory traces. Although behavioral studies implicate ABGCs in this process, the specific computational mechanisms by which they modulate activity or plasticity in downstream hippocampal circuits remain unclear. Here we performed a modified spontaneous location recognition task in mice to assess how reversible silencing of ABGCs affects behavior, and to examine how their activity, as well as that of downstream circuits, contributes to behavior. We show that silencing even a small cohort of young ABGCs disrupts pattern separation, revealing that -during their critical maturation window-, these neurons exert a disproportionately strong influence on DG-dependent behavior. We also characterize CA1 spatial maps obtained through high-throughput calcium imaging microendoscopes. Collectively, these results link behavior and population imaging to reveal how adult neurogenesis shapes hippocampal circuits involved in spatial discrimination and memory.

D-090

Plasticity changes linked to social recognition memory in mice: an electrophysiological approach

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Social recognition is the ability of an individual to identify and remember conspecifics based on previous encounters. It represents a form of social memory that is crucial for social species, as it influences decision-making, pair bonding, and the establishment of dominance hierarchies. Mice and rats are commonly used to investigate the mechanisms underlying memory formation, including Social Recognition Memory (SRM), due to their strong social behavior and natural preference for novel conspecifics over familiar ones. This behavioral trait facilitates the assessment of SRM in experimental settings. The Social Recognition Task (SRT) is a widely used paradigm to evaluate SRM. In our protocol, the experimental subject explores two unfamiliar conspecifics during a training session. One hour later, a test session is performed in which the subject is presented with one familiar and one novel conspecific. A preferential interaction with the novel conspecific is interpreted as evidence of SRM formation. One of the main goals of our work is to understand the role of oxytocin (OXT) in the formation of SRM. In this project, we focus on investigating plastic changes in brain regions that express OXT receptors, such as the hippocampus, using techniques like ex vivo extracellular recordings and patch-clamp, in mice that underwent the SRT. Elucidating the biological basis of SRM formation may contribute to a better understanding of disorders involving social cognition deficits.

D-091

Cognitive Bias in Adult Zebrafish Under Social Isolation: Preliminary Results

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Cognitive bias in decision-making under ambiguity is observed in animals and humans, and some individuals interpret ambiguous stimuli as positive (“optimistic”) while others as negative (“pessimists”). Social animals use both asocial and social cues to predict the presence of a reward or punishment in the environment through associative learning. In recent years, experimental paradigms have been developed to assess cognitive bias in various species based on their interpretation of ambiguous stimuli. The aim of this study is to determine whether a period of social isolation of adult zebrafish (Danio rerio) can affect judgement of an ambiguous stimulus, i.e. if it can bias the proportion of “optimistic”/”pessimistic” fish. We thus implemented a Go/No-Go task in a color-coordinated half-radial maze, where fish are trained to discriminate between a positive (P) and negative (N) arm. Discrimination learning is measured by the latencies to enter these arms. A Judgment Bias Score (JBS) is calculated based on latencies to enter the P, N, and an Ambiguous arm, allowing classification on an optimistic/pessimistic axis. In our preliminary results we observed two distinct behavioral phenotypes: active fish that explore all the maze and more static animals that either freeze or perform brief excursions. We also analyze how they interpret ambiguous stimuli and whether social isolation affects this cognitive process.

D-092

Zebrafish as a model to study states of consciousness.

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Understanding the neural mechanisms underlying different states of consciousness and the impact of traumatic brain injury (TBI) in humans presents major challenges in neuroscience. Mild TBIs often produce symptoms without detectable neuropathology in standard neuroimaging, while severe injuries linked to disorders of consciousness lack effective therapeutic strategies due to limited mechanistic insight and ethical constraints in clinical trials. To address these gaps, we are developing a novel research model using zebrafish to explore changes in brain dynamics during different states of consciousness. By using light-sheet microscopy to record whole-brain neuronal activity in zebrafish larvae while applying anesthesia, we emulate changes in consciousness and compare brain dynamics with global brain patterns observed in humans. Preliminary results suggest that awake zebrafish exhibit brain signatures analogous to those found in awake primates. The next step involves building a system to precisely control anesthesia levels and study transitions in brain states. Inspired by findings in humans and non-human primates where thalamic stimulation alters neural network dynamics, we are developing transgenic zebrafish lines expressing UAS-opsins targeted to the thalamus. This enables optogenetic activation or silencing of thalamic regions to assess recovery of brain patterns. This ambitious project opens new avenues to decode the neural basis of consciousness.

D-093

Does Social Isolation of Larval Zebrafish affect Behavioral Transition in a Passive Coping Assay? Preliminary Results

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Animals tend to avoid aversive or stressful experiences and select actions intended to escape from those situations. However, if the action (escape) fails to counteract the aversive event, animals may adaptively suppress the escape. Repeated failure to avoid an aversive stimulus by performing an escape can result in a deep discounting of the value of escaping. In humans, this discounting is maladaptive when manifesting as hopelessness, one of the core clinical criteria of major depressive disorder. Social isolation during early development can affect judgement of the value of negative events and is in addition a contributing factor to depression.

Our lab studies how social isolation affects behavioral decisions and particularly the judgement of negative events using zebrafish as model system. We aim to determine whether hopelessness can be affected by social isolation during early development. To do this we implemented a passive coping assay for larval zebrafish: 15 days old larvae raised either in a social context or in isolation were exposed to an inescapable aversive stimulus (mild shock) to study the transition from an active coping mechanism characterized by attempts to escape to a passive coping mechanism where mobility is reduced. Here we present our first preliminary results analyzing whether social- or isolated raised larvae show differences in reactivity to the aversive stimulus and in the transition from active to passive coping strategies.

D-094

Post Treatment Structural Correlates of Electroconvulsive Therapy in Patients with Treatment Resistant Depression

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Electroconvulsive therapy (ECT) remains one of the most effective interventions for treatment-resistant depression (TRD). This study investigated whether cortico-limbic structural connectivity following ECT is associated with symptom severity and clinical response. Twenty-three patients with TRD underwent diffusion MRI after bifrontal ECT. Structural connectivity (SC) was estimated using probabilistic tractography between seven bilateral regions previously implicated in depressive symptomatology. Symptoms were assessed with the Hamilton Depression Rating Scale. After ECT, 18 connections were significantly associated with clinical response. Specifically, greater clinical improvement was linked to lower SC within a fronto-limbic network. When restricting the analysis to responders (change in Hamilton score higher than 50%), this association remained significant, with the right anterior insula emerging as a key hub. Interestingly, this pattern was already evident in the pre-treatment SC of the right anterior insula, where higher connectivity predicted poorer response. These findings suggest distinct structural biomarkers of treatment outcome and underscore the importance of anatomically precise neuromodulation strategies to enhance ECT efficacy while minimizing adverse effects in TRD.

D-095

Neuropathic pain selectively disrupts hedonic and motivational eating behaviors

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Chronic pain disrupts affective and motivational states, yet its impact on eating—particularly the balance between homeostatic and reward-driven intake—remains unclear. Using the spared nerve injury (SNI) model in male and female mice, we examined how persistent neuropathic pain affects feeding and reward. Fourteen days post-surgery, mice underwent a binge-eating protocol, operant conditioning for chocolate pellets, and a sucrose preference test. Daily chow intake and body weight were monitored to assess homeostatic feeding. SNI induced mechanical hypersensitivity without affecting chow intake or body weight, indicating preserved homeostatic feeding. However, male SNI mice failed to escalate high-fat diet intake, showed reduced operant responding, earned fewer rewards, and had lower sucrose preference, consistent with anhedonia. Females displayed motivational deficits but not a clear binge-eating phenotype. To explore neural correlates, we assessed nucleus accumbens activation via c-Fos after high-fat diet exposure. Although diet increased activation in both control and SNI mice, no group differences were detected. These results suggest that pain-induced changes in hedonic feeding may involve non-canonical mechanisms beyond immediate early gene activation. Overall, persistent neuropathic pain selectively impairs hedonic and motivational aspects of eating, pointing to broader affective dysfunctions linked to chronic pain.

D-096

Functional role of a circular RNA in synaptic physiology

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Circular RNAs (circRNAs) are a novel category of noncoding transcripts that, despite their recent discovery, remain largely unexplored. After a circRNA screening, we selected a transcript derived from the Dtnb (Dystrobrevin Beta) gene for functional analysis. This circRNA is of particular interest as it is highly expressed in mouse brain tissue, where its levels are greater than those of the linear isoform generated from the same gene. A more detailed analysis revealed its distribution across distinct brain regions.

To explore the role of circDtnb in vivo, we generated a novel loss-of-function transgenic mouse line, subsequently characterized electrophysiologically. Patch-clamp recordings in CA1 pyramidal neurons from hippocampal slices showed preserved intrinsic properties but altered synaptic features. Stimulation of Schaefer collateral inputs revealed reduced facilitation in neurons of the transgenic animals, observed by stimulating with 2 pulses and quantifying the paired-pulse ratio, as well as when stimulating with a train-of-pulses. This finding was accompanied by an increased frequency of mEPSCs, which jointly suggests enhanced vesicular release probability. As this phenotype could be explained by fewer synaptic contacts, we are performing analyses of neuronal morphology and dendritic spine density.

The electrophysiological characterization of the circDtnb KO mouse aims to provide valuable insights into the biological role of this circRNA.

D-097

Glial GABA receptors control glia-neuron crosstalk in C. elegans

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Gamma-amino butyric acid (GABA) is the most abundant inhibitory neurotransmitter in the brain. Normal GABA function requires specialized proteins such as biosynthetic enzymes, transporters and receptors. Defects in these proteins can lead to a specific imbalance of GABA neurotransmission and lead to diseases. Recent studies have shown that both GABAergic neurons and glia cells synthesize and release GABA to maintain neural excitatory-inhibitory balance, neuroprotection, among other functions. Both neurons and glia cells express functional metabotropic and ionotropic GABA receptors, however, the role of these GABA receptors in the glia cells is still unknown. Probably the activation of these receptors in glia cells are important for neuron-glia interactions. Here, we use the model organism C. elegans to uncover the function of GABA receptors expressed in the Amsh glia cell and how these regulate the neuron-glia interactions. In particular, we focus on the study of GABAA receptors, UNC-49, LGC-36 and LGC-38, which are inhibitory chlorine-selective channels and how the activation of these receptors regulates the activity of Amsh glia and consequently the regulation of ASH neuron. We found that both GABA receptors in the Amsh glia affect the activity of these glia cells and the response to the octanol in the ASH neuron. In summary, our results show that UNC-49, LGC-36, and LGC-38 express in the Amsh glia could be an important role in the regulation of neuron-glia interaction.

D-099

Targeting Astrocytes to Help Meet Brain Energy Demands in GLUT1 Deficiency Syndrome

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Glucose transport across the blood-brain barrier and into astrocytes relies on the glucose transporter type 1 (GLUT1). Heterozygous mutations in SLC2A1 cause GLUT1 Deficiency Syndrome (GLUT1-DS), a rare disorder that affects brain energy metabolism and development. Despite the genetic cause being identified over three decades ago, no therapies beyond the ketogenic diet exist. A US-based biotechnology company has been developing a small molecule that boosts astrocytic mitochondrial ATP production; its name is withheld due to an ongoing patent application. We aim to evaluate this compound as a candidate therapy for GLUT1-DS. To mimic GLUT1-DS, primary human astrocytes were incubated in glucose-free medium and treated with the compound, β-hydroxybutyrate (βHB), or their combination. These treatments significantly increased intracellular ATP individually and had a synergistic effect when combined. Mitochondrial membrane potential was also enhanced with either treatment. Additionally, intracellular calcium imaging revealed that the compound further increased Ca^{2+} fluctuations in reactive astrocytes versus controls, suggesting enhanced responsiveness under pathological conditions. These results support the potential of this compound as a pharmacological therapy to enhance astrocyte metabolism in GLUT1-DS. Next, we will test it in hiPSC-derived astrocytes modeling SLC2A1 haploinsufficiency and in transgenic mice to generate translational preclinical evidence.

D-100

Ibogaine has an antidepressant-like effect in female rats with differential behavioural manifestations according to the estrous cycle phase

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Currently, interest in the therapeutic applications of psychedelics has expanded, with ibogaine, an atypical psychedelic derived from the root of Tabernanthe iboga, emerging as a promising candidate. Although acute ibogaine administration has been reported to induce antidepressant-like effects in male rodents, its potential in female rats remains unexplored. This knowledge gap is particularly relevant because ovarian hormones influence numerous neurotransmission systems implicated in the action of psychedelic drugs. The present study evaluated the hypothesis that ibogaine exerts an antidepressant-like effect in female rats, and that its intensity varies according to the estrous cycle phase. Adult female Wistar rats received a single intraperitoneal dose of ibogaine (40 mg/kg) or vehicle during either proestrus or metestrus, characterized by high and low plasma estrogen levels, respectively. Four hours after administration, animals were assessed in the forced swimming test. Ibogaine induced a decrease in immobility time across both phases, while an increase in swimming behavior was observed exclusively in metestrus females. These results indicate that ibogaine produces an antidepressant-like effect in female rats, with behavioral outcomes that vary depending on the estrous cycle phase, highlighting the modulatory role of the endocrine environment in shaping the response to this psychedelic compound.

D-101

Exploring the effect of Cannabis sativa on L-DOPA-induced dyskinesias: preliminary result

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L-DOPA is the gold-standard therapy for Parkinson's disease, providing a transient restoration of dopamine (DA) levels. However, chronic administration leads to the development of abnormal involuntary movements, known as L-DOPA-induced dyskinesias (LID), thereby limiting therapeutic efficacy and compromising patients' quality of life. LID are associated with the interaction between synaptic plasticity induced by dopaminergic denervation and the abnormal pulsatile stimulation of DA receptors. Structural changes in the density of dendritic spines of striatal medium spiny neurons have also been reported, suggesting an anatomical substrate for this phenomenon. The endocannabinoid system has emerged as a promising therapeutic alternative. In particular, cannabidiol, the main non-psychotropic component of Cannabis sativa (CS), exhibits neuroprotective, anti-inflammatory, and dopaminergic neurotransmission-modulating properties. To explore its therapeutic potential on LID, we conducted a pilot study in hemiparkinsonian mice, which received daily CS extract (60 or 120 mg/kg) together with dyskinesiogenic doses of L-DOPA for 14 days. Preliminary results indicate that only the lower dose tends to reduce LID severity. Ongoing studies will evaluate whether CS administration attenuates these dyskinesias and whether its effects are associated with synaptic microarchitectural plasticity in the striatum.

D-102

Integrating PPARγ Modulation with Cellular and Acellular Nanotherapies in Peripheral Neuropathy

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Peripheral neuropathies are prevalent disorders with limited therapies, where unresolved inflammation drives neuropathic pain. This project explores strategies combining pharmacological modulation of PPARγ with cellular and acellular nanoplatforms. In a sciatic nerve crush model, bone marrow multipotent cells (BMMC) migrated to the injury site within 24 h, a process inhibited by indomethacin. Indomethacin also reduced PGE_2 and increased PGJ_2, an endogenous PPARγ ligand, highlighting its regulatory role. Molecular analyses revealed that injury induced the expression of COX-1 and PPARγ, which was sustained by BMMC transplantation, whereas rosiglitazone did not significantly alter PPARγ expression. PLGA nanocapsules carrying rosiglitazone, with or without magnetic nanoparticles, were synthesized and are being optimized for stability and compatibility. Extracellular vesicles (EVs) from adipose-derived multipotent cells, functionalized with magnetic nanoparticles, were preliminarily validated for neuronal uptake without cytotoxicity. Behavioral assays showed partial pain relief with rosiglitazone and additive effects with BMMC. Altogether, results suggest PPARγ as a therapeutic target and the potential of bio-nanotechnological strategies to enhance regeneration and manage neuropathic pain in peripheral nerve injury.

D-103

Two Novel Tetracycline-Based Strategies for the Sensitive Detection of α-Synuclein Aggregates

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During the progression of Parkinson's disease (PD), aggregated α-synuclein (aSyn) species appear early, making them crucial biomarkers for early diagnosis. However, their detection remains challenging. Building on our previous research showing that certain tetracycline derivatives selectively bind to aggregated aSyn but not to its monomeric form, we evaluated two modified doxycycline derivatives using an immunoassay and developing a biosensor. The immunoassay successfully distinguished between aggregated and monomeric aSyn in PBS (phosphate-buffered saline), and we further tested it in cerebrospinal fluid (CSF) to assess potential interferents. We identified $NaHCO_3$ as the main interferent and evaluated strategies to mitigate its effect, such as dialysis and pH adjustment, which restored the signal attenuated by the interferent in CSF samples spiked with aggregated aSyn. In the biosensor approach, a thiolated doxycycline derivative was used to form a monolayer on the gold electrode and characterized by Cyclic Voltammetry (CV) and Electrochemical Impedance Spectroscopy (EIS). We evaluated its binding to aggregated aSyn at increasing concentrations by measuring changes in the oxidation peak with CV. Both approaches enable sensitive detection of aggregated aSyn, providing cost-effective and scalable alternatives for PD diagnostics. These strategies hold promise for clinical translation, including point-of-care applications that could support earlier diagnosis and intervention.

D-104

Linking motor behavior to synaptic microarchitecture in a mouse model of haloperidol-induced dyskinesia

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Tardive dyskinesias (TD) are frequently associated with long-term antipsychotic treatment, especially in schizophrenic patients. These movement disorders have a high prevalence and can become irreversible, frequently persisting after medication is discontinued. The main hypotheses regarding the pathophysiological mechanisms underlying the development of TD include hypersensitivity of dopaminergic receptors, decreased GABAergic activity, and/or the presence of structural abnormalities in the striatum. We propose to study whether medium spiny neurons (D1- and D2-MSNs) undergo structural plastic changes after the development of TD. We established a vacuous chewing movement (VCM) model in wild type mice by administering haloperidol daily for 60 days (1.5 mg/kg for 30 days, followed by 2 mg/kg for another 30 days). Behavioral assessments were conducted throughout the treatment period and for an additional 30 days. Orofacial movements were recorded: protrusion of the tongue, wide-range chewing movements, subtle chewing movements, and jaw tremors. Mice developed low or high levels of VCM, that lasted even after the pharmacological treatment was discontinued. Using D1-tomato transgenic mice, treated with this protocol and injected with an adenoviral vector expressing GFP, we are analyzing the number of dendritic spines in MSNs. These results will allow us to determine whether the severity of VCM is associated with a differential remodeling of the dendritic spines of D1- and D2-MSNs.

D-105

Deciphering Serotonergic Signaling in Drosophila Hematopoiesis Through a Single-Cell Transcriptomic Approach

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The Drosophila larval lymph gland is the main hematopoietic organ. It is composed of multiple lobes, with the primary lobe being the main site of hematopoiesis. This lobe is compartmentalized into the medullary zone (MZ) where blood progenitors are maintained, and the cortical zone (CZ), where differentiated cells, plasmatocytes and crystal cells, are accumulate. While intrinsic regulatory mechanisms have been extensively characterized, the contribution of external signals to progenitor fate decisions remains less explored. Previous studies have reported that neurotransmitters such as GABA and dopamine modulate blood cell differentiation. However, the potential role of serotonergic signaling in this process has not yet been investigated.

In this study, we performed an in silico analysis of single-cell RNA-seq data from the lymph gland, to assess the expression of key genes involved in serotonergic signaling, including receptors, transporters, and biosynthetic enzymes. By characterizing their distribution across different cell populations, we aim to explore whether serotonin could act as a novel extrinsic regulator of hematopoietic differentiation. Our findings may provide new insights into the neuro-immune interface in Drosophila and contribute to understanding conserved mechanisms of hematopoietic regulation.

D-106

Divergent roles of hypothalamic proopiomelanocortin (POMC) neuronal subpopulations in energy homeostasis: dissecting cholinergic circuit and dorsomedial hypothalamus (DMH) projection.

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Obesity and diabetes are major public health challenges that arise from metabolic imbalance. The hypothalamic arcuate nucleus (Arc) is a key integrative hub in the regulation of energy and glucose homeostasis. Arcuate proopiomelanocortin (POMC) neurons reduce food intake and promote energy expenditure. However, they comprise distinct subpopulations defined by their neurotransmitter profile and projection targets. We previously found that GABAergic Arc-POMC projection to the dorsomedial hypothalamic nucleus (DMH) contributes to the regulation of feeding and body weight. Yet, the functional relevance of other Arc-POMC subpopulations remains poorly understood. A small subset of POMC neurons co-releases acetylcholine, but whether these cholinergic neurons participate in the control of energy and glucose balance is still unknown. In the present study, we investigated the role of POMC expression in cholinergic neurons and in neurons projecting to the DMH and the paraventricular nucleus (PVN). Our preliminary results suggest that whereas cholinergic POMC neurons may play a specific role in glucose regulation, POMC neurons projecting to the DMH and PVN, regardless of the neurotransmitter they co-release, may have a role in energy balance. Defining the physiological functions of distinct Arc-POMC subpopulations could uncover novel pathways linking central circuits to peripheral metabolism and pave the way for targeted therapeutic strategies against obesity and diabetes.

D-107

Physiological Disruption of Avian Respiration by Aircraft-Generated Noise

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Noise pollution is increasing at an unprecedented rate, with documented impacts on reproduction and development across taxa. While recent research has focused on anthropogenic noise's effects on avian vocal behavior, particularly in high-noise environments like airports, its physiological impacts and consequences for avian sleep remain poorly understood. This study investigates how aircraft noise from Jorge Newbery Airport affects the respiratory activity of adult male canaries (Serinus canaria domestica) during both wakefulness and nocturnal sleep. Our results demonstrate that perturbations in respiratory rhythm are closely correlated with the positive rate of change in ambient noise levels caused by passing aircraft, with the most significant behavioral changes occurring at the peak of the noise derivative. Furthermore, we show that air sac pressure and respiratory rate exhibit similar response patterns but are affected differently. Collectively, these findings indicate that the physiological impacts of anthropogenic noise pollution are potentially more harmful than previously recognized.

D-108

Kinematic Analysis of Intersegmental Coordination in Leech Crawling

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Crawling is a fundamental locomotor pattern that leeches use to move in shallow water. It consists of sequential waves of body elongation and contraction, alternating with attachment and detachment of the anterior and posterior suckers. This stereotyped behavior requires precise coordination across multiple body segments. Previous studies have shown that individual ganglia contain the neural circuitry to generate the sequential elongation–contraction movements, but how these oscillators coordinate across segments remains unknown.

To provide a behavioral framework for physiological experiments, we recorded crawling in freely moving leeches. Using ad hoc markers and DeepLabCut, we tracked the kinematics of multiple body regions. We found that elongation in the anterior regions occurred while posterior regions were still elongating, and that elongation propagated from head to tail with deceleration, whereas contraction propagated with acceleration. These findings suggest that elongation and contraction are coordinated by distinct mechanisms.
By calculating the displacement of the center of mass during crawling, we found that it shifted backward during elongation and forward during contraction. This suggests that proprioceptive signals may influence how long the leech remains supported by the anterior or posterior sucker.

D-109

Exploring the 3D organization of the lateral protocerebrum of the semiterrestrial crab Neohelice granulata.

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Semiterrestrial crabs possess a highly developed visual system and exhibit conspicuous visually-guided behaviors. Within their eyestalks lie the optic lobes, which house the visual neuropils—lamina, medulla and lobula complex—as well as the lateral protocerebrum. The visual neuropils are highly organized, comprising thousands of retinotopically arranged columns. In contrast, the lateral protocerebrum contains a collection of neuropils and globular structures that remain largely undescribed. Previous studies in Neohelice using Golgi impregnation identified 29 types of lobula columnar neurons (LCs), classified by their arborizations within the lobula. However, due to the stochastic nature of the technique, the actual number of LC types is likely higher. In arthropods, LCs typically project their axons to cell-type-specific target regions within the lateral protocerebrum, known as optic glomeruli. In this study, we combine multiple anatomical techniques—including massive dextran-conjugated dye staining, synapsin immunohistochemistry, intracellular staining, Golgi impregnation, and Bodian staining—to describe the architecture of the lateral protocerebrum. Through Neurolucida-based reconstructions, we present a 3D model that encompasses 83 optic glomeruli, the terminals of four types of lobula giant neurons (LGs), medulla tracts, and previously unrecognized neuropils. These structures are contextualized alongside known components such as the mushroom and reniform bodies.

D-110

Mechanical sensitivity during the estrous cycle in leptin-deficient mice: role of HCN channels.

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Hyperpolarization-activated currents (IH), mediated by HCN channels, play a critical role in regulating thalamic oscillations and neuronal excitability. We examined their functional expression in the ventrobasal nucleus (VB) of female mice and the modulatory effects of sex hormones and metabolic state. Whole-cell recordings were performed in VB neurons from wild-type (WT) and leptin-deficient (ob/ob) mice. Estrous cycle phases, ovariectomy, and mechanical sensitivity assessed with the Von Frey test were evaluated. IH shows sexual dimorphism: in WT females, IH increases with age (25-30 PND: n=8; 35-60 PND: n=12; $P<0.05$, T-test), while ovariectomy (OVX) prevents this enhancement (sham: n=7; OVX: n=11; $P<0.05$, T-test), highlighting a key role for sex hormones. In ob/ob females, whose estrous cycle is disrupted, IH remains stable and resembles the male pattern (n=11,12). Behavioral analyses revealed mechanical sensitivity is similar between WT (n=7) and ob/ob (n=6) females, whereas ob/ob males (n=8) show higher sensitivity than WT males (n=15) (MLM). WT females show no estrous cycle–dependent differences in mechanical sensitivity, while ob/ob females display increased sensitivity during diestrus vs estrus (n=7,5; $P<0.05$, T-test). These findings indicate HCN channel function in VB neurons is regulated by sex hormones and leptin, and that metabolic and hormonal imbalances differentially shape thalamic excitability and somatosensory processing in a sex- and cycle-dependent manner.

D-111

Exploring D5 receptors as a therapeutic target in striatal cholinergic interneurons in Parkinson's disease

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Striatal cholinergic interneurons (SCIN) are the main source of striatal acetylcholine. In Parkinson´s disease (PD), dopaminergic neurons that innervate the striatum degenerate, increasing the cholinergic function that contributes to PD symptoms. The preferred PD therapy is L-dopa administration, but prolonged treatment may result in dyskinesia. Recent studies showing that selective modulation of SCIN activity reduces motor deficits have renewed the interest in understanding the mechanisms that alter SCIN function. SCIN become hyperexcitable in Parkinsonian and dyskinetic mice due to an increased ligand-independent activity of D5 receptors (D5R). Reducing D5R ligand-independent activity with D5 inverse agonists restores SCIN's physiology. Our aim is to clarify the role of D5R in SCINs activity and evidence the potential therapeutic value of reducing the expression of the D5R in these interneurons. For this, we remove the D5R in SCIN: ChAT-Cre+/-; D5+/+ mice, to induce D5R ablation from all cholinergic neurons during development, and D5+/+ mice injected with a ChAT-Cre viral vector in the striatum after initiation of the L-dopa, which would allow a selective ablation from SCIN in adulthood. Our preliminary results show that L-dopa induces less dyskinesia in KO mice than the control group. Further experiments are needed to confirm these findings and assess the effect of selectively eliminating D5R in SCIN after L-dopa treatment, which could make D5R a therapeutic target for PD.

D-112

Benchmarking Brain Age Prediction Models Across External MRI Datasets: Robustness, Biases, and Interpretability

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Brain age prediction from T1-weighted MRI has emerged as a promising biomarker for detecting deviations from normative aging trajectories. A key challenge, however, is ensuring robustness across external datasets, a requirement for future clinical translation.
We evaluated four pre-trained brain age prediction models (BrainAgeNeXt, Pyment, DeepBrainNet and ENIGMA) across four external datasets: ADNI, a local cohort including long-COVID patients and controls, and two healthy cohorts (JUK, RRIB) from the OpenNeuro platform, totaling 1,634 subjects. Models based on 3D convolutional neural networks showed lower bias (MAE = 3.7 and 4.6 years vs. 6.2 and 12.3 years) and variance (ASTD = 2.9 and 3.7 years vs. 4.7 and 8.9 years), with performance improving with larger and more diverse training sets.

A regression-to-the-mean effect in brain age prediction was observed across all models, with strong age underestimation in ADNI controls. Within datasets, individuals with neurodegenerative conditions consistently exhibited older brain age compared to controls. Finally, analysis on explainability maps highlighted voxels from the lateral ventricles as the most influential in model predictions, consistent with previous reports.

D-113

Is it necessary to update meta-analyses on the neurogenic theory of depression?

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Systematic reviews (SR) and meta-analyses (MAN) about the neurogenic theory of depression concluded there is evidence of pro-neurogenic effects of monoaminergic drugs, mainly fluoxetine. However, conclusions may become obsolete as new studies are published. Living Systematic Reviews (LSRs) are tools for incorporating new evidence as it emerges quickly in the literature. Trial Sequential Analyses (TSAs) are an approach that helps determine when conclusive evidence is achieved in an LSR and whether future updates are needed. Samples (k=20) from a database (k=677, https://osf.io/q9bk3) were used to construct the cumulative z-curve adjusted by tau2, DerSimonian-Laird, REM, spending α esOF:Lan&DeMets, O'Brien-Fleming boundaries, α=5%, β=10%(1-β=90%) and empirical I2 in the "RTSA" package. The evidence was considered conclusive when the cumulative z-curve crossed the boundaries for statistical significance (Z =1.96, p = 0.05), monitoring boundaries, and required information. The available information indicated that k=11, k=19, and k=46 studies would be needed to establish the current level of conclusive evidence in the MAN on the effects of antidepressants on hippocampal neurogenesis; neurogenesis and behavior; or behavioral and neurogenic effects of fluoxetine, respectively. Available information for the third MAN is insufficient, and further studies are necessary. For other MANs, it is sufficient to suggest the preliminary termination of the updates, avoiding additional studies.

D-114

Emergent Anticipatory Coding and Self-Organized Path Integration

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Navigating without landmarks relies on path integration (PI), the ability to estimate position by integrating self-motion cues. Grid cells in the entorhinal cortex (EC) are thought to perform this computation by combining speed and direction signals. Classical models fall into two camps: continuous attractor neural networks (CANNs), which assume fixed connectivity and externally supplied velocity inputs, and self-organizing feedforward models, which explain grid formation but not PI. In this work, we introduce a self-organizing CANN model for unidirectional PI, which, unlike other self-organizing approaches, makes PI an intrinsic property of the system's stable solution rather than an externally imposed feature.
From structured spatial input, the network learns feedforward weights via Hebbian plasticity and competition, forming a representation of a one-dimensional manifold. Stacking multiple feedforward layers yields predictive coding of future positions—predicted analytically, confirmed in simulations, and consistent with reports of predictive grid cells in superficial medial EC. The same principles allow recurrent connectivity to self-organize, enabling continuous attractor dynamics. With an independent additive speed-modulated input current, the model adapts its dynamics to running speed, achieving PI. Analytical tractability offers mechanistic insight and a unified framework that bridges learning, prediction, and navigation, paving the way for two-dimensional extensions.

D-115

Can we support the user-learning process in active brain-computer interfaces?: a multi-session prospective preliminary analysis

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Rehabilitation therapy based on motor imagery brain-computer interfaces (MI-BCIs) has been shown to produce lasting improvements in upper limb motor functions after stroke. However, a significant percentage of novice users (10% to 30%) fail to adequately control them, partly because traditional stimulation protocols are neither enganing nor transparent, hindering learning. Furthermore, these protocols do not provide meaningful real-time feedback that allows the user to efficiently adjust the brain activity modulation strategy employed.

Here we present a videogame-based stimulation protocol for MI-BCIs with applications for upper limb rehabilitation that integrates supportive backward adaptation (SBA) to provide informative online feedback reflecting how well the instructed MI task was performed. Using this protocol, to date a database comprising five sessions from four healthy users (three males and one female, 21-33 years old, right-handed) has been acquired. This database was recorded using affordable, portable, non-clinical-grade acquisition systems.

Preliminary results indicate that higher task accuracy was associated with lower median and deviation values of the SBA support index, suggesting that this metric can serve as an indicator of user progress across sessions. Participants reported being highly engaged in the task, considered the feedback clear and useful, and the game motivating.

D-116

Deep neural networks for decoding behavioral information from in vivo neuronal spiking activity

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Neuronal decoding is the process of using mathematical and computational techniques to interpret and extract meaningful information from measured brain activity, mapping brain responses back to the stimuli, behavioral and/or cognitive events. We provide an example of how deep neuronal networks can be trained to decode behavioral information latent in the spiking activity of neural populations.
For this, we analyze neuronal activity in the primary olfactory cortex of a mouse trained in a behavioral task where associations of spatial locations and particular odors are linked to rewards. Using artificial dense feed-forward and recurrent neuronal networks, we could successfully decode the spatial location of the mouse and its running speed, trial by trial.
Moreover, despite not being traditionally involved in spatial cognition, olfactory cortex decoding accuracy reached similar performance to decoding in the hippocampus, a well-known hub for spatial information.
These results indicate that complex and multiplexed information is already present at early sensory processing stages, supporting a distributed and decentralized view of brain organization.

D-117

Transition-specific representations emerge from next-state prediction in recurrent neural networks

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While interacting with their environment, animals form memories of state-action-outcome sequences that enable them to predict future states and make informed decisions. How this capability emerges in neural populations remains an open question. We investigated this issue by training vanilla recurrent neural networks (RNNs) to navigate randomly generated graphs and assessing whether a simple next-state prediction objective is sufficient to induce generalizable transition representations. The RNNs explored graphs by randomly choosing actions that triggered movement between adjacent nodes, while learning to predict the next node from current node-action pairs. Critically, we created training and test sets with non-overlapping (node, action) transitions, forcing the network to generalize rather than memorize. Despite never seeing test transitions during training, the RNN achieved near-optimal performance on small graphs. Analysis of hidden states revealed that the network spontaneously formed transition-specific representations: the same (node, action) pair encountered across different exploration sequences maped to similar hidden states, producing distinct clusters in representational space. These results suggest that a next-state prediction objective may support generalizable representations subserving world models in biological neural networks.

D-118

From Correlations to Connectivity: Path Decomposition and Bayesian Inference of Partial Correlations in Neural Populations

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Understanding the structure of neural circuits requires distinguishing between correlations that arise from direct interactions between neurons and those mediated by the broader network. Pairwise correlations are straightforward to measure experimentally, yet they conflate direct and indirect dependencies. Partial correlations, by contrast, quantify pairwise dependencies conditioned on the activity of all other neurons and thus provide a more principled route to inferring network connectivity. We present a theoretical framework that bridges these two measures by showing that the total correlation between any two neurons can be decomposed into a sum over all network paths connecting them. Each path contributes additively, with its weight defined as the product of the corresponding partial correlations along the route. This decomposition establishes a direct link between experimentally accessible correlations and the underlying conditional dependencies. Building on this insight, we introduce a Bayesian method for estimating partial correlations in high-dimensional neural data. We validate our approach in neural simulations, demonstrating its ability to recover network structure and disentangle direct from indirect interactions. Our results provide a principled strategy for moving from correlation-based observations to connectivity-based interpretations in population neuroscience.

D-119

Can AI tell what my mouse is doing?

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Our research group studies the role of molecular mechanisms in learning and memory. Manual scoring of rodent exploratory behavior is time-consuming and prone to bias. To address this, we developed RAINSTORM (Real and Artificial Intelligence for Neuroscience — Simple Tracker for Object Recognition Memory), an open-source, AI-driven behavioral labeling tool designed to learn from experimenters labeling criteria. RAINSTORM processes pose estimation data (e.g., from DeepLabCut) to automate precise quantification of object exploration, and easily extends to many other tasks.
We performed a series of experiments on object recognition in mice, focusing on the localized administration of ERK pathway inhibitors and activators. Our protocol goes beyond simply measuring exploration time by revealing drug-induced changes in mobility and exploration dynamics. This enables a comprehensive assessment of effects on mouse training, learning, and memory that were previously overlooked.
By standardizing and accelerating analysis, RAINSTORM enhances reproducibility and comparability of results. Fully available on GitHub, it fosters collaboration to validate and expand its utility. We believe open, collaborative AI tools, especially in the current scientific climate, are key to revolutionizing behavioral neuroscience.

D-120

A Flexible Tool for Analysing Biological Oscillations

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Biological rhythms govern a wide range of behaviours and physiological processes. In behavioural neuroscience, patterns such as locomotor activity or sleep-wake cycles are analysed to understand endogenous timekeeping mechanisms. In circadian rhythms, the timing of behavioural events relative to internal or external cycles is key to understanding how clocks respond to environmental cues. Under constant conditions, these rhythms most clearly reveal the intrinsic properties of the clock. Traditional analyses often focus on immediate responses to external cues, which can obscure long-term phase stability and complicate comparisons across groups, such as different genotypes or experimental conditions. To address this, we developed an analytical pipeline for assessing the behavioural phase and estimating phase lags under prolonged constant conditions. The method extracts oscillatory components directly from behavioural data without referencing external cues or imposing rigid period models. Implemented in R, with an open and extensible framework, the pipeline includes tools for data formatting, signal filtering, visualisation, statistical comparison, and guidelines for evaluating preprocessing choices. Although illustrated using Drosophila melanogaster locomotor activity, this approach applies to any oscillatory biological variable, enabling robust phase comparisons and providing a versatile and reproducible tool for studying oscillations.