Universität Regensburg | medbo® | UKR Universitätsklinikum Regensburg | GRK 2174

International Symposium

NEUROBIOLOGY OF SOCIO-EMOTIONAL DYSFUNCTIONS

May 8th - 10th 2026 | Regensburg, Germany

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Scientific Topics:

Decision-Making & Reward-Prioritisation
Neurobiology of Prosocial Behaviours
Mitochondria, Neuroplasticity & Psychopathology
Socio-Emotional Dysfunctions in Adolescence
Brain Networks of Socio-Emotional Dysfunctions: Humans and Rodents

DFG Deutsche Forschungsgemeinschaft

EBBS

Confirmed Speakers

Plenary Speaker: Elisabeth **Binder**

Johannes **Bohacek**
Eero **Castrén**
Simon **Chang**
Gustavo **Deco**
Freddy **Jeanneteau**
Tobias **Kalenscher**
Christian **Keysers**
Ewelina **Knapska**
Iris **Kolassa**
Kerstin **Konrad**
Tatiana **Korotkova**
Rohit **Menon**
Viginie **Rappeneau**
Jens **Schwarzbach**
Alexa **Veneema**
Christian **Wetzel**

Local Organisers

Inga D **Neumann**
Rohit **Menon**
Christian **Wetzel**
Oliver **Bosch**
Romuald **Brunner**
Jens **Schwarzbach**

Program & Registration

WELCOME TO REGENSBURG

We are delighted to extend a warm welcome to Regensburg to all participants of the Symposium on *Neurobiology of Socio-Emotional Dysfunctions 2026*. We are very pleased to host our Symposium in Regensburg and to bring together an international community of researchers interested in the neural mechanisms underlying socio-emotional processes.

The symposium marks the culmination of our DFG-funded Graduate Programme, which has been running under the same title since 2017. Accordingly, PhD candidates from our final cohort are actively involved—not only as speakers, session chairs, and poster presenters, but also behind the scenes as part of the organizing team.

In line with the research profile of our Graduate Programme, we have designed a series of stimulating and integrative sessions that bridge different areas of neuroscience and bring together diverse perspectives. The programme features a distinguished group of invited speakers alongside contributions from early-career researchers. Wherever possible, we have aimed to maintain a balance between presentations of both human and basic research approaches, fostering dialogue across experimental and clinical domains.

We are grateful to our sponsors and supporting institutions for their generous contributions, which have made it possible to organize and host this symposium here in Regensburg. Their commitment to fostering scientific exchange and supporting early-career researchers is deeply appreciated.

We would also like to extend our sincere thanks to the many friends, colleagues, and staff members whose dedication and hard work behind the scenes have ensured the successful organization of this symposium over the past months.

We wish you a pleasant, inspiring, and scientifically enriching stay in Regensburg, and a rewarding experience at the *Symposium on Neurobiology of Socio-Emotional Dysfunctions 2026*.

Inga D. Neumann
on behalf of the Local Organizing Committee

ACKNOWLEDGEMENTS

The organizers would like to thank the following sponsors for their financial support of the meeting, which was greatly appreciated:

DFG Deutsche Forschungsgemeinschaft

Regensburger Universitätsstiftung Hans Vielberth

EBBS European Brain and Behaviour Society

GRK 2174 UR Neurobiology of Social & Emotional Dysfunctions

PROGRAMME COMMITTEE

Inga D. Neumann
Romuald Brunner
Rohit Menon
Virginie Rappeneau
Jens Schwarzbach
Christian Wetzel

LOCAL ORGANIZING COMMITTEE

Inga D. Neumann
Oliver J. Bosch
Eva Hofmann
Melanie Kabas
Lorenz Kick
Doris Melcher
Laura Stangl

CONFERENCE INFORMATION

Conference Venue

Lecture Hall H52, Biology Building, University of Regensburg, Universitaetsstr. 31

Maps are provided in this booklet!

Name badges

Please always wear your name badge, including at lunch breaks. At the end of the meeting, please hand the name tag covers back for us to re-use them in the future.

Registration Desk

Located in front of the lecture hall H52 (Biology Building at the University).

Speaker presentation

You can either use your own computer (HDMI connection) or present from a memory stick using our desktop.

Poster presentation

Posters must be prepared as A0 portrait orientation. On Friday 8th, posters should be mounted before the meeting. The poster presentation is scheduled for the evening combined with the Welcome Reception. Pins and/or sticky tapes will be provided at the registration desk.

Coffee breaks / Lunch

During the coffee breaks, complimentary coffee, tea, and water are offered. Welcome Reception, lunch (Saturday and Sunday), and Farewell Party are included in the registration fee.

Trip to Weltenburg

On Saturday afternoon, we offer a trip to the oldest brewery situated in a monastery combined with spectacular views of the Danube Gorge (*Donaudurchbruch*), one of nature's unique spectacles. Included are bus transfers, boat trip, and dinner at the Weltenburg Monastery beer garden.

Health and safety

Participants are responsible for their own health and safety. The meeting organizers take no responsibility for theft or damage of property or accidents.

USEFUL INFORMATION ABOUT REGENSBURG

The Bavarian city of Regensburg, located 120 km northeast of Munich, where the Danube is joined by the rivers Regen and Naab, is undoubtedly one of history's wonders. It is the only Gothic city in Germany to have retained its medieval face with hardly a blemish and remains from the first Roman settlers (circa 1st Century) can still be visited.

Regensburg's Old Town skyline, unique in the intact section towards the Danube, is one of the most impressive anywhere in the world. Visible from afar, the 105 m high towers of St. Peter stand out, surrounded by towers, squares, houses, and lanes that tell the history of the Middle Ages in Germany like no others.

The oldest one in Germany, but still fully functional: the Stone Bridge has spanned the Danube for over 800 years. Even during the Middle Ages it was an example of Romanesque engineering to perfection. With its 16 square stone block arches rising towards the middle it was regarded as the eighth wonder of the world after its completion in 1145.

Eating out

Both local and international cuisine is reflected in the many restaurants and pubs in Regensburg. In the heart of the city, situated between the train station and the river Danube, you will find good restaurants, pubs and bars.

Shopping

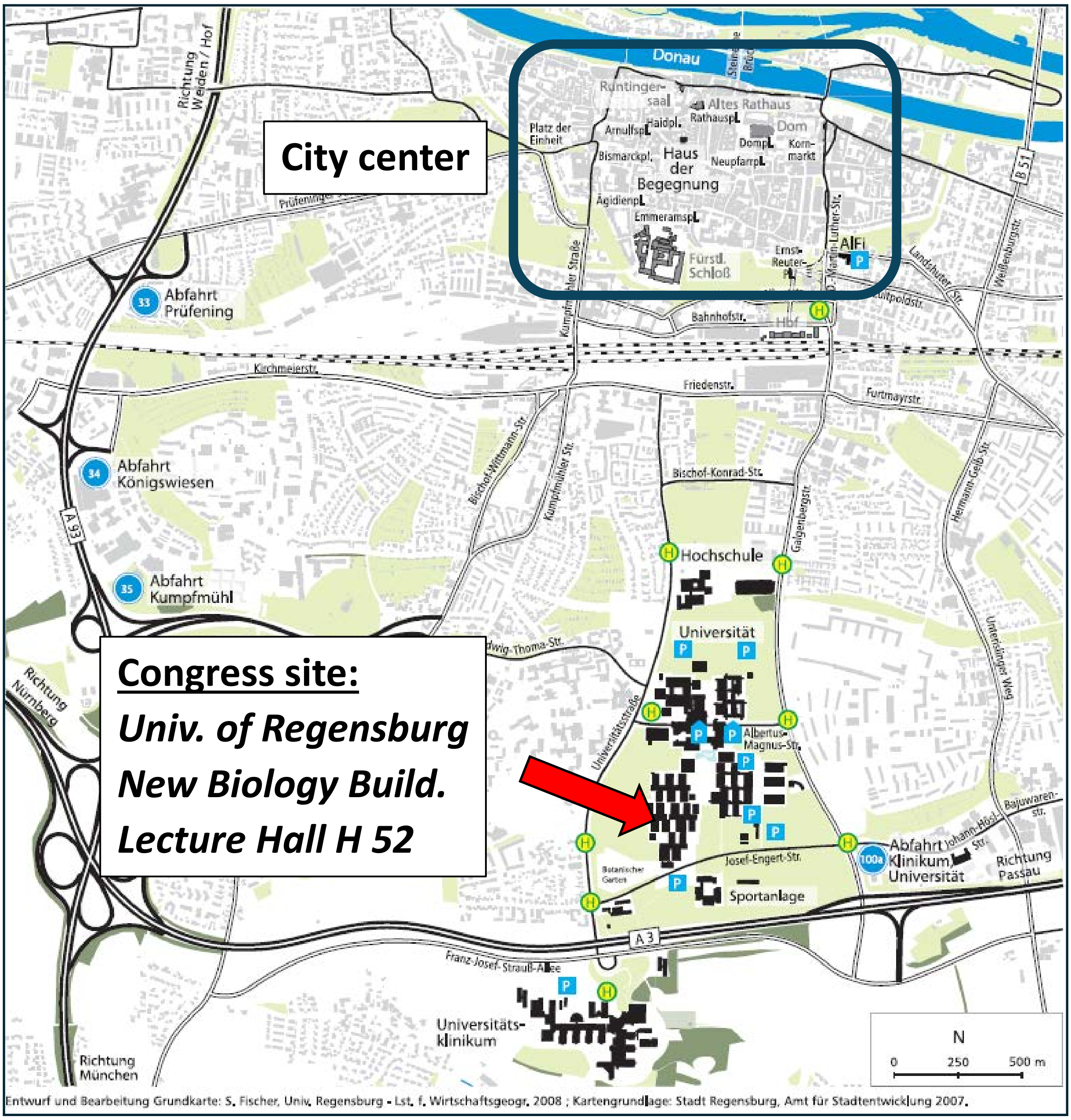
The heart of the city, particularly the narrow medieval streets of the pedestrian zone, are famous for their shops. There are two Shopping Centers within walking distance, i.e., the *Arcaden* (opposite to the train station) and the *Donau Einkaufszentrum* (east of downtown).

MAPS

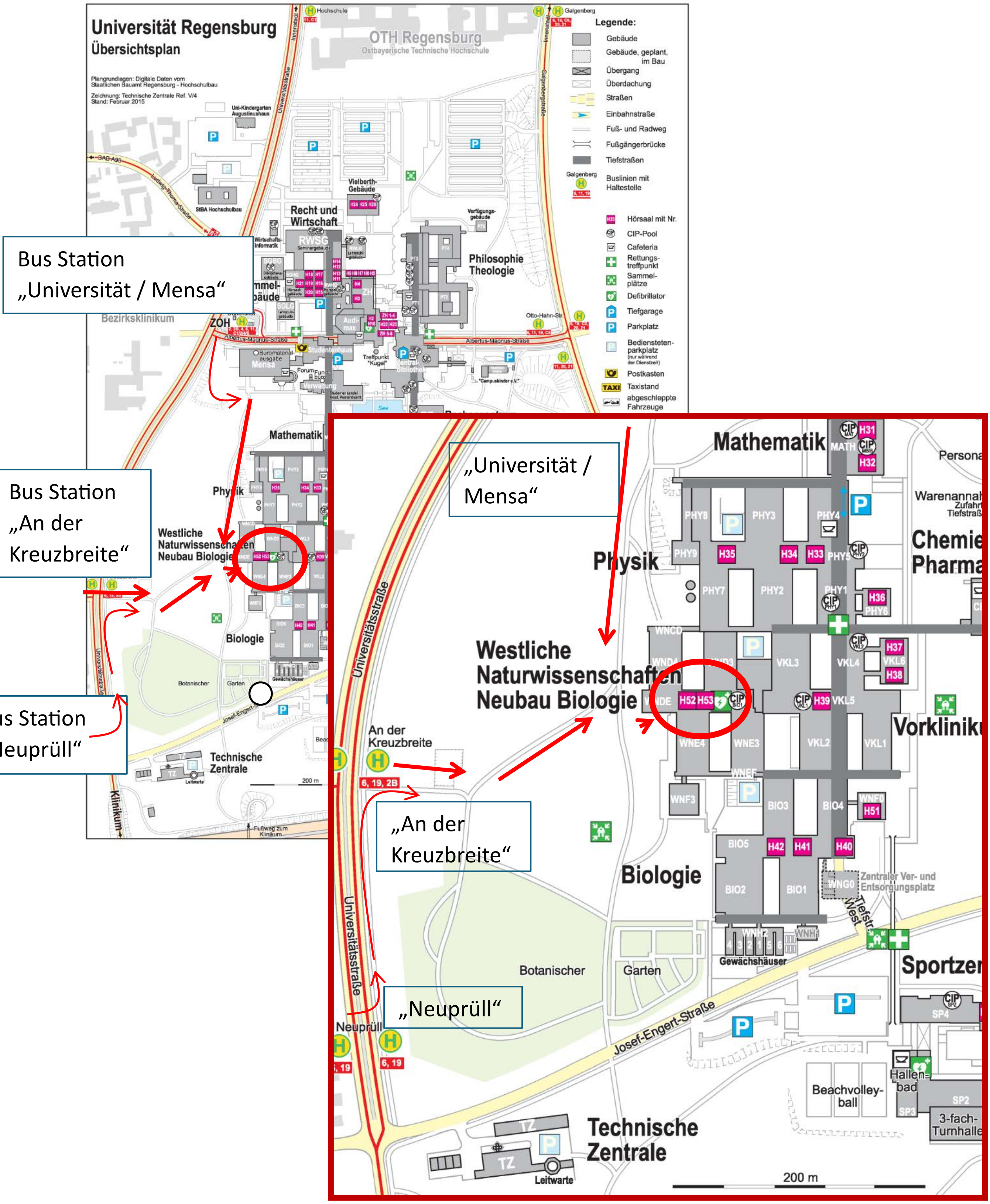
Lecture Hall H52 → located in the Biology Building of the University

You can take a taxi for approximately €15.

It is easy to take the bus, especially when you download the app and register (maps, bus times, fare payment). There are several bus stops throughout the downtown area. At any of them, you can board bus #6 towards "Klinikum". Get off at one of the following bus stops: "Universität / Mensa" (recommended, 5 min walking), "An der Kreuzbreite" (recommended, 5 min walking), or "Neuprüll" (10 min walking).



- get on bus #6, direction "Klinikum“
- get off at “Universität” or “An der Kreuzbreite” or “Neuprüll”



SCIENTIFIC PROGRAMME - OVERVIEW

FRIDAY 8TH MAY

15.00	SESSION 1 MITOCHONDRIA AND BDNF: METABOLIC DRIVERS OF NEUROPLASTICITY AND PSYCHOPATHOLOGY
17.00	BLITZ-POSTER PRESENTATION
18.00	WELCOME RECEPTION with POSTER EXHIBITION

SATURDAY 9 TH MAY

8.30	PLENARY LECTURE
9.15	SESSION 2 NEUROBIOLOGICAL MECHANISMS OF DECISION-MAKING AND REWARD PRIORITIZATION
10.45	Coffee Break with *EBBS* Young Investigator Award and group picture
11.15	SESSION 3 BRAIN NETWORKS UNDERLYING SOCIO-EMOTIONAL DYSFUNCTIONS
13.00	Lunch
13.45	TRIP TO *WELTENBURG MONASTERY*
20.30	Return

SUNDAY 10 TH MAY

9.00	SESSION 4 CIRCUITS AND NETWORK DYNAMICS UNDERLYING SOCIO-EMOTIONAL (DYS)FUNCTION
10.30	Coffee Break
11.00	SESSION 5 NEUROBIOLOGY OF PROSOCIAL BEHAVIOURS
12.45	Lunch
14.00	SESSION 6 SOCIO-EMOTIONAL DYSFUNCTIONS IN ADOLESCENCE: BRAIN STRUCTURAL AND FUNCTIONAL CHANGES
15.45	END OF SCIENTIFIC PROGRAMME
16.00	FAREWELL BBQ

SCIENTIFIC PROGRAMME

FRIDAY 8TH MAY

13.00 - 15.00 Registration

15.00 – 17.00 SESSION 1
MITOCHONDRIA AND BDNF: METABOLIC DRIVERS OF NEUROPLASTICITY AND PSYCHOPATHOLOGY
Chairs: *Nicolas Reichardt, Sara Sheibani Tezerji* (Regensburg, Germany)

15.00 Depression gets under the skin -- investigating depression in a model ranging from fibroblasts to neuronal networks

Christian Wetzel
University of Regensburg, Germany; Christian.Wetzel@klinik.uni-regensburg.de

Major depressive disorder (MDD) is a severe psychiatric disorder with a high global prevalence and a substantial impact on quality of life. Genetic and environmental factors lead to molecular and cellular alterations affecting neurotransmission, neurotrophic pathways, mitochondrial function, and neuroplasticity. Disruptions in these systems result in structural and functional brain changes across multiple levels of organization, from synapses to large-scale neural networks, ultimately impairing mental health.
To investigate cellular alterations in MDD, skin biopsies were taken from patients and healthy controls to culture primary fibroblasts. Fibroblasts from MDD patients showed reduced mitochondrial function. Reprogramming these cells into iPSCs and differentiating them into neural progenitor cells (NPCs) preserved this impaired mitochondrial phenotype. Further differentiation into neurons revealed altered electrophysiological properties, indicating that mitochondria of MDD patient cells are functionally different and that these alterations are transferred to the next lineage having functional consequences on electrophysiological properties of iPS-neurons.
To assess network-level activity, we examined the intrinsic ability of neurons to self-organize into functional networks. Using fura-2 live-cell imaging, we analyzed intracellular Ca^{2+} transients in 2D cultures of iPSC-derived neurons from patients with MDD and healthy controls and applied graph theory analysis to study network topology. To further enhance spatial and temporal resolution of network recordings, we established a high-density multi-electrode array (HD-MEA) platform, enabling large-scale electrophysiological recordings from thousands of electrodes and detailed analysis of neuronal connectivity and network dynamics.

15.30 **Effects of cortisol on bioenergetics and electrical properties of neural cells in a human cell model of depression**

Artiola Ndou, Christian Wetzel, Vladimir Milenkovic
University Clinic Regensburg, Germany; artiola.ndou@ukr.de

Major Depressive Disorder (MDD) is a leading cause of disability globally; nevertheless, its molecular underpinnings remain elusive, and treatment responses are often inadequate. Mitochondrial dysfunction is a critical risk factor, given its role in energy production, synaptic transmission, and neurogenesis. Prior work in skin fibroblasts from MDD patients demonstrated significantly reduced basal and maximal oxygen consumption rates (OCR). In iPSC-derived neuronal progenitor cells (NPCs) from MDD patients, these bioenergetic perturbations persist. In this study, we established an in vitro model using human-iPSC-derived NPCs and neurons of MDD patients and matched healthy controls. To model physiological stress, cells were exposed to cortisol acutely (30-minutes), subacutely (24-hours), and chronically (7-days). Mitochondrial respiration and calcium (Ca^{2+}) dynamics were measured. Our data show that acute cortisol exposure induces an immediate decrease in basal OCR in NPCs from both cohorts, suggesting non-genomic mitochondrial regulation. After 24-hours, both groups maintained similar suppression of basal respiration, whereas chronic exposure led to an enhancement of bioenergetic responses, weaker in MDD patients. Calcium imaging revealed that cytosolic Ca^{2+} transiently increases following 24-hours cortisol treatment, and declines again by 7-days, while mitochondrial Ca^{2+} responses vary without consistent trends across subjects. Electrophysiological analyses are currently underway to characterize ion-channel function and membrane excitability in iPSC-neurons. Furthermore, multielectrode array (MEA) recordings are planned to investigate network-level activity. Finally, pharmacological assays will test classical antidepressants and neurosteroids to assess their capacity to restore basal conditions. This study aims to uncover molecular mechanisms of MDD and guide the development of interventions.

15.45 **BDNF-TRKB signaling in neuroplasticity and antidepressant effects**

Eero Castrén
University of Helsinki, Finland; eero.castren@helsinki.fi

Neuronal plasticity is a critical mechanism underlying resilience and the antidepressant effects. Furthermore, there is evidence that restricted plasticity may be an underlying factor in the pathophysiology of mood disorders. Our previous studies have shown that antidepressant drug treatment promote neuroplasticity and resilience. Beneficial effects of are not produced by the drug treatment alone, but drug treatment needs to be combined with training that guides the changes in neural networks rendered plastic by antidepressant treatment. We have shown that many different antidepressant drugs (including typical drugs SSRIs and tricyclics, but also the rapid-acting ketamine and psychedelic compounds) act by directly binding to TrkB, the receptor for brain-derived neurotrophic factor (BDNF) and allosterically increasing BDNF signaling. The plasticity-promoting and antidepressant-like effects of various drugs known to have antidepressant effects are lost in mice with a mutation in TrkB that is otherwise silent but prevents drug binding to TrkB. We have also shown that the effects of antidepressants are predominantly mediated by TrkB receptors on the parvalbumin-expressing interneurons and that the activation of TrkB in these neurons reduces the expression of parvalbumin and the density of perineuronal nets, thereby reducing their inhibitory control of pyramidal neurons and promoting network activity through disinhibition. These findings demonstrate that antidepressants promote neuronal plasticity through BDNF-TrkB signaling and emphasize that antidepressant drug treatment needs to be combined with supporting therapy or training that guides the plastic networks towards recovery and resilience.

16.15 Modeling the Human Oxytocin System: From hiPSC-Derived Neurons to In Vivo-Integration – *Selected Talk*

Ana Zovko[1,2], Konstantinos Afordakos[1], Catello Guida[2], Alan Kania[1], Raquel Perez Fernandez[2], Jahnavi Srinidhi[3], Sreedevi Raghu[2], Sandra Horschitz[2], Quirin Krabichler[1], Valery Grinevich[1], Philipp Koch[2]
[1]Central Institute of Mental Health, Mannheim, Germany; [2]Hector Institute for Translational Brain Research; [3]University Medical Centre Mannheim; Ana.Zovko@zi-mannheim.de

Oxytocin (OT), a hypothalamic neuropeptide regulating emotional and social behaviors, is disrupted in neurodevelopmental and psychiatric disorders such as autism, anxiety, PTSD, depression, and schizophrenia. Despite its relevance, no approved therapies exist to restore OT function, and no models with high translational value are available to study its dysfunction. Approaches based on human-induced pluripotent stem cells (hiPSCs) provide a versatile framework for modeling oxytocin-related disorders and producing neurons suitable for transplantation. Here, we present four key findings advancing this approach:
(1) hiPSC-derived hypothalamic culture, patterned to generate OT-specific progenitors, arises from the Pax6-dorsal/alar hypothalamus, at the intersection of Rax1 (ventral/posterior hypothalamus), and Foxg1 (anterior/telencephalic border), characterized by the key lineage-specifying transcription factors Otp, Sim1, and Brn2 (distinct from Nkx2.1-basal/ventral plate lineage); (2) Ngn2 is a master regulatory gene that drives late-stage progenitors toward a

glutamatergic OT-producing fate; (3) Generation of first in vitro model for human oxytocin research; (4) Successful transplantation these progenitors into athymic rat brains – survival, integration and axonal projections within the host brain.

These findings provide a new tool for studying human OT biology, modeling OT-related disorders in vitro, and laying the groundwork for future cell-replacement therapies aimed at restoring neuroendocrine function in conditions involving the OT system.

16.30 **The Unity of Body and Mind: Mitochondrial Biopsychology in Clinical Psychology and Psychotherapy**

Iris T. Kolassa
University of Ulm, Germany; iris.kolassa@uni-ulm.de

Chronic, excessive, and traumatic stress accumulates across the lifespan and, in a dose-response manner, increases the risk of both mental and physical illness. At the molecular level, stress promotes oxidative damage and persistent, low-grade inflammation, ultimately disrupting mitochondrial bioenergetics. The number, duration, and intensity of childhood adversities leave lasting imprints on immunometabolic processes and shape personality development. These stress-related alterations are reflected in characteristic changes in inflammatory activity and cellular energy metabolism observed in traumatized and depressed individuals. As inflammation naturally rises with age, it may also contribute to the earlier onset of age-related diseases, particularly in those exposed to chronic stress or trauma. This talk outlines current knowledge on immunometabolic mechanisms in stress- and trauma-related disorders including depression and post-traumatic stress disorder, as well as other psychiatric conditions. It also highlights the potential of targeted lifestyle interventions to complement psychotherapy, with the aim of improving and sustaining treatment outcomes – an area that warrants further research and greater integration into clinical practice.

17.00 **BLITZ-POSTER PRESENTATION**

Chairs: *Francisca Horn, Diego Vesga* (Regensburg, Germany)

Each poster is presented by using only **one slide** within **2 min**!

18.00 **WELCOME RECEPTION with POSTER EXHIBITION**

SATURDAY 9 TH MAY

8.00 - 10.00 Registration

8.30 – 9.15 PLENARY LECTURE

Chair: *Antonia Heinrich* (Regensburg, Germany)

Moderators of the stress response: From molecular discovery to translational impact

Elisabeth Binder
Max Planck Institute of Psychiatry, Munich, Germany; binder@psych.mpg.de

Adverse and stressful experiences during sensitive developmental periods are potent risk factors for a range of psychiatric disorders. Interindividual differences in the impact of such experiences suggest a complex interplay between genetic predisposition, epigenetic regulation, and environmental exposures. This presentation will present research examining the molecular and neurobiological pathways by which stress signals might be transduced into long-lasting changes in brain function. I will present data that draws from large human cohorts, deep phenotyping, experimental models including human neural organoids and rodent models and postmortem human brain data that illustrate how genetic variants interact with adversity to influence vulnerability and resilience. A focus will be on the investigation of stress-responsive gene regulatory element using massively parallel reporter assays to probe cell-type and allele-dependent impact of stress and stress hormones on gene regulatory networks. Such context-dependent functional annotations of gene variants can be then used to construct functional gene scores that link these molecular effects to functional outcomes, such as behavior, psychopathology and brain function. Relating molecular mechanism of gene x adversity interplay may help identifying biomarkers for preventive strategies as well as novel therapeutic approaches.

9.15 – 10.45 SESSION 2

NEUROBIOLOGICAL MECHANISMS OF DECISION-MAKING AND REWARD PRIORITIZATION

Chairs: *Stefanie Kau, Niranjan Biju* (Regensburg, Germany)

9.15 Leptin-sensitive neurons in the lateral hypothalamus balance hunger, fear, and social needs

Tatjana Korotkova
University of Cologne, Germany; tatiana.korotkova@uk-koeln.de

Animals must continuously balance internal states such as hunger and thirst with competing demands including social interaction, mating, and threat avoidance. However, the neuronal mechanisms underlying the integration and prioritization of these needs remain incompletely understood. Here, combining calcium imaging in freely behaving mice with optogenetic and chemogenetic manipulations, we identify leptin receptor-expressing neurons in the lateral hypothalamus (LepR LH) as a key integrative node for adaptive behavioral control. LepR LH neurons dynamically encode metabolic states and guide behavioral choices between nutritional and social rewards. While feeding is associated with rapid inhibition of specific LepR LH subpopulations, activation of these neurons suppresses consummatory behavior and promotes social interaction even under hunger or thirst. Moreover, LepR LH neurons encode anxiogenic stimuli and predict individual anxiety levels. Under high-anxiety conditions, their activity is suppressed by prefrontal cortical input, yet their activation enables adaptive responses, including exploration and feeding in threatening environments, and reduces maladaptive behavior in an anorexia nervosa model. Together, these findings demonstrate that LepR LH neurons integrate metabolic, emotional, and social signals to flexibly prioritize competing needs and ensure adaptive behavioral output in both health and disease.

9.45 Two sides of the stress coin: Stress-Neuromodulator Effects on Social Preferences

Damon Dashti[1,2], Luca Lüpken[1], Mohammad Seidisarouei[1], Paul Forbes[1], Schnitzler Alfons[3], *Tobias Kalenscher*[1]
[1]Comparative Psychology, Heinrich-Heine University Düsseldorf, Germany;
[2]Department of Economics, University of Zurich, Zurich 8006, Switzerland;
[3]Institute of Clinical Neuroscience and Medical Psychology, Heinrich-Heine University Düsseldorf, Germany; Tobias.Kalenscher@hhu.de

How does stress affect social behavior? Does it make us more aggressive or, perhaps, more generous? Traditionally, stress was thought to trigger an antagonistic fight-or-flight response, aimed at eliminating the stressor.

However, in a series of pharmacological and behavioral studies, we and others found that acute psychosocial stress increased generosity toward others, consistent with the "tend-and-befriend" hypothesis. To reconcile these conflicting findings, we recently hypothesized that stress does not promote one response over the other; instead, it stimulates both behaviors, dissociable on the neuropharmacological level and moderated by social context. In a placebo-controlled, double-blind psychopharmacological study, we administered the drugs hydrocortisone and/or yohimbine to manipulate the actions of the main stress hormone cortisol and the sympathetic arousal transmitter noradrenaline. Participants then made decisions in an intergroup conflict task. They teamed up with others to form an ingroup and played against multiple outgroups. Notably, outgroup members initiated financial harm to the ingroup before participants made their decisions. They could choose to a) maximize personal gain, b) cooperatively increase the payoff of their ingroup, or c) increase the ingroup's payoff while competitively decreasing that of the outgroup. Consistent with our hypotheses, glucocorticoid activation increased cooperation with the ingroup, whereas noradrenergic activation increased parochial competition, i.e., prosocial ingroup support combined with hostility against the outgroup. Our findings challenge the simplistic dichotomy of fight-or-flight versus tend-and-befriend, and instead suggests that both may be co-activated, with downstream consequences for cooperation, competition, and perhaps the escalation of real-world intergroup conflict.

10.15 **Craving Company or Calories? Oxytocin's Control of Competing Needs in Male Mice**

Virginie Rappeneau
University of Regensburg, Germany; virginie.rappeneau@ur.de

Animals must balance competing drives—such as social interaction, feeding, and exploration—to adapt to changing environments. The neuronal mechanisms that prioritize rewards based on internal state and context remain incompletely understood. The neuropeptide oxytocin (OXT) and the neurotransmitter dopamine are key regulators of social and motivated behaviors, and their interaction across distributed circuits enables flexible decision-making. Dysregulation of these systems has been implicated in disorders such as autism spectrum disorder, depression, and obesity.

Here, we investigated neuronal circuits mediating choice between social interaction and food reward. Using the Social versus Food Preference test in adult male CD1 mice, motivational state was manipulated through housing conditions and food deprivation. Under ad libitum conditions, mice preferred social interaction. With increasing caloric need, group-housed mice shifted preference toward food, whereas single-housed mice engaged equally with both stimuli, suggesting conflict between competing drives.

Neuronal activity analyses revealed that PVN-OXT activation correlated with reward preference. Strong preference was associated with reduced activity in the bed nucleus of the stria terminalis and increased activity in the basolateral amygdala (BLA). Circuit tracing identified convergent input from the paraventricular thalamus (PVT) to these regions. Activity in PVT→BLA neurons predicted social preference, and chemogenetic inhibition of this pathway biased behavior toward food.

These findings identify a PVT→BLA circuit involved in prioritizing social versus metabolic needs.

Supported by GRK 2174 Neurobiology of Social and Emotional Dysfunctions, funded by the DFG.

10.45 **Coffee Break**

→ with presentation of the Young Investigator Award
sponsored by the *European Brain and Behaviour Society*

→ Group picture to be taken

11.15 – 13.00 SESSION 3
BRAIN NETWORKS UNDERLYING SOCIO-EMOTIONAL DYSFUNCTIONS
Chairs: *Alice Stephan, Rohit Menon* (Regensburg, GER)

11.15 **Lessons from Building a Molecular Stress Atlas**

Johannes Bohacek
ETH Zurich, Switzerland; johannes.bohacek@hest.ethz.ch

Our lab has catalogued the molecular changes that unfold in the mouse hippocampus in response to acute stress. These analyses reveal a highly stereotyped response that is intense but transient, and largely conserved across brain regions and stress modalities. This talk will outline how this conserved molecular stress response can be leveraged to uncover the functional drivers underlying these molecular changes, and how this perspective informs our understanding of brain function in health and disease. Moreover, this stereotyped response has provided insight into the process of stress habituation, revealing a broad suppression of gene expression following repeated stress exposures and pointing to distinct mechanisms that mediate stress habituation. Finally, the tightly regulated molecular stress response offers a framework for understanding individual differences in stress coping, with implications for stress resilience and susceptibility.

11.45 **Debugging social circuits in the Magel2 model of autism**

Freddy Jeanneteau
Universite de Montpellier, CNRS, INSERM, France;
freddy.jeanneteau@igf.cnrs.fr

Transition from social fear to social safety requires discriminating threat from ally, a mechanism failing in MAGEL2-deficient model of neurodevelopmental disorders. Magel2KO exhibit generalized social fear and persistent failure to extinguish aversive social memories.

The Lateral Septum (LS) is a regulatory hub for this transition. Within the LS, Somatostatin (SST) neurons act as neural "accelerator" for social retreat, firing upon social contact termination. In *Magel2*KO, these cells are hyperactive with exaggerated Ca^{2+} dynamics and elevated c-Fos expression. This hyperactivity is facilitated by a 30–50% depletion of septal oxytocin receptors and a breakdown of neuropeptide "brakes".

In healthy circuits, Oxytocin (OXT) and Vasopressin (AVP) silence LS SST cells via functional disinhibitory units that facilitate social approach while suppressing aggression. Mechanistically, Supraoptic Nucleus (SON) OXT neurons engaged for social safety, while Paraventricular Nucleus (PVN) OXT neurons engaged during fear conditioning. *Magel2*-deficiency hampers SON recruitment, locking mutant circuitry in social avoidance.

Adult OXT supplementation often fails due to receptor bottlenecks. Innovative "debugging" strategies-including optogenetic silencing of LS SST neurons or SSTR antagonism or postnatal OXT supplementation-restore social exploration and discrimination. This shifts the therapeutic framework toward targeted circuit modulation to restore social memory map.

12.15 **Decoding sex-specific vulnerability to adolescent social stress through homecage behavior and spatial proteomics**

Tobias Pohl, Hanna Hörnberg
Max Delbrück Center for Molecular Medicine, Germany; tobias.pohl@mdc-berlin.de

Adolescence represents a critical developmental window marked by extensive brain maturation, synaptic remodeling, and neural circuit refinement. Exposure to social instability and environmental stressors during this period can produce long-lasting effects on emotional and cognitive function. However, only a subset of individuals exposed to adolescent stress develops persistent psychopathology, suggesting that pre-existing biological differences may influence vulnerability or resilience to stress.

Here, we utilize the social instability stress model in adolescent male and female mice to investigate why some individuals develop stress-related behavioral dysregulation later in life, while others remain resilient. For this, we employ a multimodal approach combining classical behavioral phenotyping, continuous homecage monitoring, and spatially resolved single-cell proteomics. This integrative framework enables the identification of behavioral and molecular signatures associated with stress susceptibility and resilience.

We demonstrate that stress-susceptible animals exhibit distinct and persistent alterations in homecage behavior during both baseline and challenge conditions. These findings highlight continuous homecage monitoring as a sensitive tool for detecting early indicators of stress vulnerability and depressive-like phenotypes. Spatial proteomic analyses further reveal cell-type and region-specific molecular differences within the hippocampus that distinguish susceptible from resilient animals. Importantly, these molecular signatures may reflect both stress-induced adaptations and pre-existing biological features that influence individual stress responsiveness.

Together, these findings suggest that adolescent stress interacts with individual biological predispositions to shape behavioral and molecular outcomes. This work advances our understanding of the neurobiological mechanisms underlying stress vulnerability and highlights integrative behavioral and spatial omics approaches as powerful tools for studying psychiatric disease risk.

12.45 **Locus Coeruleus Degeneration Disrupts Neuromodulation and Promotes Aggression in Alzheimer's Disease – *Selected Talk***

Nicolas Landgraf[1,2], Weilin Chen[1], Giacomo Berg[1,5], Paul Feyen[1], Thomas Köglsperger[1,5], Jochen Herms[1,3,4], Lars Paeger[1,3]
[1]German Center for Neurodegenerative Diseases (DZNE), Munich, Germany; [2]Graduate School of Systemic Neuroscience (GSN), Munich, Germany; [3]Munich Cluster for Systems Neurology (SyNergy), Munich, Germany; [4]Center for Neuropathology and Prion Research, Munich, Germany; [5]Department of Neurology, University Hospital of Munich, Germany;
nicolasfriedrich.landgraf@dzne.de

Agitation and aggression are among the most prominent non-cognitive symptoms of dementia, collectively referred to as Behavioral and Psychological Symptoms of Dementia (BPSD). Despite their major clinical impact, the neuronal mechanisms underlying aggression in Alzheimer's Disease (AD) remain poorly understood.

The locus coeruleus (LC), the brain's primary source of noradrenaline, densely innervates brain regions involved in the regulation of social and aggressive behavior. Notably, the LC exhibits early tau pathology and progressive degeneration in AD. Clinical observations linking LC degeneration to neuropsychiatric symptoms suggest that noradrenaline dysfunction may contribute to aggression in AD.

To address this, we used transgenic AD mouse models expressing mutant amyloid precursor protein (APP) and tau, recapitulating key neuropathological features including Aβ plaque formation and tau deposition. Aggressive behavior was quantified using the Resident-Intruder paradigm combined with automated, machine learning-based behavioral classification. LC structural and functional integrity were assessed using immunohistochemistry and *in vivo* imaging approaches, including fiber photometry, two-photon microscopy and microendoscopy.

We found significantly increased aggression in AD mice compared to controls, accompanied by axonal degeneration of LC projections in brain regions implicated in aggression control. In a complementary mouse model exhibiting similar LC axon degeneration, we observed *in vivo* axonal dysfunction, reduced fidelity of hippocampal place cell coding and altered noradrenaline and dopamine dynamics, highlighting the widespread functional consequences of LC axon degeneration.

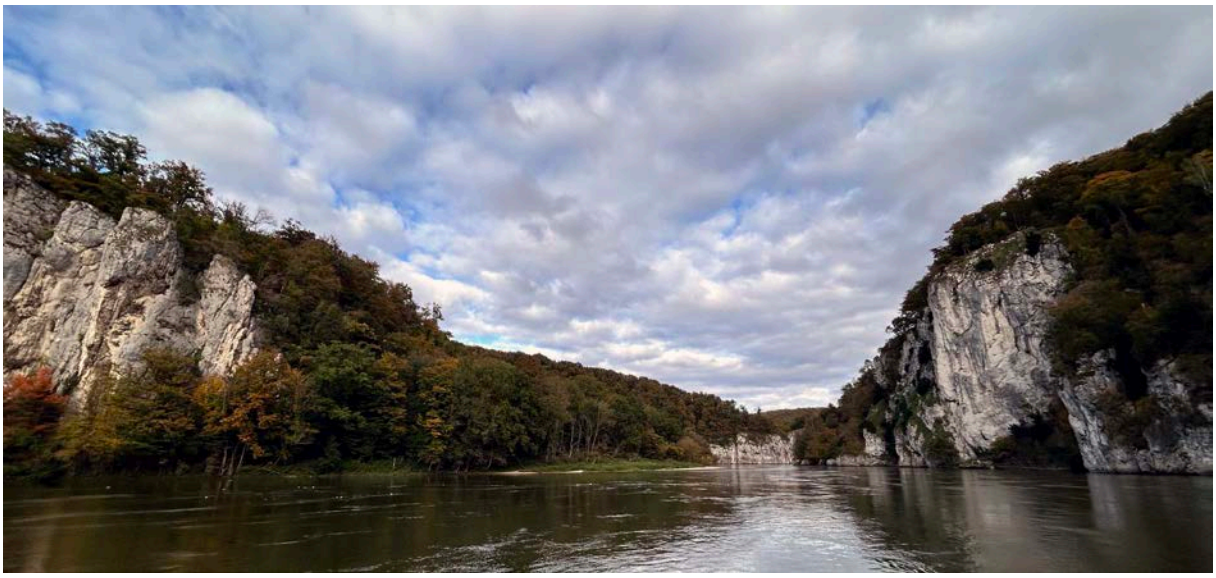
Together, our findings suggest that LC degeneration leads to dysregulated neuromodulatory signaling, which may contribute to the emergence of symptoms like aggression in AD.

13.00 Lunch

13.45 Bus & Boat through the *Danube Gorge* to *Weltenburg Monastery*

© Tourismusverband im Landkreis Kelheim e.V., Jürgen Würzinger

16.45 Dinner at *Weltenburg* beer garden



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18.30 Hike along the *Danube Gorge*

20.30 Bus to Regensburg

SUNDAY 10 TH MAY

8.30 - 9.30 **Registration**

9.00 – 10.30 **SESSION 4**

CIRCUITS AND NETWORK DYNAMICS UNDERLYING SOCIO-EMOTIONAL (DYS)FUNCTION (HUMAN)

Chairs: *Laura Stangl, Fernando Castillo Díaz* (Regensburg, Germany)

9.00 **The Computing Brain**

Gustavo Deco
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The perhaps most important unsolved problem in neuroscience is how the brain survives in a complex world by performing a rich repertoire of computation on a minimal energy budget. The brain is much better at adapting to the multiplicity of stimuli and outcomes than current generations of computers, artificial neural deep learning and reservoir model architectures. Yet, at first glance the brain appears to use a fixed anatomical architecture to perform the necessary huge variety of computations. But evolution's boldest trick is that in fact the brain's effective connectivity is constantly being updated through neuromodulation to allow the rich repertoire of computation. Inspired by this, we created a whole-brain model using empirical neurotransmitter maps modulating the underlying local regional dynamics. This NEMO (neurotransmission modulated) whole-brain model is able to flexibly compute the full task repertoire and associated functional connectivity of the neuroimaging data from 971 healthy participants. For each individual we defined a measure of 'brain computability' as the fitting of the NEMO whole-brain model to all tasks performed by the individual. Importantly, brain computability correlates with both behavioural performance on individual tasks and with a general behavioural measure of intelligence. Overall, our proposed unifying NEMO framework offers a natural way to sculpt different brain dynamics in a fixed brain architecture to compute the rich repertoire of tasks required for surviving and thriving.

9.30 **Compression of Representational Spaces as a Mechanistic Model for Socio-Emotional Dysfunction**

Jens Schwarzbach
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Humans organize their environment through relational structures linking objects, thoughts, and emotions, with perceived distances reflecting both shared organizational principles (e.g., valence, arousal, dominance) and

substantial individual variability. While distal sensory systems such as vision and audition exhibit relatively consistent organizational structures across individuals, emotional representations appear markedly more idiosyncratic. A similar individuality characterizes the chemical senses: olfactory and gustatory systems are shaped by primary sensory regions whose connectivity patterns differ fundamentally from those governing canonical sensory cortices and overlap closely with networks implicated in emotional processing.
Using representational similarity analysis (RSA), it is possible to characterize the behavioral and neural architecture of these individualized representational spaces and relate them to common underlying brain systems. Here, I propose a framework in which socio-emotional (dys)function is conceptualized as a change in representational granularity. Specifically, compression of representational spaces may reduce the ability to differentiate between stimuli or affective states, thereby impairing socio-emotional processing. By leveraging the inherently individualized structure of olfactory representations as a model system, this approach provides a tractable avenue to study emotional representations and offers a mechanistic account linking alterations in neural representational structure to dysfunction in socio-emotional behavior.

10.00 **Changes in Functional Connectivity Markers in Depressive Patients Undergoing ECT**

Lorenz Kick
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Different situations require different regions of our brains to be switched on or off. These specific configurations of brain regions that are activated or de-activated at certain time points can be called brain states. Especially important is the ability to switch between different brain states, allowing for a flexible and adaptive interaction with our surrounding.
Depression can be defined as the inability to flexibly switch between brain states, causing for example rumination and being stuck with the same thoughts over long period of times. This is coherent with cognitive theories of depression and explains deficits of patients with depression in every-day life.
In this project, functional brain networks and their interplay in depressive in-patients are investigated. Electroconvulsive Therapy (ECT) is one of the most effective methods to treat depressive disorders (60 – 80% response rates). Therefore, it can serve as a good model for understanding the neurobiological processes that underly the improvement of depressive disorders.
A preliminary dataset includes 20 patients undergoing ECT (4 female, mean age 45.05), with 16 ECT sessions in average. Patients underwent 5 MRI sessions (1 per week), consisting of 22 min resting state fMRI and T1-weighted structural imaging.

Different metrics derived from network analysis were used to longitudinally investigate static and dynamic functional connectivity of resting-state networks. Furthermore, depressive symptoms were anlayzed.
ECT treatment had a significant impact on depressive symptoms. Patients undergoing ECT also showed concurrent changes in network configuration over the course of treatment, potentially revealing underpinnings of antidepressive effects.

10.15 **A Flexible Biohybrid Synapse Array Integrating OECTs and MEAs for Advanced Neural Interfaces – *Selected Talk***

Shalini Chaudhuri[1,2,3]
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Neural interfaces are advanced electronic systems that enable direct communication with the nervous system, offering significant potential for neuroscience research and clinical therapy. Most of these systems rely on microelectrode arrays (MEAs), which provide high-resolution recording and stimulation of neural activity across in vitro, in vivo, and clinical settings. By detecting variations in local electrical potentials, these devices have improved our understanding of neuronal networks and enabled therapies such as deep brain stimulation for Parkinson's disease, cochlear implants for hearing restoration, and retinal implants for vision recovery. However, achieving seamless integration between neural interfaces and biological tissue remains a major challenge, requiring not only mechanical and material compatibility but also functional coherence so that artificial signals are interpreted as natural by the nervous system. To address this, neuromorphic approaches aim to emulate synaptic behavior through adaptive, feedback-responsive systems. Within the NeuroWin ERC project, this work presents a flexible biohybrid synapse array integrating organic electrochemical transistors (OECTs) with MEAs on a single chip. This platform enables simultaneous electrical and chemical interaction with neural tissue, including the detection of neurotransmitters such as glutamate and dopamine. The device is fabricated using flexible, biocompatible materials, including Parylene-C and PEDOT:PSS, allowing miniaturization for in vivo applications. Electrical, electrochemical, and surface characterizations were performed to evaluate device performance. This work contributes to the development of next-generation neuroelectronic systems for more natural and effective neural modulation.

10.30 **Coffee Break**

11.00 – 13.00 SESSION 5

NEUROBIOLOGY OF PROSOCIAL BEHAVIOURS

Chairs: *Inga Neumann, Alessandra Monaco* (Regensburg, Germany)

11.00 **Combining rodent and human studies to understand the neural mechanisms of emotional contagion and harm aversion**

Christian Keysers
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How does our brain make us feel what others feel? How does it motivate us to help others? Concentrating on emotions, I will show that in humans, the somatosensory, insular and cingulate cortices are activated both when experiencing pain and while witnessing others in pain. Through a series of human and rodent experiments I will ask whether such vicarious activations have causal influences on sharing the emotions of others, on deciding to help others and on avoiding to harm them. I will show the rodent cingulate cortex harbors pain mirror neurons responding to the animal's own pain and when witnessing the pain of others. Deactivating this region blocks both an animal's emotional response to the pain of others and their willingness to avoid actions that harm others. The homologies between humans and rodents suggest that emotion sharing is an evolutionarily conserved mechanism that allows animals and humans to better prepare for yet unseen dangers by tuning into the state of those that have already detected them. Finally, I will present work on psychopathic criminals and healthy volunteers that highlights that although these circuits may be biologically pre-wired, and shared with rodents, we have control on how much we use them. Empathy does not just happen to us: we can choose to empathize.

11.30 **Emotional cues in action: how rodents learn and adapt together**

Ewelina Knapska
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In social species, the emotions of others provide a rapid and efficient source of information about environmental opportunities and threats. Emotional contagion - the transfer of affective states between individuals - supports behavioral adaptation without direct experience. However, this process is not a simple reflex; rather, the brain selectively interprets socially transmitted signals to generate context-appropriate strategies.

In this talk, I will present evidence that rodents can acquire spatial knowledge through brief interactions with informed conspecifics - individuals that know the location of reward and are emotionally aroused due to recent reward consumption - demonstrating that social information extends beyond emotion to include cognitive representations of the environment. This socially acquired knowledge engages hippocampal circuits involved in planning and generates brain-wide activity patterns reflecting a partner's knowledge state. When memory-guided navigation is disrupted, animals increase their reliance on social cues, revealing how social experience dynamically shapes cognitive maps. I will further discuss how socially transmitted reward information propagates through social networks and how the prefrontal cortex regulates whether and how such information is used.

Together, these findings suggest that emotional contagion is not merely passive resonance, but a sophisticated neural mechanism that enables individuals to use others' experiences to guide adaptive decision-making. These results provide new insight into the neural basis of social cognition and its role in survival.

12.00 **Oxytocin receptor-coupled Gi signaling drives reversal of social trauma-induced avoidance in mice**

Rohit Menon[1], Theresa Schäfer[1], David Keller[2], Francisco de los Santos[2], Anna Bludau[1], Anna Krinner[1], Tatiana Korotkova[2], Inga Neumann[1]
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Successfully navigating social situations is an essential survival skill for mammals. Oxytocin (OXT) signaling in the lateral septum (LS) is a critical regulator of social behavior, but its underlying neural mechanisms remain unclear. In our study, we leverage the operant-conditioning-based social fear conditioning paradigm to examine the role of OXT signaling in the LS in regulating recovery from social trauma. First, we show that temporally precise activity of OXT receptor (OXTR) expressing neurons of the LS (LS^{OXTR}) neurons, which are heavily enriched in the caudal part of the LS (LSc), at the exact timepoint social contact predicts extinction success. LSc^{OXTR} neurons exhibit heightened inhibition at the exact timepoint of social interaction in mice with successful extinction (Responders: Res), while such modulation is absent in mice with unsuccessful extinction (Non-responders: Nres). We show that this pattern is regularly associated with enhanced social contact suggesting that inhibition of LSc^{OXTR} neurons is critical for the association of a positive emotional valence to social stimuli. In all mice, chemogenetic inhibition of LS^{OXTR} disrupts social fear extinction, whereas optogenetic stimulation has no effect, suggesting that the abovementioned temporal modulation is essential for successful extinction. Moreover, pharmacological activation of OXT signaling using synthetic OXT, TGOT (a complete OXTR agonist), or atosiban (a selective OXTR-Gi agonist) facilitates extinction of social fear. These findings indicate that precise temporal inhibition of LS^{OXTR} activity driven by OXTR-coupled Gi signaling is critical for adaptive social behavior, positioning OXT signaling in the LS as a key calibrator of social valence.

12.30 **Post-learning exposure to an enriched environment enhances the consolidation of a weak contextual threat memory in the retrosplenial cortex – *Selected Talk***

Bogomil Peshev[1], Svilen Georgiev[2], Desislava Krushovlieva[1], Pavel Rashev[1,3], Dimitrinka Atanasova[1,4], Jana Tchekalarova[1]
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The Behavioral Tagging (BT) hypothesis states that novelty and emotionality occurring before or after learning enhances the consolidation of memories for neutral or low-arousing events in memory-related areas like the dorsal hippocampus. This effect has been linked to increased dopaminergic activity and initiation of de novo synthesis of plasticity-related proteins. Considering the premises of the BT hypothesis, we decided to investigate whether novelty could also reinforce consolidation in another brain region active during the early phases of episodic-like memory formation – the retrosplenial cortex (RSC). To achieve our goal, we combined a weak version of the contextual threat conditioning paradigm (two 0.3-mA shocks) with subsequent brief exposure of the experimental animals (Wistar rats) to an environmental enrichment (EE) cage. In order to examine the role of dopamine and protein synthesis, after the completion of the behavioral protocols, rats were subjected to bilateral microinfusion of either vehicle, SCH-23390 (D1-like receptor antagonist), or anisomycin (translation inhibitor) in the anterior part of RSC. After 24 hours, the experimental animals were returned to the conditioning chamber to evaluate their context-driven freezing, a behavioral marker for long-term memory. We have shown that the experimental group exposed to the EE cage after conditioning and treated with vehicle had significantly higher freezing rates compared to rats that were returned to their home cage following vehicle infusion. This effect was also present when we compared this group with those treated with SCH-23390 and anisomycin. Thus, our results provide evidence for novelty-induced retrograde enhancement of memory consolidation in RSC.

12.45 **Lunch**

14.00 – 15.45 SESSION 6

SOCIO-EMOTIONAL DYSFUNCTIONS IN ADOLESCENCE: BRAIN STRUCTURAL AND FUNCTIONAL CHANGES

Chairs: *Irina Javers, Romuald Brunner* (Regensburg, Germany)

14.00 **Synchronizing Minds: Social Interaction and the Developing Adolescent Brain**

Kerstin Konrad
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Adolescence is a developmental period characterized by increased social-affective sensitivity, greater susceptibility to risky behaviors, and a heightened tendency toward suboptimal choices in emotionally charged contexts. This keynote will introduce the concept of interpersonal synchrony and examine how behavior, affect, physiology, and neural activity become aligned between individuals during social interaction. Particular emphasis will be placed on adolescence as a sensitive phase of social brain development, marked by heightened peer orientation, increasing sensitivity to social evaluation and exclusion, and ongoing maturation of neural systems supporting socio-emotional processing, reward, and self-regulation. The talk will present current evidence on interpersonal synchrony in peer and parent–adolescent contexts and discuss its relevance for social connectedness, emotion regulation, stress contagion, and resilience. It will also address how disrupted synchrony may contribute to socio-emotional dysfunction, including aggressive behavior, by impairing social learning, increasing interpersonal misattunement, and amplifying maladaptive patterns of responding in high-risk developmental contexts. In addition, we will critically examine whether interpersonal neural synchrony plays a causal role in shaping adolescent socio-emotional functioning and discuss candidate neurobiological mechanisms, including oscillatory brain activity, social reward networks, and neurotransmitter systems. Methodological challenges in capturing real-time, reciprocal interaction in developmental populations will also be considered. By linking developmental social neuroscience with translational perspectives, this keynote aims to show how studying synchronizing minds can deepen our understanding of adolescent mental health and open new avenues for prevention and intervention.

14.30 **The influence of psychosocial stress on functional connectivity and neuroendocrine markers in adolescents with depressive and comorbid anxiety disorders**

Ricarda Jacob[1], Irina Jarvers[1], Alexandra Otto[1], Stephanie Kandsperger[1], Angelika Ecker[1], Daniel Schleicher[1], Wilhelm M. Malloni[2], Inga D. Neumann[3], Romuald Brunner[1]
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Psychosocial stress plays a key role in the onset and maintenance of affective disorders, yet its neural and neuroendocrine mechanisms during adolescence remain poorly understood. This study examines the effects of psychosocial stress on neuroendocrine markers and fronto-limbic functional connectivity in adolescents with depressive disorders, with and without comorbid anxiety, compared to healthy controls.

Ninety-two adolescents aged 12–17 years (patients n = 58, healthy controls n = 34) were examined. Sessions included structured clinical interviews, psychometric assessments, and MRI scanning with resting-state functional connectivity (rsFC) before and after stress induction. Stress was induced via the Montreal Imaging Stress Task (MIST). Saliva samples were collected at eight time points to assess cortisol (CORT), oxytocin (OXT), and alpha-amylase (sAA). Social anxiety was included as a covariate in all analyses.

Endocrine analyses revealed significantly blunted CORT reactivity in patients compared to controls (time × group $p = .014$; AUCi $p = .006$), robust across covariate models. For OXT, no significant group differences in stress reactivity emerged, suggesting that the hypothesized attenuation is not supported in this sample. Exploratory analyses showed a decoupling of physiological and subjective stress responses in patients, as well as reduced CORT reactivity in patients with non-suicidal self-injury ($p = .015$).

MRI analyses are ongoing. We expect time × group interactions in fronto-limbic networks, with stress-induced rsFC changes correlating with endocrine reactivity, reflecting shared neuroendocrine mechanisms of stress adaptation.

14.45 **To play or not to play: How oxytocin and vasopressin regulate social play through reward neural circuitries**

Alexa Veenema
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Playing with peers in children and young animals is essential for the development of lifelong social skills. Autistic children are less engaged in social play and find these interactions less rewarding. This may impair social skill development in autistic individuals, which, in turn, may contribute to their lifelong social dysfunction. Autism is more prevalent in males than in females, suggesting sex differences in its etiology. Developing effective means to restore social play in autistic children is expected to improve their lifelong social functioning, but this requires understanding of the neural basis of social play and potential sex differences herein. In this talk I will discuss the neurobiological mechanisms driving social play behavior in male and female juvenile rats with emphasis on the roles of the evolutionarily conserved neuropeptides vasopressin and oxytocin. I will show how vasopressin and oxytocin regulate social play through distinct reward-related neural circuitries and in unique sex-specific ways. These findings may have implications for the sex-specific use of therapeutic drugs targeting the vasopressin and/or oxytocin systems and aimed at improving social play engagement in autistic children.

15.15 **Adolescent social trauma and oxytocin: Disentangling hypothalamic circuits of persistent social fear**

Melanie Kabas, Leopold Kinzel, Anna Bludau, Inga D. Neumann
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Adolescence is a sensitive developmental period during which adverse social experiences can lead to long-lasting socio-emotional dysfunctions, including social anxiety disorder. Using an adapted social fear conditioning (SFC) paradigm in mice, we investigated how adolescent social trauma shapes adult social behaviour and underlying neural mechanisms, with a particular focus on the oxytocin (OXT) system and the ventromedial hypothalamus (VMH).
Adolescent SFC induced robust social fear in both sexes; however, this phenotype persisted into adulthood selectively in males. Persistent social fear was accompanied by reduced oxytocin receptor (OXTR) binding in the VMH. Anatomical analyses revealed OXTergic fibres close to OXTR$^+$ cells in the ventrolateral VMH (VMHvl), with partial input from the paraventricular nucleus (PVN). However, PVN OXT neuron activation remained unchanged following adolescent SFC. RNAscope analyses further demonstrated that VMH cells, including the majority of OXTR$^+$ cells, are predominantly glutamatergic, suggesting that OXT modulates excitatory VMH output. Functionally,

intracerebroventricular OXT facilitated extinction of long-term social fear, whereas local VMH manipulations had limited effects. Intra-VMH OXT infusion modestly enhanced extinction, while chemogenetic activation of $OXTR^+$ neurons and viral OXTR knockdown did not alter social fear behaviour. Activity mapping using ZIF staining showed that social exposure following adolescent SFC increased VMHvl activity in males, irrespective of behavioural outcome (aggression vs. avoidance). In contrast, females exhibited increased VMHvl activity only during non-aversive social interactions, indicating sex-specific circuit engagement.
Together, these findings indicate a complex and potentially sex-dependent role of hypothalamic OXT signalling in adolescent social trauma, although its precise functional contributions require further investigation.

15.30 **Investigating the interaction between early-life stress and genetic vulnerability in the Shank3Δ11+/- mouse model – *Selected Talk***

Matteo Scaramagli[1,2], Valentina Ferretti[1,2], Cristina Bertollini[1,3], Alessia Stefanoni[4], Chiara Verpelli[4], Davide Ragozzino[1,3]
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Autism spectrum disorders (ASD) are neurodevelopmental conditions characterized by heterogeneous impairments in social communication and behavior. Their etiology arises from complex interactions between genetic susceptibility and environmental influences, however, the mechanisms through which these factors shape early brain development remain poorly understood. The postsynaptic density (PSD)-associated protein Shank3 plays a critical role in glutamatergic synapse formation and plasticity, and mutations in the *Shank3* gene have been linked to synaptic dysfunction and the emergence autistic-like phenotypes.
To investigate how early-life stress interacts with genetic vulnerability at physiological and behavioural levels, we took advantage of the transgenic $Shank3^{\Delta 11+/-}$ murine model and implemented a maternal separation paradigm consisting of two-hour daily separations from the dam and littermates for 10 consecutive days (PND 12-21).
We performed electrophysiological recordings in the medial prefrontal cortex (mPFC), a key region for the development and regulation of social and cognitive functions, consistently altered in ASDs. Recordings in pre-adolescent mice, shortly after the maternal separation protocol, revealed that excitatory synaptic transmission was selectively altered in $Shank3^{\Delta 11+/-}$ mice exposed to maternal

separation, with an increase in both miniature and spontaneous events amplitude.
Behavioral testing in young adult mice (PND60–90) revealed that Shank3$^{\Delta 11+/-}$ animals subjected to maternal separation exhibited deficits in both social odor preference and social novelty recognition, while social behavior in a free interaction test and spatial orientation were unaffected.
These findings indicate that Shank3-dependent genetic vulnerability interacts with early-life stress to selectively impair the processing of social odor cues, likely through altered maturation of prefrontal circuits.

15.45 **END OF SCIENTIFIC PROGRAMME**

16.00 **FAREWELL BBQ**

POSTERS

P1 Oxytocin receptor signaling in the medial septum: Circuit, function, and transcriptional regulation in social behavior

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Oxytocin (OXT) is a highly conserved neuropeptide that regulates a range of physiological and social processes via its receptor (OXTR), which is expressed across limbic and cortical brain regions. While OXT signaling has been extensively studied in several areas, the medial septum (MS) has only recently emerged as a potential site of modulation, and the properties of OXTR-expressing neurons in this region (MS^{OXTR}) remain poorly understood. Here, we performed a comprehensive molecular, cellular, and circuit-level characterization of MS^{OXTR} neurons. Biochemical analyses revealed that MS^{OXTR} neurons are predominantly GABAergic (~80%), although their precise inhibitory subtype remains unidentified. Using Cre-dependent anterograde tracing in OXTR-Cre mice, we identified projection targets of MS^{OXTR} neurons, including the prefrontal cortex and hippocampus, indicating their integration into broader limbic networks. To investigate the source of OXT input to the MS, we combined viral tracing with immunohistochemistry in wild-type mice. Although no direct projections from OXT-synthesizing neurons to the MS were detected, OXT^+ fibers were observed in proximity to the MS, suggesting indirect innervation of OXT-producing neurons. In parallel, multi-regional transcriptional analyses of OXT- and corticotropin-releasing factor (CRF)-related genes across sex and reproductive states revealed strong region-specific organization of these neuropeptide systems. The MS displayed intermediate transcriptional plasticity, characterized by changes in transcript abundance and receptor co-expression within individual neurons rather than alterations in cell number. Together, these findings provide a detailed characterization of MS^{OXTR} neurons and establish a foundation for understanding oxytocin signaling within septal circuits.

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P2 Sniffing out a Partner - PIEZO2 as a novel modulator of Olfaction-based Male Sexual Behaviour

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Sexual behaviour is one of the most significant driving forces influencing an individual's reproductive success. While sexual behaviour is well studied on an external ethological level, the underlying neuronal and molecular mechanisms remain opaque and highly debated. This inconclusive understanding is based on 1) the high neurobiological complexity underlying the behavioural phenomenon of reproductive drive and 2) the significant sexual dimorphism of the pathways and brain regions involved in its formation. However, a common starting point for studying sexual behaviour mostly focuses on the chemosensory detection of a possible sexual partner and of their inherent reproductive qualities.

In this current work, we present the mechanosensitive ion channel PIEZO2 as a novel molecular modulator of male sexual behaviour. Within the murine Main Olfactory Bulb, PIEZO2 expression is exclusively limited to the Mitral Cell layer. To establish a causal role for PIEZO2 in male sexual behaviour, we performed its AAV-mediated Knockdown within the Mitral Cell layer in the Main Olfactory Bulb of Male Mice. A 40% knockdown of PIEZO2 expression (as assessed by immunofluorescent staining) showed profound effects on various aspects of male sexual behaviour. PIEZO2-Knockdown impaired the preference for a receptive female partner, as measured in the Sexual Preference Test and the Bedding Test. Moreover, PIEZO2-Knockdown also impaired male sexual behaviour as evidenced by a reduced number of copulation attempts and an overall increase in the copulation frequency during the Copulation Test.

Based on these findings, we hypothesise that PIEZO2 might function as a key modulator of male sexual behaviour.

P3 Emotional Consequences of Pup Loss in Rat Mothers: Evidence for Dysregulation of OXT and CRF Systems

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The "pro-maternal" oxytocin system and the "anti-maternal" corticotropin-releasing factor (CRF) system are crucial components for the development and maintenance of the mother-infant bond, which is essential for their psychological and physical well-being. Disrupting this bond, e.g., through pup loss, is a stressful experience that affects maternal brain functions and behavior. Here, we studied rat mothers who had one day of maternal experience, after which all pups were removed, and mothers were left alone for 19 days (LD1+19). We analyzed the mRNA expression of CRF and its receptors, CRFR1 and CRFR2, in different brain areas relevant to maternal behavior. Notably, mRNA expression of CRFR2, but not of CRFR1, was elevated in the PVN and the VMH of LD1+19 mothers. In addition, these mothers showed increased passive stress coping in the forced swim test. To further examine the functional role of CRFR2 in maternal stress coping, we administered the CRFR2 antagonist Astressin-2B acutely in the VMH, which decreased passive stress-coping behavior in LD1+19 but not in non-separated mothers. Lastly, we investigated the potential role of the OXT system of the VMH in LD1+19 mothers. Our result showed that synthetic OXT administration in the VMH reinstated the passive stress-coping behaviour similar to the effect of CRFR2 antagonist administration. So far, our findings suggest that region-specific alterations in CRFR2 and OXT signaling underlie the emotional consequences of pup loss.

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P4 Stress and Individual Affective Space

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Stress and emotions are tightly intertwined: emotions can elicit stress, and stress can, in turn, modulate emotional experience. We investigated stress-related changes in the individual affective space (IAS) using an individualized assessment approach in 65 subjects. IAS was measured with a Multi-Arrangement Task (MAT) and the Self-Assessment Manikin (SAM) before and after acute stress induction with the Trier Social Stress Test (TSST). In addition, participants completed an Ambulatory Assessment (AA) to capture emotional variability in positive and negative affect during everyday life.

We examined three questions: (1) whether acute stress reduces IAS area; (2) whether IAS characteristics predict stress responses (cortisol, heart rate, and perceived stress); and (3) whether IAS area is associated with emotional variability in daily life. We did not find evidence for a shrinkage in IAS area or for associations between IAS area and emotional variability in AA. However, pre-stress SAM IAS area and pre-to-post changes in SAM IAS area predicted cortisol responses.

Not preregistered exploratory analyses using a three-dimensional IAS measure (volume) revealed a post-stress reduction in SAM IAS volume and showed that individual differences in SAM volume significantly predicted cortisol responses. Furthermore, MAT- and SAM-based IAS volume measures were associated with emotional variability in daily life.

Overall, stress-related shrinkage of the IAS emerged only when using volume-based measures, suggesting that two-dimensional representations of IAS have a reduced sensitivity to stress-related changes. SAM-based IAS characteristics were the most informative markers of stress-induced alterations in the mental representation of the affective space.

P5 From odors to emotions: Whole-brain representations of odor identity and valence using multivariate pattern analysis

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The human sense of smell is gaining increasing attention in affective neuroscience due to its direct access to the limbic system and evidence of impaired olfactory function in disorders characterized by emotional dysfunction. However, human neuroimaging studies in both olfactory and affective research have predominantly relied on mass-univariate analyses within pre-defined regions, thereby limiting insights into distributed neural representations. Our aim is to establish multivariate analysis approaches in systems relevant to affective processing, using olfaction as a model domain.

In an fMRI study, 30 participants were presented with ten odorants via an MRI-compatible olfactometer while blood-oxygen-level dependent signals were recorded. Whole-brain activation patterns were estimated for each odor. To identify regions encoding odor identity, we conducted a multivariate whole-brain searchlight analysis using 10-fold cross-validation and linear discriminant analysis. In addition, we implemented representational similarity analysis (RSA) to investigate whether neural patterns reflect concepts such as emotional valence associated with the odor stimuli.

Group-level analyses revealed above-chance odor classification not only in canonical olfactory regions such as piriform cortex, anterior olfactory nuclei, and amygdala, but also in extended areas including parietal cortex and precuneus. Furthermore, RSA results suggest that emotional valence is represented in neural patterns beyond traditionally defined olfactory regions.

These findings demonstrate that multivariate pattern-based approaches provide a powerful framework to study distributed representations in the human brain. Importantly, such methods hold strong potential to gain new insights into emotional processing and may enable the identification of neural pattern-based biomarkers for disorders characterized by olfactory and emotional dysfunction.

P6 Astrocytes phagocytic sexual dimorphism fosters Major Depressive Disorder through MEGF10 dysfunction

Diego Julian Vesga Jimenez, Eugenia Vivi, Celia Román, Anastasiia Nagumanova, Lea Seeholzer, Barbara Di Benedetto
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Major Depressive Disorder (MDD) is a prevalent global cause of disability, affecting approximately 5% of adults, and has a higher incidence in females. Despite its significant impact, its diverse symptoms and causes lack clear identification, hindering effective treatments. Understanding the underlying mechanisms is crucial. Studies in various brain areas indicate an astrocyte pathology in MDD, revealing reduced numbers and altered morphology, gene, and protein expression. Astrocytes influence synapse remodeling, which is crucial for neuronal network maturation. Notably, their phagocytic ability, facilitated by the Multiple EGF-like Domains 10 (MEGF10) signaling pathway, eliminates synapses. Abnormal synapse elimination in MDD may disrupt neuronal network balance fostering the onset of disease symptoms. Astrocytes can remodel synapses, especially during developmental stages, but also in adulthood, promoting their formation or elimination. We hypothesize that disrupted MEGF10 expression and/or subcellular localization in MDD leads to a non-functional protein, affecting different males and females, causing a synapse formation/elimination imbalance. Our study aims to assess whether MEGF10 alterations are linked to depressive-like behavior in animal models and if there are sex-dependent differences. Evaluation includes measuring MEGF10 expression, astrocytic synaptic engulfment capability, and MEGF10 subcellular localization in the different disease animal models and with both sexes. While there were no changes in expression, disease models exhibited reduced colocalization of MEGF10 with lipid rafts and decreased engulfing capacity. This suggests that MEGF10 could be a novel potential therapeutic target for developing strategies to address MDD.

P7 CRF and OXT Signalling in the Nucleus Accumbens Shape Maternal Behaviour in Lactating Rats

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Females undergo various adaptations to prepare for motherhood, resulting in a balanced interplay of various neurotransmitter systems, like corticotropin-releasing factor (CRF) and oxytocin (OXT). Their dysregulation can potentially lead to maternal neglect. The nucleus accumbens (NAc), a heterogenous brain region at the interface of the reward and maternal circuits, plays a key role in these processes. Here, we investigated the NAc CRF and OXT systems' involvement in maternal behaviour in early lactating rats. First, maternal aggression was monitored during the maternal defence test (MDT) against a virgin female intruder after bilateral, local injections of vehicle or CRF (1µg/0.5µL/side) in the NAc shell. In addition, maternal care was monitored before and after the MDT. Acute CRF infusion reduced the number of attacks and total nursing. In those brains, whole-brain c-Fos mapping revealed differential activation of various brain regions due to exposure to MDT/CRF. Microdialysis during the MDT revealed increased intra-NAc OXT release during maternal defence. Interestingly, retrodialysis of CRF, but not of UCN3, also resulted in OXT release within the NAc. Unilateral microinfusion of a retrograde transported virus in the NAc shell of virgin female rats revealed CRF+ projections descending from the basolateral amygdala, medial prefrontal cortex, and paraventricular thalamus. Using in-situ hybridisation, we further characterised the nature of CRF receptor-expressing neurons in the NAc, indicating differential expression of *Crhr1* and *Crhr2* throughout the NAc. This research highlights the critical role of NAc CRF and OXT in regulating maternal behaviour and provides novel insights into the NAc's neuronal underpinnings.
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P8 Synergistic olfactory nerve input and cholinergic neuromodulation can activate ERK in rat olfactory bulb vasopressin cells

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Social discrimination in rats relies on vasopressin cells (VPCs), neurons intrinsic to the olfactory bulb (OB). We previously observed that VPCs responded to electrical stimulation of the olfactory nerve in acute OB slices with inhibitory postsynaptic potentials (IPSPs), and that the neuromodulator acetylcholine (ACh) was able to switch these responses to excitation, often resulting in action potentials (AP). Moreover, in behaving rats exposed to conspecifics, more VPCs were immunopositive to the neural activity marker pERK (pERK$^+$ VPC) than in control rats. Is this increased ERK activation indeed related to cholinergic modulation?

Here we investigated ERK activation in acute OB slices from transgenic VP-eGFP rats upon various treatments. Both KCl and NMDA stimulation resulted in substantial pERK induction across bulbar neurons including mitral cells and caused VPC spiking, but neither increased pERK$^+$ VPC percentage. Conversely, TTX treatment further reduced VPC ERK activation compared to control, indicating spontaneous activity of VPCs, which we also observed in a subset of VPCs in electrophysiological recordings. Tetanic olfactory nerve stimulation yielded regionalized, column-like ERK activation across bulbar layers. While ACh alone had no influence on pERK induction in VPCs, the presence of ACh during tetanic stimulation substantially increased percentages of pERK$^+$ VPCs specifically within activated regions, implying that coincident cholinergic neuromodulation and afferent synaptic inputs cooperate to induce pERK in VPCs.

While depolarization alone is insufficient to trigger the pERK induction cascade in VPCs, our results thus validate pERK induction as a tool to monitor synaptic, suprathreshold VPC excitation in the OB.

P9 Bilaterian Innovation of MYRIP/MLPH Refines Secretory Actomyosin Transport in the RAB–FYVE_2 Network

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RAB GTPases are key regulators of cellular organelle transport. In response to the increased demand for signaling molecule transport to facilitate cell communication in multicellular organisms, the transition from single-celled relatives to multicellular animals is marked by a significant expansion of the secretory RAB8 small GTPase gene. The resulting RAB8, RAB3, and RAB27 proteins interact with effectors that share a common structural feature: the FYVE_2 zinc finger domain. Mammals possess four FYVE_2 domain protein families: RIM1/2, rabphilin-3A/NOC2, SYTL3/4/5, and MYRIP/MLPH, all of which are involved in exocytic and secretory transport processes. Through TBLASTN nucleotide database searches, we demonstrate that these FYVE_2 domain effector proteins co-evolved with the expansion of secretory RAB GTPases. Notably, no FYVE_2 domain proteins have been identified in unicellular holozoa.Three of the four mammalian FYVE_2 protein families—RIM, rabphilin-3A, and SYTL—are already present in early-branching animals, such as sponges and sea anemones. The MYRIP/MLPH family, however, emerged later in the evolution of bilaterians. Phylogenetic analysis, structure predictions, and interaction studies suggest that MYRIP evolved through a domain-shuffling event, which combined the basal metazoan SYTL-RAB binding domain with a myosin-7 actin motor protein binding domain. The evolution of FYVE_2 proteins in metazoans, particularly the bilaterian refinement of MYRIP/MLPH-mediated secretory actomyosin organelle transport, represents a crucial adaptation to meet the increased demands for cell communication in animals.

P10 Unrecognized Coding Potential in Social Fear Circuits: Identifying Microprotein Candidates

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Social fear is a defining feature of social anxiety disorder, a prevalent psychiatric condition that severely impairs social functioning. Despite its clinical relevance, the neurobiological mechanisms underlying social fear learning and extinction remain incompletely understood. While protein-coding genes have been extensively studied in fear-related circuits, the potential contribution of microproteins encoded by previously annotated non-coding RNAs has largely been overlooked. Here, we investigate whether such microproteins may be involved in the acquisition or extinction of social fear.

Using social fear conditioning in mice, we combined Translating Ribosome Affinity Purification (TRAP) with RNA sequencing to profile actively translated transcripts in neurons of the paraventricular nucleus of the hypothalamus (PVN) and the septum. Across comparisons of conditioned and unconditioned animals following acquisition or extinction, we identified approximately 150 transcripts showing robust differential expression, defined by a minimum twofold change. Notably, many of these transcripts are currently annotated as non-coding RNAs. Their association with ribosomes suggests that they may contain previously unrecognized open reading frames and represent microprotein-encoding candidates.

To explore this possibility further, we are establishing complementary mass spectrometry–based approaches and *in vitro* assays to assess translation and cellular function of selected candidate sequences. Together, these preliminary findings point to a previously underexplored molecular layer in social fear–related circuits and provide a foundation for future studies on the role of microproteins in socio-emotional dysfunctions.

P11 IPACL, an extended amygdala hub for social valence: anatomical connectivity and stress-induced alterations in mice

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Social interactions are fundamental to animal behavior, yet the complex neural mechanisms that encode their valence remain poorly understood. The interstitial nucleus of the posterior limb of the anterior commissure, lateral part (IPACL), region of extended amygdala, has recently emerged as a hub for integrating social and emotional information; however, its specific role in shaping social valence remains unexplored.
To address this gap, we utilized the vGATE system, an activity-dependent neuronal labelling approach to map IPACL connectivity in mice during distinct social encounters. Social behavior was evaluated using the conditioned place preference (CPP) paradigm and a free social interaction test quantified via automated pose-estimation (DeepLabCut).
Baseline analyses established a controlled framework, revealing no significant behavioral differences between encounter types. Following 21 days of Chronic Social Defeat Stress (CSDS), mice exhibited a generalized reduction in social interaction regardless of the conspecific's strain. Notably, CPP analyses demonstrated that stressed mice shifted their place preference only when conditioned with a non-aggressive CD1 mouse. This indicates that CSDS selectively modulates the motivational response to specific social targets.
Further, vGATE-based mapping revealed a stress-induced reorganization of IPACL circuits, characterized by differential changes in neuronal projections to target regions. Because whole-brain c-Fos quantification showed no significant differences across conditions, these stress-induced alterations operate through changes in synaptic strength or excitability rather than large-scale shifts in neuronal activation. Building upon established IPACL connectivity, these findings provide a characterization of its stress-induced reorganization, revealing how chronic stress selectively alters the neural processing of social valence at circuit level.

P12 Feast or Friendship: How the Brain Prioritizes Competing Needs

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Animals continuously balance competing physiological needs by prioritizing behaviors essential for survival. Despite the critical importance of need prioritization, the neuronal and molecular mechanisms remain poorly understood. Since oxytocin (OXT) modulates both social and feeding behaviors, we hypothesized that hypothalamic OXT neurons play a key role in governing preference between social interaction and food needs under competing demands. To test this, we used a recently established Social versus Food Preference test in adult CD1 male mice, manipulating social and metabolic states through housing conditions and food deprivation. Regardless of housing, all mice initially preferred social stimuli over food stimuli. However, as caloric need increased, group-housed mice shifted their preference toward food, whereas single-housed mice maintained equal preference for both stimuli, reflecting behavioral indecision when both needs remained unmet. Immunofluorescence revealed that oxytocin neuronal activity in the paraventricular nucleus (PVN) correlated with stimulus preference, with reduced PVN-OXT activity in the food-preferring group and increased activity in the social-preferring group. Additionally, animals showing strong preference exhibited lower neuronal activity in the bed nucleus of the stria terminalis (BNST) and higher activity in the basolateral amygdala (BLA). Circuit-tracing identified that both BLA and BNST regions receive input from a common upstream region, the paraventricular thalamus (PVT). Notably, animals with strong social preference showed heightened activity in PVT→BLA projecting neurons and chemogenetic inhibition of this pathway biased animals towards food preference. Together, these findings suggest that PVN-OXT activity and a PVT→BLA circuit jointly modulate behavioral prioritization between competing social and metabolic needs.