11TH BORDEAUX NEUROCAMPUS INTERNATIONAL CONFERENCE

BrainBody

1-3 October 2025
Bordeaux, France

Exploring **bidirectional crosstalk** between the **central nervous system** and the **body**

Credit: Lorena Delgado Zabalza

BORDEAUX neurocampus / université de BORDEAUX

Abstract book

About the conference

Ensuring adequate body homeostasis is a fundamental requirement for survival. The vertebrate brain has evolved intricate neural circuits and regulatory systems to precisely and constantly communicate with the body. Interoception, the sensing of internal body signals, is guaranteed through mechanisms involving crosstalk between the brain and the periphery, including sensory organs, heart, lungs, adipose tissue, and gut.
The crosstalk between the body and the brain occurs through various means, encompassing neuronal communication via the autonomous and sensory systems, but also via the humoral pathway (e.g. hormones or metabolites), and components of the immune system. Disruptions in this bidirectional communication can impact neuronal mechanisms involved in fundamental physiological processes and contribute to a spectrum of disorders, spanning psychiatric, neurological, cardiovascular, respiratory, metabolic, and immunological conditions. The interaction between the body and the brain has garnered significant general interest, as indicated by the abundance of recent articles and reviews on the topic.
The conference «Brain-body» aims to present the latest insights into these diverse mechanisms governing brain-body crosstalk, identifying commonalities and differences among the different systems. This not only advances our understanding of body-brain communication but also seeks to illuminate the concept of interoception, ultimately answering the question, "How do I feel?"

Scientific Organizers

Mario Carta - CNRS, IINS
François Georges - CNRS, Institut des Maladies Neurodégénératives
Christelle Glangetas - Institut des Maladies Neurodégénératives
Enrica Montalban – NutriNeuro
Agnès Nadjar - Université de Bordeaux - Neurocentre Magendie

About Bordeaux Neurocampus international conferences

The Bordeaux Neurocampus Conferences are a series of 3-day neuroscience meetings that take place every year in autumn at the University of Bordeaux, France, since 2014.
The purpose of the conferences is to present and discuss recent findings in a topic field in Neuroscience, bringing together leading international experts and young researchers.

Speakers

Michael BRECHT

Ann CLEMENS

Ana DOMINGOS

Ivan E. DE ARAUJO

Gérard EBERL

Amaury FRANCOIS

Giuseppe GANGAROSSA

Christelle GLANGETAS

Nadine GOGOLLA

Ali JAWAID

Zachary A. KNIGHT

Yoav LIVNEH

Arianna MAFFEI

Claire MARTIN

Enrica MONTALBAN

Francesco PAPALEO

Thomas PRADEU

Asya ROLLS

Lisa ROUX

Dana SMALL

Catherine TALLON-BAUDRY

Laure ZAGO

> More details

Program preview

Wednesday 1st October

8:30 - Registration and welcome coffee
9:00 - Session 1: *Autonomous harmony: deciphering bidirectional body-brain communication.*
12:00 - Poster session
12:45 - Lunch
14:00 - Session 2: *Savoring sensations: linking oro-nasal perception with visceral experiences.*
16:50 - Plenary lecture
17:50 - Group picture
18:00 - Wine & cheese + Poster session

Thursday 2nd October

8:30 - Welcome Coffee
9:00 - Session 3: *Mindful metabolism: exploring brain-body hormonal controls.*
11:15 - Poster Session
12:45 - Lunch
14:00 - Session 4: *Social Interoception: mechanisms shaping interindividual interactions.*
17:15 - Plenary lecture
19:30 - Gala dinner at "Le Tchanqué" (Sold out)

Friday 3rd October

9:00 - Welcome Coffee
9:30 - Session 5: Neuroimmune nexus: role of neuroimmune system in brain-body dynamics.
12:00 - Plenary lecture
13:00 - Closing remarks
13:00 - 14:00 - Lunch

Sponsors

Organization

Institutional sponsors

This project has received financial support from the French government
within the framework of the France 2030 programme IdEx université de Bordeaux.

Private supports

KARTHALA
two-photon microscopy system

KARTHALA SYSTEM is an innovative French company specializing in two-photon microscopy.
Our mission is to revolutionize the field of neurobiology through a state-of-the-art tool that enhances understanding of information processing and computation in brain regions.
Our flagship product, the AODscope, uses advanced optical deflection technology to enable rapid and precise acquisition or stimulation of neural activity, dramatically improving resolution, and the ability to monitor neural processes in real-time.
https://karthalasystem.com

THORLABS

THORLABS, a vertically integrated photonics products manufacturer, was founded in 1989 to serve the laser and electro-optics research market. As that market has spawned a multitude of technical innovations, Thorlabs has extended its core competencies in an effort to play an ever-increasing role to serve the Photonics Industry at the research end, as well as the industrial, life science, medical, and defense segments. The organization's highly integrated and diverse manufacturing assets include semiconductor fabrication of Fabry-Perot, DFB, and VCSEL lasers; fiber towers for drawing both silica and fluoride glass optical fibers; MBE/MOCVD epitaxial wafer growth reactors; extensive glass and metal fabrication facilities; advanced thin film deposition capabilities; and optomechanical and optoelectronic shops.
https://www.thorlabs.com/

WPI
WORLD
PRECISION
INSTRUMENTS
Instrumenting scientific ideas

WORLD PRECISION INSTRUMENTS (WPI) is a leading instrument and equipment manufacturer specialized in supporting neuroscience, physiology, electrophysiology and cell imaging research, as well as pioneer and leader for Trans Epithelial Electrical Resistance TEER. We offer a complete line of neuroscience equipment including microinjectors, stereotaxic systems, stimulators, amplifiers, data acquisition, micromanipulators, loupes and surgical instruments.
WPI's products for neurosciences applications have 1000s of citations and include the UMP3T-1 for stereotaxic injections, and our new Live Cell Imaging system AutoLCI.
For essential fluidic control, we offer solutions across pumping platforms including syringe, peristaltic, microfluidic and pneumatic microinjection pumps for picoliter to microliter flow.
Come and visit our booth to discuss your applications and discover our latest innovations in Neuroscience.
www.wpi-europe.com

For the general public

In french

Tuesday 30th September at 6pm

Location :
Cap Sciences – Hangar 20 – quai de Bacalan

Entrée libre et gratuite dans la limite des places disponibles.

Durée : 45 minutes suivies de question.

Par Catherine Tallon-Baudry
Directrice de recherche CNRS
Laboratoire de neurosciences cognitives et computationnelle
Ecole normale supérieure, Paris
https://sites.google.com/view/tallon-baudry-lab/people

Résumé

Tout le monde connait les 5 sens – la vue, l'ouie, le toucher, l'odorat et le gout – qui nous permette d'apprécier la richesse du monde extérieur. L'intéroception recouvre les « sens » tournés vers l'intérieur du corps, notamment les battements cardiaques ou le rythme lent de l'estomac. Alors que l'intéroception est essentielle pour un organisme en bonne santé, elle a été peu étudiée – et les résultats de ces 10 dernières années révèlent de nombreuses surprises: si nous avons des croyances bien ancrées sur nos capacités à écouter notre corps, les tests en laboratoire racontent une autre histoire. Les signaux intéroceptifs pourraient aussi jouer un autre rôle dans le cerveau, celui de créer ce que les philosophes ont appelé le « soi minimal » ou plus simplement, la personne consciente du monde qui l'entoure.

Scientific program

Wednesday 1st October

From 8:30 – Registration & welcoming coffee

9:00 – Welcoming speech

9:00 – Session 1
Autonomous harmony: deciphering bidirectional body-brain communication.

Chair:
François Georges

Yoav Livneh / 9:00 – 9:30
Brain-body interactions: sensations and predictions in the insular cortex

Nadine Gogolla / 9:30 – 10:00
Interoception in the insula shapes emotion processing

Catherine Tallon-Baudry / 10:00 – 10:30
Interoception & Cognition

Coffee break

Enrica Montalban / 11:00 – 11:30
Striatal Astrocytes Mediate Behavioral and Metabolic Adaptation in response to inflammation

Short talks:

- **Meryl Malezieux** / 11:30 – 11:45
  Cardio-insular processing shapes emotion state coding
- **Shervin Safavi** / 11:45 – 12:00
  Emergence of brain-like features in artificial neural networks trained based on homeostasis

12:00 – Poster session

12:45 – Lunch

14:00 – Session 2
Savoring sensations: linking oro-nasal perception with visceral experiences.

Chair:
Mario Carta

Arianna Maffei / 14:00 – 14:30
Neural Mechanisms for Taste Learning

Ivan E. de Araujo / 14:30 – 15:00
Psychological States and the Regulation of Mucosal Immunity

Zachary A. Knight / 15:00 – 15:30
Control of ingestion by the caudal brainstem

Coffee break

Claire Martin / 16:00 – 16:30
The impact of metabolic status on olfactory processing

Short talk:

- **Friedrich W. Johenning** / 16:30 – 16:45
  Suppression of olfactory cortex activity gates sensory satiation during hedonic feeding

16:50 – Plenary lecture

Chair:
Olga Vila

Thomas Pradeu / 16:45 – 17:45
The immunological and psychological self

17:50 – Group picture

18:00 – Wine & cheese + Poster session

Thursday 2nd October

8:30 – Welcome coffee

9:00 – Session 3
Mindful metabolism: exploring brain-body hormonal controls.

Chair :
Enrica Montalban

Dana Small / 9:00 – 9:30
Peripheral signals regulating food choice in humans

Giuseppe Gangarossa / 9:30 – 10:00
The gut-brain vagal axis governs mesolimbic dopamine dynamics and reward events

Ali Jawaid / 10:00 – 10:30
Immunometabolic basis of neuropsychiatric disorders: Across the lifespan and across generations

Coffee break

Short talk:

- **Victor Jouque** / 11:30 – 11:45
  Hypothalamic POMC satiety neurons drive the intake of hypercaloric food

12:15 – Poster session

12:45 – Lunch

14:00 – Session 4
Social Interoception: mechanisms shaping interindividual interactions.

Chair:
Christelle Glangetas

Francesco Papaleo / 14:00 – 14:30
Inter-brain circuits of emotion recognition

Ann Clemens / 14:30 – 15:00
Neurobiology of Kinship Behaviour

Amaury François / 15:00 – 15:30
Central integration of social touch

Coffee break

Short talk:

- **Laetitia Davidovic** / 16:00 – 16:15
  Microbiota and autism spectrum disorders: modulation of social behavior by the microbial metabolite p-cresol

Christelle Gangletas / 16:15 – 16:45
Insula and social behavior

Laure Zago / 16:45 – 17:15
Effects of body-centred interventions on stress-related physiological markers and psychological well-being in women.

17:15 - Plenary Lecture

Chair:
Emmat Perrot

Michael Brecht / 17:15 – 18:15
Brain representations of whiskers and genitals

19:30 - Gala dinner at "Le Tchanqué"

Until 1 am

Friday 3rd October

9:30 – Session 5
Neuroimmune nexus: role of neuroimmune system in brain-body dynamics.

Chair:
Agnès Nadjar

Ana Domingos / 9:30 – 10:00
Sympathetic neurons in obesity

Lisa Roux / 10:00 – 10:30
Breathing and hippocampal network activity during wake and sleep

Gérard Eberl / 10:30 – 11:00
Impact of acute and chronic inflammation on brain and metabolism

Coffee break

Short talks:

- **Justus Ninnemann** / 11:30 – 11:45
  Chronic systemic inflammation imposes critical thresholds in energy metabolism
- **Megan Sammons** / 11:45 – 12:00
  What's pain got to do with it: The Role of Pain in Encoding Immune Responses

12:00 – Plenary Lecture

Chair:
Léa Bonamy

Asya Rolls / 12:00 – 13:00
Immunoception: how the brain represents immunological states

12:50 – Closing remarks

13:00 – Lunch

Invited speakers

Click on the name to get to the details!

Michael Brecht, Humboldt-Universität zu Berlin, Germany
Ann Clemens, University of Edinburgh, United Kingdom
Ana Domingos, University of Oxford, United Kingdom
Ivan E. de Araujo, Max-Planck-Institut fūr biologische Kybernetik, Eberhard Karls Universität Tübingen, Germany
Gérard Eberl, Institut Pasteur, Paris, France
Amaury François, CNRS, Institut de Génomique Fonctionelle, Montpellier, France
Giuseppe Gangarossa, Université Paris Cité, Unité Biologie Fonctionnelle et Adaptative, Institut Universitaire de France, France
Christelle Gangletas, Institut des Maladies Neurodégénératives, Université de Bordeaux, France
Nadine Gogolla, Max Planck Institute of Psychiatry, Emotion Research Department, Germany
Ali Jawaid, Polish Center for Technology Development, Wroclaw, Poland
Zachary A. Knight, University of California San Francisco, USA
Yoav Livneh, Department of Brain Sciences, Weizmann Institute of Science, Israel
Arianna Maffei, Stony Brook University, NY, USA
Claire Martin, CNRS, Unité Biologie Fonctionnelle & Adaptative, Université Paris Cité, France
Enrica Montalban, Nutrineuro, Bordeaux, France
Francesco Papaleo, Genetics of Cognition Laboratory, Istituto Italiano di Tecnologia, Italy
Thomas Pradeu, CNRS, ImmunoConcept, Université de Bordeaux, France
Asya Rolls, Faculty of life science, Tel Aviv University, Israel
Lisa Roux, CNRS, Interdisciplinary Institute for Neuroscience, Université de Bordeaux, France
Dana Small, McGill University, Canada
Catherine Tallon-Baudry, CNRS, LNC2, Ecole Normale Supérieure, France
Laure Zago, CNRS, Institut des Maladies Neurodégénératives, Université de Bordeaux, France

Michael Brecht

Bernstein Center for Computational Neuroscience Berlin, Humboldt-Universität zu Berlin, Germany

Webpage

Brain representations of whiskers and genitals

Abstract

My lecture will center on the body-brain-relationship. In the first part of the lecture I will talk about early vibrissal system. I will discuss how novel X-ray based techniques allow elucidating the structure of vibrissal mechanotransduction and how large-scale neural reconstructions help us understand the mapping of afferent information onto the brain. Wire-by-wire connectivity analysis will change our understanding of body and brain. The second part of the lecture deals with sexual touch. I will show that genital representation have an unusual degree of plasticity and that genital touch affects puberty, brain and body. Finally, I discuss unexpected male-female difference in human genital representations.

Biosketch

Michael Brecht is Professor for Systems Neuroscience at Humboldt-University Berlin and Coordinator of the Bernstein Center for Computational Neuroscience Berlin. His major interest is to understand, how brain structure and activity relate to behavior. The recent research of the Brecht laboratory focused on social touch, kinship, play and elephants.

Ann Clemens

University of Edinburgh, Scotland

Webpage

Neurobiology of Kinship Behaviour

Abstract

The nervous system of vertebrate species evolved to navigate diverse social and environmental terrain. Our research group aims to understand the neurobiology of natural rodent behaviour from an ethological perspective. We are interested in how animals sense and respond to their natural environment through olfaction, somatosensation and have a particular interest in the development of social kinship behaviour. In recent years, our work in Edinburgh and the Marine Biological Laboratory has uncovered whisker sensory processing for wind stimuli (Mugnaini et al 2023) and sensory, behavioural and fur-based drying responses to environmental wetness (Attah et al 2024). We elucidated tactile mechanisms and skin sensory afferents for the rat pup transport response (Ni et al 2024) and tactile, vocal and kinship dynamics of rat pup huddling behaviour (Rocha-Almeida et al 2025). In ongoing work, we are working to understand the sensory, vocal and circuit mechanisms underlying reciprocal offspring-caregiver interactions across rodent species. We are developing research in the African spiny mouse, a gregarious, pro-social rodent with precocial offspring and the only rodent with a menstrual cycle. By pursuing a comparative approach across altricial and precocial rodent species, we aim to elucidate evolutionarily conserved and divergent neurobiological principles which will help us understand the development of the social brain and body interactions within environmental context.

Biosketch

Dr. Ann Clemens pursued her baccalaureate studies at the University of Texas at Austin and postbaccalaureate research at the National Institutes of Health in Bethesda. She returned to Austin to pursue her PhD in the laboratory of Dr Daniel Johnston, where she examined intrinsic plasticity in neurons of the hippocampus. Upon completion of her PhD, Ann moved to Berlin, Germany where she performed a postdoc in the lab of Michael Brecht. Her postdoctoral research focused on uncovering the cellular mechanisms of social-sensory processing from a structure-function perspective. Since 2016, Ann has co-led the Somatosensory/ Rat module of the Neural Systems and Behaviour Course and was a pandemic Grass Fellow in 2020 at the Marine Biological Laboratory in Woods Hole, MA. Ann is now principal investigator of the Kinship Lab at the University of Edinburgh, Simons Initiative for the Developing Brain. The research of Kinship Lab aims to understand the neurobiology and development of natural social behavior.

Ana Domingos

Department of Physiology, Anatomy & Genetics, University of Oxford, England

Webpage

X
Linkedin

Sympathetic neurons in obesity

Abstract

Obesity is a public health concern with limited treatment options, mainly focusing on sup-pressing appetite. However, reduced food intake triggers a compensatory decrease in energy expenditure (EE), hindering weight loss. Effective obesity management medications that elevate energy expenditure, such as brain-acting sympathomimetics, increase widespread sympathetic activity, raising the heart rate. This often results in their market withdrawal or rejection by regulatory agencies despite their potency in reducing body weight. Thus, cardio-protective EE-boosting drugs are an unmet medical need. My lab (1) and others have shown that directly facilitating the activity of sympathetic neurons (outside the brain) drives weight loss without suppressing food intake or inducing cardiac side effects. Our lab has demonstrated that sympathetic neurons directly burn fat (2), producing norepinephrine that simultaneously triggers lipolysis and thermogenesis, and neuropep-tide Y (NPY) that sustains the progenitors of thermogenic adipocytes3. This mechanism may explain why, in humans, defects in NPY are associated with high BMI but not with changes in feeding patterns or cardiovascular parameters (3). Fat-burning sympathetic neurons are protected by thin anti-inflammatory IL33-expressing perineurial barrier cells (4) that shed away as leptin levels gradually rise as mice fatten up (5). The leaky perineurial barrier permits the invasion of sympathetic-associated macrophages, which contribute to obesity by importing and metabolizing norepinephrine (6). Pharmacologically re-versing any of these biological processes may pave the way towards cardioprotective EE-boosting anti-obesity drugs.

1. Mahú, I., et al, Bernardes, G. J. L. & Domingos, A. I. Brain-Sparing Sympathofacilitators Mitigate Obesity without Adverse Cardiovascular Effects. Cell Metab. 2020
2. Zeng, W., et al Domingos, A. I. Sympathetic Neuro-adipose Connections Mediate Leptin-Driven Lipolysis. Cell 2015
3. Zhu, Y., et al & Domingos, A. I. Sympathetic neuron derived NPY protects from obesity by sustaining the mural progenitors of thermogenic adipocytes. Nature. 2024
4. Haberman, E. R., et al., Domingos, A. I.. Immunomodulatory Leptin Receptor+ Sympathetic Perineurial Cells Protect Against Obesity by Facilitating Neuroendocrine-Mediated Brown Adipose Tissue Thermogenesis. Immunity. 2023
5. Sarker, G., et al., Domingos, A. I. (2023) The perineurium integrates leptin with its sym-pathetic outflow to protect against obesity. Under review at Nature. bioRxiv.
6. Pirzgalska, R. M., et al Domingos, A. I. Sympathetic neuron–associated macrophages contribute to obesity by importing and metabolizing norepinephrine. Nature. Med. 2017.

Biosketch

Ana I. Domingos is a Professor of Neuroscience at the University of Oxford. Her laboratory discovered the sympathetic neuro-adipose axis mediating leptin's lipolytic effects, providing the first visualization of adipose sympathetic neurons essential for fat mass reduction via norepinephrine signaling. Her team identified Sympathetic neuron-Associated Macrophages (SAMs), contributing to obesity by metabolizing norepinephrine, findings that inspired the development of sympathofacilitators—a novel class of peripheral anti-obesity drugs free from central nervous system side effects. Domingos' research focuses on the pharmacological regulation of autonomic functions to combat obesity safely, pioneering the emerging field of Neuroimmunometabolism. Her group has extensively reviewed this field (Nature Reviews Endocrinology, Annual Review of Cell and Developmental Biology, Neuron) and organized dedicated conferences, including the Keystone Symposium (2022). Domingos serves as editor-in-chief of the American Journal of Physiology - Endocrinology and Metabolism and holds editorial roles at Cell Metabolism and eLife. Her numerous accolades include the EMBO Installation Award, Human Frontiers Science Program Award, Howard Hughes Medical Institute–Wellcome International Scholar Award, ERC-Consolidator Award, Pfizer Aspire Obesity Award, Carl Ludwig Lectureship, BBSRC Grant, and NIH Opportunity Pool Award. She has been invited to speak at over 70 international conferences.

Ivan E. de Araujo

DPhil Direktor
Max-Planck-Institut für biologische Kybernetik
Honorarprofessor
Eberhard Karls Universität Tübingen

Webpage

Psychological States and the Regulation of Mucosal Immunity

Abstract

Despite initial skepticism, it has been established that psychological states generated in the brain can affect critical body functions, such as immunity and mucosal barrier integrity. However, circuit models for such mechanisms are generally lacking. This presentation discusses recent developments in deciphering body-brain neuronal pathways, specifically those involved in the regulation of immune organs and defense against pathogens. The circuitry that enables the parasympathetic nervous system, via the vagus nerve, to regulate gut microbiome composition, mucosal permeability, and peripheral immunity will be emphasized. These circuits represent potential entry points for novel neural therapies that target the immunological effects of psychological distress.

Biosketch

Ivan De Araujo is a director at the Max Planck Institute for Biological Cybernetics in Tübingen, Germany, and an Honorary Professor of Medicine at the University of Tübingen. He is known for his investigations on sugar preference and reward functions of the gut-brain axis. After describing the taste-independent reward phenomenon, he conducted pioneering studies linking the intestine to the brain dopamine system. He defended his doctoral (DPhil) thesis at the University of Oxford, where he studied cortical mechanisms linked to pleasurable perceptions induced by food in humans. After his doctoral studies, he held a postdoctoral research position at Duke University. He then took up an assistant professor position at Pierce Laboratories at Yale University, where he was eventually appointed Professor of Psychiatry. Between 2018 and 2023, he served as a Professor of Neuroscience at Mount Sinai Hospital in New York. In May 2023, the Max Planck Society announced his appointment as a Director at the Max Planck Institute for Biological Cybernetics in Tübingen.

Gérard Eberl

Institut Pasteur, France

Webpage

Impact of acute and chronic inflammation on brain and metabolism

Abstract

The immune system and the nervous system express a large and overlapping set of receptors to sense the milieu intérieur and the environment. The two systems interact at many levels to balance the energy budget and regulate each other. I will show you how the microbiota provides information to the brain to manage the energy budget et adapt behavior in the face of potential danger, and how the brain senses the environment to regulate immune responses.

Biosketch

Gérard Eberl completed his PhD at the University of Lausanne on the structure of epitopes recognized by T cells, followed by a first post-doctoral fellowship at the Ludwig Institute for Cancer Research, also in Lausanne, on the development and function of invariant NKT cells. A second post-doctorate in New York led him to characterize the role of the nuclear hormone receptor RORgt in innate lymphoid cells (ILC). In 2005, Gérard was recruited to the Institut Pasteur in Paris to lead the Lymphoid Tissue Development Unit, which became the Microenvironment & Immunity Unit in 2015. For 10 years now, his laboratory has been studying the impact of symbiotic microbiota on the development and activity of the immune system, mainly in the gut, but also at more distant sites, such as adipose tissue and the lung. More recently, he has been interested in understanding how the microbiota also influences mouse behavior through activation of the innate immune system, and how the nervous system modulates immune responses. From 2015 to 2019, Gerard was the director of the immunology department at the Institut Pasteur.

Amaury François

Institut de Génomique Fonctionelle, CNRS

Webpage

Linkedin

Central integration of social touch

Abstract

Touch is more than a means of physical perception, it plays a vital role in shaping emotional and social behaviors, such as bonding and affiliation. While the neural circuits for tactile discrimination are well defined, the pathways mediating the emotional and motivational impact of touch remain largely uncharted.
To bridge this gap, we employed genetic and viral tools to selectively manipulate tactile afferents in mice. We pinpointed a specific class of sensory neurons—C-LTMRs—whose activation makes touch rewarding and enhances social interaction. Conversely, dampening C-LTMR activity led to a marked reduction in social behaviors.
We further mapped key integrative hubs and cortical circuits activated by pleasant touch, revealing that C-LTMR-driven pathways modulate appetitive behaviour and social motivation in unexpected ways. These findings highlight the multimodal nature of sensory processing and its influence on behavior.
These findings advance our understanding of how affective somatosensory signals are integrated into reward circuits and how their dysregulation may contribute to deficits in motivation and social functioning in pathological conditions.

Biosketch

Amaury François is a Research Fellow at the Montpellier Institute of Functional Genomics. Trained as an electrophysiologist and behaviorist, he quickly developed a passion for the study of the somatosensory system and the integration of tactile information. After defending his thesis in 2013 on the excitability of neurons responsible for pain transmission in Montpellier, he spent 4 years working on top-down pain control and endogenous opioids at Stanford University in Gregory Scherrer's team.
The research team he has been leading since 2023 focuses on the role of touch in social behavior. His team has demonstrated the existence of sensory neurons innervating the skin, the C-LTMRs, responsible for pleasant touch. These same neurons, when artificially activated, reinforce social interactions in mice, and vice versa when inhibited. The team also demonstrated a link between C-LTMR activity and sensory phenotypes associated with autism spectrum disorder. Since then, his research team has focused on identifying how the information carried by these neurons converges with social information to influence animal behavior. He is particularly interested in the somato-sensory system and the integration of tactile information in cortical areas. He is seeking to identify the neural networks linking touch to emotions.

Giuseppe Gangarossa

Université Paris Cité, Unité Biologie Fonctionnelle et Adaptative (CNRS UMR 8251)
Institut Universitaire de France (IUF), France

Webpage

X
Bluesky
Linkedin

The gut-brain vagal axis governs mesolimbic dopamine dynamics and reward events

Abstract

Reward-related processes have traditionally been ascribed to neural circuits centered on the dopamine (DA) system. While external stimuli, such as food and drugs of abuse, are well-established activators of DA-neuron activity, growing evidence indicates that interoceptive signals also play a critical role in modulating reward. Among these, the gut-brain vagal axis has emerged as a key pathway, yet its precise contribution to mesolimbic DA-dependent signaling, dynamics and behaviors remains poorly defined.
Here, we combine complementary *ex vivo* and *in vivo* approaches across multiple scales to investigate how the gut-brain vagal axis regulates DA dynamics and reward-related behaviors. We show that gut-brain vagal tone is essential for gating mesolimbic DA system activity and functions, modulating DA-dependent molecular and cellular processes, and scaling both food- and drugs-induced reinforcement.
These findings challenge the traditional brain-centric view of reward processing, supporting a more unified and integrated model in which gut-derived and vagus-mediated interoceptive signals are pivotal in intrinsically shaping motivation and reinforcement. By uncovering the influence of gut-brain vagal communication on mesolimbic DA functions, this work offers new insights into the neurobiological mechanisms underlying both adaptive and maladaptive reward processes, with broad implications for eating disorders and addiction.

Biosketch

Giuseppe Gangarossa is Professor of Neurobiology at Université Paris Cité and a member of the Unit of Functional and Adaptive Biology. His research explores the molecular and cellular mechanisms governing homeostatic and hedonic circuits that regulate energy balance and feeding behaviors, drawing inspiration from the emerging field of metabo-psychiatry.

He earned a Pharm.D. from the University of Bologna (Italy) and a European Ph.D. in Biomedical Sciences from the University of Bologna and Karolinska Institutet (Sweden) in 2011. Between 2011 and 2016, he pursued postdoctoral training in France (Montpellier, Paris) and Canada (London, Montreal). Over time, his research evolved from pure neurosciences to integrative physiology, reinforcing his belief that the brain does not act alone. In 2017, he was appointed Associate Professor at Université Paris Cité and promoted to Full Professor in 2022. His recent distinctions include the junior membership to the Institut Universitaire de France (2023) and the Humboldt Fellowship (2025). As a Humboldtian, he is also

affiliated with the Max Planck Institute for Biological Cybernetics (Tubingen, Germany), in the Body-Brain Department of Prof. Ivan de Araujo.

A committed advocate for equity in academia, Prof. Gangarossa actively works to combat discrimination and LGBTQ+ stigmatization as a member of the Alba Network.

Christelle Gangletas

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

Webpage

Bluesky
Linkedin

Insula and social behavior

Abstract

The Insula, which is known for its involvement in multisensory integration and socio-emotional processing, has a particularly robust, yet understudied, connection with its contralateral counterpart. In my recent study, I investigated the functional relevance of Insula-to-Insula ($Insula^{Ins}$) communication in modulating social behaviour and anxiety in mice. To achieve this, we employed a multidisciplinary approach, combining viral tracer neuroanatomy, *ex vivo* and *in vivo* electrophysiology, fiber photometry and targeted neural circuit manipulation.

During my presentation, I will discuss how we discovered that $Insula^{Ins}$ neurons are crucial for social discrimination following acute social isolation in mice. Our findings shed new light on the role of insular interhemispheric communication in social and emotional processing.

Biosketch

Christelle Glangetas is a senior neuroscientist working at the Institute of Neurodegenerative Diseases in Bordeaux. She is also a co-organiser of the Brain-Body conference.

She obtained a PhD in Neurosciences at the University of Bordeaux in 2014, where she investigated the role of neuronal plasticity in anxiety control. She moved to Switzerland to the team of Camilla Bellone as a postdoctoral researcher, where she worked on motor and anxiety circuits in physiopathological conditions (Huntington's disease, Autism Spectrum Disorders). Since 2018, she has joined the team of Jérôme Baufreton and François Georges at the Institute of Neurodegenerative Diseases to understand how the brain shapes emotional behaviors.

Nadine Gogolla

Max Planck Institute of Psychiatry, Emotion Research Department, Germany

Website

Interoceptive regulation of emotional homeostasis: a key role for the Insular Cortex

Abstract

Maintaining emotional balance is as crucial for survival as maintaining bodily homeostasis. The insular cortex is a key hub for interoceptive and affective processing, integrating bodily feedback signals with external sensory information to regulate both physiological and emotional states. However, the neural mechanisms underlying its dual role in homeostatic regulation and emotional modulation remain poorly understood.

Our previous work has demonstrated that the insula plays a dual role in emotional regulation, promoting the extinction of strong fear memories while maintaining weak ones, thus establishing 'emotion homeostasis'. To further elucidate this function, I will discuss ongoing research investigating the interplay between interoception and neuromodulation in the insular cortex, with a focus on the release dynamics of acetylcholine (ACh) and noradrenaline (NA) in response to changes in bodily physiology and emotional state.

Our findings reveal distinct release patterns of NA and ACh, and specific interference with their release during fear extinction learning demonstrates antagonistic effects, highlighting the crucial role of both neuromodulators in maintaining fear levels in equilibrium. These results provide new insights into the neural mechanisms underlying emotional regulation, the interoceptive basis of emotion, and have implications for understanding and treating emotional disorders.

Biosketch

Nadine Gogolla's research focuses on the neuronal underpinnings of emotion. Using the mouse as a model organism, her lab combines modern systems and circuit neuroscience tools with precise analyses of behavior and bodily physiology. In particular, Nadine's work focusses on the role of the insular cortex in interoception and emotion. Nadine obtained her PhD in Neurobiology from the University Basel, Switzerland, for her work performed in the lab of Prof. Dr. Pico Caroni at the Friedrich Miescher Institute of Biomedical Research. As a postdoc, Nadine worked in the lab of Prof. Dr. Takao Hensch at Harvard University. Between 2014 and 2021, Nadine was a Max Planck Research Group Leader at the Max Planck Institute of Neurobiology in Martinsried, Germany. In October 2021, Nadine was appointed director at the Max Planck Institute of Psychiatry in Munich, Germany, where she leads the Emotion Research Department.

Ali Jawaid

Affiliation Translational Neuropsychiatry Research Group (TREND Lab), Lukasiewicz Research Network- PORT Polish Center for Technology Development, Wroclaw, Poland

Website

X
Instagram

Immunometabolic basis of neuropsychiatric disorders: Across the lifespan and across generations

Abstract

Childhood trauma is a significant risk factor for a wide range of psychiatric and physical health disorders in adulthood. Emerging evidence suggests that the consequences of such early-life adversity may not be limited to a single generation but can also be transmitted biologically to subsequent generations. In this study, we investigate the molecular underpinnings of the long-term effects and intergenerational transmission of trauma, with a particular focus on small non-coding RNAs. We analyze serum, sperm, and breast milk samples from multiple trauma-exposed human cohorts, including Pakistani children and adult men with histories of complex childhood trauma, Polish mothers who experienced adverse childhood events, and Bosnian individuals who lived through the genocide during their formative years. To complement these human data, we employ a well-established mouse model of post-natal trauma based on unpredictable maternal separation and stress. Our cross-species analysis reveals that lipid-related circulating microRNAs (miRNAs) are significantly altered in response to early-life trauma and appear to play a critical role in mediating the transmission of trauma-related effects across generations. These findings suggest that specific miRNAs may serve as potential biomarkers for trauma exposure and transmission, offering novel insights into biological mechanisms of epigenetic vulnerability, as well as potential avenues for targeted therapeutic interventions.

Biosketch

Dr. Ali Jawaid, MD, PhD is a physician-scientist with training in both clinical and basic neuroscience. He is a Principal Research Investigator at the Research Network Łukasiewicz – PORT Polish Center for Technology Development in Wroclaw, Poland.

He completed his medical studies from Aga Khan University, Karachi, Pakistan, and followed it up with fellowship in Neuropsychiatry from Baylor College of Medicine, Houston, TX, USA. He then proceeded to complete an MD-PhD in Neuroscience from Switzerland (simultaneous PhD degrees awarded by UZH/ETH International Program in Neuroscience and UZH MD-PhD program in 2016). This was followed by a postdoc in Neuroepigenetic at ETH Zurich. Since 2020, Dr. Jawaid has been the head of Laboratory for Translational Research in Neuropsychiatric Disorders (TREND Lab) working on the immunometabolic and epigenetic basis of neuropsychiatric disorders across generations. He has authored 75+

publications, including original studies in Nature Neuroscience, Neuron, Nature Communications, EMBO Journal, and Translational Psychiatry and has a current H-index of 31.

Dr. Ali Jawaid is also is a scholar of the FENS-Kavli Network of Excellence, a platform of 30 most outstanding early to mid-career neuroscience prinicial investigators acrsso Europe. He is a fiction author, poet, and virtual-reality enthusiast outside of scientific work.

Zachary A. Knight

Professor, UCSF and Investigator, HHMI, USA

Webpage

X

Control of ingestion by the caudal brainstem

Abstract

The progress of a meal is controlled by sensory feedback generated during eating. I will describe our work investigating how these signals are integrated in the caudal brainstem and used to control behavior. A central theme is how feedback from the mouth and gut interacts both during ingestion and over longer timescales through learning.

Biosketch

Zachary Knight is a Professor in the Department of Physiology at UCSF and an Investigator of the Howard Hughes Medical Institute. His lab studies the neural mechanisms that control homeostasis, including especially the regulation of hunger and thirst. Zack received his B.A. in Chemistry from Princeton University and his Ph.D. in Chemistry from UCSF, where he performed research in the lab of Kevan Shokat. Zack then performed postdoctoral studies in physiology with Jeffrey Friedman at Rockefeller University, before returning to UCSF to start his independent lab in 2012.

Yoav Livneh

Department of Brain Sciences, Weizmann Institute of Science, Israel

Website

Bluesky
X

Brain-body interactions: sensations and predictions in the insular cortex

Abstract

The brain and body are in a continuous dialog that is essential for our physical and mental health. Yet, we still do not have a detailed neurobiological understanding of how this dialog is achieved. How do cortical activity patterns give rise to specific interoceptive sensations? How are internal and external information streams integrated to form specific interoceptive predictions? How are learned predictions transformed into top-down signals that prepare the body for anticipated physiological changes? I will present our recent work addressing these questions using non-invasive gut optogenetics for quantitative interoceptive psychophysics, combined with two-photon cortical imaging, optogenetics, and endocrine measurements. We use this combined approach to uncover an essential neural basis for perception of internal sensations in the insular cortex, as well as mechanisms of learning interoceptive predictions. I will further show that insular cortex predictions are important for anticipatory physiological control. This cortically-controlled anticipatory physiological regulation is essential for maintaining metabolic homeostasis on slower timescales long after the anticipatory phase.

Biosketch

Yoav studied Psychology and Biology for his BSc at The Hebrew University of Jerusalem. He initially planned to be a psychotherapist, but soon fell in love with Neurobiology. He then did his PhD in Neurobiology at The Hebrew University of Jerusalem in Israel with Prof. Adi Mizrahi. During his PhD he investigated the development and plasticity of olfactory adult-born neurons. His studies ranged from single synapses to cellular sensory physiology.

For his postdoctoral research, Yoav joined the labs of Profs. Mark Andermann and Brad Lowell at the Endocrinology Department of BIDMC of Harvard Medical School. There he investigated interoception of physiological needs such as hunger and thirst, and how they bias our perception of sensory cues in our environment.

Yoav started his research group at the Weizmann Institute in Israel in 2020. His lab seeks to understand brain-body communication, and its role in regulating diverse behaviors. They do so by studying cortical computations in insular cortex and how they bidirectionally relate to changes in bodily physiology. His lab focuses on prioritization of different physiological needs, perception of internal sensations, and the causal role of cortical computations in regulating bodily physiology.

Arianna Maffei

Professor, Department of Neurobiology and Behavior,
Director, PhD Program in Neuroscience,
Stony Brook University, Stony Brook, NY

Website

Neural Mechanisms for Taste Learning

Abstract

Taste experience early in life appear to influence food choices in adulthood. However, how taste preferences are established early in life and the circuit mechanisms that regulate them in adulthood have not been investigated. The gustatory cortex is involved in the detection and encoding of both sensory and emotional (hedonic) aspects of taste. Thus, it is an ideal model circuit to investigate the mechanisms regulating taste preference and determine how preference influences eating behaviors. As taste guides feeding behaviors in all mammalian species, many of the mechanisms regulating the gustatory system are shared across species.

I will present experimental evidence for the distinct circuit mechanisms regulating taste preferences early in life and in adulthood and discuss the circuit underpinning for the association of the identity of a taste with its hedonic value. I will also show how plasticity can modify the hedonic value of a taste by changing it from pleasurable to aversive and our current evidence about the mechanisms for this plasticity. The results of this work have important implications for our understanding of taste perception and taste guided behaviors. More broadly, they also inform us about how our perception of a sensory stimuli is modulated by their affective dimensions.

Biosketch

Dr. Maffei obtained her undergraduate degree in Biology (1997) and her PhD in Biophysics (2002) from the University of Pavia in Italy. During her graduate work she investigated the synaptic properties and plasticity of the mossy fiber input to cerebellar granule cells. In 2002 she joined Dr. Gina Turrigiano's group at Brandeis University for her postdoctoral training. In the Turrigiano lab, she investigated plasticity mechanisms induced by visual deprivation, focusing on homeostatic plasticity and on plasticity at GABAergic synapses. In 2008, Dr Maffei joined the faculty of the Department of Neurobiology and Behavior at Stony Brook University as an Assistant Professor. She was promoted to Associate Professor in 2014 and to Full Professor in 2020. She continued to investigate circuit mechanisms for plasticity in visual cortex and more recently shifted the interest of her lab to the analysis of circuits for taste processing, taste learning and their contribution to taste preferences.

Claire Martin

Unité Biologie Fonctionnelle & Adaptative, Université Paris Cité, CNRS UMR 8251

Webpage

Bluesky
Linkedin

The impact of metabolic status on olfactory processing

Abstract

Smell is one of the most important sensory cues for identifying food and plays a key role in food choice and consumption. Furthermore, olfaction is modulated by nutritional status. Although endocrine regulation is known to modulate olfactory physiology and behavior, it is unlikely to be the sole contributor to these effects. Despite evidence of an interconnection between the hypothalamus and the olfactory system, the impact of hypothalamic control on olfactory activity and olfactory-driven feeding behavior remains poorly understood. We investigated the influence of AgRP neurons in the arcuate nucleus of the hypothalamus on olfactory processing. Using two mouse models in which the activity of AgRP neurons was manipulated, we combined behavioral testing and in vivo calcium recording of granule cells using fiber photometry. Our data show that manipulating AgRP neuron activity affects olfactory behavior and granule cell activity independently of nutritional status. Our findings reveal a new neuronal pathway that can tune olfactory processing at the level of the olfactory bulb, in addition to endocrine regulation.

Biosketch

Claire Martin is a CNRS senior scientist working at the University Paris Cité and a member of the Unit of Functional and Adaptive Biology. She obtained her PhD in Neuroscience from Lyon in 2004, where she demonstrated the role of beta-band oscillations in olfactory behavior, particularly memory. In 2005, she joined Leslie Kay's laboratory at the University of Chicago to further investigate the networks underlying memory acquisition and expression. In 2008, she was appointed as a CNRS researcher in the laboratory Imagerie et Modélisation en Neurobiologie et Cancérologie in Orsay, where she focused her research on sensory and multisensory processing, with a particular emphasis on olfaction. In 2016, she joined the Unit of Functional and Adaptive Biology, where she develops research themes on the sensory aspects of energy balance regulation and the role of neuron-astrocyte interactions in the physiopathology of obesity.

Enrica Montalban

Striatal Astrocytes Mediate Behavioral and Metabolic Adaptation in response to inflammation

Abstract

Beyond their role in reward processing, striatal circuits emerge as key nodes in the regulation of various dimensions of eating behavior. Here I will present recent findings directly supporting this view. On the one hand, manipulation of the activity of striatal astrocytes directly affects both peripheral metabolism and cognitive flexibility in the context of obesogenic diet exposure. On the other hand, manipulation of Ankk1 expression selectively in dopamine D2 receptor-expressing neurons of the mouse striatum, recapitulate both metabolic and reward processing phenotypes of humans carrying the Taq1A polymorphism in the Ankk1 gene. Together, these findings highlight the striatum as a main structure for body–brain interactions, at the interface of metabolism and reward.

Biosketch

Enrica Montalban is currently a Research Associate in neuroscience, and her current research focuses on understanding the pathophysiology of depressive symptoms associated with inflammation. Specifically, she focuses on the comorbidity between obesity and depression and investigate how obesity-induced inflammation alters the activity of the reward system, leading to disturbances in motivational tone. In this context, her past and current research has investigated the functionality of the reward system and in particular the mechanisms underlying the regulation and dysregulation of food intake in both physiological and pathological contexts. In her early work she developed transcriptomic approaches to characterize the genetic identities of striatal neuron subpopulations in relation to reward system dysregulations. The work of her first post-doc focused on understanding the relationship between energy metabolism and reward processing in the context of obesity and genetic susceptibility to psychiatric conditions, dissecting neuronal and astrocytic implications in this interplay. Recently, she has shifted towards translational approaches and developed a project focused on exploring the role of inflammation in the development of depression, in obese subjects and preclinical models of obesity.

Francesco Papaleo

Genetics of Cognition Laboratory, Neuroscience area, Istituto Italiano di Tecnologia, 16163 Genova, Italy.

IRCCS Ospedale Policlinico San Martino, Largo Rosanna Benzi, 10, 16132 Genova, Italy

Webpage

Bluesky

Inter-brain circuits of emotion recognition

Abstract

Social interactions imply dynamic and synergic feedback loops in which actions, reactions, and internal cognitive processes of each partner are modulated by the others. Pioneering discoveries show that brains working together, couple together, through interbrain synchrony. However, it is unclear whether interbrain dynamics change in the context of altered emotions, and if they might be causative for driving social interactions.
Combining microendoscopic Ca2+ imaging in the anterior cingulate cortex, area 24, with a behavioral task for emotion recognition in mice, we are finding that somatostatin-expressing (SOM+) neurons synchronize when a mouse interact with a stressed mouse, but not with mice in a neutral state. Conversely, data suggests that pyramidal neuron activity is correlated only among mice in a neutral state. This provides a first indication of cortical inhibitory neurons involvement in social interbrain neural dynamics in the context of altered emotions.

Biosketch

Dr. Francesco Papaleo is a tenured senior researcher, group leader of the Genetics of Cognition laboratory, at the Istituto Italiano di Tecnologia (IIT), Genova, Italy. The goal of our research effort is to investigate the mechanisms underlying social and cognitive processes that are altered in neurodevelopmental and psychiatric disorders. To reach this goal, we use a cross-disciplinary approach including detailed studies in genetically modified mice and parallel clinical investigations in humans. We employ a combined approach beginning at the behavioral level and culminating at the cellular and circuit level, integrating multifunctional techniques (e.g. in vivo electrophysiology, in vivo chemogenetics, in vivo optogenetics, in vivo miniendoscopes, in vivo fiber photometry etc.). Dr. Papaleo spearheaded his line of research through previous research experience at the University of Padova (Italy, 1996-2002), University of Bordeaux (France, 2002-2005) and the National Institute of Mental Health in Bethesda (USA, 2005-2010). Dr. Papaleo laboratory provided the neuroscience community with important findings disentangling new genetic and circuital mechanisms of social and cognitive (dys)functions.

Thomas Pradeu

DR1 CNRS ImmunoConcept UMR5164, Université de Bordeaux, France
Presidential Fellow, Chapman University, USA

Website

The immunological and psychological self

Abstract

In a quite fascinating conceptual migration, immunology – one of today's most dynamic biological and biomedical fields – has borrowed the concept of 'self' from philosophy and psychology. In this talk, I will tell the story of this migration, with a particular emphasis on the role played by F. M. Burnet, who received the Nobel Prize in 1960 for his theoretical work on this issue. I will explain how immunology has revisited the related concepts of self, individuality, and identity, so much so that immunology was dubbed in the 1970s 'the science of self and nonself'. Finally, I will demonstrate that the concepts of self and nonself in their original immunological sense are inadequate in today's immunology, but I will also suggest that a renewed concept of self can fruitfully be built to shed light on crucial questions in the thriving interdisciplinary field of neuroimmunology.

Biosketch

Thomas Pradeu is a tenured Senior Investigator in Philosophy of Science at CNRS & the University of Bordeaux, France, and a Presidential Fellow at Chapman University, California, USA. He is the founder and leader of the Conceptual Biology and Medicine team in Bordeaux, and the coordinator of the Philosophy in Biology and Medicine international network. From 2008 to 2014, he was an Associate Professor in Philosophy at Paris-Sorbonne University. Starting from 2014, he became an "embedded philosopher" in the Bordeaux immunology lab (ImmunoConcept). From 2015 to 2020, he was the PI of an ERC Starting Grant project on the microbiome and biological individuality. In 2020-21, he was a CASBS Fellow at Stanford University. His research, published equally in science and philosophy of science journals, deals with immunology, cancer biology, and the microbiome, and more generally with the conceptual and theoretical foundations of today's biological and biomedical sciences. His work in the field of conceptual and theoretical immunology has explored the immune self-nonself, immunological memory, the danger theory, the discontinuity theory, the crosstalk between the microbiome and the immune system, among many other issues. In 2017, he was awarded the Lakatos Award, the most prestigious award in philosophy of science internationally.

Asya Rolls

Faculty of life science, Tel Aviv University, Israel

Webpage

Immunoception: how the brain represents immunological states

Abstract

The synchronization between the external environment and an organism's internal state is crucial for survival. The immune system serves as a sensitive indicator of disturbances caused by both internal and external factors, and plays a central role in maintaining tissue homeostasis. Accordingly, central nervous system monitoring and regulation of immune function is vital for orchestrating appropriate physiological responses. In this talk, I will explore emerging insights into how the brain represents and modulates immune states, what we term immunoception. I will present recent findings on the neural encoding of immune information and discuss the implications of this brain-immune dialogue for health and disease. Finally, I will address how these mechanisms may underlie therapeutic benefits, as well as potential risks such as the development of psychosomatic disorders.

Biosketch

Prof. Asya Rolls is a researcher at the department of neuroscince biophysics and biochemistry at Tel Aviv University. She studies how the brain regulates immunity and how mental states can reflect in the organism's ability to cope with disease. Rolls is the recipient of several ERC grants (Starting, Consolidator and POC) from the European Research Council and was selected as one of 40 International Howard Hughes Medical Institute (HHMI) investigators in collaboration with the Wellcome Trust (2018-2023).

Lisa Roux

Université de Bordeaux, CNRS UMR 5297, Interdisciplinary Institute for Neuroscience

Breathing and hippocampal network activity during wake and sleep

Abstract

Brain activity and breathing rate influence each other but it remains unclear how fine respiratory features accompany brain state transitions. By recording nasal pressure and hippocampal activity in freely-moving mice, we detected respiratory pauses nested within breathing cycles. We showed that these pauses are the key components dictating the animal's respiratory rate and that they strongly impact neuronal networks. Moreover, pause properties discriminated Wake, rapid eye movement (REM) and non-REM (NREM) sleep. We proved that the rules linking respiration and states are generalizable by predicting states based on respiratory features in animals that were not part of our training set. Transient increases in pause durations paralleled the infra-slow oscillations of noradrenaline during NREM, highlighting the classical NREM packets delineated by movement-based micro-arousals and sigma-band power troughs. As pauses after inhalation predicted subtler sigma-band variations, we proposed new packets, based on pauses, structuring NREM in ~30s windows of high sigma power. Overall, our work revealed how respiratory features highlight the macro- and micro-architectures of sleep, suggesting new relationships between brain functions and respiration.

Biosketch

Lisa Roux is a research directorat CNRS leading the team "Neurophysiology of Natural Behaviors" at the Interdisciplinary Institute for Neuroscience (IINS, CNRS UMR5297, Bordeaux University, France) since 2018. She did her PhD under the supervision of C. Giaume at the Collège de France where she worked on neuro-glial interactions in the olfactory system using primarily slice electrophysiology. Her work unraveled a bi-directional loop of interactions between neuron and astrocyte networks which could impact olfactory information processing. In 2012, Lisa Roux joined the lab of Gyorgy Buzsáki (NYU, USA) as a postdoctoral fellow. There, she used advanced in vivo electrophysiology and optogenetic approaches in freely moving miceto understand the mechanisms of hippocampal oscillations and their function in spatial memory processes. Her work also uncovered the key role played by sharp wave ripple oscillations in the maintenance of the hippocampal "cognitive map" during spatial learning. As an independent group leader at the IINS, she now aims at understanding how the dialog between olfactory and memory networks underlie the formation, consolidation and recall of long-term memory traces. Her teammainly focuses on two paleocorticesimportant for memory function: the hippocampus and the olfactory piriform cortex.

Dana Small

Professor and Canada Excellence Research Chair in Metabolism and Brain, McGill University

Webpage

Bluesky

Peripheral signals regulating food choice in humans

Abstract

It is now well-established that the critical signals underlying food reinforcement are generated in the periphery during nutrient digestion. Evidence from studies in humans and in rodents also converge to show that the sensors, pathways and central representations are distinct for fat and for carbohydrate and that these separate systems may interact to potentiate reward. Here we examine the impact of adiposity, weight loss and diet on fat and carbohydrate reinforcement in humans. Results suggest that both adiposity and diet impact sensitivity to post-oral signals for fat but not carbohydrate.

Biosketch

Dana M Small is a Professor and Canada Excellence Research Chair in Brain and Metabolism at McGill University. Professor Small received her PhD in Clinical Psychology from McGill University in 2001 and subsequently established her lab at Yale University, where she was on faculty for 20 years. Professor Small's research focuses on understanding how sensory, metabolic and neural signals are integrated to determine food choices and on how the dysregulation of these systems contribute to the development of obesity, diabetes and cognitive impairment. Her work combines neuroimaging with metabolic, psychophysical and neuropsychological methods in humans and she has established a translational - reverse translational program of research through collaborations with colleagues working in mouse models. Her work has been recognized by international awards including the Alan Epstein Award from the Society for the Study of Ingestive Behavior, the Moskowitz-Jacobs Award and the Ajinomoto Award from the Association for Chemoreception Sciences. She served as Board Member for the National Academy of Sciences Board on Behavioral, Cognitive and Sensory Science (2014-2020), Divisional Director of Nutritional Psychiatry at Yale (2018-2022), Program Chair (2016-2018) and President (2022-2023) of the Society for the Study of Ingestive Behavior.

Catherine Tallon-Baudry

CNRS, LNC2, Ecole Normale Supérieure, Paris

Website

Another role for interoception: coordination of brain dynamics to create first-person perspective

Abstract

Interoceptive signalling is key for bodily regulations. However, interoceptive-related neuronal activity is 1) observed outside regions related to bodily regulations, such as visual or auditory cortices, 2) can be relatively independent from bodily parameters, and 3) co-varies with subjective experience in humans. I propose that interoceptive signalling plays another role: the coordination of brain dynamics to create first-person perspective. While still speculative, this proposal is backed up by experimental evidence and accounts also for some findings in the emotional literature.

Biosketch

Catherine Tallon-Baudry is a CNRS senior scientist in Cognitive Neuroscience at Ecole Normale Supérieure in Paris. She earned her PhD in Neuroscience in Lyon, France in 1997, showing the existence of gamma-band oscillations in humans and their role in visual cognition, a line of research she further developed as a Marie-Curie post-doctoral fellow in Bremen, Germany, using ECoG recordings in awake monkeys. She obtained a Cnrs tenure position and moved to to Hospital Pitié-Salpêtrière in Paris to create her own research group, where she unexpectedly found a double-dissociation at the neural level between spatial attention and visual consciousness. This led her to question the view of consciousness as a high-level cognitive function. To concentrate on subjective experience, she moved to the Department of Cognitive Sciences at Ecole Normale Supérieure to create a new research group developing and testing the neural subjective frame hypothesis, which posits that the first-person perspective inherent to subjective experience and the minimal self are rooted in visceral signals, particularly from the heart and the stomach. These rhythmic, self-generated signals may provide a temporal scaffold that coordinates neural activity across different brain regions, facilitating the integration of disparate sensory and cognitive processes into a unified, self-centered experience.

Laure Zago

Institut des Maladies Neurodégénératives, UMR 5293 CNRS Université de Bordeaux

Webpage

Linkedin

Effects of body-centred interventions on stress-related physiological markers and psychological well-being in women.

Abstract

Chronic stress is a major contributor to emotional dysregulation, cognitive decline, and increased vulnerability to neurodegenerative diseases. Women are particularly affected, due to both heightened exposure to daily stressors and greater neuroendocrine sensitivity—especially during hormonally dynamic life stages. Beyond its internal physiological and brain consequences, chronic stress leaves a visible imprint on the face, the primary medium of emotional expression. Persistent muscle tension often accumulates in key facial regions involved in expressivity, resulting in reduced spontaneous facial movement, impaired emotional communication, and weakened social connection. Over time, such tension contributes to the premature emergence of visible aging markers. These external signs can fuel a negative feedback loop, further exacerbating psychological distress and promoting social withdrawal.

In this context, the development of accessible, non-pharmacological, and empirically validated interventions to alleviate both the emotional and visible manifestations of stress constitutes a growing public health priority. Here, I present findings from a pilot study investigating the acute effects of two face-centred interventions. These interventions are grounded in distinct yet complementary approaches: one employing passive, tactile-sensory myofascial stimulation through facial massage, and the other engaging interoceptive processes via audio-guided relaxation. Despite differing in modality and stimulation type, both interventions share core mechanisms of action—they are somatically anchored, follow a bottom-up regulatory model, and target the autonomic nervous system, particularly through parasympathetic activation via the vagus nerve. In a first step of the project, we examined the effects of these interventions on physiological markers of stress (HRV) and psychological indicators of subjective well-being, as well as mental and physical relaxation, in a sample of young adult women.

Biosketch

Laure Zago is a senior researcher at the French National Centre for Scientific Research (CNRS), affiliated with the Groupe d'Imagerie Neurofonctionnelle (GIN), within the Institute of Neurodegenerative Diseases (IMN) in Bordeaux. She earned her PhD in Neuropsychology and Cognitive Neuroscience from the University of Lyon in 2000. She then completed postdoctoral research at Harvard University and the Martinos Centre for Biomedical Imaging at Massachusetts General Hospital, where she investigated cognitive functions with fMRI. In 2003, she was appointed as a CNRS research fellow and established her research program at the Cyceron Centre in Caen, a major hub for biomedical imaging. In 2016, she joined the IMN, where she developed a research line focusing on brain lateralisation, cognition, and

inter-individual variability. Her current work explores brain–body interactions, with a particular emphasis on the effects of body-centred interventions on physiological and brain markers related to stress, well-being, and women's health.

Selected short talks

Laetitia Davidovic
Microbiota and autism spectrum disorders: modulation of social behavior by the microbial metabolite p-cresol

Friedrich W. Johenning
Suppression of olfactory cortex activity gates sensory satiation during hedonic feeding

Victor Jouque
Hypothalamic POMC satiety neurons drive the intake of hypercaloric food

Meryl Malezieux
Cardio-insular processing shapes emotion state coding

Justus Ninnemann
Chronic systemic inflammation imposes critical thresholds in energy metabolism

Shervin Safavi
Emergence of brain-like features in artificial neural networks trained based on homeostasis

Megan Sammons
What's pain got to do with it: The Role of Pain in Encoding Immune Responses

Laetitia Davidovic

Microbiota and autism spectrum disorders: modulation of social behavior by the microbial metabolite p-cresol

Laetitia Davidovic[1]

[1] Université Côte d'Azur, CNRS UMR7275, INSERM U1318, Institut de Pharmacologie Moléculaire et Cellulaire, Valbonne, France

Autism spectrum disorders (ASD) are increasingly associated with alterations in the microbiota-gut-brain axis and elevated levels of microbial metabolites such as *p*-cresol. Our research provides converging evidence that *p*-cresol, a phenolic compound derived from both microbial fermentation and environmental sources, plays a causal role in inducing ASD-like behaviors in mice. Chronic oral exposure to *p*-cresol in adult mice leads to marked deficits in social behavior and stereotypies, without affecting anxiety or cognitive functions. These behavioral impairments are linked to altered gut microbiota composition and reduced excitability of dopaminergic neurons in the ventral tegmental area, a core component of the social reward circuitry. Importantly, fecal microbiota transplantation (FMT) from *p*-cresol-treated animals replicates these behavioral effects in control mice and increases fecal *p*-cresol levels, while FMT from control mice restores normal social behavior and dopaminergic function in *p*-cresol-treated animals.

Extending these findings to early developmental exposure, we demonstrate that perinatal p-cresol exposure—from mid-gestation through weaning—does not affect early physical or neurological development but induces persistent social deficits and stereotypies in adulthood. This suggests that p-cresol selectively disrupts neurodevelopmental processes relevant to ASD without causing broad developmental delays.

At the molecular level, we show that *p*-cresol and its host-conjugated form, *p*-cresol sulfate, accumulate in the brainstem and inhibit catecholamine biosynthesis by targeting tyrosine hydroxylase and dopamine-β-hydroxylase. Inhibition of dopamine-β-hydroxylase alone recapitulates the observed social deficits, establishing a direct mechanistic link between *p*-cresol exposure, disrupted neurotransmitter synthesis, and altered social behavior.

Together, our findings implicate *p*-cresol as a microbiota-derived and environmental factor contributing to ASD-relevant behaviors through both microbial and host neurochemical pathways. These results underscore the potential of targeting *p*-cresol production or action for microbiota-based therapeutic strategies in ASD.

Frierich W. Johenning

Charité-Universitätsmedizin Berlin, Germany

Suppression of olfactory cortex activity gates sensory satiation during hedonic feeding

Hung Lo (羅鴻)[1,2,3,13,14]*, Walter Cañedo Riedel[4,5], Malinda L. S. Tantirigama[6,7], Anke Schoenherr[1], Laura Moreno Velasquez[1], Lukas Faiss[1,8], Amit Kumar[1,3], Aileen Hakus[1,3,8], Benjamin R. Rost[1,8], Matthew E. Larkum[6], Benjamin Judkewitz[1,2,7], Katharina Stumpenhorst[9], Marion Rivalan[7,10], York Winter[9], Eleonora Russo[5,11], Wolfgang Kelsch[4,5], Dietmar Schmitz[1,2,3,7,8,12,15] and **Friedrich W. Johenning**[1,2,3]

[1]Charité–Universitätsmedizin Berlin, Neuroscience Research Center; Charitéplatz 1, 10117 Berlin, Germany,
[2]Charité–Universitätsmedizin Berlin, Einstein Center for Neurosciences Berlin; Charitéplatz 1, 10117 Berlin, Berlin, Germany,
[3]Charité-Universitätsmedizin Berlin, Institute of Cell and Neurobiology, 10117 Berlin, Germany.
[4]Department of Psychiatry and Psychotherapy, Central Institute of Mental Health, Medical Faculty Mannheim, Heidelberg University; 68159, Mannheim, Germany
[5]Department of Psychiatry and Psychotherapy, University Medical Center, Johannes Gutenberg University; 55131, Mainz, Germany
[6]Humboldt Universität zu Berlin, Institut für Biologie; Charitéplatz 1, 10117 Berlin, Germany,
[7]Charité–Universitätsmedizin Berlin, NeuroCure Cluster of Excellence; 10117 Berlin, Germany,
[8]German Center for Neurodegenerative Diseases (DZNE) Berlin; Charitéplatz 1, 10117 Berlin, Germany,
[9]Humboldt Universität zu Berlin, Cognitive Neurobiology; Philippstraße 13, 10115 Berlin, Germany
[10]Institute of Neuroscience Paris-Saclay, CNRS, University of Paris-Saclay, 91400 Saclay, France
[11]The BioRobotics Institute, Department of Excellence in Robotics and AI, Scuola Superiore Sant'Anna, Piazza Martiri della Libertà, 33, 56127, Pisa, Italy
[12]Humboldt Universität zu Berlin, Bernstein Center for Computational Neuroscience; Philippstraße 13, 10115 Berlin, Germany
[13]Present address: Dominick P. Purpura Department of Neuroscience, Albert Einstein College of Medicine, Bronx, New York, USA

The sensory experience of food shapes not only flavor perception but also the decision to continue or terminate eating. While brainstem and hypothalamic circuits regulating satiety and energy balance have been well characterized, the contribution of cortical olfactory areas to sensory satiation remains largely unexplored. Here, we identify a novel cortical gating mechanism in the anterior piriform cortex (aPC) that dynamically modulates feeding behavior based on internal state and food value.

Using in vivo calcium imaging in freely moving mice, we find that excitatory neurons in the aPC exhibit robust, stimulus-specific responses to different food flavors during slow feeding. Remarkably, during high-value "binge" feeding, these responses are globally suppressed—a shift that is not inherited from upstream olfactory areas or mediated by local inhibition. Instead, we identify GABAergic input from the olfactory tubercle as a likely source of this suppression, with tubercle activity scaling with food palatability and correlating with the degree of aPC silencing.

Closed-loop optogenetic manipulation of aPC excitatory neurons confirms this circuit's role in sensory-driven meal termination: inhibition prolongs feeding, while activation suppresses it. Together, our findings suggest that suppression of cortical olfactory representation reduces sensory satiation and promotes hedonic overconsumption.

This work reveals a mechanistic link between oronasal perception and visceral behavior, and suggests that cortical olfactory areas act as a state-dependent gate for the sensory control of feeding—an insight with implications for the treatment of compulsive eating disorders.

Victor Jouque

Neurocentre Magendie, Bordeaux, France

Hypothalamic POMC satiety neurons drive the intake of hypercaloric food

Victor Jouque[1], Vincent Simon[1], Antonio Lopez-Gambero[1], Philippe Zizzari[1], Samantha James[1], Shim Yaeun[2], Calderon Nathan[1], FabienDucrocq[2], Pierre Trifilieff[2], Xavier Fioramonti[2], Daniela Cota[1].

[1] INSERM, Neurocentre Magendie, U1215, 33000 Bordeaux, France.
[2] INRAE, NutriNeuro, UMR 1286, 33000 Bordeaux, France

Eating is a process essential for life. Therefore, powerful brain mechanisms have evolved to allow not only the matching of the organism's energy needs with energy intake, but also the recognition of food rich in calories to guarantee survival under variable environmental food sources. In this context, neuronal circuits classically aiming at integrating information about the organism's energy status must interact with networks regulating the rewarding aspect of food intake. However, little is known about how such communication is set in place and the neuronal substrates underlying this phenomenon. Hypothalamic pro-opiomelanocortin (POMC) neurons are classically viewed as the mediators of satiety in response to metabolic and hormonal cues. However, recent evidence demonstrates that POMC neurons are highly heterogeneous, can stimulate feeding under specific conditions, and become active before food consumption. In addition, POMC neurons release the μ-opioid receptor (MOR) agonist β-endorphin known to stimulate energy-dense food intake, therefore challenging the traditional view of their satietogenic function. Here we hypothesize that, contrary to established dogma, certain subpopulations of POMC neurons drive the consumption of hypercaloric high-fat diet (HFD). By combining neuroanatomical, electrophysiological, optogenetic and chemogenetic approaches, we demonstrate that POMC neurons are rapidly activated at time of HFD consumption through orosensory processes. Activation of hypothalamic POMC neurons is specific to HFD exposure and it drives hyperphagia, as chemogenetic inhibition of POMC neurons reduces HFD intake. These effects seem to require the synthesis and release of the POMC-derived peptide β-endorphin, as its levels increase in POMC terminals upon HFD exposure, while HFD-driven hyperphagia can be selectively inhibited by MOR antagonism. Anatomical and electrophysiological mapping of POMC projections suggests that the lateral septum (LS) may act as a potential downstream brain region involved in mediating this hyperphagic response by integrating β-endorphin signaling. Functional studies are currently ongoing to fully pinpoint the brain circuit involved. These set of findings, altogether provide novel information on the complex role played by hypothalamic POMC neurons in the regulation of feeding.

Meryl Malezieux

Max Planck Institute of Psychiatry, Munich, Germany

Olfaction and feeding behaviour: Neuronal substrates underlying odour modulation of food intake regulating neuronal circuits

Meryl Malezieux[1], Jeong Yeongseok[1], Eunjae Cho[1], Andrea Ressle[1], Bianca Schmid[1], Nadine Gogolla[1]

[1] Emotion Research Dept., Max Planck Institute of Psychiatry, Munich, Germany

Emotions are conserved functional states influenced by both external stimuli and internal bodily signals. Both classical and modern theories of emotion suggest that emotions do not only drive peripheral physiological responses, but also that changes in bodily signals themselves can influence the emergence and persistence of emotion states.

One key brain region implicated in the encoding of emotions and processing of bodily signals across species is the posterior insular cortex (pInsCtx). Recent work in mice has shown that elevating heart-rate using optogenetics is anxiogenic and activates the pInsCtx[1]. Additionally, perturbing heart-to-brain communication using vagus nerve stimulation impedes fear extinction in a pInsCtx-dependent manner[2]. However, we currently lack a mechanistic understanding of how the pInsCtx processes cardiac signals and how this contributes to the emergence of emotion states.

To address this, we recorded pInsCtx activity during aversive and appetitive conditioning in mice in the presence or absence of the β-blocker metoprolol. To characterize the behavioral and physiological responses associated with positive and aversive emotion states, we monitored heart-rate, pupil diameter, facial expressions and locomotion.

Our findings reveal that heart-rate is the strongest modulator of InsCtx activity, more so than changes in arousal or locomotion. Strikingly, single neurons in the insula are precisely tuned to heartbeats, even at the membrane potential level, and heartbeat tuning increases during both aversive and appetitive emotion states. Notably, β-blockade long-lastingly reduces heartrate and, by doing so, prevents the representation of cardiac signals by the pInsCtx. This manipulation leads to strong deficits in the pInsCtx properties of sensing heart rate changes and individual heartbeats. Concomitantly, pInsCtx becomes impaired in responding to emotional cues and decoding emotional valence. Taken together, our results show that the pInsCtx precisely processes cardiac signals and that this processing shapes emotion state coding.

1. Hsueh, B., Chen, R., Jo, Y., Tang, D., Raffiee, M., Kim, Y.S., Inoue, M., Randles, S., Ramakrishnan, C., Patel, S., et al. (2023). Cardiogenic control of affective behavioural state. *Nature*

2. Klein, A.S., Dolensek, N., Weiand, C., and Gogolla, N. (2021). Fear balance is maintained by bodily feedback to the insular cortex in mice. *Science*

Justus Ninemann

Institut Pasteur, Paris, France

Chronic systemic inflammation imposes critical thresholds in energy metabolism

Justus Ninnemann[1], Gabriel Lepousez[2], Pierre-Marie Lledo[2], Gérard Eberl[1]

[1] Institut Pasteur, Université de Paris Cité, Inserm U1224, Microenvironment and Immunity Unit, 75015 Paris, France
[2] Institut Pasteur, Université de Paris Cité, CNRS UMR 3571, Perception and Memory Unit, 75015 Paris, France

The brain senses inflammatory signals upon infection and responds by inducing sickness behavior, which is characterized by fever, lethargy, social withdrawal and reduced appetite. This coordinated response of the immune system and the brain is a strategy to control infection in the host, as well as at the population level. However, it is unclear how behavior and metabolism are affected during chronic inflammation. We investigated the influence of chronic inflammation on behavior and metabolism by utilizing programmable, refillable micro-infusion pumps. By measuring behavioral parameters in real time, we found that a prolonged systemic inflammation increases the energy expenditure to critical threshold levels. To maintain a viable metabolism, physical activity is reduced, while food and water consumption is maintained. Most strikingly, the core body temperature occasionally drops to ambient temperature levels (torpors), leading to new states of equilibrium (allostasis). This inflammation dependent hypometabolic state is induced by the preoptic area (POA) of the hypothalamus, a connective hub known to be involved in thermoregulation and torpor upon fasting. We show that glucose-sensitive nuclei like the parabrachial nucleus (PBN), paraventricular nucleus (PVN) and supraoptic nucleus (SON) directly project to the POA and are highly activated during chronic inflammation induced torpor. Indeed, we confirmed that the prolonged inflammatory state reduced the serum glucose levels, employing a hypometabolic program.

Our data show that chronic inflammation imposes critical energetic thresholds via reducing available glucose levels, that force the POA to induce new states of equilibrium (allostasis) in order to maintain vital body functions.

Shervin Safavi

Max Planck Institute, Germany

Emergence of brain-like features in artificial neural networks trained based on homeostasis

Pakhi Banchalia[1,*], Harikrishna Satheesh Pillai[1,*], **Shervin Safavi[2,3]**

1 School of Computing, Amrita Vishwa Vidyapeetham, Amritapuri, India
2 Computational Neuroscience, Department of Child and Adolescent Psychiatry, Faculty of Medicine, TU Dresden, Germany
3 Department of Computational Neuroscience, Max Planck Institute for Biological Cybernetics, Germany
* Equal contribution

Regulating body homeostasis is a key principle of neural and cognitive functions [1]. The brain continuously monitors internal states and balances energy intake with physical activity, influencing behavior. Studying this relationship in animals is costly and limited by measurable neurons and behaviors. To overcome these constraints, we leverage artificial neural networks (ANNs) trained to maintain homeostasis.

Using homeostatic reinforcement learning [2], we trained ANNs in a simulated environment where agents regulate internal states defined by two nutrient variables [3]. Agents receive rewards not only for collecting resources but also for maintaining nutrient levels within a survival zone. Inputs include sensory, nutrition level, motor activity, and head orientation. To preserve complexity, the total number of resources in the environment was held constant.

Our results show that training ANNs for homeostasis leads to the emergence of a velocity-tuned neural system in ANNs and exploratory behavior, both modulated by nutrient levels. The agent learns to adapt its speed and movement according to internal state, suggesting that homeostatic regulation shapes core behavioral traits. By quantifying velocity profiles and correlating them with neural activity across ANN layers, we find distinct activation patterns tied to movement speed and nutrient status. Furthermore, the ANN exhibits nutrient-dependent exploratory behavior. We analyzed spatial coverage under varying nutrient levels (broader coverage, more exploration). In line with rodent studies [4], we found that 'hungry' ANNs explore more.

Overall, our model demonstrates the power of homeostatic reward shaping to induce adaptive, survival-oriented behavior in ANNs. The observed coupling between nutrient levels, velocity, neural dynamics, and exploration patterns highlights how internal states modulate both cognition and neural dynamics. These findings offer a novel computational framework for studying brain-body interactions and embodied intelligence.

[1] Weber et al. The interoceptive origin of reinforcement learning. Trends Cogn Sci 2025

[2] Keramati et al. Homeostatic reinforcement learning for integrating reward collection and physiological stability. Elife 2014

[3] Yoshida et al. Modeling long-term nutritional behaviors using deep homeostatic reinforcement learning. PNAS nexus 2024

[4] Kamath et al. Hunger modulates exploration through suppression of dopamine signaling in the tail of striatum. bioRxiv 2024

Megan Sammons

The Ruth and Bruce Rappaport Faculty of Medicine

What's pain got to do with it: The Role of Pain in Encoding Immune Responses

Megan Sammons[1], Margarita Sirokin[1], Tom Haran[1], Rotem Yam-Shahor[2], Rani Assa Polansky[2], Hilla Azulay-Debby[2], Liwen Deng[3], Issac Chiu[3], Asya Rolls[2]
1 Technion Institute of Technology, The Ruth and Bruce Rappaport Faculty of Medicine
2 Tel Aviv University, Faculty of Life Sciences
3 Harvard Medical School, Department of Immunology

Pain is a hallmark of inflammation, but does it serve a functional role in shaping immune processes? While growing evidence shows that peripheral sensory neurons can modulate local immune responses, it remains unclear whether the subjective experience of pain, constructed in the brain, also plays a role in immune regulation. Here, we show that pain-related information is crucial for the central representation of immune activity. Building on our previous work, we used TRAP2-Fos mice to capture neurons in the insular cortex (IC) that were activated during zymosan-induced peritonitis. By expressing DREADDs in these neurons, we were able to selectively reactivate them after recovery, thereby reinstating the inflammatory response. However, when pain was blocked during the initial inflammatory episode, using either systemic analgesics or resiniferatoxin (RTX), subsequent reactivation of these neurons led to significantly blunted immune responses, including reduced recruitment of neutrophils and monocytes to the peritoneal cavity. These findings reveal that pain is not merely a byproduct of inflammation but a critical signal for encoding immune responses in the brain. Our study positions pain as a necessary component for initiating, storing, and retrieving functional immune memory.

Posters

P1 - Brain-gut connectivity is dysregulated in disgust.
Sameer Alladin

P2 - Early Life Stress Impact on Anxiety-Related Behaviors and Anterior Insula Activity In Adult Mice
Zineb Aman

P3 – A hypothalamic circuit linking food odor perception to AgRP neuron suppression
Carlos G. Ardanaz

P4 - Prefrontal cortex interoceptive tuning can encode emotional state
Sophie Bagur

P5 - TaqIA polymorphism role in reward-associated disorders: new insights from a humanised murine model.
Fanny Bain

P6 - Mechanisms underlying cortical processing of gustatory information
Olga Barba-Vila

P7 - The role of addiction-susceptibility polymorphism TaqIA in metabolism and metabolic-associated disorders.
Simon Benoit

P8 – Dopamine D2R-neurons of the Paraventricular Thalamus Govern Feeding, Energy Balance and Body Homeostasis
Benoit Bertrand

P9 – Uncovering the neuro-immune mechanisms driving the ”memory of obesity“
Adèle Buckenmeyer

P10 – Leveraging linear models to correct interoceptive tuning for motion related activity
Ella Callas

P11 – Lateral habenula as a novel mechanism regulating systemic immune responses
Silvia Castany

P12 – Dopamine transmission in the anterior insula shapes the neural coding of anxiety
Yoni Couderc

P13 – Environmental Enrichment Modulates HPA Axis Reactivity and Anxiety in a Chronic Stress Model: Adaptive or Overloaded?
Nivea De Gusmaeo

P14 – Chemogenetic manipulation of insular cortex projections to the ventral striatum attenuates obesogenic diet-induced anxiety like behaviours
Eva-Gunnel Ducourneau

P15 – Olfaction and feeding behaviour: neuronal substrates underlying odour modulation of neuronal circuits regulating food intake
Louise Eygret

P16 – Lack of Single Amino Acids Transcriptionally Modulates Sensory Systems to Enhance Microbiota Intake
Gili Ezra-Nevo

P17 – Sex-specific metabolic and behavioral effects of ketogenic diet in a neuromelanin-based mouse model of prodromal Parkinson's disease
Sabrina Ayelén Gatti

P18 – The role of lipid metabolism and circulating miRNAs in the intergenerational transmission of the effects of parental adverse childhood experiences
Magdalena Gomolka

P19 – Miniature endoscope for high resolution electrical and optical investigation of the colon in live mice
Michalina Gora-Gioux

P20 – Induced Neural Responses to Respiration-Synchronized Olfactory Stimuli in Human EEG
Alisa Hathaway

P21 – Nicotine-induced immobility in mice reveals a coordinated brain- body recovery state post-stress
Chloé Hayhurst

P22 – The Effect of Sleep Deprivation and Nocturnal Light Exposure on Recovery Sleep and Morning Physiological Markers
Jana Koprivova

P23 – Differential involvement of the endocannabinoid system in obesogenic diet-induced memory impairments: influence of ovarian hormones
Clément Laffont

P24 – Interoceptive regulation of the fear response by a glucose-sensing circuit in the insular cortex
Christophe Lamy

P25 – Astrocytic Panx1 Channels in the Dorsal Striatum Regulate Energy Balance and Feeding Behavior
Caroline Léger

P26 – GABAergic neurons of the preBötzinger complex in the brainstem contribute to the generation of respiratory-cardiovascular coupling
Ambre Linossier

P27 – Hippocampal reactivation of aversive experience enables safety learning and slow-breathing state for stress recovery
Baptiste Maheo

P28 – The paraventricular thalamus regulates energy metabolism and glucose homeostasis
Nejmeh Mashhour

P29 – Hypothalamic PTH1R Signaling: New Therapeutic Perspectives for the Treatment of Obesity and Metabolic Disorders
Nour Mesto

P30 – Time and Mind: Electrophysiological Mechanism of Interval Timing
Tereza Nekovarova

P31 – How does the taq1a/ankk1 polymorphism influence reward related behaviours?
Asma Oummadi

P32 – Implication of neural circuits of the insular cortex in diet-induced anxiety
Léa Pages

P33 – Effect of Western Diet on Sensory Representation During Food Intake in the Mouse Olfactory Cortex
Shubhi Pal

P34 – Cardiac interoception and insular cortex: a pathway to understanding social deficits in Autism
Alessandra Panzeri

P35 – Burnout and interoceptive awareness, an exploratory research
Laure Pelloux

P36 – Implication of mesocorticolimbic dopamine transmission in behavioral flexibility: a role for dopamine and nmda receptor heteromers
Anna Petitbon

P37 – The translational functional architecture of cardiovascular interoceptors
Heloise Policet-Betend

P38 – Phascolarctobacterium faecium reverses gut motility impairment in mice with diet-induced obesity
Carlos Pomares-Diaz

P39 – Hippocampal endocannabinoid system mediates obesogenic diet-induced memory impairments in male mice
Mylene Potier

P40 – How adolescent obesity impacts social memory: the role of hippocampal CA2 and oxytocinergic system
Arielle Rakotonandrasana

P41 – Mapping the descending sympathetic innervation of spleen and pancreas
Leonor Remedio

P42 – Contribution of the anterior insular cortex in anxiolysis induced by a single injection of psychedelics
Daria Ricci

P43 – neuroIGNITOR: A flexible technology platform for stimulating and sensing peripheral nerves
Philipp Schnepel

P44 – Translational study of the protective role of indoles in psychiatric illnesses.
Toshiko Sekijima

P45 – Exploring salivary and emotional responses triggered by exposure to real foods
Carla Simoes

P46 – Infra-slow Frequency Oscillations Propagating through Multiple Organs Convey Information on Phase-specific Timing for Self-Initiated Actions
Filip Spaniel

P47 – Restoring effect of voluntary exercise on obesogenic diet-induced social memory deficits: emphasis on the oxytocin signaling in the CA2 area of the hippocampus
Emma van Loveren

P48 – Unravelling the contribution of glial cells in the insular cortex after obesogenic-diet induced anxiety
Lars Wilmes

P49 – Investigating how lung cancer alters behavior via regulating the internal sensory system
Anastasia Maria Zavitsanou

P50 – Neuromodulation in the posterior insular cortex maintains fear balance
Anna Zych

P1 - Brain-gut connectivity is dysregulated in disgust.

Alladin, S.N.B., Attwood, A., and Dalmaijer, E.S.

School of Psychological Science, University of Bristol, U.K.

Disgust is a basic emotion that helps to avoid ingesting potential toxins. Disgust has a characteristically visceral (stomach) response, typically accompanied by changes in gastric state (proto-nausea. Previous research has explored brain and stomach responses to disgust separately. But recent experimental work suggests that gastric state has a causal role in disgust experience. However, it remains unclear how brain and stomach communicate in the experience of disgust. This is particularly salient given the presence of a resting-state gastric network in the brain, which we hypothesised would be interrupted in the experience of disgust.

In the present study, we aimed to explore bidirectional stomach-brain connectivity during disgust, specifically whether and when stomach and brain signals impact each other. Participants (*N*=44) were exposed to blocks of neutral and core disgust stimuli in a passive-viewing task. Brain and stomach activity were measured using electroencephalography (EEG) and electrogastrography (EGG), respectively. Self-report disgust sensitivity and facial expression landmarks were also recorded.

Consistent with existing literature, we found normogastric power was reduced in response to disgust, whereas bradygastric and tachygastric power increased. Granger-causality revealed asynchronous connectivity between stomach and brain. Fronto-central regions of the brain predicted differences in stomach activity at an early lag, but during disgust this signal was attenuated. At later lags the stomach predicted changes in similar brain regions, which also reduced during disgust. Additionally, we found that self-reported disgust sensitivity was significantly positively correlated with somatosensory reductions in Granger-causality, indicating that higher disgust sensitivity was associated with a more dysregulated state. As a whole, these findings highlight the interaction between body-derived and brain-derived signals, with disgust serving as a model system for studying interoception. These present findings also indicate a clear relation between higher level constructs, such as disgust sensitivity, and low-level physiological processing of stimuli. Findings are discussed in the context of an active inference approach and views of exteroceptive vs. interoceptive processing.

P2 – Early Life Stress Impact on Anxiety-Related Behaviors and Anterior Insula Activity In Adult Mice

Zineb Aman, Thibault Bittar, Joeri Bordes, Anna Beyeler

Neurocentre Magendie, INSERM, Bordeaux Neurocampus, University of Bordeaux

The anterior insular cortex (anterior insula) is a critical hub for emotional integration and interoception, which was shown to be hyperactive in individuals with pathological anxiety. Early life stress is a major risk factor for anxiety disorders, yet its long-term effects on insula function remain poorly understood. In this study, we investigated how early life stress influences the dynamics of anterior insula pyramidal neuron during exposure to anxiogenic environments in adulthood.
Male and female C57BL/6J mice were subjected to a limited bedding and nesting (LBN) protocol during the early postnatal period from P02 to P09. In adulthood, pyramidal neuron activity was recorded using calcium fiber photometry during behavioral assays including the Elevated-Plus Maze (EPM), Elevated-Zero Maze (EZM), Light-Dark Box (LDB), Novelty-Suppressed Feeding Test (NSFT), social interaction tasks and a social valence assay.
The LBN early life stress increased anxiety-related behaviors of adult mice in multiple assays, along with a strong increase with locomotor activity and speed. As previously showed in our lab, mice exhibited a significant increase in anterior insula calcium activity when mice are navigating in exposed zones, such as the open arms of the EPM or the center of the OFT, reflecting heightened neural responses in dangerous contexts. Interestingly, although early life stress impacted behavioral outcomes, it did not change the amplitude of the anterior insula activity in response to dangerous areas, suggesting neurobiological compensatory mechanisms.

P3 - A hypothalamic circuit linking food odor perception to AgRP neuron suppression

Carlos G. Ardanaz[1]; Janice Bulk[1]; Joscha Schmehr[1], Sophie M. Steculorum[1]

[1] *Max Planck Institute for Metabolism Research, Cologne, Germany*

The brain exerts control over energy homeostasis by regulating food intake through signaling within the melanocortin system. Whilst we understand the role of the hypothalamus within this system, how extra-hypothalamic brain regions are involved in the control of energy balance, and how the development of these systems might be perturbed due to early influences, remains under investigation.
The melanocortin-3 receptor (MC3R) is implicated in modulating feeding behavior and body weight changes under different nutritional challenges, and MC3R deficient animals show a defective fasting response. The MC3R is highly expressed in the paraventricular nucleus of the thalamus (PVT): a brain region that integrates information about internal energy state with environmental stimuli to determine feeding and reward behaviors. Understanding the role and the development of MC3R neuronal activity within the PVT could present as an interesting nexus for food intake regulation in various contexts.
In this study we show that, in adult ad-libitum fed mice, MC3R-PVT neuronal activity follows a circadian pattern of activity. Moreover, this fluctuation of activity is dependent on food availability, as a 16-hour overnight fast alters this rhythm. Upon refeeding, however, this activity significantly increases to that seen under fed conditions. To further explore these effects in the PVT, we identified a role of early maternal overnutrition in impacting the development and maintenance of projections to the PVT and assessed changes in neuronal response to fasting established in the non-maternal diet manipulated paradigm outlined above.
In conclusion, we have identified circadian fluctuations in PVT-MC3R neuronal activity. These fluctuations are significantly impacted by energy state, as fasting results in changes to the activity pattern. We also show significant changes to the PVT in adult animals exposed to early maternal overnutrition. Further in-depth analysis of PVT MC3R neurons may yield advanced understanding of feeding-related behaviors.

P4 - Prefrontal cortex interoceptive tuning can encode emotional state

Sophie Bagur[1], Ella Callas[1], Dmitri Bryzgalov[1], Karim Benchenane[1]

[1] *Emotions, Memory, Oscillations and Brain states team – Karim Benchenane – ESPCI Paris*

Increasing evidence indicates that bodily states support emotional regulation[1,2,3]. But how does the brain "read out" body feedback to recognize the current emotional state and implement adaptive behaviors? The prefrontal cortex (PFC), positioned at the intersection of emotional and autonomic regulation[4], is well-suited to investigate this question. We recorded n=317 single units in the PFC of freely moving mice undergoing homecage sleep sessions, neutral exploration sessions and spatial fear conditioning.

We found that PFC neurons exhibit tuning to breathing (45%) and heart rate (35%), with either monotonic tuning or tuning peaking at a preferred intermediary frequency. Using generalized linear models to control for motion variables, we identified a subset of neurons specifically encoding cardiorespiratory signals, independent of movement. At the population level, breathing and heart rate could be accurately reconstructed from neural activity (cross-validated $R^2 = 0.86$ and 0.50, respectively). Remarkably, models trained on neutral wake states generalized well to stress ($R^2 = 0.74$) and sleep ($R^2 = 0.46$) for breathing. Cardiac frequency models trained in this way across wake states but not sleep. This suggests that the PFC more robustly encodes breathing—consistent with its strong respiratory phase modulation[1,5].

These observations provide strong evidence that specific prefrontal neurons encode cardio-respiratory frequency invariant to behavior and brain state, independent of motion variables with single-unit tuning curves that are reminiscent of classical senses such as audition or vision

These units provide a potential mechanism for the PFC to track bodily and emotional states. We therefore tested their functional relevance in emotional states differing in their breathing and heart rate profiles. Using our recent discrimination of two fear-related states characterized by immobility[6]—classical freezing (breathing >4Hz, high stress) and a novel recovery state (slow breathing ~2Hz, boosting resilience)—we found that PFC activity robustly discriminates between these states (89% accuracy). Importantly, the role of individual neurons in emotional state coding can be predicted from their interoceptive tuning. Accordingly, training a decoder to discriminate between high and low breathing states defined in other wake or sleep states is sufficient to decode the emotional state (76%). This demonstrates that interoceptive encoding in the PFC can support the encoding of different emotional states, providing a sensory mechanism for the brain to monitor ongoing emotional states and adapt behavior accordingly.

1. *Bagur, S. et al. Nat. Commun. 12, 2605 (2021).*
2. *Hsueh, B. et al. Nature (2023).*
3. *Klein, A. S et al. Science (2021).*
4. *McKleeven, J et al. Journal of Neuroendocrinlogy (2015)*
5. *Karalis, N. et al Nat. Commun. (2021).*
6. *Maheo et al, bioRxiv (2025)*

P5 - TaqIA polymorphism role in reward-associated disorders: new insights from a humanised murine model.

Fanny BAIN[1], Simon BENOIT[1], Anthony ANSOULT[1], Carlota NUBIOLA[1], Fabien DUCROCQ[2], Asma OUMMADI[2], Dana SMALL[3], Alberto CEBRIAN-SERRANO[4], Pierre TRIFILIEFF[2], Serge LUQUET[1]

1 Université Paris Cité, CNRS, Unité de Biologie Fonctionnelle et Adaptative, Paris, France.
2 Université de Bordeaux, INRAE, Bordeaux INP, NutriNeuro, UMR 1286, Bordeaux, France.
3 Departments of Medicine and Neurology and Neurosurgery, McGill University and Senior Scientist at the McGill University Health Centre, Montreal, Canada.
4 Institute for Diabetes and Obesity, Helmholtz Diabetes Center (HDC) Helmholtz Zentrum München and German, Center for Diabetes Research (DZD), Neuherberg, Germany.

The over-consumption of energy-dense foods is nowadays identified as one of the main causes of obesity. However, there is a substantial body of evidence that obesity and related disorders also result from an interaction between specific genetic polymorphisms and the modern food environment. In this context, the addiction-susceptibility TaqIA polymorphism is of particular interest. TaqIA is a single nucleotide polymorphism causing a change in amino acid in the gene coding for the kinase ankyrin repeat and kinase domain containing 1 (Ankk1) located in the in the vicinity of the dopamine 2 receptor (Drd2) gene. This polymorphism leads to 2 variants: A1 and A2. Approximatively 30% of the world population have one or two copies of the A1 allele. The function of the Annk1 protein remains unclear yet, but the presence of A1 leads is associated with 30% less DRD2 receptors in the striatum as well as an increased vulnerability to neurological and psychiatric disorders, eating disorders and obesity. The A1 allele is ancestral, while the A2 allele appeared more recently with the Homo taxon, impeding the understanding of its causal role in neuropsychiatric disorders.
We have then generated a single-point mutation to humanise mice with the A2 allele. This unique model offers significant translational value, allowing us to study how the A1 and A2 variants affect reward-associated disorders in a developmental context similar to humans.
Motivation of male and female mice of both homozygous for A1 or A2 allele, was assessed in operant cages where reward is a 30s-access to feeders filled with chow food. Females A2/A2 mice significantly increased their number of active presses in a Fixed-Ratio (FR) 1 paradigm compared to their A1/A1 counterparts. However, this effect is lost when the cost to get the reward is higher (FR5) or when the reward is more palatable (high-fat high-sucrose diet). Males A2/A2 significantly increased their number of feeders visits compared to their A1/A1 counterparts despite no differences in active lever press. This phenotype is heightened where the reward is more palatable, and lost when the cost to get the reward is higher (FR5).
These data confirm the importance of the role of the TaqIA polymorphism in reward-related behaviours. Our humanised murine model will then be of great importance to understand its causal role in the susceptibility induced by the TaqIA polymorphism.

P6 - Mechanisms underlying cortical processing of gustatory information

O. Barba-Vila[1], A. Rakotonandrasana[1], A. Thomas[1], M. Tortochot-Megne[1], A. Mastio[2], S. Pascal[1], M. Carta[1]

1. University of Bordeaux, CNRS, IINS, UMR 5297, Bordeaux, France
2. University of Cagliari, Department of Biomedical Sciences, Section of Neuroscience and Clinical Pharmacology, Cagliari, Italy

The primary function of the sensory cortex is to analyze incoming stimuli from the periphery, distinguishing and interpreting them to guide behavior. Among sensory stimuli, gustatory information enables animals to analyze food identity and recognize the nutritional and hedonic values (e.g., palatable as sucrose, versus aversive as citric acid) to avoid poisons and regulate feeding behaviors, making it crucial for survival. Despite the importance of gustatory processing, our knowledge of the underlying neuronal circuits and their activity remains remarkably incomplete, especially compared to other sensory modalities (e.g., somatosensation, vision, olfaction, and audition). Here, we employ a combination of slice electrophysiology, optogenetics, and activity reporters to characterize the morphology and electrophysiological properties of layer 5 pyramidal neurons in the gustatory cortex, together with their synaptic inputs from the gustatory thalamus (which provides information regarding tastant identity) and amygdala (which provides information regarding palatability). We aim to disentangle how gustatory information is integrated and processed in the mouse gustatory cortex at the synaptic and circuit levels.

P7 - The role of addiction-susceptibility polymorphism TaqIA in metabolism and metabolic-associated disorders.

Simon BENOIT[1], Fanny BAIN[1], Anthony ANSOULT[1], Carlota NUBIOLA[1], Alabama DUBURG[1], Fabien DUCROCQ[2], Asma OUMMADI[2], Dana SMALL[3], Alberto CEBRIAN-SERRANO[4], Pierre TRIFILIEFF[2], Serge LUQUET[1]

1 Université Paris Cité, CNRS, Unité de Biologie Fonctionnelle et Adaptative, Paris, France.
2 Université de Bordeaux, INRAE, Bordeaux INP, NutriNeuro, UMR 1286, Bordeaux, France.
3 Departments of Medicine and Neurology and Neurosurgery, McGill University and Senior Scientist at the McGill University Health Centre, Montreal, Canada.
4 Institute for Diabetes and Obesity, Helmholtz Diabetes Center (HDC) Helmholtz Zentrum München and German, Center for Diabetes Research (DZD), Neuherberg, Germany.

While the over-consumption of energy-dense foods is now clearly identified as one of the main causes of obesity, there is a large body of evidence supporting that the development of obesity and related disorders is also the result of an interaction between specific genetic polymorphisms and the modern food environment. In this context, the addiction-susceptibility TaqIA polymorphism is of particular importance. TaqIA is a single nucleotide polymorphism located in the Annk1 gene, leading to two alleles, A1 and A2. Approximatively 30% of the world population has one or two copies of the A1 allele. The presence of this allele increases the likelihood of developing neurological and psychiatric disorders, as well as an increased risk of obesity, indicating a role for this gene in energy homeostasis. While the A1 allele is the ancestral variant, the derived A2 variant has appeared with the Homo taxon, impeding the understanding of its causal role in neuropsychiatric and metabolic disorders. We have then generated a single point mutation to humanise mice for the A2 allele. This unique model brings strong translational value and allows us to profile how A1 and A2 variants affect energy homeostasis in a context of developmental expression of the mutation, similar to humans. Metabolism of male and female mice of both A1/A1 and A2/A2 genetic backgrounds was assessed in indirect calorimetry cages. Interestingly, only A2/A2 males displayed higher energy expenditure under both chow and habituation to a high-fat diet conditions compared to their A1/A1 counterparts, though a longer exposition to the high-fat diet makes this difference disappear. A2/A2 male mice also displayed better glucose regulation than A1/A1, maintaining similar glycaemic responses after the high-fat diet exposure. No such differences were observed in female mice. Metabolism of female mice was then assessed in different thermal conditions: room temperature, thermal neutrality and cold stress. Strikingly, A2/A2 female mice displayed higher energy expenditure under a cold stress, compensated by a higher energy intake. Altogether, these data confirm the importance of the role of the TaqIA polymorphism in the regulation of energy homeostasis, and underscore a sexual dimorphism in this context.

P8 – Dopamine D2R-neurons of the Paraventricular Thalamus Govern Feeding, Energy Balance and Body Homeostasis

Benoit Bertrand[1], Eléonore Hardy[2], Nejmeh Mashhour[1], Julien Castel[1], Oriane Onimus[1], Serge Luquet[1], Emmanuel Valjent[3], Marco Diana[2], Giuseppe Gangarossa[1]

1 Université Paris Cité, Unité de Biologie Fonctionnelle et Adaptative, 4 rue Marie-Andrée Lagroua Weill-Hallé, 75013, Paris
2 Université Paris Cité, Saints-Pères Paris Institute for the Neurosciences, 45 rue des Saints-Pères, 75006, Paris
3. Université de Montpellier, Institut de Génomique Fonctionnelle, 141 Rue de la Cardonille, 34094, Montpelier

The central control of feeding behavior has been classically ascribed to the hypothalamus, but recent studies suggest that extra-hypothalamic structures could modulate feeding behavior. The paraventricular thalamus (PVT) is a midline structure located above the hypothalamus. It is described as an interface between limbic structures which integrate and sends multiple signals in order to regulate emotional behavior and/or cognitive process. Despite many connections with the hypothalamus and other satiety nuclei (PBN and NTS), PVT is still poorly understood in feeding behavior. Here, by taking advantage of cell-type specific strategies via viral approaches, whole cell patch clamp, fiber photometry and indirect calorimetry methods, we found that PVT and notably, the neurons expressing the dopamine D2 receptor (D2R) control food intake, energy balance and body homeostasis in both physiological and obesogenic contexts. Unraveling extra-hypothalamic brain circuits, such as PVTD2R neurons, may pave the way to new insights and may provide new therapeutics solutions to fight obesity dysfunctions or metabolic disorders.

P9 - Uncovering the neuro-immune mechanisms driving the “memory of obesity”

Adèle Buckenmeyer[1], Philippe Zizzari[1], Samantha Clark[1], Fatiha Merabtene[2], Evangelia Kyriakidou[1], Stéphane Léon[1], Nathalie Dupuy[1], Frédéric Martins[1], Thierry Leste-Lasserre[1], Alexandre Brochard[1], Agnès Nadjar[1], Carmelo Quarta[1], Isabelle Dugail[2], Daniela Cota[1]

1 INSERM, Neurocentre Magendie, U1215, 33000 Bordeaux, France.
2 INSERM UMRS 1269, Sorbonne Université, NutriOmics Team, 75013 Paris, France

Weight regain after weight loss represents a medical challenge for the long-term treatment of obesity. We have recently established (*Léon S et al., Diabetes 2025*) a dietary mouse model characterized by persistently higher body weight set-point and altered metabolic responses after an episode of diet-induced obesity. We have hypothesized that these lasting changes may be related to modifications in the hypothalamus-adipose tissue axis involving changes in immune cells (microglia in the brain, macrophages in the adipose tissue) and sympathetic nervous system (SNS) functions.
Initial immunostaining analysis of different adipose tissue depots for the tyrosine hydrolase (TH) has confirmed alteration of the SNS, particularly at the level of the subcutaneous adipose tissue. We therefore tested whether the inhibition of the EPAC pathway, which is supposed to re-establish SNS function in adipose tissue (*Valentine et al., J Clin Invest 2022*) can correct the body weight set-point in previously obese mice. Our data show that this treatment significantly reduces body weight in our model. Molecular analyses in adipose tissue and hypothalamus are currently ongoing to define changes in pathways of interest. Additionally, by carrying out bulk RNAseq in both brain microglia and adipose tissue macrophages, we found molecular signatures that persist altered in previously obese mice. Additional studies are currently ongoing to explore the role of microglia in the modification of the body weight set-point and in the alteration of SNS function. These set of findings, altogether provide novel information on the modification of the body weight set-point after an episode of obesity.

P10 - Leveraging linear models to correct interoceptive tuning for motion related activity

Ella Callas[1], Karim Benchenane[1], Sophie Bagur[1]

1 Emotions, Memory, Oscillations and Brain-Body states team – Karim Benchenane – ESPCI Paris

The correlation between emotions and bodily state has long been established, and feedback from the body actively contributes to affective behaviors. Recent studies in mice have provided direct evidence that manipulating respiratory[1] (Bagur 2021) or cardiac[2,3] (Hsueh 2023, Klein 2021) feedback modifies fear behavior. The interoceptive system is thought to process these afferent visceral signals and integrate them into affective brain-body interactions. However, the precise neural encoding of interoceptive variables like heart or respiratory rate remains unknown. This may be due to the difficulty of evaluating the specific contribution of interoceptive variables to neural activity given their correlation with each other and behavioral variables. Computational approaches can help disentangle the contribution of each parameter.

In this work, we develop a framework to extract interoceptive tuning while correcting for motion biases. The data consists of prefrontal single unit activity (n=317) recorded simultaneously with motion (position, speed, head motion) and interoceptive (heart and breathing rate) variables in freely moving mice. Using Generalized Linear Models, we quantify the amount of variance explained by each measured variable as a proxy for neural sensitivity. Heart and breathing rate explain respectively 2,5 and 2% of the variance on average. Each motion variable explains around 2% of the variance, in agreement with previous studies[4,5]. This shows that interoceptive variables contribute with similar importance as motion variables to prefrontal coding. We then compute interoceptive tuning curves, defined as the firing rate of each neuron in response to specific ranges of heart or breathing rates. We observe that 35% and 45% of units are significantly tuned to heart and respiratory rate respectively. These curves shed light on the precision of single neuron interoceptive encoding. Using the motion related coefficients extracted by the linear model, we correct the firing rate of each unit for the contribution of motion activity. This yields movement corrected tuning curves. Interestingly, we find that in some units, the motion correction abolished the apparent tuning calculated from the uncorrected data, while preserving others intact. This work sheds light onto the contribution of interoceptive variables to prefrontal coding and shows the importance of correcting for misleading correlations to specifically address interoceptive related activity.

1. Bagur, S. et al. Nat. Commun. (2021).
2. Hsueh, B. et al. Nature (2023).
3. Klein, A. S., Dolensek, N., Weiand, C. & Gogolla, N. (2021).
4. Vogel, P., Hahn, J., Duvarci, S. & Sigurdsson, T. Cell Rep. (2022).
5. Brünner, H. et al. iScience (2024).

P11 - Lateral habenula as a novel mechanism regulating systemic immune responses

Silvia Castany[1] , Priscila Batista da Rosa[1], Olga Barba Vila[1], Joost Wiskerke[1], David Engblom[1]

1 Center for Social and Affective Neuroscience (CSAN), Department of Clinical and Experimental Medicine, Linköping University

Mood can have a great impact on our physical health. In our everyday life, stressful events increase our chances of becoming sick. In clinical settings, patients suffering from mood disorders such as depression have increased risk of developing comorbid inflammatory diseases, such as arthritis, diabetes, autoimmune diseases. This strongly indicates that depressed patients may have an underlying dysfunctional immune system. Understanding how brain specifically modulate the peripheral immune system in the context of depression is scarcely explore. My research is aimed at understanding how, and which brain regions can modulate systemic inflammation and peripheral immunity during affective disorders. I particularly focus on the role of the lateral habenula (Lhb), a brain region that is critical for encoding and integrating aversive signaling. Dysregulation of Lhb circuitry has been proposed to mediate depressive symptomatology in humans and rodents. Therefore, my hypothesis is that increased neuronal activity in Lhb can lead to maladaptive changes in the peripheral immunity. To address this, I used an optogenetic approach to selectively activate Lhb neurons and then evaluated the immune system function by triggering an immune challenge with LPS. Repeated optogenetic activation of Lhb induced a negative affective state in both male and female mice. It also potentiated the inflammatory response to LPS in females while dampening it in males. To understand the peripheral mechanism, we tested whether the sympathetic nervous system was involved. Sympathectomy completely blocked the increased inflammatory response to LPS in females. This data indicates that persistent activation of Lhb can induce maladaptive immune responses in a sex-specific manner. Overall, this study provides a novel neuronal mechanism regulating systemic immune responses during negative affective states.

P12 - Dopamine transmission in the anterior insula shapes the neural coding of anxiety

Yoni Couderc[1], Tanmai Dhani Reddy[1], Archi Garg[1], Giovanni Vardiero[1], Daria Ricci[1], Marion d'Almeida[1], Céline Nicolas[1], Tina Habchi[1], Camille Penet[1], Victor Le Paih[1], Yue K. Wu[2], Julijana Gjorgjieva[2], Yulong Li[3], Emmanuel Valjent[4] and Anna Beyeler[1]

1 Neurocentre Magendie, INSERM 1215, Université de Bordeaux, France
2 School of Life Sciences, Technical University of Munich, Freising, Germany
3 State Key Laboratory of Membrane Biology, Peking University School of Life Sciences, China
4 IGF, Université Montpellier, CNRS, INSERM, Montpellier 34094, France

Anxiety is an adaptive response of individuals exposed to potentially dangerous contexts. However, anxiety can persist at elevated levels regardless of the environment, becoming pathological (Belzung & Griebel, 2001). Although anxiety disorders are the most prevalent psychiatric conditions, their underlying neurobiology remain poorly understood. Numerous studies have revealed the involvement of various neuromodulators, including dopamine, in the regulation of anxiety (de la Mora et al., 2010; Hjorth et al., 2021). Concurrently, imaging studies have shown that the insular cortex (insula), particularly its anterior part, is hyperactivated in individuals with anxiety disorders, and preclinical studies showed it has anxiogenic properties (Buff et al., 2016; Nicolas et al., 2023). While it is established that dopaminergic transmission regulates anxiety levels, its specific impact on the insula remains elusive. This work explores how dopaminergic transmission within the anterior insula regulates anxiety processing.

First, we revealed a higher density of neurons expressing the type 1-like dopamine receptor (D1) in the anterior compared to the posterior insula, with a density seven times greater than those expressing dopamine type 2-like receptor (D2). Furthermore, we observed that only a few neurons coexpress D1 and D2 receptors in the anterior and posterior insula. Finally, we found that D1 neuron projection pattern closely resembled those of the overall glutamatergic population.

Second, we observed a specific increase in dopamine release within the anterior compared to the posterior insula when mice are located in exposed areas. Interestingly, while dopamine release onto both D1 and D2 neurons increased in the anterior insula when mice are in exposed areas, the amplitude of this release was positively correlated with anxiety levels only in the D1 population. Finally, we pharmacologically demonstrated that D1 signaling in the anterior insula bidirectionally regulates anxiety levels, establishing a direct causal link between dopaminergic signaling in the anterior insula and anxiety.

Finally, we examined individual neuron correlates of anxiety in the anterior insula. Using deep-learning tools, we identified distinct coding profiles when mice were in exposed or safe zones. Notably, D1 activation increased anxiety levels and concomitantly sharpened the coding specificity for exposed areas as anxiety increased.

To conclude, our findings provide a new model of neural population coding of anxiety, unravelling a D1-dependent coding mechanism of anxiety in the mouse anterior insula.

P13 - Environmental Enrichment Modulates HPA Axis Reactivity and Anxiety in a Chronic Stress Model: Adaptive or Overloaded?

De-Gusmão, N.[1,2]; Costa, G.A.[1]; Almeida, M.G. [1]; Mariano, P. [1]; Belo-Silva, A.E. [1]; Da-Rovare, V.P[1]; Rae, M.B[1]; Eichler, R.A.S. [1]; Chivers, P. [3]; Bailey, A[4]; Camarini, R[1].

[1]*Pharmacology Department, University of São Paulo, São Paulo, Brazil.*
[2]*Team Memory, Oscillations and Brain States (MOBs), ESPCI, Paris, France.*
[3]*Pharmacology Section, City St George's University of London, London, United Kingdom.*
[4]*School of Biosciences & Medicine, University of Surrey, Guildford, United Kingdom*

Chronic stress is a major risk factor for anxiety disorders[1] and disrupts neuroimmune balance through HPA axis hyperactivation[2,3] and elevated glucocorticoids, such as corticosterone (CORT) in rodents[4,5]. This dysregulation affects brain-body communication, promoting neuroinflammatory responses[6,7]. Environmental enrichment (EE) has been shown to buffer these effects, enhancing stress resilience[8,9,10]. Exploring how EE shapes neuroimmune outcomes under stress may reveal links between psychological stress and immune dysfunction in anxiety. For this, *Swiss* male mice were assigned to one of two environmental conditions: non-enriched (NE) and enriched (EE). On PND70, mice were either exposed to or spared from Chronic Unpredictable Mild Stress (CMS), creating 4 groups: (1) NENS: non-enriched and non-stressed; (2) NEST: non-enriched and stressed; (3) EENS: enriched and non-stressed; and (4) EEST: enriched and stressed. Mice were subjected to Open Field (OF), Elevated-Plus Maze (EPM) and Light-Dark Box (LDB) tests to evaluate anxiety-related behaviors. To assess HPA axis activity, CORT was measured at 3 time points: post-acclimation, after 21 days of EE, and 24 h after the last stress. On both OF and LDB, EE reduced locomotor activity, irrespective of stress exposure. On the EPM, EEST mice exhibited anxiety-like behavior, suggesting adaptive vigilance and nuanced behavioral response to stressors. EE alone initially did not alter CORT, indicating no early HPA axis modulation. However, extended EE significantly increased CORT, an effect not observed in EEST animals, suggesting that CMS blunted this increase. While prolonged HPA axis activation is often linked to neuroimmune disruption, elevated CORT in EE mice may reflect adaptive responses to complex stimuli rather than dysfunction. The increased anxiety in EEST mice may indicate functional coping rather than a pathological response. This decoupling between behavior and endocrine reactivity may signal a buffered stress response, characteristic of resilience, rather than vulnerability. Overall, our data suggest that EE promotes resilience via adaptive mechanisms involving the neuroimmune and HPA axis interplay. These results highlight the neuroendocrine systems' role in brain-body stress regulation.

[1]Hammels et al., 2015; [2]McEwen B.S., 2007; [3]Santos-Rocha et al., 2018; [4]Adams et al., 2003; [5]Heim et al., 2008; [6]Hassamal, 2023; [7]Peng et al., 2022; [8]Lehmann & Herkenham, 2011; [9]Novaes et al., 2017; [10]Zanca et al., 2015.

P14 - Chemogenetic manipulation of insular cortex projections to the ventral striatum attenuates obesogenic diet-induced anxiety like behaviours

Eva-Gunnel Ducourneau[1], A. Boutin[1], L. Pages[2], A. Beyeler[2], G. Ferreira[1]

1 *Laboratoire NutriNeuro, UMR 1286 INRAE, University of Bordeaux, Bordeaux, France*
2 *Neurocentre Magendie, U1215 INSERM, University of Bordeaux, Bordeaux, France*

Obesity, mainly due to excessive consumption of energy-dense food, is a risk factor for cardiometabolic disorders but also to brain dysfunctions with a higher prevalence of anxiety disorders compared to the general population. Clinical studies revealed that obesity and anxiety disorders are both associated with insular cortex dysfunctions. Similar to humans, rodent models of obesity induced by chronic high-fat high-sugar diet (HFSD) intake exhibit enhanced anxiety-like behaviours and using *in vivo* calcium imaging our preliminary results indicate that the neuronal activity of anterior insular cortex is higher in anxiogenic environments in HFSD-fed mice. However, it is not yet clear whether insula dysfunction causally contributes to HFSD-induced anxiety. We therefore use chemogenetic strategies to decrease insular activity and evaluate the effect on anxiety-like behaviours. Anxiety-like behaviours of adult mice, exposed to either standard diet or HFSD for 12 weeks, were assessed using elevated plus-maze, open-field, light-dark task and novelty suppressed feeding test (NSFT). Our results indicate that decreasing activity of principal neurons in the anterior insular cortex normalized anxiety-like behaviours in HFSD-fed mice, and strikingly so in NSFT. Additional controls indicate a specific effect on anxiety-like behaviour and not on motivation to eat in NSFT. As ventral striatum dysfunction mediates anxio-depressive behaviours in HFSD-fed mice, we then used an intersectional viral strategy to chemogenetically inactivate projections from insular cortex to ventral striatum. Our very preliminary data indicate that decreasing activity in this pathway also normalized anxiety-like behaviours in HFSD-fed mice. Our results indicate that insular circuit-specific neuronal populations display an increased activity after HFSD, which causally increases the level of anxiety.

P15 - Olfaction and feeding behaviour: neuronal substrates underlying odour modulation of neuronal circuits regulating food intake

Louise Eygret[1], Morgane Martin[1], Alexandra Sere[1], Farha Bouteldja[1], Vincent Simon[2], Daniela Cota[2], Xavier Fioramonti[1], David Jarriault[1]

[1] *NutriNeuro (Univ. Bordeaux, Bordeaux INP, INRAE UMR 1286), Bordeaux, France*
[2] *Neurocentre Magendie, Bordeaux, France*

A growing part of the population is aging. One of the major health concerns in elderly is decreased-food intake-associated malnutrition which can accelerate cognitive decline. Increasing feeding behaviour is a pertinent approach which could significantly improve cognitive capacities of elderly. The olfactory system plays a key role in nutrition and could offer a non-invasive approach for stimulating feeding. Food intake is mainly controlled by hypothalamic AgRP (orexigenic) and POMC (anorexigenic) neurons. We hypothesized that food-related odorant molecules modulate the activity of these hypothalamic neurons to increase food intake.
We identified diverse food-related odorant molecules which show innate attractivity in both male and female mice. Among them, bacon odour revealed a potent appetitive effect in increasing food intake measured using automated food intake monitoring experiments. Patch-clamp electrophysiological recordings from brain slices of mice exposed to bacon odour during 30 min show that it induces decreased firing of POMC neurons. No effect was observed on AgRP neuron activity despite changes in synaptic inputs. In vivo fiber photometry recordings show that bacon odour induces a rapid decrease in AgRP neuron activity and an increased activity of POMC neurons during active sniffing of this odorant.
These results replicated with other odorants suggest that attractive food-related odours modify the electrical activity of neurons within the melanocortin network. Future work will map the olfactory projections to arcuate nucleus and will investigate the efficiency of odorant stimulation in aged animals on food intake and cognitive capacities.

P16 - Lack of Single Amino Acids Transcriptionally Modulates Sensory Systems to Enhance Microbiota Intake

Ezra-Nevo, G[1,4]*, Henriques, SF[1,4], Münch, D[1,3], Francisco AP[1], Baltazar, C[1], Elias, A-P[1], Deplancke B[2], and Ribeiro, C[1]*

1 Behavior and Metabolism Laboratory, Champalimaud Foundation, Lisbon, Portugal.
2 Laboratory of Systems Biology and Genetics, Institute of Bioengineering, School of Life Sciences, Ecole Polytechnique Fédérale de Lausanne, Lausanne, Switzerland.
3 Institute of Animal Physiology, Justus-Liebig-University, Giessen, Germany
4 Authors contributed equally

* Author of correspondence: Gili.Ezra@research.fchampalimaud.org, Carlos.Ribeiro@neuro.fchampalimaud.org.

Adequate intake of dietary essential amino acids (eAAs) is critical for protein synthesis and metabolic health. In *Drosophila melanogaster*, deprivation of any single eAA drives a compensatory increase in protein appetite. However, the neural, metabolic and sensory mechanisms that translate internal nutrient needs into targeted foraging behaviors remain poorly understood.
To investigate this transformation from metabolic "need" to behavioral "want", we generated transcriptomic profiles from the heads of flies subjected to individual eAA deprivations. While each condition elicited a distinct gene expression signature, we identified a core set of genes consistently regulated across multiple deprivation states. Among these, the ionotropic olfactory receptor *Ir76a* was consistently upregulated across all deprivation conditions.
Functional imaging and genetic manipulation revealed that *Ir76a* is essential for the exploitation of gut-associated bacteria, which improves host fitness during eAA scarcity. These findings uncover a direct role for olfactory plasticity in nutrient-specific foraging behavior and demonstrate that microbiota ingestion is an integral component of dietary adaptation.
Together, our work reveals how internal nutrient status reshapes olfactory functions to guide the search for ecologically relevant nutrient sources. This establishes a mechanistic framework for understanding how sensory circuits integrate internal metabolic signals to drive adaptive feeding decisions.

P17 - Sex-specific metabolic and behavioral effects of ketogenic diet in a neuromelanin-based mouse model of prodromal Parkinson's disease

Gatti, Sabrina Ayelén[1]; Godfried Nachtigall, Eduarda [1]; Vila, Miquel [1,2,3,4]; Laguna, Ariadna[1,2]

[1] *Neurodegenerative Diseases Research Group, Vall d'Hebron Research Institute (VHIR)-Network Center for Biomedical Research in Neurodegenerative Diseases (CIBERNED), 08035 Barcelona, Spain.*
[2] *Aligning Science Across Parkinson's (ASAP) Collaborative Research Network, Chevy Chase, MD 20815, USA.*
[3] *Department of Biochemistry and Molecular Biology, Autonomous University of Barcelona, 08193 Barcelona, Spain.*
[4] *Catalan Institution for Research and Advanced Studies (ICREA), 08010 Barcelona, Spain.*

Parkinson's disease (PD) is the second most common neurodegenerative disorder, with prevalence increasing with age. While motor symptoms typically appear after 40–60% neuronal loss in the substantia nigra, non-motor symptoms, particularly gastrointestinal dysfunction, often precede them and are considered prodromal features of the disease. Our group has developed a novel transgenic mouse model of PD (tgNM), which overexpresses the tyrosinase enzyme gene under the tyrosine hydroxylase promoter, leading to neuromelanin (NM) accumulation in all catecholaminergic brain regions, as occurs in the human brain. NM buildup induces neurodegeneration affecting dopaminergic, noradrenergic, and cholinergic systems, resulting in both motor and non-motor symptoms. Disease progression in this model is defined by age as preclinical (3 months), prodromal (10 months), and early PD (18 months) stages[1]. Given recent evidence supporting ketogenic interventions as a promising non-pharmacological approach to neurodegenerative diseases[2], we evaluated sex-specific effects of a ketogenic diet (KD; 90% lipids—20% MCTs, 10% protein, 0% carbohydrates) administered for 5 months to 8-month-old tgNM mice. Results showed that tgNM males achieved deep nutritional ketosis, with elevated ketones and reduced glucose levels, while tgNM females reached only mild ketosis. They also presented different patterns of feeding regulation (water, food and calorie intake) affecting body weight. As a result of dietary adaptation, tgNM males developed a hypertrophic liver and a hypotrophic pancreas, while tgNM females exhibited the opposite pattern. Adipose tissue analysis revealed brown fat accumulation in tgNM males and white fat, in tgNM females. TgNM mice showed a trend to increase intestinal weight and reduced cecum and colon weight. Functionally, KD reduced the observed increase in intestinal permeability of tgNM mice without altering total transit time, fecal water content, or fecal output. Anxiety- and depression-like behaviors remained unchanged, but cognition improved, particularly in males. No improvements in general motor symptoms were observed. Notably, KD increased the number of vocalizations, a relevant finding since vocal impairment is common in PD. These findings highlight sex-dependent metabolic responses to KD and support the importance of personalized nutritional strategies in neurodegenerative diseases.

1 Laguna, A. et al. Modelling human neuronal catecholaminergic pigmentation in rodents recapitulates age-related neurodegenerative deficits. Nat Commun (2024)
2 Gough SM, et al. Neuroprotection by the Ketogenic Diet: Evidence and Controversies. Frontiers in Nutrition (2021).

P18 - The role of lipid metabolism and circulating miRNAs in the intergenerational transmission of the effects of parental adverse childhood experiences

Magdalena Gomółka[1,3], Weronika Tomaszewska[1,3], Sandra Binias[2], Regina Nadalinska[1], Ali Jawaid[1]

[1] Translational Neuropsychiatry Research Group (TREND Lab) Life Sciences & Biotechnology Center Łukasiewicz Research Network PORT – Polish Centre of Technology Development, Wrocław, Poland
[2] Laboratory of Sequencing, Nencki Institute of Experimental Biology PAS, Warsaw, Poland
[3] Nencki Institute of Experimental Biology PAS, Warsaw, Poland

Introduction: Childhood trauma is an important risk factor for psychiatric and physical ailments during adulthood. Emerging evidence suggests that some of its behavioural and metabolic symptoms are transmissible across generations. Intergenerational transmission of the effects of trauma is postulated to involve changes in germline non-coding RNAs. However, it is unclear how childhood trauma affects ncRNAs in the gametes. Circulating ncRNAs, such as miRNAs, majorly carried by lipid-associated factors in the body fluids, appear as important candidates for carrying the trauma effects to the gametes for intergenerational transmission.
Aim(s): Synergizing investigation in a mouse model of ACE induced via unpredictable maternal separation and unpredictable maternal stress (MSUS) and cross-injection studies, we hypothesize that lipid-associated miRNAs communicate the effects of ACE to the germline for intergenerational transmission.
Method(s): Intergenerational behavioral and metabolic phenotyping was performed, supplemented with small RNA sequencing followed by qPCR. Cross-injections of lipid-associated carriers into the tail vein of mice performed.
Results: Offspring of both MSUS- and HFD-exposed male mice showed impaired glucose tolerance, depressive-like behaviour and anxiety. Cross-injections from MSUS into CTRL mice prolonged the offspring latency to enter open arms in Elevated Plus Maze test. Cross-injections from MSUS into CTRL mice recapitulated the offspring metabolic phenotype associated with MSUS in Glucose tolerance test. Cross-injections from VE mice into MSUS mice partially mitigated the metabolic MSUS phenotype.
Conclusions: Injections of MSUS-material is sufficient and necessary to induce the intergenerational metabolic phenotype associated with MSUS while lipid-modifying interventions can potentially alter the intergenerational metabolic MSUS phenotype. This research provides proof-of-concept for a role of lipids and circulating miRNAs in communicating the effects of ACE to the germline for intergenerational sequelae.

P19 - Miniature endoscope for high resolution electrical and optical investigation of the colon in live mice

Arielle L. Planchette[1], Aleksander Sobolewski[1], Yoseline Cabara[1], Karol Wojcicki[1], Tim Hibberd[2], Lee Travis[2] Nick Spencer[3], Michel Neunlist[3], **Michalina Gora[1]**

[1]*Wyss Center for Bio and Neuroengineering, Geneva, Switzerland*
[2] *Flinders Health & Medical Research Institute, Flinders University, Adelaide, Australia*
[3]*Nantes Université, CHU Nantes, INSERM, TENS, IMAD, Nantes, France*

To address the need for in-vivo, longitudinal investigation of the functional and morphological status of the colon in preclinical research to advance our understanding of disorders of the gut-brain interaction we have developed a novel multimodal mini-endoscope for use in anesthetized small animals.
The 2mm endoscope can be inserted up to 3.5 cm into the colon of an anesthetized mouse. It has 128-channel electrode array for high-resolution electrophysiological recording from the luminal side. An inner channel allows for insertion of tools, e.g. fiber probes for Optical Coherence Tomography (OCT) imaging[1] of morphology or for neuronal optogenetic activation.
The safety and feasibility of mini-endoscopy to collect multimodal data has been so far confirmed in 30 normal mice and 3 mice with BAC-lesioned[2] colonic tissue. In vivo recordings revealed smooth muscle calcium action potentials orchestrated into complex spatiotemporal patterns, which could be modulated by intraperitoneal injections of drugs (e.g. donepezil). Morphology was imaged in-vivo with 20-microns resolution OCT visualizing changes of the gut's layers from the distal colon to rectum. After in-vivo recordings the mice were perfused the colon was collected for tissue clearing and targeted fluorescence microscopy. The origin of signals was confirmed in ex-vivo colon preparations[3] where tetrodotoxin (TTX) completely disrupted the spontaneously occurring waves. Similarly, both in-vivo functional and morphological recordings were altered following BAC treatment known to disrupt the enteric nervous system.
We believe this novel multimodal endoscopic approach represents a significant improvement over current standards (e.g. fecal pellet output). We hope that the capabilities of this device will enhance our understanding of gut function in the context of various diseases, as well as its modulation by nutrients, the microbiome, or pharmacological agents.

[1]Gora et al. Tethered capsule endomicroscopy enables less invasive imaging of gastrointestinal tract microstructure. Nat Med. 2013
[2]Qin et al. Benzalkonium chloride–treated anorectums mimicked endothelin-3–deficient aganglionic anorectums on manometry. *Journal of Pediatric Surgery*, 2010
[3]Costa et al. Characterization of alternating neurogenic motor patterns in mouse colon. *Neurogastroenterology & Motility*, 2021

P20 - Induced Neural Responses to Respiration-Synchronized Olfactory Stimuli in Human EEG

Alisa Hathaway[1], Prof. Todd Coleman[2]

1 Electrical Engineering Department, Stanford University
2 Bioengineering Department, Stanford University

This study introduces a novel, cost-effective olfactometer device that uses a nasal sensor to deliver olfactory stimuli synchronized with the natural respiration cycle. Using simultaneous electroencephalography (EEG) recordings, we assess the brain's response to olfaction without bio-behavioral paradigms that are (i) prone to within-subject variation on repeated testing and (ii) dependent upon cognitive, literary, and/or motor control abilities. Testing was conducted on subjects (N = 17) using a 128-channel Electrical Geodesics Inc EEG system. Subjects were presented with scented (peppermint, oregano, or citrus) or non-scented control (water) stimuli.

After standard EEG preprocessing, results demonstrate significant coupling between the phase of natural respiration and EEG amplitude in frontal regions within canonical frequency bands. This respiration–EEG phase–amplitude coupling (PAC) was validated against phase-shuffled null distributions, replicating prior findings that respiration can entrain cortical oscillations (Zelano et al., 2016). We then assessed condition-specific modulation of PAC by comparing each odor to the control using two-sided permutation tests. PAC was significantly enhanced in the lower gamma band (30–55 Hz) and significantly diminished in the alpha band (8-12Hz) during odor presentation compared to control (FDR-corrected $p < 0.001$), indicating that olfactory input modulates respiration–EEG coupling in certain frequency bands. To further characterize neural responses to odor, we applied mixed linear effects models independently at each time point to EEG data filtered in the lower gamma band. To isolate induced (non–phase-locked) activity, we extracted the amplitude envelope using the Hilbert transform applied to epoched EEG signals. This time-resolved modeling approach incorporated odor condition as a fixed effect and subject as a random effect, increasing sensitivity to subtle, temporally dynamic effects. This approach is consistent with prior literature implicating gamma oscillations in olfactory perceptual binding and integrative processing, suggesting that these signals reflect induced rather than purely evoked neural activity (Tallon-Baudry, 1999).

This olfactometer was designed with cost efficiency and accessibility for researchers in mind, focusing on odor presentation synchronized with a subject's natural respiration. Functionality was demonstrated at a reduced cost compared to all existing research and commercial olfactometers, allowing for application across a range of clinical and experimental contexts.

1. Tallon-Baudry et al., "Oscillatory gamma activity in humans and its role in object representation." Trends in Cognitive Sciences, 1999
2. Zelano et al., "Nasal Respiration Entrains Human Limbic Oscillations and Modulates Cognitive Function." Journal of Neuroscience, 2016

P21 - Nicotine-induced immobility in mice reveals a coordinated brain-body recovery state post-stress

Chloé Hayhurst[1], Baptiste Mahéo[1], Karim Benchenane[1]

1 eMOBBS team, Brain Plasticity Unit, ESPCI, Paris, France

Understanding how brain–body systems transition from stress to recovery is crucial to investigate adaptive defensive behaviors. Here, we characterize the physiological and neural signatures of an immobility state induced by an anxiogenic dose of nicotine in mice (0.5 mg/kg IP)[1]. We administered nicotine in both home-cage and open-field contexts. Following injection, mice exhibited prolonged immobility and clear thigmotaxis behavior in the open field. During immobility, respiratory rate slowed to 2–3 Hz, and heart rate variability markedly increased. Concurrent hippocampal recordings revealed abundant sharp-wave ripples (SWRs). This coordinated pattern - slow breathing, high heart rate variability, and SWRs - unfolded within minutes of nicotine injection and closely mirrored the recovery immobility previously identified after acute stress[2] and classical freezing associated with 4–6 Hz breathing.

These findings suggest that nicotine can be used as a fast and replicable tool to probe recovery dynamics. This work may provide a new window into the temporal structure of defensive behavior and its resolution, bridging brain rhythms, autonomic physiology, and behaviour.

1. Nguyen C, Mondoloni S, Le Borgne T, Centeno I, Come M, Jehl J, Solié C, Reynolds LM, Durand-de Cuttoli R, Tolu S, Valverde S, Didienne S, Hannesse B, Fiancette JF, Pons S, Maskos U, Deroche-Gamonet V, Dalkara D, Hardelin JP, Mourot A, Marti F, Faure P. Nicotine inhibits the VTA-to-amygdala dopamine pathway to promote anxiety. Neuron. 2021 Aug 18
2. Mahéo B, Bagur S, Bryzgalov D, Hayhurst C, Chouvaeff M, Callas E, Schmidt C, Gallopin T, Benchenane K. Hippocampal reactivation of aversive experience enables safety learning and slow-breathing state for recovery from stress. BioRxiv. 2025 June 03

P22 – The Effect of Sleep Deprivation and Nocturnal Light Exposure on Recovery Sleep and Morning Physiological Markers

Jana Kopřivová[1,4], Kateřina Skálová[1,2], Jan Maleček[3], David Kolář[1], Kateřina Červená[1], James Tufano[3], Dan Omcirk[3], Jan Padecký[3], Tomas Vetrovsky[3], Marek Piorecký[1], Martin Brunovský[1,4], Zdeňka Bendová[1,2,4]

1 National Institute of Mental Health, Klecany, Czech Republic
2 Faculty of Science, Charles University, Prague, Czech Republic
3 Faculty of Physical Education and Sport, Charles University, Prague, Czech Republic
4 Third Faculty of Medicine, Charles University, Prague, Czech Republic

Sleep deprivation increases homeostatic pressure for sleep, whereas nocturnal light exposure may disrupt circadian rhythms and affect sleep architecture and other physiological processes. The aim of this study was to compare macrostructural parameters of recovery sleep following sleep deprivation in darkness and under light, and to examine their relationship with morning melatonin and glucose levels. Twelve healthy adults participated in a within-subject protocol conducted in a sleep laboratory, consisting of two experimental blocks. Each block included a baseline night, one night of sleep deprivation (either in light or in darkness), and a subsequent recovery night. Sleep was recorded using polysomnography during the baseline and recovery nights. Sleep stages (NREM1–3, REM, wake) were scored according to standard international criteria. Sleep latency, total sleep time, sleep efficiency, and the proportion of each sleep stage were compared across the four conditions using Friedman tests. Salivary samples were collected at 07:00 following each recovery night to analyze morning melatonin and glucose concentrations. Ongoing analyses include sleep microstructure, covering markers of homeostatic sleep pressure (amount and dynamics of slow-wave EEG activity during NREM sleep), arousal (high/low frequency EEG activity during NREM sleep), and autonomic function (heart rate variability).
Both light and dark sleep deprivation conditions led to increased total sleep time, reduced proportion of light sleep (NREM1), and increased deep sleep (NREM3) during the recovery night. The increase in NREM3 was more pronounced after deprivation in darkness. Recovery nights differed highly significantly in sleep onset latency: participants took longer to fall asleep after deprivation under light ($p < 0.0001$). Recovery sleep following deprivation under light conditions was also characterized by increased wakefulness during the sleep period and lower sleep efficiency ($p = 0.011$). Morning melatonin levels were significantly lower after the recovery night following sleep deprivation under light compared to the recovery night following deprivation in darkness ($p < 0.01$). Glucose levels were higher after deprivation under light than after deprivation in darkness, although the difference did not reach statistical significance.

Supported by the ERDF-Project Brain dynamics No.CZ.02.01.01/00/22_008/0004643 and Cooperatio 38 – Neuroscience.

P23 – Differential involvement of the endocannabinoid system in obesogenic diet-induced memory impairments: influence of ovarian hormones

Laffont Clément [1], Ducourneau E.[1], N'diaye M.[1], Bakogiannis I.[1], Fermigier A.[1], Lafenetre P.[1], Bellocchio L.[2], Marsicano G.[2], Potier M.[1], Ferreira G.[1]

[1]*Laboratoire NutriNeuro, UMR 1286 INRAE, University of Bordeaux, Bordeaux, France.*
[2]*Neurocentre Magendie, U1215 INSERM, University of Bordeaux, Bordeaux, France*

In addition to cardiometabolic disorders, obesity is associated with cognitive dysfunction. This is particularly worrisome in the growing population of obese adolescents. In rodents, obesogenic high-fat high-sugar diet (HFD) consumption, in particular during adolescence, induces memory deficits. We recently demonstrated that alterations of the hippocampal endocannabinoid (ECB) system and its main receptor CB1R participate in HFD-induced memory deficits in male mice. Indeed, systemic blockade of CB1R or decrease of CB1R on hippocampal neurons improved object recognition memory task in HFD-fed males. Here, we investigated whether eCB-CB1R system contributes to HFD-induced memory impairments in females.

As in males, systemic CB1R blockade restored long-term memory in HFD-fed females, confirming its role in both sexes. However, unlike in males, hippocampal CB1R reduction failed to rescue memory deficits, while CB1R decrease in the medial prefrontal cortex (mPFC) improved memory. Our findings also revealed that hippocampal CB1R specifically on glutamatergic neurons mediated deficits in HFD-fed males, whereas mPFC CB1R on GABAergic neurons played a role in HFD-fed females. Higher levels of the eCB anandamide were found in the hippocampus and mPFC of males compared to females and systemic injection of an inhibitor of anandamide synthesis rescued memory deficits in HFD-fed males. We then investigated whether ovarian hormones mediate sex differences during puberty. Ovariectomy *per se* did not affect HFD-induced memory deficits but impede the beneficial effect of decreasing CB1R on mPFC. Remarkably, decreasing CB1R on hippocampal GABAergic neurons rescued HFD-induced memory deficits in ovariectomized females. Altogether our findings suggest that eCB-CB1R system contributes to the HFD-induced memory deficits in both sexes but that brain structures and cells types involved differ between males and females through a modulation from ovarian hormones.

P24 - Interoceptive regulation of the fear response by a glucose-sensing circuit in the insular cortex

Isabel de Araujo Salgado[1], Heloise Policet-Betend[2], Naila Ben Fredj[1,2] and **Christophe Lamy[1,2]**

1. Faculty of Medicine, University of Fribourg, Switzerland
2. Unit of Anatomy, Faculty of Medicine, University of Geneva, Switzerland

The brain relies on interoceptive mechanisms to adapt behavior to internal body states. While the contribution of subcortical circuits in the responses to metabolic challenges is well described, the involvement of cortical circuits remains less understood. Here, we identify a population of glucose-sensing neurons in the insular cortex that directly responds to hypoglycemia. This homogeneous population of large layer 5 pyramidal neurons projects to a subpopulation of PKCdelta positive cells in the lateral central amygdala (CeL). Reactivation of this cortical interoceptive pathway reduces innate defensive freezing behavior in response to a threat. Those findings reveal a direct role of the insular cortex in interoceptive sensing and in shaping emotional behavior depending of the body's energy status.

P25 – Astrocytic Panx1 Channels in the Dorsal Striatum Regulate Energy Balance and Feeding Behavior

C. Léger1, J. Castel1, A. Ansoult1, C. Franco3, D. Li3, N. Rouach2, S. Luquet1*, C. Martin1*

1Université Paris Cité, Unité de Biologie Fonctionnelle et Adaptative, Paris, France,
2Collège de France, Centre Interdisciplinaire de recherche en biologie, Paris, France,
3Sorbonne Université, Neurosciences Paris Seine, Paris, France
**Co-last and co-corresponding authors*

While the hypothalamus has long been regarded as the central regulator of energy homeostasis, emerging evidence points to a critical role for the dorsal striatum (DS), a key hub in reward-based decision-making, in modulating feeding behavior. Astrocytes, through their strategic positioning between neurons and blood vessels, are now recognized as active regulators of metabolic signaling and neuronal circuit function. In this context, our project explores the role of astrocytic pannexin1 (Panx1) channels in the DS, and how they contribute to the integration of metabolic and hedonic cues regulating energy balance.

Using a Cre-LoxP system in Panx1$^{flox/flox}$ mice, we generated astrocyte-specific Panx1 knockdown (KD) in the DS. Under short term exposure to high-fat high-sucrose (HFHS–1week) diets conditions, Panx1-KD mice exhibited decreased food intake and higher locomotor activity, associated with reduced fat mass, suggesting a change of energy expenditure regulation. Interestingly, brown adipose tissue temperature was reduced under ad libitum conditions in KD mice but increased after fasting, suggesting altered metabolic flexibility. Notably, feeding behavior following an 18 hours fast was comparable between genotypes across both CD and short term exposure to high-fat high-sucrose (HFHS – 1week) diets, pointing to a potential role of astrocytic Panx1 in modulating anticipatory or reward-driven feeding.

To dissect the underlying mechanisms, we implemented chemogenetic tools to manipulate astrocytic calcium signaling. DREADDs approaches are being used to assess how astrocyte excitability influences striatal network activity in control and Panx1-KD mice. Complementary to these studies, in vivo fiber photometry recordings of dopamine dynamics using GRAB-DA sensors in the DS revealed that astrocytic activation modulates dopaminergic responses to palatable food and associated cues. These findings support a model in which astrocytes in the DS influence feeding strategies by regulating dopamine dynamics in response to metabolic states and reward signals.

Our ongoing work aims to define the interplay between astrocytic calcium dynamics, Panx1 activity, and dopamine signaling in the DS. These insights may uncover novel glial mechanisms underlying obesity and maladaptive feeding behaviors.

P26 – GABAergic neurons of the preBötzinger complex in the brainstem contribute to the generation of respiratory-cardiovascular coupling

Ambre Linossier[1], Julie Buron[1,2], Christian Gestreau[1], Clément Menuet[1]

[1] INMED, INSERM, Aix-Marseille Université, Marseille, France
[2] UNIL, Université de Lausanne, Lausanne, Suisse.

The respiratory and cardiovascular systems act together to maintain the body's homeostasis. Their physiological efficiency is improved by respiratory-cardiovascular coupling (RCC), where heart rate (respiratory heart rate variability, RespHRV) and blood pressure (Traube-Hering waves, TH waves) oscillate in phase with respiratory activity, which optimizes pulmonary gas exchanges and cardiac energetic cost. RCC is mainly due to an interaction between neurons generating the respiratory command and neurons regulating cardiovascular activity, which are located in the brainstem. Specifically, it was shown that preBötzinger complex (preBötC) neurons, the group that generates the inspiratory rhythm, also directly modulate the activity of autonomic neurons that regulate heart rate and blood pressure. The preBötC is a highly heterogenous neuronal group, with excitatory and inhibitory neurons. Previous studies suggested that the inhibitory GABAergic neurons of the préBötC (preBötC^{GABA}) participate in the generation of RCC. To test this hypothesis and characterize preBötC^{GABA} neurons, we combined anatomical and functional approaches. Fluorescent in situ hybridization revealed that preBötC^{GABA} neurons consist of two subpopulations: neurons exclusively GABAergic and neurons co-expressing GABA and glycine. To investigate their connectome and functional roles, we injected adeno-associated viruses carrying floxed expression cassettes into the preBötC of GAD-Cre rats, allowing for selective expression of proteins for neuronal tracing (tdTomato and synaptophysin-GFP) or for bidirectional optogenetic modulation (somBiPOLES) in preBötC^{GABA} neurons. We found that preBötC^{GABA} neurons make presynaptic contacts with neurons in autonomic regions involved in regulating cardiovascular activity. Using the *in situ* Working Heart-Brainstem Preparation and *in vivo* anesthetized rats, photostimulation of preBötC^{GABA} neurons decreased the amplitude of TH waves, RespHRV, phrenic nerve inspiratory discharge, and lowered mean arterial pressure, whereas photoinhibition induced opposite effects. Systemic injection of the muscarinic antagonist atropine blocked the heart rate effects but not the blood pressure effects. This work shows that preBötC^{GABA} neurons regulate major physiological functions and contribute to their coupling through modulation of autonomic cardiovascular neurons. RCC alterations are characteristic of cardiovascular diseases, the leading cause of death worldwide. Understanding how RCC is generated under physiological conditions is essential to addressing these dysfunctions and hopefully correcting them.

P27 - Hippocampal reactivation of aversive experience enables safety learning and slow-breathing state for stress recovery

Baptiste Mahéo[1,*], Sophie Bagur[1,*], Dmitri Bryzgalov[1], Chloé Hayhurst[1], Mathilde Chouvaeff[1], Ella Callas[1], Carole Schmidt[1], Thierry Gallopin[1], Karim Benchenane[1]

1 *Brain Plasticity Unit, CNRS, ESPCI-Paris, PSL Research University, Paris, France*.

Animals face a dual challenge when confronted with threats such as predation or conspecific aggression. First, they deploy a cascade of defensive behaviours to avoid capture, such as freezing, avoidance, escape, or attack[1,2]. These strategies are essential to avoid physical harm but induce bodily and psychological stress that disrupts normal behaviours[3]. This leads to the second challenge: recovering from the stress induced by these events to restore homeostasis[4]—an underexplored process. Focusing solely on behavioural markers may overlook critical aspects of the aspects of the recovery process, such as bodily states including cardiovascular and respiratory parameters which are increasingly recognized as active participants in coping with threat. For instance, slow breathing can be artificially induced in mice to reduce stress[5] and is widely used as a relaxation technique in humans[6].
Therefore, whether animals engage in active processes of bodily and cognitive regulation leading to recovery remains unknown and their neural bases elusive.
Using a novel two-location fear conditioning paradigm in mice, we have identified a slow-breathing immobility state that emerges when animals identify safe environments after threat avoidance and participate in post-stress recovery. This immobile state was characterized by 2-4 Hz breathing and replay of the aversive experience in the hippocampus. Suppressing hippocampal sharp-wave ripples (SWRs) inhibited the emergence of this recovery state, suggesting their role in learning safe locations. Anxiolysis with diazepam directly promoted the recovery state while suppressing SWRs, showing this treatment to be a double-edged sword that facilitates immediate relief but impairs long-term safety learning. These results demonstrate the emergence of a coordinated brain-body active recovery state in which hippocampal replay plays a central role in emotional resilience and safety learning.

1. Fanselow, MS., *et al*. A functional behaviouristic approach to aversively motivated behaviour: predatory imminence as a determinant of the topography of defensive behaviour. in Evolution and learning. (1988).
2. Mobbs, D. The ethological deconstruction of fear(s). Curr. Opin. Behav. Sci. 24, 32–37 (2018).
3. Apfelbach, R., *et al*. The effects of predator odors in mammalian prey species: A review of field and laboratory studies. Neurosci. Biobehav. Rev. 29, 1123–1144 (2005).
4. Bolles, RC. *Et al*. A perceptual-defensive-recuperative model of fear and pain. Behav. Brain Sci. 3, 291–301 (1980).
5. De Sousa Abreu, R. P. et al. Episodic slow breathing in mice markedly reduces fear responses. Preprint at https://doi.org/10.1101/2024.12.09.627565 (2024).
6. Ma, D. et al. Benefits From Different Modes of Slow and Deep Breathing on Vagal Modulation. IEEE J. Transl. Eng. Health Med. 12, 520–532 (2024).

P28 - The paraventricular thalamus regulates energy metabolism and glucose homeostasis

Nejmeh Mashhour[1], Benoît Bertrand[1], Anthony Ansoult[1], Julien Castel[1], Camille De Almeida[1], Serge Luquet[1], Giuseppe Gangarossa[1].

1 Université Paris Cité, CNRS, Unité de Biologie Fonctionnelle et Adaptative, 75013 Paris, France

The control of energy homeostasis is essential for adaptation and survival. Its regulation involves a complex and dynamic process which highly depends on genetic, physiological, neurobiological, and environmental factors. Unfortunately, this homeostatic process is nowadays constantly challenged by the abundance and consumption of high-caloric meals which lead to metabolic and psychiatric disorders. While the hypothalamus and brainstem are traditionally considered as key regulators of feeding behaviors and energy homeostasis, recent studies have suggested a key role of extra-hypothalamic structures. Indeed, the paraventricular thalamus (PVT) has recently emerged as a powerful relay for cognitive, homeostatic, and visceral stimuli, therefore playing a crucial role in processes like arousal, stress, emotional memories, and drug addiction. However, its specific role in regulating feeding behavior and energy homeostasis remains poorly understood. The main objective of this study is to understand the involvement of the anterior PVT (aPVT) in scaling energy balance in both physiological and pathophysiological (obesity) conditions. Using several in vivo approaches (behavioral outputs and indirect calorimetry) combined with cell type-specific strategies (chemogenetics) and cutting-edge technologies (Ca2+ imaging), we have found that aPVT-neurons (1) dynamically respond to nutritional and metabolic states, (2) regulate nutrients partitioning and energy metabolism, (3) scale in vivo glucose dynamics and (4) are connected to hypothalamic subregions involved in the control of body's homeostasis.
In conclusion, our results uncover a new role for thalamic neurons in the coordination of homeostatic functions, therefore leading to a new understanding of unconventional brain circuits involved in metabolic disorders.

P29 - Hypothalamic PTH1R Signaling: New Therapeutic Perspectives for the Treatment of Obesity and Metabolic Disorders

Nour Mesto[1], Mathieu Galerne[2], Léa Vinel[2], Anthony Ansoult[1], François Mailliet[2], Stéphanie Moriceau[2], Franck Oury[2] & Serge Luquet[1]*

[1]*Université Paris Cité, CNRS, Unité de Biologie Fonctionnelle et Adaptative, F-75013 Paris, France.*
[2]*Institut National de la Santé et de la Recherche Médicale (INSERM) U1151, Institut Necker Enfants-Malades (INEM), Team 8, Université de Paris, 75015 Paris, France.*

Obesity, a major public health issue, results from an imbalance between energy intake and energy expenditure, and is accompanied by metabolic comorbidities such as fatty liver disease. The need to elucidate the mechanisms behind obesity and associated pathologies has sparked interest in the development of appropriate therapeutic strategies. The hypothalamus, the central regulator of energy metabolism, represents a preferential target thanks to its ability to integrate nutritional and energetic signals from peripheral organs. AgRP-neurons in the arcuate nucleus (Arc) of the hypothalamus play a central role in regulating this balance, notably via intracellular calcium signaling. One of the molecules involved in calcium homeostasis, and potentially in the regulation of food intake and energy metabolism, is the parathyroid hormone (PTH) via its receptor PTH1R.
The aim of our study is to determine the role of PTH1R signaling in AgRP-neurons in the modulation of energy metabolism in an obesity-related pathological context.
C57BL6/J mice were fed a high-fat diet (HFD) for 8 weeks. They were then stereotactically injected into the Arc with an adeno-associated virus (AAV) expressing a shRNA against Pth1r (sh-Pth1r) under the control of the AgRP neuron-specific AgRP promoter. The metabolic impact of this modulation was assessed 5 weeks after injection.
Our results show that selective down-regulation of Pth1r in AgRP-neurons in mice fed with HFD, induces a significant decrease in body weight, along with a reduction hepatic steatosis and improved glucose tolerance associated with enhanced pyruvate tolerance. The positive effect of this modulation could offer a potential new therapeutic tool for obesity and associated metabolic disorders.

P30 - Time and Mind: Electrophysiological Mechanism of Interval Timing

Tereza Nekovářová [1,2]*, Veronika Rudolfová [1,2], Kristýna Malenínská [1,2], Ondřej Škrla[1], Jakub Svoboda[1], Martin Brunovský[1], Jana Kopřivová[1], Vlastimil Koudelka[1]

1 National Institute of Mental Health, Klecany, Czech Republic
2 Faculty of Natural Science, Charles University, Prague, Czech Republic
3 Faculty of Electrical Engineering, Czech Technical University in Prague

Time perception is one of the main organizational principles of cognition shaping human life and behavior.
Temporal cognition plays a fundamental role in structuring human perception, thought, and behavior. It allows us to organize events in time, anticipate future outcomes, plan actions, and coordinate movement and speech. Time is also central to the representation of the self. The continuity of self-experience across time—often referred to as autonoetic consciousness—enables us to reflect on the past, envision the future, and maintain a stable sense of identity.
Recent research focuses on the study of oscillatory dynamics as a unifying principle for timing networks in the brain.
The present study focuses on interval timing, which involves processing durations from milliseconds to a few seconds. We investigated whether power in individual frequency bands or phase-amplitude coupling (PAC) correlates with temporal precision and accuracy in a Pair-Comparison Task. Using data from the first session, we built a linear regression model with frequency band power/PAC as predictors of temporal precision/accuracy. We then tested this model on data from a second session with the same participants.

This work was supported by the Johannes Amos Comenius Programme (OP JAK), project reg. no. CZ.02.01.01/00/23_025/0008715 and by the grant from the Ministry of Health Czech Republic (no. NU 22-04-00526).

P31 – How does the TAQ1A/ANKK1 Polymorphism Influence Reward Related Behaviours ?

Asma Oummadi[1], Fabien Ducrocq[1], Abygaëlle Da silva[1], Fanny Bain[2], Simon Benoit[2], Enrica Montalban[1], Guillaume Ferreira[1], Clémentine Bosch-Bouju[1], Serge Luquet[2] and Pierre Trifilieff[1]

[1]*Université de Bordeaux, INRAE, Bordeaux INP, NutriNeuro, 33076, Bordeaux, France*
[2]*Université Paris Cité, CNRS, Unité de Biologie Fonctionnelle et Adaptative, F-75013 Paris, France*

Psychiatric disorders, such as psychotic and affective disorders, as well as eating disorders, are clinically distinct but share similar symptomatic dimensions, suggesting comparable pathophysiological mechanisms. In particular, decreased motivation, increased impulsivity, and impaired behavioral flexibility are common behavioral features that are linked to a dysfunction of the mesocorticolimbic dopaminergic transmission. In addition to environmental factors, vulnerability to the development of mental disorders has been largely attributed to genetic variants. However, whether and how they impact behavioral dimensions of reward processes remain poorly explored.

Increased risk of obesity and addiction is associated with the Taq1A/Ankk1 polymorphism, a single-nucleotide variant (T→C, A2) in the ANKK1 gene next to the dopamine D2 receptor (D2R) gene. Present in 30–80% of the population, homozygous A1 allele carriers show a 30–40% reduction in striatal D2R. One common symptom found in these psychiatric diseases is cognitive inflexibility. Interestingly, even in non-pathological conditions, A1 carriers display alteration in reversal learning task, a well-established way to assess cognitive flexibility. Preclinical data show that Ankk1 loss-of-function, particularly in D2R-expressing neurons, disrupts dopamine signaling, reward processing, and procedural learning. However, although alterations such as cognitive inflexibility have been observed in humans carrying the Taq1A polymorphism, these behavioral dimensions remain poorly characterized at the phenotypic level in mouse models mimicking this genetic variation.

To examine how Taq1A/Ankk1 polymorphism affects reward processes, we are using a novel transgenic mouse model expressing either the A1 or A2 Taq1A variant, allowing a direct investigation of genotype-specific effects on behavior. By using an operant conditioning-based reversal learning task to assess behavioral flexibility, we found that while the A2 variant did not alter associative learning, A2/A2 mice outperformed A1/A1 individuals in a reversal learning task, suggesting enhanced cognitive flexibility. We are currently assessing other components of reward processing and executive functions in this mouse model such as motivation, impulsivity and value-sensitive behaviors.

These findings highlight the potential of the Taq1A/Ankk1 polymorphism to modulate dopamine-dependent executive functions, thereby contributing to individual vulnerability to psychiatric disorders.

P32 - Implication of neural circuits of the insular cortex in diet-induced anxiety

Léa Pages, Lena Rouzoul, Eva Ducourneau, Victor Gomez, Rim Fayad, Mathieu Lafourcade, Yoni Couderc, Thibault Bittar, Joeri Bordes, Lars Wilmes, Tina Habchi, Camille Penet, Guillaume Ferreira and Anna Beyeler

1 Neurocentre Magendie, Bordeaux Neurocampus, University of Bordeaux.
2 NutriNeuro, Bordeaux Neurocampus, University of Bordeaux.

Anxiety is an essential emotion allowing animals to avoid harmful situations. However, anxiety can become pathological, when persistent independently of the environment. In fact, anxiety disorders represent the most prevalent psychiatric conditions with an estimated yearly prevalence of 18% and a lifetime prevalence reaching 28% among adults. Although the etiology of anxiety disorders remains largely unknown, risk factors have been identified. For example, 20% of obese subjects develop an anxiety disorder. Interestingly, clinical and pre-clinical observations showed that the insular cortex (insula) is altered in patients and animal models of both obesity and anxiety disorders. Thus, we hypothesize that the insula contributes to obesity-induced anxiety.

To assess the impact of overweight on anxiety, we subjected 8-weeks-old C57BL/6J male and female mice to high caloric diet (HCD) for 12 weeks. We evaluated the anxiety level of standard-diet (SD)-fed mice or HCD-fed mice by using the elevated plus maze (EPM), the open field test and novelty suppressed feeding test. Interestingly, HFD-fed mice spent less time in open arms of the EPM, meaning they are more anxious compared to SD-fed mice. These results confirmed that HCD increases anxiety-related behaviors.

To define the role of insula circuits in HCD-induced anxiety, we used calcium imaging to record the activity of excitatory neurons of the anterior or posterior insula as well as neurons of the anterior insula projecting to the nucleus accumbens or to the basolateral amygdala (insula-NAc and insula-BLA), in both SD-fed and HCD-fed mice. As shown previously, in SD-fed mice, the activity of insula neurons was higher when mice were in open arms, selectively in the anterior insula. Interestingly, this response was amplified in HCD mice, and this activity correlated with mice anxiety levels.

Consistently, using *ex vivo* electrophysiological recordings we found that anterior, but not posterior insula pyramidal neurons were more excitable in HCD-fed mice. Overall, our work supports the hypothesis of a crucial contribution of glutamatergic neurons of the anterior insular cortex in HFD-induced anxiety in mice.

P33 - Effect of Western Diet on Sensory Representation During Food Intake in the Mouse Olfactory Cortex

Shubhi Pal[1,2*], Aileen Hakus[1], Amit Kumar[1], Hung Lo[3], Dietmar Schmitz[1], Friedrich Johenning[1**]

[1] Charité–Universitätsmedizin Berlin, Neuroscience Research Center; Institute of Cell and Neurobiology; Berlin, Germany
[2] M.Sc. Neurosciences/Neurasmus Student & Incoming PhD Candidate, University of Bordeaux, France
[3] Dominick P. Purpura Department of Neuroscience, Albert Einstein College of Medicine, Bronx, New York, USA
*Presenter: shubhi.pal@gmail.com
**Correspondence: friedrich.johenning@charite.de

Obesity is a complex metabolic condition increasingly linked to alterations in brain function, including changes in olfactory processing, which may influence eating behavior and energy regulation. In the current project, we aimed to investigate whether consumption of a western style obesogenic diet, i.e., a High-Fat and High-Sucrose (HFHS) diet, has an effect on sensory neuronal representations during feeding in the primary olfactory cortex, more precisely the anterior piriform cortex (aPC), in mice. In addition, we aimed to characterise the representation of isolated macronutrients (fat and sugar) alongside a combination solution (Ensure) in the piriform cortex. Utilizing *in vivo* Ca^{2+} imaging of excitatory neurons in the aPC, we demonstrated that, at a population level, there is feeding rate dependent modulation of aPC activity, replicating previous findings (Lo et al., 2025), and that this modulation is similar across Ensure, lipid and sucrose solutions. The representation (i.e., peak amplitude of activity) of the subset of neurons which were either activated or suppressed in response to the delivery of Ensure, lipid or sucrose solutions, seemed to scale with the caloric content of the solutions. Our findings thus suggest that the piriform cortex may also represent macronutrients such as fat and sugar, at a sensory level. A key finding of the current study was that consumption of HFHS was linked to a strong devaluation of feeding during the recording session during the period with *ad libitum* access to HFHS in the home cage, which recovered when access to the standard chow diet (SD) was restored. Exploratory and ongoing analyses on the impact of HFHS diet on sensory representations in primary olfactory cortex (aPC), in fasted conditions, were conducted, with an increase in peak amplitude of activation of Ensure responsive neurons at week 4 of HFHS consumption, however this needs to be supplemented and confirmed with increased sample size. The project sheds insight on the interplay between metabolic states and sensory systems, highlighting how metabolic states might modulate the sensory representations during food intake.

Reference:
Lo et al. (2025). Feeding-Induced Olfactory Cortex Suppression Reduces Satiation. *Neuron,* accepted 27th June 2025.

P34 - Cardiac interoception and insular cortex: a pathway to understanding social deficits in Autism

Alessandra Panzeri [1], Camilla Bellone [1]

1 Department of Basic Neuroscience, Faculty of Medicine, University of Geneva, Geneva, Switzerland

Interoception, the brain's ability to process internal bodily signals, plays a crucial role in regulating physiological states and adaptive behavior. The insular cortex, particularly its posterior region (pIC), is a key hub for integrating interoceptive and sensory information. Disruptions in these processes have been implicated in neurodevelopmental conditions, including autism spectrum disorder (ASD), where altered sensory integration and autonomic regulation are observed.
This study investigates how neuronal populations within the pIC encode interoceptive signals and their role in shaping behavior. Using a combination of in vivo imaging, behavioral paradigms, and physiological recordings in a mouse model, we examine the relationship between neural activity and cardiac dynamics in both typical and altered conditions. By identifying the circuits involved in interoceptive processing, this research may provide insights into mechanisms underlying sensory and social deficits in ASD and related disorders.

P35 - Burnout and interoceptive awareness, an exploratory research

Laure PELLOUX[1], Alexandre ZENON[1], L'BURN[2], Cécile DANTZER[3]

1 - CNRS, Université de Bordeaux, Institut de Neurosciences Cognitives et Intégratives d'Aquitaine
2 - L'BURN, Association spécialisée dans l'accompagnement de femmes en burnout, Bordeaux
3 – Laboratoire de Psychologie, LabPsy UR 4139, Université de Bordeaux, Bordeaux, France

Interoception, which is the ability to perceive, integrate, interpret and regulate internal signals (Chen et al, 2021) is implicated in various psychopathological disorders such as anxiety symptomatology, depression, panic disorders or eating disorders (Tsakiris et al., 2019). Although it is considered as a transdiagnostic process (Monestes et al., 2016), interoceptive awareness, defined as the conscious level of interoception with its multiple dimensions (Mehing et al., 2018), has received little attention in the context of burnout (BO).

The BO has been defined by the French National Authority for Health (HAS) as physical, emotional and mental exhaustion resulting from prolonged involvement in demanding work situations. According to the ICD-11, it is characterized by three criteria: (1) a feeling of exhaustion and fatigue; (2) a mental distance from one's work; and (3) a feeling of inefficiency. However, research suggests that there are several profiles of BO manifestation, some more focused on symptoms specific to BO (Berjot et al., 2017), and others, closer to certain comorbid disorders such as depression (Bauernhofer et al., 2018).

In the present research, we explore interoceptive awareness as a potential avenue to better understand the heterogeneity of BO. Indeed, as BO develops, a form of distance may emerge between the mental sphere (preoccupations, personal standard) and the perception of bodily signals (fatigue, somatization). According to Maslach (1997), during emotional exhaustion, avoiding strategies as hyperactivity can be developed to decrease the signals of fatigue. During the stage of escalation ("engrenage") and when facing a lack of performance, more cognitive and motivation efforts can be provided to trying achieving goals, and that, by ignoring or overpassing body exhaustion or pain signals (Pezé, 2022).

We hypothesize that various interoceptive modalities in term of perception, integration, interpretation or regulation, could be correlated with different profiles of BO manifestation. An initial qualitative study is currently underway to explore interoceptive awareness in women undergoing reconstruction after burnout. Initial data are currently being analyzed. We expect that an interoceptive mode combining body signals perception, attentional regulation, integration and interpretation capacities will be more expressed in people in an advanced phase of reconstruction

P36 – Implication of Mesocoticolimbic Dopamine Transmission in Behavioral Flexibility : a Role for Dopamine and NMDA Receptor Hetromers

Anna Petitbon[1], A. Contini[1], M-F. Angelo[1], R. Walle[1], M-C. Allichon[2], L. Hardt[1], M. Cazala[1], M. Depret[1], Y. Harouna[1], A. Ilie[1], F. Acedo[1], J. Correa Vazquez[1], R. Ortole[1], R. Thebeaud[1], A. Blanc[1], M. Rousseaud[1], A. Andrianarivelo[2], J. Barik[3], P. Vanhoutte[2], P. Trifilieff[1]

1 NutriNeuro UMR 1286, INRAE, Université de Bordeaux, France
2 Institut de Biologie Paris Seine, UMR 8246, CNRS, Sorbonne Université, Paris, France
3 Institut de Pharmacologie Moléculaire et Cellulaire, CNRS UMR7275, Université Côte d'Azur, Nice, France

Executive function impairments are a common symptom of many psychiatric pathologies. They are correlated with impaired dopaminergic transmission originating from the midbrain within the medial prefrontal cortex (mPFC). This mesocorticolimbic dopamine transmission is believed to be a key modulator of goal-directed behaviors and reward processing through its action on dopaminoceptive neurons expressing either D1 (D1R) or D2 receptors (D2R) in target structures. However, the precise mechanisms by which this dopamine transmission modulates goal-directed behavior remain unclear. Through a chemogenetic approach, we show that the mesocorticolimbic dopaminergic pathway is required when the animal has to adapt to changes in the association between actions and outcomes. Accordingly, using dopamine sensor coupled with fiber photometry, we show that medial prefrontal dopamine release is observed mainly during the reversal of action-outcome associations. From a mechanistic standpoint, we demonstrate, using interfering peptides, that mPFC D1/NMDA and D2/NMDA receptor heteromers constitute a central mechanism to mediate the effects of dopamine on behavioral flexibility, as their blockade spares action-outcome learning and expression but selectively impairs the animal's ability to adapt to changes in action-outcome associations. Using a calcium sensor approach coupled with fiber photometry, we characterized the neural signature of mPFC dopaminoceptive neurons expressing D1R or D2R during Pavlovian and operant conditioning paradigms and the effect of heteromer blockade on these activities. We identified specific patterns of activity during discrete cue, action and consumption phases, and found that blockade of either D1/NMDA or D2/NMDA heteromers strongly impacts activity patterns during the reversal of associations. These data provide a better understanding of the underlying mechanisms of behavioral adaptations and suggest that medial prefrontal D1/NMDA and D2/NMDA heteromers could be prime targets for the development of more specific therapeutic treatments for psychiatric symptoms.

P37 - The translational functional architecture of cardiovascular interoceptors

Héloïse Policet-Bétend[1], Christophe Lamy[1]

1 Unit of Anatomy, Faculty of Medicine, University of Geneva, Switzerland

Cardiovascular interoception plays a key role in emotional behavior. Its alteration has been linked to emotional dysregulation and anxiety. Cardiovascular states and blood composition are monitored by specialized receptors located in large vessels and the heart. However, it is unclear whether those receptors are altered in disease states linked to interoceptive dysregulation. In addition, their cellular and molecular structure is poorly characterized in humans. In this work we leveraged large scale human tissue clearing and multiplexed immunostaining to provide a detailed mapping of human cardiovascular interoceptors. We showed that they are more widely distributed and heterogeneous than presented in previous descriptions. We also traced their innervation to better understand the pathways linking them to the brain. Eventually, we studied with a similar approach equivalent structures in mice. Altogether, our data provides a detailed, functionally-relevant 3D cellular map of cardiovascular interoceptors in mice and humans which can serve as a base for mechanistic and translational research on interoception and interoceptive pathology.

P38 - *Phascolarctobacterium faecium* reverses gut motility impairment in mice with diet-induced obesity

Carlos Pomares-Díaz[1,2]; Clara Bullich-Vilarrubias[1,2]; Silvia Basili Franzin[1,2]; Giuseppe Esposito[3]; Inmaculada López-Almela[1], Alfonso Benítez-Páez[1], Verónica Tolosa-Enguís[1],Marina Romaní-Pérez[1], Yolanda Sanz[1]

[1] Microbiome Innovation in Nutrition & Health Research Unit, Institute of Agrochemistry and Food Technology, Spanish National Research Council (IATA-CSIC), 46980 Valencia, Spain.
[2] These authors contributed equally
[3] Department of Physiology and Pharmacology "V.Erspamer", Sapienza University of Rome, Italy.

The human intestinal bacterium *Phascolarctobacterium faecium* has anti-obesity properties in an animal model of diet-induced obesity (DIO) by promoting an anti-inflammatory response in the gut. Nevertheless, its impact on other intestinal pathways relevant for the energy homeostasis control, such as the modulation of the enteroendocrine system and the enteric nervous system (ENS), remains unexplored. Here, we investigated in mice whether *P. faecium* could affect these intestinal systems under an obesogenic diet and its effects on food intake, gut motility and postprandial metabolism. We demonstrated that *P. faecium* prevents hyperphagia through the anorexigenic gut hormone PYY. Independently of caloric intake, the bacterium accelerates gastrointestinal transit and reduces postprandial glycemia and intestinal lipid absorption. Immunofluorescence analysis of the ENS revealed a protective effect exerted by the bacterium on enteric neurons within the colonic myenteric plexus of mice exposed to a high-fat high-sugar diet. This study identifies the enteroendocrine system and the ENS as novel underlying targets through which the human intestinal bacterium, *P. faecium*, confers protection against obesity.

P39 - Hippocampal endocannabinoid system mediates obesogenic diet-induced memory impairments in male mice

Mylène Potier[1], E.G. Ducourneau[1], Y. Janthakhin[1], J.F. Oliveira da Cruz[2], J. Artinian[1], S. Alfos[1], J.C. Helbling[1], I. Matias[2], A. Busquets-Garcia[3], P. Trifilieff[1], G. Marsicano[2], G. Ferreira[1]

[1]INRAE, University of Bordeaux, Bordeaux INP, NutriNeuro, UMR 1286, Bordeaux, France,
[2]INSERM, University of Bordeaux, U1215 NeuroCentre Magendie, Bordeaux, France.
[3]IMIM-Hospital del Mar Medical Research Institute, Barcelona, Spain.

Obesity is a global health crisis, associated with cardiometabolic consequences but also with adverse cognitive outcomes. Adolescent obesity is increasing at a rate twice that of adults since 1990 and understanding its impact during this critical developmental period is essential, particularly on the cognitive aspect. Juvenile obesity can be modeled in rodents through high-fat and high-sugar diet (HFSD) exposure from weaning to adulthood. It is now well established that periadolescent exposure to HFSD has a detrimental effect on memory function. However, the mechanisms underlying these memory deficits remain to be elucidated. The brain endocannabinoid system and type-1 cannabinoid receptors (CB1R) control memory processes and are upregulated in obesity. In this study, we assessed in male mice whether the effects of obesogenic diet consumption on memory function are dependent on this system.

Using a pharmacological approach, we found that a systemic blockade of CB1R rescued HFSD-induced deficits of long-term object recognition memory (ORM), while also normalizing training-induced hippocampal c-Fos over-activation and aberrant in vivo long-term potentiation in CA1 of HFSD-fed mice. Following ORM training, we found an increase of endocannabinoid levels (anandamide), CB_1R expression and CB1R-dependent activation of the mTOR pathway in the hippocampus of HFSD-fed mice. Decreasing the expression of CB_1R expression on glutamatergic neurons in the hippocampus or inhibiting mTOR pathway rescued diet-induced long-term recognition memory deficits.

Together these results demonstrate that obesogenic diet consumption alters the endocannabinoid system of the hippocampus in male mice resulting in impaired activity and synaptic plasticity and eventually leads to memory deficits.

P40 - How adolescent obesity impacts social memory: the role of hippocampal CA2 and oxytocinergic system

Arielle Rakatonandrasana[1], A. Fermigier[1], M. Muller[2], E. Ducourneau[1], P. Lafenetre[1], M. Potier[1], V. Chevaleyre[2], R. Piskorowski[2], G. Ferreira[1]

1 Laboratoire NutriNeuro, FoodCircus team, Bordeaux University-INRAE, Bordeaux, France
2 Neuroscience Paris Seine, IBPS, Sorbonne University-CNRS-INSERM, Paris, France.

Obesity is a global health crisis, with adolescent obesity increasing twice as fast as in adults since 1990. Given its long-term metabolic, psychosocial, and cognitive consequences, understanding its impact during this critical developmental period is essential. Juvenile obesity can be modeled in rodents through adolescent high-fat and sugar diet (HFSD) exposure, a widely used model to study its cognitive effects. While HFSD impairs spatial, relational, contextual, and object-based memory, emerging evidence suggests it also disrupts social recognition memory (SRM). However, the underlying neural and molecular mechanisms remain unclear. We recently focused on the hippocampal CA2 area, known to be crucial for social memory, and found that HFSD-fed mice exhibit CA2 pyramidal neuron hyperactivity. Chemogenetic inhibition of these neurons rescues HFSD-induced SRM deficits. These deficits are also linked to reduced oxytocin (OT) signaling in CA2, as OT infusion during encoding restores SRM in HFSD-fed mice. However, as these findings were primarily obtained in males, we are currently investigating whether similar mechanisms are present in HFSD-fed females. Preliminary data suggest that, unlike males, HFSD-fed females show hippocampal hypoactivation and chemogenetic activation of the dorsal hippocampus rescues SRM deficits, indicating sex-dependent regulatory mechanisms. To further investigate these differences, we will assess in females SRM combined with biochemical, fiber photometry, chemogenetic, and pharmacological approaches to evaluate CA2 activity and OT signaling. Given the recent evidence implicating the supramammillary nucleus (SUM)-CA2 pathway in social novelty processing, we will also explore its role in HFSD-induced SRM deficits in both sexes. Identifying these mechanisms will underscore the importance of considering sex differences in cognitive research and may help developing future strategies to mitigate the neurocognitive impact of adolescent obesity.

P41 - Mapping the descending sympathetic innervation of spleen and pancreas

Leonor Remédio[1,2], Zack R. Lewis[3], Emma Thomas[3], Inês Rodrigues-Vaz[2], Lin T. Tun[2], Tiffany X. Chen[2], Gabriela Martins[2], Shenqin Yao[3], Kimberly Smith[3], Katie Fancher[3], Bosiljka Tasic[3], Darcy Peterka[2], Rui M. Costa[1]

1 Allen Institute for Neural Dynamics, Seattle, WA United States
2 Zuckerman Mind Brain Behavior Institute, Columbia University, New York, NY United States
3 Allen Institute for Brain Science, Seattle, WA United States

To maintain homeostasis, the immune system must trigger complex, multiorgan responses. Previous studies suggest that the nervous system regulates spleen immune function not only through peripheral circuits, but also through a broader neuronal circuitry involving the brain. The sympathetic neuronal circuits involved, and the mechanisms underlying this immune regulation, remain largely unknown.
Our whole-organ imaging revealed dense sympathetic innervation of the spleen and pancreas. Tracing studies using Cholera Toxin subunit B (CTB) demonstrated largely distinct populations of celiac-mesenteric ganglia (CMG) sympathetic neurons innervating each organ. We hypothesize that different neuronal populations exhibit distinct transcriptional profiles that correlate with their target organ's function. To investigate this, we defined CMG neuronal populations using single-nucleus sequencing and are currently characterizing those innervating spleen and pancreas.
To identify the second-order, preganglionic CMG spinal neurons, we injected CTB into the CMG. We mapped these neurons to known sympathetic regions of the thoracic spinal cord. Using a monosynaptic rabies virus approach, we identified third-order neurons in the brain projecting to preganglionic CMG spinal neurons. As expected, we found a direct communication with neurons emerging from the brainstem and hypothalamus. Surprisingly, we also observed a substantial descending cortical innervation, originating not only from the prefrontal and sensory cortices but also from the motor cortex. We found anatomical evidence of descending motor cortical axons synapsing with preganglionic CMG neurons in the spinal cord. We are exploring whether motor cortex activity modulates CMG neuronal activity and immune responses.
Taken together, our findings reveal that distinct neuronal populations in the CMG innervate almost exclusively the spleen or pancreas and receive input from specific preganglionic spinal neurons. These are innervated by third-order neurons projecting not only from the brainstem and hypothalamus but also from the cortex. We hypothesize that descending circuits from the motor cortex may influence the function of organs beyond muscles, including the spleen

P42 - Contribution of the anterior insular cortex in anxiolysis induced by a single injection of psychedelics

Daria Ricci[1], Olivia Isaac Newton[1], Benoit Eginard[1], Yoni Couderc[1], Joeri Bordes[1], Camille Penet[1], Tina Habchi[1], Anna Beyeler[1]

1. Université de Bordeaux, Neurocentre Magendie, INSERM 1215, Bordeaux, France

Anxiety is a natural, evolutionarily conserved response to perceived danger, essential for survival across species. However, when chronically high independently of the environment, it can become pathological. Anxiety disorders affect up to 18% of adults worldwide. Despite their prevalence, treatment options remain limited. SSRIs are commonly prescribed but often cause side effects, and around 30% of patients do not respond. The serotonin (5-HT) system, particularly its 1A and 2A receptor subtypes, plays a key role in mood and anxiety regulation. Psychedelics such as TCB-2 and 5-MeO-DMT, which act on these receptors, have recently gained attention for their potential to induce long-lasting anxiolytic effects. The anterior insular cortex (insula), enriched in 1A/2A receptors and involved in anxiety processing, may mediate these effects. We investigated the role of the anterior insula in the long-term anxiolytic actions of psychedelics.

Adult male and female mice (14 weeks old) received TCB-2 (3 mg/kg) or 5-MeO-DMT (20 mg/kg) intraperitoneally. Psychedelic activity was assessed by quantifying head-twitch responses over 60 minutes. Anxiety-like behaviors were measured using the elevated plus maze (EPM) and light-dark test (LDT) after short-term (60 min) and long-term (7 days) intervals.

In the short term, TCB-2 increased the time mice spent in the open arms without affecting behavior 7 days later, suggesting a transient effect. In contrast, 5-MeO-DMT showed a trend to decrease the time mice spent in the open arm in the short term ($p=0.1$) and significantly increased it after 7 days, indicating a sustained anxiolytic effect.

To explore the insula's involvement, we used fiber photometry to monitor calcium dynamics in anterior insula neurons expressing 1A or 2A receptors. 5-MeO-DMT increased the frequency of large amplitude transients (giant 'transients') in both populations, persisting at 7 days post-injection. To assess cell-type specificity, we used dual-color photometry to record glutamatergic neurons with jRGECO and monitored extracellular 5-HT with GRAB-5HT. Preliminary data suggest these giant transients are not systematically expressed by glutamatergic neurons, suggesting a contribution of GABAergic neurons.

We also performed *in vivo* electrophysiology to assess the neural impact of psychedelics. Twelve male and female mice were implanted with 16-channel electrodes to record local field potentials (LFPs) and spiking activity during behavior. Spectral analysis showed increased gamma-band power (30–80 Hz) shortly after 5-MeO-DMT injection. Single-unit analysis revealed enhanced spike–gamma phase coupling.

These findings suggest that 5-MeO-DMT induces lasting changes in anterior insula dynamics and circuit function, contributing to its anxiolytic effects and enhancing our understanding of psychedelic action on anxiety-related networks.

P43 - neuroIGNITOR: A flexible technology platform for stimulating and sensing peripheral nerves

Philipp Schnepel[1], Konstantinos Petkos[1], Shrishail Patki[1], Nicolo Rossetti[1], Vojkan Mihajlovic[1]

1 imec, Eindhoven, Netherlands

The development of more advanced and selective peripheral nerve stimulation technologies is essential for precisely modulating brain-body communication pathways, enabling deeper insights into neurophysiological mechanisms and more effective interventions for related disorders. We present a flexible stimulation and sensing benchtop system called neuroIGNITOR that is tailored to enable closed-loop neuromodulation experiments in pre-clinical settings. At the core of the system, we have developed an application specific integrated circuit (ASIC) that supports high voltage compliance (22V) and high stimulation currents (up to ±10.22mA) on 2 independent current sources that can be multiplexed to 16 output channels to enable a wide variety of peripheral nerve stimulation approaches (Xin et al. 2025). Furthermore, the ASIC provides both active and passive charge balancing capabilities and enables arbitrary waveforms along with novel, high frequency stimulation paradigms, such as i^2CS (Rossetti et al. 2025) or kHz-stimulation (up to 42kHz) in a safe manner. In order to enable closed-loop paradigms, it is integrated in a benchtop system that features additional 8 recording channels with advanced artifact blanking, built-in impedance monitoring and several digital lines for interfacing with other external devices. Finally, the hardware is controlled via a dedicated graphical user interface (GUI) for stimulation and recording, including on-the-fly visualization and pre-processing as well as a Python-based software development kit (SDK) for maximum flexibility to enable custom experimental protocols. We present results from validation experiments to showcase the potential of the system for research and pre-clinical studies by demonstrating stimulation of the medial and lateral giant fibers (MGF/LGF) in an earthworm model and simultaneous recording of the resulting evoked compound action potentials (eCAPs). Furthermore, we discuss the system components and possible applications in the neuromodulation space.

References

1. Rossetti, Nicolò, Weiguo Song, Philipp Schnepel, Naveen Jayaprakash, Dimitrios A. Koutsouras, Mark Fichman, Jason Wong, et al. 2025. “Control of Spatiotemporal Activation of Organ-Specific Fibers in the Swine Vagus Nerve by Intermittent Interferential Current Stimulation.” *Nature Communications* 16 (1): 4419.
2. Xin, Haoming, Meiyi Zhou, Roland van Wegberg, Peter Vis, Konstantinos Petkos, Shrishail Patki, Nicolò Rossetti, et al. 2025. “A 16-Output 10-V Compliant Stimulator ASIC With Sub-10-nA Mismatch and Simultaneous ETI Sensing for Selective Neural Stimulation.” *IEEE Journal of Solid-State Circuits* 60 (3): 908–20.

P44 - Translational study of the protective role of indoles in psychiatric illnesses.

Toshiko Sekijima[1], Camille Amadieu[1], Lucile Capuron[1], Quentin Leyrolle[1]

[1] *NutriNeuro, Bordeaux Neurocampus, University of Bordeaux, France*

Background: By 2030, psychiatric disorders are expected to become the leading cause of disability worldwide (Feng et al., 2020). Inflammatory processes are increasingly linked to psychiatric disorders and are known to be influenced by the gut microbiota. Among gut-derived metabolites, tryptophan-derived indoles have drawn attention for their immunomodulatory properties and potential effects on brain function (Qian et al., 2024). This project takes a translational approach to investigate the relationship between indoles, inflammation, and mood-related pathology.
Methods: Preclinical studies included *in vitro* experiments in which a selection of indoles were applied to BV2 microglial cells under inflammatory conditions (LPS) to assess anti-inflammatory effects. *In vivo*, dopaminergic activity in the nucleus accumbens was recorded via fiber photometry during a Pavlovian conditioning task to evaluate motivation-related neural dynamics. Mice received indole-3-lactic acid (ILA) either orally, through drinking water, or via intraperitoneal injection. Brain and blood samples were collected to assess ILA bioavailability and central penetration. In a clinical cohort of psychiatric patients with varying levels of depressive symptoms, circulating indole levels were measured alongside inflammatory and metabolic markers.
Preliminary Results: Data collection is ongoing. The study aims to characterize how peripheral indole levels relate to inflammation and psychiatric symptom severity in patients, assess the efficiency of brain delivery via different ILA administration routes in vivo, evaluate indole-related modulation of dopaminergic activity during reward processing, and determine the immunomodulatory effects of various indoles on microglial inflammatory responses in vitro.
Conclusion: This work integrates clinical, preclinical, and cellular approaches to examine gut-brain interactions in relation to psychiatric illness. By exploring mechanistic and translational aspects—including alternative routes of ILA administration—this project aims to study indoles potential as modulators of neuroinflammation and candidates for therapeutic development.

References
Feng et al., 2020; Qian et al., 2024

P45 - Exploring salivary and emotional responses triggered by exposure to real foods

Carla Simões[1,2], Anita Ramos[1], Ana Roque[1], Carolina Raposo[3], Elsa Lamy[1]

1 MED&CHANGE, Institute for Advanced Studies and Research (IIFA), University of Évora
2 Food Behaviour Lab, CATÓLICA LISBON School of Business & Economics, Universidade Católica Portuguesa.
3 Student at Chemistry department - University of Évora, Portugal

Our body works in a highly organized and coordinated manner to be physiologically prepared to receive food and for optimal nutrient processing - this happens even before food enters our mouth.
For example, when we see and smell a delicious chocolate cake, a cascade of different psychophysiological responses are triggered, including an increase of salivation, secretion of food-related hormones or changes in autonomic nervous system activity. These anticipatory events, collectively known to be part of the cephalic phase of digestion, play a crucial role in ingestive behaviour, may enhance appetite and encouraging us to eat.
Previous work, including studies from our lab, have reported changes at saliva secretion rate and composition level in response to visual food cues presented as pictures [1] and food odors (e.g., the smell of bread [2], vanilla and lemon [unpublished data]). These data suggest that the rapid release of saliva into our mouth, we experience during exposure to food stimuli, reflects changes in terms of composition that appear to vary according to the sensory characteristics and hedonic value of the food stimulus.
Notably, the same food stimuli may also evoke changes in our emotional states, indicating a complex interplay between physiological regulation and affective processing.
Investigating potential changes triggered by different real foods on salivary and emotional responses may provide valuable insights to elucidate physiological mechanisms underlying the regulation of ingestive behavior.
To further investigate these aspects, a total of sixteen healthy adults, from both sexes, took part in a within-subject experiment assessing salivary and emotional responses to real food stimuli.
Participants were asked to collect saliva samples, immediately before and during a 3-minute exposure to three foods, representing different calorie content and hedonic value (chocolate donut, white grapes and cream crackers) and a non-food item – a toy (car). Galvanic skin response, heart rate variability and facial expressions were also assessed to infer emotional arousal and detect affective responses. Additionally, participants self-report liking, desire to eat and rate the emotions evoked by each stimulus in study. The main results will be presented and discussed, correlating the changes in salivary biochemical composition with the activation of autonomic nervous system in response to foods.

References:
[1] Marques, E., Simões, C., Pérez-Jiménez, M., e Silva, F. C., & Lamy, E. (2025). Start looking at saliva: Effect of visualization of food images on salivary proteome. Food Research International, 209.
[2] Carreira, L., Castelo, P. M., Simões, C., Capela e Silva, F., Lamy, E. (2020). Changes in Salivary Proteome in Response to Bread Odour. 1–18.

This work was supported by Fundação para a Ciência e Tecnologia (FCT) through the PhD fellowship reference 2021.06485.BD awarded to Carla Simões.

P46 - Infra-slow Frequency Oscillations Propagating through Multiple Organs Convey Information on Phase-specific Timing for Self-Initiated Actions

Filip Španiel[1,2], Jiří Anyz[1], Dominika Grygarova[1], Jan Hubený[1], Vlastimil Koudelka[1], Ilia Shalkin[3], Jan Strobl[1,4], Jaroslav Hlinka[1,5], Tomas Nagy[4], Lea Jakob[1,2], Jakub Schneider[1,6], Jan Kudelka[1,6], Eduard Bakstein[1,6]

[1] *National Institute of Mental Health, Topolova 748, 250 67 Klecany, Czech Republic*
[2] *Third Faculty of Medicine, Charles University, Ruska 87, 100 00 Prague, Czech Republic*
[3] *Faculty of Science, Charles University, Albertov 6, 128 00 Prague, Czech Republic*
[4] *Faculty of Biomedical Engineering, Czech Technical University in Prague, nám. Sítná 3105, 272 01 Kladno, Czech Republic*
[5] *Institute of Computer Science of the Czech Academy of Sciences, Pod Vodárenskou věží 271/2, 182 07 Prague, Czech Republic*
[6] *Department of Cybernetics, Czech Technical University in Prague, Zikova 1903/4, 166 36 Prague, Czech Republic*

Cognitive functions crucial for voluntary actions fluctuate with infra-slow frequencies (ISF, <0.2 Hz), indicating their potential impact on self-driven behavior. ISF's involvement in brain–body interaction suggests that the link between ISF oscillations and self-initiated actions can be explored through low-frequency physiological readouts in peripheral organs.

We investigated whether ISF in peripheral systems relates to spontaneous motor activity. Pupillary hippus, heart rate variability (HRV), and respiration were analyzed against self-paced button presses across ISF frequency bands in 16 participants (Discovery dataset) and validated in an independent sample of 25 healthy individuals (Validation dataset).

Self-initiated actions significantly clustered within a specific phase of the slow3 ISF band (0.073–0.198 Hz) across all three organ systems ($p \leq 0.001$). Moreover, we observed phase-locking of slow3 oscillations among pupillary hippus, HRV, and respiration, indicating synchronized ISF propagation throughout the body.

These findings demonstrate that spontaneous action timing is not random: it preferentially occurs at particular phases of slow3 oscillations detectable in the autonomic nervous system. This suggests that inherent ISF rhythms modulate the propensity to act, uncovering a unified brain–body mechanism underlying the initiation of voluntary movements. Further exploration of this pathway could deepen insights into the complexities of conscious action initiation.

Supported by Ministry of Health of the Czech Republic, grant nr. NU22-04-00143

P47 - Restoring effect of voluntary exercise on obesogenic diet-induced social memory deficits: emphasis on the oxytocin signaling in the CA2 area of the hippocampus

Emma van Loveren[1], Romane Carlier[1], Guillaume Ferreira[1], Pauline Lafenêtre[1]

1 Univ. Bordeaux, INRAE, Bordeaux INP, NutriNeuro, UMR 1286, F-33000 Bordeaux, France

Obesity has emerged as a significant global health issue, with the prevalence among children and adolescents rising to 20% in 2022. During adolescence, a critical period for brain development, obesogenic diets have been shown to induce structural, cellular, and molecular alterations in multiple brain regions, particularly the hippocampus. Moreover, these diets also impair social recognition memory (SRM) which relies on the oxytocinergic system. Notably, the CA2 subregion of the hippocampus, characterized by a high expression of oxytocin (OXT) receptors, is regarded as a key hub for SRM. However, the specific role of OXT in the CA2 region on the SRM deficits induced by an obesogenic diet during adolescence remains unclear. Preliminary findings in the lab suggest that exercise during adolescence prevents these deficits, suggesting that the OXT signaling in the CA2 may be a key regulator.

The study aims at addressing the broad question of how exercise could mitigate obesity-related cognitive dysfunction.

First, we have established a dose-response of L368,899, an OXTR antagonist, in the CA2 area of control diet-fed mice in order to determine the suboptimal dose (preserving SRM) and the optimal dose (blocking SRM). Then, we locally administered these doses to assess their impact on exercise-induced SRM recovery in adolescent high-fat diet-fed mice. Besides, CA1-dependent spatial memory has been assessed via the object location memory test to evaluate the specificity of the observed effects.

Our results show that the pharmacological blockade of the OXTR in the CA2 induces deficits in the preference for social novelty but not spatial memory in control mice and that the oxytocinergic system may be involved in the beneficial effects of exercise.

P48 - Unravelling the contribution of glial cells in the insular cortex after obesogenic-diet induced anxiety

Lars Wilmes[1], Léa Pages[1], Lena Rouzoul[1], Anna Beyeler[1]

1 Neurocentre Magendie, Bordeaux Neurocampus, University of Bordeaux.

Anxiety is an essential emotion that enables animals to avoid potentially harmful situations. However, when persistent and disproportionate in regards of the environment, anxiety becomes pathological. Anxiety disorders are the most prevalent psychiatric conditions, with an estimated 12-months prevalence of 14% [1]. Although the etiology of anxiety disorders remains largely unknown, several risk factors have been identified. Notably, patients suffering from obesity show a 1.4 higher risk to develop anxiety disorders [2].
Obesity is increasingly recognized as a state of chronic low-grade inflammation, which may contribute to neuropsychiatric comorbidities [3]. Glial cells, including microglia and astrocytes, play a crucial role in mediating neuroinflammation and modulating neuronal function, thereby influencing brain circuits involved in anxiety [4]. Clinical and preclinical studies have shown alterations in the insular cortex (insula) in both obesity and anxiety, suggesting a shared neural substrate [5,6]. Based on this, we hypothesize that the insula contributes to obesity-induced anxiety.
To investigate the behavioral consequences of diet-induced obesity, we exposed 8-week-old male and female C57BL/6J mice to either a high-caloric diet (HCD) or a standard diet (SD) for 12 weeks. Anxiety-like behaviors were evaluated using the elevated plus maze (EPM). Concurrently, we examined neuroinflammatory changes in the anterior and posterior insula by quantifying both the number and morphology of microglia and astrocytes to assess their activation state.
HCD-fed mice exhibited significant weight gain and demonstrated increased anxiety-like behavior, spending less time in the open arms of the EPM compared to SD controls. In the insula, we observed a significant increase in microglial density across all cortical layers, whereas astrocyte numbers remained unchanged. This increase in microglia density following a HCD diet appears to be mainly driven by the anterior insula, with lower effect size in the posterior part. Detailed morphological analysis of these glial cells revealed an obesity-induced neuroinflammatory phenotype. Taken together, our result suggest that obesity induced anxiety is linked to microglia specific neuroinflammation in the insula. Further research will focus on elucidate the mechanisms of how neuoninflammation in the insula modulates anxiety.

[1] Craske MG et al. (2017) Nat Rev Dis Primers.
[2] Gariepy G et al. (2010) Int J Obes.
[3] Weisberg SP et al. (2003) J Clin Invest.
[4] Zhu H et al. (2023) J Neuroinflammation.
[5] Klumpp H et al. (2012). Biol Psychol.
[6] Scharmüller W et al. (2012) Neurosci Lett.

P49 - Investigating how lung cancer alters behavior via regulating the internal sensory system

Anastasia-Maria Zavitsanou[1,2,3], *Simon Downes Toney*[1,2,3], *Jake Kothandaraman*[1,3], *Simon Ogundare*[1,2,3], *Ishmail Abdus-Saboor*[1,2,3]

[1]*Zuckerman Mind Brain Behavior Institute, Columbia University, New York, NY*
[2]*Department of Biological Sciences, Columbia University*
[3]*Howard Hughes Medical Institute*

The brain receives vital sensory information from internal organs and regulates critical autonomic functions such as breathing. As such, it has been observed that lung cancer patients display an array of behavioral alterations, traditionally regulated by the nervous system, with pain being the most common one. We hypothesize that lung tumors dysregulate interoceptive signals from the lung to the brain, driving cancer-induced visceral pain.

Given that rodents cannot articulate their experiences, we use an array of behavioral tools to assess their pain state. We assess spontaneous behaviors, including body posture, facial grimace and digging in $Kras^{G12D/+};p53^{-/-}$-driven lung adenocarcinoma mice across tumor development. Using a 3D imaging and behavioral platform termed Keypoint-MoSeq, we discovered that cancer mice show more hunching and stationary movement while engaging in "turning" behaviors requiring upper body movement less frequently than healthy mice. We also employed PainFace, a deep neural network, to assess grimace in healthy and lung tumor-bearing mice as another measure of spontaneous pain. We find that lung cancer mice exhibit significantly higher grimace scores in comparison to healthy controls. Finally, digging behaviors, recently linked to visceral pain, are also altered in lung cancer mice compared to healthy mice.

To identify brain regions involved in lung cancer-associated pain, we employed chemogenetic activation of peripheral sensory neurons. Selective activation of specific lung-innervating sensory neurons was sufficient to induce pain-like behaviors that mimic those observed in lung cancer. Brain c-Fos analysis revealed that this activation drives distinct neural activity in specific regions of the brainstem, which is critical for processing visceral sensory input. Similarly, c-Fos mapping in lung cancer-bearing mice revealed overlapping patterns of brain activation. By comparing the brain regions engaged by chemogenetic nociceptor stimulation and those altered in the context of lung cancer, we identified shared circuits that likely underlie cancer-associated pain. Ongoing studies are assessing whether inhibiting these circuits with chemogenetics can reverse pain behaviors and influence tumor growth and survival.

In conclusion, we are integrating advanced behavioral tools, cellular biology methods, and lung cancer mouse models to unravel the mechanisms behind cancer-induced visceral pain, offering a novel perspective on cancer biology. Our goal is to deepen our understanding of how visceral pain signals are transmitted and to pave the way for innovative therapies in lung cancer treatment.

P50 - Neuromodulation in the posterior insular cortex maintains fear balance

Anna D. Zych[1,2], Meryl Malezieux[1], Eunjae Cho[1], Rosa-Eva Huettl[1], Andrea Ressle[1], Bianca Schmid[1] and Nadine Gogolla[1]

[1] *Emotion Research Department, Max Planck Institute of Psychiatry, Munich, Germany*
[2] *International Max Planck Research School for Translational Psychiatry, Munich, Germany*

Maintaining emotions in an adaptive range is important for survival. Our group has previously shown, that the posterior insular cortex (pInsCtx) plays a crucial role in keeping fear in balance upon extinction learning [1]. Surprisingly, we found that the pInsCtx exerts both, a fear extinction impeding, but also facilitating role depending on the strength of the acquired fear levels. However, we lack mechanistic understanding which factors may enable the dual role of the pInsCtx on fear extinction learning.

The neuromodulators noradrenaline (NA) and acetylcholine (ACh), are known to be released in response to sensory stimuli, as well as upon changes in affective states. Furthermore, they have been implicated in fear and extinction learning, as well as the regulation of bodily functions. Given these roles, we here aimed at investigating a potential contribution of these neuromodulators to the role of the pInsCtx in keeping fear in balance.

To understand the potential recruitment of NA and ACh within the pInsCtx during fear learning and extinction, we characterized their release using GRAB sensors during classical auditory fear conditioning in freely moving mice, as well as in head-fixed animals, where we obtained physiological readouts of heart rate, pupil and other arousal signals. Our results reveal specific differences in the dynamics of NA and ACh release in response to different fear-related sensory stimuli, changes in autonomic readouts and emotional behaviors. We found that NA release in the pInsCtx supports emotional learning and the maintenance of fear state, while ACh release is tightly coupled to bodily signals. Strikingly, interference with neuromodulatory release in the pInsCtx during fear extinction via optogenetic terminal inhibition resulted in bidirectional effects. While inhibition of NA terminals impaired fear extinction, inhibition of ACh terminals facilitated fear extinction. Importantly, these effects were contingent upon the fear state of an animal.

Together, our findings suggest that NA and ACh in the pInsCtx are crucial in regulating the adaptive balance of fear maintenance and extinction learning. This equilibrium is facilitated through the cholinergic integration of exteroceptive and interoceptive signals, alongside the noradrenergic encoding of fear state.

1. Klein, A.S., Dolensek, N., Weiand, C., and Gogolla, N. (2021). Fear balance is maintained by bodily feedback to the insular cortex in mice. Science (80-.). *374*, 1010–1015.

Practical Information

Venue

Centre Broca Nouvelle-Aquitaine – **Bordeaux Neurocampus**
Campus Carreire - University of Bordeaux
Bordeaux, France

Location on Google map:
Search for “Centre Broca”
https://goo.gl/maps/p4JE9mnJVMQ9hzz5A

How to get there?

Access details:
https://www.bordeaux-neurocampus.fr/en/centre-broca-nouvelle-aquitaine/

By taxi

Closest campus entrances :
40 rue Albert Marquet or 14 rue Eugène Jacquet

GPS: 44.810012 / -0.59645

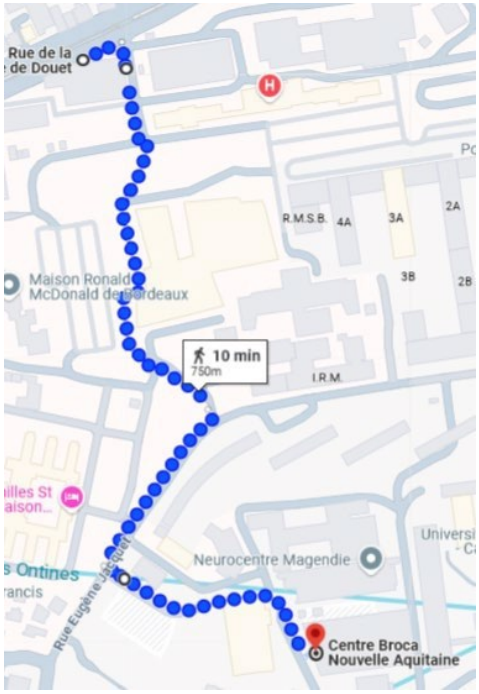
By tram

Closest tram stop: "**Saint-Augustin**".

From the airport: (20 minutes)
Take the tram A.

From the city center (around 15 minutes from the city center)
Take the tram A, direction “Mérignac”, “Le Haillan”, “Pin Galant” or “Aéroport”.

> Walking from the tram station



The Broca Centre is at around 8 min from the tramway station.

Take the **street under the chimney** and **follow the pedestrian white path** and the arrows “Centre Broca”.

By bus

Bus 24, 55 (Stop: Campus Carreire)
Bus 8, 20, 73, 80 (Stop: Bordeaux Carreire)

By car

Access: 125 rue Bethmann
Parking lot: P1

Transportation in the city

You can use your smartphone with the app TBM.

You can also buy a “m-ticket” before entering tram or bus.

All details on https://www.infotbm.com/en

To use your “m-ticket”, just validate each time you get into the tram or bus.

Posters

Sessions are organized during lunch breaks and the wine and cheese.

All posters will be presented during all sessions.

Luggage

“VIGIPIRATE” safety instructions strongly recommend to the participants to avoid to come with their luggage.
Exceptionally, the bags can be left at the front desk after being screened by a security officer.

Wifi

Wifi connection will be available.
Access through Eduroam will be possible.

Coffee and lunch

A welcome coffee will be served between every morning before the talks.
Coffee, tea and refreshments will be available during morning and afternoon breaks.
Lunches will be taken at the conference venue.
Take way will be available on Friday.

Goodies

We will not distribute notebooks, pens or totebags. Do not hesitate to go to our sponsor booths to get some!

Gala dinner

Thursday, October 2nd - From 7:30pm till 1am
« Le Tchanqué » - Bar, Restaurant, Rooftop
18 parvis des chartrons, Bordeaux

No specific dress code, but an elegant attire is welcome.





The Tchanqué Bar, Welcome to Le Tchanqué! A Bordeaux address inspired by the gentle way of life in the Arcachon Bassin, welcoming you to the heart of the chic, bohemian Chartrons district. Named 'Tchanqué' in reference to the famous oyster huts on stilts in the Arcachon Basin, this rooftop restaurant is inspired by the art of living and the gastronomy of the west coast. It's more than just a restaurant, it's a real place to live, with an atmosphere that changes throughout the day! Their menu features a majority of locally sourced produce from market gardeners, fishmongers, bakers, fish scalers and other producers in the Bordeaux region.

Located on the 7th floor, Le Tchanqué offers **a breathtaking view over the Garonne and the rooftops of Bordeaux**. Its location, attentive service and relaxed atmosphere offer the promise of memorable moments of sharing and a unique experience.

How to get there?

18 parvis des Chartrons, Bordeaux

By tram :
Tram C
Stop: CAPC - Musée d'Art Contemporain

Contact:
Neurocampus team: +33 (0)6 03 81 61 36

Contacts

Scientific Committee

Mario Carta (IINS) - mario.carta@u-bordeaux.fr
François Georges (IMN) - francois.georges@u-bordeaux.fr
Christelle Glangetas (IMN) - christelle.glangetas@u-bordeaux.fr
Enrica Montalban (NutriNeuro) - enrica.montalban@inrae.fr
Agnès Nadjar (Neurocentre Magendie) - agnes.nadjar@u-bordeaux.fr

Any questions before the event?

You can send an email to brainconf@u-bordeaux.fr or call us: +33 (0)5 33 51 47 92

Registrations:
Marine Boussicault – gestion.neurocampus@u-bordeaux.fr

Communication, abstracts:
Arnaud Rodriguez - arnaud.rodriguez@u-bordeaux.fr

Staff during the event

Marine Boussicault
Nolwenn Cloarec
Claire Delattre
Julia Goncalves
Arnaud Rodriguez

Bordeaux Neurocampus

Department of neuroscience of the University of Bordeaux

Director: Jérôme Baufreton
Deputy directors: Aude Panatier and Matthieu Wolff
Administrative team
Marine Boussicault: Administation, budget, event organization
Julia Goncalves : Scientific animation, management of the Cluster of Excellence BRAIN_2030
Arnaud Rodriguez : Communication, event organization

During the event, you can call us at:
+33 (0)6 03 81 61 36